Eur. Radiol. 10, 92–94 (2000) © Springer-Verlag 2000

European
Radiology

Case report

Vertebral sarcoidosis: imaging findings

A. Poyanli[1], **O. Poyanli**[2], **S. Sencer**[1], **K. Akan**[2], **H. Sayrak**[3], **B. Acunaş**[1]

[1] Department of Radiology, Istanbul Medical Faculty, TR-34390 Çapa, Istanbul, Turkey
[2] Department of Orthopaedics and Traumatology, Göztepe SSK Educational Hospital, Kadıköy, Istanbul, Turkey
[3] Department of Pathology, Göztepe SSK Educational Hospital, Kadıköy, Istanbul, Turkey

Received: 28 January 1999; Revised: 14 April 1999; Accepted: 25 April 1999

Abstract. Sarcoidosis is a multisystemic disease of unknown aetiology characterised by noncaseating granulomatous inflammation with varying presentation and prognosis. Osseous disease reported in 1–13 % of cases commonly involves hands and feet; however, vertebral sarcoidosis is rare. This report describes the radiologic, CT, MRI and radionuclide imaging findings of vertebral involvement of a case with sarcoidosis.

Key words: Sarcoidosis – Vertebrae – MR imaging

Introduction

Sarcoidosis is a multisystemic granulomatous disease of unknown aetiology commonly encountered in young adults affecting lungs, skin and eyes. Diagnosis is made possible by a combination of clinical, radiological and histological findings, and noncaseating granuloma is the hallmark of the disease [1].

Skeletal sarcoidosis is rare. Most common sites of osseous involvement are hands and feet. Spine, ribs, pelvis and skull are infrequently involved [2].

In this report vertebral involvement diagnosed during the progression of a case with pulmonary sarcoidosis is reported. Plain X ray, CT, MRI and radionuclide imaging findings are presented.

Case report

A 35-year-old female was admitted with cough, dyspnoea, fever and fatigue of 1-year duration. Physical examination was unremarkable. Laboratory evaluation revealed lymphopenia (650 u/l), slightly elevated erythrocyte sedimentation rate (16 mm/h), polyclonal hypergammaglobulinaemia and high serum angiotensin converting enzyme (ACE; 69 U/l, N: 8–52 U/l).

Chest X ray showed bilateral hilar enlargement. On chest CT scan the lung parenchyma was normal, but there were multiple enlarged conglomerating mediastinal, hilar and right supraclavicular lymph nodes.

Pathological examination of the lymph node excised from the right supraclavicular area revealed noncaseating granulomas pathognomonic for sarcoidosis. In the control chest CT performed 3 months after commencement of steroid therapy, there was significant reduction in the size and number of mediastinal and hilar lymph nodes. At this stage the patient started complaining about dorsal and lower back pain. Plain X-ray and CT scan of the thoracolumbar spine were performed and lytic lesions were detected in the third to fifth lumbar and second and third thoracal vertebral bodies, as well as the pedicles of the second and third thoracal and fifth lumbar vertebrae (Fig. 1 a,b).

Magnetic resonance imaging of the thoracal spine showed focal enhancing lesions hypointense on T1-weighted and of bright signal on T2-weighted and short tau inversion recovery (STIR) sequences in the second and third thoracal vertebral bodies and in the right and left pedicles of the second and third thoracic vertebrae, respectively. There was also prevertebral soft tissue extension of the destructive lesion in the anterior portion of T2 (Fig. 1 c,d). The left pedicular lesion in T3 was seen to infiltrate the ipsilateral neural foramen and invade the spinal canal. There was no intradural extension or involvement of the disk spaces, and no compression of the spinal cord.

