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Primary Osseous Amyloidoma Causing Spinal Cord Compression

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Summary: The myelographic, CT, and MR findings of a rare localized primary bone amyloidoma causing upper thoracic spinal cord compression are presented. Imaging showed a large, calcified mass centered in the posteromedial portion of the left second rib that invaded the second thoracic vertebra and the spinal canal.

Index terms: Amyloidosis; Spinal cord, compression; Spine, neoplasms

Bone involvement by amyloidosis is a well-known phenomenon. Amyloid lesions in bone are most common in patients with systemic amyloidosis, multiple myeloma, macroglobulinemia (1), and chronic hemodialysis (2). We report a case of a primary amyloidoma involving the upper thoracic spine and causing spinal cord compression. The appearance of this tumor on computed tomography (CT) and magnetic resonance (MR) imaging is presented.

Case Report

A 54-year-old man had a 3-week history of progressive bilateral lower extremity weakness, right greater than left. A chest radiograph showed a large calcified mass in the upper left part of the thorax with associated destruction of the left second rib. CT examination of the chest (Fig 1A) showed a large calcified mass with destruction of the left side of the body and left posterior elements of the second thoracic (T-2) vertebra and the posteromedial portion of the left second rib. Myelography showed a nearly complete extradural block at the T-2 level (Fig 1B). The postmyelogram CT scan (Fig 1C) showed anterior and left lateral extradural impingement by the mass on the thecal sac at the T-2 level. MR imaging (0.5-T Vista; Picker, Highland Heights, Ohio) (Fig 1D and E) showed replacement of normal marrow fat in the body and the posterior elements of T-2 with low signal intensity on T1-weighted (500/26/2 [repetition time/echo time/excitations]) and T2-weighted (2500/100/1) images. The densely calcified portion of the mass seen on the CT images demonstrated low signal

intensity on both the T1-weighted and T2-weighted MR images. There was a moderate amount of enhancement of the noncalcified portions of the mass on gadopentetate dimeglumine-enhanced T1-weighted (500/26) MR images. Technetium-99m methylene diphosphonate bone scintigraphy, performed to look for additional lesions, showed intense uptake of radiotracer in the region of the lesion in the upper left hemithorax, but no other abnormalities.

The mass was percutaneously biopsied with ultrasound guidance. Congo red staining demonstrated apple green birefringence characteristic of amyloid. The mass was surgically resected, and examination of the specimen confirmed the diagnosis of amyloidoma.

Discussion

Primary amyloidomas can involve the spine (3–8). In previously reported cases, five of these tumors were located in the thoracic spine (3, 5–8) and one was located in the cervical spine (4). Two of these patients had symptoms of spinal cord compression (paraplegia) (3, 8) and four had radicular pain (4–7). The tumor described in this report is centered to the left of the spine in the posteromedial portion of the left second rib. It probably arose from this rib and secondarily invaded the T-2 vertebra. Another case has been reported in which an amyloidoma involved a rib and the adjacent thoracic spine, but that tumor was not described in sufficient detail for us to assess whether the tumor arose primarily from rib or vertebra (3).

The densely calcified mass seen in this case is an unusual appearance for a primary bone amyloidoma because these tumors are usually lytic lesions (1). Two previously reported primary amyloidomas of the spine were described as having radiographically detectable calcifications, one with scattered calcifications shown by CT (7) and the other with calcifications seen on

