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CASE REPORT

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Periosteal gouty tophi of the anterior mid tibia

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Abstract We report the occurrence of gouty tophi in a 45-year-old man involving the surface of the mid tibia overlying the site of a remote fracture.

Key words Gout · Gouty tophus · Periosteum

Introduction

Gout is the common end point of a group of disorders that produce hyperuricemia. It is usually characterized by episodic acute attacks of monoarticular arthritis with the eventual development of tophaceous deposits of monosodium urate crystals. These tophi may appear in the articular cartilage of joints, in the periarticular ligaments, tendons, ear lobes, nasal cartilage, skin, or kidneys [1]. Their occurrence in the periosteum is rare, especially in patients with no prior history of clinical gout.

Case report

A 45-year-old man presented in February 1992 with severe pain and discomfort in both knees, both ankles, and the left anterior leg. On examination he had swollen hands, wrists, and fingers. He limped on walking. Both knees were swollen and bilateral crepitation was noted. An area of swelling and redness over the anterior left tibia was present. Tenderness on deep palpation was elicited and a fluctuant mass overlying the anterior tibia was noted. Plane radiographs of the left leg showed an old healed fracture involving the middle one-third of the tibia with a compression plate and screws in situ. Of particular note was what appeared to be an aggressive periosteal reaction along the medial distal two-thirds of the tibia adjacent to the plate. Areas of calcification or possible bone formation were seen (Fig 1).

In 1969 the patient had sustained a closed fracture of the tibia that required open reduction and internal fixation with a plate and screws. The fracture site was relatively asymptomatic until shortly before presentation at our hospital, when the patient developed pain in that region. The patient's medical history also included a diagnosis of Reiter's syndrome in 1979, with arthritis, urethritis and conjunctivitis. Since then he had suffered recurrent flare-ups in different joints and subsequently developed psoriasis. The possibility of psoriatic arthritis was actively entertained. His serology was negative for HLA B27, ANA, anti-DNA antibodies, and rheumatoid factor. Sedimentation

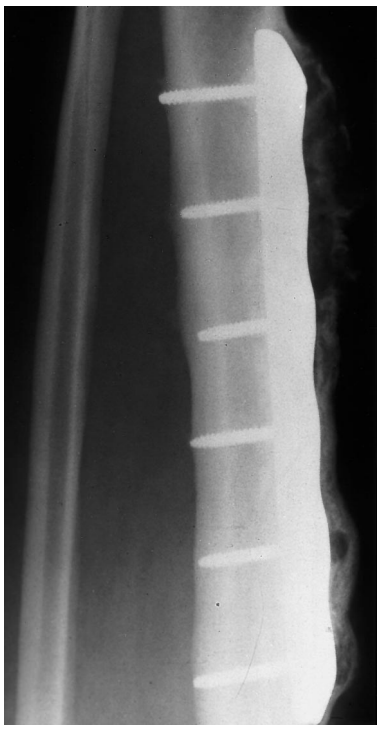

Fig. 1 The tibia and fibula with a compression plate and screws noted at the site of the old healed tibial fracture. Note the apparent aggressive periosteal reaction along the medial cortex of the tibia with what appears to be areas of new bone formation or calcification

