Girl, 5, With Fever and Hip Pain

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A 5-year-old Filipino girl was brought to a pediatric clinic for follow up of an unresolved fever and for new-onset right hip pain, which occurred intermittently for the past week and was associated with a right-sided limp. She had been experiencing nightly fevers ranging from 101°F to 105°F for the past two weeks, for which her parents had been giving ibuprofen with mixed results; she remained afebrile during daytime hours.

Using the Wong-Baker FACES pain scale, the patient rated the pain as a 4/10 in severity ("Hurts a Little More" face).[1] Standing and walking aggravated the pain but did not limit activity. Although ibuprofen decreased the fever, it did not alleviate the hip pain. Other symptoms included vomiting one to two times daily, without hematemesis, and four to five episodes of diarrhea daily, without abdominal pain, hematochezia, or melena. She also experienced decreased appetite, but her parents reported no change in her dietary or fluid intake. The patient and her parents denied additional symptoms.

Further investigation revealed that the patient had been seen a week earlier by two other clinicians in the office for complaints of fever, rash, nausea, hematemesis, and diarrhea. She had been diagnosed with a herpes simplex viral (HSV) lesion of the nose, epistaxis, and viral gastroenteritis. Her treatment plan consisted of acyclovir ointment for the HSV lesion and symptomatic support for the gastroenteritis-associated diarrhea. The complaint of hematemesis was attributed to postnasal drip from the epistaxis, and reassurance was provided to the patient and family. In addition, six weeks earlier, the patient had been treated for otitis media with a full course of amoxicillin.

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Medical history was negative for surgeries, trauma, injuries, and chronic medical conditions. She took no medications or supplements on a regular basis. Her parents denied any known drug allergies and stated that her immunizations were up to date.

The patient lived at home with her biological parents and two brothers, all of whom were healthy, without any recent infections or illnesses. Of significance, the family had travelled to the Philippines for vacation about four months earlier. Results of a tuberculin skin test done six weeks earlier (because the patient presented with respiratory symptoms shortly after traveling to the Philippines) were negative.

Physical examination revealed a well-developed, well-nourished 40-lb girl, in no acute distress, who was active and playful with her brother while in the exam room. Vital signs were significant for a fever of 101.9°F (last dose of ibuprofen was approximately six hours earlier) but were otherwise stable. Skin exam revealed that the prior HSV lesion of the nose had resolved. HEENT, cardiovascular, and pulmonary exam findings were noncontributory. Urine dipstick was negative.

Abdominal exam revealed normoactive bowel sounds in all four quadrants, and on palpation, the abdomen was soft, nontender, and without organomegaly. Specialized abdominal exams to assess for peritonitis, including those to elicit Rovsing, rebound tenderness, obturator, and psoas signs, were all negative. Bilateral extremity exams of the hips, knees, and ankles revealed full range of motion (active and passive), with normal muscle strength throughout. The only significant finding on the physical exam was mild pain of the right anterior hip at 15° of flexion, appreciated while the patient was supine on the exam table. The patient was also observed pushing off her right lower extremity when climbing onto the exam table, and she skipped down the hall when leaving the exam.

With fever of unknown origin (FUO) and a largely

TABLE 1
Case Patient's Differential Diagnosis and Laboratory Test Results

Differential diagnosis	Test	Results (reference range)
Rheumatic fever	Throat culture	Negative
	Anti-DNase B	Negative
	ASO titer	1,180 u/mL (0-640)
Juvenile idiopathic arthritis, Reiter syndrome, rheumatoid arthritis	ANA	1.2 IU/mL (< 1.0)
	ESR	110 mm/h (0-10)
	CRP	190 mg/L (0-10)
	RF	Negative
Bacteremia	CBC count	WBC count, 17.5 x10^3/μL (4-11); Platelets, 445 x10^3/μL (150-450)
	Blood culture	Negative
UTI/pyelonephritis	Urine culture	Negative
Septic joint, osteomyelitis, avascular necrosis	Hip ultrasound with fine-needle aspiration	Negative
	Bone scan	Negative

Abbreviations: ANA, antinuclear antibody; anti-DNase B, antideoxyribonuclease-B titer; ASO, antistreptolysin O; CBC, complete blood count; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; RF, rheumatoid factor; UTI, urinary tract infection; WBC, white blood cell.

