CASE REPORT

Intraosseous Lipoma: Report of 2 Cases

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ABSTRACT

Intraosseous lipoma is a rare benign bone tumour. This report presents 2 cases of intraosseous lipoma: 1 occurring in the right intertrochanteric region of a 67-year-old man and 1 at the left distal fibula of a 72-year-old woman. For the first patient, the computed tomogram showed a radiolucent intramedullary lesion with a slightly lobulated sclerotic margin and homogeneous fat density; the T1-weighted magnetic resonance image showed hyperintensity in the area of the lesion, which could be fully subtracted in the fat-saturated sequence. This patient was followed up regularly and has shown no evidence of malignant change. For the second patient, the X-ray showed pathological fractures at the distal fibula and tibia; computed tomography revealed an intramedullary, slightly expansile, osteolytic lesion at the distal end of fibula, together with endosteal scalloping and a dense tumour matrix; and histological examination revealed that this lesion consisted of necrotic adipose tissue and extensive calcification. The lipoma was resected and has not recurred. Because the overall prognosis of intraosseous lipoma is good, we suggest that once the diagnosis is established by imaging or histological studies, conservative treatment with regular follow-up scanning should be considered for asymptomatic cases.

Key Words: Bone neoplasms; Diagnosis, differential; Lipoma/radiography

INTRODUCTION

Intraosseous lipoma is a rare benign bone tumour. It consists of mature adipose tissue containing atrophic bone trabeculae. The appearance of this neoplasm on radiographs can vary depending on the degree of involution and necrosis. Computed tomography (CT) and magnetic resonance imaging (MRI) yield more definitive results than X-ray imaging. Histological examination is helpful in the diagnosis of difficult cases.

CASE REPORTS

Case 1

A 67-year-old man presented to the Department of Radiology and Imaging at the Queen Elizabeth Hospital in February 2002 because of a dull ache at the region of his right hip. He had a history of chronic gouty arthritis involving the left ankle joint and the first metatarsal-phalangeal joint of the right foot. Serial X-rays showed an approximately 3- x 4-cm radiolucent lesion with a sclerotic border at the right intertrochanteric region (Figure 1). The CT scan showed a radiolucent intramedullary lesion with a slightly lobulated sclerotic margin and homogeneous fat density; there was no obvious internal calcification or ossification (Figure 2). The coronal T1-weighted MRI scan showed hyperintensity in the area of the lesion, which could be fully subtracted in the fat-saturated sequence after gadolinium injection. No soft-tissue component, bony destruction, or contrast enhancement was visible (Figure 3). The tumour was not removed because it was benign and was reported to cause only occasional bother. The patient was followed up regularly for 2 years after the discovery of the lipoma and complained of occasional pain around the right hip.

Case 2

A 72-year-old woman presented to the Department of Radiology and Imaging at the Queen Elizabeth Hospital in September 1999 for a 5-year history of pain in her left ankle. The X-ray of her left ankle showed pathological fractures at the distal fibula and tibia.
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The CT scan showed an intramedullary, slightly expansile, osteolytic lesion at the distal end of the fibula. Endosteal scalloping could also be seen. There was a dense tumour matrix, which was suggestive of either ossification or calcification. Another well-defined cortex-based lesion with a sclerotic border and calcific matrix could be seen at the lateral aspect of the distal tibia. These features were suggestive of slow-growing or benign lesions at the distal fibula and tibia. Differential diagnoses were ossifying fibroma or cartilage-forming tumour. Pathological fractures were noted across both lesions (Figure 5). Curettage and allograft transplantation were performed. Histological examination revealed that the lesion in the distal fibula consisted of necrotic adipose tissue and extensive calcification (Figure 6) — findings consistent with the diagnosis of lipoma. The material obtained from the tibial lesion, however, was non-diagnostic. Follow-up MRI performed 1 year after the operation showed no tumour recurrence.
DISCUSSION

Intraosseous lipoma was first described in 1880. The tumour is composed of mature fat cells and varying amounts of fibrous and vascular tissues. Although intraosseous lipoma constitutes approximately 0.1% of bone tumours, its real prevalence may be higher because its usual asymptomatic nature results in the misdiagnosis of some cases as bone infarction or pseudotumour at the anterior os calcis. With the advent of MRI and CT, however, an increasing number of cases of intraosseous lipoma are being recognised and diagnosed.

