



## Case report

# A Case of Iliopsoas Bursitis With Compressive Femoral Nerve Palsy Treated With Iliopsoas Tendon Release

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## ABSTRACT

Iliopsoas bursitis is a well-established cause of groin pain after total hip arthroplasty (THA), and it can become dramatically more complex when associated with neurovascular compression. Iliopsoas bursitis may be caused by a variety of pathologies in the setting of a THA but most frequently due to a prominent acetabular component or implant wear. Here we report a rare case of a female patient presenting with iliopsoas tendonitis, an accompanying femoral nerve palsy, and debilitating pain beginning 12 years after a previously successful primary THA without apparent implant wear. Ultimately, our patient was treated successfully with iliopsoas tendon release for anterior prominence of the acetabular component.

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## Introduction

Iliopsoas tendonitis is a recognized cause of groin pain after total hip arthroplasty (THA) with a reported incidence after primary THA of up to 4.3% [1]. The iliopsoas tendon is an extra-articular structure located just anterior to the hip capsule, where it is vulnerable to irritation following THA. Established causes of THA-related iliopsoas tendonitis include acetabular cup prominence, anterior osteophyte impingement, misplaced screws, cement extravasation, and excessive change in leg length or offset [2].

Although iliopsoas pathology is a well-established cause of groin pain after THA, it is much less commonly associated with a mass, femoral nerve palsy, or vascular compression [3–6]. Cases of iliopsoas bursitis with mass effect on neurovascular structures following THA can be complex to diagnose and effectively treat. In light of this, we present an unusual case of iliopsoas tendonitis causing femoral nerve compression and debilitating pain 12 years after a previously well-functioning metal on highly cross-linked polyethylene THA.

## Case history

This case report was prepared with the full consent of the patient described herein.

A 73-year-old female presented to the emergency department (ED) with new right hip pain and weakness in 2019 following a previously successful right THA performed in 2007 and a left THA performed in 2012. Beginning in September 2019, she experienced several occasions of right-sided groin pain and paresthesias radiating distally along the anterolateral thigh and extending to the knee. She also had increasing difficulty ambulating.

This otherwise healthy patient had the right THA performed via a direct lateral approach at our institution in 2007 with a Reflection 52-mm three-hole porous acetabular shell (Smith and Nephew, Memphis, TN), one dome screw, a size E Reflection XLP liner, Synergy size-12 standard offset stem (Smith and Nephew, Memphis, TN), and a 32-mm -3 cobalt chromium femoral head. At a 2015 arthroplasty clinic follow-up, she reported no pain or problems with either THA. She ambulated with a smooth steady gait without the need for any gait aid. Both hip incisions were well healed without any tenderness or swelling. Hip range of motion active and passive was completely pain-free and symmetric with bilateral hip flexion of 120 degrees, external rotation of 45 degrees, and internal rotation of 30 degrees. Although she was not seen at our institution in any capacity between 2015 and 2019, she recalls an episode of similar right groin pain of less intensity with difficulty lifting her leg around 2017. These symptoms reportedly

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resolved with physical therapy (PT) and nonsteroidal anti-inflammatory drugs (NSAIDs).

