MINI-SYMPOSIUM

What's New in Soft Tissue Infections?

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INTRODUCTION

Soft tissue infections can be classified in (1) bacterial infections of the skin, (2) infections of the subcutaneous tissue, (3) infections of the fascial cleft, (4) infections of the muscle, (5) unusual bacterial infections of the skin, (6) fungal infections of soft tissue, (7) viral infections of the skin, and (8) protozoan and helminth infections.

A MEDLINE search (1995–1999) was performed using the keywords erysipela, cellulitis, erythrasma, folliculitis, furunculosis, carbuncle, hidradenitis suppurative, soft tissue infection and HIV, leg ulcer infection, infective bites, streptococcal necrotizing fasciitis, necrotizing fasciitis, pyomyositis, psoas abscess, rectus sheath abscess, clostridial myonecrosis, nonclostridial myonecrosis, and superficial fungal infections together with surgery. Furthermore, randomized clinical studies in soft tissue infections were retrieved.

SOFT TISSUE INFECTION AND HUMAN IMMUNODEFICIENCY VIRUS

In recent years, especially with the advent of acquired immunodeficiency syndrome, new skin disorders associated with systemic disease have been described. Pruritic papules of HIV infection, bacillary angiomatosis, and toxic strep syndrome, which is caused by group A streptococcal infection and may lead to multiorgan system dysfunction have been described in a review.[1] In a retrospective analysis of 900 consecutive admissions to a regional infection unit, more patients were admitted with soft tissue infections when compared with a study done 10 years earlier. Human-immunodeficiency-virus–related conditions contributed 4% of the admissions and 29% of the mortality.[2] In people abusing drugs parenterally, surveillance for HIV is important. Five (14%) of 35 patients tested for HIV had a positive result in a study population of 77 patients with an abscess at the site of injection caused by parenteral abuse of drugs.[3] Magnetic resonance imaging (MRI) may be an ideal technique for displaying soft-tissue abnormalities produced by infectious process.[4,5] The clinical presentation, microbiology, and treatment outcome were reported by Bergstein et al.[6] Only 28 (42%) of 66 patients were febrile, 36 (54%) had leukocytosis, and 31 (47%) had wound fluctuance. Wound cultures grew predominantly anaerobes and facultative gram-positive cocci. Simple incision and antibiotic coverage for gram-positive and anaerobic bacteria were adequate treatment.

Upper extremity infections in AIDS patients receive more attention.[7] In a study from Switzerland in patients with upper extremity infections, *Staphylococcus* and *Streptococcus* species were recovered from pus; anaerobic bacteria and yeasts were of minor importance.[8] In emergency hand service, in general, a larger percentage of HIV-positive persons may be found than HIV-negative persons seen for infection.[9] Recent reports indicate, however, that non-*typhi Salmonella* and *Candida* species may play a pivotal role in patients with HIV.[10,11]

LEG ULCER INFECTIONS, INCLUDING DIABETIC FOOT INFECTION

Limb- or life-threatening complications in patients with diabetes can be prevented with an integrated, multidisciplinary approach. If ulceration occurs, removal of pressure from the site of the ulcer and careful management of the wound will allow healing in most cases. Failure of healing may be caused by arterial insufficiency or infection, which may ask for surgical intervention and antibiotic therapy.[12,13] Foot infections may be treated empirically with antibiotics; testing may result in little improvement in outcome. A 10-week course of culture-guided oral antibiotic therapy after surgical debridement may be as effective and less costly than other approaches.[14] The necessity of bacteriological testing may be disputed; however, in patients treated for ischemic ulceration, the healing of skin grafts was better in patients with less than 50,000 bacteria/cm^2.[15] In the case of an enlarging venous leg ulcer, one should also consider unusual pathogens, such as *Pseudomonas aeruginosa*.[16] For the planning of surgical intervention, MRI was considered valuable in determining the presence and extent of infection (specificity, 77%; positive predictive

