Acute Paraplegia in a Patient with Spinal Tophi: A Case Report

Liang-Chao Wang, Yu-Chang Hung, E-Jian Lee, and Hsing-Hong Chen

Abstract: A 28-year-old man with a 5-year history of gouty arthritis suffered from an acute episode of lower back pain. He visited a rehabilitative clinic and received physical therapy following his examination. Weakness and numbness of both lower legs developed rapidly after physical therapy. He was sent to our hospital with complete paralysis of both lower limbs and complete sensory loss below the umbilicus 3 hours after the physical therapy. No peripheral tophi were found. Myelography showed an extrinsic compression of the dura sac at T10. Emergency decompressive laminectomy of T9 to T11 was performed. During the surgery, caseous material was found deposited in the ligamentum flavum and the left T9 to T10 facet joint, with indentation of the dura sac. The pathologic diagnosis was spinal tophi. After surgery, the patient's neurologic function recovered rapidly. It was suspected that inappropriate physical therapy might have aggravated acute inflammation of spinal gout and resulted in a rapid deterioration of neurologic function. Though gout is a chronic medical disease, an acute attack of spinal gout may be disastrous and requires emergency neurosurgical intervention.

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Key words:
acute paraplegia
spinal tophi
gouty arthritis

Monosodium urate crystal deposition in the joints of the appendicular skeleton is a characteristic feature of chronic gout. Reports of tophi in the axial skeleton are not common. The most common symptoms of spinal gout are back pain and progressive neurologic deficit due to chronic compression of the spinal cord or roots by tophi [1–3]. Acute paraplegia due to spinal tophi has not been previously reported. We report a case of acute paraplegia caused by spinal tophi and review the relevant literature on this condition.

Case Report

A 28-year-old obese Taiwanese male with a body height of 180 cm and body weight of 110 kg had suffered from frequent episodes of acute gouty arthritis for 5 years before this hospitalization, with the first attack occurring in his left ankle joint. His elbow and ankle joints were most affected by the disease. The frequency of acute attack was about twice per month. He had received intermittent antihyperuricemic therapy and nonsteroidal antiinflammatory drugs for acute gouty arthritis. He was not a smoker and seldom drank alcohol. Although he preferred to eat meat, he had decreased his meat consumption since hyperuricemia was diagnosed. He had a 1-year history of hypertension with irregular medical treatment. No family history of gouty arthritis was reported.

Twenty hours before admission, the patient had suffered from sudden onset of lower back pain and mild numbness in both legs and had visited a rehabilitative clinic for physical therapy. Local infrared radiation and electric stimulation were applied. Nonetheless, lower limb weakness and urine retention developed quickly after the physical therapy, and he was sent to the emergency room 3 hours after physical therapy.

On admission, both lower limbs were paralyzed with a muscle power of grade 0. Sensation of pain, light touch, temperature and proprioception were absent below the umbilicus. No peripheral tophi were found. Laboratory investigation demonstrated a serum uric acid concentration of 600.75 μmol/L, a blood urea nitrogen concentration of 3.57 mmol/L, a serum creatinine concentration of 79.65 μmol/L, a blood sugar concentration of 6.61 mmol/L, a peripheral leukocyte count of 13,700 cells/mm^3 with a shift to the left, and an alkaline phosphatase concentration of 179 units/L. Urine analysis was within the normal range. Under the

Neurosurgical Service, Department of Surgery, National Cheng Kung University Hospital, College of Medicine, National Cheng Kung University, Tainan.
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Reprint requests and correspondence to: Dr. Hsing-Hong Chen, Neurosurgical Service, Department of Surgery, National Cheng Kung University Hospital, College of Medicine, 138 Shen-Li Road, Tainan, Taiwan.

impression of a spinal stroke, spinal computerized tomography (CT) was performed to rule out spinal hemorrhage; this revealed spinal stenosis and intra-articular erosion of the T10 facet joints, but no evidence of intrinsic or extrinsic hemorrhage was found (Fig. 1). Spinal magnetic resonance (MR) imaging was desired to rule out a spinal infarct, but emergency MRI service was not available. Myelography was arranged immediately and showed complete blockage of contrast at the T10 level (Fig. 2). Under the impression of acute extrinsic compression of the thoracic spinal cord, emergency decompressive laminectomy was performed.

During surgery, the ligamentum flavum was found to be hypertrophic from T9 to T11, with caseous deposits and constriction of the dura sac. The left T9 to T10 facet joint was also enlarged by caseous deposition and the dura sac was indented. T9 to T11 laminectomy was performed. The hypertrophic liagmentum flavum as well as the enlarged portion of the left T9 to T10 facet joint were removed and the spinal cord was decompressed.

