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Uncommon primary Group A streptococcal erector spinae pyomyositis diagnosed as muscle strain in a fit young male

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Abstract

Acute lower back pain in a fit healthy male with recent strenuous physical activity is often attributed to muscle strain. We report a rare case of erector spinae pyomyositis developing in a young and otherwise healthy young adult male that was almost misdiagnosed as muscle strain. Despite admission and close monitoring, diagnosis was only confirmed on a magnetic resonance imaging of the spine later during the hospital stay. Early diagnosis in this case allowed successful treatment with intravenous antibiotics alone, without requiring further surgical drainage or development of further neurological sequelae, common in later stages of erector spinae pyomyositis.

Keywords

Anaesthesia/pain, infectious diseases, orthopaedics/rehabilitation/occupational therapy, pyomyositis, erector spinae, lower back pain, Group A streptococcus

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Introduction

Acute lower back pain (LBP) in a fit healthy male with recent strenuous physical activity is often attributed to muscle strain. Primary pyomyositis (PM) of the erector spinae muscles is a much rarer cause of LBP in this patient population, even with preceding history of trauma.[1–3]

Because of its rarity, as opposed to PM of larger muscle groups like the gluteal or thigh muscles, and its insidious onset with non-specific symptoms in the early stages, erector spinae PM is often diagnosed late when patients manifest neurological symptoms due to associated spinal epidural abscesses (SEA).[3–5] Current literature supports aggressive surgical intervention in the management of late manifestations of erector spinae PM.[5,6]

We report an uncommon case of uncomplicated erector spinae myositis developing in a young and healthy adult male, with presumptive localized muscular strain on a background of pharyngeal streptococcal infection. Despite an initial impression of a simple muscle strain, erector spinae pyomyositis (ESPM) was diagnosed early, and he was successfully treated with intravenous (IV) antibiotics alone and recovered without further neurological sequelae including chronic pain. The rapid identification and treatment prevented evolution and extension of pyomyositis to an epidural abscess, highlighting the importance of early diagnosis and intervention.

Case report

A 30-year-old Chinese male was brought to the Emergency Department (ED) for excruciating left buttock pain. With no previous history of medical or surgical problems, the otherwise fit and young man first developed pharyngitis, presenting with sore throat before developing fever three days later. During this initial period, he engaged in two strenuous gym sessions comprising primarily of isotonic strength training (free weights training, bench press, etc.). He later developed severe ipsilateral back and buttock pain, not amenable to 1650 mg of naproxen and 50 mg of tramadol given over 12 h before he presented to the ED as he had difficulty with ambulation (see Figure 1).

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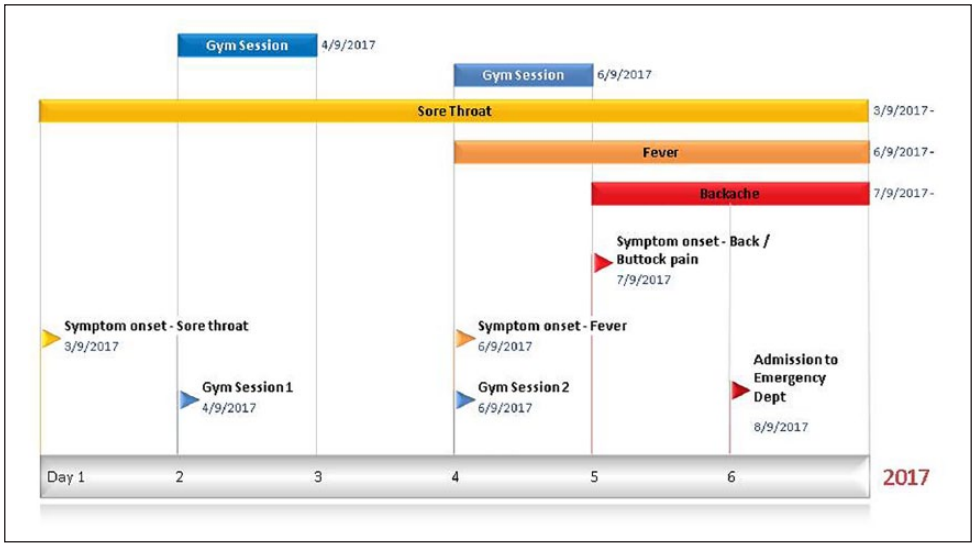


Figure 1. Presenting history and timeline of symptomatology prior to admission.

