



Management of posttraumatic soft tissue infections

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Between 10 and 12 million traumatic wounds are treated annually in emergency departments in the United States [1,2]. More than 50% of these lacerations are caused by blunt trauma. The others are caused by sharp objects, such as metal, glass, and wood. Only a small percentage of these wounds is caused by mammalian and nonmammalian bites [3,4]. Most of these lacerations occur on the face, scalp, and arms, mostly in young men [2]. Because of these locations, an important goal of management of these wounds is to avoid infection, which can lead to cosmetically and functionally unacceptable scars [5].

The current management of traumatic soft tissue injuries incorporates many of the surgical principles developed over the past century. These principles include a thorough understanding of the pathophysiology of wounding, the risk factors for infection, the basic mechanisms by which posttraumatic sepsis develops, and the appropriate methods for treatment of these injuries and the prevention of complications. This article reviews the defense mechanisms involved in soft tissue wound healing, describes the risk factors for posttraumatic wound infections, and discusses the prevention and treatment of such infections.

Physiologic effects of wounding

The human body has evolved several defense mechanisms body to protect itself from the micro-biologic invasion that causes wound infection. These mechanisms include efficient mechanical barriers to bacteria and competent biologic protection mechanisms. The mechanical barriers to bacterial invasion

include the epithelium of the skin and mucous membranes. The skin also grants chemical protection in the form of surface lipids, and the mucous membranes provide protection by surface Ig A and an acidic pH. The skin and oral mucosal surfaces are also inhabited by normal flora that can compete with potential microbial pathogens.

Biologic protection is provided in the form of various internal mechanisms that are induced by local tissue damage. These mechanisms are triggered when the mechanical line of defense is violated. Once this damage ensues, an intense chemical activity is triggered, which involves activation of the kallikrein-kinin system, the release of amines, and an increase in vascular permeability that allows for influx of humoral and cellular immunologic elements. These elements are ultimately responsible for recognition of the organisms involved and their subsequent phagocytosis [6].

Traumatic wounds carry with them a higher degree of contamination because the mechanical protective features of the skin and mucous membrane are disrupted, which allows direct invasion of micro-organisms into the deeper tissues. The internal systemic biologic mechanisms of host defense are also compromised by the effects of the trauma. For example, with major trauma there is a decrease in cellular immune functions, intracellular killing, and endothelial system function [7,8]. There is also a decrease in humoral factors, such as the immunoglobulins and the complement system [8]. If traumatic shock develops, there is also a decrease in systemic perfusion, which, in the presence of local tissue damage, may reduce blood flow to the wounded area and further compromise containment of invading bacteria [9,10].

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Risk factors for posttraumatic wound infections

Studies on management of traumatic wounds have shown that the rate of infection ranges between 2.5% and 11.5% [11–13]. This rate is influenced by several variables, which are often referred to as risk factors. Some of these factors are related to the host, the environment, and the type of wound, whereas others are related to the techniques used to manage the wounds [12,14–16]. Factors related to the wound and patient include (1) size, configuration, and depth of the wound, (2) location of the injury, (3) mechanism of injury, (4) type and amount of contamination, including presence of a foreign body, (5) time between injury and wound closure, (6) care of the wound between injury and definitive care, and (7) age and systemic condition of the patient. The technical risk factors for development of wound infection include (1) inadequate débridement of foreign bodies, further bacterial contamination, and presence of devitalized tissues, (2) inadvertent introduction of foreign materials into the wound during cleansing, (3) inadequate hemostasis and failure to eliminate dead space, which provides an environment for bacterial colonization, (4) using an excessive number of sutures to close the wound, and (5) placing excessive tension on the sutures used to approximate the tissue edges and compromising local tissue perfusion.

Prevention of posttraumatic wound infection by proper wound care

The greatest deterrent to posttraumatic wound infection is a healthy wound. This status can be accomplished by (1) thorough cleansing, (2) identification, assessment, and atraumatic débridement of all devitalized tissues, (3) removal of all foreign materials, and (4) careful handling of the tissues. Evacuation of any hematoma and obliteration of all potential dead spaces by proper wound repair and approximation of all tissue layers are also essential elements in the prevention of soft tissue infections. Appropriate use of prophylactic antibiotics and tetanus prophylaxis are also important preventive measures [17].

