World J. Surg. 22, 146–151, 1998

WORLD
Journal of
SURGERY

Soft Tissue Infections

Ronald T. Lewis, M.D.

Department of Surgery, McGill University, Royal Victoria Hospital, 687 Pine Avenue, Montreal, Quebec H3A 1A1, Canada

Abstract. Soft tissue infections vary widely in their nature and severity, and their nomenclature is confusing. A clear approach to management must allow rapid identification and treatment of the diffuse necrotizing infections because they are life-threatening. This review classifies soft tissue infections by their degree of localization and the presence of tissue necrosis. Most focal nonnecrotizing infections start in the skin or adnexae and are easily recognized and readily treated by local measures. Patients with cellulitis, the commonest diffuse nonnecrotizing infection, should be stratified from mild to severe and complicated and then treated with oral or systemic antibiotics. Focal necrotizing infections are relatively uncommon, but they are readily diagnosed on sight and effectively managed by local débridement and systemic antibiotics. In contrast, diffuse necrotizing infections may masquerade in many forms, delaying diagnosis and treatment. Edema out of proportion to erythema, subcutaneous gas, and skin vesicles are important markers. Aggressive sequential débridement and broad-spectrum intravenous antibiotics revised after 48 hours provide the best strategy for management.

Soft tissue infections are important because they are common and vary widely in severity. Most are readily recognized and easily treated; but the more severe infections may masquerade in forms similar to those of more innocent infections, causing delay in diagnosis and treatment that may result in loss of limb or life. To avoid disaster, the thoughtful physician must have a high index of suspicion and be aware of clinical and investigative clues that suggest the underlying danger.

A simple classification helps to clarify the maze of terms that have been applied to soft tissue infections. It is useful to distinguish focal from diffuse and nonnecrotizing from necrotizing infections (Fig. 1). Further classification is based on whether toxic symptoms are present, what structures are involved, and the causative organisms. This discussion considers (1) focal nonnecrotizing infections; (2) diffuse nonnecrotizing infections; (3) focal necrotizing infections; and (4) diffuse necrotizing infections.

Focal Nonnecrotizing Infections

Focal nonnecrotizing infections may involve the skin (impetigo) or adnexal skin structures (folliculitis, furuncles, and carbuncles). **Impetigo contagiosa** is a highly communicable skin infection that affects the face and extremities of children. It is usually caused by *Staphylococcus aureus* or *Streptococcus pyogenes*, which inoculate the skin through small abrasions. **Folliculitis** refers to pyodermas centered in hair follicles and caused by *S. aureus*. They may extend deeply to form **furuncles**; these lesions may coalesce and form **carbuncles**, which extend into the subcutaneous tissues. Focal infections owe their localization to poor lytic enzyme activity of the organisms.

Classic **toxic shock syndrome** (TSS) is a rare condition that exemplifies focal soft tissue infection with toxic effects. Staphylococcal vaginitis and vaginal discharge usually develop during menstruation in healthy young females using superabsorbent tampons in the vagina; they are accompanied by fever, skin rash, hypotension, and organ failure [1]. These symptoms are attributed to toxic shock syndrome toxin (TSST-1) produced by *S. aureus*. The staphylococci originate not from the tampon but from the fingers that place the tampon in the vagina. Oxygen introduced into the vagina at that time promotes formation of the toxin. Patients who develop TSS have low levels of the antibody to TSST-1 compared to these in normals. Table 1 lists criteria for the diagnosis of TSS.

Folliculitis, furuncles, and carbuncles are treated by local washing and oral cloxacillin. Larger carbuncles may require surgical drainage. To manage TSS the patient is resuscitated with intravenous lactated Ringer's solution, pressors are given if necessary, and visceral organ support is maintained. The tampon is removed by gentle vaginal examination, and a sample of the vaginal discharge is taken for culture. Cloxacillin is given for 10 days by the intravenous route.

Diffuse Nonnecrotizing Infections: Cellulitis

Cellulitis is a spreading infection of the skin and subcutaneous tissues. Erythema, warmth, tenderness, and swelling are typical signs. Nonnecrotizing cellulitis may be mild, severe, high risk, or complicated [2]. Most cellulitis is **mild** and results from infection by group A streptococci or *S. aureus*. Particularly in an extremity, lymphangitis and lymphadenitis are common. With **severe** cellulitis dominant systemic signs (fever, chills, leukocytosis) accompany the typical local signs. Underlying disease such as diabetes or limb ischemia should be sought. The diagnosis is established by needle aspiration, blood tests, cultures, and diagnostic imaging. **High risk** cellulitis occurs typically on the face or extremities or in patients with impaired host defenses. Typical examples include *Haemophilus influenzae* type B (HIB) cellulitis of the cheek in children, erysipelas with its typical painful "peau d'orange" lesions that follow streptococcal sore throat in children and the elderly, and postvenectomy cellulitis caused by non-group A streptococci

