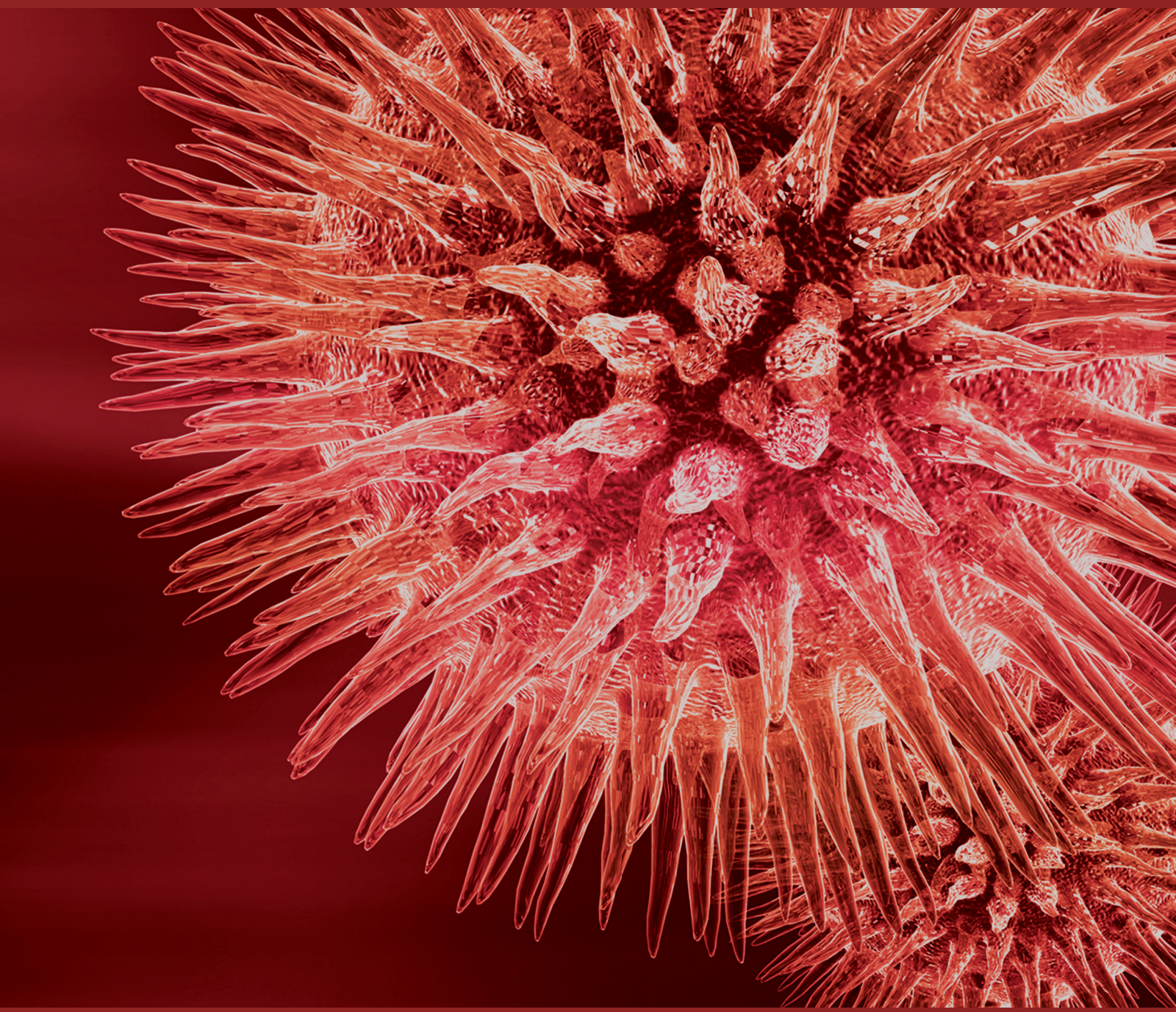
BioMed Research International

Achilles Tendinopathy: From the Basic Science to the Clinic

Guest Editors: Ying-Hui Hua, Wataru Miyamoto, Hong-Yun Li, Youichi Yasui, and Seung H. Han



Achilles Tendinopathy: From the Basic Science to the Clinic

BioMed Research International

Achilles Tendinopathy: From the Basic Science to the Clinic

Guest Editors: Ying-Hui Hua, Wataru Miyamoto, Hong-Yun Li, Youichi Yasui, and Seung H. Han

Contents

Hindawi
BioMed Research International
Volume 2017, Article ID 9534125, 2 pages
https://doi.org/10.1155/2017/9534125

Editorial

Achilles Tendinopathy: From the Basic Science to the Clinic

Hong-Yun Li,[1] Youichi Yasui,[2] Seung Hwan Han,[3] Wataru Miyamoto,[4] and Ying-Hui Hua[1]

[1]*Sports Medicine Center, Fudan University, Department of Sports Medicine and Arthroscopy Surgery, Huashan Hospital, Shanghai, China*
[2]*Hospital for Special Surgery, New York, NY, USA*
[3]*Department of Orthopaedic Surgery, Yonsei University College of Medicine, Seoul, Republic of Korea*
[4]*Department of Orthopaedic Surgery, Teikyo University School of Medicine, Tokyo, Japan*

Correspondence should be addressed to Ying-Hui Hua; hua023@hotmail.com

Received 20 February 2017; Accepted 21 February 2017; Published 30 April 2017

Chronic Achilles tendinopathy is frequent in the individuals who participate in the physical activities, especially running and jumping. This disorder could be subdivided into insertional and noninsertional tendinopathy according to the zone of the pathological disorder.

The essence of tendinopathy is a failed healing response, which includes three different and continuous stages (reactive tendinopathy, tendon disrepair, and degenerative tendinopathy). These tendinopathic lesions affect both collagen matrix and tenocytes. In a recent study, the authors found that the Achilles tendinopathy is harder than asymptomatic tendons on axial-strain sonoelastography, which indicated the decrease of resisting plastic deformation [1]. The histological studies demonstrate disorganization and fragmentation of the collagen, a decrease in collagen fiber diameter and in the overall density of collagen, an increased number of tenocytes and concentration of glycosaminoglycans in the ground substance, and neovascularization.

Metalloproteases (MMPs) are important regulators of extracellular matrix remodeling, and their levels are altered during tendinopathy. Previous studies indicated that MMP-9 and MMP-13 participate in collagen degradation process, whereas MMP-2, MMP-3, and MMP-14 participate in both collagen degradation and collagen remodeling processes. Moreover, difference kinds of growth factors and cytokines such as vascular endothelial growth factor, insulin-like growth factor, and transforming growth factor $\beta 2$ detected increased expression in the Achilles tendon, which could induce neovascularization and stimulate fibroblast and tenocytes proliferation and synthesis of collagen [2].

The reasons of pain in Achilles tendinopathy are complicated. The changes in matrix, vascular, tenocyte, cytokines, neuropeptides and neurotransmitters, and ion channels in the cells are the potential contributors to pain [3]. Moreover, pain could be evoked via nonnociceptive mechanisms through a load detection system, which could be disrupted via local or central dysfunction [3].

Various treatment options have been used for the disorder, which included conservative and surgical treatments.

Conservative treatment was the first line of treatment for Achilles tendinopathy, such as activity modification, orthotics, heel lifts, massage, hot and cold compresses, strengthening exercises, ultrasound, and nonsteroidal anti-inflammatory drugs (NSAID) or corticosteroid injection. Corticosteroid injections may have some benefit in the short term, but adverse effects were reported very high [4]. Therefore, precaution is always advised before the injection of corticosteroid in or around the Achilles tendon. In recent years, the eccentric training program, Extracorporeal Shock Wave Therapy, Deep friction massage, Sclerosing Agents and Aprotinin injection, Electrocoagulation, Prolotherapy (Intratendinous hyperosmolar dextrose), Ultrasound and Low-Level Laser Therapy, Topical Glyceryl Trinitrate, and platelet rich plasma (PRP) injections are introduced to treat Achilles tendinopathy. However, Level I, controlled, randomized studies are still needed to support the effect of these treatments. Among these conservative treatments, the most promising approach is the use of PRP to improve the healing potential of Achilles tendinopathy. However, conflicting results have been reported for PRP injection. The available data do not

support the use of PRP as a first-line treatment for chronic tendinopathy. The pharmacological rationale for using PRP in tendinopathy remains unclear. The effect of PRP injection is influenced by many factors, which included the variety of growth factors and cytokines released by the platelets, the time choice for injection, the ability of the factors passing through target tissues, the platelet concentration, and the time of centrifugation. Therefore, a standard should be established for this treatment in future research.

If conservative treatments have no effect, surgical treatments should be considered. The goals of operation are removing degenerative tissue, stimulating tendon healing, and/or augmenting the Achilles tendon with auto- or allografts. Operative treatments seem to be a good option for Achilles tendinopathy patients, when conservative treatment fails. As the lower complication rate, quick recovery, and early return to high-level sports are after surgery, minimally invasive surgery is the primary operative treatment option [5, 6], such as ultrasound + Doppler-guided arthroscopic shaving or minisurgical scraping in degenerative regions with high blood flow and nerves distributions [7]. In the future, long-term follow-ups of more patients and evaluation of tendon thickness and structure integrity are needed.

Hong-Yun Li
Youichi Yasui
Seung Hwan Han
Wataru Miyamoto
Ying-Hui Hua

References

[1] Q. Zhang, Y. Cai, Y. Hua, J. Shi, Y. Wang, and Y. Wang, "Sonoelastography shows that Achilles tendons with insertional tendinopathy are harder than asymptomatic tendons," *Knee Surgery, Sports Traumatology, Arthroscopy*, pp. 1–10, 2016.

[2] M. S. Zafar, A. Mahmood, and N. Maffulli, "Basic science and clinical aspects of Achilles tendinopathy," *Sports Medicine and Arthroscopy Review*, vol. 17, no. 3, pp. 190–197, 2009.

[3] E. Rio, L. Moseley, C. Purdam et al., "The pain of tendinopathy: physiological or pathophysiological?" *Sports Medicine*, vol. 44, no. 1, pp. 9–23, 2014.

[4] L. Hart, "Corticosteroid and other injections in the management of tendinopathies: a review," *Clinical Journal of Sport Medicine*, vol. 21, no. 6, pp. 540–541, 2011.

[5] H. Lohrer, S. David, and T. Nauck, "Surgical treatment for achilles tendinopathy—a systematic review," *BMC Musculoskeletal Disorders*, vol. 17, no. 1, article no. 207, 2016.

[6] Y. Bohu, N. Lefèvre, T. Bauer et al., "Surgical treatment of Achilles tendinopathies in athletes. Multicenter retrospective series of open surgery and endoscopic techniques," *Orthopaedics and Traumatology: Surgery and Research*, vol. 95, no. 8, supplement 1, pp. S72–S77, 2009.

[7] H. Alfredson, "Low recurrence rate after mini surgery outside the tendon combined with short rehabilitation in patients with midportion Achilles tendinopathy," *Open Access Journal of Sports Medicine*, vol. 7, pp. 51–54, 2011.

Hindawi
BioMed Research International
Volume 2017, Article ID 7021862, 4 pages
https://doi.org/10.1155/2017/7021862

Research Article

The Risk of Achilles Tendon Rupture in the Patients with Achilles Tendinopathy: Healthcare Database Analysis in the United States

Youichi Yasui,[1,2] Ichiro Tonogai,[2,3] Andrew J. Rosenbaum,[2,4] Yoshiharu Shimozono,[2] Hirotaka Kawano,[1] and John G. Kennedy[2]

[1]*Department of Orthopedic Surgery, Teikyo University School of Medicine, Tokyo, Japan*
[2]*Hospital for Special Surgery, New York, NY, USA*
[3]*Department of Orthopedic Surgery, Tokushima University, Tokushima, Japan*
[4]*Albany Medical Center, Albany, NY, USA*

Correspondence should be addressed to John G. Kennedy; kennedyj@hss.edu

Received 13 October 2016; Revised 18 January 2017; Accepted 9 February 2017; Published 30 April 2017

Academic Editor: Haining Zhang

Introduction. Disorders of the Achilles tendon can be broadly classified into acute and chronic entities. Few studies have established chronic Achilles tendinopathy as a precursor to acute Achilles ruptures. In this study, we assessed the relationship between Achilles tendinopathy and rupture, clarifying the incidence of rupture in the setting of underlying tendinopathy. *Methods*. The United Healthcare Orthopedic Dataset from the PearlDiver Patient Record Database was used to identify patients with ICD-9 codes for Achilles rupture and/or Achilles tendinopathy. The number of patients with acute rupture, chronic tendinopathy, and rupture following a prior diagnosis of tendinopathy was assessed. *Results*. Four percent of patients with an underlying diagnosis of Achilles tendinopathy went on to sustain a rupture (7,232 patients). Older patients with tendinopathy were most vulnerable to subsequent rupture. *Conclusions*. The current study demonstrates that 4.0% of patients who were previously diagnosed with Achilles tendinopathy sustained an Achilles tendon rupture. Additionally, older patients with Achilles tendinopathy were most vulnerable. These findings are important as they can help clinicians more objectively council patients with Achilles tendinopathy.

1. Introduction

Achilles tendon disorders are commonly encountered in both the athletic and general populations [1]. Disorders can be divided into two general categories: acute and more chronic overuse injuries. Acute rupture of the Achilles tendon most frequently occurs in males between 30 and 40 years of age [2]. Although it is considered an acute process, histological analyses have demonstrated that, even in the setting of acute rupture, degenerative changes are regularly found within the tendon [3–8]. Achilles tendinopathy is a more indolent and chronic process and is attributed to repetitive overuse. It is most prevalent in individuals aged 20–60 years [9]. In the setting of Achilles tendinosis, histological analysis reveals degenerative changes within the tendon [10, 11].

The histological similarities between acute Achilles tendon ruptures and chronic tendinopathy suggest that some individuals may sustain a presumed acute rupture in the setting of the more chronic tendinopathy. However, little is known about this, with studies presenting conflicting findings. While some works suggest that rupture following tendinopathy occurs in 5% to 44% of individuals [12, 13], others have shown that most patients with tendinopathy have favorable functional outcomes without tendon rupture [14–16].

Through the use of a deidentified patient database, this study clarifies the risk of Achilles tendon rupture in patients with a formal diagnosis of Achilles tendinopathy.

2. Methods

2.1. Data Source. Data was obtained from the United Healthcare Orthopedic (UHC) dataset from the PearlDiver Patient Record Database (PearlDiver Technologies, Inc., Fort Wayne,

IN, USA). This database is comprised of deidentified patients in a Health Insurance Portability and Accountability Act (HIPAA) compliant fashion [17, 18].

The UHC database consists of reported data from hospitals and/or physicians between 2007 and 2011 and has information on 20,484,172 patients. Approximately 9% of the United States (US) population younger than 65 years of age and approximately 13% of the US population with private medical insurance are represented in the database [19]. Additionally, the PearlDiver Patient Record Database includes all patients who enrolled with the insurance carrier during the desired time period before or after a specified event [17].

In the database, International Classification of Disease, Nine Codes (ICD-9), or current procedural terminology (CPT) codes are used to search for subsets of patients. Demographic information, such as age and gender, can then be assessed for these patients.

2.2. Cohort Selection. Three subsets of patients, all between 20 and 69 years old, were evaluated in this study: Group 1: patients with an acute Achilles tendon rupture; Group 2: patients with Achilles tendinopathy; and Group 3: patients with an Achilles tendon rupture following a diagnosis of Achilles tendinopathy.

The ICD-9 codes used in this study were 72767 (Achilles tendon rupture) and 72671 (Achilles tendinopathy). The number of patients was determined for each of the three cohorts. The incidence of each condition per 100,000 patients was calculated by dividing the number of patients with each disorder by the total number of patients aged 20–69 years in the UHC database (20,086,126 patients). The relationship between the disorders and demographic factors was assessed. The incidence of Achilles tendon rupture in the setting of Achilles tendinopathy was calculated by dividing the total number of patients with an Achilles tendon rupture following a diagnosis of Achilles tendinopathy (Group 3) by the total number of patients with Achilles tendinopathy (Group 2). Additionally, in each age group, the number of patients with an Achilles tendon rupture following a diagnosis of Achilles tendinopathy (Group 3) was divided the number of patients with Achilles tendinopathy (Group 2).

2.3. Statistical Analysis. Statistical analysis was performed using SAS 9.3 (SAS Institute, Cary, NC). Analysis of variance and Tukey's test were used to assess the incidence in each age and the chi-square test was applied for gender analysis. A p value of less than 0.05 was considered a statistically significant outcome.

3. Results

3.1. Group 1: Patients with an Acute Achilles Tendon Rupture. A total of 21,305 patients (106 per 100,000) were included. Individuals aged 30–39 years were most often affected, followed by those aged 40–49 years. The incidence of Achilles tendon rupture in these age groups was significantly higher than that observed in individuals aged 20–29 years and 60–69 years ($p < 0.05$) (Figure 1). Males sustained ruptures more frequently than females (male versus female; 141 per 100,000 versus 67 per 100,000, resp.; $p < 0.05$).

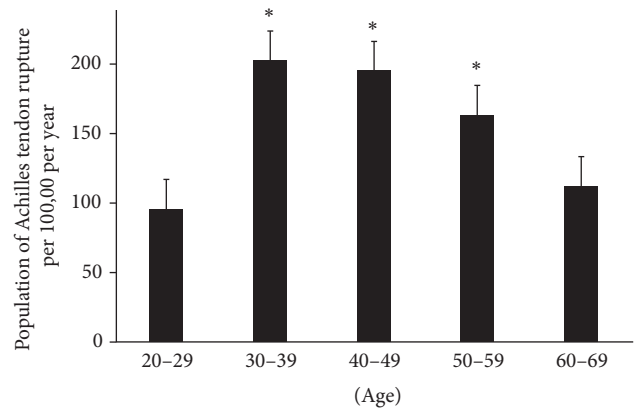


Figure 1: Group 1: population distribution in each age according to incidence of Achilles tendon rupture. *Most affected age groups.

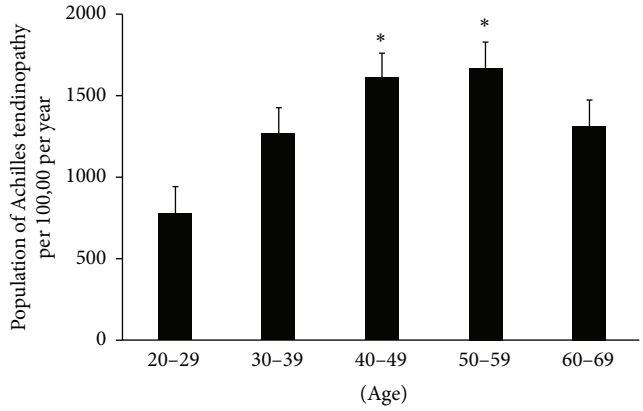


Figure 2: Group 2: population distribution in each age according to incidence of Achilles tendinopathy. *Most affected age groups.

3.2. Group 2: Patients with Achilles Tendinopathy. A total of 180,421 patients (898 per 100,000) were diagnosed with Achilles tendinopathy. Individuals aged 50–59 years were most often affected, followed by those aged 40–49 years. The incidence of Achilles tendinopathy in those groups was significantly higher than that seen in those aged 20–39 years and 60–69 years ($p < 0.05$) (Figure 2). There were no statistical differences in the incidence of Achilles tendinopathy between males and females (male versus female; 892 per 100,000 versus 840 per 100,000, resp.; *n.s.*).

3.3. Group 3: Patients with an Achilles Tendon Rupture following a Diagnosis of Achilles Tendinopathy. There were 7,232 patients (36 per 100,000) who sustained an Achilles tendon rupture following a diagnosis and treatment for Achilles tendinopathy (Figure 3). Those aged 50–59 years were most often affected, followed by those aged 40–49 years (4.3% and 3.9%, resp.). The incidence in these groups was significantly higher than that observed in those aged 20–39 and 60–69 years ($p < 0.05$) (Figure 3). There was no significant

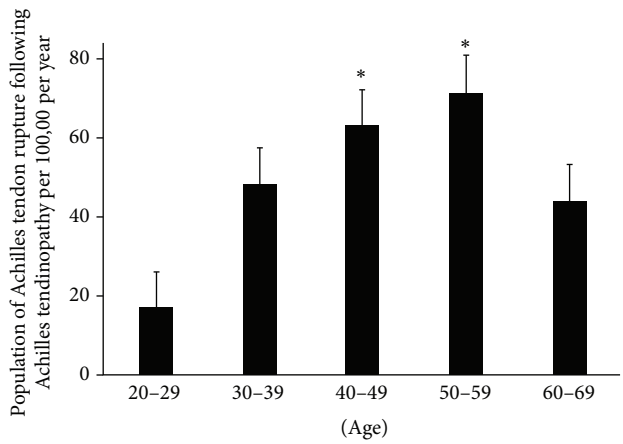


Figure 3: Group 3: population distribution in each age according to incidence of Achilles tendon rupture following Achilles tendinopathy. *Most affected age groups.

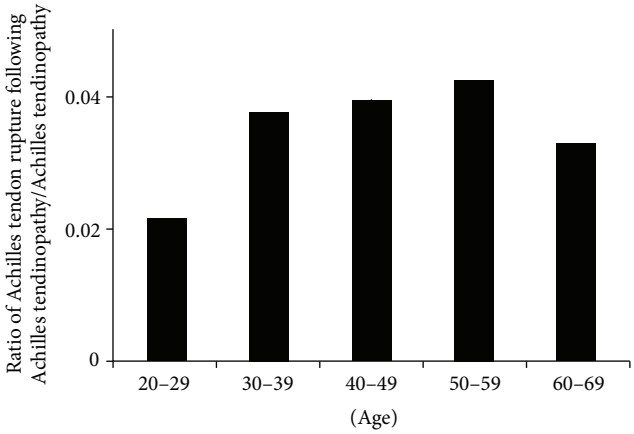


Figure 4: Distribution in each age according to ratio of incidence of Achilles tendon rupture following Achilles tendinopathy divided by incidence of Achilles tendinopathy.

difference in the incidence of Achilles tendinopathy between males and females (male versus female; 41 per 100,000 versus 29 per 100,000, resp.).

3.4. The Relationship between Achilles Tendon Rupture and Achilles Tendinopathy. Approximately 4.0% of patients with Achilles tendinopathy subsequently sustained a rupture (Figure 4). Individuals aged 50–59 years were most susceptible (4.3% incidence).

Admittedly, the time point between the diagnosis of tendinopathy and subsequent rupture was not ascertainable from current database.

4. Discussion

The present study analyzed a large, diverse population of individuals aged 20–69 years in order to determine the rate of Achilles tendon rupture in the setting of underlying Achilles tendinopathy. We found that approximately 4.0% of patients who were previously diagnosed with Achilles tendinopathy ultimately sustained a rupture. Additionally, older patients with tendinopathy were most vulnerable.

Intrasubstance degeneration of the Achilles tendon has been found in individuals with both acute rupture and chronic tendinopathy [3–8, 10, 11], suggesting that tendinopathy precedes and may even predispose individuals to Achilles tendon rupture [20]. However, studies have reported inconsistent outcomes. Maffulli found that 5% (9/176) of patients who sustained an Achilles tendon rupture had previous symptoms over their Achilles tendon [12]. In a work by Nestorson et al. [13], 44% of patients (11/25) had Achilles tendon pain before Achilles tendon rupture. These studies must be interpreted with caution as they are comprised of small cohorts with uncontrolled variables.

We found that 4.0% of patients previously diagnosed with Achilles tendinopathy suffered an Achilles tendon rupture. This is an important finding and elucidates the intimate, but complex, relationship between Achilles tendinopathy and rupture. The small percentage of patients who went on to rupture following a diagnosis of tendinopathy (4.0%) underscores the success of the various treatment modalities specific to Achilles tendinopathy (e.g., eccentric stretching).

Outcomes from the current study suggest that age may be a risk factor for Achilles tendon rupture previously diagnosed with Achilles tendinopathy. Older patients with Achilles tendinopathy had a significantly higher risk of rupture than younger individuals. This finding is supported by a recent animal study that demonstrates the relationship between advancing age and degeneration of the Achilles tendon [21].

In this study, males had a higher risk of rupture. This too is consistent with other works, such as that by Wong et al. [22]. In that study, males were found to be 4–7 times more likely to rupture their Achilles tendon [22]. Although the reasons for this finding are currently unclear, this finding should be explored in future studies.

5. Limitations

As a database study, this work has inherent limitations. Pertinent patient information, such as injury mechanism, symptom severity, duration of symptoms, medical comorbidities, the degree of tendon degeneration, use of local and systemic corticosteroids, and fluoroquinolone usage, was unavailable. Although we found that 4.0% of patients with tendinopathy go on to rupture, our data can be presented differently, as it also suggests that 33.4% of patients who sustained a rupture were previously diagnosed with Achilles tendinopathy (Group 3/Group 1). In other words, 66.6% of patients who sustained an Achilles tendon rupture were not previously diagnosed with Achilles tendinopathy. While this can be interpreted as suggesting that Achilles tendon rupture is more common in the absence of tendinopathy, we do not believe that this is accurate. It is likely that some rupture patients may have had undiagnosed tendinopathy. Furthermore, we fail to account for the mechanism of injury in our analysis. In other words, patients rupturing in the absence of a tendinosis diagnosis may have been more likely to do so because of their given activities [17]. As previously mentioned, the database

does not provide this information. The time between each diagnosis was also unknown. Another potential source of error is the possibility of any documentation or coding mistakes. We also do not know how tendinopathy patients were treated. This is important, as certain modalities (e.g., steroid injection) may have predisposed patients to rupture. Despite these limitations, we believe that the results from our large cohort of patients provide valuable insight into the relationship between Achilles tendinopathy and rupture.

6. Conclusions

In this large cohort database study, we found that approximately 4.0% of patients who were previously diagnosed with Achilles tendinopathy sustained an Achilles tendon rupture. Additionally, older patients with Achilles tendinopathy were most susceptible to rupture. These findings are important as they can help clinicians more objectively council patients following a diagnosis of Achilles tendinopathy.

Disclosure

An earlier version of this work was presented as a poster at the 6th AFFAS Triennial Scientific Meeting, 2016.

Conflicts of Interest

John G. Kennedy is a consultant for Arteriocyte, Inc.; has received research support from the Ohnell Family Foundation, Mr. and Mrs. Michael J. Levitt, and Arteriocyte Inc.; and is a board member for the European Society of Sports Traumatology, Knee Surgery, and Arthroscopy, International Society for Cartilage Repair of the Ankle, American Orthopedic Foot and Ankle Society Awards and Scholarships Committee, and International Cartilage Repair Society finance board.

References

[1] N. Levi, "The incidence of Achilles tendon rupture in Copenhagen," *Injury*, vol. 28, no. 4, pp. 311–313, 1997.

[2] S. Houshian, T. Tscherning, and P. Riegels-Nielsen, "The epidemiology of achilles tendon rupture in a Danish county," *Injury*, vol. 29, no. 9, pp. 651–654, 1998.

[3] C. Tallon, N. Maffulli, and S. W. B. Ewen, "Ruptured Achilles tendons are significantly more degenerated than tendinopathic tendons," *Medicine and Science in Sports and Exercise*, vol. 33, no. 12, pp. 1983–1990, 2001.

[4] N. Maffulli, S. W. Waterston, and S. W. B. Ewen, "Ruptured Achilles tendons show increased lectin stainability," *Medicine and Science in Sports and Exercise*, vol. 34, no. 7, pp. 1057–1064, 2002.

[5] N. Maffulli, S. W. B. Ewen, S. W. Waterston, J. Reaper, and V. Barrass, "Tenocytes from ruptured and tendinopathic achilles tendons produce greater quantities of type III collagen than tenocytes from normal achilles tendons: an in vitro model of human tendon healing," *American Journal of Sports Medicine*, vol. 28, no. 4, pp. 499–505, 2000.

[6] O. Arner, A. Lindholm, and S. R. Orell, "Histologic changes in subcutaneous rupture of the Achilles tendon; a study of 74 cases," *Acta chirurgica Scandinavica*, vol. 116, no. 5-6, pp. 484–490, 1959.

[7] L. Józsa and P. Kannus, "Histopathological findings in spontaneous tendon ruptures," *Scandinavian Journal of Medicine and Science in Sports*, vol. 7, no. 2, pp. 113–118, 1997.

[8] P. Kannus and L. Jozsa, "Histopathological changes preceding spontaneous rupture of a tendon: a controlled study of 891 patients," *Journal of Bone and Joint Surgery. Series A*, vol. 73, no. 10, pp. 1507–1525, 1991.

[9] S. De Jonge, C. Van Den Berg, R. J. De Vos et al., "Incidence of midportion Achilles tendinopathy in the general population," *British Journal of Sports Medicine*, vol. 45, no. 13, pp. 1026–1028, 2011.

[10] U. G. Longo, M. Ronga, and N. Maffulli, "Achilles tendinopathy," *Sports Medicine and Arthroscopy Review*, vol. 17, no. 2, pp. 112–126, 2009.

[11] N. Maffulli, P. M. Binfield, and J. B. King, "Tendon problems in athletic individuals," *The Journal of Bone & Joint Surgery—American Volume*, vol. 80, no. 1, pp. 142–144, 1998.

[12] N. Maffulli, "Rupture of the Achilles tendon," *Journal of Bone and Joint Surgery. Series A*, vol. 81, no. 7, pp. 1019–1036, 1999.

[13] J. Nestorson, T. Movin, M. Möller, and J. Karlsson, "Function after Achilles tendon rupture in the elderly: 25 patients older than 65 years followed for 3 years," *Acta Orthopaedica Scandinavica*, vol. 71, no. 1, pp. 64–68, 2000.

[14] H. Alfredson, T. Pietilä, P. Jonsson, and R. Lorentzon, "Heavy-load eccentric calf muscle training for the treatment of chronic achilles tendinosis," *American Journal of Sports Medicine*, vol. 26, no. 3, pp. 360–366, 1998.

[15] G. Nelen, M. Martens, and A. Burssens, "Surgical treatment of chronic Achilles tendinitis," *The American Journal of Sports Medicine*, vol. 17, no. 6, pp. 754–759, 1989.

[16] A. Coutts, A. MacGregor, J. Gibson et al., "Clinical and functional results of open operative repair for Achilles tendon rupture in a nonspecialist surgical unit," *Journal of the Royal College of Surgeons of Edinburgh*, vol. 47, no. 6, pp. 753–762, 2002.

[17] Y. Yasui, C. D. Murawski, A. Wollstein, and J. G. Kennedy, "Reoperation rates following ankle ligament procedures performed with and without concomitant arthroscopic procedures," *Knee Surgery, Sports Traumatology, Arthroscopy*, pp. 1–8, 2016.

[18] Y. Yasui, K. S. Vig, C. D. Murawski, P. Desai, I. Savage-Elliott, and J. G. Kennedy, "Open versus arthroscopic ankle arthrodesis: a comparison of subsequent procedures in a large database," *Journal of Foot and Ankle Surgery*, vol. 55, no. 4, pp. 777–781, 2016.

[19] D. Wang, N. B. Joshi, F. A. Petrigliano et al., "Trends associated with distal biceps tendon repair in the United States, 2007 to 2011," *Journal of Shoulder and Elbow Surgery*, vol. 25, no. 4, pp. 676–680, 2016.

[20] N. Maffulli, A. G. Via, and F. Oliva, "Chronic achilles tendon disorders: tendinopathy and chronic rupture," *Clinics in Sports Medicine*, vol. 34, no. 4, pp. 607–624, 2015.

[21] T. Y. Kostrominova and S. V. Brooks, "Age-related changes in structure and extracellular matrix protein expression levels in rat tendons," *Age*, vol. 35, no. 6, pp. 2203–2214, 2013.

[22] J. Wong, V. Barrass, and N. Maffulli, "Quantitative review of operative and nonoperative management of Achilles tendon ruptures," *American Journal of Sports Medicine*, vol. 30, no. 4, pp. 565–575, 2002.

Hindawi
BioMed Research International
Volume 2017, Article ID 2729807, 5 pages
https://doi.org/10.1155/2017/2729807

Research Article

UTE-T2* Analysis of Diseased and Healthy Achilles Tendons and Correlation with Clinical Score: An In Vivo Preliminary Study

Yang Qiao,[1] Hong-Yue Tao,[1] Kui Ma,[2] Zi-Ying Wu,[2] Jian-Xun Qu,[3] and Shuang Chen[1]

[1]*Department of Radiology, Huashan Hospital, Fudan University, Shanghai, China*
[2]*Department of Sports Medicine, Huashan Hospital, Shanghai, China*
[3]*GE Healthcare Applied Science Lab, Shanghai, China*

Correspondence should be addressed to Shuang Chen; chenshuang6898@126.com

Received 26 July 2016; Accepted 24 November 2016; Published 5 January 2017

Academic Editor: Haining Zhang

Objective. To compare T2* value of healthy and diseased Achilles tendons (AT) with a recently introduced three-dimensional ultrashort echo time (3D-UTE) sequence and analyze the correlation between T2* value and clinical scores. *Methods*. Ten patients with symptomatic Achilles tendon and ten healthy volunteers were investigated with 3D-UTE sequence on a 3T magnetic resonance (MR) scanner. T2* values of four regions in Achilles tendons were calculated. The clinical outcomes of patients were evaluated according to the American Orthopaedic Foot and Ankle Society (AOFAS) score and Achilles Tendon Rupture Score (ATRS). An independent sample t-test was used to compare the differences of T2* value and clinical scores between two groups. The Pearson correlation coefficient between clinical scores and T2* values was assessed. *Results*. The T2* values of Achilles tendon were statistically significantly different between patients and volunteers. The Pearson correlation coefficients between T2* and AOFAS or ATRS scores of patients were $r = -0.733$ and $r = -0.634$, respectively. *Conclusion*. The variability of T2* in healthy and pathologic AT can be quantified by UTE-T2*. T2* may be a promising marker to detect and diagnose AT tendinopathy. UTE-T2* could give a precise guidance to clinical outcome.

1. Introduction

Tendinopathy, a syndrome with tendon pain, tenderness, and swelling that limited the tendon function, is one of the most common injures in both athletic and nonathletic populations [1, 2]. Nowadays, due to a higher involvement in sports, the prevalence of Achilles tendinopathy is increasing [3]. Although the exact etiology is still uncertain, it is supposed that repetitive overload and overuse are the major causes [4]. They may lead to irreversible degenerative changes of the Achilles tendon (AT), for instance, destruction and decrease of collagen fiber in extracellular matrix, increased vascularity, and altered cellularity [5–7]. Consequently, a precise way to detect Achilles tendinopathy is highly demanded.

As a noninvasive and credible diagnostic tool, magnetic resonance imaging (MRI) has been widely used to evaluate the pathological changes of AT. But highly organized AT with very short T2 appear dark on conventional MRI sequences, for only tissues with long T2 relaxation times could be visualized [8, 9]. Thus a short echo time sequence is needed to acquire signal from the AT. Three-dimensional ultrashort echo time (UTE) imaging, with an echo time as short as 0.05–0.5 ms, provided direct visualization and quantitative T2*-mapping for short-T2* components [10–12]. Biochemical changes of early stage Achilles tendinopathy may affect T2* values of AT and thus can be caught and quantified with UTE sequences [8].

Therefore, the aim of this study was to investigate the capability of quantitative 3D-UTE-T2* in evaluating diseased AT and analyze the correlation between T2* value and American Orthopaedic Foot and Ankle Society (AOFAS) score or Achilles tendon Total Rupture Score (ATRS). We hypothesize that the pathologic AT would show increased T2* value while comparing with matched healthy samples and T2* value may be correlated with clinical score.

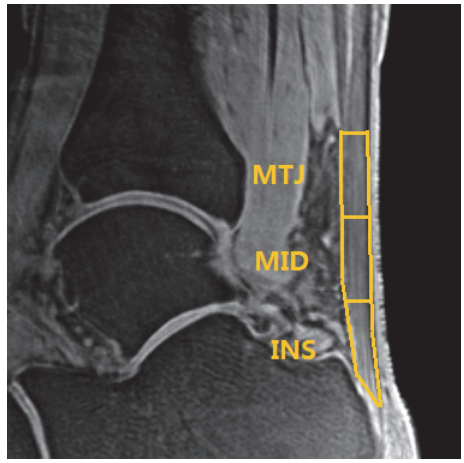


Figure 1: ROIs of Achilles tendon on sagittal UTE image acquired at TE = 0.032 ms. INS = insertion, MID = middle, MTJ = muscle-tendon junction; these three ROIs were equally divided according to the longitudinal length of Achilles tendon. The bulk ROI consisted of all the three ROIs.

2. Materials and Methods

2.1. Participants. The study was approved by the institutional review board of our hospital and all participants' informed consents were obtained. Ten patients (9 male/1 female, mean age 37.10 ± 8.60 years, BMI 23.00 ± 2.15 kg/m^2) with pain or abnormalities in the AT and ten healthy volunteers matched for sex, age, and BMI (9 male/1 female, mean age 37.40 ± 10.61 years, BMI 23.94 ± 2.32 kg/m^2) participated in the study. Participants were excluded if they had significant tendon rupture or any contraindication for MR.

2.2. MRI and Clinical Evaluation. All the participants were examined on a 3T MR scanner (Discovery 750, GE Healthcare, Waukesha, WI, USA) to get monoexponential calculation of T2* in the human AT in vivo. As a quantitative 3D-UTE sequence, four echo times (TE = 0.032, 7.5, 20.5, and 28 ms) were acquired. The parameters were set as follows: sagittal orientation, FOV = 140 × 140 mm, slice thickness = 2.0 mm, flip angle = 18, and number of excitations (NEX) = 1. Fat-saturated proton-density weighted turbo-spin echo (PD-TSE) sequence was underwent to acquire morphological assessment with the parameters: sagittal orientation, TR 2843.0 ms, FOV 180 × 180 mm, slice thickness 2.0 mm, flip angle 142, and number of excitations (NEX) = 2.

For clinical evaluation, AOFAS scoring system and ATRS were used to evaluate the patients' clinical outcome (0–100 points, worst to best).

2.3. Imaging Analysis. Images from the UTE-T2* sequence were analyzed by software in the work station of GE. The AT was segmented and divided into three parts equally according to length: insertion (INS), middle (MID), and muscle-tendon junction (MTJ) (Figure 1). These three ROIs as well as all bulk of AT regions on each echo of UTE-T2* images were drawn to get the mean MR signal. T2* value of each region is calculated by fitting the acquired signal at different echo time to a single exponential decay model (Figure 2).

2.4. Statistical Analysis. All statistical analyses were performed in SPSS 20.0 (SPSS Institute, Chicago, IL, USA). An independent sample t-test was used to compare the differences of T2* values between two groups. Pearson's correlation coefficient was used to analyze correlations between clinical scores and T2* values of patients. The difference would be statistically significant if P value < 0.05.

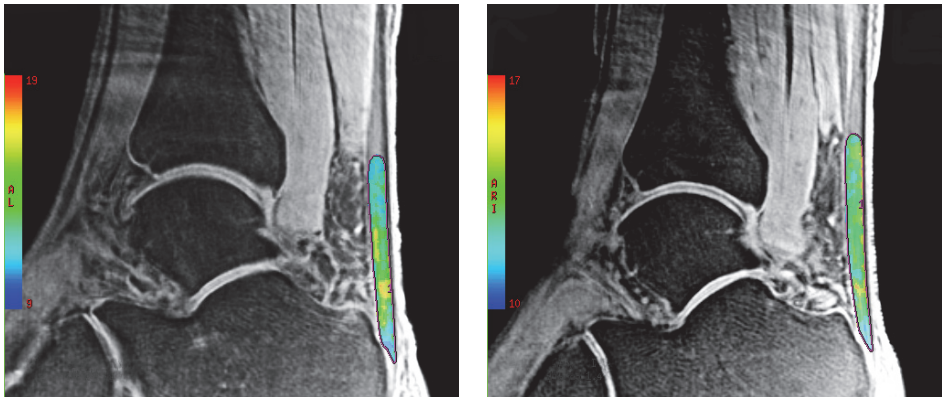
3. Results

There were no obvious tendon tears on MRI for all patients. The mean T2* value for bulk ROIs was significantly higher in patients than that in volunteers (12.508 ± 0.940 and 11.081 ± 0.297, P = 0.001) (Table 1). Separately, MTJ, MID, and INS regions of patients had statistically higher T2* value compared with the matched regions of volunteers (MTJ: 11.977 ± 0.831 and 11.005 ± 0.581, P = 0.007, MID: 12.474 ± 1.261 and 11.124 ± 0.394, P = 0.008, and INS: 13.124 ± 0.943 and 11.084 ± 0.522, P = 0.000). The difference in INS region is greater than that in MID and MTJ. In patients, the mean AOFAS and ATRS were 70.6 ± 5.58 and 52.8 ± 8.27, respectively. The T2* value for bulk region was negatively correlated with AOFAS as well as ATRS score (r = −0.733, P = 0.016, and r = −0.634, P = 0.049) (Figure 3).

In this study, T2* relaxation time in pathologic and healthy AT was measured using UTE-T2* sequence and a significant higher T2* value was observed in all four regions of diseased AT. Besides, T2* value of all bulk of AT in patients was found to be negatively correlated with AOFAS and ATRS score.

4. Discussion

UTE-T2* mapping, a novel quantitative technique, could catch the short-T2* relaxations from AT that are not well captured by standard T2 mapping [13]. In the early stages of Achilles tendinopathy, it is usually biochemical but not morphological changes that are found [14], which consist of destruction of collagen structure and increase of proteoglycan and water content [15]. UTE-T2* mapping is sensitive to these changes; thus it could be a useful tool to detect tendon disease in an early stage [12]. The results of this study suggest that the variability of Achilles tendinopathy can be quantified by UTE-T2*. The increasing of T2* value may due to disorganization of collagen structure and increasing of water content in tendons. What is more, the difference in INS region is greater. The reason could be that the enthesis is mostly involved in overuse injuries of AT [16, 17]. Gardin et al. [18] applied monoexponential calculation and showed a significant higher T2* relaxation time in symptomatic tendons compared with control tendons. In a study by Juras et al., they compared mono- and biexponential T2* analysis using variable-echo time sequence (vTE) and found that increased T2* in all parts of diseased AT with monoexponential analysis [11]. Juras et al. also reported a similar finding with bicomponent quantitative 3D-UTE. They found



(a) (b)

Figure 2: UTE T2* mapping on diseased (a) and healthy (b) Achilles tendons of a 36-year-old male acquired at TE = 0.032 ms. Color scale represents T2* values. The increase of T2* values was observed in diseased tendon.

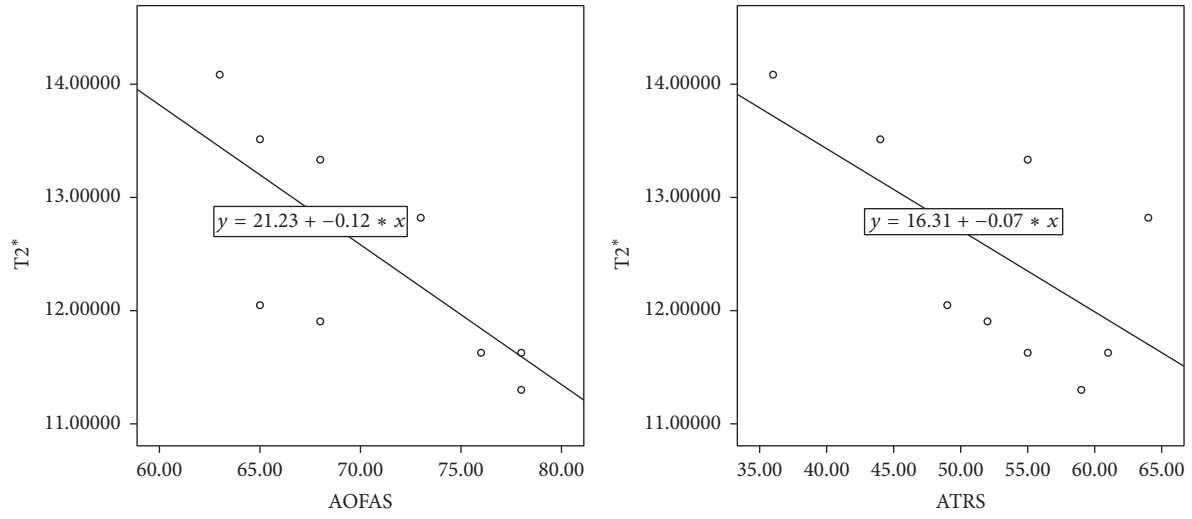


Figure 3: (a) A scatter plot of the bulk T2* and AOFAS of ten patients. The Pearson correlation coefficient was $r = -0.733$ ($P = 0.016$). (b) A scatter plot of the bulk T2* and ATRS of ten patients. The Pearson correlation coefficient was $r = -0.634$ ($P = 0.049$).

significant differences between healthy (10.28 ± 2.28 ms) and degenerated AT (12.85 ± 1.87 ms) in the long component of T2* [7]. While estimating the diagnostic value of T1 and T2* relaxation times and off-resonance saturation ratio, Grosse et al. also observed statistical significant differences between the patients with tendinopathy and controls [19].

We applied monoexponential calculation of UTE-T2*. Juras et al. [11] found that the short component of T2* reflects the changes of Achilles tendinopathy more accurately than the monoexponential T2* [11]. However, owing to the longer scanning time and higher sensitivity to movements and the magic angle, biexponential calculation is more difficult to be applied clinically [18].

To the best of our knowledge, only a few studies analyzed the correlation between T2* value and clinical score. Both AOFAS and ATRS scores are widely used in clinical practice and validated in many studies [20, 21]. They had general assessment of the AT situation. T2* value of the bulk region in patients was correlated with AOFAS and ATRS score, which suggests that T2* could give a precise guidance to clinical outcome of patients with Achilles tendinopathy. Juras et al. got a similar correlation between ATRS score and the monoexponential T2* [11].

There are also some limitations in the study. Firstly, a small number of patients which would lead to increased statistical deviation. The patient cohort would be enlarged in our next study. Secondly, monoexponential calculation of T2* reflects the mean value of all the components of relaxation time, which may lead to an underrate of T2*, especially in diseased tendons [7].

5. Conclusion

In conclusion, the differences between T2* in healthy and pathologic tendons could be observed by UTE-T2*. As the preliminary patient data suggest, UTE-T2* is an acute marker

Table 1: Comparison of T2* values between patients and volunteers. Mean (M) and standard deviation (SD) as well as *P* value were presented. The *P* value applied the significant difference in the four parts. Mean (M) and standard deviation (SD) of AOFAS and ATRS scores were also presented.

