**CASE REPORT**

**Cauda Equina Syndrome Secondary to Tophaceous Gout of the Spine**

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**ABSTRACT**

This report describes a case of cauda equina syndrome due to rupture of gouty tophus from the L3-4 facet joints into the epidural space, which caused marked epidural inflammation and compression. The patient also had carcinoma of the prostate with multiple spinal metastases. The clinical details of the symptoms, investigations, which included computed tomography and myelography, and the results of operative treatment are presented. A Medline search of the literature for the past 50 years yielded only 11 other reported cases of tophaceous gout involving the facet joints of the spine. This particular case illustrates the difficulties in making the diagnosis in the presence of concomitant spinal metastases.

**Key Words:** Cauda equina, Gout, Polyradiculopathy, Spinal cord compression, Zygapophyseal joint

**INTRODUCTION**

Gout is a common condition that results from the deposition of monosodium urate crystals. Patients with long-standing gout or a massive total body urate load may develop tophaceous gout. Tophus may develop adjacent to joints and in soft tissue structures such as the skin or the bursa.

Tophaceous gout in the axial skeleton is rare, but it may occur at any spinal level and cause myelopathy, radiculopathy, spinal osseous destruction, or cauda equina syndrome. This report describes a patient with tophaceous gout of the facet joints of the lumbar spine, which caused cauda equina syndrome.

**CASE REPORT**

A 65-year-old man was admitted to the Department of Orthopaedics and Traumatology at the Alice Ho Miu Ling Nethersole Hospital in 2002 because of the inability to pass urine and a 1-day history of right thigh pain. He had a history of diabetes mellitus hypertension, renal functional impairment, and gouty arthritis and gouty tophi in both hands and both feet. He was given oral drugs for these conditions but his compliance was poor. Urinary retention and overflow incontinence developed on the day of admission. Physical examination showed that the power of the lower limbs was normal. Perianal sensation was impaired, the anal tone was lax, and the bulbocavernosus reflex was absent.
The lower abdomen was distended because of the full bladder. Rectal examination showed hard nodules at the right lobe of the prostate. X-ray examination showed multiple osteosclerotic changes that involved the pedicles and vertebral bodies of all lumbar vertebrae (Figure 1). Myelography revealed an extradural compression at the L3-4 level (Figure 2). Computed tomographic myelography revealed multiple juxta-articular erosions and an abnormal soft tissue shadow at the L3-4 facets bilaterally (Figures 3, 4, and 5).

Blood tests showed a normal complete blood cell count and normal liver function. Renal function, however, was slightly impaired. The serum creatinine level was 167 µmol/L (normal range, 53 to 106 µmol/L), serum urea level was 5.6 mmol/L (normal range, 2.9 to 8.2 mmol/L), erythrocyte sedimentation rate was 30 mm/hour (normal range, 0 to 20 mm/hour), serum uric acid level was 0.60 mmol/L (normal range, 0.24 to 0.51 mmol/L), and serum prostate-specific antigen level was 390 ng/L (normal level, <4.0 ng/mL). The clinical diagnosis was acute cauda equina syndrome caused by spinal metastasis; the most likely primary site was the prostate gland.

Emergency posterior decompression with L3 and L4 laminectomy was performed. No tumour tissue was found inside the spinal canal. Instead, both L3-4
facet joints were distended because of chalky material; the facet joint on the right side was more severely affected than the left (Figure 6). A part of each joint was extruded into the epidural space, which caused marked inflammation of the epidural tissue, as well as cauda equina compression. The chalky material was removed from the epidural space and the facet joints, and was sent for histological examination, bacterial and acid-fast bacillus smear tests, and bacterial culture. The epidural space was irrigated thoroughly with isotonic saline. Transpedicular core biopsy of the L3 osteosclerotic lesion was performed before wound closure.

The neurological deficit gradually improved postoperatively. By the sixth month after the operation, sensation had recovered by 50%, anal tone had returned to normal, and anal sphincter function had recovered partially. The patient still required 1 to 2 episodes of intermittent catheterisation of urinary daily, as well as laxatives to assist bowel motion. Lower-limb power remained normal. Postoperative ultrasonography of the pelvis and abdomen showed cysts on the right side of the liver, chronic renal disease, bilateral renal cysts, a stone in the right kidney, and an enlarged prostate. Carcinoma of the prostate was confirmed with tissue biopsy and was subsequently treated with bilateral orchidectomy at the Department of Surgery. The bone metastasis was treated with radiotherapy.

Pathological examination revealed that the chalky material was a tophaceous deposit with an associated granulomatous response. Smear and culture tests of the chalky material did not show any bacteria or acid-fast bacilli. The bone biopsy from the L3 vertebra showed metastatic adenocarcinoma that was positive for prostate antigen. This finding was compatible with the result from the prostate tissue biopsy.

**DISCUSSION**

Tophaceous gout involving the spine is uncommon; tophaceous gout involving the facet joint is even rarer.1-11 Only 11 other cases of tophaceous gout involving the facet joint of the spine were identified in a Medline search of the literature from 1956 to 2004 (Table 1).

