
Surgery in Africa - Monthly Review

Addendum to October 2005 Surgery in Africa Review Surgical Infections - I

This Review did not clearly explain that Group A Streptococcus (GAS) can cause myositis as well as necrotizing fasciitis and toxic shock syndrome. We thank Mark Lumbasi from Kenya for reporting 4 cases of apparent GAS myositis which prompted this reconsideration. We reple below the appropriate section from Mandell, Bennett, & Dolin: Principles and Practice of Infectious Diseases, 6th ed., 2005 Churchill Living written by Mark Pasternack and Morton Swartz

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GROUP A STREPTOCOCCAL NECROTIZING MYOSITIS

In addition to producing an occasional case of typical pyomyositis with abscess formation, on rare occasions group A streptococci cause a fulminant form of myositis (peracute streptococcal pyomyositis, streptococcal necrotizing myositis, streptococcal myonecrosis or spon streptococcal gangrenous myositis). Reports such as that of 1 case of myositis and 3 cases of myositis with necrotizing fasciitis, among 20 patients in an outbreak of invasive group A streptococcal infections associated with a toxic shock-like syndrome, suggest that this form of infection may be more frequent now than in the past. The entire clinical course may be telescoped to 2 to 3 days. The clinical features are usually intense boardlike swelling of the affected muscle, and fever. The overlying skin may be uninvolved, or it may become erythematous or violaceous and contain petechiae and bullae. Most cases involve the extremities and appear to develop spontaneously without antecedent pharyngitis or Bacteremia and toxemia are prominent features and contribute to the very high mortality rate (80% to 100%). The rapid spread of infection in closed compartment of muscles can markedly raise intramuscular pressure, resulting in further necrosis of muscle. However, both processes can be simultaneously present in the same area. The compartment syndrome with group A streptococcal myositis (e.g., a tibial compartment syndrome) may develop in the absence of fascial and muscle necrosis but be associated with muscle bulging secondary to edema and serosanguine exudate.[46] Compartment pressures are elevated. The clinical features of such a syndrome include weakness of the compartment muscles, which are swollen and tender; severe pain on movement of the lower leg; and overlying cutaneous hyperesthesia.

Streptococcal necrotizing fasciitis may resemble streptococcal myositis clinically, although the presence of tense bullae and areas of skin necrosis are more suggestive of the former. Sometimes both conditions are present together. MRI may disclose the predominantly involved structure, but urgent surgical exploration, always necessary in the setting of suspected toxic shock associated with focal pain and swelling, should provide a clear answer. A rare case of acute streptococcal myositis with the toxic shock syndrome caused by group G streptococcus has been reported.

Laboratory findings include a leukocytosis and an elevated serum creatine phosphokinase level. This is in marked distinction to nonstreptococcal forms of pyomyositis, in which little if any elevation of the creatine phosphokinase concentration is seen. This disease is a medical emergency requiring prompt clinical diagnosis with verification at surgery. Distinguishing group A streptococcal necrotizing myositis from streptococcal necrotizing fasciitis and spontaneous clostridial myonecrosis may be difficult clinically, but gas in the tissue suggests spontaneous clostridial myonecrosis. In any case, all three diseases require prompt surgical exploration. Sonography, CT, or MRI scanning usually reveals muscle swelling and fluid collection in muscle compartments. If prolonged delays are encountered in the pursuit of imaging studies, proceeding directly to surgical exploration with an initial limited surgical approach for diagnostic purposes is justified. Early, aggressive surgical intervention with fasciotomy and débridement of necrotic tissue is indicated; in some instances, amputation is required. If the operative Gram stain suggests streptococcal infection, antibiotic therapy should consist of high doses of penicillin G (3 million units intravenously every 3 hours, or 4 million units every 4 hours, or adjusted appropriately for renal insufficiency) along with clindamycin (600 mg intravenously every 6 to 8 hours). Experience suggests that clindamycin has greater efficacy against group A streptococci in this life-threatening infection ("Eagle effect"), because of its greater activity against large bacterial populations in stationary phase growth, its more sustained postantibiotic effect, and its suppression of the production of toxin and other virulence factors by virtue of its inhibition of bacterial protein synthesis. The use of intravenous immunoglobulin G as an adjunct in the treatment of streptococcal toxic shock, to neutralize streptococcal exotoxins and perhaps to modulate the host immune response, has gained popularity on the basis of retrospective studies and one small prospective randomized trial, but conclusive evidence supporting its use remains limited.

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