

Septic Arthritis of the Hip Following Group B Streptococcal Psoas Abscess in a Postpartum Patient Resulting in Total Hip Arthroplasty

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Citation

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Abstract

We report the rare presentation of a septic hip in a postpartum female stemming from a Group B streptococcus (GBS) infection arising from a postpartum psoas abscess. This patient had known GBS peripartum colonization, and eventually had to undergo a total hip arthroplasty for cartilage damage caused by the GBS infection.

INTRODUCTION

The acute onset of non-traumatic hip pain in young adults is uncommon [1]. A septic joint, although rare in young adults without predisposing factors [1,2,3], must be assumed until proven otherwise due to possible cartilage damage [4]. Although *Staphylococcus aureus* and *Neisseria gonorrhoeae* remain the most frequent bacterial causes of septic arthritis of the hip, a diagnostic consideration in the female young adult should also include Group B streptococcus (GBS). GBS is a common commensal organism of the female reproductive system. GBS has been recognized as a significant cause of perinatal morbidity and mortality for at least two decades, with complications of GBS infection including urinary tract infection, amnionitis, endometritis, wound infection following delivery, and psoas abscess [5].

We present what we believe to be the first case reported in the literature of a septic hip in a postpartum female stemming from a GBS infection arising from a postpartum psoas abscess. This patient had known GBS peripartum colonization, and eventually had to undergo a total hip arthroplasty for cartilage damage caused by the GBS infection.

The patient was informed that data concerning this case would be submitted for publication.

CASE REPORT

A twenty-nine year old primigravida woman with a known peripartum history of GBS vaginal colonization and

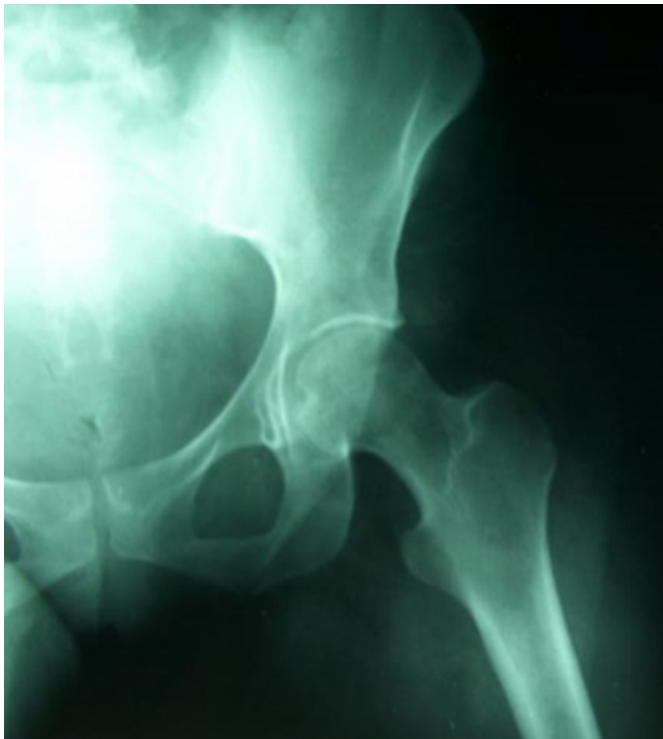
preeclampsia presented to our institution with complaints of fever, left groin, posterior thigh, and low back pain eight weeks following a spontaneous vaginal delivery at an outside institution.

The patient had received intra-partum antibiotic prophylaxis for GBS with penicillin, did not have any vaginal or cervical lacerations during delivery, and had an unremarkable past medical and surgical history (no steroid use, autoimmune disease, diabetes, chemotherapy, HIV, and/or prior pelvic procedures). Yet, on postpartum day one the patient had developed a fever and rigors which were empirically treated with intravenous vancomycin for two days prior to discharge. Two days following discharge, the patient returned to the emergency department with complaints of continued fever as well as the onset of severe left posterior thigh and groin pain. Radiographs of the hip were normal at this time (Fig. 1).

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Figure 1

Figure 1: Anteroposterior radiograph of the patient's left hip showing no abnormalities five days after delivery.



Due to the concern for infection the patient was subsequently re-admitted, and blood cultures drawn at the time of admission grew GBS sensitive to cefazolin. The patient received six days of intravenous cefazolin while in the hospital without improvement in the left posterior thigh and groin pain. As a result, a CT scan was performed which revealed a left psoas abscess which was subsequently percutaneously drained by interventional radiology (Fig. 2). Cultures of the abscess grew GBS sensitive to cefazolin, which the patient continued. Placement of a peripherally inserted central catheter (PICC) line occurred on hospital day thirteen. Discharge to home occurred on hospital day seventeen after improvement in her symptoms. A total of three weeks of cefazolin were administered to the patient.