Pathologic examination of the ligamentum flavum and facet joint revealed amorphous eosinophilic material surrounded by infiltrates of histiocytes, fibroblasts, and foreign body giant cells (Fig. 3). Under examination with a polarized light microscope, many negatively birefringent, needle-shaped crystals were found within the eosinophilic material. These were consistent with monosodium urate crystals.

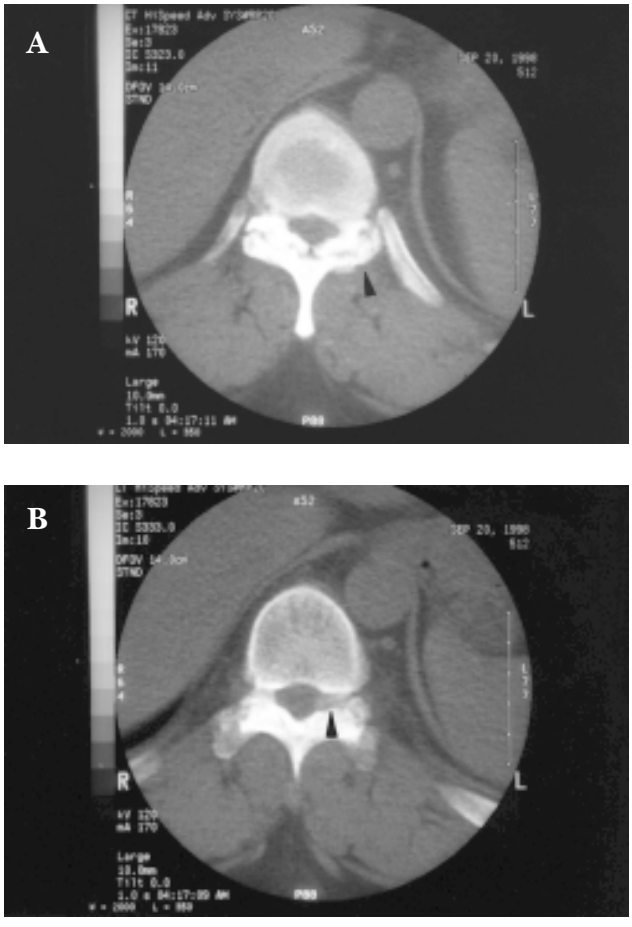

Fig. 1. *Spinal computerized tomography shows (A) erosion of left T9–T10 facet joint with a sclerotic margin (arrowhead) and (B) periarticular mass (arrowhead) compromising the spinal canal.*

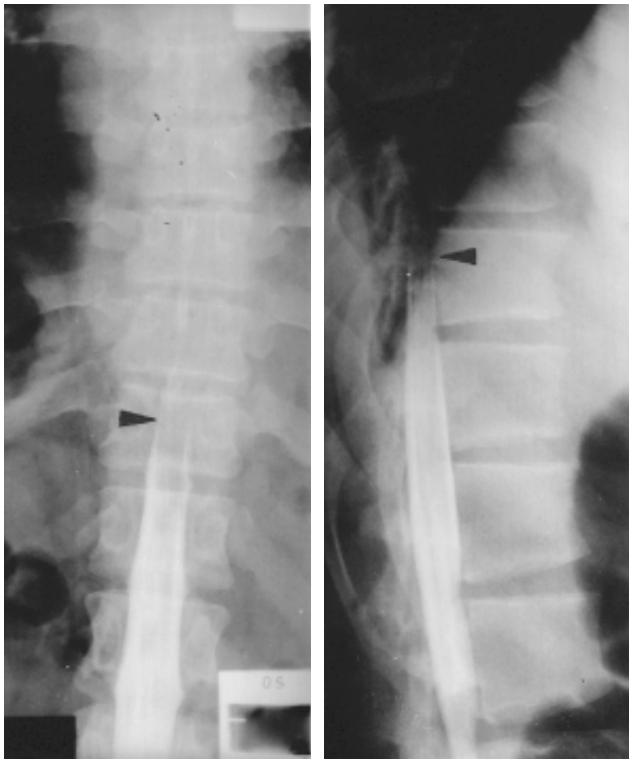

Fig. 2. *Myelography shows complete blockage of contrast medium at T10 (arrowheads).*

The decompressive surgery was performed about 20 hours after the onset of neurologic deterioration. After surgery, the patient's muscle power in both lower limbs recovered to grade 4 within 8 hours. The Foley catheter was removed on the third postoperative day. The patient continued to be free of neurologic deficit and back pain 1 year after the operation.

