CASE REPORT

Acute calcific periarthritis of the knee presenting with calcification within the lateral collateral ligament

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SUMMARY

A 71-year-old woman was admitted with acute swelling of the right knee, pain on the lateral aspect and restricted movement. There was no instability or locking. She had no history of trauma and was generally in good health. Plain radiographs demonstrated a calcific opacity adjacent to the lateral femoral condyle. This was shown to be within the lateral collateral ligament (LCL) at ultrasound and MRI. A diagnosis of acute calcific periarthritis (ACP) was made. The patient's symptoms resolved within a few weeks with simple analgesia. ACP presenting with calcification within the LCL is rare. It is important to recognise the clinical and imaging findings of this condition as it may mimic other more serious pathologies such as infection and gout. This may result in unnecessary investigations, misdiagnoses and incorrect treatments.

BACKGROUND

We describe the case of a 71-year-old woman who presented with acute pain and swelling of the right knee restricting her movements. On imaging, she was found to have calcification within the lateral collateral ligament (LCL) of the knee. A diagnosis of acute calcific periarthritis (ACP) due to calcification within the LCL was made. Anderson *et al*¹ described four patients who presented with acute and severe atraumatic knee pain with calcification in or around the LCL of the knee. Only three of the patients had calcification confirmed within the LCL. All were men, aged from 38 to 46 years, with a mean age of 42.5 years. To the best of our knowledge, ours is the first case describing ACP presenting as calcification within the LCL in a 71-year-old woman. We also describe the findings on three imaging modalities: plain radiograph, MRI and ultrasound.

ACP results from hydroxyapatite deposition in the tissues. It is characterised by periarticular inflammation and juxta-articular calcium deposition.^{2 3} The condition is typically monoarticular and can affect the hands, feet, shoulders, knees and hips. Clinically, it presents with rapid onset swelling, erythema and restriction of motion. Symptoms usually resolve within 3–4 weeks with minimal treatment.³ It is important to recognise the clinical and imaging findings of this condition as it may mimic other more aggressive pathologies such as infection, trauma, gout and pseudogout, particularly if plain radiographs have not been obtained initially.^{1 3} Misdiagnosis can therefore lead to

unnecessary and invasive investigations, as well as inappropriate treatments. Our case is important in that it describes an unusual and rare form of ACP that presents with calcification within the LCL.

CASE PRESENTATION

A 71-year-old woman presented to the orthopaedic outpatients clinic on 21 October 2014 with a 1 week onset of severe pain of the lateral aspect of the knee, associated with swelling of the whole knee, reduced mobility and her leg 'stuck' in slight flexion. She described having to lift the leg in order to move it. There was no redness. The patient initially treated herself with cold compression and rest. She obtained relief from the analgesic co-codamol. There was no preceding history of injury or other problems related to her knee. She had no systemic symptoms or complaints other than atrial fibrillation, for which she was taking rivaroxaban. On examination, she was non-feverish

Figure 1 Plain radiograph of the right knee demonstrating a dense, homogenous, rounded, calcific opacity adjacent to the lateral femoral condyle. This is the typical appearance of acute calcific periarthritis.

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and the knee was tender to palpation over the lateral joint line. The collateral ligaments were stable. The patient had a range of movement of 20–70°, which was limited by pain.