negative history and physical, the working list of differential diagnoses included

- Avascular necrosis
- Bacteremia
- Juvenile idiopathic arthritis
- Osteomyelitis
- Pyelonephritis
- Reiter syndrome
- Rheumatic fever
- Rheumatoid arthritis
- Septic joint
- Urinary tract infection

To begin the diagnostic process, a number of laboratory tests and imaging procedures were ordered. Table 1 presents the results of these studies. A tuberculin skin test was not repeated. While awaiting test results, the patient was started on naproxen oral suspension (125 mg/5 mL; 4 mL bid) for fever and pain control.

Based on findings consistent with an inflammatory pattern, the history of otitis media (of possible streptococcal origin) six weeks prior to this visit, and the elevated ASO titer, the patient was started on penicillin V (250 mg bid) and instructed to return for follow up in two days.

At the follow-up visit, no improvement was noted; the patient continued to experience nightly fevers and hip pain. Rovsing, rebound tenderness, obturator, and psoas signs continued to be negative. Physical examination did, however, reveal a mild abdominal tenderness in the right lower quadrant.

Due to this new finding, an abdominal ultrasound was ordered to screen for appendicitis. Despite the parents' appropriate concern for the child, misunderstanding about the urgent need to obtain the abdominal ultrasound led to a two-day delay in scheduling the exam. Results of ultrasonography revealed psoas abscess, and the patient was promptly admitted to the pediatric floor of the local hospital.

DISCUSSION

Psoas abscess is a collection of pus in the iliopsoas compartment, an extraperitoneal space containing the psoas and iliacus muscles.[2] It can be life-threatening if the infection progresses to septic shock. Historically, psoas abscesses were a frequent complication of tuberculosis (TB) of the spine; but with modern TB treatment, these abscesses have become rare.[2] Paradoxically, increased utilization of CT to evaluate sepsis of unknown etiology has led to

a recent increase in the frequency of psoas abscess diagnosis.[3]

Psoas abscesses are categorized as either *primary* or *secondary*, with primary infections originating in the psoas muscle and secondary infections spreading from adjacent organs.[2] In 42% to 88% of cases (depending on the study), primary psoas abscesses are caused by the hematogenous spread of *Staphylococcus aureus* from distant infection sites.[2,4,5] The psoas muscle is particularly susceptible to this mode of infection because of its rich vascular supply.[6] Children, immunosuppressed adults (ie, patients with diabetes, HIV/AIDS, or renal failure), IV drug users, and patients with a history of trauma to the muscle are most susceptible to developing a primary psoas abscess.[2,5]

Secondary psoas abscesses are caused by infections involving adjacent structures of the gastrointestinal, urinary, and skeletal systems. They are most frequently associated with intra-abdominal inflammatory processes, with the most common etiology being Crohn disease.[5] Secondary psoas abscesses, though more diverse in their bacterial flora, tend to follow certain microbiologic patterns based on the inoculating source; *Escherichia coli* is the most common pathogen in secondary abscesses caused by gastrointestinal (42%) and urinary (61%) sources, and *S aureus* the most common (35%) from skeletal origins (ie, osteomyelitis).[4,5] *Mycobacterium tuberculosis* is the more frequently found cause in developing countries but should be considered if the patient has recently travelled outside the United States.

Review of the literature suggests that the incidence of methicillin-resistant *S aureus* (MRSA) as the causative agent of psoas abscesses may be increasing. However, there is a wide variance in the incidence reported, ranging from 1.1% to 12% of confirmed microbial infections.[5,7,8]

The classic historical presentation of psoas abscess has been described as the triad of back pain, fever, and limp[5,6]; however, this triad has only been described in approximately 30% of cases.[5] The typical presentation consists of flank or lower limb pain (91%), fever (75%), anorexia (46%), and/or weakness (43%).[4] Laboratory abnormalities include leukocytosis (67%) and elevated markers of inflammation (eg, erythrocyte sedimentation rate, seen in 73% of cases).[4]

