Pyomyositis

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Abstract
Pyomyositis is a primary infection of the skeletal muscle usually caused by Staphylococcus aureus. This infectious disease is endemic in tropical areas, and sporadic in temperate climates where it mostly affects immunocompromised patients. Incidence has increased since AIDS first occurred. The first symptoms are often overlooked and evolution shows fever, sepsis, local inflammatory involvement usually located in large muscles of the lower extremities. Ultrasound, computer tomography scan (CT) and resonance magnetic imaging (RMI) confirm the diagnosis. The agent responsible can be isolated biologically from blood cultures by puncture either surgical or radio-guided. Treatment is relies on antibiotic therapy and surgical drainage.

Keywords
Staphylococcus aureus, infectious myositis, tropical disease

Definition
Infectious myositis is an infection of skeletal muscles. Pyomyositis (PM) is a primary acute bacterial infection of skeletal muscles, usually caused by Staphylococcus aureus. This clinical entity was first described in 1885 by Scriba as an endemic disease in the tropics [1]. In North America it was first reported in 1971 by Levin [2].

Differential diagnosis
Differential diagnosis varies according to the topography of PM. Appendicitis for iliopsoas, septic arthritis of the hip for iliacus muscle. Muscle infarction, sarcoma, osteomyelitis, hematoma, muscle rupture, thrombophlebitis, cellulitis are differential diagnosis for most of the localisations. Trichinosis, toxoplasmosis and coxsackie infection virus can be discussed in case of multiple abscesses and hypereosinophilia.

Epidemiology
Pyomyositis is endemic in tropical areas. For example incidence is 1/1000 in Uganda and 13% of the deaths in an emergency unit in Nigeria are due to PM [3], hence the name of tropical pyomyositis. It represents 1 to 2% of surgery hospitalization [4]. In tropical areas, PM is more frequent among men, with a female/male ratio of 3/5. It affects mostly young people and children [4]. In these areas, denutrition is frequently associated.
By contrast, in non tropical areas, PM is uncommon and affects mainly adults and elderly patients. It is worth noting that 25% of the cases were reported to have travelled recently to the tropics [5].

Trauma is found in 25-40 % of the cases [6]. Some authors have noticed its crucial role. PM could be induced, in an experimental model when trauma is associated with intravenous injection of Staphylococcus aureus [7].

PM is a feature mostly associated with various causes of immunodeficiency, especially in patients infected by human immunodeficiency virus (HIV) and with CD4 count under 150/mm3 [8]. Thus, with the increasing incidence of HIV, PM became more prevalent in tropical and non tropical countries. Thus 21% of PM patients are HIV-infected in non-tropical areas [9,10]. Several factors account for the higher prevalence of this infection in the HIV-positive population: IgG2 deficiency [11], altered phagocytosis [12], the HIV [13] virus itself or zidovudine-induced myopathy [14]. Other causes of immunodeficiency have been reported as risk factors for PM such as steroid use [15], diabetes mellitus [16], leukemia [17], neutropenia due to myelodysplasia [18], Felty’s syndrome [19], cyclic neutropenia [20], sickle cell anemia [21] and lymphoma. Several cases have also been described in HIV-negative intravenous drug abusers [22].

Clinical description

The clinical course can be divided into 3 stages:

First stage or invasive stage

During the first two weeks, the disease is subacute and symptoms are often neglected. General symptoms are variable including fever and anorexia. Local symptoms consist in swelling, erythema, mild pain and minimal tenderness.

Second stage or suppurative stage

Diagnosis is often made at this stage. General signs are more prominent with high fever, chills and septic syndrome. Local abnormalities include tenderness, swelling, fluctuance (presence of pus), myalgia and inflammatory skin.

Third stage

Systemic manifestations are severe with sepsis and fever. Local examination shows erythema, exquisite tenderness and obvious fluctuance. Further complications can occur: metastatic abscesses, arthritis, septic shock and renal failure. Toxic shock syndrome was also recently described in one patient with PM; it was caused by a non-toxin producing strain of Staphylococcus aureus [23].

