

## Involvement of the thoracic spine in tophaceous gout. A case report

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### ABSTRACT

*The presence of tophi involving the spine is an atypical complication of tophaceous gout and its diagnosis may be difficult. The authors present a case of tophaceous gout involving the thoracic spine and discuss the topic.*

### Case report

A 49-year-old Caucasian male was admitted to our hospital in August 1994, because of low back pain, muscle weakness and sensory impairment of the legs, predominantly on the right. The neurological symptoms had developed progressively over a 6-month period. He had had gout for about 5 years and presented underexcretion of uric acid. This was complicated by the rapid development of exuberant tophaceous deposits over the hands, feet, elbows and knees. He had no previous history of nephrolithiasis. He used to take colchicine, allopurinol and non-steroidal anti-inflammatory drugs on an irregular basis. At admission, a neurological examination showed the absence of Achilles and patellar deep-tendon reflexes bilaterally, decreased cutaneous sensation below the T9 level, and muscle weakness mainly involving the right leg and more evident in the distal segments. Sphincter control was normal.

Computed tomography (CT) and CT myelogram were suggestive of compression of the spinal cord by a mass from the T9 to the T10 level (Fig. 1). Magnetic resonance imaging (MRI)

showed the presence of an extradural mass inside the spinal canal. This mass was compressing the spinal cord from level T9 to T10. It was occupying approximately 50% of the spinal canal. There was isosignal intensity compared to the spinal cord on T1-weighted spin-echo and hyposignal intensity compared to cerebrospinal fluid on T2-weighted gradient-echo and spin-echo.

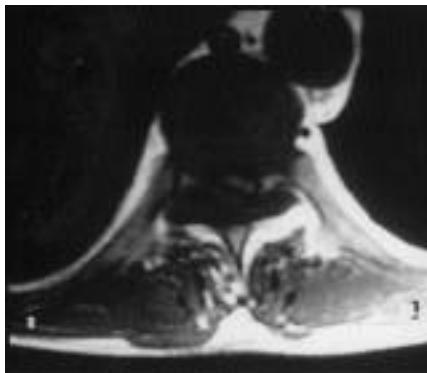
The medullar signal intensity was normal. The administration of paramagnetic contrast resulted on slightly peripheral heterogeneous enhancement (Figs. 2 and 3).

During a multi-specialist conference it was decided to adopt a non-surgical approach consisting of bedrest, along with allopurinol 600 mg/day, benzbromarone 100 mg/day, colchicine 1 mg/day and indomethacin 100 mg/day administered orally. However, he did not improve with this treatment. In July 1996 the patient underwent decompression laminectomy on the T9 to T11 levels. The surgeons observed the presence of a chalky white mass slightly adherent to the spinal cord. Analysis by polarized light microscopy revealed the presence of characteristic needle-shaped, strongly birefringent crystals of monosodium urate.

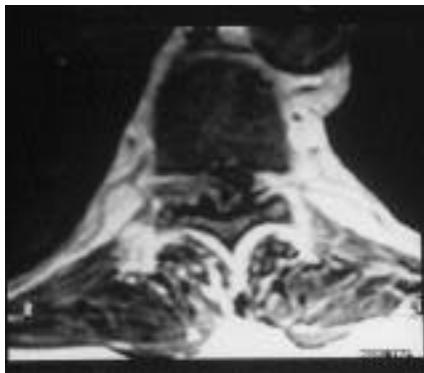
After surgery the therapeutic regimen was changed. The doses of the previous drugs were increased to 900 mg/day allopurinol per day, to 200 mg/day benzbromarone, to 2 mg/day colchicine and to 150 mg/day indomethacin. The outcome was good. There was regres-



**Fig. 1 (a)** CT scan of the thoracic spine at the T9-T10 level showing, in the soft tissue window, an extra-dural hyperdense, slightly heterogeneous mass that is compressing and pushing the dural sac to the front and to the left side. **(b)** CT myelogram of thoracic spine on T9-T10 level confirmed, on bone window, the compression on the dural sac done by the extra-dural mass. There are also zygapophyseal erosion and sclerosis.



**Fig. 2.** MRI of the thoracic spine at the T9-T10 level showed at pre-gadolinium T1-weighted spin-echo an oval mass with an intermediate signal, similar to the muscle's signal, dislocating the spinal cord to the front and to the left side.



