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Epidemiology and Diagnosis of Periodontal Diseases: Recent Advances and Emerging Trends

Guest Editors: S. Al-Mubarak, S. Ciancio, and J. K. Baskaradoss

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Editorial

Epidemiology and Diagnosis of Periodontal Diseases: Recent Advances and Emerging Trends

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Periodontal diseases, including gingivitis and periodontitis, are the most commonly occurring, yet very specific, infections of the oral cavity. When the infection is progressive, it changes from a reversible diagnosis of gingivitis to a less favorable, more difficult to reverse condition: periodontitis. Previously, research was focused on the microbiological aspects of the periodontitis. Currently, it has been well established that microorganisms alone are not sufficient for the initiation of periodontal diseases. Factors like host response, health behaviour, and stress, among other risk factors, play an important role in the presence of the disease.

Research during the past 50 years have markedly improved our understanding of the biological mechanisms of periodontal diseases. We believe that scientific partnerships among dedicated practitioners along with extensive international collaboration on research activity can further enhance existing knowledge in this field.

This special issue contains five review articles, from authors in different specialties in dentistry. In the paper entitled "*Common periodontal diseases of children and adolescents*," Al-Ghutaimel et al. present the role of periodontal/gingival diseases in children and adolescents. This paper, highlights the importance of periodontal and gingival health in the overall wellbeing of a child.

In the paper "*The effect of orthodontic therapy on periodontal health: a review of the literature*," S. Alfuriji et al. present a review on the role of periodontal health in orthodontic therapy. Knowledge about this interaction is of great importance not only for clinicians but also for researchers, as it has been shown that orthodontic forces tend to induce an inflammatory reaction in the periodontium.

The paper "*Risk factors of periodontal disease: review of the literature*," by Y. A. AlJehani, provides an in-depth review of the current evidence on the potential roles of modifiable and nonmodifiable risk factors associated with periodontal disease. This review article also briefly describes the various risk characteristics that are related to periodontal diseases.

In the paper "*A new classification of endodontic-periodontal lesions*," K. S. Al-Fouzan presents a new and modified method of classifying endo-perio lesions. The differential diagnosis of endodontic and periodontal diseases can sometimes be difficult, but it is of vital importance to make a correct diagnosis for providing the appropriate treatment.

The paper "*Diagnostic applications of cone-beam CT for periodontal diseases*," by Y. A. AlJehani, describes the advantages of using cone-beam CT (CBCT) in the diagnosis of periodontal diseases. CBCT is a relatively new imaging modality that is widely used in general and specialized dentistry. This review is highly relevant since only few studies have appraised the role of CBCT in the diagnosis of periodontal diseases. The author has highlighted the role of CBCT in the diagnosing bony craters and defects, in measuring bone levels and in the visualization of the periodontal ligament space.

The published articles are not exhaustive representation of the recent advances in the field of epidemiology and diagnosis of periodontal diseases. Nonetheless, the papers provide a rich and many-faceted knowledge, that we have

the pleasure of sharing with the readers. We would like to thank the authors for their excellent contributions and all the reviewers for their patience and cooperation.

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Review Article

Common Periodontal Diseases of Children and Adolescents

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Background. Since 2000, studies, experiments, and clinical observations revealed high prevalence of periodontal diseases among children and adolescents. Therefore, this paper was designed to provide an update for dental practitioners on epidemiology, microbiology, pathology, prevention, diagnosis, and treatment of periodontal diseases in children and adolescents. *Methods.* This paper reviews the current literature concerning periodontal diseases in pediatric dentistry. It includes MEDLINE database search using key terms: "periodontal diseases in children," "Periodontal diseasesin adolescents," "periodontal diseases risk factors," "microbiology of periodontal diseases," "classification of periodontal diseases," "epidemiology of periodontal diseases," and "treatment of periodontal diseases." Articles were evaluated by title and/or abstract and relevance to pediatric dentistry. Sixty-five citations were selected by this method and by the references within the chosen articles. A review of the comprehensive textbooks on pediatric dentistry and periodontology was done. Some recommendations were based on the opinions of experienced researchers and clinicians, when data were inconclusive.

1. Periodontium of the Primary Dentition

In medical dictionaries, the word periodontium comes from the Greek terms peri-, which means "around," and -odons, which means "tooth." Literally, it means that which is around the tooth. Periodontium includes the tissues that surround and support the teeth. Those tissues are gingiva, cementum, periodontal ligaments, and alveolar bone [1, 2]. A long time ago, it has been found that periodontium of the primary dentition differs from that of the permanent dentition in several aspects [3]. The gingiva in primary dentition appears to be more reddish, vascular, and flabby and to lack stippling [1, 4]. And the periodontal ligaments in children are wider and have less dense fibers [1, 3, 4]. The alveolar bone in primary dentition has less trabecula and calcification, more marrow spaces, and greater blood supply and lymphatic drainage [1, 3, 4]. At the molecular level, some investigators reported that periodontium of the primary dentition resorbed more easily because it contains more sialoprotein and osteoprotein, which facilitate the binding of odontoclast [1, 5–7].

2. Periodontal Diseases

2.1. Definition. Periodontal diseases constitute a group of conditions that are considered nowadays ubiquitous among children, adolescents, and adults [3]. The term "periodontal diseases" includes any inherited or acquired disorders of the tissues that are investing and supporting the teeth (gingiva, cementum, PDL, and alveolar bone) [2]. Another researcher defined periodontal diseases as chronic infectious disorders caused primarily by bacteria [14].

2.2. Epidemiology. In 1996, Albandar et al. assessed the prevalence of gingivitis among large group of adolescents in the United States and found that 82.1% of the participating subjects were having gingivitis [15]. Similar findings of high prevalence of gingivitis among children and adolescents were reported by other studies worldwide [16, 17]. Albandar et al., in another study, assessed the prevalence of early-onset forms of periodontitis among group of US adolescents and reported that 0.6% of the subjects were having juvenile periodontitis at the age of 13–15, and 2.75% of the subjects were having chronic periodontitis at the age of 16-17 [18]. Low prevalence of periodontitis among children and adolescents was reported by other studies in different populations [19]. Many researchers have observed larger amount of plaque and less inflammation in relation to the amount of plaque in children compared to the adults [3, 4, 14]. Furthermore, experts and clinicians noted that most of the periodontal

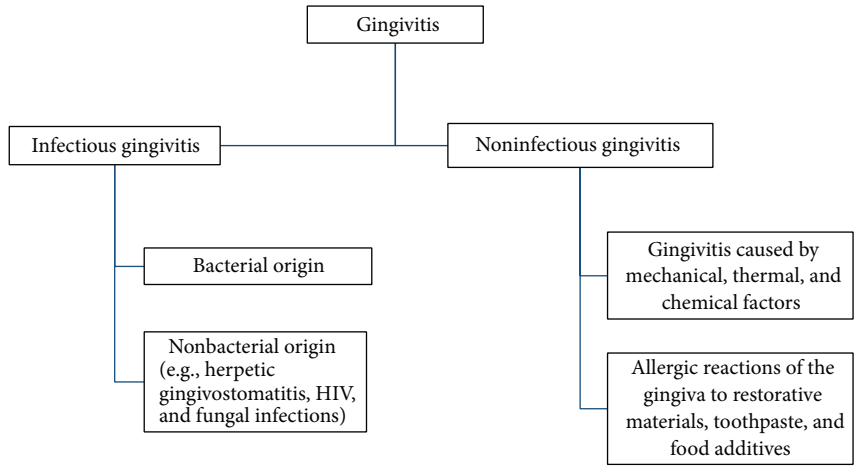

Figure 1: Classification of gingivitis.

diseases that affect children and adolescents are reversible and cause little tissue damage compared to the adults [4].

2.3. Causes. Periodontal diseases are most commonly caused by pathogenic microorganism in the oral biofilm or dental plaque that accumulated around the teeth due to poor oral hygiene [3, 4]. The evidences indicate that periodontal diseases develop when the numbers of Gram-negative bacteria and anaerobes in subgingival plaque increased [20, 21]. Numerous research efforts were implemented in order to identify bacterial species that are associated with the periodontal diseases [22, 23]. The most common periodontal-diseases-associated microorganisms were *Aggregatibacter* (*Actinobacillus*), *Porphyromonas gingivalis*, *Tannerella forsythensis*, and spirochaete *Treponema denticola* [2, 24–28]. Recent studies implicate fungi, such as *Candida albicans*, and Herpes viruses in the pathogenesis of periodontal diseases among immune-compromised children [29–31]. However, genetic, developmental, traumatic, neoplastic, and metabolic factors contributed to the cause of these diseases [9, 11, 24]. Furthermore, some systemic diseases and medications also have periodontal manifestations [2–4].

2.4. Classification. Over the last few decades, the nomenclature and classification of periodontal diseases changed periodically [3]. Regardless of the causative factors, periodontal diseases are divided into destructive and nondestructive form [14]. Gingivitis is a reversible and nondestructive form of periodontal diseases [14, 32, 33]. It is the inflammation of the marginal gingiva that may progress to include free and attached gingiva but causing no attachment loss [9, 11]. Based on clinical findings and diagnosis, gingivitis was subdivided into infectious and noninfectious forms as in Figure 1 [14, 34–37]. On the other hand, the irreversible and destructive form of periodontal diseases is periodontitis [14]. It is the inflammation of the tooth supporting tissue, which is accompanied by loss of connective tissue attachment and breakdown of the supporting alveolar bone [9, 11]. Periodontitis may progress to cause exposure of the roots, mobility, and premature loss of the teeth [9]. In 1989, the American Academy of Periodontology set criteria in order to distinguish various forms of periodontitis [3]. Those criteria are (1) age at onset, (2) distribution of the sites affected by the disease, (3) presence or absence of the systemic diseases, (4) rate of the disease progression, (5) response to treatment, and (6) presence or absence of specific host or microbial factors (the consensus of the world workshop in clinical periodontics) [3]. The most recent classification of periodontal diseases was introduced in 1999 by international workshop of periodontology and includes greater variety of periodontal diseases categories [3, 38, 39]. However, this paper will not follow specific classification system but rather will focus primarily on the periodontal diseases that are most commonly seen in pediatric dental patients.

2.4.1. Gingivitis. As mentioned earlier in this paper, gingival problems, either in acute or chronic nature, are nearly universal among children and adolescents [19]. Diagnosis of various types of gingivitis relied mainly on the clinical findings and manifestations [3, 32]. Those findings include redness and edema of the marginal gingiva and bleeding upon probing [2–4]. As disease persists, gingival margin may become rolled, interdental papilla may become enlarged and bulbous, bleeding may start spontaneously, and probing depth may increase as a consequence of gingival overgrowth (hyperplasia and hypertrophy) [2, 3, 32, 34].