In the ED, he was initially treated symptomatically for acute back strain after an otherwise unremarkable physical exam was performed. He was planned for discharge after same day outpatient physiotherapy. However, despite additional IM tramadol 50 mg in the ED, he had persistent severe pain and was unable to complete his physiotherapy. The physiotherapist thus referred him back to the emergency physician for further review where he was then admitted to the Short Stay Unit (LBP protocol). His initial blood investigations that subsequently returned revealed raised inflammatory markers: white blood cell (WBC) count 12.2×10^3 cells/μL, C-reactive protein (CRP) 148.6 mg/L and procalcitonin (procal) 5.56 μg/L. Taken together with his history of unresolving back pain, he was admitted to inpatient services the following day for further workup.

Initial plain radiographs and computed tomography (CT) scans of the abdomen and pelvis on 9 September 2017 did not reveal any abscesses or fractures in the pelvis or paraspinal region. A subsequent CT pulmonary angiogram done on 11 September 2017 in the view of desaturation and fever on day 3 of admission did not detect any pulmonary embolism but showed bilateral upper lobe consolidations. Blood cultures done on 9 September 2017 showed Gram-positive cocci in chains and IV Augmentin was switched to IV Penicillin. The subsequent full sensitivity report revealed the offending organism to be pansensitive *Streptococcus pyogenes* and he was kept on IV Penicillin.

A magnetic resonance imaging (MRI) scan of the back done on 12 September 2017 showed myositis of the left erector spinae muscle from L5 to S3 with an associated small central linear rim enhancing area suspicious of an abscess (see Figure 2). Interventional radiology was consulted, but the abscess was deemed too small for surgical drainage. He was continued instead on IV antibiotics until discharge on 21 September 2017 following evidence of down trending temperature and inflammatory markers (see Figure 3). Post discharge, he completed an additional 19 days' worth of IV Penicillin G (4MU q4 H) in the outpatient setting via a peripherally inserted central catheter (PICC).

With respect to pain during his admission, it was severe requiring regular doses of Anarex (paracetamol 450 mg, orphenadrine35 mg) two tablets three times a day (TDS), tramadol 100 mg TDS with additional oxycodone 5 mg every 4 h as necessary (q4 H PRN) for breakthrough pain. Pregabalin 75 mg twice a day (BD) was also started as an adjunct. He was prescribed all the above analgesics upon discharge, and returned 2 weeks later for review in the pain management unit. In view of some degree of residual pain, he was prescribed with pregabalin 75 mg every morning (OM), 150 mg every evening (ON), as well Etoricoxib 90 mg every morning as necessary (OM/PRN) and tramadol 100 mg twice daily as necessary (BD/PRN) for break through pain. At a further review 12 weeks later, he had minimal residual pain that was amenable to physiotherapy and lifestyle modification and did not require further pharmacotherapy.

Discussion

PM is an acute pyogenic infection involving skeletal muscle. Previously seen primarily in tropical regions (tropical pyomyositis), the prevalence of pyomyositis has increased in other regions, with these cases associated more recently with IV drug abuse and immunosuppressed states, such as patients with human immunodeficiency virus (HIV) infection, diabetes mellitus (DM), liver disease or those on immunosuppressant drugs.[1,7]

Pathophysiology

Intact skeletal muscle is normally resistant to bacterial infection due to sequestration of iron by myoglobin, limiting the growth and proliferation of bacteria. Development of

