Rehabilitation Practice and Science

Volume 39
Issue 4 *Taiwan Journal of Physical Medicine and Rehabilitation (TJPMR)*

Article 8

12-31-2011

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Recommended Citation

Lin, Chia-Chen and Meng, Nai-Hsin (2011) "Concurrent Ankylosing Spondylitis and Tophaceous Gout in Spine Causing Spinal Cord Compression: A casereport," *Rehabilitation Practice and Science*: Vol. 39: Iss. 4, Article 8.
DOI: https://doi.org/10.6315/2011.39(4)08
Available at: https://rps.researchcommons.org/journal/vol39/iss4/8

Case Report

Concurrent Ankylosing Spondylitis and Tophaceous Gout in Spine Causing Spinal Cord Compression: A Case Report

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Ankylosing spondylitis (AS) rarely coexists with tophaceous gout in the spine. We present a case of a 57-year-old businessman with AS who did not have previous history of hyperuricemia or gouty arthritis. He had a three-month history of progressive bilateral lower limb weakness caused by spinal cord compression (rooted on a tophaceous gout), leading to incomplete paraplegia. He received surgical decompressive laminectomy, which removed the hypertrophic ligamentum flavum and tophus in the level of thoracolumbar junction. There was no obvious neurologic recovery after the surgery.

We reviewed the incidence, clinical manifestations, and management of the spinal tophaceous gout coexisting with AS. Non-steroidal anti-inflammatory drugs are used widely for symptom control in patients with AS; this may lead to an under-diagnosis of gout because its clinical signs can be masked by the NSAID drugs. Spinal tophaceous gout should then be considered for patients with a history of rheumatic disorder and myeolopathy. (Tw J Phys Med Rehabil 2011; 39(4): 253 - 258)

Key Words: spinal cord compression, spinal cord injury, neurogenic bladder, gout, ankylosing spondylitis

INTRODUCTION

Ankylosing spondylitis (AS) and gout are two rheumatic disorders that don't frequently coexist. Concurrent AS and tophaceous gout in the spine is scarcely seen. Symptoms of gout tend to be masked when Nonsteroidal Antiinflammatory Drugs (NSAIDs) are prescribed to control AS. In the present paper, we present a case of spinal cord compression from the 11th thoracic spinal vertebra to the 1st lumbar spinal vertebra levels due to the coexistence of AS and spinal tophaceous gout in a patient without previous history of hyperuricemia or gouty arthritis.

CASE REPORT

The patient is a 57-year-old businessman with a 4-year history of AS under regular medication control. He complained of progressive bilateral lower limb weakness, urinary and fecal incontinence and sensory impairment for three months. Initially, he had lower back pain with bilateral toes and feet numbness, and the symptoms progressed to the proximal thigh. There was no history of

Submitted date: 8 February 2011 Revised date: 24 June 2011 Accepted date: 4 July 2011

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trauma or infection. Progressive bilateral lower limb weakness was also noted, and he later needed a walker to facilitate his mobility. Further evaluation and operation were suggested by a local clinic, but he refused and instead took NSAIDs for pain control. Urinary retention (residual urine over 1000ml within four hours) and voiding difficulty developed, and he was finally sent to our hospital because he could no longer stand up on his own (mainly caused by a deteriorated muscle weakness of the lower limbs).

A physical examination in our emergency department revealed bilateral lower limb paralysis, Medical Research Council (MRC) muscle power grade 0, decreased deep tendon reflex in the bilateral lower limbs and paresthesia from the toes to the knees. An indwelling catheter was inserted to address urine retention. No trauma history or fever was detected in the entire course. Initial lumbar spine x-ray revealed marginal syndesmophyte compatible with AS and spondylolisthesis of the L5 on S1 (Figure 1). However, a lumbar spine magnetic resonance imaging (MRI) showed erosive appearance of the end-plate at T11/12 level with bone marrow edematous change at T11, T12 vertebral body (Figure 2). A long T2 appearance of the spinal cord at T11/T12 level and an infective process with focal spinal stenosis were considered (Figure 3). Contrast enhancement was noted around T11, T12 vertebral body and posterior element and epidural space within the spinal canal. A low signal intensity in the level of T12 and vein engorgement were also noted. Tophaceous lesion is considered (Figure 4).

