S. Radke · J. Vispo-Seara · M. Walther · V. Ettl
J. Eulert

Transient bone marrow oedema of the foot

Accepted: 28 February 2001 / Published online: 13 June 2001
© Springer-Verlag 2001

Abstract We treated ten patients who on the basis of MRI were suspected to have transient bone marrow oedema. In eight cases the talus was affected, in one the cuboid and in one the navicular bone. All patients had acute onset pain at the ankle. Four were treated with core decompression and had an immediate pain relief. Six were treated conservatively and became also pain-free but with considerable delay.

Résumé Nous avons traité dix patients qui d'après l'examen IRM présentaient un oedème de la moelle osseuse transitoire. Tous avaient des douleurs de cheville à début aigu. Le talus a été en cause huit fois, le cubo une fois et l'os naviculaire une fois. Quatre ont été traités par forage avec soulagement immédiat de la douleur. Six ont été traités d'une manière conservatrice et sont aussi devenus indolores mais dans un délai beaucoup plus long.

Introduction

Transient bone marrow oedema syndrome (TBME) is an uncommon, self-limiting skeletal disease [12]. Curtiss [5] gave the first description in 1959. They referred to it as transient osteoporosis because of the osteopenic appearance of the affected bone on plain radiographs. As a result of the characteristic oedema-like signals as seen on magnetic resonance imaging (MRI) and the lack of osteoporosis in computer tomographic as well as histological studies, Wilson referred to this condition as "transient marrow oedema syndrome" [24].

S. Radke (✉) · J. Vispo-Seara · M. Walther · V. Ettl · J. Eulert
Department of Orthopaedic Surgery, Koenig-Ludwig Haus,
University of Würzburg, Würzburg, Germany
e-mail: s-radke.klh@mail.uni-wuerzburg.de
Tel.: +49-931-8031, Fax: +49-931-8031128

S. Radke
Orthopädische Klinik, Brettreichstrasse 11, 97074 Würzburg,
Germany

TBME usually affects one bone, mainly the proximal femur [2]. Reports describing an involvement of the foot, particularly the talus, are rare [4, 17]. Nevertheless TBME is an important differential diagnosis of foot and ankle pain of acute onset. The definitive diagnosis of TBME is made on MRI by excluding other causes of bone marrow oedema.

Based on histological studies, TBME of the hip is considered to be a disease of ischaemic origin and a reversible form of avascular necrosis (AVN) [8, 20]. Core decompression (CD) has therefore been used [13], shortening the natural course significantly. Later CD has also been used successfully in AVN of the talus [7]. It seems reasonable to apply CD in TBME of the foot as a therapeutic alternative to conservative measures.

The aim of this study was to report our experience with operative and non-operative treatment in TBME of the foot.

Materials and methods

Between January 1998 and June 1999 ten patients were treated for TBME of the foot (Table 1). TBME was diagnosed using MRI excluding other causes of bone marrow oedema (Table 2) in patients with acute onset of foot and ankle pain. The characteristics of a bone marrow oedema are a decrease in signals on T1w sequences

Table 1 Causes of bone marrow oedema

Ischaemic
Avascular necrosis
Reflex sympathetic dystrophy
Inflammatory
Osteomyelitis
Rheumatoid arthritis
Traumatic
Bone bruise
Stress fracture
Others
Neoplasm

Table 2 Patient list including details of treatment (A analgesic + protected weight bearing, CD core decompression)

Age (years)	Sex	Affected bone	Radiographic changes	Treatment	Histological examination	Time between onset of pain and start of treatment (months)	Time between start of treatment and CD (months)	Time between start of treatment and resolution of pain (months)
52	F	Talus	No radiolucency	CD	No necrosis	6	1	3
49	F	Talus	No radiolucency	CD	Partial necrosis	1	2	7
20	M	Cuboid	No radiolucency	CD	—	2	1	3
52	M	Talus	No radiolucency	CD	—	1	1	2
50	F	Navicular	Radiolucency	A	—	1	—	8
53	M	Talus	No radiolucency	A	—	1	—	3
52	M	Talus	No radiolucency	A	—	0	—	7
43	M	Talus	No radiolucency	A	—	1	—	5
51	M	Talus	Radiolucency	A	—	2	—	8
39	M	Talus	No radiolucency	A	—	0	—	9

and an increase in signals on T2w sequences (Fig. 1A,B). If contrast media is used, there is a subtle enhancement of the oedematous bone marrow (Fig. 2A,B).

