
CASE REPORT

Bilateral Pyogenic Psoas Abscesses with Inferior Vena Cava Thrombosis

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ABSTRACT

Psoas abscess is a rare clinical entity. Rarely, it is associated with thrombosis of the inferior vena cava. This report is of a 36-year-old woman with bilateral pyogenic psoas abscesses with spondylodiscitis complicated by thrombosis of the inferior vena cava. The pathogenesis and imaging features of this condition are discussed.

Key Words: Discitis; Infection; Psoas abscess; Thrombosis; Tomography, X-ray computed; Vena cava, inferior

INTRODUCTION

Psoas abscess is an important and potentially life-threatening infection. If the diagnosis and management is delayed, complications can result. The morbidity and mortality of psoas abscess are high.¹ This report is of a woman with bilateral pyogenic psoas abscesses with spondylodiscitis and inferior vena cava thrombosis.

CASE REPORT

A 36-year-old Indian woman presented in 2003 with a 3-week history of progressive low back pain associated with a high-grade fever. Her condition worsened 3 days prior to presentation, such that she could not walk due to severe abdominal and low back pain. She had no symptoms suggestive of tuberculosis, or any history of weight loss or urinary symptoms. She was not diabetic or immunologically compromised. Her past medical history was uneventful.

At physical examination, she was pale and dehydrated, and had a temperature of 38°C. She had generalised abdominal tenderness on palpation, although her abdomen was not distended and had normal bowel sounds. No organomegaly or palpable mass was detected. Spinal examination revealed tenderness over the lower

lumbar region, with reduced spinal movements. Straight-leg raising was restricted to 60° bilaterally. The power, tone, and reflexes were normal, and the Babinski signs were plantar bilaterally. No sensory disturbance could be elicited, and anal tone was normal with no perianal anaesthesia. Initial laboratory evaluation demonstrated mild anaemia with a haemoglobin value of 92 g/L (normal range, 120-150 g/L), leukocytosis of 16.5 x 10⁹/L (normal range, 4.5-11.0 x 10⁹/L) with neutrophilia of 0.83 (normal range, 0.56 proportion of 1.00), and a markedly elevated erythrocyte sedimentation rate of >150 mm/hour (normal range, 0-20 mm/hour).

Radiograph of the lumbosacral spine showed a reduced intervertebral disc space at L3/L4 with destruction of the adjacent end plates and erosion of most of the upper half of the L4 vertebral body and superior end plate (Figure 1). The chest radiograph was normal. Contrast-enhanced axial computed tomography (CT) of the abdomen revealed multiseptated hypodense lesions with rim enhancement in the left and right psoas muscles measuring 8.2 x 5.5 x 16.0 cm and 7.2 x 6.3 x 15.0 cm, respectively (Figure 2). There was extension into the iliacus muscle on the left side. No calcification was present. There were inflammatory changes in the surrounding soft tissues. A filling defect was present in the infrarenal inferior vena cava (IVC) at the L3/L4 level measuring 6.0 x 1.5 cm (Figure 3). The kidneys, pancreas, gall bladder, and liver were normal. A diagnosis of L3/L4 spondylodiscitis with bilateral psoas abscesses and IVC thrombosis was made. Thrombosis of the IVC was a clinically significant finding and was recognised as an unusual complication of psoas abscess.

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Figure 1. Sagittal reformat computed tomography scan demonstrating a reduced intervertebral space between L3/L4 associated with erosion of the superior end plate of L4 (arrow), in keeping with spondylodiscitis.

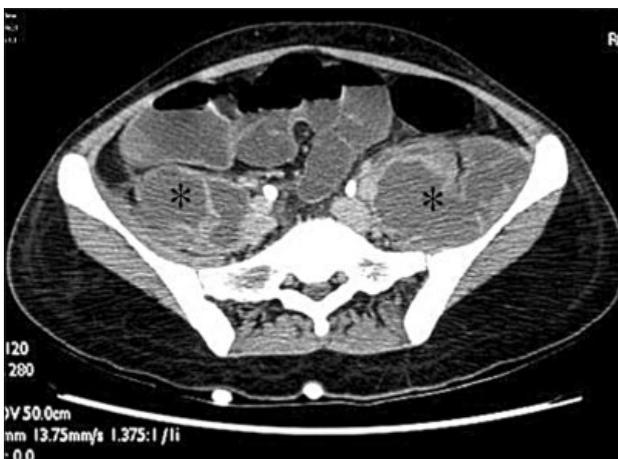


Figure 2. Contrast-enhanced axial computed tomography scan of the abdomen showing multiseptated hypodense lesions in the bilateral psoas muscles (asterisks), suggesting the presence of abscesses.

