Eur. Radiol. 7, 1215–1217 (1997)

European
Radiology

Case report

Acute calcific tendinitis of the pectoralis major insertion associated with cortical bone erosion

H. R. Dürr[1], **A. Lienemann**[2], **H. Silbernagl**[1], **A. Nerlich**[3], **H. J. Refior**[1]

[1] Department of Orthopedics and Orthopedic Surgery, Ludwig-Maximilians University, Klinikum Grosshadern, Marchioninistrasse 15, D-81 366 Munich, Germany
[2] Department of Radiology, Ludwig-Maximilians University, Klinikum Grosshadern, Marchioninistrasse 15, D-81 366 Munich, Germany
[3] Institute of Pathology, Ludwig-Maximilians University, Klinikum Grosshadern, Marchioninistrasse 15, D-81 366 Munich, Germany

Received 19 December 1996; Revision received 19 February 1997; Accepted 24 February 1997

Abstract. A case of calcific tendinitis of the pectoralis major insertion with cortical bone erosion is presented. Clinical and laboratory findings showed a significant inflammatory reaction. Both CT and MR images demonstrated the extent of the lesion providing additional information on the dimensions of inflammatory soft tissue and bone marrow reaction. Biopsy was performed and histology revealed the typical features of calcification, inflammation and giant cell reaction.

Key words: Tendinitis – Bone resorption – Cortical defect – MRI – CT

Introduction

Calcific tendinitis is a well-known and clearly defined disease [1], frequently affecting the distal supraspinatus tendon [2, 3]. Involvement of the pectoralis major insertion is a less-common location [4, 5]. Associated bone destruction raises the suspicion of neoplasm, necessitating biopsy. We report a biopsy-proven lesion which resolved spontaneously without specific treatment 7 weeks after onset of symptoms.

Case report

A 31-year-old women presented with a 4-week episode of progressive mostly nocturnal pain in the left upper arm. No history of trauma or further diseases was noted. Clinical examinations showed neither swelling nor skin lesions. A localized pressure pain in the ventrolateral proximal third of the humerus with radicular sensation to the lower arm was evident.

The radiograph of the left proximal humerus showed faint calcification at the insertion of the pectoralis major muscle (Fig. 1). The 1-mm-thin sections of the spiral CT scan clearly demonstrated a juxtacortical calcified mass with an underlying cortical erosion at the anterior aspect of the proximal humerus (Fig. 2). An MRI scan after administration of Gd-DPTA (Magnevist, Schering, Berlin, Germany) revealed a zone of increased uptake surrounding the lesion and within the adjacent bone marrow. The muscles next to the lesion were not involved (Fig. 3).

The patient's sedimentation rate (22 mm/h), the white blood cell count (16.5×10^9) and the C-reactive protein (0.87 mg/dl, normal < 0.5 mg/dl) were elevated. Because the lesion was suggestive of a parosteal sarcoma, an incisional biopsy was performed revealing macroscopically a partially thickened cortex.

Histological examination showed fibrocartilage with focal psammom-like calcifications and chronic inflammation with few giant cells (Fig. 4).

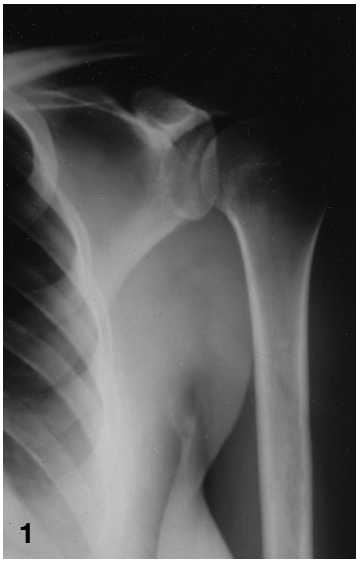
Three months later the patient was asymptomatic and the radiographs showed a total regression of the lesion.