**Table 1** Summary of reported cases of tophaceous gout in the facet joint of the spine.

<table>
<thead>
<tr>
<th>Study</th>
<th>Sex/age (years)</th>
<th>Gout</th>
<th>Duration of spinal pain</th>
<th>Neurological symptoms</th>
<th>Level of facet</th>
<th>Serum urate (mmol/L)</th>
<th>Peripheral tophi</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arnold et al1</td>
<td>F/72</td>
<td>Yes</td>
<td>Many years</td>
<td>Radiculopathy</td>
<td>L4</td>
<td>0.35</td>
<td>No</td>
</tr>
<tr>
<td>Barrett et al2</td>
<td>M/70</td>
<td>Yes</td>
<td>2 days</td>
<td>Radiculopathy</td>
<td>L4-5</td>
<td>0.61</td>
<td>No</td>
</tr>
<tr>
<td>Fenton et al3</td>
<td>M/61</td>
<td>No</td>
<td>6 months</td>
<td>Radiculopathy</td>
<td>L4-5</td>
<td>0.39</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td>F/71</td>
<td>No</td>
<td>2 years</td>
<td>Stenosis</td>
<td>L4-5</td>
<td>0.74</td>
<td>No</td>
</tr>
<tr>
<td>Hall and Selin4</td>
<td>M/51</td>
<td>No</td>
<td>None</td>
<td>None</td>
<td>L4-5, L5-S1</td>
<td>Unknown</td>
<td>No</td>
</tr>
<tr>
<td>King and Nicholas6</td>
<td>M/37</td>
<td>Yes</td>
<td>7 days</td>
<td>Radiculopathy</td>
<td>L5-S1</td>
<td>0.63</td>
<td>Yes</td>
</tr>
<tr>
<td>Miller et al7</td>
<td>F/80</td>
<td>No</td>
<td>Days</td>
<td>Radiculopathy</td>
<td>L2-3</td>
<td>Unknown</td>
<td>No</td>
</tr>
<tr>
<td>Reynolds et al8</td>
<td>M/74</td>
<td>Yes</td>
<td>Many months</td>
<td>Paraparesis</td>
<td>L4-5</td>
<td>0.63</td>
<td>Unknown</td>
</tr>
<tr>
<td>Thornton et al9</td>
<td>M/27</td>
<td>No</td>
<td>1 day</td>
<td>None</td>
<td>L3-4</td>
<td>0.71</td>
<td>No</td>
</tr>
<tr>
<td>Varga et al10</td>
<td>F/76</td>
<td>No</td>
<td>Many years</td>
<td>Radiculopathy</td>
<td>L5</td>
<td>0.61</td>
<td>No</td>
</tr>
<tr>
<td>Wang et al11</td>
<td>M/28</td>
<td>Yes</td>
<td>Many days</td>
<td>Paralysis</td>
<td>T9-10</td>
<td>Unknown</td>
<td>No</td>
</tr>
<tr>
<td>Mak et al (this report)</td>
<td>M/65</td>
<td>Yes</td>
<td>2 days</td>
<td>Cauda equina</td>
<td>L3-4</td>
<td>0.60</td>
<td>Yes</td>
</tr>
</tbody>
</table>
The diagnosis of gouty tophus in the facet joint of the spine is difficult. The classic X-ray features of gouty arthritis — a dense asymmetrical, well-defined soft tissue mass, juxta-articular erosion, and relative preservation of the joint space — may not be obvious. On the other hand, the increased density of the tophaceous deposits and the destruction of the facet joints cannot be easily differentiated from tumour metastases.

Fenton et al suggested that computed tomography allows better visualisation than X-ray, because monosodium urate has a radiographic attenuation density greater than that of the surrounding muscle but less than that of bone. King and Nicholas suggested that magnetic resonance imaging could be used to differentiate the fluid phase of a synovial cyst or infection from the solid phase of a gouty tophus. Tophi manifest as homogeneous intermediate to low signal intensity on T1-weighted images. On T2-weighted images, their appearance ranges from a homogeneous high signal intensity to homogeneous low signal intensity. With gadolinium–diethylenetriaminepenta-acetic acid enhancement, gouty tophi can manifest as homogeneous enhancement to heterogeneous peripheral enhancement. The definitive diagnosis still depends on direct observation with polarised light microscopy: gouty tophi appear as needle-shaped urate crystals with negative birefringence.

Surgical decompression should be the treatment of choice if patients have any neurological compromise. This approach has improved the neurological status in all reported patients. Furthermore, tissue specimens can be obtained for pathological examination. Conservative treatment has a very limited role in the treatment of spinal gout. Barrett et al suggested that non-steroidal anti-inflammatory drugs, colchicine, and allopurinol can be given to patients with polyarticular tophaceous gout who have no obvious infection, present with back pain, have no neurological deficit, and have imaging findings that are consistent with spinal gout. Needle aspiration of the involved facet joint or needle biopsy of the abnormal bone or disc is needed to establish the presence of monosodium urate crystals.

Although tophaceous gout of the facet joints of the spine is rare, it should be included in the differential diagnosis of patients who have back pain or a neurological deficit and gout. Computed tomography and magnetic resonance imaging may help in the establishment of the diagnosis. However, only histological proof provides a definitive diagnosis. Surgical treatment is preferred for patients with a neurological deficit. Medical treatment is an option, but it is only applicable for exceptional cases and the clinical response should be monitored very closely.

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REFERENCES

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