CASE REPORT

Spinal tophaceous gout presenting in a young adult without pain

Zaid Al-Jebaje,¹ John Mellon Elibol,² Jonathan Peters,³ Ali Alameri¹

¹Department of Internal Medicine, University at Buffalo Catholic Health, Buffalo, New York, USA

²Department of Internal Medicine, Mercy Hospital of Buffalo, Buffalo, New York, USA

³University at Buffalo School of Medicine and Biomedical Sciences, Buffalo, New York, USA

Correspondence to
Dr John Mellon Elibol,
jelibol36@gmail.com

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SUMMARY

A 26-year-old man with a medical history of gout and morbid obesity presented with a 7-day history of decreased sensation to light touch and temperature from the feet to the level of the nipples. He also noted incomplete voiding. Laboratory investigations showed an elevated serum uric acid level (10.4 mg/dL, reference range: 3.8–8.7 mg/dL) as well as negative rapid plasma reagin and rheumatoid factor. MRI showed inflammatory changes on multiple spinal levels. Laminectomy was performed, with follow-up biopsy revealing multiple multinucleated giant cells and monosodium urate (MSU) crystals. He was ultimately diagnosed with spinal gout. Patient's symptoms did not resolve immediately after surgery. Yet with the administration of intravenous glucocorticoids and a course of non-steroidal anti-inflammatory drugs, he slowly regained sensation, leaving the hospital with complete resolution of symptoms.

BACKGROUND

Spinal gout is a relatively rare condition, with only 113 cases reported in the literature from 1950 to 2012.¹ Yet with an observed doubling in the prevalence of gout in the USA, this unlikely manifestation should be on the differential in patients with presumed spinal pathology.² This case is especially interesting because unlike 75.8% of reported cases of spinal gout, this patient did not present with pain and a neurological deficit. Further, this 26 year-old patient fell well below the mean patient age of 60.3 years.¹

This patient received a laminectomy, which resulted in the acute loss of sensation to the level of the sternum and new-onset weakness in the arms. Only after a 2-day course of intravenous steroids his neurological symptoms resolve. Other case studies have shown medical treatment efficacious in alleviation of symptoms.^{3,4}

CASE PRESENTATION

A 26-year-old Caucasian man with a medical history of gout and morbid obesity (body mass index 52.9) presented with an ascending loss of sensation in his lower extremities for 7 days from the feet to the level of his nipples. He denied weakness, pain, confusion, recent trauma or illness or any sick contacts. He did mention that he had recently developed urinary retention. The patient reported that 2 years prior he experienced right hand and finger pain, stiffness and swelling that resolved

spontaneously after 1 week. He stated that he never received a definitive diagnosis at that time. These symptoms did not recur. The patient also reported a family history of gout in his father. The patient's diet was marked by heavy consumption of red meat and cheese. The patient reported occasional alcohol use, approximately 3–4 beers per week.

On physical examination, decreased light touch and temperature sensation to the lower abdomen were noted. 4/5 weakness in the right triceps and right deltoid were also noted. Reflexes were intact throughout. Other aspects of the examination were within normal limits.

INVESTIGATIONS

At the time of presentation, the patient's haemoglobin was 12 g/dL, white cell count $0.0084 \times 10^9/L$, creatinine 0.93 mg/dL (range: 0.9–1.3 mg/dL) and blood urea nitrogen 17 mg/dL (range: 7–25 mg/dL). His temperature was 98.5°F. His C-reactive protein was 22.3 mg/L and erythrocyte sedimentation rate was 102 mm/hour. Rheumatoid factor, rapid plasma reagin and human leukocyte antigen-B27 were all negative. Uric acid was elevated at 10.4 mg/dL.

An MRI of cervical (figure 1), thoracic and lumbar spine (figure 2) with and without contrast revealed multilevel spondylitic change and disc disease at C6–C7, C7–T1, T12–L1 as well as spinal stenosis at C7–T1 and T12–L1. Also noted were marked inflammatory changes affecting the sacroiliac and facet joints.

DIFFERENTIAL DIAGNOSIS

On initial presentation, the differential diagnosis included acute transverse myelitis, Guillain-Barre syndrome (GBS), osteomyelitis and sensory neuropathy. After the MRI was read, inflammatory changes were noted and reactive arthritis had to be ruled out as well as demyelinating disease, GBS and transverse myelitis. Prior to the first laminectomy, the preoperative diagnosis was spinal stenosis C3–T1 with herniated disk at C6–C7. Final diagnosis of spinal tophaceous gout was only reached after the biopsy results returned (figure 3 and 4).

