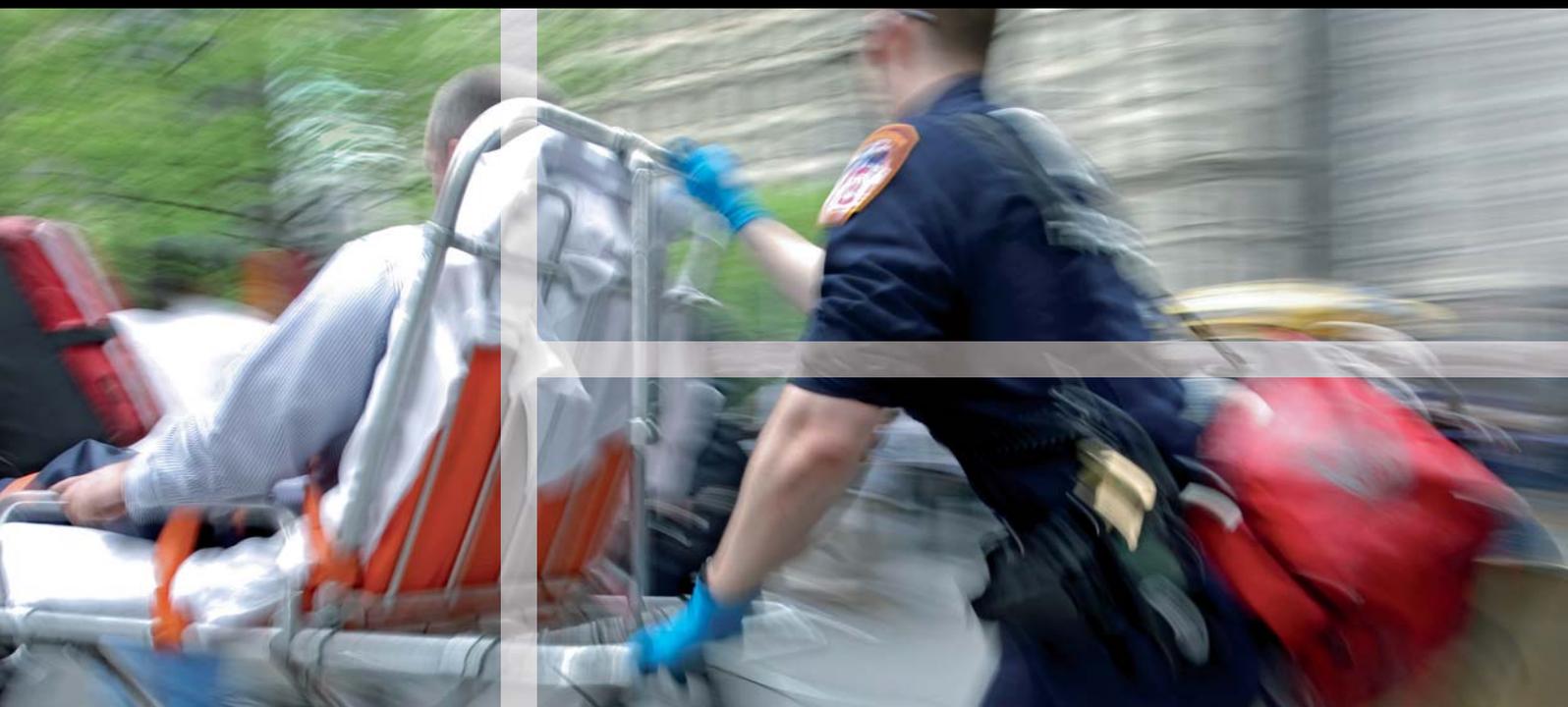


IMAGING AND RESUSCITATION IN TRAUMA

GUEST EDITORS: ARISTOMENIS K. EXADAKTYLOS, MARC A. DE MOYA, FIONA LECKY,
PETER DRISCOLL, HEINZ ZIMMERMANN, LEE ALAN WALLIS, AND ROBERT A. NOVELLINE





Imaging and Resuscitation in Trauma

Emergency Medicine International

Imaging and Resuscitation in Trauma

Guest Editors: Aristomenis K. Exadaktylos, Marc A. de Moya,
Fiona Lecky, Peter Driscoll, Heinz Zimmermann,
Lee Alan Wallis, and Robert A. Novelline



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Editorial

Imaging and Resuscitation in Trauma

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Injury is expected to become the second leading cause of death by 2020 [1].

The initial evaluation of a critically injured polytrauma patient is a challenging task and every minute can make the difference between life and death. It has been shown that mortality decreases as the time from injury to diagnosis and treatment is shortened [1]. Over the past few years, the assessment of trauma patients has evolved, due to the improved understanding of the mechanisms that contribute to morbidity and mortality in trauma, and this has led to the development of advanced trauma life support.

Because hemorrhagic shock is the second leading cause of death after trauma, the optimal treatment for bleeding has become a major priority. In their paper, V. Jeger et al. "The role of thromboelastography in multiple trauma" describe thromboelastography (TEG) as a very useful tool in managing bleeding patients, and we suspect that this procedure will become a gold standard of resuscitation during the next decade. Since fluid resuscitation is one of the major tasks in the management and treatment of severely burned patients, the paper by M. Stander et al. "The emergency management and treatment of severe burns" is really significant, as it presents a simple guideline for the initial management of severe burns, as utilised by the South African Burn Society.

But what would treatment be without imaging? Quick and cost-effective imaging has become one of the cornerstones of modern trauma management. R. Kaewlai et al.

"Blunt cardiac injury in trauma patients with thoracic aortic injury" and P. D. Levy et al. "Micropower impulse radar: a novel technology for rapid, real-time detection of pneumothorax" present some very promising data on detecting injuries of the heart and lungs. Micropower impulse radar, for example, is a novel technology for the rapid detection of pneumothorax, with the power to simplify decision making in the prehospital sector as well as in the trauma bay and to reduce the burden of radiation for patients and staff. Since no technology is perfect and any 100% sensitivity and specificity margin for diagnosing injuries is a myth, we focus on the importance of the followup of trauma patients and raise the awareness for even rare but potentially life-threatening complications.

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Review Article

The Role of Thrombelastography in Multiple Trauma

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Hemorrhage and traumatic coagulopathy are major causes of early death in multiply injured patients. Thrombelastography (TEG) seems to be a fast and accurate coagulation test in trauma care. We suggest that multiply injured trauma patients would benefit the most from an early assessment of coagulation by TEG, mainly RapidTEG, to detect an acute traumatic coagulopathy and especially primary fibrinolysis, which is related with high mortality. This review gives an overview on TEG and its clinical applications.

1. Introduction

Hemorrhage is a major cause of early death in multiply injured patients. One of the reasons of uncontrolled hemorrhage may be acute traumatic coagulopathy. It has been first discussed by Brohi and colleagues, and it is now thought to be induced by trauma and hypoperfusion [1, 2]. The pathomechanism of acute traumatic coagulopathy is extensively reviewed by Hess et al. [3] 25% of major trauma patients suffer from coagulopathy at admission to the hospital, and its presence is associated with a fourfold increase in mortality [2].

The initial treatment of bleeding trauma patients is not limited any more to damage control surgery but to damage control resuscitation, using a balanced administration of blood products in the ratio of red blood cells: fresh frozen plasma as 1 : 1 or 1 : 2, which is able to correct hypovolemia, anemia, and, to a certain degree, the acute traumatic coagulopathy [4, 5]. Additionally, the acute traumatic coagulopathy may also occur in absence of acute bleeding, for example, due to massive blunt injury and hypothermia. This pattern is typical for our patients population in a Level 1 trauma center in Switzerland, where we face mainly car accidents and injuries related to outdoor sports (skiing, climbing, base jumping, avalanches, etc.). In situations where coagulopathy is frequent but less obvious at admission of the patient to the resuscitation bay, the decisions should rely on evidence based point of care devices to correct coagulopathy.

In reality, the trauma physician is somehow blinded to the current state of coagulation because of long turnover times of standard coagulation screening from the lab and he/she has to base decisions on experience and gut feeling [6].

The search for appropriate point of care devices in trauma care brought thrombelastography (TEG) back in focus in 1997 by Kaufmann et al. after the technique had been used for years in cardiac and liver surgery [7, 8].

2. Thrombelastography—Assessing the Viscoelastic Properties of the Thrombus

The concept of thrombelastography had been first described by Hartert in 1948 [9]. Only its development to a compact device in combination with a computer made its manipulation easier and its measurements reproducible. The technique had been described before and should only be mentioned briefly [7, 10, 11]. A small volume of blood (340–360 μL) is placed in an oscillating cup kept at 37°C or at the patient's current temperature. A pin is suspended in the cup from a torsion wire with an electrical transducer. Initially there is no clot, the motion of the cup does not affect the pin, and a straight line trace is obtained. As the blood in the cup clots however, the motion of the rotating cup is transmitted to the pin and its oscillation is recorded (Figure 1) [11]. The procedure can be accelerated by activating coagulation of the sample with kaolin, a celite, which increases the surface (intrinsic activation), or with tissue factor (extrinsic

activation, RapidTEG). Additionally, heparinase coated cups and pins can be used to monitor coagulation properties of the patient's blood underneath heparin therapy. Finally, there are also kits to measure platelet function or fibrinogen in special situations, which are not discussed in this paper.

The most important TEG parameters are the reaction time " r ", which measures the initiation of coagulation until a first pin oscillation of 2 mm and represents initiating coagulation factors, " K " and " α angle" represent the dynamic of the clot formation and are measures of time from the end of " r " to 20 mm of pin oscillation, and the slope between " r " and " K " respectively. They correspond mainly with fibrinogen concentration. The maximal amplitude (MA) represents clot strength and is therefore an estimation of platelet function. Finally, LY30, which is the lysis of the clot 30 minutes after MA, measures the rate of fibrinolysis. These parameters reflect the strength of thrombelastography to monitor not only one step of the cascade-like conventional coagulation tests, which focus only on fibrin formation. It gives a rapid overview on the main players in the cascade which are important to the trauma physician like initiation factors, fibrinogen, platelet function, and fibrinolytic components.

