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Tropical pyomyositis

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ABSTRACT

Tropical pyomyositis is a serious infectious disease characterised by the formation of abscesses in the skeletal muscles and is primarily caused by *Staphylococcus aureus*, with an increasing incidence in non-tropical regions. The disease primarily affects men and young adults, often following minor trauma, with an increasing incidence in immunocompromised individuals. Immunocompromised hosts are more likely to be affected by Gram-negative organisms, *Mycobacterium tuberculosis*, opportunistic infections such as fungal pathogens, non-tuberculous mycobacteria, and *Nocardia* species. Diagnosis is complicated by non-specific symptoms and the low yield of blood cultures, so imaging studies such as Magnetic Resonance Imaging (MRI) are required for accurate identification. Treatment focuses on controlling the source through drainage, tailored antibiotic therapy, and supportive care, especially in patients with complications such as multi-organ dysfunction. Given the complex clinical manifestations, heightened awareness and a collaborative approach to education and resource provision are critical to improving outcomes in patients with tropical pyomyositis.

1. Introduction

Tropical pyomyositis or primary pyomyositis is a serious infectious condition characterized by the formation of abscesses within skeletal muscles [1]. It is believed to be hematogenous in origin, arising from transient or concurrent bacteremia due to a remote or unknown source. In contrast, myositis caused by infection spreading from a nearby site or resulting from penetrating trauma is known as secondary myositis [2]. While tropical pyomyositis is predominantly observed in tropical regions such as sub-Saharan Africa, Southeast Asia, and the Caribbean, an increasing number of cases have emerged in non-tropical areas [3–7]. This trend prompts a re-evaluation of its nomenclature; the term "primary" or "infective pyomyositis" may be more appropriate.

The existing literature largely consists of case reports, case series, and retrospective studies, which contribute to a fragmented understanding of the disease's complexities. The nonspecific clinical presentation, combined with limited diagnostic and therapeutic resources in endemic regions, complicates timely diagnosis and effective management. This chapter aims to provide a comprehensive review of the epidemiology, pathophysiology, clinical presentation, diagnosis and treatment strategies for pyomyositis.

2. Epidemiology

The incidence of tropical pyomyositis exhibits considerable geographic variation. In endemic regions, particularly in Africa,

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Southeast Asia, and Latin America, pyomyositis accounts for approximately 1–4% of all hospital admissions [8–10]. This condition is slightly more prevalent in males and shows a peak incidence among children and young adults aged 10–30 years. The increased frequency in these demographics is attributed to higher levels of physical activity, which raises the likelihood of muscle micro-trauma—a proposed factor in the disease's pathogenesis [11,12]. Patients often report a history of blunt trauma occurring days or even weeks prior to the onset of symptoms. Notably, there has been a significant rise in cases among immunocompromised individuals, including those with diabetes, HIV, malnutrition, hematological malignancies, intravenous drug abuse and patients undergoing treatment with monoclonal antibodies [13–15].

3. Etiology and pathogenesis

Staphylococcus aureus is the most common bacterial pathogen associated with pyomyositis; however, other bacteria, including *Streptococcus, Escherichia coli, Klebsiella* spp*, Salmonella* spp*, anaerobes, Candida* spp*,* and *Mycobacterium* spp, may also be implicated especially in children and immunocompromised hosts [16–21]. The emergence of methicillin-resistant Staphylococcus aureus (MRSA) raises significant concerns due to its resistance profile, necessitating more complex treatment strategies [22,23].

Skeletal muscles are typically resistant to infection, and muscle involvement is rarely observed in autopsies of disseminated staphylococcal infections [24]. Myoglobin within muscle tissue sequesters iron, which is essential for bacterial growth, thereby limiting bacterial proliferation. It is hypothesized that bacterial pathogens may enter muscle tissue through transient bacteremia, particularly following minor trauma that disrupts muscle structure. Contributing factors such as malnutrition, vitamin deficiencies, and immunosuppression create a more favorable environment for bacterial proliferation within muscle tissue [13–15,25–27].

The pathogenesis of pyomyositis, particularly involving *Staphylococcus aureus*, is increasingly well understood. *Staphylococcal* cytotoxins, notably alpha-toxin and Panton-Valentine leukocidin (PVL), play critical roles in this process [28]. Alpha-toxin, a potent cytolysin, directly damages muscle cells by forming pores in their membranes, leading to cell lysis and necrosis [29]. This contributes not only to tissue destruction but also to an inflammatory response that exacerbates local damage. Additionally, alpha toxin induces the release of pro-inflammatory cytokines, intensifying the characteristic inflammatory milieu of pyomyositis.

