

CASE REPORT

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A case of psoas abscess in a patient with systemic sclerosis and rheumatoid arthritis

Received: August 8, 2000 / Accepted: January 9, 2001

Abstract We report a case of psoas abscesses associated with systemic sclerosis and rheumatoid arthritis. A 61-year-old woman had been suffering from high fever. A computed tomography (CT) scan revealed bilateral psoas abscesses from which *Peptostreptococcus* spp. were detected by a culture of the pus. The abscesses were ameliorated by performing CT-guided percutaneous drainage and using appropriate antibiotics. Although psoas abscess is relatively rare, it can be a cause of fever of unknown origin in collagen diseases.

Key words Psoas abscess · Rheumatoid arthritis · Systemic sclerosis

Introduction

Psoas abscess is a rare life-threatening infection, the diagnosis of which tends to be delayed because of the nonspecific clinical symptoms. Reports of this entity from the early and mid-1900s uniformly showed *Mycobacterium tuberculosis* to be the predominant pathogen, but recently *Staphylococcus aureus* and *Escherichia coli* have been reported to be the predominant pathogens.¹

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Here we report a case of psoas abscesses associated with systemic sclerosis (SSc) and rheumatoid arthritis (RA). The patient was successfully treated with computed tomography (CT)-guided percutaneous drainage and appropriate antibiotics. A high degree of suspicion for unusual sites of infection must be maintained when one is caring for immunocompromised patients.

Case report

A 61-year-old woman with systemic sclerosis of 6 years' duration and seropositive rheumatoid arthritis of 5 years' duration was admitted with spike fever on March 31, 2000. Before being admitted to our hospital, she had been in another hospital for 6 months and had had intermittent fever which was ameliorated by the empirical use of antibiotics (piperacillin, levofloxacin, cefditoren pivoxil, cefmetazole, isepamicin, ceftiofuran, cefotiam, and minocycline). She was immobile in bed because of severe muscle atrophy from disuse, and had become anorexic and weak. She had received 10–20 mg prednisolone daily for 5 years because of the arthralgias. Actarit (300 mg daily), sulfasalazine (1000 mg daily), and mizoribine (150 mg daily) had also been given.

On admission she was semiconscious and dehydrated, with a high fever of over 40°C. Examination revealed a fine crackle in the lower lung field and psoas signs. Neurological findings were normal.

Initial test results were as follows: hemoglobin 8.9 g/dl; WBC $19.9 \times 10^3/\text{mm}^3$; platelets $47.2 \times 10^4/\text{mm}^3$; creatinine 0.25 mg/dl; c-reactive protein (CRP) 30.4 mg/dl. A urine specimen had 31–40 WBC/hpf and gram-positive cocci.

The patient was rehydrated and started on piperacillin after blood and urine cultures. Organism culture from the blood was negative, but the urine was positive for *Enterococcus faecalis*, *Enterococcus faecium*, and *Candida albicans*. Prednisolone (10 mg daily) was continued intravenously, but actarit, sulfasalazine, and mizoribine were stopped because she could not take medicines.



Fig. 1. Computed tomography (CT) scan of the abdomen showing bilateral psoas abscesses 12 days after admission

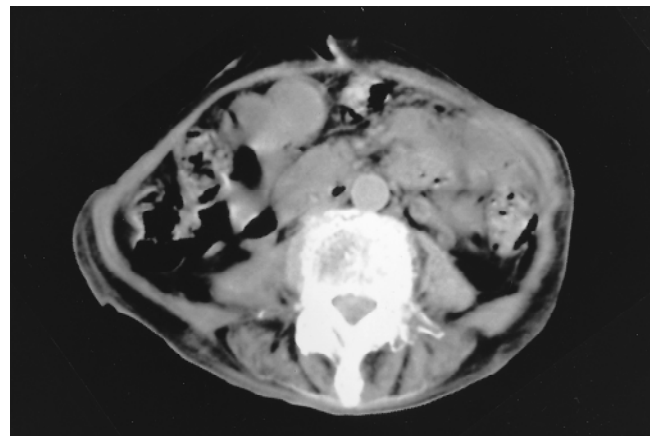


Fig. 2. CT scan of the abdomen showing intact psoas muscles 52 days after admission

Her temperature was lowered to between 37°C and 38°C by using piperacillin, and her CRP dropped to 9.4 mg/dl on day 7. Seven days after admission, a metallic sound was clearly heard on abdominal auscultation, and abdominal X-rays showed marked small packets of intestinal gas. These conditions were soon ameliorated by the use of metoclopramide and panthenol.

