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# Neuroimaging Highlight

Editor: David Pelz

## Gouty Arthropathy of the Axial Skeleton Causing Cord Compression and Myelopathy

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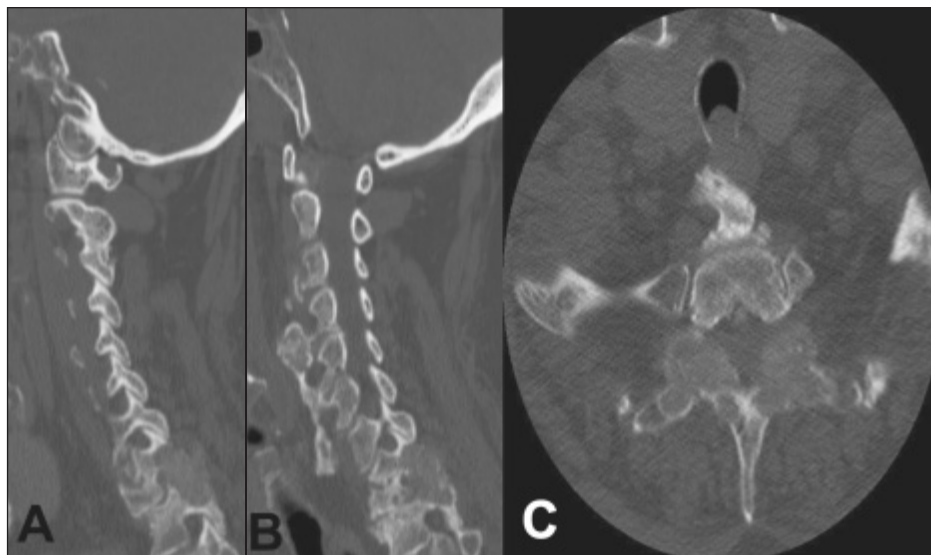
A 70-year-old Caucasian female with a past medical history of hypertension, chronic renal insufficiency, dyslipidemia, hypothyroidism, anemia, congestive heart failure, recent humeral fracture, and longstanding gout, presented with a one-year history of progressive weakness and falls, increasing “clumsiness” in her hands and weakness of the lower extremities. Bowel and bladder function were intact.

On examination, resistance in the lower extremities was reduced, sensation was diminished bilaterally, and hyperreflexia with sustained ankle clonus noted.

Computed tomogram (CT) and magnetic resonance imaging (MRI) of the spine (Figures 1 and 2) revealed a bilateral

destructive process involving the posterior elements of T1 and T2, centered on the facet joints. There was anterior listhesis of T1 on T2 and increased T2 signal within the spinal cord. She underwent posterior decompression with instrumented fusion. Surgical pathology of the spinal mass was sent for histology and crystal analysis (Figure 3). Monosodium urate crystals were detected. The patient’s deficits gradually improved post-operatively and the management of her gout (Serum Uric Acid [SUA] at presentation 792  $\mu\text{mol/L}$ ) was optimized.

Gouty arthropathy of the axial skeleton is well-described, affecting up to 17% of patients with chronic gout and is typically localized to the sacroiliac joints and the facet joints of spinal



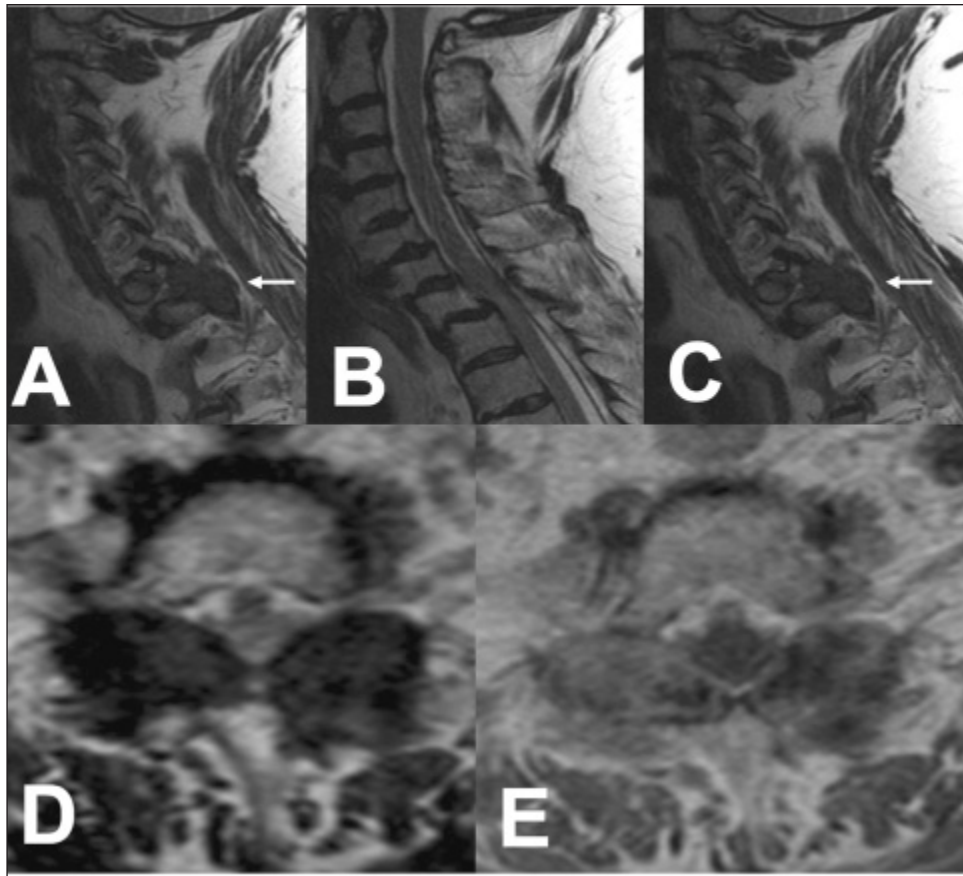
**Figure 1:** Computed tomography of the cervical spine showing (A) left; and (B) right facets in sagittal section; as well as (C) axial section at the level of T1. These demonstrate symmetric destructive processes of bilateral T1 and T2 facets. There is subtle increased attenuation within involved facets.

