Fatal meningitis secondary to undetected bacterial psoas abscess

Report of three cases

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Three unusual cases of fatal meningitis secondary to undetected bacterial psoas abscess occurred at this institution over an 11-year period. All three patients had suffered chronic debilitating disorders before the abscess formation. The superimposed variable clinical presentations led to the initial diagnosis of a progressing cerebrovascular accident in one case, herniated nucleus pulposus at the L3-4 level in another, and osteomyelitis of the hip joint in the third. Analysis of these cases revealed that before the meningeal dissemination, all of the patients had shown evidence of intra-abdominal pathology with positive psoas signs. Diagnostic and therapeutic guidelines are discussed.

KEY WORDS • psoas abscess • meningitis

THREE cases of fatal meningitis caused by the extension of undiagnosed psoas abscesses have been seen at this institution in the past 11 years. This complication of a psoas muscle infection was not encountered in our literature review. An analysis of these three patients is presented.

Case Reports

Case 1

This 55-year-old man was readmitted in July, 1974, with a traumatic fracture of the left hip. His past medical history included diabetes mellitus, chronic alcoholism, chronic obstructive pulmonary disease, seizure disorder, and malabsorption syndrome presumed to be secondary to pancreatic insufficiency. Before this admission, he had been treated for two episodes of E. coli urinary tract infections, recurrent perianal abscesses (staphylococcal), and bilateral aspiration pneumonia. Ten years before admission, a left subphrenic abscess caused by E. coli was drained surgically.

The left comminuted intertrochanteric fracture was repaired by open reduction with internal fixation. The subsequent course was complicated with intractable infection at the nailing site with development of osteomyelitis and destruction of the femoral head, despite removal of the nail and treatment with antimicrobial agents. Bone biopsy as well as a deep aspirate of the draining sinus from the left hip grew E. coli, Staphylococcus aureus (coagulase-positive), and beta hemolytic streptococcus (non-group A). The patient was treated with intravenous...
methicillin for 6 weeks with simultaneous intramuscular gentamicin, and then changed to oral cloxacinilin with probencid. On this regimen he became afebrile and there was gradual but complete resolution of the draining sinus. A concomitant sacral decubitus ulcer also resolved, and he remained afebrile throughout the course of wound healing except for a brief period when he developed documented urinary tract infection treated successfully with a suprapubic cystostomy.

Nearly a year later he developed right hip pain associated with daily temperature spikes of 101° to 102° F, with night sweating episodes. Oral cloxacinilin, which had been continued in the therapy of his osteomyelitis, was discontinued. Urine and two blood cultures were taken. X-ray films of the chest and right hip were unremarkable, but radiographs of the left hip revealed persistent osteomyelitis. Urine cultures grew more than 100,000 colonies of Enterobacter aerogenes. Physical examination revealed a chronically ill, uncooperative man who was alert and fully oriented. No abdominal or meningeal signs were found. A healing decubitus ulcer and a suspicious lesion in the perirectal area were noted. Two blood cultures grew Gram-negative rods and Gram-positive cocci with a morphology consistent with anaerobic flora.

An intra-abdominal or pelvic abscess of anaerobic etiology was suspected, and the patient was immediately started on chloramphenicol (4 gm/day) along with 6 gm of intravenous methicillin (1 gm every 4 hrs). During the next 3 to 4 hours he developed a stiff neck, and became unresponsive with fixed dilated pupils. Laboratory data at that time revealed a leukocytosis of 18,000 with 86% polymorphonuclear cells (PMN’s) and arterial blood gases revealed respiratory alkalosis. Ruptured brain abscess was the presumptive diagnosis, but an emergency brain scan showed no evidence of a focal lesion. A lumbar puncture disclosed extremely foul-smelling purulent cerebrospinal fluid (CSF). The CSF protein was 1.8 gm/100 ml, there were 100,000 white blood cells (WBC), with 75% PMN’s, and 17,000 red blood cells (RBC), and a glucose of 7 mg/100 ml. Gram stains revealed organisms identical to those found in the previous blood cultures.

