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# Gout: A Possible Cause of Lumbal Canal Stenosis. Cases Report in Sub-Saharan Area and Literature Review

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## Abstract

**Introduction:** Gout is defined as an arthritic condition resulting from the deposition of monosodium urate crystals in and/or around joints, following long-standing hyperuricemia. This may cause gouty arthritis in joints and tophi in soft tissues. Spinal gout is rare and never mentioned in our context. It can appear as acute back pain, radiculopathy, spinal cord compression, spondylodiscitis or neoplasic/infectious epiduritis. Our aim was to share our surgical experience and proceed of a Literature review. **Cases Presentation:** Between January and August 2022, two patients male were surgically cared, aged of 42 and 60 years old. The gout was unknown in the youngest and poorly followed in the eldest. There was no past medical history of tuberculosis or immunodeficiency in both. The early diagnosis retained was unspecific lumbar spondylodiscitis due to clinical features: Patients complained both of lower back pain with initial fever. It was of a progressive left L5S1 deficit with erectile defect and dysuria in the first case and a progressive paraplegia without sphincter disorders in the second case. We proceeded with a lumbar laminectomy with a biopsy on both patients. The spinal tophus was ligamentous in one case and arthro-ligamentous in the other. There was a progressive motor recovery from postoperative Day-2 till postoperative Month-1. A probabilistic antituberculosis treatment was promptly initiated postoperatively based on radioclinic features while waiting for histologic proof. The *Polymerase Chain Reaction* (*PCR*) *of Mycobacterium tuberculosis* was negative and the histology was of a chronic calcified osteitis with dense fibrosis in both. The anti-gout treatment was implemented after 15 days with blood test evidence.

A rheumatologic follow-up was also initiated and adjuvant physio-therapy. The results were very satisfactory from 4 - 6 months with independent walking. **Discussion Conclusion:** Spinal Gout may be suggested in 40-male-old faced with any acute rachialgia with neuro deficit with dubious neuro-imaging.

## Keywords

Gout, Lumbar Canal Stenosis, Neurosurgery, Spinal Gout

## 1. Knowledge Status

- Spinal Gout quite frequently corresponds to an ultimate evolved step of Gout.
- It can be caused by neurological deficit pictures.
- Surgical radicular-spinal decompression associated with long-term gout treatment improves the short-term functional prognosis.
- The positive diagnosis is essentially histological.

## 2. Contribution of Study

- Spinal gout has been mentioned very little in our context although quite frequent.
- Surgery improves the functional disability created early.
- The bioclinical gout profile must be established in the presence of any disabling or deficient inflammatory back pain.

## 3. Introduction

Spondyloarthritis and rheumatoid arthritis are the two main chronic inflammatory rheumatisms affecting the spine, even in Sub-Saharan Africa. Chronic gout is, although frequent is of less-reflex in the diagnosis of narrowed spinal canal or spinal cord compression. Chronic gout is a chronic tophaceous polyarthritis, characterized by a ubiquitous deposit of monoscopic urates, causing atypical locations such as the spine, ENT, pelvis-abdomen, kidney, myocardium, etc… Spinal (or axial) gout may interest all vertebral structures, including bodies, pedicles, discs, ligamentum flavum, facet joints, epi/intradural space; realizing spondylodiscitis or epiduritis or facet arthritis. The clinical expression is several: intense spinal pain (acute rachialgia), radiculo-cord compression, and peripheral neurogenic deficit syndrome. In 2017, Zang *et al.* listed 287 cases reported in the literature and from 2017 til 2022, *McConville and al* analysed 38 further cases [1]; notwithstanding the 5 African cases by then reported in the Literature [2] [3] [4]. In fact, before this paper, only four African studies have yet been published on spinal gout from 1999 til 2022 [2] [3] [4]. It was for us to report our neurosurgical experience—the first 2 cases from Sub-Saharan Africa operated and followed—to increase diagnostic awareness for this pathology and to do a literature review. The two patients were collected by their onset period and the

clinical and diagnostic similarities.

