ISSN 2564-0127 (Print)
ISSN 2564-0135 (Online)

Canadian Journal of Medicine 6(2024) 62-68

Canadian Journal of Medicine

WWW.CIKD.CA

journal homepage: https://www.cjm.cikd.ca

Bilateral Psoas Abscess Extended into Spinal Canal in a Patient with Long-Standing Poorly Controlled Diabetes Mellitus – A Case Report

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Keywords:
Psoas abscess, Methicillin-resistant staphylococcus aureus, Spondylodiscitis

Received
11 July 2024
Received in revised form
21 July 2024
Accepted
22 July 2024

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ABSTRACT

Psoas abscess is a relatively rare condition with varying clinical presentation. Therefore, the diagnosis and treatment are frequently delayed. Psoas abscess can extend into the spine and cause spinal infection or spinal infection can be a source for secondary psoas abscess. It has 100% mortality if left untreated. Broad-spectrum antibiotics and drainage of pus by either percutaneous drain insertion or open surgery are the treatment modalities. A 62-year-old patient, male patient with long-standing diabetes and ischemic heart disease presented with fever and lower back pain for four days duration without any systemic focus of infection. He was hemodynamically stable, and neurological examinations of the lower limb were normal on admission. He developed bilateral lower limb weakness on the 7th day of hospital stay, and neurological examinations of the lower limb revealed flaccid paralysis. His Contrast Enhanced Computerized Tomography (CECT) of the abdomen revealed a bilateral psoas abscess. Both pus culture and blood culture were positive for Methicillin-resistant staphylococcus aureus. Later, his Magnetic Resonance Image (MRI) spine revealed infective multilevel spondylodiscitis, arachnoiditis, radiculitis, and early infective myelitis. The abscess was drained, and a broad-spectrum antibiotic was started. Unfortunately, he passed away despite maximal medication intervention due to septicemia, acute kidney injury, and septic shock. Even though rare, psoas abscess should be suspected in a patient with back pain, fever, and high inflammatory markers due to its high mortality and morbidity. Early diagnosis and treatment can reduce mortality and morbidity. However, advanced age, presence of bacteremia, and poorly controlled diabetes carry poor prognosis.

Introduction

Psoas abscess is a collection of pus in the iliopsoas compartment [1]. The incident is rare, but with the improvement in imaging technology, the frequency of diagnosis of this condition has increased [2]. The diagnosis is frequently delayed due to its rarity and varying nonspecific presentations [3]. It is commonly caused by *staphylococcus aureus* including methicillin-resistant *staphylococcus aureus* (MRSA), followed by streptococci and *Escherichia coli*. *Mycobacterium tuberculosis* is a frequent cause of psoas abscess in endemic areas [4]. Imaging studies confirm the diagnosis of psoas abscess. In this case report, we present a rare case of bilateral primary psoas abscess, which extended into the spinal canal and caused flaccid paralysis of the bilateral lower limb.

Case Presentation

A 62-year-old male patient with long-standing diabetes mellitus with poor control and ischemic heart disease with an ejection fraction of 60 percent and negative exercise tolerance test presented with fever associated with chills and rigors for four days duration. He also had lower back aches for the same duration. There were no urinary, respiratory, or gastrointestinal symptoms. He did not have any lower limb weakness on admission. He owned a fish market with a possibility of leptospirosis exposure.

On examination, he was found to be afebrile with normal vital signs. His respiratory, cardiovascular, and abdominal were unremarkable. He had severe tenderness over the coccygeal area.

His full blood count revealed neutrophil leukocytosis, microcytic hypochromic anemia, and thrombocytopenia. His inflammatory markers were high, with C-reactive protein (CRP) of 206.8mg/L and an Erythrocyte Sedimentation Rate (ESR) of 90mm/h. His creatinine and serum electrolytes were normal on admission, but later, he developed acute kidney injury due to sepsis. The liver functions test revealed marginally elevated transaminases, hypoalbuminemia, elevated alkaline phosphatase (ALP), and elevated gamma-glutamyl transferase (GGT) (Table 1). His capillary blood sugar levels were high.