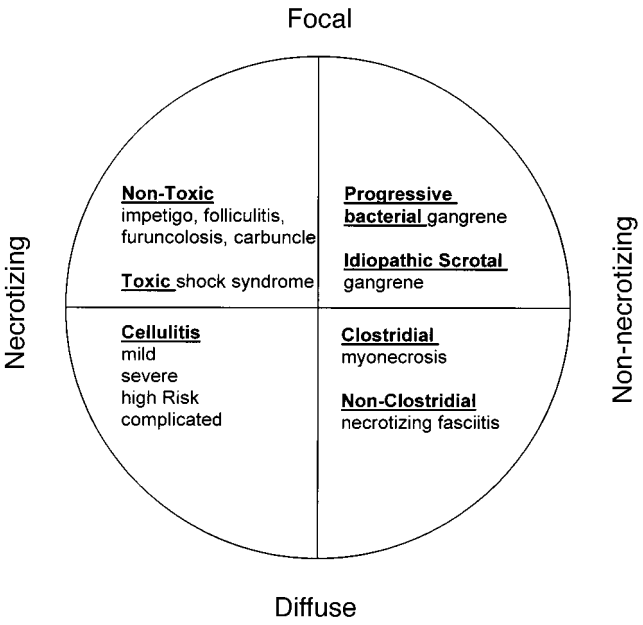

Fig. 1. Classification of soft tissue infections.

Table 1. Criteria for diagnosing toxic shock syndrome.

Hypotension <90 mmHg
Diffuse erythematous rash
Desquamation of rash 1 to 2 weeks later, especially palms, soles
Organ failure
Diarrhea, vomiting, hyperemic mucosa
Myalgias: creatine phosphokinase (×2)[a]
Hepatitis: enzymes (×2)
Renal: serum creatinine (×2)
Coagulopathy: platelets < 100,000/mm^3
Neurologic: disorientation or diminished consciousness

[a] ×2: exceeding twice the upper limit of normal.

in lymphedematous limbs of patients after coronary bypass. Less common is erysipeloid in the fingers of handlers of fish, meat, and poultry; it is caused by the gram-positive bacillus *Erysipelothrix rhusiopathiae*. Deep infections and tenosynovitis are caused by the gram-negative bacillus *Eikenella corrodens* following human bites of the hand. In patients with impaired host resistance high risk cellulitis is caused by unusual organisms such as *Streptococcus pneumoniae* and gram-negative bacteria. With **complicated** cellulitis, deep abscess or infections of bone or joint may develop and should be sought by clinical evaluation, plain radiographs, and bone scans.

With cellulitis, the spread of infection through the tissues is facilitated by toxins and enzymes, especially those produced by *S. aureus* and group A streptococci. Despite the marked inflammatory response, fewer than 30% of cases submitted to deep aspiration yield organisms [3]. This disparity previously was attributed to lymphatic overload or to an exaggerated inflammatory response to denatured protein derived from killed bacteria. More recently it has been ascribed to the dendritic cells of Langerhans in the stratum spinosum of the skin, which release cytokines such as interleukin-1 and tumor necrosis factor when exposed to bacterial components [4]. The cytokines increase phagocytosis and clearance of bacteria, but they also increase the inflammatory response.

Mild cellulitis responds well to oral cloxacillin or cephalexin and local cleansing. Severe cellulitis initially requires intravenous antibiotic therapy in hospital using a combination of antibiotics such as cloxacillin and an aminoglycoside or by monotherapy with cephalosporins, ticarcillin clavulanate, or quinolones. Later, outpatient once-daily intravenous therapy may be employed, using long-acting cephalosporins such as ceftriaxone, or oral quinolones may be given. Some high risk cellulitis is treated with specific antibiotics. Intravenous penicillin or cefuroxime are recommended for HIB cellulitis; and cloxacillin, but not erythromycin, is required for human bites. Other high risk cellulitis is treated as severe cellulitis. Complicated cellulitis usually requires surgical drainage and prolonged antibiotic therapy.

Focal Necrotizing Soft Tissue Infections

Progressive Bacterial Synergistic Gangrene

Progressive bacterial synergistic gangrene, described by Brewer and Meleney in 1926 [5], presents 1 to 2 weeks after injury or surgery as an ulcerating gangrenous lesion that consists of three concentric zones: an outer advancing zone of erythema and swelling, merging with a violaceous mid-zone, and a central ulcerated necrotic zone. Fever and wasting are commonly associated. The lesion is now seen most often in pericolostomy infections and decubitus ulcers. Meleney determined that it results from synergy between a microaerophilic nonhemolytic streptococcus and hemolytic *S. aureus* [6] or a gram-negative bacillus (often *Proteus* sp.). As indicated by its name, the lesion spreads rapidly and progressively in ever-widening circles unless it is treated effectively.