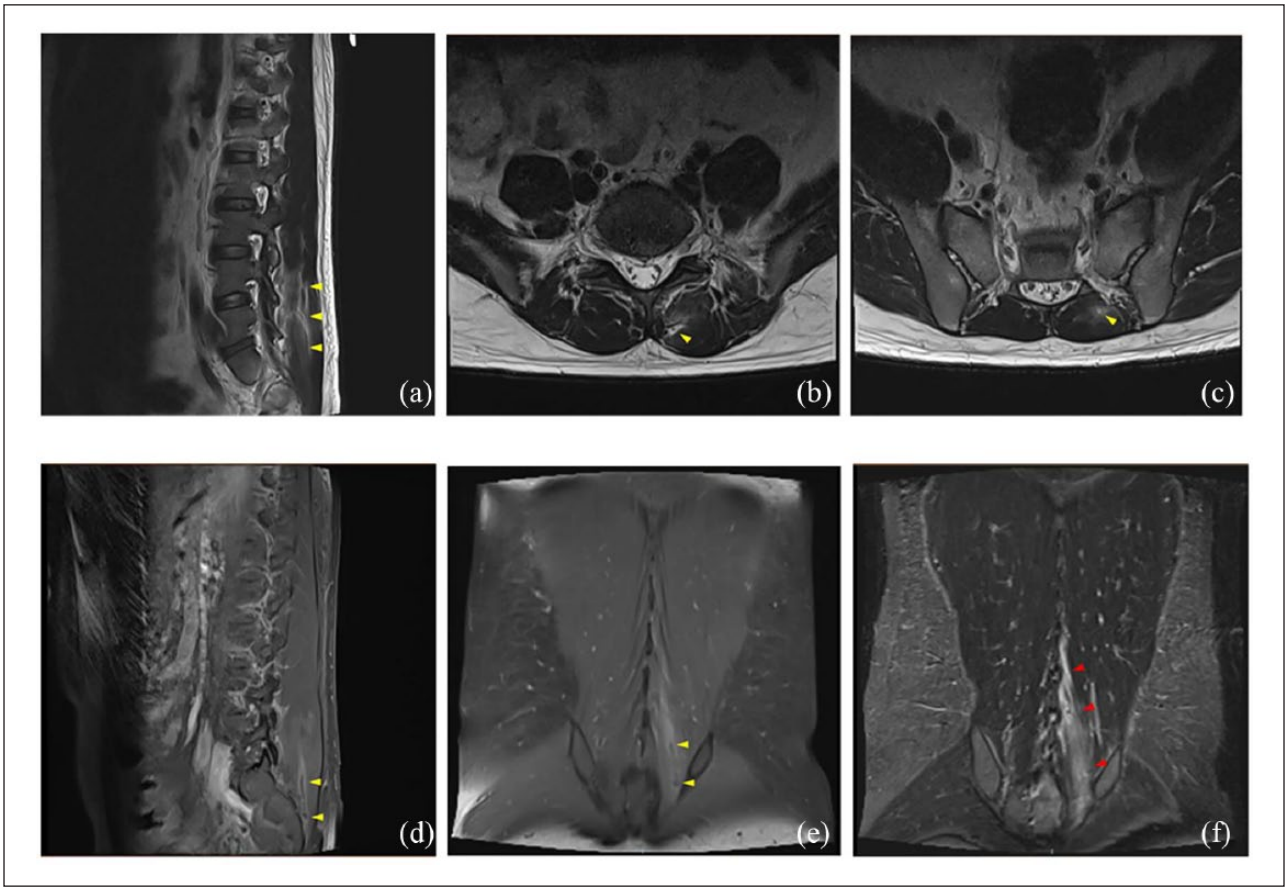
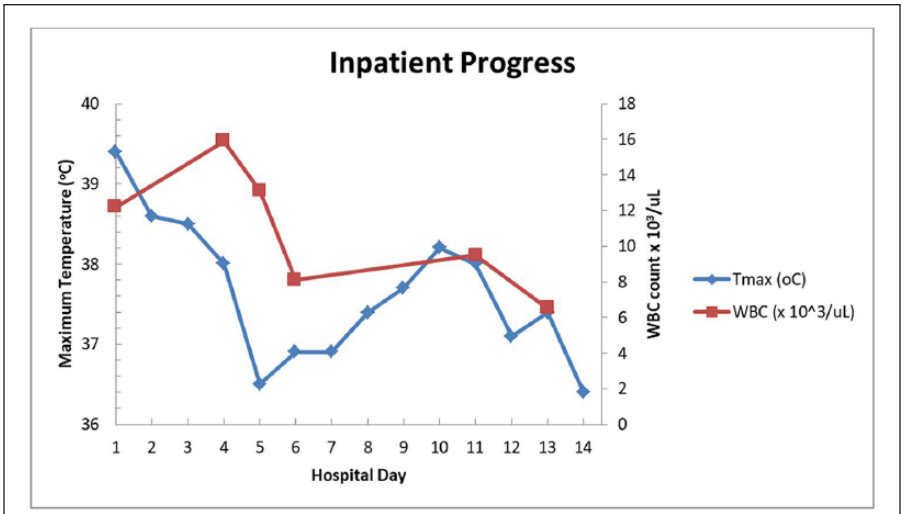