## 4. Cases Report

Two patients were gathered between *January and August* 2022.

### 4.1. Case 1

It was of a 42-year-old male, admitted in January 2022 for acute rachialgia and walking disorder. He was unknown goutic without a history of tuberculosis or immunodeficiency and without any cardiovascular risk factors (Table 1). The fever and a right L5 intractable neuropathic pain were present initially, limiting positions on the bed with excruciating pain on mobilization.

Clinic features and neuro-imaging evoked lumbar spondylodiscitis with compressive epiduritis L4L5S1 (Figure 1): progressive paraparesis of 3/5 with erectile defect and dysuria. A laminectomy with biopsy was performed revealing a spinal arthro-flavo-ligamentous tophus. It was a firm crystallized matrix of chalky-white appearance on the hypertrophied ligamentum flavum and whitish-granulated tissue on the articular. The compressive epiduritis was cleavable anteriorly. The motor recovery was progressive, from Day-2 postoperative to Month-1 postoperative. Polymerase Chain Reaction (*PCR*) to *Mycobacterium tuberculosis* (*BK*) was negative. Probabilistic tuberculosis treatment was started preoperatively according to radioclinical findings and stopped after histological results on Day-15 postoperatively. This latter, performed under a microscope without polarized light, was in favor of chronic osteitis with calcified sequestra and dissociated dense fibrosis in both cases. Consequently, a gout treatment (*Allopurinol* 100 *milligrams per day and Colchicine* 200 *milligrams per day*) was instituted after bioclinical evidence: persistent hyperuricaemia and arthritic flare (Table 1); as much as a regular rheumatological follow-up, with satisfactory clinical results from the 1st month.

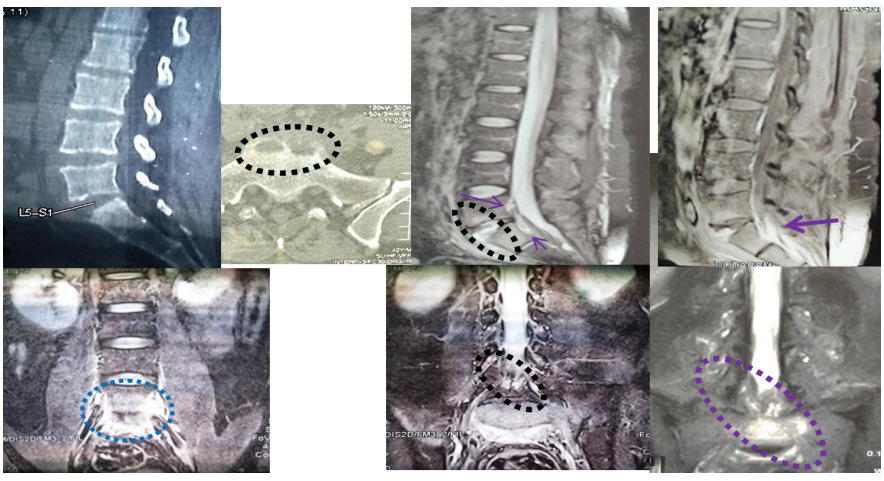


Figure 1. CT-Scan (*top and left*) & MRI (*top and right, down*) of the youngest patient showing L5S1 spondylodiscitis with geodes on S1 body (*black dotted circle*), the anterior epidermis (*arrows and blue dotted circle*)and a crystalloid matrix in L5S1 both (*pink dotted circle*).

