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Case Report: Deep Myositis Presenting as Acute Painful Limp in a Child – A Diagnostic Challenge

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Abstract

Deep myositis, also known as pyomyositis, is a bacterial infection of skeletal muscle characterised by localised inflammation and potential abscess formation. Early diagnosis is essential, as its clinical presentation often mimics other serious conditions such as septic arthritis or osteomyelitis, leading to diagnostic uncertainty and potential delays in treatment. We report the case of a 10-year-old girl who presented with acute severe pain in the left thigh and inability to bear weight following a recent febrile illness. Laboratory investigations revealed elevated inflammatory markers as indicated by high levels of C-reactive protein and leukocytosis, while MRI demonstrated oedema involving the deep femoral musculature without abscess formation or bone involvement. The patient was treated with intravenous meropenem and linezolid followed by oral linezolid, resulting in progressive clinical and laboratory improvement. This report emphasises the importance of differentiating this benign entity from septic arthritis and osteomyelitis to avoid unnecessary invasive treatment.

Keywords: NIL

Introduction

Deep myositis or pyomyositis, is a primary bacterial infection of skeletal muscle, most commonly associated with Staphylococcus aureus. However, other organisms, including Streptococcus species and Gram-negative bacteria like Escherichia coli, Klebsiella pneumoniae, etc., may be implicated. [1,2] The condition is usually self-limiting. It progresses through three stages:

1. Invasive stage – diffuse muscle inflammation and oedema without abscess
2. Suppurative stage – abscess formation
3. Late stage – systemic toxicity and complications

The condition is increasingly recognised in children and may follow transient bacteremia, minor trauma, or preceding infection. [3, 4] Early-stage deep myositis presents with localised muscle pain, limp, and elevated inflammatory markers, although it may lack signs such as swelling or abscess formation.

MRI is the gold standard investigation, allowing differentiation from septic arthritis, osteomyelitis, and inflammatory myopathies. Early diagnosis and prompt antibiotic therapy are essential to prevent progression to abscess formation, sepsis, and permanent functional impairment. [5]

Case Presentation

A 10-year-4-month-old girl presented with five days of progressively worsening pain in the left thigh and inability to bear weight. The child was afebrile and hemodynamically stable. She had experienced a fever three days prior following an upper respiratory tract infection. There was no history of trauma, intramuscular injection, or preceding exertion. On physical examination, the child appeared uncomfortable due to pain. The left lower limb was held in an adducted and internally rotated posture, and there was marked tenderness over the anterior and medial aspect of the thigh. Importantly, there was no visible swelling, erythema, increased local temperature, or skin discolouration. Hip and knee movements were painful and restricted, but distal neurovascular examination was normal.

Laboratory investigations over the course of hospital stay are summarised in Table 1.

Table 1: Laboratory investigations throughout the course of hospital stay

Parameter	27 Jan 2026	29 Jan 2026	1 Feb 2026	3 Feb 2026	Unit
Hemoglobin	113	118	114	111	g/L
Packed cell volume	0.365	0.352	0.354	0.338	L/L
Total leukocyte count	9.8	22.3	6.2	4.2	$\times 10^9$/L
Neutrophils (absolute count)	8.3	8.8	5.6	4.4	$\times 10^9$/L
Lymphocytes (absolute count)	1.4	0.9	2.5	4.2	$\times 10^9$/L
Platelet count	284	296	326	304	$\times 10^9$/L
ESR	35	44	41	39	mm/h
C-reactive protein	61.2	177	182	65	mg/L
Procalcitonin	0.05	0.11	0.27	<0.05	µg/L

Laboratory investigations over the course of hospital stay are summarised in Table 1.

Initial laboratory investigations demonstrated elevated inflammatory markers with C-reactive protein of 61.2 mg/L and ESR of 35 mm/hr, while the total leukocyte count was within normal limits with neutrophilic predominance. Serum creatine kinase and procalcitonin levels were normal, and blood cultures remained sterile. Autoimmune screening, including Antinuclear antibody was negative. In view of the acute limp with raised inflammatory markers, imaging was pursued to exclude septic arthritis and osteomyelitis.

Magnetic resonance imaging (MRI) of the left thigh and hip revealed diffuse hyperintense signals within the femoral musculature with adjacent marrow oedema, but there was no evidence of abscess formation, periosteal reaction, cortical destruction, or joint effusion. These findings were suggestive of early-stage deep myositis involving the deep muscle compartments.

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Initially, the patient was managed conservatively with nonsteroidal anti-inflammatory drugs (NSAIDs) and supportive care. However, in view of persistent severe pain, progressive elevation of inflammatory markers, and MRI evidence of deep muscle inflammation, empirical intravenous antibiotic therapy was initiated with meropenem and linezolid to cover possible deep-seated bacterial infection, including resistant organisms. Following initiation of intravenous antibiotics, the patient showed significant clinical improvement, with gradual reduction in pain, improved range of motion, and restoration of mobility.