Initially, a presumptive diagnosis of leptospirosis was made due to acute febrile illness with leukocytosis, thrombocytopenia, high CRP, and exposure history in an endemic area. He was treated with intravenous ceftriaxone and oral as overnight intravenous fluids with monitoring of input and urine output. His blood sugar was managed with soluble insulin. An ultrasound scan (USS) of the abdomen was carried out due to deranged liver functions and revealed features of chronic liver cell disease with left sided psoas muscle collection (7*3cm) (Figure 1). Once the psoas abscess was detected, intravenous metronidazole was added. Ultrasound-guided percutaneous aspiration of pus was carried out, and an interventional radiologist placed a pigtail catheter in situ to allow further drainage.

As there was an inadequate response to antibiotic therapy indicated by ongoing fever spikes and inadequate drop of CRP, a CECT scan of the abdomen was carried out and demonstrated bilateral psoas abscess (Right – 1.8cm*3.7cm*1cm and left – 12.7cm*3cm (Tr)*4cm (AP)) and degenerative changes in bone window of the vertebral column. By that time, both blood and pus cultures were positive for MRSA and sensitive to vancomycin, chloramphenicol, and teicoplanin. The antibiotic was changed to the renal dose of teicoplanin.

On the 7th day of the hospital stay, he developed bilateral lower limb weakness without upper limb weakness. His lower limb neurological examination revealed a normal tone with a power of 1/5 and absent reflexes bilaterally (flaccid paralysis). There was no sensory level, and Babinski was negative bilaterally. An urgent MRI of the spine was carried out and revealed previously demonstrated bilateral psoas abscess (Figure 2) and multilevel infective spondylodiscitis, arachnoiditis, radiculitis, and early infective myelitis without abscess formation (Figure 3). Neurosurgical opinion was taken and said neurosurgical intervention wasn't necessary considering the absence of abscess formation or spinal cord compression.

He developed oliguric acute kidney injury with hyperkalemia due to sepsis resulting in several cycles of intermittent hemodialysis. Later, he also developed septic shock and started on noradrenaline. There was a poor response to antibiotics, and his blood sugar control was poor despite maximal therapy. Even though he was treated with maximal medical therapy, he passed away after 10 days of admission due to severe septicemia and acute kidney injury.

Table 1. Investigations

White Blood Cell	13,510/μL
Neutrophil	81.2%
Lymphocyte	8%
Hemoglobin	8.0g/dl
Mean Corpuscular Volume	74.2fL
Platelet	95,000/μL
Blood picture	Ongoing severe infection or inflammation Mild mixed deficiency anemia, predominantly iron deficiency Features of liver impairment
ESR	90mm/h
CRP	206.8mg/L
Renal functions test	Creatinine – 101.6 μmol/L → 434.5 μmol/L Urea – 40.3mg/dl → 196.lmg/dl
Serum electrolyte	Sodium– 136mmol/L Potassium – 4.6mmol/L
Liver functions test	AST – 45U/L, ALT – 41U/L, ALP – 153U/L, GGT – 132U/L, Protein – 5.5g/dl, Albumin – 3.3g/dl, Globulin – 2.2g/dl, Total bilirubin 12.8 μmmol/L, Direct bilirubin – 8.1 μmmol/L, Indirect bilirubin – 4.7 μmmol/L
APTT	34.1 sec
INR	1.17
USS abdomen	Appearance could represent a left psoas abscess (7*3cm) Early chronic liver cell disease changes
Full report of pus	Colour – reddish, appearance – turbid, protein – 4530mg/dl, lymphocyte – 00 mm3, polymorphs – field full, red cells – 00mm3
Blood culture	*Staphylococcus aureus* (MRSA) isolated
Pus culture	*Staphylococcus aureus* (MRSA) isolated
CECT Abdomen	An intramuscular fluid collection in left psoas major muscle measuring 12.7cm * 3cm (Tr) * 4cm (AP). Smaller collection within mid right psoas muscle measuring 1.8cm * 3.7cm * 1cm in size Liver cirrhosis with evidence of portal hypertension Bone window revealed degenerative changes
Contrast MRI spine	The appearances are in favour of multilevel infective spondylodiscitis (L2 to L4) There is presence of inflammatory soft tissue in the spinal canal, probably causing an arachnoiditis and radiculitis No current evidence of an epidural abscess The abnormal signal within the conus medullaris without enhancement is likely representing oedema or early infective myelitis Bilateral psoas abscess

