Extraskeletal ossifying chondroma in Hoffa's fat pad: an unusual cause of anterior knee pain

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ABSTRACT

Hoffa's disease is an obscure cause of anterior knee pain. A misconception about its rarity is very common among clinicians, and hence it is often misdiagnosed and treated as meniscal pathology. Increased awareness is required to diagnose and treat the condition appropriately. These diagnostic uncertainties commonly result in increased patient morbidity and mismanagement. In spite of a widely-accepted common occurrence of Hoffa's disease, ossification of the Hoffa's fat pad is seldom reported. We report a giant extraskeletal ossifying chondroma in a 55-year-old man, presented as chronic knee pain and successfully treated by excision. The anatomy, pathology, histology, radiological features and management of the disease are described, to increase awareness in the orthopaedic community of this common, interesting but rarely discussed condition.

Keywords: chondroma, extraskeletal chondroma, Hoffa's disease, infrapatellar fat pad

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INTRODUCTION

Hoffa's disease is a clinical condition characterised by infrapatellar anterior knee pain secondary to inflammation and impingement of the Hoffa's fat pad (HFP). It was first described by Albert Hoffa in 1904 when he reported 21 patients affected with this clinical condition.[1] In spite of being a common cause of anterior knee pain, only few case reports, clinical observations and case series have been described in literature, showing the gross underestimation of its incidence.[2,3] Its precarious location makes HFP vulnerable to be affected by a range of pathological insults. The present case illustrates a good example where increased wareness can facilitate a timely intervention.

CASE REPORT

A 55-year-old male builder presented with a history of progressively worsening knee pain for one year. The pain was especially severe while climbing stairs and lifting heavy weights. He injured his knee the year before, when he fell on a concrete floor. After primary management in casualty, he was discharged on analgesics. In spite of initial transient improvement, he continued to have knee pain off and on. The frequent bouts of incapacitating knee pain forced him to seek repeated consultations. The general practitioner finally referred him to an orthopaedic clinic for specialist advice. On examination, the left knee revealed moderate infrapatellar swelling with tenderness, atrophy of quadriceps and anterior compartment crepitus. He had painful, restricted range of movement from 0 to 90 degrees. There was no laxity of collaterals with normal McMurray's, Lachmann's and drawer tests. Patellar tracking was normal. Radiographs of the knee revealed calcification in the infrapatellar fat pad (Fig. 1). The rest of the bony architecture was unremarkable. The

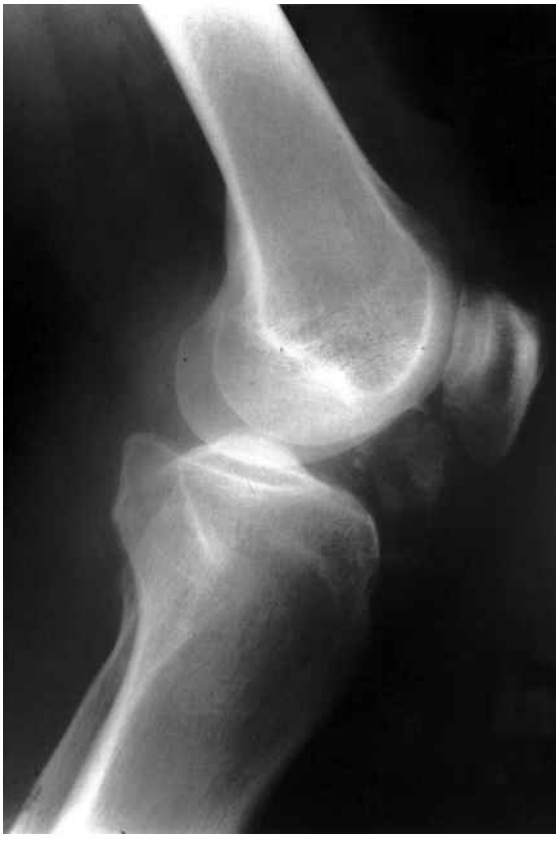

Fig. 1 Lateral radiograph of the knee shows calcification in Hoffa's fat pad.