		Patients		Volunteers		*P* values
		M	SD	M	SD	
T2* values	Bulk	12.508	0.940	11.081	0.297	0.001
	MTJ	11.977	0.831	11.005	0.581	0.007
	MID	12.474	1.261	11.124	0.394	0.008
	INS	13.124	0.943	11.084	0.522	0.000
AOFAS score		70.6	5.58			
ATRS score		52.8	8.27			

to detect AT tendinopathy in the early stage and it gives a precise guidance to clinical outcome. By further investigation in larger cohort of patients, different terms of follow-up after treatments are required to define the exact role of UTE-T2* for monitoring the change of AT.

Competing Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

Authors' Contributions

Yang Qiao and Hong-Yue Tao contributed equally to this work and should be considered co-first authors.

Acknowledgments

This project was subsidized by the National Science Foundation for Distinguished Young Scholars of China (no. 81501440) and the Project of Shanghai Municipal Science and Technology Commission (16ZR1404600).

References

[1] S. G. Rees, C. M. Dent, and B. Caterson, "Metabolism of proteoglycans in tendon," *Scandinavian Journal of Medicine and Science in Sports*, vol. 19, no. 4, pp. 470–478, 2009.

[2] A. Möller, M. Åström, and N. E. Westlin, "Increasing incidence of Achilles tendon rupture," *Acta Orthopaedica Scandinavica*, vol. 67, no. 5, pp. 479–481, 1996.

[3] S. L. James, B. T. Bates, and L. R. Osternig, "Injuries to runners," *The American Journal of Sports Medicine*, vol. 6, no. 2, pp. 40–50, 1978.

[4] P. Kannus and L. Jozsa, "Histopathological changes preceding spontaneous rupture of a tendon: a controlled study of 891 patients," *Journal of Bone and Joint Surgery A*, vol. 73, no. 10, pp. 1507–1525, 1991.

[5] M. Aström and A. Rausing, "Chronic Achilles tendinopathy. A survey of surgical and histopathologic findings," *Clinical Orthopaedics and Related Research*, no. 316, pp. 151–164, 1995.

[6] T. Movin, A. Gad, F. P. Reinholt, and C. Rolf, "Tendon pathology in long-standing achillodynia. Biopsy findings in 40 patients," *Acta Orthopaedica Scandinavica*, vol. 68, no. 2, pp. 170–175, 1997.

[7] V. Juras, S. Zbyn, C. Pressl et al., "Regional variations of T_2^* in healthy and pathologic achilles tendon in vivo at 7 Tesla: preliminary results," *Magnetic Resonance in Medicine*, vol. 68, no. 5, pp. 1607–1613, 2012.

[8] S. Peto, P. Gillis, and V. P. Henri, "Structure and dynamics of water in tendon from NMR relaxation measurements," *Biophysical Journal*, vol. 57, no. 1, pp. 71–84, 1990.

[9] M. D. Robson, P. D. Gatehouse, M. Bydder, and G. M. Bydder, "Magnetic resonance: an introduction to ultrashort TE (UTE) imaging," *Journal of Computer Assisted Tomography*, vol. 27, no. 6, pp. 825–846, 2003.

[10] P. D. Gatehouse and G. M. Bydder, "Magnetic resonance imaging of short T2 components in tissue," *Clinical Radiology*, vol. 58, no. 1, pp. 1–19, 2003.

[11] V. Juras, S. Apprich, P. Szomolanyi, O. Bieri, X. Deligianni, and S. Trattnig, "Bi-exponential T2* analysis of healthy and diseased Achilles tendons: an in vivo preliminary magnetic resonance study and correlation with clinical score," *European Radiology*, vol. 23, no. 10, pp. 2814–2822, 2013.

[12] M. D. Robson, M. Benjamin, P. Gishen, and G. M. Bydder, "Magnetic resonance imaging of the Achilles tendon using ultrashort TE (UTE) pulse sequences," *Clinical Radiology*, vol. 59, no. 8, pp. 727–735, 2004.

[13] E. Y. Chang, J. Du, and C. B. Chung, "UTE imaging in the musculoskeletal system," *Journal of Magnetic Resonance Imaging*, vol. 41, no. 4, pp. 870–883, 2015.

[14] S. Tom, J. Parkinson, M. Z. Ilic, J. Cook, J. A. Feller, and C. J. Handley, "Changes in the composition of the extracellular matrix in patellar tendinopathy," *Matrix Biology*, vol. 28, no. 4, pp. 230–236, 2009.

[15] G. H. Filho, J. Du, B. C. Pak et al., "Quantitative characterization of the Achilles tendon in cadaveric specimens: T1 and T2* measurements using ultrashort-TE MRI at 3 T," *American Journal of Roentgenology*, vol. 192, no. 3, pp. W117–W124, 2009.

[16] M. Kvist, "Achilles tendon injuries in athletes," *Sports Medicine*, vol. 18, no. 3, pp. 173–201, 1994.

[17] M. Benjamin, H. Toumi, J. R. Ralphs, G. Bydder, T. M. Best, and S. Milz, "Where tendons and ligaments meet bone: attachment sites ('entheses') in relation to exercise and/or mechanical load," *Journal of Anatomy*, vol. 208, no. 4, pp. 471–490, 2006.

[18] A. Gardin, P. Rasinski, and J. Berglund, "T2* relaxation time in achilles tendinosis and controls and its correlation with clinical score," *Journal of Magnetic Resonance Imaging*, vol. 43, pp. 1417–1422, 2016.

[19] U. Grosse, R. Syha, T. Hein et al., "Diagnostic value of T1 and T2* relaxation times and off-resonance saturation effects in the evaluation of achilles tendinopathy by MRI at 3T," *Journal of Magnetic Resonance Imaging*, vol. 41, no. 4, pp. 964–973, 2015.

[20] L. J. Lapidus, B. A. Ray, and P. Hamberg, "Medial Achilles tendon island flap-a novel technique to treat reruptures and neglected ruptures of the Achilles tendon," *International Orthopaedics*, vol. 36, no. 8, pp. 1629–1634, 2012.

[21] R. S. Kearney, J. Achten, S. E. Lamb, C. Plant, and M. L. Costa, "A systematic review of patient-reported outcome measures used to assess Achilles tendon rupture management: what's being used and should we be using it?" *British Journal of Sports Medicine*, vol. 46, no. 16, pp. 1102–1109, 2012.

Hindawi Publishing Corporation
BioMed Research International
Volume 2016, Article ID 9364170, 8 pages
http://dx.doi.org/10.1155/2016/9364170

Clinical Study

A Prospective Study of Platelet-Rich Plasma as Biological Augmentation for Acute Achilles Tendon Rupture Repair

Jian Zou,[1] Xiaolian Mo,[2] Zhongmin Shi,[1] Tanzhu Li,[2] Jianfeng Xue,[1] Guohua Mei,[1] and Xiaolin Li[1]

[1]*Shanghai Jiaotong University Affiliated Sixth People's Hospital, Shanghai 200233, China*
[2]*The People's Hospital of Shigatse City, Shigatse, Tibet 857000, China*

Correspondence should be addressed to Zhongmin Shi; 18930177323@163.com

Received 1 July 2016; Revised 26 November 2016; Accepted 7 December 2016

Academic Editor: Hong-Yun Li

Acute Achilles tendon rupture is one of the most common tendon injuries in adults. We hypothesized that Platelet-Rich Plasma (PRP) can be used as biological augmentation for surgical treatment of acute Achilles tendon rupture. Our study is a prospective randomized controlled trial. Patients with acute Achilles tendon rupture undergoing surgical repair were randomly assigned into either control group or PRP group. End-to-end modified Krackow suture was performed in both groups. In the PRP group, PRP was injected into the paratenon sheath and around the ruptured tissue after the tendon was repaired. Postoperatively we evaluated isokinetic muscle strength at 3, 6, 12, and 24 months. In addition, ankle ROM, calf circumference, Leppilahti score, and the SF-36 score were evaluated at 6, 12, and 24 months after operation. At 3 months, the PRP group had better isokinetic muscle. The PRP group also achieved higher SF-36 and Leppilahti scores at 6 and 12 months. At 24 months, the PRP group had an improved ankle range of motion compared to the control group. Our study results suggest that PRP can serve as a biological augmentation to acute Achilles tendon rupture repair and improves both short and midterm functional outcomes.

1. Introduction

The acute Achilles tendon rupture is one of the most common tendon injuries in adults. The incidence of Achilles tendon injury is higher among the middle-aged population because of their participation in high-level sports [1–3]. Surgical treatment will accelerate the healing process, reduce rerupture, and improve the quality of life.

The use of Platelet-Rich Plasma (PRP) in the treatment of orthopedic injuries has been widely reported in recent years [4–7]. PRP is defined as a high concentration of platelets in plasma after special processing. Platelets are known to contain more than 300 bioactive proteins, including vascular endothelial growth factor (VEGF), insulin-like growth factor (IGF), fibroblast growth factor, platelet-derived growth factor (PDGF), platelet-derived epidermal growth factor (PD-EGF), transforming growth factor beta (TGFb), and epidermal growth factor (EGF) [8–10]. Thus, using PRP promotes postoperative healing and can potentially improve functional outcomes by concentrating these platelet-derived bioactive proteins at the side of injury.

The objective of this study was to evaluate clinical outcomes and calf muscle strength recovery after surgical repair of acute Achilles tendon repair with PRP augmentation.

2. Materials and Methods

2.1. Patient Recruitment. The current study was approved by Shanghai Sixth People's Hospital Ethics review board. All participants signed informed consent prior to entering the study.

Patients who presented with acute Achilles tendon rupture and underwent surgical repair at the Department of Orthopaedics, Shanghai Sixth People's Hospital, from January 2013 to January 2014, were recruited in the study. These patients were diagnosed with acute Achilles tendon rupture by the presence of a palpable gap, a positive Thompson squeeze test and ultrasonography were included.

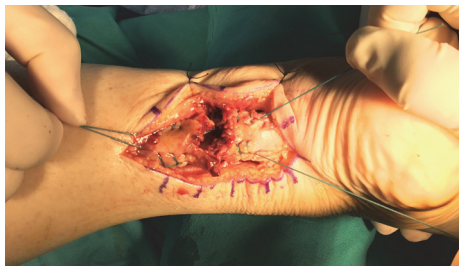


Figure 1: Modified Krackow suture technique was performed.

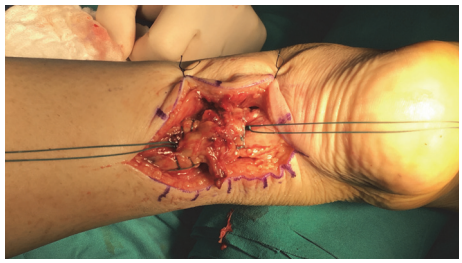


Figure 2: Modified Krackow suture technique was performed and maintained lacerated tissue.

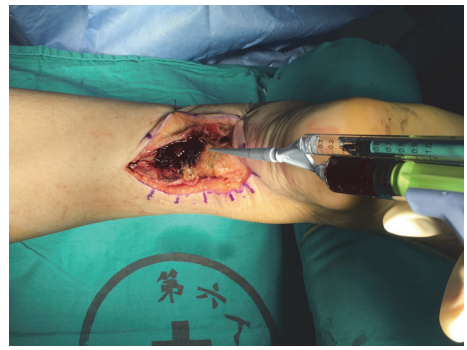


Figure 3: PRP was injected in rupture ends and paratenon sheath.

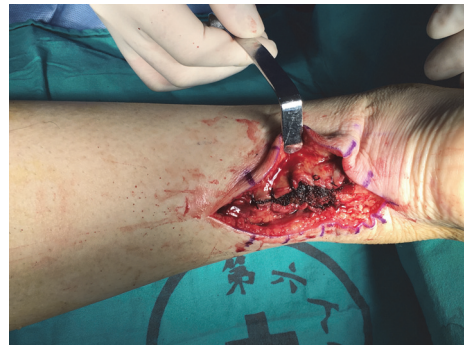


Figure 4: Close paratenon sheath and preserve PRP in it.

Included patients were aged 18–45 years, with a confirmed closed acute Achilles tendon rupture due to noncontact sport injuries. Major exclusion criteria included previously incurred Achilles tendinopathy, prior Achilles tendon rupture, patients with previous surgical procedures on the affected or contralateral side, open Achilles tendon rupture, rupture more than 3 weeks prior to presentation, pathological rupture, smokers, and polytrauma. In addition, patients with diabetes, paraplegia, or any peripheral neuropathy that could impair healing were also excluded. Our study is a prospective randomized controlled trial. Patients diagnosed with acute Achilles tendon rupture were randomly assigned to control group or PRP group. Computer generated a blinded randomization number. If generated number was an odd number, the patient was assigned to PRP group. If generated number was an even number, the patient was assigned to control group. RICE principle (rest, ice, compression, and elevation) was used to decrease leg swelling in all patients before the surgery.

2.2. PRP Preparation. The PRP was prepared using the WEGO Platelet-Rich Plasma Preparation Kits (WEGO Ltd., Shandong, China) [11]. Approximately 40 mL of blood was taken from patient via venipuncture and then placed in Kits and spun in a potable centrifuge (WEGO LTD, Shandong, China) at 2000 rpm for 10 minutes twice. This standard process produces 3 to 4 ml of plasma that has 6 times higher platelet concentration than normal physiological value. The leukocytes in PRP are also 4 times higher in concentration than normal blood.

2.3. Operation Procedure. All patients were operated on under epidural anesthesia. They were placed in prone position with thigh tourniquet applied. In both groups, end-to-end modified Krackow suture technique [12] was performed after longitudinal incision of the fascia and paratenon. To maintain the Achilles length in PRP group, only blood clots were removed. The ruptured tissue was preserved at both ends. The ruptured Achilles tendon was repaired with a number 2 nonabsorbable Ethibond suture (Ethicon, Somerville, NJ) (Figures 1 and 2). PRP was injected into the paratenon sheath and the surrounding lacerated tissue (Figures 3 and 4). Then, the paratenon was carefully closed with number 3-0 braided polyglycolic absorbable suture (Vicryl, Ethicon). The skin was closed with number 4-0 Prolene suture.

In the control group, the same modified Krackow suture technique was performed after the rupture ends of the tendon were debrided without PRP injection.

2.4. Postoperative Care. Patients in both groups underwent the same postoperative protocol. All patients had postoperation follow-up assessment at 3 weeks and 3, 6, 12, and 24 months. The surgical site was examined at 3 weeks postop. If there was no evidence of infection, skin sutures were removed. Postoperatively, the foot was immobilized in an anterior splint for 3 weeks, followed by nonweight-bearing walking boot with heel lifts and daily active range of motion for the first 6 weeks. Then the heal lift height was decreased by 1 mm every day. The patient was allowed gradual return to full weight bearing walking over another 6 weeks. Full weight bearing was permitted in 3 months, and full activities were

permitted as tolerated in 6 months. Patients were reassessed at 12 and 24 months after surgery.

2.5. Outcome Measures. The primary outcome measures were the Leppilahti score at 6, 12, and 24 months after surgery. The secondary outcome measures including the Short Form (36) Survey (SF-36) score, ankle range of motion (ROM), and calf circumference were evaluated at 6, 12, and 24 months after surgery. In addition, Isokinetic evaluation was performed at 60 degrees per second, 120 degrees per second and 240 degrees per second angular speeds with the Multi-Joint Isokinetic Dynamometer Prima DOC (Firenze, Italy) at 3, 6, 12, and 24 months. The calf circumference and the isokinetic calf muscle strength were measured and compared with the contralateral normal side. An independent clinical observer performed all evaluations.

2.6. Leppilahti Score. The Leppilahti score includes subjective factors (pain, stiffness, muscle weakness, footwear restriction, and subjective outcome) and objective factors (range of ankle active motion and isokinetic calf muscle strength score). The isokinetic muscle strength score is calculated from the plantar flexion and dorsiflexion peak torques of 3 different ankle test speeds as described by Leppilahti et al. [13]. The maximum Leppilahti score is 102 points. A total score of 87 to 102 points was graded as excellent, 72 to 86 as good, 57 to 71 as fair, and 56 or less as poor. Patients completed the subjective part of the Leppilahti score independently without any supervision or instruction.

2.7. Ankle Range of Motion (ROM). Bilateral ankle dorsal flexion was assessed at 6, 12, and 24 months after operation. The physiotherapist calculated an average of three readings with a hand-held goniometer that measured the maximum ankle dorsiflexion.

2.8. Strength Measurements. The isokinetic strength of both ankles in plantar flexion and dorsiflexion was assessed 3, 6, 12, and 24 months after surgery. An independent athletic therapist, who is blinded to both groups, used isokinetic dynamometer (CSMI HUMAC Norm, Stoughton, Massachusetts, USA) to measure muscle strength. During the measurements as patients sat on the positioning chair their feet were fixed with Velcro straps. All patients were encouraged to achieve a maximal effort during testing. Five isokinetic plantar flexion and dorsiflexion cycles were performed at a speed of 60 degrees per second, 120 degrees per second, and 240 degrees per second. The peak torque of the injured leg as well as uninjured leg was measured. The relative performance of the injured limb was calculated as follows: injured side/healthy side $*$ 100%.

2.9. Complications. The complication rates included the rerupture rates, superficial and deep infection rates, and sural nerve injury. All complications were recorded during the follow-up assessment.

2.10. Statistical Methods. Descriptive statistics included mean ± standard deviation, median (interquartile, IQR), frequency distribution, and percentage. Pearson's chi-square test and Fisher's exact test were used to evaluate the categorical variables. The fitness of the variables to normal distribution was evaluated using visual (histogram and probability graphs) and analytic methods (Shapiro-Wilk Test). In the presence of a significant difference between two independent groups, Student's t-test was used for normal distributed variables. For variables not distributed normally, Mann–Whitney U test was used to compare two independent groups. Repeated-measures analysis of variance (ANOVA) was employed to determine if there were any significant differences between the time points (3, 6, 12, and 24 months after surgery) or between the groups. Post hoc Bonferroni analysis was performed to identify the mean differences within the existence of a statistically significant P value ($P < 0.05$) in time or between groups. Two-tailed tests of significance are reported and P value <0.05 were considered statistically significant. Statistical analyses were performed using SPSS statistical software program (version 11.0.0, SPSS, IBM).

Table 1: The basic information of two groups.

	PRP group ($n = 16$)	Control group ($n = 20$)
Age (yrs)	30.2 ± 5.8	28.9 ± 5.7
Sex		
Male	16	19
Female	0	1
Affected leg		
Left	6	7
Right	10	13
Causes of rupture		
Sport	15	19
Others	1	1
Location of rupture		
Insertion	0	0
Midtendon	16	20
Enthesis	0	0

3. Results

3.1. Demographic and Anatomical Characteristics of Achilles Tendon Rupture. A total number of 52 cases of acute Achilles ruptures were recruited during the study period. However, 9 patients refused operative treatment and 7 patients refused postoperative assessment. Therefore, only 36 patients were included in the study. There were 16 cases in the PRP group and 20 cases in the control group. Most of patients were males and had sports related tendon rupture. All ruptures happened at middle portion of the tendon. There were no significant differences in demographic data or pattern of tendon rupture between two groups (Table 1).

3.2. Postoperative Complications. In control group, 18 cases healed within 3 months. Among them, there were 2 superficial infections and that resolved with antibiotic treatment.

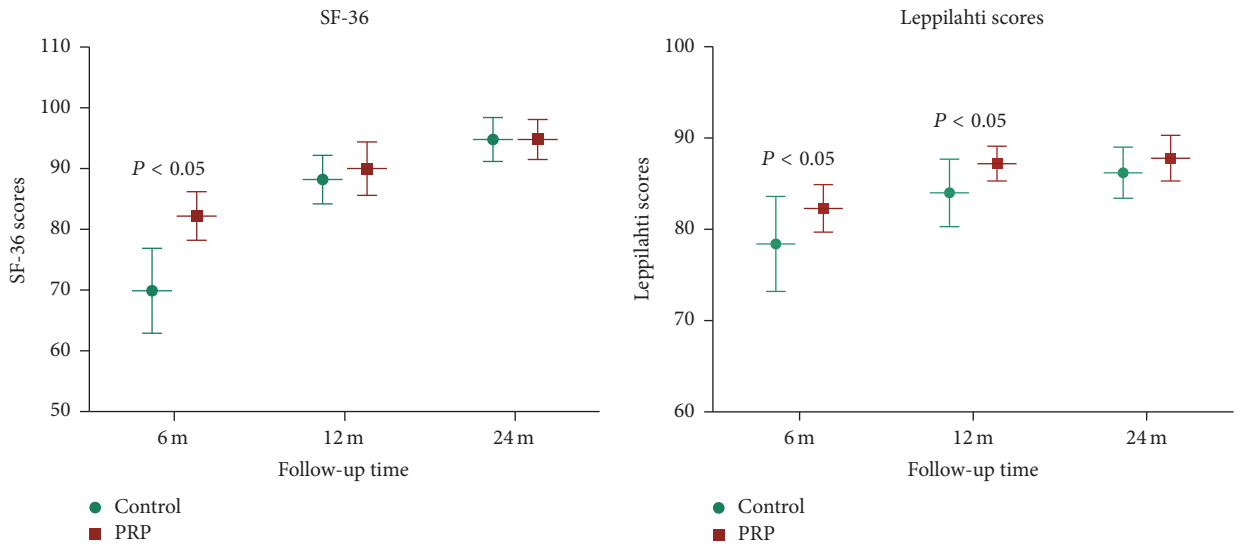


Figure 5: The SF-36 scores and Leppilahti scores were evaluated at 6, 12, and 24 months after operation. PRP group had higher scores in SF-36 and Leppilahti scores than control group for up to postoperative 6 and 12 months, respectively.

Table 2: Ankle range of motion (ROM).

	Control group (mean ± SD) (°)	PRP group (mean ± SD) (°)	*P* value
Plantar flexion			
6 months	−4.5 ± 0.5	−3.0 ± 0.3	<0.001
12 months	−2.2 ± 0.4	−0.9 ± 0.4	<0.001
24 months	−2.0 ± 0.4	−1.1 ± 0.3	<0.001
Dorsiflexion			
6 months	−4.4 ± 0.4	−2.6 ± 0.4	<0.001
12 months	−2.2 ± 0.3	−1.1 ± 0.5	<0.001
24 months	−1.9 ± 0.4	−1.0 ± 0.4	<0.001

One had deep infection and one had a rerupture. The deep infection was because of allergic reaction to nonabsorbable suture. After debridement, the wound healed at 4 months from the first operation. The rerupture was caused due to a mechanical fall and it was managed successfully nonoperatively with walking boots for one month. In the PRP group, all 16 cases healed uneventfully in 3 months after surgery. There was neither infection nor rerupture. For both groups, there was no sural nerve injury or skin adhesion.

3.3. Patient Reported Outcome Measures. Both the Leppilahti scores and SF-36 scores were evaluated at 6 months, one year, and two years after the operation. For the SF-36 scores, although there were no significant differences between two groups at one and two years of follow-up, the scores in the PRP group were significantly higher than in control group at 6 months postop. Similar results were seen in Leppilahti scores with significantly higher scores in PRP group at 6 months and 1 year after the surgery. Finally, both the groups had similar Leppilahti scores at 2-year postoperative follow-up (Figure 5). The results were excellent for 12 patients (60%), good for 7 patients (35%), and fair for 1 patient (5%) in control group. In PRP group, scores were excellent for 13 patients (72%) and good for 5 patients (28%) at 2-year postoperative follow-up.

3.4. Ankle and Leg Function Tests. The ankle ROM was measured for plantar flexion and dorsiflexion at 6-month, one-year, and two-year postoperative follow-up. Patients in the PRP group had significantly better range of motion at all time points (Table 2).

When compared to the normal (contralateral) side of calf circumferences, calf circumferences at the injured side were smaller, but there were no significant differences in the calf circumferences between two groups at all time periods (Table 3).

The isokinetic calf muscle strengths were evaluated in percentage of plantar flexion strength and the percentage of dorsiflexion strength for 60 degrees, 120 degrees, and 240 degrees at 3-month, 6-month, one-year, and two-year postoperative follow-up. At 3-month postoperative follow-up, PRP group had significantly higher percentage in all

Table 3: Calf circumference (%).

	6 months	1 year	2 years
Control	85.3 ± 4.0	95.8 ± 3.3	96.2 ± 3.1
PRP	88.1 ± 4.9	95.5 ± 3.0	96.8 ± 2.2
t	1.9	0.3	0.6
P	0.07	0.73	0.64

measured degrees than those in the control group. However, after 6-month follow-up both groups were similar (Tables 4 and 5 and Figure 6).

4. Discussion

PRP has been used to enhance bone and soft tissue healing for several years, previously being mainly used in oral and maxillofacial surgery [4, 14]. The use of PRP in orthopedics and sports medicine is a novel concept. It has been used as an agent to improve the healing of muscle, bone, cartilage, tendon, and ligaments [15]. Previous laboratory studies have shown PRP as an activator of circulation-derived cells for the improvement of the initial tendon healing process. The major findings of our study were to find out the role of PRP in short and midterm outcome of surgical repair of acute Achilles tendon rupture. Both subjective patients reported scores and objective functional tests results demonstrated that PRP group has better early-midterm outcomes. In addition, the PRP group showed better ankle ROM for up to two years after operation.

Our results were consistent with other studies. Sánchez et al. [16] demonstrated an early recovery of ROM and running after the application of PRP in Achilles tendon suture. The functional performances in isokinetic calf muscle strength were better in the PRP group. In our study, at postoperative 3-month follow-up, we also found that the calf circumference proportion was similar in both groups. This implicated the similar levels of leg muscle decrease after the injury and operation between two groups. However, the muscle performance was statistically different between two groups for up to 6-month postoperative follow-up. This could be due to the biological augmentation effect of PRP on early-midterm tendon healing. The underlying mechanism is likely increased vascular activity after direct administration of PRP into tendon body.

However, one potential limitation of our study is the different surgical preparation of the rupture ends of the tendon in the two groups. In the control group, the ruptured tendon was debrided before end-to-end repair. This may have caused shortening of the Achilles tendon, while in the PRP group, there was no tendon debridement performed; therefore, tendon length could be maintained. We need further studies to evaluate whether the difference in final tendon length influenced our current study results.

The functional performances in isokinetic calf muscle strengths were better in the PRP group at postoperative 3-month follow-up. This result was also similar to Sánchez et al.'s study [16], where they proved that PRP injection after surgical repair of Achilles tendon rupture could facilitate patients' return to sports activities and training faster than others.

Apart from ankle ROM, other midterm outcomes of the PRP injection as an adjuvant of acute Achilles tendon surgical repair were similar to control group in our study. Those results were consistent with other studies as well. De Carli et al. [17] showed a substantial equivalent structural and functional results in Achilles tendon ruptures surgically treated with and without PRP at 6 months and 24 months after surgery.

When measuring patient reported outcome, we used both SF-36 and the Leppilahti scores. The SF-36 score is a validated, commonly used tool in postoperative recovery. It contains both physical and psychological evaluation. In our study, patients who had PRP injection had higher scores in SF-36 at 3 months after the surgery when compared to patients in control group. The Leppilahti scores is the measurement for subjective report outcome after ankle, tendon treatment. The scores were higher at 3- and 6-month postoperative follow-up in PRP group than in control group. These subjective outcomes were consistent with our functional (objective) outcomes but were different from other studies. De Carli et al. [17] found that the Foot and Ankle Outcome Score (FAOS) and VISA-A showed no difference between the PRP and control groups at 1, 3, 6, and 24 months after operation of acute Achilles tendon rupture. The Leppilahti score system has been used in our hospital for years. Because of the different scales, it would be difficult to compare our results with others.

The complication after surgical treatment of acute Achilles tendon rupture could delay the healing process. The common complications include infection and rerupture. Bartel et al. [18] conducted a systematic review showing that the wound site infection rate was 4% in open surgery and the risk of rerupture was 3.4%. Among our participants, the rerupture rate was only 1% in control group and none in PRP group. The infection rate was 1.5% in control group. Despite there being no statistical difference between the two groups, our small sample size may give rise to a false negative result. Recent research [5, 19] suggests that PRP has significantly high serum growth factor levels, and it also has potential anti-infection effects when applied to tissue. The concentration of PRP obtained in our study using the WEGO apparatus is 6 times the concentration of platelets in baseline blood and 4 times the white cells of normal value. This concentration has been found to be effective both in vitro and in vivo studies in proliferation and promote angiogenesis of the tissue [20, 21].

PRP is most effective in the treatment of chronic tendon injuries, such as Tennis elbow. But the effectiveness in treating chronic noninsertional Achilles tendinopathy is controversial. Gaweda et al. [22] treated Achilles tendinopathy with local injection of autologous PRP. They found that PRP was effective in elimination of clinical symptoms, normalization of tendon thickness in the region of intrasubstance tears, decreased peritendineum and tendon thickening, and resolution of hypoechogenic lesions. On the contrary, De Vos et al. [6] reported that a PRP injection did not have improvement in pain and function of patients with chronic

Table 4: Percentage of plantar flexion strength (%).

	3 months	6 months	1 year	2 years
60°/s				
Control	64.2 ± 7.0	86.0 ± 4.7	95.1 ± 2.8	94.4 ± 3.3
PRP	68.8 ± 3.3	89.6 ± 6.4	96.0 ± 2.3	95.0 ± 2.3
t	3.6	2.3	0.6	1.1
P	0.022	0.059	0.28	0.54
120°/s				
Control	62.8 ± 5.5	83.2 ± 4.6	92.9 ± 3.5	93.8 ± 3.5
PRP	66.1 ± 2.9	86.1 ± 4.0	93.5 ± 3.2	93.5 ± 3.6
t	2.103	1.996	0.4899	0.2953
P	0.043	0.054	0.63	0.77
240°/s				
Control	61.4 ± 9.2	80.6 ± 5.1	92.6 ± 2.0	94.1 ± 3.4
PRP	67.8 ± 5.5	83.1 ± 3.1	92.5 ± 2.7	94.2 ± 3.0
t	2.4	3.9	0.7	0.2
P	0.021	0.091	0.9	0.94

Table 5: Percentage of dorsiflexion strength (%).

	3 months	6 months	1 year	2 years
60°/s				
Control	65.6 ± 6.6	86.6 ± 6.6	95.5 ± 2.5	95.7 ± 2.4
PRP	69.7 ± 4.0	90.6 ± 6.0	96.1 ± 2.6	96.2 ± 1.9
t	2.2	1.8	0.6	0.7
P	0.035	0.072	0.52	0.46
120°/s				
Control	63.0 ± 5.3	83.4 ± 5.9	93.1 ± 3.0	94.7 ± 2.6
PRP	67.9 ± 4.4	84.9 ± 5.9	94.0 ± 3.3	94.4 ± 3.2
t	3.2	3.1	0.2	0.1
P	0.006	0.44	0.39	0.74
240°/s				
Control	61.4 ± 3.2	80.45 ± 4.4	94.9 ± 2.6	95.0 ± 3.1
PRP	67.9 ± 4.1	83.5 ± 5.0	94.7 ± 3.5	95.6 ± 2.8
t	5.3	1.9	0.2	0.5
P	<0.001	0.061	0.83	0.61

Achilles tendinopathy. De Jonge et al. [23] also showed no clinical or ultrasonographic superiority of Platelet-Rich Plasma injection over placebo injection in chronic Achilles tendinopathy at 1-year follow-up.

There are several limitations in our study. Firstly, the number of patients in our study was relatively small and so the power is limited. This could result in either false positive or false negative results. Secondly, the two groups had different surgical preparation of the ruptured tendon that caused difference in tendon length. This may be responsible in differences in ankle ROM. Thirdly, the time interval between follow-up times may be too long, especially during early and midtime period after the surgery. If we could evaluate our patients more frequently, we may be able to find out how quick the PRP effect and how long it could last. Such results may be more useful for the clinical use of PRP.

5. Conclusion

PRP is safe and effective as a biological augmentation agent for surgical repair of acute Achilles tendon rupture. It may improve early-midterm postoperative functional recovery. Further studies to determine the long-term effects of PRP on functional outcomes after Achilles tendon repair and the cost-benefit analysis of using PRP are recommended.

Competing Interests

All authors state that they have no conflict of interests.

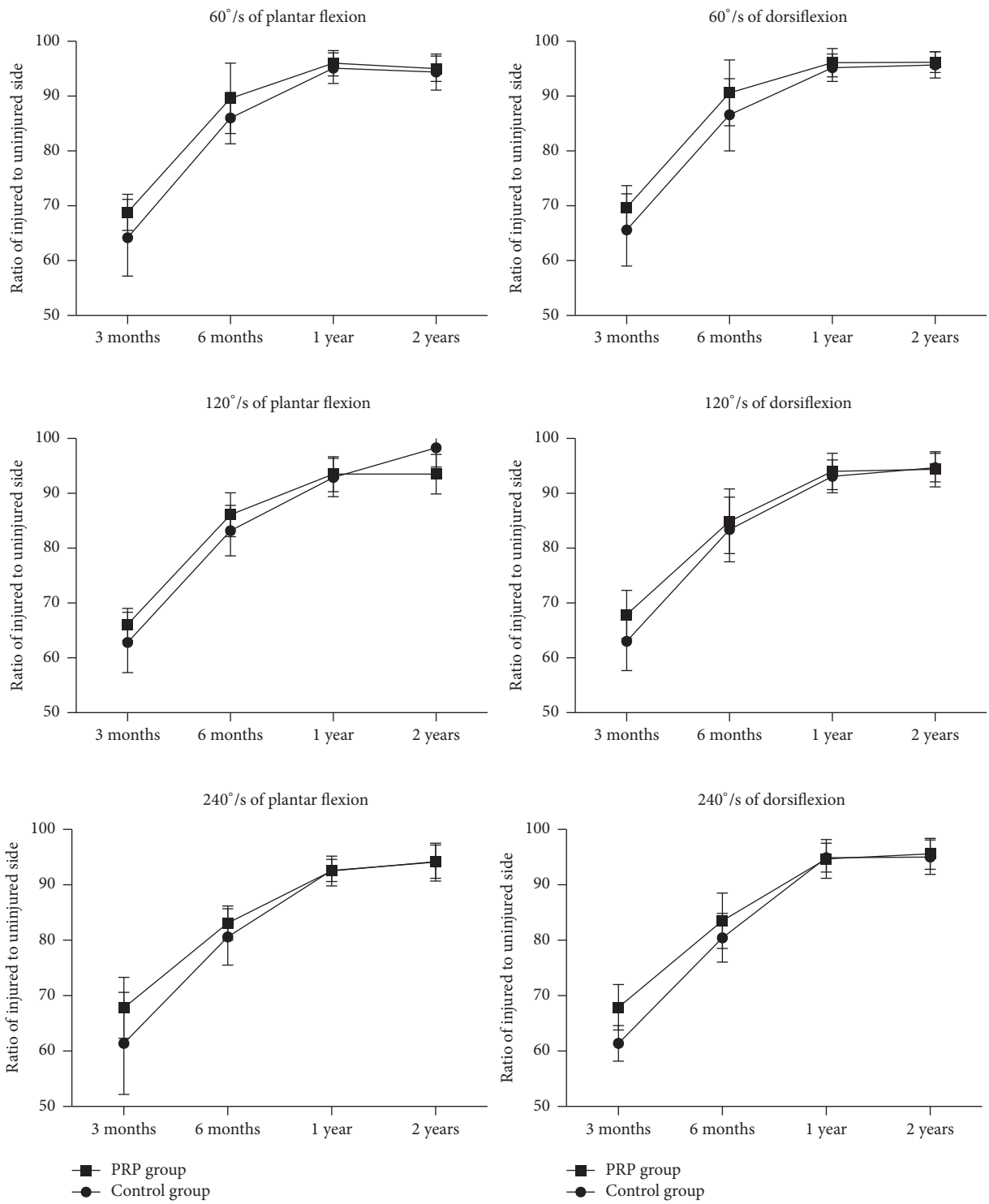


Figure 6: Demography of isokinetic calf muscle strength.

Authors' Contributions

Jian Zou and Xiaolian Mo contributed equally to this study and should be regarded as first authors.

Acknowledgments

Selected data from this study were presented at the Shanghai Sixth People's Hospital, Shanghai, China.

References

[1] N. Olsson, M. Petzold, A. Brorsson, J. Karlsson, B. I. Eriksson, and K. G. Silbernagel, "Predictors of clinical outcome after acute achilles tendon ruptures," *The American Journal of Sports Medicine*, vol. 42, no. 6, pp. 1448–1455, 2014.

[2] J. F. Keating and E. M. Will, "Operative versus non-operative treatment of acute rupture of tendo Achillis: a prospective randomised evaluation of functional outcome," *The Journal of Bone & Joint Surgery—British Volume*, vol. 93, no. 8, pp. 1071–1078, 2011.

[3] F. Neumayer, E. Mouhsine, Y. Arlettaz, G. Gremion, M. Wettstein, and X. Crevoisier, "A new conservative-dynamic treatment for the acute ruptured achilles tendon," *Archives of Orthopaedic and Trauma Surgery*, vol. 130, no. 3, pp. 363–368, 2010.

[4] B.-H. Choi, S.-J. Zhu, B.-Y. Kim, J.-Y. Huh, S.-H. Lee, and J.-H. Jung, "Effect of platelet-rich plasma (PRP) concentration on the viability and proliferation of alveolar bone cells: an in vitro study," *International Journal of Oral and Maxillofacial Surgery*, vol. 34, no. 4, pp. 420–424, 2005.

[5] L. Creaney, A. Wallace, M. Curtis, and D. Connell, "Growth factor-based therapies provide additional benefit beyond physical therapy in resistant elbow tendinopathy: a prospective, single-blind, randomised trial of autologous blood injections versus platelet-rich plasma injections," *British Journal of Sports Medicine*, vol. 45, no. 12, pp. 966–971, 2011.

[6] R. J. De Vos, A. Weir, H. T. M. Van Schie et al., "Platelet-rich plasma injection for chronic Achilles tendinopathy: a randomized controlled trial," *JAMA*, vol. 303, no. 2, pp. 144–149, 2010.

[7] M. P. Hall, P. A. Band, R. T. Meislin, L. M. Jazrawi, and D. A. Cardone, "Platelet-rich plasma: current concepts and application in sports medicine," *Journal of the American Academy of Orthopaedic Surgeons*, vol. 17, no. 10, pp. 602–608, 2009.

[8] J. Alsousou, M. Thompson, P. Harrison, K. Willett, and S. Franklin, "Effect of platelet-rich plasma on healing tissues in acute ruptured Achilles tendon: a human immunohistochemistry study," *The Lancet*, vol. 385, supplement 1, p. S19, 2015.

[9] H. Rajabi, H. Sheikhani Shahin, M. Norouzian, D. Mehrabani, and S. Dehghani Nazhvani, "The healing effects of aquatic activities and allogenic injection of Platelet-Rich Plasma (PRP) on injuries of achilles tendon in experimental rat," *World Journal of Plastic Surgery*, vol. 4, no. 1, pp. 66–73, 2015.

[10] E. Rubio-Azpeitia, P. Sánchez, D. Delgado, and I. Andia, "Three-dimensional platelet-rich plasma hydrogel model to study early tendon healing," *Cells Tissues Organs*, vol. 200, no. 6, pp. 394–404, 2015.

[11] T. Yuan, S.-C. Guo, P. Han, C.-Q. Zhang, and B.-F. Zeng, "Applications of leukocyte- and platelet-rich plasma (L-PRP) in trauma surgery," *Current Pharmaceutical Biotechnology*, vol. 13, no. 7, pp. 1173–1184, 2012.

[12] S. A. Labib, R. Rolf, R. Dacus, and W. C. Hutton, "The "Giftbox" repair of the Achilles tendon: a modification of the Krackow technique," *Foot & Ankle International*, vol. 30, no. 5, pp. 410–414, 2009.

[13] J. Leppilahti, P. Siira, H. Vanharanta, and S. Orava, "Isokinetic evaluation of calf muscle performance after Achilles rupture repair," *International Journal of Sports Medicine*, vol. 17, no. 08, pp. 619–623, 1996.

[14] A. Mishra and T. Pavelko, "Treatment of chronic elbow tendinosis with buffered platelet-rich plasma," *American Journal of Sports Medicine*, vol. 34, no. 11, pp. 1774–1778, 2006.

[15] F. Franceschi, R. Papalia, E. Franceschetti, M. Paciotti, N. Maffulli, and V. Denaro, "Platelet-rich plasma injections for chronic plantar fasciopathy: a systematic review," *British Medical Bulletin*, vol. 112, no. 1, pp. 83–95, 2014.

[16] M. Sánchez, E. Anitua, J. Azofra, I. Andía, S. Padilla, and I. Mujika, "Comparison of surgically repaired Achilles tendon tears using platelet-rich fibrin matrices," *The American Journal of Sports Medicine*, vol. 35, no. 2, pp. 245–251, 2007.

[17] A. De Carli, R. M. Lanzetti, A. Ciompi et al., "Can platelet-rich plasma have a role in Achilles tendon surgical repair?" *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 24, no. 7, pp. 2231–2237, 2016.

[18] A. F. P. Bartel, A. D. Elliott, and T. S. Roukis, "Incidence of complications after achillon® mini-open suture system for repair of acute midsubstance achilles tendon ruptures: a systematic review," *Journal of Foot and Ankle Surgery*, vol. 53, no. 6, pp. 744–746, 2014.

[19] T. Gosens, J. C. Peerbooms, W. Van Laar, and B. L. Den Oudsten, "Ongoing positive effect of platelet-rich plasma versus corticosteroid injection in lateral epicondylitis: a double-blind randomized controlled trial with 2-year follow-up," *The American Journal of Sports Medicine*, vol. 39, no. 6, pp. 1200–1208, 2011.

[20] P. Aspenberg and O. Virchenko, "Platelet concentrate injection improves Achilles tendon repair in rats," *Acta Orthopaedica Scandinavica*, vol. 75, no. 1, pp. 93–99, 2004.

[21] G. Weibrich, T. Hansen, W. Kleis, R. Buch, and W. E. Hitzler, "Effect of platelet concentration in platelet-rich plasma on peri-implant bone regeneration," *Bone*, vol. 34, no. 4, pp. 665–671, 2004.

[22] K. Gaweda, M. Tarczynska, and W. Krzyzanowski, "Treatment of Achilles tendinopathy with platelet-rich plasma," *International Journal of Sports Medicine*, vol. 31, no. 8, pp. 577–583, 2010.

[23] S. De Jonge, R. J. De Vos, A. Weir et al., "One-year follow-up of platelet-rich plasma treatment in chronic achilles tendinopathy: a double-blind randomized placebo-controlled trial," *The American Journal of Sports Medicine*, vol. 39, no. 8, pp. 1623–1629, 2011.

Hindawi Publishing Corporation
BioMed Research International
Volume 2016, Article ID 8684231, 5 pages
http://dx.doi.org/10.1155/2016/8684231

Research Article

The Effect of Sodium Hyaluronate on Ligamentation and Biomechanical Property of Tendon in Repair of Achilles Tendon Defect with Polyethylene Terephthalate Artificial Ligament: A Rabbit Tendon Repair Model

Shengkun Li, Kui Ma, Hong Li, Jia Jiang, and Shiyi Chen

Department of Sports Medicine, Huashan Hospital, 12 Wulumuqi Zhong Road, Shanghai 200040, China

Correspondence should be addressed to Hong Li; lihongdr@163.com

Received 30 June 2016; Revised 16 October 2016; Accepted 1 November 2016

Academic Editor: George Babis

The Achilles tendon is the most common ruptured tendon of human body. Reconstruction with polyethylene terephthalate (PET) artificial ligament is recommended in some serious cases. Sodium hyaluronate (HA) is beneficial for the healing of tendon injuries. We aimed to determine the effect of sodium hyaluronate in repair of Achilles tendon defect with PET artificial ligament in an animal tendon repair model. Sixteen New Zealand White rabbits were divided into two groups. Eight rabbits repaired with PET were assigned to PET group; the other eight rabbits repaired with PET along with injection of HE were assigned to HA-PET group. All rabbits were sacrificed at 4 and 8 weeks postoperatively for biomechanical and histological examination. The HA-PET group revealed higher biomechanical property compared with the PET group. Histologically, more collagen tissues grew into the HA-PET group compared with PET group. In conclusion, application of sodium hyaluronate can improve the healing of Achilles tendon reconstruction with polyethylene terephthalate artificial ligament.

1. Introduction

The incidence of Achilles tendon rupture increased during the past few years [1]. In some serious cases, direct repair is impossible. In that case, reconstruction of the tendon is required. There are many methods to treat the Achilles tendon rupture, including autograft reconstruction [2, 3], allograft reconstruction [4], and artificial ligament reconstruction [5]. However, the autograft tendon has the donor site problem, while the allograft tendon may increase the risk of disease transfusion or immunological rejection.

Artificial ligament is frequently used in the reconstruction of ligament rupture management like anterior cruciate ligament reconstruction [6], acromioclavicular joint reconstruction [7], also the Achilles tendon reconstruction [5], etc. Artificial ligaments can promise a fast functional recovery and avoid complications. In addition, there are various treatments applied for helping the healing of tendon injuries such as platelet-rich plasma (PRP) [8, 9] and sodium hyaluronate (HA) [10]. Sodium hyaluronate (HA) is polysaccharide, which is found in all extracellular matrix of vertebrates and in some bacteria as well [11]. In this way, it may be helpful to the wound healing process by generating proper environment for growth, leading to the accumulation of several matrix proteins [12].

Based on the background before, we applied the sodium hyaluronate (Bausch & Lomb Freda Co., Ltd., Shandong, China) after the repair procedure of Achilles tendon defect with PET artificial ligament. The aim of this study was to analyze the effect of sodium hyaluronate on ligamentation and biomechanical property of tendon in repair of Achilles tendon defect with PET in a rabbit tendon repair model.

2. Materials and Methods

2.1. Experimental Design. The animal experiment was approved by the Animal Care and Use Committee of our college. Two groups were involved in this study; the group

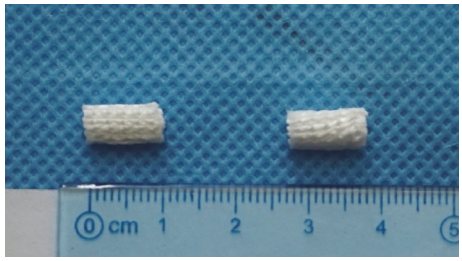


Figure 1: Optical photograph of artificial ligament graft.

treated with sodium hyaluronate (HA) was set as an experimental group, while another group untreated with sodium hyaluronate was prepared as a control group.

2.2. Animals. Total 16 fifteen-week-old male New Zealand White rabbits (mean weight 2.6 ± 0.2 kg) were randomly selected for experiments. In the study, 8 rabbits were assigned to the experimental group, while another 8 rabbits were used for the control group. Animals were sacrificed at the time points of weeks 4 ($n = 4$) and 8 ($n = 4$) after surgery for both the experimental group and the control group.

