Lumbar Spinal Stenosis Induced by Rare Chronic Tophaceous Gout in a 29-year-old Man

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abstract

Spinal gout is rare in patients younger than 45 years, occurring most commonly between ages 45 and 80 years. This article describes a 29-year-old man with a history of gout initially observed more than 20 years previously who presented with lower back pain and left lower limb weakness. Computed tomography and magnetic resonance imaging revealed severely damaged facet joints and laminae surrounding L4-S1, and materials with a granular, yellow, cheese-like appearance were observed under direct vision. Postoperative histological examination confirmed spinal gout. Complete posterior decompression was performed concurrently with interbody fusion at L4-L5 and L5-S1. Seventeen-month follow-up revealed good recovery.

The mechanism by which urate crystals form preferentially in the spine as opposed to more common soft tissue sites is not well understood. Most reported spinal gout cases were located at L4-S1, which were believed to be the segments with high stresses. These cases occurred in older patients with degenerative spinal diseases and had trauma as indicators. The current authors presume that gout is more likely to exist in the mobile regions with high pressure, such as L4-S1, even in individuals with an initially healthy spine, especially with a long period of abnormal renal dysfunction. Although it is often overlooked in young patients, this condition may be more common than the literature suggests due to the high potential for misdiagnosis in relatively young patients.

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Figure: Photographs showing the spine after decompression and lesion debridement (A) and granular, yellow, cheese-like deposits (B). All facet joints and associated ligamentum flavum from L4-S1, the L4-L5 disks, and L5-S1 were completely removed. The left L5 nerve root was completely decompressed. Interbody fusion was then completed using Capstone intervertebral fusion cages and pedicle screws (Medtronic Danek, Memphis, Tennessee).
Gout occurs in approximately 0.2% to 0.4% of the population, and the global annual incidence is approximately 0.01% to 0.015%, with the majority of cases occurring in older patients. The risk of uric acid buildup in the blood leading to joint inflammation associated with spinal gout commonly increases with age, often resulting in the formation of monosodium urate crystals, generally referred to as tophi or tophaceous gout.

In contemporary spinal gout treatment, symptoms are generally assessed using computed tomography (CT) scanning, wherein areas of sclerotic bony erosion can be easily identified by their distinctive punched-out appearance. Magnetic resonance imaging (MRI) findings are nonspecific in most cases, although they may provide supporting evidence for diagnosis by other means. Few reports indicate the occurrence of gout in adults younger than 45 years, with the majority of patients ranging between 45 and 80 years. The current article describes a case of a young patient with lumbar spinal stenosis secondary to chronic tophaceous gout examined by CT scanning, indicating that spinal involvement may be a greater concern in younger gout patients than previously indicated.

**Case Report**

A 29-year-old man presented with severe pain, paresthesia, and acratia of the left lower limb. Symptoms were first observed more than 1 year previously, showing progressive behavior and increasingly impacting the quality of life. The patient reported a history of chronic gout symptoms over the past 20 years without evidence of positive family gout, degenerative spinal disease, or traumatic spinal injury history. The patient had a height of 168 cm, weight of 85 kg, and body mass index of 30.5 kg/m². Furthermore, the patient reported an uncommonly high alcohol intake spanning many years, including generally heavy beer drinking to the point of intoxication.

Initial daily treatment with allopurinol failed to affect improvement. Physical examination revealed obvious tenderness in the lumbar spine during palpation. Multiple tophi were noted superior to the digits of the hand and feet (Figure 1). Furthermore, the left tibialis anterior and hallucis longus muscles showed severely weakened power (grade III/V) and decreased sensitivity to stimulation by touch, particularly prominent in the anterior crus region. Reflexes were intact and within normal ranges using the straight-leg raise test. Initial observations of elevated levels were made of serum uric acid at 0.509 mmol/L (range, 0.1-0.42 mmol/L), creatinine at 146 µmol/L (range, 50-130 µmol/L), and blood urea nitrogen at 11.7 mmol/L (range, 2.5-7 mmol/L), with no other evidence suggesting primary renal dysfunction, such as hematuria, proteinuria, hypertension, elevated blood electrolyte level, and glomerulonephritis history, providing clear indications of potentially symptomatic gout-induced impairments in normal renal function.

