Acute Severe Hip Pain Associated With Labral Calcific Deposition Disease

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abstract

Calcific tendinitis is a term used to describe radiographic evidence of calcific deposition within a tendon. This condition, also known as calcium deposition disease, has been described in the gluteus maximus, the peroneus longus tendon, the popliteus tendon, the longus colli muscle in the neck, and the tendon of the rectus femoris. However, most of the literature on calcific tendinitis relates to crystal deposition within the rotator cuff of the shoulder. The peri-articular pain related to calcium deposition may be indolent and chronic, and patients can have varying degrees of functional deficit. Patients also may present with an acute inflammatory event, with severe incapacitation and restricted passive range of motion and a clinical picture that is concerning for septic arthritis. Severe pain associated with calcific tendonitis usually occurs during the resorptive phase, where there is vascular infiltration of the calcium deposits and histologic evidence of phagocytosis. The authors report a case of calcium deposition disease found within the hip labrum with a clinical presentation of acute, atraumatic, debilitating pain in a patient with underlying femoroacetabular impingement. This clinical picture is similar to that described during the resorptive phase seen in calcific tendonitis of the shoulder. The authors attribute this presentation to acute rupture of the calcium deposit into the intra-articular joint space of the hip. To the authors’ knowledge, there are no other reports of this clinical presentation in the literature. [Orthopedics. 2014;37(12):e1137-e1140.]

Figure: Preoperative coronal magnetic resonance image of the pelvis showing an acute effusion of the left hip.
Calcific tendinitis, also known as calcium deposition disease, is a term used to describe radiographic evidence of calcific deposition within a tendon. Calcific tendinitis has been described in the gluteus maximus, the peroneus longus tendon, the popliteus tendon, the longus colli muscle in the neck, and the tendon of the rectus femoris.\textsuperscript{1-7} However, most of the literature on calcific tendinitis relates to crystal deposition within the rotator cuff of the shoulder.\textsuperscript{8,9} It is estimated to occur in approximately 10% of the population; however, most patients are asymptomatic.\textsuperscript{8,10} Its peak incidence occurs in the fifth decade of life, and it is almost exclusively found in adults older than 30 years.\textsuperscript{11,12}

Peri-articular pain as a result of calcific tendinitis can be indolent and chronic, and patients can have varying degrees of functional deficit. Patients may also present with an acute inflammatory event and have severe incapacitation, with restricted passive range of motion and a clinical picture that is concerning for septic arthritis.\textsuperscript{11,12} Severe pain associated with calcific tendinitis usually occurs during the resorptive phase, where there is vascular proliferation along the margin of the deposit, with infiltration of macrophages and multinucleated giant cells that digest the apatite crystals and release hyperalgesic cytokines.\textsuperscript{13-15}

The authors report a case of calcific tendinitis found within the hip labrum with a clinical presentation of acute, atraumatic, debilitating pain, with radiographic follow-up showing resorption of the calcific foci. The clinical picture is similar to what is described during the resorptive phase seen in calcific tendinitis of the shoulder. To the authors’ knowledge, only 1 other case series by Jones\textsuperscript{16} in 1955 reported this entity and there is no description of it in the current literature.

**CASE REPORT**

A healthy 41-year-old woman presented to an unaffiliated emergency department for evaluation of sudden-onset, severe left hip pain of 1 day’s duration. On presentation, she was unable to bear weight, but had no history of trauma, fever, or chills. Evaluation included orthogonald radiographs (Figure 1) and magnetic resonance imaging scan of the left hip (Figure 2). Laboratory studies, including complete blood count, erythrocyte sedimentation rate, and C-reactive protein value, were normal. There was no evidence of acute fracture or infection; therefore, she was provided with crutches and narcotic pain medication and was instructed to obtain follow-up in an outpatient setting. This patient was made aware that her medical history and images would be submitted for publication in a scientific journal, and informed consent was obtained.

The next day, she presented to the authors’ clinic, still unable to bear weight on the left lower extremity. She had no associated fever or chills. She reported previous intermittent groin pain with athletic activities, particularly with deep flexion of the hips. The groin pain before the acute episode, although bothersome, did not limit her daily activity. Vital signs taken in the clinic showed a hemodynamically stable patient with no current fever. Findings on physical examination were remarkable for inability to bear weight on the left lower extremity. She held the hip in slight flexion and had severe pain referred to the left groin region with gentle range of motion. Findings on neurovascular examination were normal, with intact pulses, good motor strength, and no sensory deficits.