**Fig. 3.** MRI of the thoracic spine at the T9-T10 level after the administration of intravenous gadolinium showed a heterogeneous peripheral enhancement of the mass.

sion of the neurological symptoms, the tophaceous deposits decreased, and the peripheral arthritis improved. The patient's uric acid level now is below 5 mg/dL and the MRI after surgery presented a laminectomy from the T9 to T11 levels and replacement of muscle layers by fat tissue (Fig. 4).

#### Discussion

The clinical presentation of gout is predominantly appendicular. There are very few reports of spinal involvement with gout (1). Thirty-seven similar case reports have been described so far (2). Spinal involvement by tophi has ranged from the level of C1 to S1 (3). The majority of patients who develop axial tophi usually have a longstanding history of severe polyarticular gout (4). The spinal involvement may be asymptomatic, as has been demonstrated by

an autopsy study (5). However, the patient may also present with a wide variety of symptoms ranging from isolated cervical, thoracic or lower back pain to neurological symptoms secondary to compression of the spinal cord or nerve root. These neurological symptoms can include paraparesis, quadripareisis and cauda equina syndrome (6, 7).

Compression of the neural structures is usually observed in patients with long-standing and polyarticular tophaceous gout. The presence of neurological symptoms in this subset of patients should promptly receive attention due to the possibility of compression of the

spinal cord or of nerve roots by a tophi (8). In patients with spinal involvement of tophaceous gout, conventional radiography may reveal erosions at the zygapophyseal joints, pedicle and on the vertebral plateau, or only signs of secondary degenerative changes, reactive bone sclerosis and space disc narrowing. Myelography may show spinal canal stenosis by an extra-dural mass on the levels involved by the tophi and may also evidence spinal cord or nerve root compression (9).

The CT scan is useful to show bony erosions with sclerotic margins inside the joint or peripherally. These erosions are usually associated with a surrounding lobulated hyperdense mass (10). MRI can provide complete information about the extension of the process and also about the involvement of soft tissue, synovium, cartilage and bone. The pattern of the lesions seems to be similar to that observed in storage diseases, like amyloidosis. There is a range from hypo- to hypersignal intensity on T2-weighted spin-echo images. Also, there should be intermediate signal, similar to the muscles' signal, on T1-weighted and on proton density imaging. This wide range of signals observed on T2-weighted images are probably related the amount of calcium on the tophi. The enhancement of the lesion after the administration of paramagnetic contrast is probably due to perilesional inflammation (9, 11).

In this case, the primary investigation with myelogram and CT myelogram suggested spinal cord compression by a mass whose presence was confirmed by MRI. The differential diagnosis should include a gouty tophus, a spinal tumor, and an epidural infection. As the patient had been suffering from tophaceous gout, the mass was considered to be a gouty tophus and he was managed clinically. There was hope for the regression of the tophi as a response to therapy. At the time of the diagnosis the treatment had been conservative in the only case reported in the literature, but in that case the treatment was not purely medical; the patient presented quadripareisis secondary to a type II odontoid fracture in the context of a tophaceous gout and the fracture was suc-



(a)



(b)

**Fig. 4.** Post-operative MRI on axial (a) and sagittal sections (b) and on T2-weighted images. Signs of laminectomy from the T9 to T11 levels and fatty tissue replacing the muscle layers can be seen. The spinal canal, dural sac and medulla emit a normal signal.

cessfully stabilized with the use of a collar, which resulted in the regaining of normal muscle strength (12). The first case of regression of a lesion secondary to urate deposits following medical treatment was reported only in 1997. The success of the use of allopurinol was observed by comparison of the MRI findings at the beginning of the treatment and one year later (13). Unfortunately, the response of our patient post-medical treatment was not favorable. Therefore, the medical team decided to conduct a decompression laminectomy along with medical therapy. Decompression laminectomy was the procedure of choice in most cases reported in the literature for the diagnosis and treatment of spinal gout with neurological symptoms (1,14). Therefore, it is advisable to consider a surgical approach, when dealing with a patient with a neurological deficit in the context of tophaceous gout, in order to avoid permanent incapacity. Nevertheless, the rarity of this gouty manifestation does not allow us to draw definite conclusions, because the experience in the literature is based on uncontrolled reports.

This case report should serve to remind

clinicians and rheumatologists of the possibility of spinal involvement in patients with longstanding tophaceous gout who present neurological symptoms. It also summarizes the main radiological features, which could be useful as a guide for the proper diagnosis. A correct diagnosis may require biopsy of the mass and therapy should include medical and surgical management.

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