
Tophaceous Gout of the Lumbar Spine Mimicking an Epidural Abscess: MR Features

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Summary: We present the MR findings in a case of tophaceous gout of the lumbar spine with cauda equina compression, mimicking an epidural abscess. The diagnosis was clinically unsuspected and confirmed at laminectomy. Tophus material in the paraspinal soft tissues appeared intermediate in signal intensity and showed heterogeneous contrast enhancement.

Index terms: Spine, abscess; Spine, magnetic resonance

Involvement of the spine by tophaceous gout is an uncommon occurrence; we found 23 reported cases (1–23), of which 16 resulted in spinal cord or radicular compression, necessitating surgical decompression (2, 6–11, 13–16, 18–21, 23). In many instances, the preoperative diagnosis was unsuspected despite the fact that the majority of patients had chronic polyarticular gouty arthritis. Two previous publications have documented cord compression by gouty tophus on magnetic resonance (MR) images (21, 23). Our case demonstrates cauda equina compression with MR features that simulate an epidural abscess.

Case Report

A 76-year-old man with long-standing peripheral gouty arthritis had a 1-week history of persistent fever and severe lower back pain. Physical examination revealed a fever of 40°C and marked tenderness to palpation on the right side of the lower back and flank. Multiple tophi were also noted over the elbows, hands, left knee, and both big toes. Purulent discharge emanated from the right big toe, raising the possibility of sepsis from this source. Blood cultures were found to be positive for staphylococcus species and the patient was subsequently started on intravenous ampicillin and gentamicin. Computed tomography (CT) of the lumbar spine showed spinal stenosis at the L3-4 and L4-5 levels. Laboratory investigations revealed leukocytosis (cell count of $11.4 \times 10^9/L$) and a serum uric acid level

of 491 $\mu\text{mol/L}$ (normal range, 202 to 416 $\mu\text{mol/L}$). Seventeen days after admission, despite continued antibiotic treatment and repeated negative cultures, the patient still had severe back pain and a low-grade fever but was neurologically intact.

A lumbar spine MR study showed an abnormal anterior epidural collection extending from the L-3 to the L-4 levels, compromising the spinal canal and causing cauda equina compression (Fig 1A and B). This collection was of soft-tissue intensity on the T1-weighted sequence and appeared relatively hyperintense on the T2-weighted images, particularly posterior to the L-3 vertebral body. Both L3-4 and L4-5 disks as well as the adjacent L-4 and L-5 vertebral endplates were abnormally hyperintense on the long-repetition-time sequence (Fig 1B). The intervertebral disk space at L3-4 was maintained, whereas at L4-5 it was moderately narrowed. After administration of contrast material, the collection appeared multiloculated with several zones showing marginal enhancement (Fig 1C and D). Focal enhancement was also noted at the posterior aspect of the L4-5 disk and in the adjacent vertebral endplates. These findings were highly suggestive of an infectious spondylodiskitis with an epidural abscess.

A laminectomy at the L3-4 and L4-5 levels did not reveal an abscess. Rather, a chalky granular sandlike substance was found scattered diffusely in the epidural and paraspinal soft tissues, including the bones, muscles, and ligaments. An L3-4 discectomy was performed and multiple open biopsy samples were taken from the adjacent locations. Gram stain of this material did not reveal bacteria, but polarizing microscopy later showed the presence of negatively birefringent uric acid crystals. Histologic examination disclosed a granulomatous infiltrate consisting of multinucleated giant cells and histiocytes surrounding an amorphous material, consistent with tophaceous gout (Fig 1E). The patient was subsequently treated with colchicine followed by allopurinol. The fever subsided and all antibiotics were discontinued. No further episode of fever was noted and the patient was discharged in stable condition.