2.3. Preparation of PET Sheet. PET sheets (Figure 1) were taken from the Ligament Advanced Reinforcement System (LARS) ligament and then soaked in 75% alcohol solution to remove foreign material and contamination for 4 hours, cleaned with a large amount of deionized water and then dried in air for 24 hours [13, 14].

2.4. Operative Procedure. Anesthesia was induced by the intravenous administration of 3% pentobarbital (30 mg/kg) [13]. The animal was placed prone on the operating table. After preparation of skin and disinfection, a longitudinal skin incision was made. Then, two-thirds of vertical plane of the Achilles tendon was cut by scissors to create a partial tendon injury. To help the Achilles tendon to heal, the PET sheets were implanted into the defect to strengthen the tendon defect with 5–0 sutures in the experimental group with the injection of 2 ml sodium hyaluronate (10 mg/ml) over the surface of the tendon defect before the skin incision was closed routinely [13]. The control group was performed in the same way without the injection of sodium hyaluronate. The animals were sent back to their cages and allowed free cage activity without immobilization after surgery. All animals were given buprenorphine (0.05 mg/kg) subcutaneously for 3 days for pain control [15]. The rabbits were, respectively, sacrificed at weeks 4 and 8 after surgery to harvest tissue specimens for histological and biomechanical analysis.

2.5. Histological Analysis. Identically located longitudinal tendon samples ($n = 1$ tendon of each group at each time) were collected randomly from the injured and normal tendons [16]. After fixation in 10% formalin for 24 h, the tendon samples were washed, dehydrated, cleared, and embedded in paraffin wax. The samples were then sectioned into layers with a thickness of 5 um perpendicular to the longitudinal axis of the tendon using a microtome (SM2500, Leica) [17]. These sections were stained with hematoxylin and eosin (H&E) staining and Masson trichrome staining [18]. The slides were examined by light microscopy (Olympus Optical Co., Tokyo, Japan) for initial evaluation and photography [13, 16–18]. Two evaluators who performed the histological observation were blinded to treatment group of the sections.

2.6. Biomechanical Analysis. The repaired tendons ($n = 3$ tendons of each group at each time) were harvested after sacrifice and subjected to mechanical testing within 6 h, using a biomechanical analyzer (AGS-X 5 kN, Shimadzu Co., Japan). The dimension of the test tendon sample is approximately 4 cm. And all the samples were prepared for testing without being frozen. Both ends of the tendon were sutured by number 5 Ethibond suture for traction. Then, the sutures were fixed firmly in the biomechanical analyzer. Care was taken to keep the longitudinal tendon samples parallel to the testing axis. After preconditioning, the ultimate load-to-failure was performed with the extension rate of 5 mm/min. The load-to-failure (N) and the stiffness (N/mm) were measured, while the load-distortion curve was recorded. For each sample, testing ends when the tendon ruptured.

2.7. Statistical Analysis. Results are presented as mean and standard deviation. The paired Student t-test was used to compare the experimental group with control group to determine the significant differences. If $p < 0.05$, the differences were considered significant statistically.

3. Results

3.1. Mechanical Examination Results. No tendon rupture occurred for all specimens of both 4 and 8 weeks. The most common failure mode is the slippage of rope from the test tendon. At 4 weeks, there was no statistically significant difference in the load-to-failure between the HA-PET group and the control group (171.7 ± 38.1 N for HA-PET group and 156.7 ± 12.7 N for controls; $p > 0.05$), while at 8 weeks there was also no statistically significant difference between two groups (175.3 ± 44.7 N for HA-PET group and 137 ± 65.8 N for controls; $p > 0.05$) (Figure 2). Similarly, at 4 weeks there was no statistically significant difference in the stiffness between the HA-PET group and the control group (4.2 ± 2.4 N/mm for HA-PET group and 2.1 ± 0.9 N/mm for controls; $p > 0.05$). Meanwhile at 8 weeks there was also no statistically significant about stiffness difference between two groups (4.5 ± 3.8 N/mm for HA-PET group and 2.1 ± 0.3 N/mm for controls; $p > 0.05$) (Figure 3).

3.2. Histological Results. Four weeks after surgery it appeared that there was no tissue surrounding the graft fibers in both the PET and HA-PET groups. After 8 weeks, thick collagen tissue with some vasculature covered the grafts in the HA-PET group, while there was little tissue infiltration of the graft fibers in the PET group (Figures 4 and 5). Some cells from the fibrous tissue infiltrated the graft site, and the collagen fibers tended to orient along the axis of the tendon.

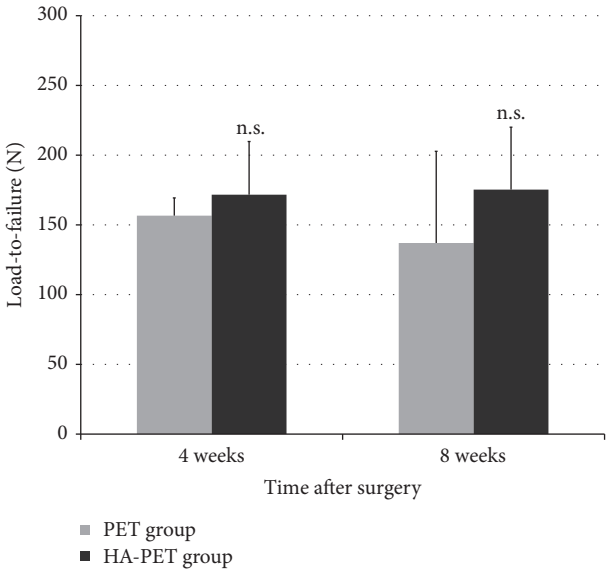


Figure 2: Comparison of maximal failure load for Achilles tendon healing in a rabbit model of the PET group and HA-PET group at each time after surgery.

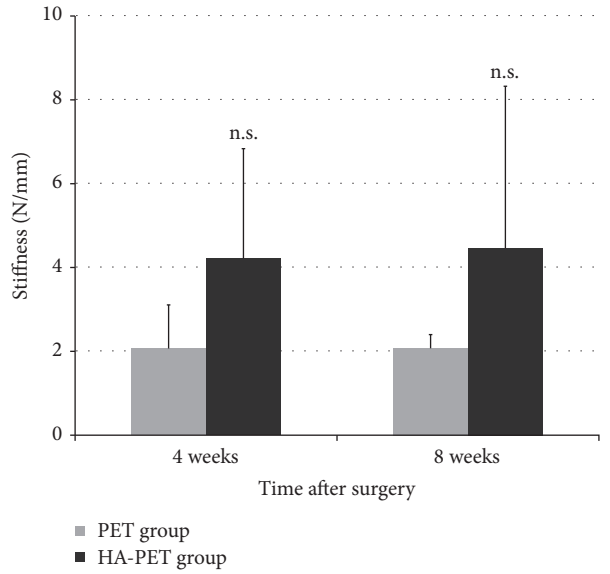


Figure 3: Comparison of stiffness at failure for Achilles tendon healing in a rabbit model of the PET group and HA-PET group at each time after surgery (n.s. indicates there is no significant difference between groups).

4. Discussion

Achilles tendon is the most common ruptured tendon of our human body [19]. Reconstruction procedure must be taken using allograft, autograft, or artificial ligament for some serious cases if direct repair is impossible. Artificial ligaments like LARS can provide enough intensity, while it is not fully bioactive for ligamentization. Additional treatment like sodium hyaluronate (HA) can facilitate the process

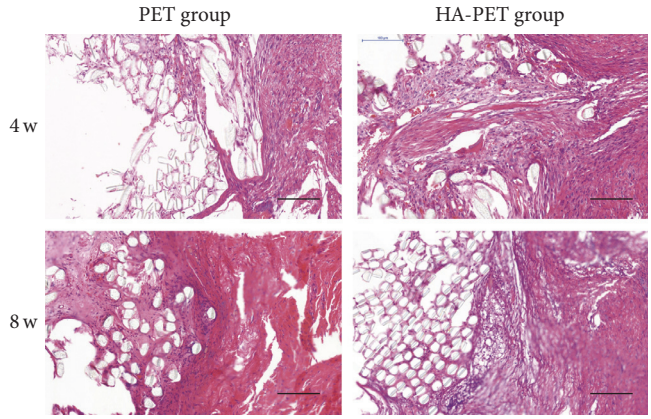
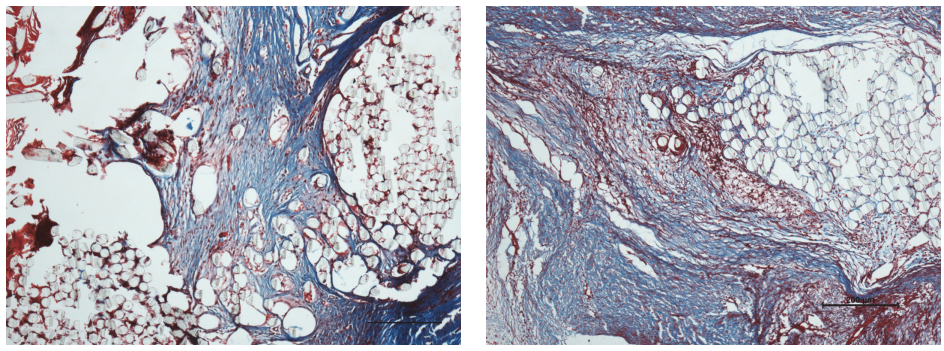


Figure 4: Histological images (HE staining) of the PET group and the HA-PET group at 4 weeks and 8 weeks after surgery. Bar = 100 μm.

of graft healing including inflammation, cellular adhesion and proliferation, vascular proliferation and pruning, and collagen remodeling [20]. In this study, we are trying to find out the effect of sodium hyaluronate for Achilles tendon reconstruction using PET.

Sodium hyaluronate partly contributes to cell adhesion and growth as well as inflammation suppressing. In a previous study, Li et al. found out that polyethylene terephthalate (PET) artificial ligament grafts coated with sodium hyaluronate can enhance cell adhesion, facilitated cell growth, and suppressed the expression of inflammation-related genes relative to a pure PET graft for ACL reconstruction [21]. Another study done by Nakamura et al. demonstrated that HA possesses anti-inflammatory and antiadhesive activities in tendon and synovial fibroblasts derived from RCT [22]. In addition, Salamanna et al. find out that, after repeated peripatellar injections of HA to rats, a significantly higher proliferation rate and viability, along with increased synthesis of C-terminal-propeptide of type I collagen, fibronectin, aggrecan, tenascin-c, and matrix-metalloproteinase-3 show respect to control group [23]. In this study, the administration of HA improved collagen formation which is good for tendon healing. The molecular weight and the concentration of NaHA influence the rate of the elimination process from the tendon sheath after local injection [24]. There are investigations suggesting that the injection of high-molecular-weight NaHA in high concentrations around the tendons improves tendon healing and decreases adhesion formation [24–26]. In our study, the weight-average molecular weight of NaHA is within $6 \times 10^5 \sim 1.5 \times 10^6$ which improves tendon healing to a certain extent. There are several different explanations on the effects of NaHA. Some researchers find out that NaHA acts as a physical barrier around the repaired tendon location, while others suggest that the main effect of NaHA may be developed pharmacologically or physiologically [24–26]. However, the exact mechanism of effect of NaHA is not clear.

The histological results especially the Masson staining in this study show that sodium hyaluronate can facilitate new collagen formation. Li et al. suggest that PET graft



(a) (b)

Figure 5: Histological images (Masson staining) of the PET group and the HA-PET group at 8 weeks after surgery. Bar = 200 μm.

coated with HA can enhance not only cell adhesion and proliferation but also stimulating collagen generation [21]. Oryan et al. demonstrated that, after surgical treatment of tendon rupture, application of sodium hyaluronate to the lesions can improve the density of collagen fibers, almost similar to the normal contra-lateral tendons. Ultrastructural morphometric analyses of the number, diameter (nm), and density (%) of the collagen fibrils show significant differences with control group on both the number and the diameter of collagen fibrils [27]. In another study Halici et al. suggested that, after 6 weeks of repetitive administration of HA during the rabbit tendon healing process, type IV collagen expression increased significantly, and they believe it is part of the angiogenesis along with the increase of VEGF [28].

In our experiment, the mean load-to-failure and stiffness of HA-PET group appeared higher than those of the PET group. However, there are no significant differences in the mechanical examination results both on the 4 weeks' and 8 weeks' terminal point. Deng et al. [13] investigated the feasibility of using adipose derived stem cells (ASCs) for engineered tendon repair in vivo in a rabbit Achilles tendon model, and they found no difference in tensile strength at 5 weeks but found significant difference at the time points of 12 weeks, 21 weeks, and 45 weeks. Some other studies also show treatment of HA strongly improved the biomechanical properties of the injured tendons and the ultimate strength [10, 27]. One conceivable possibility is that, unlike with the normal Achilles tendon, PET artificial ligament has much better characteristic of strength and stiffness, and injection of HA may not add significant improvement of biomechanical property to PET artificial ligament. Only 16 rabbits were investigated in our study, which may be somehow insufficient on sample size.

5. Conclusion

Collectively, the histological results of our study demonstrate that application of sodium hyaluronate can improve collagen formation, which is beneficial for the healing of Achilles tendon reconstruction with polyethylene terephthalate artificial ligament.

Competing Interests

The authors report that they have no conflict of interests.

Authors' Contributions

Shengkun Li and Kui Ma contributed equally to this work.

Acknowledgments

This project was subsidized by the Natural Science Foundation of China (81401812).

References

[1] V. Gulati, M. Jaggard, S. S. Al-Nammari et al., "Management of achilles tendon injury: a current concepts systematic review," *World Journal of Orthopaedics*, vol. 6, no. 4, pp. 380–386, 2015.

[2] R. Arriaza, R. Gayoso, E. López-Vidriero, J. Aizpurúa, and C. Agrasar, "Quadriceps autograft to treat Achilles Chronic tears: a simple surgical technique," *BMC Musculoskeletal Disorders*, vol. 17, article 116, 2016.

[3] K. Nilsson-Helander, L. Swärd, K. G. Silbernagel, R. Thomeé, B. I. Eriksson, and J. Karlsson, "A new surgical method to treat chronic ruptures and reruptures of the Achilles tendon," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 16, no. 6, pp. 614–620, 2008.

[4] J. M. Deese, G. Gratto-Cox, F. D. Clements, and K. Brown, "Achilles allograft reconstruction for chronic achilles tendinopathy," *Journal of Surgical Orthopaedic Advances*, vol. 24, no. 1, pp. 75–78, 2015.

[5] M. S. Dhillon, D. Chauhan, V. Kumar, and U. C. Saini, "Reconstruction of bilateral spontaneous chronic idiopathic Achilles tendon rupture using LARS ligament: case report," *Foot*, vol. 20, no. 4, pp. 154–157, 2010.

[6] C. Legnani, A. Ventura, C. Terzaghi, E. Borgo, and W. Albisetti, "Anterior cruciate ligament reconstruction with synthetic grafts. A review of literature," *International Orthopaedics*, vol. 34, no. 4, pp. 465–471, 2010.

[7] G. M. Marcheggiani Muccioli, C. Manning, P. Wright, A. Grassi, S. Zaffagnini, and L. Funk, "Acromioclavicular joint reconstruction with the LARS ligament in professional versus non-professional athletes," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 24, no. 6, pp. 1961–1967, 2016.

[8] E. Circi, Y. E. Akman, E. Sukur, E. R. Bozkurt, T. Tuzuner, and Y. Ozturkmen, "Impact of platelet-rich plasma injection timing on healing of Achilles tendon injury in a rat model," *Acta Orthopaedica et Traumatologica Turcica*, vol. 50, pp. 366–372, 2016.

[9] M. Sánchez, E. Anitua, J. Azofra, I. Andía, S. Padilla, and I. Mujika, "Comparison of surgically repaired Achilles tendon tears using platelet-rich fibrin matrices," *American Journal of Sports Medicine*, vol. 35, no. 2, pp. 245–251, 2007.

[10] H. B. Tosun, S. A. Gumustas, M. Kom, A. Uludag, S. Serbest, and Y. Eroksuz, "The effect of sodium hyaluronate plus sodium chondroitin sulfate solution on peritendinous adhesion and tendon healing: an experimental study," *Balkan Medical Journal*, vol. 33, no. 3, pp. 258–266, 2016.

[11] J. R. E. Fraser, T. C. Laurent, and U. B. G. Laurent, "Hyaluronan: its nature, distribution, functions and turnover," *Journal of Internal Medicine*, vol. 242, no. 1, pp. 27–33, 1997.

[12] K. R. Kirker, Y. Luo, J. H. Nielson, J. Shelby, and G. D. Prestwich, "Glycosaminoglycan hydrogel films as bio-interactive dressings for wound healing," *Biomaterials*, vol. 23, no. 17, pp. 3661–3671, 2002.

[13] D. Deng, W. Wang, B. Wang et al., "Repair of Achilles tendon defect with autologous ASCs engineered tendon in a rabbit model," *Biomaterials*, vol. 35, no. 31, pp. 8801–8809, 2014.

[14] H. Li, Y. Ge, Y. Wu et al., "Hydroxyapatite coating enhances polyethylene terephthalate artificial ligament graft osseointegration in the bone tunnel," *International Orthopaedics*, vol. 35, no. 10, pp. 1561–1567, 2011.

[15] L. V. Gulotta, D. Kovacevic, L. Ying, J. R. Ehteshami, S. Montgomery, and S. A. Rodeo, "Augmentation of tendon-to-bone healing with a magnesium-based bone adhesive," *American Journal of Sports Medicine*, vol. 36, no. 7, pp. 1290–1297, 2008.

[16] A. Oryan, A. Moshiri, and A. R. Raayat, "Novel application of theranekron® enhanced the structural and functional performance of the tenotomized tendon in rabbits," *Cells Tissues Organs*, vol. 196, no. 5, pp. 442–455, 2012.

[17] H. Li, Y. Ge, P. Zhang, L. Wu, and S. Chen, "The effect of layer-by-layer chitosan-hyaluronic acid coating on graft-to-bone healing of a poly(Ethylene Terephthalate) artificial ligament," *Journal of Biomaterials Science, Polymer Edition*, vol. 23, no. 1–4, pp. 425–438, 2012.

[18] J. Buschmann, A. Müller, F. Nicholls et al., "2D motion analysis of rabbits after Achilles tendon rupture repair and histological analysis of extracted tendons: can the number of animals be reduced by operating both hind legs simultaneously?" *Injury*, vol. 44, no. 10, pp. 1302–1308, 2013.

[19] N. Maffulli, S. W. Waterston, J. Squair, J. Reaper, and A. S. Douglas, "Changing incidence of Achilles tendon rupture in Scotland: A 15-Year Study," *Clinical Journal of Sport Medicine*, vol. 9, no. 3, pp. 157–160, 1999.

[20] S. M. Joshi, A. N. Mastrangelo, E. M. Magarian, B. C. Fleming, and M. M. Murray, "Collagen-platelet composite enhances biomechanical and histologic healing of the porcine anterior cruciate ligament," *American Journal of Sports Medicine*, vol. 37, no. 12, pp. 2401–2410, 2009.

[21] H. Li, J. Jiang, Y. Ge et al., "Layer-by-layer hyaluronic acid-chitosan coating promoted new collagen ingrowth into a poly(ethylene terephthalate) artificial ligament in a rabbit medical collateral ligament (MCL) reconstruction model," *Journal of Biomaterials Science, Polymer Edition*, vol. 24, no. 4, pp. 431–446, 2013.

[22] H. Nakamura, M. Gotoh, T. Kanazawa et al., "Effects of corticosteroids and hyaluronic acid on torn rotator cuff tendons in vitro and in rats," *Journal of Orthopaedic Research*, vol. 33, no. 10, pp. 1523–1530, 2015.

[23] F. Salamanna, A. Frizziero, S. Pagani et al., "Metabolic and cytoprotective effects of in vivo peri-patellar hyaluronic acid injections in cultured tenocytes," *Connective Tissue Research*, vol. 56, no. 1, pp. 35–43, 2014.

[24] L. Hagberg and B. Gerdin, "Sodium hyaluronate as an adjunct in adhesion prevention after flexor tendon surgery in rabbits," *Journal of Hand Surgery*, vol. 17, no. 5, pp. 935–941, 1992.

[25] S. Işik, S. Öztürk, S. Gürses et al., "Prevention of restrictive adhesions in primary tendon repair by HA-membrane: experimental research in chickens," *British Journal of Plastic Surgery*, vol. 52, no. 5, pp. 373–379, 1999.

[26] J. A. Miller, R. L. Ferguson, D. L. Powers, J. W. Burns, and S. W. Shalaby, "Efficacy of hyaluronic acid/nonsteroidal anti-inflammatory drug systems in preventing postsurgical tendon adhesions," *Journal of Biomedical Materials Research*, vol. 38, no. 1, pp. 25–33, 1997.

[27] A. Oryan, A. Moshiri, and A.-H. Meimandiparizi, "Effects of sodium-hyaluronate and glucosamine-chondroitin sulfate on remodeling stage of tenotomized superficial digital flexor tendon in rabbits: a clinical, histopathological, ultrastructural, and biomechanical study," *Connective Tissue Research*, vol. 52, no. 4, pp. 329–339, 2011.

[28] M. Halici, S. Karaoglu, O. Canoz, S. Kabak, and A. Baktir, "Sodium hyaluronate regulating angiogenesis during Achilles tendon healing," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 12, no. 6, pp. 562–567, 2004.

Hindawi Publishing Corporation
BioMed Research International
Volume 2016, Article ID 1895948, 7 pages
http://dx.doi.org/10.1155/2016/1895948

Clinical Study

The Double-Row Suture Technique: A Better Option for the Treatment of Haglund Syndrome

Yiqiu Jiang,[1] Yang Li,[2] Tianqi Tao,[2] Wang Li,[2] Kaibin Zhang,[2] Jianchao Gui,[2] and Yong Ma[1]

[1]*Nanjing University of Chinese Medicine, Nanjing, China*
[2]*Department of Orthopedics, Nanjing First Hospital, Nanjing Medical University, Nanjing, China*

Correspondence should be addressed to Jianchao Gui; gui1997@126.com and Yong Ma; zhongyi-my@263.net

Received 29 June 2016; Accepted 26 October 2016

Academic Editor: Ying-Hui Hua

Purpose. The purpose of this study is to investigate whether double-row suture technique is a better option for the treatment of Haglund syndrome than single-row suture technique regarding the surgical outcomes. *Methods*. Thirty-two patients with Haglund syndrome were recruited in this study. Patients were divided into Group 1 (treated with single-row suture technique) and Group 2 (treated with double-row suture technique). There were 16 patients in each group. The AOFAS-ankle-hindfoot scale, VISA-A scores, and Arner-Lindholm standard were used to assess the clinical outcomes. The pre- and postoperative X-rays were used to assess the radiological outcome. *Results*. Both AOFAS-ankle-hindfoot scale score and VISA-A score had varying degrees of improvement in both groups. In latest follow-up assessment, the Arner-Lindholm standard investigation showed there were 7 excellent, 7 good, and 2 bad outcomes in Group 1 and 12 excellent and 4 good outcomes in Group 2. In Group 2 patients, there were no more posterosuperior bony prominence of the calcaneum in post-op X-rays and there were no recurrent cases. The ankle-related scale score was statistically significantly higher in Group 2 than in Group 1 ($P = 0.029$). *Conclusion*. The double-row suture technique seems to be a better option to treat Haglund syndrome than single-row suture technique.

1. Introduction

Posterior heel pain is a common presentation in outpatient clinics and there are many different causes [1]. In 1928, Swedish orthopedic surgeon Haglund firstly described a posterior heel pain caused by a prominent posterosuperior corner of the calcaneus in combination with wearing a rigid low-back shoe [2]. This report made people realize such special posterior heel disease, which we called Haglund syndrome.

Actually, Haglund syndrome is an enlargement of the posterosuperior prominence of the calcaneus, which is frequently associated with insertional Achilles tendinitis, bursal projection, and Achilles bursitis [3–5]. Haglund syndrome can cause mechanical impingement to the retrocalcaneal bursa and Achilles tendon. Patients with the syndrome will present with posterior heel pain and pain on passive ankle motion. Haglund syndrome can also induce inflammation and the degeneration of the Achilles tendon [6, 7], because of the abnormal high pressure between the bursal projection of calcaneus, the Achilles tendon, and the bursal impingement of the Achilles. If there is concomitant immediate reverse tension, Haglund syndrome may even cause acute Achilles tendon rupture. Unfortunately its distinct pathogenesis is still unknown. Haglund syndrome is also called "pump bump" disease because the rigid back of pump-style shoes could create pressure that stimulate the enlarged prominence during walking [8].

Haglund syndrome can be treated conservatively or surgically. Conservative treatment included the avoidance of rigid heel counter shoes, use of heel cushions, softer uppers or pads for elevation of the heel, activity modification, or local block treatment. Medication included nonsteroidal anti-inflammatory drugs or corticosteroid injection into retrocalcaneal bursa are also recommended for acute cases. However direct intratendinous steroid injections might weaken the

Table 1: The detailed information of the 32 patients.

Group	Gender		Average age	Right or left		Mean follow-up duration
	Male	Female		Right	Left	
Group 1	06	10	50.6 ± 3 years (range, 21 to 59 years)	8 (50.0%)	8 (50.0%)	3.5 ± 0.8 years (range, 24 to 60 months)
Group 2	05	11	52.1 ± 2 years (range, 33 to 68 years)	9 (56.3%)	7 (43.7%)	3.5 ± 0.5 years (range, 24 to 60 months)

Group 1: the patients had traditional single-row suture method.
Group 2: the patients had double-row suture technique.

tendon and cause tendon rupture [9]. For many cases, the effectiveness of the conservative management is low; moreover there is higher recurrent rate in conservative treatment group [10, 11]; those patients who fail conservative treatment of more than 6 months are indicated for surgical treatment [12–14].

Before 2010, a traditional single-row suture method was used to treat Haglund syndrome, by which 50–70% of the Achilles insertion was detached without compromising the tendon. After excision the calcified and the inflamed tendon, a suture anchor was used to reattach and repair the Achilles tendon [15]. However, the results of this operation were not always satisfactory. Its recurrence rate, the Achilles tendon instability, and residual heel pain limited its use and popularity.

Recently, we treated the Haglund syndrome with the double-row suture technique and obtained good clinical results. In this study, the clinical results, the safety, and efficacy of this procedure were analyzed to evaluate whether this method could give a better long-term result than single-row suture technique.

2. Materials and Methods

2.1. Patient Population. Thirty-two patients with Haglund syndrome from February 2008 to February 2014 were retrospectively reviewed; all MRI showed the posterosuperior calcaneal prominence or Achilles tendinitis. The detailed information of all 32 patients could refer to Table 1.

Patients were selected according to the following criteria:

(1) Diagnosed as Haglund syndrome.

(2) All treated surgically (either the single-row or double-row suture technique).

(3) Follow-up more than 24 months.

(4) Having both preoperative and postoperative X-rays and preoperative MRI done.

Exclusion criteria included the following:

(1) Old injuries.

(2) Patients with any kinds of inflammatory arthritis (such as rheumatoid arthritis).

(3) Fracture or other concomitant disorders in the foot and ankle area.

(4) Patients who had other comorbidities such as diabetes, severe heart disease, morbid obesity, or peripheral vascular disease who were also excluded to avoid severe surgical complications.

2.2. Surgical Procedure. During the surgical procedure, patient was in prone position with a thigh tourniquet. A longitudinal skin incision lateral to Achilles tendon was made. During the surgical procedure, we found most of the patients had degenerative change and inflammation with calcification scattered in their Achilles tendons.

For Group 1, in order to ensure the continuity of Achilles tendon, only 50–70% of the Achilles insertion was detached by sharp-pointed knife. After excision of the bony prominence, the degeneration tissue, scar tissue, calcified and inflammatory tissue in the field of vision, and the detached portion of the Achilles tendon was reattached to the newly created cancellous surface of the calcaneus using one suture anchor. Two sutures connected to the anchor screw were tied with equal tension. The skin was closed with 3-0 nonabsorbable suture. In this operation, the split tendon healed in the form of point-to-point (Figure 1).

For Group 2, Achilles insertion was completely detached from the insertion site. After excising the whole bony prominence and the diseased tendon, the first suture anchor was inserted in the proximal calcaneal insertion. Krackow suture technique was used to suture the detached Achilles tendon with the 4 stitches (Knot 1). The next step was to assess the size of the posterosuperior calcaneal prominence and assess whether there was any impingement syndrome by the impaction test. Osteotome was then used to resect the posterosuperior calcaneal prominence. The second suture anchor was inserted in the distal point of calcaneum resection surface (Knot 2). The stitches passed through the terminal part of the Achilles tendon and were tied with the first 4 stitches by the double-row suture technique (Knot 3) (Figure 2). After that, the skin was closed with 3-0 nonabsorbable suture. For all Group 2 patients, no one needed flexor hallucis longus tendon transfer nor proximal V-Y advancement of the gastrocnemius fascia.

2.3. Postoperative Management. All patients were put on a short leg plaster cast with ankle in equinus position for 6 weeks immobilization. They were instructed on non-weight-bearing walking for 6 weeks, before full weight bearing walking was allowed. Passive dorsiflexion and active resistive plantar flexion ankle exercises were started at 6 weeks after surgery. Usually at 3 months' time, patients could participate in normal daily activities.

2.4. Evaluation Methods. The American Orthopaedic Foot and Ankle Society (AOFAS) ankle-hindfoot scale, the Victorian Institute of Sport Assessment-Achilles (VISA-A) scores,

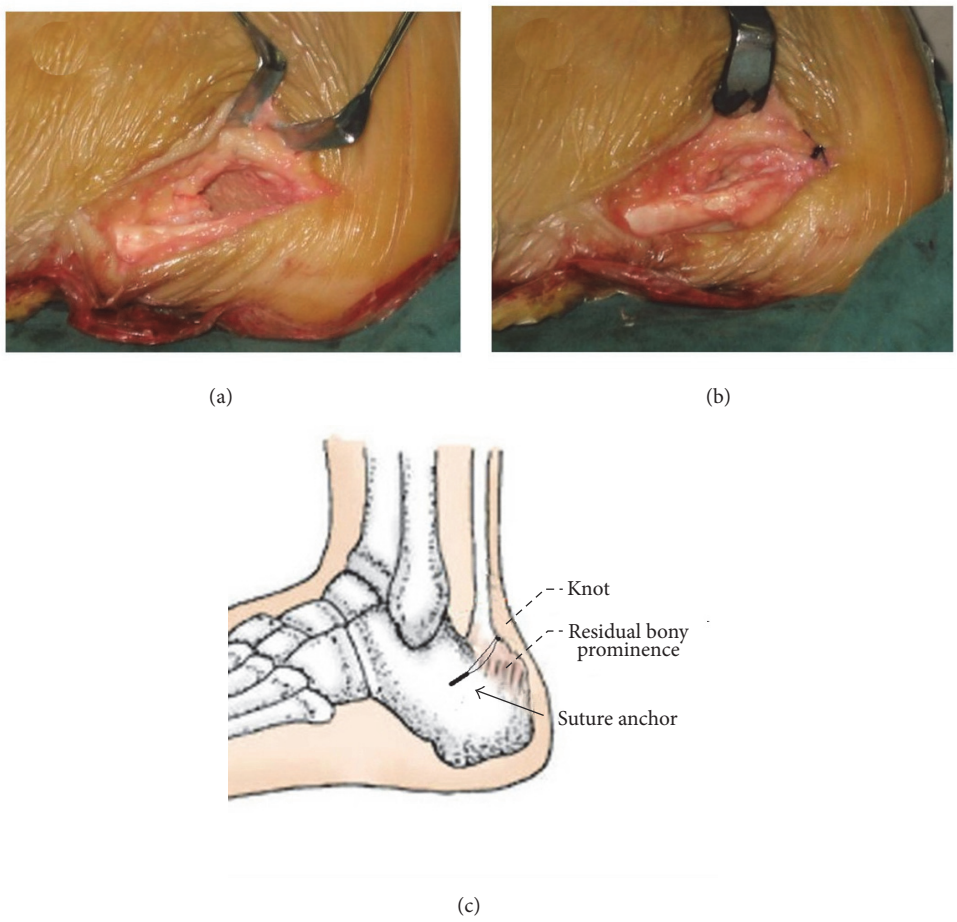


Figure 1: (a) 50–70% of the Achilles insertion was detached without compromising the tendon. The calcified lesions were excised. (b) A suture anchor was inserted to reattach and repair of Achilles tendon. (c) The diagram of single-row suture technique.

and Arner-Lindholm standard were used to evaluate the surgical outcomes of the patients. The preoperative and postoperative radiological features of calcaneal shapes were also assessed on the standing lateral foot X-ray.

2.5. Statistical Analysis. The SPSS software (version 18.0) was used for statistical analyses. The independent sample t-test was used for comparison of the preoperative and postoperative data. The Wilcoxon signed-rank test was used to compare the ankle-related scale score varieties between two groups. The statistical significance was set at P value < 0.05.

3. Results

All 32 patients in both groups achieved primary healing without anchor loosening, displacement, or rupture of the Achilles tendon. In Group 1, two patients had recurrent symptoms and five patients had mild residual posterior heel pain; those residual symptoms decreased patients' satisfaction. In Group 2, there were no recurrent cases. 15 patients regained normal range of motion of the ankle joint at 12 weeks and resume low impact sports at 6 mouths without posterior heel pain. One patient had delayed recovery up to one year because of the relative low threshold to pain and inadequate rehabilitation exercise. In Group 2, all patients eventually achieved satisfactory results.

The mean AOFAS ankle-hindfoot scale score, the VISA-A score, and the Arner-Lindholm standard could be refer to in Table 2. The ankle-related scale score varieties were statistically significant higher in Group 2 than in Group 1 ($P = 0.029$).

Radiologically, there was no posterosuperior bony prominence in the calcaneus in Group 2. And there was no impingement syndrome in all patients. The preoperative and postoperative comparison of the X-ray film could refer be to in Figures 3 and 4.

4. Discussion

Haglund syndrome, firstly described by Swedish orthopedic surgeon Haglund in 1928 [2], is the general description of syndrome which included posterosuperior calcaneal bony prominence, insertional Achilles tendinitis, bursal projection, and Achilles bursitis [4, 5]. From the lateral foot

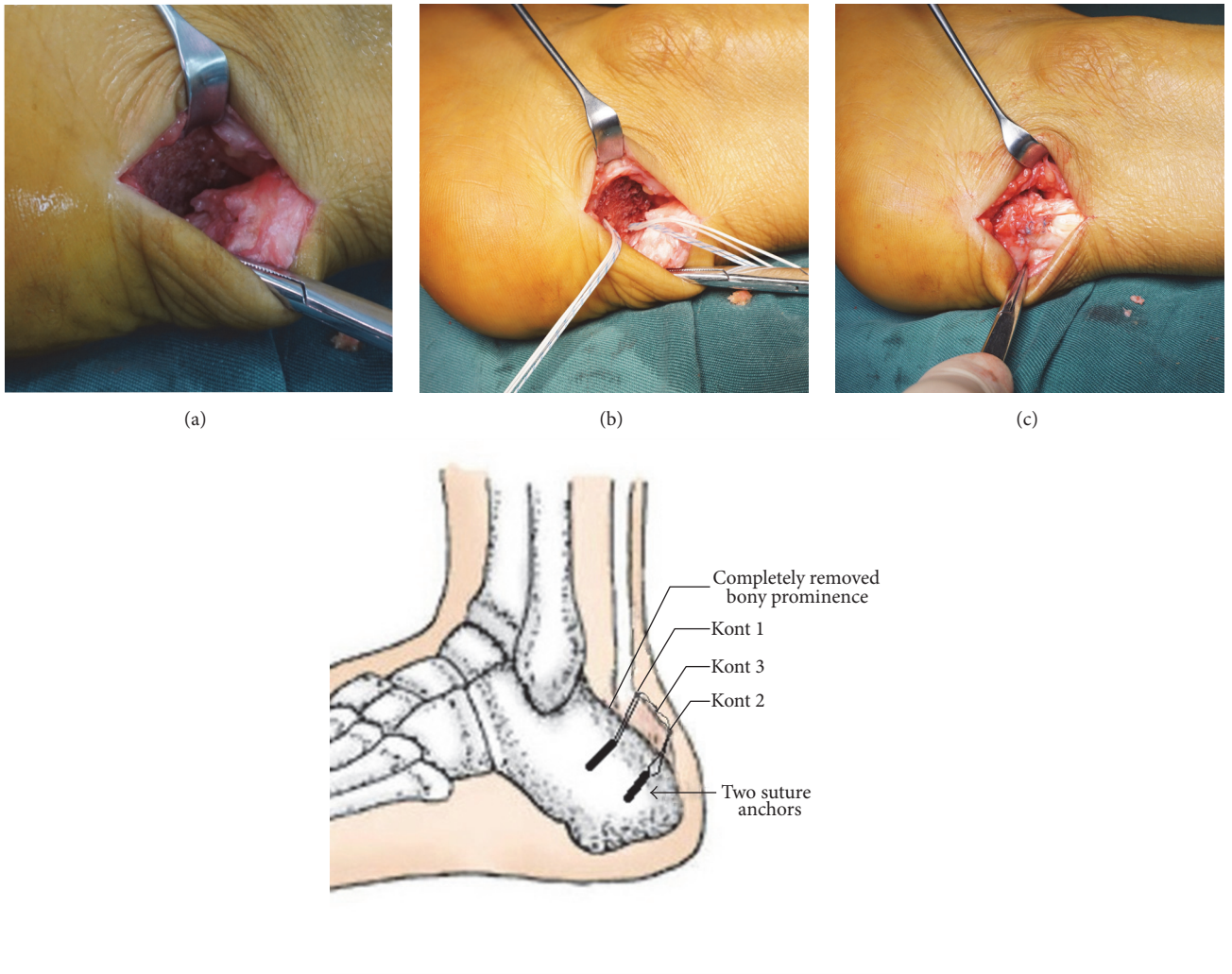


Figure 2: (a) Achilles tendon insertion was completely detached. The calcified tendon was completely excised. (b) Two suture anchors were inserted to reattach and repair the Achilles tendon. (c) The tendon was repaired with the double-row suture technique. (d) The diagram of double-row suture technique.

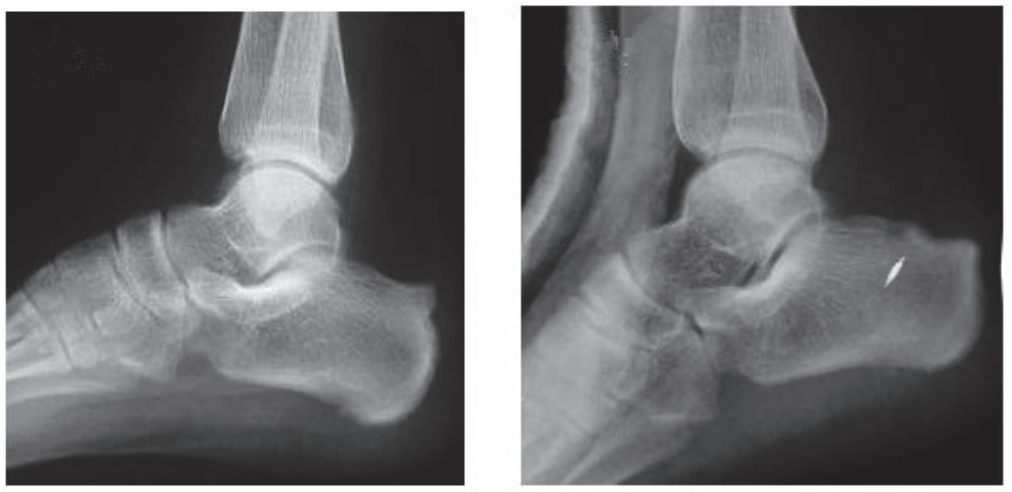
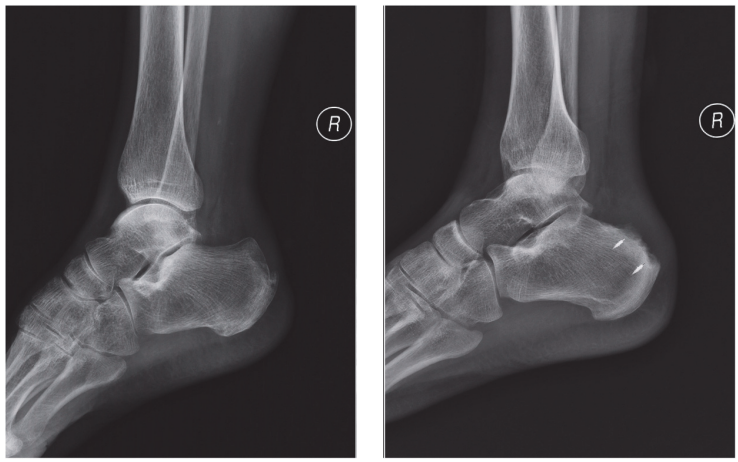


Figure 3: The standing lateral foot X-ray preoperatively (a) and postoperatively (b) showed the calcaneal prominence was excised.

Table 2: Comparison of functional scores pre- and postoperatively in 2 groups ($N = 16$ patients in each group).

	Scale	Preoperative score	Latest follow-up score	P value*
Group 1	AOFAS ankle-hindfoot scale score	56.1 ± 4.1	81.3 ± 6.5	0.0441
	VISA-A score	52.6 ± 5.2	84.1 ± 3.9	0.0408
	The Arner-Lindholm standard		7 excellent, 7 good, 2 bad	
	Recurrence rate		2 (12.5%)	
	Residual heel pain		5 (31.3%)	
Group 2	AOFAS ankle-hindfoot scale score	59.2 ± 6.7	91.1 ± 4.2	0.0228
	VISA-A score	50.6 ± 3.2	90.6 ± 3.4	0.0158
	The Arner-Lindholm standard		11 excellent, 5 good, 0 bad	
	Recurrence rate		0	
	Residual heel pain		0	

AOFAS: American Orthopaedic Foot and Ankle Society.
VISA-A: Victorian Institute of Sport Assessment-Achilles.
Data presented as mean ± standard deviation.
Group 1: the patients had traditional single-row suture technique.
Group 2: the patients had double-row suture technique.
*Independent sample t-test.



(a) (b)

Figure 4: The standing lateral foot X-ray preoperatively and postoperatively showed complete excision of posterosuperior calcaneal prominence. (a) Preoperative X-ray showed obvious posterosuperior calcaneal prominence. (b) Postoperative X-ray showed the location of two suture anchors and the complete excision posterosuperior calcaneal prominence.

radiograph, the prominent calcaneal bursal projection, retrocalcaneal bursitis, and thickening of the Achilles tendon could all be seen [16]. This disease had been postulated to cause posterior heel pain resulting from mechanical impingement of the retrocalcaneal bursa [17]. Meanwhile, insertional Achilles tendinitis was regarded as an overuse phenomenon resulting in inflammation and accelerating the degeneration of the Achilles tendon at insertion site [18]. The combination of such pathologies wound seriously affected patients' walking. In worse situation the Achilles tendon might rupture with a mild trauma event.

Patients with Haglund syndrome varies in age from young to elderly, and it is more commonly seen in women [19]. The exact pathogenesis is still unknown. The possible causes include inheritance factor, injuries related to the sport, inappropriate shoe wear, and sequelae of calcaneal fractures [20]. It is usually diagnosed clinically and radiologically. The standing lateral foot radiograph is useful to assess the presence of the posterosuperior calcaneal bony prominence (Haglund deformity) [16, 21]. MRI has superior soft tissue and bone marrow signal sensitivity, which facilitate it to make diagnosis of Haglund syndrome [22], especially for those ambiguous or clinically equivocal cases.

To date, the management of Haglund syndrome included conservative and surgical treatments [23]. Conservative treatment included the avoidance of rigid heel counter shoes, use of heel cushions, softer uppers or pads for elevation of the heel, activity modification, or local block treatment. Medications including nonsteroidal anti-inflammatory drugs or corticosteroid injection into retrocalcaneal bursa are also recommended for acute cases. Although the bursitis could be controlled by these methods, the posterosuperior calcaneal

bony prominence could not be removed. Such mechanical impingement causes persistent heel pain. It is controversial whether the inflammation in the bursa or tendon could be relieved by local block treatment, as the persistent inflammation in the bursa or tendon could lead to rupture of the Achilles tendon [9].

If conservative treatment failed, surgical intervention should be recommended [24–26]. It has been believed that traditional one suture anchor with single-row suture technique, by which 50–70% of the Achilles insertion was detached from the insertion, repaired, and reattached after the procedure, can improve the heel pain. However, concerning such method, inadequate bone resection could lead to recurrence of heel pain. Moreover, point-to-point tendon healing may not restore the full strength and the stability of the Achilles tendon, which may weaken it or even cause rupture of the Achilles tendon.

In view of such disease, we conclude that the treatment principles should include excision of the Haglund deformity, relieving the mechanical impingement, and restoring the continuity of Achilles tendon [13]. Comparing these principles with the rotator cuff repair technique [27], we derived a concept of double-row suture technique, which obtained good clinical outcomes and high patients' satisfaction. During the operation, the first step was to excise the degenerative and scar tissue, thorough debridement of the calcified and the inflamed tendon. Secondly, two suture anchors were inserted in the proximal and distal point of Haglund deformity bone resection surface. The detached ends of Achilles tendon were repaired by the sliding suture of the anchor. Thirdly, through the double-row suture technique the sutures on each anchor were tied over with the another. With such repair method, it provided larger contact area between tendon and bone surface and promotes the healing of the Achilles tendon.

In this study, we obtain long-term satisfactory outcomes in an average follow-up period of 3.5 years. Compared to the single-row suture group, all patients obtained primary healing. There are no significant complications, such as spur recurrence or residual heel pain. An analysis of the various ankle-related scores consists of the American Orthopedic Foot and Ankle Society (AOFAS) ankle-hindfoot scale [28], Victorian Institute of Sport Assessment-Achilles (VISA-A) scores [29], and Arner-Lindholm standard [30] which were also conducted to evaluate the clinical effect. All these scores obtained satisfactory results. The AOFAS ankle-hindfoot scale score improved from 59.2 ± 6.7 preoperatively to 91.1 ± 4.2 at the latest follow-up visit, and the VISA-A score improved from 50.6 ± 3.2 to 90.6 ± 3.4. The Arner-Lindholm standard investigation at the latest follow-up visit showed 11 excellent, 5 good, and no bad outcomes. Postoperative X-rays showed complete excision of the Haglund deformity. Whilst in the single-row suture group, the Arner-Lindholm standard investigation at the latest follow-up visit showed 7 excellent, 7 good, and 2 bad outcomes. Two of the patients had recurrence and five patients had residual posterior heel pain.