The patient underwent laminectomy in the T11, T12, and L1 vertebrae levels. The operation revealed a white chalky material (known as tophus) in the epidural space and bamboo spine. The neurosurgeon removed the hypertrophic ligamentum flavum and tophus for decompression.

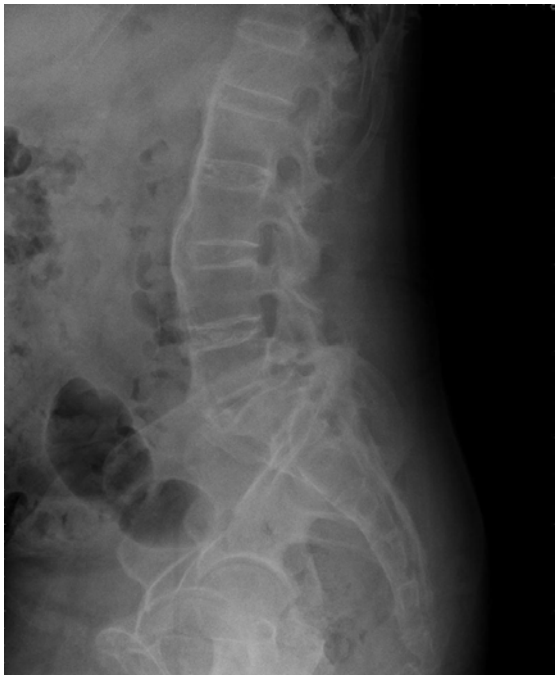

Figure 1. Lumbar spine routine showed: angulation at the sacrococcygeal junction, grade I spondylolisthesis of L5 on S1. Obliteration of the sacroiliac joint. Presence of marginal syndesmophyte compatible with AS.

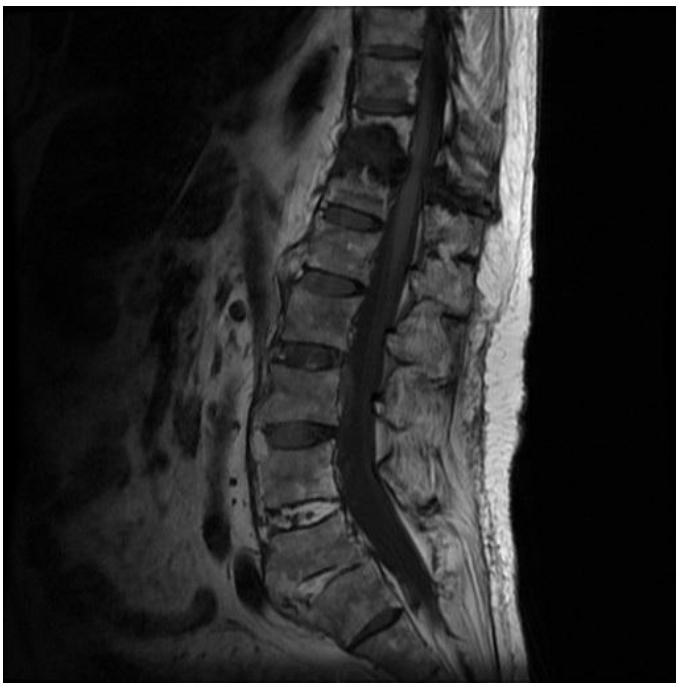

Figure 2. The sagittal T1 weighted MRI images of patient's spine. Erosive appearance of the end-plate at T11/12 level with bone marrow edematous change at T11, T12 vertebral body was seen.

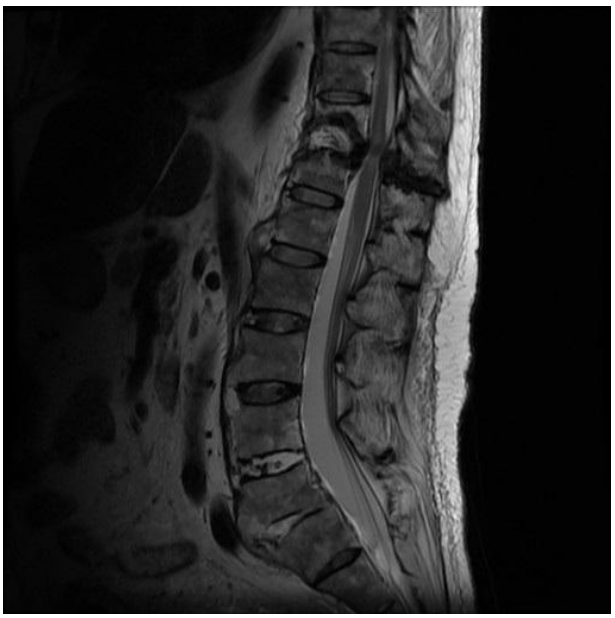

Figure 3. Sagittal T2 weighted images of the patient's spine. Long T2 appearance of the spinal cord at T11/T12 level and an infective process with focal spinal stenosis was considered.