Panton-Valentine leucocidin (PVL), on the other hand primarily targets leukocytes, particularly neutrophils. By disrupting the membrane integrity of these immune cells, PVL impairs their function, hindering the body's ability to mount an effective immune response against the infection [30]. This immune evasion allows *Staphylococcus aureus* to proliferate within muscle tissues, facilitating the development of abscesses. The combined effects of alpha-toxin and PVL not only lead to significant tissue damage but also create an environment conducive to bacterial survival and replication (Fig. 1). The high prevalence of nasal colonization by PVL-positive Staphylococcus aureus in sub-Saharan Africa partially explains the region's elevated incidence of pyomyositis [31,32].

4. Clinical presentation

Tropical pyomyositis typically progresses through three stages, each with distinct clinical features [2,33]. During the initial invasive phase, patients may experience mild, localized muscle pain, swelling, and stiffness, often without pronounced systemic symptoms. Fever may or may not be present, making early diagnosis challenging. Frequently, patients are initially diagnosed with musculoskeletal pain and treated with painkillers and muscle relaxants. The absence of fever in this stage often complicates the recognition of a possible infection.

This is followed by a suppurative phase, during which patients present with increased pain, swelling, and erythema over the affected area, often accompanied by fever and malaise. As the infection progresses, an abscess forms within the muscle. The condition can mimic cellulitis, arthritis, deep vein thrombosis, and sometimes a soft tissue tumour [34]. It is during this phase that most patients seek medical care, as the pain becomes severe and functionally limiting. If patients do not seek medical attention or fail to respond to

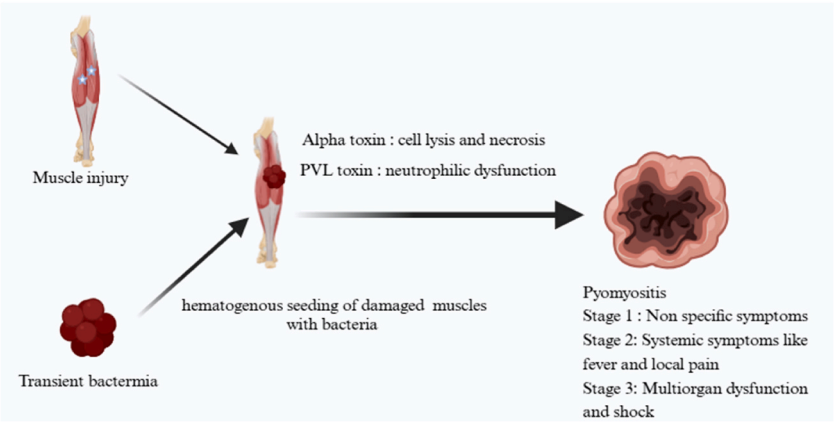

Fig. 1. Pathogenesis and clinical stages of pyomyositis.

treatment, they may progress to the dissemination phase, characterized by disseminated disease, multiorgan dysfunction, and vasoplegic shock, which is associated with high mortality despite treatment.

While subacute presentations are common, some patients may progress rapidly, presenting with full-blown sepsis and multiple organ dysfunction syndrome (MODS) [35,36]. In tropical regions, where pyomyositis is more prevalent, clinicians should maintain a high index of suspicion for this diagnosis in any patient with unexplained muscle pain and fever. A thorough history, including recent muscle trauma, infection, or travel to endemic areas, is invaluable.

5. Blood and microbiological workup

Initial blood investigations often reveal leukocytosis with a neutrophilic response, alongside elevated inflammatory markers such as C-reactive protein (CRP) and ferritin. In patients with hematological malignancies or solid organ tumours undergoing chemotherapy, neutropenia may also be observed [37]. Creatine phosphokinase (CPK) levels typically remain within normal ranges during the early stages of tropical pyomyositis, which helps differentiate it from inflammatory myopathy or rhabdomyolysis. The yield of blood cultures is generally low, with positivity rates reported between 5% and 30% [2,4,28,33]. While blood cultures may become positive in the later stages of disseminated infection, aspiration of collections and subsequent pus cultures are crucial for microbiological confirmation. Pus samples should be sent for Gram staining, as well as aerobic and anaerobic bacterial cultures, fungal cultures, acid-fast bacilli (AFB) staining, GeneXpert testing, and Mycobacterium Growth Indicator Tube (MGIT) cultures. It is important to note that up to 30% of pus cultures may yield negative results.

