CASE REPORT

Lipoma Arborescens of the Knee Joint after Anterior Cruciate Ligament Injury

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Introduction

Lipoma arborescens (LA) is a rare, benign, intra-articular condition characterized by subsynovial proliferation of mature fat cells^{1,2}. The resultant fatty mass produces characteristic frond-like and villous synovial projections that give rise to a macroscopic appearance reminiscent of a leafy tree, hence the name arbor (tree in Latin). The condition is commonly seen in the knee joint but has also been described in the hip, glenohumeral, wrist and ankle joints^{1,2}. The cause of this benign condition is unknown. Associated conditions have included osteoarthritis, rheumatoid arthritis, psoriatic arthritis, trauma, diabetes and popliteal cysts. It is not clear whether these conditions are causative or merely co-incidental¹⁻⁴. Though trauma is reportedly a cause of this entity, we found no descriptions of a specific ligamentous injury in our search of published articles in English. We present here a case of LA that developed a few months after injury to anterior cruciate ligament. We obtained consent from the patient to use his clinical details and imaging for presentations and publication.

Case Report

A 38-year-old man presented to us with pain and swelling in the left knee following an injury while playing recreational football. Radiographs were normal. We aspirated 100 mL of blood from the knee and immobilized it with a knee brace. Three weeks later, he was free of pain, the swelling had resolved and his range of motion in the affected joint was from 0° to 130°. Examination revealed positive anterior drawer and Lachmann tests. An MRI scan confirmed the diagnosis of anterior cruciate ligament (ACL) tear (Figs 1 and 2). The patient refused ACL reconstruction and opted for non-operative management and was subsequently lost to follow-up. Eight months after the patient's index injury, he reported that his left knee had been swollen for another 4 months. He had returned to

playing the sport in which he had sustained the original injury and denied having episodes of instability.

On examination of the left knee there was swelling in the supra- and para-patellar regions (Fig. 3). The patellar ballot test confirmed effusion and the cross fluctuation test was positive. The affected knee had a range of motion from 0°–120° flexion and we could elicit no signs of ligament instability because of the effusion. We detected no other joint abnormalities. Routine blood investigations, erythrocyte sedimentation rate, C-reactive protein, serum uric acid and rheumatoid arthritis factor were normal.

We aspirated 120 mL of amber yellow synovial fluid from the left knee under aseptic conditions. Biochemistry and cytological examination of the fluid were normal and we made a diagnosis of post-traumatic chronic synovitis of the knee. We recommended a compression bandage, knee brace and non-steroidal anti-inflammatory drugs for two weeks, with quadriceps strengthening exercises. The patient returned with a recurrent painless swelling of the left knee. An MRI scan confirmed the presence of a large joint effusion and villous proliferation of the suprapatellar pouch synovium. In addition, there was a frond-like mass arising from the anterior aspect of the synovium. The signal intensity was similar to that of fat on all pulse sequences (Figs 4 and 5). Accordingly, we diagnosed LA and scheduled the patient for open synovectomy and ACL reconstruction.

We excised thickened synovium and fibrofatty tissue from the supra patellar pouch, medial and lateral para-patellar gutters and between the femoral and tibial condyles. The anterior cruciate ligament was torn, whereas the posterior cruciate ligaments and menisci were intact. We found cartilage fibrillation over the medial tibial condyle. We reconstructed the ACL using hamstring grafts and immobilized the patient in a plaster slab for two weeks, after which we instituted an ACL

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Fig. 1 Sagittal T₁ MRI 4 weeks after index injury showed ACL tear.

Fig. 2 Sagittal T₂ MRI 4 weeks after index injury showed ACL tear.

rehabilitation protocol. Histopathological examination of resected tissue showed large villi and mature adipose tissue interspersed with chronic inflammatory cells (Fig. 6), confirming the diagnosis of lipoma arborescens. At two-year follow-up, the patient was clinically asymptomatic with no instability or swelling.