The largest reported series of intraosseous lipomas was by Milgram. Campbell et al conducted a meta-analysis of a total of 206 cases, including those in Milgram’s study. The meta-analysis showed that the mean age at presentation was about 43 years; both of the patients in our cases were older than this age. The sex distribution of the cases reviewed was nearly equal. Both the axial and the appendicular skeleton were affected; the most frequent sites were the lower limbs (71%) — particularly the os calcis (32%), the femur (20%) and the fibula (6%). The tumours in our cases were in the femoral neck and the distal fibula.

Patients are frequently asymptomatic and their lesions are discovered incidentally. Some patients may present with pain, as did the patient in our first case. Some may present with swelling or even a pathological fracture, as did the patient in our second case.

Figure 4. Radiograph of the left ankle of patient 2 showing pathological fractures at the distal fibula and tibia.

Figure 5. Axial computed tomogram of the distal tibia and fibula of patient 2 showing an intramedullary osteolytic lesion at the distal end of the fibula that had internal calcification or ossification, and a well-defined cortex-based lesion with a sclerotic border and calcific matrix at the lateral aspect of the distal tibia; the fracture line extended across both lesions.

Figure 6. Photomicrograph of tissue from the lesion from the distal fibula of patient 2 showing necrotic adipose tissue and extensive calcification (haematoxylin and eosin; original magnification, x10).
Milgram has proposed 3 stages of intraosseous lipoma. Stage 1 consists of radiolucent expansile lesions, viable lipocytes on histological examination, and some fine bone trabeculae. Stage 2 lesions are transitional cases that exhibit partial fat necrosis and focal calcification, but also have regions of viable lipocytes. Stage 2 lesions are radiographically similar to stage 1 lesions, but they frequently have areas of increased density because of central calcification and ossification. Radiographs of stage 3 lesions show a reactive ossified rim, some central cysts, and calcification. Furthermore, complete fat necrosis is visible during histological examination. Therefore, each stage shows radiographical features that can be correlated with the histopathological findings and that vary with the degree of involution and necrosis.

For difficult cases, CT and MRI can be helpful. In CT scans, the lesion displays fat attenuation without a soft tissue component. In MRI studies, the lesion is isointense to the subcutaneous fat on T1-weighted images and shows low signal intensity and fat suppression on T2-weighted images. A thin circumferential rim of low signal intensity is visible in T1- and T2-weighted images because of reactive sclerosis surrounding the lesion. Low signal intensity areas within the central portion of lesion on T1- and T2-weighted images are consistent with calcification. Areas of fat necrosis have a variable signal on T1-weighted scans and increased signal on T2-weighted ones.

The differential diagnosis varies with different stages of disease. For stage 1 lesions, any expansile lesion can resemble intraosseous lipoma radiographically. For stage 2 and 3 lesions, the differential diagnosis includes bone infarction, enchondroma, chondromyxoid fibroma, and fibrous dysplasia. Distinction between intraosseous lipoma and bone infarction is difficult, but lipoma shows expanded contours of bone, resorption of pre-existing bone, a wider peripheral sclerosis, and calcification and ossification throughout the lesion. In contrast, calcification and ossification in a bone infarct are seen at the periphery.

According to Milgram’s staging system, our first case was a stage 1 lesion and our second case was a stage 3 lesion. Milgram suggested that there was no preponderance of the involuted stage 2 and 3 lesions in elderly patients.

Once the diagnosis of intraosseous lipoma has been established by means of CT, MRI, or histological examination of a tissue specimen in suspicious cases, a conservative treatment protocol with regular follow-up scanning is suggested in asymptomatic patients. Surgical treatment with curettage and bone grafting in symptomatic cases has been proposed.

Malignant transformation or recurrence after surgery appears to be very rare. The recurrence rate is unknown. Milgram reported 4 cases of presumed malignant transformation in which lipoma developed into malignant fibrous histiocytoma or liposarcoma; 1 had been a stage 1 lesion and 3 had been stage 3 lesions. The cause of intraosseous lipoma itself is unknown. Risk factors and sex or site predilection have not been addressed in the literature. Because the overall prognosis of intraosseous lipoma is good, we suggest that once the diagnosis is established by imaging or histological studies, conservative treatment with regular follow-up scanning should be considered for asymptomatic cases. The 2 patients in our cases have been followed up regularly and have shown no evidence of malignant change (patient 1) or recurrence after resection (patient 2).

REFERENCES