Fracture blisters: a study of 13 cases and review of the literature

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Introduction

Fracture blisters are a relatively rare entity manifested by vesicles or bullae that arise on markedly swollen skin overlying a fracture[1]. They commonly form following fractures caused by high energy trauma or severe twisting type of injuries and occur in characteristic locations[1]. Reports concerning fracture blisters are limited in the literature and here we report the clinical characteristics of fracture blisters seen in 13 patients.

Method

During the period from July 2010 to June 2011, 13 patients who developed blisters after acute injuries were referred to the Dermatology Unit of National Hospital of Sri Lanka. These patients were prospectively followed up. Demographic data and data concerning the mechanism of injury, fracture type, time course in the development of lesions, clinical charac-teristics, their management and impact of blisters on the fracture management and outcome were studied.

Results

There were thirteen patients who developed fracture blisters during this period; ten males and three females with the age ranging from 14 to 72 years. Twelve patients had lower extremity fractures and one had upper extremity fracture. Two patients had multiple fractures involving the same extremity and one had fractures in both lower limbs. No one had other organ involvement.

Past medical history was significant in eight patients. This included insulin dependent diabetes mellitus in one, non-insulin dependent diabetes mellitus in five (one was newly diagnosed), hypertension in two and affective disorder in one whose medications included sodium valproate. Two of them were smokers and alcohol abusers.

The mechanism of fracture injury was a high energy trauma in most cases. Eight were involved in road traffic accidents and struck by a motor vehicle. Four were due to fall from height and one sustained injuries when a heavy metal bar had fallen from the top of a heavy vehicle.

Fracture patterns associated with fracture blister formation in this series included two tibial-plateau fractures, two tibial-plateau fractures with proximal fibular fractures, one tibial bi-condylar fracture, four tibial shaft fractures with fracture fibula, two tibial malleolar fracture, one calcaneus fracture associated with metatarsal bone fracture and one supracondylar fracture of humerus.

Fracture blisters were found to be located around the knee, over the tibia and on dorsum of foot and ankles in patients with lower extremity fractures and near the elbow in the patient with supracondylar fracture. Number of blisters, their size and position about each fracture were variable. One patient had single blister and the others had multiple blisters with the number ranging from 2 to more than 10. Blister size ranged from few millimeters to as larger as 10 cm in diameter. Blisters were discrete and unilocular in eight patients but in patients who had more than 10 blisters, several tiny vesicles localized to one area were also noted, some of them coalesced to form large bullae.

Blister fluid was either clear or haemorrhagic. Some had combination of both clear and haemorrhagic blisters. In addition, some of the blisters which contained clear fluid initially became haemorrhagic after 3-4 few days as they became older; and in one instance a blister with clear fluid became blood-stained when it refilled after its content had been aspirated for analysis.

Blisters were tense when they appear and became flaccid as they become older.

Marked skin and soft tissue swelling were seen in all patients. Seven patients had associated erythema, five had ecchymoses and four had superficial skin abrasions. Compartment syndrome occurred in one patient. The blisters were asymptomatic in all.

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In most patients blisters developed within 24 - 48 hours of acute injury; the interval ranged from 4 hours to 7 days. Blisters occurred within 24 hours in nine, between 24 to 48 hours in two and 48-72 in one. In two patient blisters developed postoperatively. Blisters were discovered as an incidental clinical finding at initial presentation or dressing change or in the surgical theatre. Hence the exact time interval between the injury and the appearance of fracture blister was difficult to be determined in every case. In two patients new blisters occurred daily over the period of 2-3 days.

Blister fluids aspirated from intact blisters in nine patients were found to be sterile on microbial studies. Skin biopsy samples in one displayed intra epidermal and sub epidermal blisters without inflammation and in another patient showed only sub corneal separation.

Blisters were left intact and allowed to heal spontaneously in ten patients. When blisters ruptured spontaneously they were covered with non adherent sterile dressing with/without topical antibiotic cream. Blister bed re-epithelialization occurred in 13 to 20 days (mean of 15.3 days). In three patients blisters were ruptured after 15, 7 and 2 days (but not deroofed) under sterile conditions and covered with povidone iodine paint and dry dressing, and re-epithelialization noted after 6 days, 8 days and 11 days respectively.

When blisters occurred preoperatively, they had an impact on the patient management, causing delay in surgery, change in operative plan, and prolonging the hospital stay. Four patients underwent open reduction and internal fixation and seven patients were managed non-operatively when soft tissue swelling had subsided and blister bed re-epithelialized. The overall mean delay in definitive management from the time of presentation was 17.9 days, range 12-28 days. Patient management was unaffected when blisters developed postoperatively.