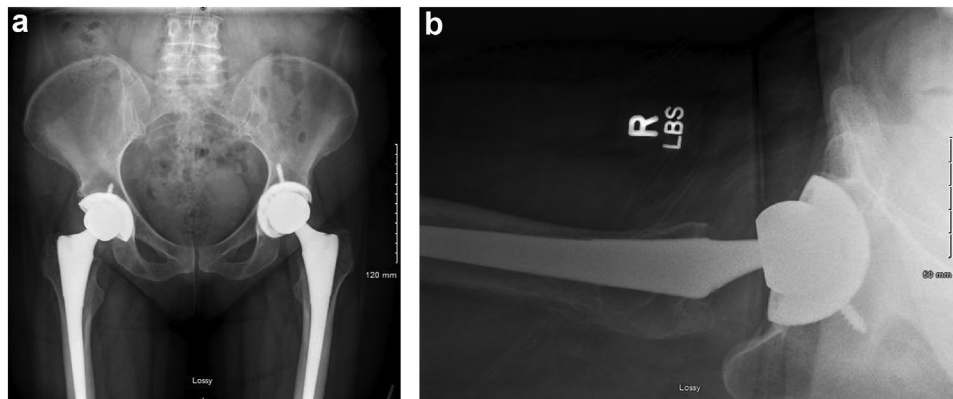
Her first visit to the ED in September 2019 was after a ground-level fall earlier that day due to weakness. She had been experiencing right-sided groin pain for several days prior, but since falling, she had new right thigh numbness. Workup at this initial presentation revealed discomfort with internal and external rotation of the right hip and diminished sensation over the anterolateral thigh with a normal distal neurovascular exam. She was unable to perform a right straight leg raise due to groin pain but could actively flex the right hip and knee. Patella reflexes were 0/4 on the right and 3+/4 on the left. Anterior-posterior (AP) and lateral radiographs of the right hip showed no evidence of dislocation, fracture, or loosening of hardware (Fig. 1a and b), and she was sent home with instructions to use NSAIDs, PT, and primary care provider follow-up. Three days later, the patient returned to the ED with refractory right-sided pain despite recent treatment with NSAIDs, tizanidine, and a single dose of prednisone prescribed at an urgent care. Diagnostic testing was notable for normal C-reactive protein of <7 mg/L (normal <10 mg/L), complete blood count, and basic metabolic panel. Coagulation labs were not obtained at this time, but she had not been taking any anticoagulants and had a prior international normalized ratio of 1.0 (normal 0.9–1.1). Lumbar magnetic resonance imaging (MRI) demonstrated degenerative disc disease and facet arthropathy, mild spinal canal stenosis at L4–L5 with effacement of the subarticular zones, and possible impingement of the L5 nerve roots. She was discharged home with a small quantity of opioid medication for pain control and instructed to follow up with the primary care provider. She again returned to the ED the following day with persistent right-groin pain, and this time, the orthopedic service was first consulted.

Initial orthopedic evaluation in the ED was notable for minimal tenderness in the right anterior groin without erythema or fluctuance, decreased sensation of the anteromedial distal thigh, and 1/5 strength in knee extension and 2/5 hip flexion. MRI of the right hip demonstrated a multilobulated fluid collection with well-circumscribed borders immediately anterior to the right prosthetic femoral head measuring  $3.1 \times 3.3 \times 7.8$  cm with mass effect on the iliopsoas muscle (Fig. 2a and b). Clinically, it was decided that her symptoms were most consistent with femoral neuropraxia secondary to traumatic hematoma compressing the femoral neurovascular bundle based on the radiographic density and the trauma from falling. An occult fracture, trunnionosis-related pseudotumor, and iliopsoas bursitis from local tendon irritation were also considered. She was then referred for outpatient evaluation in our arthroplasty clinic expecting that her symptoms might improve as the hematoma resorbed.

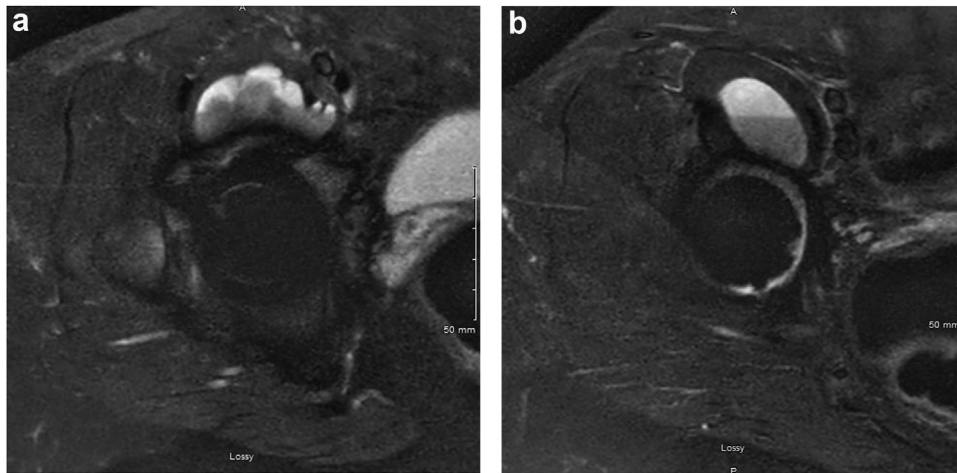
The following day, she presented to an outside hospital with uncontrollable pain at which point she was given fentanyl and gabapentin with some relief, but ultimately, she was transferred and admitted to our medicine service for pain control. On repeat evaluation by our service, there was no change in her sensory and strength exam, the right patellar deep tendon reflex remained absent, C-reactive protein remained normal at <7 mg/L, and sedimentation rate was normal at 26 mm/h (normal 0–30 mm/h). Interventional radiology aspirated the right iliopsoas fluid collection and obtained 3cc of dark bloody fluid, which was sent for fluid analysis. A requested synovial fluid cell count was not performed. Aspirate culture had no growth, and cobalt and chromium ion levels were undetectable in the synovial fluid. Serum cobalt was <0.2 ng/mL (normal 0–0.9 ng/mL), and serum chromium was 0.1 ng/mL (normal <0.3 ng/mL), indicating trunnionosis was unlikely. While admitted, the patient became dependent on a walker due to weakness and subjective instability of the right hip without any other change to her clinical examination. Adequate pain control was achieved, and it was determined that she was safe to be discharged home without the need for further immediate intervention. Initially she did well at home but then returned to the ED again with severe pain, numbness, and muscle spasms days later.