On lumbar spine MRI, hypointense nodular lesions were detected on T1-weighted sequence in the peridiskal compartments of third to fifth lumbar vertebral bodies. The same lesions enhanced peripherally after IV Gd-DTPA (0.1 mmol/kg body weight; Schering, Berlin, Germany) injection on T1-weighted images and with high signal on T2-weighted sequence (Fig. 1 e,f). There was neural foramen and left prevertebral soft tissue involvement of the lesion detected in the body of L4. The

Correspondence to: A. Poyanli

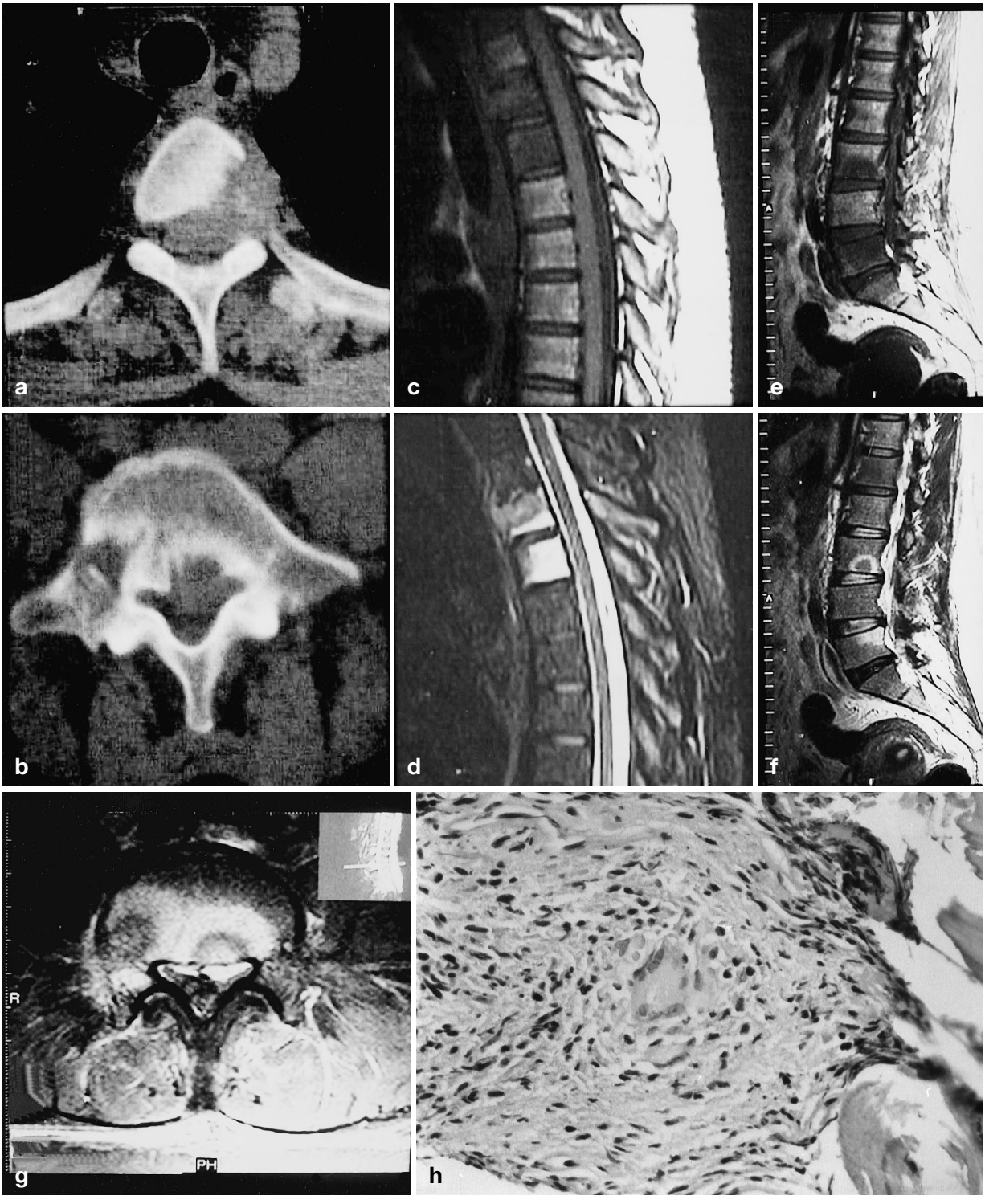

Fig. 1. **a** Destructive lesion is shown in the left half of T3 on CT. **b** Lytic lesion with sclerotic margins is shown on CT posteriorly in the body and in the right pedicle of L5. Destructive mass lesion in the anterior portion of T2 and extending into the prevertebral region and focal signal changes in T2 and T3 is shown to be dark on **c** sagittal T1-weighted and bright on **d** sagittal short T1 inversion recovery sequences. In the sagittal MRI of the lumbar region, lesions in L3 and L5 are hypointense on **e** T1-weighted and peripherally hyperintense on **f** fat-suppressed T2-weighted sequences. **g** Enhanced T1-weighted MRI shows peripherally enhancing lesion in the body and right pedicle of L5 and epidural extension most prominent on the left side. **h** Marrow biopsy from L5, paraffin block section. Haematoxylin and eosin stain. Granuloma consisting of multinucleated giant cells and epithelioid cells almost surrounded by a ring of collagen fibrosis

extensive lesion detected in the vertebral body and right pedicle of L5 invaded intraspinal epidural fat planes (Fig. 1 g).

The first two phases of a radionuclide triple-phase bone scan with 20 mCi of IV Tc-99 m tagged MDP were normal. The third phase showed pathological focal and regional uptake in the second and third thoracal as well as the fourth and fifth lumbar vertebral bodies.

A transpedicular bone biopsy was performed in L5. On pathological examination the diagnosis of sarcoidosis was made upon detection of typical noncaseating granulomas (Fig. 1 h).

Discussion

Rieder was the first to define the radiological features of sarcoidosis in 1910 [3]. Although there are now numerous reports of osseous involvement in sarcoidosis defining its radiographic characteristics, there is still controversy about the incidence of skeletal involvement in the disease, with radiographic evidence of osseous involvement varying from 1 to 13 % [4].