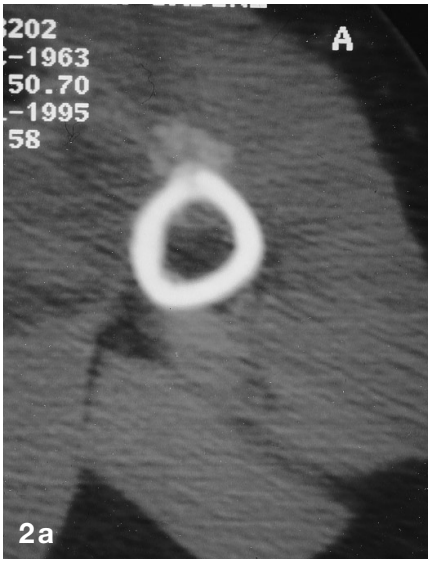
Discussion

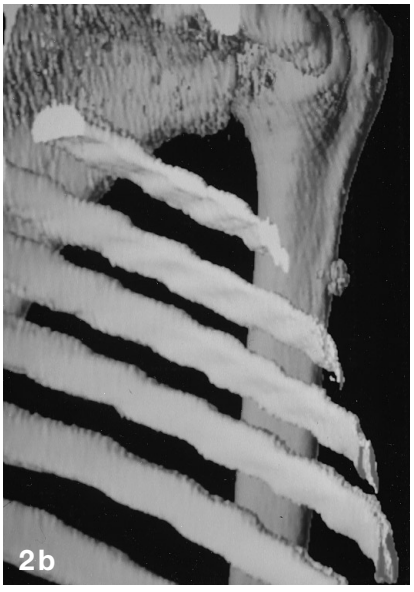
Calcific tendinitis is a common disorder [6] which can occur within or around tendons and at the insertion of muscle. As far as we know, reports on six cases of calcific tendinitis of the pectoralis major insertion have been published [3–6]. The characteristic location at the lateral lip of the distal portion of the bicipital groove, as seen in our case (Fig. 2b) in combination with a more or less calcified juxtacortical mass, should draw attention to this specific disorder. Clinical onset and features, as well as laboratory findings, may vary; however, MRI and histology, as in our case, are confirmatory.

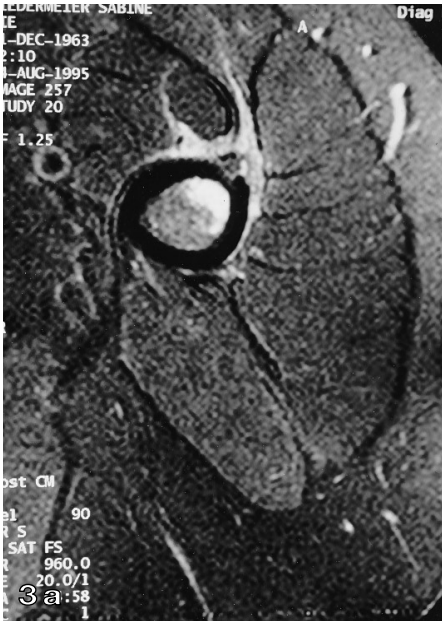
The pathogenesis is unknown, although soft tissue degeneration [8], soft tissue necrosis [9], trauma [10, 11] and hypoxia-induced metaplasia of the less perfused

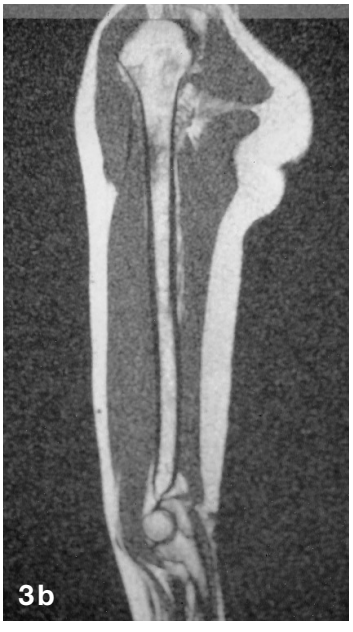
Correspondence to: H. R. Dürr











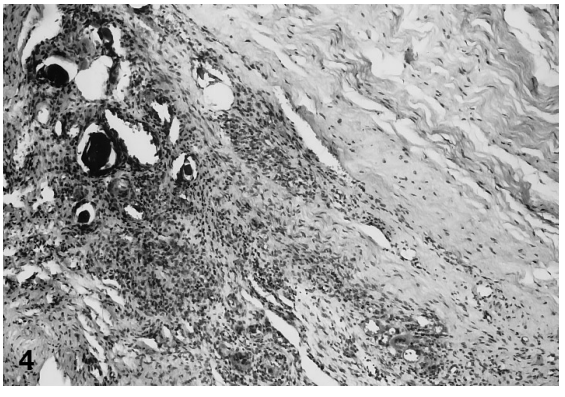


Fig. 1. Anteroposterior-view radiograph showing the calcification within the proximal third of the left humerus

Fig. 2 a, b. A CT scan demonstrates the fan-like juxtacortical soft tissue calcifications. An adjacent cortical erosion with extension into the bone marrow is noted. On the 3D surface reconstruction the lesion is located at the anterolateral aspect of the humerus beneath the bicipital groove

Fig. 3 a, b. Axial fat-suppression spin-echo images (TR 855 ms, TE 20 ms, field of view 200 mm) after Gd-DPTA **(b)** show an inflammatory reaction surrounding the low-signal calcification at the insertion as well as within the bone marrow. On the sagittal T1-weighted spin-echo image (TR 650 ms, TE 20 ms) a hypointense zone indicative of oedema is noted within the bone marrow

Fig. 4. Histomorphological aspect of the biopsy specimen showing focal round calcifications *(left side)* surrounded by a moderate infiltrate of lymphocytes and monohistocytes, focal multinucleated giant cells and increased vascularization of the otherwise typically fascicular tendon *(right side)*. (Original magnification × 60, haematoxylin and eosin)

tendon insertion to (fibro)cartilage with consecutive calcifications [12] have been discussed.