The patient was readmitted and re-evaluated by our service for persistent pain and numbness. In the context of suspected traumatic hematoma and worsening symptoms, we ordered a computed tomography (CT) of the hip to evaluate for an expanding hematoma causing further neurovascular compromise. CT of the hip showed a largely unchanged heterogenous collection with maximal transaxial measurement of  $4.0 \times 2.5$  cm tracking along the anterior aspect of the right hip with extension proximally into the iliopsoas bursal space (Fig. 3). Given the extent of her symptoms, diagnostic difficulty and nonspecific nature of our imaging studies, several additional diagnostic avenues were discussed with the patient including electromyograph, repeat interventional radiology aspiration, and surgical intervention. The patient decided to go home but then soon returned to the ED. In the ED, she had persistent pain with ultrasonography of the right hip demonstrating a  $5.6 \times 3.2 \times 3.4$ -cm heterogenous, avascular collection most consistent with a hematoma. She was discharged home with a plan to follow up in the outpatient arthroplasty clinic for definitive management of her symptoms.

All the previously described admissions and ED visits occurred over a short period such that she was seen in our outpatient arthroplasty clinic 2 weeks after the initial ED consultation by orthopedics. At that visit, she had persistent decreased sensation of the right medial thigh, 0/5 strength of the quadriceps, and was unable to actively flex her right hip; yet she had only mild pain with



**Figure 1.** September 2019 preoperative (a) anterior-posterior (AP) and (b) cross-table lateral radiographs showing no evidence of wear or significant component loosening of the right total hip with minimal anterior prominence of the right acetabular component.

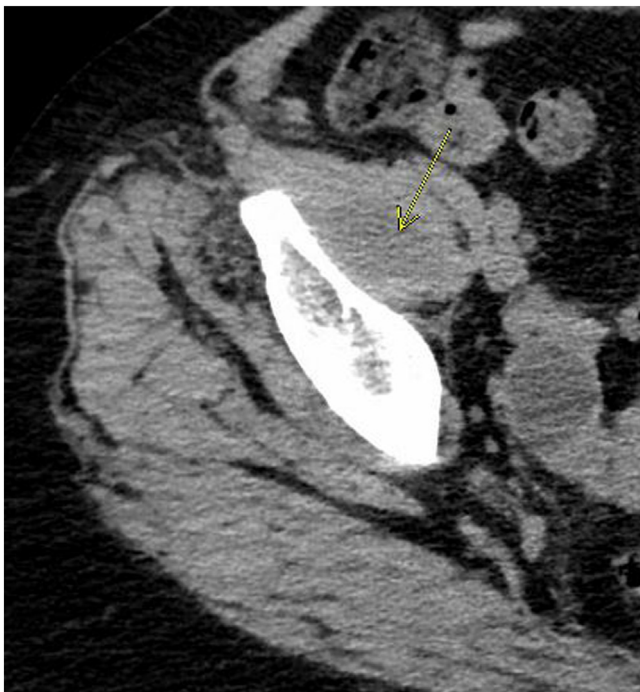


**Figure 2.** (a and b) Two separate images of an axial STIR metal suppression MRI demonstrating an iliopsoas-associated fluid collection anterior to the right prosthetic femoral head.

