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GRAND ROUNDS

Tophaceous gout of the lumbar spine mimicking a spinal meningioma

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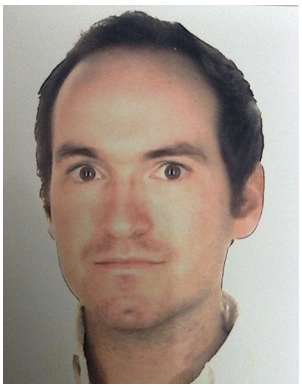
Abstract



Purpose Although gout is a common metabolic disorder, it usually affects distal joints of the appendicular skeleton. Axial spine involvement is rare, with only 131 cases reported in the literature. The authors report a rare case of lumbar spinal gout mimicking a spinal meningioma.
Methods A 77-year-old man with a history of gout presented with chronic low back pain and progressive paraparesis. Imaging revealed a lumbar spine compressive mass lesion with a dural tail signal. The differential diagnosis was thought to be straightforward favoring a spinal meningioma. Tophaceous gout was never considered. The presence of a dural tail associated with the lesion is an interesting detail of this case, that strongly misguided it and to the best of our knowledge it is the first one reported in the literature.
Results The patient underwent surgery and intra-operative findings were surprisingly different from those expected, revealing a chalky white mass lesion firmly adherent and compressing the dural sac. It was completely excised, leaving the dura intact. Histopathology confirmed the diagnosis of tophaceous gout. The patient was sent to physical therapy and had a complete remission of pain and neurological deficit, regaining his walking capacity.
Conclusion Although spinal gout is rare, it should be considered in the differential diagnosis for patients presenting with symptoms of spinal stenosis, a suspicion of neoplastic lesion of the spine, and a previous history of gout. Early diagnosis can ensure proper and timely medical management, perhaps avoiding neurological compromise and the need for surgery.

Keywords Tophaceous gout · Lumbar spine · Spinal meningioma · Low back pain

Case presentation

A 77-year-old man presented with a 6-month progressive paraparesis resulting in gait impairment for one week at admission. He also complained of increasing non-mechanic, low back pain, mostly at night, He denied history of recent trauma, radiating pain, and bowel or bladder dysfunction. There was also no history of recent infection or neoplastic disease. He had a medical history of gout with episodic pain crisis involving his first right metatarsal-phalangeal joint, arterial hypertension and dyslipidaemia. He did not have a rigorous compliance of his usual medical prescription of allopurinol and several times forgot to take

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it. Physical examination revealed significant tenderness in his lower back, mild paraparesis (motor strength grade 4/5), slightly worse on the left. He also exhibited bilateral decreased reflexes in both lower limbs and bilateral flexor plantar reflex. No sensorial deficit was found. He had a right 1st toe with a deformed joint.

Diagnostic imaging

Lumbar magnetic resonance imaging revealed a 3.7 cm dorsal extradural mass lesion in the spinal canal at the level of L3–L4 compressing the thecal sac posteriorly. The mass lesion was isointense on T1, hypointense on T2 and with heterogeneous peripheral enhancement after gadolinium administration (Fig. 1). Due to the location inside the spinal canal and the pattern of gadolinium enhancement similar to a dural tail (Fig. 1d), the first diagnosis suggested was a lumbar meningioma. The hypothesis of an abscess was also taken in consideration, mostly due to the periarticular enhancement of the L3–L4 and L4–L5 facet joints, after gadolinium administration, probably of inflammatory or infectious nature.

Review

Tophaceous gout is a common metabolic disorder in which abnormal production or impaired excretion of uric acid results in the deposition of monosodium urate crystals in the distal joints of the appendicular skeleton and soft tissues. The involvement of the axial spine is rare, with only 131 cases of cervical, thoracic and lumbar spine gout reported in the literature [12]. More than half of the cases reported (73 cases) are located in the lumbar spine. Because of its rareness, diagnosis is often delayed, even in cases of patients with a long history of gout. The diversity in the location of urate deposits implies that they can mimic neoplasm or abscess by mass effect, or cause instability and pain frequently seen in degenerative