Discussion

Hoffa first described of LA in 1904 (Albert Hoffa, German surgeon, 1859–1907; paper presented at the annual session of the American Medical Association, Atlantic City, NJ, 1904), attributing it to post-traumatic adipose tissue proliferation⁵. The exact cause of this condition remains unknown. However, it has been theorized that it is a nonspecific reactive change in the synovial membrane to joint trauma, chronic synovitis or arthritis-like rheumatoid arthritis, rather than a neoplastic process^{1,5}. There is characteristically infiltration of lymphocytes and plasma cells amidst the adipocytes. Although our patient's knee initially evidenced no arthritic changes, sub-clinical instability secondary to an untreated ACL injury caused chronic irritation in the synovial lining, which may have activated proliferation of subsynovial fat cells. Alternatively, subsynovial connective tissue may have undergone metaplastic proliferation to fatty tissue, eventually resulting in LA. Weitzman described a case of LA in a 26-year-old army recruit who had a definite history of trauma³. However, at arthrotomy the menisci, ACL and articular surfaces were

normal³. Although this disease has been reported in multiple other joints, the knee is by far the most commonly involved joint^{1,2,5}. Aydin *et al.* reported bilateral LA in two sisters; however, they ruled out the possibility of genetic association⁶. Thus, there are two distinct categories of LA: an idiopathic “primary” type affecting younger patients, and a much more common “secondary” variety associated with arthritis^{1,5,7}.

Before the era of MRI, the diagnosis of LA was extremely difficult to confirm radiologically⁸. In fact, by the mid-1990s, only 13 cases had been reported⁸. The nonspecific clinical presentation and conventional radiographic findings required

Fig. 3 Knee swelling at second presentation.

Fig. 4 Sagittal T₂ proton density turbo spin echo image at second presentation.

surgical and pathologic correlation. Conventional arthrography offered improved sensitivity but lacked specificity, the differential diagnosis including other causes of intra-articular filling defects such as synovial chondromatosis and pigmented villonodular synovitis. Although the role of CT has also been explored, MR imaging has proved to be the ideal modality for identifying the disease because of its excellent contrast resolution and sensitivity for fatty tissues^{2,5,8}. The radiologic community was quick to agree that LA has a pathognomonic MRI appearance^{2,5,8}. Characteristic features include a synovial mass with an arborescent, frondlike architecture and a signal intensity similar to that of fat on all pulse sequences, with suppression of the signal on short T1 inversion recovery sequences or pre-saturation of the fat; associated joint effusion; potential chemical shift artifacts at the fat–fluid interface in the joint; and absence of magnetic susceptibility effects associated with hemosiderin, as seen in pigmented villonodular synovitis. Furthermore, the synovial mass does not enhance after i.v. administration of contrast medium^{2,5,8}. Three distinct patterns of LA have been identified, the most common of which is a diffuse villous proliferation; the other two are a discrete mass lesion and a mixed pattern^{2,5,8}. Joint effusion is universally present^{2,5,8}. Degenerative changes are observed in 87% of patients and meniscal tears in 72%⁸.

The differential diagnosis of a soft boggy swelling in the suprapatellar pouch includes pigmented villonodular synovi-

Fig. 5 Fat suppressed image at second presentation.

tis, synovial lipoma, synovial chondromatosis, rheumatoid arthritis, synovial hemangioma, amyloid arthropathy, xanthomata and LA¹. A number of published reports have described patients who have been treated for diagnoses of rheumatoid or

Fig. 6 Histopathology showing large villi and mature adipose tissue interspersed with chronic inflammatory cells HE × 100.

other arthritides for 3 to 12 years and only has MRI scanning resulted in a diagnosis of LA⁸⁻¹⁰. Aydin *et al.* suggest that osteoarthritis develops secondary to LA⁶. Therefore, they stress that early diagnosis of LA is crucial⁶. Some reports suggest a correlation between the severity of osteoarthritis and the duration of LA^{6, 7}. In developing countries, tuberculosis is a public health problem and most tuberculous arthritis of the knee presents with indolent joint swelling due to synovial hypertrophy and fluid accumulation that can persist for years before diagnosis¹¹. Thus, it is imperative to use MRI at an early stage to aid in diagnosing this entity, especially with refractory knee swellings.

