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### **Case Report**

# A unique presentation of acute tophaceous gout in the lumbar spine causing cauda equina syndrome $^{\Rightarrow, \Rightarrow \Rightarrow, \star, \star, \dagger}$

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#### ABSTRACT

Gout is a common metabolic disease characterized by the deposition of monosodium urate (MSU) crystals and typically affects the peripheral joint, rarely involving the axial skeleton. We present a rare case of acute tophaceous gout in the lumbar spine causing cauda equina syndrome. A 60-year-old man with a history of gout and prior admissions for polyarticular gout flare presented with acute onset of bilateral lower limb numbness and weakness. He underwent surgical decompression with drainage of the epidural collection, with histology consistent with tophaceous gout. The patient made a full recovery postoperatively and was discharged uneventfully. Due to the high initial suspicion for gout, early spinal decompression surgery was performed, and the patient was started on medical therapy. Spinal tophaceous should be considered in the list of different diagnoses of spinal epidural masses especially in the context of a history of gouty arthritis.

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#### Introduction

Gout is an inflammatory arthritis associated with the presence of monosodium urate (MSU) crystal deposits in the synovial fluid or soft tissue. Gouty tophi are soft tissue masses of urate crystal deposits in and around the joints, which form in the chronic phase of the disease. Gout can also involve the axial joints, although this is relatively rare [1]. Spinal gout cases can present similarly to other spinal pathology—such as radiculopathy, paraparesis, or back pain—making early detection challenging. We present a case report of tophaceous

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<sup>\*</sup> No patient identifiers are present in the case report.

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gout in the spinal canal causing cauda equina syndrome. In this case, the patient's clinical presentation and past medical history played a critical role in reaching the diagnosis radiologically.

#### **Case report**

A 60-year-old gentleman with a background history of hypertension, hyperlipidemia, and chronic gout was admitted to our hospital for emergency neurological evaluation.

He suffered from acute onset bilateral lower limb weakness and numbness for 1 day. This was associated with acute urinary retention. Physical examination revealed a temperature of 36.5°C, heart rate of 68, respiratory rate of 17, and blood pressure of 137/78 mm Hg. The patient demonstrated weakness and numbness in the bilateral lower limbs, with loss of deep tendon reflex of both knees and ankles. Further history and physical examination revealed saddle anesthesia and a laxed anal tone.

His laboratory data indicated raised inflammatory markers of CRP 144 mg/L ( $\leq$  4.9), ESR > 130 (1-10 mm/h), and total white blood cell count at 23.02/L (2.00-10.00 × 10(9)/L). Uric acid level was elevated, 526 umol (202-417). Imaging studies including plain film and magnetic resonance imaging (MRI) of the lumbar spine were performed.

Lumbar spine radiograph showed lumbar spondylosis and mild anterolisthesis of L4 over L5. MRI showed L3-4 and L4-5 facet joint effusion and posterior epidural collection extending from L2 to S1 resulting in severe stenosis and causing equina compression (Fig. 1). There were lobulated foci in the epidural collection manifesting as hypointense signal on T1, intermediate signal on T2, and hyperintense and marginal enhancement after gadolinium contrast administration (Figs. 2 and 3).

The initial differential diagnoses were epidural abscess and spinal tophaceous gout. Given the preoperative suspicion for tophaceous gout in view of the patient's past medical history, the patient underwent emergency decompression and laminotomy, medial facetectomy, flavectomy, drainage of tophaceous material, and decompression of cauda equina from L2 to S1 the next day.

Intraoperative findings revealed tophaceous material in the facet joints bilaterally extending into the spinal canal causing severe compression of the cauda equina. Histology revealed crystal deposits consistent with gouty tophi (Fig. 5). The patient was subsequently referred to rheumatology, and started on oral Colchicine 500 mcg OM and Febuxostat 80 mg OM. The patient was discharged well.

#### Discussion

Gouty tophi in the spinal canal are relatively rare, with a prevalence of 14% [1]. Axial gout has also been reported to be more prevalent in males, comprising of up to 73% in a study [1]. The lumbar spine is the most commonly affected region, followed by the cervical and then the thoracic spine [2–4]. Spinal gout can affect any part of the vertebra, for example, the epidural space, facet joint, pedicle, or ligamentum flavum [5,6].

Spinal imaging can be useful in diagnosing spinal gout. Radiograph is usually the first line of investigation for pain related to spine. Radiographic features of spinal gout are often nonspecific and may include degenerative spondylosis, discovertebral erosions, bone destruction causing joint subluxation, spinal deformity, spontaneous fusion, and occasionally pathological fractures [6].

MR features of gouty tophi usually present as low or intermediate signal on T1-weighted images and variable intensity on T2-weighted images. Gouty tophi deposition may lead to spinal canal stenosis, narrowing of the neural foramina, and hypertrophy of the ligamentum flavum. There may also be juxta-articular bony erosion of the facet joints with associated effusion. Postcontrast administration, spinal gouty tophi may



Fig. 1 – Spinal tophaceous gout in a 60-year-old man presenting with cauda equina syndrome. Posterior epidural collection and severe spinal canal stenosis with crowding of the cauda equina demonstrated along with the presence of bilateral L3-4 facet joint effusions with rim-enhancement. (A) Sagittal TIRM image of the lumbar spine showing facet joints effusion. (B) Axial T2-weighted image at the level of L3-4 showing posterior epidural collection displacing the nerve roots. (C) Axial T1-weighted fat-saturated image showing facet joints effusions with rim-enhancement at the level of L3-4.



