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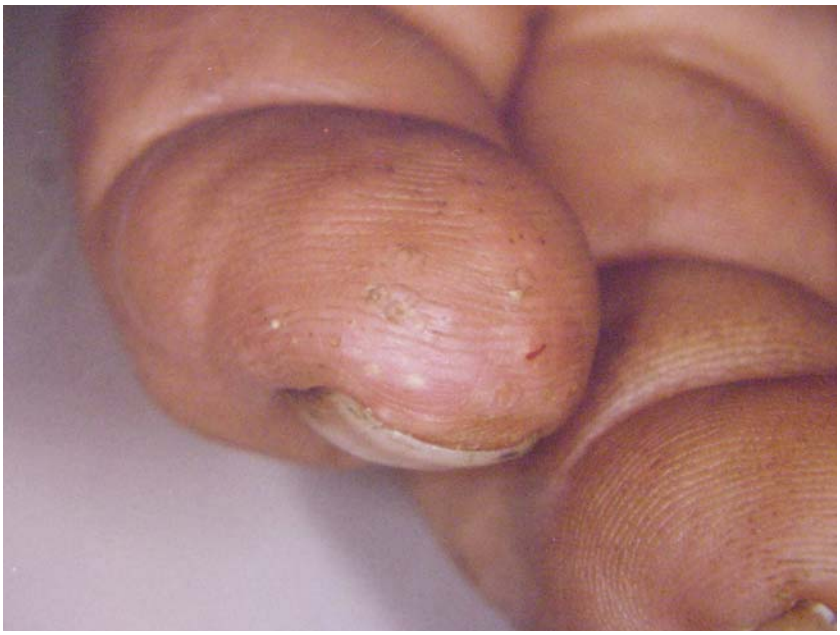

A 49-year-old obese male mechanic presents with slightly tender papules on the first 3 fingers of each hand. This eruption has been present for a few years and was treated with high-potency topical steroids without relief. The patient also has a history of gout, hypertension, chronic proteinuria, and severe coronary artery disease, for which he takes atorvastatin, isosorbide, and metoprolol.

What is your diagnosis?

PLEASE TURN TO PAGE 205 FOR DISCUSSION

The Diagnosis: Intradermal Tophaceous Gout

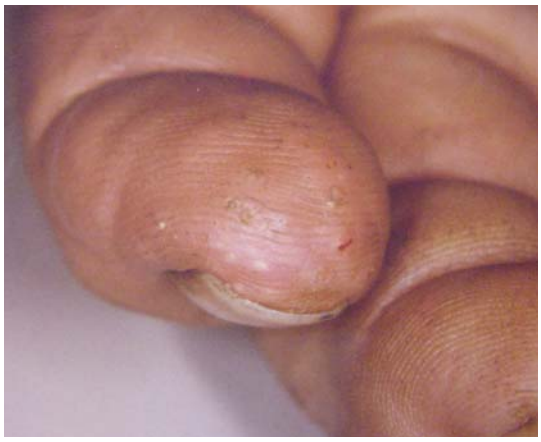

Figure 1. Small tophi on the finger pads.

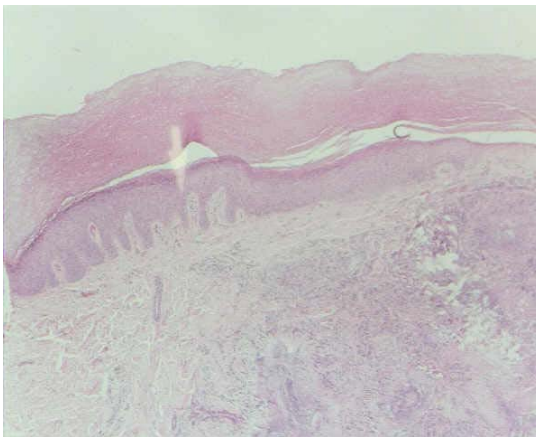

Figure 2. Amorphous urate deposits in the dermis (H&E, original magnification ×20).

Gout is the common endpoint of a group of metabolic disorders that result in hyperuricemia. Gout is characterized by transient attacks of painful acute arthritis caused by crystallization of urate in and around articular joint spaces, most commonly the first metatarsophalangeal joint. Prolonged hyperuricemia may eventually lead to chronic gouty arthritis, as well as deposition of urate crystals in other sites creating tophi. Gouty tophi are nodular collections of monosodium urate crystals that can appear as firm, nontender, yellowish-white or salmon-pink papules that mimic pustules or subcutaneous nodules that are sharply circumscribed from surrounding tissues (Figure 1).[1,2]

Gout is a disease that primarily affects middle-aged or older males who are obese and have a family history of gout. When it does appear in women, it is usually after menopause.[3] The prevalence of gout increases with age, is higher in black patients than in white patients, and is extraordinarily high in Filipinos and South Pacific Islanders.[4] Risk factors associated with gout include alcohol use, lead exposure, diuretic use, long-term glucocorticoid use, hypertension, hyperlipidemia, and impaired renal function.[1,4,5]