**Table 1.** Summary of clinical and radiological signs of reported cases.

| Radioclinic Parameters | Youngest Patient | Elder Patient |
|---|---|---|
| Tuberculosis history | No | Yes |
| Immunocompetence | Yes | Yes |
| Cardiovascular risk factors | Any | • High blood pressure<br>• Glycaemic Instability |
| Fever | Initially | Initially |
| Uncontrollable neuropathic pain | Yes | Yes |
| Motor deficit | Progressive Paraparesia | Progressive Paraplegia |
| Sensitive deficit | L5S1 right Hypoesthesia | L3 Hypoesthesia |
| Genito-Sphincters disorders | Erectile Defect & Dysuria | Any |
| Radiology | L5S1 Spondylodiscitis + Anterior epiduritis + L5 rightfacet jointarthritis | L4L5 Spondylodiscitis + Antero-posterior epidurals |
| Surgery | L4L5S1 Laminectomy + Biopsy | L3L4L5 Laminectomy + Biopsy |
| Macroscopic aspect of the lesion | • Flavo-ligament tophus in white-chalky crystallized matrix<br>• Articular tophus L5 granulated-whitish<br>• Anterior epidurals | • Hypertrophied chalky-white compressive ligament tophus<br>• Antero-posterior epiduritis |
| Postoperative Polyarthritis | • 2 knees<br>• 2 ankles<br>• Cervical Pain | • 2 knees<br>• 2 ankles<br>• 2 Hallux |
| Hyperuricemia Pre- (**a**) & Postoperative (**b**) | **(a)** 58 mg/l = 345.1 µmol/l<br>**(b)** 111.8 mg/l = 665.8 µmol/l | **(a)** 71 mg/l = 422.5 µmol/l<br>**(b)** 107 mg/l = 636.7 µmol/l |
| Histology | • Non-specific Inflammatory tissue<br>• Negative PCR-MT | • Non-specific Inflammatory tissue<br>• Negative PCR-MT |
| Evolvement | • Sensorimotor recovery<br>• Normal gait at 3 months | • Sensorimotor recovery<br>• Normal gait à 6 months |

### 4.2. Case 2

The second patient was also a male of 60 years old, admitted in *August* 2022 for lower limb deficit and sharp back pain, making him sad and whiny. He was known to gout poorly and there was no history of tuberculosis or immunodeficiency found (Table 1). He also has poorly monitored arterial hypertension and glycemic instability. He had a fever in started with neuropathic pain in both limbs. The clinic was in favor of a progressive 1/5 paraplegia without sphincter disorders and the radiology showed lumbar spondylodiscitis with compressive epiduritis L3L4L5 (Figure 2). The laminectomy with biopsy was performed and a spinal flavo-ligamentous tophus. It was also a firm crystallized matrix of chalky-white appearance on the hypertrophied ligamentum flavum and whitish-granulated tissue on the articular. The compressive epiduritis was cleavable anteroposteriorly. The pain had strongly regressed immediately after surgery and the motor recovery was progressive, from Day-4 postoperative to Month-2 postoperative. Polymerase Chain Reaction (*PCR*) to *Mycobacterium tuberculosis* (*BK*) was negative. The tuberculosis treatment also previously started preoperatively was stopped on Day-20 postoperative. It was of the same modalities of laboratory exam evoking chronic osteitis with calcified sequestra and dissociated dense fibrosis. Gout treatment was therefore instituted samely after bioclinical evidence: persistent hyperuricaemia and arthritic flare (Table 1) (Figure 3); as much as a regular rheumatological follow-up, with satisfactory clinical results seen ambulatory at 1 and half months.