Using our technique, we can overcome the previous complications. The complete excision of posterosuperior calcaneal bony prominence (Haglund deformity) can effectively relieve the heel pain and prevent the recurrence. The larger contact surface between tendon and bone will facilitate tendon healing and stability. The shorter period of immobilization (plaster cast after surgery) allowed early functional exercise and reduced the joint stiffness. Early activity also maintains gastrocnemius muscle capacity and minimizes the plantar flexor muscle strength deficit. Therefore, the double-row suture technique can improve clinical outcome of Haglund syndrome.

5. Conclusion

For those patients with the Haglund syndrome, the double-row suture technique could be a better option for its satisfactory surgical outcomes than traditional single-row suture technique.

Competing Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

Authors' Contributions

Yiqiu Jiang and Yang Li contributed equally to this work.

References

[1] J. E. Martin, J. C. Hosch, W. P. Goforth, R. T. Murff, D. M. Lynch, and R. D. Odom, "Mechanical treatment of plantar fasciitis. A prospective study," *Journal of the American Podiatric Medical Association*, vol. 91, no. 2, pp. 55–62, 2001.

[2] P. Haglund, "Beitrag zur Klinik der Achillessehne," *Zeitschrift für Orthopädie und Unfallchirurgie*, vol. 49, pp. 49–58, 1928.

[3] F. Rossi, F. La Cava, F. Amato, and G. Pincelli, "The Haglund syndrome (H.s.): clinical and radiological features and sports medicine aspects," *Journal of Sports Medicine and Physical Fitness*, vol. 27, no. 2, pp. 258–265, 1987.

[4] A. A. Schepsis and R. E. Leach, "Surgical management of Achilles tendinitis," *The American Journal of Sports Medicine*, vol. 15, no. 4, pp. 308–315, 1987.

[5] S. Kang, D. B. Thordarson, and T. P. Charlton, "Insertional Achilles tendinitis and Haglund's deformity," *Foot and Ankle International*, vol. 33, no. 6, pp. 487–491, 2012.

[6] F. W. Ortmann and A. M. McBryde, "Endoscopic bony and soft-tissue decompression of the retrocalcaneal space for the treatment of Haglund deformity and retrocalcaneal bursitis," *Foot and Ankle International*, vol. 28, no. 2, pp. 149–153, 2007.

[7] M. R. Clain and D. E. Baxter, "Achilles tendinitis," *Foot and Ankle*, vol. 13, no. 8, pp. 482–487, 1992.

[8] E. H. Y. Hung, W. K. Kwok, and M. M. P. Tong, "Haglund syndrome—a characteristic cause of posterior heel pain," *Journal of the Hong Kong College of Radiologists*, vol. 11, no. 4, pp. 183–185, 2009.

[9] J. C. Kennedy and R. B. Willis, "The effects of local steroid injections on tendons: a biomechanical and microscopic correlative study," *The American Journal of Sports Medicine*, vol. 4, no. 1, pp. 11–21, 1976.

[10] J. Jerosch and N. M. Nasef, "Endoscopic calcaneoplasty—rationale, surgical technique, and early results: a preliminary

report," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 11, no. 3, pp. 190–195, 2003.

[11] P. E. Scholten and C. N. van Dijk, "Endoscopic calcaneoplasty," *Foot and Ankle Clinics*, vol. 11, no. 2, pp. 439–446, 2006.

[12] J. A. Heneghan and H. Pavlov, "The Haglund painful heel syndrome. Experimental investigation of cause and therapeutic implications," *Clinical Orthopaedics and Related Research*, vol. 187, pp. 228–234, 1984.

[13] M. Pauker, K. Katz, and Z. Yosipovitch, "Calcaneal osteotomy for Haglund's disease," *Journal of Foot and Ankle Surgery*, vol. 31, pp. 558–589, 1992.

[14] P. Angermann, "Chronic retrocalcaneal bursitis treated by resection of the calcaneus," *Foot and Ankle*, vol. 10, no. 5, pp. 285–287, 1990.

[15] M. S. Ballal, C. R. Walker, and A. P. Molloy, "The anatomical footprint of the Achilles tendon: a cadaveric study," *The Bone and Joint Journal*, vol. 96, no. 10, pp. 1344–1348, 2014.

[16] H. Pavlov, M. A. Heneghan, A. Hersh, A. B. Goldman, and V. Vigorita, "The Haglund syndrome: initial and differential diagnosis," *Radiology*, vol. 144, no. 1, pp. 83–88, 1982.

[17] M. R. Vega, D. J. Cavolo, R. M. Green, and R. S. Cohen, "Haglund's deformity," *Journal of the American Podiatry Association*, vol. 74, no. 3, pp. 129–135, 1984.

[18] W. G. Clancy, "Runners' injuries. Part two. Evaluation and treatment of specific injuries," *The American Journal of Sports Medicine*, vol. 8, no. 4, pp. 287–289, 1980.

[19] Ü. Dündar, H. Pusak, and V. Kavuncu, "A rare cause of heel pain: Haglund's syndrome," *Turkiye Fiziksel Tip ve Rehabilitasyon Dergisi*, vol. 54, no. 1, pp. 33–35, 2008.

[20] C. N. van Dijk, M. N. van Sterkenburg, J. I. Wiegerinck, J. Karlsson, and N. Maffulli, "Terminology for Achilles tendon related disorders," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 19, no. 5, pp. 835–841, 2011.

[21] L. J. W. Burhenne II and D. G. Connell, "Xeroradiography in the diagnosis of the Haglund syndrome," *Canadian Association of Radiologists Journal*, vol. 37, no. 3, pp. 157–160, 1986.

[22] J. A. Narváez, J. Narváez, R. Ortega, C. Aguilera, A. Sánchez, and D. E. Andía, "Painful heel: MR imaging findings," *Radiographics*, vol. 20, no. 2, pp. 333–352, 2000.

[23] V. Gulati, M. Jaggard, S. S. Al-Nammari et al., "Management of achilles tendon injury: a current concepts systematic review," *World Journal of Orthopaedics*, vol. 6, no. 4, pp. 380–386, 2015.

[24] E. J. Sella, D. S. Caminear, and E. A. McLarney, "Haglund's syndrome," *Journal of Foot and Ankle Surgery*, vol. 37, no. 2, pp. 110–114, 1998.

[25] D. Jones, "Retrocalcaneal bursitis and insertional Achilles tendinitis," *Sports Medicine and Arthroscopy Review*, vol. 2, pp. 301–309, 1994.

[26] G. Nelen, M. Martens, and A. Burssens, "Surgical treatment of chronic Achilles tendinitis," *The American Journal of Sports Medicine*, vol. 17, no. 6, pp. 754–759, 1989.

[27] M. H. Baums, T. Kostuj, H.-M. Klinger, and R. Papalia, "Rotator cuff repair: single- vs double-row. Clinical and biomechanical results," *Orthopade*, vol. 45, no. 2, pp. 118–124, 2016.

[28] W. Schneider and S. Jurenitsch, "Normative data for the American Orthopedic Foot and Ankle Society ankle-hindfoot, midfoot, hallux and lesser toes clinical rating system," *International Orthopaedics*, vol. 40, no. 2, pp. 301–306, 2016.

[29] J. V. Iversen, E. M. Bartels, and H. Langberg, "The victorian institute of sports assessment—achilles questionnaire (visa-a)—a reliable tool for measuring achilles tendinopathy," *International Journal of Sports Physical Therapy*, vol. 7, no. 1, pp. 76–84, 2012.

[30] L.-L. Liu, B.-J. Xie, W.-L. Wang, M.-H. Dai, G.-J. Yang, and C.-X. Tang, "Therapeutic effects of suture anchors for the reconstruction of distal tendo achillis rupture," *Zhongguo Gu Shang*, vol. 23, no. 3, pp. 177–179, 2010.

Hindawi Publishing Corporation
BioMed Research International
Volume 2016, Article ID 6043638, 8 pages
http://dx.doi.org/10.1155/2016/6043638

Research Article

Application of Computed Tomography Processed by Picture Archiving and Communication Systems in the Diagnosis of Acute Achilles Tendon Rupture

Hai-Peng Xue, Xin-Wei Liu, Jing Tian, Bing Xie, Chao Yang, Hao Zhang, and Da-Peng Zhou

Department of Orthopedics, General Hospital of Shenyang Military Area Command of Chinese PLA, Rescue Center of Severe Wound and Trauma of Chinese PLA, Shenyang, Liaoning 110016, China

Correspondence should be addressed to Da-Peng Zhou; me3210@163.com

Received 30 June 2016; Revised 19 September 2016; Accepted 26 September 2016

Academic Editor: Hong-Yun Li

The applications of CT examination in the diagnosis of the acute Achilles tendon rupture (AATR) were investigated. A total of 36 patients with suspected acute Achilles tendon rupture were tested using physical examination, ultrasound, and 3DCT scanning, respectively. Then, surgery was performed for the patients who showed positive result in at least two of the three tests for AATR. 3DVR, MPR, and the other CT scan image processing and diagnosis were conducted in PACS (picture archiving and communication system). PACS was also used to measure the length of distal broken ends of the Achilles tendon (AT) to tendon calcaneal insertion. Our study indicated that CT has the highest accuracy in diagnosis of acute Achilles tendon complete rupture. The length measurement is matched between PACS and those actually measured in operation. CT not only demonstrates more details directly in three dimensions especially with the rupture involved calcaneal insertion flap but also locates the rupture region for percutaneous suture by measuring the length of distal stump in PACS without the effect of the position of ankle. The accuracy of CT diagnosis for Achilles tendon partial rupture is yet to be studied.

1. Introduction

Acute Achilles tendon rupture is the most common tendon injures of lower limbs [1]. Physical examination, color Doppler ultrasound (US), and magnetic resonance imaging (MRI) have high rates of diagnosis, but each one has its drawbacks and limitations, so there is still no golden standard for diagnosis. Thompson's sign test is not very obvious in some patients with AATR for the intact plantar tendon or more proximal rupture. Ultrasound is often chosen to diagnose AATR, because of high accuracy and being economical and easy to use. However, complete rupture is usually misdiagnosed by ultrasound as partial rupture which will influence the strategy of treatment [2]. MRI also has high accuracy, but it may have few days' appointment for examination, and some authors even suggest MRI is unnecessary for diagnosing AATR [3].

CT is usually used for checking lungs, brain, and internal organs. Three-dimensional computed tomography (3DCT) has been used to diagnose the osteopathy for its high spatial resolution which can appraise bones in any views. It has been reported that 3DCT was used for tendon inspection two decades ago [4], but the results appeared unsatisfactory due to technology limitations. Thus, CT diagnosis of soft tissue has not been widely used. However, CT technology has been developing rapidly in recent years. Hardware has developed from the 4 rows scan to 256 MDCT and even dual energy CT [5] as well as gemstones CT [6]. Postprocessing technology has also been improved greatly such as the emergence of shaded surface display (SSD), maximum intensity projection (MIP), multiplanar reconstruction (MPR), three-dimensional-volume rendering (3DVR) [7–11], and kinematic 4DCT [12]. The new technologies not only reduce the diagnostic time significantly, but also demonstrate more distinction and intuitive information than before. Recently, some authors have reported the diagnosis of tendon disease with computed tomography [13, 14]. However, the accuracy of

(a) (b)

Figure 1: (a) PACS with four windows in left display three orthogonal MRP images and one 3DVR image, and module list in right prepare for displaying various tissues. (b) Select the module we have created before to display bone and tendon in one window. The fibula, calcaneus, peroneus longus and brevis muscles, and Achilles tendon can be seen clearly.

CT technology for diagnosis of acute Achilles tendon rupture has not been reported.

PACS has been widely used in most of large-scale hospitals. It can receive and store archive images from medical imaging modalities (including CT, MR, CR, DX, and any other DICOM device, except US) and distributes them to DICOM devices [15]. DICOM data saved in PACS servers will not deform images. The images can be stored for a long time and can be retrieved anytime in any computer where PACS client has been installed. Surgeons trained in a few hours can process DICOM by PACS like radiologists and are able to get more useful information and details of injures directly. In this work, we expect to explore the advantages and disadvantages of CT diagnosis processed by PACS of AATR.

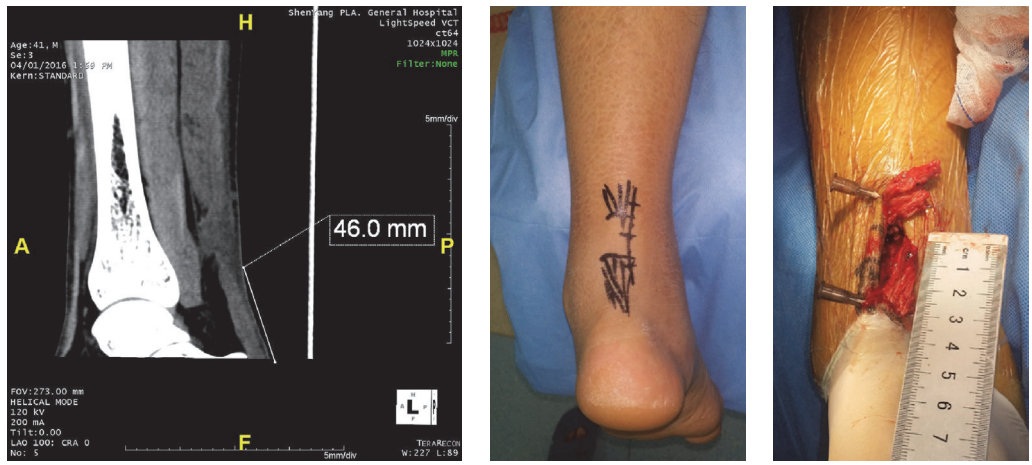
2. Materials and Methods

2.1. Patients Selection and Diagnosis. From February 1, 2014, to May 1, 2016, patients who had a history of acute sudden pain of Achilles tendon were included. Cases such as opening injure, Achilles tendinitis, and chronic rupture were excluded. The patients were examined by US and 3DCT scanning after one orthopedist finished the physical examination. And then, another orthopedist processed the DICOM data with 3DVR and MPR modules of PACS and got a CT diagnosis.

2.2. CT Examination. GE LightSpeed VCT, KVP 120 kV, X-ray tube current 200–230 mA, slice thickness 0.625 mm, spacing between slices 5.0, and 64 Spiral CT were used in this study. Dual energy CT can better display calcium deposits in the blood vessels, but not as good as ordinary CT on showing the tendon [16]. Gemstone CT can effectively reduce the shadow of implants, but not improve tendon images [17]. As we all know, CT scanning parameters are different for different tissues. The greater the tube current the better the image which displays the details of the tissues while the amount of radiation is larger. There are no common CT conditions for the tendon scanning. The parameters for the bone scanning are used for tendon scanning though a clear image of Achilles tendon can be obtained [13, 14]. Hopefully, scholars can design the parameters specifically for Achilles tendon scanning. So the radiation absorption can be reduced and clear images can be obtained.

2.3. PACS. The information of patient scanned by CT was saved as DICOM and was translated to PACS (Aquarius Net, Terarecon, Foster City, CA, USA) servers. We can retrieve those data by searching ID or names in PACS which has many useful modules like 3DVR, MPR, MIP, VR, CPR, and other image processing functions. Each module has many templates. We can switch and demonstrate various tissues in one window by choosing different templates of 3DVR. We can also create a new template to demonstrate many tissues in one picture (Figure 1(a)). In this research, we have created template which can display bone and tendon at the same time to observe the relationship between them. The measurement function allows us to measure the distance between any points or lines in 2D or 3D images and can be accurate to 0.1 mm.

2.4. 3DVR Postprocessing. 3DVR Reconstruction is the most common CT three-dimensional displaying method [18]. The attenuation values (or Hounsfield units) are different for different tissue at CT scan. HU of air is defined as −1000, water HU is defined as 0, the fat is about −100 HU, muscle 40 HU, Achilles 100 HU, and bone 200–500 HU [13]. Different tissues can be displayed in one image distinguishingly when different HU threshold values are assigned to different colors. The front of the Achilles tendon is fat and the skin is at the back. A three-dimensional image of the tendon and bone can be obtained after hiding the skin and fat based on the HU values. After we got the data of patients by PACS, choose the bone 3DVR template (window width 500, window level 400, opacity 1.0, and white color) and add a tendon tissue bar (window width 200, window level 100, opacity 0.8–1.0, and red color), or choose the template we had created before. It is important to note that the voltage can affect HU value.



(a) (b) (c)

Figure 2: 41-year-old male. (a) Adjusting the axis of images though the MPR module and measuring the length of distal part. (b) Locating the rupture position by the distance from the calcaneal insertion of AT measured in CT MPR images. (c) Measuring the length of distal part intraoperation directly.

The higher the voltage is, the higher the HU value is [19], so fine-tune is needed to get clear outline of Achilles tendon (Figure 1(b)).

2.5. MPR Postprocessing. When a patient lays on his back for CT scan, legs are in a swing state. The coronal and sagittal images based on torso axis are not lower limb axial images (Figure 1(a)). The coronal and sagittal images of lower limb can be obtained through MPR reconstruction by adjusting the angle of the axis. The images of coronal, sagittal, and tangential are shown in mutually perpendicular plane, respectively. The axis of each plane is adjusted to get real axial, sagittal, and coronal images of AT. Better contrast between tendons and other soft tissues can be obtained by window width and window level through MPR module. You can also adjust the axis to vertical or horizontal to the tibia, tendons, or other tissues to reduce the position requirements for CT scan. More details of the damage can also be learned from the different levels [9]. We can observe the continuity and tension of AT in 3DVR and MPR images and get a CT diagnosis. If the diagnosis is rupture, the length of distal broken ends to tendon calcaneal insertion would be measured (Figure 2(a)).

2.6. Treatments. A surgery will be conducted if a patient shows positive result in at least two of those three tests: (A) Thompson's sign test in clinical examination; (B) complete rupture diagnosed by ultrasound; (C) complete rupture diagnosed by CT. The length of distal stump measured in CT MPR images was used as a reference in a mini-incision (Figure 2(b)). An operation would be conducted to fix the rupture tendons, and the real length of distal stump would be measured in the operation (Figure 2(c)). An above-knee cast with ankle plantar flexion is placed after the operation for 3 weeks and then shifted to below-knee cast for another 3 weeks.

Table 1: Comparison of Thompson's sign test, US, CT diagnosis, and intraoperation findings.

		Thompson's sign test		Total
		Positive	Negative or WP	
		33	3	36
US	CR	29	3	32
	PR	4	0	4
CT	CR	33	3	36
	PR	0	0	0
IO	CR	33	3	36
	PR	0	0	0

WP: weakly positive; CR: complete rupture; PR: partial rupture; IO: intraoperation.

3. Result

3.1. Patients Information. There are 33 males and 3 females in the study. The oldest is 81 years old, and the youngest is 17 years old. The average age is 37.6 years. The shortest time between diagnosis and injury was 0.5 hours and the longest time was 48 hours. For the causes of injuries, 11 patients were injured from playing basketball, 6 patients were injured during playing badminton, 4 patients were injured due to playing jump rope, 4 patients were injured because of playing soccer, 3 patients were injured during running, 4 patients were injured in the training, and 5 patients were injured due to other causes.

3.2. Accuracy of CT Diagnosis. As listed in Table 1, there are total of 36 patients. 33 patients showed positive result in Thompson's sign test. 4 were diagnosed as Achilles tendon partial rupture (PR) and 32 were diagnosed as complete Achilles tendon rupture (CR) in Doppler ultrasound diagnosis. CT results showed all 36 patients had acute Achilles

Table 2: The Pearson correlation coefficient of the LODS measured by CT and surgeries.

Correlations			
		CT	IO
CT	Pearson correlation	1	0.963
	Sig. (2-tailed)		0.000
	N	36	36
IO	Pearson correlation	0.963	1
	Sig. (2-tailed)	0.000	
	N	36	36

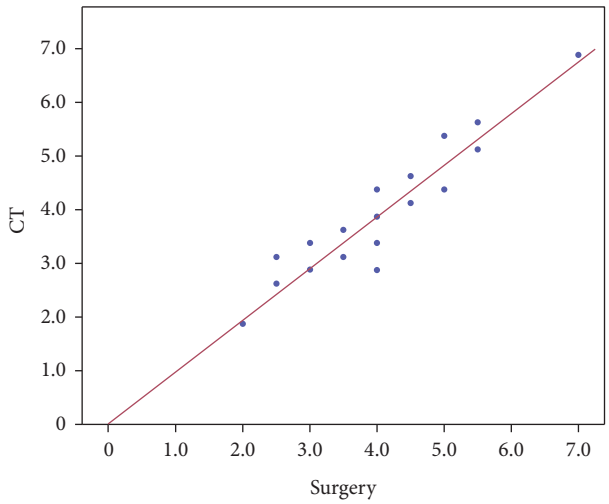


Figure 3: The correlation between CT measurements and the measurements in surgeries on the length of distal stump.

tendon complete rupture. In the operation, all the Achilles tendons were completely ruptured. Based on these results, CT diagnosis is the most accurate method to diagnose acute Achilles tendon complete rupture. Diagnostic accuracy of CT for acute Achilles tendon partial rupture is to be studied.

3.3. Accuracy of CT Measurement. The length of distal stump (LODS) measured in CT is 3.71 ± 1.16 cm (range 0–7.0 cm), and LODS measured intraoperation is 3.83 ± 1.17 (range 0–7.2 cm) (Figure 3). Pearson correlation coefficients show a high correlation ($r = 0.963$, $p < 0.01$) (Table 2).

4. Discussion

AATR is the most common tendon of lower limbs [1], although the AT is one of the strongest tendons of human [20]. It usually happens in patients between 30 and 40 years old [21] and significantly more men than women [22]. The incidence rate has trended higher [23, 24] recently. Rupture is located in the middle distance at the tendon insertion 2–6 cm [25]. In our study, the ratio of men to women is close to 11 : 1 with an average of 37.6 years old. Most incidences happened when patients took part in various sports activities who are middle-aged and fat. The distance between the rupture site to the tendon stump position has an average of 3.8 cm as some other studies reported.

Clinical examination is the first and most important step to diagnose the Achilles ruptures: ecchymosis and swelling occur rapidly in the first 24 hours after injury. A palpable gap can be touched at the rupture site and plantar flexion power is more often diminished [26]. Thompson's sign test has been a routine examination since it is described by Thompson and Doherty in 1962 [27]. However, Thompson's sign test is not very obvious for the intact plantar tendon, and the diminished plantar flexion strength may be less with a more proximal rupture [28], and the palpable gap will be overlooked with soft tissue swelling. In this study, Thompson's sign was weakly positive for 3 patients, and these 3 patients had the plantar tendons intact. Therefore, some other diagnostics are necessary as supplements of Thompson's sign test. Doppler ultrasound and magnetic resonance imaging are recognized to be the most useful technique to diagnose the AATR [29, 30].

Margetić et al. [2] study showed that ultrasound has an excellent result. But 8 patients with complete rupture had been diagnosed as partial rupture, though it was found that only 2 patients had partial rupture in the operation. The misdiagnosis might result in entirely different therapeutic strategies and prognosis. Coincidentally, the accuracy of ultrasound diagnosis will be also influenced by experience and some other subjective factors of the sonographer. Otherwise, orthopedist can only be given a result of the diagnosis such as Achilles tendon complete rupture. There are not any other details like the region, range of rupture, or the relationship with the bone around it which could be observed by imaging examination.

In this study, 4 patients were diagnosed by ultrasound as partial rupture. but Thompson's test showed positive sign, and they were diagnosed by CT as Achilles tendon complete rupture. It can be seen in CT 3DVR images that the tendon was thickening, rather than thinning or missing at broken ends (Figure 4(a)). They were caused by the overlap of broken end and hematomas near the surrounding areas, where the HU value is approximate to tendons. So it looks like Achilles Tendinitis or old Achilles tendon injury [13]. Furthermore, 3DVR images showed bending and thickening of the tendon, and MPR images were even more obvious because the tension disappears and the distal part of tendon retreats slightly for the AATR (Figure 4(b)).

In addition, the accuracy can be even improved if comparing with the contralateral limb (Figures 4(c) and 4(d)). This can be done by increasing the scanning window and getting data of both lower extremities. This does not increase radiation exposure time or radiation damage because it is only one scan and the absorbed radiation for distal limb is much less than the abdomen. The only cost is to need more storage to save the data.

MRI has a higher accuracy than ultrasound in the diagnosis of Achilles tendon rupture, especially chronic ones [30, 31]. But Garras et al. [3] suggested MRI is unnecessary for diagnosing AATR because it would take so many days to obtain MRI scanning after the injury, which is time consuming and expensive and may lead to treatment delays.

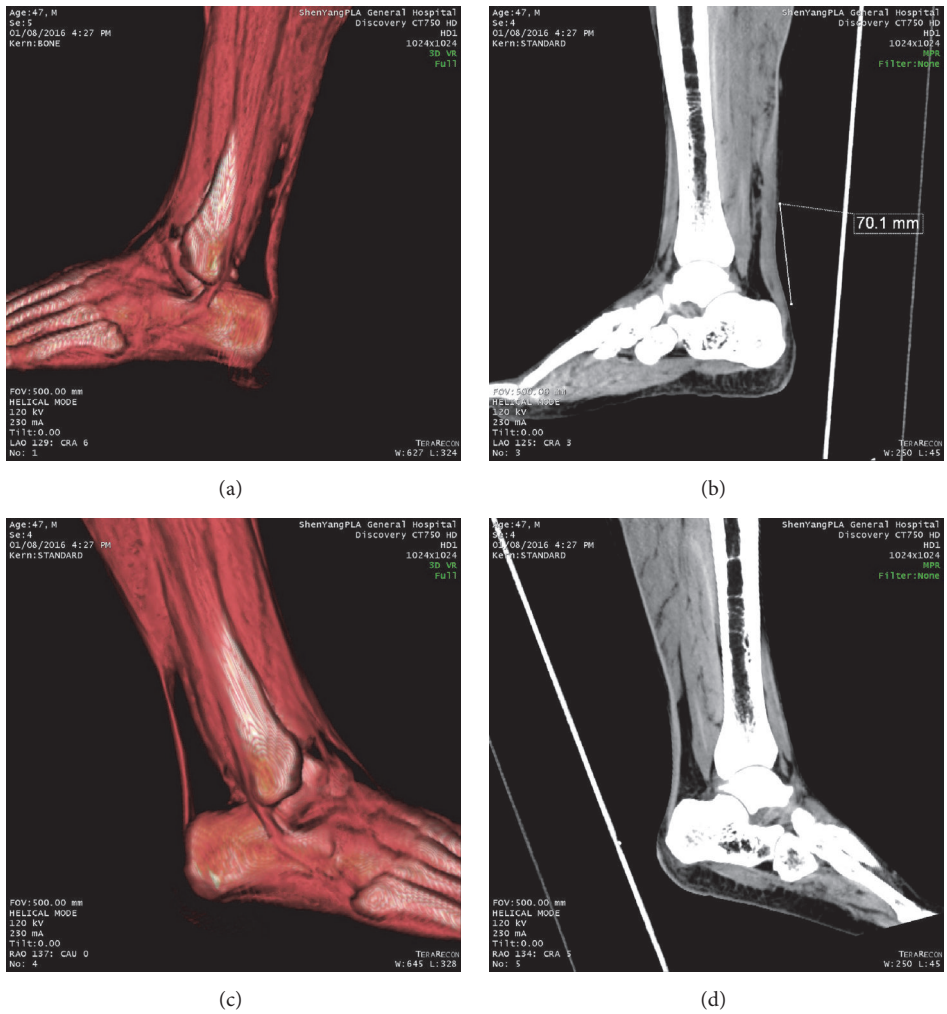
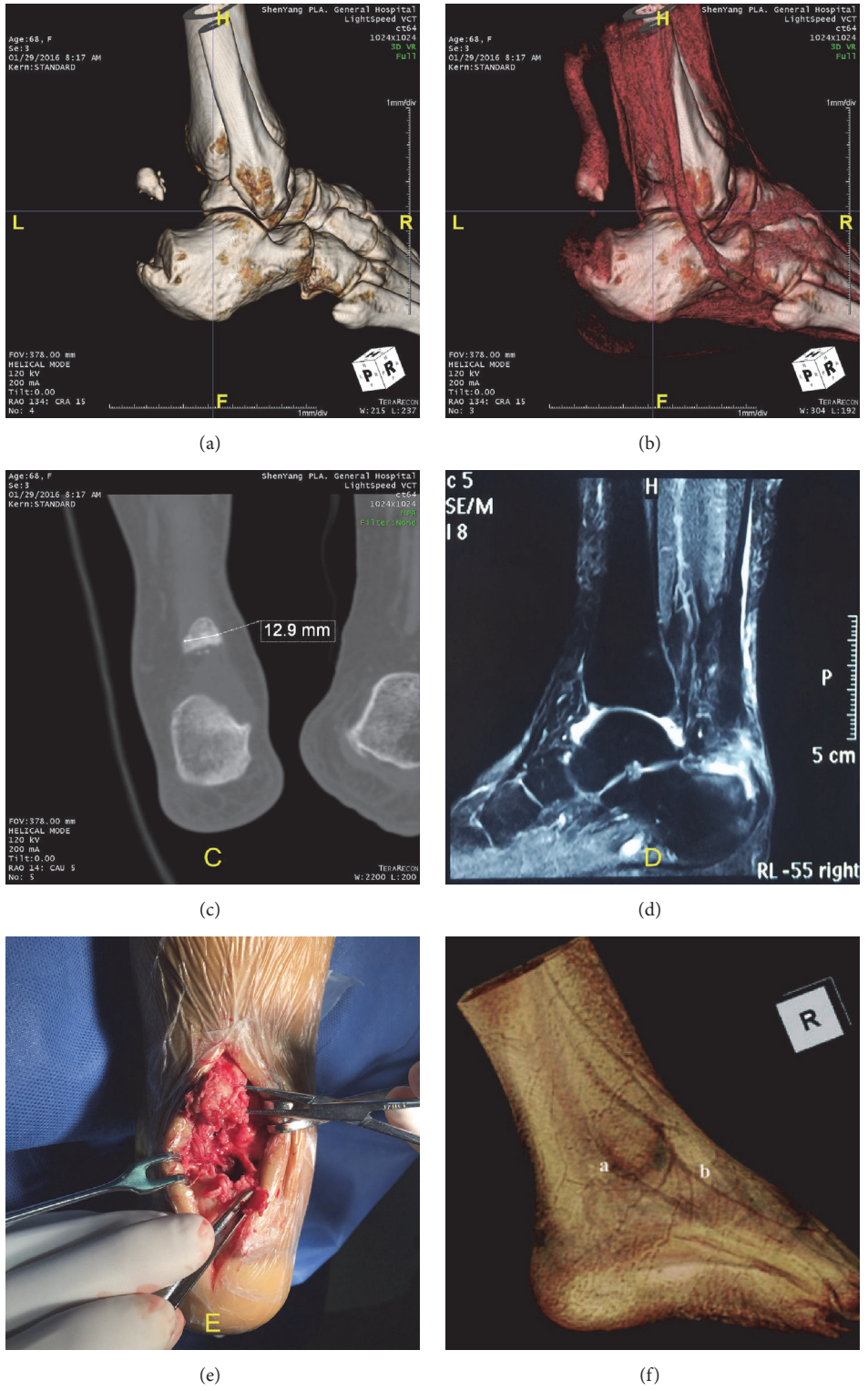


Figure 4: Male patient, 47 years old, was injured while playing badminton. (a) The rupture of Achilles tendon is close to near end and the distance to Achilles point is 7 cm. 3DVR outline image shows the broken ends of the tendon have thickened. (b) Ipsilateral MPR images showed distal tendon thickened and slightly bent due to tension disappearance. Contralateral 3DVR image (c) and MPR image (d) showed good tendon tension but thinner compared with the patients. It is easy to find the difference with affected side.

MRI can even be contraindicated if patients have metal hardware or a history of claustrophobia or obesity [7]. In our hospital, MRI check has been occasionally used as diagnosis for AATR. Patients usually need to wait for 1–3 days. After that soft tissue is at the peak of edema and the distance of broken end increases due to broken end shrinking back gradually. These caused difficulty of microsurgery, and also increased risk of incision healing. Therefore, we do not use MRI diagnosis unless other examinations do not work. By contrast, CT is more efficient. It is easily to take CT scanning, and we can get the information by PACS quickly, when the patients may not leave the scanning room.

Clinicians can use PACS System observing any position of an injury intuitively in 3DVR images, rather than getting only one conclusion from ultrasound and hard to understand details of the damage. CT's advantage is more significant when Achilles tendon calcaneal insertion breaks and combined with avulsed bone (Figures 5(a)–5(e)). MRI data can also be processed using 3DVR, but it is hard to get clearer images. It is difficult to hide the surrounding tissues from tendon based on HU values, because water content in the Achilles tendon is similar to water content in the surrounding tissues and they have similar HU values [32] (Figure 5(f)).

Minimally invasive percutaneous suture technique has been accepted by the majority of doctors because it can greatly reduce the complications of the surgical incision of Achilles tendon problems [33–37]. The precise positioning of the broken end is the basis of minimally invasive surgery. After Achilles tendon ruptures, it is difficult to determine the accurate position because proximal muscles contract. Despite the fact that distal tendinous length has smaller change, it is still difficult to accurately locate the broken position because the tendon sheath remained relatively intact and distal tendon can slide up and down in the lumen of the sheath as well as due to local swelling [38]. Ultrasound can position broken end in surface of projection location, but errors may



(a) (b) (c) (d) (e) (f)

Figure 5: Female patient, 68 years old, hurt in the shower. (a) 3DVR image displays free bone at the back of calcaneus. (b) Widened tendon tissue image, visible Achilles tendon insertion avulsion fragment, Achilles tendon tension disappearing, and visible small tear of the Achilles tendon stump. (c) Design procedures based on bone size through PACS System. (d) The avulsion fragment cannot be seen in MRI images. (e) Achilles tendon avulsion of bone and torn tendon of distal stump observed in the surgery. (f) MRI 3DVR processed image, hard to get 3D image of Achilles tendon due to similar water content in Achilles tendon and the surrounding tissue (image from [32]).

also occur with ankle joint plantarflexion or dorsiflexion [39]. Some doctors used ultrasound for positioning during surgeries, but they need to have ultrasound knowledge which makes it hard to promote this practice [40]. Accordingly, it was mostly relying on the doctor's experience to determine the location of broken end.

Another significant advantage of CT in the diagnosis of AATR is to determine the rupture location. The length of distal stump of Achilles tendon is relatively stable. We can locate the rupture position with the distance to the calcaneal insertion measured predetermined in CT MPR images whenever ankle joint is plantarflexion or dorsiflexion. So the accurate measurement is very helpful to locate the rupture position in the surgeries. Figure 3 indicated that there is high correlation between CT measurements and the measurements in the surgeries on the distal stump length.

In this study, there were two orthopedists to make the clinical diagnosis and CT diagnosis independently to eliminate subjective factors, but CT still showed excellent accuracy. Our sample size is small, and there is no acute Achilles tendon partial rupture. So the result may be more convinced when the sample size is larger and contains more types of rupture.

Some scholars also believe that there is no difference between normal image of Achilles tendon and the image of the partial rupture of Achilles tendon [13]. We did not see Achilles tendon partial rupture in our study. Thus, CT diagnosis of acute Achilles tendon partial rupture is yet to be studied.

In some cases, CT images of AATR are similar with Achilles Tendinitis or old Achilles tendon injury, so medical history is necessary for diagnosis.

5. Conclusions

CT diagnosis has higher accuracy than physical examination or color US for the acute Achilles tendon complete rupture. The details of the injury can be reviewed readily based on CT postprocessing data using PACS System. The relationship of Achilles tendon and the surrounding bone tissues can be shown when the broken end involves the avulsed fragment. The position and length can be accurately measured by PACS, which can provide strong support for minimally invasive surgery. But the accuracy of CT diagnosis for Achilles tendon partial rupture is yet to be studied.

Competing Interests

The authors declare that they have no financial and personal relationships with other people or organizations that can inappropriately influence their work; there is no professional or other personal interests of any nature or kind in any product, service, and/or company that could be construed as influencing the position presented in, or the review of, the manuscript entitled.

References

[1] S. F. Habusta, "Bilateral simultaneous rupture of the Achilles tendon: a rare traumatic injury," *Clinical Orthopaedics and Related Research*, no. 320, pp. 231–234, 1995.

[2] P. Margetić, D. Miklić, V. Rakić-Eršek, Z. Doko, Z. I. Lubina, and B. Brkljačić, "Comparison of ultrasonographic and intraoperative findings in achilles tendon rupture," *Collegium Antropologicum*, vol. 31, no. 1, pp. 279–284, 2007.

[3] D. N. Garras, S. M. Raikin, S. B. Bhat, N. Taweel, and H. Karanjia, "MRI is unnecessary for diagnosing acute achilles tendon ruptures: clinical diagnostic criteria foot and ankle," *Clinical Orthopaedics and Related Research*, vol. 470, no. 8, pp. 2268–2273, 2012.

[4] R. J. Bellon and S. M. Horwitz, "Three-dimensional computed tomography studies of the tendons of the foot and ankle," *Journal of Digital Imaging*, vol. 5, no. 1, pp. 46–49, 1992.

[5] T. Xu, J. L. Ducote, J. T. Wong, and S. Molloi, "Dynamic dual-energy chest radiography: a potential tool for lung tissue motion monitoring and kinetic study," *Physics in Medicine and Biology*, vol. 56, no. 4, pp. 1191–1205, 2011.

[6] Z. Zhu, X. M. Zhao, Y. F. Zhao, X. Y. Wang, and C. W. Zhou, "Feasibility study of using gemstone spectral imaging (GSI) and adaptive statistical iterative reconstruction (ASIR) for reducing radiation and iodine contrast dose in abdominal CT patients with high BMI values," *PLoS ONE*, vol. 10, no. 6, article e0129201, 2015.

[7] J. Fritz, E. K. Fishman, F. Corl, J. A. Carrino, K. L. Weber, and L. M. Fayad, "Imaging of limb salvage surgery," *American Journal of Roentgenology*, vol. 198, no. 3, pp. 647–660, 2012.

[8] B. S. Kuszyk, D. G. Heath, D. F. Bliss, and E. K. Fishman, "Skeletal 3-D CT: Advantages of volume rendering over surface rendering," *Skeletal Radiology*, vol. 25, no. 3, pp. 207–214, 1996.

[9] J. H. Lim, B. C. Jun, and S. W. Song, "Clinical feasibility of multiplanar reconstruction images of temporal bone CT in the diagnosis of temporal bone fracture with otic-capsule-sparing facial nerve paralysis," *Indian Journal of Otolaryngology and Head and Neck Surgery*, vol. 65, no. 3, pp. 219–224, 2013.

[10] B. J. Whyms, H. K. Vorperian, L. R. Gentry, E. M. Schimek, E. T. Bersu, and M. K. Chung, "The effect of computed tomographic scanner parameters and 3-dimensional volume rendering techniques on the accuracy of linear, angular, and volumetric measurements of the mandible," *Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology*, vol. 115, no. 5, pp. 682–691, 2013.

[11] G. Cutroneo, D. Bruschetta, F. Trimarchi et al., "In vivo CT direct volume rendering: a three-dimensional anatomical description of the heart," *Polish Journal of Radiology*, vol. 81, pp. 21–28, 2016.

[12] J. T. Shores, S. Demehri, and A. Chhabra, "Kinematic '4 dimensional' CT imaging in the assessment of wrist biomechanics before and after surgical repair," *Eplasty*, vol. 13, article e9, 2013.

[13] S. Demehri, M. Chalian, S. J. Farahani, E. K. Fishman, and L. M. Fayad, "Detection and characterization of tendon abnormalities with multidetector computed tomography," *Journal of Computer Assisted Tomography*, vol. 38, no. 2, pp. 299–307, 2014.

[14] A. Abe, H. Ishikawa, A. Murasawa, and K. Nakazono, "Extensor tendon rupture and three-dimensional computed tomography imaging of the rheumatoid wrist," *Skeletal Radiology*, vol. 39, no. 4, pp. 325–331, 2010.

[15] W. Tzeng, K. Kuo, H. Lin, and T. Chen, "A Socio-technical assessment of the success of picture archiving and communication systems: the radiology technologist's perspective," *BMC Medical Informatics and Decision Making*, vol. 13, no. 1, article 109, 2013.

[16] D. G. Lohan, K. Motamedi, K. Chow et al., "Does dual-energy CT of lower-extremity tendons incur penalties in patient radiation exposure or reduced multiplanar reconstruction image quality?" *American Journal of Roentgenology*, vol. 191, no. 5, pp. 1386–1390, 2008.

[17] Y. Jia, J. Zhang, J. Fan et al., "Gemstone spectral imaging reduced artefacts from metal coils or clips after treatment of cerebral aneurysms: a retrospective study of 35 patients," *British Journal of Radiology*, vol. 88, no. 1055, 2015.

[18] A. H. Mahnken, J. E. Wildberger, A. M. Sinha et al., "Value of 3D-volume rendering in the assessment of coronary arteries with retrospectively ECG-gated multislice spiral CT," *Acta Radiologica*, vol. 44, no. 3, pp. 302–309, 2003.

[19] A. Parsa, N. Ibrahim, B. Hassan, A. Motroni, P. Der Van Stelt, and D. Wismeijer, "Influence of cone beam CT scanning parameters on grey value measurements at an implant site," *Dentomaxillofacial Radiology*, vol. 42, no. 3, Article ID 79884780, 2013.

[20] M. DeMaio, R. Paine, and D. J. Drez Jr., "Achilles tendonitis," *Orthopedics*, vol. 18, no. 2, pp. 195–204, 1995.

[21] R. J. Khan and R. L. Carey Smith, "Surgical interventions for treating acute Achilles tendon ruptures," *Cochrane Database of Systematic Reviews*, vol. 9, Article ID CD003674, 2010.

[22] G. S. Karin, B. Annelie, O. Nicklas, B. I. Eriksson, and K. Jon, "Sex differences in outcome after an acute achilles tendon rupture," *The Orthopaedic Journal of Sports Medicine*, vol. 2, no. 2, 2014.

[23] A. Ganestam, T. Kallemose, A. Troelsen, and K. W. Barfod, "Increasing incidence of acute Achilles tendon rupture and a noticeable decline in surgical treatment from 1994 to 2013. A nationwide registry study of 33,160 patients," *Knee Surgery, Sports Traumatology, Arthroscopy*, 2015.

[24] T. T. Huttunen, P. Kannus, C. Rolf, L. Felländer-Tsai, and V. M. Mattila, "Acute achilles tendon ruptures: incidence of injury and surgery in Sweden between 2001 and 2012," *American Journal of Sports Medicine*, vol. 42, no. 10, pp. 2419–2423, 2014.

[25] M. J. Coughlin, R. A. Mann, and C. L. Saltzman, *Charles Saltzman: Surgery of the Foot and Ankle*, 8th edition, 2007.

[26] G. W. C. Ma and T. G. Griffith, "Percutaneous repair of acute closed ruptured Achilles tendon: a new technique," *Clinical Orthopaedics and Related Research*, vol. 128, pp. 247–255, 1977.

[27] T. C. Thompson and J. H. Doherty, "Spontaneous rupture of tendon of Achilles: a new clinical diagnostic test," *The Journal of Trauma*, vol. 2, pp. 126–129, 1962.

[28] N. Giannestras, *Foot Disorders: Medical and Surgical Management*, Lea & Febiger, Philadelphia, Pa, USA, 1973.

[29] M. Paavola, T. Paakkala, P. Kannus, and M. Järvinen, "Ultrasonography in the differential diagnosis of achilles tendon injuries and related disorders: a comparison between preoperative ultrasonography and surgical findings," *Acta Radiologica*, vol. 39, no. 6, pp. 612–619, 1998.

[30] K. M. Khan, B. B. Forster, J. Robinson et al., "Are ultrasound and magnetic resonance imaging of value in assessment of Achilles tendon disorders? A two year prospective study," *British Journal of Sports Medicine*, vol. 37, no. 2, pp. 149–153, 2003.

[31] A. F. Sadek, E. H. Fouly, M. A. Laklok, and M. F. Amin, "Functional and MRI follow-up after reconstruction of chronic ruptures of the Achilles tendon Myerson type III using the triple-loop plantaris tendon wrapped with central turndown flap: a case series," *Journal of Orthopaedic Surgery and Research*, vol. 10, no. 1, article 109, pp. 1–10, 2015.

[32] G. Anastasi, G. Cutroneo, D. Bruschetta et al., "Three-dimensional volume rendering of the ankle based on magnetic resonance images enables the generation of images comparable to real anatomy," *Journal of Anatomy*, vol. 215, no. 5, pp. 592–599, 2009.

[33] H. Krueger and S. David, "The effectiveness of open repair versus percutaneous repair for an acute achilles tendon rupture: a critically appraised topic," *Journal of Sport Rehabilitation*, 2016.

[34] A. Mavrodontidis, M. Lykissas, P. Koulouvaris, D. Pafilas, V. Kontogeorgakos, and C. Zalavras, "Percutaneous repair of acute Achilles tendon rupture: a functional evaluation study with a minimum 10-year follow-up," *Acta Orthopaedica et Traumatologica Turcica*, vol. 49, no. 6, pp. 661–667, 2015.

[35] H. Chen, X. Ji, Q. Zhang, X. Liang, and P. Tang, "Erratum to: channel-assisted minimally invasive repair of acute Achilles tendon rupture," *Journal of Orthopaedic Surgery and Research*, vol. 11, article 15, 2016.

[36] B. Shi and Z. Y. Li, "Double minimally invasive incisions for surgical repair of the acute closed Achilles tendon rupture," *Zhongguo Gu Shang*, vol. 28, no. 9, pp. 820–823, 2015.

[37] Z. Y. He, M. X. Chai, Y. J. Liu et al., "Percutaneous repair technique for acute achilles tendon rupture with assistance of kirschner wire," *Orthopaedic Surgery*, vol. 7, no. 4, pp. 359–363, 2015.

[38] M. H. Amlang, H. Zwipp, A. Friedrich, A. Peaden, A. Bunk, and S. Rammelt, "Ultrasonographic classification of achilles tendon ruptures as a rationale for individual treatment selection," *ISRN Orthopedics*, vol. 2011, Article ID 869703, 10 pages, 2011.

[39] A. A. Qureshi, T. Ibrahim, W. J. Rennie, and A. Furlong, "Dynamic ultrasound assessment of the effects of knee and ankle position on achilles tendon apposition following acute rupture," *Journal of Bone and Joint Surgery—Series A*, vol. 93, no. 24, pp. 2265–2270, 2011.

[40] S. Lacoste, J. M. Féron, and B. Cherrier, "Percutaneous Tenolig® repair under intra-operative ultrasonography guidance in acute Achilles tendon rupture," *Orthopaedics and Traumatology: Surgery and Research*, vol. 100, no. 8, pp. 925–930, 2014.

