FRACTURE BLISTERS

Richard J. Zirm, D.P.M.

The treatment of fractures complicated by overlying trauma blister formation remains controversial as well as intimidating. There is very little objective data in the literature regarding their characteristics and management. Previous recommendations have ranged from benign neglect with operative management through the blister, to surgical delay until blister reepithelialization has occurred.¹ The most serious concern is the potential increased risk for infection that the fracture area presents. Intelligent clinical decisions regarding the treatment of fractures involving fracture blisters should be made to prevent untoward complications.

ETIOLOGY

Fracture blisters occur after high-energy trauma, and are especially associated with a shear or torque component. Falls from heights and motor vehicle accidents are prime examples. Varela² identified fracture blisters in 2.9% of acute fractures. The time after such a fracture, especially within the first 6 and up to 36 hours, is marked by swelling around the injury. The initial swelling is largely due to hemorrhage from torn blood vessels that cause hemarthrosis or hematoma. After 6 hours, interstitial edema becomes a more significant contributor and continues for the next 36 hours before beginning to subside. Soft tissue injury produces increased vasodilation while perfusion to traumatized tissue decreases. This favors the formation of interstitial edema. This edema can separate the epidermis from the dermis. The deficit fills with fluid and becomes the fracture blister. The blister demonstrates a cleavage injury at the dermalepidermal junction.

According to Varela's study, fracture blisters were most likely to occur 24 to 48 hours following acute injury.² Varela's study cited the incidence of fracture blisters associated with ankle fractures as 4.2%, calcaneal fractures 10.9%, and pilon fractures 29.4%.

The skin around the ankle demonstrates several characteristics which place this area at high risk for blister formation. The epidermal papillae over the malleoli tend to be flatter than elsewhere in general. This signifies a lack of extensive arterioles between the rete ridges. Second, the subcutaneous fat is sparse, especially over the medial malleolus and the lateral calcaneus where the skin is relatively thin. There is an absence of well-formed adipose or muscular layers which, when present elsewhere protect the skin and deeper structures. The skin over the medial malleolus is usually 1.5 to 2.5 mm thick compared to 5 mm on the soles and palms.³ Additionally, there is extensive arborization of veins over the medial malleolus. Rupture of the venous plexuses causes extensive hemorrhage, venous obstruction and arterial spasm. Finally, there is a significant variation in the amount of hair follicles surrounding the foot and ankle. This may have important consequences in the re-epithelialization of wounds since the hair follicle is a major source of epithelial cells. It has also been postulated that the hair follicle helps to anchor the dermal-epidermal junction.⁴

It has also been postulated that the hair follicle helps to anchor the dermal-epidermal junction.⁴ A biomechanical study performed by Giordano⁵ demonstrated dermal-epidermal separation in 60 cadaver skin specimens when the skin was strained to 152% or greater. Lesions that demonstrated partial separation developed clear fluid blisters whereas complete separation of the dermis and epidermis developed blood-filled blisters. It is theorized that the blood-filled blister represents a slightly deeper injury than the clear fluid-filled blister and has a higher risk of poor healing of a surgical incision. The more serious "red blister" represents a more severe injury and is probably the result of papillary vascular injury leaking blood into the fracture blister. These blood-filled blisters take on average four more days to re-epithelialize than the clear fluid blisters. The fluid-filled blisters in Giordano's study⁶ reepithelialized in a mean of 12 days (range, 9 to 13 days); blood filled blisters healed in 16 days (range 11 to 19 days). (Fig. 1)

The blister fluid was initially found to be a sterile transudate. Once the blister ruptures colonization, primarily with skin pathogens, occurs and continues until reepithelialization. The fluid can be positive for HIV antibody.

TREATMENT



Figure 1. Red, blood-filled blister on the distal forefoot. Clear fluid-filled blister on the lateral ankle.

PREVENTION

Compression and elevation remain the mainstays of initial treatment and prevention of acute fracture blisters. RICE therapy should be instituted immediately after the initial evaluation is performed. Compartment syndrome may be coexistent and needs to be treated or ruled out of the clinical picture. Intermittent pneumatic compression may have a place in selected injuries. Compression with a properly applied Jones compression soft cast is utilized widely by faculty members of the Podiatry Institute. This dressing may incorporate a decreasing pressure gradient from distal to proximal. Incorporating a temperature controlled device (Hot Ice) into the dressing has also been found to be helpful in controlling edema and reducing pain.

Surgery is often delayed for a myriad of reasons including polytrauma with life-threatening injuries, medical problems, scheduling difficulties, NPO status of the patient, obtaining laboratory or imaging studies, a delay in initial triage or assembly of a surgical team. These factors can delay operative time 24 to 48 hours or more. The subsequent delay may represent the least optimal time to operate on such patients because of the potential for massive edema, hemorrhage and interstitial edema. The Swiss AO group recommends that if operative procedures cannot be performed on the ankle within 6 to 8 hours and significant swelling is present, surgery should be delayed for 6 or more days, until soft tissue circulation and resistance to infection have returned to near normal.