The patient was given intravenous cloxacillin 1 g every 6 hours. However, her condition deteriorated when she developed dyspnoea and tachycardia with poor oxygen saturation. A clinical diagnosis of pulmonary embolism was made, which was found to be unsubstantiated on CT pulmonary angiogram. Further laboratory evaluation revealed evidence of disseminated intravascular coagulopathy with thrombocytopenia and prolonged coagulation profiles. Liver function tests showed hypoalbuminaemia of 17 g/L (normal range, 35–50 g/L).

Fluoroscopy-guided IVC filter insertion was performed via the right internal jugular vein. A Gunther Tulip retrievable vena caval filter (Cook; Bloomington, USA) was positioned at the L2/L3 level under fluoroscopic

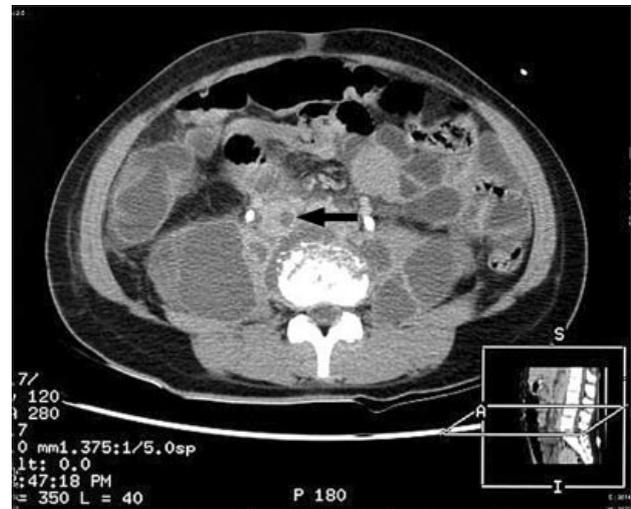


Figure 3. Contrast-enhanced computed tomography scan of the abdomen showing a filling defect in the inferior vena cava (arrow), in keeping with inferior vena cava thrombosis.

guidance. In view of the patient's worsening condition, the abscesses were drained surgically. Drainage was performed on the third day after admission and 1 L of greenish pus was drained. The culture grew methicillin-sensitive *Staphylococcus aureus* that was sensitive to cloxacillin. Blood culture grew the same organism. Examination for tuberculosis was negative. The dose of cloxacillin was increased to 2 g every 4 hours. Unfortunately, the patient's condition progressively deteriorated and she died of septicaemia 2 days after surgery.

DISCUSSION

Psoas abscess can be classified as either primary or secondary.² Primary abscess occurs as a result of haematogenous spread of infection,² and occurs in conditions in which patients are immunocompromised, such as diabetes mellitus, renal failure, intravenous drug abuse, and acquired immunodeficiency syndrome.² Primary psoas abscess occurs mostly in children and young people.² The predominant pathogen isolated in a primary abscess is *S aureus*, in 80% to 90% of patients.³

Secondary abscess are associated with local pathology.² Common causes include Crohn's disease, diverticulitis, appendicitis, urinary tract infection, and septic arthritis.² The pathogens are mixed. The predominant organisms are *Escherichia coli*, *Bacteroides* spp, *Staphylococcus* spp, and *Streptococcus* spp.³

Primary psoas abscess has a better prognosis than secondary psoas abscess, with mortality rates of 2.4% and 18.9%, respectively.³ The major reason for mortality is delayed or inadequate therapy. With delayed management,

a psoas abscess can result in extension of infection into adjacent anatomy such as the kidneys or spine. Another complication is septicaemia, which occurred in this patient only 3 weeks after the onset of symptoms.¹ Thrombosis of the IVC is an extremely unusual consequence of the condition.