Hindawi Publishing Corporation
BioMed Research International
Volume 2016, Article ID 6315846, 6 pages
http://dx.doi.org/10.1155/2016/6315846

Clinical Study

Outcome of Extracorporeal Shock Wave Therapy for Insertional Achilles Tendinopathy with and without Haglund's Deformity

Ziying Wu, Wei Yao, Shiyi Chen, and Yunxia Li

Department of Sports Medicine, Huashan Hospital, Fudan University, Shanghai, China

Correspondence should be addressed to Yunxia Li; yunxialihuashan@163.com

Received 30 June 2016; Revised 26 October 2016; Accepted 30 October 2016

Academic Editor: Giuseppe Filardo

Purpose. To compare the results of extracorporeal shock wave therapy (ESWT) for insertional Achilles tendinopathy (IAT) with or without Haglund's deformity. *Methods.* Between September 2014 and May 2015, all patients who underwent ESWT were retrospectively enrolled in this study. A total of 67 patients were available for follow-up and assigned into nondeformtiy group ($n = 37$) and deformtiy group ($n = 30$). Clinical outcomes were evaluated by VISA-A Score and 6-point Likert scale. *Results.* The VISA-A score increased in both groups, from 49.57 ± 9.98 at baseline to 83.86 ± 8.59 at 14.5 ± 7.2 months after treatment in nondeformity group ($P < 0.001$) and from 48.70 ± 9.38 at baseline to 67.78 ± 11.35 at 15.3 ± 6.7 months after treatment in deformity group ($P < 0.001$). However, there was a greater improvement in VISA-A Score for the nondeformity group compared with deformity group ($P = 0.005$). For the 6-point Likert scale, there were decreases from 3.92 ± 0.80 at baseline to 1.57 ± 0.73 at the follow-up time point in nondeformity group ($P < 0.001$) and from 4.0 ± 0.76 at baseline to 2.37 ± 1.03 at the follow-up time point in deformity group ($P < 0.001$). There was no significant difference in improvement of the 6-point Likert scale between both groups ($P = 0.062$). *Conclusions.* ESWT resulted in greater clinical outcomes in patients without Haglund's deformity compared with patients with Haglund's deformity.

1. Introduction

Insertional Achilles tendinopathy (IAT) is among the most common posterior heel conditions while walking and running and is located at the insertion of the Achilles tendon onto the calcaneus, involving pain and swelling of the Achilles tendon itself, the formation of bone spurs, and calcifications at the insertion site [1]. Nonoperative management consists of rest, activity modification, anti-inflammatory medication, physical therapy, eccentric exercise, and corticosteroid injections [2]. Recent several studies have shown that extracorporeal shock wave therapy (ESWT) for the treatment of IAT has achieved good functional and clinical outcomes [3, 4]. Several fundamental studies have shown biological effects of ESWT for IAT. van der Worp et al. claimed that the nonexclusive theories about the mechanisms of ESWT involve pain relief, tissue regeneration, and destruction of calcifications [5]. Waugh et al. observed the increase of IL-6 and IL-8 which could promote fibroblast production of collagen and ECM components and demonstrated that the mechanical stimulus provided by ESWT might contribute to injured tendon tissue remodeling in tendinopathy [6]. Moreover, improved blood supply and early vascularity make use of ECM-degrading enzymes to promote the initial leukocyte infiltration and the subsequent metabolism of the fibers in the damaged tendon area [7]. The ESW-driven transitory increase in TGF-ß1 expression and persistent IGF-I expression could lead to some important changes including controlled inhibition of macrophages-induced ECM degradation and inflammation and an enhanced ECM and collagen type I synthesis [8]. The other therapeutic effects of ESWT consist of tendon cells proliferation and endogenous lubricin production by fibroblasts and tenocytes resulting from growth factors stimulation [9]. Therefore, the ESWT ultimately effectively bring about promotion of cell metabolism, and the latter may accelerate healing process in the pathological Achilles tendon tissue [10].

IAT may be associated with a Haglund's deformity, which is defined as a complex of symptoms involving a superolateral calcaneal prominence, retrocalcaneal bursitis, and superficial

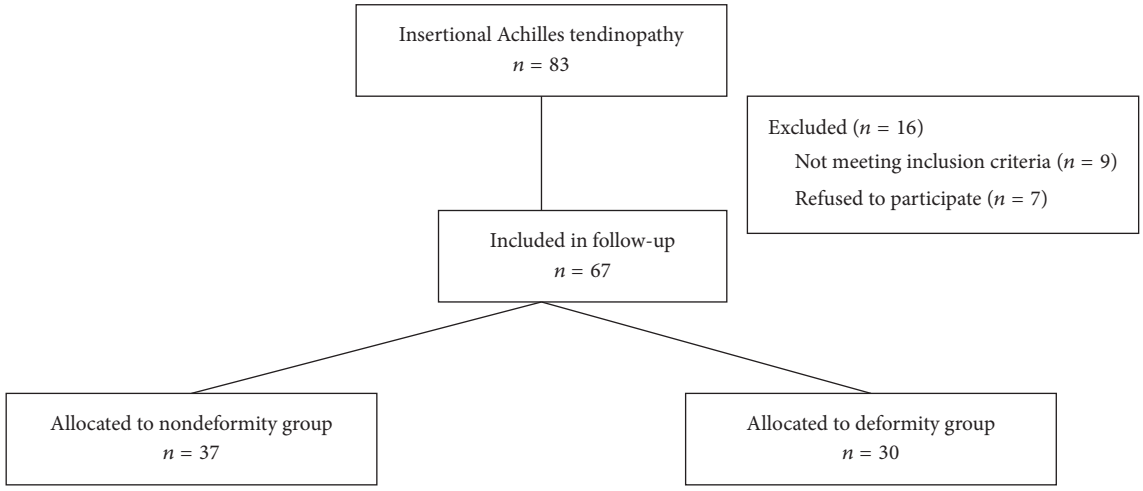


Figure 1: Flow chart of patients included in follow-up. Deformity: Haglund's deformity.

adventitious Achilles tendon bursitis [11–13]. The study of Sundararajan and Wilde exhibited that Haglund's deformity was present in 25% of IAT patients [13].

To our knowledge, it remains unclear if IAT concomitant with Haglund's deformity could achieve great clinical efficacy or not when treated with ESWT. Therefore, the purpose of this study was to reveal clinical outcomes following ESWT between IAT patients with or without Haglund's deformity. We hypothesized that patients with Haglund's deformity will have inferior clinical outcomes compared with patients without Haglund's deformity.

2. Materials and Methods

2.1. Participants. This study was approved by the Ethics Committee of Fudan University. Between September 2014 and May 2015, participants who received shock wave therapy for IAT were recruited retrospectively in this study. The diagnosis of IAT was confirmed by the following definition: pain and localized tenderness at the insertion region of the Achilles tendon and decreased activity levels secondary to Achilles tendon pain [3, 14–16]. All patients underwent preoperative lateral radiograph of ankle to identify Haglund's deformity. The diagnosis of Haglund's deformity was confirmed by the following definition: remarkable osseous prominence at the region of the posterosuperior part of the calcaneus and Fowler-Philip angle of >75° on lateral plain radiographs [3, 11]. According to the presence of Haglund's deformity, all patients were retrospectively classified into two groups: deformity group if patients have concomitant Haglund's deformity and nondeformity group if patients have no Haglund's deformity (Figure 1).

All patients included in this study failed to respond to nonsurgical treatment for at least six months. Nonoperative treatment included activity modification ($n = 65$), physiotherapy ($n = 35$), nonsteroidal anti-inflammatory drugs ($n = 62$), and the use of orthotics ($n = 27$). Steroid injections were not used because of the risk of Achilles tendon rupture and, in some cases, unwillingness to try. Exclusion criteria for this research included prior Achilles tendon rupture, previous surgery of the ankle or the Achilles tendon on the involved side, ankle arthritis, radiculopathy, or systemic neurological conditions, congenital or acquired deformities of the knee and ankle, peripheral neuropathy, lumbar radiculopathy, and inability to comply with the recommended treatment regimen.

2.2. Shock Wave Therapy. The shock wave therapy was performed with the patient in the prone position and was administered once a week, for 5 sessions. All patients completed all 5 sessions. And no patients received more sessions. A radial shock wave device (EMS Swiss Dolor-Clast, Munich, Germany) was used. Radial shock wave is created ballistically with the pressurized air source accelerating a bullet to strike a metal applicator. The kinetic energy produced is transformed into radially expanding shock waves from the application site into the tissue to be treated [3]. At each treatment session, 2000 pulses with an energy flux density of 0.12 mJ/mm^2 and a rate of 8 pulses per second were applied. The applicator of hand-piece was located on the maximal tenderness point and was properly placed and adjusted according to patients' feedback during treatment if necessary [17]. No local anesthetic was applied.

2.3. Clinical Evaluation. Patients, researchers evaluating the clinical outcome, and treating physicians were blinded to the presence of deformity or not. Clinical functional evaluation included VISA-A score (see Figure 2) and 6-point Likert scale (see Figure 3) collected before treatment and at the follow-up time point. As a self-administered questionnaire, the VISA-A score (Appendix) is used to evaluate the severity of Achilles tendinopathy. It has previously been shown to be valid, reliable, and clinically relevant [18]. The content of VISA-A questionnaire is as follows: pain (questions (1)–(3)), function (questions (4)–(6)), and activity (questions (7) and (8)). VISA-A scores have a range of 0 to 100. The 6-point Likert scale is interpreted as success if patients rate themselves 1 or 2 and as failure if patients rate themselves 3, 4, 5, or 6 [19].

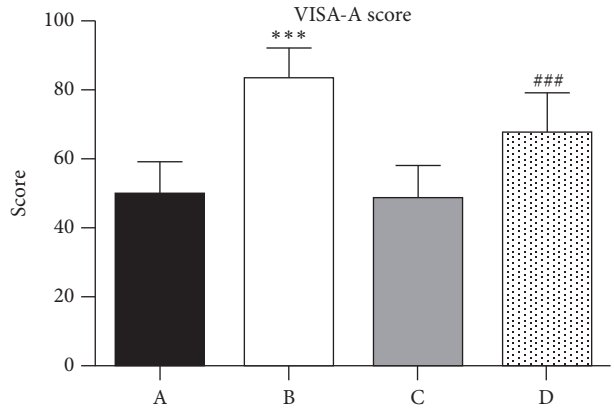


Figure 2: Results of VISA-A score for both groups. A: at baseline in nondeformity group. B: at follow-up in nondeformity group. C: at baseline in deformity group. D: at follow-up in deformity group. $^{***\&\#\#\#}P < 0.001$.

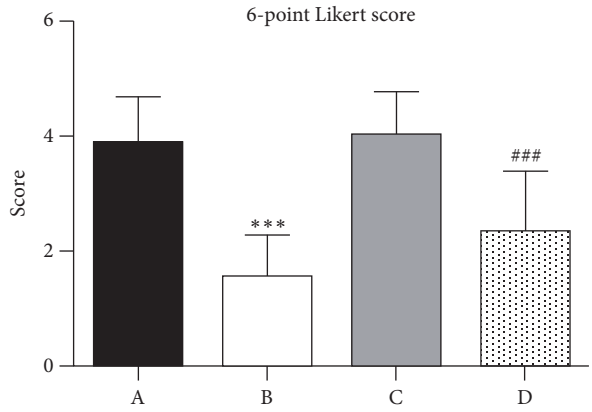


Figure 3: Results of 6-point Likert score for both groups. A: at baseline in nondeformity group. B: at follow-up in nondeformity group. C: at baseline in deformity group. D: at follow-up in deformity group. $^{***\&\#\#\#}P < 0.001$.

2.4. Statistical Analysis. Data analysis is performed using Stata 10.0 software (StataCorp, College Station, TX, USA) and all data are expressed as mean and standard deviation (SD) for description.

The improvement between pretreatment and at the follow-up time point was calculated by paired t-test (see Figure 3). The difference between groups was compared by t-test. Statistical significance was set at $P < 0.05$. The odds ratio (OR) was calculated to figure out the influence of deformity on treatment failure. The 95% confidence interval (CI) was also calculated.

3. Results

3.1. Patient Demographics. At the follow-up time point, a total of 67 patients were available for follow-up, 30 patients in deformity group and 37 in nondeformity group. Participants' demographic data are shown in Table 1. The two groups did not differ significantly in age, body mass index, sex, therapeutic side, and follow-up time. All patients underwent extracorporeal shock wave therapy (ESWT).

3.2. Clinical Outcomes. At the follow-up time point, the functional outcomes with regard to VISA-A score and 6-point Likert scale achieved significant improvements in both groups. However, there was a greater improvement in VISA-A score for the nondeformity group compared with deformity group ($P = 0.005$) (Tables 2 and 3). According to the 6-point Likert scale grading system, there were 34 graded as success and 3 graded as failure in the nondeformity group, and there were 23 graded as success and 7 graded as failure in the deformity group. There were no serious complications including infection and Achilles tendon rupture, except transient reddening of the skin in all patients.

The OR was 3.45 with a 95% CI of [0.81, 14.74], indicating that patients with deformity had a 3.45 times higher risk to experience treatment failure compared with those who are without. However, the difference was not significant ($P = 0.09$).

4. Discussion

The current study validated that ESWT for IAT concomitant with or without Haglund's deformity exhibited improved clinical outcomes. However, the VISA-A scores in patients with Haglund's deformity were inferior to those in patients without Haglund's deformity. The results of this study suggest that Haglund's deformity may worsen therapeutic effect of the ESWT for IAT.

Recent numerous studies have reported that satisfactory clinical results in the treatments of IAT could be achieved with the use of ESWT [3, 4]. Furia concluded that shock wave therapy could obtain satisfactory clinical outcome in the treatment of the chronic insertional Achilles tendinopathy. Follow-up was performed at 1, 3, and 12 months after treatment, and the mean visual analog score for the nonoperative therapy and ESWT groups were 8.2 and 4.2 ($P < 0.001$), 7.2 and 2.9 ($P < 0.001$), and 7.0 and 2.8 ($P < 0.001$), respectively. Twelve months after treatment, more patients in the ESWT group (83% of ESWT group patients) have successful Roles and Maudsley scores compared to those in the control group [4]. In addition, in a randomized, controlled study, Rompe et al. showed shock wave therapy could provide more favorable results compared to eccentric loading for chronic IAT. At 4 months after treatment, both groups have improvement in the mean VISA-A score, increasing from 53 to 63 points in eccentric loading group and 53 to 80 points in shock wave therapy group. Moreover, this clinical result after shock wave therapy remained stable at the one-year follow-up evaluation [3]. In our present study, all the participants in both groups obtain pain relief and significant improvement in clinical outcomes (see Figure 4).

However, significant differences between both groups were observed in the current study, suggesting that Haglund's deformity in the posterior calcaneus has negative influence on clinical results of ESWT for IAT. The calcaneal posterosuperior prominence is called Haglund's deformity. Repetitive squeezing of the retrocalcaneal bursa at dorsal

Table 1: Participant demographic data of the study groups.

Variable	Nondeformity group (n = 37)	Deformity group (n = 30)	P value	95% CI
Age, mean ± SD, y	37.6 ± 9.2	35.8 ± 7.4	0.228	−2.17–5.17
Sex, mean ± SD, n			0.789	0.43–3.04
Male	21	18		
Female	16	12		
Body mass index, mean ± SD, kg/m^2	23.7 ± 2.0	22.9 ± 2.2	0.591	−0.22–1.82
Therapeutic side, mean ± SD, n			0.818	0.42–2.89
left	15	13		
right	22	17		
Follow-up time, mean ± SD, months	14.5 ± 7.2	15.3 ± 6.7	0.705	−4.14–2.54

CI: confidence interval.

Table 2: Clinical outcome scores for both groups.

Outcome Score	Nondeformity group		P value	95% CI	Deformity group		P value	95% CI
	Baseline	Follow-up			Baseline	Follow-up		
VISA-A	49.57 ± 9.98	83.86 ± 8.59	<0.001	30.05–38.53	48.70 ± 9.38	67.78 ± 11.35	<0.001	13.81–24.35
6-point Likert	3.92 ± 0.80	1.57 ± 0.73	<0.001	2.00–2.70	4.0 ± 0.76	2.37 ± 1.03	<0.001	1.17–2.09

CI: confidence interval.

Table 3: Comparison of differences in improvement of clinical outcome scores between nondeformity and deformity group.

	Improvement of clinical outcome scores			
	Nondeformity group	Deformity group	P value	95% CI
VISA-A	34.30 ± 11.96	19.08 ± 7.08	0.005	10.61–19.83
6-point Likert	2.29 ± 0.90	2.00 ± 0.64	0.062	−0.08–0.66

CI: confidence interval.

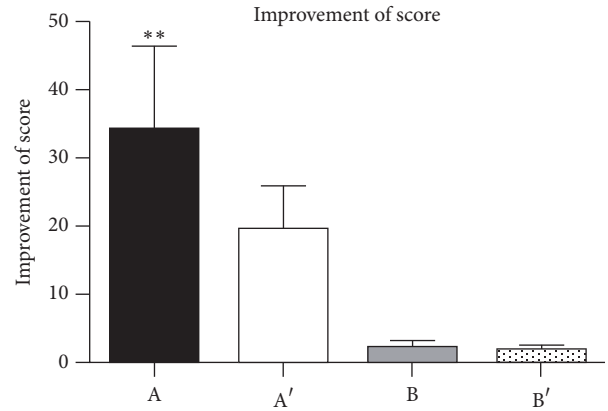


Figure 4: Improvement of clinical scores between nondeformity and deformity group. A: improvement of VISA-A in nondeformity group. A′: improvement of VISA-A in deformity group. B: improvement of 6-point Likert score in nondeformity group. B′: improvement of 6-point Likert score in deformity group. $^{**}P$ = 0.005.

flexion of ankle, resulting from impingement between the posterosuperior prominence of the calcaneus and the anterior aspect of the Achilles tendon, can sometimes cause painful bursitis [20]. Sundararajan and Wilde found 25% frequency of Haglund's syndrome within the IAT population according to both the clinical examination and magnetic resonance imaging [13]. The previous study has reported that the presence of Haglund's deformity has influence on the clinical outcomes of other treatments including eccentric training, which was consistent with the results of our current study. Fahlström et al. found that only 32% of the patients with insertional Achilles tendon pain had good clinical results with usage of painful eccentric training beyond plantar grade. The unfavourable outcomes in patients with insertional Achilles tendon pain may be attributable to mechanical impingement between the prominent calcaneus and the tendon and bursa, when the ankle was in the dorsiflexed position [15]. Following the findings from Fahlström et al., Jonsson et al. made modification and used an eccentric training model without dorsiflexion in the ankle joint to avoid possible mechanical impingement, which resulted in 67% satisfied patients compared with the study of Fahlström et al. [21]. The presence of Haglund's deformity could explain the poorer clinical evaluation in deformity group compared with nondeformity group in the current study.

There are still some limitations in our study. The first is the small number of patients. Larger patient series are needed to verify whether the clinical outcomes could be undermined by Haglund's deformity. Secondly, a longer follow-up is necessary to determine whether ESWT for IAT with or without Haglund's deformity may persist in symptomatic relief. Lastly, a major limitation of the current study is the retrospective study-design, which makes it hard to perform

a power analysis to ensure the statistical efficacy. Future trials with higher quality are required to reveal the influence of deformity on failure rates. The odds ratio (OR) was calculated to figure out the influence of deformity on treatment failure. We found that the OR was 3.45 with a 95% CI of [0.81, 14.74], indicating that patients with deformity had a 3.45 times higher risk to experience treatment failure compared with those who are without. However, the difference was not significant ($P = 0.09$). Future trials with higher quality are required to reveal the influence of deformity on failure rates.

5. Conclusion

The clinical results of ESWT for IAT with and without Haglund's deformity showed significant improvement. However, IAT without Haglund's deformity had significantly greater VISA-A score compared with IAT with Haglund's deformity.

Appendix

The VISA-A Questionnaire: An Index of the Severity of Achilles Tendinopathy

In this questionnaire, the term pain refers specifically to pain in the Achilles tendon region

(1) For how many minutes do you have stiffness in the Achilles region on first getting up?

100 mins ☐☐☐☐☐☐☐☐☐☐☐ 0 mins Points ☐

0 1 2 3 4 5 6 7 8 9 10

(2) Once you are warmed up for the day, do you have pain when stretching the Achilles tendon fully over the edge of a step? (keeping knee straight)

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain Points ☐

0 1 2 3 4 5 6 7 8 9 10

(3) After walking on flat ground for 30 minutes, do you have pain within the next 2 hours? (If unable to walk on flat ground for 30 minutes because of pain, score 0 for this question).

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain Points ☐

0 1 2 3 4 5 6 7 8 9 10

(4) Do you have pain walking downstairs with a normal gait cycle?

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain Points ☐

0 1 2 3 4 5 6 7 8 9 10

(5) Do you have pain during or immediately after doing 10 (single leg) heel raises from a flat surface?

Strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ No pain Points ☐

0 1 2 3 4 5 6 7 8 9 10

(6) How many single leg hops can you do without pain?

0 ☐☐☐☐☐☐☐☐☐☐☐ 10 Points ☐

0 1 2 3 4 5 6 7 8 9 10

(7) Are you currently undertaking sport or other physical activity?

0 ☐ Not at all Points

4 ☐ Modified training ± modified competition ☐

7 ☐ Full training ± competition but not at the same level as when symptoms began

10 ☐ Competing at the same or higher level than when symptoms began

(8) Please complete *EITHER (A), (B) or (C)* in this question.

(i) If you have *no pain while undertaking Achilles tendon loading sports* please complete *Q8a only*.

(ii) If you have *pain while undertaking Achilles tendon loading sports but it does not stop you from completing the activity*, please complete *Q8b only*.

(iii) If you have *pain that stops you from completing Achilles tendon loading sports*, please complete *Q8c only*.

(A) If you have *no pain* while undertaking *Achilles tendon loading sports*, for how long can you train/practise?

Points

NIL	1–10 mins	11–20 mins	21–30 mins	>30 mins	
☐	☐	☐	☐	☐	☐
0	7	14	21	30	

OR

(B) If you have some pain while undertaking *Achilles tendon loading sport*, but it does not stop you from completing your training/practice for how long can you train/practise?

Points

NIL	1–10 mins	11–20 mins	21–30 mins	>30 mins	
☐	☐	☐	☐	☐	☐
0	4	10	14	20	

OR

(C) If you have *pain that stops* you from completing your training/practice in *Achilles tendon loading sport*, for how long can you train/practise?

Points

NIL	1–10 mins	11–20 mins	21–30 mins	>30 mins	
☐	☐	☐	☐	☐	☐
0	2	5	7	10	

Total score (/100) ☐ (%)

Competing Interests

All authors declare that there is no conflict of interests regarding the publication of this paper.

Authors' Contributions

Ziying Wu and Wei Yao contributed equally to this work.

References

[1] C. N. van Dijk, M. N. van Sterkenburg, J. I. Wiegerinck, J. Karlsson, and N. Maffulli, "Terminology for Achilles tendon related disorders," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 19, no. 5, pp. 835–841, 2011.

[2] A. J. Roche and J. D. F. Calder, "Achilles tendinopathy: a review of the current concepts of treatment," *The Bone and Joint Journal*, vol. 95, no. 10, pp. 1299–1307, 2013.

[3] J. D. Rompe, J. Furia, and N. Maffulli, "Eccentric loading compared with shock wave treatment for chronic insertional achilles tendinopathy: a randomized, controlled trial," *Journal of Bone and Joint Surgery—Series A*, vol. 90, no. 1, pp. 52–61, 2008.

[4] J. P. Furia, "High-energy extracorporeal shock wave therapy as a treatment for insertional Achilles tendinopathy," *The American Journal of Sports Medicine*, vol. 34, no. 5, pp. 733–740, 2006.

[5] H. van der Worp, I. van den Akker-Scheek, H. van Schie, and J. Zwerver, "ESWT for tendinopathy: technology and clinical implications," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 21, no. 6, pp. 1451–1458, 2013.

[6] C. M. Waugh, D. Morrissey, E. Jones, G. P. Riley, H. Langberg, and H. R. C. Screen, "In vivo biological response to extracorporeal shockwave therapy in human tendinopathy," *European Cells and Materials*, vol. 29, pp. 268–280, 2015.

[7] G. Bosch, M. de Mos, R. V. van Binsbergen, H. T. M. van Schie, C. H. A. van de Lest, and P. R. van Weeren, "The effect of focused extracorporeal shock wave therapy on collagen matrix and gene expression in normal tendons and ligaments," *Equine Veterinary Journal*, vol. 41, no. 4, pp. 335–341, 2009.

[8] V. Visco, M. C. Vulpiani, M. R. Torrisi, A. Ferretti, A. Pavan, and M. Vetrano, "Experimental studies on the biological effects of extracorporeal shock wave therapy on tendon models. A review of the literature," *Muscles, Ligaments and Tendons Journal*, vol. 4, no. 3, pp. 357–361, 2014.

[9] Y. E. Zhang, "A special issue on TGF-β signaling and biology," *Cell and Bioscience*, vol. 1, no. 1, article 39, 2011.

[10] Y.-J. Chen, C.-J. Wang, K. D. Yang et al., "Extracorporeal shock waves promote healing of collagenase-induced Achilles tendinitis and increase TGF-β1 and IGF-I expression," *Journal of Orthopaedic Research*, vol. 22, no. 4, pp. 854–861, 2004.

[11] C. N. van Dijk, G. E. van Dyk, P. E. Scholten, and N. P. Kort, "Endoscopic calcaneoplasty," *American Journal of Sports Medicine*, vol. 29, no. 2, pp. 185–189, 2001.

[12] C. A. Uquillas, M. S. Guss, D. J. Ryan, L. M. Jazrawi, and E. J. Strauss, "Everything achilles: knowledge update and current concepts in management: AAOS exhibit selection," *The Journal of Bone & Joint Surgery—American Volume*, vol. 97, no. 14, pp. 1187–1195, 2015.

[13] P. P. Sundararajan and T. S. Wilde, "Radiographic, clinical, and magnetic resonance imaging analysis ofinsertional Achilles tendinopathy," *Journal of Foot and Ankle Surgery*, vol. 53, no. 2, pp. 147–151, 2014.

[14] J. R. McCormack, F. B. Underwood, E. J. Slaven, and T. A. Cappaert, "Eccentric exercise versus eccentric exercise and soft tissue treatment (Astym) in the management of insertional achilles tendinopathy: a randomized controlled trial," *Sports Health*, vol. 8, no. 3, pp. 230–237, 2016.

[15] M. Fahlström, P. Jonsson, R. Lorentzon, and H. Alfredson, "Chronic Achilles tendon pain treated with eccentric calf-muscle training," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 11, no. 5, pp. 327–333, 2003.

[16] M. K. Sayana and N. Maffulli, "Insertional achilles tendinopathy," *Foot and Ankle Clinics*, vol. 10, no. 2, pp. 309–320, 2005.

[17] S. Rasmussen, M. Christensen, I. Mathiesen, and O. Simonson, "Shockwave therapy for chronic Achilles tendinopathy: a double-blind, randomized clinical trial of efficacy," *Acta Orthopaedica*, vol. 79, no. 2, pp. 249–256, 2008.

[18] J. M. Robinson, J. L. Cook, C. Purdam et al., "The VISA-A questionnaire: a valid and reliable index of the clinical severity of Achilles tendinopathy," *British Journal of Sports Medicine*, vol. 35, no. 5, pp. 335–341, 2001.

[19] N. Smidt, D. A. W. M. van der Windt, W. J. J. Assendelft, W. L. J. M. Devillé, I. B. C. Korthals-de Bos, and L. M. Bouter, "Corticosteroid injections, physiotherapy, or a wait-and-see policy for lateral epicondylitis: a randomised controlled trial," *Lancet*, vol. 359, no. 9307, pp. 657–662, 2002.

[20] Z. Wu, Y. Hua, Y. Li, and S. Chen, "Endoscopic treatment of Haglund's syndrome with a three portal technique," *International Orthopaedics*, vol. 36, no. 8, pp. 1623–1627, 2012.

[21] P. Jonsson, H. Alfredson, K. Sunding, M. Fahlström, and J. Cook, "New regimen for eccentric calf-muscle training in patients with chronic insertional Achilles tendinopathy: results of a pilot study," *British Journal of Sports Medicine*, vol. 42, no. 9, pp. 746–749, 2008.

Hindawi Publishing Corporation
BioMed Research International
Volume 2016, Article ID 2580969, 5 pages
http://dx.doi.org/10.1155/2016/2580969

Clinical Study

Utility of Ultrasonography in Assessing the Effectiveness of Extracorporeal Shock Wave Therapy in Insertional Achilles Tendinopathy

Yi Cheng,[1] Jian Zhang,[2] and Yehua Cai[1]

[1]*Department of Ultrasound in Medicine, Huashan Hospital Affiliated to Fudan University, Shanghai, China*
[2]*Department of Sports Medicine, Huashan Hospital Affiliated to Fudan University, Shanghai, China*

Correspondence should be addressed to Yehua Cai; sunnei@msn.com

Received 24 June 2016; Accepted 30 October 2016

Academic Editor: Seung H. Han

Introduction. The aim of this study was to investigate the utility of ultrasonography (US) for predicting and assessing the effectiveness of extracorporeal shock wave therapy (ESWT) in insertional Achilles tendinopathy (IAT). *Methods*. A total of 42 patients with an established diagnosis of chronic IAT were examined by US before ESWT and at 4 weeks and 12 weeks after ESWT. The thickness and cross-sectional area (CSA) of the Achilles tendon, size of calcific plaques, tendon structure score, and neovascularization score were measured at each time point. *Results*. After therapy, Victorian Institute of Sport Assessment-Achilles (VISA-A) scores increased significantly, and the size of calcific plaques decreased ($P < 0.05$). Neovascularization scores increased at the 4th week and then decreased at the 12th week ($P < 0.05$). The thickness, CSA, and structure of the Achilles tendon did not change. Variables observed by US at baseline were not associated with changes in VISA-A scores at follow-up. However, the changes in calcific plaque size and neovascularization scores were related to the improvement of VISA-A scores between pre- and posttherapy ($P < 0.01$). *Conclusion*. Ultrasonography can reveal some changes in the insertion of the Achilles tendon after ESWT, but the outcome of ESWT in IAT cannot be predicted by the variables observed by US.

1. Introduction

Achilles tendinopathy (AT) is a frequent overuse problem in athletes and is also common in the general population [1]. From a functional perspective, it is helpful to classify AT occurring at the bone-tendon junction as insertional and those that occur more proximally as noninsertional. Insertional Achilles tendinopathy (IAT) is clinically characterized by tendon pain and swelling in the posterior heel with impaired performance [2].

Insertional Achilles tendinopathy is often difficult to treat, and there is no agreement on the best form of management. Recently, extracorporeal shock wave therapy (ESWT) has been proposed as an effective intervention and should be considered for IAT when other nonoperative treatments have failed [2–6]. The effectiveness of ESWT is assessed by questionnaires, such as the Victorian Institute of Sport Assessment-Achilles (VISA-A) questionnaire and the visual analog score (VAS) [3, 4, 6].

As a convenient, safe, and inexpensive examination, ultrasonography (US) has been widely used in the diagnosis of Achilles tendinopathy [7–11]. However, whether US imaging can reveal changes in the Achilles tendon after ESWT or predict or measure the outcome of ESWT remains unclear. The purpose of this study was to investigate the utility of US for predicting and assessing the effectiveness of ESWT in IAT.

2. Methods

2.1. Patients. Patients with an established diagnosis of chronic IAT who were eligible for ESWT were recruited from the sports medicine department of a large teaching hospital. The diagnosis of IAT was made clinically by a sports medicine doctor. For this study, IAT was defined as symptoms of moderate to severe posterior heel pain located at the bone-tendon junction that extended no more than 2 cm proximal from the base of the heel, swelling, and impaired

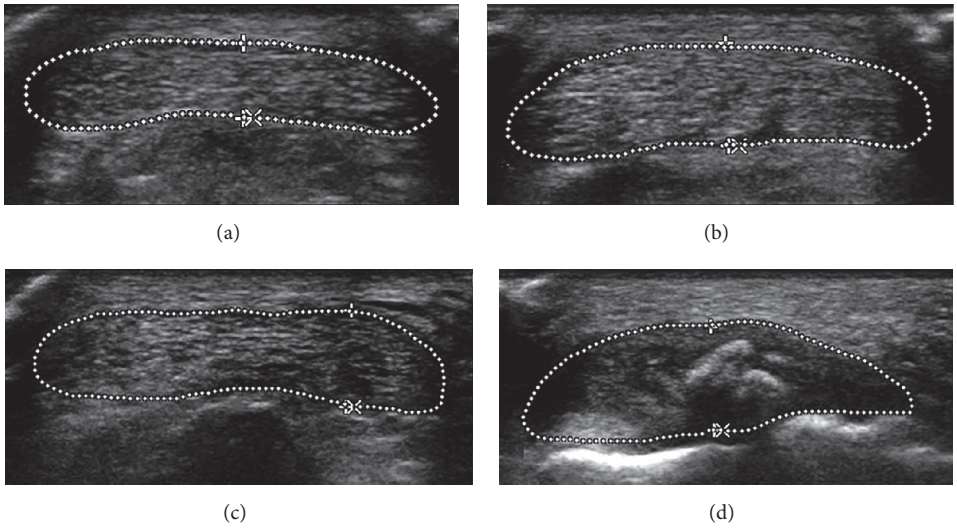


Figure 1: Transverse sonograms show 4-grade scale of tendon structure (outline of Achilles tendon traced by dotted line). (a) Grade 0—normal structure (homogenous echogenicity). (b) Grade 1—light structural changes (discrete hypoechogenic areas). (c) Grade 2—moderate structural changes (some well-defined hypoechogenic areas). (d) Grade 3—severe structural changes (extended hypoechogenic areas).

function [2]. All patients had an established diagnosis of IAT for at least 6 months and had failure with at least 3 forms of traditional nonoperative treatments (such as rest, nonsteroidal anti-inflammatory drugs, physical therapy, and injections) for a minimum of 6 months. Patients were excluded if they had systemic illness or any other condition that could contribute to posterior ankle pain, such as ankle arthritis or radiculopathy. Patients with deformities of the ankle or a history of previous Achilles tendon rupture or surgery were also excluded. The study was approved by the local medical ethics committee, and informed consent was obtained from each subject prior to the investigation.

2.2. ESWT. All patients received ESWT with a radial shock wave device (EMS Swiss Dolor-Clast, Munich, Germany). In our study, ESWT was performed once a week for five weeks. At each of the five sessions, 2000 impulses (0.16 mJ/mm^2) at 6–8 Hz were applied. With this device, starting at the area of maximal tenderness, shock waves were focused on the bone-tendon insertion and extended in a circumferential pattern.

2.3. Ultrasonographic Examination. Ultrasonography scanning was performed with an iU22 ultrasonography unit (Philips Medical Systems, Bothell, Washington) by a radiologist who was blinded to the clinical status of the patients. A wide-frequency linear array transducer (5–17 MHz) was used. The patients were examined in a prone position with the foot hanging over the edge of the examination couch. All Achilles tendons were scanned in the transverse and longitudinal planes within 2 cm above the tendon insertion. Particular attention was paid to maintaining the ultrasound beam perpendicular and minimal probe pressure. The anteroposterior thickness and cross-sectional area (CSA) of the Achilles tendon were measured in the transverse plane 1 cm above the lowest rim of bone-tendon junction. We used a 4-grade scale to evaluate the tendon structure: 0—normal structure (homogenous echogenicity), 1—light structural changes (discrete hypoechogenic areas), 2—moderate structural changes (some well-defined hypoechogenic areas), and 3—severe structural changes (extended hypoechogenic areas) (Figure 1) [12]. A standardized, preprogrammed scanning protocol was used to obtain low blood flow. The color gain was adjusted to the maximum level, creating no clutter or noisy artifacts, and the pulse repetition frequency was set at low. Neovascularization was assessed based on the appearance of vessels inside the tendons: 0—no neovascularization, 1—1 vessel mostly in the anterior part, 2—2 vessels throughout the tendon, 3—3 vessels throughout the tendon, and 4—>3 vessels throughout the tendon [13]. If there was calcific plaque within 2 cm above the tendon insertion, the maximal diameter of the calcific plaque was measured.

Before the first session of ESWT, the baseline measurement was established. Follow-up examinations were performed at 4 weeks and 12 weeks after the whole sessions.

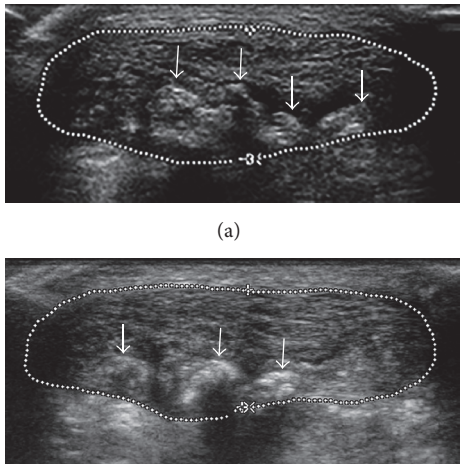
2.4. VISA-A Score. Every patient completed the VISA-A score at baseline and 4 weeks and 12 weeks after the treatment. The VISA-A questionnaire is valid and reliable to measure the severity of Achilles tendinopathy. The VISA-A score contains eight questions that cover the three domains of pain, function, and activity (a maximum score of 100) [14].

2.5. Statistical Analysis. Statistical analysis was performed using the SPSS 16.0 software program. A paired Student t-test or the Wilcoxon signed-rank test was used to compare the difference between baseline and posttreatment effects. Spearman's correlation was used to assess the relationship between outcome variables. Statistical significance was specified as P less than 0.05.

Table 1: Outcome after therapy.

Variables	Baseline	Week 4	P	Week 12	P
VISA-A scores	54.0 ± 8.0	78.3 ± 5.5	0.00	82.6 ± 5.6	0.00
Thickness of the Achilles tendon (mm)	3.8 ± 0.8	3.7 ± 0.9	NS	3.7 ± 0.9	NS
CSA of the Achilles tendon (mm^2)	76.5 ± 17.4	76.7 ± 21.2	NS	73.1 ± 21.4	NS
Size of calcification (mm)	8.5 ± 8.4	7.3 ± 7.3	0.00	7.2 ± 7.2	0.00
Structure scores	2.1 ± 0.7	2.1 ± 0.7	NS	2.0 ± 0.7	NS
Neovascularization scores	1.3 ± 1.1	0.7 ± 0.9	0.00	0.6 ± 0.9	0.00

Note. Values are the mean ± SD; P value, comparison of data before and after treatment. P less than 0.05 was considered statistically significant. NS: not significant.



(a)

(b)

Figure 2: (a) Transverse sonogram shows plentiful calcific plaques (arrows) in the insertion of the Achilles tendon before therapy (outline of Achilles tendon traced by dotted line). (b) The calcific plaques reduced by the 4th week after therapy.

3. Result

Forty-two consecutive patients (29 males and 13 females) with a mean age of 37 years (range, 22–66 years) were enrolled in this study. The average duration of the disease was 10.2 ± 8.7 months (range, 6–37 months). Before ESWT, the mean VISA-A score was 54.0 ± 8.0 (range, 42–67). In the sonographic images, a hypoechoic degenerative region within the thickened insertion of the Achilles tendon was documented in all patients; in addition, 52.4% of patients had calcific plaques, and 73.8% of patients had neovascularization at the end of the Achilles tendon.

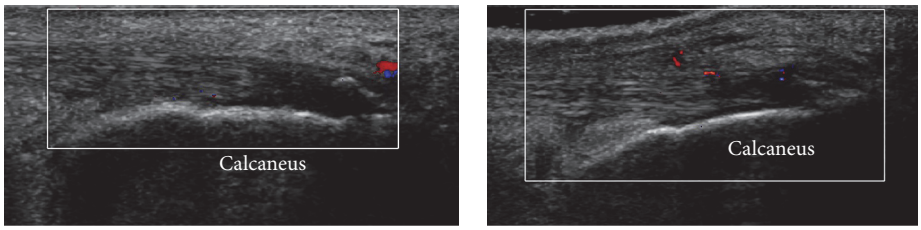
At the 4th and 12th week after therapy, there were significant improvements in VISA-A scores ($P < 0.01$). The mean diameters of calcific plaques decreased at follow-up, as shown by sonography ($P < 0.05$) (Figure 2). The percentage of patients with neovascularization increased to 90.5% at the 4th week and then decreased to 81.0% at the 12th week (Figure 3). The neovascularization scores increased at the 4th week ($P < 0.01$). However, the thickness and CSA of the Achilles tendon and the tendon structure score were unchanged ($P > 0.05$) (Table 1).

The increased VISA-A scores at follow-up did not differ between patients with and without neovascularization or calcific plaques (Figures 4 and 5). The neovascularization scores or the size of calcific plaques at baseline was also not related to the increased VISA-A scores at follow-up. The thickness, CSA, and structure scores of the Achilles tendon at baseline were not associated with the changes in VISA-A scores at follow-up. However, the changes in calcific plaque size or neovascularization scores were related to the changes in VISA-A scores between pre- and posttherapy. There was a correlation between the decreased calcific plaque size and increased VISA-A scores at the 4th week ($r = 0.71$) and the 12th week ($r = 0.68$) ($P < 0.01$). The increased neovascularization scores at the 4th week were related to the increased VISA-A scores at the 4th week ($r = 0.51$) and the 12th week ($r = 0.50$) ($P < 0.01$). At the 12th week, the neovascularization scores decreased compared with the 4th week ($P < 0.01$). The difference in the neovascularization scores was not associated with the difference in VISA-A scores between week 12 and the baseline evaluation or between week 12 and week 4 ($P > 0.05$).

4. Discussion

There is no agreement on the best management for Achilles tendinopathy, with ESWT recently proposed as a viable treatment option [2–6]. In animal and in vivo experiments, some studies have reported that ESWT stimulated the ingrowth of neovascularization at the tendon-bone junction and promoted the inflammatory and catabolic processes [15, 16]. Others have demonstrated that ESWT significantly decreased the nonmyelinated sensory fibers and increased tenocyte proliferation, collagen synthesis, glycosaminoglycan (GAG) content, protein synthesis, and transforming growth factor- (TFG-) β1 [17–19].

In this study, the VISA-A scores of patients increased significantly after ESWT. Changes were also detected by US, such as increased neovascularization scores and decreased size of calcific plaques. In an animal study, Vetrano et al. [17] observed a significant increase in neovessels by biopsy at 4 weeks, and this increase in neovessels persisted until 12 weeks after ESWT. This result is comparable with that we detected at the 4th week. However, we observed a decrease in the neovascularization scores at the 12th week compared with the 4th week. The reduction of neovascularization did not influence the outcome. It is hypothesized that the



(a) (b)

Figure 3: (a) Color Doppler shows no vessels in the insertion of the Achilles tendon before therapy. (b) There was neovascularization at the 4th week after therapy.

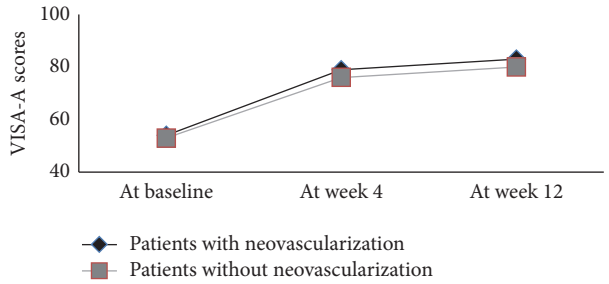


Figure 4: Changes in the VISA-A scores of patients with and without neovascularization at baseline, week 4, and week 12.

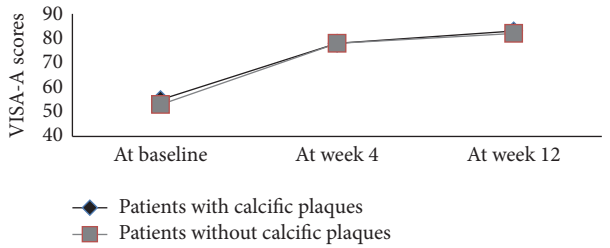


Figure 5: Changes in the VISA-A scores of patients with and without calcific plaques at baseline, week 4, and week 12.

number of blood vessels of the tendon was not reduced, but the percentage of slow blood flow, which cannot be detected with Color Doppler US, increased. The mechanical force of ESWT can disintegrate calcium deposits partially or completely, as applied to the treatment of chronic calcific tendinitis of the shoulder [20]. In our study, the size of calcific plaques also became smaller significantly at the 4th week. Ultrasonography revealed a trend of reduction in the thickness and CSA of Achilles tendon after therapy. However, the difference in the changes between pre- and posttherapy was not statistically significant, in accordance with the results of a study about ESWT for patellar tendinopathy [21]. The short duration of follow-up may be one reason for this result. The tendon structure was not changed significantly in this study. The hypoechoic areas in the tendons observed by US are caused by increased interfibrillar glycosaminoglycans and neovascularization, which can be induced by ESWT [22].

We attempted to evaluate the predictive value of US in ESWT in this study. Unfortunately, none of the categorical and continuous variables of US observed at baseline were associated with the changes in VISA-A scores at follow-up. Thus, we could not use US to predict the outcome of ESWT beforehand. However, at the 4th week, the increased neovascularization scores were related to the increased VISA-A scores. The neovascularization may improve the blood supply, leading to tissue regeneration in tendinopathy.

A limitation of this study is that the length of follow-up was short. In this study, we only used B-mode and Color Doppler US. Recently, sonoelastography was used to evaluate the mechanical properties of tendons [11]. We encourage further studies to assess outcome of therapy with sonoelastography.

5. Conclusion

Ultrasonography can reveal changes in the insertion of the Achilles tendon after ESWT, and the changes in calcific plaque size and neovascularization scores were related to the improvement of VISA-A scores at follow-up. However, the outcome of ESWT in IAT cannot be predicted by the variables observed by US.

Competing Interests

The authors confirm that there is no conflict of interests associated with this publication and there has been no financial support for this work that could have influenced its outcome.

Authors' Contributions

Yi Cheng and Jian Zhang contributed equally to this study.

References

[1] N. Maffulli, J. Wong, and L. C. Almekinders, "Types and epidemiology of tendinopathy," *Clinics in Sports Medicine*, vol. 22, no. 4, pp. 675–692, 2003.

[2] J. P. Furia, "High-energy extracorporeal shock wave therapy as a treatment for insertional achilles tendinopathy," *The American Journal of Sports Medicine*, vol. 34, no. 5, pp. 733–740, 2006.