Computed tomography scans revealed severe bilateral erosions directly over the L5-S1 facet joints and periarcticular bone and the presence of soft tissue masses and calcifications in the spinal canal (Figure 2). Magnetic resonance images further confirmed the presence of a large quantity of tophus masses possessing similar characteristics and dimensions localized around the L5-S1 region and resulting in significant compression of the spinal contents toward the dural sac and left L5 roots (Figure 3). No obvious disk degeneration was observed by CT or MRI. The diagnosis of spinal gout was based on the patient’s gout history and the presence of lower-extremity pain, severe bone erosions revealed by CT, and masses...
causing compression of the lumbar cord at L5-S1 without obvious disk hernia revealed by MRI. These findings were considered in combination with abnormal serum uric acid levels; normal white blood cell count (8.99×10⁹), polymorphonuclear neutrophil rate (58.3%), and C-reactive protein level (5.81 mg/L); and slightly elevated erythrocyte sedimentation rate level (59 mm/h), potentially due to gout. This diagnosis excluded lumbar disk hernia, infection, and tuberculosis. Tumor diagnosis was excluded according to postoperative histological testing, further confirming the diagnosis of spinal gout.

Intraoperatively, the space between the L4-L5 facet joints was characterized by the abnormal presence of a substance with a white, chalky, and granular appearance, presenting an overall appearance reminiscent of a coarse, sand-like material. In addition, materials with a granular, yellow, cheese-like appearance were confirmed as urate deposits (Figure 4). The facet joints of L4-S1 and the pars interarticularis of L5 showed signs of severe damage (Figure 4), necessitating surgical laminectomy and facetectomy for lamina and spinal nerve decompression, respectively. Furthermore, fusion of the vertebrae from L4-S1 was required for full spinal reconstruction (Figure 5). All facet joints and associated ligamentum flavum from L4-S1, the disks of L4-L5, and L5-S1 were completely removed. The left L5 nerve root was completely decompressed. Interbody fusion was then completed using Capstone intervertebral fusion cages and pedicle screws (Medtronic Danek, Memphis, Tennessee).

Following postoperative recovery, function of the affected lower limb improved with regular allopurinol use, resulting in assignment of power grade IV at final follow-up 17 months postoperatively.

Tissue biopsies of the abnormal white, granular material and surrounding tissue were taken for further analysis. Histological examination revealed negatively birefringent crystals showing positive Alcian Blue staining, indicating the likely occurrence of chronic synovitis. Examination also confirmed the presence of gout tophi previously indicated by MRI and CT scanning (Figure 6). At final follow-up, MRI and CT scans indicated successful solid fusion of the spine exhibiting excellent spinal morphological performance and stability, associated with notable reductions in the initial symptoms (Figure 7).

**DISCUSSION**

Gout tophi in the spinal region, particularly those located around prominent neurological structures, may contribute to symptomatic spi-
nal stenosis that may potentially affect numerous bodily processes by interfering with normal nerve function. The accumulation of urate crystals in the spine occurs most commonly in the intervertebral disk space, ligamentum flavum, and facet joints. These depositions usually form peripheral tophi in the spine, although exceptions have been reported. The mechanism by which urate crystals form preferentially in the spine instead of more common soft tissue sites, particularly the para-articular regions and articular cartilage of the limbs, is poorly understood; however, symptoms of such spinal tophi are often more severe.

Numerous risk factors are associated with gout and have widespread effects on critical bodily functions. Common manifestations include progressive and chronic renal failure, positive family history, and cyclosporin A therapy. Although tophaceous gout of the spine is uncommon compared with its occurrence in soft tissues,6 it is more uncommon for the condition to manifest in young adults. Numerous risk factors are associated with gout and have widespread effects on critical bodily functions. Common manifestations include progressive and chronic renal failure, positive family history, and cyclosporin A therapy. Although tophaceous gout of the spine is uncommon compared with its occurrence in soft tissues,6 it is more uncommon for the condition to manifest in young adults.

