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ORIGINAL PAPER

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Intraosseous lipoma: retrospective analysis of 29 patients

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Abstract We reviewed 29 patients with intraosseous lipoma treated between 1985 and 2002. Mean patient age was 48 (20–75) years. According to Milgram's classification, ten cases were classified as stage I, 14 as stage II, and three as stage III. All patients were initially treated by curettage. In 11 cases an additional phenolization was performed. The average follow-up was 32 (6–208) months. At the last follow-up, none had any clinical or radiological signs of recurrence. The adequate treatment of a symptomatic intraosseous lipoma is curettage and bone grafting. In the current study, phenolization showed no added benefit. An asymptomatic intraosseous lipoma without impending pathological fracture can be treated conservatively.

Résumé Nous avons examiné 29 malades avec un lipome intraosseux traité entre 1985 et 2002. L'âge moyen des malades était de 48 ans (20–75). D'après la classification de Milgram, dix cas ont été classés comme stade I, 14 comme stade II, et trois comme stade III. Tous les malades ont été traités initialement par curetage. Dans 11 cas une phénolisation supplémentaire a été faite. Le suivi moyen était de 32 mois (6–208). Àu dernier examen aucun n'avait de signe clinique ou radiologique de récidive. Le traitement adéquat d'un lipome intraosseux symptomatique est un curetage avec greffe osseuse. Dans cette étude, la phénolisation n'a montré aucun avantage supplémentaire. Un lipome intraosseux asymptomatique sans menaçe de fracture pathologique peut être traité d'une manière conservatrice.

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Introduction

The intraosseous lipoma is a benign bone lesion, and this tumor is said to be one of the rarest primary bone tumors [7, 24]. The etiology of intraosseous lipomas is discussed controversially [3]. Hypotheses range from a primary benign neoplasm [5, 19, 20, 24] to a reactive bone lesion following trauma or bone infarct [12]. In two publications hyperlipoproteinemia has been suggested to be a possible reason for the intraosseous lipoma [8, 11]. Another hypothesis is that a conglomeration of fatty marrow, as commonly seen in calcaneus or vertebral bodies, simply is the reason for the lesion [2]. The intraosseous lipoma can be clinically silent, and then the lesion may be found incidentally during radiological investigation following an injury in the same region [2, 20]. However, some intraosseous lipomas can be symptomatic [19, 20, 25]. The purpose of this retrospective, multicenter study was to evaluate our results of surgically treated patients with an intraosseous lipoma.

Material and methods

Between 1985 and 2002, 29 cases of intraosseous lipoma were diagnosed at three orthopaedic centers. The criterion for inclusion in this retrospective study was a histologically verified intraosseous lipoma. Demographic data, including age, bodyweight, and body-mass index (BMI),

were calculated. A serological testing of cholesterol and triglycerides was done before the operation in 15 patients. Preoperatively, patients were investigated using conventional radiographs in two planes of the affected region. Additional examination was done with computerized tomography (CT) in eight patients and magnetic resonance imaging (MRI) in 20. In ten patients the radiolucent lesions were located in the calcaneus, in ten patients in the tibia, in three patients in the femur, in the two patients in the metacarpal bones, in two patients in the humerus, in one patient in the ilium, and in one patient in the radius (Table 1). The operative treatment was curettage. The bone defect was filled with autogenous bone graft in 18 cases, with homogenous bone graft in five, with a mixture of both in four, and a synthetic bone substitute in one. An additional phenolization of the bone cavity was performed in 11 patients. Three patients had additional plating due to weakening of the bone architecture. In two asymptomatic patients, the radiological appearance was unclear.

Lesions were categorized according to the radiological and histological Milgram classification system [19, 20]. Stage I contains viable lipocytes and appears as a radiolucent lesion. Stage II has increased local radiographic density due to calcified fat; histologically, there are calcifications surrounded by viable lipocytes. Stage III has involution often combined with ossification around calcified necrotic fat. We studied the histologic features and reviewed the medical records. All patients were invited for a follow-up visit. The clinical investigation consisted of a physical examination including conventional radiographs in two planes.

Results

There were 15 female and 14 male patients. Mean age at the time of operation was 48 (20–75) years, mean body weight was 76 (57–108) kg, and mean BMI was 26 (23–33). Twenty-five patients complained of pain without preceding trauma, and three patients also had recurrent swelling. Symptoms preceded the diagnosis with a mean of 8 (1–24) months. In four patients a radiolucent lesion was found incidentally. Tumor sites and patient characteristics are listed in Table 1. In 15 patients serum cholesterol was on average 266 (196–565) mg/dl and serum triglycerides 240 (58–783) mg/dl before surgery. In 11 patients either one or both parameters were elevated. None of the patients received cholesterol-lowering agents. After