The timing of surgery in acute fractures is of utmost importance in preventing complications associated with fracture blisters. Since the majority of fracture blisters develop 24 hours after injury it would make perfect sense to treat all fractures with a high risk of developing fracture blisters within 6 to 8 hours or even 24 hours if possible. This treatment, albeit in a perfect world with no delays to the operating room, would mimic the protocol established for emergency treatment for open fractures. It is unknown how many fracture cases have avoided fracture blisters and compromised skin conditions with prompt surgery.

A further advantage of immediate surgery is that rapid surgical decompression at the time of open reduction may actually decompress the soft tissue, drain the subcutaneous hematoma and prevent fracture blisters. Besides early release of the hematoma, early surgical intervention, (within 6 to 8 or even 24 hours) allows for reapproximation of the disrupted soft tissues, ligation of bleeding vessels, fixation of bleeding fracture surfaces and placement of drains. The resultant reduction in swelling makes for a vastly improved environment for soft tissue viability.

However, there remain cases where blisters will have already formed before surgery can be performed. Options include: delaying surgery until the fracture blisters heal, proceeding with ORIF despite their presence, avoiding or modifying the incision line to avoid the blister(s), and keeping the blisters intact until they can be deroofed under sterile conditions in the operating room.

There have been numerous treatment protocols described for fracture blisters. Basically they involve draining, deroofing or debridement of the blister followed by the application of an antibacterial agent (povidone-iodine, silver sulfadiazine, chlorhexidine gluconate, or even normal saline). A nonadherent gauze (xeroform, adaptic) is then applied and periodically changed. There is no generally accepted technique, and there is little difference in time to reepithelialize.

A popular technique involves draining the blister with a needle in the clinic or at bedside, painting with povidone-iodide, maintaining the blister roof as a biological dressing and treating the wound base like a second degree burn with silver sulfadiazine. A nonadherent gauze is applied and



Figure 2A. Fracture blisters 48-hours following a crush injury.

changed daily. The author has had success with a similar treatment that used a wet to dry dressing that employs diluted chlorhexidine gluconate with sterile saline. It is felt that silver sulfadiazine is toxic to keratinocytes and can inhibit epithelialization. There has been a trend away from using Silvadene cream for second degree burns for this reason.

If large blood-filled blisters have formed prior to surgery, the blisters should be treated as described previously. Closed manipulation may be performed at this time as well as the application of an external fixator. It is prudent to avoid or postpone open surgery in the presence of bloodfilled blisters until granulation and signs of epithelialization occur. (Fig. 2)

Another major consideration about the timing of surgery should be an evaluation of the extent of swelling and overall health of the skin overlying the fracture. One or two blisters over an edematous ankle may appear innocuous, but they are the tip of the iceberg of diminished dermal-epidermal viability in the involved area. A valuable clinical pearl that tests for skin viability is pinching the skin. If the skin wrinkles, the turgor is returning to within normal limits and the edema is resolving. At this point an incision can be made through a blister with less chance of complications.

The dilemma of surgical timing is complicated by the process of bone healing. Because a



Figure 2B. Blisters are drained, the roof left intact, painted with betadine, and a compression dressing is applied.



Figure 2C. Complete healing with re-epithelialization, 14 days later.

considerable amount of fibrosis may develop after 14 days, and the fact that it may take this amount of time or longer to heal fracture blisters, surgical intervention may need to be performed before blisters are completely resolved. Significant fibrosis makes fracture fragments more difficult to reduce. This is especially true for comminuted calcaneal fractures after two weeks. Granulation tissue, hematoma organization and bone resorption can hinder the goals of fracture reduction and internal fixation.⁷

Continued debate regarding the perioperative treatment of patients with fracture blisters continues to prevent universal guidelines from being established. However it usually takes a physician who treats trauma patients regularly, only one significant skin healing complication before he changes his protocol and becomes more conservative. The following observations may be helpful.

First and foremost, edema and not the presence of trauma blisters should dictate the timing of surgery. The skin should be supple with normal turgor and skin lines present. Secondly, it is more conservative not to make an incision through a fracture blister. It may be advantageous to allow the blister to reepithelialize before surgery. If this is not possible, then treat the blister for several days (3 to 7 days) with wound care to allow for some granulation and resistance to infection to occur. It is best not to incise through a healing blister. If an incision has to be made through a fracture blister, it is best to avoid the red, blood-filled fracture blisters whenever possible as these have been associated with a higher rate of complications.

In extenuating circumstances where an incision must be made through a fracture blister, even after edema has been treated and skin turgor has returned to near normal, healing complications must be anticipated. They may be minimized with meticulous, sterile postoperative wound care. The blister bed is dressed with a moist dressing daily starting the first postoperative day. Antibiotic creams (Bactroban) may be used as well. Intravenous antibiotics that began preoperatively are continued and adjusted to cultures. No incision should be made through compromised dermis.

In the event of severe skin compromise, where the viability of the skin is in question, closed treatment of the initial injury must be employed. The surgeon's judgment about the extent of skin damage, ability to heal an incision and potential for complications is of paramount importance. Arthrodesis or late reconstruction for a malunion can provide an acceptable result when surgery must be delayed.

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