Imaging via abdominal ultrasound may be helpful to screen for psoas abscess; however, its utility is limited by a low diagnostic yield of 60% or less.[2,4] Direct visualization of the retroperitoneal structures, for example, can be problematic due to the presence of bowel gas.[9] Abdominal CT is considered the gold standard for the definitive diagnosis of psoas abscess due to its high sensitivity (100%) and specificity (77%); it can also be used simultaneously to guide percutaneous drainage to treat the abscess if needed.[7] However, some clinicians prefer abdominal MRI because of its ability to enhance soft-tissue visualization without requiring use of IV contrast.[2,4]

The approach to treating psoas abscess varies from a strictly antibiotic regimen to percutaneous drainage, and in rare circumstances, open surgical drainage. Antibiotic therapy without drainage or surgical intervention is a sufficient starting point for treatment of abscesses less than 3 cm in size.[3]

The antibiotic regimen choice depends on the suspected pathogen. In cases of suspected *S aureus,* empiric antistaphylococcal antibiotics should be initiated while culture results are pending.[2,4] Secondary psoas abscesses thought to be derived from a urinary or gastrointestinal source should prompt use of a broader spectrum antibiotic due to the higher probability of gram-negative, anaerobic, or polymicrobial involvement.[2,4]

Once final culture and sensitivity results are obtained, antibiotic therapy should be modified to target the isolated pathogen(s). Treatment duration is typically six weeks but may vary, depending on serial culture results and the inoculating source.[4] Review of the literature reveals that abscesses resulting from skeletal sources have traditionally been treated longer, usually with antibiotics alone, than those from urinary or gastrointestinal sources, which are often treated with the combination of antibiotics and percutaneous drainage.[4]

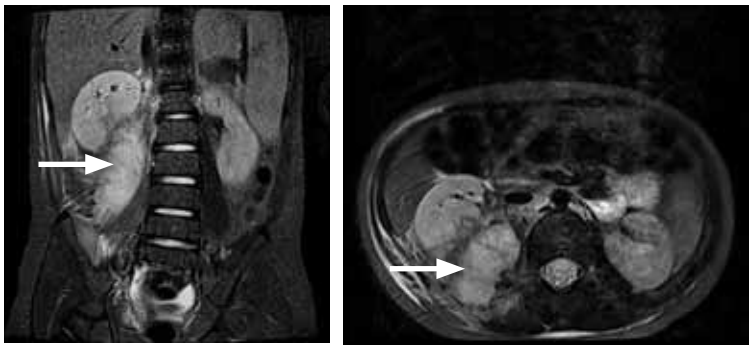
In cases of psoas abscesses larger than 3 cm, management should include both appropriate antibiotics and percutaneous drainage of the abscess.[2] Percutaneous drainage is preferred to open surgical drainage because outcomes are similar, it is less invasive, and there is less risk of spreading abscess contents.[2-4] In a retrospective analysis by Dietrich et al, 50% of patients treated with antibiotics and percutaneous drainage responded after one drainage, but the success rate increased to 100% after a second drainage.[7] In addition, percutaneous drainage was associated with a lower mortality rate and a shorter hospital stay when compared to open surgical drainage.[7]

Open surgical drainage is rarely performed and usually only considered if the patient is not respond-

ing to a combination of focused antibiotic treatment and percutaneous drainage or has associated comorbidities, such as Crohn ileocolitis.[2-4] In a retrospective analysis by Tabrizian et al, percutaneous drainage served as a bridge to open surgical drainage in nearly all patients with a gastrointestinal origin, such as Crohn disease, diverticulitis, appendicitis, and/or pancreatitis.[6]

Treatment of psoas abscesses has an overall failure rate of 15.8%, with an associated mortality rate of less than 7%.[4] Overall prognosis is good, but outcomes can be negatively affected by such factors as advanced age, delay in diagnosis, bacteremia, and other comorbidities.[4]

FIGURE 1



MRIs of the patient's abdomen with contrast (left, anteroposterior view; right, transverse view), performed at the time of hospital admission, demonstrate abnormal enhancement of the right psoas muscle (arrows), indicating right psoas abscess.

OUTCOME FOR THE CASE PATIENT

The patient required an 11-day hospitalization; her day-by-day course is described briefly below.