All these patients were empirically treated with antibiotics. There were no major complications directly related to the presence of blisters. One patient with NIDDM and tibial plateau fracture who underwent ORIF developed pseudomonas infection of the surgical wound which responded to appropriate antibiotics without any sequelae. All blisters healed without any scarring. None had any complications related to fracture union.

Discussion

The incidence of fracture blisters has been reported as 2.9% of all acute fractures requiring hospitalization[1]. They usually appear within 24-48 hours of acute injury but may occur as late as 3 weeks after the trauma and may occur postoperatively as well. In this series the shortest time interval observed was 4 hours and in majority cases (69.2%) blisters occurred within 48 hours of injury. Fracture blisters rarely occur with open fractures. One patient with compound fracture and compartment syndrome developed blisters after ORIF and fasciotomy.

They occur in characteristic locations, most commonly over the tibia, ankle, elbow and knee – where there is little soft tissue between bone and skin or in areas of restricted skin motility. All the blisters in our patients were confined to sites distal to mid-shaft of humerus in upper extremity and distal to knee in lower extremity as observed by varela *et al*[1] in their series. Patients with existing co-morbidities like diabetes mellitus, hypertension, peripheral vascular disease, smoking history, alcohol abuse and lymphatic obstruction may be at increased risk of developing fracture blisters[1]. Ten out of thirteen patients in this series had one or more comorbid conditions and it is interesting to note that six of them had diabetes as a comorbid condition. The exact role played by these conditions in the formation of fracture blisters is not known. Undue joint or limb manipulation, dependent positioning, heat application in patients at risk may produce fracture blister in an otherwise relatively minor injury[2].

Fracture blisters may occur singly or in multiples and the size can vary from few mm to several cm. Clinically the blister may contain either clear/serous or haemorrhagic fluid. They may be associated with skin and soft tissue swelling, erythema and ecchymoses. Generally they are tense blisters but older blisters tend to become more flaccid and are more likely to contain haemorrhagic fluid. Blister fluid within the intact blister has been shown to be sterile transudate[1].

Histologically, location of the blister may be intra epidermal or at dermo epidermal junction. Skin biopsies were done only in few cases in our study and findings were similar to the observations made by other authors. Biopsy examination of 15 blisters by Varela *et al*[1] showed sub corneal blister located superficial to the granular layer in 13 and sub epidermal blister in 2. In a clinical and histological study performed by Giordano *et al*[3], the authors showed cleavage injuries at dermoepidermal junction. There was complete separation of dermis from epidermis in haemorrhagic blisters whereas there were scattered areas of retained epidermal cells on the dermis in clear blisters which authors believed contributed to rapid re-epithelialization and less morbidity seen with clear blisters.

