PST PEDIATRIC SURGERY IN TROPICS

Pediatric Surgery in Tropics
2024 (Jan-March); Volume 1, Issue 1: Pages 36-46
DOI: 10.5281/zenodo.10648219

Tropical surgery series

Tropical Pyomyositis

Venkatachalam Raveenthiran

Department of Pediatric Surgery, Government Cuddalore Medical College, Chidambaram 608002, Tamilnadu, India.

Keywords
Intramuscular Abscess
Myositis
Bacterial infection

Abbreviations
PVL - Panton-Valentine leukocidin
SA - Staphylococcus aureus
TPM - Tropical Pyomyositis

Abstract

Tropical pyomyositis (TPM) is primarily an inflammatory disorder of skeletal muscles that is characterized by coagulative necrosis and superadded bacterial infection. It is common in tropical weather but not necessarily in a tropical country. Staphylococcus is the most common causative organism. TPM has 3 clinical stages. Local signs of inflammation such as redness and warmth are often deceptively missing. Streptococcal TPM is more aggressive disease. Mortality of stage-2 TPM is less than 2% while that of stage-3 approaches 20%. This article is a descriptive review of recent advances in this neglected tropical disease.

INTRODUCTION

Tropical pyomyositis (TPM) is primarily an inflammatory disorder of skeletal muscles that is characterized by coagulative necrosis and superadded bacterial infection. This definition excludes other suppurative infections of skeletal muscles that are secondary to penetrating injuries, osteomyelitis, septicemia or spread of infection from adjacent organs. It also excludes intermuscular abscesses, specific granulomatous infections such as tuberculosis and clostridial myolysis.[1] As its name implies, TPM is common in tropical climate; but not necessarily in tropical countries.

HISTORY

In 1858, Gelle of France, described the first known case of TPM in his paper entitled "Suppurative myositis following muscle fatigue".[2] Subsequently De Salvia (1866) from Brazil[3] and Scriba (1885) from Tokyo[4] reported additional cases. In 1904, Miyake of Japan[5] established its pathogenesis by animal experiments. Interestingly, all these seminal papers of a disease named 'tropical' had come from temperate countries.

NOMENCLATURE

TPM is known by a variety of synonyms. (Box 1) Native Africans called it Bungpagga disease or Bungura.[6] Rudolph Virchow is credited for coining the most popular term 'Tropical pyomyositis'.[1,7] In addition to these generic terms, site specific names such as ilio-psoas abscess, thigh or leg abscess and abscess of rectus abdominis are also frequently used. Some authors prefer 'Non-tropical Pyomyositis'[8] or 'Temperate Pyomyositis'[9] over TPM when the disease occurs in temperate countries. Such hypercritical semantic perfection is unnecessary for two reasons: (1) The adjective 'tropical' may mean 'tropical weather' rather than 'tropical geographic location'. High humidity combined with high environmental temperature appears to be essential for the

pathogenesis of TPM. For example, TPM is rare in Middle East countries with hot arid climate.[10] By corollary, TPM can occur in temperate countries when tropical weather prevails there. (2) Traditionally diseases are named on the basis of their high prevalence in a particular geographical locality. Rocky Mountain spotted fever, Kyasanur forest disease and Minamata disease are named with geographic identity, although they can occur at other places.

Box 1: Synonyms of Tropical Pyomyositis

Acute suppurative myositis
Bungpagga disease (Bungura)
Epidemic abscess
Myositis infectiosa
Myositis purulenta tropica
Myositis tropicans
Non-tropical pyomyositis
Primary intramuscular abscess
Primary pyomyositis
Primary suppurative (bacterial) myositis
Purulent infectious myositis
Pyomyositis tropicans
Spontaneous bacterial myositis
Suppurative myositis
Tropical myositis
Tropical skeletal muscle abscess

ETIO-PATHOGENESIS

Skeletal muscles are relatively immune to infections, because myoglobin in them avidly binds elemental iron that is essential for bacterial growth.[9] This is supported by the fact that intramuscular abscesses are extremely rare even in frank septicemia.[11,12] In a series of 201 cases of staphylococcal bacteremia, none developed TPM.[11] Among 327 cases of fatal staphylococcal sepsis, [12] there were only 2 cases of TPM, even which appears to be the cause rather than the effect of septicemia. Intrigued by these facts, Miyake[5] conducted rabbit experiments by intravenous injection of sublethal doses of *Staphylococcus aureus (SA)*. Intramuscular abscess did not occur unless the muscle was made vulnerable by prior trauma such as pinching or electrical stimulation. It was hypothesized that muscle injury liberates sequestrated iron from myoglobin. Iron needed for microbial growth is also likely to be released from hemoglobin of traumatic intramuscular hematomas. Miyake's experiment proved that two concomitant factors are essential to cause TPM: (1). Presence of bacteremia, (2) A pre-existing muscle injury or defect that localizes the circulating bacteria. Miyake's conclusion is consistent with Knudson's two-hit hypothesis of pathogenesis which says that two concomitant factors are essential to cause any disease. Several such predisposing factors have been identified. (Box 2)

In tropical climates, high salt content of sweat and its inhibitory effect promotes selective colonization of skin with SA. This may explain as to why TPM is common in hot humid periods. Predisposing muscle damage may be traumatic or non-traumatic. Severity of injury may vary from unrecognizable subtle tear of fibers to overt penetrating injuries. Subclinical injuries as a result of strenuous exercise or sports activities occurring in 5-39% of TPM may actually be ignored as 'muscular fatigue'.[13, 14, 15]

