The case:

A 69-year-old man presented with acute and severe back pain that radiated to his left lower leg. Computed tomography and magnetic resonance imaging scans were performed to assess for potential lesions.

Figure 1: Sagittal T1-weighted (A) and T2-weighted (B) magnetic resonance images of the spine.

Your diagnosis?

For answer see page 434
Diagnosis:

Vertebral Gout

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A 69-year-old man presented with acute, severe back pain that radiated throughout the lower left extremity. No fever, weight loss, nephropathy, or diabetes mellitus was noted, but he had heart failure. Computed tomography (CT) and magnetic resonance imaging (MRI) were performed to assess for potential lesions, and a large destructive mass involving the body of L2 was found. The mass extended to adjacent intervertebral spaces and the inferior plateau of L1, as well as the nearby soft tissues, L1-L2 intervertebral foramen, and vertebral canal (Figure 2).

On CT, the mass displayed soft tissue density with scattered focus of hypodense attenuation (Figure 2E). On MRI, the mass had a hypointense signal on all sequences (Figure 2A-C). Postcontrast images showed heterogeneous enhancement in the mass, including areas with marginal enhancement (Figure 2D).

Because the mass was of low signal on all MRI sequences and close to a facet joint, the prime consideration was pigmented villonodular synovitis, a reactive synovial lesion that typically shows a low signal on all MRI sequences. However, the calcifications noted on CT excluded this diagnosis. Despite the size and contrast enhancement, the absence of bright signal within the mass on the T2-weighted images and the absence of overt osseous destruction for the size of the mass spoke against a malignant neoplasm.

A needle biopsy of the mass (3 samples) revealed a whitish, chalky material with firm and rubbery consistency mixed with serous fluid. Culture of the material resulted in no bacterial growth. Microscopic images demonstrated the presence of urate crystals and cells compatible with a gouty tophus. Further testing revealed that the patient had hyperuricemia (9.4 mg/dL). Because the patient had heart failure and was unable to undergo surgery, conservative pharmacologic treatment was initiated. The patient reported that he experienced partial reduction of pain after 1 month of treatment.

Definition and Epidemiology

Gout, a purine metabolism disorder, is caused by long-standing hyperuricemia that results in urate crystal deposition in the joints, soft tissues, bones, and urinary tract. Untreated, gout may result in tophus formation, as seen in this patient. A tophus is the deposition of monosodium urate crystals surrounded by chronic mononuclear and giant-cell reactions, and tophi can be seen anywhere in the body. The skin overlying a tophus may ulcerate and the crystals extrude as a white, chalky material.

The prevalence of gout has increased in the world, and it is now one of the most common causes of inflammatory arthritis, with a prevalence of...
radiologic case study

approximately 5.2 cases per 1000 individuals in the United States in 1999. Gout typically affects the peripheral joints of the appendicular skeleton but is also known to involve the axial joints. In one study, 17% of 143 patients with gout had axial involvement, specifically of the sacroiliac joints. Another report found evidence of spinal gout in 14% of 64 patients with gouty arthritis who had undergone a spinal CT examination.

Gout can result from primary or secondary hyperuricemia. Primary hyperuricemia is related to congenital enzymatic defects, whereas secondary hyperuricemia results from a preexisting or acquired condition linked to dietary excess (a diet containing high quantities of meat, seafood, and sugar-sweetened soft drinks), alcohol consumption, obesity, hypertension, hyperlipidemia, insulin resistance, chronic kidney disease, cardiovascular disease, or diuretic use. Osteoarthritis appears to predispose patients with gout to local urate crystal deposition but is not a risk factor for gout.

Studies of spinal gout have demonstrated risk factors and triggers similar to those in patients without axial gout, including renal failure, diuretic use, and progressive cardiovascular disease. Sex and age also affect a patient’s risk for developing gout. At all ages, men have higher urate levels than women and are more likely to develop gout. Younger women rarely develop gout, presumably because of the uricosuric effect of estrogen; gout is more likely to occur in postmenopausal women. Aging individuals also have an increased risk of developing gout, possibly because patients may accrue multiple risk factors for gout as they age. Spinal gout, similar to peripheral gout, has been reported to be more common in men (3:1) and in the fifth decade of life.