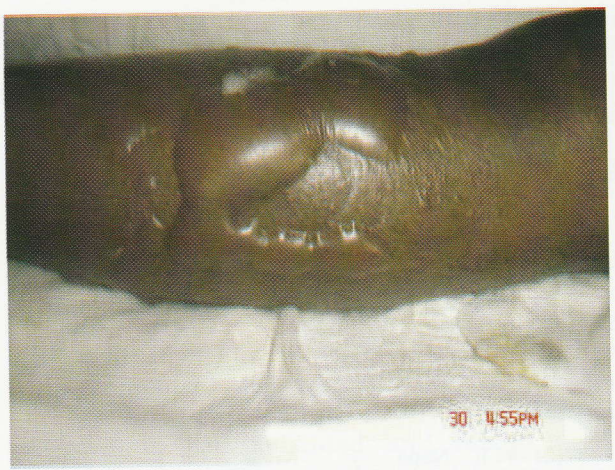
Table 1. Salient clinical features

Serial number	*Age and Sex*	*Type of fracture*	*Blister characteristics no, size, type, site*	*Soft tissue injury*	*Time interval before blister*	*Associated illnesses*	*Blister management*	*Delay in surgery*
1	48/M	Tibial plateau and fibula	Multiple (4), large (3-4cm), clear and blood, on knee and shin	Marked Oedema. ecchymoses	18 h (<24h)	Diabetes mellitus	None. Dry dressing only.	18 days (ORIF)
2	60/M	Tibia and fibula segmental	Multiple (14), varying size (few mm-4cm), clear and blood, on knee, shin	Oedema, ecchymoses and abrasions.	20 h (<24h)	None	Ruptured after 15 days then povidone iodine dressing.	22 days (ORIF)
3	42/M	Tibial plateau and proximal fibula	Multiple veicled and bullae, varying size (few mm -7cm) clear and blood, on leg	Erthema, Oedema, ecchymoses and abrasions	(<24 h)	Diabetes mellitus	Ruptured after 15 days then povidone iodine dressing	15 days (POP cast)
4	37/M	Tibial plateau fracture	Multiple (4), lense, 1-3 cm, clear and blood, on knee and upper leg.	Erythema, oedema, ecchymoses	20 h (<24 h)	Depression on sodium valproate	None. Dry dressing only	24 days (ORIF)
5	14/M	Supracondylar fracture of left humerus	Single, 2 cm, initially clear → haemorrhagic later. on elbow	Oedema	24 - 48 h	None	Dry dressing	18 days. MUA and POP back slap
6	30/M	Calcaneus and metatarsal bone Lis Franc fracture dislocations	Multiple (5), 1-3 cm, clear → blood on dorsum of foot.	Oedema	24 -48 h	None	None	18 days (MUA)
7	53/M	Upper tibia and fibula (compound)	Multiple (>10), few mm – 7 cm clear and blood knee, leg (medial)	Marked oedema, compartment syndrome.	<24 h (Blisters appeared after ORIF and fasciotomy)	Diabetes mellitus	Left intact. povidone iodine dressing	Blisters appeared after ORIF and fasciotomy
8	32 /M	Tibial bimalleolar fracture	Multiple (2), large 3-4 cm tense haemorrhagic ankle	Oedema	07 days after ORIF	Diabetes mellitus	Dry dressing	Blisters appeared 07 days after ORIF
9	45/M	Tibial and fibiual shaft	Multiple (6), large 1-4 cm tense clear → haemorrhagic	Oedema	<24 h	Smoker, alcohol abuse	Left intact. povidone iodine dressing	14 days MUA and POP

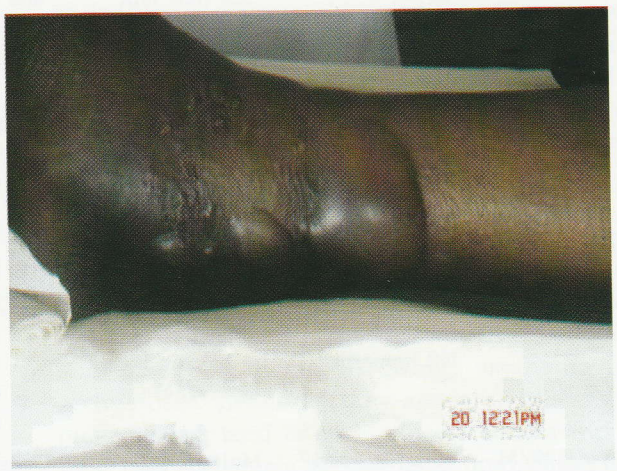
(Continued)

10	54 /M	Tibial plateau fracture	Multiple (6), large 1 - cm tense clear →haemorrhagic	Oedema, abrasions	48 - 72 h	Smoker, alcohol abuse	Left intact. Povidone iodine dressing	15 days MUA and POP
11	72/F	Tibial and fibiual shaft	Multiple > 30, feuumm - 10 cm tense clear → haemorrhagic	Oedema, erythema	24 h	Diabetes mellitus, hypertension	Povidone iodine dressing	2 days POP cast
12	34/F	Tibial plateau fracture	Multiple (10), tense clear → haemorrhagic	Oedema, erythema	24 h	Hypertension	Ruptured. Povidone iodine dressing	13 days POP
13	47/F	Medial mellitus	Multiple (4) tense clear → haemorrhagic	Oedema, erythema, abrasions, ecchymoses	<24 h	Diabetes mellitus	Ruptured. Povidone iodine dressing	28 days ORIFL

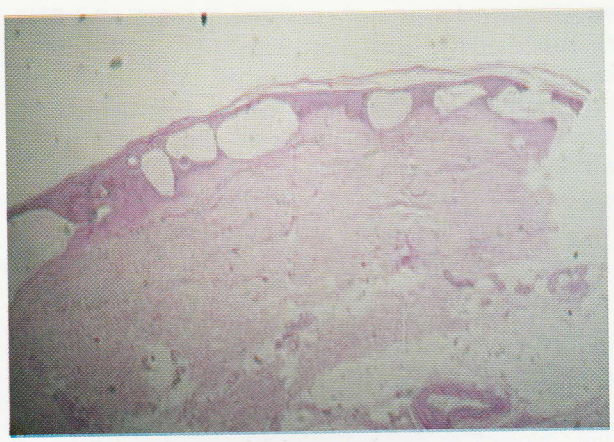
M – Male, F – Female, ORIF – Open Reduction and Internal Fixation , MUA – Manipulation Under Anaesthesia, POP – Plaster of Paris