Radiological and CT imaging findings in vertebral sarcoidosis include a lytic appearance with or without peripheral sclerosis, mixed lytic and sclerotic lesions and purely sclerotic lesions [5, 6, 7, 8, 9]. Multiplicity of lesions, disk-space preservation and extension into the pedicles have been reported. In our case there were lytic vertebral body lesions with sclerotic margins often involving the pedicles, findings compatible with those in the literature. Disk spaces were normal.

Although MRI is a very sensitive modality in the detection of osseous sarcoidosis, the findings are not specific and may mimic osteomyelitis or neoplasm, even in the presence of proven systemic sarcoidosis. It is well known that osteolytic lesions of sarcoidosis have long T1 and T2 values and enhance strongly [5, 10, 11]. Bushara et al. reported a case of vertebral sarcoidosis with normal plain X-rays and spinal CT showed sclerotic lesions [9]. Magnetic resonance imaging of the same case revealed nonenhancing lesions hypointense on T1-weighted and isointense to hypointense on T2-weighted sequences. These signal characteristics have been attributed to osteosclerotic sarcoidosis. In our patient MRI findings were in the form of hypointense lesions in the lumbar and thoracal vertebral bodies on T1-weighted sequences. T2-weighted and STIR-sequence signals of the same lesions were uniformly hyperintense in the thoracic region, and peripherally hyperintense in the lumbar region. Enhancement on T1-weighted series was diffuse in the thoracic region and peripheral in the lumbar region.

Reports on radionuclide imaging of vertebral sarcoidosis is rare [12, 13]. Cinti et al. detected multiple lesions in the ribs and skull showing uptake of Tc-99 m MDP [13]. They also reported that the uptake in the ribs resolved completely after steroid therapy, whereas skull uptake decreased significantly. In our case triple-phase bone scintigraphy showed increased focal uptake in the involved thoracic and lumbar vertebral bodies in the late phase.

In conclusion, radiological, CT, MRI and radionuclide imaging findings are nonspecific in vertebral sarcoidosis. Multiple levels of involvement and sparing of intervertebral disks are suggestive; however; precise diagnosis is only possible with histopathological verification.

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