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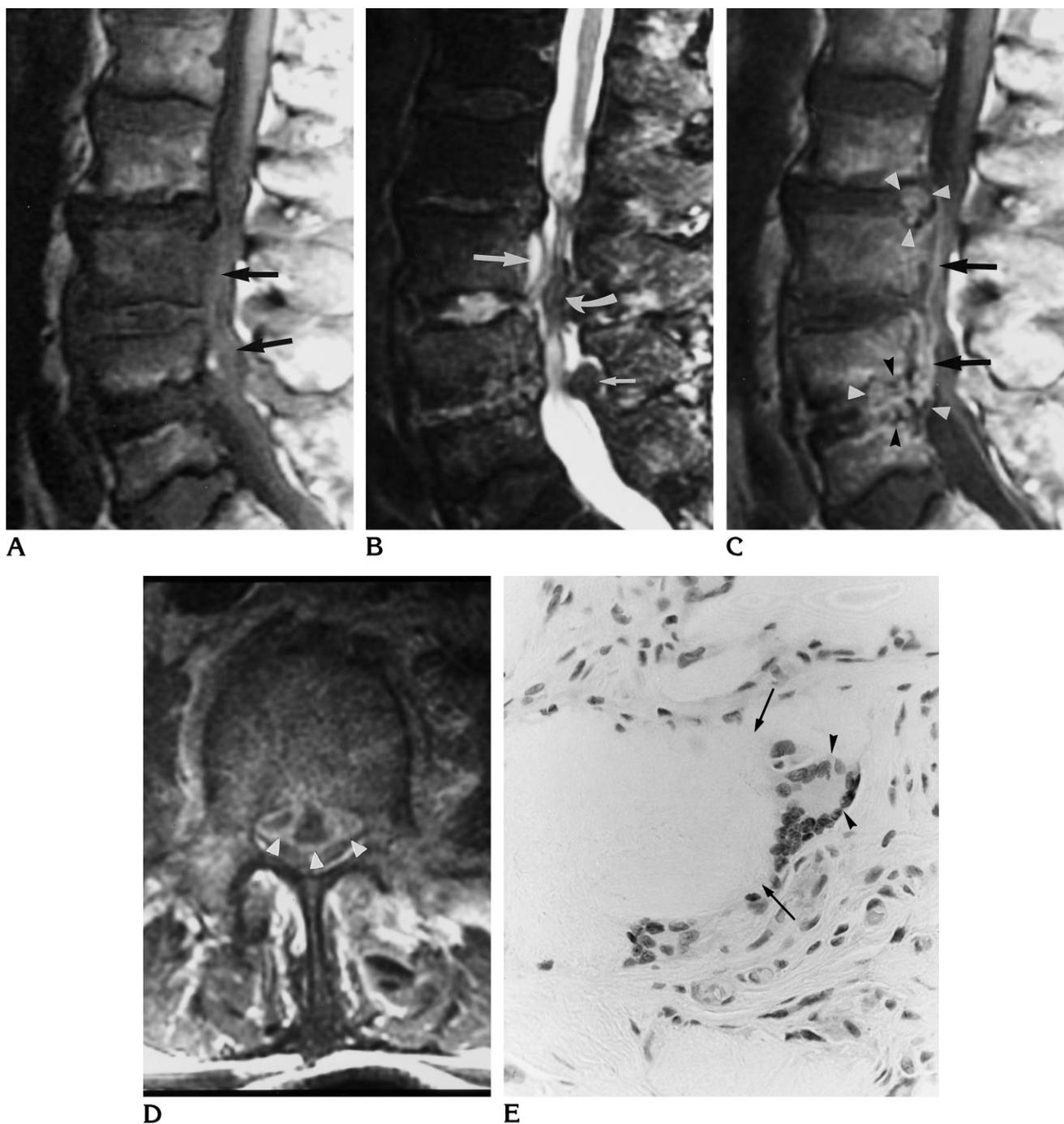


Fig 1. Seventy-six-year-old man with long-standing peripheral gouty arthritis and 1-week history of persistent fever and severe lower back pain.

A, Sagittal spin-echo T1-weighted (500/20 [repetition time/echo time]) MR image of the lumbar spine shows cauda equina compression by an epidural soft-tissue-intensity lesion at L-3 and L-4 (arrows). Note also herniation of the L2-3 and L4-5 disk and erosive changes at the L-4 and L-5 vertebral endplates.

B, Sagittal fast spin-echo T2-weighted (4000/102) MR image (echo train length, 8) of the lumbar spine shows abnormal hyperintense signal of the epidural collection at L-3 (long arrow) and of the L3-4 and L4-5 intervertebral disks. Note the hypointense appearance of the compressed roots of the cauda equina (curved arrow). The hypointense structure at the posterior aspect of the spinal canal at L4-5 represents partial volume averaging of the posterior elements (short arrow).

