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REVIEW ARTICLE

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Microbiology and management of myositis

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Abstract This review summarizes the microbiology, management, and prevention of myositis. Muscular infections frequently occur in areas of the body that have been compromised or injured by a foreign body, trauma, ischemia, injection of illicit drug, malignancy, or surgery. These infections can develop very rapidly to life-threatening systemic illness. The predominant pathogens are *Staphylococcus aureus*, Group A streptococci (GAS), gram-negative aerobic and facultative bacilli, and the indigenous aerobic and anaerobic cutaneous and mucous membranes local microflora. Pyogenic myositis can be classified into either GAS necrotizing myositis, clostridial myonecrosis (gas gangrene), or nonclostridial (crepitant) myositis. Intensive surgical and medical therapy that includes the administration of intravenous fluids and antimicrobial therapy is an essential element in management of muscle infection.

Résumé Cette révision résume la microbiologie, la gestion et la prévention des myosites. Les infections musculaires se produisent fréquemment dans une région du corps qui a été concernée ou blessée par corps étranger, traumatisme, ischémie, injection de drogue illicite, malignité ou chirurgie. Ces infections peuvent se développer très rapidement en maladie systémique potentiellement mortelle. Les pathogènes prédominants sont des staphylocoques dorés, des streptocoques de groupe A (GAS), des aérobies Gram-négatif et la flore microscopique locale aérobie et anaérobie de la peau et des muqueuses. Les myosites pyogènes peuvent être classées soit dans les myosites nécrosantes du GAS, les gangrènes gazeuses ou les myonécroses nonclostridiennes. La thérapie chirurgicale et médicale intensive qui inclut l'administration de fluides intraveineux et la thérapie anti-microbienne sont des éléments essentiels dans la gestion des infections musculaires.

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Introduction

Myositis is a rare infection that may occasionally lead to serious and potentially life-threatening local and systemic complications. The infection can progress rapidly, and early recognition and proper medical and surgical management is, therefore, the cornerstone of therapy. Myositis often occurs in body sites that have been compromised or injured by a foreign body, trauma, ischemia, malignancy, or surgery. The predominant pathogens are *Staphylococcus aureus* Group A streptococci (GAS), gram-negative aerobic and facultative bacilli, and the indigenous aerobic and anaerobic cutaneous and mucous membranes local microflora. Anatomic sites that are subject to fecal or oral contamination are particularly at risk [10]. Muscle infections complicating surgery or trauma are often polymicrobial aerobic and anaerobic synergistic infections. Some of the clues to the anaerobic origin of such infections are putrid discharge, gas production, and extensive tissue necrosis with a tendency to burrow through subcutaneous and fascial planes [9]. This review describes the microbiology and management of myositis.

Microbiology

Staphylococcus aureus is the predominant cause of tropical and nontropical infection [5]. Other pathogens are GAS nd other groups (B, C, and G), as well as *Streptococcus pneumoniae* and *Streptococcus anginosus*. Gram-negative aerobic and facultatives have also been rarely recovered. These include *Enterobacteriaceae*, *Yersinia enterocolitica*, *Salmonella* spp., *Pseudomonas* spp., *Haemophilus influenzae*, *Neisseria gonorrhoeae*, and *Aeromonas* spp. (Table 1)

Table 1 Bacterial etiology of myositis

Staphylococcus aureus
Groups A, B, C and G streptococci
Enterobacteriaceae
Yersinia enterocolitica
Pseudomonas spp.
Aeromonas spp.
Clostridium spp. (especially *Clostridium perfringens*)
Peptostreptococcus spp.
Bacteroides spp.

Anaerobic bacteria including *Bacteroides*, *Fusobacterium*, *Clostridium*, and *Peptostreptococcus* spp. have also been recovered in adults [4] and children [2] in studies where proper methods for their isolation were employed . Cryptococcal myositis has been rarely reported in patients with human immunodeficiency virus (HIV) infection [15].

Pyogenic myositis can be classified into these major groups according to the organisms recovered [5, 9, 10]:

1. GAS necrotizing myositis
2. Clostridial myonecrosis (gas gangrene)
3. Nonclostridial (crepitant) myositis

Clostridium perfringens accounts for 80–95% of cases of gas gangrene, *Clostridium novyi* for 10–40%, and *Clostridium septicum* for 5–15%. Rarely, other clostridial species can be isolated: *Clostridium bifermentans*, *Clostridium fallax*, and *Clostridium histolyticum*. Other organisms such as *Escherichia coli*, *Enterococcus* and *Enterobacter* spp. can also be recovered with these clostridia.