As mentioned above, TEG has evolved in an automated device which only needs 2-3 pipetting manipulations and not more than 2 minutes to start. It is possible to train physicians and nurses in only one or two short sessions, even with no previous lab experience, to run a TEG accurately. A step by step protocol, as provided by the manufacturer, is helpful, especially in smaller trauma units, where TEG is not performed every day. Quality control test runs should be performed regularly and may be attributed to the quality control experts for point-of-care devices of your hospital. The site of blood sampling seems not to be neglectable, especially when comparing patients in a study or if there is repeated measurements on the same patient. It has been shown that there are differences between the sampling sites, not related to the oxygen content but to the calculated shear forces in the corresponding blood vessel [12].

Two different but very similar devices exist to measure thrombelastography: TEG 5000 Analyzer from Haemonetics (Braintree, MA, USA) and ROTEM from TEM (Munich, Germany). This paper did focus specially on the TEG device, but the conclusions from the current literature are applicable for ROTEM as well.

3. Animal Studies—Trauma Models and Coagulopathy

In trauma research, the main limitation, even in large multicenter studies, is the high variability of the studied patient sample. There is a heterogeneity in the injury pattern, age, comorbidities, mechanism of injury, and in many more. This is the reason why trauma studies focusing on hemodynamics, hemorrhage control, and coagulopathy are performed with animal models. Some recent publications are mentioned here, which all showed that TEG is a reasonable coagulation test in the early assessment of trauma patients.

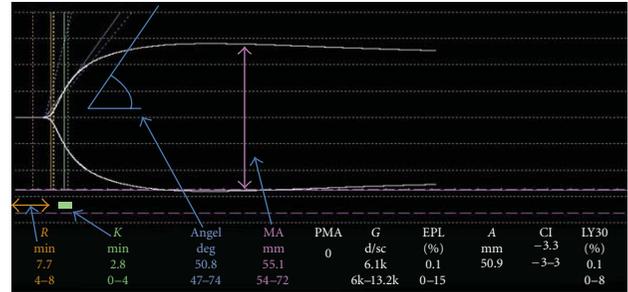


FIGURE 1: Normal TEG tracing with arrows highlighting the most important TEG parameters (r , K , angle and MA).

That pigs are a good model for research in coagulation and fibrinolysis has been shown by Velik-Salchner and colleagues [13]. White et al. worked with a pig model for traumatic hemorrhagic shock consisting of soft tissue injury and femur fracture. They randomized 23 pigs in 18 hemorrhage and 5 controls. With the beginning of hemorrhagic shock, fibrinogen concentration decreased rapidly. With increasing shock, TEG MA was reduced from 68.8 (SE: 0.9) to 64.7 (SE: 0.9). All other TEG parameters and conventional coagulation tests remained unchanged during hemorrhage [14]. Martini and colleagues were interested in the effect of hypothermia or hemorrhage on coagulation. Therefore, they randomized 24 pigs in four groups (control, hemorrhage with resuscitation, hypothermia, hemorrhage with resuscitation, and hypothermia). PT and aPTT did not change by hemorrhage or hypothermia. However, TEG parameters were affected by both: hypothermia prolonged r -time and K and decreased the angle α , hemorrhage only decreased MA. As only TEG was able to differentiate the mechanism of coagulopathy, the authors suggested, that TEG may be a suitable test to guide treatment of clotting alterations associated with hypothermia and hemorrhagic shock [15]. Kheirabadi et al. had a similar conclusion in a rabbit model of coagulopathy: 21 rabbits were randomized in three groups (control, warfarin treated, hemodiluted hypothermia). All animals underwent splenic injury. PT was valid to monitor warfarin-induced coagulopathy but failed to be reliable as a screening test for dilutional and hypothermic coagulopathy. TEG measurements of blood clotting rate represented better coagulopathic bleeding and mortality [16]. Finally, in a very complex pig model of multiple injury, preclinical phase and clinical phase, Alam and colleagues induced an acute traumatic coagulopathy. Different protocols of resuscitation had an effect on coagulation, which was measured most accurately with TEG: Hetastarch worsened coagulopathy, but it was rapidly reversed with the administration of blood components, especially FFP [17].

4. Clinical Studies—Describing the Trauma Patient

Kaufmann and colleagues studied the use of TEG in 69 blunt trauma patients and found that only Injury Severity Score (ISS) and TEG were predictive for transfusion. They

described that the majority of their patients studied was hypercoagulable [8]. Schreiber et al. did also discover that their trauma patients ($n = 65$) were hypercoagulable. They used noncitratd whole blood for the kaolin-activated TEG measurements. Mainly on day 1 (62%) compared to day 4 (26% of patients) after admission to the hospital. Women were more hypercoagulable than men. Like in animal studies described above, the authors did not detect pathologic conventional coagulation test values, which were within normal limits throughout the study [18]. However, the finding that trauma patients show mainly hypercoagulable changes had not been found by others. One explanation may be the small sample size and the high variability among trauma patients. Rugeri et al. used the ROTEM device in their trauma study ($n = 88$), which uses citrated whole blood. Four consecutive measurements had been done over the first 24 hours after admission to the ER. One third of the patients presented a coagulopathy at the first measurement after admission to ER. The authors showed strong correlations of conventional coagulation screening with the corresponding ROTEM parameter. However, the authors did not show the data of the evolution of ROTEM values over time, as they pooled all the ROTEM values together to compare and correlate them with the corresponding conventional coagulation parameter [19].

A larger sample of trauma patients ($n = 161$) had been studied by Carrol and colleagues: TEG had been performed either tissue factor activated or using platelet mapping. One sample was collected onsite and the second in the emergency department (ED). The authors did not detect changes from onsite to ED. No differences had been found between 22 patients who received blood products compared to the other patients. Only by using the platelet mapping assay, differences in adp responsiveness were obtained. When they had a closer look to the 14 fatalities in their sample, they found a difference in r value and MA compared to survivors. Hyperfibrinolysis was detected in three patients of whom two died. The authors concluded that TEG should be used as an additional tool to standard coagulation tests, especially to monitor hyperfibrinolysis, which is related to high mortality [20].

TEG had also been evaluated and applied in combat support hospitals by the U.S. Army in Iraq and Afghanistan. Plotkin et al. described 44 patients with combat injuries. A TEG was run with noncitratd whole blood samples (kaolin activated) within 24 hours after admission to the hospital. Conventional coagulation tests were suggesting hypocoagulation but did not correlate with blood product use. However, TEG values, especially MA correlated with blood product use platelet count [21].

To evaluate the best ratio of packed blood cells (PBC) to fresh frozen plasma (FFP), Davenport et al. used a ROTEM device to assess the coagulation status of 50 trauma patients who received more than 4 units PBC. The authors stated that a ratio of 1 : 2 or 3 : 4 should be achieved to preserve coagulation and a ratio of 1 : 1 did not show to be better. However, if the ratio is $<1 : 2$, patients presented hypocoagulable ROTEM results [5].

We started a few years ago to introduce tissue factor activated TEG (RapidTEG) in our resuscitation bay of a

level 1 trauma center in Switzerland. We found that using noncitratd whole blood from samples drawn at admission of the patient to resuscitation bay provided the trauma physician with fast and accurate results about the patient's coagulation status. This may help to guide focused blood product administration early in the treatment [11]. Kashuk and colleagues were studying the use of RapidTEG in trauma as well. In a first study ($n = 44$), they were interested if the use of noncitratd whole blood would have advantages compared to citrated whole blood. The authors correlated RapidTEG results with corresponding conventional coagulation measurements and found slightly higher correlation between noncitratd TEG values with CCT compared to citrated TEG results. This led the authors to the assumption that noncitratd samples may be more accurate. However, they stated that it is difficult to compare a static (conventional) with a dynamic test (TEG) [22]. In a further study, analyzing the data of 61 trauma patients, Kashuk et al. found that 34% of patients requiring massive transfusion suffered from primary fibrinolysis. 64% of these patients sustained penetrating injuries. RapidTEG tests had been run with noncitratd whole blood. The authors suggested the G-value as a measure of clot strength, (dynes/cm^2) as a predictive parameter for identification of fibrinolysis, and death as early as one hour after injury.

5. Conclusion

According to the current literature, thrombelastography seems to be a fast and accurate coagulation test in trauma care. We suggest that multiply injured trauma patients with an ISS > 16 would benefit the most from an early assessment of coagulation by TEG, mainly RapidTEG, to detect an acute traumatic coagulopathy and especially primary fibrinolysis, which is related with high mortality. Where the TEG should be run depends on the staff resources of your emergency department. We suggest to draw citrated whole blood at the admission of the trauma patient to the resuscitation bay and to send it to the central hematologic lab to run the test. The results may then be presented on a screen at the bed side. Tieu et al. did also share this opinion and stated that it may be difficult to run a TEG on noncitratd whole blood, especially due to the handling time of less than 4 minutes after blood sampling. This may demand multiple TEG devices in many critical areas of a hospital. Although some effects of citrated blood on TEG values had been observed, it may be an alternative to run a TEG on citrated whole blood in a central lab like conventional coagulation tests [23]. This may prevent the trauma physicians from an additional manipulation and could give them TEG values with a better quality control and reproducibility.

Interventional multicenter studies are needed to establish the use of TEG in the trauma setting. May early TEG results influence blood product administration and would this change the outcome of trauma patients? Do we use the correct TEG transfusion thresholds? Hopefully, we are able to answer some of these questions soon to improve the outcome of trauma patients.

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Review Article

The Emergency Management and Treatment of Severe Burns

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Burn injuries continue to cause morbidity and mortality internationally. Despite international collaborations and preventative measures, there are still many cases reported in high- and low-income countries. The treatment of these patients is often protracted and requires extensive resources. The adequate resuscitation of these patients coupled with meticulous wound care can have a huge impact on their outcome. The authors present a simple guideline for the initial management of severe burns which is utilised by the South African Burn Society and is based on the guidelines of the American Burn Association and the Australian and New Zealand Burn Association.