For the purpose of excluding other causes of bone marrow oedema, all patients had a thorough clinical examination and the findings were rated according to the Weber ankle score modified by Bray. Laboratory tests [including white blood cell count, erythrocyte sedimentation rate (ESR), c-reactive protein, rheumatoid factor] and radiographs of the foot and ankle were done.

All patients with TBME were initially treated conservatively with an analgesic and protected weight bearing. Additionally all patients were given the possibility of CD as a further therapy option. All patients were followed up for 1 year.

Results

All patients had an acute onset of ankle pain. Five patients complained of constant ankle pain, five only of pain on exertion. The time of presentation after the onset of symptoms varied as seen in Table 2.

The Weber ankle score modified by Bray calculated before treatment was on average 52 (25–65) points. The radiographic changes are shown in Table 2. On MRI eight patients had bone marrow oedema of the talus, one of the navicular bone (Fig. 3) and one of the cuboid. Initially all our patients were treated symptomatically with an analgesic and limited weight bearing. Four patients chose to have CD after 1–2 months of conservative therapy. All of them had immediate pain relief after CD and limited weight bearing for 6–8 weeks. The average Weber ankle score was 93 points 2–6 months after CD. In two patients with TBME of the talus biopsies were taken during CD. One biopsy showed normal bone tissue. The other revealed partially vital and partially necrotic bone tissue. Clinically this patient was not completely pain-free until 6 months after CD at which time MRI still showed oedema of the talus. The other patient with biopsy had an MRI 3 months postoperatively (Fig. 4A,B). At the follow-up examination 1 year postoperatively the patients had hardly any pain and a significant improvement

Fig. 1 A T1w coronal section of the ankle joint. TBME in the medial part of the talus (arrow). B T2w coronal section in fat suppression. TBME in the medial part of the talus (arrow)

Fig. 2 A T1w sagittal section of the ankle joint: TBME marked by a hypointense area reaching from the dome of the talus to the neck of the talus (arrow). B T1w + i.v. contrast agent in sagittal section. In contrast to A, there is uptake in the area of TBME

in range of motion. Radiographically there was no sign of necrosis. Patients treated conservatively had a Weber ankle score of 95 points 3–9 months after treatment start. A decrease in range of motion was not observed.

Discussion

Transient bone marrow oedema is a rare cause of acute onset ankle pain. There is currently no data regarding the

Fig. 3 T1w coronal section of the forefoot. Hypointense area of the os naviculare indicating TBME (arrow)

incidence. In our clinic we see around 1200 patients per year with foot and ankle pain and we found ten patients within a 2-year period with TBME.

There have been few reports on regional migratory osteoporosis [18, 23], a special form of TBME involving the foot. TBME of the talus [4, 17] is rare. In our study there were however eight patients with TBME of the talus.

Patients with TBME of the hip typically complain of an acute onset of pain, present on activity, not at rest, a disproportional functional disability and an antalgic gait. This was also observed in this study and is in contrast to the course of TBME of the hip [22] with three phases, starting with rapid aggravation of pain and functional disability, lasting for approximately 1 month, followed by a phase where symptoms reach a plateau in intensity over 4 months and ending in a phase of regressing symptoms.