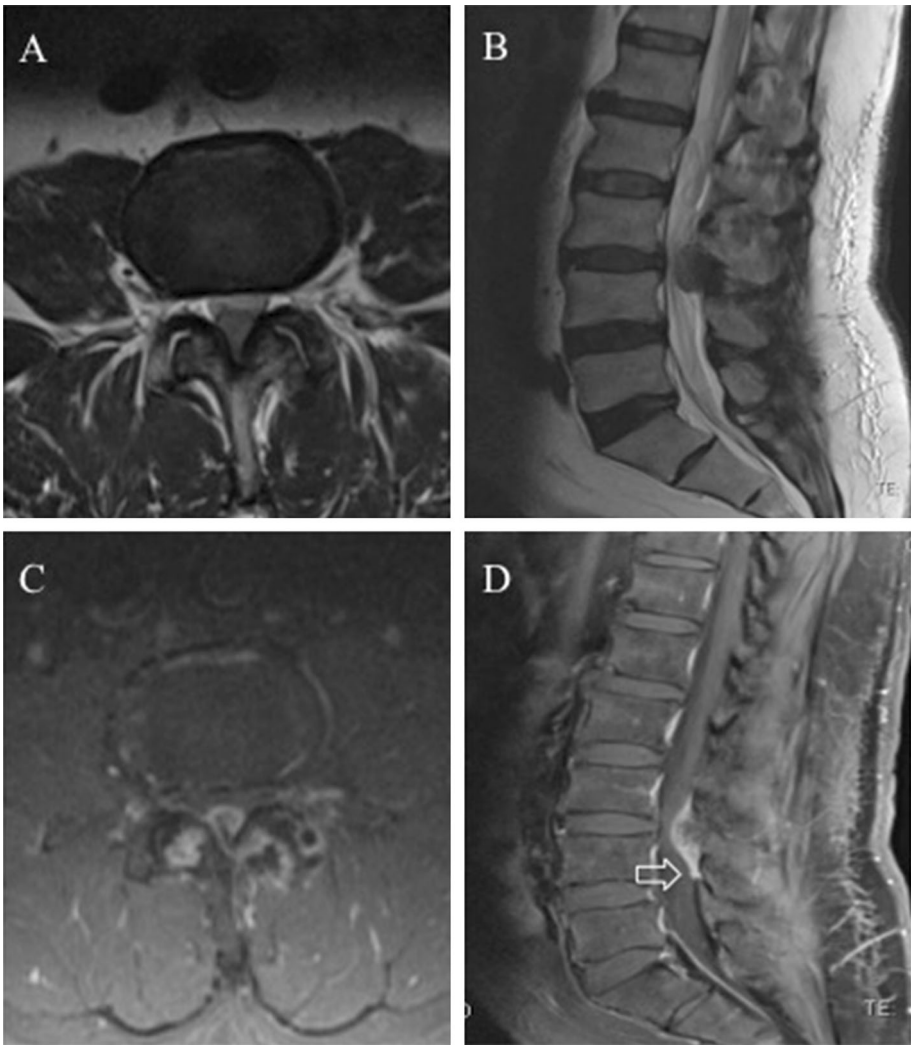

Fig. 1 Magnetic resonance imaging findings. T_2-weighted axial (**a**) and sagittal (**b**) images, and gadolinium contrast-enhanced (**c**) and **d** images, this last one depicting the dural tail enhancement pattern (*arrow*)

processes [3]. Therefore, diagnosis is only possible after examination of a biopsy specimen [6].

Gout is mainly a disease of middle-aged men, although asymptomatic hyperuricemia is tenfold more common than gout [10]. In the majority of the cases reported patients are between 44 and 74 years of age [12].

Although the specific pathophysiology of crystal deposition in the axial spine is not clear, authors like Bonaldi et al. [1] have hypothesized that degenerative disease of the spine may be a predisposing factor, as suggested by their observation that most cases involve the lumbar spine at the lumbosacral junction. This is in line with the review by Hout et al. [3], where most of the patients with lumbar disease involved L4 through S1 consistent with the possibility that inflammation associated with motion-related damage may create a favorable environment for urate deposition. Facet joints were also involved in the majority of cases.

Patients with spinal gout may present with acute, subacute, or chronic symptoms. In the review by Toprover et al. [12] of 131 patients with spinal gout in different locations, the most common presentation was back pain, which was present in 89 (68.5%) cases, usually in the area corresponding to the spinal level affected by urate deposition. In 85 (65.4%) patients some form of neurological deficit was present, ranging from radiculopathy, loss of sensation, motor weakness, bowel/bladder dysfunction, or quadriparesis. Surprisingly two patients were asymptomatic and were only diagnosed during autopsy for other causes of death [2, 7]. This means that in early stages or if hyperuricemia is well controlled by medication, patients can be asymptomatic. In the same review the duration of symptoms ranged from 1 day to 6 years. The fact that some patients can present with an acute neurological compromise suggests that the rate of crystal deposition can be fast and early diagnosis and treatment are essential [3].

Regarding imaging characteristics, spinal gout has been found in essentially every component of the vertebrae, including the facet joints, pedicles, lamina, vertebral body, intervertebral disc, epidural space (dorsal and ventral), ligamentum flavum, and rarely, in the intradural space [8].

Gouty tophi normally appear as a hypointense, homogenous mass associated with a joint on T1- and T2-weight MRI, which enhances with gadolinium because of vascularized reactive tissue in the tophus [1, 5].

