# **Case Report**

# Piriformis Myositis Secondary to Staphylococcus Aureus Bacteraemia

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#### Abstract

Piriformis myositis is an uncommon condition, defined as a form of pyomyositis affecting the piriformis muscle. It usually manifests clinically with back or buttock pain impairing mobility and a marked inflammatory response. In our case, we describe an immunosuppressed 48-year-male who developed piriformis myositis secondary to staphylococcus aureus bacteraemia, who later developed complications including native aortic valve infective endocarditis and sciatica. Timely initiation of treatment in the form of intravenous antibiotics, with or without surgical drainage, will help to minimize long-term morbidity in patients.

Keywords: Piriformis myositis, Pyomyositis, Piriformis syndrome.

#### Introduction

Pyomyositis refers to a bacterial infection of skeletal muscles which often results in intramuscular abscess formation. Primary pyomyositis occurs following bacteraemia, most commonly due to staphylococcus species. Secondary pyomyositis refers to myositis secondary to direct trauma or contiguous spread. Primary pyomyositis typically favours larger muscle groups such as the iliopsoas, quadriceps or gluteal muscle bulk but infrequently may affect smaller muscles of the hip such as the piriformis. Pyomyositis of the piriformis is uncommon, with only 27 reported cases in English literature<sup>1</sup>. We herein describe an uncommon entity of piriformis pyomyositis complicated by abscess formation in an immunosuppressed patient.

#### Case

A 48-year-old male laborer presented to the emergency department with a 3-day history of focal left sided buttock and lumbar spine pain with associated rigors. He denied any symptoms of sciatica, with the pain severely limiting his mobility. This is on the background of poorly controlled type 2 diabetes mellitus and a previous splenectomy in the setting of idiopathic thrombocytopenic purpura, for which he was not on antibiotic prophylaxis for, nor up to date with his immunization schedule. He denied any recent travel history nor any history of intravenous drug use.

Bloods on admission revealed a C-reactive protein level of 196 mg/L and a white cell count of  $30 \times 10^9$  units/L with predominant neutrophilia. Creatinine kinase level was not elevated. He experienced intermittent rigors with recorded temperatures as high as  $38.6^{\circ}$ C. Plain radiographs of the hip and lumbosacral spine were unremarkable. A CT lumbosacral spine and hip was performed which revealed a non-specific volume of free fluid in the presacral region with no radiological evidence of acute fracture or compressive neuropathy. A subsequent contrast MRI study revealed a high T2 signal of the left piriformis suggestive of myositis.

Blood cultures grew methicillin-sensitive staphylococcus aureus (MSSA) within 16 hours of collection. This was treated initially with 2 grams of flucloxacillin 6-hourly with a single dose of

vancomycin. Daily blood cultures for the following 3 days each returned positive with a similar organism, with minimal clinical improvement in pain or mobility despite all sets of blood cultures being sensitive to flucloxacillin.

A repeat CT hip was obtained on day 3 of presentation, which showed progression and formation of abscesses within both the piriformis and iliacus muscles measuring 74 x 37mm and 17 x 11m respectively. His intravenous flucloxacillin dose was increased to 2 grams four hourly. An initial transthoracic echocardiogram was performed but negative for infective endocarditis. A transesophageal echocardiogram (TOE) was performed in the setting of persistent bacteremia, which showed findings suggestive of native aortic valve infective endocarditis.

Interventional radiology was involved on day 5, where 2mL of purulent fluid grew MSSA with similar sensitivities. A 10Fr catheter was inserted, with 60 mL draining within the first 24 hours, followed by a further 55mL and 35mL in the subsequent consecutive 24-hour periods. This drain was eventually removed on day 3 post insertion.

His pain was poorly responsive to regular paracetamol and opioids (subcutaneous fentanyl and oxycodone), but attained the most analgesia from suppository non-steroid anti-inflammatories – in this case 25mg of peri-rectal indomethacin.

Three consecutive days of daily blood cultures were eventually collected from days 6 to 9 following commencement of therapy. A midline catheter was inserted, with the patient being discharged home to receive a further 6 weeks of IV flucloxacillin. He was followed up in the outpatient department following this, and continued to have raised inflammatory markers. His flucloxacillin dose was continued for a further 2 weeks, for a total of 8 weeks, for a total of 8 weeks, for a total antibiotic treatment time of 12 weeks. Three months on from presentation, he has had an improvement in mobility but has developed symptoms of sciatica in the left limb.

IV Vancomycin (21/07 - 1 dose) IV Flucloxacillin 2g 6 hourly (21/07 --> 25/07) IV Flucloxacillin 2g 4 hourly (25/07 - 12/9/23) (Extended due to rising CRP and subsequent admission) PO Flucloxacillin 1g QID 12/9 - (EDD 10/10)

20/07, 22/07 x2, 23/07 x2, 24/07, 25/07: MSSA 26/07 onwards: No growth to date.

