CASE REPORT

Spinal tophaceous gout encasing the thoracic spinal cord

Nagina Subrati, Melissa C Werndle, Christos M Tolias

Department of Neurosurgery,
Kings College London,
London, UK

Correspondence to
Nagina Subrati, nagina.
subrati@gmail.com

Accepted 17 September 2016

SUMMARY

Gout rarely compresses the thoracic spinal cord. A 43-year-old man presented with lower limb paraparesis. MRI showed a soft tissue swelling at the level of T10/T11. He was managed with a laminectomy and evacuation of a presumed abscess and started on intravenous antibiotics. However, histology confirmed tophaceous gout.

BACKGROUND

Patients rarely present with fever and thoracic cord compression secondary to tophaceous gout in the spine. Gout usually manifests as acute inflammation in peripheral joints. The classic triad of fever, spinal pain and neurological deficit is often associated with an epidural abscess. Our case report describes an unexpected diagnosis of tophaceous gout in the thoracic spine.

CASE PRESENTATION**History**

A 43-year-old man presented with a 4-week history of worsening back pain. He had a 2-week history of progressive lower limb weakness and frequent falls. On admission he was unable to walk and incontinent of urine. There was no preceding trauma.

He had a past medical history of hypertension, gout, obesity (weight 107 kg and height 183 cm) and paranoid schizophrenia. Gout was diagnosed 4 years ago; he reported acute flares every 6 months, which he self-managed with ibuprofen and naproxen. He did not attend regular rheumatology clinics. His regular medications were olanzapine (poor adherence), co-dydramol and non-steroidal anti-inflammatory drugs (NSAIDs). He could not recall and did not take his antihypertensive.

There was no significant family history. He had a high alcohol intake and usually consumed half a bottle of vodka per day; however, he denied drinking alcohol for 2 months. He was unemployed, a non-smoker, an ex-drug misuser and had poor health seeking behaviour.

Examination

On general examination, he was mildly confused, lungs were clear to auscultation, respiratory rate was 26 breaths per minute, oxygen saturation was 96%, there were no heart murmurs, heart rate was 96 bpm, blood pressure was 161/80 mm Hg, temperature was 37.7°C.

Upper limb and cranial nerve examination was unremarkable. Lower limb examination revealed increased tone, reduced sensation, hyperreflexia and positive Babinski sign. There was weakness throughout the lower limbs; Medical Research Council (MRC) grade 1/5 above the knee and grade 0/5 below the knee bilaterally. He had reduced anal tone with faecal loading and required a urinary catheter. Sensation was reduced to the level of his umbilicus.

On later examination of our patient, we noted tophi at the olecranon bursa of both elbows and the first MTP joints. There were no acutely swollen joints or tophi around the ears.

INVESTIGATIONS

He underwent urgent imaging of the lumbar and lower thoracic spine ([figure 1](#)). On sagittal and axial T2-weighted MRI at the T10/11 level, there is evidence of bilateral mixed intensity abnormal soft tissue, which appears to involve predominantly the left T11 pedicle and, to a lesser extent, the right pedicle. The soft tissue has caused moderate thoracic cord compression at this level. Contrast was not administered due to acute kidney injury (AKI).

The patient was investigated for a source of infection. Inflammatory markers were raised on admission; C reactive protein (CRP) was 70 mg/L and white cell count (WCC) was $13 \times 10^9/L$. Serum uric acid and erythrocyte sedimentation rate (ESR) raised, 7.57 mg/dL and 111 mm/hour, respectively. Blood cultures were negative and no acid-fast bacillus organisms were detected. No further serology was requested.

He presented with AKI; creatinine was 126 $\mu\text{mol/L}$, urea was 31.7 mmol/L and estimated glomerular filtration rate (eGFR) was 55 mL/min. Chest X-ray, abdominal ultrasound and echocardiogram demonstrated no abnormalities.

DIFFERENTIAL DIAGNOSIS

The radiological and neurosurgical differential diagnosis included an epidural abscess or metastasis. At this point, tophaceous gout was not considered.

