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Tophaceous gout of the first costochondral junction in a heart transplant patient

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Abstract We report the case of a 49-year-old man with a 10-year history of gout, who presented with a painful left first costochondral junction mass. A computed tomography (CT)-guided biopsy of the mass revealed foreign body giant cell reaction and crystalline deposition consistent with tophaceous gout.

Introduction

Gout is a common inflammatory disorder caused by hyperuricemia and deposition of urate crystals in and around joints and soft tissues [1, 2]. Gout primarily affects the peripheral skeleton and has two major stages of disease. Gouty arthritis results from acute inflammation secondary to urate crystal deposition within the joint. Chronic tophaceous gout develops from established disease and is secondary to urate, protein matrix and inflammatory cell deposition in cartilage, bone, tendon and other soft tissues. Recent studies have demonstrated an increased incidence of gout in transplant recipients, especially in those patients who have undergone heart transplants. Risk factors for developing gout in this patient population are renal insufficiency and cyclosporine use [3–5]. Reports of uncharacteristic deposition of gouty tophus have been described in the spine [6], intestine [7] and carpal tendons [8, 9]. We report a case of a heart transplant recipient who developed tophaceous gout of the costochondral junction. To our knowledge, this has not been described previously in the English language literature.

Case report

The patient was a 49-year-old man who had undergone orthotopic heart transplant for viral cardiomyopathy 18 years prior to presentation. Following the transplant the

patient developed chronic polyarticular tophaceous gout primarily in his knees and ankles with tophaceous deposits over the dorsal surface of his right fourth metatarsal. The patient had been using cyclosporine for immunosuppression until 6 months prior to admission, when his regimen was changed secondary to progressive renal failure. His current transplant-related immunosuppression regimen consisted of oral doses of Rapamune 2 mg and Prednisone 5 mg daily, and Cellcept 500 mg three times daily.

Forty-eight hours prior to admission the patient complained of pleuritic chest pain and had a fever of 39.5 °C. Local examination of the chest revealed no focal mass or erythema. A chest radiograph (Fig. 1) revealed left upper lung zone opacity, and the patient was admitted for presumptive pneumonia and started on intravenous antibiotics. Laboratory results included: WBC 10,190/μl (normal <9.3), blood urea nitrogen 47 mg/dl (normal <20), creatinine 2.8 mg/dl (normal <1.2), serum uric acid 8.6 mg/dl (Normal <8.9).

A non-contrast chest CT scan (Fig. 2A) was performed which demonstrated an irregular 4.5 cm x 4.9 cm mass arising from the left first costochondral junction. It showed soft tissue density (Fig. 2B) and adjacent rib destruction. No involvement of the left sternoclavicular joint was evident from imaging. There was no evidence of pulmonary airspace disease.

A CT-guided core biopsy was performed. Histologic examination with H&E stain (Fig. 3A) disclosed fibrous tissue with foreign body giant cell reaction. Examination

Fig. 1 Posteroanterior chest radiograph demonstrated a density over the left lung (arrow)

under a polarized light filter (Fig. 3B) revealed birefringent needle-shaped crystals consistent with the monosodium urate crystals of gout. The biopsy was negative for malignancy, and cultures for bacteria, fungi and acid-fast bacteria were also negative.

To treat the patient's acute gout, his Prednisone dose was increased to 15 mg daily. Colchicine was increased from 0.6 mg daily to twice daily with careful observation of his renal function. The patient's allopurinol dose was maintained at 100 mg daily with a long-term plan to titrate the dose to keep serum urate levels below 6.0 mg/dl. His pain gradually improved and the patient was discharged a week after the biopsy.

Discussion

Burack et al. [3] have demonstrated that up to 80% of cardiac transplant patients develop hyperuricemia and up to 10% develop gout. The onset of acute gouty arthritis occurred after a mean of less than 1.5 years of asymptomatic hyperuricemia, with progression to polyarticular tophaceous gout in nearly half of afflicted patients within less than 3 years after the first episode of acute gout. Review of three retrospective studies ($n=47-182$) revealed an average of 10–25% of post-cardiac transplant patients developed acute gouty arthritis within 1.5–3 years after transplantation [3–5].

The high prevalence of gout in these individuals reflects concomitant renal insufficiency and diuretic use, as is the case with our patient. It has also been suggested that cyclosporine can cause a specific renal tubular defect in the handling of uric acid [3, 10]. Hyperuricemia is a central factor in the development of gout, but not the sole determinant [2]. Although our patient had a normal serum uric acid level on admission, his average serum uric acid on 33 previous tests was 11.2 mg/dl (normal <8.9 mg/dl).

Considering the location of the lesion, a strong consideration for the diagnosis in this case was chondrosarcoma [11]. The patient's immunosuppressed status also raised concern for fungal infection [12], post-sternotomy infection [13] and tuberculosis [14]. Chondrosarcoma has been commonly described to occur near the costochondral junction with a lobulated mass containing scattered amorphous calcifications [12].

While the imaging appearance of gout on radiographs has been well described, more recent articles have described the CT findings. Gerster et al. [15] reported the CT appearance of round and oval masses with an average density of 160 Hounsfield units (HU) as being specific to tophus deposition. The average density of the mass in our patient measured 160 HU. The density of monosodium urate crystal is less than that of calcium crystal, which is

Fig. 2 A CT image demonstrates a lobulated first costochondral junction mass measuring 4.9×4.5 cm. The appearance is worrisome for neoplasia. **B** CT image with soft tissue windows demonstrates a soft tissue component to the mass

Fig. 3 A H&E-stained photograph showing foreign body giant cell reaction (20 × objective). **B** Examination with polarized filter reveals birefringent crystals with gout (original magnification ×20)

usually around 450 HU, but higher than that of soft tissues or xanthomatous deposits [16]. Gerster et al. [17] suggested increased x-ray attenuation of tophaceous deposits, relative to soft tissues, may be secondary to a high concentration of sodium atoms.

Acute attacks of manubriosternal joint gout have been previously described [18, 19], and gout has been reported

in the sternoclavicular joint [20]. To our knowledge, however, this is the first reported case in the English literature of gout at the costochondral junction.

In conclusion, gouty tophus in an uncharacteristic location should be considered when a mass lesion is seen in a heart transplant recipient patient.

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