Discussion

Gouty arthritis is a chronic inflammatory process resulting from deposition of monosodium urate crystals in joints and periarticular connective tissue. Tophi are typically found in the joints of the appendicular skeleton and reports of spinal tophi are uncommon. Being a chronic inflammatory process, the most common presenting symptom of spinal tophi is chronic back pain. Among the 26 reported cases [1–15], 21 patients had suffered from back pain or neck pain. Chronic deposition of monosodium urate crystals in the epidural space, ligamentum flavum, disc, or facet joints may

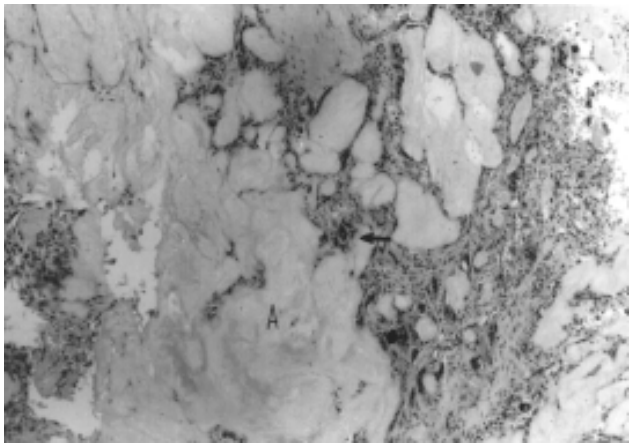

Fig. 3. *Pathologic examination shows amorphous eosinophilic material (A) surrounded by infiltrates of histiocytes, fibroblasts, and foreign body giant cells (arrow) (hematoxylin & eosin, original magnification, x 100).*

cause insidious compression of the spinal cord, resulting in the gradual development of neurologic deficits. The reported neurologic complications included radiculopathy (9 cases), paraparesis (4 cases), and quadriparesis (3 cases). None of the reported patients presented with acute paraplegia. Although peripheral tophi can be found in most patients with severe gouty arthritis, they are not always present in patients with spinal tophi. In our patient, as well as in seven other reported cases, no peripheral tophus was found [1, 3–7].

The clinical diagnosis of spinal gout depends on a high index of suspicion in patients with a history of gouty arthritis. CT and MR imaging are very helpful in the diagnosis of spinal gout. The classical findings of spinal gout on CT described by Fenton et al are lobulated juxta-articular masses with attenuation density greater than that of the surrounding muscle, well-defined intra-articular and juxta-articular osseous erosions with sclerotic borders, normal bone density, and relative preservation of synovial joint spaces [1]. MRI findings of spinal gout include low-signal-intensity gouty tophi and surrounding fibrous tissue on T2-weighted images, sharply delineated erosion without a surrounding inflammatory infiltrate, and a segmental pattern of disk involvement [8]. However, these radiologic characteristics are not specific for spinal gout and pathologic examination is necessary for diagnosis.

Surgical decompression is the treatment of choice for patients with neurologic compromise. Of the six reported patients who received medical treatment for spinal gout [1, 5, 9–12], only four had neurologic improvement [1, 9, 10, 12]. Surgical decompression provided improvement of neurologic compromise in all reported cases. Further stabilization was performed in one case [13].

Our patient presented with acute atraumatic paraplegia with rapid deterioration of neurologic function within several hours after receiving physical therapy for lower back pain. Most cases of acute atraumatic paraplegia result from spinal-cord strokes, such as those involving hemorrhage or spinal arterial occlusion. Neoplasms with hemorrhage or vertebral collapse are other common causes of acute paraplegia. Spinal gout has not been previously included in the differential diagnosis of acute paraplegia.

The initial lower back pain and numbness of lower limbs in our patient suggested that his spinal canal had been compromised before the physical therapy. The present case demonstrates that an acute inflammation, aggravated by inappropriate physical therapy, may decompensate the spinal cord and result in rapid deterioration of neurologic function within several hours.

Emergency decompressive surgery is the only treatment to rescue neurologic function in patients with acute paraplegia due to extrinsic spinal cord compression. Although MR imaging is a very powerful tool and can replace myelography and CT in assessing acute paraplegia, it is still expensive and is not always readily available for emergency service. Myelography is an invasive and primitive examination, but nevertheless is still a useful tool in diagnosing acute spinal cord compression. If there is strong evidence of extrinsic compression of the spinal cord on myelography, time should not be wasted waiting for MR imaging to justify emergency decompressive surgery, unless an emergency MR imaging service is available.

Our patient had a rare clinical presentation of spinal gout. Acute spinal cord compression by gouty tophi can be disastrous. When treating patients with gouty arthritis with back pain, the possibility of spinal gout should be kept in mind. This case also reminds us that myelography, like many other primitive examinations, should not be abolished in the era of advanced technology such as MR imaging.

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