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# Unusual Presentation of Recurrent Pyogenic Bilateral Psoas Abscess Causing Bilateral Pulmonary Embolism by Iliac Vein Compression

Authors' Contribution:  
Study Design A  
Data Collection B  
Statistical Analysis C  
Data Interpretation D  
Manuscript Preparation E  
Literature Search F  
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**Conflict of interest:** None declared

**Patient:** Male, 47  
**Final Diagnosis:** Bilateral psoas abscess • acute lower extremity deep vein thrombosis • bilateral pulmonary embolism  
**Symptoms:** Progressive left leg swelling • productive cough with whitish sputum • right flank pain  
**Medication:** Antibiotics and anticoagulation  
**Clinical Procedure:** CT-guided percutaneous drain placement  
**Specialty:** Internal Medicine/Critical Care

**Objective:** Unusual presentation  
**Background:** Psoas abscesses are a known cause of back pain, but they have not been reported as a cause of acute lower extremity thromboses and bilateral pulmonary emboli. We report a patient with bilateral psoas abscesses causing extensive pulmonary emboli through compression of the iliac vein.





**Case Report:** A 47-year-old man presented with bilateral leg swelling over 4 weeks. Physical examination revealed a thin male with bilateral leg swelling, extending to the thigh on his left side. He had hemoglobin of 10.5 g/dl, leukocytosis of 16 000/ml, and an elevated D-dimer. A computed tomography (CT) angiogram of his chest showed extensive bilateral pulmonary emboli and infarcts. He remained febrile with vague flank pain, prompting a CT of his abdomen and pelvis that showed large, multiloculated, septated, bilateral psoas abscesses with compression of the left femoral vein by the left psoas abscess and a thrombus distal to the occlusion. Two liters of pus was drained from the left psoas abscess by CT-guidance, and although the Gram staining showed Gram-positive cocci in clusters, cultures from the abscess and blood were negative. A repeat CT showed resolution of the abscesses, and the drain was removed. He was discharged to a nursing home to complete a course of intravenous antibiotics and anticoagulation.

**Conclusions:** Although the infectious complications of psoas abscesses have been described in the literature, the mechanical complications of bilateral psoas abscesses are lacking. It is important to assess for complete resolution of psoas abscesses through follow-up imaging to prevent venous thromboembolic events.

**MeSH Keywords:** Psoas Abscess • Pulmonary Embolism • Venous Thrombosis

**Abbreviations:** CT – computerized tomography; PE – pulmonary emboli; DVT – deep venous thrombosis; ICU – intensive care unit; IV – intravenous; MRI – magnetic resonance imaging; ADA – adenosine deaminase; PCD – percutaneous catheter drainage

**Full-text PDF:** <http://www.amjcaserep.com/abstract/index/idArt/894206>

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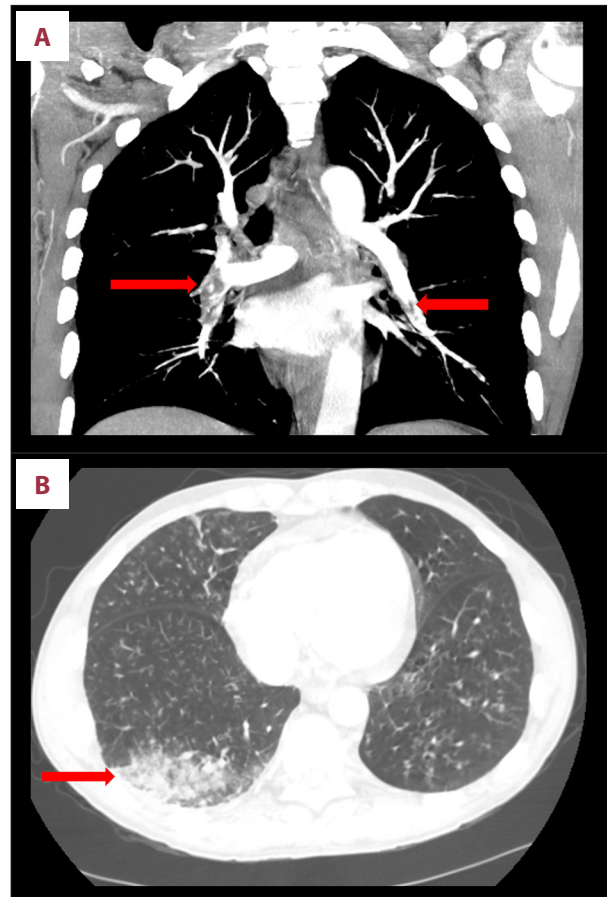
## Background