[3] J. D. Rompe, J. Furia, and N. Maffulli, "Eccentric loading compared with shock wave treatment for chronic insertional

achilles tendinopathy: a randomized, controlled trial," *The Journal of Bone and Joint Surgery—Series A*, vol. 90, no. 1, pp. 52–61, 2008.

[4] R. Fridman, J. D. Cain, L. Weil Jr., and L. Weil Sr., "Extracorporeal shockwave therapy for the treatment of Achilles tendinopathies: a prospective study," *Journal of the American Podiatric Medical Association*, vol. 98, no. 6, pp. 466–468, 2008.

[5] A. Saxena, S. Ramdath, P. O'Halloran, L. Gerdesmeyer, and H. Gollwitzer, "Extra-corporeal pulsed-activated therapy ('EPAT' sound wave) for achilles tendinopathy: A Prospective Study," *The Journal of Foot and Ankle Surgery*, vol. 50, no. 3, pp. 315–319, 2011.

[6] M. C. Vulpiani, D. Trischitta, P. Trovato, M. Vetrano, and A. Ferretti, "Extracorporeal shockwave therapy (ESWT) in Achilles tendinopathy. A long-term follow-up observational study," *The Journal of Sports Medicine and Physical Fitness*, vol. 49, no. 2, pp. 171–176, 2009.

[7] J. L. Y. Leung and J. F. Griffith, "Sonography of chronic Achilles tendinopathy: a case-control study," *Journal of Clinical Ultrasound*, vol. 36, no. 1, pp. 27–32, 2008.

[8] X. Yang, N. D. Pugh, D. P. Coleman, and L. D. M. Nokes, "Are Doppler studies a useful method of assessing neovascularization in human Achilles tendinopathy? A systematic review and suggestions for optimizing machine settings," *Journal of Medical Engineering and Technology*, vol. 34, no. 7-8, pp. 365–372, 2010.

[9] K. Knobloch, "The use of a neovascularization score to predict clinical severity in Achilles tendinopathy," *The American Journal of Sports Medicine*, vol. 36, no. 2, pp. 395–397, 2008.

[10] R.-J. de Vos, A. Weir, L. P. J. Cobben, and J. L. Tol, "The value of power doppler ultrasonography in Achilles tendinopathy: a prospective study," *The American Journal of Sports Medicine*, vol. 35, no. 10, pp. 1696–1701, 2007.

[11] C. C. Ooi, M. E. Schneider, P. Malliaras, M. Chadwick, and D. A. Connell, "Diagnostic Performance of axial-strain sonoelastography in confirming clinically diagnosed achilles tendinopathy: comparison with B-mode ultrasound and color doppler imaging," *Ultrasound in Medicine & Biology*, vol. 41, no. 1, pp. 15–25, 2015.

[12] K. Sunding, M. Fahlström, S. Werner, M. Forssblad, and L. Willberg, "Evaluation of Achilles and patellar tendinopathy with greyscale ultrasound and colour Doppler: using a four-grade scale," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 24, no. 6, pp. 1988–1996, 2016.

[13] L. Öhberg and H. Alfredson, "Ultrasound guided sclerosis of neovessels in painful chronic Achilles tendinosis: Pilot study of a new treatment," *British Journal of Sports Medicine*, vol. 36, no. 3, pp. 173–175, 2002.

[14] J. M. Robinson, J. L. Cook, C. Purdam et al., "The VISA-A questionnaire: a valid and reliable index of the clinical severity of Achilles tendinopathy," *British Journal of Sports Medicine*, vol. 35, no. 5, pp. 335–341, 2001.

[15] C.-J. Wang, F.-S. Wang, K. D. Yang et al., "Shock wave therapy induces neovascularization at the tendon-bone junction. A study in rabbits," *Journal of Orthopaedic Research*, vol. 21, no. 6, pp. 984–989, 2003.

[16] C. M. Waugh, D. Morrissey, E. Jones, G. P. Riley, H. Langberg, and H. R. C. Screen, "In vivo biological response to extracorporeal shockwave therapy in human tendinopathy," *European Cells and Materials*, vol. 29, pp. 268–280, 2015.

[17] M. Vetrano, F. d'Alessandro, M. R. Torrisi, A. Ferretti, M. C. Vulpiani, and V. Visco, "Extracorporeal shock wave therapy promotes cell proliferation and collagen synthesis of primary cultured human tenocytes," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 19, no. 12, pp. 2159–2168, 2011.

[18] Y.-J. Chen, C.-J. Wang, K. D. Yang et al., "Extracorporeal shock waves promote healing of collagenase-induced Achilles tendinitis and increase TGF-β1 and IGF-I expression," *Journal of Orthopaedic Research*, vol. 22, no. 4, pp. 854–861, 2004.

[19] G. Bosch, M. de Mos, R. V. van Binsbergen, H. T. M. van Schie, C. H. A. van de Lest, and P. R. van Weeren, "The effect of focused extracorporeal shock wave therapy on collagen matrix and gene expression in normal tendons and ligaments," *Equine Veterinary Journal*, vol. 41, no. 4, pp. 335–341, 2009.

[20] R. R. Bannuru, N. E. Flavin, E. Vaysbrot, W. Harvey, and T. McAlindon, "High-energy extracorporeal shock-wave therapy for treating chronic calcific tendinitis of the shoulder: a systematic review," *Annals of Internal Medicine*, vol. 160, no. 8, pp. 542–549, 2014.

[21] C.-J. Wang, J.-Y. Ko, Y.-S. Chan, L.-H. Weng, and S.-L. Hsu, "Extracorporeal shockwave for chronic patellar tendinopathy," *American Journal of Sports Medicine*, vol. 35, no. 6, pp. 972–978, 2007.

[22] T. Movin, A. Gad, F. P. Reinholt, and C. Rolf, "Tendon pathology in long-standing achillodynia. Biopsy findings in 40 patients," *Acta Orthopaedica Scandinavica*, vol. 68, no. 2, pp. 170–175, 1997.

Hindawi Publishing Corporation
BioMed Research International
Volume 2016, Article ID 6492597, 9 pages
http://dx.doi.org/10.1155/2016/6492597

Review Article

Achilles Tendinopathy: Current Concepts about the Basic Science and Clinical Treatments

Hong-Yun Li and Ying-Hui Hua

Sports Medicine Center of Fudan University, Department of Sports Medicine and Arthroscopy Surgery, Huashan Hospital, Shanghai 200040, China

Correspondence should be addressed to Ying-Hui Hua; hua_cosm@aliyun.com

Received 30 June 2016; Accepted 20 September 2016

Academic Editor: Jiang Du

Achilles tendinopathy is one of the most frequently ankle and foot overuse injuries, which is a clinical syndrome characterized by the combination of pain, swelling, and impaired performance. The two main categories of Achilles tendinopathy are classified according to anatomical location and broadly include insertional and noninsertional tendinopathy. The etiology of Achilles tendinopathy is multifactorial including both intrinsic and extrinsic factors. Failed healing response and degenerative changes were found in the tendon. The failed healing response includes three different and continuous stages (reactive tendinopathy, tendon disrepair, and degenerative tendinopathy). The histological studies have demonstrated an increased number of tenocytes and concentration of glycosaminoglycans in the ground substance, disorganization and fragmentation of the collagen, and neovascularization. There are variable conservative and surgical treatment options for Achilles tendinopathy. However, there has not been a gold standard of these treatments because of the controversial clinical results between various studies. In the future, new level I researches will be needed to prove the effect of these treatment options.

1. Introduction

The clinical symptoms of pain, swelling, and impaired physical function of Achilles tendon are common in sports and daily life. Traditionally, many terms have been used to describe the disorders, including tendinitis, tendinosis, and paratenonitis. However, recent histopathological studies have found these disorders as a result of a failed healing response, which may cause degenerative changes in the tendon. The failed healing response includes three different and continuous stages (reactive tendinopathy, tendon disrepair, and degenerative tendinopathy) [1–3]. However, inflammatory response is not found in the three stages. In 1998, Maffulli et al. suggested to use the term tendinopathy in order to describe these intratendinous disorders [4].

The two main categories of Achilles tendinopathy are classified according to anatomical location and broadly include insertional (at the calcaneus-Achilles tendon junction) and noninsertional (2 to 6 cm proximal to the insertion of the Achilles tendon into the calcaneus) tendinopathy [5].

2. Epidemiology

Achilles tendinopathy is one of the most frequently ankle and foot overuse injuries [6]. This disorder is more likely to be found in the individuals who participate in the physical activities such as running and jumping. It may affect 9% of recreational runners and cause up to 5% of professional athletes to end their careers [7]. In an epidemiologic investigation of 1394 nonathletes, Achilles tendinopathy was found in 5.6% of the subjects (4% insertional, 3.6% noninsertional, and 1.9% both forms) [8]. In another research, Kvist found that 20% to 25% of Achilles tendinopathy patients had insertional disorder, 66% had noninsertional, and 23% had either retrocalcaneal bursitis or insertional tendinopathy [9].

Chronic Achilles tendinopathy is more common in older people than in young people. In Kvist's study, among 470 patients who had Achilles tendinopathy, only 25% of the patients were young athletes and 10% were younger than 14 years [9]. Moreover, insertional tendinopathy tends to occur in more active persons, whereas noninsertional tendon injury

tends to occur in older, less active, and overweight persons [10].

3. Etiology

The risk factors of Achilles tendinopathy can be divided into intrinsic and extrinsic factors, either alone or combination. Intrinsic factors include biomechanical abnormalities of the lower extremity such as leg length discrepancy hyperpronation, varus deformity of the forefoot, pes cavus and limited mobility of the subtalar joint [10], and systemic conditions such as increasing age [11, 12], inflammatory arthropathies, corticosteroid use, diabetes, hypertension, obesity, gout, hyperostotic conditions [13–16], lipidaemias, aromatase inhibitors, and quinolone antibiotics [17]. Extrinsic factors include excessive mechanical overload and training errors such as increased interval training, abrupt changes in scheduling, excessive hill training, training on hard or sloping surfaces, increased mileage, increased repetitive loading, poor shock absorption, and wedging from uneven wear [10, 15, 17].

4. Tendon Anatomy and Physiology

4.1. Histology and Anatomy. The Achilles tendon originates from the merging of the soleus muscle with the two bellies of the gastrocnemius and it is inserted distally onto the calcaneus. The normal tendon is seen as a fibrillar and generally rounded structure that is white and elastic, because most of them are avascular. Two kinds of cells, tenoblasts and tenocytes, account for 90–95% of the cellular element of the tendon. The cells in a normal Achilles tendon are well organized. The remaining 5–10% of cells are chondrocytes at the entheses and a few synovial cells in the synovial tendon sheath [18–20]. The extracellular matrix between the collagen fibers and tenocytes is composed of glycosaminoglycans, glycoproteins, and proteoglycans, whose high hydrophilicity contributes to the elasticity of the tendon [19, 21]. The tendon is mainly composed of collagen fibers, which make up 90% of the tendon protein or 70% to 80% of the dry weight of the tendon. Type I collagen is the commonest; it forms 95% of tendon collagen, and most of the collagen fibers are aligned longitudinally [22]. Other types of collagen, such as type III, type V, and type XII, are also important in the tendon [21]. Type III collagen has an important role in the healing and developing process. As type III collagen tends to produce smaller, less organized fibrils, this has implications for the mechanical strength of the tendon. Type V collagen is intercalated into the core of the type I collagen fibril, where it forms a template for fibrillogenesis and modulates fibril growth [23]. Collagen type XII has been postulated to integrate adjacent matrix components due to its ability to bind proteoglycans, fibromodulin, and decorin, while interacting with collagen type I fibrils [24]. Elastin accounts for only about 2% of the dry mass of tendon and can undergo up to 200% strain before failure [18]. Collagen forms microfibrils, fibrils, and fibers. A group of fibers constitutes a fascicle. The fascicles unite to form bundles, and they are surrounded by the endotenon, which carries blood vessels, lymphatics, and nerves [20].

The normal blood supply to the tendon is variable between different age and area. The Achilles tendon receives blood supply from three sources: the muscle-tendon junction, the bone-tendon junction, and the length of the tendon. The blood supply to middle portion of the tendon is by way of the surrounding paratenon (the most important blood supply to the tendon) [25, 26]. The most abundant blood supply zone in the tendon is at the tendon insertion, whereas, in the people who are older than 30 years, the most intensely vascularized zone is at the tendon origin. The area of the tendon approximately 2 to 6 cm above the insertion into the calcaneus is the least vascularized zone at all ages [26–29], resulting in limited reparative ability at times of stress or injury.

The nerve supply to the tendon comes from the overlying superficial nerves or from nearby deep nerves, the tibial nerve and its branches [30]. The nerves travel along with the blood vessels. Four types of afferent receptors are found either on the surface or in the tendon. The receptors are (a) Ruffini corpuscles, pressure receptor; (b) Vater-Pacinian corpuscles, movement receptor; (c) Golgi tendon organs, mechanoreceptor; and (d) free nerve endings, acting as pain receptors [26]. Golgi organs are the most abundant receptor at the tendon attachments, which can detect pressure and stretching changes in the Achilles tendon [31].

5. Pathology

In the Achilles tendinopathy patients, the tendon turns out to be thickened, uneven, and brownish. Histological examination of the affected tissue shows no macrophages, neutrophils, or other inflammatory cells. Therefore, traditional terms tendinitis and tendonitis are inappropriate for designating this tendon disorder [32]. As the histological studies demonstrate an increased number of tenocytes and concentration of glycosaminoglycans in the ground substance, disorganization and fragmentation of the collagen, and neovascularization, the term "tendinopathy" seems preferable. The tenocytes present at the site of degeneration have an irregular shape and a higher rate of apoptosis [10, 32–34]. These tissue changes progress to chronic mucoid and/or lipoid degeneration of the tendon with a variable amount of fibrocartilaginous metaplasia and calcium hydroxyapatite deposits [17, 35]. In chronic Achilles tendinopathy, major molecular changes include increased expression of type III collagen, fibronectin, tenascin C, aggrecan, and biglycan. These changes are consistent with repair, but they might also be an adaptive response to changes in mechanical loading because repeated minor strain is thought to be the major precipitating factor in tendinopathy [36].

Healthy tendons are relatively avascular. Neovascularization, a descriptive term for the appearance of abnormal vessels, is one feature of Achilles tendinopathy [37, 38]. Neovascularization and the accompanying neonerves have been hypothesized to be the source of pain in chronic midportion Achilles tendinopathy [3, 39–43]. Moreover, postcapillary venous filling pressures are increased at both the midportion

Achilles tendon and the paratendon tissues. However, the tissue oxygen saturation in tendon does not show any difference between Achilles tendinopathy and normal tendon tissue [44]. Moreover, mechanoreceptors and nerve-related components such as glutamate N-methyl-D-aspartate (NMDA) receptors are present in association with blood vessels in tendinopathic tendons [45–47]. In a rabbit model of tendinopathy, the increased expression of angiogenesis factor (vascular endothelial growth factor (VEGF)) was detected and the pattern of vascularity showed an increase in the number of tendon blood vessels [48].

5.1. Natural Healing of Tendinopathy. Tendon repair involves a sequence of three phases. The first inflammatory phase lasts a few days. Erythrocytes and inflammatory cell migrate to the injury site within the first 24 h. Vasoactive and chemotactic factors are released with increased vascular permeability, initiation of angiogenesis, proliferation of tenocyte, and production of collagen fiber [49]. After a few days, the proliferative phase begins. Synthesis of type III collagen reaches a peak during this stage, which lasts for a few weeks [49]. Water content and glycosaminoglycan concentrations remain high during this stage. Tendon repair coincides with tenocyte proliferation in the epitenon and endotenon, as well as in the tendon sheath [17]. Finally, after approximately 6 weeks, the modeling stage starts. The healing tissue is resized and reshaped [49]. Syntheses of cellularity, collagen, and glycosaminoglycan are decreased. This remodeling phase starts with a fibrous consolidation process. In this period, the repair tissue changes from cellular to fibrous and the collagen fibers align along the direction of the loads applied to the tendon. Then, after the tenth postinjury week, the maturation phase occurs, with gradual change of fibrous tissue to scar-like tendon tissue over the course of one year [46].

Matrix metalloproteinases (MMPs) are a family of proteolytic enzymes, which are classified according to their substrate and primary structure. Previous studies indicated that the MMPs are important in regulating the extracellular matrix network remodeling, and their amounts are changing during Achilles tendon healing process [46].

The quality of the tissue is weakened because of the abnormal healing process, with disordered proliferation of tenocytes, degeneration change of tendon cells, disruption of collagen fibers, and eventually increasing of noncollagenous matrix. If the source of the tendon injury persists, the area of degeneration or the tear may persist or worsen over time [20].

The sources of pain in Achilles tendinopathy are very complicated. The pain may originate from multiple factors. Increased production of prostaglandins (PGs) in matrix, neovascularization in tendon body, tenocyte changes in structure and function, and metabolites changes in tendinopathy are thought to be the sources of pain [47]. Chemical irritants, including cytokines (tumour necrosis factor-alpha (TNFa) and interleukin- (IL-) 1b), signalling molecules (Ca^{2+}, adenosine triphosphate (ATP)), neuropeptides (substance P (SP), neuropeptide Y), and neurotransmitters such as glutamate, have been found to be elevated in tendinopathy and proposed as causing pain [47, 48]. Recent research indicated that the nonneuronal cholinergic system also has been implicated as a factor of pain in chronic tendinopathy [49].

6. Treatment

6.1. Nonoperative Management. In the acute phase, initial rest is the most important. The use of braces and immobilization with a cast or pneumatic walking boot are combined with modified activity [10]. Immobilization is frequently used in the acute setting to control exacerbating factors, but prolonged immobilization should be avoided. Modification of training regimes and specific exercises are necessary. Most patients will be able to return to previous activities. Orthotics may be helpful alongside other modalities of treatment if there is an identifiable malalignment, whereas braces or splints do not appear to improve outcomes in Achilles tendinopathy [50]. A graduated shoe raise or heel lift can alleviate pressure on the insertion by plantar flexing the heel. Hindfoot malalignment associated with insertional disorders can be corrected by insoles, if it is thought to be a provocative factor [51].

Ultrasound is a common prescribed program of physical therapy. In animal studies, ultrasound could stimulate collagen synthesis in tendon fibroblasts and cell division during the period of rapid cell proliferation [52]. Therapeutic ultrasound has been shown to reduce the swelling in the acute inflammatory phase of soft-tissue disorders, relieve pain, and increase function in patients with chronic tendon injuries and may enhance tendon healing [53, 54]. However, due to a lack of new, high quality data on the effect of ultrasound on Achilles tendinopathy, the evidence remains insufficient to support its clinical use [55, 56].

Low level laser therapy (LLLT) could reduce the expression of proinflammatory markers such as IL-6 and TNF-α in gene level [57]. In the cellular level, LLLT may increase collagen production [58], stimulate tenocyte proliferation [59], downregulate MMPs, decrease the capillary flow of neovascularization, and finally preserve the resistance and elasticity of tendon [60, 61]. In a placebo-controlled, double-blind, prospective study of twenty-five patients with a total of forty-one digital flexor tendon repairs, laser therapy could reduce postoperative edema but did not relieve the pain and increase the grip strength or functional results compared with those in control group [62]. Another randomized controlled clinical study indicated that LLLT was not used in treating midportion Achilles tendinopathy [63]. A meta-analysis indicated that LLLT could potentially be effective in treatment of tendinopathy when recommended dosages were used [64]. In the future, new, high quality researches will be needed to prove the effect of LLLT in treatment of Achilles tendinopathy.

The scientific basis of NSAIDs used in chronic tendinopathy is questionable, because the histological examination in the tendinopathic tissue shows no inflammatory cells [21]. The benefits of NSAIDs use are reliving pain in the acute phase and reducing the possibility of leg stiffness [65]. However, there are some studies that indicated that the NSAIDs may inhibit tendon cell migration and proliferation

and impair tendon healing [66]. Some authors believed that NSAIDs had little or no effect on the clinical outcome [5, 67].

Corticosteroid injections are reported to reduce pain and swelling and improve the ultrasound appearance of the tendon. The mechanism behind any positive effect of local steroids on chronic Achilles tendinopathy remains unclear. Some authors have hypothesized that any beneficial effects of corticosteroids in this condition are owed to other local steroid effects rather than suppression of inflammation, including lyses of tendon and peritendon adhesions or alteration of the function of pain generating nociceptor in the region [68]. Corticosteroid injections may have some benefit in the short term, but adverse effects were reported in up to 82% of corticosteroid trials [69]; these include tendon atrophy [69], tendon rupture [70], and decreased tendon strength [71, 72]. Corticosteroid injections could cause vasoconstriction through prostacyclin and adrenoceptors and inhibit nitric oxide synthase, which may be the source of adverse effects [20]. Any possible benefit of corticosteroid injection appears to be outweighed by potential risks. Overall, the evidence to support the injection of corticosteroid in or around the Achilles tendon is insufficient.

Many studies and systematic reviews have found that eccentric exercises are beneficial in the early treatment of noninsertional Achilles tendinopathy [56, 68, 73], but the mechanism of how this exercise works is poorly understood. Theoretically, the reasons of eccentric exercise in reducing pain and improving healing process include more rapid strengthening of the calf muscle, stiffening and lengthening of the myotendinous unit, and decreasing of neovascularization in the tendon [68]. The tensile force generated within the tendon during the exercise temporarily ceases blood flow through the neovessels. With repetition over time, the neovessels are obliterated, along with their associated pain receptors, which lead to the relief of symptoms [55]. The potential harms of eccentric exercise include delayed-onset muscle soreness, exacerbation of the tendinopathy, and muscle injuries [50]. This injury may increase in subjects who are unaccustomed to exercises and if sufficient recovery periods are not allowed. Muscle injuries often occur when athletes are fatigued and strength recovery may take up to 24 hours after exercise [73]. Therefore, the exercise should be taught and monitored by a health professional, such as a physiotherapist or sports medicine physician, capable of ensuring correct biomechanics and of supervising the gradual increase of tendon loading [50].

Conflicting results have been reported for extracorporeal shockwave therapy (ESWT) [20]. How ESWT works is still poorly understood, but it is known to cause selective dysfunction of sensory unmyelinated nerve fibers, alteration in the dorsal root ganglia, and cavitation in interstitial and extracellular disruption, which could promote the healing response [74]. Recently, an in vivo study found that mechanical stimulus provided by ESWT might aid the initiation of tendon regeneration in tendinopathy by promoting proinflammatory and catabolic processes that are associated with removing damaged matrix constituents [75]. In recent research, series randomized placebo-controlled trials have confirmed the high evidence of efficacy of ESWT in chronic Achilles tendinopathy [74]. However, the most effective dose and duration of ESWT are still unknown.

The introduction of platelet-rich plasma (PRP) at the site of tendon injury is thought to facilitate healing because it contains several different growth factors and other cytokines that can stimulate healing of soft tissue [55]. Animal studies indicated that PRP could increase the expression of collagen types I and III and vascular endothelial growth factor and improve the healing and remodeling process of the tendon [76, 77]. A lot of studies demonstrating improved tendon healing using PRP compared with controls, but controversial clinical results exist when using PRP to treat Achilles tendinopathy [78–84]. Ultrasound guidance allows injected PRP into the tendon with great accuracy [79, 84]. However, the evidence to support the use of PRP in the management of Achilles tendinopathy is insufficient and level I, controlled, randomized studies are still needed in the future researches.

Intratendinous hyperosmolar dextrose (prolotherapy) is thought to produce a local inflammatory response and increase in tendon strength. Clinical results indicated that intratendinous injections of hyperosmolar dextrose could reduce the pain at rest and during tendon-loading activities in patients with chronic of the Achilles tendinopathy [85, 86]. Moreover, after injection of dextrose, there were reductions in the size and severity of hypoechoic regions and intratendinous tears and improvements in neovascularity [86].

Nitric oxide is a small-free radical generated by a family of enzymes, the nitric oxide synthases. It can induce apoptosis in inflammatory cells and cause angiogenesis and vasodilation. Moreover, oxygen free radicals can stimulate fibroblast proliferation [87]. Nitric oxide can enhance tendon healing. Inhibition of nitric oxide synthase can reduce healing process, which resulted in a decreased cross-sectional area and reduced failure load [48]. Theoretical, topical glyceryl trinitrate (GTN) application could increase local tissue nitric oxide concentrations, which are believed to improve fibroblast function and wound healing [68]. Acute GTN facilitates capillary venous outflow in painful Achilles tendons. However, in another study, capillary blood-flow and tendon oxygenation remain unchanged following GTN application [88]. A randomized double-blind placebo-controlled study demonstrated improvements in symptoms using GTN patches [89]. Another randomized controlled trial found no difference between groups in scores between the two groups for pain or disability. Moreover, there has not appeared to be any histological or immunohistochemical change between groups [90].

Cryotherapy might play a role in reducing the increased capillary blood flow in Achilles tendinopathy, reducing the metabolic rate of the tendon and applying for relief of pain [91, 92]. However, recent evidence in upper limb tendinopathy indicated that the addition of ice did not offer any advantage over an exercise program consisting of eccentric and static stretching exercises [93].

The sclerosing agent that selectively targets the vascular may cause thrombosis of the vessel. As the concomitant sensory nerves have been implicated as possible pain generators, to destroy local nerves adjacent to neovessels may decrease pain [55]. As vessel number has been shown to

correlate with tendon thickness, treatment that decreases vessel number is likely to also affect the tendon thickness. Moreover, the sclerosing agent injected at multiple sites around the tendon and neovessels initiates a local inflammatory response, which induces a proliferation of fibroblasts and synthesis of collagen. Therefore, a stronger, more organized tendon could be produced [52]. Early reports using sclerosing agent injected under Doppler ultrasound guidance into the abnormal vessels on the ventral aspect of the Achilles tendon demonstrated significant improvements in pain and function scores [54, 94]. However, due to the conflicting results, high quality evidence to make a recommendation for sclerosing injections is needed.

Deep friction massage (DFM) and tendon mobilization may also be helpful in the treatment of Achilles tendinopathy. DFM has been advocated for tendinopathy and paratendinopathy. Friction has been shown to increase protein output of tendon cells [95]. In combination with stretching, deep friction massage helps to restore tissue elasticity and reduce the strain in the muscle-tendon unit [52, 95]. Future randomized comparison studies are necessary to compare DFM in isolation with other modes of treatment.

Aprotinin is a broad spectrum serine protease inhibitor capable of blocking trypsin, plasmin, kallikrein, and a range of MMPs [96]. Most previous studies using Aprotinin injection in the management of Achilles tendinopathy showed a trend towards improved clinical results [97, 98]. The major potential negative of using Aprotinin is the side effect of allergy [51], but the allergic reactions can be reduced by minimizing repeat injections and recommending a delay of at least 6 weeks between injections [96].

7. Surgical Treatment

7.1. Noninsertional Achilles Tendinopathy. The goal of surgery is to resect degenerative tissue, stimulate tendon healing by means of controlled, low-grade trauma and/or augment the Achilles tendon with grafts. It has been suggested that noninvasive treatment should be tried for at least 4 months prior to operative interventions [10]. Conventional surgical treatment has consisted of open release of adhesions with or without resection of the paratenon. If >50% of the tendon has been debrided, augmentation or reconstruction is recommended [10].

Several operative treatments include percutaneous longitudinal tenotomies, minimally invasive tendon stripping, open tenosynovectomies, open debridement and tubularization, and tendon augmentation with flexor hallucis longus (FHL). Long-term results of operative interventions are promising but need more fair evidence to support the clinical use.

Complications are common in surgical procedure. In a large series of 432 consecutive patients, Paavola et al. reported wound necrosis in 3%, superficial infection in 2.5%, and sural nerve injury in 1%, with further complications including haematoma, seroma, and thrombosis. Therefore, the overall complication rate was 11% and reoperation rate was 3% [99].

7.2. Insertional Achilles Tendinopathy. Patients who do not respond to conservative treatment may need operative manage. No consensus exists regarding the duration before surgery, though most clinicians consider 3 to 6 months the minimum time necessary to evaluate the effect of conservative treatment [100].

The operative strategy for insertional Achilles tendinopathy is removal of the degenerative tendon and associated calcification, excision of the inflamed retrocalcaneal bursa, resection of the prominent posterior calcaneal prominence, reattachment of the insertion as required, and/or augmentation of the tendo-Achilles with a tendon transfer/graft [101, 102]. Calcaneoplasty and resection of the retrocalcaneal bursa can be performed endoscopically [102].

Biomechanical and clinical data suggest that 50% of the tendon attachment can be safely debrided without compromise of the insertion strength or risk of rerupture. When greater than 50% of the Achilles tendon is detached from the calcaneus, suture anchors are recommended to reattach the residual tendon. With extensive insertional, Achilles tendon disease and/or when greater than 75% of the tendon is excised, augmentation with local tissue, such as the flexor hallucis longus tendon and semitendinosus tendon, is advisable [102].

8. Conclusion

Achilles tendinopathy is a clinical syndrome characterized by the combination of pain, swelling, and impaired performance. The etiology of Achilles tendinopathy is multifactorial including intrinsic and extrinsic factors. The histological studies demonstrate an increased number of tenocytes and concentration of glycosaminoglycans in the ground substance, disorganization and fragmentation of the collagen, and neovascularization. The sources of pain in Achilles tendinopathy are very complicated. The pain may originate from multiple factors. There are variable conservative and surgical treatment options for Achilles tendinopathy. However, there is no gold standard of the treatments because of the controversial clinical results between various studies. In the future, more new level I researches are needed to prove the effect of these treatment options.

Competing Interests

The authors declare that there are no competing interests regarding the publication of this paper.

References

[1] M. Abate, K. G. Silbernagel, C. Siljeholm et al., "Pathogenesis of tendinopathies: inflammation or degeneration?" *Arthritis Research & Therapy*, vol. 11, no. 3, p. 235, 2009.

[2] J. L. Cook and C. R. Purdam, "Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy," *British Journal of Sports Medicine*, vol. 43, no. 6, pp. 409–416, 2009.

[3] U. G. Longo, M. Ronga, and N. Maffulli, "Achilles tendinopathy," *Sports Medicine and Arthroscopy Review*, vol. 17, no. 2, pp. 112–126, 2009.

[4] N. Maffulli, K. M. Khan, and G. Puddu, "Overuse tendon conditions: time to change a confusing terminology," *Arthroscopy*, vol. 14, no. 8, pp. 840–843, 1998.

[5] L. C. Almekinders and J. D. Temple, "Etiology, diagnosis, and treatment of tendonitis: an analysis of the literature," *Medicine & Science in Sports & Exercise*, vol. 30, no. 8, pp. 1183–1190, 1998.

[6] S. Sobhani, R. Dekker, K. Postema, and P. U. Dijkstra, "Epidemiology of ankle and foot overuse injuries in sports: a systematic review," *Scandinavian Journal of Medicine & Science in Sports*, vol. 23, no. 6, pp. 669–686, 2013.

[7] J. Lysholm and J. Wiklander, "Injuries in runners," *American Journal of Sports Medicine*, vol. 15, no. 2, pp. 168–171, 1987.

[8] U. Waldecker, G. Hofmann, and S. Drewitz, "Epidemiologic investigation of 1394 feet: coincidence of hindfoot malalignment and Achilles tendon disorders," *Foot and Ankle Surgery*, vol. 18, no. 2, pp. 119–123, 2012.

[9] M. Kvist, "Achilles tendon injuries in athletes," *Annales Chirurgiae et Gynaecologiae*, vol. 80, no. 2, pp. 188–201, 1991.

[10] T. A. Irwin, "Current concepts review: insertional Achilles tendinopathy," *Foot and Ankle International*, vol. 31, no. 10, pp. 933–939, 2010.

[11] R. Strocchi, V. De Pasquale, S. Guizzardi et al., "Human achilles tendon: morphological and morphometric variations as a function of age," *Foot and Ankle*, vol. 12, no. 2, pp. 100–104, 1991.

[12] D. J. Tuite, P. A. F. H. Renström, and M. O'Brien, "The aging tendon," *Scandinavian Journal of Medicine and Science in Sports*, vol. 7, no. 2, pp. 72–77, 1997.

[13] M. Fahlström, R. Lorentzon, and H. Alfredson, "Painful conditions in the Achilles tendon region: a common problem in middle-aged competitive badminton players," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 10, no. 1, pp. 57–60, 2002.

[14] G. B. Holmes and J. Lin, "Etiologic factors associated with symptomatic achilles tendinopathy," *Foot and Ankle International*, vol. 27, no. 11, pp. 952–959, 2006.

[15] B. Murtaugh and J. M. Ihm, "Eccentric training for the treatment of tendinopathies," *Current Sports Medicine Reports*, vol. 12, no. 3, pp. 175–182, 2013.

[16] M. Paavola, P. Kannus, T. A. H. Järvinen, K. Khan, L. Józsa, and M. Järvinen, "Achilles tendinopathy," *Journal of Bone and Joint Surgery A*, vol. 84, no. 11, pp. 2062–2076, 2002.

[17] T. Kirchgesner, A. Larbi, P. Omoumi et al., "Drug-induced tendinopathy: from physiology to clinical applications," *Joint Bone Spine*, vol. 81, no. 6, pp. 485–492, 2014.

[18] E. Ippolito, P. G. Natali, F. Postacchini, L. Accinni, and C. De Martino, "Morphological, immunochemical, and biochemical study of rabbit Achilles tendon at various ages," *The Journal of Bone & Joint Surgery—American Volume*, vol. 62, no. 4, pp. 583–598, 1980.

[19] P. Kannus and L. Józsa, "Histopathological changes preceding spontaneous rupture of a tendon: a controlled study of 891 patients," *The Journal of Bone and Joint Surgery A*, vol. 73, no. 10, pp. 1507–1525, 1991.

[20] A. J. Roche and J. D. F. Calder, "Achilles tendinopathy: a review of the current concepts of treatment," *The Bone & Joint Journal*, vol. 95, no. 10, pp. 1299–1307, 2013.

[21] W. J. Ribbans and M. Collins, "Pathology of the tendo Achillis: do our genes contribute?" *The Journal of Bone and Joint Surgery B*, vol. 95, no. 3, pp. 305–313, 2013.

[22] N. Maffulli, P. M. Binfield, and J. B. King, "Tendon problems in athletic individuals," *The Journal of Bone & Joint Surgery—American Volume*, vol. 80, no. 1, pp. 142–144, 1998.

[23] D. T. Kirkendall and W. E. Garrett, "Function and biomechanics of tendons," *Scandinavian Journal of Medicine & Science in Sports*, vol. 7, no. 2, pp. 62–66, 1997.

[24] M. O'Brien, "The anatomy of the achilles tendon," *Foot and Ankle Clinics*, vol. 10, no. 2, pp. 225–238, 2005.

[25] I. M. Ahmed, M. Lagopoulos, P. McConnell, R. W. Soames, and G. K. Sefton, "Blood supply of the achilles tendon," *Journal of Orthopaedic Research*, vol. 16, no. 5, pp. 591–596, 1998.

[26] J. M. McShane, B. Ostick, and F. McCabe, "Noninsertional Achilles tendinopathy: pathology and management," *Current Sports Medicine Reports*, vol. 6, no. 5, pp. 288–292, 2007.

[27] V. Niculescu and P. Matusz, "The clinical importance of the calcaneal tendon vasculature (tendo calcaneus)," *Morphologie et Embryologie*, vol. 34, no. 1, pp. 5–8, 1988.

[28] M. O'Brien, "Functional anatomy and physiology of tendons," *Clinics in Sports Medicine*, vol. 11, no. 3, pp. 505–520, 1992.

[29] S. M. Lephart, D. M. Pincivero, J. L. Giraldo, and F. H. Fu, "The role of proprioception in the management and rehabilitation of athletic injuries," *American Journal of Sports Medicine*, vol. 25, no. 1, pp. 130–137, 1997.

[30] C. J. Pearce, M. Ismail, and J. D. Calder, "Is apoptosis the cause of noninsertional achilles tendinopathy?" *The American Journal of Sports Medicine*, vol. 37, no. 12, pp. 2440–2444, 2009.

[31] B. Dallaudière, M. Lempicki, L. Pesquer et al., "Efficacy of intra-tendinous injection of platelet-rich plasma in treating tendinosis: comprehensive assessment of a rat model," *European Radiology*, vol. 23, no. 10, pp. 2830–2837, 2013.

[32] J. G. P. Williams, "Achilles tendon lesions in sport," *Sports Medicine*, vol. 16, no. 3, pp. 216–220, 1993.

[33] J. S. Yu, J. E. Popp, C. C. Kaeding, and J. Lucas, "Correlation of MR imaging and pathologic findings in athletes undergoing surgery for chronic patellar tendinitis," *American Journal of Roentgenology*, vol. 165, no. 1, pp. 115–118, 1995.

[34] H. Alfredson, D. Bjur, K. Thorsen, and R. Lorentzon, "High intratendinous lactate levels in painful chronic Achilles tendinosis. An investigation using microdialysis technique," *Journal of Orthopaedic Research*, vol. 20, no. 5, pp. 934–938, 2002.

[35] K. Knobloch, "The role of tendon microcirculation in Achilles and patellar tendinopathy," *Journal of Orthopaedic Surgery and Research*, vol. 3, no. 1, article 18, 2008.

[36] D. Kader, A. Saxena, T. Movin, and N. Maffulli, "Achilles tendinopathy: some aspects of basic science and clinical management," *British Journal of Sports Medicine*, vol. 36, no. 4, pp. 239–249, 2002.

[37] P. Sharma and N. Maffulli, "Understanding and managing Achilles tendinopathy," *British Journal of Hospital Medicine*, vol. 67, no. 2, pp. 64–67, 2006.

[38] P. Sharma and N. Maffulli, "Tendinopathy and tendon injury: the future," *Disability and Rehabilitation*, vol. 30, no. 20–22, pp. 1733–1745, 2008.

[39] A. M. Vora, M. S. Myerson, F. Oliva, and N. Maffulli, "Tendinopathy of the main body of the achilles tendon," *Foot and Ankle Clinics*, vol. 10, no. 2, pp. 293–308, 2005.

[40] M. S. Zafar, A. Mahmood, and N. Maffulli, "Basic science and clinical aspects of Achilles tendinopathy," *Sports Medicine and Arthroscopy Review*, vol. 17, no. 3, pp. 190–197, 2009.

[41] K. Knobloch, R. Kraemer, A. Lichtenberg et al., "Achilles tendon and paratendon microcirculation in midportion and

insertional tendinopathy in athletes," *The American Journal of Sports Medicine*, vol. 34, no. 1, pp. 92–97, 2006.

[42] H. Alfredson, K. Thorsen, and R. Lorentzon, "In situ microdialysis in tendon tissue: high levels of glutamate, but not prostaglandin E2 in chronic Achilles tendon pain," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 7, no. 6, pp. 378–381, 1999.

[43] G. Andersson, P. Danielson, H. Alfredson, and S. Forsgren, "Nerve-related characteristics of ventral paratendinous tissue in chronic Achilles tendinosis," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 15, no. 10, pp. 1272–1279, 2007.

[44] C. Spang, V. M. Harandi, H. Alfredson et al., "Marked innervation but also signs of nerve degeneration in between the Achilles and plantaris tendons and presence of innervation within the plantaris tendon in midportion Achilles tendinopathy," *Journal of Musculoskeletal & Neuronal Interactions*, vol. 15, no. 2, pp. 197–206, 2015.

[45] G. Andersson, S. Forsgren, A. Scott et al., "Tenocyte hypercellularity and vascular proliferation in a rabbit model of tendinopathy: contralateral effects suggest the involvement of central neuronal mechanisms," *British Journal of Sports Medicine*, vol. 45, no. 5, pp. 399–406, 2011.

[46] P. Sharma and N. Maffulli, "Biology of tendon injury: healing, modeling and remodeling," *Journal of Musculoskeletal Neuronal Interactions*, vol. 6, no. 2, pp. 181–190, 2006.

[47] E. Rio, L. Moseley, C. Purdam et al., "The pain of tendinopathy: physiological or pathophysiological?" *Sports Medicine*, vol. 44, no. 1, pp. 9–23, 2014.

[48] P. Sharma and N. Maffulli, "Tendon injury and tendinopathy: healing and repair," *The Journal of Bone and Joint Surgery A*, vol. 87, no. 1, pp. 187–202, 2005.

[49] S. Forsgren, H. Alfredson, and G. Andersson, "Further proof of the existence of a non-neuronal cholinergic system in the human Achilles tendon: presence of the AChRα7 receptor in tendon cells and cells in the peritendinous tissue," *International Immunopharmacology*, vol. 29, no. 1, pp. 195–200, 2015.

[50] A. Scott, E. Huisman, and K. Khan, "Conservative treatment of chronic Achilles tendinopathy," *CMAJ: Canadian Medical Association journal*, vol. 183, no. 10, pp. 1159–1165, 2011.

[51] N. J. Rukin and N. Maffulli, "Systemic allergic reactions to aprotinin injection around the Achilles tendon," *Journal of Science and Medicine in Sport*, vol. 10, no. 5, pp. 320–322, 2007.

[52] J. D. Rompe, J. P. Furia, and N. Maffulli, "Mid-portion Achilles tendinopathy-current options for treatment," *Disability and Rehabilitation*, vol. 30, no. 20–22, pp. 1666–1676, 2008.

[53] T. M. Best, B. Moore, P. Jarit, C. T. Moorman, and G. K. Lewis, "Sustained acoustic medicine: wearable, long duration ultrasonic therapy for the treatment of tendinopathy," *The Physician and Sportsmedicine*, vol. 43, no. 4, pp. 366–374, 2015.

[54] L. Willberg, K. Sunding, L. Öhberg, M. Forssblad, M. Fahlström, and H. Alfredson, "Sclerosing injections to treat midportion Achilles tendinosis: a randomised controlled study evaluating two different concentrations of Polidocanol," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 16, no. 9, pp. 859–864, 2008.

[55] X. F. Courville, M. P. Coe, and P. J. Hecht, "Current concepts review: noninsertional Achilles tendinopathy," *Foot and Ankle International*, vol. 30, no. 11, pp. 1132–1142, 2009.

[56] V. Rowe, S. Hemmings, C. Barton, P. Malliaras, N. Maffulli, and D. Morrissey, "Conservative management of midportion achilles tendinopathy: a mixed methods study, integrating systematic review and clinical reasoning," *Sports Medicine*, vol. 42, no. 11, pp. 941–967, 2012.

[57] R. Torres-Silva, R. A. B. Lopes-Martins, J. M. Bjordal et al., "The low level laser therapy (LLLT) operating in 660 nm reduce gene expression of inflammatory mediators in the experimental model of collagenase-induced rat tendinitis," *Lasers in Medical Science*, vol. 30, no. 7, pp. 1985–1990, 2014.

[58] M. Xavier, R. A. De Souza, V. A. Pires et al., "Low-level light-emitting diode therapy increases mRNA expressions of IL-10 and type I and III collagens on Achilles tendinitis in rats," *Lasers in Medical Science*, vol. 29, no. 1, pp. 85–90, 2014.

[59] W.-C. Tsai, J.-W. Cheng, J.-L. Chen et al., "Low-level laser irradiation stimulates tenocyte proliferation in association with increased NO synthesis and upregulation of PCNA and cyclins," *Lasers in Medical Science*, vol. 29, no. 4, pp. 1377–1384, 2014.

[60] R. L. Marcos, G. Arnold, V. Magnenet, R. Rahouadj, J. Magdalou, and R. Á. B. Lopes-Martins, "Biomechanical and biochemical protective effect of low-level laser therapy for Achilles tendinitis," *Journal of the Mechanical Behavior of Biomedical Materials*, vol. 29, pp. 272–285, 2014.

[61] R. L. Marcos, E. C. P. Leal-Junior, G. Arnold et al., "Low-level laser therapy in collagenase-induced Achilles tendinitis in rats: analyses of biochemical and biomechanical aspects," *Journal of Orthopaedic Research*, vol. 30, no. 12, pp. 1945–1951, 2012.

[62] N. Özkan, L. Altan, Ü. Bingöl, S. Akin, and M. Yurtkuran, "Investigation of the supplementary effect of GaAs laser therapy on the rehabilitation of human digital flexor tendons," *Journal of Clinical Laser Medicine & Surgery*, vol. 22, no. 2, pp. 105–110, 2004.

[63] S. Tumilty, S. McDonough, D. A. Hurley, and G. D. Baxter, "Clinical effectiveness of low-level laser therapy as an adjunct to eccentric exercise for the treatment of Achilles' tendinopathy: a randomized controlled trial," *Archives of Physical Medicine and Rehabilitation*, vol. 93, no. 5, pp. 733–739, 2012.

[64] S. Tumilty, J. Munn, S. McDonough, D. A. Hurley, J. R. Basford, and G. D. Baxter, "Low level laser treatment of tendinopathy: a systematic review with meta-analysis," *Photomedicine and Laser Surgery*, vol. 28, no. 1, pp. 3–16, 2010.

[65] J. Maquirriain and A. Kokalj, "Management of acute Achilles tendinopathy: effect of etoricoxib on pain control and leg stiffness," *Georgian Medical News*, no. 222, pp. 36–43, 2013.

[66] W.-C. Tsai, C.-C. Hsu, S.-W. Chou, C.-Y. Chung, J. Chen, and J.-H. Pang, "Effects of celecoxib on migration, proliferation and collagen expression of tendon cells," *Connective Tissue Research*, vol. 48, no. 1, pp. 46–51, 2007.

[67] M. Åström and N. Westlin, "No effect of piroxicam on achilles tendinopathy: a randomized study of 70 patients," *Acta Orthopaedica Scandinavica*, vol. 63, no. 6, pp. 631–634, 1992.

[68] R. A. Magnussen, W. R. Dunn, and A. B. Thomson, "Nonoperative treatment of midportion achilles tendinopathy: a systematic review," *Clinical Journal of Sport Medicine*, vol. 19, no. 1, pp. 54–64, 2009.

[69] L. Hart, "Corticosteroid and other injections in the management of tendinopathies: a review," *Clinical Journal of Sport Medicine*, vol. 21, no. 6, pp. 540–541, 2011.

[70] R. S. Kearney, N. Parsons, D. Metcalfe, and M. L. Costa, "Injection therapies for Achilles tendinopathy," *The Cochrane Database of Systematic Reviews*, vol. 5, Article ID CD010960, 2015.

[71] B. T. Haraldsson, H. Langberg, P. Aagaard et al., "Corticosteroids reduce the tensile strength of isolated collagen fascicles,"

The American Journal of Sports Medicine, vol. 34, no. 12, pp. 1992–1997, 2006.

[72] R. Hugate, J. Pennypacker, M. Saunders, and P. Juliano, "The effects of intratendinous and retrocalcaneal intrabursal injections of corticosteroid on the biomechanical properties of rabbit Achilles tendons," *The Journal of Bone & Joint Surgery—American Volume*, vol. 86, no. 4, pp. 794–801, 2004.