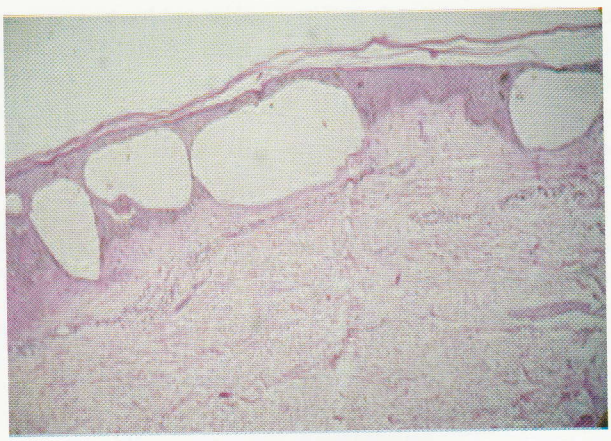


Medial view right leg one day after injury show in marked oedema and injury clear vesicles and bullae.



Lateral view left ankle one day after demonstrating marked edema, ecchymoses, and haemorrhagic bullae.





The pathophysiology of fracture blisters is multifactorial. Fracture blisters are thought to be caused by dermal-epidermal separation secondary to strains created in the skin during the initial fracture deformation. When a critical strain is reached during the fracture mechanism, the differing elasticity and vesico-elastic properties of the dermis and epidermis cause the two layers to separate and then the combination of the inflammatory cascade and Starling forces causes the fluid to pass into the potential spaces between the dermis and epidermis. The results of the biome-chanical study involving uniaxial strain test at several levels on cadaver ankle skin specimens performed by Giordano *et al* support this hypothesis[4].

Early stabilization of fractures and elevation of limbs to reduce oedema and vascular congestion may decrease the incidence of fracture blisters. Early surgical intervention before blister develops may reduce the incidence of blister formation. In one study Varela *et al*[1] have shown that that patients who underwent open reduction and internal fixation within 24 hours of injury had a significantly lower incidence of fracture blister formation (2%) compared to those patients whose surgery delayed for >24 hours (8%).

Presence of fracture blisters may delay the surgery /alter the operative plan and/may be associated with increased risk of complications like infection, skin breakdown, and delayed wound healing, scarring, and problems in fracture union[1]. Haemorrhagic blisters appear to be associated with increased risk of complications. In one study, Varela *et al*[1] found major postoperative wound infections when blisters were present at the time of surgery and recommends avoiding surgical incisions through fracture blisters and associated damaged soft tissues. However Giordano *et al*[5] observed no skin or wound complications when incisions were made through clear-filled blisters or adjacent to either type of blisters. But wound healing complications developed in two patients in whom incisions were made through blood-filled blisters.

When fracture blisters have occurred variety of treatment options are available, including:

1. Sterile deroofing and application of silver sulfadiazine and/or non-adherent dressings,
2. Sterile aspiration alone (with maintenance of overlying roof), and
3. Leaving the blister intact.

In ten of our patients blisters were left intact and in three blisters were ruptured (but not deroofed), at the discretion of treating surgeon, after varying intervals. Number of patients was small and no standard protocol was followed to make any stastical comparisons.

Giordano and Koval[5] compared these three methods of managing fracture blisters in 53 patients and found no significant difference in the outcome. The authors recommended leaving blisters intact and to deroof and cover only those blisters that have spontaneously ruptured. Strauss Eric *et al*[6], using a prospective protocol, deroofed all blisters at presentation and treated with silvasulfadiazine dressings. They found this was successful in minimizing soft tissue complications by promoting re-epitheliazation in all non diabetic patients; two diabetic patients developed full thickness skin breakdown at blister bed. There is no compelling evidence to support any method over another[7]. If feasible the blisters may be allowed to resolve spon-taneously which may take 10 to 14 days and, surgical treatment can be delayed; or alternatively they can be treated aggressively which may produce faster resolution, within 5 to 10 days[8,9]. It appears reasonable to avoid incisions through a non-epithelialized blister bed, particularly a haemorrhagic blister, if possible[7].

The clinical characteristics of fracture blisters observed in our patients are similar to those described in previous studies. Six patients had diabetes mellitus as a co-morbidity. No one developed any blister bed complications. But postoperative wound infection occurred in one diabetic patient who underwent open reduction and internal fixation. Further studies with large number of patients and prospective treatment protocol are needed to compare the outcome of various treatment methods.

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