TREATMENT

Patient was admitted to the neurosurgery service, which performed two emergent decompressive laminectomies, the first of C3–T1, removing both the lamina and spinous processes on hospital day 3. The surgeon noted a crystalline substance at every level, and was surprised to find that the herniation

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Figure 1 MRI of cervical spine without contrast revealed multilevel cervical spondylitic change and disc disease with the major finding noted at C6–C7 where there is tophus formation leading to severe central canal and right C7 foraminal stenosis. There was also posterior osteophyte formation seen at C2–C3 causing an impression on the anterior aspect of the thecal sac.

noted at C6–C7 was in fact this very same substance. On hospital day 23, the patient returned to surgery for a laminectomy of T7–T11; the surgeon again removed the lamina and spinous processes. No intraoperative complications were noted during either procedure.

OUTCOME AND FOLLOW-UP

The patient developed new onset weakness of the upper and lower extremities and loss of sensation to the level of the sternum immediately following the first surgery. He was urgently transferred to the intensive care unit. The patient was then started on 4 mg of intravenous Dexamethasone every 6 hours and reported significant improvement in sensation over the following 2 days. Allopurinol 150 mg PO daily for 90 days was added to his medication regimen for symptom prevention. He was also placed on a course of non-steroidal anti-inflammatory drugs (NSAIDs) 375 mg two times per day for 90 days to manage the acute flare and to prevent further flares, as introduction of xanthine oxidase

Figure 2 Postcontrast MRI of lumbar spine at the level of sacroiliac joint. Bilateral sacroiliac joint inflammatory involvement noted with white arrows.

Figure 3 Processed tissue under polarised light. Magnification 200x. Mixed needle-shaped and clumped birefringent crystals consistent with monosodium urate.

inhibitors may precipitate new events.⁵ Colchicine was considered as a second-line agent but ultimately not used.

C3–T1 biopsy demonstrated ‘fibrous tissue associated with abundant foreign body giant cells.’ Likewise, biopsy specimen from T7–T11 showed monosodium urate (MSU) crystals, confirming gout (figure 3 and 4).

Postlaminectomy, CT spine without contrast revealed adequate decompression of the thecal sac and no perivertebral fluid collection. By hospital day 10, his strength returned to 4+/5 bilaterally.

He was discharged on hospital day 29. He is currently undergoing physical therapy at a long-term care facility. He will require additional spinal stabilisation surgeries in the future.

Figure 4 Unstained (unprocessed) slide under polarised light. Magnification 200x. Needle-shaped negatively birefringent monosodium urate crystals consistent with gout.

DISCUSSION

Gout is a deposition of MSU crystals in the bone, joint space or skin, which presents with arthritic symptoms of pain and decreased range of motion.⁶ It generally manifests in the extremities, and can form tophi, which are simply collections of these crystals.⁶ Pain in the first metatarsal joint is often the initial symptom. Spinal gout is a much more rare type of gout, one that has only been described in 113 cases between 1950 and 2012.¹ Yet a recent cross-sectional study showed CT changes consistent with axial gout in 35% of patients with chronic appendicular gout (n=48).⁷ It is thought to develop through a similar mechanism as peripheral gout, with the facet joint being the first site to develop MSU crystallisation.⁸

In a review of cases of spinal gout, the most common presenting symptom was pain with neurological deficit (76%).¹ For this patient, the tophi primarily led to spinal compression, leading to symptoms of sensation loss, urinary retention and weakness.^{9 10} Uric acid was elevated in 70% of patients.¹ MRI and CT investigations are considered non-specific, but may show homogenous changes on T1 and T2 imaging.¹¹ Likewise, dual energy CT, which is used for identifying appendicular gout, is not ideal for imaging of spinal gout due to poor sensitivity for MSU crystals.¹² The gold standard diagnosis is through biopsy of the spine and pathological analysis showing MSU crystals that are negatively birefringent under polarised light.¹ Treatments are either medical or surgical. Medical treatment includes both intravenous glucocorticoids and an NSAID such as colchicine to manage the acute flare and allopurinol to reduce production of uric acid chronically.^{8 13} Spinal gout has been successfully managed without surgery if treated in early stages or if it is a benign presentation.¹⁴ Spinal decompression is generally a

second-line approach to managing spinal gout, but it is necessitated in the presence of emergent symptoms.^{8 14 15} As with this patient, medical treatment is typically continued after surgery, improving symptoms and reducing the risk of future flares.¹⁴

While infection or trauma is a more common cause of sensation loss, spinal gout should be a consideration in patients with new-onset claudication or weakness.

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Learning points

- While gout is well known to affect the lower extremity joints, tophi can appear in unexpected places with unexpected symptoms.
- A spinal MRI finding of multilevel inflammatory changes should trigger suspicion of spinal gout, especially in patients with a previous history or multiple risk factors.
- Spinal gout is a treatable manifestation of a chronic disease, best handled by a multidisciplinary team including both internists and surgeons.
- We present a case of spinal gout in a patient with an uncommon presentation.

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