The patient’s condition continued to deteriorate and he died that night. At autopsy a large right psoas abscess was found extending posteriorly to the gluteus maximus. Direct extension into the spinal canal at the level of L3-4 was demonstrated with a resultant ascending purulent meningitis. No other sources of infection were located, nor was there communication from the psoas abscess to either hip. The multiple cultures obtained from this case are summarized in Table 1.

Case 2

This 47-year-old alcoholic and chronic schizophrenic man was admitted on June 30, 1972. His chief complaint was a “nagging toothache-like pain” in the left flank. Night sweats had occurred daily for 2 weeks, and he noted that coughing, sneezing, and extending the left thigh increased the back pain. This painful discomfort began 4 weeks previously and in the last 3 to 4 days had become excruciating. In the last few days he had also

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**TABLE 1**

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<th>Origin of Specimen</th>
<th>Gram-Stain Morphology</th>
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<td>Gram-neg. rods and</td>
<td>Peptostreptococcus</td>
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<td>Gram-pos. cocci</td>
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<td>psoas abscess</td>
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Meningitis secondary to psoas abscess

noticed episodes of visual “blackout” without unconsciousness that lasted only a few seconds.

Past medical history disclosed a diagnosis of Gaucher’s disease in 1953, confirmed by lymph node and liver biopsy. He had undergone splenectomy in 1953.

Physical examination showed this patient to be in acute distress with a temperature of 99° F, pulse 88/min, and blood pressure 130/80. The pupils reacted normally, and the funduscopic examination was benign. The liver was palpable below the right costal margin. There was marked tenderness on the left side of the abdomen, with abnormal motor function due to weakness of the left iliopsoas muscle. Admission laboratory data included 17,700 WBC with 75% PMN’s. All other initial values were normal including a negative purified protein derivative (PPD) and blood cultures.

Several days after admission the patient’s temperature was 103° F. Chest x-ray films were normal. Temperature later fell to 98° F and a myelogram was attempted to rule out lumbar disc; this was unsuccessful, with two dry taps. The following day a myelogram was again attempted and a small amount of bloody fluid obtained. Following attempted myelography the patient’s temperature rose to 103° F, and he became notably disoriented, incontinent of urine and stools, and had weakness of the legs and arms. Over the next 48 hours he responded only to painful stimuli. Examination of the CSF obtained at the second myelogram attempt revealed a glucose of 52 mg/100 ml, WBC 1050/cu mm with 75% PMN’s, and RBC 25,000/cu mm; the CSF cultures grew Diplococcus pneumoniae.

Examination of the lumbar spine radiographs taken at admission revealed that the patient’s calcified aorta was displaced anteriorly by a mass at the L3-4 level (Fig. 1). Pain was elicited by percussion at the lumbar region. Straight leg raising also induced pain bilaterally, and neck stiffness was present. Diplococcal meningitis secondary to a paravertebral abscess was diagnosed, and the

Fig. 1. Lumbar spine film in Case 1. Left: Film taken 1 year before admission. Right: Film taken on admission, showing anterior displacement of the calcified aorta by a mass lesion.
patient's condition remained critical with a few signs of improvement on antibiotics and medical management. Subsequently, transabdominal drainage of a psoas abscess was performed with evacuation of 200 ml of odorless pus from the left iliopsoas muscle. Bacterial cultures taken at surgery revealed no growth. The patient's condition slowly deteriorated and he died. Mycobacterial and fungal cultures taken from the psoas abscess at 8 weeks were negative.

At autopsy there was extensive meningitis involving the cerebral hemispheres, spinal cord, and ependymitis of the lateral ventricles. Prominent edema of the brain and cord was present with enlargement of the fourth ventricle. Bilateral uncal herniation had occurred and there was also focal encephalomalacia in the putamen.

Case 3

A 69-year-old man was admitted on April 19, 1965, with confusion and progressive motor disability as a consequence of a "stroke" that occurred 1 year previously. The patient had been a heavy user of ethanol for many years, but there was no history of recent antibiotic therapy or other drug intake, and he had no problem with sphincter control.

