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Tropical Pyomyositis

S. S. Chauhan, S. Chauhan, K. K. Singh

Introduction

The term ''tropical pyomyositis'' (TP) is referred to "primary muscle abscess arising within the skeletal muscles". This strictly excludes (a) intermuscular abscesses, (b) abscesses extending into muscles from adjoining tissues such as bone or subcutaneous tissues and (c) those secondary to previous septicemia.

Historically, first case of TP was described by Scriba in 1885, though some authors believe that Virchow was the first one to describe this entity.[1,2] Since TP was predominantly seen in tropics, various terms like *tropical pyomyositis, myositis tropicans,* and *tropical myositis* are used, but nowadays this entity is no longer confined only to tropics. Levine in 1971, reported the first case of TP from temperate area. In the last 3 decades there has been a surge of cases from temperate areas, often affecting individuals who are immunocompromised.[3,4,5] As the entity is becoming increasingly recognized in nontropical areas, it has been suggested that term TP be renamed as infectious myositis or spontaneous bacterial myositis, but till present this entity is still commonly referred to as TP.

Prevalence and Incidence

TP was earlier thought to be confined to tropical areas, but there has been a resurgence of this disease in non tropical areas. Immunodeficient conditions like human immunodeficiency virus (HIV), diabetes, organ transplantation and iatrogenic immunosuppression by corticosteroids, chemotherapy, or immunomodulating agents, rheumatological diseases, sickle cell, renal failure, lung diseases, chronic liver disease and malignancies have been implicated in the development of pyomyositis in temperate regions in up to 75% of cases and another 9% have history of travel or have migrated from tropical areas.[6,7] However in temperate regions more and more cases are being reported from individuals who are not immunocompromised.

In some tropical countries, TP accounts for 1-4% of all hospital admissions.[8,9] The exact incidence and prevalence from India is not known but we come across these cases frequently during the clinical practice.[6]

Etiology

Bacteriologic diagnosis of pyomyositis is traditionally made from cultures of surgical specimens or blood cultures. Staphylococcus aureus is the causative organism in up to 90% of cases from tropical areas and 75% from temperate areas followed by Group A Streptococci in up to 5% of cases.[10] Recently virulent strains of methicillin resistant staphylococcus have been isolated from

cases of TP. Less common organisms include streptococci (Group B, C, G), pneumococcus, neisseria, hemophilus, aeromonas, pseudomonas, klebsiella, escherichia.[10,11,12] There are anecdotal case reports of organisms like mycobacterium, fungal, anerobes, salmonella, vibrio causing TP both in immunocompromised and immunocompetent hosts.[13,14,15] Patients who have suppressed immune systems or underlying medical conditions, such as diabetes, may be more susceptible to developing infections caused by unusual organisms. However, a review of pyomyositis cases in the United States found that the distribution of causative organisms were similar among cohorts that were HIV-positive, HIV-negative with underlying medical conditions, or HIV-negative with no underlying medical conditions.

Pathogenesis

The exact pathogenesis has not been elucidated till date. Diminished local resistance in the setting of transient bacteremia is usually invoked. Local predisposition to infection, or ''locus minoris resistentiae,'' may be attributed to antecedent trauma. Generally, skeletal muscles are intrinsically very resistant to infections. This is probably due to sequestration of iron by myoglobin. Iron is essential for rapid growth and proliferation of organisms. Relative lack of iron in these tissues, results in slowing the growth of bacteria's and more time for effective host defences against invading organisms. This is borne out by the fact that of all cases where staphylococcus septicemia, the incidence of infectious myositis is less than 1%. Even direct inoculation of staphylococcus in skeletal muscles failed to produce abscess.[16] Trauma as in exercise or blunt injury may facilitate hematogenous access to the muscle and provide a critical bacterial nutritional requirement in the form of iron from myoglobin. Formation of a small hematoma may provide a favorable site for the binding of staphylococci and other bacteria, and the surrounding damaged and devitalized tissue might also impede the host immune response. The permissive role of minor muscle damage is suggested by the numerous reports of pyomyositis after vigorous exercise and athletic activity in previously healthy individuals in temperate regions In upto 20-50% of cases there is preceding history of blunt trauma or vigorous exercise. Some authors have postulated that in some of these patients the immunity against staphylococcus may be lacking as is evidenced by defective T lymphocyte responses.[17]

Other factors implicated include preceding viral and protozoal infections but definite cause and effect relationship is lacking. More so in these cases the abscesses are not typical of TP.