Histologically, ulceration of the sulcular epithelium was observed both in children and in adolescents [32, 34]. However, researchers have noted predominance of T-lymphocyte infiltrate in gingivitis in children compared to B-cell (plasma cells) infiltrate in gingivitis in adolescents (Ranney et al., 1981, and Page and Schroeder, 1976) [3, 4]. Although the microbiological picture of gingivitis in children and adolescents has not been completely characterized, certain bacterial species have been found in experimental studies [4]. Those species were *Aggregatibacter (Actinobacillus)* sp., *Capnocytophaga* sp., *Leptotrichia* sp., and *Selenomonas* sp. [27, 28].

Gingival problems that are commonly seen in children and adolescents are as follows.

(1) Eruption Gingivitis. Some gingival inflammation normally accompanies eruption process [4, 9, 11]. Poor oral hygiene by neglect or as a consequence of malalignment of the erupting teeth will aggravate gingival inflammation [2–4, 9, 11]. Usually, the condition will subside as the oral hygiene improves and the tooth reaches normal occlusion [2, 4, 9, 11]. Plaque control regimen is the treatment of eruption gingivitis [4].

(2) Pubertal Gingivitis. Pubertal gingivitiswhich isalso called steroid hormone-related gingivitis [3] is defined as exacerbation of gingivitis by fluctuation in gonadotrophic hormone levels during puberty [3, 4, 11]. A similar condition is seen during pregnancy (Loe, 1965) and in females taking contraceptives (Kalkwarf, 1978) [2–4, 11]. The phenomenon of this condition can be explained as any increase in the levels of estrogen and progesterone in the gingival tissues resulting in vasodilatation and proliferation, increase in gingival vascularity, and increase in susceptibility of inflammation in the presence of local factors [4, 11, 40, 41].

Pubertal gingivitis is characterized by swelling of the interdental papilla, with spontaneous gingival hemorrhage [3, 4]. Professional prophylaxis and removal of the local factors combined with good oral hygiene regimen at home will result in major improvements [4]. In some cases, gingival swelling becomes fibrotic and necessitates surgical excision in the future [3].

(3) Gingivitis Related to Mouth-Breathing. Mouth-breathing causes desiccation of the oral tissue and consequently gingival inflammation and halitosis [4, 11]. Immediate management for the problem includes (1) maintaining good oral hygiene, (2) lubrication of the tissue, and (3) the use of the oral screen to cover the tissue during sleeping [4]. Elimination of the problem requires comprehensive treatment plan by an orthodontist and an otolaryngologist [3, 26].

(4) Drug Induced Gingival Overgrowth. Certain classes of medications have been approved to cause gingival overgrowth and aggravate gingival inflammation in the presence of local factors [3, 4, 41]. Those medications are cyclosporine (immune-depressant), phenytoin (anticonvulsant), and calcium channel blockers (antihypertensive) [2, 3, 11, 41]. Gingival overgrowth was noted in 30% of patients taking cyclosporine, 50% of patients using phenytoin, and 15% of patients medicated with calcium channel blockers such as nifedipine, verapamil, and amlodipine [3, 41, 42]. This kind of gingival overgrowth usually starts at the interdental area and then spreads to include marginal gingiva [3, 4, 41]. Occasionally, it can be so severe to cover the incisal and occlusal surfaces of the teeth [11, 41]. However, its severity is closely related to the amount of accumulated plaque [4, 11, 41].

The pathogenesis of this condition is uncertain yet [11]. However, the interaction between those drugs and/or metabolites and fibroblast will lead to fibroepithelial gingival overgrowth, epithelial acanthosis, increase in fibroblast number, and increase in collagen production [43].

The management of this condition starts from improving patient's oral hygiene by both mechanical and chemical plaque control techniques [4, 11]. In addition, professional scaling and polishing are required to remove all the local aggravating factors [2–4, 11]. Sometimes, gingivectomy and gingivoplasty are needed for gingival recontouring in order to improve esthetic and hygiene [3, 4]. Dentist should not try to stop or replace patient medications [4]. However, a consultation with the patient's physician can be done to determine the possibility of drug replacement [3, 44].

(5) Gingivitis Associated with Malnutrition. There is strong evidence that hypovitaminoses and mineral deficiency associated with specific manifestation in oral and perioral area may lead to periodontal diseases [2, 11]. For example, vitamin C deficiency will cause scurvy, which is manifested as a decrease in the production and maintenance of collagen [11]. Oral scurvy is characterized by painful gingival swelling, gingival edema, and hemorrhage on slight provocation [11, 45]. "Scorbutic gingivitis" results when severe vitamin C deficiency is combined with poor oral hygiene [11, 46, 47]. However, it is characterized by ulcerative gingivitis, fetid odor, rapid development of periodontal pocket, and tooth loss [46, 47].

(6) Acute Necrotizing Ulcerative Gingivitis (ANUG). Trench mouth or Vincent's infection is an acute gingival inflammation caused mainly by a special bacterial species called *Borrelia vincentii* [4, 9, 11]. Occasionally, other anaerobes and spirochetes such as *Fusobacterium* spp., *Selenomonas* spp., *Prevotella* spp., and *Treponema* spp. are observed in microbiological culture [48]. The risk factors include poor oral hygiene, stress, decreased host resistance, and HIV infection [4, 11]. ANUG is characterized by punched-out interdental papilla that is covered with a grayish-white pseudomembrane, which may extend to cover marginal gingiva [4, 48, 49]. Patients are usually suffering from strong continuous pain and fetid odor as a result of bacterial reaction's end products, bacterial toxins, and tissue necrosis [4, 49]. Generalized systemic manifestation including low-grade fever, lymphadenopathy, and malaise is often accompanying ANUG [48, 49].

Both local and systemic therapy are needed for the treatment of ANUG [4, 48, 49]. The first step is professional gentle scaling to remove local deposits as well as necrotic tissue [4]. Patients are instructed to follow strict daily oral hygiene regimen [4, 49]. Oxidizing mouthwash such as chlorhexidine may help to restore microbial balance [4, 48, 49]. 250–500 mg per dose of penicillin or erythromycin was recommended for five days [4, 48, 49]. Flagyl (metronidazole) is approved by evidence to help in eliminating the acute symptoms rapidly [11].

(7) Primary Herpetic Gingivostomatitis. Primary herpetic gingivostomatitis is defined as an acute gingival condition that is caused by Herpes simplex virus type I [3, 4]. Its clinical picture is characterized by a painful gingival inflammation and vesicles that are formed mainly on the dorsum of the tongue, hard palate, and gingiva [4]. Those vesicles ruptured eventually, leaving a painful ulcer with a

Table 1: Systemic and genetic disorders associated with periodontal diseases in children and adolescents.

Systemic or genetic disorder	Nature of the disorder	Periodontal and other manifestations
Insulin dependent diabetes mellitus (IDDM)	Decrease in insulin secretion or availability caused by genetic defect in pancreatic beta-cells [8–10].	(i) Gingivitis, attachment loss, and bone loss are more prevalent in poorly controlled cases [4]. (ii) Reduced PMNs functions (chemotaxis, adhesion, and phagocytosis) [3, 11]. (iii) Decreased collagen synthesis and increased collagenase activity [4]. (iv) Delayed wound healing [3, 4]. (v) Increased susceptibility to infections [8–10].
HIV/AIDS	HIV/AIDS develops as a result of infection with human immunodeficiency virus [3].	(i) Linear gingival erythema [3, 4]. (ii) Acute necrotizing ulcerative gingivitis [3, 4, 11]. (iii) Acute necrotizing periodontitis [12, 13].
Leukocyte adhesion deficiency (LAD)	Inherited as autosomal recessive condition in which glycoprotein adhesion in leukocyte molecules is severely reduced [3, 11].	(i) Poor immune response to bacterial infections [3, 4]. (ii) Acute inflammation and rapid bone loss [3, 4, 11]. (iii) Recurrent bacterial infections [3]. (iv) Poor wound healing [3, 4]. (v) Associated with prepubertal periodontitis [3, 8, 11].
Leukemia	Uncontrolled proliferation of white blood cells [3, 4].	(i) Gingival hyperplasia and hypertrophy [3, 4]. (ii) Gingival pallor [3, 4, 11]. (iii) Spontaneous gingival hemorrhage and petechiae [3, 8].
Neutropenia	The number of PMNs in peripheral blood is below $1000/mm^3$ in infants and $1500/mm^3$ in children [3, 4].	(i) Severe gingivitis, gingival ulcerations, and periodontitis [3, 4]. (ii) Recurrent infections such as otitis media and upper respiratory infections [3, 9, 11].
Acrodynia	Acrodynia is caused by mercurial toxicity reaction (mercury poisoning or idiosyncrasy to mercury) [3, 4, 11].	(i) Gingival and mucosal hyperplasia [3]. (ii) Alveolar bone loss [3, 4]. (iii) Early loss of primary teeth [3, 4]. (iv) Profuse salivation and sweating [3, 11].
Histiocytosis X	Disturbance of the reticuloendothelial system includes defects in PMNs and monocyte [3, 4, 11].	(i) Increased susceptibility to bacterial infections [11].
Hypophosphatasia	Genetic disorder characterized by low level of serum alkaline phosphatase and excretion of phosphoethanolamine in urine [3, 4, 11].	(i) Premature loss of deciduous teeth and skeletal deformity [3, 4, 11]. (ii) Defective bone/tooth mineralization [3, 4, 11]. (iii) Cementum hypoplasia/aplasia [3, 4, 11].
Chediak-Higashi syndrome	Autosomal recessive disorder characterized by impaired function of cytoplasmic microtubules in PMNs [3, 4, 11].	(i) Recurrent infections [3]. (ii) Severe gingivitis and periodontitis [4]. (iii) Intraoral ulcerations [3, 11].
Papillon-Lefevre syndrome	Autosomal recessive condition associated with impaired neutrophil functions [3, 4, 11].	(i) Palmoplantar hyperkeratosis [3]. (ii) Early-onset periodontitis affecting both primary dentition and permanent dentition [3].
Down syndrome	Trisomy 21, mongolism, and autosomal chromosomal anomaly associated with impaired PMNs functions, connective tissue disorders, and gingival hyperinnervation [3, 11].	(i) Gingivitis and periodontitis especially in lower anteriors [11]. (ii) Enamel hypoplasia [3, 4, 11]. (iii) Microdontia [3, 4, 11]. (iv) Macroglossia [3, 4, 11]. (v) Fissured tongue [3, 4, 11].
Ehlers-Danlos syndrome	Collage disorder affecting joints and skin. Ten type; type VIII is autosomal dominant and has periodontal implications [11].	(i) Aggressive early-onset periodontitis [11]. (ii) Prolonged bleeding [3]. (iii) Easily traumatized mucosa. [11]

yellow gray floor and red halo [3, 4, 11]. Lymphadenopathy, fever, and malaise are common systemic features for herpetic gingivostomatitis [48, 49]. It is commonly affecting children under the age of ten with a peak incidence at 2–4 years of age [4]. The condition is self-limiting and required symptomatic treatment only [3, 4]. However, systemic antiviral therapy is needed in immunocompromised patients [11].