Anesthesia

Before one begins definitive wound management, adequate patient comfort should be ensured not only to permit maximum wound repair but also to allow thorough examination, cleansing, and débridement of the wound. In most patients, comfort can be accomplished with local anesthesia (lidocaine with

1:100,000 or 1: 200,000 epinephrine). Inserting the needle through the open part of the laceration can decrease the pain of the injection [18,19]. Use of a field block is beneficial in reducing distortion of the operative site when accurate approximation of the wound edges is necessary. If there are skin or tissue flaps of doubtful viability, the use of lidocaine without epinephrine is desirable to avoid further impairing circulation [20].

Local anesthesia can be supplemented with analgesics or sedatives, if necessary. General anesthesia is preferable for uncooperative or combative patients and for children. General anesthesia is also often indicated in patients who require prolonged procedures, as in extensive lacerations, lacerations that require flap rotation or grafts for closure, and concomitant repair of facial bones fractures [21]. Patients with confirmed or suspected involvement of important structures, such as joints, nerves, or tendons, also may be better treated in an operating room setting [5,19,22].

Wound preparation

Once the patient is comfortable and the wound is adequately anesthetized, a complete and methodical cleansing of the wound should be achieved. The process involves irrigation and débridement of the wound before draping of the operative site in preparation for wound closure. Irrigation is essential in preventing infection because it removes debris, dirt, microorganisms, and devitalized tissue from the wound, which results in a reduction in infection rate [3]. Irrigation with normal saline solution using a 50-mL syringe and a 16-gauge needle is adequate for most lacerations [23,24]. In general, 250 to 500 cc of solution provides adequate irrigation for small wounds [24]. High-pressure irrigation is occasionally indicated for large wounds and when a high degree of contamination is present [17]. High-pressure irrigation has been shown to decrease the bacterial count of wounded tissues and decrease the rate of infection [25]. Vigorous irrigation in general, however, and high-pressure irrigation in particular may force debris into the wound and can cause further tissue damage [26,27]. For these reasons, high-pressure irrigation should be used with discretion and reserved for heavily contaminated wounds in which its benefits may outweigh its risks [17]. Similarly, use of concentrated povidone-iodine, hydrogen peroxide, and detergents may cause significant tissue damage and should be avoided [28]. Some authors even questioned the value of any form of wound irrigation in noncontaminated facial and scalp wounds [29].

Decontamination of the skin around the wound is another step in preventing wound infection. Decontamination can be accomplished effectively with mild soap and water or with a povidone iodine or hexachlorophene solution. Use of chemical solutions should be limited to the adjacent skin surface, and caution should be used to avoid getting them into the wound because these agents are noxious to tissues. Because shaving may damage hair follicles and allow for bacterial access and increase the risk of wound infection, it should be kept to a minimum. In areas such as the eyebrows, it should be avoided totally [23].

Débridement is considered by many to be the most important step in avoiding infection. One goal of wound débridement is to remove all foreign materials that may be attached to, or embedded in, the edges of the laceration or abrasion. Removing foreign material can be accomplished by thorough irrigation of the wound, vigorous scrubbing, or even surgical excision using a surgical blade or small curette. The second goal of débridement is the removal of devitalized tissue. The general rule regarding débridement of devitalized tissue is that all crushed or frayed edges that may become necrotic, all obviously nonviable tissue, and all grossly contaminated tissue should be removed. Exceptions to this rule involve certain anatomic areas, such as the eyebrow and vermilion border of the lip, where such débridement may compromise accurate realignment of the tissues and in instances in which tissue débridement would result in excessive tension on the suture line. In these instances, it is preferable to avoid extensive trimming of the tissue and to approximate the irregular edges without tension [30]. In the management of facial wounds, only minimal débridement is generally required because of the excellent blood supply. When tissue loss is present, it is recommended to save all tissue that has a chance to survive to provide the basis for secondary reconstruction at a later time [22].

Wound closure

Once the devitalized tissue has been removed, a decision must be made regarding the timing and type of wound closure. There are at least three major choices [16]: (1) primary closure and healing by primary intention, (2) leaving the wound open, treating it with frequent dressing changes, and allowing it to heal by secondary intention and wound contraction, and (3) using delayed primary closure, in which the wound is splinted in a position of rest with an occlusive dressing and is closed in 3 to 5 days when it is free of infection and necrotic tissue. The choice of

method is generally based on the assessment for risks of infection. It is often difficult to determine which treatment to use for a given wound, and the length of the “golden period” within which it can be closed primarily varies [31]. In general, there is a direct correlation between the time from injury to closure of the wound and the risk of infection. It is generally accepted that wounds with a high risk of infection should be closed as soon as possible (within the first 6–8 hours), whereas wounds with low risk of infection, such as those in the head and neck area, can be closed primarily within the first 18 to 24 hours after injury [17,24]. After 24 hours, for most wounds, consideration should be given to packing them open and performing a secondary repair 4 to 8 days later [17,30].