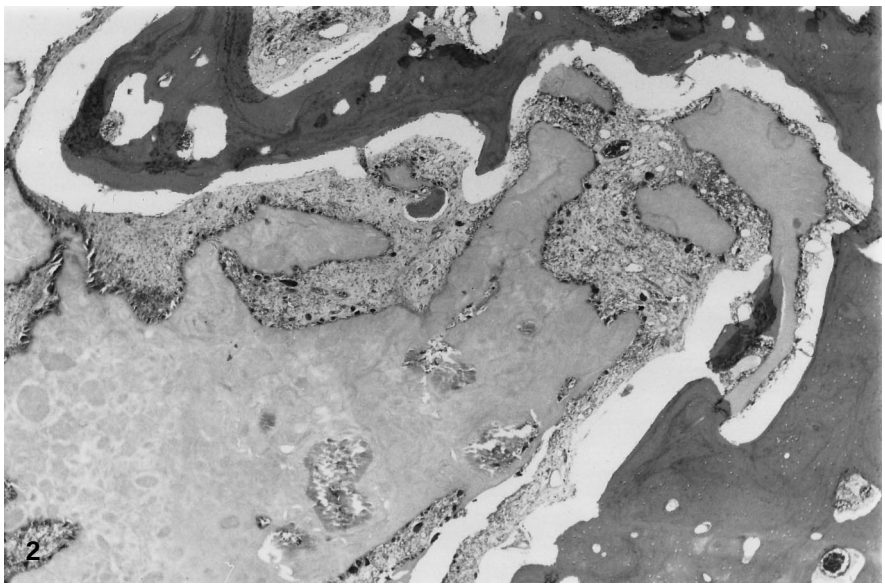

Fig. 2 Low-power magnification showing tophaceous deposits with irregular geographic outlines surrounded by bone

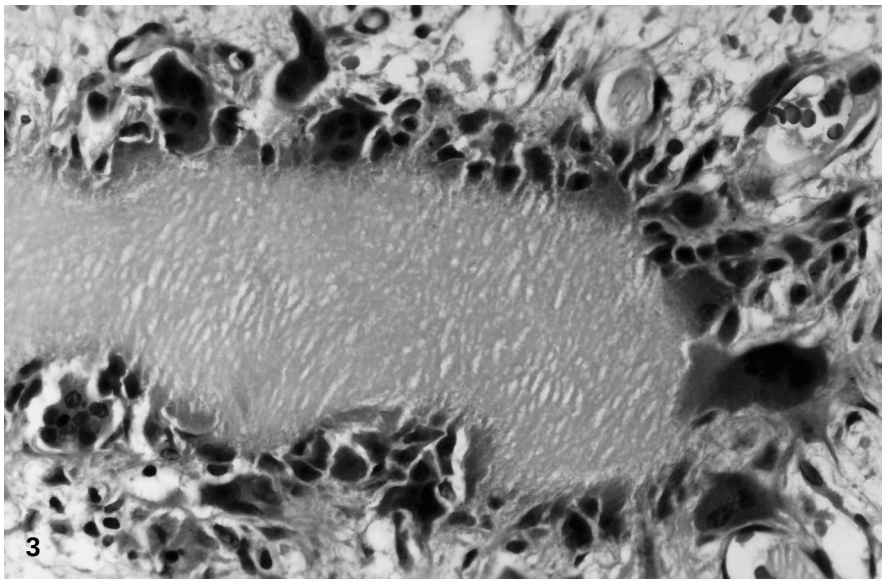

Fig. 3 High-power magnification of tophaceous deposits showing central crystalline and proteinaceous material rimmed by mononuclear macrophages and multinucleated giant cells

rate was elevated. Uric acid was elevated at 579 mmol/l (normal range for males 210–450 mmol/l), which was attributed to the psoriasis. His family history was significant in that two of his brothers had gout. Despite an elevated serum urate, the patient himself did not present with clinical gout. The patient underwent a biopsy of the periosteal lesion of the anterior left tibia.

Preoperative diagnostic considerations from the radiographs included chronic infection and periosteal osteosarcoma or chondrosarcoma. The possibility of metastatic disease was also entertained. The diagnosis of gouty tophi was not considered radiologically prior to surgery.

At operation soft white “cheese-like” material found around the lower end of the plate extruded through the surgical incision. This material was curetted and sent for histological examination. Representative material was sent for culture. Bone had overgrown in that area and the excess bone was osteotomized. The plate and screws were removed with difficulty.

Histological examination of the bony tissue shaved from the tibial surface showed typical gouty tophi surrounded by a mixture of mature and immature woven sclerotic bone (Fig. 2). This bone showed remodelling activity with osteoblastic rimming and osteoclastic resorption related to the tophi. The latter showed primarily pale-pink, amorphous material with aggregates of brown, negatively birefringent uric acid crystals rimmed by numerous multinucleated giant cells and histiocytes (Fig. 3).