2.4.2. Periodontitis

(1) Chronic Periodontitis (Incipient). Although this form of periodontitis is considered more prevalent in adults, it can be seen occasionally in children and adolescents [9]. Comparing to aggressive periodontitis, chronic periodontitis is characterized by a low to moderate rate of progression that may include episodes of rapid destruction [9, 11]. It is subdivided according to the percentage of the involved sites into localized (<30%) and generalized (>30%) [2, 3, 9]. Furthermore, it can be subdivided according to the severity of the disease into mild (1-2 CAL), moderate (3-4 CAL), and severe (≥5 CAL) [9, 11].

(2) Aggressive Periodontitis. Aggressive periodontitis which is also called "juvenile periodontitis" is considered to be prevalent in children and adolescents during circumpubertal period [3, 10, 11]. It is characterized by rapid loss of connective tissue attachment and alveolar bone with familial aggregation [11]. It is caused by both pathogenic microflora and abnormality in host defense mechanisms [3]. Aggressive periodontitis can be subdivided into localized (LAgP) and generalized form (GAgP).

Localized aggressive periodontitis patients have interproximal attachment loss on no more than two teeth other than first permanent molars and incisors [3, 10, 11]. At the microbiological level, up to date, no single species of microorganism has been found in all cases of LAgP [9, 50]. However, *Aggregatibacter* (*Actinobacillus*) sp. in combination with *Bacteroides*-like sp. and *Eubacterium* sp. has been isolated from most of LAgP cases [51–54]. It is well documented that LAgP is associated with a variety of functional defects in neutrophils [10, 55].

Generalized aggressive periodontitis patients have interproximal attachment loss on at least three teeth that are not permanent first molars or incisors [3, 10]. It is usually affecting the entire dentition and is considered as a disease of adolescents and young adults [10]. At the microbiological level, *Porphyromonas gingivalis* and *Treponema denticola* were isolated from most GAgP cases [3, 10]. Patients with GAgP have defective neutrophil functions and reduction in GP-110 [10, 56]. Furthermore, alteration in IgG was reported to be present in both forms of aggressive periodontitis [10]. IgG is known to have a protective and disease-limiting effect [10].

The successful treatment of aggressive periodontitis includes surgical or nonsurgical periodontal therapy in combination with systemic antibiotic therapy [10, 57]. According to the number of studies, the most successful antibiotic in the treatment of aggressive periodontitis is tetracycline alone or with metronidazole [10, 57–59], followed by metronidazole in combination with amoxicillin in the presence of tetracycline resistance [10, 60].

(3) Periodontitis as a Manifestation of Systemic and Genetic Disorders. Include a group of rare diseases that predispose the affected individual to highly destructive periodontal infections [8, 9, 12]. Those diseases were characterized by defective functions of neutrophils and/or other immune cells [9]. The most common systemic diseases and genetic disorders that are associated with periodontal conditions are listed in Table 1.

The treatment of periodontitis as a manifestation of systemic diseases includes a combination of surgical and nonsurgical therapy in addition to antibiotic therapy [8, 9, 61, 62]. However, the success of treatment of periodontitis as a manifestation of systemic diseases is considered unpredictable [8, 9].

Conflict of Interests

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

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Review Article

The Effect of Orthodontic Therapy on Periodontal Health: A Review of the Literature

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Objectives. This review aims to evaluate the effect of orthodontic therapy on periodontal health. *Data.* Original articles that reported on the effect of orthodontic therapy on periodontal health were included. The reference lists of potentially relevant review articles were also sought. *Sources.* A literature search was conducted using the databases, Medline, EMBASE, Cochrane Library, Web of Science, Google Scholar, and Scopus databases for relevant studies. The search was carried out by using a combined text and the MeSH search strategies: using the key words in different combinations: "periodontal disease," "orthodontics" and "root resorption." This was supplemented by hand-searching in peer-reviewed journals and cross-referenced with the articles accessed. Articles published only in English language were included. Letters to the Editor, historical reviews and unpublished articles were not sought. *Conclusions.* Within the limitations of the present literature review, it was observed that there is a very close inter-relationship between the periodontal health and the outcome of orthodontic therapy.

1. Introduction

Orthodontic treatment ensures proper alignment of the teeth and improves the occlusal and jaw relationship. This not only aids in better mastication, speech, and facial aesthetics, but also contributes to general and oral health, thereby improving the quality of life. Like any other treatment modalities, orthodontic treatment, in addition to its benefits, has also associated risks and complications. However, the risk and complication associated with treatment are reported to be considerably lower compared to other surgical or nonsurgical interventions [1].

However, the most commonly reported adverse effects of orthodontic treatment can be both local and systemic. This includes, tooth discolorations, decalcification, root resorption, periodontal complications, psychological disturbances, gastrointestinal complications, allergic reactions, infective endocarditis, and chronic fatigue syndrome [1–4]. It has been shown that orthodontic forces represent a physical agent capable of inducing an inflammatory reaction in the periodontium [5]. This reaction is necessary for orthodontic tooth movement [6]. One of the challenges of orthodontics is to finish the orthodontic treatment with the least effects on the root and periodontium.

This review aims to highlight the main coordinates of risk issues like periodontal complication and root resorption in orthodontics.

2. Periodontal Complication

Periodontal health is an important factor that may be used to evaluate the success of orthodontic therapy. Periodontal complications are reported to be one of the most common side effects linked to orthodontics [7]. Also, properly aligned teeth are easier to clean, and perhaps correct occlusion may promote healthier periodontium. The periodontal complications associated with orthodontic therapy mainly include gingivitis, periodontitis, gingival recession or hypertrophy, alveolar bone loss, dehiscences, fenestrations, interdental fold, and dark triangles [1, 2, 8, 9]. Presence of microbial plaque is reported to be the most important factor in the initiation, progression, and recurrence of periodontal disease in reduced periodontium [10].

The reasons behind these periodontal complications involve patient factors and the technique used in the treatment [11]. Patient factors include past periodontal condition, increased susceptibility, and poor oral hygiene. Smoking is also a known factor that affects the periodontal support [12, 13].

Orthodontic treatment and the procedures are known to induce both positive and negative local soft-tissue reactions in the gingiva. The negative reaction is mainly associated with gingivitis.

The presence of plaque is the considered as one of the main factors in the development of gingivitis [11, 12]. Orthodontic brackets and elastics might interfere with effective removal of dental plaque, thereby increasing the risk of gingivitis. Few clinical studies also reported poor periodontal health and greater loss of clinical attachment level distally in the dental arches. This could be a result of poor oral hygiene in molar regions and the presence of molar bands, which favors food lodgment [14]. However, as a result of the orthodontic treatment a shift in the composition and type of bacteria can be expected [15]. Orthodontic treatment is known to affect the equilibrium of oral microflora by increasing bacteria retention. In a study done by Ristic et al. [16] an increase in the value of periodontal indices and growth of periodontopathogenic bacteria were observed in adolescent patients undergoing fixed orthodontic treatment. In the majority of the patients, following placement of a fixed appliance, small amount gingival inflammation is visible, which could be transient in nature and does not lead to attachment loss [17]. Some reports support the fact that the fixed orthodontic treatment may result in localized gingivitis, which rarely progresses to periodontitis [18]. Gingival inflammation around orthodontic bands leads to pseudopockets, which usually disappear with debanding of the brackets. However, this is usually resolved within weeks of debanding. However, some of the published researches have reported reduced risk of gingivitis in the absence of plaque, orthodontic forces, and tooth movements [7, 19, 20]. If the orthodontic forces kept within the adequate limits in healthy reduced periodontal tissue support regions, the chances of gingival inflammation will be minimal [21]. Alexander [14] in his results has also reported lack of periodontal destruction over a longer period of time among patients wearing fixed appliances.

Published reports on human periodontal tissues state that the orthodontic banding performed with great care and proper maintenance of oral hygiene can prevent permanent periodontal destruction [22].

3. Pathophysiology

The periodontal ligament mainly consists of type I collagen, although type III collagen fibres are also present. The main function of PDL is sending proprioceptive signals to the brain and withstanding compressive forces during chewing movements. Various studies have reported significant recruitment of mononucleated cells, macrophages, dendritic cells, and MHC class II Ia-expressing cells in the pressure zone incident to orthodontic tooth movement [23, 24]. In the tension zone, however, minimal changes in the number and distribution of immune cells have been reported [25]. Under stress from the orthodontic treatment, there would be changes to the blood flow [26].

Neuropeptides are released from the periodontal nerve endings, which causes neurogenic inflammation in the compressed periodontal ligament [27]. Furthermore, various immunoregulatory molecules, such as interleukin-1 a, interleukin-6, and tumour necrosis factor-a, are released during inflammation and participate in the remodelling of the periodontium [28].

3.1. Root Resorption. One of the challenges of orthodontics is to finish the orthodontic treatment with the least effects on the root and periodontium. Root resorption is considered as undesirable but unavoidable iatrogenic consequence of orthodontic treatment [29]. Individual biologic variability, genetic predisposition, and the effect of mechanical factors are believed to influence apical root resorption [30, 31]. This undesirable complication of orthodontic treatment may result in tooth mobility and even permanent tooth loss [32]. It is an inflammatory process resulting in an ischemic necrosis in the periodontal ligament when the orthodontic force is applied [33]. It appears that apical root resorption is not just a result of orthodontic force but instead a combination of individual biological variability and the effect of mechanical factors [31].

Root resorption is a common consequence associated with orthodontic treatment. It has received considerable attention because of medicolegal exposure. It appears that apical root resorption results from a combination of individual biological variability and the effect of mechanical factors [31]. Loss of apical root structure is not predictable; when it progresses reaching the dentine is considered irreversible [34]. The cause of root resorption is still unknown, but the possible etiological factors are known and considered to be complex and multifactorial [30].

Severe root resorption after orthodontic treatment compromises the outcome of successful orthodontic treatment.

Generally, root resorption can affect the longevity of the dentition. However, the majority of orthodontic root resorption does not affect the functional capacity of the dentition [35–37].

Studies of root resorption date back to more than 150 years. Bates in 1856 was the first to discuss root resorption of permanent teeth [38]. Several studies on root resorption have been published in the last 20 years [30, 31, 34, 39–41]. During this period, a better understanding of the process of root resorption has been achieved. The terms used to describe root resorption in the literature were variable like (root shortening, idiopathic root resorption, frequent complication, and common consequence).