Pathology

As already mentioned the term TP should be strictly restricted to primary muscle abscess arising within the skeletal muscle and not for intermuscular abscesses or abscesses extending into muscles from adjoining tissues or those secondary to previous septicemia.

Microscopically, there is edematous separation of muscle fibers with interfiber infiltration by lymphocytes and plasma cells. The process is not diffuse but patchy myocytolysis with inflammatory infiltrate consisting of lymphocytes and plasma cells is seen with areas of complete disintegration. This is followed by either a reparative process or complete degeneration with suppuration. It is again reemphasized here that demonstrating myositis and not abscess in biopsied specimen of muscle is diagnostic for TP.[18]

Clinical features

No age and gender is immune from TP, but it has a slight preponderance in active young males in age group of 10-40 years. Usually a single muscle is affected but, it is not uncommon to see multiple muscles being involved (12-40% of cases).[19] The most common site of pyomyositis is the thigh, with other bulky muscles like gastrocnemious, glutei, pectorals, biceps, also being commonly involved.[20] The abdominal wall, chest wall, paraspinal muscles, and pelvic muscles are occasionally affected.[11] The

lower extremity is four times more likely than the upper extremity to be involved]. Clinically the progression is divided into three stages:

Invasive stage: This stage is characterised by painful swelling. Fever and leukocytosis may be present. Examination reveals a tender area of wooden consistency with or without erythema. Aspiration at this stage will not yield pus. This stage may resolve remaining undiagnosed in majority or progress to next stage.

Suppurative Stage: This is seen in cases which progress from invasive stage. High spiky fevers beginning between 2nd and 3rd weeks mark the onset of this stage. It is characterized by abscess formation. However since the infection is deep seated, classical characteristics of abscess such as erythema and fluctuation may be lacking, but there is extreme pain and tenderness in the affected part. Aspiration yields pus. Regional lymphadenopathy if present, suggests an alternative diagnosis.

Late Stage: If suppurative stage remains untreated, the infection may disseminate leading to multiple abscesses, septicemia, septic shock and multiorgan system failure.

Differential Diagnosis

TP can have myriad presentations in addition to the typical course described above. These include only local symptoms, only fever and leukocytosis, acute fever with chills, pyrexia of unknown origin (in absence of local symptoms), compartment and compression syndromes and septic shock.[21,22,23]

TP is a great mimicker with many diseases having similar presentation to TP, often leading to diagnostic dilemma. The list of differential diagnosis includes deep vein thrombosis, cellulitis, muscle contusion, muscle hematoma, muscle or tendon rupture, septic arthritis, osteomyelitis, osteosarcoma, polymyositis, spontaneous gangrenous myositis trichinosis, Cysticercus cellulose and leptospirosis. Therefore a high degree of suspicion is required, lest the diagnosis be missed resulting in adverse outcomes of a potential treatable entity.

Diagnosis

Early diagnosis and treatment is of utmost importance but in clinical practice this is not so. The reasons are manifold and include lack of awareness, atypical manifestations and wide range of differential diagnosis.

Aspiration and culture of the pus remains the standard diagnostic method but in early stages when there is minimal or no suppuration and pus may not be aspirated. Pus cultures may not yield any organism in upto 30% of cases. Muscle biopsy with culture of tissue remains the gold standard.[18] Besides, confirming the diagnosis of TP, other diagnosis like polymyositis, osteosarcoma, and intermuscular abscesses can be confidently excluded. Blood cultures may be supportive.

Short of muscle biopsy, noninvasive radiological methods like ultrasound, computed tomogram (CT) or magnetic resonance imaging (MRI) are useful in confirming the diagnosis and monitoring the patient during follow up. Plain radiographs are not sensitive, but in a few cases may suggest muscle enlargement, loss of muscle definition, obliteration of deep fat planes, gas in soft tissues, and reactive changes in adjacent bone. Plain radiographs are more useful in excluding other processes, such as osteomyelitis or bone sarcoma Ultrasound remains the initial screening tool being easily available and cost effective. It shows hypoechoic areas with increased muscle bulk but early invasive stage may be missed.[24]