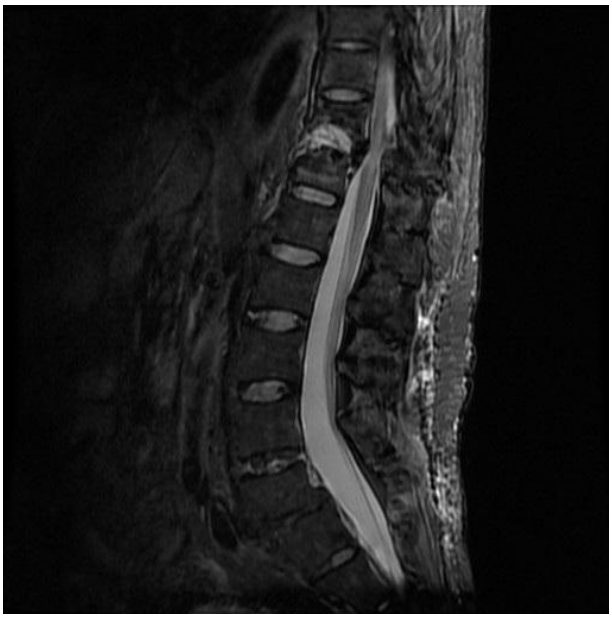

Figure 4. Sagittal T2 weighted images of the patient's spine with contrast. Contrast enhancement around T11, T12 vertebral body and posterior element and epidural space within the spinal canal. Low signal intensity in the level of T12 and vein engorgement are noted. AS with pseudoarthrosis, even the infective process at T11/T12 level with focal spinal cord edematous change is noted . Tophaceous lesion is considered.

There was no obvious neurological recovery soon after the operation. Physical examination one month after the operation failed to show any improvement in the bilateral lower limb strength power (which remained zero below the L2 level), and there was still a severe sensory impairment below the hip girdle area (T12 level). His bladder sensation and anal sphincter tone were severely decreased. Therefore, spinal cord injury at neurological level T12, paraplegia, paresthesia, and neurogenic bladder were indicated in the diagnosis.

Laboratory data did not show signs of uric acid fluctuation and acute onset of gout during the entire hospital course. His white blood cell count remained within the normal range, the C-reactive protein (CRP) was less than 5 mg/dL, the erythrocyte sedimentation rate (ESR) was less than 40 mm/hr, and his uric acid was 6.5-7.4 mg/dL. There were no peripheral signs of gout and history of gouty arthritis.

The patient was transferred to the rehabilitation ward for further training after the operation. He received physical and occupational therapy that focused on training for independence in mobility and daily activities. A urodynamic study arranged for the neurogenic bladder revealed an areflexic bladder. Intermittent self-catheterization was instructed to manage the neurogenic bladder. At discharge, the patient already exhibited a fair to good sitting balance, and could dress himself with minimum assistance. He could eat and take medication by himself but needed other people to prepare the meal for him. He needed moderate assistance when grooming and keeping personal hygiene. He needed maximum assistance when transferring from the bed to the wheel chair. He could not stand on his own and needed a wheelchair for ambulation. He could not control urinary and fecal discharge; therefore, intermittent catheterization was performed, and he needed to wear a disposable diaper for bowel incontinence. No posture hypotension was detected through the entire clinical course.