Postoperative wound care

For most wounds, patients should be instructed to keep them covered with a nonadherent dressing for at least 24 to 48 hours to protect the wound from gross contamination. After this period, the patient should wash, but not scrub or soak, the wound. Once the wound is left open, it should be cleaned two to three times a day with a cotton applicator stick and hydrogen peroxide or soap and water. The patient also should be instructed to place a topical antibiotic ointment on the wound. Prospective and retrospective studies have shown the value of topical antibiotic agents in decreasing infection in certain wounds [32,33]. Their benefit beyond day 5 of wound closure remains controversial, however [19]. A topical antibiotic ointment also keeps the wound moist, which speeds the rate of wound epithelialization [34].

The routine use of systemic prophylactic antibiotics to prevent infection of soft tissue wounds is not recommended [35]. Because traumatic wounds are, by definition, contaminated wounds, however, some of them are prone to infection despite all attempts to follow the principles of appropriate wound care. In such instances, and when the consequences of wound infection may be devastating, prophylactic antibiotics may be indicated [16]. The use of prophylactic antibiotics should be tailored individually based on factors such as the degree of bacterial contamination and the presence of the predisposing risk factors discussed previously. Several studies suggest that the administration of an antibiotic to prevent infection of the soft tissues after trauma may be of more value when given prophylactically within the first 3 to 4 hours of the injury and continued until the wound is sutured than when given

for a therapeutic course postoperatively [16]. Some authors recommend that prophylactic antibiotics be continued for 24 hours [36], however, with additional doses if the operative procedure is prolonged.

Tetanus continues to occur worldwide despite the availability of an effective vaccine. Consideration always should be given to administration of tetanus prophylaxis at the time of initial wound examination. A summary guide to tetanus prophylaxis is available through the Centers for Disease Control (Table 1) [37].

Treatment of posttraumatic soft tissue wound infections

Because traumatic wounds are caused by different types of trauma, the resulting soft tissue damage often ranges from blunt damage to penetrating and complex tissue injuries. The spectrum of microbiologic soft tissue contamination also varies and ranges from simple wound colonization without invasion to frank tissue infection and necrosis. Contamination of post-traumatic wounds can occur from introduction of endogenous normally nonpathogenic bacteria into the wound, from missiles or other objects, or from contact with various surfaces at the time of injury. The variation in degree of tissue injury and microbial invasion can result in a wide spectrum of wound infections, including general inflammation, a cellulitis or simple abscess, a diffuse inflammation and spreading cellulitis with or without signs of systemic toxicity, or a progressive necrotizing soft tissue infection [16]. Once wound infection occurs, it results in the immediate release of inflammatory cytokines from the monocytes and macrophages, which causes a delay in wound healing. These cytokines also may result in the release of growth factors that stimulate fibrosis and result in localized scar hypertrophy [20].

Regardless of the mechanism of injury and the degree of suspected wound contamination, once the signs and symptoms of wound infection become clinically evident, the goals of treatment should be to optimize tissue perfusion and nutrition, remove devitalized tissue, prevent further spread of the infection or further tissue destruction, and achieve wound closure [38]. To attain these goals, Fields et al described a five-phase unified approach to treatment of posttraumatic soft tissue infections [6]. The phases of this systematic approach that are applicable to wounds in the head and neck are phase 1: early recognition of infection; phase 2: rapid initiation of empiric antibiotics; phase 3: immediate surgical incision and drainage or débridement, when necessary; and phase 4: early wound closure.

Early recognition

Early recognition and diagnosis are essential to treating wound infections successfully and minimizing morbidity [16]. The diagnosis of infection should be based on the clinical signs and symptoms, the laboratory findings, and identification of predisposing factors. A definitive diagnosis can be made later based on culture of the microorganisms and examination of histologic specimens if débridement was performed.

Because a minor inflammatory reaction, pain, and swelling are normal components of the early stages of wound healing, recognition of infection based on these cardinal signs can be difficult and wound infections can be missed [16]. Changes in these signs on frequent examination of the wound and observation of increased swelling, tenderness, induration, discoloration, and fluctuation should increase the index of suspicion [6,16,39]. Systemic signs of infection, such as malaise, fever, tachycardia, and leukocytosis, may be present in patients with infected