1. Introduction

Burn wounds and injuries are often devastating. They can have severe long-term consequences for the victims and they continue to be a major problem affecting communities worldwide [1]. The treatment of these patients is often protracted, and large amounts of resources are often needed to achieve the medical and psychological healing that needs to occur. Prevention is the vital factor that will have an impact on decreasing the morbidity and mortality associated with burns [2–4]. Education and training are vital steps to empower communities to help them protect themselves, and also the most vulnerable of burn victims are children. There have been studies into the different epidemiological factors related to burn injuries [5–11] with the subsequent introduction of training programmes, community outreach and social development, and the development of safe and effective household practices. These include initiatives like the Global Alliance for Clean Cookstoves [12]. International organisations like the World Health Organisation's Department of Violence and Injury Prevention and Disability (VIP) and the International Society for Burns Injuries (ISBI) strive to ultimately decrease this significant scourge by improving data collection, research collaborations, and preventative strategy development [13].

Statistics from the WHO demonstrate that there are over 300,000 deaths per year from fires alone with many more

from scalds, electrical burns, and other sources but there is still no accurate global data to confirm these numbers [13]. Over 95% of fatal fire-related burns occur in low- and middle-income countries [13]. Multitudes more patients have survived their injuries but are often left disfigured and destitute. Children and the elderly remain the most vulnerable groups with the highest mortality [13]. Intensive and specialised burn centres are in existence all over the world but are very often situated in high-income countries. These innovative and expensive treatment modalities play an important part, but the way in which a burn patient is initially managed carries an equally important role. Simple adherence to the basics including adequate resuscitation and meticulous wound care go a long way to achieving favourable outcomes and even in influencing mortality rates [14]. The following guidelines are based on the South African Burn Society management guidelines [15] which in turn are based on the American Burn Association [16] and Australian and New Zealand Burn Association guidelines [17].

2. Minimal Criteria for Transfer to a Burn Centre

Burn injury patients who should be referred to a burn unit include the following:

- (i) all burn patients less than 1 year of age;

- (ii) all burn patients from 1 to 2 years of age with burns >5% total body surface area (TBSA);
- (iii) patients in any age group with third-degree burns of any size;
- (iv) patients older than 2 years with partial-thickness burns greater than 10% TBSA;
- (v) patients with burns of special areas—face, hands, feet, genitalia, perineum or major joints;
- (vi) patients with electrical burns, including lightning burns;
- (vii) chemical burn patients;
- (viii) patients with inhalation injury resulting from fire or scald burns;
- (ix) patients with circumferential burns of the limbs or chest;
- (x) burn injury patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality;
- (xi) any patient with burns and concomitant trauma;
- (xii) paediatric burn cases where child abuse is suspected;
- (xiii) burn patients with treatment requirements exceeding the capabilities of the referring centre;
- (xiv) septic burn wound cases.

3. Treatment Protocol

3.1. Remove any Sources of Heat

- (1) Remove any clothing that may be burned, covered with chemicals, or that is constricting.
- (2) Cool any burns less than 3 hours old with cold tap water (18 degrees centigrade is adequate) for at least 30 minutes and then dry the patient.
- (3) Cover the patient with a clean dry sheet or blanket to prevent hypothermia.
- (4) Use of Burnshield [18] is a very effective means of cooling and dressing the injury for the first 24 hours.
- (5) Rings and constricting garments must be removed.

3.2. Assess Airway/Breathing

- (1) Careful airway assessment must be done where there are flame or scald burns of the face and neck. Intubation is generally only necessary in the case of unconscious patients, hypoxic patients with severe smoke inhalation, or patients with flame or flash burns involving the face and neck. Indications for airway assessment include the presence of pharyngeal burns, air hunger, stridor, carbonaceous sputum, and hoarseness.
- (2) All patients with major burns must receive high-flow oxygen for 24 hours.

- (3) Always consider carbon monoxide poisoning in burn patients. They may have the following symptoms: restlessness, headache, nausea, poor co-ordination, memory impairment, disorientation, or coma. Administer 100% oxygen via a non-rebreathing face mask; if possible, measure blood gases including carboxyhaemoglobin level.
- (4) If breathing seems to be compromised because of tight circumferential trunk burns, consult with the burn centre surgeons immediately regarding the need for escharotomy.

Circulation

- (1) Stop any external bleeding.
- (2) Identify potential sources of internal bleeding.
- (3) Establish large-bore intravenous (IV) lines and provide resuscitation bolus fluid as required in all compromised patients, using standard ATLS protocols [19]. Perfusion of potentially viable burn wounds is critical.

Estimate the Percentage Total Body Surface Area (%TBSA Burned (See Figure 1). Initially, use the Rule of Nines. In the case of all paediatric patients and for a more accurate assessment, use the Berkow diagram; alternatively, the patient's unstretched open hand represents 1% of TBSA.

Reminder. Accurate estimation of burn size is critical to ongoing fluid replacement and management.

3.3. Ongoing Losses (Once the Patient Has Been Stabilised)

- (1) Patients with <10% TBSA burns can be resuscitated orally (unless the patient has an electrical injury or associated trauma). This needs ongoing evaluation and the patient may still require an IV line.
- (2) In the case of patients with burns 10–40% TBSA, secure a large-bore IV line; add a second line if transportation will take longer than 45 minutes.
- (3) Burns >40% TBSA require 2 large-bore IV lines.
- (4) If the transfer will take less than 30 minutes from the time of call, do not delay transfer for an IV line.

Reminder. IV lines may be placed through the burned area if necessary (suture to secure). Avoid the saphenous vein if at all possible, and avoid cut-downs through unburned skin if possible. An intraosseous line is an excellent alternative in children.

- (5) Initiate fluids for ongoing resuscitation and fluid losses using the Parkland formula

$$4 \text{ mL crystalloid} \times (\text{kg of body weight}) \times (\% \text{ burn}) = \text{mL in first 24 hours,} \quad (1)$$

<u>Patient name and date of birth</u>	<u>Date completed</u>	<u>Type of burn</u>
	<u>Date and time of burn</u>	

<u>Superficial</u>				<u>Superficial</u> (pink, painful, moist)
_____ %				
+		=		
<u>Indeterminate or deep</u>				<u>Total % burn</u>
_____ %				_____ %
				<u>Indeterminate or deep</u> White, mottled, dark red or black, leathery

Paediatric adjustments

(i) Weight approximated to $(8 + \text{age} \times 2)$

(ii) <1 year—head and neck are 18% and each leg 14% of BSA

(iii) >1 year—for each year of life

(a) Head decreases by 1% of BSA

(b) Leg increases by 0.5% of BSA

Fluids

- (i) Total % burn _____ × weight _____ × 3.5 mL = total fluid in 24 hours _____.
- (ii) Total fluid in 24 hours _____ / 2 = volume in first 8 hours since burn _____.
- Volume in next 16 hours since burn _____.
- (iii) In children, add maintenance fluid to the above calculated volume _____.

Note:
If urine output is not adequate, increase fluids for the next hour to 150% of calculated volume until urine output is adequate.

FIGURE 1: South African Burn Society Burn Assessment Form.

with half of this total given in the first 8 hours after injury (note that this is the time from burn, not from presentation to healthcare services). Children must have their daily maintenance fluids added to these replacement fluids (including dextrose).

Example 3.1. In the case of a patient weighing 70 kg with a 50% TBSA burn, $(4 \times 70 \times 50) = 14\,000$ mL needed in the first 24 hours. Half is needed in the first 8 hours after injury.

Example 3.2. The fluid requirements of a child weighing 15 kg with a TBSA burn of 40% $(4 \times 15 \times 40) = 2\,400$ mL in the first 24 hours plus maintenance requirements of 1250 mL $(1000 \text{ mL} + 250 \text{ mL}) = 3\,650$ mL in the first 24 hours. Half is needed in the first 8 hours after injury.

Reminder. Do not give dextrose solutions (except for maintenance fluids in children)—they may cause an osmotic

diuresis and confuse adequacy of resuscitation assessment. Ideally, use Ringer’s lactate or normal saline for replacement fluid and a 5% dextrose-balanced salt solution for the child’s maintenance.

This is only a guide, and ongoing evaluation is essential as patients may need more fluids than calculated. Use the patient’s vital signs and, most importantly, urine output to guide ongoing requirements.

3.4. Assess Urine Output (This Is the Best Guide to Resuscitation)

- (1) Insert a Foley catheter in patients with burns >15% TBSA. Adequate urine output is 0.5 mL/kg/h in adults and 1.5 mL/kg/h in children.

Reminder. Lasix and other diuretics must not be given to improve urine output; increase IV fluid rates to increase urine output.

- (2) Observe urine for burgundy colour (seen with massive injuries or electrical burns). There is a high incidence of renal failure associated with these injuries, requiring prompt and aggressive intervention.

Reminder. If the urine is red or brown consult a burn centre.

3.5. Insert a Nasogastric Tube. Insert a nasogastric tube in any patient with burns >30% TBSA, or any patient who is unresponsive, shocked, or with burns >20% if preparing for air or long-distance transportation.

3.6. Decompression Incisions (Escharotomy). Assess for circumferential full-thickness burns of the extremities or trunk. Elevate the burned extremities on pillows above the level of the heart. If transfer will be delayed, discuss indications and methods for decompression incisions (escharotomies) with a burn surgeon.

3.7. Medication

- (1) Give tetanus immunisation.
- (2) After fluid resuscitation has been started, pain medication may be titrated in small intravenous doses (not intramuscular). Blood pressure, pulse, respiratory rate, and state of consciousness should be assessed after each increment of IV morphine.

3.8. Wound Care

- (1) Debridement and application of topical antimicrobials are usually unnecessary. Initial wound care needs to ensure that the burn is kept covered and the patient is kept warm. Plastic food wrap (such as Gladwrap) is ideal.
- (2) Apply a thin layer of silver sulfadiazine to open areas if transportation will be delayed for more than 12 hours.
- (3) Use of Burnshield is a very effective means of cooling and dressing the injury in the first 24 hours.

3.9. General Items

- (1) A history, including details of the accident and preexisting diseases/allergies, should be recorded and sent with the patient.
- (2) Copies of all medical records, including all fluids (calculation of fluids administered) and medications given, urine outputs, and vital signs must accompany the patient. These specific details may be recorded on the back of the burn size assessment sheet.
- (3) The burn centre will arrange transport if appropriate.
- (4) In the case of paediatric patients not accompanied by a parent, obtain consent in consultation with your burn centre.