Although the etiopathogenesis of urate crystal accumulation in the axial skeleton is not completely understood, it has been previously reported that degenerative diseases of the spine occurring in the L5-S1 region or traumatic damage to these areas may increase the risk for tissue necrosis, triggering the abnormal accumulation of crystals in these damaged regions. This hypothesis provides a likely explanation for the prominence of spinal gout in patients older than 45 years because older age increases the rate of occurrence of degenerative spinal diseases. With the exception of a 27-year-old man who previously underwent renal transplantation due to complications associated with spinal tophi formation, no cases were identified in the literature occurring in patients younger than 45 years within the past 4 decades. This may be due to the rarity of these cases and the fact that spinal gout is difficult to properly diagnose, which may have resulted in delayed or incorrect treatment for these patients.

In the current patient, several potential causative factors may have played a role in the occurrence of spinal stenosis of the lumbar region resulting from the chronic effects of tophaceous gout. Spinal gout has been previously reported to be most common in the lumbar region, especially between levels L4 and S1. This area, characterized by high facet joint stress, is believed to be the most prominent region of occurrence for degenerative spinal diseases. Increased stresses in this region

Figure 6: Photomicrograph of the surgical specimens from the L5-S1 facet joints showing the amorphous character of the gouty lesions, indicating an active reaction of multinucleated giant cells (×200).

Figure 7: Seventeen-month follow-up anteroposterior (A) and lateral (B) plain radiographs, sagittal computed tomography scans (C, D), and axial magnetic resonance images (E, F), showing excellent bony fusion without nerve compression and spinal deformation.
may promote localized crystal formation, even in healthy spines presenting no evidence of degenerative spinal disease.

Renal dysfunction plays a significant role in raising the uric acid levels of the serum. Primary or secondary (eg, induced by gout) renal function promotes uric acid levels, causing tophi deposition in the spine and mild spinal stenosis with abnormal nerve compression. In turn, the elevated serum acid level further impairs renal function and contributes to the worsening of toph deposition, which is supported by Chonchol et al. The elevation of serum creatinine and blood urea nitrogen provided evidence of such a mechanism of kidney malfunction in the current case. Because the current case presented no other indicators, including hematuria, proteinuria, hypertension, elevated blood electrolyte level, and glomerulonephritis history, which may provide evidence for causative factors associated with primary renal dysfunction, and furthermore combined with symptom alleviation after spinal surgery, it may be inferred that preoperative abnormal serum blood urea nitrogen and creatinine levels were likely due to the effects of spinal gout. The current patient’s more than 20-year history of gout likely contributed to the progressive formation of gouty tophi. This extended duration also increased the risk for tophi buildup in uncommon locations, such as the spinal canal. Previous studies suggested that the formation of tophi in uncommon locations is most prevalent in patients with a several-year history of gout or asymptomatic gout of undetermined duration, as reported by Nygaard et al in the case of a 75-year-old patient. In each case, evidence existed of prolonged elevation of serum uric acid levels, likely contributing to a progressive increase in tophi formation.

Lifestyle choices often affect the occurrence and progression of symptomatic cases of tophaceous gout. Obesity and alcohol intake contribute to the development and progression of many metabolic diseases, including gout. Consequently, patients who exhibit a higher-than-average body mass index coupled with regular intake of excessive amounts of alcohol may have a metabolic background that contributes to the development of spinal gout.

The success of allopurinol, a xanthine oxidase inhibitor, to control serum uric acid level only after spinal surgery in the current case supports the diagnosis of tophaceous gout of a progressive nature. These findings suggest that the treatment of gout patients, particularly young adults, should be customized based on patient age, lifestyle, body type, and relevant comorbid conditions. Gout patients who exhibit severe compression symptoms with or without spinal instability may benefit from surgical decompression and fusion because this operation may be critical to the alleviation of symptoms and improvement of overall quality of life. Particularly in relatively young patients, fusion should be considered for the removal of gout tophi because it can often be completed without significantly disturbing spinal stability.

**CONCLUSION**

Although spinal gout is uncommon, clinicians should be aware of its possible occurrence to assign the correct treatments at the earliest time possible. Because gout tophi in the spine may result in spinal stenosis with the potential to affect many organ and tissue functions, patients who present with general neurological symptoms should be assessed for gout early in treatment. Although this is often overlooked in young patients, the condition may be more common than the current literature suggests due to the high potential for misdiagnosis in relatively young patients. Surgical intervention may be necessary for the effective long-term alleviation of serious symptoms of spinal compression or spinal instability induced by the presence and continued growth of tophaceous gout, particularly in the lumbar spine.

**REFERENCES**