Unusual presentation of gouty spondylitis following antituberculosis therapy

Sirs,

Gout usually affects the peripheral joints but there have been a few reports of spinal involvement (1–4). Cervical, thoracic, and lumbar sacral spine involvement have all been reported with the lumbar spine being most commonly affected site. The clinical features vary, ranging from axial back pain alone to various neurological symptoms, including radiculopathy, neurogenic claudication, myelopathy and cauda equina syndrome. We encountered a case of gouty spondylitis induced by antituberculosis therapy.

A 69-year-old man presented with acute low back pain and polyarthralgia in association with fever. He had a prior history of gout and pulmonary tuberculosis. He had been treated with antituberculosis drugs, which included ethambutol and pyrazinamide. Severe tenderness and swelling was noted in the area of the lumbar spine and over both the 1st–3rd metatarsophalangeal and ankle joints. The laboratory test revealed the following: a white blood cell count of 12,000/mm³, a hemoglobin level of 11.2 g/dL, a platelet count of 741,000/mm³, an erythrocyte sedimentation rate (ESR) of 120 mm/h, a C-reactive protein (CRP) level of 17.9 mg/dL and a serum uric acid level of 12.9 mg/dL. The results of the blood and urine cultures were negative. A plain lumbar spine radiograph showed a narrowing of the L3-L4 disc space with sclerosis and an erosion of the end plates. Magnetic resonance imaging (MRI) was performed to further evaluate the back pain. Fat-suppressed T2-weighted MR image showed heterogeneous increased signal intensity lesions involving both the L3 and L4 bodies (Fig. 1). Empirical antibiotic therapy was begun under the impression of infection and confirm gouty spondylitis. There are few reports of gouty arthritis induced by antituberculosis drugs (5, 6). However, there are no reports of an association between gouty spondylitis and tuberculosis medication. To our knowledge, this is the first case of gouty spondylitis induced by antituberculosis therapy with ethambutol and pyrazinamide. The mechanism underlying hyperuricemia by antituberculosis drugs is an interference of the renal tubular excretion of urate (7). Hyperuricemia and arthralgia induced by antituberculosis drug usually disappears after stopping the treatment (8).

This case shows that colchicine prophylaxis and hypouricemic therapy may be required to prevent an attack in patients with gout undergoing antituberculosis therapy. Gouty spondylitis may be often difficult to distinguish from infectious spondylitis due to the similar clinical signs (9, 10). Furthermore, gout and infection can also be coexistent. However, a prior history of gout, the presence of peripheral tophi and a negative culture result would suggest spinal gout. Spine MRI shows abnormal T1- and T2-weighted signal intensities with the gadolinium-enhanced images. However, these changes are nonspecific and may also be found in infectious spondylitis (10). Therefore, a closed-needle aspiration under CIT guidance may be needed to rule out a spinal infection and confirm gouty spondylitis.

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References