Note. AST – aspartate aminotransferase, ALT – alanine aminotransferase, APTT – activated partial thromboplastin time, INR – international normalization ratio

Figure 1. Ultrasound of Psoas muscle – on the left side, collection was noted in the left psoas muscle, and on the right side, no collection was noted in the right psoas muscle

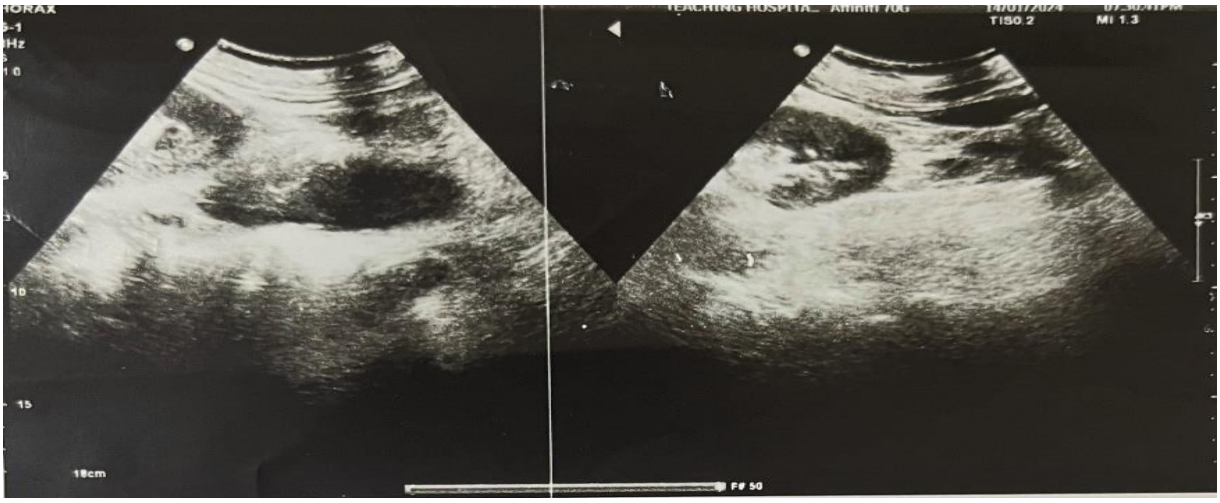


Figure 2. t1 fse image of MRI showing bilateral psoas abscess – on left coronal view and right transverse view