Idiopathic Scrotal (Fournier's) Gangrene

Idiopathic scrotal gangrene refers to the condition described by Fournier in 1883 [7] in five patients with fever and scrotal edema that progressed to gangrene within 24 to 30 hours. The slough then separated rapidly from the testes. Despite a thorough search, Fournier could find no underlying local or systemic cause for the lesion [8]. Later Coenen and Przedborski found that anaerobic streptococci were the main causative organisms [9], although secondary overgrowth of gram-negative bacteria may occur later. The name **Fournier's gangrene** is now used for many types of infective scrotal gangrene, especially the perineal form of necrotizing fasciitis [10].

Treatment

Focal necrotizing soft tissue infections are best treated by intravenous antibiotics, wide surgical excision of all involved tissue, and delayed skin grafting. Cloxacillin and an aminoglycoside may be used or a single agent such as a third-generation cephalosporin, ticarcillin/clavulanate, or a quinolone antibiotic. Repeated débridement may be necessary. With classic Fournier's gangrene the testes are typically spared by the disease process but require skin cover when granulations are present.

Diffuse Necrotizing Soft Tissue Infections

Diffuse necrotizing infections are the most treacherous soft tissue infections, particularly because they may masquerade as simple cellulitis, thereby delaying diagnosis and treatment. They include classic gas gangrene, Meleney's hemolytic streptococcocal gangrene, necrotizing fasciitis as described by Wilson, and the gram-negative synergistic necrotizing cellulitis of Stone. Generally one condition cannot be distinguished from another at the time of diagnosis; indeed, lately they have all been referred to by the generic term necrotizing fasciitis. A high index of suspicion is required to ensure prompt recognition and early treatment. Suspicion is heightened when the clinical setting suggests impaired host resistance or underlying conditions such as diabetes mellitus and peripheral vascular disease. The recently described group A streptococcal necrotizing fasciitis with associated toxic shock occurs typically in healthy young subjects.

The earliest clinical clues to recognition of diffuse necrotizing infections are edema out of proportion to skin erythema, gas in the subcutaneous tissues seen on plain radiographs of the part or identified later as clinical crepitus, and the presence of skin vesicles. Lymphangitis and lymphadenitis, commonly associated with nonnecrotizing cellulitis, are usually absent. If the early signs are missed, local skin anesthesia and skin necrosis occur; and systemic progression may present as fever resistant to antibiotic therapy or hypotension. These findings should provoke prompt surgical exploration and administration of broad-spectrum antibiotic therapy. The specific bacteriologic diagnosis may be determined later, but the initial frozen section or Gram stain of the tissues may be helpful for differentiating histotoxic clostridial infection (gas gangrene), with which little inflammatory reaction is present, from the predominantly nonclostridial causes of diffuse necrotizing infection, with which inflammation is prominent [11].

With Inflammation Present

Streptococcal Gangrene. Streptococcal gangrene was first described during the American Civil War but was identified and studied by Meleney in 1924 [12]. He focused on the rapidity of development and evolution, from erythema, fever, and toxicity in 24 hours to vascular thrombosis in the subcutaneous tissues and patchy gangrene of the skin within 4 to 5 days. The causative organism is the group A β-hemolytic *Streptococcus*.

Clostridial Cellulitis. Clostridial cellulitis, usually caused by *Clostridium perfringens*, occurs in diabetics and in patients with ischemic tissue due to peripheral vascular disease. Unlike gas gangrene, it is confined to the subcutaneous tissues and skin and is characterized by a foul-smelling purulent discharge. It also exhibits abundant gas in the subcutaneous tissues. The lesions are painful, but neither the pain nor the toxicity compares with that of gas gangrene. These distinctions were emphasized by Qvist in 1941 [13] but are sometimes forgotten today.