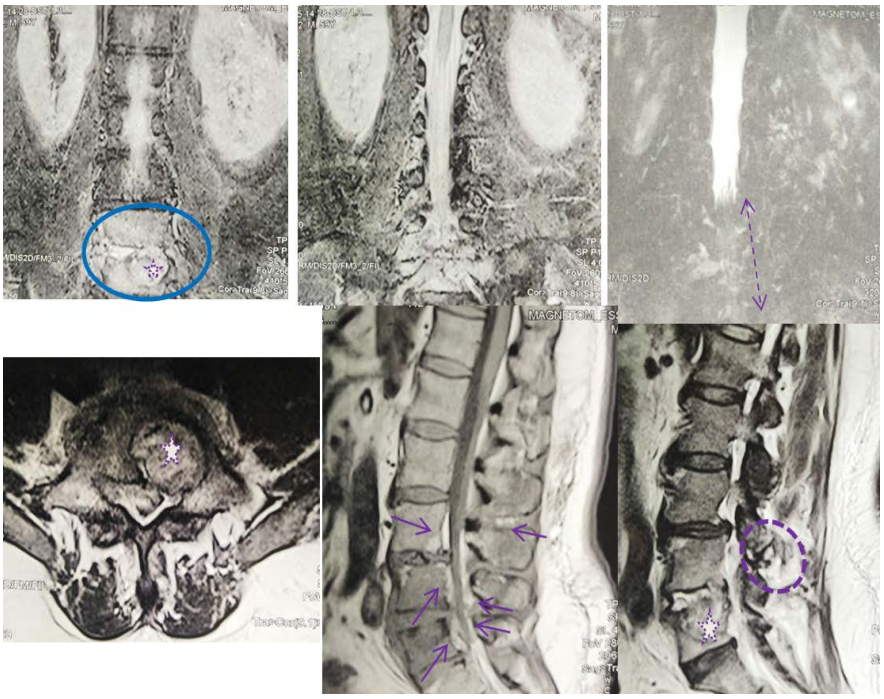

Figure 2. MRI features of elder patient showing L3L4L5 spondylodiscitis with left L4L5 cluster (*white star and blue circle*), the anterior epidermis with facet deposit (*arrows and pinkdotted circle*) and anteroposterior lumbar canal stenosis (*pinkdotted arrows*).

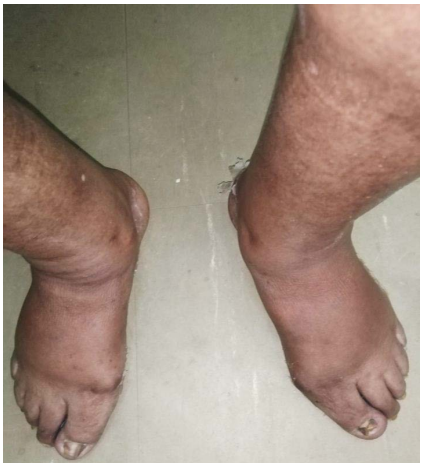

**Figure 3.** Gout polyarthritis (knees, ankles, hallux) occurs in postoperative in the elder patient.

## 5. Discussion

Polyarticular tophaceous chronic gout is the fourth and final progressive stage of gouty disease [5]. The first case of axial gout was described in 1950 by *Kersley GD* on an autopsy case of subluxation by tophic erosion of the Atlas (*C*1) with protrusion of the Odontoid in the Foramen Magnum [6] [7] [8]. *Koskoff YD et al.* described the first gouty myelopathy 3 years later and *Reynolds AF Jr.* in 1976, the first 3 neurosurgical cases of spinal gout caused by compressive flavo-ligament (yellow ligament) tophi [6], as found in our 2 cases. The first African case of spinal gout published in 1999 was a South African patient from Cape Town, performed for progressive T8 spinal compression over 18 months by posterior epidermis and spondylitis [3]. The most recent case of spinal gout found in the literature dates from March 2023, the most recent African case having been published in *January* 2022 [2] [9] (**Table 2**).

The current prevalence of spinal gout reaches 35% of gout patients and due to its atypical presentations; it can be confused with Diffuse Idiopathic Skeletal Hyperostosis (*DISH*), Vertebral Osteomyelitis, Neoplastic or Infectious Epiduritis [10]. Our patients were all male over the age of 40, corroborating the literature which evoked in this condition the male predominance of 85%, an average age of 55 years and the same conditions of onset of gout in Africans or Westerners [1] [8] [11] [12]. The risk factors listed are hyperuricaemia > 68 mg/l (404.6 *µmol/l*), cardiovascular conditions, obesity, blood diseases, and alcoholism… [10]. In Africa, it is associated with homozygous sickle cell disease, particularly in young patients (*mean* = 32 *years*) [11]. In 15% of cases, spinal gout may exist without any clinical history of hyperuricaemia or gout, as seen in the youngest of our patients, and in 30% it may be indicative of gout [8] [12].

