Tophaceous gout producing spinal cord compression

Case report


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A case of thoracic paraplegia secondary to extradural tophaceous gout is presented. The ability of gout to compromise bone elements, periarticular tissues, and neural elements in the vertebral column is discussed.

KEY WORDS • hyperuricemia • gout • extradural tophus • paraplegia

The cause of hyperuricemia remains unknown. When manifest clinically, it presents as gout, typified by episodes of acute peripheral arthritis. As knowledge of this disease increases, its potential to involve nearly all organ systems becomes apparent. This case of tophaceous gout with associated spinal cord compression prompted a review of the literature. Gout is implicated as the underlying pathological process in an expanding and heterogeneous group of spinal disorders, ranging from chronic backache to tetraplegia. Such involvement further emphasizes the importance of long-term therapy in reducing serum uric acid levels.

Case Report

This 33-year-old man presented with a 5-year history of documented hyperuricemic gout involving the metatarsophalangeal, ankle, knee, shoulder, and elbow joints, with right pretibial and bilateral olecranon subcutaneous tophi. There was no family history of gouty or other arthritides, and no secondary cause for hyperuricemia was demonstrated. Previous treatment had consisted of intermittent courses of colchicine and indomethacin for acute episodes of arthritis. Allopurinol or uricosuric agents were not prescribed. Prior to his admission, the patient had experienced a typical exacerbation of his gout with painful swelling of the right elbow and right ankle joints. This was subsequently overshadowed by the gradual onset of constant severe pain at the midthoracic, midline area, which did not respond to conventional therapy or acupuncture. There had been mild constipation for several days, followed by the development of paresthesias and progressive weakness in both lower limbs, culminating in acute urinary retention which prompted his presentation.

Examination. The patient was febrile, with typical stigmata of both quiescent and active gout, consisting of periarticular synovial thickening, subcutaneous and auricular tophi, and acutely inflamed right elbow and ankle joints. In addition, there was incomplete motor and sensory paraplegia of the upper motor neuron type, with a sensory level demonstrated at T-7 and a distended urinary bladder.

Serum uric acid content was 0.56 mM/liter (normal 0.18–0.48 mM/liter) with increased 24-hour urinary uric acid excretion. Lumbar and cisternal myelography demonstrated a complete obstruction to contrast flow at T7–11, consistent with extradural compression (Fig. 1). Skeletal radiological survey reported typical changes of gout in the first metatarsophalangeal joints, with subcutaneous calcified tophi.

Operation. Thoracic laminectomy was performed, with unroofing of the extradural space from T-7 to T-11, inclusive. Following initial removal of the T-9 lamina, a sudden discharge of creamy white amorphous material was seen, and further exposure revealed a poorly defined fibrous mass lying within the extradural space anterolaterally on the right side of the cord. This mass extended from T-7 to T-11, with
Cord compression by tophaceous gout

significant cord compression at T-9, and erosion of the T-9 and T-10 pedicles on the right. No attachment to the dura mater or extradural tissues was demonstrated, and the tissue was removed in a piecemeal fashion.

Histological section showed crystals of uric acid, multinucleate giant cells, and histiocyte proliferation typical of gouty tophus. Postoperatively, the patient improved progressively; he regained bladder and bowel control and was walking with a stick prior to discharge.

Discussion

Urate crystal deposition in vertebral periarticular tissues may be considered as an extension of articular involvement of the peripheral joints. How often this actually occurs is open to speculation. Tkach reported back pain in up to 75% of patients with peripheral gout in his series. Pathological evidence of vertebral column involvement is infrequently reported. Involvement of the cervical vertebrae may produce bone erosion and ligament softening, as seen in the periphery, with the additional complication of potential subluxation producing spinal cord compression. Thoracic vertebrae have been noted to develop distinctive right-sided osteophytosis, so-called "hyperostotic spondylosis," with thoracic lipping. Vertebral bodies and intervertebral discs from both thoracic and lumbar levels containing tophaceous gouty deposits have been reported. The posterior articulations of the lumbar spine may exhibit urate deposits in the capsule, ligaments, and articular cartilage as demonstrated in the periphery. The bodies, pedicles, and laminae at this level may be eroded by extradural intraspinal tophus. Compromise of central nervous system tissues by gout is a much rarer entity. Sass, et al., described juvenile hyperuricemia with urate deposition in the brain and meninges producing a specific syndrome. Extensive pathological deposition of urate crystals in the ligamentum flavum has produced chronic cauda equina compression with paraplegia.

True spinal cord compression by extradural tophaceous gout was until recently a unique entity; however, cases with involvement at both the cervical and thoracic levels are now documented. Some inferences may be drawn from these reports and the case presented here. In every instance, spinal cord compression occurred in the clinical setting of chronic polyarticular tophaceous gout, suggesting that it is this subgroup of the gout population which is predisposed to develop extradural tophi. Age, sex, race, and relative diameter of the spinal canal are apparently not relevant.

The extradural focus of tophus formation remains unclear. Tophi have been demonstrated thus far at the cervical and thoracic levels, and apparently lie ventral and lateral to the cord on each occasion. In our case and that of Magid, et al., acute paraplegia was associated with active peripheral gout and back pain, perhaps suggesting that the tophus was undergoing a phase of active acute inflammation, with cellular infiltration and inflammatory exudate formation producing pain and resulting in a critical size increase, sufficient to produce clinical evidence of cord compression.

Although there was active peripheral gout in the situation of cervical cord involvement, there was no neck pain and the compression was chronic in nature. In the original case described by Koskoff, et al., the peripheral gout was quiescent at the time of presentation, the compression was chronic but associated with thoracic pain.

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References


Fig. 1. Left: Lumbar myelography demonstrating complete obstruction to contrast flow at T-11. Right: Cisternal myelography showing failure of contrast flow below T-7.


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