Recently, a new imaging modality for gout called dual-energy CT (DECT) scanning has emerged. It has high sensitivity (91.9%) and specificity (85.4%) [4] identifying gout tophi. Further studies are warranted to determine if this new modality will be sufficient for diagnosis in the presence of a spinal mass on imaging and this way precludes the need for an invasive procedure and tissue diagnosis. At the moment, though, DECT is not generally accessible and most radiologists lack training interpreting the images [4].

Due to its diverse location and involvement of the several components of the spine, the differential diagnosis of spinal gout include epidural abscess, spondylodiscitis, metastatic disease, rheumatoid arthritis and dialysis-related amyloid spondyloarthropathy, just to name the most common [13].

In the majority of cases, definitive diagnosis of gout can only be made by histological examination of the biopsy material. Further confirmation by polarized light microscopy of negative birefringent urate crystals is mandatory for firmly establishing the diagnosis of gouty arthritis [9, 11].

Rationale for treatment

Standard management of gout is stabilization of acute attacks by colchicine, non-steroidal anti-inflammatory drugs or both, with urate-lowering therapy using allopurinol [9]. In spinal gout, patients with acute progressive neurological deterioration and radiographic evidence should be considered and recommended to undergo timely surgical decompression, and if necessary, surgical stabilization. These patients who underwent surgery had a favorable outcome [3].

In patients without neurological compromise, image-guided needle biopsy plays an important role as it helps confirmation of diagnosis and initiation of medical conservative therapy, preventing unnecessary surgical risks [3].

Regardless of surgical or conservative treatment, compliance with maintenance treatment of gout is mandatory for disease control.

Our patient had a progressive lower limb weakness for 6 months with subacute deterioration resulting in gait impairment and also lumbar axial pain. Since a subacute neurological deterioration was identified, surgical decompression was warranted.

The patient was also advised to better comply with his baseline medical treatment for gout disease with allopurinol.

Procedure

An L3–L4 laminectomy was made, removing chalky white material from the interlaminar space that spread into the spinal canal invading the yellow ligament and forming a firmly adherent lesion to the dura mater. The joint facets were also infiltrated and destroyed bilaterally. The extradural mass lesion compressing the dural sac was completely excised, leaving the dura intact (Fig. 2). The specimen was sent to pathology.

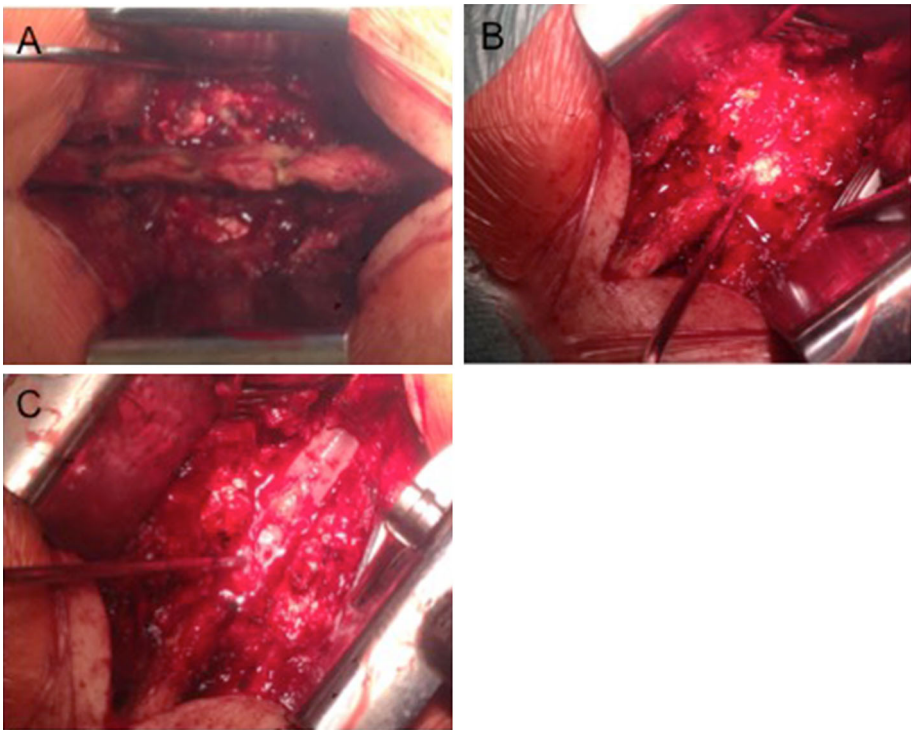

Fig. 2 Intraoperative photographs. Chalky white material infiltrating the facet joints, the interlaminar space (**a**) and spreading into the spinal canal invading the yellow ligament and forming a firmly adherent mass lesion to the duramater (**b**). The depression in the dural sac after total removal of the gout tophus (**c**)