Necrotizing Fasciitis. The term necrotizing fasciitis is now used in a generic sense to include all diffuse necrotizing soft tissue infections except gas gangrene, although it was coined by Wilson to refer to infections characterized by necrosis of the deep layer of superficial fascia with sparing of the deep fascia and muscle [14]. Because the focus of infection is deep in the subcutaneous tissues the clinical onset is typically slower than that of hemolytic streptococcal gangrene and the skin is initially spared. The infection usually results from synergy between gram-positive cocci, such as non-group-A β-hemolytic streptococci, and gram-negative bacilli, particularly *Proteus* sp., *Pseudomonas* sp., or *Enterobacter* sp. Far less common is a pure group A streptococcal infection [15]. Recently, new varieties of necrotizing fasciitis have been described caused by halophilic marine vibrios, especially *Vibrio vulnificus* [16], and by phycomycoses, particularly *Rhizopus arrhizus* [17]. Clinical presentation of *Vibrio* necrotizing fasciitis is similar to that of streptococcal gangrene, occurring in persons with minor wounds exposed to seawater or sustained while cleaning seafood. In contrast, the clinical presentation of phycomycotic necrotizing fasciitis is insidious. With classic necrotizing fasciitis, tissue necrosis and spread of infection along the deep layer of superficial fascia is facilitated by synergy between bacteria and by the enzymes and toxins they produce. An anaerobic environment favors bacterial growth. Skin necrosis occurs late as a result of thrombosis of subcutaneous arteries.

Table 2. Comparing Meleney's streptococcal gangrene, necrotizing fasciitis of Wilson, and group A streptococcal necrotizing fasciitis with toxic shock syndrome.

Parameter	Streptococcal gangrene	Necrotizing fasciitis	Necrotizing fasciitis with toxic shock
Patients	Healthy	Medical problems	Healthy
Organisms	Hemolytic streptococci	Non-group-A streptcocci, gram-negative bacilli, others	Group A streptococci
Pathogenesis	Toxins	Toxins + synergy	Toxins, superantigens
Course	Abrupt and rapid	Slow, insidious	Rapid
Mortality	20%	20%	20–60%

Necrotizing Fasciitis with Toxic Shock Syndrome. Over the last 15 years, a significant increase in cases of necrotizing fasciitis associated with toxic shock syndrome (StrepTSS) has occurred, caused by a highly invasive strain of group A streptococcus [18]. The increase is truly epidemic, and the condition has been dramatized by characterizing these group A streptococci as "flesh-eating bacteria"; but the disease is still uncommon: fewer than 20 cases per 100,000 population. Table 2 summarizes the characteristics of StrepTSS compared with those of streptococcal gangrene and necrotizing fasciitis.

The pathogenesis of this devastating syndrome is especially interesting and is related to the role of streptococcal pyrogenic exotoxins (spe) produced by specific strains of *Streptococcus pyogenes*. These exotoxins include *spe*A found most often in StrepTSS in the United States, and *spe*B and *spe*C seen in StrepTSS in Europe and Canada [19]. Their production is stimulated by M proteins, particularly types 1 and 3 which also decrease phagocytosis of streptococci by polymorphonuclear leukocytes so the bacteria are more invasive. The *spe* exotoxins exert their effects in two ways: They stimulate mononuclear cells, and they act as "superantigens," which interact with T lymphocytes [20].

Mononuclear Effect. The exotoxins stimulate antigen-presenting mononuclear cells (APCs) to produce monokines, such as tumor necrosis factor-α (TNFα), interleukin (IL)-1β, and IL-6. In