Figure 2. Magnetic resonance imaging (MRI) of the lumbosacral spine showing the erector spinae pyomyositis abscess complex. T2W images in the (a) sagittal and (b) axial planes at L5/S1 and (c) S2 show hyperintense signal in the left erector spinae muscle extending from L4 to S3 with small central area of post contrast peripheral rim enhancement representing the abscess (yellow arrows). T1W images in the (d) sagittal and (e) coronal views demonstrate similar features. Fat suppressed STIR imaging reveals entire extent of pyomyositis involving the erector spinae (f, red arrows).



Day of Admission	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Tmax (oC)	39.4	38.6	38.5	38	36.5	36.9	36.9	37.4	37.7	38.2	38	37.1	37.4	36.4
WBC (x 10^3/uL)	12.2			15.9	13.1	8.1					9.5		6.5	
CRP (mg/L)	148.6			239.8		54.3					30.6		28.2	
Procal (ug/L)	5.56			4.12		1.48					0.32		0.19	

Figure 3. Inpatient clinical and biochemical markers trend during admission.

pyomyositis is hence dependent on preconditions of (a) muscle injury from trauma, overuse or underlying disease; coupled with (b) concurrent bacteremia, resulting in haematogenous seeding into damaged muscle tissue. Risk factors for increased risk of pyomyositis include increased bacteremia in immunosuppressed states (HIV, DM, liver disease, drugs) or due to direct introduction (IV drug use, atopic dermatitis with broken skin), for example.[7,8]

Previous articles have suggested young athletes may develop pyomyositis due to overuse injuries with concurrent asymptomatic bacteremia.[2] In some patients, superficial skin abrasions during physical activity are thought to allow entry of bacteria. *Staphylococcus aureus* remains the most common causative agent in these cases. Group A streptococcal infection, such as in our patient, is postulated to result from haematogenous spread from the pharynx.[9,10] A history of trauma or muscle injury has only been determined in up to 50% of reported cases. The exact inciting event leading to development of pyomyositis remains unknown in most patients.[11]

Infection most commonly involves large muscle groups in the lower extremity such as quadriceps and gluteal muscles.[12] Primary ESPM remains infrequent and poorly diagnosed. Cases are often identified late as a result and are frequently associated with progression, developing into communicating spinal epidural abscess complexes as seen in various published case reports.[3–5] Isolated ESPM is considerably rarer, likely due to the product of increased suspicion and vigilance, together with early imaging to aid diagnosis.

Clinical features

Primary PM typically progresses through three phases.[6] In the initial invasive phase, patients present with generalized, non-specific symptoms such as malaise and low grade fevers. This represents systemic infections and may last up to 4 weeks. Concurrent myalgia or localized swelling may hint towards initial muscle infection and inflammation. Depending on the presenting symptoms, the diagnosis is most often confused with muscle strains, contusions and haematomas, or even osteomyelitis in the presence of fever.

In the second suppurative phase which can occur over the next week or longer, muscle infection leads to ongoing necrosis with resultant collection of pus, in particular with Group A streptococcal necrotizing myositis.[9] Symptoms of worsening and more localized pain are associated with localized swelling and surrounding erythema, especially with superficial muscle infection, allowing for most cases of PM to be diagnosed in this phase. ESPM may however remain masked due to its rarity and relatively deeper location.[3]

If not treated adequately or expediently, PM progresses on towards the third phase with systemic symptoms including sepsis, or with abscess extension beyond the primarily infected muscle. ESPM in particular can progress with direct spread of the abscess anteriorly through the intervertebral foramina or ligamentum flavum towards the epidural space, resulting in debilitating spinal-epidural abscesses (ESPM–SEA complexes). If diagnosed and treated late, the greater the number spinal segments involved, the greater the likelihood of irreversible myelopathy and radiculopathy even after successful surgical drainage.[4,5] Furthermore, streptococcal myositis is also thought to have high case fatality due to toxin production inducing release of cytokines leading to septic shock and eventual multi-organ failure.[9,10]