3.10. Special Considerations with Chemical Burns (Consult Burn Centre)

- (1) Remove all clothing.
- (2) Brush powdered chemicals off the wound, then flush chemical burns for a minimum of 30 minutes using copious volumes of running water. Be careful to protect yourself.

Reminder. Never neutralise an acid with a base or vice versa; the heat generated can worsen the burn.

- (3) Irrigate burned eyes using a gentle stream of saline. Follow with an ophthalmology consultation if transportation is not imminent.
- (4) Determine what chemical (and what concentration) caused the injury.

3.11. Special Considerations with Electrical Injuries (Consult Burn Centre)

- (1) Differentiate between low-voltage (<1000 v) and high-voltage (>1000 v) injuries.
- (2) Attach a cardiac monitor; treat life-threatening dysrhythmias as needed.
- (3) Assess for associated trauma; assess central and peripheral neurological function.
- (4) Administer Ringer's lactate; titrate fluids to maintain adequate urine output or to flush pigments through the urinary tract (see urine output above). Useful laboratory test: arterial blood gas levels with acid/base balance.
- (5) Using pillows, elevate burned extremities above the level of the heart. Monitor distal pulses.

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Research Article

Blunt Cardiac Injury in Trauma Patients with Thoracic Aortic Injury

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Trauma patients with thoracic aortic injury (TAI) suffer blunt cardiac injury (BCI) at variable frequencies. This investigation aimed to determine the frequency of BCI in trauma patients with TAI and compare with those without TAI. All trauma patients with TAI who had admission electrocardiography (ECG) and serum creatine kinase-MB (CK-MB) from January 1999 to May 2009 were included as a study group at a level I trauma center. BCI was diagnosed if there was a positive ECG with either an elevated CK-MB or abnormal echocardiography. There were 26 patients (19 men, mean age 45.1 years, mean ISS 34.4) in the study group; 20 had evidence of BCI. Of 52 patients in the control group (38 men, mean age 46.9 years, mean ISS 38.7), eighteen had evidence of BCI. There was a significantly higher rate of BCI in trauma patients with TAI versus those without TAI (77% versus 35%, $P < 0.001$).

1. Introduction

Blunt cardiac injury (BCI) is a very rare, but potentially fatal, condition that accounts for 12%–32% of trauma-related fatality [1–3]. Ruptured cardiac cavities, coronary arteries, or intrapericardial portion of major vessels typically result in death at the scene of the collision [2, 4]. Victims of relatively less severe cardiac injuries such as myocardial contusion, hemopericardium due to contusions or lacerations, valvular regurgitation, or myocardial infarction due to coronary artery injury may survive the initial trauma and thus present to the emergency department (ED) for evaluation [1, 5, 6]. Among those who arrived alive at the ED, BCI is associated with a high mortality rate of 89% [7]. The frequency of reported BCI in trauma patients is difficult to determine. It varies widely from 0.045% to 86%, depending on patient population, subpopulation, and a variety of factors that were considered to make the diagnosis [1, 4, 5, 7–10]. Patients who suffer BCI usually have multiple severe concomitant injuries of other organs, including the thoracic aorta. By itself,

thoracic aortic injury (TAI) is considered a potentially fatal condition if left untreated [1, 11–15]. Therefore, a surgical or endovascular intervention is usually performed as a definitive treatment if the patient's condition allows. When an open thoracotomy or endovascular repair is expected, it is critical for treating physicians to evaluate all associated injuries to determine the need for immediate interventions and for preoperative risk assessment [16]. With the widespread use of CT in polytrauma patients, the diagnosis of TAI and coexisting injuries to the lungs, pleura, chest wall, and abdomen can quickly and accurately be made on a single scan [13, 17]. However, concurrent injuries to the heart in patients with TAI may go undetected on clinical examinations and on imaging due to the subtlety of the injuries. Existing data suggests a wide range of incidence of BCI in trauma patients with TAI between 3.6% and 63% [11, 14, 16]. By using diagnostic criteria of BCI based on abnormal ECG, elevated Creatine Kinase-MB (CK-MB), and/or abnormal echocardiography, we performed a retrospective investigation of trauma patients with TAI to (1) determine

the frequency of BCI in trauma patients with TAI; (2) assess whether BCI was significantly more prevalent among trauma patients with TAI than those without TAI in whom the abbreviated injury scales (AIS) were comparable.

2. Materials and Methods

2.1. Subjects. Our hospital institutional review board approved the investigation, which was deemed HIPAA compliant. Patients with a diagnosis of TAI from January 1999 to May 2009 were identified from the hospital's trauma registry. Patients with available ECG and serum Creatine Kinase-MB (CK-MB) performed within 24 hours of arrival at the hospital were included in the investigation. TAI was either diagnosed preoperatively with contrast-enhanced computed tomography (CT) of the thorax, with angiography, or at surgery. The diagnosis of TAI was made on CT scan when there was aortic pseudoaneurysm and/or intimal flap involving the thoracic aorta. All patients with TAI, diagnosed on CT were confirmed at the time of therapeutic angiography or surgery.

Patient's age, gender, history of cardiac disease, mechanism of trauma, abbreviated injury scales (AIS), injury severity scores (ISS), treatment of TAI and outcome including survival at 24 hours and at 30 days of arrival to the hospital were acquired from electronic medical records. Results of electrocardiography (ECG) performed within 24 hours of arrival, cardiac enzyme assays performed within 72 hours of arrival, and echocardiography performed within one week of arrival were collected for evidence of BCI. Patients with TAI were compared with a control population who did not have TAI. A list of potential control population was acquired by a search of the trauma registry for consecutive trauma patients from January 2006 to April 2009. Based on this list, we selected two control cases per one TAI patient by matching the abbreviated injury severity (AIS) for head (± 1), chest (± 1), extremity (± 1), face (± 2), abdomen (± 2), and external (± 1) to include in the control group.

The diagnosis of blunt cardiac injury in both case and control populations was based on ECG, serum CK-MB, and/or echocardiography. BCI was considered present if there was an ECG abnormality with either CK-MB elevation or abnormal echocardiography. ECG abnormality was considered a necessary component of diagnosing BCI. *Abnormal admission ECG* was defined as presence of nonspecific ST-T changes, premature atrial contractions, premature ventricular contractions, heart block, or ischemic changes in trauma patients without underlying heart disease. In patients with underlying heart disease, these changes were considered positive when they were not present on prior ECGs. *Abnormal CK-MB level* was found to correlate directly with cardiac complications from blunt cardiac trauma requiring treatment, therefore it was used as a criterion in this investigation. Abnormal CK-MB was defined as an elevated level above normal (0.0–6.9 ng/mL) [18]. Some patients underwent serial CK-MB assay, in these patients we recorded only the peak CK-MB within the first 24 hours of admission. *Abnormal echocardiography* was defined as at least mild degree of valvular insufficiency, presence of thrombus, wall

hypokinesia, pericardial effusion, and septal rupture [19, 20]. If there was a prior echocardiography, changes were considered abnormal if it was new. When present, BCI was graded according to the American Association for the Surgery of Trauma Organ Injury Scale (OIS) [21], which is based on ECG and pathology. Because none of our patients had autopsy, we utilized data from echocardiography as a pathological criteria. The classification is summarized in Table 1.

2.2. Statistical Calculation. Mean, standard deviation, and range were calculated for continuous variables, using Microsoft Excel (Microsoft Corp., Redmond, Wash, USA). Frequencies and relative frequencies were calculated for noncontinuous variables. Differences of categorical variables and continuous variables were tested by Fisher's exact and student *t*-tests, respectively, using QuickCalcs (GraphPad Software Inc., La Jolla, Calif, USA).

3. Results

From January 1999 to May 2009, there were 39 patients diagnosed with TAI. Twenty-five cases were diagnosed on CT and confirmed at endovascular treatment or surgery. The remaining were diagnosed with angiography (7 cases) and surgery (7 cases). Of these, 26 patients (19 men, 7 women, mean age 45.1 years, SD 21.2, range 20–98 years) had available admission ECG and serum CK-MB; therefore they were included in the investigation as the *study group*. The mean AIS for the head, chest, extremity, face, abdomen, and external were as follows: 1.62, 4.31, 2.04, 0.5, 1.62, and 0.73, respectively. The mean injury severity score (ISS) was 34.4 (SD 11.1, range 17–57).

There were 52 control cases (38 men, 14 women, mean age 46.9 years, SD 15.7, range 17–81). The mean AIS for the head, chest, extremity, face, abdomen, and external were as follows: 1.81, 4.17, 1.83, 0.37, 1.29, and 0.67, respectively. The mean injury severity score (ISS) was 32.6 (SD 11.1, range 16–59).

The detailed characteristics of study and control groups are provided in Table 2. There was no significant difference in mean age, gender, presence of underlying cardiac disease, mean AIS, and mean ISS between the two groups. Patients in the study group had a higher rate of trauma from motor vehicle collision and a longer mean length of stay than those in the control population. The abnormalities found on ECG, cardiac enzyme assay, and echocardiography are provided in Table 3. Frequency of ECG abnormalities in the study group was not statistically different from the control groups (80.8% versus 73.1%, $P = 0.787$). However, there was a higher rate of abnormal CK-MB in the study group (24/25, 96%) than in the control group (27/52, 51.9%, $P < 0.001$). Echocardiography was utilized in 15 study patients and 10 control patients. They were considered abnormal in three cases of the study group and two of the control group (20%). Among these five cases, echocardiography contributed to one additional case of BCI in the study group and none in the control group. Using criteria defined above, blunt cardiac injury was diagnosed in 20 patients with TAI (77%) and

TABLE 1: Grading of cardiac injury by the American Association for Surgery of Trauma. Adapted from [21].