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  0149-7944/99/$20.00
PII S0149-7944(99)00164-6

value, 77%; sensitivity, 91%; negative predictive value, 91%).[17] Surgical treatment may consist of ligation of incompetent perforating veins with a success rate of more than 90%.[18] Preliminary results are available on the effect of minimal invasive surgery. Although the clinical situation improved after subfascial endoscopic perforator surgery in 88% of the cases and recurrence only occurred in 3%, a significant number of complications developed, such as wound infections, superficial thrombophlebitis, cellulitis, and saphenous neuralgia. Prospective evaluation of long-term results is warranted before a general recommendation of this technique can be given.[19,20] In a randomized trial, 19 patients were treated by open exploration and 20 patients by endoscopic exploration. The incidence of wound infections after open exploration was 53%, compared with 0% in the endoscopic group. The results of ulcer healing were 90% in the open group and 85% in the endoscopic group. The authors concluded that endoscopic division of incompetent perforating veins is equally effective as open surgical exploration, but it may lead to fewer wound complications.[21] It may be fair to say then that a wound infection rate of 53% in a clean operation should be reason enough to evaluate the open surgical procedure, so that more surgical treatment of incompetent perforating veins may be done in outpatient settings. Lateral venous ulcers may be caused by a short saphenous vein insufficiency, and ligation of the vein may be the optimal treatment.[22] One report indicates the effect of the granulocytecolony-stimulating factor (G-CSF) in diabetic foot infection. It has been demonstrated that neutrophil superoxide production is impaired in diabetes and may thus be relevant in diabetic foot infection. Granulocyte colony-stimulating factor–treated patients (n = 20) were associated with an earlier eradication of pathogens from infected ulcers, a quicker resolution of cellulitis, a shorter hospital stay, a shorter duration of intravenous antibiotic treatment, and a higher neutrophil superoxide production. Although an improved clinical outcome is reported in this study, more clinical studies with a larger patient population are necessary before the treatment with G-CSF should be recommended.[23] Instead of parenteral administration of antibiotics, oral administration may be preferred. Serum and tissue concentration of ofloxacin exceeded the minimal inhibitory concentration (MIC 90) of commonly involved pathogens.[24] Further improvement in therapy of diabetic foot infections and venous ulcers may be achieved by a diagnostic and therapeutic algorithm. In a prospective study of 293 patients seen in a regional wound care center, the overall healing rate of diabetic foot infections was 68% and 74% in venous ulcer. Mean treatment time was 4.5 months. Relevant prognostic factors were localization of ulcer, ischemia, age, and compliance. Ulcer grading had no effect on healing.[25] In a retrospective analysis of 360 patients with diabetes (wound depth, sensory neuropathy, vascular insufficiency, infection), however, outcomes deteriorated with increasing grade and stage of wounds using the University of Texas Wound Classification System.[26]

GROUP A *STREPTOCOCCUS* INFECTIONS

Reports indicate an increasing incidence of group A *Streptococcus* infections without obvious explanation. Streptococcal pyrogenic exotoxins that were characterized as superantigens are considered as major triggers initiating the immune response. Three types of invasive infections were classified as follows.

- Necrotizing fasciitis.
- Streptococcal toxic shock syndrome.
- Myositis.[27]

Necrotizing Fasciitis

Necrotizing fasciitis is a rare and potentially fatal infection characterized by rapid and progressive involvement of the fascia and subcutaneous tissue. It can occur in any region of the body; however, the abdominal wall, perineum, and extremities are the most common sites of infection. Early diagnosis, aggressive initial debridement followed by planned redebridements, and nutritional and antibiotic support remain the mainstay of therapy. Mortality rate may be as high as 76%.[28] Necrotizing fasciitis may occur after a variety of clinical situations, such as paronychia,[29] pilonidal sinus disease,[30] inguinal herniorrhaphy,[31] and perforated colonic carcinoma.[32] Even in outpatient procedures, such as liposuction, however, which is considered to be a safe and effective procedure, necrotizing fasciitis has been reported as a complication.[33,34]