passive hip flexion. The acetabular component was deemed to have relatively little anteversion (10 degrees on the axial CT) with an exposed anterior surface thought to be a possible source of iliopsoas irritation (Fig. 4). The maximum anterior prominence of the acetabular component was measured to be 2 mm on the cross-table lateral radiograph and 6 mm on the axial CT images. Surgical options were discussed in detail with the patient including debridement, iliopsoas tendon release (open or arthroscopically), and acetabular component revision to prevent ongoing iliopsoas irritation by increasing the cup anteversion through a variety of possible surgical approaches. She was also counseled that because the anterior cup prominence was mild, reorienting the cup may not be required for symptom relief. She decided to proceed with an anterior-approach modular component exchange with possible iliopsoas release as the primary plan. An anterior approach was

selected due to a combination of surgeon preference and concern for posterior dislocation if the cup position was maintained with a posterior approach. Appropriate consent was obtained for urgent surgery in a week.

In October 2019, she underwent a right THA femoral head and polyethylene liner exchange via a direct anterior approach through a new incision. Within the hip joint, there was some normal-appearing synovial fluid, but no significant fluid collection or mass was encountered on exploration. The iliopsoas recess was inspected, and suction was performed along the iliopsoas without any discernible hematoma. The head and liner were removed. No wear was evident on the acetabular liner which was intact but discolored (Fig. 5). The head and trunnion had no corrosion. No periprosthetic osteolysis was observed. A new liner, which was identical to the initial one (Smith and Nephew Reflection size E XLP 0 degree) was inserted, and a new 32-mm -3 Oxinium (Smith and Nephew, Memphis, TN) head was impacted. The iliopsoas tendon

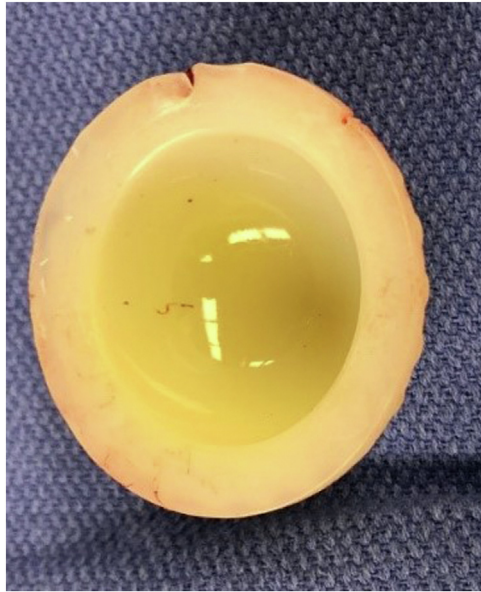


**Figure 3.** Computed tomography (CT) of right hip demonstrating proximal extension of iliopsoas collection highlighted with an arrow.



**Figure 4.** Computed tomography (CT) of the right hip demonstrating minimally exposed anterior edge of the acetabular cup.





**Figure 5.** Removed polyethylene Smith and Nephew Reflection size E XLP 0-degree liner demonstrating no evidence of significant wear and slight yellow discoloration.

was isolated and transected. Five tissue samples from the joint had no growth on culture. Pathology of the hip synovium demonstrated granulation tissue, fibrosis, and hemosiderin deposition that could be consistent with a prior hematoma and scattered foci of lymphocytic perivascular inflammation.

Over the first year following the surgery, she had significant pain relief and progressive functional improvement. On the first postoperative day, she continued to have 0/5 right quadriceps strength but improved overall pain. On postoperative day 2, she demonstrated a slight improvement to 1/5 right quadriceps strength. At the 1-month follow-up, the patient reported complete resolution of numbness and some improvement in right leg strength. At 4 months following revision, she had no pain but had continued hip flexor weakness causing her to occasionally fall. One-year after surgery, she had no numbness and full strength (5/5 hip flexion and knee extension). She felt that the right hip was similar to the left and completely pain-free allowing her to again garden and mow her lawn. Patient-reported outcome scores were a hip disability and