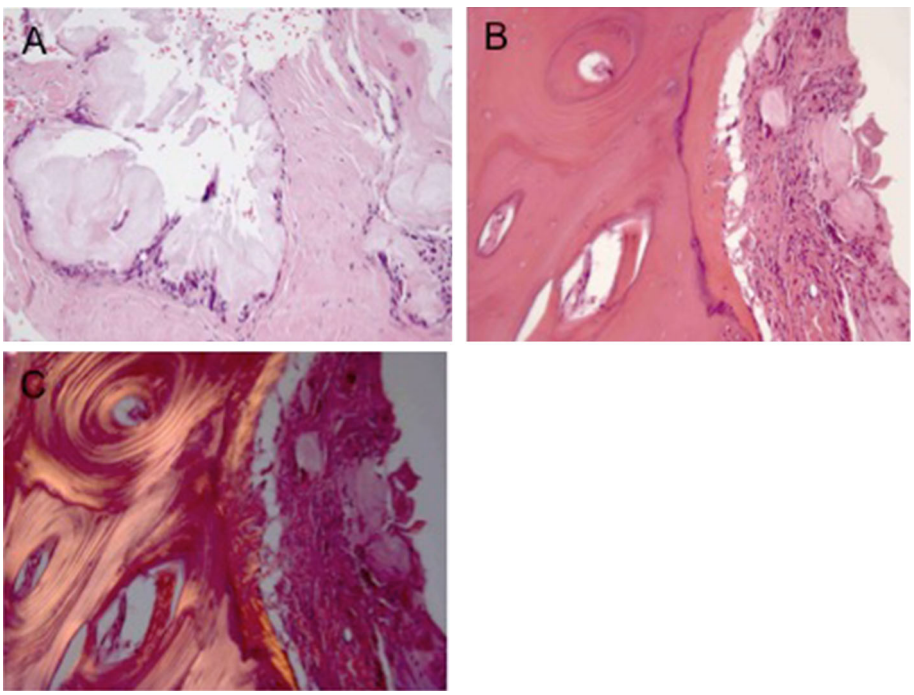

Fig. 3 Photomicrographs showing frozen sections H&E ×200, extensive deposition of an amorphous, basophil material in relation with a multinucleated foreign-body giant cell inflammatory reaction—Tophus (**a**). H&E ×100, transition from normal osseous tissue (on the *left*) to urate crystals deposition, tophus (on the *right*) (**b**), Polarized light microscopy ×100, positive birefringence of osseous tissue on the *left* vs. negative birefringence tophaceous material on the *right* (**c**)

Histological examination of the resected specimen revealed extensive deposition of an amorphous, basophil, non-birefringent material, characteristic of gout, in relation with a multinucleated foreign-body giant cell inflammatory reaction, within the osseous tissue, paravertebral and intracanalar soft tissues (Fig. 3). These findings allowed the diagnosis of tophaceous gout of the lumbar spine.

Outcome

The patient was sent to physical therapy and made a good recovery with a complete remission of pain and neurological deficit, regaining his walking capacity. The 3-month follow-up lumbar MRI confirmed complete removal of the epidural mass lesion with no signs of spinal

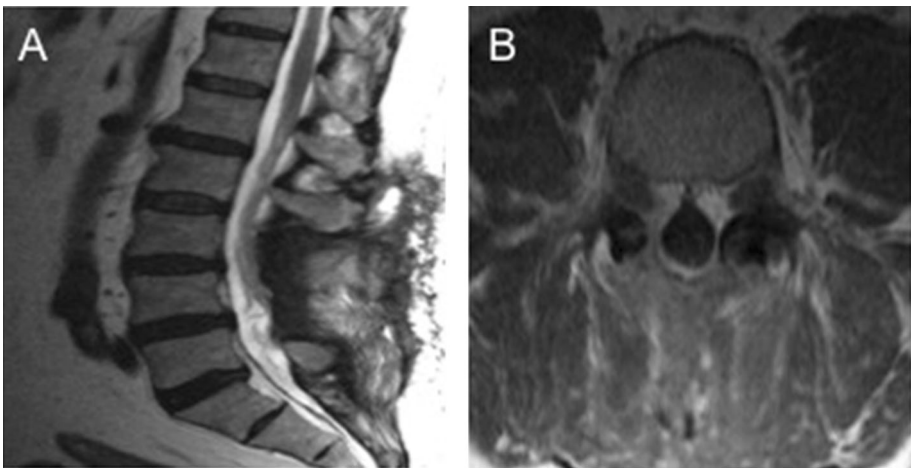

Fig. 4 3-month follow-up lumbar MRI. T_2-weighted sagittal (**a**) and, gadolinium contrast-enhanced axial (**b**) images showing complete removal of the epidural mass lesion

instability (Fig. 4). He resumed his medication for hyperuricemia according to his physician.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflicts of interest.

Informed consent The patient has consented to the submission of the case report to the journal.

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