Piriformis abscess culture (26/07): MSSA

- # Community acquired MSSA bacteraemia
- Complicated by:
- > L) piriformis & L iliacus muscle abscess.

- > Native aortic valve infective endocarditis.
- Likely skin source.

- IR drainage (26/07): Collections thought to communicate. Catheter inserted into piriformis collection.

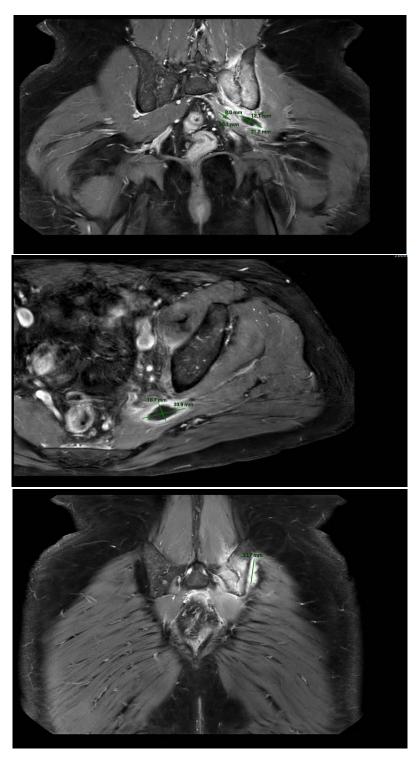
- Drain removed on 29/07.

- IE MDT (02/08):? Small lesion on AV on TOE. Treat empirically as IE considering prolonged bacteraemia and indeterminate lesion.

- Readmitted 11/8/23 due to worsening pain and fevers

- MRI showed sacroiliac joint osteomyelitis, no drainable collection

- Discharged with plan to complete flucloxacillin in the community as previously planned



There is significant bone marrow oedema and post contrast enhancement involving left sacroiliac joint with intra-articular T2 hyperintense signal and cortical irregularity representing acute/subacute septic arthritis and associated established osteomyelitis. There is a 7 x 27 x 31 mm fluid collection along posterior sacroiliac joint, not well seen on prior CT.

There is persistent rim enhancing abscess in left piriform muscle, approximately measuring  $31 \times 12 \text{ mm}$  on coronal images (previously  $41 \times 15 \text{ mm}$ ). Adjacent small pocket of abscess measures  $18 \times 8 \text{ mm}$  with a thin communication between the collections. This shows interval decrease in size as compared to prior imaging.

There is significant T2 SPAIR hyperintensity and post contrast enhancement in obturator internus, obturator externus, left iliacus and gluteus medius muscles however no discrete abscess noted. These findings are in favour of acute myositis. Psoas muscle appear unremarkable. Left sciatic nerve is seen traversing through the inflamed pyriform however no compression noted. No epidural abscess/discitis identified in the lumbosacral spine. No extension of the disease in the facet joints noted. Preserved bone marrow signal of vertebral bodies.

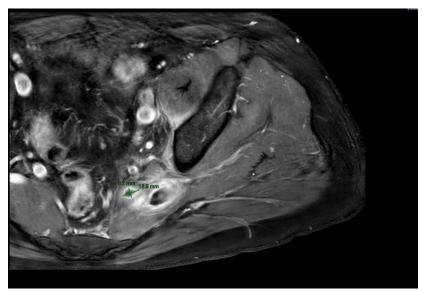
Minimal fluid seen in the presacral space. No vascular complication noted. No abscess identified in subcutaneous tissue.

Rest of the visualized abdominopelvic viscera are unremarkable.

# Conclusion

Findings are of acute septic arthritis and osteomyelitis involving left sacroiliac joint with minimal/marginal interval decrease in size of abscess in left piriform muscle. No evidence of epidural abscess/discitis.

Acute myositis involving left obturator muscles, left iliacus and gluteus medius with no discrete collection/abscess seen in these muscles.



Repeat CT on 10/9/23:

Comparison is made with a previous examination of 27/08/2023. There is a very subtle area of asymmetry in the piriformis muscle, where the abscess was previously demonstrated. There is no longer any cavity demonstrated and no areas of enhancement. The abscess appears to have completely healed. The surrounding musculature is normal. There are no new areas of enhancement or cavitation. Large anterior fat containing hernia seen in the anterior left iliac fossa. No bowel obstruction. The visible kidneys are normal. No bony abnormality.

#### Discussion

Staphylococcus aureus species appear to be the predominant causative organism for piriformis myositis. Using PubMed with keywords "piriformis myositis", we were able to identify 27 reported cases of piriformis myositis available in English as published literature from 1973 [1]. Of these cases, 13 of the 27 cases were attributable to a staphylococcus aureus species. [2][3][4][5][6][7][8][9][10][11][12][13].