Root resorption is defined as the destruction of the cementum or dentin by cementoclastic or osteoclastic activity; it may result in the shortening or blunting of the root [42]. Root resorption is also defined as microscopic areas of resorption lacunae visualized with histological techniques [43]. Root resorption is a general term that describes the general pathologic process which does not include any expression of the etiological factors.

Several etiological factors for root resorption are known (trauma, periodontal diseases, etc.), with almost similar outcome of root structure loss. Orthodontic root resorption is unique as compared to other types of root resorption. Brezniak and Wasserstein in 2002 suggested a new and more descriptive term of orthodontic root resorption based on the actual process and termed it orthodontically induced inflammatory root resorption (OIIRR) [44].

Orthodontically induced inflammatory root resorption (OIIRR) is a sterile inflammatory process that is extremely complex and composed of various disparate components including forces, tooth roots, bone, cells, surrounding matrix, and certain known biological messengers [44].

3.2. Prevalence and Diagnosis. Histological studies report greater than 90% occurrence of root resorption in orthodontically treated teeth with varying degree [45]; in most cases the loss of root structure is minimal and clinically insignificant. When diagnostic radiographic techniques are used lower percentages are reported of root resorption. Other studies reported that the average OIIRR is usually less than 2.5 mm when using panoramic or periapical radiographs [46, 47].

Using graded scales, OIIRR is usually classified as minor or moderate in most orthodontic patients. Severe resorption defined as exceeding 4 mm or 1/3 of the original root length, is seen in 1-5% of orthodontically treated teeth [48, 49]. The risk group where severe resorption may occur compromises one to three percent of the population [50].

Lupi et al reported the incidence of root resorption before orthodontic treatment 15% and after orthodontic treatment 73% [48].

3.3. Etiological Factors

3.3.1. Treatment Duration. Most studies have concluded that the risk and severity of external apical root resorption increase as the duration of orthodontic treatment increases [39, 44, 51–56]. Sameshima and Sinclair looked at a sample of 868 patients collected from 6 different specialist practitioners and found longer treatment times to be significantly associated with increased root resorption for maxillary central incisors [54]. The reasons for the longer duration in treatment may also have had an influence on the increased levels of root resorption seen in these patients.

However, others found no significant association between OIIRR and treatment duration [51, 57, 58].

Many variables are associated with treatment duration such as complicated treatment plans or lack of the patient compliance and these variables may also contribute to OIIRR.

3.3.2. Appliance Type. Fixed appliances have been shown to cause more root resorption than removable appliances which can be explained by the increased range of tooth movement afforded by fixed appliances [52]. The risk of root resorption associated with different bracket designs has yielded inconclusive results [59, 60].

It is generally agreed that the use of a rapid maxillary expander is associated with increased levels of root resorption [61–64]. There are no other strong studies that investigated this correlation, but a case report has shown a significant OIIRR outcome with aligner treatment [44].

Kinzinger et al. studied the correlation between bonded Herbst functional appliance and root resorption. They concluded that banded Herbst appliance might deliver unphysiologic forces to immediate anchor teeth, thereby exposing these to a higher risk of root resorption than in other teeth incorporated into the anchorage either directly via bands or indirectly via occlusal or approximal contacts [65].

3.3.3. Treatment Mechanics. When comparing straight wire and standard edgewise techniques, no statistically significant differences in the amount of tooth root loss or prevalence of root resorption were observed between groups [60]. Some studies have suggested that the Begg technique may induce more root resorption [60, 66, 67]. Other studies showed no significant difference between Begg, Tweed, or various straight wire edgewise techniques on root resorption [58, 59, 68].

L. Linge and B. O. Linge suggested that the use of intermaxillary elastics increased the amount of root resorption [52], but Sameshima and Sinclair did not find any correlation [54]. No difference has been found between the use of sectional and continuous mechanics [68].

Bioefficient therapy using contemporary orthodontic materials was found to produce less root resorption than the standard edgewise systems. The use of heat-activated and superelastic wires and a smaller rectangular stainless steel wire during incisor retraction and finishing played a role in this finding [69]. When comparing conventional edgewise systems to self-ligating systems, three studies concluded that there are no statistically significant differences in root resorption between systems [43, 70–72].

3.3.4. Force Magnitude. Human and animal studies agree that there is an increase in severity of root resorption with

increasing force magnitude [63, 73–78]. Harry and Sims used a scanning electron microscope to examine extracted human premolar teeth that had 50 g, 100 g, and 200 g of intrusive force. They concluded that higher forces increased root resorption through an increase in the stress to the root surface which increased the rate of lacunae development [74]. The more recent studies have confirmed that the higher forces increase the amount of external root resorption, thus confirming the previous studies. Chan and Darendeliler used a volumetric analysis of resorption craters on extracted human teeth to compare controls with a force of 25 g or 225 g, with buccal displacement [79] or intrusion [80]. Reitan, on the other hand, found that external root resorption was poorly correlated with force magnitude. He examined 72 premolars after application of 25 g to 240 g of intrusive, extrusive, and tipping movement over a period of 10 to 47 days [81].

A series of studies by Owman-Moll et al. agreed with the findings of Reitan. They looked at tooth movement with regard to force magnitudes of 50 g, 100 g, and 200 g. They found that there was a large interindividual variance, but no significant differences in the frequency and severity of root resorption could be detected. They concluded that root resorption was independent of force magnitude, but that individual reactions may be more important [82].

3.3.5. Force Duration. Debate exists as to whether more root resorption is associated with continuous or intermittent forces. Many believe that discontinuous forces produce less root resorption because the pause in tooth movement allows the resorbed cementum to heal [83–88].

Acar et al. examined 22 human teeth. The patients were exposed to a continuous tipping force of 100 g on one side and on the other side an intermittent force was applied through elastics for 12 hours per day over a period of 9 weeks. Their results showed that the intermittent forces resulted in less root resorption. The accuracy of these results is questionable because the intermittent forces were subject to patient compliance [87].

Weiland [88] studied 84 premolars from patients which had been moved buccally with an orthodontic appliance. On one side of the mouth, force on the premolar was applied with a stainless steel wire (0.016 inch), while force on the contralateral premolar was applied with a superelastic wire (0.016 inch). Their results support the findings of Acar et al. [87] that continuous forces cause more resorption. They showed that the teeth activated with the super elastic wire moved significantly more but had 140% more resorption than the teeth with stainless steel wire.

Contrary to these reports Owman-Moll et al. [82] found no difference in the amount or severity of root resorption between forces applied continuously or intermittently after application of a buccally directed force of 50 g to human premolars.

3.3.6. Direction of Tooth Movement. Intrusion has been consistently implicated as the most likely type of tooth movement to cause root resorption [57, 81]. Displacement of the root apex horizontally or torquing has been proven beyond doubt to produce root resorption [54, 89]. The highest incidence of root resorption is reported to occur when 3 to 4.5 mm of torquing movement was performed [54]. Reitan and Thilander and colleagues suggested that the stress distribution associated with tipping movements is more likely to cause root resorption than the stress distribution associated with bodily movement [64, 83].

3.3.7. Amount of Tooth Movement. Sameshima and Sinclair [54] found that severe root resorption occurred in their samples when the root apex was displaced lingually, with a mean difference of 1 mm more than the control group. They concluded that root resorption is directly related to the distance moved by the tooth roots. Maxillary incisors tend to be moved more than other teeth in orthodontic treatment and therefore this is a possible explanation for why maxillary incisors are at a high risk of root resorption.

3.3.8. Timing of Orthodontic Therapy. It is generally recommended that orthodontics be preceded by periodontal therapy based on the belief that orthodontics in the presence of inflammation can lead to rapid and irreversible breakdown of the periodontium [90]. Scaling, root planning (if necessary, by open flap debridement procedures for access), and gingival augmentation should be performed as appropriate before any tooth movement. The corrective phase of periodontal therapy, that is, osseous or pocket reduction/elimination surgery, ought to be delayed until the end of orthodontic therapy, because tooth movement may modify gingival and osseous morphology [91].

3.3.9. Extraction. Sameshima and Sinclair [54] examined the relationship of the extraction pattern in detail as a factor affecting the resorption process. They concluded that extraction procedures (all first premolars, all second premolars, mandibular incisors, and asymmetric extractions) have the potential to produce root resorption during space closure. They observed a statistically significant difference in the resorption process when extraction and nonextraction groups were compared; among the extraction groups, the extraction of all first premolars showed the greatest resorption potential. Other studies that examined this factor did not find it to be significant [92, 93].

There are many etiological possible factors that may increase susceptibility for OIIRR. The current evidence available is conflicting and inconclusive. Weltman et al. [41] conducted a systematic review to this topic, where the factors have been grouped into likely, unlikely, and unclear risk-relationship categories.

4. Conclusion

Periodontal health is essential for any form of dental treatment. Adult patients must undergo regular oral hygiene instruction and periodontal maintenance in order to maintain healthy gingival tissue during active orthodontic treatment. Close monitoring of adults with reduced periodontal

support is mandatory. Orthodontic treatment is usually contraindicated in patients with active periodontal disease or poor periodontal health as the chance of further periodontal deterioration is high in such case. Therefore, a thorough assessment of the periodontal health and level of attached gingival is recommended prior to the orthodontic treatment. Also, it is equally important to lay emphasis on the necessity of good oral hygiene in order to achieve the best treatment outcome. Oral hygiene instructions should be given before the start of orthodontic treatment and it should be reinforced during every visit.

Conflict of Interests

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

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Review Article

Risk Factors of Periodontal Disease: Review of the Literature

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Objectives. This paper aims to review the evidence on the potential roles of modifiable and nonmodifiable risk factors associated with periodontal disease. *Data*. Original articles that reported on the risk factors for periodontal disease were included. *Sources*. MEDLINE (1980 to Jan 2014), PubMed (using medical subject headings), and Google Scholar were searched using the following terms in different combinations: "periodontal disease," "periodontitis," "risk factors," and "causal." This was supplemented by hand-searching in peer-reviewed journals and cross-referenced with the articles accessed. *Conclusions*. It is important to understand the etiological factors and the pathogenesis of periodontal disease to recognize and appreciate the associated risk factors. As periodontal disease is multifactorial, effective disease management requires a clear understanding of all the associated risk factors.

1. Introduction

Periodontitis is one of the most ubiquitous diseases and is characterized by the destruction of connective tissue and dental bone support following an inflammatory host response secondary to infection by periodontal bacteria [1, 2]. Severe periodontitis, which may result in tooth loss, is found in 5–20% of most adult populations worldwide [3–5]. Children and adolescents can have any of the several forms of periodontitis such as aggressive periodontitis, chronic periodontitis, and periodontitis as a manifestation of systemic diseases [6–8].