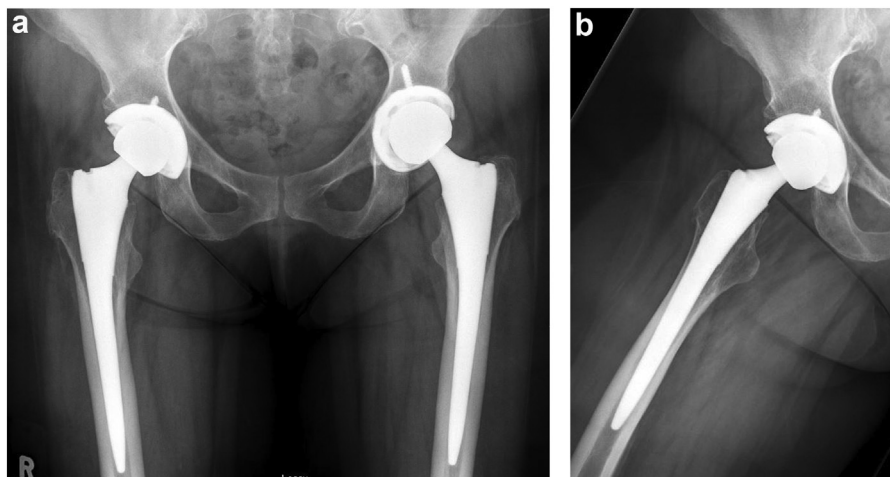
osteoarthritis outcome score, joint replacement of 50 prior to surgery, which improved to 85 at 4 months and then to 100 at 1 year after the revision [7]. The 1-year postoperative radiographs demonstrate an intact revised right THA (Fig. 6a and b).

## Discussion

This case illustrates the diagnostic complexity of iliopsoas pathology in the setting of a THA. Initially, it is essential to eliminate multiple other potential causes of hip pain such as acute dislocation, fracture, infection, adverse tissue reaction to metal, and implant loosening [2,8]. Tendinopathies including iliopsoas tendonitis or bursitis, greater trochanteric pain syndrome, and snapping hip syndrome are becoming increasingly recognized as common causes of persistent groin pain after THA [2]. It is important to understand the diagnosis and treatment of iliopsoas tendonitis along with the rare but serious potential for neurovascular compression.

Most often, iliopsoas tendonitis after THA is related to direct irritation of the tendon by the anterior edge of the acetabular cup, retained cement, penetration of long screws, or a prominent femoral collar [8,9]. Less common causes of iliopsoas tendonitis include trunnionosis in modular prostheses or bearing surface wear [10]. Rarely as in our case, iliopsoas tendonitis with accompanying bursal distention causes neurovascular compromise. The iliopsoas bursa is situated anterior to the hip joint capsule, at the thinnest aspect of the joint and extending proximally between the iliacus muscle and iliopsoas tendon [6,11,12]. Bursal distention has also been recognized in the context of hip joint pathology including degenerative arthropathy, inflammatory arthropathy, snapping hip syndrome, synovial chondromatosis, arthroplasty-related particle disease, iliacus muscle hematoma, lipohemarthrosis, and repetitive trauma [6,11].

The iliopsoas bursa is present bilaterally in roughly 98% of adults [6]. It varies in size and protects the iliopsoas tendon from abrasion. The bursa has been described to directly communicate with the synovial membrane of the hip joint through an opening of the capsular ligament [12]. In 2002, Wunderbaldinger et al. reported that upon investigating 18 patients with iliopsoas bursitis, they found direct communication of the bursa with the hip joint in 56% of cases by ultrasound, 60% of cases by CT, and 100% of cases by MRI [13]. The frequency of direct bursal communication with the hip joint varies with 15% of adults having anatomical communication, but direct bursal communication with the joint is overwhelmingly



**Figure 6.** (a) Low AP pelvis view and (b) frog lateral view of the right total hip 1 year after revision.

present among patients with iliopsoas bursitis [6,13]. Furthermore, it has been suggested that development of bursal communication may be attributed to disruption of the anterior hip joint capsule by the overlying tendon, either via capsular attrition or direct injury to the joint [4,13,14]. Provided a direct communication between the bursa and hip joint is present in the majority of patients with symptomatic iliopsoas bursitis, it has been hypothesized this communication plays a major role in the pathophysiology of the condition [13].

Those presenting with iliopsoas tendonitis often present with unremitting groin pain with or without palpable mass worsened by daily activities, specifically by getting in and out of cars or climbing stairs. Pain often follows a period of pain-free mobility, most commonly presenting 2–96 months after THA [2]. It is often difficult or impossible for the patient with iliopsoas irritation to lift the symptomatic leg in a seated position, especially against resistance, while passive hip flexion may be minimally painful. Iliopsoas bursal distention may also be palpable as an inguinal or lower abdominal mass [6]. The anatomical location and proximity of the iliopsoas bursa to the femoral neurovascular bundle (femoral nerve, femoral artery, and femoral vein) is well documented in the literature [11,15–17]. It is because of this anatomic proximity that neurovascular compromise can occur by mass effect with bursal distention [3].