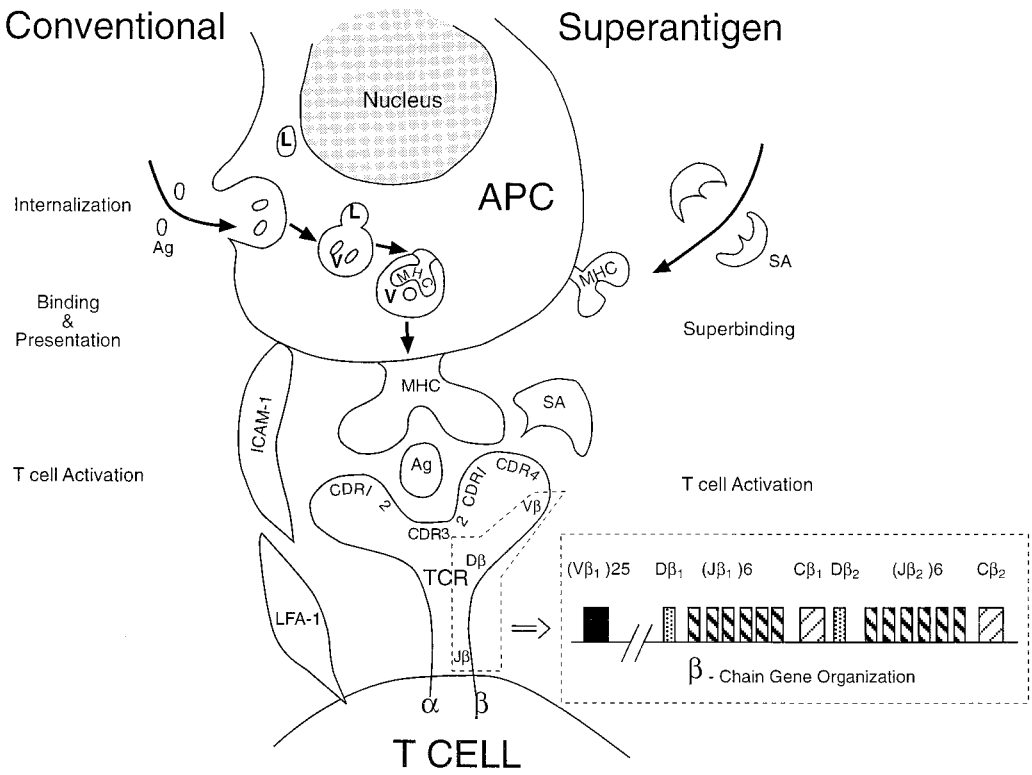

Fig. 2. Activation of T lymphocytes by superantigens compared with that by conventional antigens. Ag: antigen; APC: antigen-presenting (mononuclear) cell; L: lysosome; V: vesicle; MHC: major histocompatibility class II molecule; SA: superantigen; ICAM: intracellular adhesion molecule; LFA: lymphocyte functional associated molecule; CDR: complementarity determining region; TCR: T cell receptor. **Inset**. Gene organization on β-chain of the T cell receptor. V: variable region; C: constant region; D: diversity segment; J: junctional zone.

particular, TNFα is a potent mediator of the fever, shock, and tissue injury typical of StrepTSS.

Superantigen Effect. It has been proposed that the exotoxins act as superantigens, differing from conventional antigens in their interaction with T lymphocytes. Conventional antigens are phagocytosed by monocytes and *preprocessed* into small peptide antigen determinants; they then pass into small vesicles and form complexes with major histocompatibility class II molecules (MHC) in a special *binding* groove; finally the MHC–peptide complexes pass to the surface of the monocyte to be sampled by *specific sequences* of amino acids in the variable regions (V) of the α and β chains of the T cell receptor (TCR). These variable regions are separated from constant regions by diversity segments and junctional zones. Thus T cell activation by conventional antigen is limited by the need for preprocessing, restriction of complexing to a specific binding groove, and the specificity of the TCR and MHC–peptide complexes.

In contrast, superantigens require no preprocessing; and they are less restricted, in that binding occurs on the surface of the MHC molecule rather than in a specific binding groove. Interaction of the superantigen with the T cells is governed solely by the Vβ region of the TCR and does not require complementarity with specific segments of the α and β chains. Thus superantigens can react with *all* T cells expressing Vβ elements (5–20% of T lymphocytes), whereas conventional antigens react with only 0.01% of T lymphocytes. For example, if 2 of the 25 Vβ families can recognize a particular superantigen, 8% of all T cells can be activated by that superantigen. This massive activation results in clonal proliferation of the whole T cell subset and release of an immense load of lymphokines, especially IL-2, TNFβ, and interferon-γ. The magnitude of the response helps explain the increased virulence of group A streptococci in StrepTSS and the severity of the systemic effects. Figure 2 illustrates the differences between conventional and superantigen interactions; the inset highlights the organization of the β-chain of the TCR.

In addition, activation of T cells by superantigen is aided by APC-associated co-stimulatory molecules such as the B7 and the intracellular adhesion molecule (ICAM-1), with their respective ligands on the T cell, CD28, and lymphocyte functional associated (LFA) molecule-1 (Fig. 2) [21]. In turn, T cell interaction brings the ligands and co-stimulatory molecules closer together, facilitating transduction of signals that direct T cell activation and clonal proliferation.

Gram-Negative Bacterial Synergistic Necrotizing Cellulitis. There is a form of necrotizing infection caused by synergy between gram-negative aerobic and anaerobic bacteria; it is localized mainly in the deep fascia and therefore presents even more slowly than classic necrotizing fasciitis [22]. Usually *Bacteroides fragilis* or peptostreptococci and Enterobacteriaceae are involved. The deep location not only delays presentation but favors proliferation of anaerobic bacteria. Extensive necrosis that includes the underlying muscle has usually occurred by the time focal patchy necrosis of the skin signals the presence of more superficial infection. Today the perineal form of this condition is generally called Fournier's gangrene [10], though it differs from idiopathic scrotal gangrene in etiology, extent, and clinical presentation. In particular, it is polymicrobic from the outset, frequently extends onto the abdominal wall, and presents insidiously.

Streptococcal Myositis. Streptococcal myositis is an interesting variant of necrotizing fasciitis in which muscle inflammation occurs without necrosis [23]. Most cases present like streptococcal gangrene with pain and bacteremia, although gas in the tissues is uncommon. The usual bacteria are group A streptococci associated with anaerobic streptococci or occasionally with *S. aureus* or gram-negative bacilli.

