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CLINICAL VIGNETTE

Refractory Psoas Abscess due to Group A Strep

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Introduction

Iliopsoas abscess (IPA) is a rare condition defined as a collection of pus in the iliopsoas compartment. It is classified by pathogenesis into primary and secondary types. Primary IPA occurs as the result of hematogenous or lymphatic spread of a causative organism from a distant site. Secondary IPA results from direct extension of infection from adjacent structures. Patient factors that can increase the risk of developing primary IPA include diabetes mellitus, immunosuppression, HIV, intravenous drug use, and renal failure.¹

As physical exam findings can be vague and laboratory abnormalities non-specific, diagnosis requires computed tomography (CT), and treatment is dependent on obtaining culture data. The most common causative organism in primary IPA is staphylococcus, whereas secondary IPA is most commonly caused by *E coli* and polymicrobial infections.² Treatment consists of percutaneous drainage using ultrasound or CT-guidance or surgical drainage for source control, as well as initial empiric antibiotics, adjusted based on culture results. We present a rare case of psoas abscess caused by Group A streptococcus from an unclear source, complicated by osteomyelitic fracture, despite pathogen-specific antimicrobial therapy.

Case Report

A 68-year-old man with hypertension, hyperlipidemia, and poorly controlled type 2 diabetes mellitus presented to the Emergency Room with eight days of left-sided back and hip pain that radiated down his left leg. Ten days prior, the patient had tripped and fallen on his left leg while pushing a wheelbarrow, which after two days, resulted in rapidly increasing severe pain. At that time, he was evaluated as a musculoskeletal injury and discharged home with pain medication. However, the patient re-presented eight days later due to persistence in his pain despite analgesics, and with increasing difficulty ambulating.

On physical exam, the patient was holding his left thigh in a flexed position. Initial labs included a white blood cell count of 18.3 k/cumm, hemoglobin of 11.3 g/dL, platelet count of 376 K/cumm, glucose of 293 mg/dL, alkaline phosphatase of 362 U/l, AST of 55 u/l, ALT of 79 u/l. Vital signs included at temperature of 36.9 degrees Celsius, heart rate of 77 beats per minute, respiratory rate of 21 breaths per minute, and blood pressure of 108/56 mmHg. He had severe pain with passive hip flexion, extension, and internal and external rotation. A CT scan

of the abdomen and pelvis showed heterogeneous enlargement of the left psoas and left iliacus muscles with subtle peripheral enhancement and adjacent fat stranding (Figure 1). These findings were concerning for a developing left psoas abscess with extension and involvement of the left iliacus. No gastrointestinal abnormality was noted other than fatty atrophy of the pancreas.

The patient was admitted and immediately started on broad spectrum antibiotics with vancomycin, ceftriaxone, and metronidazole. Interventional Radiology (IR) was consulted and performed image-guided percutaneous drainage of the abscess. On hospital day 2, a catheter was placed for continued drainage. Cultures obtained from the abscess grew *Streptococcus pyogenes* (Group A streptococcus). Infectious Disease (ID) recommended antibiotics be narrowed to ceftriaxone based on presumed sensitivity of the causative organism. However, as there was no clear mechanism for how this infection entered the patient's psoas muscle, ID recommended future colonoscopy to rule out contiguous spread from the gastrointestinal tract.

The patient reported improvement in symptoms and was soon able to ambulate with a walker. He continued the course of intravenous ceftriaxone for one week and then transitioned to oral amoxicillin for four weeks. On hospital day 10, repeat CT scan of the abdomen and pelvis showed resolution of the abscess, and the percutaneous drain was removed. The patient was discharged with close outpatient follow-up with the ID service.

Sixteen days later, the patient re-presented to the emergency room for recurrent left hip pain. Nine days prior, he was working with physical therapy and felt a "pop" in his left hip. ED CT scan imaging of the abdomen and pelvis showed a left femoral neck fracture as well as progression of the left psoas abscess (figure 2), indicating poor source control despite the treatment course described above. The patient was readmitted and IV ceftriaxone was restarted per ID recommendations. IR was consulted again, but it was determined that the recurrent abscess was too small for percutaneous drainage. Management of the hip fracture was deferred to Orthopedics, who performed a left hip hemiarthroplasty two days later. Intraoperatively, a significant amount of purulence was expressed after passage of a sigmoid sucker into the sheath of piriformis and up into the pelvis, but the surgery was otherwise uncomplicated. ID assessed that the left femoral neck fracture was likely the result of

native expansion of the psoas abscess, causing osteomyelitis of the left hip, and recommended the patient complete a six-week course of intravenous ceftriaxone. The patient was discharged to a skilled nursing facility to complete the antibiotic course with further rehabilitation.