Grade	ECG	Pathology
I	Nonspecific ST or T wave change, premature atrial or ventricular contraction, persistent sinus tachycardia	Blunt or penetrating pericardial wound without cardiac injury, tamponade, or herniation
II	Heart block, ischemic changes without cardiac failure	Penetrating tangential cardiac wound up to but not extending through endocardium without tamponade
III	Sustained or multifocal ventricular contractions	
IV		Blunt or penetrating cardiac injury with septal rupture, pulmonary or tricuspid incompetence, papillary muscle dysfunction, or distal coronary artery occlusion producing cardiac failure
		Blunt or penetrating cardiac injury with aortic or mitral incompetence
		Blunt or penetrating cardiac injury of the right ventricle, right or left atrium
V		Blunt or penetrating cardiac injury with proximal coronary artery occlusion
		Blunt or penetrating left ventricular perforation
		Stellate injuries <50% tissue loss of the right ventricle, right or left atrium
VI		Blunt avulsion of the heart

18 patients without TAI (35%) with a P value of less than <0.001. Of note, the death rate in patients with combined TAI and BCI did not significantly differ from those with BCI alone (7.7% versus 3.8%, $P = 0.606$).

Analysis of patients who were excluded from the study group ($n = 13$) showed that they were predominantly male ($n = 9$), had a mean age of 45.2 years (SD 20.5, range 17–92), and with mean AIS scores for the head, chest, extremity, face, abdomen, and external of 1.62, 4.62, 2.31, 0.23, 1.31 and 0.38, respectively. Six patients had serum CK-MB performed, three had echocardiography, and none had ECG. All CK-MB levels and echocardiography were abnormal. For the treatment of TAI, 4 patients underwent surgery, 6 had endovascular therapy, and three received supportive measures only. All patients who had endovascular therapy survived but the remaining patients deceased within their first 24 hours of admission. Autopsy was not performed in any of the deceased patients.

4. Discussion

Blunt cardiac injury is a potentially lethal injury that may occur in trauma patients with thoracic aortic injury. A high clinical suspicion of BCI in this patient subset is important to achieve the diagnosis [10, 19, 22, 23]. The incidence of combined TAI and BCI varies widely from 4% to 61.5% [11, 14, 16] depending on diagnostic criteria. Fabian et al. [11] reported 4% prevalence of BCI in 274 patients with TAI. Cook et al. [14] showed 18%–26% frequency of BCI in patients with TAI, diagnosed with echocardiography, serum troponin I, operative description, need for inotropic support, and ongoing treatment for angina pectoris to conclude the presence of cardiac injury. Kram et al. [16] described eight (out of 13) trauma patients with TAI who

had concomitant BCI diagnosed by ECG, cardiac enzyme assay, and radionuclide ventriculography. Our investigation confirmed, by utilizing a case-control study, that patients with TAI were significantly more likely to have concomitant blunt cardiac injuries with a frequency of 77%, compared with 35% in those without TAI who had comparable degree of traumatic injuries.

Our investigation has several limitations. The criteria used to define blunt cardiac injury have often been challenged because neither ECG nor cardiac enzymes are specific for cardiac injury in blunt trauma [22, 24–27]. Albeit, in clinical practice, they are commonly performed as it was evident in our investigation. Serum troponin I, believed to be highly specific for cardiac injury [28–30], was not consistently obtained in our patients during the investigation period. Therefore, we opted to use such criteria for diagnosis of blunt cardiac injury in our cases. We excluded approximately one third of patients with TAI from the analysis because they did not undergo ECG and serum CK-MB at the time of admission. The reasons excluded patients did not receive these diagnostic tests were unclear but it might be due to factors such as variation of care from one to another physician, acuity of patient's presentation eluding the ability to perform these tests before operative interventions, or a lack of standardized protocol to assess for BCI in our institution at the time of investigation. More patients in the study group had echocardiography performed compared with the control group (15 of 26 in the study group, 10 of 52 in the control group), which may introduce detection bias into the investigation. Given a small sample size and retrospective nature, we cannot conclude that the presence of BCI in TAI patients would alter clinical management or patient outcome. In our patient population, the majority of patients with TAI had concomitant BCI; therefore, there was

TABLE 2: Comparison between patients with TAI (study group) and without TAI (control group).

	Study group (<i>n</i> = 26)	Control group (<i>n</i> = 52)	<i>P</i> value
Mean age (years) (SD, range)	45.1 (21.2, 20–98)	46.9 (15.7, 17–81)	0.673
Male gender	19	38	1.000
Mechanism of trauma			
Motor vehicle collision	23	26	0.001
Fall	0	18	
Others	3	8	
Presence of underlying cardiac disease	4	2	0.091
Mean length of stay (days) (mean, SD, range)	25.1 (21.9, 0–84)	12.5 (10.9, 0–43)	0.001
Mean abbreviated injury scales (SD, range)			
Head	1.62 (1.9, 0–5)	1.81 (2.0, 0–5)	0.689
Chest	4.31 (0.5, 4–5)	4.17 (0.5, 3–5)	0.247
Extremity	2.04 (1.2, 0–3)	1.83 (1.2, 0–4)	0.469
Face	0.50 (0.8, 0–2)	0.37 (0.7, 0–2)	0.463
Abdomen	1.62 (1.6, 0–5)	1.29 (1.5, 0–4)	0.373
External	0.73 (0.5, 0–2)	0.67 (0.5, 0–2)	0.619
Mean injury severity score (SD, range)	34.42 (11.1, 17–57)	32.60 (11.1, 16–59)	0.497
Treatment of TAI			
Surgery	12	N/A	
Endovascular	12		
Medical	2		
Outcome			
Survive	23	50	0.326
Died within 24 hours of arrival	2	2	
Died within one month	1	0	

TABLE 3: Details of cardiac test results including ECG, CK-MB assay, and echocardiography.

	Study group (<i>n</i> = 26)	Control group (<i>n</i> = 52)	<i>P</i> value
Number with ECG abnormalities	21 (80.8%)	38 (73.1%)	0.787
Type of ECG abnormalities*			
Nonspecific ST-T changes	17	33	
Heart block	8	10	
Ischemic changes	2	1	
Atrial arrhythmias	2	7	
Number with abnormal CK-MB level	24 (24/25, 96%)**	27 (51.9%)	<0.001
Mean CK-MB level when elevated (ng/mL) (SD, range)	95.2 (309, 8.6–1544)	25.8 (23.5, 7.2–109.3)	0.109
Number with abnormal echocardiography***	3 (3/15, 20%)	2 (2/10, 20%)	1.000
Type of echocardiographic abnormalities			
Valvular insufficiencies	3	1	
Pericardial effusion	0	1	
Wall motion abnormalities	0	1	
Number with blunt cardiac injury	20 (20/26, 77%)	18 (18/52, 35%)	<0.001

* Numbers are not mutually exclusive. **CK-MB level was not tested in one subject, in whom an echocardiography was performed. ***Echocardiography was performed in 15 and 10 patients in the study and control groups, respectively.

not enough number of patients with isolated TAI to perform a meaningful comparison. The presence of BCI in patients with TAI, however, had been described in the literature as associated with high morbidity and mortality [16], high rate of hypotension [31], ICU admission [32], and increased risk of cardiac or operative intervention [18]. Although our data

suggested considering an outcome as short-term survival, death rates of patients with combined TAI and BCI were not different from those with BCI alone. We were unable to differentiate BCIs that were clinically significant from those that were not based on our present data. The diagnosis of BCI was based on clinical tests, not pathological evaluation

of the heart. Issues remain for the management of those who had blunt cardiac injuries, whether the knowledge of BCI changes the management plan or patient outcome. Further investigation with prospective design and a larger patient cohort would be required to answer these important questions.

In conclusion, blunt cardiac injury (diagnosed by a combination of ECG, serum CK-MB, and/or echocardiography) was significantly more frequent in patients with thoracic aortic injury than in patients without thoracic aortic injury.

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Review Article

Micropower Impulse Radar: A Novel Technology for Rapid, Real-Time Detection of Pneumothorax

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Pneumothorax detection in emergency situations must be rapid and at the point of care. Current standards for detection of a pneumothorax are supine chest X-rays, ultrasound, and CT scans. Unfortunately these tools and the personnel necessary for their facile utilization may not be readily available in acute circumstances, particularly those which occur in the pre-hospital setting. The decision to treat therefore, is often made without adequate information. In this report, we describe a novel hand-held device that utilizes Micropower Impulse Radar to reliably detect the presence of a pneumothorax. The technology employs ultra wide band pulses over a frequency range of 500 MHz to 6 GHz and a proprietary algorithm analyzes return echoes to determine if a pneumothorax is present with no user interpretation required. The device has been evaluated in both trauma and surgical environments with sensitivity of 93% and specificity of 85%. It is has the CE Mark and is available for sale in Europe. Post market studies are planned starting in May of 2011. Clinical studies to support the FDA submission will be completed in the first quarter of 2012.

1. Introduction

The ability to rapidly identify a pneumothorax (PTX) at the point of care for trauma victims can be life saving. This is particularly true in the military setting where undetected tension PTX is thought to contribute to death in up to 4% of fatal combat cases [1]. Accurate diagnosis of PTX in the prehospital setting depends on physical examination skills which include the ability to look for respiratory distress, jugular venous distension, or tracheal deviation, listen for diminished lung sounds, and feel for crepitus or broken ribs. Detection of such findings however can be challenging [2–4] even when physician providers are involved in patient assessment [5]. Consequently, prehospital protocols often incorporate a low-threshold for intervention when a PTX is suspected clinically [6–10].

Needle decompression, the procedure most commonly performed prehospital for emergent treatment of PTX, is not benign and has the potential to induce substantial morbidity when applied inappropriately [6, 7]. The existence of a quick, practical, easy to use method of diagnosing PTX would

greatly improve the margin of error for prehospital providers and facilitate the use of precise, directed intervention for individuals with thoracoabdominal injury. Portable lung ultrasound (US) has high sensitivity and specificity for detection of PTX [11–14] and has been proposed as modality capable of fulfilling this need [15–18]. Performance of lung US, however, requires advanced training and its accuracy is highly operator dependent making it suboptimal for use by basic field medics [19]. Moreover, there are issues with existing portable US equipment including cost, weight, and durability which preclude broad adoption by regionally funded emergency medical service (EMS) providers.