Nonclostridial myositis can be divided into these subgroups [5, 9, 10]:

1. Anaerobic streptococcal myonecrosis—which is a mixed infection of GAS or *S. aureus* with *Peptostreptococcus* spp.
2. Synergistic nonclostridial anaerobic myonecrosis—due to polymicrobial flora
3. Infected vascular gangrene—due to *Bacteroides* spp. and other anaerobes plus *Proteus* spp.
4. *Aeromonas hydrophila*—myonecrosis
5. Psoas abscess—generally due to *S. aureus* or polymicrobial aerobic–anaerobic flora

Pathogenesis

Muscular infections frequently occur in areas of the body that have been compromised or injured by a foreign body, trauma, ischemia, injection of illicit drug, malignancy, or surgery [11]. Because the indigenous local bacterial flora is often responsible for these infections, anatomic sites that are subject to fecal or oral contamination are particularly at risk. These include wounds associated with surgery of the intestine or pelvic tract, human bites, decubitus ulcers in the perineal area, pilonidal cysts, omphalitis, and cellulitis around the fetal monitoring site [16].

Infectious myositis caused by bacteria can invade from contiguous sites such as skin and subcutaneous abscesses, ulcers, penetrating wounds, and osteomyelitis or through hematogenous spread. Trauma is a common predisposing cause in children [2, 3, 9]. Vascular insufficiency in an extremity can also facilitate the process. However, primary muscle abscess can also occur in the absence of a predisposing site of infection [12]. No conclusive evidence exists that relates tropical pyomyositis causality to predisposing conditions unique to the tropics (i.e., filariasis, malaria, arbovirus). However, about two thirds of tropical myositis cases have a predisposing condition that include diabetes, alcoholism, corticosteroid therapy, immunosuppressive therapy, hematological illnesses, and HIV infection [5].

Pyomyositis is an increasingly recognized infection of the striated muscles in HIV-infected patients. It affects almost exclusively males with advanced HIV infection. Most cases are due to *S. aureus* (67%). These infections add a significant morbidity and mortality in affected individuals [21]. The increased susceptibility of HIV patients to pyomyositis is believed to be due to the combination of the underlying cell-mediated immunodeficiency, defective neutrophils activity, and the potential of muscle injury (HIV myopathy, zidovudine-associated mitochondrial myopathy, and concomitant bacterial infection).

Clostridial myonecrosis usually follows muscle injury and contamination by dirt or during surgery. Contamination of the muscle can occur as a result of compound fracture, penetrating war wounds [14], surgical wounds—especially following bowel or biliary tract surgery, arterial insufficiency of an extremity [6], and, rarely, after parenteral injection of medication, especially epinephrine in oil.

Psoas abscess generally develops as a result of spread from an adjacent structure, either as an extension of intra-abdominal infection (appendicitis, diverticulitis, Crohn's disease), perinephric abscess, or infected retroperitoneal hematoma [1]. It can also originate from vertebral tuberculosis or *S. aureus* osteomyelitis. Osteomyelitis of the ilium or septic arthritis of the sacroiliac joint can produce iliaci or psoas abscess.

Diagnosis

The recovery of fastidious organisms depends on employment of proper methods for collection, transportation of specimen, and cultivation of organisms. Since many potential pathogens are part of the normal skin or mucous membrane flora, specimens should be obtained using methods of collection that will bypass the normal skin and mucous membrane flora. Therefore, disinfecting the skin followed by deep-tissue or surgically obtained aspirates will yield reliable specimens [20].

Radiological studies can reveal the presence of free gas in the tissue. This can assist in the differentiation between infection due to streptococcal or mixed polymicrobial

aerobic–anaerobic infection, and also signify the presence of gas-forming bacteria in other types of necrotic infections. A feathery linear pattern of gas can be observed in infected muscles in clostridial myonecrosis.

The presence of osteomyelitis as a cause of subcutaneous abscess or sinus tract can be discovered by radiological and radionuclide scanning studies. Plain radiograph can show osteopenia or osteolytic lesions, periosteal elevation, and periosteal new bone formation. Sclerotic lesions can be seen when the infection has been present for longer than a month.

Radionuclide scanning is useful in early diagnosis of osteomyelitis. Technetium-labeled methylenediphosphonate isotope is used most frequently, since its uptake by infected bone is enhanced with increased osteoblastic activity. In some cases, decreased uptake can be observed, reflecting compromised vascular supply to the bone.

