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# Gouty Tophi in the Lumbar Spinal Canal: A Case Report Managed with Unilateral Biportal Endoscopic Decompression

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## 1. Abstract

Gout is a systemic metabolic disorder mainly caused by problems with purine metabolism and issues with uric acid excretion. It is marked by recurrent acute or chronic arthritis, deformed joints, and severe pain. High uric acid levels in the blood cause urate crystals to build up in joints, ligaments, tendons, and subcutaneous tissues, which damages the tissues [1].with the rapid economic development and better quality of life, the prevalence of gout among adults in areas like China is between 0.68% and 3.90%, and there's a trend of it increasingly affecting younger people [2-4]. The first gout attack usually happens in the big toe, followed by the hands, wrists, elbows, and knees [5]. The lamina, ligaments, epidural, and intradural spaces of the spine can be affected. Among them, the lumbar spine is the most common site for gout of the spine. When gouty tophi compress the nerves in the lumbar spinal canal, it can be hard to tell apart from space-occupying lesions such as lumbar disc herniation, lumbar canal stenosis, and infections in the intervertebral space. In recent years, reports of gouty tophi in the lumbar spinal canal have been on the rise both in China and abroad, with most treatments focusing on open surgery, while there are fewer reports on endoscopic minimally invasive surgery. In November 2024, our hospital admitted a patient with gouty tophi in two lumbar spine segments, and after removing the tophiusing the unilateral biportal endoscopy (UBE) technique, we saw good results. Here's a report on this case.

#### 2. Clinical Data

The patient is a 28-year-old male, presenting with complaints of lower back pain and left lower limb pain, which have limited his activity for the past 6 months. He was admitted to the hospital in November 2024. Six months ago, he started having lower back pain without any obvious triggers, along with left lower limb pain. Despite resting and taking oral medications, he didn't find much relief, and the symptoms gradually worsened recently, which led him to seek medical help at our hospital.Outpatient MRI (Figures 1a, b, c) showed irregular bone structure in the L3-5 facet joints, with diffuse patchy and cystic long T1 and T2 signals around the facet joints, some protruding into the lumbar canal, with infectious lesions not ruled out and signs of lumbar canal stenosis. He was diagnosed with "lumbar canal stenosis" and admitted to our department. The patient has a 9-year history of gout, taking febuxostat 20mg once daily, which he effectively manages with ibuprofen or naproxen during acute gout attacks. He has a history of type 2 diabetes for the past week, fatty liver disease for 2 years, and he had an appendectomy 4 years ago.

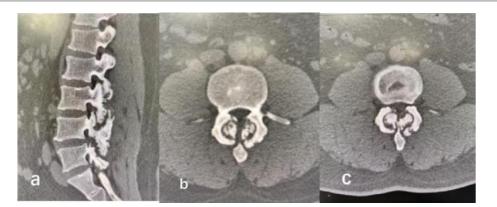
#### 2.1. Admission Examination

Walked into the clinic with a limping gait, tightness and spasms in the lower back muscles, tenderness along the spinous processes of the lower back, slight limitation in movement, tenderness in the left

piriformis muscle and where it exits, normal sensation in both legs, left foot dorsiflexor strength rated 3-4, right foot dorsiflexor strength rated 4, left straight leg raise test positive at 30 degrees, Bragard's test is positive, no knee-ankle reflexes were elicited in either leg, Babinski sign is negative on both sides, and pulses in the dorsalis pedis arteries are palpable in both feet. The patient's lumbar spine CT shows: irregular bone structure in the small joints of lumbar 3-5, with cystic low-density areas under the joint surfaces, increased bone density in the small joints and the lamina (Figures 2a, b, c), perisynovial inflammation and possibly synovial cysts around the small joints of lumbar 3-5? Lumbar 3-5 intervertebral disc protrusion, which is causing secondary spinal canal stenosis. Test results were as follows: blood uric acid 498 µmol/L (normal range at our hospital 202-416 µmol/L), blood glucose 7.6 mmol/L, total cholesterol 5.57 mmol/L (normal range at our hospital 0.01-5.20 mmol/L), other parameters were within the normal range. Considering the patient's age, no history of lumbar trauma, elevated uric acid levels, and mAfter 2 days of conservative treatment with uric acid-lowering and nonsteroidal anti-inflammatory drugs, the patient didn't feel any relief from the pain in the lower limbs and waist, which got worse when he walked. Considering the severe compression of the dural sac in spinal canal and after excluding various surgical the contraindications, a one-sided approach for both sides of the neuroforamen at L3-5 was chosen. They put him under general anesthesia and placed him in a prone position. They used a C-arm Xray to check the surgical area, followed by routine disinfection and covering. A 2.0 mm K-wire was anchored at the junction of the L4 facet joint and the inferior facet joint to help guide the surgery. They made cuts 1.5 cm above and below the left L4-5 space, 0.5 cm lateral to the spinous process, with the upper cut measuring 0.5 cm (viewing channel) and the lower cut measuring 1.2 cm (working channel). An endoscope was placed in the viewing channel, and a disposable bipolar radiofrequency electrode was inserted into the working channel. The anchored K-wire was located under the endoscope to confirm the correct surgical segment. The radiofrequency electrode was used to treat the left L4 inferior lamina, inferior facet joint, and L5 superior lamina soft tissue. They used a bone chisel to take out the L4 inferior lamina to the point of the yellow ligament and one-third of the inferior facet joint, as well as the lower edge of the L5 lamina and one-third of the medial aspect of the L5 superior facet joint. They noticed a chunk of white material between the superior and inferior facet joints (Figure 3), with a lot of white, plastery stuff stuck to the surface of the dural sac and severe adhesions, along with partial ossification of the soft tissue on the dural surface. Using a special tool called Kerrison rongeurs, they took out the ossification and white plastery deposits on the same side (Figure 4), and the L5 nerve root was carefully freed from adhesions, avoiding dural tearing (Figure 5). Several pieces of white material were excised for pathological examination, and they adjusted the