Skeletal muscle damage may also occur in dietary deficiencies and infectious diseases. In Japan, the incidence of TPM dropped with the disappearance of dry-beriberi which is known to cause hyaline degeneration of muscles. TPM is also common in areas where polished rice devoid of thiamine is eaten. By animal experiments, Osawa confirmed the role of vitamin B_1 deficiency in the etiology of TPM.[16,17]. In Uganda, protein energy malnutrition and muscle wasting was noted in 94% of TPM.[18] Tiny intramuscular hematomas of vitamin-C deficiency (Scurvy) was suspected to favor bacterial invasion; but it could not be proved.[17,19] Although deficiencies of Vitamin E, Vitamin A and selenium were shown to cause TPM in cattle, none is proved in human beings.[14, 17, 19] In Japan, TPM is common during the seasons of sweet potato cultivation. In India, it is common during the

Box 2: Predisposing factors of Tropical Pyomyositis

Source infection of bacteremia

- Tropical weather favoring selective colonization of the skin with *SA*
- Pyoderma (e.g. boils, eczema, chicken pox, paronychia)
- Otitis media
- Pneumonia
- Traumatic wounds
- Crohn's disease, Ulcerative colitis

***I*mmuno-compromised state that promotes bacteremia**

- Cancers (e.g. Leukemia, lymphoma) and their chemotherapy
- Type-1 Diabetes mellitus
- HIV infection
- Immunosuppressive / myelosuppressive medications (e.g. Corticosteroids, post-transplant medications, anticancer drugs)
- Chronic liver or renal failure
- Malnutrition
- Intravenous drug abuse
- Agammaglobulinemia
- Congenital immune deficiency disorders (e.g. C_3 complement deficiency, Myeloperoxidase deficiency, IgM deficiency)
- Myelodysplasia
- Atopic dermatitis

Muscle defects that facilitate localization of circulating bacteria

- Hemoglobinopathies (Beta-Thalassemia, Sickle cell anemia)
- Dermatomyositis
- Polyangitis
- Connective tissue disorders (e.g. Rheumatoid disease, systemic sclerosis, Systemic lupus erythematosis, Felty's disease)
- Syphilis
- HIV myositis
- Viral myositis of exanthematous fevers (e.g. Influenza, coxsackie virus B, measles, herpes, arenavirus, picornavirus, arbovirus, leptospirosis)
- Blunt trauma (e.g. bicycle injuries and sports injuries)
- Penetrating injuries (e.g. Intramuscular injections and surgical incisions)
- Strenuous exercise, sports injuries, muscle fatigue
- Intramuscular parasites (e.g. *Trichuris*, *Cysticercus*, *Ancylostoma*, *Dracunculus*, Filarial worm, *Trypanosoma*, *Toxocara*)

SA - Staphylococcus aureus; HIV - Human Immunodefeciency Virus; IgM - Immunoglobilin-M

harvesting season of rice and wheat. In New Guinea and Uganda, TPM is common after pork feast. From these observations, Shepherd[14] hypothesized that abrupt change in dietary habit may precipitate bacteremia by altering the colonic flora. High quality evidences are missing to support this hypothesis.

Several infectious diseases are known to cause pre-disposing muscle damage of TPM. Coinciding

of mosquito breeding season, malarial epidemic and TPM outbreak raised a suspicion that TPM could be a vector born muscle pathology.[14,17] Zenker's degeneration myopathy of typhoid and myopathy of human immuno-deficiency virus (HIV) are incriminated in the pathogenesis of TPM.[14,20] The association between HIV and TPM appears to be real with an odds ratio of 4.82.[20] Eosinophilia, which is unusual in acute bacterial infection, is seen in 5% of TPM. This prompted a search for parasitic etiology. In 7-63% of TPM patients stool was positive for *Ancylostoma* eggs.[21] Intra-muscular helminthes such as the hookworms, *Trichuris*, cysticercus and *Dracunculus* were thought to render the muscle susceptible for bacterial invasion.[17,21,22] However, microscopic examination of affected muscle or pus did not show any evidence of worm infestations. Often, a predisposing factor may not be easily evident.[17]

Exact mechanism, as to how the circulating bacteria get localized at the site of muscle damage, is not known. Trogocytosis is recently proposed to play a role. It is a phenomenon by which circulating lymphocytes bind bacteria by immune complex conjugation and transmit them to the recipient myocytes.[15,23] Increased risk of TPM in patients receiving monoclonal antibodies such as certolizumab, tocilizumab and infliximab may be attributed to immuno-complex conjugation analogous to trogocytosis.[26]

Clinical severity of TPM is linked to Panton-Valen tine leukocidin (PVL), an exotoxin secreted by certain strains of *Staphylococcus* for their survival advantage.[24] It causes leukocyte-mediated tissue destruction and necrosis.[25] PVL, by binding CD45 and C5a receptor, causes lysis of granulocytes and release of proteolytic enzymes which leads to autodigestion of tissues.[26] PVL is now recognized as a critically essential factor in the pathogenesis and outcome of TPM in tropical countries.[24] PVL positive SA cause severe disease and leads to prolonged hospitalization.[27] PVL positive SA can cause TPM even without antecedent muscle damage.[27]