Day 1. Upon admission, abdominal MRI was ordered (see Figure 1) and empiric piperacillin/tazobactam IV was initiated. C-reactive protein (CRP) level and white blood cell (WBC) counts were elevated (see Table 2). Infectious disease, surgery, and urology consults were obtained.

Day 2. Fine-needle aspiration of the abscess was performed for cultures, and 40 mL of purulent fluid was drained. Piperacillin/tazobactam administration was continued, but the patient experienced ongoing fever and vomiting.

Day 3. Preliminary aspirate culture results revealed *S aureus* infection. Piperacillin/tazobactam was discontinued, and vancomycin IV was started. CRP levels and WBC counts decreased, as did fever and vomiting.

Day 4. Final aspirate culture results identified MRSA infection, sensitive to clindamycin. Vancomycin was discontinued, and clindamycin IV was started. Although the patient's condition improved somewhat, fever and vomiting persisted.

Day 5. Both CRP levels and WBC counts increased from day 3. A surgical consult was sought.

Day 6. Repeat abdominal MRI revealed a decrease in the size of the abscess (see Figure 2, page 30. CRP levels and WBC counts remained high, with persistent fever and vomiting.

Day 7. The clinical team, in consultation with the parents, determined that placement of a peripherally inserted central catheter (PICC) line for drainage of the abscess was necessary.

Day 8. A 10-French pigtail catheter was inserted into the abscess, 20 mL of purulent fluid was drained, and a PICC line was inserted. Clindamycin IV was continued and, eight hours after the catheter was placed, fever and vomiting resolved.

Day 9. Both CRP levels and WBC counts dropped by half (WBC count was normal), while 10 mL of clear fluid drained from the catheter. The patient re-

TABLE 2
Laboratory Test Results During Patient's Hospitalization

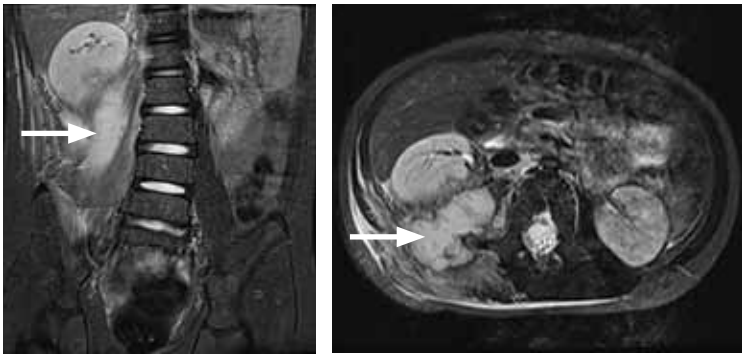
Study	Day 1	Day 3	Day 5	Day 6	Day 9	Day 10
C-reactive protein, mg/L (reference range, 0-10 mg/L)	211	146	188	149	77	35
White blood cell count, x 10^3/μL (reference range, 4-11 x 10^3/μL)	18.6	14.8	16.7	16.4	8.4	n/a

mained afebrile, without nausea or vomiting, on clindamycin IV.

Day 10. After 36 hours of clear drainage, the catheter was removed. CRP level further decreased. Clindamycin IV was discontinued, and the patient, now asymptomatic, was started on oral clindamycin.

Day 11. The patient was discharged on a regimen of oral clindamycin for six weeks, with weekly abdominal ultrasounds. She completed her entire course of antibiotics and fully recovered from the infection.

FIGURE 2



MRIs of the abdomen with contrast (left, anteroposterior view; right, transverse view), performed on hospital day 6, demonstrate improvement in the right psoas muscle (at arrows) compared to hospital admission images (see Figure 1).

CONCLUSION

Since children generally compensate well during times of increased stress on the body, it is vital that persistent FUOs continue to be evaluated until a definitive source is identified, especially in this population. Early diagnosis and treatment of psoas abscess is essential for better outcomes, since delay is associated with a greater risk for sepsis.

While the likelihood of developing psoas abscess is low, it is worth keeping the diagnosis in mind for cases of unexplained lower abdominal pain, flank pain, or hip pain when more common etiologies have been excluded. This is especially important in the setting of recent travel to a developing country due to the fact that a psoas abscess can be a complication of TB of the spine. **CR**

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