A novel alternative to PTX detection has been developed by PneumoSonics Inc. (Cleveland, OH, USA). Based on a technology called micropower impulse radar (MIR), the “PneumoScan” (Figure 1) is a portable device that emits ultrashort radar pulses (<1 ns) with pulse repetition rates on the order of 4 MHz. The device utilizes the same ultrashort pulse circuitry for time gating, with a 33 gigasample-per-second transient digitizer that allows the detection of reflective surfaces in air with spatial accuracy of approximately

5 mm (Figure 2). The radar return signals are digitized and immediately stored for real-time analysis using a proprietary algorithm.

2. Micropower Impulse Radar Technology

Micropower impulse radar technology has been licensed by PneumoSonics from Lawrence Livermore National Laboratories for use in medical devices. The technology uses very short ultrawideband (UWB) pulses that penetrate the body cavity. Returned echoes are a result of the type of medium that is encountered along the path of the pulse. The magnitude of the reflection and transmission coefficients depends on the relative permittivity of the structures. Using the fact that air has a different permittivity than normal body habitus, it is possible to use MIR for the detection of a pneumothorax. Also, as long as the tissues involved (and their permittivity) are known, the distances between reflections can be calculated by measuring the time and performing simple mathematics. The advantages of producing and detecting very brief radar impulses are considerable.

- (i) The target echoes return a tremendous amount of information. With short pulses, the system operates across a wide band of frequencies, giving high resolution and accuracy. The system is also less susceptible to interference from other radars.
- (ii) Battery current is drawn only during the short time the system is pulsed, so power requirements are extremely low (<0.1 Watt).
- (iii) The microwave power associated with pulsed transmission is exceedingly low (averaging tens of microwatts) and is medically safe.

The advantages of using UWB itself are significant in that it is possible to penetrate differing tissue densities. By optimizing the center frequency, UWB signals are able to distinguish tissue types (e.g., fat, muscle, bone, pooled blood) from air and each other. Additionally, the system readily penetrates clothing allowing the device to be used in the field or in the hospital setting quickly without having to disrupt the patient.

3. Using the PneumoScan

Use of the PneumoScan is straightforward and total scan time is less than 1 minute. The device is operated by acquiring signals from a transceiver placed at eight pre-specified sites along the anterior thorax (Figure 3). These locations are designed to allow rapid scanning of the entire chest cavity to isolate the PTX to a specific side of the body. Data are sent in real time to be analyzed by a program housed in the connected, hand-held computer. Skin contact is unnecessary as the MIR pulses can penetrate through clothing. The device emits an audible and visual signal when a scan is complete. Once all scans are completed, the PneumoScan analyzes the echoes and immediately displays results to the user (Figure 4) indicating the presence and location (side) of a PTX.



FIGURE 1: PneumoScan device.

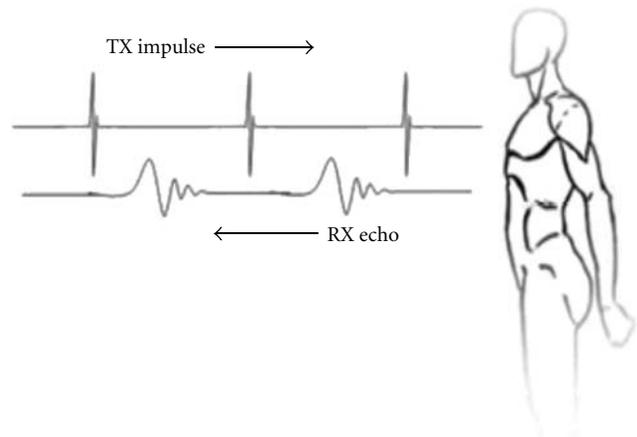


FIGURE 2: Micropower impulse radar signaling.

4. Simulated Pneumothorax

Initial study of MIR technology for PTX detection was conducted using a phantom system (concentric discs filled with water or air) developed specifically to simulate pneumothoraces of varying thickness. As shown in Figure 5, MIR signals change as a function of air pocket thickness (tested range: 3 to 12 mm). The root mean square deviation of the response signal obtained in phantoms with and without simulated PTX was calculated. A log normal relationship between the thickness of the PTX and the deviation from the control phantom was noted. These MIR transmission/reflection characteristics of the system can be applied to a one-dimensional projection of PTX volume. Each scan was then compared against the phantom system with no PTX present. The differences (Figure 6) show up at approximately point 281, which corresponds to the starting depth of the pneumothorax in the phantom based on time of flight calculations. Using proper filters and analysis methods, we can ascertain the depth of the PTX, how large the pneumothorax is based on the time of travel to the next

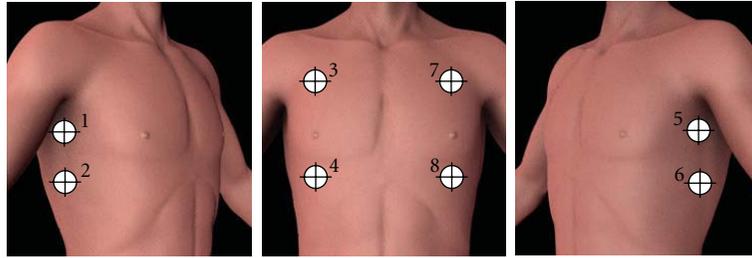


FIGURE 3: Pneumoscan data acquisition points.

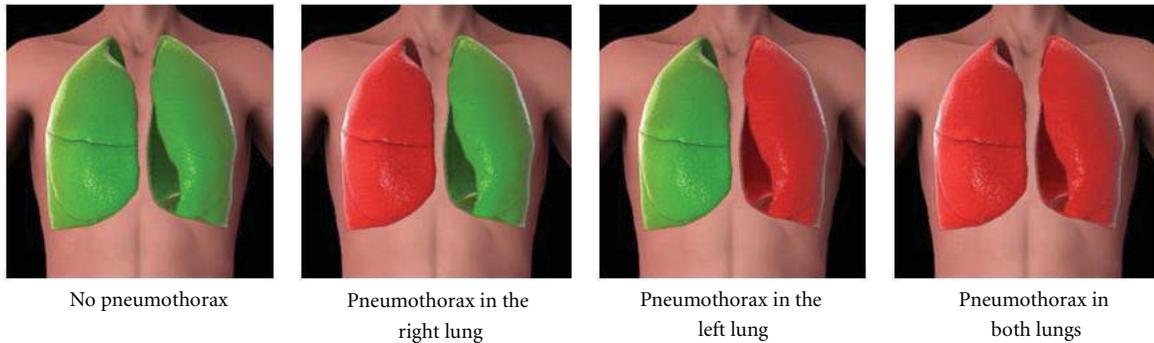


FIGURE 4: Example of real-time Pneumoscan data interpretation and report.

dielectric layer, and detect edges to allow reconstruction of the pneumothorax shape and size.

5. Preliminary Clinical Data

Initial clinical testing was performed on patients who presented to either of two Level 1 Trauma centers (Detroit Receiving Hospital and Sinai-Grace Hospital, both located in Detroit, MI, USA) with a blunt or penetrating chest injury. Using a prototype of the PneumoScan, a reading was taken prior to any intervention by the Emergency Department staff and confirmed with a chest X-ray (CXR) or computed tomography (CT) scan. We then evaluated the device’s diagnostic capabilities based on the following definitions.

- (i) True positive: MIR device identifies presence of a clinically significant PTX and the correct lung, as verified by CXR or CT.
- (ii) True negative: Both MIR and CXR or CT determine no clinically significant PTX.
- (iii) False positive: MIR identifies presence of a clinically significant PTX, while CXR or CT does not.
- (iv) False negative: CXR or CT identifies a clinically significant PTX but MIR does not or MIR identifies the incorrect lung when a PTX is present while not detecting the proper lung.

Reasonable sensitivity (93%) and specificity were found (Table 1) prompting further device refinement and a follow-

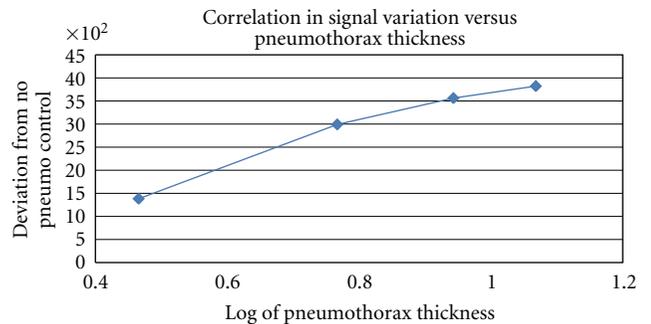


FIGURE 5: Correlation of scan analysis as a function of simulated pneumothorax thickness.

up study in patients scheduled for elective cardiothoracic surgery, who, by nature of their procedure, were at risk for development of a PTX. Using the present, commercially developed version of the PneumoScan, a reading was taken at all 8 acquisition points during the pre-, intra-, and postoperative period and the presence of a PTX was visually confirmed by the operating surgeon (used, for purposes of this study, as the gold-standard for diagnosis). Data were processed off line with double blinding of clinical findings and device results. Sensitivity was equivalent to the preliminary trauma study (Table 1) but specificity was slightly lower (Table 1). Of note, sensitivity of the PneumoScan in both studies was far better than that which has been reported for CXR (28–75%) [13, 20–23].

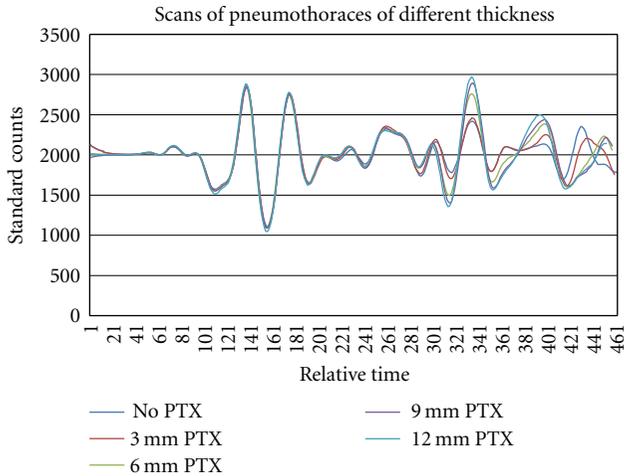


FIGURE 6: Scan results of various thicknesses simulated pneumothoraces.