Diagnosis

Laboratory investigations are generally suggestive of systemic infection, but otherwise non-specific. Leukocytosis with left shift is common except in patients with depressed immune systems. Inflammatory markers such as erythrocyte sedimentation rate (ESR), CRP and procalcitonin are frequently elevated, as was the case in our patient (Figure 3). While non-specific for diagnosis, decreasing trends in inflammatory markers (as demonstrated in our patient) are useful in suggesting if antibiotic therapies are effective while awaiting full microbiological investigations. Regardless, microbiological cultures, when positive for organisms (in up to 30% of cases), are paramount in guiding subsequent antibiotic therapy and may determine eventual outcome.[12,13]

Simple radiographic imaging is non-specific and limited to indirect demonstration of soft tissue swelling. Radionuclide imaging, while sensitive for diagnosis of PM, is unable to delineate anatomy adequately. Ultrasonography may be useful in diagnosis of superficial PM in the later suppurative stages and beyond, as well as aid in aspiration for microbiological investigations.[14] CT imaging is useful once myositis is established, with involved muscle groups showing abnormal decreased attention or accumulation of gas or fluid again in the second phase of PM.

MRI, with or without Gadolinium enhancement, remains the modality of choice for soft tissue infections as it is more sensitive compared to CT in early detection of PM. It also allows for more precise anatomical localization of involved soft tissues, which is potentially useful if further surgical drainage is required.[14,15]

As seen in our patient, early contrasted CT done on day 3 of admission failed to diagnose early PM, and only the MRI done a further 3 days later picked up the diagnosis of ESPM in the early suppurative phase. In combination with the early presentation of severe symptoms, this allowed for early accurate diagnosis and expedient treatment when the disease was only in the early suppurative phase, preventing further progression to ESPM–SEA.

Treatment

While IV antibiotics alone may suffice for stage 1 pyomyositis, current literature supports urgent and aggressive surgical debridement and drainage of muscular abscesses

in later stages of ESPM. This is especially in necrotizing pyomyositis seen in Group A streptococcal infections, as well as PM in unique locations, such as ESPM, where extension of abscess can cause devastating and irreversible neurological injury. Monotherapy with IV antibiotics in early ESPM thus requires greater vigilance and surveillance for failure of therapy.[5,4]

The choice of antibiotics for initial therapy, before cultures and sensitivity studies are available, should cover *S. aureus* and be administered intravenously.[13] In the case of non-tropical PM, the presence of methicillin-resistant *S. aureus* (MRSA) warrants the use of vancomycin, and patients may need concurrent broad-spectrum antibiotics to cover Gram-negative and anaerobic organisms.

For Group A streptococcal PM, high-dose penicillin remains the drug of choice, as was the case in our patient. Concurrent use of clindamycin is recommended in some centres as it is thought to suppress M protein (streptococcal virulence factor) synthesis and toxin production, preventing systemic effects of the infection.[16]

Conclusion

We report a rare case of stage 2 Group A streptococcal ESPM successfully treated with IV antibiotics alone in an otherwise healthy, young male with a clear prodromal phase (pharyngitis) and precipitating muscle injury (from overuse at the gym). Rapid progression and severity of symptoms suggested an initial misdiagnosis of muscle strain and warranted further hospital stay. Primary PM was eventually diagnosed on MRI scan, which early CT imaging failed to do so.

Apart from a protracted recovery period complicated by acute pain requiring oxycodone initially as well as issues of subacute lower back pain requiring low dose weak opioids (tramadol) and anti-epileptics (pregabalin). The patient otherwise recovered well with no obvious neurological sequelae.

Primary ESPM remains a poorly diagnosed entity due to its non-specific symptoms and can rapidly progress to potentially debilitating ESPM–SEA with subsequent neurological impairments. Physicians must maintain a high degree of suspicion especially when symptoms are out of proportion to the presumptive diagnosis.

Declaration of conflicting interests

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Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

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Informed consent

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