A psoas abscess is a rare condition with a variable clinical presentation [1]. It can be either primary or secondary and various etiologies have been reported [2]. Primary psoas abscess is commonly seen in immunocompromised states, including AIDS, diabetes mellitus, renal failure, and transplant patients. Contiguous spread from intra-abdominal structures, either infective or inflammatory conditions, results in secondary psoas abscesses. Bilateral psoas abscesses are rare and can lead to significant complications, including thrombosis (by compressing venous vasculature); hydronephrosis and hydroureter (by compression of the ureters); small bowel obstruction, and septic arthritis and osteomyelitis of the vertebrae, hip, and sacroiliac joints [3]. The major etiologies of PE include either inherited or acquired disorders like blood clotting proteins deficiency and other hypercoagulable states due to malignancy, pregnancy, trauma, major surgery, immobilization, and oral contraceptive usage [4]. We report a rare case of DVT of the common femoral vein and subsequent extensive PE caused by bilateral psoas abscesses through compression of the iliac vein. We illustrate the clinical presentation, pathophysiology, and management of this rare condition, along with a review of the literature.

## Case Report

A 47-year-old man presented to our hospital complaining of left flank pain with progressive left leg swelling for 4 weeks. The left flank pain was dull in nature, aggravated by movement, and with no radiation. He had limited exercise tolerance because of left leg swelling and later even became wheelchair bound 1 month prior to admission. He had a history of epidural abscess with *Streptococcus mitis* bacteremia at another institution about 3 months prior to the current admission and had undergone treatment with antibiotics for 4 weeks. He did not report fever, chills, chest pain, coughing of blood, dizziness, loss of consciousness, palpitations, or any urinary or bowel complaints. There was no history of any sick contacts, recent travel, or weight loss. He denied smoking cigarettes, drinking alcohol, or illicit drugs use. He had no history of tuberculosis or malignancy. He was not taking any medications and had no relevant family or surgical history. He was born and raised in the United States. He was single, sexually inactive, and had no children. He had no known allergies.

Examination revealed a heart rate of 132 beats/min, blood pressure of 138/89 mm Hg, respiratory rate of 20 breaths/min, temperature of 98°F, and 96% oxygen saturation on room air. He had a body mass index of 19.2 kg/m<sup>2</sup>. There were decreased breath sounds at the right lung base. Cardiovascular and abdominal examination was unremarkable. The lower

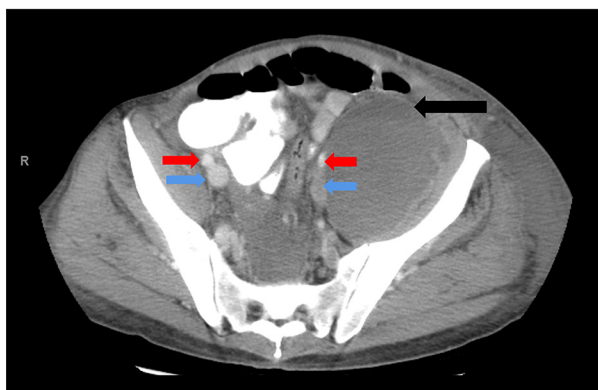


**Figure 1.** (A) CT angiogram showing filling defects within the right middle lobe, right lower lobe, and left lower lobe segmental and sub-segmental branches suggestive of extensive segmental pulmonary emboli (red arrows). Small filling defects are also seen within the left upper lobe sub-segmental branches. (B) Peripheral ground glass opacities in the right middle and lower lobes are suggestive of pulmonary infarcts (red arrow).

extremities showed bilateral pitting edema, more on the left side, extending to his thigh.

His laboratory evaluation in the emergency room showed hemoglobin of 10.5 g/dl, leukocytosis of 16 000/ml, and a neutrophil predominance of 88.9%. Thyroid, liver, and kidney function tests were normal, as was an erythrocyte sedimentation rate. The QuantiFERON TB gold test result was indeterminate. Blood cultures were negative. HIV test was negative. Chest x-ray showed bibasilar atelectasis without infiltrates or effusions.