The joint involved in an acute gout attack is usually inflamed, warm, swollen, red, and so tender that the affected limb is unusable.[3] If left untreated, the symptoms may persist for days to weeks but will gradually subside. Chronic intradermal tophaceous gout lacks the marked inflammation associated with acute gout in the joints.[4] This condition is characterized by aggregates of crystal in the connective tissue (tophi) that appear most commonly on the helix of the ear, olecranon bursa of the elbow, and digits of the hands and feet.[3] In rare cases, tophi may be the initial manifestation of gout. However, this presentation tends to occur in older women with decreased renal function who are taking diuretics and generally affects the fingers rather than the feet.[6] Eight previous cases of finger pad tophi as a presenting lesion of gout have been reported in the literature.[2]

The pathogenesis of gout begins with hyperuricemia. Uric acid is produced by purine metabolism and excreted by the kidneys. A plasma uric acid level above 7 mg/dL is considered elevated because it exceeds the saturation value for blood pH and urate at normal body temperature. The hyperuricemia of gout results from a heterogeneous group of biochemical and physiologic abnormalities. Overproduction of urate, diminished renal excretion, or a combination of both can cause hyperuricemia. Secondary hyperuricemia may be caused by renal failure; medications such as thiazide and loop diuretics that interfere with urate excre-

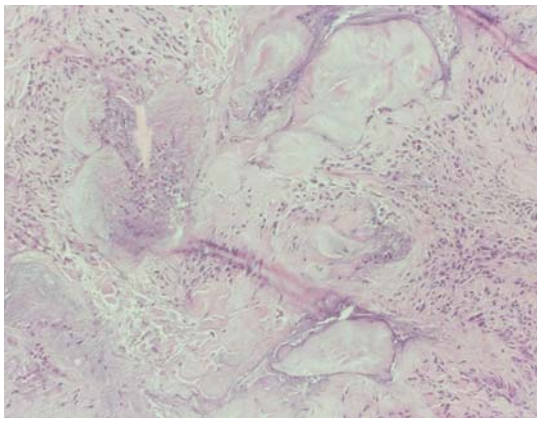

Figure 3. Giant cells surround urate deposits (H&E, original magnification ×20).

tion; myeloproliferative disorders; or diseases with high tissue turnover, such as psoriasis. Measuring 24-hour urine urate excretion may differentiate the underlying cause of hyperuricemia.[4] Not all patients with hyperuricemia develop gout, but virtually all gouty patients have a serum urate level above 7 mg/dL.[3]

Deposits of uric acid crystals in the joints stimulate an inflammatory reaction via production of chemotactic factors by monocytes, leading to an accumulation of macrophages. Phagocytosis of crystals by these neutrophils triggers cell damage, induces release of lysosomes, and thereby heightens the inflammation. This process leads to further deposition of urate crystals, and the cycle continues.[4] The histologic changes related to urate deposition are essentially that of a foreign body reaction (Figures 2 and 3).[7] In formalin-fixed tissue with hematoxylin and eosin stain, urate deposits appear as amorphous, amphophilic feathery material with stellate empty spaces surrounded by multinucleated giant cells.[8] The yellow needle-shaped crystals with negative birefringence are more visible with alcohol fixation and a polarized light microscope.[4]

The clinical features of an acute attack and appropriate response to colchicine therapy within 1 to 2 days are sufficient for the presumptive diagnosis of gout.[3] This diagnosis may be supported by evidence of hyperuricemia, leukocytosis, or an elevated erythrocyte sedimentation rate.[4] The diagnosis of gout is definitively confirmed when light microscopy demonstrates urate crystals in fluid from synovial effusion or material in a subcutaneous tophus.

The differential diagnosis of an acute gout attack should include bacterial cellulitis or septic arthritis. Gouty tophi must be distinguished from osteoarthritis and chondrocalcinosis (pseudogout), which may exhibit deposition of calcium pyrophosphate crystals.[3]

Treatment of acute episodes includes colchicine to inhibit neutrophil phagocytosis of urate crystals, indomethacin and other nonsteroidal anti-inflammatory drugs to relieve pain and inflammation. Occasionally, systemic corticosteroid therapy or narcotic analgesics are used when colchicine and nonsteroidal anti-inflammatory drugs are not effective. Long-term control of gout is achieved through lifestyle modifications and/or chronic medication to maintain normal levels of serum urate. Weight control, decreased alcohol intake, and a high fluid/low purine diet is advised for all gout patients.[4] Probenecid is a uricosuric that is used to decrease reabsorbtion of urate in underexcreters of uric acid. Allopurinol is a xanthine oxidase inhibitor that decreases uric acid production in urate overproducers. Tophi can be expected to clear within 6 to 12 months after normalization of serum uric acid.[9]

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