

CORD COMPRESSION IN A YOUNG WOMAN SECONDARY TO SPINAL TOPHI

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INTRODUCTION

Tophi occur in poorly managed long-standing gout. They usually form around the joints, helix of the ear, bursae and tendons.

CASE REPORT

A 39-year-old woman was transferred to Hospital Al-Sultan Abdullah (HASA) from district hospital with a 2-week history bilateral lower limbs paralysis associated with fever, polyarthritis and back pain. Gout was diagnosed 10 years ago and she was maintained on allopurinol 300mg until the current presentation. Her serum uric acid ranged between 520 - 760 $\mu\text{mol/L}$ since the last 2 years. She had been taking prednisolone regularly for gout flares. She also had hypertension, diabetes mellitus and hyperlipidemia.

On examination, BMI was 40 kg/m^2 . She was Cushingoid. She was tender all over especially over the joints and tophi (hands, elbows, wrists, knees, and ankles), some of which had ulcerated. The muscle power of the lower limbs was 1/5 with hypoesthesia from T8 level downward. Tone and reflexes were not performed due to severe pain but Babinski reflexes were negative. There was a grade 4 sacral sore.

The blood culture grew *Staphylococcus* and *Streptococcus* organisms. Ultrasound of the left ankle showed a multiloculated deep subcutaneous collection and joint aspiration yielded sterile chalky substance. Contrasted CT TAP revealed multiple tophi and multiple rim-enhancing collections (Fig 1A-1D). A T2WI axial MRI of the vertebrae showed tophi at T8 causing severe spinal stenosis and cord compression and presence of multiloculated rim-enhancing collections. (Fig 2A-2C)

On Day 22 admission, posterior sacroiliac fusion at the T6/T7 and T10/T11 levels and laminectomy at T8-T9 were performed. Intra-operative findings revealed numerous tophi and pale spinal cord which were non-pulsatile after tophi removal. Postoperatively, there was no neurological recovery. Histopathologic analysis confirmed the presence of monosodium crystal deposits. The sacral sore was debrided on D29 admission and the tissue culture revealed *Pseudomonas aeruginosa*. Two weeks later, another blood culture revealed *Candida albicans*.

She was treated with multiple courses of antibiotics, hydrocortisone for adrenal insufficiency and severe polyarticular gout, and multiple analgesia including oxycodone, morphine and gabapentin. She passed away on D53 admission due to catheter related bloodstream infection caused by *Klebsiella pneumoniae*.

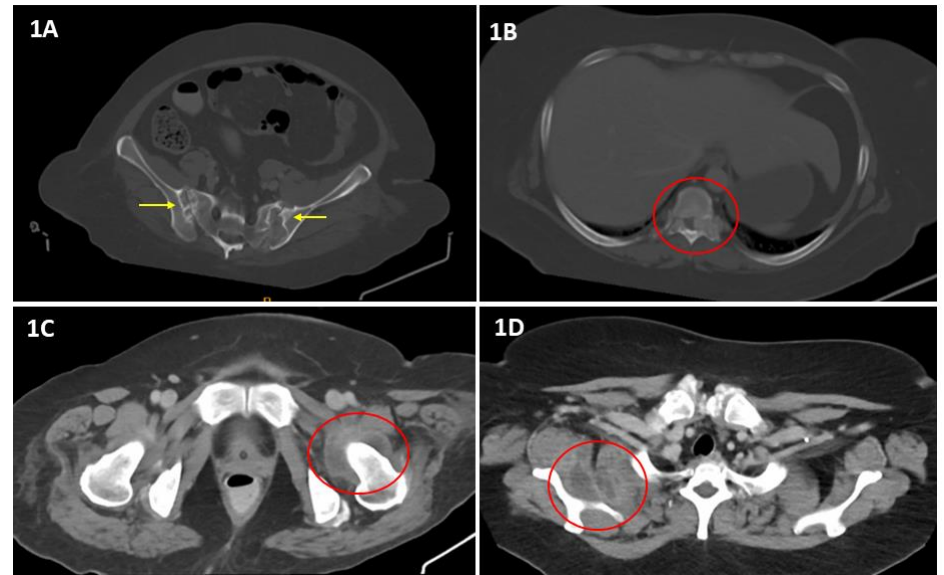


Fig 1: CT images of the contrasted CT thorax, abdomen and pelvis on axial view. Well-defined subchondral erosions with punched out appearances and sclerotic margins are seen at bilateral sacroiliac joints on bone window (A). Multilevel irregular dense spinal tophi, most significant at bilateral T8 pedicles and more pronounced on the left, with intraspinal extension resulting in spinal canal stenosis. This is well demonstrated on bone window (B). On soft tissue window, multiple intramuscular rim-enhancing collections are demonstrated in the left hip (C) as well as right subscapularis and adjacent levator scapulae muscles (D).

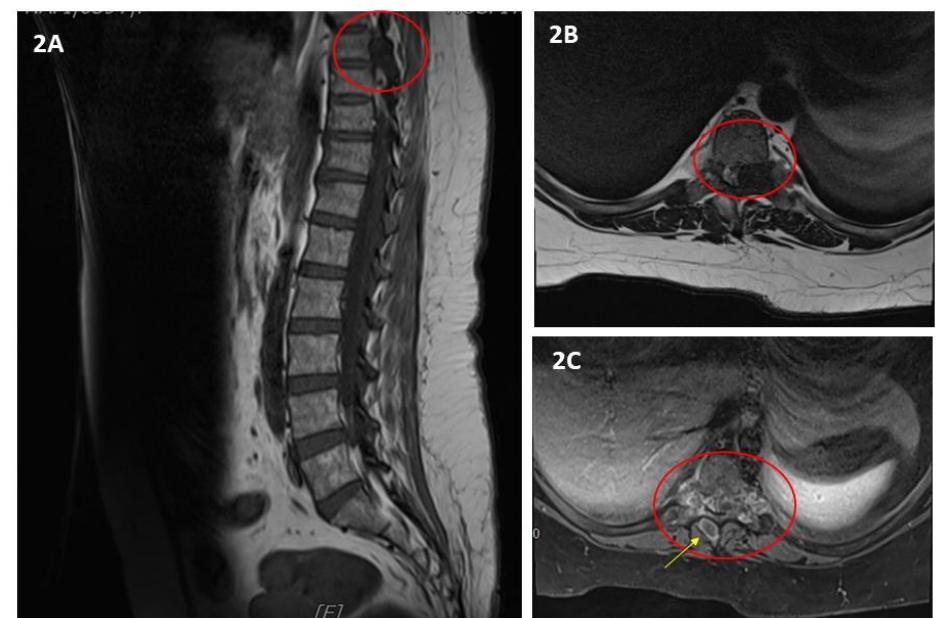


Fig 2: A few representative images of the contrasted whole spine MRI. T2WI axial MRI at T8 vertebral levels shows low signal tophi at bilateral pedicles, particularly on the left side causing severe spinal stenosis and cord compression (2A). The tophus shows similar low signal on T1WI which is shown on sagittal MRI (2B) and following contrast administration, it shows heterogeneous enhancement with a small rim enhancing collection seen in the right multifidus muscle posteriorly (2C). Bilateral exiting nerve roots are not appreciated, likely to be compressed.

CONCLUSION

Gout is curable but this case illustrated a severe gap in gout management. The importance of implementing treat-to-target strategy must be emphasized.