6. Imaging studies

Ultrasonography is a useful, rapid, and cost-effective imaging modality for detecting fluid collections within muscles and guiding diagnostic aspiration. Computed Tomography (CT) though helpful in diagnosis can miss changes associated with early pyomyositis [38]. It is less commonly used but can help visualize abscesses and guide aspiration or drainage, particularly in facilities where Magnetic Resonance Imaging (MRI) is unavailable. MRI is the preferred imaging technique, providing excellent soft tissue contrast and the ability to detect early-stage muscle inflammation [39]. MRI can effectively distinguish pyomyositis from similar conditions such as cellulitis, osteomyelitis, and deep vein thrombosis (Fig. 2).

7. Differential diagnosis and approach to the diagnosis

Tropical pyomyositis is often confused with several conditions, including septic arthritis, spondylodiscitis and osteomyelitis, particularly when patients present with localized pain and fever. It may also be misdiagnosed as inflammatory myositis, post-viral myositis, trichinellosis, or rhabdomyolysis during the dissemination stage. A thorough history and clinical examination are essential for differentiating these conditions.

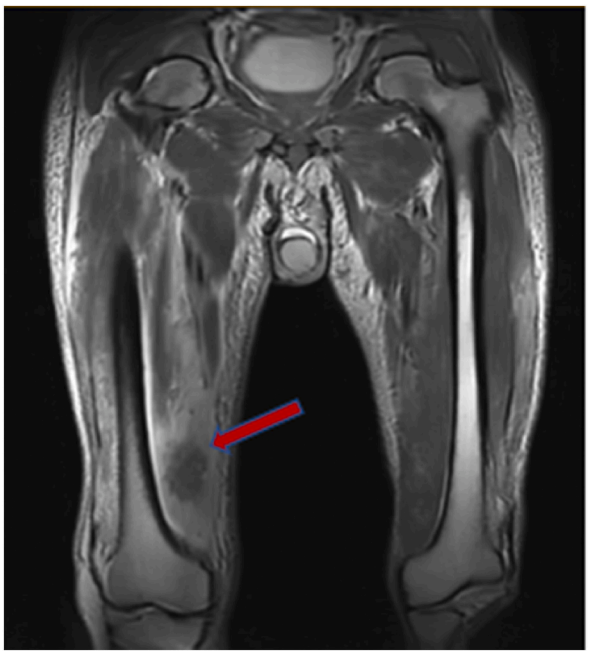

Fig. 2. Focal area of an evolving abscess in the vastus medialis muscle in a kidney allograft recipient (indicated by an arrow).

For instance, a history of consuming raw or poorly cooked meat, along with recent diarrhea and periorbital oedema with peripheral eosinophilia can suggest trichinellosis [40]. Recent flu-like symptoms followed by the onset of myositis may indicate influenza-associated myositis [41]. Additionally, a history of rash, arthritis, photosensitivity, or specific skin manifestations—such as Gottron's papules, heliotrope rash, palmar papules, and alopecia—can point towards autoimmune myositis. In patients with inflammatory myopathy who are on immunosuppression, pyomyositis may be misdiagnosed as a flare of the underlying myopathy. However, asymmetrical muscle involvement accompanied by localized pain and tenderness should prompt suspicion of infective myositis. In cases where there is an absence of movement restriction and no bony tenderness, this favours a diagnosis of tropical pyomyositis over septic arthritis or osteomyelitis.

Gangrenous myonecrosis is a critical differential diagnosis that necessitates urgent surgical intervention (Table 1). Microbiologically, gangrenous myositis is often associated with anaerobic infections like *clostridium spp*, whereas tropical pyomyositis is predominantly caused by *Staphylococcus aureus, Streptococcus* and Enterobacteriaceae [42–46].

Distinguishing between gangrenous myositis and tropical pyomyositis requires careful consideration of clinical features, imaging studies, and laboratory findings. Gangrenous myositis typically presents with rapid progression, severe pain, systemic toxicity, and signs of necrosis. In contrast, tropical pyomyositis generally begins with localized pain and swelling, often following muscle trauma, and is associated with milder systemic symptoms. Imaging studies, such as ultrasound and MRI, can reveal abscess formation, while gangrenous myositis usually demonstrates extensive necrosis and gas formation.

