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Spinal cord compression: an unusual neurological complication of gout

We report the case of a 60-yr-old dental technician who presented in 1997 with a 6-week history of progressive right leg weakness. In addition he complained of damp underwear for 2–3 weeks and 3 days of nocturnal incontinence. He had previously been seen in 1989 with progressive weakness of all four limbs; was found to have a quadraparesis and was diagnosed as having both cervical and lumbar canal stenoses. Anterior decompression of the cervical spine at the C5/6 level was performed with some improvement in upper limb function. Because of persistent leg weakness, he underwent L2–4 decompressive lumbar laminectomy the following year and made a good functional recovery, and was able to return to work. He was known to have gout, for which he took allopurinol 300 mg daily, and had not suffered an acute attack for 10 yr. He had had polio as a child and had been left with residual left leg weakness. General examination revealed a wheelchair-bound middle-aged man who smelt of urine. There were no peripheral stigmata of gout. Examination of the cranial nerves and upper limbs was normal. Examination of the lower limbs demonstrated a wasted left leg with reduced muscle tone and MRC grade 3 weakness, compatible with the early history of polio. Muscle tone was also decreased in the right leg, power was well preserved proximally but he was MRC grade 3 distally. Sensory testing demonstrated a level to pinprick on the left below T4 and some impairment of joint position sense in the right leg. Lower limb reflexes were bilaterally absent and his right plantar was extensor. His bladder was painlessly distended and his anal sphincter tone was reduced. Because of the demonstrated hemisensory level on the left and the new right-side leg weakness and right extensor plantar, the Brown–Séquard syndrome was diagnosed.

Magnetic resonance imaging (MRI) of the entire spine was performed and showed a degree of lumbar arachnoiditis assumed to be the result of previous surgery. In addition to this, right-sided extradural spinal cord compression was demonstrated at the T1–2 level (Fig. 1). The patient underwent a T1–2 laminectomy the following day. At operation a greyish, tough, fibrous mass was found indenting the spinal cord on the right.

There appeared to be no point of attachment and it was removed easily. Postoperatively, sensation improved on his left side but the right leg power and bladder function did not improve.

Histological examination revealed a thick-walled cystic structure containing dense eosinophilic debris surrounded by a layer of partly palisaded histiocytes and multinucleated foreign body giant cells. A single needle-shaped urate crystal was seen, and the lesion was identified as a degenerate tophus. Subsequently his serum urate was measured but found to be normal.

The first case of myelopathy secondary to cord compression by a gouty tophus was reported by Koskoff et al. in 1953 [1]. There have been occasional further reports since that time [2]. The compression has been almost exclusively extradural in position but intradural deposition of urate has been noted [3].

In previous reports, males predominated as the disease itself is commoner among this group. Hyperuricaemia was present in most but not all cases [2, 4]. Symptomatic axial skeletal deposits are usually thought to represent a complication of long-standing, poorly controlled hyperuricaemia. Previous authors have emphasized the importance of long-term serum urate control to prevent the complication of spinal cord compression, but the complication may occur even if this is
achieved. It is significant that in none of the previous reports, as in this case, were tophi suspected as the cause of the patient’s symptoms. It is noteworthy that tophaceous gout of the spine may occur in the patient with no peripheral tophi [5] and may rarely be the presenting feature of the disease [4].

Most cases have been treated with surgery, decompressive laminectomy with occasional spinal fixation being the procedure most commonly performed. However, suppression of serum urate with allopurinol may arrest the pathological deposition of uric acid with regression of tophi [6], and successful non-surgical management in a patient with quadriplegia secondary to tophaceous involvement of the odontoid process and C1–2 instability has been described [7]. At surgery, a characteristic chalky, cheese-like, friable mass is usually identified. The deposit may occasionally be tough and fibrous in nature [8]. Histologically, a fairly uniform pattern is described with a cellular infiltrate of multinucleated giant cells and histiocytes surrounding cellular eosinophilic debris. Occasionally, only a shadow of crystal may be seen because of the dissolution of urate in the aqueous phase of fixation, but usually the needle-shaped, negatively birefringent crystals are observed. Symptomatic gouty atrophy of the spine is rare but should be included in the differential diagnosis of patients with suspected cord compression who are known to have or are suspected of having gout.

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