On physical examination the majority of our patients had a subtle swelling and tenderness over the dorsal side of the foot. The routine laboratory investigations are usually unremarkable [2, 23]. There may be a mild increase of ESR [12]. On radiographs the characteristic changes are a focal increase in radiolucency of one bone 4–8 weeks after the onset of pain. There are no narrowing of the joint space, no bone erosions or subchondral fractures. Remineralisation usually occurs within 2 years [15]. Radionuclide bone scans demonstrated an increased uptake in the first few days after the onset of

Fig. 4 **A** T1w GRE coronal section of the ankle joint. Hypointense area of the talus indicating TBME (arrow). **B** T1w GRE coronal section of the ankle joint taken 3 months after CD. Hypointense ringlike structure in the center of the talus indicating the drill hole after CD (arrow). No signs of TBME

symptoms, even before the radiographic changes are seen [3, 10, 16, 19]. The uptake is completely homogeneous [22] but non-specific. The diagnostic value of scintigram in TBME is therefore limited. Computed tomography is only a complementary investigation. It shows the joint space integrity, the extent of local demineralization and excludes other pathological conditions

Table 3 Characteristics of transient bone marrow oedema in comparison with avascular necrosis (AVN) and reflex sympathetic dystrophy

	Reflex sympathetic dystrophy	TBME	AVN (Ficat stage II)
History			
Onset of symptoms	Slow, progressive	Acute	Insidious
Pain at rest	Often	Uncommon	Common
Pain on exertion	Often	Often	Often
Previous trauma	Often	Uncommon	Common
Examination			
Migration	Uncommon	Often	None
Recurrence	Uncommon	Often	Uncommon
Trophic changes	Often, progressive	Uncommon	Uncommon
Personality	Labile	Stable	–
Diagnostics			
Radiographs	Diffuse, radiolucency	Homogenous, radiolucency	Osteosclerosis, subchondral collapse
MRI			
Signal alterations	Patchy	Homogenous	Double line sign
Localization	Multiple bones	One bone	Parts of one bone
Soft tissue involvement	Often	Uncommon	None
Treatment			
Core decompression	Contra- indication	Immediate pain relief	Immediate pain relief
Prognosis	Usually remaining disability	Reversible	Reversible

[9]. MRI is the diagnostic method of choice for TBME [1, 6, 11, 21]. Forty-eight hours after the start of the symptoms the characteristic oedema-like signal pattern of the bone marrow can be observed. In some cases there is also a joint effusion [1]. The diagnosis of TBME by MRI is one by exclusion. The different causes of a bone marrow oedema on MRI listed in Table 1 have to be excluded. Difficulties arise in the differentiation of reflex sympathetic dystrophy, early stage AVN (Ficat II) and TBME (Table 3).

Because of the clinical findings Lequesne [18] considered TBME to be a non-traumatic form of Südeck's atrophy. As a result of histological studies [8] showing an ischaemic origin of TBME, Hofmann et al. [14] recently postulated a common pathophysiological pathway between TBME and AVN. According to this theory, there is a complete repair of the ischaemic damage in TBME compared to an incomplete repair in AVN. This theory might explain the prolonged symptoms in our patient, who demonstrated partially necrotic tissue in the biopsy taken during CD.

Most of the treatments used for TBME have been evaluated in TBME of the hip. None have any effect on the course of disease. This was also confirmed in our study. Since TBME is a reversible disease, symptomatic treatment with non-steroid anti-inflammatory drugs and limited weight bearing is considered to be the treatment of choice [17]. Only CD is able to shorten the natural course of disease, which Hofmann et al. [13] was the first to observe in patients with TBME of the hip. Calvo et al. [4] – as well as the results of our study – confirmed this observation also in patients with TBME of the foot.