Histopathologically, the differential diagnosis of LA includes other fat-containing, benign synovial disorders like synovial lipoma and Hoffa disease. Hoffa disease is a syndrome in which the infra-patellar fat pad impinges between the femoral condyle and patella. Microscopically, LA is characterized by diffuse replacement of the subsynovial layer by mature fat cells with moderate infiltration of mononuclear inflammatory cells. Intra-articular lipoma, which may be covered by

synovium, does not arise from or replace the subsynovial layer. In addition, intra-articular lipoma lacks a villous frond-like appearance¹². The treatment of LA is either arthroscopic synovectomy or open synovectomy, both having short term morbidity. Recurrence of lesions following synovectomy is uncommon. After undergoing synovectomy, our patient required no further treatment.

It seems logical to conclude that our patient had post-traumatic monoarticular LA caused by a documented ACL injury. The absence of other joint involvement and no laboratory investigations suggestive of inflammatory arthritis support the conclusion that trauma was the cause of LA in our patient. LA is an increasingly recognized articular disease process. Given its pathognomonic appearance, it should be readily diagnosed by the radiologist on musculoskeletal MR images. The purpose of this report is to document the first case of LA after ACL injury reported in English and to highlight the importance of early diagnosis to enable treatment of this potential cause for development of early osteoarthritis.

References

1. Kloen P, Keel SB, Chandler HP, Geiger RH, Zarins B, Rosenberg AE. Lipoma arborescens of the knee. *J Bone Joint Surg Br*, 1998, 80: 298–301.
2. Das D, Sallomi DF, Howlett DC. A case of chronic knee pain and swelling. *Br J Radiol*, 2011, 84: 92–95.
3. Weitzman G. Lipoma arborescens of the knee. report of a case. *J Bone Joint Surg Am*, 1965, 47: 1030–1033.
4. Hubscher O, Costanza E, Elsner B. Chronic monoarthritis due to lipoma arborescens. *J Rheumatol*, 1990, 17: 861–862.
5. Venkatanarasimha N, Suresh SP. AJR teaching file: an uncommon cause of knee swelling. *AJR Am J Roentgenol*, 2009, 193 (3 Suppl): S53–S55.
6. Aydin G, Keles I, Tosun A, *et al.* Lipoma arborescens in childhood: a report of two sisters. *Turk J Rheumatol*, 2012, 27: 56–62.
7. Al-Hsmail K, Torreggiani WC, Al-Sheikh F, Keogh C, Munk PL. Bilateral lipoma arborescens associated with early osteoarthritis. *Eur Radiol*, 2002, 12: 2799–2802.
8. Coll JP, Ragsdale BD, Chow B, Daughters TC. Best cases from the AFIP: lipoma arborescens of the knees in a patient with rheumatoid arthritis. *Radiographics*, 2011, 31: 333–337.
9. Cil A, Atay OA, Aydingöz U, Tetik O, Gedikoğlu G, Doral MN. Bilateral lipoma arborescens of the knee in a child: a case report. *Knee Surg Sports Traumatol Arthrosc*, 2005, 13: 463–467.
10. Santiago M, Passos AS, Medeiros AF, Sá D, Correia Silva TM, Fernandes JL. Polyarticular lipoma arborescens with inflammatory synovitis. *J Clin Rheumatol*, 2009, 15: 306–308.
11. Hoffman EB, Allin J, Campbell JA, Leisegang FM. Tuberculosis of the knee. *Clin Orthop Relat Res*, 2002, 398: 100–106.
12. Yan CH, Wong JW, Yip DK. Bilateral knee lipoma arborescens: a case report. *J Orthop Surg (Hong Kong)*, 2008, 16: 107–110.