True muscle necrosis occurs in patients with streptococcal necrotizing fasciitis with toxic shock syndrome. The condition known as gangrenous streptococcal myositis [24] is similar to clostridial myonecrosis but differs in the absence of gas in the tissues. It may be caused by hematogenous spread of the bacteria to muscle primed for necrosis by coincidental prodromal viral inflammation. A protease has also been identified that causes muscle necrosis in this syndrome.

Treatment. Nonclostridial necrotizing infections are best treated by resuscitation, systemic broad-spectrum antibiotics, and early exploration and radical débridement. With classic necrotizing fasciitis, the skin can often be spared if débridement is prompt, but planned reexploration for further débridement is advisable. The gray-white necrotic fascia readily allows dissection in that plane, accounting for the undermining typical of this lesion. Muscle débridement is unnecessary for classic necrotizing fasciitis but may be required for gram-negative bacterial synergistic cellulitis and in StrepTSS. After 48 hours the antibiotic regimen may be revised on the basis of clinical progress and results of tissue culture. Broad-spectrum coverage is generally maintained in more severe cases, but specific therapy for *B. fragilis* should be prolonged only when that organism is present, particularly in gram-negative synergistic necrotizing cellulitis. With StrepTSS, intravenous high-dose penicillin and clindamycin are now recommended to suppress exotoxin production by killing the streptococci and impairing M protein synthesis; human immunoglobulin [25] is given to neutralize the exotoxin already present. Intravenous amphotericin B may be prescribed if the presence of hyphae on Gram stain or on histologic section suggests phycomycotic necrotizing fasciitis. Delayed skin grafting is necessary for most severe cases of nonclostridial infection.

Inflammation Absent

Clostridial Myonecrosis. The clinical picture of clostridial myonecrosis, commonly called gas gangrene, is well known. It was recognized by Hippocrates and Celsus, then forgotten, and later redescribed by Quesnay in 1745 [26]. The four main characteristics are (1) an abrupt onset in young men following trauma or in the elderly with diabetes and peripheral vascular disease; (2) intense wound pain; (3) marked swelling; and (3) severe toxicity manifested by mental confusion and tachycardia out of proportion to the rise in body temperature. The typical wound discharge is watery and has a mousy odor. Skin vesicles, gangrene, and tissue gas are late signs. The details of presentation depend on the bacteria involved [27]. The usual causative organisms are *Clostridium perfringens* (70% of cases), *Clostridium novyi* (40%), and *Clostridium septicum* (10%). Rarely *Clostridium histolyticum*, *Clostridium bifermentans*, or *Clostridium fallax* is the cause.

The hallmarks of gas gangrene—muscle necrosis and systemic toxicity—are caused by exotoxins produced by the bacteria. The most important are the α-toxin lecithinase, which destroys cell membranes and causes hemolysis, and the θ-toxin, which causes hemolysis, muscle necrosis, and cardiac toxicity. *C. perfringens* produces at least 12 exotoxins, *C. novyi* 8, and *C. septicum* 4. The low redox potential in untidy or deep traumatic wounds and in the subcutaneous fat of ischemic limbs favors conversion of clostridial spores to vegetative forms that produce the exotoxins.

Treatment. Early, wide débridement of necrotic muscle and other tissue is of critical importance in arresting gas gangrene. High-dose penicillin is required to reduce toxicity and should be started once the Gram stain has revealed the typical spore-bearing rods in a patient with clinical features of gas gangrene. As with necrotizing fasciitis, it is preferable to start antibiotic therapy with empirically chosen broad-spectrum intravenous antibiotics such as penicillin and an aminoglycoside, a third-generation cephalosporin, ticarcillin/clavulanate, or a quinolone and to revise therapy after 48 hours.

The use of hyperbaric oxygen therapy in patients with gas gangrene is controversial, though it is of more specific value than when used for necrotizing fasciitis. In special centers it clearly adds to the effect of treatment by antibiotics and surgical débridement [28]. In experimental animals production of the α-toxin ceases at PO_2 240 mmHg [29], and survival is improved [30]. Hyperbaric oxygen, however, cannot take the place of wide surgical débridement.

Conclusions

Soft tissue infections encompass a wide spectrum of diseases that vary in severity from the trivial to the life-threatening. The classification provided simplifies the approach and facilitates rapid diagnosis and treatment. The focal nonnecrotizing infections that involve the skin and adenexae are readily diagnosed on sight and are easily treated by proper hygiene, local measures, and antibiotics. Toxic shock syndrome carries a low mortality of 5% only when managed by prompt diagnosis, aggressive systemic treatment, and organ support. A systematic approach to cellulitis that adds needle aspiration and culture, blood testing, and diagnostic imaging to thoughtful clinical evaluation avoids misdiagnosis and allows appropriate categorization by risk, setting the stage for optimal treatment. Focal necrotizing infections are uncommon but require aggressive treatment to reduce morbidity and avoid mortality.

