TOMATOCIOUS GOUT OF THE AXIAL SKELETON

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Abstract:
A 72-year-old female with a recent episode of podagra, without peripheral lumps, presented with nocturnal back pain and a destructive lesion of the right lamina of L4. A tophus was excised at laminectomy. Evidence of an axial skeletal tophus causing an irritative radioluphritis via mass effect is presented. (Aust NZ J Med 1988; 18: 865-867.)

Key words: Gout, skeletal tophus.

CASE REPORT
A 72-year-old female was referred for the investigation of back and right calf pain, and a destructive lesion of the right lamina and inferior articular process of the L4 vertebra. The patient had experienced lower back pain for one year prior to presentation, with nocturnal pain in the month prior to admission. She had become bedfast as a result of pain. There was a history of right shoulder pain consistent with an irritative radioluphritis in the three weeks prior to admission. Past medical history was notable for hyperension, untreated primary hyperparathyroidism,-controlled cardiac failure and chronic obstructive pulmonary disease. A right nephrectomy had been performed four years previously for adenocarcinoma. Asymptomatic hyperurnemia was first noted 12 years previously. This had remained untreated until two months prior to admission when she experienced right sided podagra treated with indomethacin, and subsequently allopurinol. Therapy included frusemide, allopurinol, colchicin, probactin, potassium supplements, dextroamphetamine.

She was grossly obese, and no peripheral tophi were noted. Percussive tenderness was present over the L4 vertebra, but no dural tension or meningeal signs were present. The mictococeal scar was free of recurrence, and there was no evidence of lymphadenopathy or hepatosplenomegaly. The appendicular skeleton was normal, anal tone was normal and all deep tendon reflexes were present and symmetrical.

Laboratory investigations showed an ESR of 14 mm/hr and peripheral blood count was normal. Serum creatinine was 0.12 mmol/L, serum uric acid was 0.35 mmol/L, serum free calcium was 2.85 mmol/L and serum phosphatase was 0.95 mmol/L. A skeletal survey showed no areas of bone destruction, however a computed tomogram of the lumbar spine showed a destructive lesion of the right L4 lamina (Fig. 1). 99mTc bone scintigraphy showed increased uptake on the right side of the L4 vertebra (Fig. 2). On two occasions, attempted fine needle biopsy failed to yield diagnostic material.

To establish the nature of the lesion, an L4 laminectomy was performed, disclosing a 4x4 cm tophaceous mass which was easily shelled out from the residual right L4 lamina, the apophyseal joint being largely destroyed. Histological examination showed multiple crystaline deposits within the subchondral bone and adjacent fibrocartilage, surrounded by histocytes and multinucleated giant cells. Polarized light microscopy demonstrated monosodium urate crystals.

The post-operative course was complicated by an episode of polyarticular gout affecting both knees, ankles and first metatarsophalangeal joints. Joint aspiration revealed intra-articular monosodium urate crystals, and the attack responded to intra-articular corticosteroids.

DISCUSSION
This patient presented with the diagnostic problem of a destructive lesion of the L4 vertebral lamina on the background of left breast adenocarcinoma. The recent onset of nocturnal pain was suggestive of metastatic disease. Attempted fine-needle aspiration biopsy of this lesion which was demonstrated by bone scintigraphy and computer tomography twice failed to establish the diagnosis. A gouty tophus was found at operation, and a polyarticular attack of gout occurred post-operatively.
the diagnosis being confirmed by the demonstration of intratubular monosodium urate crystals in the involved joints.

Our patient is the 14th such case of tophaceous gout involving the axial skeleton, with the diagnosis having been established ante-mortem since Kersney’s initial report.1 It is only the fourth case of axial skeletal tophaceous gout occurring in a patient without perihyal tophi.5 Axial skeletal tophi have been reported on several occasions in the cervical,3,4 thoracic5,6 and lumbar sacral spine.5,7 All but one of these cases have occurred in patients with longstanding tophaceous disease.

The incidence of axial skeletal involvement in gout is difficult to estimate. From these anecdotes, which are supported by only two well documented post-mortem examinations,3,4 it is impossible to meaningfully estimate the prevalence of axial involvement in gout. Liebow and colleagues5 reported 11 autopsy cases, commenting in detail upon one patient in whom asymptomatic urate deposits were found in the intervertebral discs, and contiguous vertebral bodies. Details of the axial skeletons of the other 10 cases is however, lacking. Hall and Sellin7 reported one case of asymptomatic tophi involving the posterior facets of L4/5 and L5/S1 in a patient with chronic tophaceous gout. Other than these two reports, no other studies are available to allow the frequency of spinal tophi to be estimated.

Thach8 surveyed 100 patients with proven gout to estimate the incidence of acute crystal arthropathy of the axial skeleton in such patients who developed back pain. Significant episodes of pain in the cervical, lumbar and thoracic spine were reported in 74, 72 and 55% of cohorts, respectively. The etiology of these painful episodes in this study is however, unclear. It is likely that other common causes of regional pain such as cervical spondylosis, diffuse intervertebral skeletal hyperostosis (DISH) and simple mechanical back pain may have been responsible for pain in this instance, rather than acute axial gout. Similarly Jovic9 reviewed 54 patients with documented gouty arthritis, 32% of whom had experienced episodes of acute back pain lasting between seven to 14 days, noting DISH in 34%. As was the case in our patient, destructive lesions of the lumbar vertebrae were seen, but the nature of these destructive lesions was not confirmed by biopsy.

It is clear that chronic tophaceous gout may involve the axial skeleton in both the symptomatic and asymptomatic hyperuricemic population. Such patients may harbour asymptomatic tophi, recognised only when significant mass effects resulting in a compressive or irritative radiculopathy ensue. This may result in a substantial underestimate of the prevalence of axial skeletal involvement in gout. More extensive post-mortem studies may help to clarify this issue. The question as to why so few patients experience axial disease is largely unanswered. It is important to consider tophi in the differential diagnosis of destructive lesions of the axial skeleton in patients with and without chronic tophaceous gout.

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References