8. Management

The management principles for tropical pyomyositis include source control, antibiotic therapy, and the identification and management of complications (Table 2). The most critical aspect is source control. Less invasive pus drainage using a pigtail catheter is ideal; however, if the pus is loculated and there is widespread muscle necrosis or compartment syndrome, surgical debridement should be considered. Patients with extensive involvement may be critically ill and carry significant operative risks. A delay in achieving source control is associated with poor outcomes [1].

The choice of antibiotics depends on the patient's age, comorbidities, and the severity of illness (Table 2). In adult patients without comorbidities, the most likely pathogens are *Staphylococcus and Group A Streptococcus*. These patients can be started on empiric therapy with Vancomycin or Daptomycin or Linezolid until culture and susceptibility results are available [47].

In immunocompromised patients with multiple comorbidities, there is a risk of gram-negative and anaerobic infections. In such cases, broad-spectrum gram-negative coverage with Meropenem or a β-lactam/β-lactamase inhibitor combination should be considered, along with Vancomycin or Daptomycin or Linezolid [17,37]. Antibiotics can be adjusted based on susceptibility reports once they are available.

In cases of *Group A Streptococcus* infection with toxic shock syndrome, the addition of Clindamycin is recommended to provide additional antitoxin activity. The duration of antibiotic therapy should be individualized based on the clinical response. If source control is achieved, a treatment duration of 2–4 weeks is typically sufficient. If source control is incomplete, antibiotic therapy may need to be extended. In patients with toxic shock syndrome, early initiation of adjunctive Intravenous Immunoglobulins (IVIG) can be helpful [47].

Supportive care for primary myositis involves several critical components, particularly in patients with multiple organ dysfunction syndrome (MODS) and acute respiratory distress syndrome (ARDS). Lung-protective ventilation strategies should be implemented to avoid ventilator induced lung injury in patients with ARDS [48]. It is essential to optimize vasopressor use, as inadvertent high infusion rates can exacerbate muscle ischemia. In malnourished patients, hypocaloric feeding should be carefully administered, with close monitoring for the potential development of refeeding syndrome. Given the possible role of thiamine and vitamin C deficiency, supplementation can be considered in malnourished patients [49]. Strict glycaemic control is imperative for diabetic patients to prevent complications. Additionally, organ support measures, such as dialysis, may be necessary, along with vigilant monitoring for signs of compartment syndrome.

Summary: Tropical pyomyositis is a complex and potentially life-threatening condition that presents significant diagnostic and management challenges, particularly in endemic regions. Its increasing incidence in non-tropical areas necessitates heightened awareness among healthcare professionals. A comprehensive understanding of its epidemiology, pathophysiology, and clinical presentation is crucial for timely diagnosis and intervention. Effective management hinges on early source control, appropriate antibiotic

Table 1
Comparison of tropical pyomyositis and gas Gangrene.

Clinical Feature	Tropical Pyomyositis	Gas Gangrene
Etiology	Typically caused by *Staphylococcus aureus*, *Streptococcus* spp, Enterobacteriaceae	Caused primarily by *Clostridium perfringens*, *Clostridium septicum*, *Clostridium* spp
Inciting event	Often in healthy individuals without trauma or minor injuries	Often occur after penetrating injuries or surgical wounds though spontaneous gas gangrene is possible
Clinical presentation	Localised muscle pain, swelling and fever. Crepitus is usually not present	Severe pain, gas production and systemic toxicity. Crepitus is present due to gas formation within the tissues
Progression	Gradual onset; develops over weeks to months	Rapid progression, often within hours of infection
Management	Surgical drainage and antibiotics	Emergency debridement, antibiotics, hyperbaric oxygen therapy
Mortality	Low with prompt treatment	High mortality despite treatment

Table 2
Common Microorganisms, Host factors, and Geographic Distribution.

