ABSTRACT

Medial tibial plateau osteonecrosis is a disease that lacks distinguishing signs and symptoms, especially in the early stage, and requires clinicians to exercise a high degree of suspicion to prevent disease progression. We present a case of spontaneous osteonecrosis of the medial tibial plateau in a 59-year-old woman. Within 5 months of the onset of symptoms, the entire medial tibial plateau collapsed down into the metaphyseal region, causing severe varus deformity, instability, and inability to walk without crutches and a hinged knee brace. Initial symptoms of medial joint line pain and generalised swelling of the knee were attributed to early degenerative changes. Subsequent radiographs were misinterpreted as simple medial compartment arthritis. Due to severe bone loss and significant lateral collateral ligament attenuation, a total joint arthroplasty was required, using a stemmed tibial component with medial metal block and bone graft augmentation. The speed of bone collapse and the extension into the metaphysis, requiring complex joint arthroplasty, makes this case unique.

Key words: diabetes mellitus, type 1; knee; osteonecrosis

CASE REPORT

In July 2003, a 59-year-old wheelchair-bound woman presented to the Droitwich Knee Clinic in the United Kingdom with subtotal collapse of the medial tibial plateau (MTP) and condyle. Her symptoms had started insidiously about 5 months earlier and she had suffered progressive anteromedial knee discomfort, accompanied by swelling and increasing varus deformity. Two months after the onset of discomfort, she had developed disabling pain, losing her ability to bear weight without the use of walking aids. Pain and deformity further increased over the following 3 months.
months, leaving her wheelchair bound. She appeared generally fit and well apart from a 38-year history of well-controlled insulin-dependent diabetes. There was no indication of any sensory deficit in and around the knee, despite a mild degree of distal peripheral neuropathy limited to the sole of the foot.

The patient was treated initially by a general practitioner with non-steroidal anti-inflammatory drugs. Progressive swelling of her lower leg suggested a deep vein thrombosis but this proved negative on ultrasound investigation. Radiographs obtained 3 months after onset of symptoms were interpreted as early osteoarthritic changes (Fig. 1). Increased density of the subchondral plate of the MTP, representing early signs of osteonecrosis, were overlooked. Clinically the patient was unable to fully extend the leg and to squat. The knee showed a grade-2 effusion and a 25° varus deformity which increased to 30° on weight bearing (Fig. 2). The deformity was passively correctible to 10°. The lateral collateral ligament presented significant laxity but was difficult to assess due to the medial collapse (Fig. 3).

Figure 1  Posteroanterior radiograph of the left knee in 45° flexion taken 6 weeks after the onset of symptoms, showing signs of subchondral sclerosis and generalised increased density across the medial tibial plateau. Moderate medial joint space narrowing is visible.

Figure 2  The left knee has developed a 25° varus deformity 5.5 months after onset of symptoms, which increases to 30° on weight bearing.

Figure 3  Anteroposterior radiograph of the left knee taken 5.5 months after the onset of symptoms showing total collapse of the medial tibial plateau and condyle into the metaphysical area. Margins of the defect appear sclerotic. Significant varus mal-alignment and lateral joint opening is demonstrated.
Radiographs revealed total collapse of the MTP and medial condyle with extension into the metaphyseal region (Fig. 3). Magnetic resonance imaging (MRI) also highlighted sclerotic margins and surrounding bone marrow oedema (Fig. 4). The knee was aspirated and yielded blood-stained synovial fluid that did not grow any organisms. A bone scan demonstrated increased focal uptake in the medial femoral condyle and part of the proximal tibia (Fig. 5). Neurological examination of the knee and lower leg did not reveal any deficit or impairment, apart from a minor degree of hypoaesthesia affecting the sole of the foot.

No further bone loss was noted during 6 weeks of observation while the knee was kept in a stabilising brace. It was assumed that the process of osteonecrosis had come to a standstill or ‘burned out’. The patient underwent a posterior-stabilised left total knee arthroplasty with a stemmed revision prosthesis. To re-create a physiological joint line, the medial tibial component had to be built up by using a 10 mm block augmented with a bone graft obtained from the femoral condyles. At postoperative 6-month follow-up, the patient had a range of movement of 0° to 110° flexion and no radiological signs of further bone collapse (Figs. 6 and 7). At 36-month follow-up, her range of movement measured -5° to 130°, equalling the opposite side (Fig. 8). She was pain free and presented a normal walking pattern. Her only limitation was inability to kneel comfortably. Radiographs confirmed bone graft integration under the medial tibial plateau and no radiological signs of loosening.

