(ii) Open fractures

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Abstract

Open fractures remain a significant cause of morbidity and, despite improvements in practice, unresolved questions and significant challenges in management remain. Much of the evidence base derives from work on open tibial fractures, which can be considered a worst case scenario since the risks of both non-union and infection in these injuries is particularly high. Evidence based guidelines exist for the management of open tibial fractures and much of this work can be extrapolated to include other open injuries. This article aims to present a comprehensive overview of open fracture assessment and management with reference to the available evidence and current controversies.

Keywords antibiotic prophylaxis; Gustilo and Anderson classification; open fractures; wound debridement

Introduction

A fracture is considered open if the fracture fragments or fracture haematoma communicate with the outside environment. Any wound on the same limb segment as a fracture should lead to the injury being considered an open fracture until proven otherwise. These injuries imply high energy transfer, potential contamination of the wound, soft tissue stripping and devascularization of both soft tissue and bone, along with the potential for neurovascular injury. 30% of patients with an open fracture are multiply injured.

Historically, mortality due to infection was a common outcome following open fracture. With the introduction of antibiotics, outcomes were much improved but significant morbidity is still associated with these injuries. Non-union rates of 18% and infection rates of up to 50% have been reported in the most severe injuries[1] and the long-term morbidity, in terms of pain and loss of function due to bone and soft-tissue injury, cannot be underestimated. Outcomes have been found to correlate with the number of pre-existing co-morbidities, age and smoking as well as the severity of injury.[2]

The aim of treatment is to achieve a healed fracture, freedom from infection, satisfactory restoration of the soft tissue envelope and the return of function. The basic management principles established in the latter half of the twentieth century[1] remain essentially unchanged:

- initial emergent treatment; temporary stabilization of the fracture, wound dressing, antibiotic therapy, tetanus immunization
- primary surgical treatment; debridement, irrigation and fracture stabilization
- delayed surgical treatment; wound closure/cover within an appropriate time scale
- rehabilitation and follow-up.

Other adjuvant treatment modalities may be applied where appropriate, for example local administration of antibiotics, bone graft substitute, vacuum assisted therapy or flap closure.

Most studies concentrate on tibial fractures, since they are the most common. They also render the highest rates of infection and non-union, so it is reasonable to extend treatment recommendations derived from experience of open tibial fractures to include fractures in other locations. Areas of controversy include the timing of surgery, the type and duration of antibiotic administration, type of irrigation fluid and indications for the use of newer adjuvant treatments.

Classification

Open fractures are commonly classified according to the system developed by Gustilo and Anderson.[1] Their original paper appeared in the Journal of Bone and Joint Surgery in 1976, providing a practical classification system, which could serve as an aid to treatment and guide to prognosis. It also emphasized the importance of antibiotic administration and advised against the primary closure of type III wounds because of the high risk of contamination and subsequent infection. According to this system, type-I open fractures are characterized by a wound of <1 cm with minimal contamination, comminution, and soft-tissue damage. Type II features lacerations of >1 cm and moderate soft-tissue injury, but wound coverage is still adequate after debridement and periosteal stripping is not extensive. Type-III open fractures were further divided into three subtypes in a later modification by Gustilo et al.[3] Type IIIA is characterized by high-energy trauma, extensive soft-tissue damage, and substantial contamination, but wound coverage remains adequate after débridement has been completed. Type IIIB is similar to IIIA, except that the soft tissues will not permit closure without additional coverage procedures. Type IIIC is an open fracture associated with an arterial injury that requires repair to maintain limb viability (Table 1).

Studies have observed relatively high levels of inter-observer error using this system and most authors emphasize the importance of surgical debridement prior to assigning a classification grade to the injury. Despite this, the Gustilo and Anderson system has been shown to correlate well with the risk of infection and other complications (Table 2). Other classification systems have been devised but since the large majority of the literature references Gustilo and Anderson they are not discussed in this article.

Assessment

Open fractures are frequently associated with high-energy trauma and thus other life-threatening injuries may be present. It is imperative to assess the patient according to Advanced Trauma Life Support protocols and avoid being distracted by the obvious open injury. Early adequate resuscitation and stabilization may help to minimize secondary local injury from hypoxia, hypovolaemia and hypothermia. Co-morbidities, allergies and

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Classification system of Gustilo et al.[1,3]

Type	Definition
I	Wound <1 cm; minimal contamination, comminution, and soft-tissue damage
II	Wound >1 cm; moderate soft-tissue damage, minimal periosteal stripping
IIIA	Severe soft-tissue damage and substantial contamination; coverage adequate
IIIB	Severe soft-tissue damage and substantial contamination; coverage inadequate
IIIC	Arterial injury requiring repair

Table 1

tetanus immunization status should be established where possible. Accurate neurological and vascular assessment should be performed and documented, and a photograph of the wound prior to the application of the dressing may prevent repeated exposure of the wound by multiple examiners (Figure 1). This should be considered in the context of Departmental guidelines within the treating establishment. The risk of compartment syndrome cannot be underestimated and clinical examination should be repeated regularly. The presence of an open wound does **not** prevent the development of a compartment syndrome.