DEMOGRAPHY

True TPM is relatively rare even in tropical countries.[14] It has been reported from almost all countries, with increased frequency from tropical areas.[15] A history of recent travel to a tropical country is obtained only in 9% of cases.[9,17] Its incidence varies from 1-in-1000 (tropics) to 1-in-5000 (non-tropics) population per year.[21] TPM constitutes 1-4% of all admissions in African hospitals.[21] In Ugandan hospitals as many as 1000 cases per year were seen.[14] Within North America it is more common in tropical belt (e.g. Mexico and Texas) than in Canada or Iowa.[25] There are some data to suggest that the incidence is falling in tropics while increasing in temperate countries. For example, annual incidence of TPM in Mulago Hospital, Kampala fell from 250 to 71 during 1948 to 1961.[28] On the contrary, its incidence in an Australian hospital increased from 2.04 cases per 10,000 emergency admission in 2002, to 8.7 cases in 2012 (with a peak of 13.5 cases in 2008).[29] Increased awareness about TPM, better diagnostic imaging, effect of global warming, ease of international travels and antibiotic misuse could be the reason for this changing incidence.

Slight male preponderance of TPM in tropics (M:F - 1.4:1 to 2.3:1) is probably attributable to gender related bulkiness of skeletal muscles or increased chances of sports injuries in boys.[25,30] In non-tropical areas, male-female ratio is as high as 4:1.[31] Cook noted a chronological trend of narrowing sex-ratio. He found male-female ratio of 5.5:1 in 1948 which became 2.7:1 in 1961.[28]

The two peaks of age incidence are 2-9 years and 20-40 years.[10,14] In pediatric age group, Royston noted 51% occurring between 3-6 years of age while 31% occur between 9-12 years.[32] No age

including neonatal period is immune to TPM.[33,34] The youngest patient reported in literature was a 6-days-old newborn.[33] Fewer than 7 cases have been reported in neonates.[33,35]

Different races appear to vary in their susceptibility to TPM, although no genetic correlation could be proved. Seventh Day Adventist of Solomon Islands are said to be immune to TPM as they do not eat pork.[14,36] In tropics, Caucasians migrants are more affected than natives.[14,17] Even in the same race different patterns of dietary habit is said to be associated with varying frequency of TPM. For example, among Africans incidence of TPM is high in Eastern Uganda where the staple diet is cassava and sweet potato while it is low in Western Kenya where maize is the main course.[14]

TPM is common during July to October when humidity is high.[30] It coincides with harvesting season of rice and guinea corn.[25,37] It is rarely seen above an altitude of 4000 feet from sea level.[37]

PATHOLOGY

Involvement of single muscle is more common than multifocal lesions (10-40%).[21,22,25] Right handedness of majority population and hence the increased risk of subclinical injury to muscles could be the reason behind higher incidence of TPM on the right side (R:L - 3:2).[1,15,17] Frequency of individual muscles affected is directly proportional to the bulkiness of the muscle and to strenuous activities.[21,25,30,38](Fig 1) However, even small muscles are not spared. Shepherd cited a case of TPM of orbital muscles that presented as proptosis.[14] The amount of pus drained from a single lesion may be as high as 300 ml to 2 liters.[21,26] Despite bacterial nature of the infection, regional lymph nodes are seldom enlarged.[14]

Histologically the affected muscles show Zenker's degeneration with infiltration of acute inflammatory cells including monocytes and eosinophils. Electron microscopy of the affected muscles show degenerative changes in apparently healthy looking areas well away from the lesion.[39] These

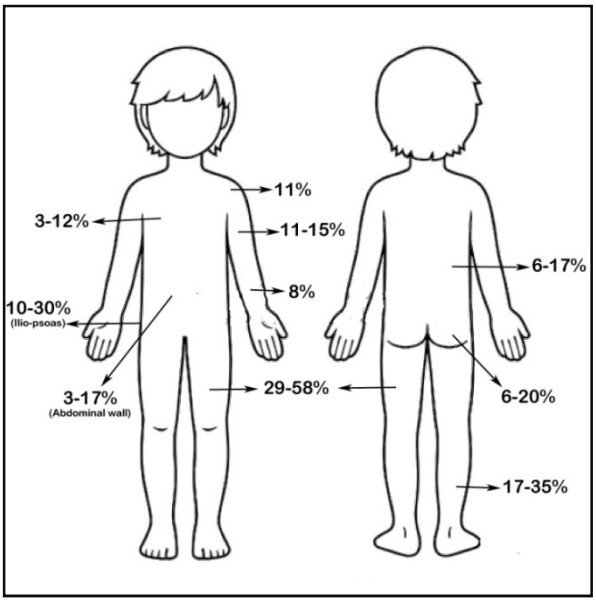

Fig 1: *Anatomical distribution of tropical pyomyositis in children (Data from Chinda[38] Ansaloni[21] Verma[25,30])*

changes are not easily appreciated in light microscopy.[39] Even distant muscles such as the myocardium may show submicroscopic changes.[39] This indicates that TPM is primarily a degenerative disease of muscle to which hematogenous bacterial infection is superadded.