**Figure 1**: Preoperative MRI sagittal and axial views of the lumbar spine. a: The sagittal view of the lumbar MRI shows a cystic lesion with T2 signal, protruding into the spinal canal (indicated by the white arrow), which corresponds to compression of the dural sac, lumbar canal stenosis, and redundant cauda equina. b:The axial view of the L3-4 intervertebral disc shows irregularities in the bone structur reof the facet joints, with surrounding diffuse patchy areas, and narrowing of the spinal canal due to compression. c: The axial view of the L4-5 intervertebral disc shows hyperplasia of the right-sided facet joint (indicated by the white arrow) compressing the nearby nerve.



**Figure2**:PreoperativeCTsagittalandcross-sectionalviewsofthelumbarspine.a:Thesagittalviewofthe lumbar spine CT shows multiple Schmorl's nodes, osteophyte formation at the vertebral body margins, and irregular bone growth at the upper and lower articular processes. b: The cross-sectional view of the L3-4 intervertebral disc shows higher bone density in the articular processes and lamina, with a local bulge into the spinal canal. c: The cross-sectional view of the L3-4 intervertebral disc shows joining and overgrowth of the articular processes.

angle of the working sleeve to locate the central fissure of the yellow ligament, revealing the contralateral yellow ligament endpoint. The yellow ligament was completely excised, and the contralateral proliferative white material was removed with a bone knife. They carefully checked and relieved pressure on the spinal cord and both nerve roots without any compressive material, and they made sure to stop any bleeding. Methylprednisolone was injected around the nerve roots, and the working and viewing channels were withdrawn. They did the same thing for the left L3-4 spinal canal, checking and freeing up the spinal cord and nerves, and removing white material. They stitched up the cuts in layers, with a total surgical duration of 150 minutes. The white chalky material was sent for pathological examination, showing some extra bone tissue with some weird deposits consistent with gout stones (Figure 6). Postoperatively, the patient kept taking febuxostat to lower uric acid and oral celecoxib for anti-inflammatory pain relief. The day after surgery, the patient said the pain was a lot better and could walk normally with a back brace, with muscle strength in both feet rated at 5. A CT scan three days after surgery showed good decompression of the spinal canal, with no major pressure on the duralsac (Figures 7a, b). They checked the patient's blood uric acid level after surgery, and it was 371 µmol/L. More than 9 years of gouty arthritis, the MRI T2 images show high signal in the ligamentum flavum in the spinal canal. The patient refused a biopsy, so it's highly suspected that the lumbar disc compression is related to gout. The diagnosis is:spinal stenosis, gouty

tophi in the lumbar canal, lumbar disc herniation, gouty arthritis, and type 2 diabetes.

#### 3. Discussion

In simple terms, gout is a long-term condition caused by the deposition of monosodium urate crystals (MSU). Elevated urate levels in the blood are the main risk factor for both MSU crystal deposition and gout development [6], Other risk factors include obesity, hypertension, chronic kidney disease, drinking alcohol, sugar- sweetened beverages, and some medications (like thiazide diuretics or loop diuretics) [7]. With the improvement of living standards and changes in dietary structure, the incidence of hyperuricemia has been increasing every year, especially in cities along the coast. For example, the overall prevalence of hyperuricemia among residents of coastal cities in Shandong Province (Yantai, Weihai, Rizhao, Dongying, Qingdao) is 13.19%, with 18.32% of males and 8.56% of females, which means that men are 2.5 times more likely to have the disease than women. [8]. Spinal Gout (SG) is caused by the deposition of urate crystals in the spinal column, which causes pain and inflammation. Studies have shown that risk factors like dyslipidemia, obesity, and a sedentary lifestyle can result in poor blood supply to the cells of the intervertebral discs. This, in turn, contributes to the formation of urate crystals [9]. In 1950, Kersley[10] and colleagues reported the first case of spinal gout, which refers to gout affecting the spine. Later, Rukmini and her team conducted a cross-sectional research study on spinal gout and noted that it mainly affects the lumbar spine, then the cervical spine,

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and lastly the sacroiliac joints [11].Gout affecting the spine can show up with a variety of symptoms. These can include mild spinal pain and spinal cord compression, which can lead to more serious issues [12].In serious cases, it can lead to a partial dislocation of the joint between the first two vertebrae in the neck [13]. Pain in patients with gout of the spine is similar to that caused by a herniated disc. This



Figure3: Showsawhitelump sticking out from the upper and lower joints that was seen during the endoscopy.