He developed a fever of 102°F on day 1 of admission and was started on intravenous (IV) Ceftriaxone and Azithromycin with the suspicion of community-acquired pneumonia. Because of the presence of an elevated D-dimer assay and sinus tachycardia on his admission electrocardiogram, a CT angiogram of his chest was ordered in the emergency room. It showed



**Figure 2.** Large left psoas abscess (black arrow). The left common iliac artery (red arrow) and left common iliac vein with a thrombus in the lumen (blue arrow) are being compressed by the large psoas abscess (compare with vessels on right side). There is fluid centered around the vertebral body with sclerotic changes and endplate erosions, suggestive of osteomyelitis and discitis.

extensive segmental and sub segmental PE bilaterally with peripheral ground glass opacities in the right middle and lower lobes, suggestive of a pulmonary infarct (Figure 1A, 1B). To establish the etiology of PE, ultrasound of the lower extremities was done, which showed an acute deep venous thrombosis (DVT) involving the left common femoral vein down to the level of the left popliteal vein. The patient was admitted to the intensive care unit (ICU) and was started on IV unfractionated heparin. He continued to be febrile in the ICU and because of his flank pain and history of psoas abscess in the past, a CT of his abdomen and pelvis with contrast was done, which showed very large multiloculated, septated, bilateral retroperitoneal, primarily psoas, abscesses as well as findings in the lower lumbar spine worrisome for osteomyelitis and discitis from L3 to S1. Moreover, the left-sided abscess was pressing on the left common iliac vein (Figure 2), resulting in its occlusion and distal thrombus formation. Antibiotics were changed to IV vancomycin and IV piperacillin-tazobactam. Given his previous bacteremia, an echocardiogram was done, which showed no vegetations. Osteomyelitis of L3 to S1 was confirmed by the contrast-enhanced magnetic resonance imaging (MRI) of the lumbar spine.

A CT-guided drain was placed in the left psoas collection, yielding a total of 2 liters of pus. The Gram staining of the purulent drainage showed Gram-positive cocci in clusters. The cultures from the drainage showed few Gram-positive cocci in pairs initially, but final growth was negative. Repeat CT showed resolution of abscess and the drain was removed. Anticoagulation and antibiotics were continued, and the patient was discharged to a nursing home to complete his treatment on day 16 of hospitalization. After discharge, his adenosine deaminase (ADA) level from the fluid was elevated to 257 U/L but acid-fast bacteria were not found in staining or culture of the abscess.

During hospitalization, records from another institution showed that he had been admitted with similar complaints about 3 months ago and he had been diagnosed with lumbosacral osteomyelitis with epidural and bilateral psoas abscesses. He had undergone drainage of the left psoas abscess. Blood cultures showed *Streptococcus mitis* and psoas abscess cultures were sterile. He was treated with IV Ceftriaxone for 4 weeks. There was no report of DVT or PE during that admission. Given a previous history of epidural abscess, *Streptococcus mitis* bacteremia and presence of Gram-positive cocci in clusters on Gram staining of the fluid in our hospital suggests a bacterial etiology of the abscesses. He was treated with antibacterials for 6 weeks with the plan to consider treatment for tuberculosis if the abscesses recurred, given the high ADA level in the fluid. On follow-up at 4 months after the drainage of the abscesses, an MRI lumbosacral spine showed no evidence of osteomyelitis or any collection to suggest psoas abscess.

## Discussion

The iliopsoas muscle originates from the anterior aspect of the lumbar vertebrae. Caudally, it inserts into the lesser trochanter of the femur. During its course, it crosses the kidneys, the ureters, the pancreas, and the small intestine [1]. A retrofascial psoas abscess is a rare condition that has been described as a fluid collection within the fascia of the psoas muscle, containing leukocytes and microorganisms [5]. Iliopsoas or psoas abscesses can either be primary or secondary. Primary abscesses occur from hematogenous spread or lymphatic seeding of an occult focus of infection and are most commonly caused by *Staphylococcus aureus*, followed by *Streptococci* species and *E. coli*. Blood cultures are only positive in 42% of patients with a primary psoas abscess, which might explain the low mortality rate with bacteremia due to this abscess as compared to other staphylococcal bacteremias [2]. Secondary abscesses result from spread of an infective focus in surrounding structures. Crohn's disease is the commonest cause, but conditions like colon diverticulitis, appendicitis, pancreatitis, urinary tract infections, septic arthritis, and vertebral osteomyelitis are usually associated with secondary psoas abscesses [2,5]. Other causes of secondary psoas abscesses include surgery and instrumentation of structures adjacent to the muscle [3,6]. The usual etiologic agents of secondary abscesses are mixed infections, with a predominance of enteric bacteria [2].

Primary psoas abscesses are more prevalent in males than in females, while there is no sex difference in secondary psoas abscess formation [7]. Unilateral psoas abscesses can occur on either side, with roughly equal frequency. The presence of bilateral psoas abscesses is uncommon and is reported to be 1–5% in most studies, but as high as 30% in other studies [2,8–10]. Psoas abscesses usually present with back or flank

pain, fever, a mass in the inguinal region, inability to walk, anorexia, and weight loss. The classic triad of fever, abdominal or back pain, and limitation of hip joint movements may be present in only 35% of patients [11]. Fever, weight loss, and constitutional symptoms are observed in less than 40% of cases [12]. Complications of psoas abscesses include sepsis, DVT [7,13], hydronephrosis and/or bowel ileus, septic arthritis, and osteomyelitis of the vertebrae, hip, and sacroiliac joints [5].