TREATMENT**Operative management**

He underwent an urgent T10 laminectomy and an evacuation of a suspected epidural abscess. During the laminectomy frank pus was noted. White solid material was found in the epidural space with surrounding membrane adherent to the dura mater. Specimens were obtained and sent for neuropathology and microbiology. The thoracic spinal cord was

CrossMark

To cite: Subrati N, Werndle MC, Tolias CM. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2016-214869

Figure 1 Sagittal T2-weighted MRI spine (left) and axial T2-weighted MRI spine (right). Red arrows indicate areas of pathology.

adequately decompressed with no intraoperative complications. Our microbiology team advised we start intravenous vancomycin, ceftriaxone and metronidazole for the treatment of an epidural abscess.

OUTCOME AND FOLLOW-UP

Postoperatively he regained some power in the lower limbs (MRC grade 2/5 above the knee on the right, 1/5 above the knee on the left and 0/5 below the knee bilaterally).

He was started on an antihypertensive. The AKI improved on stopping NSAIDs and encouraging rehydration (creatinine 50 $\mu\text{mol/L}$, urea 4.7 mmol/L and eGFR 90 mL/min). The inflammatory markers also improved (CRP 9.8 mg/L and WCC $4.83 \times 10^9/\text{L}$).

The psychiatrist increased his dose of olanzapine from 15 mg daily to 20 mg daily for on-going paranoia. We suspect he was not taking his medication in the community.

Neuropathology demonstrated no tumour, pus or microorganisms. Multiple islands of eosinophilic amorphous material surrounded by partly palisading histiocytes with few multinucleated giant cells suggestive of gouty tophi were noted. There was birefringence seen under polarised light. Histology confirmed tophaceous gout (see figure 2).

The rheumatologist advised starting 100 mg of allopurinol daily with 500 μg of colchicine twice a day (this was given to prevent a flare of gout while starting the allopurinol). The

Figure 2 H&E stain. Multiple islands of eosinophilic amorphous material surrounded by partly palisading histiocytes with few multinucleated giant cells.

allopurinol was titrated up every 3–4 weeks with an aim for a serum uric acid level of $<5 \text{ mg/dL}$. NSAIDs were contraindicated in this patient as he was recovering from AKI secondary to the over use of NSAIDs.

The patient was transferred to his local hospital and referred to the spinal injury unit for rehabilitation.

DISCUSSION

Gout is a common inflammatory arthropathy. Epidemiological studies show that the prevalence of gout is increasing worldwide. In the UK the prevalence is estimated to be 1.4%. Gout predominantly affects people in the fourth decade of life and it commonly affects males more than females (3–4:1).¹

Gout usually affects the appendicular skeleton, including the first metatarsal, ankle, knee and elbow joints. It rarely affects the spine and even more rarely the thoracic spine. Clinical manifestations of gout in the spine can include myelopathy, radiculopathy and, in some cases, fever.¹

A literature review in 2010 identified 75 published cases of tophaceous gout in the spine. Spinal gout is probably under diagnosed. A study that imaged the spine in patients with gout demonstrated a high prevalence of asymptomatic spinal gout. The most commonly affected region is the lumbar spine (55%), followed by the cervical spine (24%) and thoracic spine (21%). A literature search demonstrated 14 published cases of symptomatic thoracic gout.^{2–3}

Gout is a disorder of purine metabolism. Elevated levels of serum uric acid cause the formation and deposition of monosodium urate (MSU) crystals in and around the synovial joints. The MSU crystals exhibit strong negative birefringence under polarised light, which is characteristic of gout and the gold standard for diagnosis. These crystals form more readily at lower temperatures, thus gout commonly affects peripheral joints. The deposition of MSU crystals triggers a local inflammatory reaction within the joint.

Long-standing hyperuricaemia results in urate deposits in joints called tophi, which appear macroscopically as chalky white nodules. Tophi are generally found in articular, periarticular, bone, bursal and cutaneous tissue.^{2–4}

The increase in conditions that promote hyperuricaemia, such as hypertension, type 2 diabetes mellitus, metabolic syndrome, alcoholism, hyperlipidaemia, obesity and chronic kidney disease have contributed to the higher prevalence of gout.⁴