References

1. Bloem JL (1988) Transient osteoporosis of the hip: MR imaging. *Radiology* 167:753–755
2. Bramlett KW, Killian JT, Nasca RJ, Daniel WW (1987) Transient osteoporosis. *Clin Orthop* 222:197–202
3. Bray ST, Partain CL, Teates CD, Guilford WB, Williamson BR, McLaughlin RC (1979) The value of the bone scan in idiopathic regional migratory osteoporosis. *J Nucl Med* 20:1268–1271
4. Calvo E, Alvarez L, Fernandez YD, Vallejo C (1997) Transient osteoporosis of the foot. Bone marrow edema in 4 cases studied with MRI. *Acta Orthop Scand* 68:577–580
5. Curtiss PH (1959) Transitory demineralization of the hip in pregnancy: a report of three cases. *J Bone Joint Surg [Am]* 41:1327–1333
6. Daniel WW, Sanders PC, Alarcón GS (1992) The early diagnosis of transient osteoporosis by magnetic resonance imaging. A case report. *J Bone Joint Surg [Am]* 74:1262–1264
7. Delanois RE, Mont MA, Yoon TR, Mizell M, Hungerford DS (1998) Atraumatic osteonecrosis of the talus [published erratum appears in *J Bone Joint Surg [Am]* 1999 81:296]. *J Bone Joint Surg [Am]* 80:529–536
8. Dihlmann W, Delling G (1985) Is transient hip osteoporosis a transient osteonecrosis? *Z Rheumatol* 44:82–86
9. Dihlmann W, Thomas W (1983) Diagnostischer Algorithmus für die transitorische Hüftosteoporose unter Einbeziehung der Computertomographie. *ROFO Fortschr Geb Röntgenstr Nuklearmed* 138:214–219
10. Gaucher A, Colomb JN, Naoun A, Pourel J, Robert J, Faure G, Netter P (1980) Radionuclide imaging in hip abnormalities. *Clin Nucl Med* 5:214–226
11. Grimm J, Higer HP, Benning R, Meairs S (1991) MRI of transient osteoporosis of the hip. *Arch Orthop Trauma Surg* 110:98–102
12. Guerra JJ, Steinberg ME (1995) Distinguishing transient osteoporosis from avascular necrosis of the hip. *J Bone Joint Surg [Am]* 77:616–624
13. Hofmann S, Engel A, Neuhold A, Leder K, Kramer J, Plenk HJ (1993) Bone-marrow oedema syndrome and transient osteoporosis of the hip. An MRI-controlled study of treatment by core decompression [see comments]. *J Bone Joint Surg [Br]* 75:210–216
14. Hofmann S, Schneider W, Breitenseher M, Urban M, Plenk HJ (2000) “Transient osteoporosis” as a special reversible form of femur head necrosis. *Orthopade* 29:411–419
15. Hunder GG, Kelly PJ (1968) Roentgenologic transient osteoporosis of the hip. A clinical syndrome? *Ann Intern Med* 68:539–552
16. Hunder GG, Kelly PJ (1971) Bone scans in transient osteoporosis. *Ann Intern Med* 75:134
17. Judd DB, Kim DH, Hrutkay JM (2000) Transient osteoporosis of the talus. *Foot Ankle Int* 21:134–137
18. Lequesne M (1968) Transient osteoporosis of the hip. A non-traumatic variety of Sudeck’s atrophy. *Ann Rheum Dis* 27:463–471
19. O’Mara RE (1970) Bone scanning in regional migratory osteoporosis. *Radiology* 97:579–581
20. Plenk HJ, Hofmann S, Eschberger J, Gstettner M, Kramer J, Schneider W, Engel A (1997) Histomorphology and bone morphometry of the bone marrow edema syndrome of the hip. *Clin Orthop* 334:73–84
21. Potter H, Moran M, Schneider R, Bansal M, Sherman C, Markisz J (1992) Magnetic resonance imaging in diagnosis of transient osteoporosis of the hip. *Clin Orthop* 280:223–229
22. Schapira D (1992) Transient osteoporosis of the hip. *Semin Arthritis Rheum* 22:98–105
23. Swezey R (1970) Transient osteoporosis of the hip, foot, and knee. *Arthritis Rheum* 13:858–868
24. Wilson AJ, Murphy WA, Hardy DC, Totty WG (1988) Transient osteoporosis: transient bone marrow edema? *Radiology* 167:757–760