The vertebral and/or spinal cord location of urate crystals in clusters, called tophus, surrounded by an inflammatory reaction is the princeps definition of spinal gout. The spinal tophus can be single or plural to all vertebral structures,

**Table 2.** Summary of published african cases of spinal gout.

| N° | Authors & Publications | Countries | Year of Publication | Number of Cases Reported | Reported Cases |
|---|---|---|---|---|---|
| 1 | **Kaye PV and al.** *Cytopathology.* 1999 *Dec*; 10(6): 411-4. | **South Africa** | **1999** | 1 | *Yes* |
| 2 | **Ntsiba H and al.** *Joint Bone Spine. 2010* Mar; 77(2): 187-8. | **Congo** | **2010** | 1 | *No* |
| 3 | **Hounsounou M and al.** *Médecine d'Afrique Noire.* 2020; 67(11): 613-16. | **Ivory Coast** | **2020** | 1 | *No* |
| 4 | **Onana Y and al.** *Jaccr Africa* 2022; 6(1): 56-9. | **Cameroon** | **2022** | 1 | *No* |
| 5 | **Jibia A and al.** | **Cameroon** | **2023** | 2 | *Yes* |

also intra- and epidural spaces [7] [8]. In our observations, the tophi were articular, ligamentous and epidural. Spinal gout can truly mimic infectious spondylodiscitis and/or epiduritis or, conversely, be asymptomatic [13] [14]. In the 2 cases reported, the initial clinical and radiological semiology raised the suspicion of pottic etiology; hence the anti-tuberculosis treatment was started early and then stopped after negative histology. Rachialgia is quite common in axial gout, readily hyperalgesic and febrile, associated with radiculalgia (25% *of cases*) or spinal cord compression (25% *of cases*) [12]. The lumbosacral topography is the most frequent (56%), as observed in our 2 cases. The other cervical and thoracic sites remain at 22% each [1] [10] [12].

Neuroimaging is valuable for diagnosis: computed tomography without/with contrast and especially *DECT = Dual Energy Computed Tomography*. The latter sufficiently differentiates the deposits of sodium urates from the surrounding structures by its attenuation properties; it captures images at 2 different energy levels for comparison as well as color displays. Tophi appearing in hypo-HU (*houndsfield units*): Low dual-energy index (160 - 170 *HU*) compared to the high index of calcium deposits (450 *HU*) [15] [16]. Magnetic resonance imaging is irreplaceable, especially to refine compressive epidurals, spondylodiscitis or vascularized reactive tissue around the tophus. Discovertebral biopsy can be associated with imaging to establish the diagnosis [10] [12].

The diagnostic certainty remains the histology which uses several determinants: the fixation of the sample with absolute alcohol to avoid the dissolution of the urate crystals, noticeable by an optical vacuum; the polarized light microscope for Hematoxylin-Eosin staining and finally De Galantha staining allowing the preservation and visualization of urate crystals or [8] [10] [12]. The surgical

specimens of our 2 cases were fixed with 10% formalin diluted with saline, stained with hematoxylin-eosin and read under a microscope without polarized light.

Surgery promises a better and faster functional prognosis, as noted in our study [8] [15] [16]. It is especially indicated in cases of neuro deficit and is used in several ways: laminectomy + vertebral biopsy ± pleurectomy ± spinal stabilization. Gout and hypouricemic treatment jointly are mandatory to optimize healing, as well as functional physiotherapy.

## 6. Conclusion

Spinal gout is less rare. Diagnostic error or ignorance could explain the scarcity of publications on African cases. Spinal gout should be considered in our sub-Saharan context after 40 years face of any chronic febrile back pain with significant neurological deficit and initial neuroimaging that is not very contributive. Surgery is of short-term diagnostic and prognostic utility.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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