There are no specific radiological features for gout in the spine. On the plain radiograph, gout may appear as bony erosions and vertebral degeneration. On CT and MRI, features can include bone erosions; joint subluxation; spinal deformity; osteophytes and pathological fractures. The abnormal soft tissue at the level of T10/T11 in our patient was of mixed intensity on T2-weighted MRI. Others have found, on T1-weighted MRI, tophi may appear isointense to hypointense, on T2-weighted imaging tophi signal intensity can vary and on T1-weighted MRI plus gadolinium tophi enhances. The high protein content in the centre of tophi may cause the hyperintensity on T2-weighted MRI and the low intensity may be secondary to calcifications, urate crystals and fibrous tissue.²

The initial treatment of an acute flare of gout depends on the patient's comorbidities. Patients without contraindications (eg, chronic kidney disease or peptic ulcer) can be started on an NSAID. The use of non-selective NSAIDs is recommended, as these are inexpensive and easily obtained. NSAIDs have been found to be most effective when started within 48 hours of an acute flare. The dose of NSAIDs can be tapered down when symptoms begin to improve.⁵

Where NSAIDs cannot be tolerated, a low dose of colchicine can be administered. Colchicine has been shown to be most effective 12–24 hours after the onset of symptoms. Colchicine can be continued for the duration of the acute flare and can be stopped when symptoms subside.⁵

In patients who cannot take NSAIDs or colchicine, glucocorticoids can be considered. Glucocorticoids can be administered orally, intra-articularly or parenterally. In polyarticular disease, oral glucocorticoids are more likely to be beneficial. When one or two joints are affected and there are no contraindications for joint injections, the intra-articular route can be used. In patients who cannot take oral glucocorticoids and would not benefit from intra-articular injection, the parenteral route is an option; however, this is more difficult and costly to carry out.⁵

Gout needs to be adequately managed as long-standing hyperuricaemia can have catastrophic effects as demonstrated by this case. First and foremost patient education is key. The American

College of Rheumatology (ACR) recommends education on diet, lifestyle and comorbidities (eg, weight loss). Next, first-line urate lowering therapy is suggested; this can be either allopurinol or febuxostat. The serum urate level should be lowered enough to improve signs and symptoms. If the serum urate level remains high with continuing disease, then the advice is to add on uricosuric. If there is still no improvement, then consider pegloticase.⁵

In an acute flare of gout, the patient can present with fever and raised inflammatory markers. Acute tophaceous gout may simulate the macroscopic appearance of pus making it difficult to distinguish between gout and an epidural abscess.³ Our patient was surgically managed for cord compression and initially treated for an epidural abscess. On receiving the neuro-pathology findings, the patient was managed for gout and started on colchicine and allopurinol.¹

Acknowledgements NS would like to acknowledge her husband for all his support.

Contributors NS has written the case report submitted. MCW has edited the report and CMT was the responsible consultant for the patient and has also overviewed the case report.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Roddy E, Doherty M. Epidemiology of gout. *Arthritis Res Ther* 2010;12:223.
- 2 Chan ATS, Leung JLY, Sy ANL, et al. Thoracic spinal gout mimicking metastasis. *Hong Kong Med J* 2009;15:143–5.
- 3 Bonaldi VM, Duong H, Starr MR, et al. Tophaceous gout of the lumbar spine mimicking an epidural abscess: MR features. *AJNR Am J Neuroradiol* 1996;17:1949–52.
- 4 Neogi T, Jansen TLTA, Dalbeth N, et al. 2015 Gout classification criteria: an American College of Rheumatology/European League against Rheumatism Collaborative Initiative. *Arthritis and Rheumatology* 2015;67:2557–68.
- 5 Khanna D, Fitzgerald JD, Khanna PP, et al., American College of Rheumatology. 2012 American College of Rheumatology for management of gout. Part 1: systematic nonpharmacologic and pharmacologic therapeutic approaches to hyperuricemia. *Arthritis Care and research* 2012;64:1431–46.

Learning points

- ▶ The prevalence of gout is increasing but symptomatic tophaceous gout in the spine is still uncommon.
- ▶ If gout is poorly managed, it can lead to hyperuricaemia and deposition of tophi anywhere in the body that can have catastrophic results.
- ▶ Previous case reports describe gout in the spine, which may be mistaken for metastasis, an epidural abscess or spondylodiscitis.
- ▶ This case report highlights the rare causes of thoracic cord compression.
- ▶ Although we still recommend surgical management in these patients, it reminds us that in the feverish patient, with a history of gout, we should consider tophi in the spine to enable prompt rheumatology involvement and treatment.

Copyright 2016 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow