Chronic tophaceous gout presenting as nonhealing ulcers

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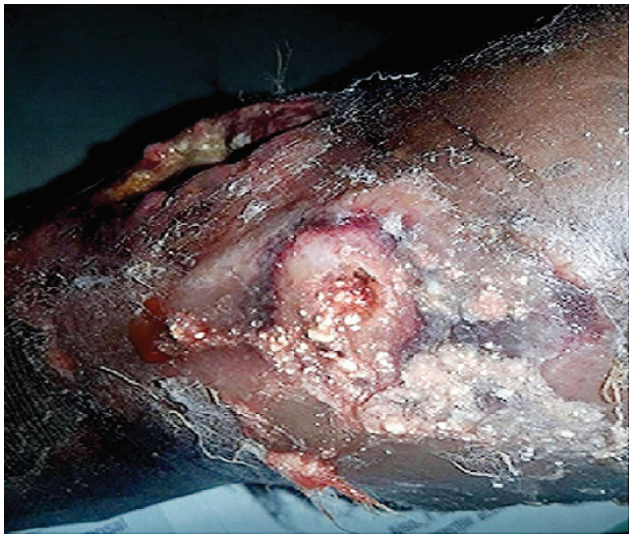
A 31-year-old male from rural India presented with multiple swellings on both feet and hands for the previous 2 years. The swellings were firm in consistency, immobile, variable in size and not attached to the skin. A biopsy had been performed that had resulted in a nonhealing ulcer at the dorsum of right foot with oozing of chalky white material from the wound (Figure 1). Biopsy confirmed it to be a tophus. The histopathology slide shows a central area of amorphous proteinaceous deposits surrounded by inflammatory aggregates and haemorrhages (Figure 2). Thereafter, the patient was referred to rheumatologist for further management. The patient had presented 1 month after the biopsy was performed. He was diagnosed with chronic tophaceous gout (CTG) with secondary infection of the ulcerated wound. His uric acid levels were 9.5 mg% (565 µmol/l) and creatinine was 1.2 mg% (106.1 µmol/l). His concern was related to the nonhealing ulcer and increasing size and number of tophi, rather than acute pain.

Figure 1 Ulcerated tophi on the dorsum of right foot



The patient was treated with allopurinol and antibiotics according to the sensitivity report of the swab from the infected wound. The ulcers healed in 3 months (Figure 3).

Most of the gouty tophi in CTG can be treated with medical therapy. Surgery is usually not indicated. Indications for surgical treatment include restriction of joint movements, compression of adjacent structures, pain and recurrent infection.[1] Surgery for gouty tophus has a high rate of complications, mainly delayed healing, especially if the tophus is infected and if the patient has numerous comorbidities.[2]

The diagnosis of gout is usually confirmed by demonstration of monosodium urate (MSU) crystals from the joint aspirate. However, diagnosis can be difficult if the MSU crystal deposition is in extra-articular tissues, such as bursae and tendon. Even with MSU deposition in typical joints, the diagnosis could be difficult in atypical presentations, such as in elderly, those with genetic predisposition and those with immunosuppression.

Secondly, joint aspiration may be difficult if there is inadequate fluid or if the joint is inaccessible. Serum urate levels could help, but are not always diagnostic, especially in acute attacks. Plain radiographs are helpful to differentiate CTG from rheumatoid arthritis. Erosions in gout are characteristically punched out with overhanging sclerotic margins and are situated away from joint margins, sometimes outside the joint capsule. Ultrasound findings