INVESTIGATIONS

- ▶ Plain radiographs of the right knee demonstrated a dense, homogenous, calcific opacity adjacent to the lateral femoral condyle, also minor valgus angulation at the knee but no other significant abnormality (figure 1).
- ▶ MRI scan on 21 October 2014 demonstrated an area of low-signal intensity within the LCL of the right knee, adjacent to the lateral femoral condyle and corresponding with the calcification seen on the plain radiographs. There were areas of oedema and inflammation within and around the LCL (figures 2–4).
- ▶ Ultrasound scan on 8 December 2014 demonstrated calcification lateral to the lateral femoral condyle and within the LCL of the right knee. This was surrounded by a rim of low echogenicity, consistent with inflammation (figure 5).
- ▶ Ultrasound-guided aspiration of the area of calcification and surrounding soft tissues was carried out on 11 December 2014. Histological analysis confirmed the absence of gout or any other crystal arthropathy.
- ▶ Blood test results on 3 November 2014 were normal:
 White cell count $7.1 (4\text{--}11 \times 10^9/\text{L})$
 Haemoglobin 147 (130–180 g/L)
 Platelets 351 ($150\text{--}400 \times 10^9/\text{L}$)
 Neutrophils 4.7 ($2\text{--}7.5 \times 10^9/\text{L}$)
 Sodium 142 (133–145 mmol/L)
 Potassium 4.8 (3.5–5.5 mmol/L)
 Creatinine 74 (45–84 mmol/L)
 Estimated glomerular filtration rate 67 ($>90 \text{ mLs/min/1.73 m}^2$)
 C reactive protein 4 ($<5 \text{ mg/L}$)
 Bilirubin 7 (1–17 mmol/L)
 Total protein 72 (60–80 g/L)
 Globulin 24 (15–35 g/L)
 Alanine transaminase 30 (7–55 IU/L)
 Alkaline phosphatase 87 (35–104 IU/L)

Figure 2 MRI scan of the right knee. Coronal proton density fat-suppressed image of the right knee, demonstrating a lobulated, predominantly low-signal intensity nodular focus within the lateral collateral ligament. The low-signal intensity is mainly at the edges and rim of the nodule, and is consistent with the calcification seen on the plain radiograph.

Figure 3 MRI scan of the right knee. Coronal proton density fat-suppressed image of the right knee, demonstrating a lobulated, predominantly low-signal intensity nodular focus within the lateral collateral ligament. The low-signal intensity is mainly at the edges and rim of the nodule and is consistent with the calcification seen on the plain radiograph.

Calcium 2.51 (2.2–2.6 mmol/L)

Adjusted calcium 2.47 (2.2–2.6 mmol/L)

Thyroid stimulating hormone 1.99 (0.3–5 mIU/L)

Random glucose 3.9 (3.3–7.7 mmol/L)

Plasma viscosity 1.78 (1.5–1.72 mpas), repeat plasma viscosity (4.12.14) 1.65 mpas

DIFFERENTIAL DIAGNOSIS

Differential diagnoses of the clinical presentation include infection or fracture, due to the pain, swelling and restricted movement. This was discounted by investigations including normal blood results, such as erythrocyte sedimentation rate (ESR) and white cell count (WCC). Other differentials clinically included gout, pseudo-gout and other crystal arthropathies. All were discounted based on histological analysis of the aspirated sample of the area of calcification and surrounding soft tissues. Differential diagnosis from the imaging findings of calcification

Figure 4 MRI scan of the right knee. Sagittal T1-weighted image demonstrating low-signal intensity within the lateral collateral ligament (LCL), corresponding with the calcification within the LCL.

Figure 5 Ultrasound scan of the right knee demonstrating a calcific opacity within the lateral collateral ligament. This is surrounded by hypoechogenicity consistent with inflammation and oedema.

adjacent to the knee joint included chronic renal failure, hypercalcaemia and hyperparathyroidism. These were also discounted by the blood results and the absence of joint pathology.

TREATMENT

The patient was managed conservatively. She was unable to take non-steroidal anti-inflammatory tablets due to her other medication (rivaroxaban). She continued to take co-codamol when required.

OUTCOME AND FOLLOW-UP

After a week of analgesia, the patient was able to weight bear. At 2 months' follow-up, the knee swelling had completely subsided and movement was fully restored. She was able to walk for 25 min without pain, experiencing minor discomfort only after a long walk.