Abdominal ultrasound is the usual initial imaging technique when a psoas abscess is suspected. It can detect hypo echoic lesions suggestive of psoas abscess in about 60% of patients. The gold standard imaging modality is intravenous contrast-enhanced spiral CT. However, MRI has 90% sensitivity and 80% specificity in diagnosing psoas abscess, gives a better view of the spinal canal, and provides complete evaluation of the spinal pathology [14,15].

The psoas abscess should be treated with drainage and appropriate antibiotics [16]. Drainage can be performed percutaneously under radiological guidance or surgically. Percutaneous catheter drainage (PCD) is less invasive and has low mortality, morbidity, and complication rates [16,17]. Surgical drainage is usually reserved for patients with persistence of an abscess even after PCD, or in those who have contraindications to PCD or who will undergo surgical treatment for another abdominal pathology [17]. According to Buttaro et al. [3] mortality is 2.4% in primary abscesses and 19% in secondary abscesses. Ricci et al. [2] suggested that mortality in untreated patients approaches 100%.

In our patient, the etiology of the vein thrombosis is speculated to be venous stasis and endothelial damage. The venous stasis was caused by extrinsic compression of the iliac vein by the psoas abscess. The endothelial damage was due to adherence and migration of leukocytes to the vessel walls and the resultant desquamation of these endothelial cells and exposure of the sub-endothelial structures due to sepsis [18,19]. The end result was thrombus formation. In their review, Rosenfeld et al. showed that 2% of the fatal PEs that had DVTs associated with decreased blood flow in the lower extremity veins were caused by external pressure from pelvic masses [20]. The usual treatment for PE is usually with a quick-onset anticoagulant followed by warfarin for 3 months except in patients with high risk of recurrence, where more extended duration of treatment is required [21]. Newer oral anticoagulants, which include factor Xa inhibitors (rivaroxaban and apixaban) and factor II inhibitor (dabigatran), are approved in the USA for treatment of PE [22]. For patients with PE in whom anticoagulation is contraindicated or the risk of bleeding is very high, an inferior vena cava filter can be placed [22]. For hemodynamically unstable patients, IV thrombolysis is recommended [21].

What led to the psoas abscess in the first place is unknown, as our patient was not an intravenous drug user, was

immunocompetent, and had intact skin. Further elicitation for a possible etiology was non-revealing. The spread of the infection in our patient could be explained by the secondary invasion of the retrofascial space from supportive lymph nodes from an epidural abscess, dating back to his prior admission.

Interestingly, ADA level measured from the abscess was markedly elevated. ADA is an enzyme that plays a role in the purine catabolism. It is involved in the differentiation of T-lymphocytes [23]. Hence, ADA has been thought to be a marker of cell-mediated immunity, particularly T cell activation. There are several different isoenzymes of ADA; the 2 most important are ADA1, which is found in all cells, including lymphocytes and monocytes, while ADA 2 is only found in monocytes [23]. Ungerer et al. reported that ADA 1 is elevated in pyogenic bacterial infection of the pleural cavity, while ADA 2 is elevated in tuberculous pleurisy [24].

A microbial conclusion is achieved in most psoas abscess collection through cultures of drained samples. In our case, the drained purulent fluid from the abscess in both hospitalizations yielded no microorganisms, and there was no lymph node enlargement on CT scan to attribute to secondary spread of psoas abscess, suggesting the relapse could be a paradoxical reaction to treatment [25]. At the end of treatment our patient had significantly improved and was independently performing activities of daily life.

Upon review of the literature, we did find a case of right-sided psoas abscess leading to DVT in the common iliac vein [7] and a left-sided psoas abscess leading to DVT in the left popliteal vein [13] but neither of these reported associated PE. Few cases of pelvic masses compressing on iliac veins leading to DVT and PE have been reported [4]. Our case of a bilateral psoas abscesses leading to lower extremity DVT and extensive bilateral PE is very rare considering that our careful literature review found no such case report.

## Conclusions

Clinicians should have a high index of suspicion for a psoas abscess in a febrile patient presenting with back pain and should be vigilant of the size effect such an abscess may have, leading to complications. A psoas abscess should be appropriately managed with complete drainage and intravenous antibiotics. The patients should be followed up at the end of treatment with CT scan or MRI for resolution of the abscess and early detection of complications if drainage has been incomplete.

## Conflict of interest

The authors of the manuscript have no conflicts of interest to declare.

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