MICROBIOLOGY

Staphylococcus aureus (*SA*) is the major offender in 70-90% cases.[40] Phage type-2 SA, recovered from 60% of TPM, was originally thought to be of pathogenic significance;[40] but subsequent studies did not support this hypothesis. Other microbes occasionally reported in TPM include *Streptococcus* (5-10%),[36] *Pneumococcus,*[41] *Enterococcus,*[42] *Klebsilella,*[43] *Proteus, Escherichia,*[44] *Salmonella,*[45] *Citrobacter, Enterobacter, Morganella, Haemophilus, Aeromonas, Serratia, Pseudo-*

monas, Nocardia and *Yersinia*.[15] Often mixed infection of anaerobes such as *Clostridium difficile*, *Peptostreptococci , Bacteroides fragilis, Fusobacterium*, *Prevotella* and *Veillonella* is seen.[15,46] Although other microbes such as *Mycobacterium tuberculosis*, *Mycobacterium avis*, *Candida albicans* and *Cryptococcus neoformans* have been reported in the literature,[15] by strict definition those specific infections should not be included under TPM.

In recent years, an increasing proportion (46-63%) of *Staphylococcal* isolates are community acquired; of which 75% are methicillin resistant SA (MRSA) and 25% are methicillin susceptible SA (MSSA).[27,30] PVL positive strains and USA300 clones affect relatively younger patients, have shorter duration of bacteremia and cause larger abscess than PVL negative SA.[27]

CLASSIFICATION

TPM is classified in a number of ways such as 'primary *versus* secondary'; 'axial (paraspinal or thoraco-abdominal) *versus* appendageal (limbs)'; and 'focal *versus* multiple'. These classifications are not popular as they lack clinical significance. However, tropical TPM significantly differ from non-tropical TPM.[9] In temperate countries TPM occurs mostly in immuno-compromised adults (60%) while in tropics it predominantly affects healthy young children. Male preponderance in temperate climate (4:1) is much exaggerated than that of tropics (1.4:1). Rarity of TPM in temperate countries often leads to diagnostic delays.[47] The usual delay in diagnosis is 12-16 days while it may be as long as 3 months.[21] Christin reported North American patients with an unusual delay of 1 year.[17] Blood cultures are more often positive (35%) in temperate countries than in tropical centers (5%).[1,9] *Staphylococcus* is more common in tropics (90%) while it accounts for only 75% in temperate countries.[9] PVL strains are more often seen in tropical TPM than in non-tropical TPM.[48]

CLINICAL FEATURES AND STAGING

Three distinct clinical stages of TPM have been described.[1,17,21,25,31] They are as follows:

Stage 1 (Stage of invasive myositis)

In this stage bacterial invasion of muscle causes features of inflammation which include low grade fever, local muscle pain, fusiform diffuse swelling of the involved muscle, muscle spasm and flexion deformity of the associated joint. This stage usually lasts for 7-21 days. Symptoms are often less dramatic and vague (e.g. dull muscle ache, anorexia, general malaise and cramps). The affected muscle may be woody hard in consistency due to tense edema within investing fascia (epimysium). Tenderness of the involved muscle may be mild or absent. As the inflammation is deep to deep-fascia, skin over the lesion may look deceptively normal.(Fig 2) Diagnostic needle aspiration would be negative. Very few patients (<5%) present at this stage and they are often misdiagnosed due to their non-specific clinical features.[31]

Fig 2. *Clinical appearance of tropical pyomyositis of the right thigh. Overlying skin may deceptively look normal. (Representative illustration generated by AI technology using 'wepik' software)*

Stage 2 (Stage of suppuration / abscess)

In this stage the localized infection progresses to suppuration of muscle resulting in an abscess. Inflammatory signs will be more prominent (e.g. high grade fevers with chills, local tenderness, severe crippling of muscle function and sick

appearance). Pseudofluctuation of overlying muscle belly should not be mistaken for pus collection. It lasts for 1-12 days. Diagnostic aspiration with a wide bore needle would yield pus. Overlying skin may still look deceptively normal. Rarely, the skin may be warm, red, edematous and shiny. About 90% of patients present at this stage.[31]

Stage 3 (Stage of dissemination)

Clinical features of this stage are similar to stage-2 with the addition of systemic manifestations of sepsis and circulatory shock. The muscle infection may spread to adjacent and distant organs. Mortality is usually 2-10%. About 2-3% of children present at this stage. [31]

DIFFERENTIAL DIAGNOSIS

Vague symptoms of stage-1 are easily mistaken for a variety of conditions including non-specific myalgia, sciatica, backache, viral fever, poliomyelitis, dermatomyositis, toxoplasmosis, deep vein thrombosis, thrombophlebitis, trypanosomiasis and Perthes disease.[15,21,25] Morison's aphorism says, "Cellulitis occurring in children is never primarily in the cellular tissues but secondary to an underlying bone infection".[49] Hence, many a TPM are mistaken for osteomyelitis or vice versa. Nearly 32% of children suspected to have septic arthritis of hip turned out to be TPM. In true arthritis, joint movements are restricted in all directions while in TPM it is restricted in only one axis.

Swelling of stage-2 lesion may be mistaken for muscle hematoma, soft tissue sarcoma, nodular fasciitis or infantile fibromatosis.[50] Conversely rhabdomyosarcoma has been mistaken for TPM.[51] Psoas abscess of right side may mimic acute appendicitis or appendicular abscess.[52]

INVESTIGATIONS

Blood investigations[21,25,14] may show leucocytosis (49%), eosinophilia (5%), elevated ESR (91%) and increased C-reactive protein level (93%). Blood cultures are rarely (5%) positive. They are more likely to be positive if the organism is *Streptococcus* rather than *Staphylococcus*.[17] Pus cultures are positive in 10-40% cases.[25,30] High rate of negative culture is attributed to partial treatment with antibiotics prior to sampling. Recently, polymerase chain reaction technique has been introduced to overcome this problem and to increase the diagnostic yield.[15, 53] Coexisting HIV in 31% and intestinal hookworm in 17-63% should also be investigated.[20,21]

Inflammatory obliterated muscle planes in plain radiographs are highly non-specific.[54] However, x-rays may be useful in excluding osteomyelitis as a differential diagnosis. Ultrasonography (USG) is useful in differentiating stage-1 and stage-2 of TPM.[32,55] Hypo-echoic shadows within the muscle and floating debris or gas bubbles are diagnostic of abscess. However, it must be remembered that sometimes edematous muscle of stage-1 TPM may sonographically mimic an abscess. USG is often useful in guiding needle aspiration and in excluding septic arthritis.