**DISCUSSION**

The natural history of MTP osteonecrosis varies
from complete reconstitution to progressive joint degeneration. Extensive collapse of the tibial plateau is rare; complete collapse of the MTP with metaphyseal extension, as described in the present case, has not been reported in the literature.

In the early stages of the disease, non-operative treatment, including brace application and restrictions in weight bearing, is generally indicated and most cases follow a benign course. Complete or partial resolution of symptoms usually occurs within 12 months. Surgical options include arthroscopic Pridie drilling, debridement, and in more advanced cases unicompartmental or total knee arthroplasty.

Patients typically present with an insidious onset of anteromedial knee discomfort, which is progressive and often independent of load bearing. The aetiology remains unclear; proposed causes include a vascular ischaemic event, intra-osseous hypertension, or localised microfracture. The latter is believed to be the most likely mechanism, based on the fact that affected patients are mostly elderly and osteoporotic. Minor trauma may cause a microfracture with a breach in the subchondral plate. Fluid subsequently enters the narrow space, resulting in an increase in intra-osseous pressure followed by ischaemia and pain. MRI scans in these patients classically show an increased signal on T2-weighted images indicative of subchondral bone oedema. Coagulation disorders may be present and are believed to be associated with osteonecrosis.

The diagnosis is easily missed in the early stages as plain radiographs are often normal apart from minor pre-existing degenerative changes (Fig. 1). MRI is more sensitive and specific than radiography, and capable of depicting bone marrow oedema, which is pathognomonic to osteonecrosis, in the early stages of the disease process. T1-weighted images usually show a hypo-intense signal whilst the signal on T2-weighted or fast scan images is often hyper-intense. A fat suppression image sequence (STIR) may be helpful to exclude neoplasms and to determine the extent of disease involvement. Nonetheless, the MRI may be normal in the early course of the disease, with false negative results being reported in up to 20% of cases.

Based on MRI findings, tibial osteonecrosis has been classified into 4 types. Type A lesions show low signals on T1-weighted MRIs and are small and well localised. Type B lesions are similar to type A lesions but are more diffuse and extend down into the tibial metaphysis. Type C lesions represent true osteonecrosis of the bone and are characterised by a well-circumscribed low signal surrounded by a rim of reactive bone. Type D lesions are more extensive,
are associated with subchondral collapse and can also be seen on plain radiographs.

In symptomatic patients with no conclusive radiographic or MRI findings, a radionuclide bone scan should be considered. This is a highly sensitive tool demonstrating focal uptake over the affected area even before changes are apparent on other imaging modalities.4,9

There are limited reports in the literature regarding MTP osteonecrosis. Conservative management generally yields a high success rate but complete resolution of symptoms is usually not observed before 9 to 12 months.1 Satku et al.2 reported 3 distinct outcome patterns in 18 cases. 12 showed rapid progression to varying degrees of osteoarthritis within 2 years. Complete resolution was noted in 4 patients, and 2 suffered partial collapse of the medial tibial plateau.

Osteonecrosis has recently been linked with insulin-dependent diabetes mellitus, after a patient with severe peripheral neuropathy developed pain-free osteonecrosis and Charcot joint arthropathy.5 In the present case, however, no sensory impairment was noted around the knee and pain was the predominant presenting feature making the diagnosis of Charcot’s disease unlikely.

Total knee arthroplasty has been reported to have a favourable outcome in patients presenting with extensive bone collapse due to osteonecrosis, with 97% of patients enjoying a successful clinical outcome 9 years following arthroplasty.10 Cemented implants with or without stem augmentation provide reliable long-term fixation and represent the treatment of choice in most cases.4,10,11 Unicompartment knee arthroplasty prostheses may be indicated in cases where the lesion is small and limited to the medial compartment.4,12

CONCLUSION

Osteonecrosis of the MTP is a rare condition usually seen in women in their late 50s.9,11,13 A high index of suspicion and careful examination of the patient’s history and physical condition along with imaging studies are essential to make an accurate diagnosis and to instigate appropriate treatment.

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