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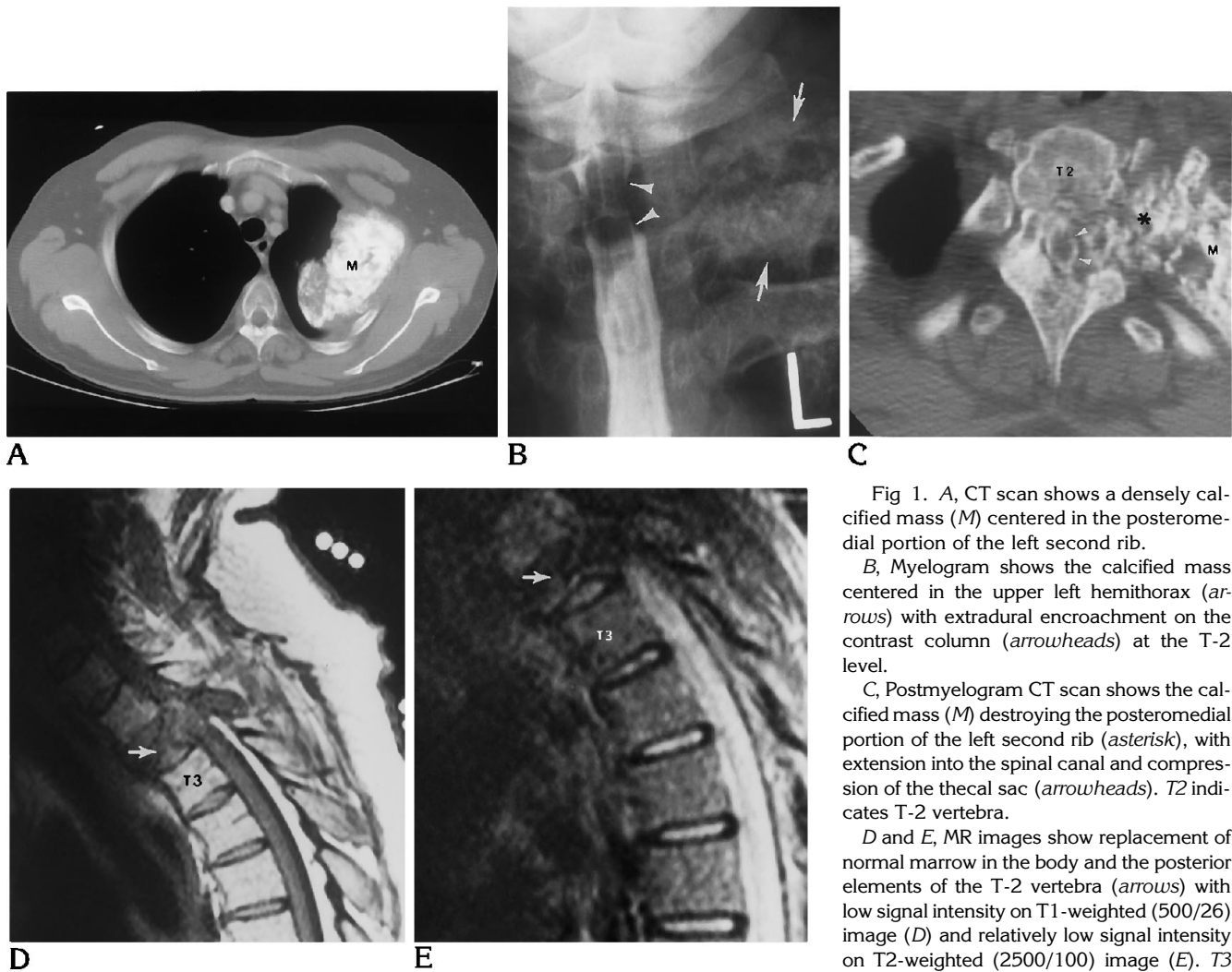


Fig 1. A, CT scan shows a densely calcified mass (M) centered in the posteromedial portion of the left second rib.

B, Myelogram shows the calcified mass centered in the upper left hemithorax (arrows) with extradural encroachment on the contrast column (arrowheads) at the T-2 level.

C, Postmyelogram CT scan shows the calcified mass (M) destroying the posteromedial portion of the left second rib (asterisk), with extension into the spinal canal and compression of the thecal sac (arrowheads). T2 indicates T-2 vertebra.

D and E, MR images show replacement of normal marrow in the body and the posterior elements of the T-2 vertebra (arrows) with low signal intensity on T1-weighted (500/26) image (D) and relatively low signal intensity on T2-weighted (2500/100) image (E). T3 indicates T-3 vertebra.

thoracic spine radiographs. However, as radiographs for the latter case were not shown, we are unable to compare that case directly with ours (8). Focal amyloidomas of the head and neck have been described to frequently contain calcifications detectable with CT (9), but that does not apply to the widespread, dense calcifications seen in the amyloidoma described here. On the basis of the plain radiographic and CT findings in our case, the differential diagnosis included chondrosarcoma, osteogenic sarcoma, and a calcified metastatic tumor, such as mucinous adenocarcinoma of the colon.

The intense uptake of bone radiotracer by the tumor is not surprising, because amyloid deposits, which form ectopic ossifications and calcification, have been previously described as

demonstrating avid uptake of bone radiotracer (10).

The MR signal characteristics of solitary amyloidomas in the head and neck region have been previously described as being similar to those of skeletal muscle (9, 11). The signal characteristics in the noncalcified portion of the mass in our case are also similar to those of skeletal muscle. An amyloidoma of the nasopharynx has been reported that demonstrated a moderate degree of enhancement with gadopentetate dimeglumine, again similar to the tumor we describe (11).

The imaging findings in amyloidoma are not specific and can simulate neoplasm. Biopsy is required for diagnosis. The presence of calcification and low signal intensity on T2-weighted

images should, however, suggest the possibility of primary amyloidoma.

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