A single group of muscles is usually involved and 25% of the cases are described with multiple abscesses [4]. The groups most frequently involved are large muscles of the lower extremities, mostly thigh and trunk muscles. The right side is more frequently involved than the left one. Lesions of abdominal muscles are rarely found but may represent a challenging differential diagnosis with acute appendicitis.

Leukocytosis is frequent and eosinophilia is commonly described in patients with PM living in tropical countries. Eosinophilia is rare in patients living in temperate climates probably because of the low prevalence of parasitic infections in these areas [4]. Muscle enzymes levels are usually normal and markers of denutrition such as hypoalbuminemia can be found [4].

Etiology

Staphylococcus aureus is responsible for 95 % of the cases in tropical areas and 70% of the cases in non-tropical areas [9]. Several other bacterial pathogens have also been described: Other Gram-positive cocci than Staphylococcus aureus: Staphylococcus epidermidis [24], Streptococcus pneumoniae [25], Streptococcus pyogenes [26], Streptococcus dysgalactiae [27] Gram-negative bacilli: Proteus mirabilis [28], Klebsiella oxytoca [29], Klebsiella pneumoniae [30] Yersinia enterocolitica [31], Salmonella species [32,33], Aeromonas hydrophila [34], Escherichia Coli [35], Haemophilus Influenzae [36], Citrobacter freundii [37]; Stenotrophomonas maltophilia [38], Gram-positive bacilli: Nocardia asteroides [39]; Gram-negative cocci: Neisseria gonorrhoea [40], Anaerobic bacteria such as Prevotella melaninogenica [41], Bacteroides fragilis [42], Eubacterium lentum [43] may also rarely trigger PM. One case of polymicrobial PM has been described [44] and at least eight cases of Mycobacterium tuberculosis PM have been reported [45-49]. Parasitic (Filaria, nematodes) and viral causes (other than HIV) (Herpes, Picornavirus, Coxsackie virus, Arenaviruses, Arbovirus) have also been involved [4,9]. A recent study demonstrated a link between PM and toxocariasis [50].

The causative agent can be isolated from ultrasound or computed tomography-guided biopsy or surgical puncture or from blood cultures (positive in 5 % of the cases in tropical cases, and 38% in non tropical cases [51]).

The main causative agent of PM in HIV patients is also Staphylococcus aureus (16 out of 18 HIV patients with PM) [9]. Cases of PM due to Mycobacterium tuberculosis and one case of polymycrobial PM have been reported in those patients.
Diagnostic methods
Ultrasound can help as a diagnostic tool showing muscular heterogeneity or a purulent collection. It also can guide puncture [52]. However, it is not useful during the first stage of the disease. CT can confirm the diagnosis before abscesses occur with enlargement of the involved muscles and hypodensity. When abscess is present, heterogeneous attenuation and fluid collection with rim enhancement can be found [53]. MRI is useful to assess PM and determine its localization and extension. It shows hyperintense signal in T2-weighted images, hyperintense rim on enhanced T1-weighted images and peripheral enhancement after Gadolinium-diethylenetriamine pentaacetic acid (Gd-DTPA) [54]. MRI is currently the most sensitive radiological procedure. Gallium scintigraphy is the reference method to document multiple localizations of muscle abscesses. This isotope imaging procedure must not be carried out after a RMI with gadolinium injection because of the interference between the 2 components during 48-72 hours (responsible for false negative interpretations) [55,56] CT and ultrasound can also help diagnosis and therapeutic punctures.

Treatment
Surgical or radio-guided drainage of the abscesses must be associated to intravenous antibiotic therapy which is effective on Staphylococcus aureus penicillin-resistant. The treatment is adapted to the results of Gram-staining smear, cultures and the antibiogram. No specific duration has been established for treatment.

References
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