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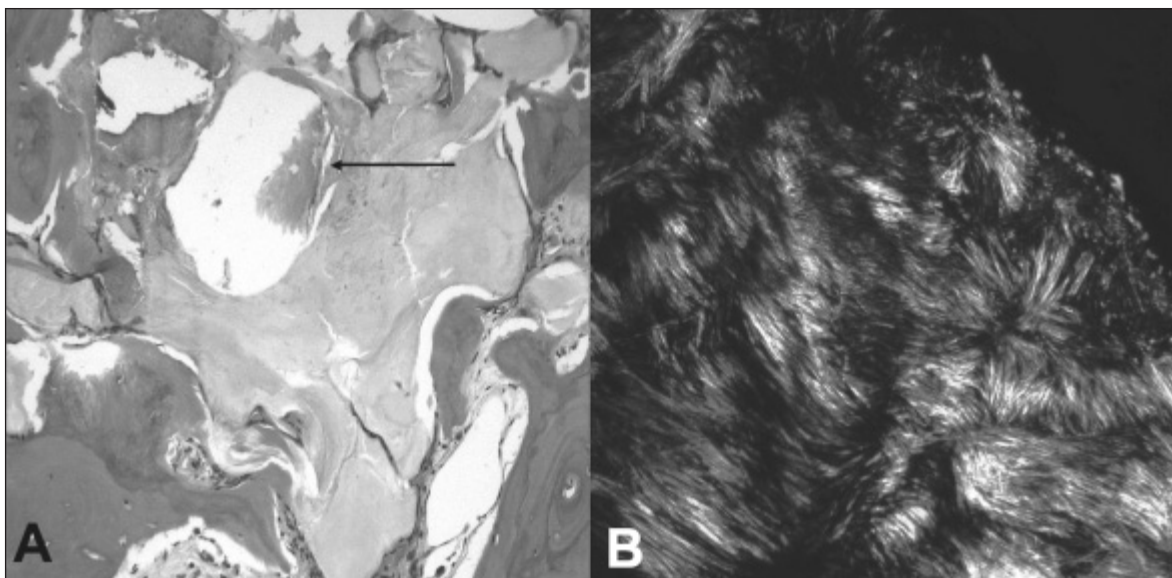
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**Figure 2:** Magnetic resonance imaging showing (A) left parasagittal; (B) mid-sagittal; and (C) right parasagittal section of the cervical spine; as well as (D) T2-weighted; and (E) gadolinium-enhanced T-1 weighted axial sections at the level of T1. The lesions are symmetric, hypointense on T2-weighted images and demonstrate subtle enhancement after gadolinium administration. There is obliteration of the subarachnoid space adjacent to the cord, subtle cord indentation and minimal focal increased T2 signal within the cord.



**Figure 3:** Surgical pathology. (A) Hematoxylin phloxine saffron stain (x100) with arrow indicating gout material in bone marrow spaces; and (B) sample showing negatively birefringent gout crystals with needle-like morphology under polarized light.

vertebrae<sup>1</sup>. The lumbar spine is most commonly affected, followed by the cervical and thoracic segments. While the majority are incidental findings on imaging, the most common presenting symptoms include back pain and radiculopathy<sup>1</sup>. Gouty arthropathy remains an uncommon cause of cord compression and myelopathy.

On MRI, appendicular gout typically shows intermediate to low signal intensity on T1- and T2- weighted with variable heterogeneous enhancement<sup>2</sup>. Diagnostic adjuncts include fluorodeoxyglucose-positron-emission tomography (FDG-PET), where lesions are hypermetabolic<sup>3</sup>. The hallmark of diagnosis is the presence of monosodium urate crystals in the pathological specimen as demonstrated in this case<sup>4</sup>. If gout is clinically suspected prior to surgery, on the basis of the history and imaging features, tissue sampling is imperative, and the specimen must be collected appropriately (in alcohol) and examined rapidly after collection. Formation and solubility of crystals are affected by temperature and pH<sup>5</sup>.

This patient's gout was poorly controlled for many years, as evidenced by markedly elevated SUA at presentation, and history of tophaceous deposits. In patients without neurological compromise, medical treatment of gout has been associated with regression of gouty tophi on serial MRI scans<sup>6</sup>. This is also the mainstay of treatment after decompression, as surgery alone does not change the natural course of the disease. This very unusual presentation of a progressive myelopathy demonstrates, that in the appropriate clinical setting, gouty arthropathy should be considered in the differential diagnosis of destructive spinal lesions.

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