[73] A. Meyer, S. Tumilty, and G. D. Baxter, "Eccentric exercise protocols for chronic non-insertional Achilles tendinopathy: how much is enough?" *Scandinavian Journal of Medicine and Science in Sports*, vol. 19, no. 5, pp. 609–615, 2009.

[74] L. Gerdesmeyer, R. Mittermayr, M. Fuerst et al., "Current evidence of extracorporeal shock wave therapy in chronic Achilles tendinopathy," *International Journal of Surgery*, vol. 24, pp. 154–159, 2015.

[75] C. M. Waugh, D. Morrissey, E. Jones, G. P. Riley, H. Langberg, and H. R. C. Screen, "In vivo biological response to extracorporeal shockwave therapy in human tendinopathy," *European Cells and Materials*, vol. 29, pp. 268–280, 2015.

[76] J. C. González, C. López, M. E. Álvarez, J. E. Pérez, and J. U. Carmona, "Autologous leukocyte-reduced platelet-rich plasma therapy for Achilles tendinopathy induced by collagenase in a rabbit model," *Scientific Reports*, vol. 6, article 19623, 2016.

[77] J.-F. Kaux, P. Drion, J. L. Croisier, and J. M. Crielaard, "Tendinopathies and platelet-rich plasma (PRP): from preclinical experiments to therapeutic use," *Journal of Stem Cells and Regenerative Medicine*, vol. 11, no. 1, pp. P7–P17, 2015.

[78] B. Di Matteo, G. Filardo, E. Kon, and M. Marcacci, "Platelet-rich plasma: evidence for the treatment of patellar and Achilles tendinopathy—a systematic review," *Musculoskeletal Surgery*, vol. 99, no. 1, pp. 1–9, 2015.

[79] G. Ferrero, E. Fabbro, D. Orlandi et al., "Ultrasound-guided injection of platelet-rich plasma in chronic Achilles and patellar tendinopathy," *Journal of Ultrasound*, vol. 15, no. 4, pp. 260–266, 2012.

[80] G. Filardo, E. Kon, B. Di Matteo et al., "Platelet-rich plasma injections for the treatment of refractory Achilles tendinopathy: results at 4 years," *Blood Transfusion*, vol. 12, no. 4, pp. 533–540, 2014.

[81] M. Guelfi, A. Pantalone, D. Vanni, M. Abate, M. G. B. Guelfi, and V. Salini, "Long-term beneficial effects of platelet-rich plasma for non-insertional Achilles tendinopathy," *Foot and Ankle Surgery*, vol. 21, no. 3, pp. 178–181, 2015.

[82] C. D. Murawski, N. A. Smyth, H. Newman, and J. G. Kennedy, "A single platelet-rich plasma injection for chronic midsubstance Achilles tendinopathy: a retrospective preliminary analysis," *Foot and Ankle Specialist*, vol. 7, no. 5, pp. 372–376, 2014.

[83] V. Salini, D. Vanni, A. Pantalone, and M. Abate, "Platelet rich plasma therapy in non-insertional Achilles tendinopathy: the efficacy is reduced in 60-years old people compared to young and middle-age individuals," *Frontiers in Aging Neuroscience*, vol. 7, article 228, 2015.

[84] K. Mautner, R. E. Colberg, G. Malanga et al., "Outcomes after ultrasound-guided platelet-rich plasma injections for chronic tendinopathy: a multicenter, retrospective review," *PM&R*, vol. 5, no. 3, pp. 169–175, 2013.

[85] N. J. Maxwell, M. B. Ryan, J. E. Taunton, J. H. Gillies, and A. D. Wong, "Sonographically guided intratendinous injection of hyperosmolar dextrose to treat chronic tendinosis of the Achilles tendon: a pilot study," *AJR. American Journal of Roentgenology*, vol. 189, no. 4, pp. W215–W220, 2007.

[86] M. Ryan, A. Wong, and J. Taunton, "Favorable outcomes after sonographically guided intratendinous injection of hyperosmolar dextrose for chronic insertional and midportion achilles tendinosis," *American Journal of Roentgenology*, vol. 194, no. 4, pp. 1047–1053, 2010.

[87] A. R. Bokhari and G. A. C. Murrell, "The role of nitric oxide in tendon healing," *Journal of Shoulder and Elbow Surgery*, vol. 21, no. 2, pp. 238–244, 2012.

[88] R. Osadnik, J. Redeker, R. Kraemer, P. M. Vogt, and K. Knobloch, "Microcirculatory effects of topical glyceryl trinitrate on the Achilles tendon microcirculation in patients with previous Achilles tendon rupture," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 18, no. 7, pp. 977–981, 2010.

[89] J. A. Paoloni, R. C. Appleyard, J. Nelson, and G. A. C. Murrell, "Topical glyceryl trinitrate treatment of chronic noninsertional achilles tendinopathy. A randomized, double-blind, placebo-controlled trial," *The Journal of Bone & Joint Surgery—American Volume*, vol. 86, no. 5, pp. 916–922, 2004.

[90] T. P. C. Kane, M. Ismail, and J. D. F. Calder, "Topical glyceryl trinitrate and noninsertional Achilles tendinopathy: a clinical and cellular investigation," *The American Journal of Sports Medicine*, vol. 36, no. 6, pp. 1160–1163, 2008.

[91] K. Knobloch, R. Grasemann, M. Spies, and P. M. Vogt, "Intermittent KoldBlue cryotherapy of 3x10 min changes mid-portion Achilles tendon microcirculation," *British Journal of Sports Medicine*, vol. 41, no. 6, article e4, 2007.

[92] K. Knobloch, R. Grasemann, M. Spies, and P. M. Vogt, "Midportion achilles tendon microcirculation after intermittent combined cryotherapy and compression compared with cryotherapy alone: a randomized trial," *The American Journal of Sports Medicine*, vol. 36, no. 11, pp. 2128–2138, 2008.

[93] P. Manias and D. Stasinopoulos, "A controlled clinical pilot trial to study the effectiveness of ice as a supplement to the exercise programme for the management of lateral elbow tendinopathy," *British Journal of Sports Medicine*, vol. 40, no. 1, pp. 81–85, 2006.

[94] B. Lind, L. Öhberg, and H. Alfredson, "Sclerosing polidocanol injections in mid-portion Achilles tendinosis: remaining good clinical results and decreased tendon thickness at 2-year follow-up," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 14, no. 12, pp. 1327–1332, 2006.

[95] M. F. Joseph, K. Taft, M. Moskwa, and C. R. Denegar, "Deep friction massage to treat tendinopathy: a systematic review of a classic treatment in the face of a new paradigm of understanding," *Journal of Sport Rehabilitation*, vol. 21, no. 4, pp. 343–353, 2012.

[96] J. Orchard, A. Massey, J. Rimmer, J. Hofman, and R. Brown, "Delay of 6 weeks between aprotinin injections for tendinopathy reduces risk of allergic reaction," *Journal of Science and Medicine in Sport*, vol. 11, no. 5, pp. 473–480, 2008.

[97] R. Brown, J. Orchard, M. Kinchington, A. Hooper, and G. Nalder, "Aprotinin in the management of Achilles tendinopathy: a randomised controlled trial," *British Journal of Sports Medicine*, vol. 40, no. 3, pp. 275–279, 2006.

[98] N. Maffulli, F. Spiezia, U. G. Longo, V. Denaro, and G. D. Maffulli, "High volume image guided injections for the management of chronic tendinopathy of the main body of the Achilles tendon," *Physical Therapy in Sport*, vol. 14, no. 3, pp. 163–167, 2013.

[99] M. Paavola, S. Orava, J. Leppilahti, P. Kannus, and M. Järvinen, "Chronic achilles tendon overuse injury: complications after surgical treatment. An analysis of 432 consecutive patients," *The*

American Journal of Sports Medicine, vol. 28, no. 1, pp. 77–82, 2000.

[100] R. Kearney and M. L. Costa, "Insertional achilles tendinopathy management: a systematic review," *Foot and Ankle International*, vol. 31, no. 8, pp. 689–694, 2010.

[101] J. I. Wiegerinck, G. M. Kerkhoffs, M. N. van Sterkenburg, I. N. Sierevelt, and C. N. van Dijk, "Treatment for insertional Achilles tendinopathy: a systematic review," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 21, no. 6, pp. 1345–1355, 2013.

[102] M. J. DeOrio and M. E. Easley, "Surgical strategies: insertional achilles tendinopathy," *Foot & Ankle International*, vol. 29, no. 5, pp. 542–550, 2008.

Hindawi Publishing Corporation
BioMed Research International
Volume 2016, Article ID 1416971, 8 pages
http://dx.doi.org/10.1155/2016/1416971

Clinical Study

Surgical Strategy for the Chronic Achilles Tendon Rupture

Yangjing Lin, Liu Yang, Li Yin, and Xiaojun Duan

Center for Joint Surgery, Southwest Hospital, Third Military Medical University, Chongqing 400038, China

Correspondence should be addressed to Xiaojun Duan; dxj9@163.com

Received 29 June 2016; Accepted 4 October 2016

Academic Editor: Ying-Hui Hua

Background. Chronic Achilles tendon rupture is usually misdiagnosed and treated improperly. This study aims to better understand the treatment of chronic Achilles tendon rupture. *Methods*. Patients who were not able to perform a single-limb heel rise were chosen. Pre- and postoperative magnetic resonance imaging (MRI) were conducted. By evaluating the presence or absence of Achilles tendon stumps and the gap length of rupture, V-Y advancement, gastrocnemius fascial turndown flap, or flexor halluces longus tendon transfer were selected for tendon repair. The function of ankle and foot was assessed by American Orthopaedic Foot & Ankle Society (AOFAS) ankle-hindfoot scores and Achilles Tendon Total Rupture Score (ATRS). *Results*. Twenty-nine patients were followed up. One patient had superficial incision infection, which was healed after debridement and oral antibiotics. Three months postoperatively, MRI showed some signs of inflammation, which disappeared at one or two years postoperatively. All patients were able to perform a single-limb heel rise. Mean AOFAS scores and ATRS scores were increased at the latest follow-up. *Conclusion*. Surgical options can be determined by evaluating the presence of the Achilles tendon stumps and the gap length, which can avoid using the nearby tendon and yield satisfactory functional results.

1. Introduction

The Achilles tendon is one of the most commonly ruptured tendons of the lower extremity [1–4]. Clinically, acute Achilles tendon rupture can be easily diagnosed and cured; however, a significant number of cases are still neglected without treatments. Chronic Achilles tendon rupture is usually defined as the rupture that occurs in 4 to 6 weeks after injury [3]. The symptoms of chronic Achilles tendon rupture include pain, decreased strength, fatigue, and ankle stiffness. During physical examination, a palpable gap between the rupture ends can be observed. Chronic Achilles tendon rupture often occurs 2 to 6 cm proximal to the stumps, but it sometimes can also be observed at the stumps [5]. Usually, small gaps (less than or equal to 2 mm) of chronic Achilles tendon rupture can be directly closed in an end-to-end manner [6]. However, there is still no standard treatment for chronic Achilles tendon rupture with large gaps [7]. Recently, Den Hartog [6] used an flexor halluces longus tendon (FHLT) transfer for all defects over 2 cm. But Park and Sung [8] deemed that gaps greater than 4 cm in chronic Achilles tendon rupture that underwent various reconstruction methods depending on the state of the remaining could achieve good outcomes.

Magnetic resonance imaging (MRI) has been a reliable medical image tool for diagnosing Achilles tendon rupture and other joint diseases preoperatively [9], and it has a strong hint in the individualized rehabilitation treatment and judgment of residual pain [10]. However, there were rare researches reporting clinical follow-up of chronic Achilles tendon rupture by MRI.

Carrying on retrospective study on patients with chronic Achilles tendon rupture in our department and postoperative evaluation by MRI, we have provided the reference of the standardized treatment for future.

2. Materials and Methods

Retrospective analysis of the chronic Achilles tendon rupture cases in our department between January 2004 and July 2015 was conducted. Inclusive criteria were as follows: firstly, there is history of trauma at Achilles tendon; secondly, the interval from rupture to surgery was more than 4 weeks; thirdly, patients were not able to perform a single-limb heel rise; fourthly, MRI confirmed the final clinical diagnosis. Exclusion criteria were as follows: firstly, open Achilles tendon rupture; secondly, the history of local infection near the

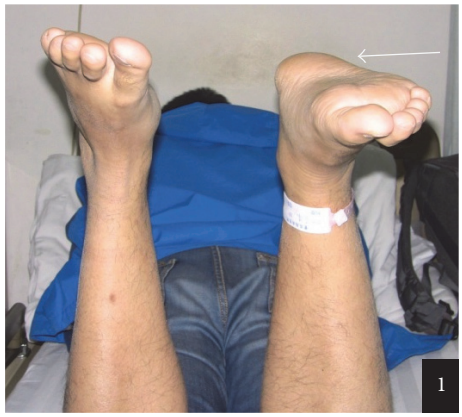


Figure 1: The right foot (arrow) was dropped because of Achilles tendon rupture when patient took prone position.

Table 1: Characteristics of patients.

Characteristics	Patients (n = 29)
Sex	
Male	23
Female	6
Age (yr)	40.3 (19.2 to 71.5)**
Side	
Right	13 (44.8%)*
Left	16 (55.2%)*
Reasons for chronic rupture	
Neglected	12 (41.4%)*
Failed treatment	17 (58.6%)*
Time from injury to surgery (wk)	13 (4 to 104)**
Length of gap (mm)	56 (25 to 100)**
Follow-up (mo)	31 (13 to 68)**

*The values are given as the number of patients, with the percentage in parentheses. **The values are given as the mean, with the range in parentheses.

Achilles tendon rupture; thirdly, concomitant diseases with fracture, blood vessels rupture, or nerve rupture; fourthly, the patients who could not accept regular follow-up. The main preoperative physical signs included the following: firstly, there is localized tenderness; secondly, when the patient lied prone with the knee bent at 90°, the static position of ankle dorsiflexion was different between the normal and injury ankles (Figure 1); thirdly, with further squeezing the calf on both sides, passive plantar flexion should be present on the healthy side but absent on the injured side; fourthly, patients were not able to perform a single-limb heel rise with the injured lower extremity. X-ray test was the routine before operation and it could rule out the chance of fracture. Preoperative MRI was conducted with 0.2 T Artoscan C (Italy) by using dedicated coil, and all MRIs showed that the Achilles tendon was not in continuity in transaxial and sagittal planes.

In 12 cases, acute Achilles tendon rupture had been neglected after the first injury. The other 17 cases with correct first-time diagnosis had Achilles tendon rupture after failure of conservative treatment. Of all the involved patients, 23 cases were male and 6 cases were female, with the mean age of 40.3 years (range 19.2–71.5 years) at surgery. 16 cases had left Achilles tendon rupture and the other 13 cases had right Achilles tendon rupture (Table 1).

2.1. Surgical Strategy. Appropriate method for tendon reconstruction was chosen based on preoperative MRI results (e.g., presence or absence of Achilles tendon stumps) as well as the defect gap measured during operation. If the tendon stumps had enough integrity of the Achilles tendon, ruptures of the gap less than 2 cm could be repaired directly by Krakow method, while the gap greater than 2 cm could be addressed through V-Y advancement or gastrocnemius fascial turndown flap. If the tendon stumps did not have enough integrity of the Achilles tendon, FHLT transfer could be considered for reconstruction (Figure 2).

2.2. Surgical Technique. All operations were performed by two senior orthopedic surgeons. The patient was placed prone on the operating table. Anesthesia of lumbar plexus-sciatic nerve block and thigh tourniquet were used. A posterolateral or posteromedial incision was made over the position of the Achilles tendon rupture. The Achilles tendon was adequately exposed. The surrounding tissue and distal tendon stumps were carefully preserved. For the V-Y advancement [11, 12], V-shape part was designed in aponeurosis; limbs of the V-shape part should be attached to soleus as much as possible; the tendon should be slowly torn with caution. For the gastrocnemius fascial turndown flap [13, 14], the gap of ruptures should be measured; the length of the turndown flap was then determined as 2 cm in addition to the length of gap. FHLT could be considered for reconstruction. The main operation technique included harvesting the FHLT and transferring it into the bone tunnels drilled in posterior calcaneus. Hydroxyapatite composite screws (Smith & Nephew) were used to fix the FHLT to the bone tunnel [15, 16]. The ankle was kept in appropriate position. After reconstruction of Achilles tendon rupture, the wound was closed in layers.

2.3. Postoperative Treatment. The ankle was kept in plantar flexion (up to 20°) for 4–6 weeks using a below-knee cast; then the cast angle in plantar flexion was decreased in nonpain conditions. Patients were encouraged to perform physical exercises under rehabilitation guidelines. 6 weeks after operation, partial-weight-bearing crutch ambulation was allowed with ankle-foot boot. At 12 weeks postoperatively, patients were allowed to participate in riding and swimming without restrictions. Patients can do strenuous sports such as running and jumping at 1 year postoperatively.

2.4. Statistical Analysis. All patients were asked to accept regular follow-up, during which the MRI and physical examination were applied. The Achilles tendon function was evaluated by integrity, pain, ankle strength, and range of motion. Postoperative complications such as wound healing problems were also observed. At the latest follow-up,

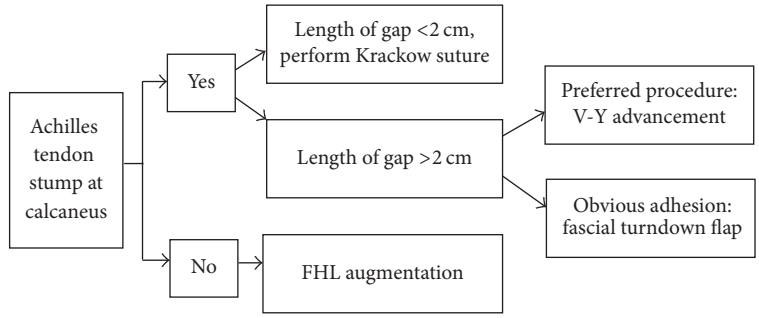


Figure 2: Treatment strategy for chronic Achilles tendon rupture.

subjective outcomes including the American Orthopaedic Foot & Ankle Society (AOFAS) ankle-hindfoot scores [8] and Achilles Tendon Total Rupture Score (ATRS) [14] were reevaluated. Comparisons between the preoperative and postoperative AOFAS and ATRS scores were performed with use of paired-sample t-tests in a commercial statistics package (version 18.0; IBM, Chicago, IL, USA). Significance level was set as $P < 0.05$.

3. Results

No rupture gap was less than 2 cm. Gaps greater than 2 cm (18 cases) were addressed through V-Y advancement, including 7 cases whose rupture gaps were more than 6 cm (Figure 3). 8 cases received gastrocnemius fascial turndown flap, including 4 cases whose rupture gaps were more than 6 cm (Figure 4). 3 cases had undergone FHLT transfer (Figure 5). The mean follow-up period was 31 months (range 13–68 months).

All patients were followed up. One patient had superficial incision infection, which was healed after debridement, dressing change, and oral antibiotics. All patients were able to perform a single-limb heel rise and had returned to their preinjury level of sports participation. Three months after operation, MRI showed some signs of inflammation, which disappeared at one and two years postoperatively. At the latest follow-up, MRI showed continuity of the Achilles tendon. Mean AOFAS scores increased from 60.13 ± 10.76 points preoperatively to 94.63 ± 4.02 points at the latest follow-up ($P < 0.05$). Mean ATRS score also showed significant improvements from 43.83 ± 11.78 points preoperatively to 92.62 ± 7.77 points at the latest follow-up ($P < 0.05$).

4. Discussion

The treatment of chronic Achilles tendon rupture is a challenge for most orthopedic surgeons [17–19]. It is different from the acute Achilles tendon rupture in pathophysiology. Chronic Achilles tendon ruptures with large gaps may lead to ankle dysfunction [1–3, 20, 21]. If the gap of rupture is bridged by scar tissue, ankle weakness and gait disturbances may occur due to severely infiltrated fat composition [7]. Though chronic Achilles tendon rupture is not unusual, it is frequently misdiagnosed. According to previous study, neglected Achilles tendon ruptured can occur at rate as high as 20% clinically [22]. After Achilles tendon rupture occurred, the strength of plantar flexion is reduced [23], and the patients are not able to perform a single-limb heel rise with the injured lower extremity. The sign is the vital indication for reconstruction surgery. In the current study, the modified Thompson test was used to diagnose the Achilles tendon rupture. When the patient lied prone with the knee bent at 90°, Thompson test could get higher positive rate than the knee being straight. Surgical reconstruction could restore full strength of the Achilles tendon and thus improve the activity level of patients [7]. However, the reconstruction and augmentation of chronic Achilles tendon rupture are complex, and they might affect the choice of procedures. In the literature, no optimal treatments for chronic Achilles rupture had been documented. Our study provided a simple and useful treatment strategy for chronic Achilles tendon rupture. Based on the presence or absence of Achilles tendon stumps, the appropriate reconstruction methods can be determined. Our results in the study supported the surgery integrity.

The optimal technique for treating chronic Achilles tendon rupture was controversial [24–27]. V-Y advancement flap was first introduced by Abraham and Pankovich, as a treatment for neglected Achilles tendon rupture [11]. In our study, V-Y advancement was used in 18 cases, in which the maximal gap was 9 cm (range from 3 to 9 cm). In these patients, the AOFAS and ATRS scores showed significant improvements at latest follow-up and no serious complications were observed. Ahmad et al. [7] deemed that gaps greater than 6 cm in chronic Achilles tendon rupture could be a big challenge to surgeons. In their study, defect up to 6 cm could be repaired; 75% of the patients had regained full tendon strength and could perform heel raises [11]. Khiami et al. [25] suggested that V-Y advancement flap was appropriate for gaps of 3 to 5 cm when the reconstruction was delicate. McClelland and Maffulli [18] reported that V-Y advancement could achieve satisfactory results in treating chronic Achilles tendon ruptures measured over 6 cm in length. Our results also supported these conclusions.

Gastrocnemius fascial turndown flap is also a useful technique in repairing chronic Achilles tendon ruptures with great defect. In 1931, Christensen [23] first reported his method in which the defect was filled using a fascial turndown flap sized 2 cm by 10 cm. In our study, the maximal

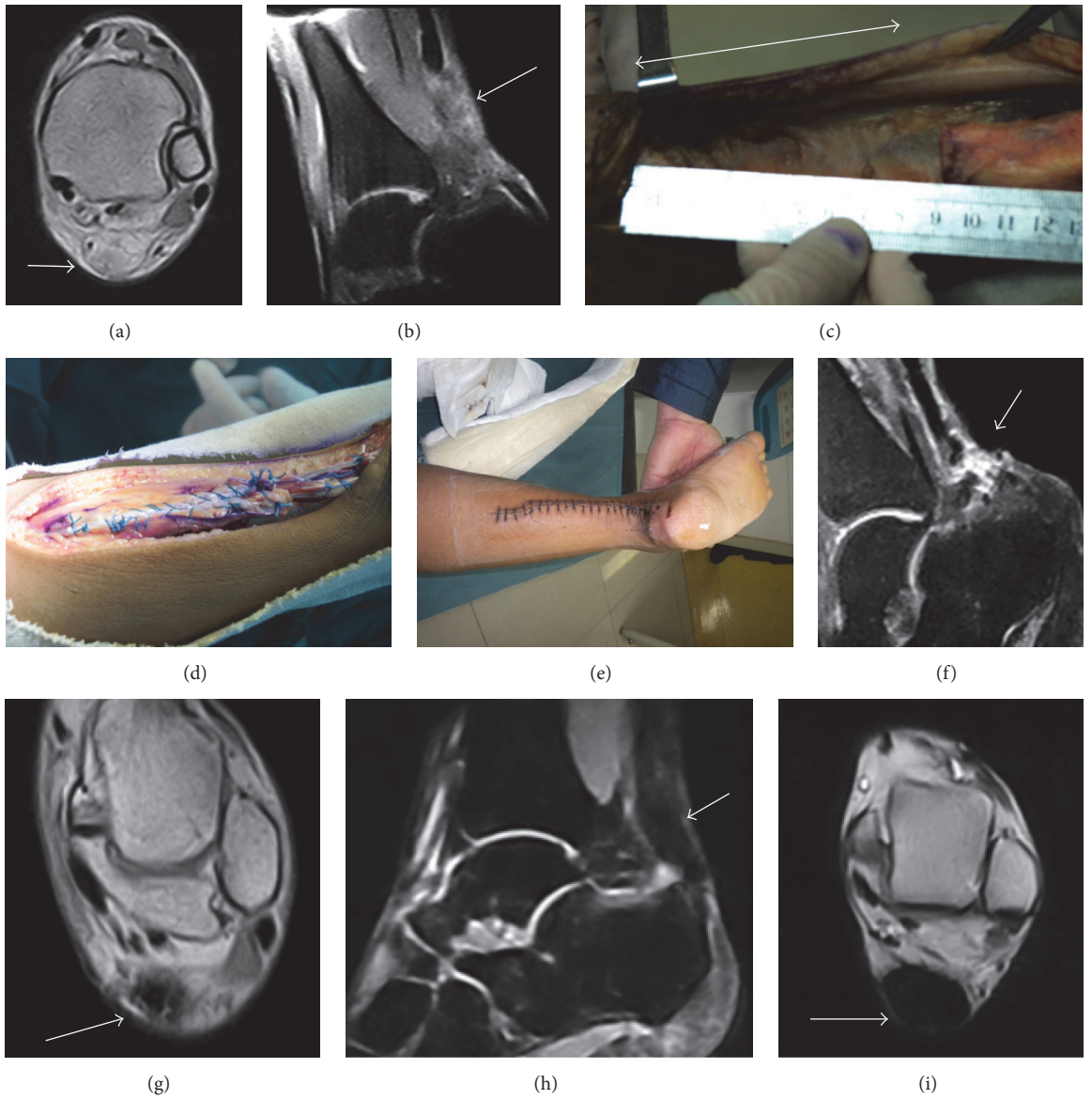


FIGURE 3: A 23-year-old male. The interval from rupture to surgery was 7 weeks. Preoperative MRI showed chronic Achilles tendon rupture (arrow) (a, b). Length of gap (double-headed arrow) was 9 cm after scar tissue debridement (c). V-Y advancement performed (d). Wound healed (e). MRI showing inflammation (arrow) at 6 weeks of follow-up (f, g). MRI showing fusiform-shaped tendon thickening and homogeneous low-signal at 1 year postoperatively (arrow) (h, i).

gap treated with this technique was 10 cm, and the patient had achieved satisfactory ankle function. Other authors have also described successful repair of chronic Achilles tendon ruptures with modified gastrocnemius fascial turndown flaps. Tay et al. [13] reported that chronic Achilles tendon rupture was treated with two turndown flaps and FHL augmentation yielded satisfactory results during two-year follow-up. Peterson's et al. study [5] also revealed similar outcomes when treating central defect of approximately 12 cm.

Three aspects should be considered in V-Y advancement reconstruction for chronic Achilles tendon rupture. First, V-Y advancement is more suitable for acute chronic Achilles tendon rupture; second, V-Y advancement should be used in young patients; third, soleus muscle can provide revascularization for the tendon of V-Y advancement so that the Achilles tendon rupture can heal faster. In comparison, gastrocnemius fascial turndown flap was suitable for all kinds of patients, but the rupture may heal more slowly because soleus muscle cannot directly provide revascularization. In our opinion, V-Y advancement should be chosen with priority for the ruptures with a gap greater than 2 cm. When V-shape part was not enough in the operation and the soleus cannot attach to V-shape part, fascial turndown flap was a choice for reducing the operative wound.

FHLT transfer was first used to treat chronic Achilles tendon rupture about 20 years ago [16]. It had developed to

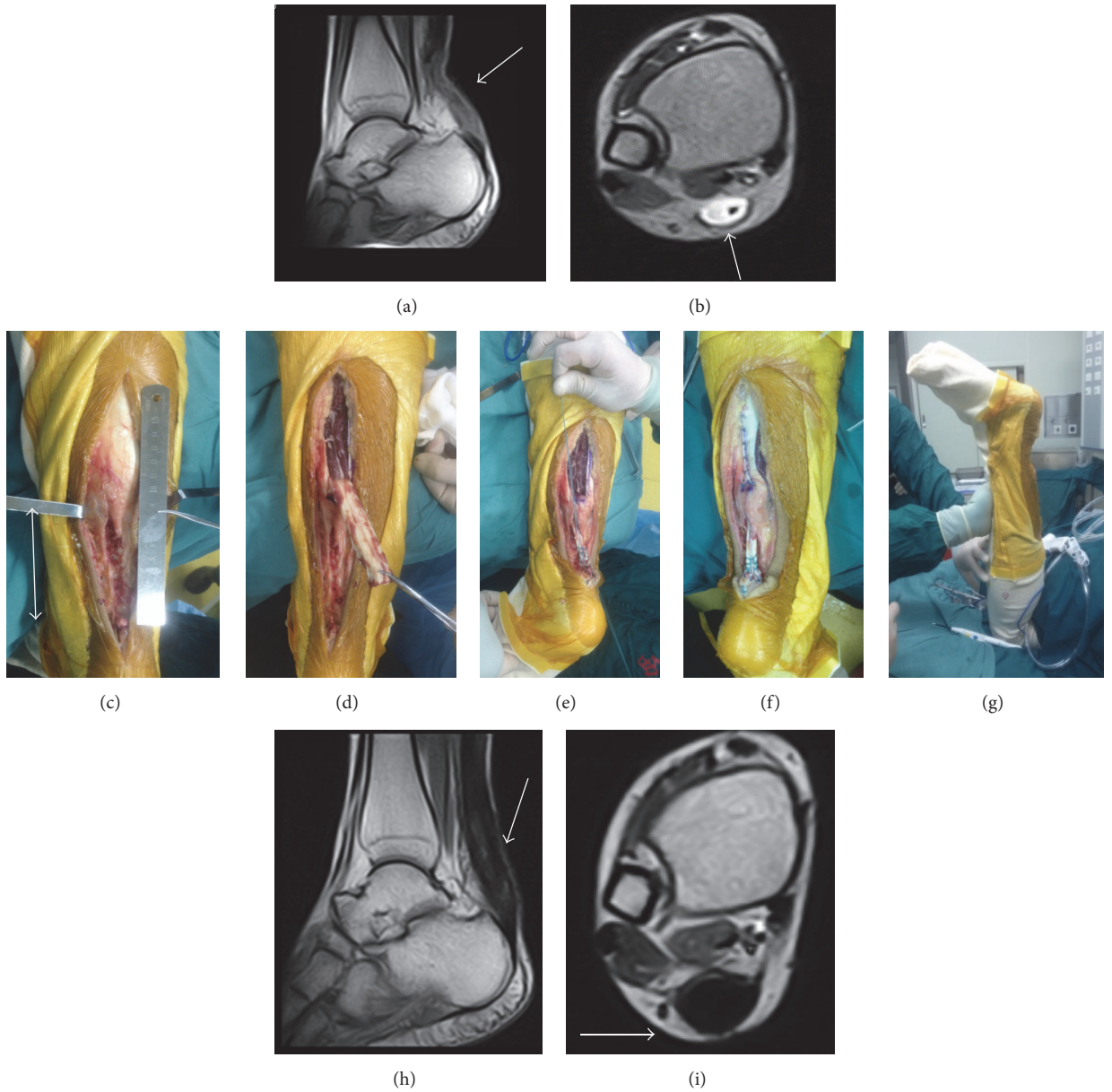


Figure 4: A 30-year-old male. The interval from rupture to surgery was 22 weeks. Preoperative MRI showed chronic Achilles tendon rupture (arrow) (a, b). Length of gap (double-headed arrow) was 9 cm, a lot of scar tissue located stump area, and it was difficult to perform V-Y advancement (c). Gastrocnemius fascial turndown flap performed (d, e). Sutured with the stump and adjusted the tension (f). The position of ankle was similar to the other side after surgery (g). MRI showing fusiform-shaped tendon thickening and homogeneous low-signal at 1 year postoperatively (arrow) (h, i).

a widespread application in tendon stumps reconstruction. When the tendon stumps did not have enough integrity of the Achilles tendon, we can consider FHLT for surgical reconstruction. The main operation technique included harvesting the FHLT and transferring the FHL into the bone tunnels made in posterior calcaneus. Therefore, the FHL muscle can generate enough strength to raise the heel and substitute the function of Achilles tendon. FHLT has four advantages in the chronic Achilles tendon reconstruction. First, it is close to the Achilles tendon; second, the FHL muscle has the same function as the triceps surae; third, it has adequate strength; fourth, it has the same axis of moving with the Achilles tendon [16]. Since FHLT has the above advantages in the chronic Achilles tendon reconstruction, more and more surgeons preferred this surgical technique and had reported positive outcomes. Yeoman et al. [15] treated 11 patients with chronic Achilles tendon rupture using FHL technique and interference screw fixation, showing reliable outcomes and low complication morbidity. Oksanen et al. [28] reported that mean hypertrophy of 52% of the FHL muscle was observed by MRI after FHL. Similar phenomenon was also observed in the current study. This may demonstrate that the FHL had good

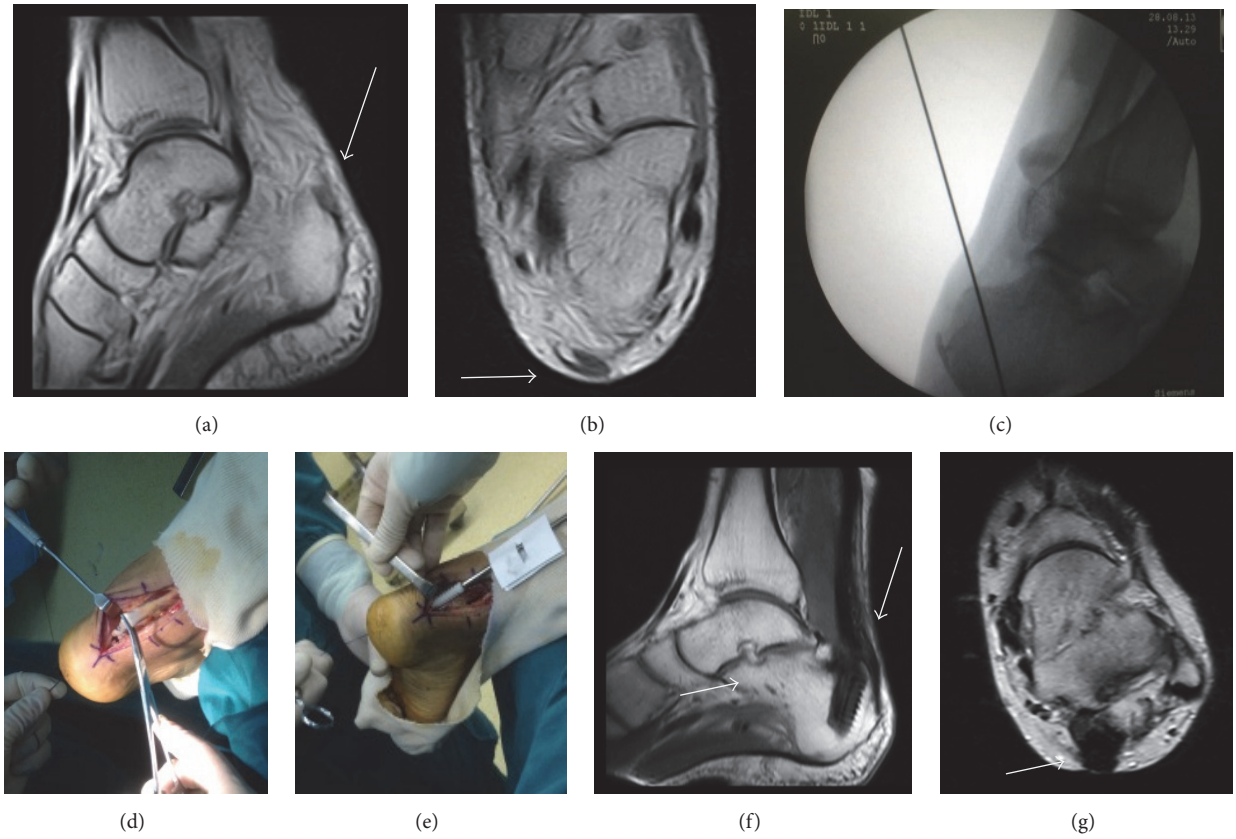


Figure 5: Preoperative MRI showed chronic Achilles tendon rupture and no stump at calcaneus (a, b). FHLT transfer performed (c, d). Using the screws of hydroxyapatite composition to fix FHL into the bone tunnels (e). MRI showing the continuity of the Achilles tendon and homogeneous low-signal at 34 months postoperatively (arrow) (f, g).

adaptation capacity [28]. Coull et al. [29] found that though the active range of motion of interphalangeal joint might bet lost, it did not impair functions such as walking, running, and stair climbing. But, sometimes, this technique can reduce the function of halluces; therefore, it should not be routinely recommended for young patients. Besides, Achilles tendon stump is very important for Achilles tendon reconstruction because it has the normal tendon bone interface structure and Sharpey's fiber [30], whereas, in FHLT transfer, there would be a rather long process of interface structure reconstruction [31]. Thus, we suggested that FHLT transfer only be used for increasing strength when there were not enough tendon stumps.

We also found that MRI could be a useful tool for diagnosing Achilles tendon rupture. It can assess the integrity of the Achilles tendon stumps, acquire the information of Achilles tendon healing, and so on. No patient was misdiagnosed by MRI in our study. Postoperative follow-up MRI had a strong hint in the individualized rehabilitation treatment and judgment of residual pain. In our study, three months after operation, MRI showed some signs of inflammation, which disappeared at one and two years postoperatively.

One limitation of the current study was that it was a retrospective study with small number of cases. Also, we did not have isokinetic strength analysis of the patients, nor did we conduct comparative study among the three techniques. Future study with larger number of cases and longer follow-up time would be able to provide stronger evidence in a multicenter randomized control clinical trial.

5. Conclusion

A clear treatment strategy can be determined by evaluating the presence or absence of the Achilles tendon stumps and the gap length of rupture after Achilles tendon rupture, which can avoid using the nearby tendon and yield satisfactory functional results by making the most of the local Achilles tendon and gastrocnemius fascial. MRI is used in the healing process observation and is a useful tool for postoperative rehabilitation.

Competing Interests

The authors declare that there are no competing interests regarding the publication of the paper.

Authors' Contributions

Yangjing Lin and Liu Yang made equal contribution to the manuscript.

Acknowledgments

This study was supported by the General Program of Chongqing Natural Science Foundation of China (no. CSTC 2016shmszx0630). The authors thank Xin Chen for proofreading services.

References

[1] T. Movin, Å. Ryberg, D. J. McBride, and N. Maffulli, "Acute rupture of the achilles tendon," *Foot and Ankle Clinics*, vol. 10, no. 2, pp. 331–356, 2005.

[2] N. Maffulli and A. Ajis, "Management of chronic ruptures of the achilles tendon," *The Journal of Bone & Joint Surgery—American Volume*, vol. 90, no. 6, pp. 1348–1360, 2008.

[3] J. H. Flint, A. M. Wade, J. Giuliani, and J.-P. Rue, "Defining the terms acute and chronic in orthopaedic sports injuries: a systematic review," *American Journal of Sports Medicine*, vol. 42, no. 1, pp. 235–241, 2014.

[4] E. Taşatan, T. Y. Emre, D. T. Demircioğlu, B. Demiralp, and V. Kırdemir, "Long-term results of mini-open repair technique in the treatment of acute achilles tendon rupture: A Prospective Study," *The Journal of Foot and Ankle Surgery*, vol. 55, no. 5, pp. 971–975, 2016.

[5] K. S. Peterson, M. J. Hentges, A. R. Catanzariti, M. R. Mendicino, and R. W. Mendicino, "Surgical considerations for the neglected or chronic Achilles tendon rupture: a combined technique for reconstruction," *Journal of Foot and Ankle Surgery*, vol. 53, no. 5, pp. 664–671, 2014.

[6] B. D. Den Hartog, "Surgical strategies: delayed diagnosis or neglected achilles' tendon ruptures," *Foot and Ankle International*, vol. 29, no. 4, pp. 456–463, 2008.

[7] J. Ahmad, K. Jones, and S. M. Raikin, "Treatment of chronic achilles tendon ruptures with large defects," *Foot & Ankle Specialist*, vol. 9, no. 5, pp. 400–408, 2016.

[8] Y.-S. Park and K.-S. Sung, "Surgical reconstruction of chronic achilles tendon ruptures using various methods," *Orthopedics*, vol. 35, no. 2, pp. e213–e218, 2012.

[9] M. M. B. Morrow, M. G. Van Straaten, N. S. Murthy, J. P. Braman, E. Zanella, and K. D. Zhao, "Detailed shoulder MRI findings in manual wheelchair users with shoulder pain," *BioMed Research International*, vol. 2014, Article ID 769649, 7 pages, 2014.

[10] T. Yasuda, H. Shima, K. Mori, M. Kizawa, and M. Neo, "Direct repair of chronic achilles tendon ruptures using scar tissue located between the tendon stumps," *The Journal of Bone & Joint Surgery*, vol. 98, no. 14, pp. 1168–1175, 2016.

[11] E. Abraham and A. M. Pankovich, "Neglected rupture of the Achilles tendon: treatment by V-Y tendinous flap," *Journal of Bone and Joint Surgery—Series A*, vol. 57, no. 2, pp. 253–255, 1975.

[12] B. Guclu, H. C. Basat, T. Yildirim, O. Bozduman, and A. K. Us, "Long-term results of chronic achilles tendon ruptures repaired with V-Y tendon plasty and fascia turndown," *Foot & Ankle International*, vol. 37, no. 7, pp. 737–742, 2016.

[13] D. Tay, H. A. Lin, B. S. A. Tan, K. W. Chong, and I. S. Rikhraj, "Chronic Achilles tendon rupture treated with two turndown flaps and flexor hallucis longus augmentation—two-year clinical outcome," *Annals of the Academy of Medicine Singapore*, vol. 39, no. 1, pp. 58–60, 2010.

[14] N. Maffulli, F. Spiezia, V. Testa, G. Capasso, U. G. Longo, and V. Denaro, "Free gracilis tendon graft for reconstruction of chronic tears of the Achilles tendon," *The Journal of Bone & Joint Surgery—American Volume*, vol. 94, no. 10, pp. 906–910, 2012.

[15] T. F. M. Yeoman, M. J. C. Brown, and A. Pillai, "Early post-operative results of neglected tendo-Achilles rupture reconstruction using short flexor hallucis longus tendon transfer: a prospective review," *Foot*, vol. 22, no. 3, pp. 219–223, 2012.

[16] S. Rahm, C. Spross, F. Gerber, M. Farshad, F. M. Buck, and N. Espinosa, "Operative treatment of chronic irreparable achilles tendon ruptures with large flexor hallucis longus tendon transfers," *Foot and Ankle International*, vol. 34, no. 8, pp. 1100–1110, 2013.

[17] R. Cetti, S.-E. Christensen, R. Ejsted, N. M. Jensen, and U. Jorgensen, "Operative versus nonoperative treatment of Achilles tendon rupture. A prospective randomized study and review of the literature," *American Journal of Sports Medicine*, vol. 21, no. 6, pp. 791–799, 1993.

[18] D. McClelland and N. Maffulli, "Neglected rupture of the Achilles tendon: reconstruction with peroneus brevis tendon transfer," *Surgeon*, vol. 2, no. 4, pp. 209–213, 2004.

[19] T. K. Dabak, O. Sertkaya, N. Acar, B. O. Donmez, and I. Ustunel, "The effect of phospholipids (surfactant) on adhesion and biomechanical properties of tendon: a rat achilles tendon repair model," *BioMed Research International*, vol. 2015, Article ID 689314, 6 pages, 2015.

[20] Y.-B. Liu, S.-Y. Jiang, L. Zhao, Y. Yu, X.-C. Tao, and D.-H. Zhao, "Functional assessment of the foot undergoing percutaneous achilles tenotomy in term of gait analysis," *BioMed Research International*, vol. 2016, Article ID 1973403, 6 pages, 2016.

[21] N. Maffulli, A. G. Via, and F. Oliva, "Chronic Achilles tendon disorders: tendinopathy and chronic rupture," *Clinics in Sports Medicine*, vol. 34, no. 4, pp. 607–624, 2015.

[22] S. A. Razik Ibrahim, "Surgical treatment of chronic Achilles tendon rupture," *Journal of Foot and Ankle Surgery*, vol. 48, no. 3, pp. 340–346, 2009.

[23] I. Christensen, "Rupture of the Achilles tendon; analysis of 57 cases," *Acta Chirurgica Scandinavica*, vol. 106, no. 1, pp. 50–60, 1953.

[24] M. J. Barnes and A. E. Hardy, "Delayed reconstruction of the calcaneal tendon," *The Journal of Bone & Joint Surgery—British Volume*, vol. 68, no. 1, pp. 121–124, 1986.

[25] F. Khiami, M. Di Schino, E. Sariali, D. Cao, E. Rolland, and Y. Catonné, "Treatment of chronic Achilles tendon rupture by shortening suture and free sural triceps aponeurosis graft," *Orthopaedics and Traumatology: Surgery and Research*, vol. 99, no. 5, pp. 585–591, 2013.

[26] S. Hollawell and W. Baione, "Chronic Achilles Tendon rupture reconstructed with Achilles tendon allograft and xenograft combination," *Journal of Foot and Ankle Surgery*, vol. 54, no. 6, pp. 1146–1150, 2015.

[27] Z. Wu, Y. Hua, H. Li, S. Chen, and Y. Li, "Biomechanical comparison of three methods for distal Achilles tendon reconstruction," *Knee Surgery, Sports Traumatology, Arthroscopy*, vol. 23, no. 12, pp. 3756–3760, 2015.

[28] M. M. Oksanen, H. H. Haapasalo, P. P. Elo, and H.-J. Laine, "Hypertrophy of the flexor hallucis longus muscle after tendon transfer in patients with chronic Achilles tendon rupture," *Foot and Ankle Surgery*, vol. 20, no. 4, pp. 253–257, 2014.

[29] R. Coull, R. Flavin, and M. M. Stephens, "Flexor hallucis longus tendon transfer: evaluation of postoperative morbidity," *Foot and Ankle International*, vol. 24, no. 12, pp. 931–934, 2003.

[30] Y.-J. Ju, T. Muneta, H. Yoshimura, H. Koga, and I. Sekiya, "Synovial mesenchymal stem cells accelerate early remodeling of tendon-bone healing," *Cell and Tissue Research*, vol. 332, no. 3, pp. 469–478, 2008.

[31] P. P. Y. Lui, G. Ho, W. T. Shum et al., "Inferior tendon graft to bone tunnel healing at the tibia compared to that at the femur after anterior cruciate ligament reconstruction," *Journal of Orthopaedic Science*, vol. 15, no. 3, pp. 389–401, 2010.