Radionuclide (^{67}Ga) scanning can be used in the diagnosis of pyomyositis. It shows diffuse uptake in the involved area but does not differentiate intramuscular abscess from necrotizing myositis or necrotizing fasciitis. Computed tomography (CT) can show low-density areas with muscle loss and a surrounding rim of contrast enhancement typical of pyomyositis. Magnetic resonance imaging (MRI) can detect alteration in soft tissue and is particularly useful in differentiating cellulitis from pus and abscess formation. MRI can show enlargement of involved muscles and areas of signal attenuation suggestive of fluid collection. Sonography or CT can be used to guide diagnostic aspiration [13].

CT scanning is the most rapid and sensitive method to diagnose psoas and iliacus muscle infection. It can show diffuse enlargement of the involved muscle and may demonstrate the presence of gas within the muscle suggesting the presence of an abscess [8]. MRI is more sensitive in showing early inflammatory changes prior to development of frank abscess cavity and can show enlarged muscles [7]. MRI with gadolinium injection, in addition to helping to make the diagnosis, may help differentiate between early and late stages that help guide treatment [17]. Some infections can develop very rapidly to life-threatening systemic illness, and definitive diagnosis of the nature and extent of the myositis is made only on surgical exploration.

Management

An emergency surgical exploration is often warranted. This is done in order to define the nature of the infective process, which is accomplished by direct examination of the involved muscles. Furthermore, surgical intervention is needed to perform appropriate debridement. Immediate extensive surgery is necessary to treat gas gangrene. The muscles involved should be removed, and fasciotomies to decompress and drain the swollen fascial compartment are performed. Complete amputation may sometimes be necessary.

Intensive surgical and medical therapy that includes the administration of intravenous fluids and antimicrobial therapy is an essential element in management of muscle infection. Establishing the bacterial etiology initially by gram stain and later by culture followed by bacterial susceptibility testing can allow for selection of proper antimicrobial therapy. Often, however, the initial therapy is empiric, based on epidemiological, historical, and clinical features.

In infection due to GAS parenteral penicillin is often used. The occasional failure of penicillin despite the universal in vitro susceptibility has been explained by the "Eagle effect." Animal work demonstrated the superiority of clindamycin therapy in treating the infection [18]. This observation generated the recommendation for administration of a combination of penicillin and clindamycin in the treatment of GAS as well as in *Clostridium* spp. infections. If staphylococcal infection is suspected or when no initial clue for an etiology is available, a penicillinase-resistant penicillin (e.g., oxacillin, methicillin) is given. Macrolides, vancomycin, or linezolid can be used in penicillin-allergic individuals, and an aminoglycoside, a quinolone, or a fourth generation cephalosporin (i.e., ceftazidime, cefepime) can be administered when a gram-negative aerobe bacilli is suspected.

Prevotella spp. and *Fusobacterium* spp. that were previously susceptible to penicillins are currently exhibiting increased rates of resistance to these and other antimicrobial agents. The production of the enzyme β-lactamase is one of the main mechanisms of resistance to penicillins by these and other gram-negative anaerobic bacilli, including members of the *B. fragilis* group. Complete identification and testing for antimicrobial susceptibility and β-lactamase production are, therefore, essential for the management of infections caused by these bacteria.

Antimicrobial therapy for mixed aerobic and anaerobic bacterial infections is required when polymicrobial infection is suspected [6]. Antimicrobial agents that generally provide coverage for *S. aureus* as well as anaerobic bacteria include cefoxitin, clindamycin, a carbapenem (i.e., imipenem, meropenem), and the combinations of a β-lactamase inhibitor (i.e., clavulanic acid) and a penicillin (i.e., ticarcillin) and the combination of metronidazole plus a β-lactamase-resistant penicillin. Cefoxitin, the carbapenems, and a penicillin plus a β-lactamase inhibitor also provide coverage against members of the family *Enterobacteriaceae*. However, agents effective against these organisms (i.e., aminoglycosides, fourth generation cephalosporins, and quinolones) should be added to the other agents when treating infections that include these bacteria.

The use of hyperbaric oxygen therapy for clostridial myonecrosis is controversial [6, 19]. No controlled studies were performed, and the published reports do not provide evidence of beneficial effect. The potential toxicity of hyperbaric oxygen is also of concern. The most important limitation of utilizing hyperbaric oxygen therapy is the lack of hyperbaric chambers in most hospitals. Transpor-

tation of a seriously ill patient to a facility with a hyperbaric unit is hazardous, as the separation from immediate care for the unstable patient is risky. However, the use of hyperbaric oxygen should be considered when the involved area cannot be completely excised surgically, as may be the case in the paraspinal or abdominal wall.

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