To see whether there were any apparent lesions in the peritoneal space that could be the cause of intestinal obstruction, an abdominal CT scan was performed 12 days after admission. The scan showed bilateral hypodense lesions in the psoas muscles (Fig. 1). On day 31, the lesions were aspirated under CT guidance and about 100 ml and 50 ml of thick yellow pus were removed from the right and left psoas abscesses, respectively. Drainage catheters were inserted in both abscess cavities. Organism culture from the aspirated fluid was positive for *Peptostreptococcus* spp. *Mycobacterium tuberculosis* was not identified by polymerase chain reaction and organism culture of the specimen.

The patient was started on ampicillin in accordance with the sensitivity test for antibiotics. On the 47th day, her WBC count had decreased to $7.6 \times 10^4/\text{mm}^3$ without a leftward shift, and her CRP had decreased to 1.2 mg/dl. On the 52nd day, CT showed that the abscesses were no longer present (Fig. 2) and the drainage catheters were removed.

Discussion

Psoas abscess remains a relatively uncommon clinical entity. Since Mynter's original description of pyogenic iliopsoas abscess in 1881,² more than 400 cases have been reported in the world literature.^{3,4} Although *Mycobacterium tuberculosis* was the predominant pathogen in the early and mid-1900s, *Staphylococcus aureus* and *Escherichia coli* are now the most common organisms in primary and secondary psoas abscess, respectively.¹

In the present case, the clinical symptom relating to the psoas abscesses was fever. Arthralgias and back pain were the patient's usual complaint up to that time, and had been suspected to be due to rheumatoid arthritis and her long-term bedridden condition. Her fever was diagnosed as due to a urinary tract infection, because urine tests showed moderate pyuria and gram-positive cocci, and piperacillin was effective.

A CT scan has become the standard means of diagnosing psoas abscess, with a sensitivity of over 90% and specificity of over 80%.⁵ Ultrasound is also useful as a screening test in that it is cheap and rapid. An additional advantage is that CT or ultrasound guidance allows safe percutaneous needle placement and the collection of samples from the psoas abscess. It is very important to determine which antibiotics are effective against the pathogens responsible. We performed a CT scan to investigate the cause of ileus and discovered the bilateral psoas abscesses. An organism culture from the aspirated fluid was positive for *Peptostreptococcus* spp., which was the predominant pathogen, and was isolated from 43.9% (18/41) of retroperitoneal abscesses in children.⁶

SSc has various gastrointestinal manifestations⁷ in which the esophagus is frequently involved. The involvement of the small intestine and colon is less common, but may lead to life-threatening complications such as pseudoobstruction or pneumatosis cystoides intestinalis (PCI). PCI, a condition characterized by multiple gas-filled cysts in the submucosa and/or subserosa, may be the cause of the psoas abscesses because it is often complicated by a spontaneous pneumoperitoneum, which may arise from mucosal microperforation. However, in the present case it was not likely that the psoas abscesses were associated with PCI or microperforation since the CT scan of the abdomen did not show pneumoperitoneum or a gas-filled wall of small intestine. In addition, the bacteria identified from the patient's abscesses was *Peptostreptococcus* spp. rather than *Escherichia coli*, which is most often identified in secondary psoas abscesses caused by penetration or perforation of the small intestine and colon to the psoas muscle.

Ricci et al.⁴ reported that primary abscesses occurred equally in the right and left psoas muscles (46.5% vs. 52.1%, respectively, bilateral 1.4%, 142 patients), while secondary abscesses occurred predominantly in the right muscle (68.0% vs. 29.2%, respectively, bilateral 2.8%, 72 patients). In addition, there was a distant focus of infection in 16% of the patients with primary abscesses. Based on this microbial profile and etiology, we speculated that the abscesses in our case arose as a result of hematogenous dissemination from an unknown distant focus of infection.

Regarding psoas abscesses associated with autoimmune disease, Isdale et al.⁸ reported only two patients with a psoas abscess in RA. However, some patients with autoimmune diseases may have psoas abscesses when they suffer from fever of unknown origin. There should be a strong suspicion of an unusual site of infection when one is caring for immunocompromised patients.

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