Table 1
Recommendations for prophylaxis against tetanus

	After clean, minor wounds		After all other wounds ^a	
	Tetanus-diphtheria toxoid	Tetanus immunoglobulin	Tetanus-diphtheria toxoid	Tetanus immunoglobulin
History of tetanus immunization				
Number of previous doses <3 or not known	Yes	No	Yes	Yes
Number of previous doses ≥3				
Timing of last dose				
Within 5 y	No	No	No	No
Within 5–10 y	No	No	Yes	No
>10 y ago	Yes	No	Yes	No

^a Examples of these wounds include contaminated wounds, puncture wounds, avulsions, burns, and crush injuries.

wounds. Severe infections also may be associated with altered glucose metabolism, respiratory distress, altered mental status, and hypotension [40]. These signs and symptoms are nonspecific, however, especially in critically ill patients. When such signs and symptoms and local signs of wound infection are present, they are indicative of a serious problem and may be associated with progression of the infection into deeper tissue. Bacterial growth on culture provides essential information for treatment, although this is not essential for the diagnosis of posttraumatic wound infection. Because bacteria can colonize traumatic wounds even in the absence of infection, interpretation of culture results must be conducted with care. Finally, when there is an associated open fracture, differentiation between infection that originates in soft tissues and infection that is related to the fracture should be made by surgical exploration, microscopic examination of tissue samples, culture of organisms from the bone, or radiographic examination [41].

Also important in the recognition and diagnosis of posttraumatic infection is differentiation between superficial, or local soft tissue infection, and deep infections that involve fascia and muscle. Risk factors for the presence of deep soft tissue infection include systemic diseases, such as diabetes, immunocompromised status, underlying malignancy, and local factors, such as gross contamination and delay of wound closure.

Empiric antibiotics

The choice of empiric antibiotics should be based on the location and depth of the wound and the systemic status of the patient. For most posttraumatic head and neck soft tissue infections, first-generation, second-generation, and third-generation cephalosporins remain the drugs of choice for *Staphylococcus aureus* [42,43]. In most of the severe posttraumatic soft tissue infections, a combination of antibiotics is often used until data from culture and antibiotic sensitivity testing become available on day 2 or 3 [6].

Fungal infections are uncommon in posttraumatic wounds. *Candida albicans* and, less commonly, *Phycomycetes* are opportunistic organisms that may cause secondary infection after systemic antibiotic therapy, however. Fluconazole and amphotericin B are the most commonly used agents for treatment of these fungal infections [33,43]. Viral infections are also rare in posttraumatic wound infections but can be present in immunocompromised patients, patients with major multisystemic trauma, and burn patients [44]. The presence of these infections increases the

likelihood of sepsis from bacterial infection. Viral infections should be treated with such agents as systemic acyclovir.

Débridement

The sine qua non of treatment of wound infection is to provide wide drainage of any purulent material and débride all necrotic tissue [16]. Devitalized tissue acts as a culture medium for bacteria, creates an anaerobic environment, and impairs the cellular and humoral immune defenses [6,45]. It is imperative that débridement be performed if nonviable tissue is present on the margins of the wound. In instances in which tissue necrosis continues beyond the time of injury, such as in blast gun shot wounds, repeated débridement may be necessary. Frequent cleansing of the wound and repeated dressing changes should be instituted to assist in removal of bacteria, exudate, and devitalized tissue. It is important that a representative tissue specimen or a sample of purulent discharge be submitted for microbiologic identification of the offending organisms and for diagnosis of the extent of tissue necrosis. These specimens are best obtained from beneath the intact skin, away from the wound, because bacteria recovered from these sites are more likely to represent the true pathogens and not part of the wound colonization [6].

Wound closure

Wound closure should be performed when all the infection is resolved and healthy granulation tissue is present [6,46]. This goal usually can be accomplished by secondary intention or primary closure if the wound is small. Large wounds may require split-thickness skin grafting or flaps, however.

Management of bite wounds

Each year 1% of all visits to emergency rooms (approximately 300,000 visits) are related to bite injuries [47,48]. Ninety percent of these injuries are dog and cat bites, and the rest are human or other animal bites. Although most of these bites involve the hand, a significant percentage (16%) of dog bites are in the face and scalp, whereas only a small percentage (2%) of cat bites are in these regions [4,48].

Approximately 3% to 18% of dog bites and 28% to 80% of cat bites become infected [48,49]. The risk of infection is greatest for crush injuries, puncture wounds, and wounds to the hand [47]. Human bites,

although less common than dog and cat bites, were believed to be more prone to infection than those inflicted by animals. Such reports are biased by emphasis on human bites of the hand that present late with infection already present, however [50]. The generally reported poor prognosis in such cases is probably caused by the delay in treatment and location of the wound rather than the cause of injury. Human bites to the face, lips, and ears have a lower risk of infection (less than 3%), than human bites elsewhere (10%–12%) if treated properly [50,51]. With proper wound care, the rate of dog bite infections in the head and neck is also as low as 1.4%, even when prophylactic antibiotics were not used [52]. Wound infection from cat bites to the face is 3%, whereas it is 19% in the hand and 18% in the lower extremities [53].