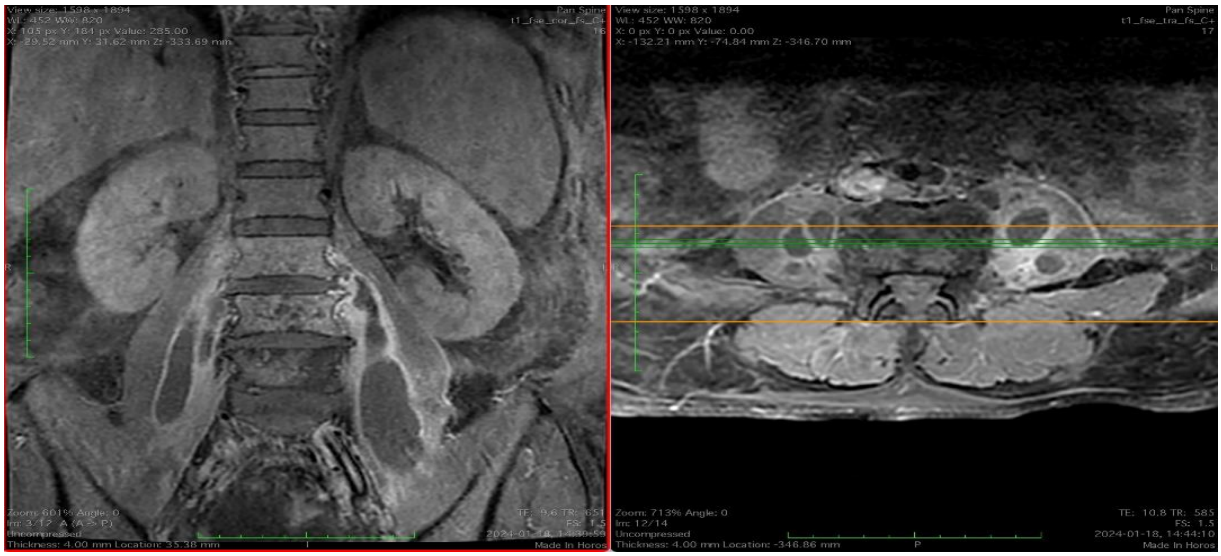
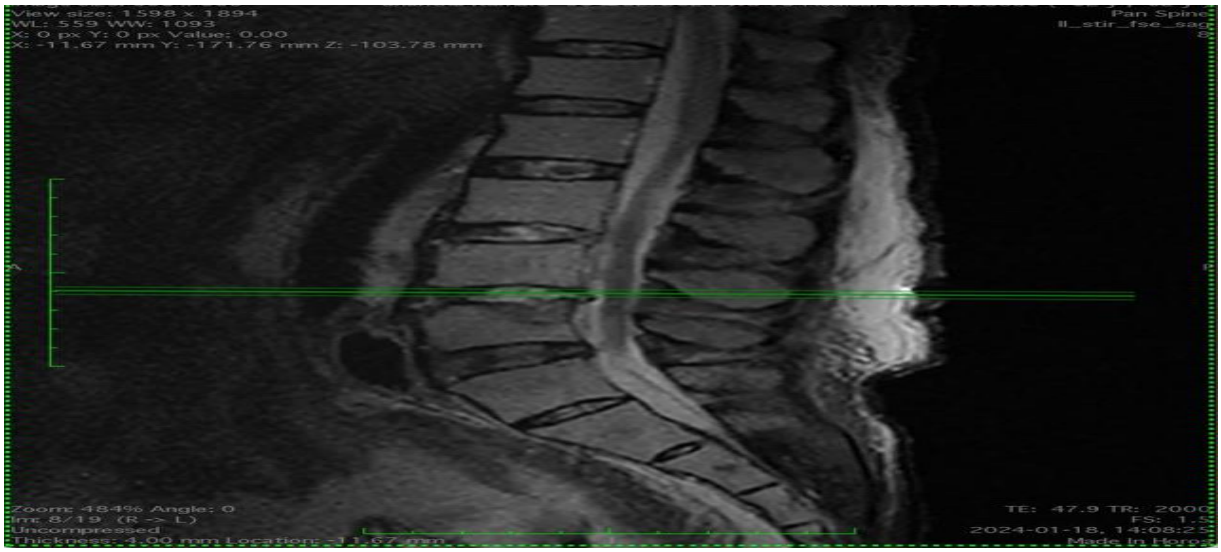


Figure 3. Sagittal – SRIR sequence of MRI spine showing spondylodiscitis in L2 to L4



Discussion

The iliopsoas and iliacus muscles form the iliopsoas compartment, which is located retroperitoneally. The Psoas muscle originates from the transverse and lateral aspect of vertebral bodies from the 12th thoracic vertebrae to the 5th lumbar vertebrae, passes downwards, and is inserted into the lesser trochanter of the femur. The iliacus muscle joints the psoas to insert via the same tendon. The psoas muscle has an anatomical relation with vertebral bodies, abdominal aorta, sigmoid colon, appendix, jejunum, ureters, kidneys, pancreas, and iliac lymph node. Therefore, the infection can spread between these structures and the psoas muscle [1].