Computed Tomography may show low attenuation of abscess collection, gas bubbles within the muscle and peripheral contrast enhancement of abscess cavity. Point-of-care ultrasonography (POCUS) is comparatively better than CT in diagnosing TPM.[56] Magnetic resonance imaging (MRI) is more sensitive than USG and CT scan.[57,58,59,60] It is claimed to effective in diagnosing TPM within 3-5 days of onset.[57] Hyperintense signals in T_2 weighted images and hyperintense rim on enhanced T_1 weighted images are characteristic of intramuscular abscess. Often the lesion is larger in MRI than that is appreciated clinically. Gallium scintigraphy[61,62], although a sensitive method, lack precision and specificity. Positron emission tomography (PET-CT) or Single photon emission computed tomography (SPECT) scans are said to be useful in multiple or occult

lesions.[63,64] In resource poor settings, the diagnosis of abscess is often confirmed by repeated diagnostic aspiration with a wide bore (16G or larger) needle rather than by imaging.

Despite extensive myolysis, muscle enzymes such as lactate dehydrogenase (LDH), creatine phosphokinase (CPK), and aldolase are not elevated. If they are elevated, it may suggest either multifocal TPM[65] or an alternate diagnosis such as polymyositis. In school going children TPM occurring as a complication of influenza is associated with marked rise in CPK.[30] Electromyography (EMG) may show short duration, low amplitude polyphasic potentials; but it is not recommended in fear of spreading the infection.

TREATMENT

Stage-1 TPM is treated with appropriate antibiotics that can cover *Staphylococcus*. Initially antibiotics are given intravenously for 7-10 days followed by oral antibiotics. Antibiotics are usually needed for prolonged period (average 3 to 4 weeks with a maximum of 94 days reported by Christin).[15,17,35] Combination therapy is preferable over monotherapy to avoid development of bacterial resistance. Cloxacillin, nafcillin, oxacillin, flucloxacillin, ceftazidime, cefepime, piperacillin-tazobactam, carbapenem, aminoglycosides, third generation cephalosporins, clindamycin, penicillin are the commonly used drugs. Anaerobic coverage by metronidazole is given during the first few days of therapy. Vancomycin, linezolid, teicoplanin and daptomycin are reserved for Methicillin or Vancomycin resistant *SA* (MRSA or VRSA respectively). Quinolones (ciprofloxacin, ofloxacin), doxycycline and fusidic acid are rescue drugs that are rarely used in older children. In case of multidrug resistant organism, rifampicin may be considered. There are some evidences to suggest that macrolides, lincosamides, rifampicin or oxazolidinone inhibit production of PVL, thereby facilitate quicker recovery.[66] In stage-3 TPM intravenous immunoglobulins may reduce the mortality of toxic shock syndrome.

When pus is localized, surgical drainage is essential in addition to antibiotics. Although occasional papers[67] reported success with antibiotics alone without surgical drainage of pus, author of this review does not subscribe to that view. The standard surgical dictum *"Ubi pus, ibi evacua"* should never be violated. Usually open drainage is preferred over percutaneous tube drainage or image-guided therapeutic aspiration. Multiple loculi of the abscess cavity cannot be effectively broken by percutaneous drainage techniques. A drainage tube is left *in situ* as long as it drains. Surgical access is determined by the location and size of abscess. Ilio-psoas abscesses are drained either by transabdominal extraperitoneal route or by laparoscopy.[68]

Mutilating surgeries[69,70] such as the excision of rectus femoris and amputation are no longer required. Such radical operations may be occasionally needed especially in severe streptococcal TPM.[70]

Failure of clinical symptoms to resolve with antibiotic treatment may be due to incomplete drainage of pus, occult abscess elsewhere, antibiotic resistance or drug-induced fever. Endpoints of antibiotic therapy include resolution of clinical symptoms, fall of erythrocyte sedimentation rate (ESR) to normal level, return of C-reactive protein to normalcy.

Topical nasal application of mupirocin cream is sometimes used as a therapeutic adjunct or prophylaxis to eradicate nasal carrier state of *Staphylococcus*.[1]

COMPLICATIONS AND OUTCOME

Although a majority of patients recover uneventfully without any significant residual deformity or recurrence, mortality of stage-2 TPM is 0.8-2%

and that of stage-3 is 2-23%.[10, 20] Streptococcal TPM is more serious than staphylococcal TPM.[34] PVL positive SA causes more deaths than PVL negative infections.[71] Average hospital stay is 14 days (range 7 days to 90 days).[26] Recurrences are exceptionally rare. Complications of TPM vary between 9 to 66%.[15] They include osteomyelitis (5-41%), septic or reactive arthritis (7-25%), pneumonia (18%), septicemia (3-5%), toxic shock syndrome (2%), multi-organ dysfunction syndrome (<1%), spinal epidural abscess, pyopericardium, myocarditis or endocarditis, meningitis, brain abscess and empyema thorax.[25] Osteomyelitis may be contiguous (adjacent bone) or hematogenous (distant bone). Compartment syndrome and compression neuropathy [72] have also been reported. Myoglobinuria due to extensive myolysis may precipitate renal failure. [14]

(Endnote: Peculiarities of site specific pyomyositis will be addressed separately in this series. Randomized controlled trials are non-existent and the only one meta-analysis available examines the association of HIV and TPM. Thus, recommendations made in this article are derived from case series and isolated case reports)

REFERENCES

[1] Chauhan S, Jain S, Varma S, Chauhan SS. Tropical pyomyositis (myositis tropicans): current perspective. Postgrad Med J. May; 80(943):267-70.