The management of bite wounds remains somewhat controversial because there is considerable variation in the patients studied and in wound severity, and there is a relatively small number of cases in the studies reported [47]. There is general agreement, however, that the most effective method for preventing infection of these wounds is through proper initial management, which should include thorough irrigation with a copious volume of normal saline, débridement when necessary, and closure of the wound when appropriate. The wound also should be cultured. Radiographic examination of the adjacent facial bones may be indicated in cases in which a fracture is suspected. Proper selection and administration of antimicrobial therapy, which is based on the suspected flora and the risk of the wound for infection, are also important elements of treatment.

The most commonly isolated organism from infected dog and cat bites is *Pasteurella* species, 50% and 75%, respectively [48,49]. *Pasteurella canis* is the most common isolate from dog bites, whereas *P. multocida* subspecies *multocida* and *septica* are the most common isolates from cat bites. Other common aerobic organisms isolated from dog and cat bites include streptococci, staphylococci, *Moraxella*, and *Neisseria* [48]. Common anaerobic organisms that are isolated from mixed infections include *Fusobacterium*, *Bacteroides*, *Porophyromonas*, and *Prevotella*. Isolates from human bites include *S. aureus*, *Eikenella corrodens*, *Haemophilus influenzae*, and beta-lactamase-producing oral anaerobic bacteria [48,49].

Based on this spectrum of organisms, the empirical therapy for animal bites should be directed against *Pasteurella*, streptococci, staphylococci, and anaerobes. Commonly used empiric antibiotic

therapy for outpatients includes penicillin or dicloxacillin, ampicillin, or a first-generation cephalosporin [49,54,55]. A recent multicenter study of infected animal bites concluded that optimal therapeutic agents for this purpose include a combination of a β -lactam antibiotic and β -lactamase inhibitor, a second-generation cephalosporin with anaerobic activity, or combination therapy with either penicillin and a first-generation cephalosporin or clindamycin and fluoroquinolone [48]. Based on their in vitro activity, azithromycin, trovafloxacin, and ketolide antibiotics are also effective against all of the common aerobic and anaerobic isolates from these bites [56–58]. For treatment of high-risk human bites, empiric therapy includes penicillin and anti-staphylococcal agents, such as dicloxacillin and nafcillin [59]. An alternative to this combination is oral amoxicillin-clavulanic acid, which provides excellent in vitro coverage of the suspected pathogens [57,60]. Ertapenem also has an excellent potency against the full range of animal and human bite pathogens [58].

Use of prophylactic antibiotics for prevention of bite wound infection remains an area of considerable controversy. Some authors argue that such use is cost effective in selected cases. These cases include wounds seen more than 8 hours after injury, wounds that affect the hand, wounds that involve bone and joints, cat bite puncture wounds, especially near a joint or joint prosthesis, and wounds in patients with a compromised immune system. In such cases, the use of prophylactic antibiotic may decrease the rate of infection from 15% to 20% to approximately 5% [47,49,54,61]. Excluding these instances, most authors believe that prophylactic antibiotics are not indicated, especially for patients with bite wounds in the head and neck and patients who present within the first few hours after injury and show no evidence of infection [51,53,54,62,63]. This conclusion is based on the fact that most studies that showed a benefit of prophylactic antibiotics used diverse treatment protocols for wounds that were not of similar severity. Most of these studies also were biased by inclusion of hand wounds, which have a higher incidence of infection. Several studies have shown that with early surgical intervention using irrigation, débridement, and primary closure, the wound infection rate in the face, with and without the use of prophylactic antibiotics, is similar and routine antibiotic prophylaxis is not justified [52,53,55,64,65].

The timing of closure of bite wounds also continues to be controversial. In general, the time of wound closure is influenced by the period between injury and presentation for care. Researchers gen-

erally agree that bite wounds in the face, where cosmesis is a major concern, can be closed primarily or covered with skin graft when necessary—even 4 days after injury—with an acceptably low risk for infection because of the rich blood supply [51,63,66].

Summary

With improvements in surgical techniques and the availability of effective systemic antibiotics, the incidence of posttraumatic soft tissue wound infections is relatively low. When such infections occur, however, they can result in significant morbidity. These complications can be reduced by prompt initial management of the wound and proper treatment if infection occurs.

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