Table 1 Demographic data of the 29 cases of intraosseous lipoma from the current study. *M* male, *F* female, *MD* missing data

Patient	Age (years)	Gender	Location	Clinical appearance	Surgery	Follow-up (months)
1	73	M	Calcaneus	Incidental	Curettage, bone graft,	8
2	50	M	Calcaneus	Pain	Curettage, bone graft, phenol	36
3	20	M	Calcaneus	Incidental	Curettage, bone graft, phenol	48
4	51	F	Calcaneus	Pain, tenderness	Curettage, bone graft, phenol	28
5	49	F	Calcaneus	Pain	Curettage, bone substitute	11
6	43	F	Calcaneus	Pain	Curettage, bone graft	21
7	33	M	Calcaneus	Pain	Curettage, bone graft	22
8	60	M	Calcaneus	Pain	Curettage, bone graft	6
9	37	Ff	Calcaneus	Pain	Curettage, bone graft	6
10	30	M	Calcaneus	Pain	Curettage, bone graft	12
11	29	M	Tibia	Incidental	Curttage, bone graft, phenol, plate	37
12	47	F	Tibia	Pain	Curettage, bone graft, phenol	9
13	52	F	Tibia	Incidental	Curettage, bone graft	md
14	39	M	Tibia	Pain, swelling	Curettage, bone graft	23
15	30	F	Tibia	Pain	Curettage, bone graft, plate	56
16	45	F	Tibia	Pain	Curettage, bone graft	28
17	60	F	Tibia	Pain	Curettage, bone graft	16
18	53	F	Tibia	Pain	Curettage, bone graft, phenol	75
19	37	M	Tibia	Pain	Curettage, bone graft	14
20	21	F	Tibia	Pain	Curettage, bone graft	26
21	55	M	Femur	Pain	Curettage, bone graft, phenol	80
22	50	M	Femur	Pain	Curettage, bone graft	12
23	61	F	Femur	Pain	Curettage, bone graft, plate	6
24	59	F	Metacarpale	Pain, swelling	Curettage, bone graft, phenol	MD
25	26	M	Metacarpale	Pain, swelling	Curettage, bone graft	6
26	63	F	Humerus	Pain	Curettage, bone graft, phenol	23
27	72	M	Humerus	Pain	Curettage, bone graft, phenol	12
28	58	M	Radius	Pain	Curettage, bone graft, phenol	36
29	74	F	Ilium	Pain	Curettage	208

Fig. 1 **a** Radiograph of the right femur of a 61-year-old woman (patient 23) with a radiolucent lesion with a well-defined margin. **b** MRI appearance of the intraosseous lipoma in the femur shows a hyperintensity. **c** Histology showed mature fat cells of a lipoma without any signs of calcifications (original magnification ×40). Both radiograph and histology corresponds to a stage I intraosseous lipoma according to the Milgram classification.

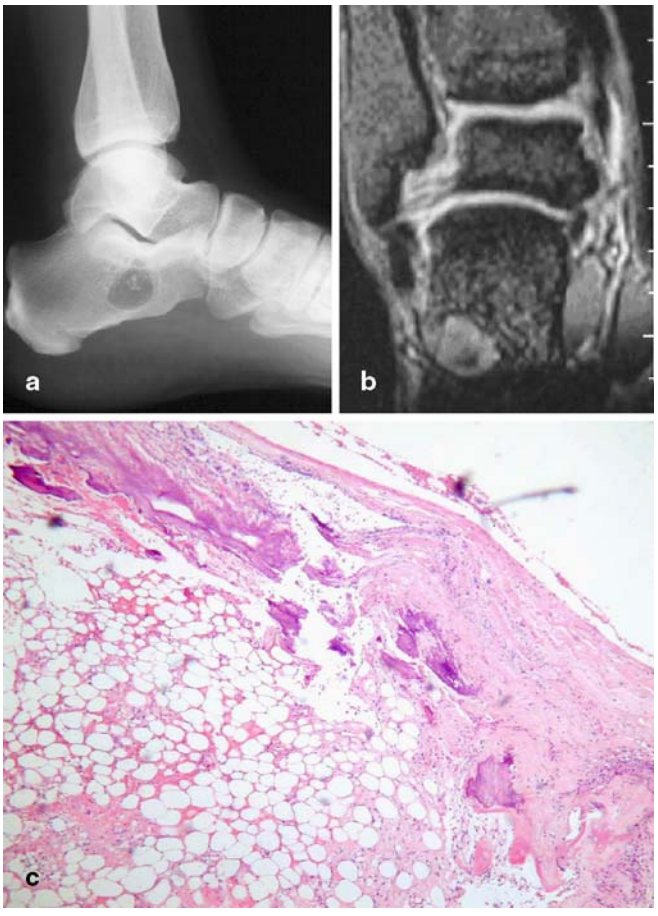

Fig. 2 **a** Lateral radiograph of the left calcaneus of a 43-year-old female patient (patient 6) with a radiolucent lesion with slight central calcifications. **b** MRI revealed a hyperintense lesion surrounded by thin hypointense areas and a central hypodensity corresponding with the calcification on the radiograph. **c** Histology showed an intraosseous lipoma with sclerotic fatty tissue, local fibrosis, and incipient signs of calcifications (original magnification ×40), which corresponds to a stage II intraosseous lipoma.

a mean observation period of 32 (6–208) months, there was no tumor recurrence. Two patients were lost to follow-up.