Fig. 2 – Spinal tophaceous gout in a 60-year-old man presenting with cauda equina syndrome. Images show heterogeneous collection in the posterior epidural space from L2 to S1 which contain a few lobulated low signal foci, possibly calcification or gouty deposits. There is marginal contrast enhancement seen. (A) Sagittal T1-weighted image of the lumbar spine. (B) Sagittal T2-weighted image of the lumbar spine. (C) Sagittal TIRM image of the lumbar spine. (D) Sagittal T1-weighted image postcontrast.



Fig. 3 – Spinal tophaceous gout in a 60-year-old man presenting with cauda equina syndrome. Images show a low signal focus in the epidural collection, possibly representing calcification or gouty tophus, causing compression on the cauda equina. (A) Axial T1-weighted image at the level of L2-3. (B) Axial T2-weighted image at the level of L2-3.

show homogeneous enhancement representing vascularized tissue within the tophus or peripheral enhancement due to hypervascular tissue surrounding the tophus [2,7–11].

In our case, MRI showed lobulated lesions in the posterior epidural space from L2 to S1 which exhibits low intensity on T1-weighted images, intermediate intensity on T2-weighted images, and marginal contrast enhancement after gadolinium administration (Figs. 2 and 3).

CT is superior to MRI for the detection of spinal gout as it can detect tiny erosions caused by tophi [2,8]. The characteristic CT findings are articular erosions with sclerotic margins and tophi formation. Recent development of spectral imaging such as dual-energy CT (DECT) enables detection of MSU crystal deposition. DECT also permits a quantitative assessment of deposits that can be used for follow-up of the patients. CT however was not performed in our case.

Histological or cytological analysis remains the gold standard for a definite diagnosis [12]. The sample is usually obtained intraoperatively and should be handled fresh or preserved in alcohol as the MSU is soluble and is destroyed in formalin fixation. Strongly negative birefringent urate crystals seen under the polarizing microscope are pathognomonic of gout. In our case, the sample was unfortunately submitted in formalin therefore was not examined under the polarizing microscope. The specimen, however, showed pockets of crystalloid material surrounded by inflammatory cells with areas of calcifications, which were consistent with tophaceous gout (Fig. 5).

Patients can be asymptomatic or they may present with a variety of neurological symptoms such as radiculopathy, backache, or paraparesis secondary to root or cord compression. Given the broad range of presenting symptoms, these may mimic degenerative, inflammatory, or a neoplastic process, confounding diagnosis.

One of the mimickers of spinal gout is degenerative spondylosis. This is often seen in the cervical and lumbar spine. Although facet joint effusion and endplate changes can occur in degenerative spondylosis, abscess, or epidural collection is not a feature of degenerative spondylosis. Nonetheless, it should be noted that patients with spinal gout may have concomitant degenerative changes of the spine.

Another common mimicker of spinal gout is infection such as vertebral osteomyelitis and spondylodiscitis, which should be urgently excluded. Both conditions can present similarly with fever and back pain but can be easily differentiated with DECT, along with patient history and histological findings.



Fig. 4 – Spinal tophaceous gout in a 60-year-old man presenting with cauda equina syndrome. No convincing restricted diffusion of the epidural collection. (A) Sagittal DWI sequence. (B) Sagittal ADC sequence.

Although both can show peripheral contrast enhancement on MRI, diffusion-weighted imaging (DWI) usually reveals restricted diffusion in infective collection but not in gouty spine. In our case, the epidural collection did not demonstrate restricted diffusion on DWI which was therefore more in favor of gouty spine (Fig. 4).

Spinal tumor is another possible mimicker of spinal gout. This can be differentiated based on biochemical markers, histology, and past medical history. Depending on the type of the tumor, some may show internal enhancement which can be differentiated from spinal gouty tophus.

Spinal hematoma is also a differential of spinal gout, and should be considered in the context of trauma or iatrogenic injury. Both can show blooming artifact on MRI gradient echo imaging but spinal gout tophus demonstrates enhancement on postcontrast sequences, which is not commonly seen in the case of a hematoma.

The initial working diagnosis of this patient was septic arthritis of the facet joints with an epidural abscess because there were facet joint effusions and posterior epidural collection. The patient's inflammatory markers were also elevated. Gouty arthritic involvement of the spine was also a consideration given the patient's past medical history.



Fig. 5 – H&E histology staining of epidural mass lesion of a 60-year-old man presenting with spinal tophaceous gout. (A) Well-defined deposits of lightly basophilic fibrillary material. (B) Ligamentous tissue with well-defined deposits of lightly basophilic fibrillary material. (C) Fragmented deposits of crystalloid deposits surrounded by inflammatory cells. (D) Crystalloid material surrounded by inflammatory cells with areas of calcifications.

Radiological suspicion of gout etiology will guide the intraoperative handling of histological specimens. In this case, the strong suspicion for gouty tophi in the spine resulted in early decompression surgery for cauda equina syndrome.

#### Conclusion

Spinal gouty tophus should be considered in the different diagnoses of spinal epidural lesions, especially in patients with a history of chronic tophaceous gout. Although there are many mimickers of gouty spine, the patient's past medical history, clinical presentation, and the relevant imaging findings were pivotal in helping to reach the correct diagnosis. Surgical intervention is the mainstay of treatment for cord decompression. Such patients should also be reviewed for urate-lowering medical management.

#### Patient consent

Informed consent to publish the case and photographs without patient identifiers was obtained from the patient.

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