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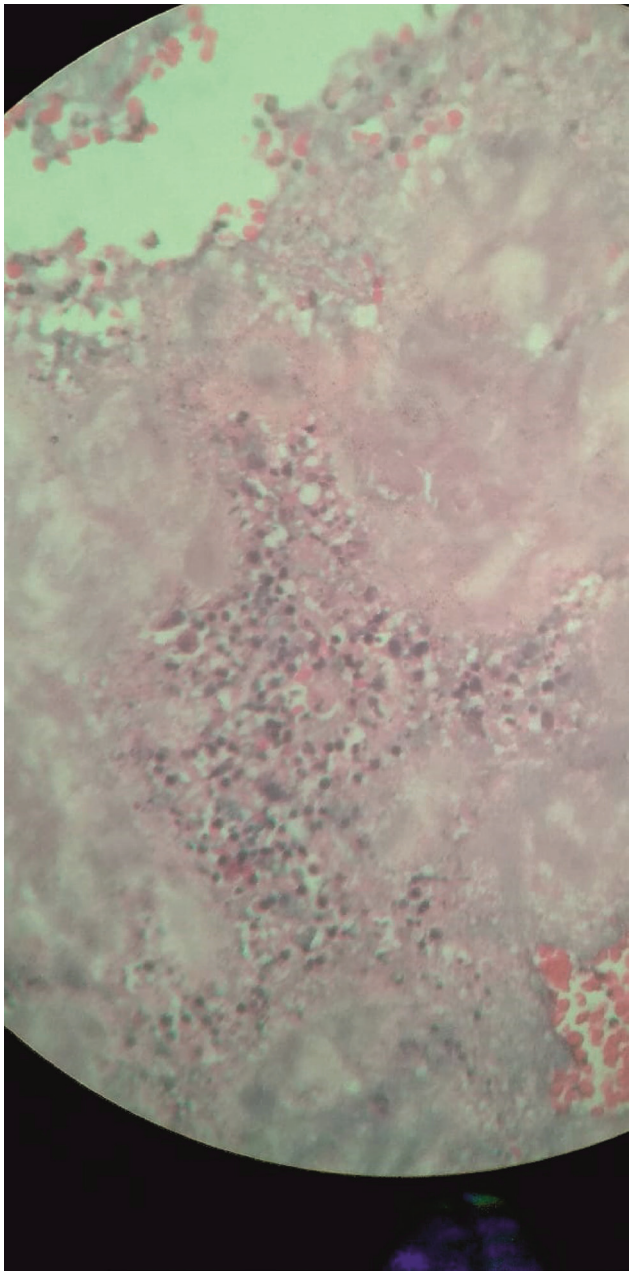
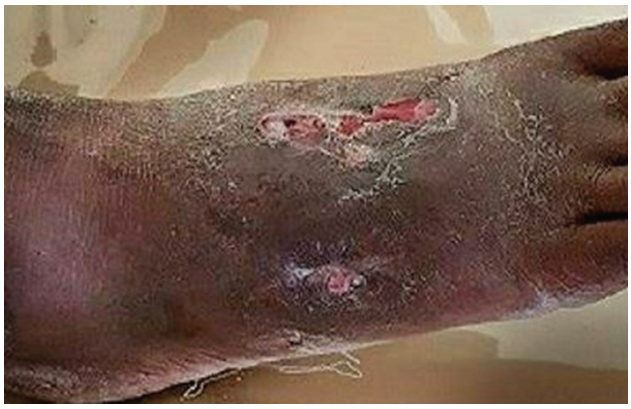
Figure 2 Hisopathology showing central amorphous material surrounded by inflammatory cells and red blood cells



in gout include joint effusion, synovitis and erosions. The characteristic hyperechoic floating aggregates of MSU crystals (microtophi) can sometimes be seen in the joint,

Figure 3 Healed ulcers after appropriate treatment



described as a 'snowstorm appearance'. Double contour appearance is another sign in ultrasonography of the gout arthropathy. This is seen as an echogenic line on the outer surface of the joint cartilage parallel to the subchondral bone, secondary to deposition of MSU crystals on the surface of hyaline articular cartilage.

Diagnosis also becomes difficult in gout mimics, periarticular tumours and in gout coexisting with other arthropathies.

In such situations, dual-energy CT (DECT) has evolved to be a useful diagnostic modality. With its high sensitivity and specificity, DECT has shown to be a valuable tool in the noninvasive diagnosis of gout with many potential clinical applications.[3]

CTG is treated with urate-lowering therapy, allopurinol being the first-line treatment in patients with normal kidney function. In cases of intolerance to allopurinol or if the target serum uric acid is not achieved with maximum dosages adjusted to creatinine clearance, patient is switched to febuxostat or a uricosuric drug (benzbromarone or probenecid) is added to allopurinol.[4] In patients with crystal-proven severe debilitating CTG and with a poor quality of life, in whom the target serum uric cannot be reached, pegloticase is indicated.[4] In contrast to the previous guidelines, current guidelines on management of CTG also include addressing issues such as obesity, cardiovascular disease, kidney failure and mortality.[4]

Acknowledgement

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