Cortical erosion can be associated with calcific tendinitis [4]. In a review of the literature, Hayes et al. [3] found no previous cases of calcific tendinitis with radiographic evidence of underlying bone erosion. In their reported five-patient study, two showed identical radiographic aspects as in our case. Similar cases were reported by Chadwick [4]. In haemodialysis patients, tendon rupture and bone erosion at the insertion site was found in 6 of 169 cases [13]. The pathomechanism in this particular condition might be linked to severe secondary hyperparathyroidism, but also structural disorders of collagen due to chronic acidosis have been shown [14].

The MRI images, as in our case, showed an inflammatory reaction with increased signal intensity due to a high uptake of Gd-DPTA. The reaction is limited to the insertion without any mass effect or infiltration of the surrounding muscles. Of interest is the transcortical

extent with involvement of the underlying bone marrow and oedema (Fig. 3).

Bone resorption may be due to active inflammation and associated increased local vascularization at the tendon insertion, or alternatively be the consequence of the mechanical effect of muscular traction at the inflamed insertion [3]. Fritz et al. favoured the induction of bone resorption by mononuclear phagocytosis of bone minerals and consecutive by induced prostaglandin and cytokine (interleukin-1, interleukin-6) release [5]. The typical picture of resorptive inflammation with foreign-body reaction, as is also shown in Fig. 4, seems to support this theory.

The main differential diagnosis is a malignant bone tumour also sharing the atypical location and extended cortical erosion, but also periostitis/myositis ossificans may be taken into consideration. Although calcific tendinitis seems to be a rare disorder, the exclusion of a malignant tumour must be achieved, eventually by biopsy. In some instances, however, particularly when the location at the anatomical insertion of a tendon is present and a significant soft tissue mass is lacking, the diagnosis can be made by radiographic methods alone. In such cases surgical intervention can be avoided.

Acknowledgements. We thank W. Knoll for corrections. This paper was supported by the Curt-Bohnewand Fund.

References

1. Uhthoff HK, Sarkar K (1989) Calcifying tendinitis. Baillieres Clin Rheumatol 3: 567
2. Holt PD, Keats TE, Gazarian A, Foucher G (1992) Calcific tendinitis: a review of the usual and unusual. Skeletal Radiol 11: 14
3. Hayes CW, Rosenthal DI, Plata MJ, Hudson TM (1987) Calcific tendinitis in unusual sites associated with cortical bone erosion. Am J Roentgenol 149: 967
4. Chadwick CJ (1989) Tendinitis of the pectoralis major insertion with humeral lesions. A report of two cases. J Bone Joint Surg [Br] 71: 816
5. Fritz P, Bardin T, Laredo JD, Ziza JM, DÁnglejan G, Lansaman J, Bucki B, Forest M, Kuntz D (1994) Paradiaphyseal calcific tendinitis with cortical bone erosion. Arthritis Rheum 37: 718
6. Welfling J, Kahn MF, Desroy M, Paolaggi JB, Seze S (1965) Les calcifications de l'epaule. II. La maladie des calcifications tendineuses multiples. Rev Rhum Mal Osteoartic 32: 325
7. Resnick D (1995) Calcium hydroxyapatite crystal deposition disease. In: Resnick D (ed) Diagnosis of bone and joint disorders, 3rd edn. Saunders, Philadelphia, p. 1615
8. Sandstrom C (1938) Peritendinitis calcarea: a common disease of middle life, its diagnosis, pathology, and treatment. Am J Roentgenol Radium Ther Nucl Med 40: 1
9. Peterson RR (1979) Prevention! A new approach to tendinitis. Occup Health Nurs 27: 19
10. Gondos B (1957) Observation on periarthritis calcarea. Am J Roentgenol Radium Ther Nucl Med 77: 93
11. Husson JL, Masse A (1983) Geode osseuse dystrophique polymicro-traumatique par arrachement tendino-musculaire chez le motocrossman. Acta Orthop Belg 49: 233
12. Uhthoff HK, Sarkar K, Maynard JA (1976) Calcifying tendinitis: a new concept of its pathogenesis. Clin Orthop 118: 164
13. Meneghello A, Bertoli M (1983) Tendon disease and adjacent bone erosion in dialysis patients. Br J Radiol 56: 915
14. Hahn TJ, Avioli LV (1970) Effect of chronic uremia on collagen metabolism in skin and bone. Arch Intern Med 126: 882