It is now generally agreed that almost all forms of periodontal disease occur as a result of mixed microbial infections within which specific groups of pathogenic bacteria coexist [9–11]. Evidence is reviewed on the potential roles of modifiable and nonmodifiable risk factors associated with periodontal disease. An understanding of risk factors is essential for clinical practice.

1.1. Search Strategy. MEDLINE (1980 to Jan 2014), PubMed (using medical subject headings), and Google Scholar were searched using the following terms in different combinations: "periodontal disease," "periodontitis," "risk factors," and "causal." This was supplemented by hand-searching in peer-reviewed journals and cross-referenced with the articles accessed.

2. Risk Factors of Periodontal Disease

2.1. Modifiable Risk Factors

2.1.1. Microorganisms and Periodontal Disease. The oral bacterial microbiome includes over 700 different phylotypes, with approximately 400 species found in subgingival plaque [12, 13]. The subgingival microflora in periodontitis can harbor hundreds of bacterial species but only a small number has been associated with the progression of disease and considered etiologically important. Subgingival plaque from deepened periodontal pockets is dominated by gram-negative anaerobic rods and spirochetes [14, 15]. Strong evidence has implicated *Porphyromonas gingivalis* [16] and *Aggregatibacter actinomycetemcomitans* [17, 18] to the pathogenesis of adult periodontitis. In addition, *Bacteroides forsythus* [19], *Prevotella intermedia* [18], *Peptostreptococcus micros* [20], and *Fusobacterium nucleatum* [21] have been strongly linked with the progression of adult periodontitis.

2.1.2. Tobacco Smoking. There is accumulating evidence for a higher level of periodontal disease among smokers [22, 23]. Tobacco smoking exerts a substantial destructive effect on the periodontal tissues and increases the rate of periodontal disease progression [24]. Risk factors including tobacco smoking modify the host response to the challenge of bacteria

in microbial dental plaque [25, 26]. Smokers with periodontal disease seem to show less signs of clinical inflammation and gingival bleeding compared to nonsmokers [27]. That could be explained by the fact that nicotine exerts local vasoconstriction, reducing blood flow, edema, and clinical signs of inflammation [28]. Nicotine acetylcholine receptor has been found to play an important role in the development of nicotine related periodontitis [29].

2.1.3. Diabetes Mellitus. One of the important oral signs of diabetes is gingivitis and periodontitis. Patients with undiagnosed or poorly controlled diabetes mellitus type 1 or type 2 are at higher risk for periodontal disease. There are many studies that demonstrate an association between diabetes and an increased susceptibility to oral infections including periodontal disease [30–34]. Periodontitis also progresses more rapidly in poorly controlled diabetics [35], and early age of onset of the disease is seen as a risk factor for more severe diseases [36]. Conversely, most well-controlled diabetic patients can maintain periodontal health and will respond favorably to periodontal therapy [37].

Despite discrepancy regarding this issue in the scientific literature, it seems that the effect of glycemic control is related to the mode of periodontal therapy [38]. Many studies addressed the effect of periodontal treatment on glycemic control of diabetes patients [39–46].

2.1.4. Cardiovascular Disease. The biological plausibility of the association between periodontal diseases and cardiovascular diseases is well studied and it includes some of the following possible mechanisms: high concentrations of cholesterol and the action of oral bacteria in the process of atherosclerosis or the participation of acute-phase proteins that may increase in chronic periodontitis [47, 48]. Several biological mechanisms have been proposed to explain the relationship between periodontal diseases and cardiovascular diseases. Therefore, periodontitis can probably elicit a systemic inflammatory response and it deserves more attention [49].

Periodontal disease is capable of predisposing to vascular disease due to the rich source of subgingival microbial species and host's response. Furthermore, we must be aware that these diseases share many risk factors and there are evident similarities to the basic pathogenic mechanisms [50].

Periodontitis is associated with the increase in the level of C-reactive protein and fibrinogen, irrespective of coronary diseases. Furthermore, there is evidence that suggests that the increase in the levels of systemic markers of inflammation, such as the C-reactive protein (CRP) and interleukin-6 (IL-6), is associated with cardiovascular diseases [51].

Bacteremia from periodontitis and dental disease is known to be the primary cause of infective endocarditis [52]. In particular, patients who have undergone heart valve surgery have a significant risk of life-threatening infective endocarditis. Epidemiological and microbiological studies have lent credence to the concept that periodontal disease may be a separate risk factor for cardiovascular disease, cerebrovascular disease [53], and preterm delivery of low birth weight infants [54].

Wu et al. [55] have shown that periodontal disease is another putative and independent risk factor for cerebrovascular disease, particularly for ischemic stroke. Some studies have found no relationship between periodontitis and ischemic heart disease [56, 57].

2.1.5. Drug-Induced Disorders. Some medications significantly decrease salivary flow. These include antihypertensives, narcotic analgesics, some tranquilizers and sedatives, antihistamines, and antimetabolites. Other drugs, particularly those in liquid or chewable form that contain added sugar, alter the pH and composition of plaque, making it more able to adhere to tooth surfaces [58].

Drugs can be a contributing factor in periodontal diseases. Drugs such as anticonvulsants, calcium channel blocking agents, and cyclosporine may induce gingival overgrowth [59].

2.1.6. Stress. Patients with inadequate stress behavior strategies (defensive coping) are at greater risk for severe periodontal disease [60–65]. Stress is associated with poor oral hygiene, increased glucocorticoid secretion that can depress immune function, increased insulin resistance, and potentially increased risk of periodontitis [66]. Men who reported being angry on a daily basis had a 43% higher risk of developing periodontitis compared with men who reported being angry seldom [66]. Studies have found some periodontal disease indicators such as tooth loss and gingival bleeding to be associated with work stress [66, 67] and financial strains [68].

2.1.7. Obesity. Obesity has been reported to be an important risk factor for periodontal disease [69, 70]. Several explanations for the association between obesity and periodontal disease [71–73] in younger adults have been provided. Younger people may have different dietary patterns than older study participants. Research in dietary trends in adolescent's ages from 11 to 18 reveals a significant decrease in raw fruit and nonpotato vegetables, which are sources of vitamin C. In addition, adolescents have decreased their calcium intake and increased their intake of soft drinks and noncitrus juices. This is important to oral health because low dietary intake of calcium and vitamin C has been associated with periodontal disease [74]. People who consume less than the recommended dietary allowance (RDA) for calcium and vitamin C have slightly higher rates of periodontal disease [74].

2.2. Nonmodifiable Risk Factors

2.2.1. Osteoporosis. Many of the studies conducted to date suggest there is a relationship between skeletal osteoporosis and bone loss [75–80] to the extent that postmenopausal osteoporosis may result in dental osteopenia involving the jaws, and particularly the mandible [81]. Osteoporosis was significantly associated with severe alveolar crestal bone loss and the prevalence of periodontitis cases in postmenopausal

women [82]. A review of the relationship between osteopenia, oral bone loss, and periodontal disease [83, 84] concluded that osteopenia does play a role in the expression of periodontal disease. The review indicated a direct association between skeletal and mandibular osteopenia and loss of alveolar crestal height and tooth loss in postmenopausal women. Taguchi et al. [85] have stressed that it is important to distinguish among osteopenia, which has been defined in general terms as a decrease in normal mineralized bone, postmenopausal osteoporosis, which is a disease caused by the cessation of estrogen production and characterized by spinal fractures that occur between the ages of 50 and 70 years, and osteoporosis, which affects an older population and results in proximal femur fractures [86]. Periodontitis and osteopenia may have common etiological agents that may either directly influence or modulate both disease processes [87].

2.2.2. Hematological Disorders. Hemorrhagic gingival overgrowth with or without necrosis is a common early manifestation of acute leukemia [88]. Patients with chronic leukemia may experience similar but less severe periodontal changes. Chemotherapy or therapy associated with bone marrow transplantation may also adversely affect the gingival health [89, 90].

2.2.3. Host Response. Chronic periodontitis involves complex interactions between microbial factors and susceptible hosts [91, 92]. The bacterial components such as lipopolysaccharides and cytokines activate the macrophages to produce cytokines such as interleukin (IL)-1 and tumor necrosis factor (TNF) [39, 93]. These cytokines activate the fibroblasts that reside in the periodontal tissues to the matrix metalloproteinases (MMPs), a plasminogen activator, which can activate plasmin. Plasmin, in turn, can activate some other types of latent MMPs, while tissue inhibitors of metalloproteinases (TIMPs) can inactivate the active MMPs [94]. Among susceptible individuals, the prolonged and excessive bacterial promotions of the MMPs induce the enhanced degradation of collagen, which is a primary component of the periodontal matrix. MMP-8 and -9 are released from the polymorphonuclear leukocytes (PMN) and are responsible for a substantial part of the destruction caused by the host response. MMP-13 also facilitates bone resorption by degrading the collagenous matrix of the bone after the bone is demineralized by osteoclasts [95]. Marcaccini et al. reported increased plasma levels of MMP-8 and MMP-9 in chronic periodontitis patients and emphasized the importance of periodontal treatment to avoid elevated MMP-8 and -9 levels which are associated with many systemic diseases, particularly cardiovascular disorders [96]. A recent review on the modifiable risk factors concluded that smoking and excess caloric intake contribute to increases in systemic markers of inflammation and can modify gene regulation through a variety of biologic mechanisms [97].

2.2.4. Female Hormonal Alterations. Hormonal fluctuations in the female patient may alter the status of periodontal health [98]. Such changes may occur during puberty, the menstrual cycle, pregnancy, or menopause. Changes may also be associated with the use of oral contraceptives. The most pronounced periodontal changes occur during pregnancy, as a significant proportion of pregnant women suffer from pregnancy gingivitis. Women on hormonal replacement therapy (HRT) and oral contraceptives experience increased gingival inflammation [81, 99]. With oral contraceptives, this increase in gingival inflammation is mainly related to the duration of use as it has been suggested that prolonged use of oral contraceptives may detrimentally affect the periodontium.

2.2.5. Pregnancy. Offenbacher et al. [92] found significantly more periodontal attachment loss among mothers of PLBW infants compared with mothers of normal-term infants.

Similarly, several other studies have suggested an adverse influence of periodontal disease on the course of pregnancy [100–103]. It has been suggested that periodontal disease may increase the risk of having preterm low birth weight (PLBW) infants [104, 105]. This outcome is thought to be the effect of biologic mediators of inflammatory processes such as prostaglandins E2 and TNF. The common bacterial product lipopolysaccharide also may have a triggering role in adverse change of the course of pregnancy.