C, Contrast-enhanced sagittal spin-echo T1-weighted (500/20) MR image of the lumbar spine shows marginal enhancement of the epidural collection (arrows) and focal enhancement of the L2-3 and L4-5 disks and the L-4 and L-5 vertebral endplates (arrowheads). The L-5 vertebral body is mildly compressed.

D, Contrast-enhanced axial spin-echo T1-weighted (500/20) MR image of the lumbar spine at L-3 shows severe dural sac compression by the multilobulated epidural collection representing gouty tophaceous deposits (arrowheads).

E, Photomicrograph of biopsy specimen from the L2-3 disk shows a large giant cell (arrowheads) adjacent to a tophaceous deposit (arrows). Several other multinucleated giant cells are also present (original magnification $\times 200$).

Discussion

A radiologic description of gouty involvement of the spine was published in 1947 by Bauer and Klemperer (24) but the pathologic basis of these lesions was not established until the report by Kersley et al in 1950 (1). These authors described the autopsy findings of a case of cord compression due to atlantoaxial subluxation associated with bony destruction by gouty tophus. We found 22 additional cases in the literature, all proved at autopsy, by surgical laminectomy, or by percutaneous biopsy findings (2–23). Why urate crystals rarely accumulate in the spine as opposed to the appendicular skeleton is unclear. The pathogenesis of tophus deposition may relate to local tissue changes, which may differ in the spine and peripheral joints. It has been suggested that irrespective of the tissues affected, a previous injury or tissue necrosis is a prerequisite for urate deposition (25). Degenerative disease of the spine may also be a predisposing factor, as most cases involving the lumbar spine predominate at the lumbosacral junction. The prevalence of spine involvement in gout is, however, probably underestimated. Patients who may have asymptomatic tophi are not routinely imaged, and such lesions even if diagnosed are not pathognomonic. In addition, unlike peripheral gouty arthritis, the lack of readily available synovial fluid or biopsy material for demonstration of urate crystals renders the diagnosis of spinal gout more difficult.

Some inferences may be drawn from a review of the cases reported to date. The majority of patients (82%) have chronic polyarticular tophaceous gout and hyperuricemia, with a mean duration of disease of 14 years (range, 0 to 35 years). These findings are independent of age and sex. There is a wide spectrum of clinical symptoms, varying from absence of to acute quadriplegia (9). Many patients experience back pain, but this could be attributed to coexisting degenerative disease rather than direct spinal involvement by gouty arthritis. All segments of the spine are affected in approximately equal distribution. The frequency of neural compromise is 73%, and, in most cases, this leads to an acute neurologic deficit requiring emergency surgical decompression. In cases of isolated pain, colchicine therapy produces rapid relief of the symptoms (5, 22). Our case is similar to the one reported by Wald et al (8), in that the patient was neurologically intact de-

spite imaging evidence of cauda equina compression.

Radiographic features of spinal gout are well documented and include vertebral erosions (mainly at the diskovertebral junction), bone destruction causing joint subluxation, spinal deformity, pathologic fractures, and osteophyte formation. MR imaging findings have been reported in two recent cases of cord compression at the C1-2 and C3-5 levels (21, 23). In our case, epidural tophus extended beneath the posterior longitudinal ligament over two vertebral levels and also involved the intervening intervertebral disks and portions of the adjacent endplates. This is expected, as urate deposits are known to accumulate at the diskovertebral junction (12). Its multicocular appearance following administration of contrast material further suggested the diagnostic possibility of an epidural abscess resulting from spondylodiskitis. Magid et al (10) reported that even on gross inspection at surgery, gouty tophus could simulate the appearance of purulent material. Other granulomatous conditions should also be considered in the differential diagnosis, including tuberculous and fungal infections (given the pattern of subligamentous epidural extension and relative preservation of the intervertebral disk spaces) as well as rheumatoid arthritis. The latter more commonly involves the upper cervical spine and can be distinguished from gouty arthritis only by the isolation of urate crystals in the epidural tophus deposits.

Our case illustrates the MR features of intraspinal epidural tophus deposits mimicking an epidural abscess. Such demonstration in patients with peripheral gouty arthritis with no neurologic symptoms should forestall surgical intervention and prompt adequate medical treatment.

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