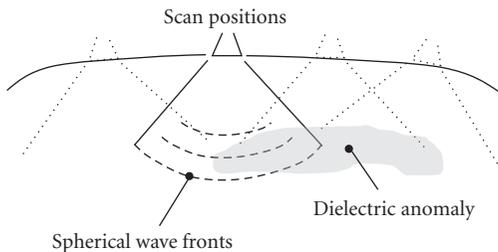


FIGURE 7: Microimpulse radar signal propagation in the body.

TABLE 1: Preliminary device performance.

	Clinical testing results	
	Trauma study	Surgical study
Total patients (Sides)	53 (106)	50 (100)
Sensitivity	93%	93%
Specificity	89%	84%
Location (left/right) accuracy	93%	100%

6. Device Availability

While the PnuemoScan has not yet been approved by the Food and Drug Administration (FDA) for use in the United States (USA), it is CE Marked for distribution in Europe. Postmarket release studies are planned for Europe in May of 2011, which will provide important data on real-world performance of the device when used in the trauma setting. Submission to the FDA is targeted for January of 2012, pending completion of a definitive, double-blinded, pre/posttrial of the PnuemoScan in patients undergoing lung biopsy procedures. Set to begin at three academic medical centers in the USA, this trial is designed to demonstrate device sensitivity and specificity of 95% with confidence interval of 88.7% to 98.4%; assuming a 3% dropout rate, a total of 345 subjects will be prospectively enrolled.

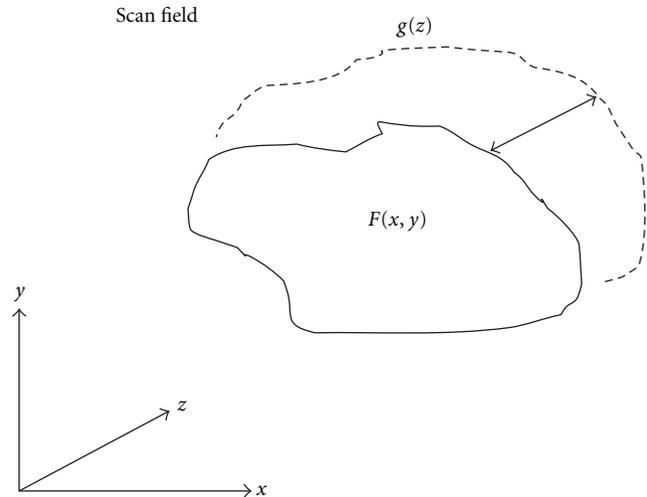


FIGURE 8: Microimpulse radar scan field and volumetric scatter.

7. Future Directions

With improved antenna design and an advanced algorithm, the PnuemoScan may be able to provide information on the specific location and volume of a PTX. Further refinements can also provide within-patient monitoring to allow rapid assessment of changes in a patient’s status and more precise quantification of posttreatment PTX resolution. The latter could dramatically reduce the need for repeated CXR thus minimizing compounded exposure to potentially harmful ionizing radiation.

At present, the UWB MIR antenna and electronics provide data that represents a one-dimensional volumetric representation of dielectric gradient into the body. However, the nature of the mechanism that couples the energy into the body with the horn type of antenna currently used in the PnuemoScan is such that the “direction” of propagation is essentially hemispheric. That is, a spherical wavefront penetrates the body as shown in Figure 7 centered at the antenna face, and propagates radially, returning a reflection from dielectric differences in the form of an expanding spherical half-sphere and its intersection with those differences. Anomalies in the dielectric as typified by a PTX are traversed radially across their entirety or a portion of their volume, depending upon their size.

Scans can be taken across the surface of the body and the inversion of their response can provide an accurate depiction of the volume of the anomaly. Inverse scattering can then be used to recover volume and to some extent, the image of any scattering volume within the body, given an appropriate choice of data acquisition and processing techniques. As shown in Figure 8, the form of a dielectric object can be generated by assuming a scattering area $F(x, y)$ that is nearly planar with a gradient in the z -direction, $g(z)$. The processing itself is quite geometric but unlike the Radon or backprojection methods required in CT, it is based on volumetric integration, not full tomographic inversion.

Additional scan positions provide more detail and can help clarify geometrically complex volumetric interactions. Optimization of volume assessment with PnuemoScan, therefore, may require a refinement in how data are acquired, specifically the number and location of scan performance. To better understand this, a pilot study of trauma patients is currently being conducted at Massachusetts General Hospital (Boston, MA, USA) which compares PnuemoScan data with PTX volume as quantified from multi detector CT images using a proprietary computer-aided volumetry scheme [24].

8. Conclusion

PSI has developed a point-of-care, noninvasive, portable, lightweight, low-power, diagnostic tool for detecting PTX. Based on novel, MIR technology, preliminary data for the PnuemoScan are encouraging with a sensitivity of 93% and a specificity of at least 85%. Further study of the PnuemoScan is in progress and submission for FDA approval is planned for early 2012. The PnuemoScan has been CE Marked and is presently available for clinical use in Europe.

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Case Report

Skull Base Osteomyelitis in the Emergency Department: A Case Report

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Skull base osteomyelitis (SBO) is a rare clinical presentation and usually occurs as a complication of trauma or sinusitis. A 5-year-old child presented to the emergency department with a three-week history of fever associated with drowsiness and left parietal headache, and a week's history of swelling on the left frontoparietal soft tissue. He had suffered a penetrating scalp injury four months ago. On physical examination, there was a tender swelling with purulent stream on the lateral half of his scalp. His vital signs are within normal limits. Plain X-ray of the skull showed a lytic lesion on the left frontoparietal bone. A cranial computed tomography (CT) scan demonstrated a large subgaleal abscess at the left frontoparietal region. SBO possesses a high morbidity and mortality; therefore, prompt diagnosis and appropriate treatment are mandatory to prevent further complications and to reduce morbidity and mortality significantly.

1. Introduction

Osteomyelitis can affect any bone. The common sites are the long bones especially the tibia and fibula. Skull base osteomyelitis (SBO) is a rare clinical presentation [1]. Osteomyelitis of the frontal bone associated with subperiosteal abscess collection is termed Pott's puffy tumour [2]. Sir Percivall Pott described Pott's puffy tumour in 1768 as a local subperiosteal abscess and osteomyelitis of the frontal bone resulting from trauma [3]. The prevalence of SBO is about 1.5% of all osteomyelitis [1]. The overall incidence of skull base osteomyelitis ranged from 57 to 95 cases annually [4].

SBO, which is a true bony infection, originates mostly from a chronic infection, which has been inadequately treated [5]. It can affect the calvarium or the base of the skull. In children trauma is the commonest predisposing factor [1]. Etiology of SBO may result from trauma, bone surgery, bacteremia, or a contiguous infectious focus and is further influenced by various diseases which affect the vascularity of bone, as well as by systemic diseases that can produce an alteration of host defenses. Radiation,

malignancy, osteoporosis, osteopetrosis, and Paget's disease are all conditions that decrease the vascularity of bone and, therefore, cause a predisposition to infection [1, 4, 5]. Mortality from complications in SBO is 20–40%. Early diagnosis and appropriate management of SBO can prevent neurologic deficits and reduce morbidity and mortality significantly [1]. We described such a rare case in a 5-year-old child presenting with skull base osteomyelitis secondary to penetrating scalp injury.

2. Case Report

A 5-year-old child presented to the emergency department (ED) on July 2, 2010 because of a three-week history of fever associated with drowsiness, and left parietal headache, and a week's history of swelling on the left frontoparietal soft tissue. The patient had suffered a penetrating scalp injury resulting from motor vehicle-pedestrian accident four months ago. He had never admitted to the hospital nor received a medical treatment. There was no previous history of any other concurrent medical conditions. On physical examination, there was a tender swelling associated with



FIGURE 1: A view of purulent stream and tender swelling on the patient's lateral half of scalp.

purulent stream on the lateral half of his scalp (Figure 1). Initial ED evaluation revealed a hemodynamically stable patient with an oral temperature of 38.9°C, a blood pressure of 120/85 mmHg, a heart rate of 96 beats/min, and a respiratory rate of 17 breaths/min. He had minimal neck stiffness but, Brudzinski's and Kernig's signs were negative. Furthermore, his pupils were equal in size and reactive. Examination of his respiratory, abdominal, and cardiovascular systems were normal. Plain X-ray of the skull showed a lytic lesion on the left fronto-parietal bone (Figure 2). A cranial computed tomography (CT) scan demonstrated a large subgaleal abscess at the left frontoparietal region (Figure 3). The leukocyte count, erythrocyte sedimentation rate and C-reactive protein (CRP) were 24900/uL, 40 mm/h, and 5.7 mg/dl, respectively. Other laboratory studies including blood chemistry and urine analysis were within normal ranges. He was started on intravenous ceftriaxone and clindamycin. By day 7 the treatment with oral amoxicillin/clavulanic acid (45 mg/kg/day divided every 12 hours) for 10 days was continued. Debridement of infected soft tissues and bone was performed by a neurosurgeon. The patient was discharged from the hospital on the 14th day following admission with no residual neurologic deficits, to be followed up in neurosurgery outpatient clinic.

3. Discussion

Osteomyelitis of skull bones is uncommon particularly in children. It can affect the calvarium or the base of the skull [1]. Anatomically, the bones involved in osteomyelitis of the skull include the mandible, frontal bone, maxilla, nasal bone, temporal bone, and skull base bones [4]. In the present case, left frontoparietal region of the skull was affected.

Etiology may result from trauma, bone surgery, bacteremia, or a contiguous infectious focus and is further influenced by diseases that affect the vascularity of bone, as well as by systemic diseases that produce an alteration of host defenses. Systemic diseases that reduce host defenses include diabetes, anemia, radiation, malignancy, and malnutrition

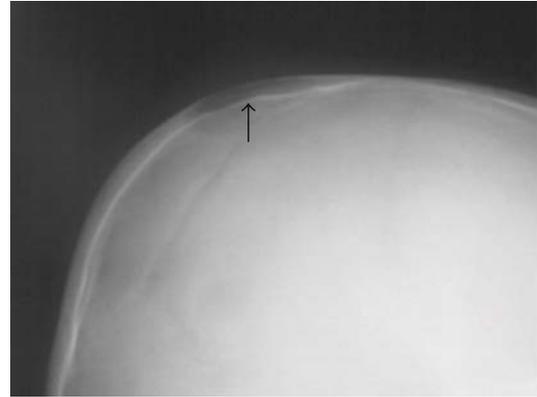


FIGURE 2: Plain radiograph of the skull taken at time of presentation showing a lytic lesion on the left frontoparietal bone.