3. Risk Characteristics

3.1. Age. Several studies show that the prevalence and severity of periodontal disease increase with age [87, 106–112]. Papapanou et al. demonstrated that the mean annual rate of bone loss among the initially 70-year-old subjects was 0.28 mm compared to 0.07 on the 25-year-old individuals [112]. The increased severity of periodontal disease and bone loss with age is probably related to the length of time, where the periodontal tissues have been exposed to bacterial plaque, and is considered to reflect individual's cumulative oral history [113]. More studies carried out in some of the developed countries show changing patterns of periodontal disease progression. These studies have shown that advanced periodontal destruction and bone loss are seldom seen in individuals under the age of 40 [109, 114]. A similar finding has been observed even in the elderly population. Studies among the elderly have shown that advanced periodontal disease affects only a small fraction of this age group [108, 114]. However, among those with advanced disease, further breakdown does occur with increasing age.

3.2. Sex. Numerous studies reported higher periodontal destruction among males compared to the female population [87, 115–118]. The reasons for these sex differences are not clear, but they are thought to be related to the ignorance of oral hygiene, which is usually observed among males [118, 119]. However, the relationship observed between sex and the disease is not apparent and is not considered as strong and consistent. Thus, sex may be a demographic factor, which may interfere with the effects of other factors and it must be controlled for investigating the disease.

3.3. Socioeconomic Status (SES). The possible relationship between periodontal disease and socioeconomic status was

found in several studies [108, 110, 120–122]. Gingival condition is clearly related to lower SES, but the relationship between SES and periodontitis is less direct. It can be certain that gingival health is better among individuals with higher education and with more secure income. SES is a modifiable factor and it can be examined in multivariate models for the disease.

3.4. Education and Race. Periodontal disease has a reciprocal relationship with educational level. The higher the educational level, the lower the periodontal diseases (Department of Health Education and Welfare, 1966). Several studies involving different racial populations have found some difference in the expression of periodontal disease [108]. Once again, race is not a modifiable factor, and some discrepancies in disease expressions may be explained by the differences in other risk factors between populations.

3.5. Genetic Considerations. Studies show genetic risk factors associated with periodontitis [123–129]. McDevitt et al. demonstrate that the composite IL-1 genotype is significantly associated with the severity of adult periodontitis. They also confirmed that both IL-1 genotyping and smoking history provide objective risk factors for periodontal disease in a private practice environment [130]. Currently, there are two major forms of periodontitis-chronic and aggressive periodontitis [6]. Risk for periodontitis is not shared equally by the population. It is clear that periodontitis severely affects a high-risk group representing around 10–15% of the population, in whom the disease quickly progresses from chronic gingivitis to destructive periodontitis [131]. This differential risk for periodontitis is consistent with heritable elements of susceptibility, but direct evidence for a differential genetic contribution to periodontitis comes from several sources.

Many works of the literature report familial aggregation of periodontal diseases, but due to different terminology, classification systems, and lack of standardized methods of clinical examination, it is difficult to compare reports directly. Although periodontal disease nosology has changed many times over the timeframe of these reports, most familial reports for periodontitis are for early-onset forms now called aggressive periodontitis [132–139]. Reports of the familial nature of chronic forms of periodontitis are less frequent, although German studies of the familial nature of chronic forms of periodontitis from the early 20th century have been reviewed by [140]. This aggregation within families strongly suggests a genetic predisposition. It must be borne in mind that familial patterns may reflect exposure to common environmental factors within these families. Thus, it is important to consider the shared environmental and behavioral risk factors in any family. These would include education, socioeconomic grouping, oral hygiene, possible transmission of bacteria, incidence of chronic diseases such as diabetes, and environmental features, such as passive smoking and sanitation. Some of these factors, such as lifestyle, behavior, and education, may be under genetic control and may influence the standards of oral hygiene. The complex interactions between genes and the environment must also be considered in the evaluation of familial risk for the periodontal diseases.

In chronic periodontitis, the phenotype or disease characteristics do not present significantly until the third decade of life, whereas, in the aggressive forms of periodontal disease, the presentation can occur in the first, second, third, and fourth decades. This variability in presentation of significant signs of disease makes diagnosis difficult, not only in declaring if a patient suffers from the disease but also in detecting patients who do not suffer from the disease and differentiating between adult and aggressive forms of periodontitis. The problems associated with the clinical differentiation of periodontal disease are not uncommon in medical genetics, since similar problems arise in the study of other delayed-onset hereditary traits [133].

3.6. C-Reactive Protein. Cholesterol has long been known to play a crucial role in predicting risk for heart attack in seemingly healthy people. But half of all heart attacks occur among people who do not have high cholesterol. Also, the classical risk factors of CVD cannot account for all the variation in the incidence of CVD cases [141]. As a result there is a growing interest to identify additional markers of coronary risk. One likely candidate is the C-reactive protein (CRP), although this protein is part of the body's normal response to infection and inflammation. Some of the recent studies have reported elevated CRP levels among those with periodontitis [142–145]. In a study by Ebersole et al., they reported significantly higher levels of CRP among those with adult periodontitis, especially among those having more active sites [146]. The participants of the MI Life Study [143] also reported positive association between elevated levels of CRP (>3 mg/L) and severity of periodontitis. Periodontitis is an inflammatory reaction of the supporting tissues of the teeth like the periodontal ligament, cementum, and alveolar bone to gram-negative anaerobic bacteria. As a response to bacterial endotoxins, the local host inflammatory mediators are activated [94, 147] that in turn initiate localized inflammatory response [148, 149] and finally result in serum antibody response to the bacteria [150, 151]. Bacterial infections may often provide a strong stimulus for a systemic acute phase response that may result in increased production of acute-phase proteins like CRP, macroglobulin, and serum amyloid [152]. In a recent study, it was found that the concentrations of hs-CRP and IL-6 were significantly higher in the sera of patients with periodontitis and periodontal treatment decreased the levels of serum hs-CRP and IL-6 [153]. Elevation of CRP levels among those with periodontitis indicates that periodontitis may also have systemic cytokine mediated effects that may in turn participate in atherogenesis. This may in turn help to explain conditions where dental infections may stimulate systemic inflammatory response, thereby placing "apparently healthy" people at increased risk of cardiovascular disease.

Many works in the literature report familial aggregation of periodontal diseases, but due to different terminology, classification systems, and lack of standardized methods of clinical examination, it is difficult to compare reports directly. Reports of the familial nature of chronic forms of periodontitis are less frequent, although German studies of the familial nature of chronic forms of periodontitis from the early 20th

century have been reviewed by [140]. This aggregation within families strongly suggests a genetic predisposition. It must be borne in mind that familial patterns may reflect exposure to common environmental factors within these families.

4. Conclusion

It is important to understand the etiological factors and the pathogenesis of periodontal disease to recognize and appreciate the associated risk factors. As periodontal disease is multifactorial, effective disease management requires a clear understanding of all the associated risk factors.

Conflict of Interests

The author declares that there is no conflict of interests regarding the publication of this paper.

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Review Article

A New Classification of Endodontic-Periodontal Lesions

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The interrelationship between periodontal and endodontic disease has always aroused confusion, queries, and controversy. Differentiating between a periodontal and an endodontic problem can be difficult. A symptomatic tooth may have pain of periodontal and/or pulpal origin. The nature of that pain is often the first clue in determining the etiology of such a problem. Radiographic and clinical evaluation can help clarify the nature of the problem. In some cases, the influence of pulpal pathology may cause the periodontal involvement and vice versa. The simultaneous existence of pulpal problems and inflammatory periodontal disease can complicate diagnosis and treatment planning. An endo-perio lesion can have a varied pathogenesis which ranges from simple to relatively complex one. The differential diagnosis of endodontic and periodontal diseases can sometimes be difficult, but it is of vital importance to make a correct diagnosis for providing the appropriate treatment. This paper aims to discuss a modified clinical classification to be considered for accurately diagnosing and treating endo-perio lesion.

1. Introduction

The periodontal-endodontic lesions have been characterized by the involvement of the pulp and periodontal disease in the same tooth. This makes it difficult to diagnose because a single lesion may present signs of both endodontic and periodontal involvement. There is a general agreement today that the vast majority of pulpal and periodontal lesions are the result of bacterial infection. This suggests that one disease may be the result or cause of the other or even originated from two different and independent processes which are associated with their advancement [1]. Diagnosis is complicated by the fact that these diseases are too frequently viewed as independent entities. However, it is critical to recognize the interrelationship for successful management of these lesions. The pathways for the spread of bacteria between pulpal and periodontal tissues are still a subject of controversy [2–6].

The apical foramen is the main access route between the pulp and the periodontium, with the participation of all root canal system: accessory, lateral, and secondary canals, as well as the dentinal tubules through which the bacteria and its products contaminate the medium [7, 8]. It is known that the main cause of the periodontal lesions is the presence of the bacterial plaque, formed by aerobic and anaerobic microorganisms [9–12]. Pulp exposures, periodontitis, and caries lesions are of significant importance in the development of periodontal-endodontic lesions. If the lesions are not well treated and the canals are not disinfected and sealed completely, they will house bacterial necrotic rests, which account for the progression of the lesion or even for the endodontic reinfection [13–15]. Another form of the interrelationship is because of the iatrogenic perforations due to either rotary instruments or improper handling of the endodontic instruments [16].

Vertical root fractures and cracks may serve as a "bridge" for pulp contamination. If the periodontium had a previous inflammation, it may lead to dissemination of the inflammation which can result in pulp necrosis [17].

Several authors, through their studies, diverge on the contamination routes. Rubach and Mitchell [18] suggested that the periodontal disease may affect the pulp health when the accessory canal exposure occurs, allowing the periodontopathogenic bacteria to cause inflammatory reactions followed by pulp necrosis.

Lindhe [19] also reported that bacterial infiltrates of the inflammatory process may reach the pulp when there

is accessory canal exposure, through apical foramens and canaliculi of the furcation area. Adriaens et al. [8] demonstrated that bacteria coming from the periodontal pockets have the capacity of reaching the root canals towards the pulp, suggesting that the dentinal tubules may serve as a reservoir for these microorganisms and that a recolonization of the treated root surface may occur.

It is highlighted that the root planning and scaling may result in the rupture of the vessels and destruction of the neurovascular bundle in the lateral canals, provoking a reduction of the blood supply and consequently leading to pulp alterations.

Knowledge of these disease processes is essential in coming to the correct diagnosis. This is achieved by careful history taking, examination, and performing special tests.

This paper is an attempt to provide a rational classification to the endo-perio question in order to scientifically diagnose and treat these lesions with predictable success.