DISCUSSION

AS and gout seldom coexist, and if they do, they affect mostly the peripheral distal joints such as the knees, feet, hands, wrists and elbows.[1-7] The concurrence of AS and spinal tophaceous gout is seldom noticed. The case raised an important issue on the masked symptoms and signs of spinal tophaceous gout when NSAIDs are administered for AS control. The diagnosis of gout may be missed among patients with AS, particularly those who have spinal gout but without signs of peripheral lesions. The symptoms and signs of spinal gout are similar to those of spinal cord lesions and may include infection, inflammation, trauma, tumor, spinal apoplexy, degenerative change of spine, demyelination, and vascular deformities.[6]

Spinal tophaceous gout should be considered for the differential diagnosis of patients with AS who also develop symptoms and signs of spinal cord compression. A study involving 125 patients[7] showed that lumbar involvements in spinal gout ranked first (40% of the patients), sacroiliac joint involvement came second (28%), and cervical and thoracic involvement shared the same percentage (about 15%).[7]

Gout in the spine may not be detected easily through plain radiograph because some patients with spinal gout may have a normal plain radiograph.[8-10] The preferred method is MRI, which shows homogeneous and low-signal intensity on T1 images as well as heterogeneous and various signal intensities on T2 images.[11,12] The variation on the T2 images may be caused by the different contents of the tophus. The low signal intensity on the T2 images implies calcified content, whereas the intermediate to high signal intensity on T2 images implies water or protein content.[12]

Spinal gout can be prevented and treated as other gouty arthritis in the appendicular skeleton. Several dietary factors, such as alcohol consumption, seafood intake and diseases comorbidities increase the risk of incident gout. Specific medication usage such as loop diuretics is also associated with higher rate of incident gout and flare ups.[13-15] Spinal gout may also cause inflammatory changes, and anti-inflammatory agents are needed. Anti-inflammatory medications are required in addition to uric acid-lowering agents because spinal gout may also cause inflammatory changes. Lowering the blood uric acid level can suppress the deposition of crystals and control the progression of tophus.[16] Patients with a higher blood uric acid level were previously reported to have higher risks of acquiring concurrent AS and gout than those with normal blood uric acid level.[16] No exact cut point is identified as to how gout and blood uric acid should be controlled strictly in AS patients. However, our case showed a normal blood uric acid level. We should then consider tophaceous gout for patients with AS who also develop symptoms and signs of spinal cord compression. Surgery is the first choice for the treatment of spinal cord compression. Most cases undergo decompressive surgery whereas some require spinal fixation.[3] Early diagnosis and treatment of spinal gout can prevent surgical intervention.

Gout and AS are two distinct disorders but they share some characteristics such as predominant occurrence in men, familial and genetic predisposition, involvement of the joints in entheses and good response to NSAIDs therapy.[1] The tophaceous spinal gout was an incidental finding in our patient who had been taking NSAIDs for AS control.[1] The onset of AS usually precede the onset of gout,[1] and NSAIDs therapy usually starts before gout is diagnosed. Some patients with rheumatic diseases may even choose to control the symptoms by taking over-the-counter NSAIDs. Consequently, the clinical signs of gout can be masked easily by the medications, delaying the diagnosis of gout among patients with rheumatic diseases.[1]

CONCLUSION

The concurrence of AS and spinal tophaceous gout is rare. Spinal tophaceous gout should be considered for patients with a history of rheumatic disorder and myeolopathy because the symptoms of gout may be masked by NSAIDs often taken to control AS.

ACKNOWLEDGMENT

Thanks to Dr. Pei-Yu Yang, department of Physical Medicine and Rehabilitation, for supervising the article and makes it more specific for further research.

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僵直性脊椎炎併發脊椎痛風石造成脊髓壓迫：病例報告

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這是一位有僵直性脊椎炎的 57 歲男性，過去並無痛風的病史，主訴近三個月下肢有漸進性的無力，感覺異常，及大小便失禁。患者接受脊髓減壓手術，並將壓迫物移除，手術報告爲痛風石所造成之神經壓迫。我們發現雖然痛風石影響中軸骨骼的機會較少，但是仍爲每位背痛患者的鑑別診斷之一，當僵直性脊椎炎與痛風石壓迫脊髓同時並存，由於病患爲了治療僵直性脊椎炎而長期服用非類固醇抗發炎藥物，導致痛風的症狀不易被發現，尤其是在脊椎的痛風石。如此造成之併發症十分嚴重。甚至造成嚴重脊神經損傷，無論在有無痛風病史之疑似脊髓損傷患者，都應將痛風石壓迫的原因列爲考慮。除了手術治療外，將來也需長期控制血中尿酸並接受復健治療，方可及早恢復生活功能。（台灣復健醫誌 2011；39(4)：253 - 258)

關鍵詞：脊髓壓迫(spinal cord compression)，脊髓損傷(spinal cord injury)，神經性膀胱(neurogenic bladder)，痛風(gout)，僵直性脊椎炎(ankylosing spondylitis)

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