[2] Gelle G. Myosite suppuree suite de fatigue musculaire. Bull Soc Anat Paris 1858; 33: 414-7.

[3] De Salvia B. Myosita suppurada dos musculos rectos do abdomen. Rev Curso Prac Theor Facul Med Rio de Janeiro 1866; 3: 49-52.

[4] Scriba J. Beitrag zur aetiologie der myositis acuta. Deuts. Z. Chir. 1885; 22: 497–502.

[5] Miyake H. Beitrag zur Kenntniss des sogenannten Myositis infectiosa. Mitteilungen zur Grenzgebiet Der Medizin Und Chirurgie 1904; 13:155-98.

[6] Patton CR. Preliminary note on a disease called "Bungpagga", found in the northern territories of the Gold Coast. Br Med J. 1916 Apr 1; 1(2883):483-4.

[7] Traquair RN. Pyomyositis. J Trop Med Hyg. 1947 May; 50 (5): 81-9..

[8] Messerli FH. Tropische und nicht-tropische Pyomyositis. Schweiz Med Wochenschr. 1972; 102: 1383-6.

[9] Small LN, Ross JJ. Tropical and temperate pyomyositis. Infect Dis Clin North Am. 2005 Dec; 19(4): 981-9.

[10] Abdulla MA, Sanie A. Tropical pyomyositis in Saudi Arabia. Ann Trop Paediatr. 1982 Mar; 2(1): 45-6.

[11] Faber V, Jessen O, Rosendal K, Eriksen KR. Staphylococcal bacteraemia. Clinical and bacteriological observations in 201 cases. Br Med J. 1960 Dec 24; 2 (5216): 1832-6.

[12] Smith IM, Vickers AB. Natural history of 338 treated and untreated patients with staphylococcal septicaemia (1936-1955). Lancet. Lancet. 1960 Jun 18; 1(7138): 1318-22.

[13] Unnikrishnan PN, Perry DC, George H, Bassi R, Bruce CE. Tropical primary pyomyositis in children of the UK: an emerging medical challenge. Int Orthop. 2010 Feb; 34 (1): 109-13..

[14] Shepherd JJ. Tropical myositis: is it an entity and what is its cause? Lancet. 1983 Nov 26; 2(8361):1240-2.

[15] Habeych ME, Trinh T, Crum-Cianflone NF. Purulent infectious myositis (formerly tropical pyomyositis). J Neurol Sci. 2020 Jun 15; 413: 116767.

[16] Engel D. Tropical pyomyositis, a thiamine-deficiency disease. Med Hypotheses. 1981 Mar; 7(3): 345-52.

[17] Christin L, Sarosi GA. Pyomyositis in North America: case reports and review. Clin Infect Dis. 1992 Oct; 15 (4): 668-77.

[18] Kitara DL, Bwangamoi PO, Wabinga H, Odida M. High prevalence of malnutrition among the above thirteen with primary pyomyositis in Northern Uganda. Br J Med (Med Res). 2015; 6(8): 814-822.

[19] Idoko JA, Jimoh O, Onyewotu II. Has lack of vitamin C any role in the aetiology of tropical pyomyositis? J Infect. 1990 Jul; 21(1): 7-9.

[20] Ngor C, Hall L, Dean JA, Gilks CF. Factors associated with pyomyositis: A systematic review and meta-analysis. Trop Med Int Health. 2021 Oct; 26(10): 1210-1219.

[21] Ansaloni L. Tropical pyomyositis. World J Surg. 1996 Jun; 20 (5): 613-7.

[22] Anand SV, Evans KT. Pyomyositis. Br J Surg. 1964 Dec; 51: 917-20.

[23] Joly E, Hudrisier D. What is trogocytosis and what is its purpose? Nat Immunol. 2003 Sep; 4(9): 815.

[24] Young BC, Earle SG, Soeng S, Sar P, Kumar V, Hor S, Sar V, Bousfield R, Sanderson ND, Barker L, Stoesser N, Emary KR, Parry CM, Nickerson EK, Turner P, Bowden R, Crook DW, Wyllie DH, Day NP, Wilson DJ, Moore CE. Panton-Valentine leucocidin is the key determinant of *Staphylococcus aureus* pyomyositis in a bacterial GWAS. Elife. 2019 Feb 22; 8: e42486.

[25] Verma S. Pyomyositis in Children. Curr Infect Dis Rep. 2016 Mar; 18(4): 12.