Psoas abscess can be primary or secondary according to the pathogenesis. Primary abscess is due to the hematogenous or lymphatic spread of organism from distant sites [1,4-6]. Diabetes, intravenous drug abuse, acquired immunodeficiency syndrome, renal failure, and immunosuppression are the risk factors for primary abscess. The secondary abscess is due to a direct spread of infection from an adjacent structure. Chron's disease is the most common cause of secondary psoas abscess. Other secondary causes are appendicitis, ulcerative colitis, colonic cancer, diverticulitis, and vertebral osteomyelitis [1]. In some circumstances, it may be difficult to distinguish between primary and secondary psoas abscess.

Primary psoas abscesses are most frequently caused by a single organism [7]. The most common organism is *Staphylococcus aureus* including MRSA followed by *Escherichia coli* and *streptococcus* [5,7,8]. Secondary psoas abscesses are more frequently polymicrobial. Enteric bacteria are usually responsible for secondary psoas abscesses [4]. *Mycobacterium tuberculosis* is a common cause of psoas abscess in developing countries [1].

In the reported cases, psoas abscess is mostly unilateral, accounting for 95-97% [9]. Psoas abscess occurs on the right and left sides with roughly equal frequency, but bilateral psoas abscesses are uncommon [4,10,11] Psoas abscess has a classic triad of clinical features with fever, back pain, and psoas spasm. However, triad accounts for approximately 30% of patients. Most of the patients present with nonspecific symptoms and with its rare occurrence, the diagnosis is frequently delayed [12]. Laboratory findings include leukocytosis, anemia, and elevated inflammatory markers [8].

Imaging studies confirm the diagnosis of psoas abscess. Ultrasound has poor sensitivity and specificity for the diagnosis of psoas abscess [13]. It may be diagnostic only in 50% of cases [8]. In our patient, an ultrasound scan only detected a unilateral psoas abscess. Therefore, a CT scan is the imaging modality for the diagnosis [8,14]. MRI appears to be no better than a CT scan for diagnosis of psoas abscess and, given its availability and scan time, often not the imaging modality of choice [15]. However, an MRI is better than a CT scan, the imaging of the spine for the complete evaluation of back pain.

Management of psoas abscess consists of two aspects: broad-spectrum antibiotics and drainage of pus [1,10,11]. In the case of primary psoas abscess, anti-staphylococcal antibiotics should be started as *Staphylococcus aureus* is the main causative organism. But in a patient suspected to have secondary psoas abscess, a broad spectrum antibiotic effective against bowel flora should be used. Later, antibiotics can be changed according to sensitivity pattern. Imaging-guided percutaneous drainage is an initial approach to draining the pus. Surgical drainage of pus by either open surgery or laparoscopy is used where percutaneous drainage is unsuccessful or in complicated cases [1,11].

Psoas abscess carries high mortality and morbidity. The mortality rate is 2.4% for primary abscess and 19% for secondary abscess [1]. Mortality rate may approach 100% in untreated cases [4]. Delayed or inadequate treatment, advanced age, renal impairment, and presence of bacteremia are the risk factors for mortality [8,11,16]. Our patient had bacteremia and renal impairment.

Conclusion

Even though rare, psoas abscess should be suspected in a patient with back pain, fever, and high inflammatory markers due to its high mortality and morbidity. Early diagnosis and treatment can reduce mortality and morbidity. However, advanced age, presence of bacteremia, and renal impairment carry a poor prognosis.

Declarations

Acknowledgements

We would like to express our deepest gratitude to the wife and son of the patient for allowing us to report and publish this case.

Disclosure Statement

No potential conflict of interest was reported by the authors.

Ethics Approval

Not applicable.

Funding Acknowledgements

Not applicable.

Citation to this article

Sathees S, Amarasekara D, Jayasekara P, Rasangika C, Ekanayaka S. Bilateral psoas abscess extended into spinal canal in a patient with long-standing poorly controlled diabetes mellitus – A case report. Canadian Journal of Medicine. 2024 July 22;6(2):62-68. doi: 10.33844/cjm.2024.6038

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