Of these 13 cases, 7 cases involved patients aged 19 years or younger, each with no previous documented medical conditions or known pre-disposing risk factors for immunosuppression. 2 cases were attributed to the perinatal period, including one case of unsafe abortion in an 18-year-old female, and one involving pregnancy in the early trimester [2].

All 13 cases reported some form of inflammatory response through either one or a combination of documented fever (temperature >38°C), raised white cell counts (> 11 x 10<sup>9</sup> cells/L) and/or raised C-reactive protein or erythrocyte sedimentation rates.

Of these cases, five required a form of direct source control through direct aspiration [2][3][7][8][10]. One case described in 1992 involved a posterior open surgical approach with direct drainage to good clinical effect [9]. All cases were treated with some form of intravenous antibiotics over a period of at least 3 weeks, some cases recording stepdown therapy using oral antibiotics for a total treatment of 11 weeks. Antibiotic regimens varied vastly between cases, most commencing with broad-spectrum antibiotics such as ceftriaxone and carbapenems, before adjusting to sensitivities once cultures returned, of which the most common agent used was penicillin (either flucloxacillin or cloxacillin).

Reassuringly all cases reported ongoing recovery over a period of weeks to months, although it is unknown if recurrence or continued resolution of symptoms were sustained due to lack of reported formal follow-up.

Our patient displayed several risk factors for developing bacteraemia with subsequent piriformis myositis immunosuppression in the setting of prior splenectomy and poorly controlled diabetes mellitus in addition to occult risk factors for muscular trauma and skin abrasions as a manual labourer. The use of IV flucloxacillin 2 grams QID as a single agent appeared to be effective in treating his bacteremia, but not as effective in managing his buttock pain.

It would be pertinent for clinicians to consider patient risk factors for pyomyositis and consider the appropriate workup accordingly not to dismiss cases as so easily as such as merely musculoskeletal pain. From our review of literature, it would be reasonable for patients presenting with back pain to have routine blood work, particularly looking for raised inflammatory markers such as an elevated white cell count, C-reactive protein and/or erythrocyte sedimentation rate. Whilst the sample size of a rare disease entity is relatively small all reported cases to our knowledge displayed biochemical or an otherwise objective form of evidence of an inflammatory response.

Direct drainage of the localised abscess is often indicated, particularly in cases where there is a lack of clinical response to an appropriate intravenous antibiotic. In our case, direct aspiration with drain insertion in addition to an increase in flucloxacillin dosage were performed concurrently considering the lack of clinical and biochemical improvement. Consequently, it is difficult for us to ascertain whether a synergistic or individual change in management was largely responsible for improvement. Previous cases preferred more broad-spectrum antibiotic coverage such as carbapenems and 3<sup>rd</sup> generation cephalosporins, before titrating therapy to a narrow-spectrum agent following return of microbial sensitivities.

In our case, the presentation of native valve endocarditis was sub-clinical and discovered via TOE in the setting of persistent bacteraemia despite appropriate intravenous antibiotic treatment. The guidelines surrounding whether a TOE is necessary after a negative TTE are controversial. Sekar et, al in 2017 reported an increased sensitivity in TOE based upon 119 cases of staphylococcus aureus bacteraemia where both a TOE and a TTE were performed - 29 of the 119 cases were diagnosed with IE (24%), and of these 25 were seen on TOE compared to 6 on TTE [14]. Only one of the cases involved S. aureus bacteremia reported an echocardiogram being performed and negative [2]. To date, we have only found one other documented case of pyomyositis with concurrent bacterial endocarditis in literature [15]. Lo et.al, described a case in 1998 of a 48-yearold male intravenous drug user who presented with methicillin sensitive staphylococcus aureus bacteremia, complicated by mitral-valve infective endocarditis and right calf pyomyositis.

# Conclusion

Pyomyositis is an uncommon condition that has potential for clinical morbidity. Prompt consideration of pyomyositis in patients presenting with focal back pain with associated fevers and/or raised inflammatory markers can aid with commencing empirical treatment early with the view of reducing complications such as abscess development or in cases involving bacteraemia, subsequent infective endocarditis. MRI with contrast is the gold-standard imaging modality. The mainstay of treatment appears to commence with broad spectrum antibiotics, with clinical adjustment to microbial sensitivities. Duration of antibiotics of at least 6 weeks appears to be the mainstay of treatment. Primary drainage is indicated if a concurrent abscess is present. Reassuringly, all reported cases appear to document resolution of clinical symptoms with nil reported fatalities.

**Conflict of interest:** The authors declare that they have no conflict of interest to declare.

**Declaration of role played by each co-author.** K. Tang was the first author responsible for collection of and interpretation of data. A. Al-Heilfi was responsible for supervision and first editing of the manuscript.

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