[26] Shittu A, Deinhardt-Emmer S, Vas Nunes J, Niemann S, Grobusch MP, Schaumburg F. Tropical pyomyositis: an update. Trop Med Int Health. 2020 Jun; 25(6): 660-665.
[27] Pannaraj PS, Hulten KG, Gonzalez BE, Mason EO Jr, Kaplan SL. Infective pyomyositis and myositis in children in the era of community-acquired, methicillin-resistant Staphylococcus aureus infection. Clin Infect Dis. 2006 Oct 15; 43(8): 953-60.
[28] Cook J. Pyomyositis. East Afr Med J. 1963 Nov: 40: 574-9.
[29] Moriarty P, Leung C, Walsh M, Nourse C. Increasing pyomyositis presentations among children in Queensland, Australia. Pediatr Infect Dis J. 2015 Jan; 34 (1): 1-4.
[30] Verma S, Singhi SC, Marwaha RK, Singhi P, Singh S, Singh M. Tropical pyomyositis in children: 10 years experience of a tertiary care hospital in northern India. J Trop Pediatr. 2013 Jun; 59(3): 243-5.
[31] Drosos G. Pyomyositis. A literature review. Acta Orthop Belg. 2005 Feb; 71(1): 9-16.
[32] Royston DD, Cremin BJ. The ultrasonic evaluation of psoas abscess (tropical pyomyositis) in children. Pediatr Radiol. 1994; 24(7): 481-3.
[33] Falesi M, Regazzoni BM, Wyttenbach M, Wyttenbach R, Bianchetti MG, Riavis M. Primary pelvic pyomyositis in a neonate. J Perinatol. 2009 Dec; 29(12): 830-1.
[34] Maddox JL Jr, Riordan TP, Odom RB. Pyomyositis in a neonate. J Am Acad Dermatol. 1984 Feb; 10(2 Pt 2): 391-4.
[35] Barchi L, Fastiggi M, Bassoli I, Bonvicini F, Silvotti M, Iughetti L, De Fanti A. Pyomyositis associated with abscess formation caused by streptococcus pneumoniae in children: a case report and review of literature. Ital J Pediatr. 2023 Jun 14; 49(1): 73.
[36] Sayer EG. Tropical myositis and muscle abscess. Trans Royal Soc Trop Med Hyg 1930; 23: 385-400.
[37] Taylor JF, Templeton AC, Henderson B. Pyomyositis. A clinico-pathological study based on 19 autopsy cases, Mulago Hospital 1964-1968. East Afr Med J. 1970 Oct; 47 (10): 493-501.
[38] Chinda J, Ameh EA, Chirdan LB. Pyomyositis. In: Ameh EA, Bickler SW, Lakhoo K, et al (ed). Paediatric Surgery: A Comprehensive Text for Africa. Vol-1 Global Help publishers (www.global-help.org). 2016. pp 120-123
[39] Taylor JF, Fluck D, Fluck D. Tropical myositis: ultrastructural studies. J Clin Pathol. 1976 Dec; 29 (12): 1081-4.
[40] Foster WD. The bacteriology of tropical pyomyositis in Uganda. J Hyg (Lond). 1965 Dec; 63(4): 517-24.
[41] Tuerlinckx D, Bodart E, de Bilderling G, Nisolle JF. Pneumococcal psoas pyomyositis associated with complement deficiency. Pediatr Infect Dis J. 2004 Apr; 23 (4): 371-3.
[42] Perez-Rodriguez MT, Sopeña B, Longueira R, Lamas JL, Martinez-Vazquez C. Calf pyomyositis caused by Enterococcus faecalis. QJM. 2011 Jun; 104(6): 527-9.
[43] Wang TK, Wong SS, Woo PC. Two cases of pyomyositis caused by Klebsiella pneumoniae and review of the literature. Eur J Clin Microbiol Infect Dis. 2001 Aug; 20 (8): 576-80.
[44] Fujioka K, Takeuchi S, Tayama T, Takei M, Ono A, Shono M, Shichijo K, Narita T, Kondo S. *Escherichia coli* pyomyositis in a patient with Down syndrome: A case report. ID Cases. 2021 May 9; 24: e01158.
[45] Collazos J, Mayo J, Martínez E, Blanco MS. Comparison of the clinical and laboratory features of muscle infections caused by Salmonella and those caused by other pathogens. J Infect Chemother. 2001 Sep; 7(3): 169-74.
[46] Brook I. Pyomyositis in children, caused by anaerobic bacteria. J Pediatr Surg. 1996 Mar; 31(3): 394-6.
[47] Mignemi ME, Menge TJ, Cole HA, Mencio GA, Martus JE, Lovejoy S, Stutz CM, Schoenecker JG. Epidemiology, diagnosis, and treatment of pericapsular pyomyositis of the hip in children. J Pediatr Orthop. 2014 Apr-May; 34 (3): 316-25.
[48] Hoppe PA, Holzhauer S, Lala B, Bührer C, Gratopp A, Hanitsch LG, Humme D, Kieslich M, Kallinich T, Lau S, Leistner R, Niebank M, Pokrywka A, Ringe H, Schaper AS, Schröder JT, Schwarz C, Staab D, Stegemann MS, Thee S, Varnholt V, von Bernuth H, Weber-Carstens S, Wendt A, Krüger R. Severe infections of Panton-Valentine leukocidin positive Staphylococcus aureus in children. Medicine (Baltimore). 2019 Sep; 98(38): e17185.
[49] Lumley JSP, D'Cruz AK, Hoballah JJ, Scott-Conner CEH. Hamilton Bailey's Demonstrations of Physical Signs in Clinical Surgery. (19 edn) CRC Press, London 2016. p73
[50] Pittam MR. Pyomyositis mimicking soft-tissue sarcoma. Eur J Surg Oncol. 1988 Oct; 14(5): 459-61
[51] Kumar S, Chougle A, Meadows TH. Rhabdomyosarcoma presenting as a calf pyomyositis. J Pediatr Orthop B. 2005 May; 14(3): 226-7.
[52] Wysoki MG, Angeid-Backman E, Izes BA. Iliopsoas myositis mimicking appendicitis: MRI diagnosis. Skeletal Radiol. 1997 May; 26(5): 316-8.
[53] Gabas T, Podglajen I, Cheminet G, Gagnard JC, Wyplosz B. Diagnostic accuracy of 16S rDNA PCR in bacterial pyomyositis: A prospective study. J Infect. 2019 Nov; 79 (5): 462-470.
[54] Pretorius ES, Hruban RH, Fishman EK. Tropical pyomyositis: imaging findings and a review of the literature. Skeletal Radiol. 1996 Aug; 25(6): 576-9.
[55] Matsuda M, Tagaya M, Kikawa Y, Shigematsu Y, Konishi Y, Sudo M. Diagnosis of iliopsoas pyomyositis by ultrasonography and CT. Acta Paediatr Jpn. 1990 Oct; 32 (5): 555-8.