Histologically all cases were diagnosed as intraosseous lipoma. In two patients the preoperative X-rays were not available. In 27 cases the tumor was staged according to Milgram [19, 20]. There were ten stage I (Fig. 1), 14 stage II (Fig. 2), and three stage III lesions (Fig. 3).

Discussion

The intraosseous lipoma is a benign bone tumor, which proliferates from mature lipocytes [7]. Although lipoma and liposarcoma are very common, the intraosseous lipoma presents one of the rarest bone tumors [24]. Some authors stated that intraosseous lipomas are more frequent because often there are no symptoms and the tumor remains latent. However, with increasing use of MRI and CT evaluations, intraosseous lipomas will be more accurately diagnosed [7, 20, 24].

Some intraosseous lipomas are said to be difficult to diagnose radiologically [16]. Therefore, confusion with other bone lesions can occur [7]. Due to this possible misdiagnosis, we included only cases with histologically verified intraosseous lipoma in the analysis of the current study.

Milgram, who presented the largest series, found a slight male preponderance [19] while a very recent meta analysis showed a nearly even gender distribution [6]. Tumor frequently occurs around the fourth age decade, but intraosseous lipomas are reported in patients aged from 4 to 85 years [6, 19]. A very similar gender and age distribution as presented in the studies mentioned above has been found in the cases of the current report. Nearly every bone can be affected by the lesion although the

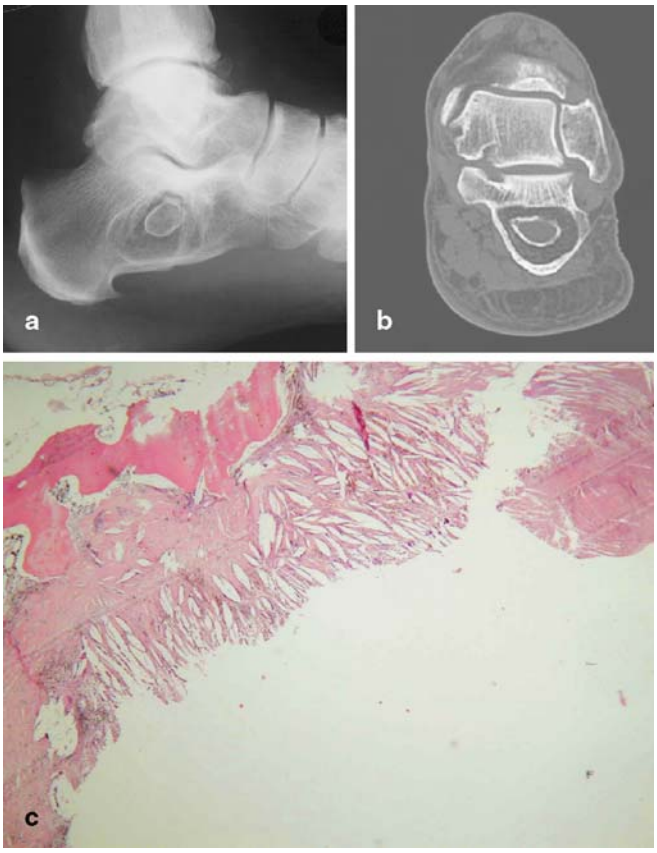

Fig. 3 **a** Lateral radiograph of the left calcaneus of a 73-year-old male patient (patient 1) with a radiolucent lesion and a central calcification. **b** CT examination revealed a well-circumscribed radiolucent lesion in the calcaneus surrounded by bone sclerosis. **c** Histology showed a massive ossification and calcification of necrotic fat (original magnification ×40). Both radiograph and histology correspond to a stageIII intraosseous lipoma

favored sites of the intraosseous lipoma are the calcaneus [22] and the metaphysis of long bones such as the femur, tibia, fibula, and humerus [6, 19]. The epiphysis or diaphysis of long bones, the ilium, sacrum, vertebral bodies, and skull bones are less frequent locations [6, 19].

About half of the patients with an intraosseous lipoma present no symptoms, and the tumor is found incidentally [19, 20]. However, in symptomatic patients, signs associated with the tumor are not specific [22]. Pain, swelling, and tenderness are the most frequent symptoms [19, 22]. Rarely, a pathologic fracture occurs [19, 25].