Plain radiographs of the limb must be obtained but supplementary imaging (eg CT scan) should not delay primary surgical treatment.

Treatment

The basic principles of open fracture management in the emergency department should then be applied; analgesia, antibiotics, fracture reduction, wound dressing and splintage. In smaller units, emergent transfer to the nearest trauma centre may reduce the risk of complications, particularly in injuries requiring combined orthopaedic and plastic surgical input.[4] However, in the presence of vascular compromise or compartment syndrome, emergency surgical treatment should not be delayed.

If the patient's immune status is uncertain, or their last tetanus booster was more than 10 years previously, tetanus toxoid should be administered. Tetanus immunoglobulin should be considered for patients who are inadequately immunized and have grossly contaminated wounds.

Prophylactic parenteral antibiotic therapy should be initiated in the emergency department. The surgical management should be scheduled taking into consideration the severity of the injury and the availability of appropriately qualified staff. The timing of surgical debridement and definitive wound closure, choice and duration of antibiotic treatment, type of irrigation fluid and mode of fracture stabilization must all be considered when planning the management of the patient.

Infection rates reported in the literature

Fracture type	Infection rates %
I	0–2
II	2–5
IIIA	5–10
IIIB	10–50
IIIC	25–50

Table 2

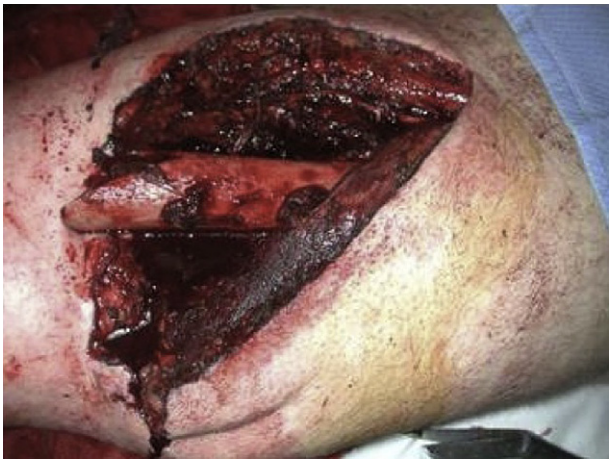

Figure 1 An open fracture of the femur. A photograph may be taken of the injury before dressings are applied to avoid the need for repeated removal of dressings for wound examination before debridement.

Antibiotics

Systemic antibiotic therapy

There is consistent, good quality evidence in the literature demonstrating that systemic antibiotic therapy plays a key role in the prevention of infection in open fractures. Burke, in 1961,[5] performed tissue-contamination studies to determine the effective period of antibiotic administration. Experimentally created lesions were compared with controls for animals given antibiotics 1 h before to 6 h after injection of the bacterial inoculum. There was no increase in bacterial colony formation in those animals given antibiotics within 3 h from the time of contamination.

Patzakis published the first randomized controlled trial of antibiotic use in this context in 1974[6] and more recently a Cochrane review showed that antibiotics reduced the overall risk of infection by 59% (relative risk 0.41)[7]. It is also clear that early administration has a significant effect on infection rate; Patzakis et al showed infection rate of 4.7% in 364 patients given antibiotics within 3 h compared with 7.4% in those given antibiotics after 3 h (661).[7] The most common organisms implicated in infection are gram positive *Staphylococci* and gram negative rods.

Broad spectrum antibiotics (eg first generation cephalosporins) have been shown to reduce infection rates in grades I and II fractures by as much as seven-fold in a number of studies. Additional gram negative cover is recommended for grade III fractures with either an aminoglycoside or a fluoroquinolone. In macroscopically contaminated wounds or farmyard injuries, cover for Clostridial sp. is required (penicillin or metronidazole) (Table 3).

It has previously been recommended that microbiological cultures should be obtained at initial debridement, but this is not supported by evidence in the literature. Infective organisms are usually nosocomial rather than initial contaminants. In a study of pre-debridement cultures only 8% of 226 organisms cultured caused infection. Of 106 patients with negative cultures 7% went