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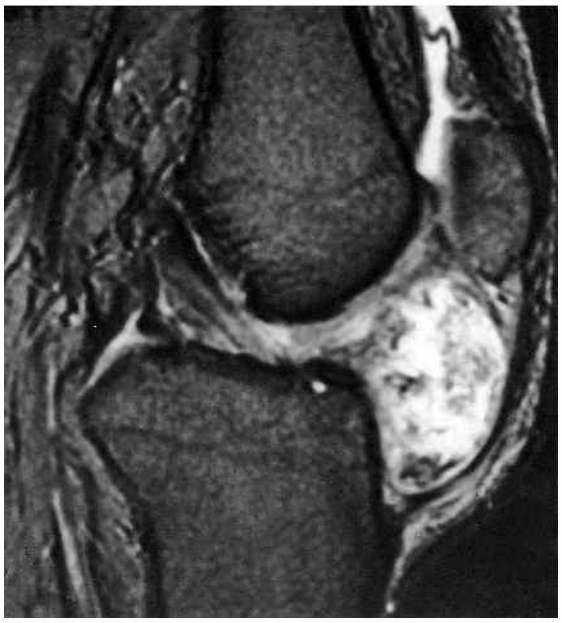

Fig. 2 Sagittal T2-W MR image shows a heterogeneous mass consistent with extraskeletal chondroma within the Hoffa's fat pad.

haematological profile was within physiological limits with normal inflammatory markers. Due to the presence of pathological calcification, a provisional diagnosis of Hoffa's disease was made, and magnetic resonance (MR) imaging was done to assess the lesion in detail. MR imaging suggested a large infrapatellar soft tissue mass containing chondroid calcification with erosion of inferior pole of patella (Fig. 2). Fine-needle aspiration was done prior to surgical intervention, which did not show any evidence of malignancy.

Arthroscopy of the knee revealed grade 2 arthritic changes throughout the joint. Biopsy taken at that time proved traumatic necrosis of the HFP and calcification with no evidence of malignancy. Histology delineated a well-circumscribed lesion composed of lobular proliferation of cartilage, lamellar bone and vascular fibrous connective tissue. An open excision of HFP was done as a second procedure by anterior midline approach, as the calcified mass was not amenable to arthroscopic excision. The excision HFP showed a very large area (60 mm × 45 mm × 50 mm) of totally calcified HFP, which was well encapsulated. The cut surface had a partly cartilaginous and partly bony appearance. Postoperatively, the patient was kept weight bearing as tolerated and was referred to physiotherapy. His symptoms resolved completely in four weeks. At the three-year follow-up, the patient remained completely asymptomatic with no evidence of recalcification.

DISCUSSION

The knee joint has many well-defined fat pads which are intracapsular but are extrasynovial in location.

Table I. Pathological spectrum of Hoffa's disease.

Intrinsic
Hoffa's disease
Intracapsular chondroma
Localised nodular synovitis
Post-arthroscopy / post-surgery fibrosis
Shear injury
Extrinsic
Intra-articular
Joint effusion
Intra-articular bodies
Meniscal cyst
Ganglion cyst
Cyclops lesions
Synovial
Pigmented villonodular synovitis
Synovial haemangioma
Primary synovial chondromatosis
Chondrosarcoma
Rheumatoid, seronegative arthritis
Synovitis secondary to osteoarthritis
Extracapsular
Patellar fracture
Patellar tendon rupture
Sinding-Larsen-Johansson disease
Deep infrapatellar bursitis
Osgood-Schlatter disease

Three anterior knee fat pads are anterior suprapatellar (quadriceps), posterior suprapatellar (prefemoral) and infrapatellar (Hoffa).[3] Albert Hoffa first described Hoffa's disease as characterised by anterior knee pain due to inflammation of the infrapatellar fat pad secondary to impingement. His classical description was a stable knee with pain over the anteromedial side of the joint.[1] HFP is pyramidal in shape and is surrounded by the patella superiorly, tibia and infrapatellar bursa inferiorly, patellar ligament and capsule at front, and synovium of the knee joint posteriorly. It is stabilised by its attachment superiorly to the intercondylar notch with plica ligamentum mucosum, and inferiorly to the anterior horn of the meniscus and periosteum of tibia. Lateral margins of HFP are known as alar plicae that protrude in the joint and project posteriorly along the anterior horn of the meniscus.[2,3]

HFP, because of its intracapsular and extrasynovial location, is affected by a wide spectrum of disorders (Table I). Pathological involvement of HFP could be due to intrinsic or extrinsic factors.[3] Intrinsic causes are due to the primary pathology in HFP. Extrinsic causes constitute involvement of HFP by intra-articular or synovial pathologies.[1] Hoffa's disease occurs in normal knees while Hoffa's syndrome is associated with hypertrophy of HFP due to meniscal, ligamentous or capsular lesions.[2] Hoffa's disease has two clinical phases and can be classified as acute and chronic. Acute cases are post-traumatic and are characterised by non-specific

symptoms such as pain, swelling, bruising, and functional impairment with flexion deformity of the knee.[1,2] In the chronic phase, symptoms are infrapatellar discomfort or pain which is exacerbated when going up and down stairs and lifting heavy weights. Movements are usually well preserved. It may occasionally be associated with patellar crepitus and with signs of instability.[2,4] Hoffa's test is a useful aid in diagnosis. In this test, the examiner takes up the flexed knee and presses the thumbs of both hands deeply along the sides of the patellar tendon just below the patella. A positive sign elicits a sharp pain at the terminal extension while extending the flexed knee.[1-3]