Chow et al. hypothesized that proliferating fat cells would cause pressure in the bony cavity followed by an involution with secondary changes in the lipoma and the surrounding bone [7]. However, the tumor also can remain as a stage I lesion [19, 20]. In some cases intraosseous lipomas appear to undergo a spontaneous involution and can perform as a benign, self-limiting tumor [20]. Based on his study, Milgram concluded that curettage is usually sufficient therapy for stage I and II lesions whereas the involuted intraosseous lipomas, which represent the stage III lesion, are treatable with observation alone [20]. Therefore, an accurate assessment of the degree of involution is of clinical relevance and underlines the need for the application of Milgram's classification system. The classification system should be applied in all future patients with intraosseous lipoma to achieve best comparability.

The intraosseous lipoma is characterized by involution of the tumor, which gives several different appearances to this lesion [7, 19, 20]. The differential diagnosis of the intraosseous lipoma includes mainly benign tumors and varies with the stage of the lesion [20]. Malign neoplasm also should be included in the differential diagnosis [15, 19–21]. In the calcaneus region, the most frequent differential diagnoses are the pseudocyst, which is formed by the major weight bearing bone trabeculae at the junction of the neck and body of the calcaneus (Ward's triangle) [3] and the bone infarct [4, 20]. Several other differential diagnosis are emphasized in the literature: fibrous dysplasia [20], osteoblastoma [20], enchondroma [12, 20], chondroblastoma [12], chondrosarcoma [12], solitary bone cysts [23], Brodie abscess [17], nonossifying fibroma [6], and giant cell tumor [6].

The majority of intraosseous lipomas present classic radiographic features, which can be suggestive of or even diagnostically relevant to this rare lesion. On plain radiographs the stage I intraosseous lipoma presents as a purely radiolucent lesion with a well-defined margin, which is formed by the remodelled bone around the slowly growing lesion [20]. The stage II lesion typically has some increased radiographic density due to calcification or ossification inside the lesion [20]. At stage III, the tumor's typical feature appears as a radiolucent bone lesion with reactive woven bone formation around the calcified fat combined with cystic regions [20].

The primary role of the MRI in identifying the intraosseous lipoma is to visualize fat within the lesion [4]. Magnetic resonance tomography shows high signal intensity on T1-weighted images similar to that of subcutaneous fat, and in areas of necrosis or cystic formations, high signals are demonstrated in T2-weighted images, and on the short inversion time inversion recovery images (STIR) a lack of signal [4, 16]. These imaging techniques should be applied to find the diagnosis and to reach a conclusion, as a biopsy and operative treatment can be avoided in most cases [4].

The need of a surgical treatment for intraosseous lipomas is discussed controversially [1, 6]. Supported by the current literature, the authors feel that in asymptomatic cases with no signs of impending fracture, a nonoperative treatment with clinical and radiographic follow-up is a wise approach [9, 14, 16, 18, 20]. However, diagnosis should include the degree of involution of the tumor [20]. Some authors even have stated that the intraosseous lipoma belongs to the "leave me alone" group of bony lesions and that any invasive method is unnecessary [17]. In symptomatic lesions and in cases with impending pathological fracture, an operative intervention should be the recommended treatment [14, 20]. Previously published studies of large series have shown that curettage and bone grafting is an adequate therapy in intraosseous lipomas

and that after the procedure, the tumor does not recur [20]. In contrast to the above-mentioned findings, recurrence of the tumor was reported in two cases [13, 23]. Furthermore, a malignant transformation of an intraosseous lipoma has been described in five cases [15, 21].

Unfortunately, the serological testing of our patients according to cholesterol and triglycerides was performed only in 15 of the 29 cases. Therefore, a possible pathogenetic association remains only speculation although this interesting finding was found in cases with multiple intraosseous lipomas [8, 11] as well as in multiple subcutaneous lipomas [10]. In order to obtain more data on this interesting issue, all future patients with this lesion should be screened with special regard to hyperlipoproteinemia.

There is a weak point in the data presented in this article. We have included only surgically treated patients in the analysis of the cases from the three orthopaedic centers. This has been done to enroll only histologically verified cases although this tumor has been suspected by radiological means in some other asymptomatic cases, which have been treated nonoperatively.

The results of our cases in conjunction with the literature support the notion that curettage and bone filling of a symptomatic intraosseous lipoma is an adequate therapy. Phenolization as an adjuvant for local tumor control did not show any benefit in the current study; therefore, it cannot be recommended in this type of tumor. In cases of a suspected intraosseous lipoma, the MRI is the investigation of choice, and if the diagnosis and classification of the tumor can be clearly achieved by radiological imaging and there is no danger of pathological fracture, a nonoperative treatment in asymptomatic cases is justified. Regular radiological follow-up should be performed in these cases.

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