A multidisciplinary team involving paediatrics, orthopaedics, radiology, and rheumatology reviewed the clinical and radiological findings and reached a consensus in favour of deep inflammatory myositis over septic pathology.

Over the subsequent five days, the child showed gradual but definite clinical improvement. She began to bear weight with minimal support, tenderness reduced significantly, and inflammatory markers demonstrated a downward trend with CRP decreasing from 181 mg/L to 65 mg/L. Creatine kinase and procalcitonin remained within normal limits throughout the admission.

Repeat MRI scans demonstrated a reduction in muscle oedema and regression of lymphadenopathy, with persistent but non-progesterone marrow oedema and no evidence of cortical thickening or periosteal reaction. Clinically, the child remained afebrile, systemically well and haemodynamically stable with full and painless range of movement of the left lower limb and good oral intake.

After completing nine days of intravenous antibiotic therapy and demonstrating sustained clinical and laboratory improvement, the patient was transitioned to oral antibiotic therapy consisting of linezolid for a total duration of 21 days and cefixime. Naproxen was continued for symptomatic pain relief. Physiotherapy and gradual mobilisation were initiated to facilitate functional recovery. At the time of discharge, the child was independently ambulatory, had minimal to no residual pain, stable vital signs, and no functional limitation.

Discussion

Deep myositis results from muscle inflammation triggered by bacterial antigens. Distinguishing it from septic arthritis is critical, as unnecessary arthrotomy or prolonged antibiotics carry significant morbidity. Over the course of treatment, multiple differential diagnoses were considered and ruled out.

Septic arthritis was the most urgent concern due to severe pain and inability to bear weight. However, it was ruled out based on the absence of joint effusion on MRI, no local warmth, erythema, or systemic toxicity, normal procalcitonin and sterile blood cultures and pain localised to thigh muscles rather than the joint capsule.

Acute Osteomyelitis can present with raised CRP and limb pain, but MRI showed no cortical destruction, periosteal elevation or localised bone abscess.

Post-viral inflammatory myositis was initially considered initially due to preceding febrile illness, but progressively rising inflammatory markers and severity of symptoms suggested infectious etiology.

Autoimmune Myositis was also considered unlikely as ANA was negative, CK remained normal and the abrupt onset after bacterial infection favoured reactive rather than autoimmune etiology.

This case highlights several important clinical lessons. Normal creatine kinase levels do not exclude clinically significant myositis, as inflammatory muscle involvement may occur without overt muscle necrosis. [6] MRI remains the investigation of choice for evaluating such patients, as it effectively differentiates diffuse inflammatory muscle edema from abscess formation, septic arthritis, or osteomyelitis.

In this case, there was evidence of significant localized muscle infection, as demonstrated by marked leukocytosis, elevated inflammatory markers, and MRI-confirmed deep muscle edema involving the femoral musculature. Although procalcitonin levels remained within normal or minimally elevated range, this does not exclude localized bacterial infection, as procalcitonin is more sensitive for systemic infection and sepsis rather than localized musculoskeletal infections. [7] In early-stage deep myositis, infection may remain confined to muscle tissue without systemic dissemination, resulting in a limited procalcitonin response. Importantly, MRI findings confirmed deep muscle involvement, indicating a deep-seated infectious process with risk of progression to suppurative pyomyositis, abscess formation, or contiguous osteomyelitis if inadequately treated.

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Current literature recommends prolonged antibiotic therapy, typically for 3–6 weeks, to ensure complete eradication of infection and prevent relapse, particularly in deep-seated musculoskeletal infections where antibiotic penetration may be limited. [8,9] The decision to continue antibiotic therapy in this patient was further supported by the severity of initial clinical presentation, radiological findings, and sustained clinical and laboratory improvement following antimicrobial treatment. CRP trends also correlated well with treatment response and were used to guide duration of therapy. [9]

Conclusion

Deep myositis should be considered in children presenting with acute limb pain, inability to bear weight, and elevated inflammatory markers, even in the absence of abscess formation. This case highlights that normal creatine kinase and procalcitonin levels do not exclude localised muscle infection, as early-stage disease may not involve significant muscle necrosis or systemic inflammatory response. MRI plays a pivotal role in early diagnosis by identifying deep muscle inflammation and excluding other differential diagnoses such as septic arthritis and osteomyelitis. Prompt initiation of appropriate antibiotic therapy, guided by clinical and radiological findings, can prevent disease progression and result in complete clinical and functional recovery.

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