Physical examination showed a small, emaciated, and chronically ill elderly man with a temperature of 98.6°F, pulse 130/min, blood pressure 120/80, and respirations 36/min. His extremities were flexed, and examination of the head revealed slight neck stiffness. Movement of the legs and the left arm was painful. Deep tendon reflexes were decreased, and a grasp reflex was noted in the left hand. Bilateral Babinski signs were present. The chest x-ray film taken on admission showed emphysematous changes, and there was an incomplete right bundle branch block on electrocardiogram. Initial laboratory data showed hematocrit 35%, WBC 3600, stool negative for occult blood, blood urea nitrogen 48 mg%, blood glucose 144 mg/100 ml, alkaline phosphatase 7.2 King-Armstrong (KA) units, and acid phosphate 2.8 KA units.

Shortly after admission the patient's temperature increased to 101°F, and progressive mental obtundation, rigidity of the extremities, and incontinence of urine and stool followed. The patient's condition rapidly deteriorated and he became responsive only to deep pain, despite massive penicillin therapy. On the second day after admission he died.

At autopsy an unexpected acute left-sided psoas abscess was found. Cultures revealed the offending organisms to be Klebsiella and Streptococcus fecalis. An acute, extensive, purulent meningitis was the cause of death.

Discussion

Psoas abscesses represent approximately 6% of all intra-abdominal purulent collections, and are usually caused by inflammatory processes that extend directly into the iliopsoas muscle. Diverticulitis, appendicitis, and Crohn's disease are the most frequent associated disorders. However, suppurrative lymphadenitis, direct extension of peritoneal abscesses into the retroperitoneal space, and septicemic dissemination of bacteria (especially when previous trauma to the muscle has occurred) are other means by which a septic focus may be established within the muscle.

The most common pathogens are Staphylococcus aureus, Escherichia coli, and Enterobacter-Klebsiella species; anaerobic organisms, as reported here, are very unusual. After infection of the psoas muscle, dissemination into the subarachnoid space may occur either by direct extension as in Case 1, or through the invasion of the paravertebral venous plexus. As described by Batson and others, this venous plexus lacks valvular structures and the blood can flow within it in either direction, depending only on intrathoracic and intra-abdominal pressure changes such as those elicited during straining, sneezing, and coughing.

Meningitis caused by dissemination of a psoas abscess is in itself a very unusual complication, and a review of the literature provided no similar case reports. One case of a spinal epidural abscess arising from a primary psoas infection has been reported recently by Baker, et al.

The salient clinical features of psoas abscess presentations are as follows. The usual chief complaint is pain localized to the lower back which radiates ipsilaterally either to the hip, or toward the inguinal region. Full extension of the thigh and abdominal exertion during coughing, sneezing, or straining worsens this pain. As the condition deteriorates, the patient is unable to walk, and
remains bedridden with his legs flexed. Minimal extension or lateral rotation of the thigh induces severe pain (psoas sign). A tender, fluctuating mass may be palpated just above Poupart's ligament. Some cases may not reach this last stage, or the abscess may extend posteriorly into the gluteus maximus (Case 1), making clinical diagnosis even more difficult. Fever, leukocytosis, and an elevated sedimentation rate frequently co-exist, which may lead to a differential diagnosis of intra-abdominal inflammatory or infectious disorders. In other instances the diagnosis may be focused upon hip or pelvic bone disorders such as osteomyelitis (Case 1). Radiation of the pain following either the femoral nerve distribution to the inguinal and proximal thigh area, or that of the sciatic nerve following the buttock, hip, and posterior aspect of the thigh, mimic radiculopathies occurring at L2–3 and L4–S1 levels, respectively.

Radiographic examination after a barium enema or an intravenous pyelogram may reveal a mass displacing the intra-abdominal structures. Plain lumbar spine films may show clouding of the psoas shadow or on rare occasions reveal anterior displacement of a calcified aorta (Case 2). Gallium isotope scanning of the abdomen is the diagnostic procedure of choice, since it makes inflammatory and neoplastic lesions of the abdominal cavity readily apparent.

Surgical evacuation of an uncomplicated non-tuberculous psoas abscess usually leads to full recovery. Immediate Gram-stain examination of effusions and bacterial cultures for aerobic and anaerobic organisms should be initiated. Based on clinical and experimental data, a combination of both aerobes and Gram-negative anaerobes may be expected; the antibiotics of choice in uncomplicated psoas abscess are gentamicin and clindamycin. If meningitis has occurred, then a combination of penicillin with chloramphenicol should be used.

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References


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