CASE REPORT

Calcification of the Lateral Collateral Ligament of the Knee: A Rare Cause of Acute Knee Pain

Imran Khan[1] and Muhammad Iftikhar Rashid[2]

ABSTRACT

Calcification of the lateral collateral ligament is a rare phenomenon, which can cause acute knee pain. The management is usually conservative and there is subsequent resolution of the calcification seen on initial radiographs. It is important to exclude more sinister pathology such as septic arthritis as a cause of pain. We report the case of an elderly lady who presented with acute knee pain. Initial radiographs showed calcification at the lateral aspect of the knee joint. The pain settled with conservative management. Resorption of calcification was demonstrated on subsequent radiographs.

Key words: *Calcification. Knee. Lateral collateral ligament. Radiographs.*

INTRODUCTION

Calcification of the lateral collateral ligament is a rare phenomenon that can cause acute knee pain. The lateral collateral ligament, also known as the fibular collateral ligament, arises from the lateral femoral condyle. It inserts on the lateral aspect of the middle third of the fibular head, occasionally joining the biceps femoris tendon. It requires visualization in sequential coronal images on MR as its posterior and oblique course makes visualization on a single coronal image difficult. The insertion of the lateral gastrocnemius tendon is posterior to the lateral collateral ligament.[1] The management is usually conservative and there is subsequent resolution of the calcification seen on initial radiographs.

We report this rare condition with radiographic findings and discussion.

CASE REPORT

A 64-year-old lady presented in emergency with severe pain on the lateral aspect of her left knee joint, associated with difficulty in weight bearing. Her symptoms had a sudden onset of mild niggling pain with gradual worsening over 48 hours. There was no history of trauma.

Her past medical history included hypertension, hypercholesterolemia and osteoporosis. Current medications included an anti-hypertensive, a statin and a bisphosphonate. She was otherwise quite fit and active for her age and denied any history of previous systemic illnesses or joint problems.

On examination, her body temperature was normal and there were no signs or symptoms of any systemic illnesses. Her left knee joint was slightly warm compared to the right, and was markedly tender on palpation on the lateral aspect. Joint examination showed intact ligaments. There were active and passive movements with full extension and flexion up to 90 degrees. There was no joint effusion, swelling, overlying skin erythema or neurovascular compromise. The patient could bear weight but it was painful. Blood tests showed slightly elevated CRP and white cell count, with neutrophilia. Initial radiographs (Figure 1) showed calcification at the lateral aspect of the left knee joint. The bones and joint were otherwise unremarkable. After review by the Orthopaedic team, it was felt that the patient did not have septic arthritis and was discharged home on oral anti-inflammatory medications and crutches to help with mobility. She was advised to return if the symptoms did not settle or deteriorated.

The patient's symptoms started to subside 48 hours after she started taking anti-inflammatory medication. She was seen by the Orthopaedic team 7 days after her casualty department visit. On evaluation, she was mildly tender at the lateral aspect of the joint. A small mass was palpable at the level of the fibular head, which moved freely over the underlying bone. The patient's mobility was back to normal and she no longer required analgesics.

Further imaging was performed on 1.5 Tesla MRI Unit after 5 weeks. Magnetic resonance imaging showed mild thickening of the lateral collateral ligament at its fibular attachment (Figure 2). The lateral collateral ligament was otherwise intact. Mild swelling and oedema was seen around the ligament and the area corresponded to the calcification seen on the plain film. A degenerative horizontal tear was noted through the

Department of Radiology, West of Scotland Radiology Trainee, Glasgow, UK.
Department of Radiology, Wishaw General Hospital, Wishaw, UK.

Correspondence: *Dr. Imran Khan, 116 Elder Crescent, Cambuslang, Glasgow, G72 7GL, UK.*
E-mail: drkhan02@yahoo.com

Received December 08, 2010; accepted December 19, 2011.

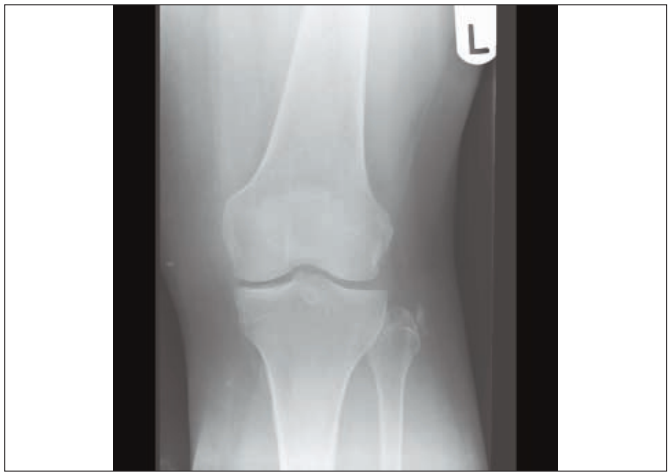

Figure 1: Plain X-ray of the left knee showing lateral collateral ligament calcification.

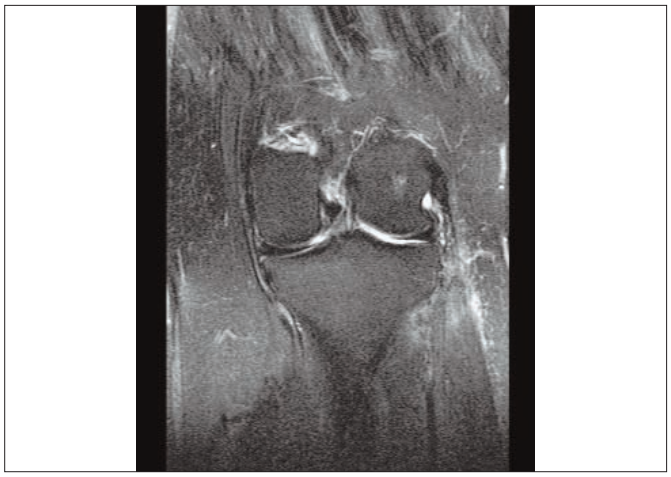

Figure 2: T2WI MRI coronal section showing thickening and oedema in the lateral collateral ligament region.

posterior horn of the medial meniscus, which was not considered to be a source of the patient's symptoms. The bones and all other ligaments were unremarkable.