Workup often includes radiographs of the hip to exclude more common causes of pain, followed by MRI and local ultrasound, although both are minimally useful in the clinical assessment of iliopsoas tendon pathology [2]. Most useful for clinical assessment is combined diagnostic and therapeutic intervention with image-guided injection of anesthetics and corticosteroids into the iliopsoas tendon [2,9]. Iliopsoas tendonitis is initially managed non-operatively with PT, NSAIDs, or local injections although it may require operative management with tendon debridement, tenotomy, or full acetabular component revision [2,9]. Notably, management is dependent on the severity of the patient's symptoms and acetabular component position.

Our patient represents a rare and diagnostically interesting case of symptomatic iliopsoas bursitis complicated by compression femoral nerve palsy 12 years after primary THA. We believe that the most likely underlying pathology in this case was too little anteversion of the original acetabular component, which caused the anterior edge of the cup to be prominent. The uncovered anterior acetabular component was likely a significant factor in the development of iliopsoas tendonitis/bursal distention and local bleeding as the tendon glided over the rough edge of the cup. It is curious, however, that her hip was asymptomatic for so many years after THA. Perhaps the acute trauma from the initial fall contributed to acute tendonitis and local bleeding in the setting of mostly latent subclinical iliopsoas tendonitis. This theory is supported by the onset of a brief episode of tendonitis that occurred 2 years prior to the dramatic presentation and by the increasingly severe symptoms of iliopsoas tendonitis this patient described in the days leading up to the fall followed by progressive femoral neuropathy. We hypothesize that an additional factor in this case is that the primary THA was performed via a direct lateral approach with anterior capsulectomy. The anterior hip capsule in this case had been excised with the original THA and replaced with scar tissue, which certainly altered the local anatomy in the vicinity of the iliopsoas bursa. However, it is difficult to attribute this as a primary causative factor in this case because surgical approach is not independently recognized as a risk factor for iliopsoas tendonitis. Both anterior- and posterior-approach THAs may be associated with iliopsoas tendonitis especially when the anterior edge of the acetabular component is prominent. This case is particularly interesting because of the delayed onset of dramatic symptoms in the absence of adverse local tissue reaction to metal debris or

polyethylene wear. Our decision to perform iliopsoas tendon release is consistent with the treatment recommendations of Chalmers et al. to perform isolated iliopsoas release if there is less than 8 mm of anterior acetabular prominence [9]. This treatment was successful in resolving her iliopsoas tendonitis and functionally limiting femoral nerve compression. An alternative treatment option for symptomatic iliopsoas tendonitis after THA is arthroscopic iliopsoas tendon release, which is documented in multiple series to successfully relieve symptoms [18–21]. We are not aware of arthroscopic treatment for iliopsoas-related femoral neuropathy and know of only one published case of iliopsoas bursitis mass effect causing femoral nerve palsy [14]. In this case, the authors reported nerve compression at 10 years following a ceramic-on-metal THA that was treated with iliopsoas bursectomy via an invasive modified iliofemoral approach.

## Summary

Our patient's experience with femoral nerve compressive iliopsoas bursitis years after a seemingly successful THA is rare and dramatic. While iliopsoas tendonitis is a well-established phenomenon, it can produce serious neurovascular compromise as demonstrated in this case. Surprisingly, anterior prominence of the acetabular component in isolation can be associated with delayed onset of femoral nerve palsy many years later. Fortunately, even with associated compressive femoral neuropathy, standard treatment of iliopsoas tendonitis such as iliopsoas tendon release can provide complete symptom resolution.

## Conflicts of interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: N. J. Nelms is in the editorial board of *Arthroplasty Today* and in the AAOS Knee Program Evaluation Committee.

For full disclosure statements refer to <https://doi.org/10.1016/j.artd.2022.02.004>.

## Informed patient consent

The authors confirm that written informed consent has been obtained from the involved patient and that she has given approval for this information to be published in this case report.

Please refer to Elsevier's policy regarding written patient consent requirements.

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