S. No	Microbiology	Host factors	Geographical distribution
1.	Staphylococcus aureus	Most common cause in immunocompetent host	90% of cases in tropical countries 75% of cases in temperate countries
2.	Group A streptococcus	More common in children and young adults	25% of pyomyositis in pediatrics age group
3.	Gram negative bacilli *Escherichia coli*, *Klebsiella* spp, *Pseudomonas* spp and anaerobes	Immunocompromised hosts	Not limited to any specific geographic region
4.	Tuberculosis	Immunocompromised hosts, particularly those with advanced HIV infection or receiving steroids, anti-TNF-alpha inhibitors, or Janus kinase (JAK) inhibitors."	In Tb endemic countries
5.	Fungal (Candida, Aspergillus spp, Mucorales)	Immunocompromised hosts especially in neutropenic hosts and poorly controlled diabetes	Not limited to any specific geographic region
6.	Histoplasmosis	Immunocompromised hosts, particularly those with advanced HIV infection or receiving steroids, anti-TNF-alpha inhibitors, or Janus kinase (JAK) inhibitors."	Nitrogen-rich soil, typically found along riverbanks
7.	Dematiaceous fungi	Immunocompromised hosts, especially renal transplant recipients	Not limited to any specific geographic region
8.	Nocardia	Immunocompromised hosts especially in poorly controlled diabetes and patients on steroids	Not limited to any specific geographic region

Table 3
Summary of management for tropical pyomyositis.

Management strategy
Definite care
1. **Source control:** - Pigtail catheter for less invasive drainage if no loculation. - Surgical debridement for loculated pus, widespread necrosis, or compartment syndrome. 2. **Empiric antibiotic therapy:** **Adults without comorbidities and hemodynamically stable**- Vancomycin, Daptomycin, or Linezolid **Immunocompromised patients or hemodynamically unstable** - Meropenem or β-lactam/β-lactamase inhibitors + Vancomycin/Daptomycin/Linezolid. *Addition of Clindamycin for Group A Streptococcus with toxic shock syndrome (antitoxin activity). *Adjust antibiotics as per susceptibility results - Antibiotics duration: 2–4 weeks if source control is achieved, extended if not. - Early IVIG for toxic shock syndrome to reduce toxin activity.
Supportive care
- Lung-protective strategies in ARDS. - Carefully monitor and adjust vasopressor infusions to avoid muscle ischemia. - Hypocaloric feeding in malnourished patients, with close monitoring for refeeding syndrome. - Consider thiamine and vitamin C supplementation in malnourished patients. - Strict glycemic control in diabetic patients to prevent complications. - Dialysis if necessary, and monitor for compartment syndrome.

therapy, and robust supportive care. Given the diverse clinical manifestations and potential complications, maintaining a high index of suspicion is essential, especially in patients with recent trauma or immunocompromising conditions. As research progresses, refining diagnostic criteria and treatment protocols will be vital for improving patient outcomes. Ultimately, a collaborative approach emphasizing education, surveillance, and resource allocation will enhance the management of tropical pyomyositis and mitigate its public health impact.

9. Practice points

1. Maintain a high index of suspicion for tropical pyomyositis in patients presenting with unexplained muscle pain and fever, particularly those with a history of recent trauma or immunocompromising conditions.
2. Prioritize the aspiration of abscesses for microbiological confirmation, including Gram staining and cultures, since blood cultures often yield low positivity rates in pyomyositis cases.
3. Initiate empiric antibiotic therapy based on patient demographics and clinical severity, adjusting based on culture results; consider broad-spectrum coverage for immunocompromised patient

4. Implement prompt source control measures, such as drainage of abscesses, and consider surgical debridement in cases of extensive muscle necrosis or compartment syndrome to improve patient outcomes.

10. Research agenda

- Investigate immune responses in immunocompetent vs. immunocompromised patients, focusing on cytokine profiles, immune cell function, and genetic predispositions.
- Characterize clinical presentations, including atypical and rapidly progressing forms across different demographics and immunological statuses.
- Assess rapid diagnostic techniques like GeneXpert and next-generation sequencing for pathogen identification and resistance profiling.
- Facilitate multinational cohort studies and RCTs to improve diagnostic and management strategies across diverse regions.
- Study the impact of malnutrition, vitamin deficiencies, and glucose control on treatment outcomes.

CRediT authorship contribution statement

Praveen Kumar Tirlangi: Writing – review & editing, Writing – original draft, Methodology, Formal analysis, Conceptualization. **Anjely Sebastian:** Writing – review & editing, Conceptualization. **Mukhyaprana Prabhu M:** Supervision, Conceptualization.

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