Discussion

Psoas abscess remains an uncommon infection that presents with nonspecific findings, and varied etiology. Most are treated successfully with source control and appropriate antimicrobials. Whereas primary psoas abscesses most commonly are due to staphylococcus, the most common sources of secondary infection include Crohn's disease followed by appendicitis, colorectal cancer or colon infection, and other intraabdominal infections, with *E coli* and polymicrobial enteric organisms most commonly isolated.² Historically, IPA was commonly attributed to spinal tuberculosis, but as overall tuberculosis rates have significantly declined in developed countries, hematogenous spread has become far more common.³

IPA often presents with fever, limp, and pain, often in the back, anterior thigh, or groin. Onset can be subacute, with nonspecific symptoms of weight loss and malaise manifesting even months before diagnosis.⁴ Infection typically extends along the psoas sheath, rarely penetrating this sheath to involve more distant structures.⁵ The abscess can occasionally amass below the inguinal ligament to cause the characteristic "psoas sign," where lower abdominal pain is elicited upon extension of the hip.⁶ Laboratory findings suggestive of psoas abscess include leukocytosis, thrombocytosis, and elevation of erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and alanine aminotransferases (ALT)^{1,6}.

This report of a psoas abscess is, notable for a highly unusual causative organism, *Streptococcus pyogenes* or group A streptococcus (GAS). In a worldwide literature review, *Staphylococcus aureus* was isolated in over 88 percent of primary cases, followed by streptococcal species (4.9%) and *E. coli* (2.8%). Cultures from secondary IPA were most often polymicrobial with enteric organisms.² While this study is dated, the findings have been supported by others, including a case series by Lopez et al that also found *S. aureus* to be the most common organism in patients with primary abscesses (42.9%) and *E. coli* the leading organism in secondary abscesses.⁷ Among the minority of streptococcal cases, pneumoniae and viridans have been documented, but reports of pyogenes are sparse. While GAS is commonly associated with skin and soft tissue infections, including necrotizing fasciitis, it rarely involves the underlying muscular structures⁸. The first reported case of a GAS primary psoas abscess was documented at a Hong Kong hospital in 2002 in a healthy immunocompetent 31-year-old female with no predisposing risk factors.⁸ Two other cases published in 2010, identified GAS psoas abscesses in a 35-year-old woman with a history of IV drug use and a 50-year-old man, both secondary to significant cutaneous lesions.⁹

Aside from the limited documentation of GAS psoas abscess, our case is unique with respect to etiology. While the patient's medical history was remarkable for poorly controlled type 2 diabetes, increasing his susceptibility to infections, he had no other typical risk factors, such as IV drug use or immunocompromising conditions. Notably, the ground-level fall that he sustained 10 days prior to hospital presentation did not produce any notable cutaneous injuries that could have served as potential portals of entry for pathogens. The Hong Kong case reported a comparable presentation of a GAS psoas abscess without a clear inciting event. However, this report noted acute pharyngitis one week preceding the onset of the patient's symptoms, which could be considered the entry point for invasive GAS.⁸ Although the reason for our patient's development of psoas abscess is, unclear, his mechanical fall remains the most likely causative event. Another case by Moriarty and Baker reports a 19-year-old male college athlete with no past medical history who developed a psoas abscess four days following a twisting injury of his anterior hip.¹⁰ This abscess was thought to have stemmed from a traumatic hematoma or strain of the psoas muscle, which may have been the case for our patient as well.

Finally, our case presents an interesting sequela of a psoas abscess: osteomyelitis of the hip resulting in a femoral neck fracture. Rarely does the bacterial infection of the psoas compartment spread to adjacent bony structures to cause osteomyelitis after adequate drainage and antibiotic therapy. This complication underscores the utility of close monitoring for recurrent symptoms even after completion of appropriate treatment.

Overall, while the exact incidence of psoas abscesses is not known, diagnosed cases are estimated to be increasing in recent years due to improved CT imaging.⁸ Thus, this condition should consistently be included in differential diagnoses for hip, low back, thigh, and groin pain, especially for patients with risk factors and pain refractory to conservative analgesics. Unfortunately, signs and symptoms for psoas abscess are nonspecific, yet a thorough musculoskeletal exam of the hip joint is an appropriate starting point. When suspicion for psoas abscess is present, CT scan offers prompt diagnosis, with drainage and antibiotics remaining mainstays of treatment.

Figures



Figure 1: Transverse view of CT abdomen displaying iliopsoas abscess from initial presentation.