Direct trauma to the knee is the most common cause of Hoffa's disease and constitutes 85% of cases. In the rest of the patients, repetitive microtrauma to HFP is the causative factor.[2] This explains an increased prevalence in patients involved in sporting activities such as basketball, volleyball and jumping.[2] Ligamentous laxity leading to genu recurvatum predisposes a patient to develop Hoffa's disease. It is shown to be more common in females than males.[2] Hoffa's disease has a predilection for young patients, especially in their twenties.[2] Initial pathogenesis described by Hoffa was attributed to inflammation hypertrophy and fibrosis of the infrapatellar fat pad, which impinged between the tibia and femur during extension.[1] This hypothesis is also supported by recent arthroscopic examination by some authors.[2,4] Cadaveric studies support this theory but also implicate repetitive hyperextension, genu recurvatum and rotational sprains as contributing factors.[2,5-7] Repetitive microtrauma leads to metabolic changes in the lipocytes resulting in alteration in properties of cell membrane and release of vasoactive substances, including histamine, serotonin and quinine. These chemicals sensitise the synovial membrane and neighbouring fat tissues, causing necrosis of lipocytes.[5,8] This mechanism sustains the cycle of the pathological changes, making the disease chronic.[2,5,8] Wickham et al illustrated the derivation of multipotent stem cells from HFP.[9] They also demonstrated the ability of these cells to differentiate into chondrocytes and osteoblasts under appropriate culture conditions. The formation of extraskeletal ossifying chondroma is a result of metaplasia in capsule or adjacent connective tissues, which leads to the deposition of osteoid material in HFP.[1-3] Deposition of primitive haematopoietic cells from the blood stream has also been suggested.[10]

A high index of suspicion, awareness and appropriate imaging are imperative in the early diagnosis of Hoffa's disease. Radiographs might not show any changes, but calcification in HFP may be seen if present, as in our case. If there is clinical suspicion, arthroscopy or MR imaging must be performed in order to identify the condition. Arthroscopy may show an enlarged HFP with a harder consistency. HFP may also develop adhesions with ligamentum mucosum, meniscus and tibial plateau.[2] T2-weighted MR images may show an increased signal due to the presence of oedema and haemorrhage in HFP in the acute stage.[3] Bowing of the tendon may be seen due to mass affect. A small effusion may also be seen in the joint. Both T1- and T2-weighted MR images may show low signals in the subacute and chronic phases due to the presence of fibrin and haemosiderin.[3] Ossification may also have a low signal on MR imaging, and hence radiographical findings are important for differentiation.[3] T2-weighted MR images of the intracapsular chondroma may show a heterogeneous mass within the fat pad. Chondroid matrix and oedema represent a high signal while calcification or ossification represents a low signal.[3] Arthroscopy can also be done as it has both diagnostic and therapeutic value.[2,11] Initially, there are foci of haemorrhage with subsynovial vascularisation on histology. Later there may be evidence of deposition of fibrin and haemosiderin. Mononuclear cells and macrophages may transform into giant cells as part of the immune response. Hyperplasia of synovial membrane is also seen occasionally.[2] Degeneration of lipocytes is also evident with fibroblastic proliferation, which may lead to deposition of collagen. These organise to form fibrous tissue which may transform into fibrocartilaginous tissue that may rarely ossify, as in our case.[2]

Symptomatic treatment is given in the acute phase of any soft tissue or ligamentous injury in the form of rest, ice, elevation, painkillers and non-steroid anti-inflammatory agents. Splints may be used for immobilisation if the patient has significant pain.[2] In the chronic phase, lifestyle modification and physiotherapy are useful. Heavy lifting, excessive walking and stairs must be avoided as much as possible. Physiotherapy to strengthen the quadriceps muscle, especially vastus medialis, helps to relieve the symptoms.[2] Extra-articular steroid injection may also be given in order to alleviate the symptoms. Chronic stage with persistent symptoms or failure to conservative trail may require arthroscopic or open resection of the hypertrophic fat pad.[2] Resection includes excision of hypertrophic and sclerotic borders of HFP. Standard anterolateral arthroscopic portals do not visualise HFP adequately, and portals might have to be readjusted.[11] Open excision may be required, as in our case, with associated ossification or calcification when it is not possible to excise the mass by arthroscopy. In conclusion, Hoffa's disease is very common but rarely diagnosed. It may mimic meniscal pathology. Lack of awareness may lead to incorrect or late diagnosis. Increased awareness of this interesting clinical condition is necessary for early diagnosis and institution of appropriate management in order to avoid unnecessary morbidity.

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