**Figure4**: showswhitegouty crystalsspread through the endoscoped uring surgery.

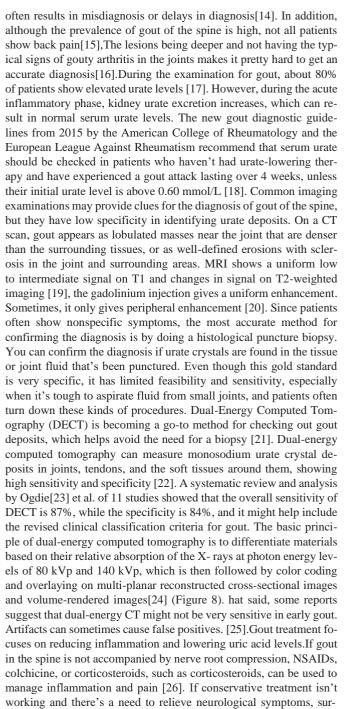




Figure5:Separating adhesions with an endoscoped uring surgery.



**Figure6**: showspostoperativepathology with eosinophilstructureless, which isconsistent with gout.

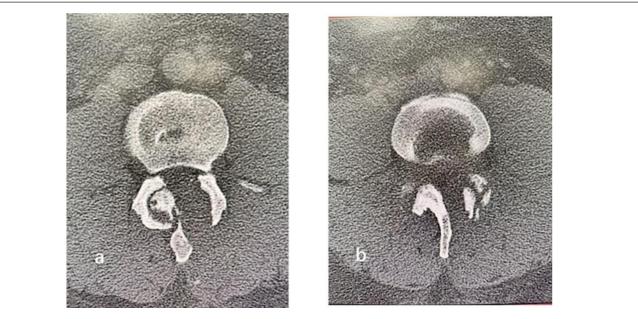
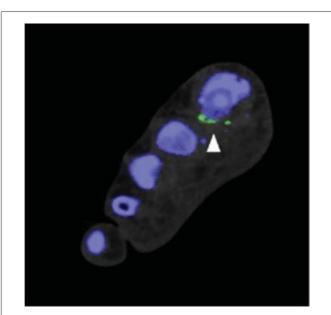


Figure7:CTscanofthelumbarspine3dayspost-surgery.a)L3-4,b)L4-5;theleftsidelaminadecompressionis sufficient.



**Figure8**<sup>[24]</sup>: Thepost-processingimageofthecoronalplanefromthe dual-energyCTdisplaystypicalmonosodiumurate(MSU) depositsontheoutersideofthefirstmetatarsophalangeal joint, as shown by the arrow .In this im age, green pixels represent MSU, blue pixels show cortical bone, and purple shows trabecular or cancellous bone.

gery may be considered, including laminectomy to relieve pressure, mass removal, screw fixation, and interbody fusion, making sure to follow the guidelines and create a personalized treatment plan based on the severity of the patient's condition and overall status. No matter if surgery or conservative treatment is chosen, uric acid levels should be controlled. In this case, the patient is young and has significant compression in the L3-5 spinal canal, left lateral recess stenosis, and minimal damage to the facet joints from gout stones in the canal, which hasn't caused lumbar instability. Given the big trauma, heavy bleeding, and long recovery time that come with open surgery, as well as the patient's wishes, it was ultimately decided to use UBE (Ultraminimally Invasive Endoscopic) technology for laminectomy. UBE has advantages like a smaller incision and a one-sided approach that allows for decompression on both sides, avoiding damage to the contralateral lumbar soft tissue and spinal stability structures, and ensuring adequate decompression of the spinal canal, combined with osteotomy using a bone knife for higher surgical efficiency. The gout stones in this patient were closely adhered to the tissue, and the magnification function of the spinal endoscope could better avoid damage to the nerves and the dural sac. During the procedure, they used a burr and laminectomy forceps to make a big bone window to observe the starting and ending points of the gout stones in the spinal canal, then carefully removed using nucleotomy forceps and a radiofrequency knife. If there is severe adhesion to the dural sac or nerves, a probe can be used for separation, and complete removal is not necessary; just getting the pressure off is enough. It's important to fully remove where the yellow ligament and lamina attach to prevent urate buildup and lower the chance of it comingback. After surgery, they should keep taking medication to control uric acid, treat acute gout attacks, and get advice on diet and lifestyle. The cases reported in this article only developed low back pain and neurological dysfunction in the late stages of lumbar gout, highlighting the difficulty of early diagnosis. Therefore, for patients presenting with significant low back pain, neurological dysfunction, or spinal canal issues, clinicians should thoroughly review the medical history and carry out a thorough analysis of symptoms, signs, and tests to avoid missing or misdiagnosing. If a patient has elevated uric acid levels or a history of gout flare-ups, doctors should consider lumbar gout. Hopefully, this report will help clinicians with the future management and treatment of spinal gout.

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