Figure 2

Figure 2: CT scan image in the axial plane of the abdomen/pelvis demonstrating a left psoas abscess (white arrow)



Three weeks after the completion of antibiotic therapy, the patient returned to the emergency department with complaints of continued fever, left groin, posterior thigh, and low back pain. An MRI obtained upon this presentation demonstrated a left hip effusion, extensive soft tissue edema, a flattened femoral articular surface with signal changes and edematous bone, loss of overlying articular cartilage, and a fluid collection within the iliopsoas consistent with an abscess (Fig. 3 and 4). Intravenous vancomycin was started and the patient was transferred to our institution for further management of a septic hip. An aspiration of the hip joint prior to the initiation of antibiotics was not performed at the outside institution.

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Figure 3

Figure 3: T1 weighted MRI image of the pelvis in the axial plane demonstrating increased signal in the region of the left iliopsoas and femur, consistent with extensive inflammatory edema, iliopsoas abscess, and infection.

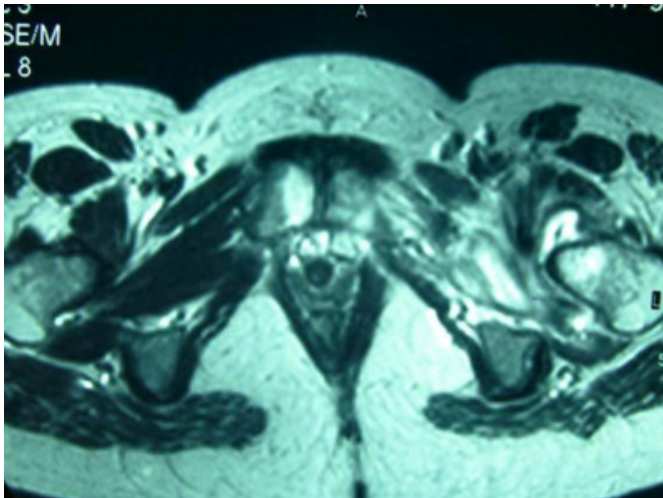
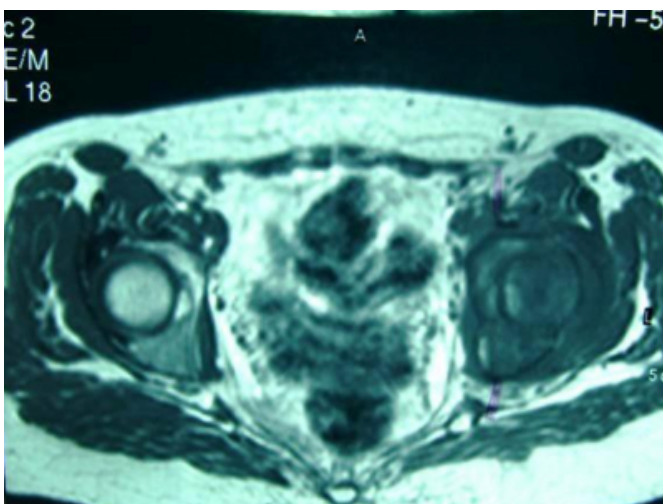


Figure 4

Figure 4: T1 weighted MRI image of the pelvis in the axial plane demonstrating a distended hip joint, edematous femur and acetabulum, a flattened femoral articular surface, and loss of hyaline cartilage over the articular surface of the femur and acetabulum; consistent with an infectious process destroying the hip joint.



Upon presentation to our institution, the patient was complaining of tenderness along the left posterior thigh and buttock. The patient denied any trauma. On physical exam, the patient was afebrile, and very tender to palpation along the left posterior thigh and gluteal region. Passive range of motion was limited secondary to pain. Active hip extension and abduction were limited, however, the patient was able to flex the left hip to 90 degrees with minimal discomfort. The

patient had no neuromuscular or vascular deficits.

Laboratory values obtained at the time of admission included a white blood cell count of 9,000/mm³ and an erythrocyte sedimentation rate of 55 mm/hour.

Intravenous vancomycin was discontinued on hospital day two, and three days later interventional radiology aspirated the left hip effusion. Aspirate and blood cultures produced no organisms. Due to the sterile aspiration and the patient's improvement, the decision was made to not proceed with surgical drainage. A PICC line was placed, and the patient was discharged home for a six week course of intravenous ceftriaxone (per the infectious disease service). This was done with the hope of placing a hip arthroplasty in the future as treatment for the patient's ambulatory dysfunction as a result of extensive joint degeneration from the infection.

The patient returned to clinic after her six week antibiotic course, and was doing well. Plain films demonstrated extensive damage to the left hip joint (Fig. 5), and the patient subsequently underwent a left hip total arthroplasty (Fig. 6) one month later without complications. The patient is currently six years out from her joint replacement and is doing well.

Figure 5

Figure 5: Anteroposterior radiograph of the patient's pelvis six weeks after completion of antibiotic therapy demonstrating extensive damage to the articular surface of the left hip joint.