On CT scan, areas of pyomyositis are visualized as areas of low attenuation with loss of muscle plain surrounded by a rim of contrast enhancement, but it cannot differentiate abscess from swollen muscles.[25] MRI is more specific and sensitive as compared to CT scan. On MRI T1 weighted images show hyperintense rim with increased signal intensity of involved muscles which on T2 weighted with gadolinium enhancement show uniform hyperintensity with a low intensity rim.[24,25,26] MRI is also more useful in detecting pyomyositis in pelvic region and to rule out other pathologies effectively.[27]

For inconclusive cases, gallium scintigraphy helps to localize inflammation, but it is nonspecific and expensive.[28]

Anemia, leukocytosis with elevated ESR and CRP are commonly present. Eosinophilia if present, suggests parasitic infection. Blood cultures are sterile in upto 90% of cases from tropical areas and upto 70% from temperate regions, but are not specific to TP. Diffuse myalgia's with deranged liver and renal functions favor the diagnosis of leptospirosis. Bilateral symmetrical involvement of proximal muscle groups, elevated muscle enzymes (creatine phosphokinase, aldolase) and characteristic electromyography (low amplitude polyphasic potential) is consistent with polymyositis, but muscle biopsy is diagnostic. Elevated creatine phosphokinase is not a feature of TP.

Once diagnosis is made, common immuno-suppressive states (diabetes, HIV, rheumatological and malignancies) must be ruled out along with immunoglobulin levels.

Treatment

Treatment should be immediately initiated on confirmation of diagnosis. Although, rarely diagnosed during invasive stage, antibiotics alone will suffice at this stage. Once the abscess has formed, drainage is mandatory. Traditionally surgical incision and drainage have been used but newer methods like CT guided percutaneous drainage or continuous suction with primary closure of wound are equally effective. However, if significant necrosis with large areas of involvement is present, traditional incision and drainage is the best alternative.

As with any serious infection, antibiotic therapy cannot be delayed and must be guided by the treating physician's judgment. Impending culture and sensitive reports, it is advised that cloxacillin be the first line therapy with first generation cephalosporins like cefazolin as an alternative in penicillin sensitive individuals. Coverage for methicillin resistant Staphylococcus aureus (MRSA) using vancomycin or teicoplanin should be considered in severely ill patients, who have high risk of MRSA or the culture sensitivity report shows MRSA.[29] Linezolid or dalfopristine-quinapristine are reserved for vancomycin intermediate sensitive Staphylococcus (VISA).[29] Adding a second drug against Staphylococcus does not confer any benefit.

Penicillin remains the first line antimicrobial agent for streptococcus. Gram negative organisms require two drugs, usually a combination of third generation cephalosporin with aminoglycosides. Suspicion of anerobes warrants addition of metronidazole. If patient is immunosuppressed or very toxic, empirical treatment is started with broad spectrum antibiotics covering staphylococcus, streptococcus, gram negative and anerobes. In such situations vancomycin with an antipseudomonal carbapenems or β lactam combination are the most appropriate therapy. Other antimicrobials, aztreonam fluoroquinolone, aminoglycoside, cephalosporin or clindamicin, alone or in combination have been used successfully.

Treatment is continued for about a week after patient becomes afebrile, blood counts normalize and wound is healthy, but for metastatic infections it is recommended that treatment be continued for 4-6 weeks. Failure of fever to normalize indicates metastatic infection, drug resistance or drug fever. Radiology help may be taken to assess the course of the disease and to find out metastatic infection.

Although no recommendation are available, it is believed that eliminating nasal carriage of staphylococcus by using topical mupirocin or systemic rifampicin in patients with past episodes of pyomyositis, staphylococcus septicemia and in immunocompromised persons, further episodes of pyomyositis can be prevented.[30]

Prognosis

With heightened awareness, newer diagnostic modalities and effective chemotherauptic agents, the mortality in TP has been considerably reduced.

The fatality rate still varies from as low as 0.5% to as high as 10%. In patients who recover even from severe disease, surprisingly there is little or no dysfunction in the affected part.

Summary

Despite an agreement on the definition of evidence-based medicine (EBM), there remains considerable debate around what constitutes an evidence-based care. In the current review, we discuss the clinical application of EBM including challenges in retrieving relevant medical information, critically reviewing the data and applying it to the patient. Also discussed are the technics and issues surrounding patient- physician communication. Among the current updates in EBM we highlight the '5S' model of retrieving best evidence, use of hand held devices for point of care information and describe future directions and use of computer based decision support, ehealth, electronic medical records and evidence based management to improve quality of health care. Several methods are described to enhance risk communication and evidence-based practice.

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