The diffuse necrotizing infections are the real threat to life and limb. They are increasing in frequency; and with the emergence of virulent group A streptococci that cause necrotizing fasciitis with toxic shock they have become more complex. An aggressive unified approach to diagnosis and initial treatment is the key to simplified, successful management. Careful monitoring and modification of therapy based on the initial pathologic findings and on a clear understanding of the natural history of the several variants of clostridial (inflammation absent) and nonclostridial (inflammation present) categories gives the best result. Specific therapies related to the pathogenesis of StrepTSS are beginning to emerge and may reduce the high morbidity and mortality of this condition further.

Résumé

Les infections des parties molles variant dans leur nature et dans leur sévérité, et leur classification n'est pas claire. Une thérapeutique bien visée nécessite l'identification et le traitement rapides des infections nécrosantes diffuses en raison de la menace pour le pronostic vital qu'elles représentent. Cette revue classe les infections selon leur localisation et selon la présence ou l'absence de tissus nécrosés. La plupart des infections focales, non-nécrosées, débutent dans la peau ou ses annexes et sont facilement reconnues et traitées par des mesures locales. La cellulite, l'infection diffuse, non-nécrosante la plus répandue, doit étre classée pour la gravité allant de l'infection peu sévère à l'infection sévère et compliquée, et ensuite traitée par des antibiotiques oraux ou systémiques. Les infections focales nécrosantes sont peu fréquentes. Elles sont volontiers diagnostiquées cliniquement, traitées par excision locale et des antibiotiques par voie systémique. L'infection nécrosante diffuse peut simuler d'autres affections rendant son diagnostic difficile et retardé. Un oedéme sans rapport avec l'érythème. Ia présence de gaz sous-cutané ainsi que des vésicules au niveau de la peau sont des indications importantes. Un traitement chirurgical vigoureux, répété, et des antibiotiques intraveineux à large spectre, éventuellement réadaptés après 48 heures, sont les meilleurs garants d'une thérapeutique efficace.

Resumen

Las infecciones de los tejidos blandos exhiben una amplia variación en cuanto a su naturaleza y su gravedad. Además, su nomenclatura es confusa. Una estrategia de manejo bien definida debe permitir la identificación rápida y el tratamiento inmediato de las facsitis necrotizantes difusas, por cuanto éstas representan real amenaza de muerte. La presente revisión establece una clasificación de las infecciones de los tejidos blandos según el grado de localización y la presencia de necrosis tisular. La mayoría de las infecciones focales no necrotizantes se inician en la piel o en sus anexos, y son fácilmente reconocibles y tratables con medidas locales. Los pacientes con celulitis, que es la más común de las infecciones difusas no necrotizantes, deben ser estratificados por categorias leves a severas y complicadas, y luego tratados con antibióticos orales o sistémicos. Las infecciones focales necrotizantes son relativamente raras, pero fácilmente diagnosticables a la inspección visual y pueden ser manejadas en forma efectiva con desbridación local y antibióticos sistémicos. En contraste, las infecciones necrotizantes difusas pueden aparecer enmascaradas en diversas formas, lo cual demora el diagnóstico y el tratamiento. El edema desproporcionado ante el eritema, el gas subcutáneo y las vesículas cutáneas son marcadores importantes; el desbridamiento agresivo y secuencial y los antibióticos de amplio espectro por vía intravenosa, con revisión cada 48 horas, constituyen la mejor estrategia de tratamiento.