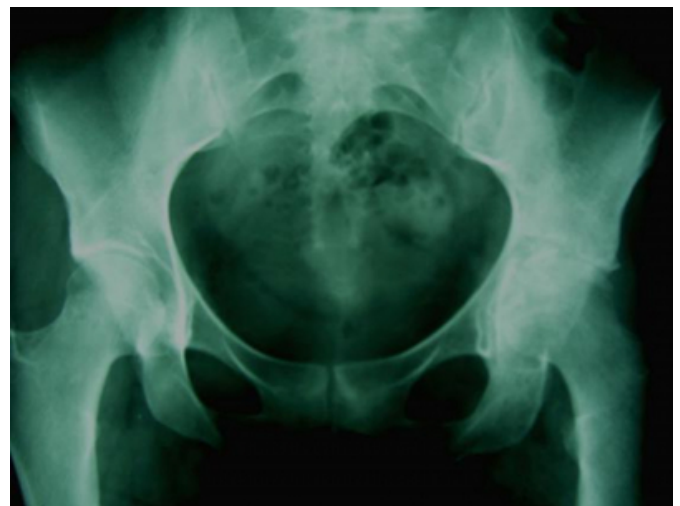


Figure 6

Figure 6: Anteroposterior radiograph of the patient's left hip after total hip arthroplasty for extensive degenerative disease of the left hip as a result of septic arthritis.



DISCUSSION

Psoas abscesses are a rare entity, and have been described in a limited fashion within the literature [6,7]. They are typically classified as primary or secondary, with primary abscesses lacking a definitive etiology; perhaps resulting from hematogenous spread [8]. Secondary psoas abscesses generally form from infections in adjacent organs/tissue and can stem from a variety of conditions including Crohn's disease, appendicitis, colonic inflammation or neoplasm, disc infections, as well as a variety of intra-abdominal and retroperitoneal infections which are in close proximity to the psoas musculature [8]. Given the known vaginal colonization in our patient, our psoas abscess can be classified as secondary.

Initial presentation of a patient with a psoas abscess includes back, hip, or flank pain, fever, leukocytosis (although not always present), elevated erythrocyte sedimentation rate, elevated C-reactive protein, and positive blood cultures [9]. Most signs and symptoms of the disease are non-specific, hindering prompt diagnosis [10]. Therefore, these findings should lead to further investigation with imaging studies, including CT scan and MRI. CT scan has a reported specificity of 77% and a sensitivity of 100% [6].

The patient described in our paper exhibited a delay between the initial presentation and treatment, and then a repeat flare 3 weeks after the completion of the first round of antibiotic therapy. This latency period in abscesses formed by GBS infection has also been described by Brandenberger and Hauser et al [11]. They describe a patient with a postpartum periarticular hip abscess caused by GBS [11]. After initial antibiotic treatment, the patient's symptoms seemingly resolved then reappeared again four months after delivery, this time causing a septic hip joint.

Antibiotics as well as adequate drainage via a percutaneous approach or an open or arthroscopic procedure are essential in the management of psoas abscesses [12]. In fact, aggressive incision, debridement, and drainage is recommended by many authors [9,13,14,15,16] in lieu of a percutaneous approach. In our patient, the lack of an aggressive approach to the abscess (i.e. lack of surgical intervention) may have been the reason the psoas abscess was able to persist and provide a nidus for infection into the hip. This led to a septic joint and extensive cartilage damage in a young patient which eventually resulted in total joint arthroplasty.

Yet, septic arthritis of the hip following a psoas abscess is a rare entity, with only a few cases described in the literature [13,14,17]. In fact, septic arthritis of the hip in general after pregnancy is extremely uncommon [18]. Furthermore, we are not aware of any cases in the literature which specifically describe the formation of a septic hip from a postpartum GBS psoas abscess stemming from a woman with known GBS vaginal colonization. Not only is intrapartum hematogenous spread of GBS from vaginal colonization rare [11], but also subsequent development of postpartum psoas abscesses from an intrapartum seeding even more unlikely with only a few cases described in the literature [7,19,20].

If a psoas abscess does develop, a pathway exists for direct extension into the hip joint via the iliopsoas bursa; which is postulated to connect with the hip joint in approximately 15% of patients [15,21,22]. As a result, aggressive treatment of psoas abscess is warranted via incision, debridement, and drainage as described above. Some surgeons have even recommended prophylactic hip arthrotomy during the drainage of a psoas abscess to ensure that the hip is debrided [15].

If a septic hip does develop from a GBS psoas abscess, we believe the treatment algorithm should remain the same as

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any other septic joint: diagnosis via aspiration (cell count, gram stain, and cultures), appropriate antibiotics, and performing incision and drainage when the patient's clinical picture is worsening or if fluid from repeated aspiration show a persistent infection. In our patient, the aspiration performed at our institution (after antibiotic therapy was initiated at the outside hospital) was sterile, and the patient was clinically improving. As a result, surgical intervention was not performed. If extensive cartilage damage is the sequela of a hip infected via an adjacent psoas abscess (as in our patient), total hip arthroplasty can be performed in patients after the infection has cleared via appropriate medical and/or surgical treatment [13,14].

This case highlights the importance of recognizing that females with GBS vaginal colonization are at risk for the development of GBS psoas abscesses after delivery. Due to the fact that the psoas abscess can provide a nidus for the development of a septic hip, these abscesses need to be aggressively treated early on; particularly with aggressive surgical incision and drainage. Delay in treatment can lead to an infected native joint, with a high risk for extensive cartilage damage at a young age, and subsequent total joint arthroplasty.

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