



## CALCIFICATION OF THE LATERAL COLLATERAL LIGAMENT OF THE KNEE JOINT: A RARE ABNORMALITY AND CAUSE OF ACUTE KNEE PAIN

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<p><b>Article Info</b>  <i>Received 15/02/2015</i>  <i>Revised 27/03/2015</i>  <i>Accepted 12/04/2015</i></p> <p><b>Key words:</b>            Calcification, Knee,            Lateral Collateral            Ligament.</p>	<p><b>ABSTRACT</b>            Calcification of the lateral collateral ligament (LCL) of the knee is a rare cause of knee pain and is thought to reflect underlying hydroxyapatite deposition. The management is usually conservative and there is subsequent resolution of the calcification. This rare abnormality, to our knowledge, has never been previously shown on Computerised Tomography (CT) imaging with 3D reconstruction. We report the case of an elderly lady who presented with this condition treated conservatively and relevant CT images of the LCL calcification.</p>
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### INTRODUCTION

Calcification of the lateral collateral ligament (LCL) of the knee is a rare phenomenon to cause acute knee pain. This, to our knowledge, has never been previously reviewed on Computerised Tomography (CT) Scan with 3D reconstruction. Very few reports have been published in literature of calcification of the LCL and Popliteus tendon. The management is usually conservative and there is subsequent resolution of the calcification. We report this rare condition with radiographic and CT findings and discussion

### Case History

A Fifty year old lady presented for an Orthopaedic consult with complaints of pain on the lateral aspect of her left knee joint since two days. Her pain was sudden in onset and was aggravated on weight bearing and on sitting in crossed legged or squatting position. Her symptoms gradually worsened over 48 hours. There was no history of trauma or massage. Her past medical history was irrelevant and no features suggestive of sepsis or any systemic illness. She denied any history of joint problems in the past.

On Examination, her left knee joint was slightly warm compared to her right knee with fullness of the joint and extreme tenderness on palpation. Knee flexion and extension was associated with extreme pain on the lateral aspect of the knee, however there was no restriction in range of movement. Joint examination showed intact ligaments.

Anteroposterior radiograph of the knee joint revealed a large calcific deposit involving the lateral side of the knee joint in the region of the lateral collateral ligament. Computerised Tomographic (CT) scan showed calcification of the lateral collateral ligament along with several benign soft tissue calcifications along the lateral capsule of the knee, overlying the popliteus tendon.

The patient was treated conservatively with rest, analgesics and ice fomentation. The patient returned for follow up 5 days later with good symptomatic relief. Patient was advised the same regimen for another week. 2 weeks later, patient came for her second follow up completely symptom free. Anteroposterior radiographs taken at this juncture revealed complete resolution of the calcification. The patient was reviewed every monthly for



the next 3 months, during which she did not complain of any recurrence of symptoms or any new joint problems. Anteroposterior radiographs taken at the follow up visits

did not show any new calcification or recurrence of the calcification seen on initial x rays.

**Figure 1. Plain Anteroposterior Radiograph of the left knee showing calcification on the lateral aspect of the knee.**



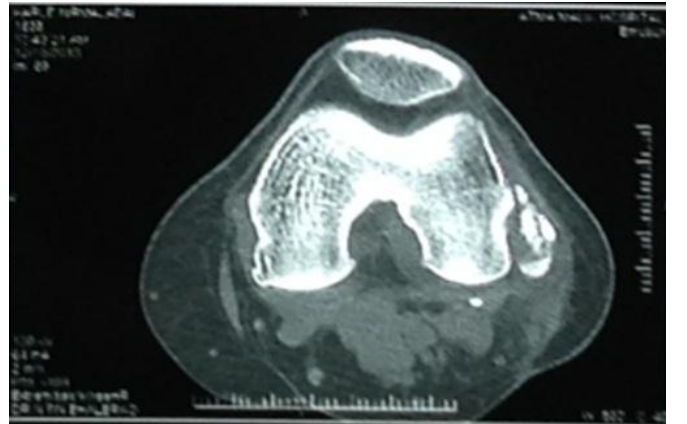
**Figure 2. Computerised tomographic images with 3D Reconstruction of the knee joint (Coronal images) showing characteristic calcific deposits within the body of the lateral collateral ligament and along the lateral joint capsule and the popliteus tendon**