Hindawi Publishing Corporation
BioMed Research International
Volume 2016, Article ID 7123769, 4 pages
http://dx.doi.org/10.1155/2016/7123769

Clinical Study

Low-Energy Extracorporeal Shock-Wave Therapy in the Treatment of Chronic Insertional Achilles Tendinopathy: A Case Series

Vito Pavone, Luca Cannavò, Antonio Di Stefano, Gianluca Testa, Luciano Costarella, and Giuseppe Sessa

Dipartimento di Chirurgia Generale e Specialità Medico Chirurgiche, Sez. Ortopedia e Traumatologia A. O. U. Policlinico-Vittorio Emanuele, Catania, Italy

Correspondence should be addressed to Luca Cannavò; lucacannav@hotmail.it

Received 30 June 2016; Accepted 4 October 2016

Academic Editor: Ying-Hui Hua

Introduction. We report the results of a series of 40 patients with chronic insertional Achilles tendinopathy treated with low-energy ESWT after the failure of a 3-month program of eccentric exercises alone. *Methods and Materials*. 40 patients, 28 (70%) males and 12 (30%) females, were treated between January and December 2014. All patients were previously treated with only eccentric exercises for a 3-month period. The treatment protocol included 4 sessions of ESWT with a 2-week interval, from 800 shots in each one (4 Hz, 14 KeV), together with eccentric exercises. Visual Analogue Scale (VAS) and American Orthopedic Foot and Ankle Society (AOFAS) Hindfoot score were recorded. *Results*. At the 12-month follow-up, 26 (65.0%) patients did not complain about pain (VAS < 2), 11 (27.5%) patients got back to normal activities despite residual pain (VAS 2–4), and 3 (7.5%) of the patients still complained about pain (VAS > 4). There was no significative improvement in both scores after eccentric exercises alone. Mean VAS improvement was 5.8 ± 1.3 SD points ($P < 0.001$). Mean AOFAS Hindfoot score improvement was 19.8 ± 5.0 SD points ($P < 0.001$). *Conclusions*. ESWT is recommended, in combination with an eccentric exercise program, in patients with chronic Achilles tendinopathy being both insertional and not.

1. Introduction

Chronic Achilles tendinopathy is one of the most frequent injuries during sport and physical practice representing 5%–18% of all running injuries [1]. This condition, characterized by chronic pain, has a great impact, especially in professional athletes [2]. Chronic pain and long recovery time have stimulated interest around the various treatment options. Among the various therapies offered, one with Extracorporeal Shock-Wave Therapy (ESWT) seems to be an effectiveness support to other noninvasive therapies [3] like eccentric exercises, although there is no consensus in literature around the ESWT application protocol (number of strokes, energy, and use of anesthetics). In this article, we report the results of a series of 40 patients suffering from resistant chronic insertional Achilles tendinopathy, treated with a low-energy ESWT protocol, after the failure of a 3-month program of eccentric exercises alone.

2. Methods and Materials

40 patients, 28 (70%) males and 12 (30%) females, were treated between 1 January and 31 December 2014 for chronic insertional Achilles tendinitis according to *International Classification of Diseases, 10th Revision* procedure codes (code M76.6) [4]. The study-enrolled patients showed persistent pain for more than 3 months in the insertional region of Achilles tendon, especially during walking and physical activities.

The study was approved by local ethical committee and it is in accordance with the Declaration of Helsinki. The enrolled patients came from Outpatient Department of the Orthopedics Clinic, University of Catania (Catania, Italy). All the treatments were performed by two of us (V.P and L.Co.), the data were abstracted by two of us (A.Ds and G.T.) and the outcome measurements and statistical analysis were undertaken by one of us (L.Ca.).

The mean age was 41.0 years (range 32–56). The right tendon was involved in 25 (62.5%) cases and the left in 15 (37.5%) cases (Table 1). Before starting ESWT treatment, an X-ray exam of the two feet in anteroposterior and lateral view was carried out.

Patients started our protocol after at least 3 months of therapy with Non-Steroidal Anti-inflammatory Drugs (NSAIDs) for 4–7 days and stretching and eccentric exercises for 50 min a day four times a week. Before treatment with ESWT, 23 (57.5%) patients were treated with therapy and 17 (42.5%) with diathermic treatment.

The overuse and the possibility of tendon-shoe friction were taken into consideration before starting treatment.

The treatment protocol included stretching and eccentric exercises for 50 min a day four times a week, use of ice before and after every session, and treatment with low-energy radial ESWT. ESWT was carried out in 4 sessions, with a 2-week interval. Electromedical device OssaTron®, at a frequency of 4 Hz and a voltage of 14 KeV with 800 shots each session, was used for the treatment.

Visual Analogue Scale (VAS) to measure pain and American Orthopedic Foot and Ankle Society (AOFAS) Hindfoot score, to evaluate alignment and functional outcome, were performed. VAS and AOFAS Hindfoot score were registered at the end of the eccentric exercises period, at the beginning of ESWT + eccentric exercises protocol, and then at 2, 6, and 12 months.

Patients with cardiac disease, with pacemaker, in treatment with anticoagulants, in treatment or with chronic history of neoplasm, diabetes, or rheumatologic diseases, in treatment with corticosteroids, or with fluoroquinolones in the last 2 years and patients with vascular diseases of the lower extremities were excluded from the study.

Two-tailed Student's t-test was performed to compare the difference between VAS and AOFAS Hindfoot score along the study period. For statistical analysis, Ministat was employed.

Limits of the study were the small sample size and the absence of control group together with the short-term follow-up.

3. Results

At the 12-month follow-up, 26 (65.0%) patients did not complain about pain (VAS < 2), 11 (27.5%) patients got back to normal daily activities and sports despite residual pain (VAS 2–4), and 3 (7.5%) of the patients still complained about pain (VAS > 4).

Mean VAS value at the beginning of eccentric exercises program was 7.7 ± 0.5 DS and after eccentric exercises was 7.4±0.6 SD ($P > 0.05$). At the beginning of ESWT + eccentric exercises treatment, VAS score was 7.6 ± 0.6 DS, 3.8 ± 0.7 SD at 2 months, 2.8 ± 0.7 SD at 6 months, and 1.9 ± 1.2 SD after 12 months, with a mean improvement at the end of treatment of 5.8 ± 1.3 SD points. The results observed were statistically significant ($P < 0.001$).

Mean AOFAS score at the beginning of eccentric exercises program was 71.5 ± 5 SD and it was 71.0 ± 4.1 SD at the end ($P > 0.05$). Mean AOFAS Hindfoot score at the beginning of treatment was 71.4 ± 4.6 SD, 85.2 ± 4.1 SD at 2 months, 89.2 ± 3.6 SD at 6 months, and 91.3 ± 3.8 SD at 12 months, with a mean improvement of 19.8 ± 5.0 points ($P < 0.001$).

At X-ray examination, in 10 (25%) cases, calcification in the insertional region and, in 15 (37.5%) cases, the presence of Haglund's deformity were reported.

Table 1: Study group.

Patients	40
Feet	40
Males	28 (70%)
Females	12 (30%)
Mean age	41.0 (range 32–56)
Right	25 (62.5%)
Left	15 (37.5%)
Laser therapy before ESWT treatment	23 (57.5%)
Diathermic therapy before ESWT treatment	17 (42.5%)

4. Discussion

Therapy with low-energy ESWT has been shown, in our series, to be effective in both reducing pain and function recovery after the failure of only eccentric exercises program and occasional NSAID treatment. The last 10 years' literature supports our conclusions (Table 2).

In 2005, Costa et al. published a randomized clinical trial in which they verified the use of ESWT administered 1 time per month for 3 months [9]. The study did not show a real beneficial effect on pain and the use of ESWT was not supported, probably because the time between the different sessions was too long. After this first trial, the trend in literature started to change. In fact, a randomized trial, published by Rasmussen et al. in 2008, revealed a beneficial effect of ESWT on functional recovery despite not significant results on pain [5]. The study did not differentiate between insertional and noninsertional Achilles tendinopathy. The protocol of treatment used was 2,000 strikes (0 : 12 to 0 : 51 mJ/mm^2, 50 Hz) in 4 sessions once a week. They explain the poor effect on pain with the earlier recovery to a full level activity. Simultaneously, between 2006 and 2008, Furia published two case-control trials [7, 10]. The author evaluated the effectiveness of the treatment, respectively, on insertional and noninsertional Achilles tendinopathy, using a single dose of high-energy ESWT: 3000 shots (0.21 mJ/mm and 604 mJ/mm). These studies showed a beneficial effect on pain and supported the use of ESWT in both insertional and nooinsertional Achilles tendinopathy. Then, in 2007, Rompe et al. published a randomized case-control trial comparing eccentric exercises, low-energy ESWT, and "wait and see" in treatment of chronic tendinopathy of Achilles tendon body. The ESWT protocol was delivered in 3 sessions of 2000 weekly interval shots (0.1 mJ/mm^2; 8 Hz) [11]. Improvement in pain symptoms in the group with eccentric exercises and in the group treated with ESWT in comparison with simple observation was found. In 2008, Rompe et al. again in treating chronic Achilles insertional tendinopathy released another randomized case-control trial comparing the effect of

Table 2: Case series, VAS score during activity.

Authors	N. cases	Follow-up (months)	Treatment	ESWT parameters	Pain improvement	Functional improvement
Rasmussen et al. 2008 [5]	48 (24 + 24)	3	ESWT versus placebo	Low-energy radial ESWT, 4 sessions, 2,000 shots, 0.12–0.51 mJ/mm^2, 50 Hz	/	AOFAS +18 points ($P < 0.05$)
Rompe et al. 2008 [6]	50 (25 + 25)	12	ESWT versus eccentric exercises	Low-energy ESWT, 3 sessions, 2000 shots, 0.12 mJ/mm^2	NRS −3.0 ± 2.3 (0–8)	VISA-A +27 points ($P < 0.001$)
Furia 2006 [7]	68 (35 + 33)	12	ESWT versus control	High-energy ESWT, 1 dose, 3000 shocks, 0.21 mJ/mm^2, total energy flux density, 604 mJ/mm^2	VAS 7.0 (control) and 2.8 (ESWT) ($P < 0.001$)	Roles-Maudsley ($P < 0.001$)
Taylor et al. 2016 [8]	56 (46)	24	ESWT	Low-energy radial ESWT, 3 sessions, 2000 shots, 10 Hz, 1.5–2.5 bar	VAS −4.8 (NAT) and −3.9 (IAT) ($P < 0.01$)	VISA-A +66 points ($P < 0.001$)
The present study	40	12	ESWT + eccentric exercises	Low-energy radial ESWT, 4 sessions, 800 shots, 4 Hz, 14 KeV	VAS −5.8 ± 1.2 SD ($P < 0.001$)	AOFAS +19.8 ±5.0 SD ($P < 0.001$)

eccentric exercise versus low-energy radial ESWT [6]. Three sessions of 2000 strikes once a week (0.1 mJ/mm^2; 8 Hz) were administered. Excellent results in terms of both pain and recovery were observed in both groups, with significantly better results in ESWT group compared with only eccentric exercises. Recently, a very interesting prospective study was published by Taylor et al. in 2016, who evaluate the effectiveness of ESWT in the treatment of refractory Achilles tendinopathy with a 24-month follow-up [8]. They make a distinction between noninsertional Achilles tendinopathy (NAT) and insertional Achilles tendinopathy (IAT) and analyze the results among different age groups. They found pain and functional improvement across all age groups. Unfortunately no control group was compared in this study.

If we summarize the results of these various studies, it is worth mentioning two meta-analyses. The first, by Al-Abbad and Simon in 2013, evaluated the effectiveness of ESWT in treating chronic tendinopathy Achilles [1]. Six studies are examined. With the selection of 6 studies, the authors concluded that the ESWT was effective with a level of evidence 1, particularly when associated with an eccentric exercise program, in the treatment of chronic Achilles tendinopathy. The second meta-analysis, published by Gerdesmeyer et al. in 2015, examined 6 clinical trials [3]. Also in this study authors conclude that ESWT is effective, in terms of pain and functional recovery, in the treatment of Achilles chronic tendinopathy of both insertional and noninsertional condition. No evidence of a specific recommended protocol was reported.

5. Conclusions

The ESWT is recommended, especially in combination with an eccentric exercise program and in association with other not invasive treatments of chronic Achilles tendinopathy that are both insertional and not. Further studies are needed requirements to establish the most effective protocol.

Competing Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

References

[1] H. Al-Abbad and J. V. Simon, "The effectiveness of extracorporeal shock wave therapy on chronic Achilles tendinopathy: a systematic review," *Foot and Ankle International*, vol. 34, no. 1, pp. 33–41, 2013.

[2] T. A. H. Järvinen, P. Kannus, N. Maffulli, and K. M. Khan, "Achilles tendon disorders: etiology and epidemiology," *Foot and Ankle Clinics*, vol. 10, no. 2, pp. 255–266, 2005.

[3] L. Gerdesmeyer, R. Mittermayr, M. Fuerst et al., "Current evidence of extracorporeal shock wave therapy in chronic Achilles tendinopathy," *International Journal of Surgery*, vol. 24, pp. 154–159, 2015.

[4] World Health Organization: International Classification of Diseases (ICD), http://www.who.int/classifications/icd/en/.

[5] S. Rasmussen, M. Christensen, I. Mathiesen, and O. Simonson, "Shockwave therapy for chronic Achilles tendinopathy: a double-blind, randomized clinical trial of efficacy," *Acta Orthopaedica*, vol. 79, no. 2, pp. 249–256, 2008.

[6] J. D. Rompe, J. Furia, and N. Maffulli, "Eccentric loading compared with shock wave treatment for chronic insertional achilles tendinopathy: a randomized, controlled trial," *Journal of Bone and Joint Surgery—Series A*, vol. 90, no. 1, pp. 52–61, 2008.

[7] J. P. Furia, "High-energy extracorporeal shock wave therapy as a treatment for insertional achilles tendinopathy," *American Journal of Sports Medicine*, vol. 34, no. 5, pp. 733–740, 2006.

[8] J. Taylor, S. Dunkerley, D. Silver et al., "Extracorporeal shockwave therapy (ESWT) for refractory Achilles tendinopathy: a prospective audit with 2-year follow up," *The Foot*, vol. 26, pp. 23–29, 2016.

[9] M. L. Costa, L. Shepstone, S. T. Donell, and T. L. Thomas, "Shock wave therapy for chronic Achilles tendon pain: a randomized placebo-controlled trial," *Clinical Orthopaedics and Related Research*, vol. 440, pp. 199–204, 2005.

[10] J. P. Furia, "High-energy extracorporeal shock wave therapy as a treatment for chronic noninsertional Achilles tendinopathy," *American Journal of Sports Medicine*, vol. 36, no. 3, pp. 502–508, 2008.

[11] J. D. Rompe, B. Nafe, J. P. Furia, and N. Maffulli, "Eccentric loading, shock-wave treatment, or a wait- and-see policy for tendinopathy of the main body of tendo Achillis: a randomized controlled trial," *American Journal of Sports Medicine*, vol. 35, no. 3, pp. 374–383, 2007.

Hindawi Publishing Corporation
BioMed Research International
Volume 2016, Article ID 1973403, 6 pages
http://dx.doi.org/10.1155/2016/1973403

Research Article

Functional Assessment of the Foot Undergoing Percutaneous Achilles Tenotomy in Term of Gait Analysis

Yu-Bin Liu,[1] Shu-Yun Jiang,[2] Li Zhao,[1] Yan Yu,[2] Xu-Chen Tao,[2] and Da-Hang Zhao[1]

[1]*Department of Pediatric Orthopaedics, Xin-Hua Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai 200092, China*
[2]*Gait Lab, Yueyang Hospital, Shanghai University of Traditional Chinese Medicine, Shanghai 200437, China*

Correspondence should be addressed to Li Zhao; orthzl@126.com

Received 27 June 2016; Accepted 4 August 2016

Academic Editor: Ying-Hui Hua

Background. This study was designed to evaluate the function of the foot undergoing the procedure of percutaneous Achilles tenotomy (PAT) in case of clubfoot management in terms of gait analysis. *Methods.* Nineteen patients with unilateral clubfeet were retrospectively reviewed from our database from July 2012 to June 2016. The result in all the cases was rated as excellent according to the scale of International Clubfoot Study Group (ICSG). The affected sides were taken as Group CF and the contralateral sides as Group CL. Three-dimensional gait analysis was applied for the functional evaluation of the involved foot. *Results.* Statistical difference was found in physical parameters of passive ankle dorsiflexion and plantar-flexion. No statistical difference was found in temporal-spatial parameters. There was statistical difference in kinematic parameters of total ankle rotation, ankle range of motion, and internal foot progression angle and in kinetic parameters of peak ankle power. No statistical difference was found in other kinematic and kinetic parameters. *Conclusions.* It is demonstrated that the procedure of PAT is safe and efficient for correcting the equinus deformity in case of clubfoot management and preserving the main function of Achilles tendon at the minimum of four-year follow-up.

1. Introduction

The Achilles tendon, that is, heel cord, is a tendon of the back of the leg and the strongest and thickest tendon in the human body. It serves as the function of connecting the soleus and gastrocnemius muscles to the os calcis to allow plantar flexion of the foot at the ankle and provides elastic energy storage in hopping, walking, and running [1]. The Achilles tendon is the most frequently ruptured tendon in the human body [2] with a higher incidence in males than females [3]. It is recommended that the intervention of Achilles tendon rupture should be undertaken soon, whether surgical or conservative treatment, to obviate late disability, pain, and healthcare cost [4]. However, it is still controversial whether operative or nonoperative procedure should be undertaken in the management of Achilles tendon rupture [5, 6].

Percutaneous Achilles tenotomy (PAT) is an important procedure in the clubfoot management using Ponseti method [7–9]. As reported, over 90% of cases required this procedure for correcting the equinus deformity [7, 8]. However, the debates still remain as to the healing and efficacy of Achilles tenotomy in the professional community of orthopaedists because of the established opinion that the orthopaedic intervention of the ruptured Achilles tendon should be undertaken soon. The evidence of the healing of Achilles tendon has been obtained from the ultrasonographic and MRI studies [10, 11]. However, it was considered in the previous studies that the Achilles tendon, undergoing the rupture and healing of Achilles tendon, would never be as strong as the original one because it was supposed that the gap of tendon rupture was filled with fibrous tissues even after healing [12]. It was revealed that the rerupture rate with conservative treatment was from 12% [13] to 13.4% [14]. As to the functional evaluation of Achilles tendon, clinical questionnaire consisting of the items as pain, functional activity level assessments, and muscle strength testing was established to assess the recovery of injured tendons in vivo [15]. In clinical practice, two typical tests, including

Thompson sign and heel raise test, were conducted to evaluate the Achilles tendon function. The outcome measurement of unilateral clubfoot treated with the Ponseti method including the PAT procedure with a particular reference to the data of gait analysis has been poorly reported in the literature. Percutaneous Achilles tenotomy has been the critical procedure over the last decades in the management of congenital clubfoot. It is assumed that this procedure is taken as the model of Achilles tendon healing in a child. This study was designed to evaluate the function of the foot in which the Achilles tenotomy was performed for correcting the equinus deformity in case of clubfoot management in terms of clinical assessment and gait analysis.

2. Materials and Methods

The medical records were retrospectively reviewed from our database to identify patients with idiopathic clubfeet treated with Ponseti method during the period between July 2012 and June 2016. All the clubfoot cases were treated by a single orthopedist. The patients, who had the unilateral clubfoot, had undergone the procedure of PAT, and had finished the recommended bracing phase and the evaluation of gait analysis, were included for the present study. The exclusion criteria were as follows: (1) the postural, syndromic, and neurological clubfeet, (2) the cases still remaining in the course of treatment without the evaluation using gait analysis, and (3) the cases, which underwent the surgical treatments such as the procedures of transfer of anterior tibialis tendon (TATT), Ilizarov technique, and/or extensive soft tissue release. Informed consent was obtained from all the parents. This study was approved by the institutional ethics committee.

The result in all the cases (16 boys and 3 girls) was rated as excellent according to the Functional Rating Scales of International Clubfoot Study Group (ICSG). There were 14 clubfeet on the right side and 5 clubfeet on the left side. The mean age was 4.5 years (range, 4 to 6 years) at the time when the test of gait analysis was undertaken, with mean body height of 108.6 cm (range, 103 to 122 cm) and mean body weight of 18.9 kg (range, 16 to 23.5 kg). Fourteen patients firstly came to visit us at our clinic with the mean presentation age of 82.9 days (range, 11 days to 7 months) while five patients received the previous treatments (mainly casting and PAT procedure) before referral to our clinic with the mean presentation age of 26 months (range, 8 months to 3 years). The mean Pirani score was 4.2 points (3 to 6 points) in the 15 feet from fifteen patients with age younger than 1 year while the remaining four feet from 4 patients with age older than 1 year were classified as moderate according to Dimeglio classification [16]. The initial complete correction was obtained in all the cases (100%), requiring mean numbers of 4.2 (2 to 6) casts before PAT procedure was performed. The affected sides of treated patients with clubfoot were grouped as Group CF while the contralateral normal sides were grouped as Group CL. A foot abduction orthosis (FAO) brace, which consisted of a pair of straight high-top shoes and a connected bar, was prescribed to prevent the relapse of deformity. The brace protocol was full-time use for the first 3 months and then 16 to 18 hours until the patient was 2 years old and then 14 to 16 hours until the patient was 4 years old. All the patients were followed up at the minimum of 4.5 years old.

All the subjects, who had finished the whole course of clubfoot management using Ponseti method, were regularly recommended to undertake the test of gait analysis for the functional evaluation in our partner institute-gait lab at Shanghai Yueyang Hospital. Anthropometric data, including height, weight, passive ankle dorsiflexion angle with knee extension, passive ankle plantar flexion angle, and thigh foot angle, were documented. Twenty-two passively reflective markers were placed on the standardized and specific anatomical landmarks. After the retroreflective markers were applied to the patients, they were instructed to walk barefoot at a self-selected speed over a 10 m walkway while the data capture was undertaken. Three-dimensional gait data was collected with the motion analysis system (Motion Analysis Corporation, USA). Four force plates (Advanced Mechanical Technology, Inc., Watertown, MA) were used for kinetic analysis. The spatial locations of the individual markers were recorded using twelve cameras (60 Hz) as the subjects walked. Temporal-spatial parameters, kinematic parameters, and kinetic parameters were collected as the gait summary measures for analysis. Calibration of the motion analysis system was performed each time before the subjects were taken for the test.

2.1. Statistical Analysis. Statistical analysis was performed using the statistical package of SPSS 17.0. Comparisons of groups in terms of clinical and physical evaluation, temporal-spatial parameters, and kinematic and kinetic variables were performed using paired t-test. Statistical significance was set at $p < 0.05$.

3. Results

3.1. Physical Examination. Anthropometrical characteristics of these two groups were presented in Table 1. Passive ankle dorsiflexion angle with knee extension in Group CF ($15.20 \pm 9.68^\circ$) was smaller than that in Group CL ($26.40 \pm 9.19^\circ$) with the statistically significant difference ($p = 0.000$). Significant difference was also statistically found in the parameter of passive ankle plantar flexion angle between these two groups ($42.13 \pm 7.28^\circ$ for Group CF and $47.93 \pm 6.70^\circ$ for Group CL, $p = 0.000$). There was no significant difference in the physical parameter of thigh foot angle ($9.53 \pm 9.83^\circ$ for Group CF and $10.07 \pm 10.61^\circ$ for Group CL, $p = 0.752$).

3.2. Temporal-Spatial Parameters. Table 2 shows the comparison of temporal-spatial parameters between Group CF and Group CL. The parameter of stride length in Group CF (73.10 ± 11.58 cm) was a little smaller than that in Group CL (73.59 ± 11.42 cm); however, no significant difference was found between these two groups ($p = 0.092$). There was no significant difference in the parameter of forward velocity between these two groups (79.72 ± 20.27 cm/s for Group CF and 79.93 ± 20.33 cm/s for Group CL,

Table 1: Physical parameters.

Parameters	Group CF	Group CL	t value	p value
Passive ankle dorsiflexion angle (deg)	15.20 ± 9.68	26.40 ± 9.19	$t = -4.836$	$p = 0.000$
Passive ankle plantar flexion angle (deg)	42.13 ± 7.28	47.93 ± 6.70	$t = -5.908$	$p = 0.000$
Thigh foot angle (deg)	9.53 ± 9.83	10.07 ± 10.61	$t = -0.323$	$p = 0.752$

Table 2: Temporal-spatial parameters.

Parameters	Group CF	Group CL	t value	p value
Stride length (cm)	73.10 ± 11.58	73.59 ± 11.42	$t = -1.777$	$p = 0.092$
Forward velocity (cm/s)	79.72 ± 20.27	79.93 ± 20.33	$t = -0.779$	$p = 0.446$
Cadence (steps/min)	129.96 ± 16.65	129.98 ± 16.37	$t = -0.076$	$p = 0.940$
Single support time (%)	37.56 ± 3.41	38.16 ± 3.45	$t = -1.470$	$p = 0.159$

Table 3: Kinematic parameters.

Parameters	Group CF	Group CL	t value	p value
Total ankle rotation (deg)	19.91 ± 5.10	16.87 ± 4.91	$t = 2.879$	$p = 0.011$
Peak ankle dorsiflexion (deg)	9.99 ± 4.90	10.85 ± 4.82	$t = -0.832$	$p = 0.416$
Peak ankle plantar flexion (deg)	−9.21 ± 6.57	−11.10 ± 6.84	$t = 1.565$	$p = 0.135$
Ankle range of motion (deg)	19.20 ± 4.53	21.95 ± 5.58	$t = -2.509$	$p = 0.022$
Internal foot progression angle (deg)	−1.38 ± 6.64	−8.09 ± 6.09	$t = 3.613$	$p = 0.002$

$p = 0.446$). The parameter of cadence in Group CF (129.96 ± 16.65 steps/min) was nearly identical to that in Group CL (129.98 ± 16.37 steps/min, $p = 0.940$). No significant difference was found in the parameter of single support time (%) between these two groups (37.56 ± 3.41 for Group CF and 38.16 ± 3.45 for Group CL, $p = 0.159$).

3.3. Kinematic Parameters. Table 3 shows the comparison of kinematic parameters between Group CF and Group CL. The total ankle rotation angle in coronal plane in Group CF (19.91 ± 5.10°) was significantly greater than that in Group CL (16.87 ± 4.91°, $p = 0.011$). There was no significant difference in the parameters of peak ankle dorsiflexion and peak ankle plantar flexion ($p > 0.05$, Figure 1). However, the parameter of total ankle range of motion was significantly greater in Group CL (21.95 ± 5.58°) than that in Group CF (19.20 ± 4.53°) ($p = 0.022$). Significant difference was also found in the parameter of internal foot progression angle (−1.38 ± 6.64° for Group CF and −8.09 ± 6.09° for Group CL, $p = 0.002$).

3.4. Kinetic Parameters. Table 4 shows the comparison of kinetic parameters between Group CF and Group CL. There was no significant difference in the parameters of peak ankle plantar flex moment between these two groups (0.61 ± 0.27 Nm/kg for Group CF and 0.71 ± 0.13 Nm/kg for Group CL, $p = 0.197$). No significant difference was found in the parameters of peak vertical ground reaction force (GRF) (1.03 ± 0.12 N/kg for Group CF and 1.06 ± 0.12 N/kg for Group CL, $p = 0.265$) and peak frontal propulsion force (0.15 ± 0.07 N/kg for Group CF and 0.15 ± 0.04 N/kg for Group CL, $p = 0.882$) between these two groups either. Significant difference was found in the parameter of peak ankle power (0.55 ± 0.23 Watts/kg for Group CF and 0.91 ± 0.47 Watts/kg for Group CL, $p = 0.001$) between these two groups (Figure 2).

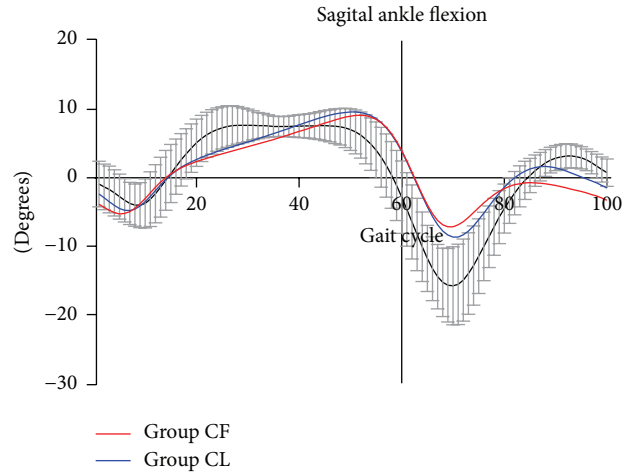


Figure 1: Averaged ankle range of motion in sagittal plane for Group CF (red) and Group CL (blue) over one complete gait cycle was compared. Positive values represent dorsiflexion, and negative values represent plantar flexion. Decreased plantar flexion at initial swing phase (toe off phase) was found for both Group CF and Group CL.

4. Discussion

Gait analysis has been used as a useful tool for assessing the functional outcomes by quantitatively measuring the motion of walking in terms of body movements, body mechanics,

Table 4: Kinetic parameters.

Parameters	Group CF	Group CL	t value	p value
Peak ankle plantar flex moment (Nm/kg)	0.61 ± 0.27	0.71 ± 0.13	$t = -1.352$	$p = 0.197$
Peak vertical GRF (N/kg)	1.03 ± 0.12	1.06 ± 0.12	$t = -1.160$	$p = 0.265$
Peak frontal propulsion (N/kg)	0.15 ± 0.07	0.15 ± 0.04	$t = -0.151$	$p = 0.882$
Peak ankle power (Watts/kg)	0.55 ± 0.23	0.91 ± 0.47	$t = -3.982$	$p = 0.001$

GRF: ground reaction force.

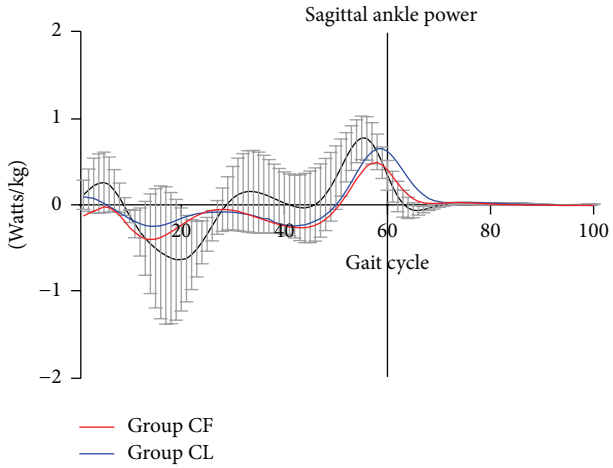


Figure 2: Averaged ankle power in sagittal plane for Group CF (red) and Group CL (blue) over one complete gait cycle was compared. Positive values are power generation (Gen) and negative values are power absorption (Abs). Ankle power at push-off phase was significantly decreased for Group CF in comparison with that for Group CL.

and the activity of the muscles to evaluate treatment outcome of clubfeet functionally and objectively over the last decades [17–20]. It is controversial as to the issue whether the Achilles tendon should be completely transected. As it was warned, the tendon continuity must be maintained with percutaneous Achilles tendon lengthening rather than Achilles tenotomy [21, 22] fearing the spastic gastrocnemius-soleus muscle contraction of the proximal tendon end, which is supposed to prevent the tendon-to-tendon healing and lead to myotendinous incompetence. However, Berg [23] reported that inadvertent Achilles tenotomy could rebuild the tendon continuity as long as the patients (3 children and 2 adults) were immobilized for a minimum of 2 months in a short leg walking cast. From the viewpoint of pediatric orthopedists in their practice of clubfoot management, the procedure of PAT has since been a safe and effective procedure to correct the equinus deformity once the forefoot is adequately abducted [24]. As to the issue of heel cord healing, Agarwal et al. [10] demonstrated that functional continuity of the Achilles tendons could be rebuilt at 4 weeks' time after tenotomy in terms of clinical and ultrasonographic measurements. Mangat et al. [25] reported that the complete healing of transected Achilles tendons required the time of at least twelve weeks although there was evidence of continuity of the Achilles tendon at three weeks after tenotomy. Saini et al. [11] demonstrated that the continuity of the Achilles tendon in all cases could be reestablished at the end of 6 weeks in terms of clinical assessment and 6 months of the tenotomy in terms of MRI imaging evaluation. All the data from ultrasonographic and MRI studies supported that the Achilles tendon did regenerate following the procedure of PAT. In our clinical practice, the healing of Achilles tendon was recognized from the clinical evaluations including heel raise test and walking gait based on the general observation. However, the specific difference of foot function between the feet with and without the procedure of PAT procedure has been poorly reported using the detailed methods of gait analysis. In the present study, we aimed to quantify the foot function of unilateral clubfoot, which underwent the procedure of PAT in comparison with the contralateral foot in terms of gait analysis.

It is suggested based on the findings of the present study that the majority of the affected feet have obtained the satisfactory gait and function following the procedure of PAT in terms of the temporal-spatial parameters during the complete gait cycle. The values of passive ankle dorsiflexion and plantar flexion angles presented the significant reduction angles for Group CF in comparison with those for Group CL ($p < 0.05$). However, the angles of ankle passive dorsiflexion and plantar flexion for Group CF were great enough to complete the whole gait cycle without any limitation (Table 3, $p > 0.05$). Dynamic ankle range of motion for Group CF was found to present the dramatic reduction (Table 3, $p < 0.05$). Mindler et al. [26] also reported significantly decreased range of motion at the ankles in the Ponseti group in comparison with that in the controls. This may be attributed to the underlying musculoskeletal pathologies as well as the long time casting immobilization. The mean foot progression angle ($-1.38 \pm 6.64^\circ$) for Group CF also presented the significant reduction in comparison with that for Group CL ($-8.09 \pm 6.09^\circ$). However, it was more externally oriented comparing with that reported by Karol et al. [19] and more internally oriented comparing with that reported by Mindler et al. [26]. We have found that the subjects of Group CF walked similarly to those of Group CL in terms of temporal-spatial parameters such as stride length, forward velocity, cadence, and single support time ($p > 0.05$). The similar results were reported in previous studies [27–29]. Aksahin et al. [30] found that clubfoot patients, who underwent posteromedial release surgery, walked slower than those from the control group with short steps. The kinetic parameters were comparable between these two groups except for the parameter of peak ankle power. The mean value of the peak ankle power in the Group CF undergoing the procedure

of PAT (0.55 Watts/kg) was 60.4% of that in Group CL (0.91 Watts/kg). Reduced ankle power percentages relative to controls have been reported in some other studies [19, 26, 31]. In the present study, peak ankle powers of Group CF (0.55 Watts/kg) and Group CL (0.91 Watts/kg) presented the great reduction comparing with those in the previous studies (2.2 Watts/kg for Ponseti group and 2.6 Watts/kg for control group [26]; 1.19 watts/kg for surgical group and 1.25 for control group [30]) as to the outcome evaluation of clubfoot treatment. This may be attributed supposedly to the variations in regions, races, and demographic characteristics among the different reports. Ankle power is affected by both range of motion and muscle strength [19]. Because normal ankle plantar flexion is requisite for foot push-off, insufficient ankle plantar flexion may be an important factor for difficulty of foot propulsion. In the present study, peak ankle plantar flexion along with ankle plantar flex moment was comparable between Group CF and Group CL ($p > 0.05$). The reduced ankle power for Group CF may be supposedly attributed to the procedure of PAT. However, it was difficult to draw the conclusion because the smaller gastrosoleus muscle in the bulk of the most clubfeet patients may lead to relative weakness and reduced ankle power. Previous studies also supported an association between clubfoot and diminished ankle power [29, 32, 33]. No significant difference was found in other kinetic parameters such as peak ankle plantar flex moment, peak vertical ground reaction force (GRF), and peak frontal propulsion force.

There were a few limitations in the present study. Assuming that the foot could be taken as a single rigid segment, pivoting at the ankle joint, the traditional single-segment foot model (Helen Hayes model) was used for gait analysis in the present study. However, multisegment foot model may enhance the accuracy of the gait analysis data and provide valuable additional information among the different foot segments including Milwaukee Foot Model, Oxford Foot Model, and Leardini (IOR) Foot Model [34]. Either way it is not unusual that the child does not walk in their "normal" fashion while he or she is undergoing the test of gait analysis. Finally, the threshold for what is considered as pathologic versus functional gait in children is still controversial. However, we applied paired t-test for statistical analysis and observed the gait difference in the children with unilateral clubfoot undergoing the procedure of PAT in comparison with that in contralateral normal foot. This may be supposed to reduce the bias among the groups.

As revealed in this study, the feet following the procedure of PAT showed no obvious gait deviation in outcome measure with the reference to the temporal-spatial, kinematic, and kinetic parameters of gait analysis. Based on the findings from this study, it is demonstrated that good or excellent results can be achieved in the foot undergoing the procedure of PAT at the minimum of four-year follow-up. The Achilles tendon healed well in terms of clinical assessment and gait analysis. It is recommended that the procedure of PAT be safe and efficient for correcting the equinus deformity in case of clubfoot management and preserving the main function of Achilles tendon.

Competing Interests

The authors declare that they have no competing interests.

Authors' Contributions

Yu-Bin Liu and Shu-Yun Jiang contributed equally to this article.

Acknowledgments

This study was supported by the Municipal Hospital Emerging Frontier Technology Joint Research Program, Shanghai Hospital Development Center, no. SHDC12015130. This study was conducted in both Xin-Hua Hospital, Shanghai Jiao Tong University School of Medicine, and Yueyang Hospital, Shanghai University of Traditional Chinese Medicine, Shanghai, China.

References

[1] G. A. Lichtwark and A. M. Wilson, "*In vivo* mechanical properties of the human Achilles tendon during one-legged hopping," *The Journal of Experimental Biology*, vol. 208, part 24, pp. 4715–4725, 2005.

[2] N. Maffulli, S. W. Waterston, J. Squair, J. Reaper, and A. S. Douglas, "Changing incidence of Achilles tendon rupture in Scotland: a 15-year study," *Clinical Journal of Sport Medicine*, vol. 9, no. 3, pp. 157–160, 1999.

[3] A. Möller, M. Åström, and N. E. Westlin, "Increasing incidence of Achilles tendon rupture," *Acta Orthopaedica Scandinavica*, vol. 67, no. 5, pp. 479–481, 1996.

[4] S. Thomopoulos, W. C. Parks, D. B. Rifkin, and K. A. Derwin, "Mechanisms of tendon injury and repair," *Journal of Orthopaedic Research*, vol. 33, no. 6, pp. 832–839, 2015.

[5] V. Gulati, M. Jaggard, S. S. Al-Nammari et al., "Management of achilles tendon injury: a current concepts systematic review," *World Journal of Orthopaedics*, vol. 6, no. 4, pp. 380–386, 2015.

[6] D. M. van der Eng, T. Schepers, J. C. Goslings, and N. W. L. Schep, "Rerupture rate after early weightbearing in operative versus conservative treatment of Achilles tendon ruptures: a meta-analysis," *The Journal of Foot and Ankle Surgery*, vol. 52, no. 5, pp. 622–628, 2013.

[7] D. Zhao, H. Li, L. Zhao, J. Liu, Z. Wu, and F. Jin, "Results of clubfoot management using the Ponseti method: do the details matter? A systematic review," *Clinical Orthopaedics and Related Research*, vol. 472, no. 4, pp. 1329–1336, 2014.

[8] J. A. Morcuende, L. A. Dolan, F. R. Dietz, and I. V. Ponseti, "Radical reduction in the rate of extensive corrective surgery for clubfoot using the Ponseti method," *Pediatrics*, vol. 113, no. 2, pp. 376–380, 2004.

[9] M. B. Dobbs, J. R. Rudzki, D. B. Purcell, T. Walton, K. R. Porter, and C. A. Gurnett, "Factors predictive of outcome after use of the Ponseti method for the treatment of idiopathic clubfeet," *The Journal of Bone & Joint Surgery—American Volume*, vol. 86, no. 1, pp. 22–27, 2004.

[10] A. Agarwal, N. A. Qureshi, P. Kumar, A. Garg, and N. Gupta, "Ultrasonographic evaluation of Achilles tendons in clubfeet before and after percutaneous tenotomy," *Journal of Orthopaedic Surgery*, vol. 20, no. 1, pp. 71–74, 2012.

[11] R. Saini, M. S. Dhillon, S. K. Tripathy et al., "Regeneration of the Achilles tendon after percutaneous tenotomy in infants: a clinical and MRI study," *Journal of Pediatric Orthopaedics B*, vol. 19, no. 4, pp. 344–347, 2010.

[12] J. M. Webb and G. C. Bannister, "Percutaneous repair of the ruptured tendo Achillis," *The Journal of Bone & Joint Surgery—British Volume*, vol. 81, no. 5, pp. 877–880, 1999.

[13] I. K. Y. Lo, A. Kirkley, B. Nonweiler, and D. A. Kumbhare, "Operative versus nonoperative treatment of acute achilles tendon ruptures: a quantitative review," *Clinical Journal of Sport Medicine*, vol. 7, no. 3, pp. 207–211, 1997.

[14] R. Cetti, S.-E. Christensen, R. Ejsted, N. M. Jensen, and U. Jorgensen, "Operative versus nonoperative treatment of Achilles tendon rupture. A prospective randomized study and review of the literature," *The American Journal of Sports Medicine*, vol. 21, no. 6, pp. 791–799, 1993.

[15] L. N. Zhang, W. B. Wan, Y. X. Wang et al., "Evaluation of elastic stiffness in healing achilles tendon after surgical repair of a tendon rupture using in vivo ultrasound shear wave elastography," *Medical Science Monitor*, vol. 22, pp. 1186–1191, 2016.

[16] A. Dimeglio, H. Bensahel, P. Souchet, P. Mazeau, and F. Bonnet, "Classification of clubfoot," *Journal of Pediatric Orthopedics Part B*, vol. 4, no. 2, pp. 129–136, 1995.

[17] K. A. Jeans, A. L. Erdman, C. H. Jo, and L. A. Karol, "A longitudinal review of gait following treatment for idiopathic clubfoot: gait analysis at 2 and 5 years of age," *Journal of Pediatric Orthopaedics*, vol. 36, no. 6, pp. 565–571, 2016.

[18] H. P. Gottschalk, L. A. Karol, and K. A. Jeans, "Gait analysis of children treated for moderate clubfoot with physical therapy versus the Ponseti cast technique," *Journal of Pediatric Orthopaedics*, vol. 30, no. 3, pp. 235–239, 2010.

[19] L. A. Karol, K. Jeans, and R. ElHawary, "Gait analysis after initial nonoperative treatment for clubfeet: intermediate term followup at age 5," *Clinical Orthopaedics and Related Research*, vol. 467, no. 5, pp. 1206–1213, 2009.

[20] R. El-Hawary, L. A. Karol, K. A. Jeans, and B. S. Richards, "Gait analysis of children treated for clubfoot with physical therapy or the Ponseti cast technique," *Journal of Bone and Joint Surgery A*, vol. 90, no. 7, pp. 1508–1516, 2008.

[21] M. J. Moreau and D. M. Lake, "Outpatient percutaneous heel cord lengthening in children," *Journal of Pediatric Orthopaedics*, vol. 7, no. 3, pp. 253–255, 1987.

[22] R. N. Hatt and T. A. Lamphier, "Triple hemisection: a simplified procedure for lengthening the Achilles tendon," *The New England Journal of Medicine*, vol. 236, no. 5, pp. 166–169, 1947.

[23] E. E. Berg, "Percutaneous achilles tendon lengthening complicated by inadvertent tenotomy," *Journal of Pediatric Orthopaedics*, vol. 12, no. 3, pp. 341–343, 1992.

[24] M. T. Alam, E. B. Akber, Q. S. Alam et al., "Outcome of percutaneous tenotomy in the management of congenital talipes equino varus by Ponseti method," *Mymensingh Medical Journal*, vol. 24, no. 3, pp. 467–470, 2015.

[25] K. S. Mangat, R. Kanwar, K. Johnson, G. Korah, and H. Prem, "Ultrasonographic phases in gap healing following Ponseti-type Achilles tenotomy," *The Journal of Bone & Joint Surgery—American Volume*, vol. 92, no. 6, pp. 1462–1467, 2010.

[26] G. T. Mindler, A. Kranzl, C. A. M. Lipkowski, R. Ganger, and C. Radler, "Results of gait analysis including the oxford foot model in children with clubfoot treated with the ponseti method," *The Journal of Bone & Joint Surgery—American Volume*, vol. 96, no. 19, pp. 1593–1599, 2014.

[27] L. A. Karol, M. C. Concha, and C. E. Johnston II, "Gait analysis and muscle strength in children with surgically treated clubfeet," *Journal of Pediatric Orthopaedics*, vol. 17, no. 6, pp. 790–795, 1997.

[28] H. T. Hee, E. H. Lee, and G. S. Lee, "Gait and pedobarographic patterns of surgically treated clubfeet," *The Journal of Foot and Ankle Surgery*, vol. 40, no. 5, pp. 287–294, 2001.

[29] T. C. Davies, G. Kiefer, and R. F. Zernicke, "Kinematics and kinetics of the hip, knee, and ankle of children with clubfoot after posteromedial release," *Journal of Pediatric Orthopaedics*, vol. 21, no. 3, pp. 366–371, 2001.

[30] E. Aksahin, H. Y. Yuksel, G. Yavuzer, H. H. Muratli, L. Celebi, and A. Bicimlioglu, "Quantitative gait characteristics of children who had successful unilateral clubfoot operation," *Acta Orthopaedica et Traumatologica Turcica*, vol. 44, no. 5, pp. 378–384, 2010.

[31] C. M. Duffy, J. J. Salazar, L. Humphreys, and B. C. McDowell, "Surgical versus ponseti approach for the management of CTEV: a comparative study," *Journal of Pediatric Orthopaedics*, vol. 33, no. 3, pp. 326–332, 2013.

[32] J. Aronson and C. L. Puskarich, "Deformity and disability from treated clubfoot," *Journal of Pediatric Orthopaedics*, vol. 10, no. 1, pp. 109–119, 1990.

[33] T. N. Theologis, M. E. Harrington, N. Thompson, and M. K. D. Benson, "Dynamic foot movement in children treated for congenital talipes equinovarus," *Journal of Bone and Joint Surgery B*, vol. 85, no. 4, pp. 572–577, 2003.

[34] A. C. Novak, D. J. Mayich, S. D. Perry, T. R. Daniels, and J. W. Brodsky, "Gait analysis for foot and ankle surgeons—topical review, part 2: approaches to multisegment modeling of the foot," *Foot and Ankle International*, vol. 35, no. 2, pp. 178–191, 2014.