Radiographs (Figure 1) showed evidence of large labral calcifications, consistent with underlying femoroacetabular impingement. Magnetic resonance imaging scan (Figure 2) showed similar mor-
phologic findings along with a large effusion.

The patient underwent ultrasound-guided aspiration the same day. The aspirated fluid was cloudy, and given the potential diagnosis of a septic joint, no intra-articular steroids were given at the time of aspiration. The fluid had a nucleated cell count of 7650, with 94% polymorphonuclear leukocytes and no crystals. The authors requested that laboratory services centrifuge the excess fluid for further evaluation. The laboratory staff described the pellet as having a “toothpaste-like” consistency. Gram stain showed no organisms, and the final cultures were negative for growth. Findings of repeat complete blood count and inflammatory markers were unremarkable.

During the course of 2 months, the patient was progressively able to bear weight and eventually returned to her baseline discomfort before the acute event. However, she continued to have impingement-type pain that was refractory to conservative measures. She underwent elective hip arthroscopy, with excision of the residual calcium deposits in addition to acetabuloplasty, labral repair, and femoral osteochondroplasty. Intraoperative photographs (Figure 3) showed calcium deposits within the labrum that had a “toothpaste-like” consistency.

**Discussion**

The deposition of calcium has been described in various locations throughout the musculoskeletal system. In the 1970s, Uhthoff et al hypothesized that calcific deposition was caused by a reactive calcification process as opposed to a degenerative process. Uhthoff and Loehr later described the disease process in 4 separate histopathologic stages.

The precalcific stage is marked by a decrease in oxygenation and infiltration of the area with chondrocyte-like cells that have elevated alkaline phosphatase activity. The calcific stage is subdivided into formative and resting phases. In the formative phase, calcium crystals are deposited within vesicles of the extracellular matrix and coalesce to form calcifications. During the resting phase, large deposits may cause mechanical symptoms that cause indolent, chronic pain. Radiographs obtained during both phases show distinct calcifications with well-defined borders.

The resorptive phase occurs after the calcification has been surrounded by vascularization that allows for an influx of macrophages that phagocytize the calcium crystals. This is radiographically apparent as a calcium deposit with blurred margins. Patients with active calcium resorption or rupture may present with acute, incapacitating pain because of the release of hyperalgesic cytokines. The final stage, the reparative phase, histologically shows infiltration of fibroblasts that repair the injured tendon. This final phase is most often radiographically and clinically imperceptible.

The patient presented similarly to patients seen in the resorptive phase of calcifying tendinitis. The authors believe that intra-articular rupture of the calcium deposit likely caused the patient’s symptoms. Her presentation was consistent with synovitis, and workup consisted of fluoroscopic-guided hip aspiration to evaluate for infectious and noninfectious causes. Findings of cell count, Gram stain, and culture were unremarkable for an infectious process. Analysis showed no evidence of crystals, ruling out the likelihood of gout or pseudogout. Although no crystals were noted on microscopic examination, a sample of the aspirate, when centrifuged, formed a pellet noted to have a “toothpaste-like” consistency. Calcium phosphate crystals, otherwise known as calcium hydroxyapatite, have been identified as the collections in calcifying tendinitis. The rectangular crystalline structures, measuring 1400×120 nm on average, are only visualized with scanning or transmission electron microscopy techniques and thus were not seen on standard light microscopy analysis.

**Conclusion**

This report highlighted a clinical presentation of hip labral calcification that has not been described in the orthopedic literature. Para-acetabular periarthritis calcarea has been described in the radiology literature as well as in the orthopedic literature as it pertains to femoroacetabular impingement. Although the radiographic findings in these reports appear to coincide with calcifying tendinitis, the calcium deposit may have been secondary to underlying femoroacetabular impingement.
impingement because the patient did not have acute-onset, atraumatic hip pain. The authors believe that acute rupture of the calcium deposit in the current case likely caused the acute symptoms of severe hip pain. The incapacitating pain that ensued likely was the result of macrophage-mediated phagocytosis of the intra-articular debris and subsequent release of hyperalgesic cytokines, coinciding with the resorptive phase of calcifying ten-dinitis. This must be considered when a patient presents with acute onset of pain and radiographically apparent labral calcifications.

References