Increasing evidence exists that necrotizing fasciitis may also occur after laparoscopic operations.[35–38] The clinical diagnosis of necrotizing fasciitis may often be unreliable, and intensive care therapy may be of limited value.[39–41] None of the clinical factors, including temperature, heart rate, systolic blood pressure, white blood cell count, base deficit, albumin level, PO2, or APACHE II score could predict mortality or the requirement for extensive debridement.[42] Polymicrobial necrotizing fasciitis is usually caused by enteric pathogens, whereas monomicrobial necrotizing fasciitis is usually caused by skin flora. In rare cases, *Aspergillus* species, unencapsulated *Haemophilus influenzae* infection may cause necrotizing fasciitis.[43,44] Platelet count, PT, and PTT are readily available parameters that may provide substantial information on the outcome of necrotizing fasciitis. Surgery seemed to correct coagulopathies. In the case of alteration of platelet counts, PT and PTT together, however, no difference existed between surgical and medical treatment with regard to mortality.[45] The authors suggest that in this case surgery may not be necessary. Early recognition and expeditious initial wide excision and debridement serve to decrease morbidity and mortality. Patients with a delay in therapy or inadequate preliminary therapy had a higher mortality (38%) than patients receiving aggressive therapy from the onset (mortality, 4.2%).[46] For wound coverage in abdominal wall reconstruction, the use of tissue expanders and other methods of covering the abdomen may be used.[47–50]

Streptococcal Toxic Shock Syndrome

Only a few reports are available regarding streptococcal toxic shock syndrome. Patients present with fever, leukocytosis, severe pain, and develop rapidly shock and organ failure.[51] Cause of this newly defined entity may be minor injury, crural ulcer, or abscess after intravenous immunoglobulins injection.[52] The treatment consists of aggressive surgery and antibiotics. In a literature review, it was observed that 2 patients have been successfully treated with intravenous immunoglobulins.[53]

Myositis

Clostridium myonecrosis infection (gas gangrene) is an uncommon sequelae of traumatic injury. Infection with *Clostridium perfringens* in devitalized tissue is the most common cause. This disease may be caused by cholecystectomy,[54] laparoscopic cholecystectomy,[55] or malignant or

hematologic disease[56] presenting as nontraumatic gas gangrene.[57]

The successful immune response in patients surviving myonecrosis has been associated with detectable IgG to alpha toxin.[58] Therapy consists of aggressive surgery and antibiotic treatment. The addition of hyperbaric oxygen therapy has been shown to have a synergistic effect in reducing morbidity and mortality in animal models.[59] Only a few reports are available, however, on the clinical efficacy of hyperbaric surgery.[60] Further studies are warranted to establish the role of hyperbaric oxygen.

Psoas Abscess

Psoas abscess is often complicated by delayed diagnosis or misdiagnosis caused by variable and nonspecific symptoms. Fever, flank pain, and limitation of hip movement may be observed only in 30% of patients.[61] Patients may be admitted with signs of sepsis, peritonitis, or shock. In a number of cases, psoas abscess was observed as a complication of Crohn's disease.[62] Psoas abscess may occur after ileo-anal pouch surgery[63,64] or as an early complication of extracorporeal shock-wave lithotripsy.[65] The isolated pathogens are mostly of intra-abdominal origin; however, *Streptococcus pneumoniae* infection and tuberculosis may cause psoas abscess.[66,67] The diagnosis is made by ultrasound, computed tomography (CT) scan, or surgery. The treatment options are surgery or percutaneous drainage.[68]

Cellulitis

Cellulitis may occur after tissue expansion in plastic surgery[69] or after breast conservation therapy in breast cancer.[70] A definite pathogen in cellulitis after breast conservation therapy has not been isolated. Local inflammatory signs will clear after antibiotic treatment; the possibility of persistent tumor recurrence should be considered. In the pathogenesis, lymph stasis is probably involved.[71] In late cellulitis factors related to vascular and skin integrity modifications, for example, surgery and radiotherapy may show a causal relationship.[72] Risk factors are obesity and older age. Cefazolin prophylaxis reduced the surgical site infection rate after breast surgery from 10.8% or 2.6% to 0.9%.[73] Diagnosis of soft tissue infection, for example, cellulitis, may be difficult. Recent reports support CT and MRI for the delineation of soft tissue infections.[74] Cellulitis may also occur after varicose vein surgery, especially after minimally invasive endoscopic Linton operations.[75,76] Cellulitis was also reported after ventral hernia repair and in burn scars.[77,78] In most cases, cellulitis is treated with antibiotics covering gram-positive pathogens. Regarding the antibiotic treatment of soft tissue infections, surprisingly few clinical randomized studies are reported.[79] In a study of 194 patients admitted to an emergency department, no difference existed in outcome determined by the ratio of the involved infected area on initial and last treatment and the frequency of failure of treatment between ceftriaxone and cefazoline treatment.[80]

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