Discussion

Gout, a constitutional and frequently hereditary disease of uric acid metabolism, is characterized by hyperuricemia. It is one of the more common crystalline deposition diseases. Urates and metabolites are produced in amounts exceeding the body's ability to eliminate them [2]. A variety of conditions producing hyperuricemia are known. Those patients presenting with clinical gout caused by primary enzyme defects are labelled as suffering from "primary gout", while those individuals in whom the cause of the hyperuricemia is known, increased nucleic acid turnover for example, are designated as suffering from "secondary gout". The pathologic findings are related to the deposition of this needle-shaped, negatively birefringent crystal of monohydrate in joint fluid, in articular cartilage, in subchondral bone, in synovium, in joint capsules, in para-articular tissues including bursae and tendons, and in other tissues of the body such as the kidneys [3, 4]. Less commonly, gout may present as solitary bone neoplasms [5] or skeletal cysts [6]. It can present in association with osteonecrosis [7] and can be the underlying lesion leading to a pathologic fracture [8]. Tophaceous gout may simulate an infection about orthopedic hardware [9] and has been demonstrated as a contributing cause of aseptic loosening of a hip prosthesis [10].

The presentation of tophaceous gouty deposits in the periosteum is unusual. One wonders whether the micro-environment at the site of the previous fracture promoted deposition of uric acid, as has been postulated in instances of tophaceous gout accompanying osteonecrosis [7, 11]. Necrotic bone at a fracture site along with the accompanying reparative process may actually alter the surrounding tissues by lowering tissue pH and favoring the deposition of monosodium urate crystals [7]. Low pH is thought to play a role in initiating urate deposition in tophi and synovial gouty arthritis [12]. The osseous tissue surrounding the tophi in this case was a mixture of mature lamellar and immature woven bone. We speculate that the continued presence of uric acid crystals at the site of the fracture callus accounted for the ongoing remodelling of the surrounding periosteal bone. This would explain the lack of complete maturation of the woven bone during this long period of time.

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References

1. Cotran RS, Kumar V, Robbins SL. Skeletal system and soft tissue tumors. In: Schoen FJ (ed) Pathologic basis of disease. Philadelphia: Saunders, 1994: 1255–1258.
2. Bogumill GP, Schwamm HA (eds). In: Metabolic diseases of bone. Orthopaedic pathology. Philadelphia: Saunders, 1984: 280–292.
3. Milgram JW. Crystalline deposition diseases. In: Gruhn JG (ed) Radiologic and histologic pathology of nontumorous diseases of bones and joints. Illinois: Northbrook, 1990: 779–781.
4. Catto M. Pathology of gout. Scott Med J 1973; 18: 232–238.
5. Cope R, Marsan R, Castelli MJ. Case report 732. Skeletal Radiol 1992; 21: 269–270.
6. Foucar E, Buckwalter J, El-Khoury GY. Gout presenting as a femoral cyst. J Bone Joint Surg Am 1984; 66: 294–297.
7. Cooper A, Minutello R, Vigorita VJ, Bryke E. Case report 763. Skeletal Radiol 1992; 21: 555–558.
8. Greenberg DC. Pathological fracture of the patella secondary to gout. J Bone Joint Surg Am 1986; 6: 1286–1288.
9. Torke MD, Henderson RC. Tophaceous gout simulating infection about a stainless steel plate. J Orthop Trauma 1990; 4: 463–465.
10. Ortman BL, Pack LL. Aseptic loosening of a total hip prosthesis secondary to tophaceous gout: a case report. J Bone Joint Surg Am 1987; 69: 1096–1099.
11. Mielants H, Veys EM, DeBussere A, Van Der Jenght J. Avascular necrosis and its relation to lipid and purine metabolism. J Rheum 1975; 2: 430–436.
12. Howell DS. Preliminary observation on local pH in gouty tophi and synovial fluid. Arthritis Rheum 1965; 8: 736–740.