The patient was reviewed again after 5 months. She did not complain of any recurrence of her symptoms or any new joint problems. Plain radiographs were performed which showed complete resolution of the calcification seen on the initial X-rays.

DISCUSSION

There have been cases reported in the literature of acute atraumatic knee pain associated with radiographic evidence of calcification at the lateral aspect of the knee joint. Structures described include popliteal tendon, iliotibial tract, vastus lateralis and bursae around the knee joint.[2-5] Anderson *et al*. were the first to describe 4 cases of acute lateral knee pain associated with calcification seen at the lateral aspect of the knee joint on plain radiographs, and MRI findings confirming the calcification in the proximal portion of an intact lateral collateral ligament.[6] The clinical history revealed no trauma or systemic illnesses. The pain appeared inflammatory in nature and worsened at night. MRI performed showed calcification within a thickened but intact lateral collateral ligament with adjacent soft tissue oedema. Only one of the 4 cases had additional calcification between the lateral collateral ligament and the biceps femoris tendon. There was resorption of calcification in all the cases. Only one patient underwent joint aspiration, which did not show any organisms. All patients underwent successful conservative management. Though a definite diagnosis was not made, the cause was presumed to be calcium hydroxyapatite deposition disease, as the exact cause remained unknown. One of the patients in their series returned with calcific tendinitis of the rotator cuff muscle after 2 years.

Calcium hydroxyapatite deposition disease is one of the common causes of periarticular calcification, usually occuring in middle-age group.[7] The disease can be primary (idiopathic) or may occur secondary to chronic renal failure and collagen vascular disease. It usually presents as amorphous calcification in tendons, close to their site of insertion, but may also affect joint capsules and bursae.[8] Calcification may also be present away from the joint.[4] The aetiology is uncertain. However, Gondos observed that there was a higher incidence in joints with a higher physiologic range of motion.[9] Shoulder joint is most commonly affected though it can also present at the hip, elbow, wrist and knee joints. Patients usually present with chronic or acute pain though asymptomatic deposits in the shoulder joint have been documented.[7] Disappearance of the calcification is a recognized phenomenon. Another variant of this disease causes intra-articular calcification leading to severe destruction of the joint. This most commonly manifests as the Milwaukee shoulder syndrome seen in elderly females.[7]

The clinical presentation of hydroxyapatite deposition disease can mimic an infectious process.[10] Therefore, it is important to keep septic arthritis as a differential in mind as misdiagnosis can have serious consequences for the patient. Other differential diagnoses for peri-articular and soft tissue calcifications including gout, CPPD disease, scleroderma, dermatomyositis, hyper-parathyroidism and dystrophic calcification secondary to trauma. However, there are other associated findings visible on the plain radiograph due to these disease entities such as chondrocalcinosis and peri-articular erosions.[7,8]

This patient's history and symptoms were similar to those presented in the case series by Anderson *et al*. The only differences were in the age group, where our patient was older, and the fact that our patient's pain did not get worse at night. No aggressive or invasive procedures were performed in our case, as there was no suspicion of septic arthritis or presence of a joint

effusion on examination. The course of the disease and the subsequent resorption of calcification tend to favour the diagnosis of calcium hydroxyapatite deposition disease.

In summary, calcification of the lateral collateral ligament is a rare phenomenon that can present as acute knee pain. Its cause is uncertain though the most likely aetiology is considered to be calcium hydroxyapatite deposition disease. Once septic arthritis has been excluded, conservative management is advised which leads to successful resolution of the pain and resorption of calcification seen on initial radiographs.

REFERENCES

1. Haims AH, Medvecky MJ, Pavlovich R Jr, Katz LD. MR imaging of the anatomy of and injuries to the lateral and posterolateral aspects of the knee. *AJR Am J Roentgenol* 2003; **180**:647-53.
2. Holden NT. Deposition of calcium salts in the popliteus tendon. *J Bone Joint Surg* 1955; **37**:446-7.
3. McCarty DJ, Gatter RA. Recurrent acute inflammation associated with focal apatite crystal deposition. *Arthritis Rheum* 1966; **9**:804-19.
4. Rehak DC, Fu FH. Calcification of the vastus lateralis: a case report. *Am J Sports Med* 1992; **20**:227-9.
5. Norley T, Bickel WH. Calcification of the bursae of the knee. *J Bone Joint Surg Am* 1949; **31**:417-20.
6. Anderson SE, Bosshard C, Steinbach LS, Ballmer FT. MR imaging of the calcification of the lateral collateral ligament of the knee: a rare abnormality and a cause of lateral knee pain. *AJR Am J Roentgenol* 2003; **181**:199-202.
7. Hayes CW, Conway WF. Calcium hydroxyapatite deposition disease. *Radiographics* 1990; **10**:1031-48.
8. Grainger AJ, McGonagle D. Imaging in rheumatology. *Imaging* 2007; **19**:310-22.
9. Gondos B. Observations on periarthritis calcarea. *Am J Roentgenol Radium Ther Nucl Med* 1957; **77**:93-108.
10. Claudepierre P, Rahmouni A, Bergamasco P, Montazel JL, Larget-Piet B, Chevalier X. Misleading clinical aspects of hydroxyapatite deposits: a series of 15 cases. *J Rheumatol* 1997; **24**:531-5.