The periodontal-endodontic lesions have received several classifications, among which is the classification of Simon et al. [20] separating lesions involving both periodontal and pulpal tissues into the following groups:

(i) primary endodontic lesions,

(ii) primary endodontic lesions with secondary periodontal involvement,

(iii) primary periodontal lesions,

(iv) primary periodontal lesions with secondary endodontic involvement,

(v) true combined lesions.

From the point of view of treating these cases efficaciously, another clinical classification was provided by Torabinejad and Trope in 1996 [21], based on the origin of the periodontal pocket:

(i) endodontic origin,

(ii) periodontal origin,

(iii) combined endo-perio lesion,

(iv) separate endodontic and periodontal lesions,

(v) lesions with communication,

(vi) lesions with no communication.

Another classification was recommended by the world workshop for classification of periodontal diseases (1999) [22], Periodontitis Associated with Endodontic Disease:

(i) endodontic-periodontal lesion,

(ii) periodontal-endodontic lesion,

(iii) combined lesion.

Based on these classifications, the most widely used classification of endodontic-periodontal lesions is the one that has been classified by Simon et al. [20], according to the primary cause of disease. One of the main classification items was primary endodontic disease, which we believe should be modified, since it has no periodontal relationship. A new endodontic-periodontal interrelationship classification, based on the primary disease with its secondary effect, is suggested as follows:

(1) retrograde periodontal disease:

  (a) primary endodontic lesion with drainage through the periodontal ligament,

  (b) primary endodontic lesion with secondary periodontal involvement;

(2) primary periodontal lesion;

(3) primary periodontal lesion with secondary endodontic involvement;

(4) combined endodontic-periodontal lesion;

(5) iatrogenic periodontal lesions.

(1) Retrograde Periodontal Disease. It could be of two subcategories.

(a) Primary Endodontic Lesion with Drainage through the Periodontal Ligament. A deep narrow probing defect is noted on just one aspect of the tooth root. Acute exacerbation of a chronic apical lesion on a tooth with a necrotic pulp may drain coronally through the periodontal ligament into the gingival sulcus. This condition may mimic, clinically, the presence of a periodontal abscess. In reality, it is a sinus tract from pulpal origin that opens through the periodontal ligament area. For diagnostic purposes, it is imperative for the clinician to insert a gutta-percha cone into the sinus tract and to take one or more radiographs to determine the origin of the lesion. When the pocket is probed, it is narrow and lacks width. Primary endodontic diseases usually heal following root canal treatment.

(b) Primary Endodontic Lesion with Secondary Periodontal Involvement. There is a more extensive periodontal pocket which has occurred as a result of the drainage from noxious agents present in an infected root canal system. Long-term existence of the defect has resulted in deposits of plaque and calculus in the pocket with subsequent advancement of the periodontal disease.

The integrity of the periodontium will be reestablished if root canal treatment is done properly. If a draining sinus tract through the periodontal ligament is present before root canal treatment, resolution of the probing defect is expected.

(2) Primary Periodontal Lesion. The periodontal disease has gradually spread along the root surface towards the apex. The pulp may remain vital but may show some degenerative changes over time. In such cases, it is advisable to treat the periodontal tissues only.

(3) Primary Periodontal Lesion with Secondary Endodontic Involvement. Progression of the periodontal disease and the pocket leads to pulpal involvement via either a lateral canal foramen or the main apical foramen. The pulp subsequently

becomes necrotic and infected. In such cases, it is advisable to treat both tissues [23].

(4) Combined Endodontic-Periodontal Lesion. The tooth has a pulpless, infected root canal system and a coexisting periodontal defect. A simpler classification would be to define any situation with both endodontic and periodontal diseases as being a "combined endodontic-periodontal lesion." An attempt should be made to identify the primary cause of a combined lesion but this may not always be possible. In such cases, it is not essential to determine which disease entity occurred first as the treatment will involve both endodontic and periodontal management. If only one of the problems was treated, then it would be expected that the lesion would not heal adequately. It is generally advisable to treat both tissues concurrently in order to create the most favorable environment for healing.

(5) Iatrogenic Periodontal Lesions. Lesions produced as a result of treatment modalities include the following.

(A) Root Perforations. Iatrogenic root canal perforations: they are serious complications during dental treatment and have a rather poor prognosis [24]. Perforations may be produced by powered rotary instruments during the attempt to gain access to the pulp or during preparation for a post. Improper manipulation of endodontic instruments can also lead to a perforation of the root. When root perforation occurs, communications between the root canal system and either periradicular tissues or the oral cavity may often reduce the prognosis of treatment. At the site of perforation, an inflammatory reaction in periodontal ligament occurs and leads to the formation of a lesion which can progress as a conventional primary endodontic lesion.

(B) Coronal Leakage. It is the leakage of bacterial elements from the oral environment along the margin of the restoration to the endodontic filling. Studies have indicated that this factor may be an important cause of endodontic treatment failure [25–27]. Root canals may become recontaminated by microorganisms due to delay in placement of a coronal restoration and fracture of the coronal restoration and/or the tooth. Madison and Wilcox [13] found that exposure of root canals to the oral environment leads to coronal leakage, and in some cases along the entire length of the root canal. Ray and Trope [14] reported that defective restorations and adequate root canal fillings had a higher incidence of failures than teeth with inadequate root canal fillings and adequate restorations.

(C) Dental Injuries or Trauma. They may take on many shapes but generally can be classified as enamel fractures, crown fractures without pulp involvement, crown fractures with pulp involvement, crown-root fracture, root fracture, luxation, and avulsion [28]. Treatment of traumatic dental injuries varies depending on the type of injury and it will determine pulpal and periodontal ligament healing prognosis [17, 29–33]. The most common cause of vertical root fracture in endodontically treated teeth is the excessive force used during lateral condensation of gutta-percha. Mild pain or discomfort and swelling are the major clinical symptoms, and solitary pocket around one aspect of the suspected tooth is the major clinical sign.

(D) Chemicals Used in Dentistry. They have the potential to cause root resorption. Clinical reports [34–36] have shown that intracoronal bleaching with highly concentrated oxidizing agents, such as 30–35% hydrogen peroxide, can induce root resorption. The irritating chemical may diffuse through the dentinal tubules, and when combined with heat, they are likely to cause necrosis of the cementum, inflammation of the periodontal ligament, and subsequently root resorption [36, 37]. Replacement resorption or ankylosis occurs following extensive necrosis of the periodontal ligament with formation of bone onto a denuded area of the root surface. This condition is most often seen as a complication of luxation injuries, especially in avulsed teeth that have been out of their sockets in dry conditions for several hours. The potential for replacement resorption was also associated with periodontal wound healing. Granulation tissue derived from bone or gingival connective tissue may induce root resorption and ankylosis [17, 31].

(E) Vertical Root Fractures. The artificial pathways between periodontal and pulpal tissues are vertical root fractures. Vertical root fractures are caused by trauma and have been reported to occur in both vital and nonvital teeth. In vital teeth, vertical fractures can be continuations of coronal fractures in the "cracked tooth syndrome" or can occur solely on root surfaces [30, 31].

2. Discussion

It is known that both the pulp and the periodontium are closely linked to each other, through the apical foramen, accessory canals, and dentinal tubules of the root, and one can interfere on the integrity of the other. Although there is existence of these communication routes, the mechanism of direct transmission of the periodontal infection to the pulp is still controversial. Some authors such as Rubach and Mitchell [18] affirmed that the periodontal disease may affect the pulp when there is exposure of the accessory canals through the apical foramina and the canaliculi in the furcation. Adriaens et al. [8] reported that the bacteria coming from the periodontal pockets may contaminate the pulp through the dentinal tubules that would be exposed during root planning and scaling, serving as a microorganism reservoir resulting in the recolonization of the treated root surface. Some studies [2, 38] have contradicted this idea, because even with the removal of the cementum during the periodontal therapy in vital teeth, the pulp tissue will be protected against the harmful agents through forming reparative dentin. Moreover, the dentinal fluids move towards the exterior, thereby reducing the diffusion of the harmful products of the bacteria on the exposed dentin. On the other hand, Langeland et al. [6] affirmed that only pulp would be affected by the periodontal disease if the apical foramen is involved.

The differential diagnosis of endodontic and periodontal diseases can sometimes be difficult but it is of vital importance to make a correct diagnosis so that the appropriate treatment can be provided. Endodontic-periodontal lesions present challenges to the clinician as far as diagnosis and prognosis of the involved teeth are concerned. Etiologic factors such as bacteria, fungi, and viruses as well as other various contributing factors such as trauma, root resorptions, perforations, and dental malformations also play an important role in the development and progression of such lesions.

The endo-perio lesion is a condition characterized by the association of periodontal and pulpal disease in the same dental element. This highlights the importance of taking the complete clinical history and making the right diagnosis to ensure correct prognosis and treatment. Taking into consideration all these factors and the divergences regarding the origin and direction that these infections developed, the new modified classification of these lesions has been justified.

3. Conclusions

Based on the current classification, it can be concluded that it is of extreme importance that the dentist should know how to differentiate between the origins of the periodontal-endodontic lesions, including all the routes of communication between the pulp and the periodontium which act as possible "bridges" for the microorganisms, thereby enabling the dissemination of the infection from one site to another.

Through this knowledge, the dentist will achieve the correct diagnosis and adequate treatment, resulting in greater chances of obtaining success in the treatment of the periodontal-endodontic lesions.

Due to the complexity of these infections, an interdisciplinary approach with a good collaboration between endodontists, Periodontist, and microbiologists is recommended.

Conflict of Interests

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

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Review Article

Diagnostic Applications of Cone-Beam CT for Periodontal Diseases

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Objectives. This paper aims to review the diagnostic application of cone beam computed tomography (CBCT) in the field of periodontology. *Data*. Original articles that reported on the use of CBCT for periodontal disease diagnosis were included. *Sources*. MEDLINE (1990 to January 2014), PubMed (using medical subject headings), and Google Scholar were searched using the following terms in different combinations: "CBCT," "volumetric CT," "periodontal disease ," and "periodontitis." This was supplemented by hand-searching in peer-reviewed journals and cross-referenced with the articles accessed. *Conclusions*. Bony defects, caters, and furcation involvements seem to be better depicted on CBCT, whereas bone quality and periodontal ligament space scored better on conventional intraoral radiography. CBCT does not offer a significant advantage over conventional radiography for assessing the periodontal bone levels.

1. Introduction

Periodontal disease is a chronic bacterial infection that affects the gingiva and bone supporting the teeth [1]. Treatment of patients with advanced periodontal diseases requires not only extensive clinical recording but also radiological examination [2]. Radiography provides vital information on the amount and type of damage to the alveolar bone [3]. The current diagnostic approaches including clinical probing and intraoral radiography have shown several limitations in their reliability [4–6].