References

1. Shanks, K.N., Schmid, G.P., Dan, B.B., Blum, D., Guidotti, R.J., Hargrett, N.T., Anderson, R.L., Hill, D.L., Broome, C.V., Band, J.D., Fraser, D.W.: Toxic shock syndrome in menstruating women: association with tampon use and *Staphylococcus aureus* and clinical features in 52 cases. N. Engl. J. Med. *303*:1436, 1980
2. Lewis, R.T.: Approach to the patient with cellulitis and other soft tissue infections. In Textbook of Internal Medicine, W.M. Kelley, editor. Philadelphia, Lippincott-Raven, 1997, pp. 1550–1555
3. Hook, E.W., III, Hooton, T.M., Horton, C.A., Coyle, M.B., Ramsey, P.G., Turck, M.: Microbiologic evaluation of cutaneous cellulitis in adults. Arch. Intern. Med. *146*:295, 1986
4. Streilein, J.W.: Skin-associated lymphoid tissue (SALT): origins and functions. J. Invest. Dermatol. *80*(Suppl.):12, 1983
5. Brewer, G.E., Meleney, F.L.: Progressive gangrenous infection of the skin and subcutaneous tissues, following operation for acute appendicitis. Ann. Surg. *84*:438, 1926
6. Meleney, F.L.: Bacterial synergism in disease processes with confirmation of the synergistic bacterial etiology of a certain type of progressive gangrene of the abdominal wall. Ann. Surg. *94*:961, 1931
7. Fournier, J.A.: Gangrène foudroyante de la verge. Sem. Med. *3*:345, 1883
8. Fournier, J.A.: Etude clinique de la gangrène foudroyante de la verge. Sem. Med. *4*:69, 1884
9. Coenen, T.E., Przedborski, J.: Die gangrän des penis und scrotums. Beitr. Klin. Chir. *75*:136, 1911
10. Rouse, T.M., Malangoni, M.A., Schulte, W.J.: Necrotizing fasciitis: a preventable disaster. Surgery *92*:765, 1982
11. Dellinger, E.P.: Severe necrotizing soft tissue infections: multiple disease entities requiring a common approach. J.A.M.A. *246*:1717, 1981
12. Meleney, F.L.: Hemolytic streptococcus gangrene. Arch. Surg. *9*:317, 1924
13. Qvist, G.: Anerobic cellulitis and gas gangrene. B.M.J. *2*:217, 1941
14. Wilson, B.: Necrotizing fasciitis. Am. Surg. *18*:416, 1952
15. Giuliano, A., Lewis, F., Hadley, K., Blaisdell, F.W.: Bacteriology of necrotizing fasciitis. Am. J. Surg. *134*:52, 1977
16. Howard, R.J., Pesa, M.E., Brennaman, B.H., Ramphal, R.: Necrotizing soft tissue infections caused by marine vibrios. Surgery *98*:126, 1985
17. Patino, J.F., Castro, D., Valencia, A., Morales, P.: necrotizing soft-tissue lesions after a volcanic cataclysm. World J. Surg. *15*:240, 1991
18. Schwartz, B., Facklam, R.R., Brieman, R.F.: Changing epidemiology of group A streptococcal infection in the USA. Lancet *336*:1167, 1990
19. Stevens, D.L., Tanner, M.H., Winship, J., Swartz, R., Ries, K.M., Schlievert, P.M., Kaplan, E.: Severe group A streptococcal infections associated with a toxic shock-like syndrome and scarlet fever toxin A. N. Engl. J. Med. *321*:1, 1989
20. Wanatabe-Ohnishi, R.D.E., Low, A., McGeer, D.L., Stevens, D.L., Schilivert, P.M., Newton, D., Schwartz, B., Kreiswirth, B., Kotb, M.: Selective depletion of Vβ bearing T cells in patients with invasive group A streptococcal disease and streptococcal toxic shock syndrome: implications for a novel superantigen. J. Infect. Dis. *171*:74, 1995
21. Sigal, L.H., Yacov, R.: Ontogeny and differentiation of B lymphocytes. In Immunology and Inflammation. New York, McGraw-Hill, 1994, pp. 87–112
22. Stone, H.H., Martin, J.D., Jr.: Synergistic necrotizing cellulitis. Ann. Surg. *129*:702, 1975
23. MacLennan, J.D.: Streptococcal infection of muscle. Lancet *1*:582, 1943
24. Yoder, E.L., Mendez, J., Khatib, R.: Spontaneous gangrenous myositis induced by *Streptococcus pyogenes*: case report and review of the literature. Rev. Infect. Dis. *9*:382, 1987
25. Takei, S., Arora, Y.K., Walker, S.M.: Intravenous immunoglobulin contains antibodies inhibitory to the activation of T cells by staphylococcal toxin superantigens. J. Clin. Invest. *91*:602, 1992
26. Altemeier, W.A.: Gas gangrene. Int. Abstr. Surg. *84*:507, 1947
27. MacLennan, J.D.: Histotoxic clostridial infections in man. Bacteriol. Rev. *26*:177, 1962
28. Hitchcock, C.R., Demello, F.J., Haglin, J.J.: Gangrene infection, new approaches to an old disease. Surg. Clin. North Am. *55*:1403, 1975
29. Brummelkamp, W.H.: Considerations on hyperbaric oxygen therapy at three atmospheres absolute for clostridial infections type *welchii*. Ann. N.Y. Acad. Sci. *117*:688, 1965
30. Demello, F.J., Haglin, J.J., Hitchcock, C.R.: Comparative study on experimental *Clostridium perfringens* infection in dogs treated with antibiotics, surgery, and hyperbaric oxygen. Surgery *73*:936, 1965