Acute IVC thrombosis is a life-threatening condition. Virchow described the triad of stasis, vessel injury, and hypercoagulability as the foundation of the pathophysiology of deep vein and IVC thrombosis.³ Extrinsic compression of the IVC from any intra-abdominal space-occupying lesions, such as the extensive right psoas abscess in this patient, results in distortion of the normal vena cava anatomy and induces both venous stasis and turbulent flow, which facilitates thrombus formation.³ Another possible aetiology for this patient is disruption of balance between the coagulation and fibrinolytic systems secondary to disseminated intravascular coagulopathy, which can also result in IVC thrombus formation.³

Although a patient with IVC thrombosis may present with a spectrum of signs and symptoms, the symptomatology depends on the anatomic location of the thrombus and the degree of lumen stenosis.³ The classic features are bilateral lower extremity oedema with dilated superficial abdominal veins. However, one study reported that 60% of patients did not have this feature.³ If the thrombus is confined to the vena cava and does not involve the iliac or femoral system, collateral pathways form along the posterior abdominal wall. Thrombosis at the level of the renal veins commonly suggests a nephrotic syndrome or renal cell carcinoma. A thrombus occluding the IVC at the juxtarenal level may affect renal function by altering renal perfusion. Patients with IVC thrombosis may also present with pulmonary embolism as the first sign.³

Contrast venography, ultrasound, CT, and magnetic resonance imaging (MRI) are the currently available imaging modalities for diagnosing IVC thrombosis.⁴ Contrast-enhanced CT can provide information about the localisation and upper extent of an IVC thrombus.⁵ However, a hyperdense recent thrombus may not be appreciated on contrast-enhanced CT due to its density, as also occurs with contrast medium in the vena cava.⁴ Zerhouni et al described 3 features of a thrombosed vein on CT: enlargement of the thrombosed vein, low-density lumen, and a sharply defined wall.⁶ False-positive results sometimes occur due to pseudothrombosis of the infrarenal IVC, which is thought to result from variable contrast in

the vena cava above and below the renal veins. Another possible cause is collapse of the IVC at the diaphragm when the patient is in the supine position.⁵

The current treatment options for IVC thrombosis are either medical treatment or IVC filter insertion. Medical treatment includes anticoagulation using anticoagulating and thrombolytic agents.⁶ The absolute indications for IVC filters are contraindications to anticoagulation, recurrent thromboembolic disease despite anticoagulation therapy, and complications of anticoagulation therapy.⁷ Currently, IVC filters are also inserted for prophylaxis for patients at high risk for thrombosis, for example, patients who have experienced severe trauma, and those with a history of thromboembolic disease who are to undergo surgery.⁷

In the patient described in this report, an IVC filter was inserted, as medical therapy was contraindicated due to the deranged coagulation profiles. Vena cava filters do not provide absolute protection against pulmonary embolism,⁸ and can even become a source of pulmonary embolism, particularly in patients with large IVC clots that propagate through the filter. Anticoagulation is commonly used as an adjuvant to a vena cava filter for patients without contraindications for anticoagulation.⁸

Psoas abscess needs to be recognised as a rare life-threatening disorder with multiple complications. Prompt management must be instituted to save life.

REFERENCES

1. Walsh TR, Reilly JR, Hanley E, Webster M, Peitzman A, Steed DL. Changing etiology of iliopsoas abscess. *Am J Surg.* 1992; 163:413-6.
2. Santaella RO, Fishman EK, Lipsett PA. Primary vs secondary iliopsoas abscess: presentation, microbiology and treatment. *Arch Surg.* 1995;130:1309-13.
3. Desandre AR, Cottone FJ, Evers ML. Iliopsoas abscess: etiology, diagnosis and treatment. *Am Surg.* 1995;61:1087-91.
4. Sonin AH, Mazer MJ, Powers TA. Obstruction of the inferior vena cava: a multiple modality demonstration of causes, manifestations and collateral pathways. *Radiographics.* 1992; 12:309-22.
5. Castillo M, Nunez D, Morillo G. Review of computed tomography findings in thrombosis of the major abdominal venous pathways. *J Comput Tomogr.* 1986;10:205-14.
6. Zerhouni EA, Barth KH, Siegelman SS. Demonstration of venous thrombosis by computed tomography. *AJR Am J Roentgenol.* 1980;134:753-8.
7. Kinney TB. Update on inferior vena cava filters. *J Vasc Intervent Radiol.* 2003;14:425-40.
8. Streiff M. Vena caval filters: a comprehensive review. *Blood.* 2000;95:3669-77.