DISTRIBUTION

Spinal gout occurs most commonly in the lumbar region (78% of cases). The sacroiliac joints, cervical spine, and thoracic spine can also be affected. The crystal depositions are generally found at the facet joint, but the vertebral body, disk space, pedicle, lamina, facets, ligamentum flavum, and epidural space can also be affected. One case report described intradural involvement of the filum terminale.

CLINICAL PRESENTATION

Spinal gout lesions can be found in asymptomatic patients. Patients with symptomatic spinal gout may present with a variety of symptoms, including back pain, radiculopathy, neurogenic claudication, cauda equina syndrome, paralysis, myelopathy, and fever. Symptoms may last for a day or can be chronic; no specific duration seems to be characteristic of this condition. Most patients with spinal gout present with a history of polyarticular gout or an unclear arthritis. On physical examination, peripheral tophaceous deposits may be present.

LABORATORY STUDIES

The demonstration of crystals during an acute attack is diagnostic of gout; however, because the spine lacks available synovial fluid, the diagnosis of spinal gout is more difficult. Serum uric acid levels are often elevated in patients.
with spinal gout. However, serum uric acid can have normal values during an acute attack. The proportion of men with a normal serum uric acid during a gouty attack has been reported as 49%, 39%, and 43% when the serum uric acid upper limit was 7.5, 8, or 8.1 mg/dL, respectively. However, some consider serum uric acid levels >6 mg/dL abnormal; only 14% of the patients with acute gout present with values below this normal level. Inflammation markers, such as C-reactive protein and erythrocyte sedimentation rate, can be elevated, and leukocytosis may also occur.

On macroscopic evaluation of pathological specimens, gout presents as a whitish, chalky material (100% alcohol is required for preservation). Under a polarized light microscope, monosodium urate crystals are large (10 to 20 mm long), needle-shaped crystals that show strong light intensity and a negative sign of birefringence.

**IMAGING**

The major role of imaging is to evaluate structural changes, crystal deposition, and chronic inflammation in patients with gout. Imaging studies may also assess the patient’s response to urate-lowering therapy because crystal dissolution can occur after a long period of therapy and result in a subsaturating serum urate level. The appropriate imaging study will depend on the question to be answered and the cost.

Bony changes caused by gout may take years to be visible on radiographs, and urate crystals are not radiographically opaque—only if the urate deposits undergo calcification will they be characteristic. Although radiographs lack sensitivity and have limited use in identifying axial gout, they are low cost and may be used to initiate screening and to evaluate vertebral stability.

Computed tomography and MRI are more effective in evaluating soft tissue extension and neuronal compression in patients with gout. When calcified, the crystal deposits can be delineated on CT images as a hyperdense focus (Figure 2E). On MRI, the crystals have a nonspecific appearance, with low or intermediate signal on T1-weighted and variable signal on T2-weighted images (Figure 2A-C).

Postcontrast MRIs also result in a nonspecific appearance, showing diffuse contrast enhancement among areas without enhancement; the latter may be related to crystal deposition (Figure 2D). This entity should be considered when a noncalcified mass shows low signal on T2-weighted MRIs, particularly in locations associated with gout such as extensor surfaces. In the reported case, the MRIs showed a large mass in the vertebral body of L2, with low signal on both T1- and T2-weighted images (Figure 2A-C). This appearance on MRI may be useful in distinguishing gout from other etiologies because it appears in only a few conditions, such as calcification, fibrous tissue (may be relative to acellular-ity and abundant collagen), protein-rich fluid, gas, and flow void, and in the presence of paramagnetic substances such as hemosidin (e.g., pig-mented villonodular synovitis and chronic hemorrhage), de-oxymyoglobin, intracellular methemoglobin, iron, ferritin, and melanin.