FIGURE 3: Axial computed tomography scan of the head (taken at time of presentation) through the left frontoparietal region shows a large subgaleal abscess.

[3, 4]. Most cases of skull osteomyelitis are related to trauma [6]. In the present case, there was no prior history of bone surgery to the face or no comorbid diseases except malnutrition, but he had been sustained a penetrating scalp injury four month ago. He had never admitted to the hospital or received any treatment.

Acute osteomyelitis may present as a routine infection with several signs including fever, malaise, pain, and facial cellulites [7]. The main clinical findings include headache, sometimes associated with edema and spontaneous drainage if a sinocutaneous fistula has formed [8]. There may not be any associated noticeable radiographic changes [9]. Radiologic diagnosis of skull base osteomyelitis should be fast and accurate [10]. A cranial computed tomography (CT) and magnetic resonance imaging (MRI) can be used for early detection [4]. Early features are seen as islands of normal bone with increased or diminished density. Advanced features are seen as lytic lesions [1]. It may take up to 10 to 12 days for bone loss to be apparent radiographically [6]. CT scan shows contrast-enhancing rim

with a non-enhancing hypodense center [1, 6]. A cranial CT scan combines X-ray images taken from many different angles, creating detailed cross-sectional views of a person's internal structures [11]. In the present case, lytic lesions in the left frontoparietal bones at plain X-ray of the skull and a large subgaleal abscess at CT scan of the skull were demonstrated.

Useful laboratory values include elevated white blood cell count, certainly in the acute stages. Elevated erythrocyte sedimentation rate (ESR) and elevated C-reactive protein (CRP) may also be useful markers in both the diagnosis and treatment of osteomyelitis [4]. Monitoring of the ESR or CRP is one of the key investigations that can help to guide how long antibiotic therapy is continued, and its normalization would appear to be a good indicator that the infection has resolved [12]. In the present case, body temperature fell after the start of parenteral antibiotic therapy and debridement of involved soft tissue. Also, the clinical course in the present case was correlated with ESR and CRP levels. The patient responded quite well to the therapy of broad-spectrum antibiotics and surgical debridement with decreased activity levels in both ESR and CRP.

Acute osteomyelitis may be primarily managed with antibiotics [9]. Before the era of systemic antimicrobial therapy, skull base osteomyelitis was almost universally fatal [10, 12]. Broad-spectrum antibiotics are strongly recommended because the sites of primary infections vary and many different organisms can be the cause of the abscess formation. Brain abscess is the commonest complication of skull osteomyelitis. This is usually associated with subperiosteal abscess. The source of the infection must be eradicated [1]. Surgical treatment is usually focused on debridement of involved soft tissue and bone. Delay in surgical intervention has been associated with prolonged hospitalization [13]. Due to the implementation of effective antibiotics and early surgical intervention, the patient was discharged from hospital with no residual neurological problems.

4. Conclusion

Skull base osteomyelitis is a rare condition in children that usually require prompt diagnosis and treatment to avoid neurologic deficits and permanent disability and to reduce mortality. A combination of effective surgical debridement with prolonged appropriate antibiotic therapy in early term of skull base osteomyelitis might provide a complete resolution in all cases. Plain X-ray of the skull is helpful in establishing a diagnosis of osteomyelitis, but cranial CT is even more useful for determining the extent of the abscess.

Conflict of Interests

None of the authors have any financial or other conflict of interests related to this manuscript.

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Case Report

Isolated Transverse Sacrum Fracture: A Case Report

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Sacral fracture commonly results from high-energy trauma. Most insufficiency fractures of the sacrum are seen in women after the age of 70. Fractures of the sacrum are rare and generally combined with a concomitant pelvic fracture. Transverse sacral fractures are even less frequent which constitute only 3–5% of all sacral fractures. This type of fractures provide a diagnostic challenge. We report a unique case of isolated transverse fracture of sacrum in a young man sustained low-energy trauma. The patient presented to our emergency department after several hours of injury, and diagnosed by clinical features and roentgenogram findings.

1. Introduction

Reports of transverse fractures of the sacrum in the literature are not common. This issue has been attributed mostly to the challenge of obtaining diagnostic roentgenograms but also to the fact that this type of a fracture rarely is suspected [1]. A longitudinal fracture of the sacrum can be associated with approximately 45% of pelvic fractures. However, a transverse fracture of the sacrum is even less frequent, and accounted for only 4.5% of sacral fractures in humans [1, 2]. Lower sacral transverse fracture are often due to direct traumatic force against coccyx, and it is related to fall and resulting in break at the kyphos of sacrum mostly through the body of the lower 4-5 sacral vertebrae. Rarely, neurological deficit may accompany these fractures [2, 3]. There is often a delay in definitive diagnosis, if the quality of the roentgenograms is not adequate and if they are not examined specifically for the lesion. Most neurological insults associated with delays in diagnosis [1, 3]. Here, a case of lower fourth sacral vertebrae fracture associated with low-energy fall, who was diagnosed by clinical suspicion and appropriate roentgenogram, is reported.

2. Case Report

A 37-year-old male was admitted to the emergency department (ED) with low back pain and tenderness following

injury. He fell on his back onto a rough surface three hours ago. On examination, there were no weakness or anaesthesia in both the lower limbs, and inability to void urine, and to control bowel function. Roentgenogram of the pelvis revealed irregularity in the arcuate lines of the lower two sacral foramina associated with a transverse sacrum fracture through fourth sacral vertebrae (Figure 1). The fracture line from the anterior to the posterior aspect of the fourth sacral segment without narrowing of the sacral canal was confirmed later by lumbosacral tomography. As the patient had no neurological deficit, he was discharged home on the same day with recommendations for bed rest and analgesics.

3. Discussion

Isolated fractures of the sacrum are rare and generally these occur in combination with pelvic rim fractures [2]. Sacral fracture commonly results from high-energy trauma. Isolated sacral fractures which occur by shear forces on the pelvic ring are seen less commonly, and they are commonly transversely oriented [4]. Most fractures of the sacrum occur in women (94,3%) of advanced age (mean age : 70,6 years). A complete and careful physical and neurological examination will contribute a definitive diagnosis in suspecting this rare injury. Fracture of the sacrum should be suspected in the presence of low back or sacral pain and tenderness [5]. In addition, depending on the degree of root involvement, there



FIGURE 1: The anteroposterior roentgenogram of the lumbosacral area demonstrating a transverse fracture line at the right and anterior aspect of the fourth sacral vertebrae.

may be hypesthesia or segment provides sensation to the posterior aspect of the thigh, the posterolateral aspect of the calf, and the sole of the foot. The third sacral segment involves not only the superior medial aspect of the thigh but also part of the sacral region. The perianal region, anus, penis, posterior part of the scrotum, and posterior portion of the labia majora are innervated through the fourth and fifth sacral-nerve roots. These are the areas that must be tested carefully to elicit evidence of a sacral fracture [1, 5]. In the present case, despite he was young man and sustained low-energy trauma due to fall on a ground, the injury led to a stable sacral fracture associated with lower back pain and sacral tenderness. Transverse sacral fractures have been classified as upper and lower fractures. Lower sacral transverse fracture are often resulting from direct traumatic force. Rarely, it can produce neurological damage [1]. The muscles of the lower limb are supplied by multiple peripheral nerve roots, predominantly above the second sacral level. Therefore, no extensive paralysis will be developed in patients who have only a sacral fracture, as in the present case. If such paralysis does exist, other associated injuries should be suspected. Some of the patients with transverse sacral fractures demonstrates a neurological deficit of importance which mainly concerns the bowel or bladder [1, 2]. Furthermore, attention should always be paid to the bladder because the neurological deficit may not be apparent immediately after the injury. Lumbalgia, pain of the lower limbs, functional disability of the bladder, and bowel, seem due to a narrowed lumbar canal, a disc-nervous root conflict or a vertebral fracture [2]. No neurological deficit in the present case was demonstrated on arrival and during the 12-hour observation in the ED. As described by Rowell [6], transverse sacral fractures almost always involve the lower three segments of the sacrum (S3–S5). The ligamentous support of this region is achieved by the sacroiliac ligament, the sacrospinous ligament, and, more importantly, the sacrotuberous ligament. The coccyx may act as a lever arm on the body of the sacrum. The force so

applied is resisted primarily by the ligamentous structures just mentioned. In the present case, the fractured fourth sacral vertebrae, injured by either of the mechanisms pointed out above, was driven forward, resulting in injury to the sacral region [1, 6]. Diagnosis is often late, or sometimes is not even made. Standard roentgenograms of lumbosacral region with an adequate quality can able to predict ordinary degenerative lesions or fracture displacement narrowing the sacral canal in every case, and seem sufficient to make a definitive diagnosis of sacral fractures. If the quality of the roentgenograms is not adequate, fractures of sacrum are discreet, without displacement, often hidden by gas, stercoral stasis, or vascular calcifications. In this setting, a computed tomography of the lumbosacral region is always mandatory to confirm the diagnosis [1]. Treatment of transverse sacral fractures uncomplicated by neurological deficits should be conservative. However, in case of sacral root injuries with displaced sacrum fracture, decompression such as gibbectomy is recommended [1, 2, 7]. Outcomes of operative decompression are debatable, and even conservative treatment has been advocated [2].

4. Conclusion

Because isolated transverse sacrum fractures are rarely seen, and it can be a challenge of obtaining appropriate roentgenograms, the early diagnosis might be overlooked. The ED physician should be suspected of this type of fractures in the presence of low back or sacral pain and tenderness. Conventional roentgenograms can allow for visualization of a transverse sacral fracture, if the quality of the roentgenograms is adequate. In most instances, treatment is consisted of analgesia, sedation, and bed rest.

Conflict of Interests

None of the authors have any financial or other conflict of interests related to this paper.

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