[56] Oduyoye O, Thomas E. Point of Care Ultrasound (POCUS) in Bedside Diagnosis of Pyomyositis. POCUS J. 2022 Apr 21; 7(1): 134-136.
[57] Tharmarajah H, Marks M. Early use of MRI for suspected pyomyositis. J Paediatr Child Health. 2015 Jun; 51 (6): 651-2.
[58] Chotai PN, Hsiao MS, Pathak I, Banh D, Abdou M, Abdelgawad AA. Pediatric pelvic pyomyositis: initial MRI can be misleading. J Pediatr Orthop B. 2016 Nov; 25 (6): 520-4.
[59] Trusen A, Beissert M, Schultz G, Chittka B, Darge K. Ultrasound and MRI features of pyomyositis in children. Eur Radiol. 2003 May; 13(5): 1050-5.
[60] Gordon BA, Martinez S, Collins AJ. Pyomyositis: characteristics at CT and MR imaging. Radiology. 1995 Oct; 197(1): 279-86.
[61] Datz FL, Lewis SE, Conrad MR, Maravilla A, Parkey RW. Pyomyositis diagnosed by radionuclide imaging and ultrasonography. South Med J. 1980 May; 73(5):649-51.
[62] Schiff RG, Silver L. Tropical pyomyositis. Demonstration of extent and distribution of disease by gallium scintigraphy. Clin Nucl Med. 1990 Aug;15(8):542-4.
[63] Harisankar CN, Mittal BR, Kashyap R, Bhattacharya A, Singhi S. (18)F-FDG Positron Emission Tomography in Multifocal Pyomyositis. World J Nucl Med. 2012 Jan; 11 (1): 22-3.
[64] Abdullah ZS, Khan MU, Kodali SK, Javaid A. Pyomyositis mimicking osteomyelitis detected by SPET/CT. Hell J Nucl Med. 2010 Sep-Dec; 13(3): 277-9.
[65] Kallen P, Nies KM, Louie JS, Keller M, Worthen N, Bayer AS. Tropical pyomyositis. Arthritis Rheum. 1982 Jan; 25 (1): 107-10.
[66] Dumitrescu O, Boisset S, Badiou C, Bes M, Benito Y, Reverdy ME, Vandenesch F, Etienne J, Lina G. Effect of antibiotics on Staphylococcus aureus producing Panton-Valentine leukocidin. Antimicrob Agents Chemother. 2007 Apr; 51(4): 1515-9.
[67] Miller NJ, Duncan RD, Huntley JS. The conservative management of primary pyomyositis abscess in children: case series and review of the literature. Scott Med J. 2011 Aug; 56 (3): 1-4.
[68] Singh N, Nelson JM, Sawyer MA. Laparoscopic management of abdominopelvic abscesses in tropical pyomyositis. JSLS. 2002 Jan-Mar; 6(1): 85-8.
[69] Echeverria P, Vaughn MC. "Tropical pyomyositis." A diagnostic problem in temperate climates. Am J Dis Child. 1975 Jul;129(7):856-7.
[70] Comegna L, Guidone PI, Prezioso G, Franchini S, Petrosino MI, Di Filippo P, Chiarelli F, Mohn A, Rossi N. Pyomyositis is not only a tropical pathology: a case series. J Med Case Rep. 2016 Dec 21; 10(1): 372.
[71] Mitchell PD, Hunt DM, Lyall H, Nolan M, Tudor-Williams G. Panton-Valentine leukocidin-secreting Staphylococcus aureus causing severe musculoskeletal sepsis in children. A new threat. J Bone Joint Surg Br. 2007 Sep; 89 (9): 1239-42.
[72] Brik R, Braun J, Bialik V, Zuckerman N, Berant M. Non-tropical pyomyositis in children with report of severe neurological complications. Acta Paediatr Scand. 1989 Mar; 78(2): 331-4.

Address for communication: *Dr. V. Raveenthiran, Email: vrthiran@gmail.com*

Received 20 November 2023
Accepted 15 December 2023

Acknowledgements : None
Conflicts of Interest : None declared by authors
Source of Funding : None
Ethical Concerns : None (Literature Review)

Citation: *Raveenthiran V. Tropical pyomyositis. Pediatr Surg Trop 2024; 1(1):36-46.*