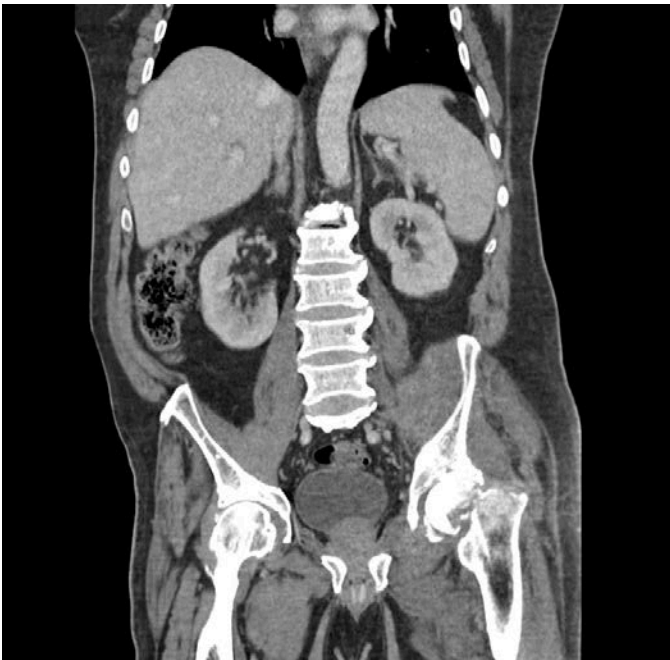


Figure 2: Coronal view of CT abdomen on re-presentation, revealing recurrence of abscess as well as left femoral hip fracture.

REFERENCES

1. **Mallick IH, Thoufeeq MH, Rajendran TP.** Iliopsoas abscesses. *Postgrad Med J.* 2004 Aug;80(946):459-62. doi: 10.1136/pgmj.2003.017665. PMID: 15299155; PMCID: PMC1743075.
2. **Ricci MA, Rose FB, Meyer KK.** Pyogenic psoas abscess: worldwide variations in etiology. *World J Surg.* 1986 Oct;10(5):834-43. doi: 10.1007/BF01655254. PMID: 3776220.
3. **van den Berge M, de Marie S, Kuipers T, Jansz AR, Bravenboer B.** Psoas abscess: report of a series and review

of the literature. *Neth J Med.* 2005 Nov;63(10):413-6. PMID: 16301764.

4. **Shields D, Robinson P, Crowley TP.** Iliopsoas abscess-- a review and update on the literature. *Int J Surg.* 2012;10(9):466-9. doi: 10.1016/j.ijsu.2012.08.016. Epub 2012 Sep 5. PMID: 22960467.
5. **Zhou Z, Song Y, Cai Q, Zeng J.** Primary psoas abscess extending to thigh adductors: case report. *BMC Musculoskelet Disord.* 2010 Aug 6;11:176. doi: 10.1186/1471-2474-11-176. PMID: 20691069; PMCID: PMC2921354.
6. **Li Y, Funakoshi H, Shiga T, Fujitani S.** Iliopsoas abscess. *Cleve Clin J Med.* 2017 Nov;84(11):833-834. doi: 10.3949/ccjm.84a.17002. PMID: 29173253.
7. **López VN, Ramos JM, Meseguer V, Pérez Arellano JL, Serrano R, Ordóñez MAG, Peralta G, Boix V, Pardo J, Conde A, Salgado F, Gutiérrez F; GTI-SEMI Group.** Microbiology and outcome of iliopsoas abscess in 124 patients. *Medicine (Baltimore).* 2009 Mar;88(2):120-130. doi: 10.1097/MD.0b013e31819d2748. PMID: 19282703.
8. **Lau SK, Woo PC, Yim TC, To AP, Yuen KY.** Molecular characterization of a strain of group a streptococcus isolated from a patient with a psoas abscess. *J Clin Microbiol.* 2003 Oct;41(10):4888-91. doi: 10.1128/JCM.41.10.4888-4891.2003. PMID: 14532252; PMCID: PMC254351.
9. **Routier E, Bularca S, Sbidian E, Roujeau JC, Bagot M.** Abscès du psoas à streptocoques bêta-hémolytique du groupe A à point de départ cutané : deux cas [Two cases of psoas abscesses caused by group A beta-haemolytic streptococcal infection with a cutaneous portal of entry]. *Ann Dermatol Venereol.* 2010 May;137(5):369-72. French. doi: 10.1016/j.annder.2010.02.020. Epub 2010 Apr 2. PMID: 20470918.
10. **Moriarty CM, Baker RJ.** A Pain in the Psoas. *Sports Health.* 2016 Nov/Dec;8(6):568-572. doi: 10.1177/1941738116665112. Epub 2016 Aug 20. PMID: 27542388; PMCID: PMC5089355.