DISCUSSION

ACP is characterised by “peri-articular inflammation associated with juxta-articular deposits of basic calcium phosphate crystals”.² It has been described in various parts of the body; mainly the hands and shoulders, but also the hips, feet, elbows and knees. The calcification may be within the joint, the surrounding soft tissues, tendons, ligaments or bursae.³ We have described a rare case of calcification within the LCL of the knee of an elderly woman. ACP should be suspected in any patient presenting with an acutely swollen and painful joint with limited movement. It is important for clinicians to be aware of its clinical presenting features in order to avoid mistaking it for more serious conditions such as infection, gout or other inflammatory arthropathies.³ It is also important to be aware of its imaging findings. The main appearance on plain radiographs is of dense, homogenous, amorphous, cloud-like, generally round or oval calcific deposits, with no cortex or internal trabecular pattern. This is confirmed at MRI and ultrasound. The calcification may be juxta-articular, intra-articular, intratendinous or within the surrounding soft tissues. For the unsuspecting, this may be interpreted as due to other pathology and result in investigations including biopsies or surgery, or inappropriate treatment. ACP is self-limiting and typically symptoms resolve within a few weeks. The calcifications also typically resolve, or greatly reduce, over a period of several weeks.³

ACP is part of the spectrum of hydroxyapatite deposition disease. Hydroxyapatite is deposited into psammoma-like bodies

surrounded by extensive inflammatory cells, in particular neutrophils.³ The mechanisms resulting in the formation of the calcifications are not yet fully understood. The initial insult may be trauma and necrosis, leading to poor blood supply and injury.⁴ This may result in local hypoxia within the tendon and soft tissues, followed by metaplasia and formation of fibrocartilage, finally resulting in deposition of dystrophic calcifications.⁵ The reason for the clinical presentation of swelling, pain and restricted movement is postulated to be the result of rupture of these calcific deposits, followed by an inflammatory reaction. ACP normally occurs as an isolated condition with no obvious triggering factors or underlying cause. Laboratory results, including serum calcium, phosphorus, alkaline phosphatase and uric acid, are typically normal. Occasionally, WCC and ESR may be elevated, but they are otherwise usually normal. However, patients with hypothyroidism, rheumatoid arthritis and systemic sclerosis,³ as well as those with metabolic conditions including diabetes, gout and pseudogout, may also get ACP.⁶ These conditions can be excluded by the appropriate blood results. In our patient, all relevant blood tests were normal.

There is very little in the published literature regarding ACP of the LCL. Anderson *et al*¹ described three patients with the condition confirmed by MRI. Despite this, all were male and aged from 38 to 46 years, with a mean age of 42.5 years. The case we present, to the best of our knowledge, is the first describing ACP with calcification within the LCL of a 71-year-old woman.

Learning points

- ▶ Acute calcific periarthritis (ACP) is a self-limiting condition that normally resolves with minimal treatment within a few weeks.
- ▶ ACP with calcification in the lateral collateral ligament is rare, but has now been described in adult patients, both male and female.
- ▶ ACP is normally mono-articular, presenting with pain and swelling, with restricted movement. The clinical picture may mimic other conditions such as fracture and infection.
- ▶ Radiologically, ACP demonstrates juxta-articular calcification, or calcification within tendons, bursae and ligaments. On imaging, it may therefore be mistaken for other conditions including gout, pseudo-gout, or systemic conditions such as hyperparathyroidism and renal failure.

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Competing interests None declared.

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REFERENCES

- 1 Anderson SE, Bosshard C, Steinbach LS, *et al*. MR imaging of calcification of the LCL of the knee: a rare abnormality and a cause of lateral knee pain. *AJR Am J Roentgenol* 2003;181:199–202.
- 2 Gálvez J, Linares LF, Villalón M, *et al*. Acute calcific periarthritis of the fingers. *Rev Rhum Engl Ed* 1995;62:602–4.

- 3 Dumas C, Vazirani RM, Clifford PD, *et al.* Acute calcific periarthritis of the hand and wrist: a series and review of the literature. *Emerg Radiol* 2007;14:199–203.
- 4 Dilley DF, Tonkin MA. Acute calcific tendinitis in the hand and wrist. *J Hand Surg Br* 1991;16:215–16.
- 5 Unthoff HK, Sarkar K, Maynard JA. Calcifying tendinitis: a new concept of its pathogenesis. *Clin Orthop Relat Res* 1976;118:164–8.
- 6 Lee KB, Song KJ, Kwak HS, *et al.* Acute calcific periarthritis of proximal interphalangeal joint in a professional golfer's hand. *J Korean Med Sci* 2004;19:904–6.

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