Recently, dual-energy CT (DE-CT) scanning has been proposed as a way to detect and monitor urate crystal deposition in patients with gout. Dual-energy CT systems scan at 2 different energy levels (at 80 and 140 kVp) and create 2 sets of images that can, with postprocessing, distinguish tissues with different chemical compositions if they have different attenuations at high and low radiography energies. Because uric acid crystals, soft tissue, bone, and calcium show different attenuations at the 2 kVps, DE-CT allows accurate and specific characterization of uric acid.

Besides the potential to allow specific, noninvasive diagnosis of tophaceous de- posits, DE-CT can also estimate the total body urate burden through whole body scans. A recent case report confirmed that a DE-CT scan could better identify uric acid depositions at the facet joint than conventional CT images that showed mildly increased attenuation. Further investigations are required to determine the role DE-CT will play in the management of gout.

**DIFFERENTIAL DIAGNOSIS**

The differential diagnosis of spinal gout includes osteomyelitis, Pott’s disease (vertebral tuberculosis), diskitis, epidural abscess, metastatic disease, anklyosing spondylitis, and spinal stenosis. Gout should be considered as a possible diagnosis in all cases of spinal cord impingement in patients with risk factors for gout. Fever, leukocytosis, elevated sedimentation rate, and granulomatous reaction on histology may occur in patients with gout or infection, and these conditions may also coexist.

To rule out infection, blood cultures should be taken, and fine-needle aspiration or biopsy of the lesion under CT guidance is generally recommended. Needle biopsy is not always diagnostic; however, it may also help to exclude other disease processes, thus preventing unnecessary surgery. As with other presentations of gout, the definitive diagnosis of spinal gout is made by means identifying urate crystals on polarized light evaluation of histological specimens.

**TREATMENT**

Early recognition and pharmacological treatment of spinal gout are important to avoid spinal surgery. Medical therapy alone has been reported to reduce intraspinal tophi and therefore should be the treatment of choice for patients without neurologic compromise.

Therapy of patients with acute gout should aim to rapidly relieve the pain and disability caused by intense inflammation. Management of acute attacks may include...
the use of nonsteroidal anti-inflammatory drugs, colchicine, glucocorticoids, and possibly corticoterpin.\textsuperscript{21} For chronic treatment, the goal is to lower serum urate levels and prevent acute flares and the development of tophi. Drugs approved for this purpose include xanthine oxidase inhibitors, uricosuric agents, and uricase agents.\textsuperscript{21}

For patients with neurologic symptoms, neurosurgical decompression is usually necessary.\textsuperscript{3,10} A review of the literature found that almost all patients with spinal gout and neurological symptoms who underwent decompressive laminectomy with or without stabilization had good results.\textsuperscript{8}

**Prognosis**

Gout is considered a progressive disease that can be divided into the following phases: initial asymptomatic hyperuricemia, recurrent acute gout attacks intermingled with asymptomatic intercritical periods, and chronic symptomatic tophaceous gout. When not treated effectively in long-term therapy, gout may progresses to severe tophaceous gout in as quickly as 5 years and cause joint damage and significant functional impairment. Not all patients with hyperuricaemia develop gout, and a long period of asymptomatic hyperuricaemia may precede the first acute attack.\textsuperscript{9} Medical maintenance of gout is the mainstay of therapy. Modification of risk factors should be pursued whenever possible. Frequent follow-ups and imaging studies may help minimize complications.\textsuperscript{8}

**Conclusion**

Spinal gout is more common than generally perceived and should be included in the differential diagnosis for patients with destructive and aggressive lesions of the spine who have gout or risk factors for gout. Although gout often has a nonspecific appearance on MRI and CT scans, when a vertebral lesion demonstrates calcifications on CT or a low signal on T1- and T2-weighted MRI images, spinal gout should be considered.\textsuperscript{10}

**References**