**Figure 3. Computerised tomographic images with 3D Reconstruction of the knee joint (Coronal images) showing characteristic calcific deposits within the body of the lateral collateral ligament and along the lateral joint capsule and the popliteus tendon**



**Figure 4. Computerised tomographic image of the knee joint (Axial image) showing thickening and calcification of the lateral collateral ligament**



## DISCUSSION AND CONCLUSION

Radiographic evidence of calcification at the lateral aspect of the knee joint has been reported in the literature. The structures reported include popliteal tendon, vastus lateralis, iliotibial tract and also, in a few cases, the lateral collateral ligament. In 1952, Lamb [1] was the first to describe presence of calcium salts in the knee joint, but these deposits were seen in the medial aspect of the joint.

In 1955, Holden [2] was the first to describe the radiographic evidence of presence of calcium deposits in the lateral aspect of the knee joint. He reported 2 cases with history of pain on the outer aspect of the knee joint with swelling and restriction of movement. In the first patient, 64 years old male, as the symptoms were not relieved by rest, the knee was explored which showed

presence of a whitish mass within the substance of the popliteus. Pathological examination revealed chiefly acellular collagen with a low calcium content. In the second patient, a 60 years old female, radiograph showed calcium deposits on lateral side of the joint. This pain rapidly subsided with bed rest and a follow up radiograph taken six weeks later showed decrease in the size of the mass.

In 2002, Tibrewal [3] reported three cases of acute onset pain over lateral side of knee with locking of the knee joint. Radiographs revealed well circumscribed calcifications within the tendon of popliteus. A similar study was done by Shenoy et al in 2009[4] where a patient presented with severe pain in lateral aspect of the knee joint without locking of the knee joint. MRI showed the



calcification present intra articularly near the popliteus tendon. In the first study patients responded to intra lesional corticosteroid injections and gained immediate pain relief. In the second study arthroscopic partial synovectomy followed by excision and biopsy was done. Histopathological evaluation revealed presence of Hydroxyapatite crystals.

The first ever case report of calcification of the lateral collateral ligament was given by Anderson et al [5] in 2003. In this report four patients presented with acute lateral knee pain that worsened at night. Plain radiographs showed calcification over lateral aspect of the knee joint. MRI confirmed the presence of calcification in lateral collateral ligament. All four patients responded to conservative management.. Khan et al [6] in 2009 reported a similar case of an elderly lady with calcification in the lateral collateral ligament which resolved completely on conservative management. In both the studies although cause was unknown Hydroxyapatite Deposition Disease (HADD) was presumed to be the cause.

HADD is characterized by presence of calcium phosphate crystals predominantly hydroxyapatite in the peri articular soft tissues especially the tendons. It most frequently involves the shoulder but numerous other sites including the knee have been recognized [7]. Thee etiology

of HADD is not clearly understood. In 1934 Codman [8] suggested the presence of these calcifications in relation to pressure and compression over the tendon. Sandstrom [9] postulated this disease to decrease the vascularity and preexisting tissue degeneration. Many more postulations have been made, however to date the exact etiology remains unknown. In plain radiographs HADD deposits appear as homogenous, amorphous densities without trabeculations with well or ill-defined margins. Characteristic radiologic feature that helps distinguish HADD from other calcific lesions is the location of the deposit within tendons close to their site of insertion, but may also affect joint capsules and bursae. Patients usually present with sudden onset knee pain with swelling of the joint; with conservative treatment the pain subsides and the calcium deposits completely disappear.

Differential diagnosis for such calcifications include gout, scleroderma, CPPD disease, dystrophic calcification secondary to trauma and hyperparathyroidism [7,10].

In our study the presenting features of the patient were consistent with HADD and similar to those presented by Anderson et al and Khan et al. The course of the disease was self-limiting and subsequent resorption of the calcification was seen.

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