Intraoral radiography is the most commonly used imaging technique for the diagnosis of periodontal bone defects. However, intraoral radiography provides only a 2-dimensional (2D) view of 3-dimensional (3D) structures which can lead to underestimation of bone loss and errors in identifying reliable anatomical reference points [4, 5, 7]. Three-dimensional (3D) diagnostic imaging of the jaws has been of interest from the introduction of computerized tomography (CT) as a clinical tool. However, due to the factors like high cost and high radiation dosage, use of this technology in dentistry has been limited.

Cone-beam computed tomography (CBCT) is a relatively new imaging modality and with the introduction of dedicated dentomaxillofacial CBCT scanners in the late 1990s [8, 9], there has been an explosion of interest in these devices in the field dentistry. It has the obvious advantage of relatively low-cost and low-dose [10].

CBCT differs from CT in that it uses a single X-ray source that produces a cone beam of radiation (rather than a fan beam, as with CT) [11]. CBCT uses a single, relatively inexpensive, flat-panel or image intensifier radiation detector. CBCT imaging is performed using a rotating platform to which the X-ray source and detector are fixed. As the X-ray source and detector rotate around the object, it produces multiple, sequential, and planar images that are mathematically reconstructed into a volumetric dataset. A single rotational sequence would capture enough data for volumetric image construction. The entire scanning of the target region is performed in a single rotation thereby significantly reducing the radiation exposure. Further, the exposure is reduced by 50% (0.0037 mGy) if a 180° scan is performed instead of 360° [12]. In comparison, the radiation exposure in a digital panoramic radiograph is around 0.0063 mGy and around 0.0012 mGy in a periapical radiograph [13]. It has been reported that for an intraoral status of the entire dentition an effective dose ranging from 33 to 84 Sv is required [14].

CBCT as a diagnostic tool is widely used in dentoalveolar surgeries [15–17], implantology [18, 19], general/specialized dentistry (orthodontics, endodontics, periodontics, and forensic dentistry) [20–23], and otolaryngology [24]. The currently available CBCT devices are capable of providing panoramic and cephalometric images. Additionally, the low footprint of these devices makes it suitable for dental office placement, therefore producing high quality images of specific regions of interest.

Few studies have appraised the role of CBCT in the diagnosis of periodontal diseases. This review aims to assess the diagnostic application of CBCT in the field of periodontology.

Search Strategy. MEDLINE (1990 to January 2014), PubMed (using medical subject headings), and Google Scholar were searched using the following terms in different combinations: "CBCT," "volumetric CT," "periodontal disease," and "periodontitis." This was supplemented by hand-searching in peer-reviewed journals and cross-referenced with the articles accessed.

2. CBCT in Periodontology

The success of periodontal therapy depends on many factors. One of the most important factors is an accurate image of the morphology of periodontal bone destruction to plan the treatment plan [25]. Radiographs are necessary to determine the extent and severity of the periodontal lesions [5, 6]. To view the periodontal structures, intra- and extraoral imaging modalities are available. The more commonly used method is the intraoral radiographs which provide a two-dimensional view. The extraoral panoramic radiographs are also used, especially to view larger areas. The major disadvantage of this method is the distortion of the images and the blurring of anatomical structures. Also, three-dimensional information is represented in a two-dimensional plane, thus losing essential diagnostic details [7]. When compared to periodontal probing and 2D intraoral radiography, 3D CBCT scanning was found to be more effective in assessing periodontal structures [26]. CBCT had better potential of detecting periodontal bone defects in all directions compared with periapical radiographs and were as reliable as radiographs for interproximal areas [26]. Misch et al. [26] reported that CBCT is as accurate as direct measurements using a periodontal probe and as reliable as intraoral radiographs for interproximal areas. Also, since buccal and lingual defects could not be diagnosed with intraoral radiography, CBCT could be considered a superior technique. Considering the various benefits, CBCT is currently being considered as a superior diagnostic tool for applications in periodontology [27].

2.1. CBCT in Diagnosing Furcations, Caters, and Bony Defects. Detection of bone defects or furcation involvement poses significant challenges for the practitioner [28]. Earlier studies have shown that computed tomography (CT) assessment of periodontal bone height and intrabony defects is reasonably accurate and precise [29–32]. However, the higher radiation exposure could not always be adequately justified.

Noujeim et al. [33] created periodontal lesions of different depths in dried human hemimandibles and analyzed them using intraoral radiography and CBCT. They found that CBCT was more accurate in detecting the defects than the conventional radiograph [34]. Similarly, other studies have reported higher precision in diagnosing periodontal defects, particularly, in the orovestibular orientation using CBCT compared with conventional radiograph [25, 34].

Stavropoulos and Wenzel [35] evaluated the accuracy of CBCT scanning with intraoral periapical radiography for the detection of periapical bone defects. CBCT was found to have better sensitivity compared to intraoral radiography. Various in vitro studies have stated that CBCT is effective in identifying and measuring artificially created defects on samples [26, 31].

Leung et al. [36] evaluated the accuracy and reliability of CBCT in the diagnosis of naturally occurring bone defects by comparing the difference between the CBCT measurements and measurements made directly on the skulls. They reported that CBCT measurements were not as accurate as direct measurements on skulls. A certain discrepancy between direct measurements and estimated measurements on radiographs has to be considered as clinically acceptable [34]. Further, with the development of advanced equipment and software the diagnostic ability of CBCT has improved. A recent study reported on an improved quantification of periodontal bone defects based on CBCT datasets using a new software [20]. These studies provide promising data promoting the use of CBCT for the detection of periodontal bony defects.

Vandenberghe et al. [34] studied thirty periodontal bone defects of 2 adult human skulls using intraoral digital radiography and CBCT. Periodontal bone levels and defects on both imaging modalities were assessed and compared to the gold standard. The study concluded that the intraoral radiography was significantly better for contrast, bone quality, and delineation of lamina dura, but CBCT was superior for assessing crater defects and furcation involvements.

2.2. CBCT in Measuring Periodontal Bone Levels. Sufficient alveolar bone volume and favorable architecture of the alveolar ridge are essential to obtain ideal functional and esthetic prosthetic reconstruction [37]. Studies of the extent of vertical alveolar bone defects from radiographs and from exploratory surgery have also indicated a good agreement between the radiographic and the clinical findings [38, 39]. Persson et al. [39] reported that conventional radiographic images provided a better resolution of the bone levels than what can be achieved from computer screen images.

Infrabony defects are the main cause of tooth loosening and loss and are often overlooked in researchers pertaining to the validation of radiographic modalities for periodontal diagnosis [2, 40]. CBCT provides high resolution images that can be used to gather diagnostic and quantitative information on periodontal bone health. The 3D images are ideal for evaluating the infrabony defects and assessing the treatment outcomes. Mol and Balasundaram [27] compared the image quality between CBCT and conventional radiography in the assessment of alveolar bone levels. They found that CBCT provided slightly better diagnostic and quantitative

information on periodontal bone levels in three dimensions than conventional radiography. They found that the accuracy in the anterior aspect of the jaws is limited in both imaging techniques, obtained with traditional means.

Alternatively, numerous studies have reported that the CBCT images provided comparable measurements of periodontal bone levels and defects as intraoral radiography [26, 41]. Vandenberghe et al. [34] reported that CBCT images demonstrated more potential in the morphological description of periodontal bone defects, while the digital radiography provided more bone details.

2.3. CBCT in the Visualization of Periodontal Ligament Space. A break in the continuity of lamina dura and a wedge-shaped radiolucent area at the mesial or distal aspect of the periodontal ligament space are one of the earliest signs of periodontal disease [42]. However, it is clear that this does not occur until sometime after the loss of soft tissue attachment [43]. Therefore, only a sensitive imaging technique would be able to detect the earliest changes in the periodontal ligament space. The conventional intraoral radiographs have some significant disadvantages including the overlap of anatomical structures due to the positioning of the X-ray tube. Also, there could be errors related to the chemical processing and patient positioning [44]. Considering the potential advantages of using CBCT for assessing the periodontal structures, only very few studies have used it for visualization of the periodontal ligament space [45, 46]. In terms of image quality, the CBCT scans were found to be superior to the CT scans with particular reference to the periodontal ligament space [32]. Various studies have stated that conventional radiographs are better than CBCT in assessing the periodontal ligament space [34, 47]. Özmeric et al. [47] created a phantom model with artificial periodontal ligament space to compare between CBCT and conventional radiographs. They found that the CBCT was inferior to conventional radiographs in terms of the clarity of the artificial periodontal ligament space. However, conflicting views were reported in an in vitro study that found CBCT to be better than conventional radiography in visualizing the periodontal ligament space [48]. A phantom mimicking variable periodontal ligament spaces was radiographed using CBCT and intraoral radiographs [48]. This study found that CBCT provided better visualization of simulated periodontal ligament space in this phantom.

2.4. Other Periodontal Applications of CBCT. Even within the field of periodontology, CBCT has found numerous applications. CBCT has been widely used in the detection of periapical pathology. Various studies have reported on the effectiveness of using CBCT for the diagnosis of periapical pathology [49–51]. The literature shows that CBCT images are superior for the detection of apical periodontitis than conventional radiographs [49–51]. Apical periodontitis is one of the most common endodontic diseases, and it is considered to be the primary indication for root canal treatment and a sequela of inadequate or failing treatment [52]. CBCT has found application even in epidemiological surveys. A recent study [53] used CBCT images from a database to determine the prevalence of apical periodontitis. Dutta et al. [23] investigated the prevalence of periradicular periodontitis using CBCT scans in a retrospective cross-sectional epidemiological study in a Scottish subpopulation. CBCT is a radiological technique that has been more successful in detecting periradicular changes than conventional radiography [54]. In a recent case-report, CBCT was used in the diagnosis of a palatogingival groove [55]. Another recent study evaluated bone resorption at the extraction sites of a group of patients under orthodontic treatment using CBCT to evaluate the periodontal and bone support loss after tooth extraction [56]. CBCT was preferred due to its higher precision in detecting bone changes.

3. Conclusion

As the radiation dosage of CBCT is substantially higher than that of other routine dental imaging techniques, appropriate patient selection criteria must be adopted [57]. Also, the influence of the technical conditions on the image quality is relatively higher for CBCT. CBCT has the potential to gather accurate diagnostic and quantitative information about periodontal bone condition. Bony defects, caters, and furcation involvements seem to be better depicted on CBCT, whereas bone quality and periodontal ligament space scored better on conventional intraoral radiography. CBCT does not offer a significant advantage over conventional radiography for assessing the periodontal bone levels. Decision pertaining to the use of CBCT in the field of periodontology should be taken after careful consideration of its advantages, limitations, and risks.

Conflict of Interests

The author declares that there is no conflict of interests regarding the publication of this paper.

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