

Gout

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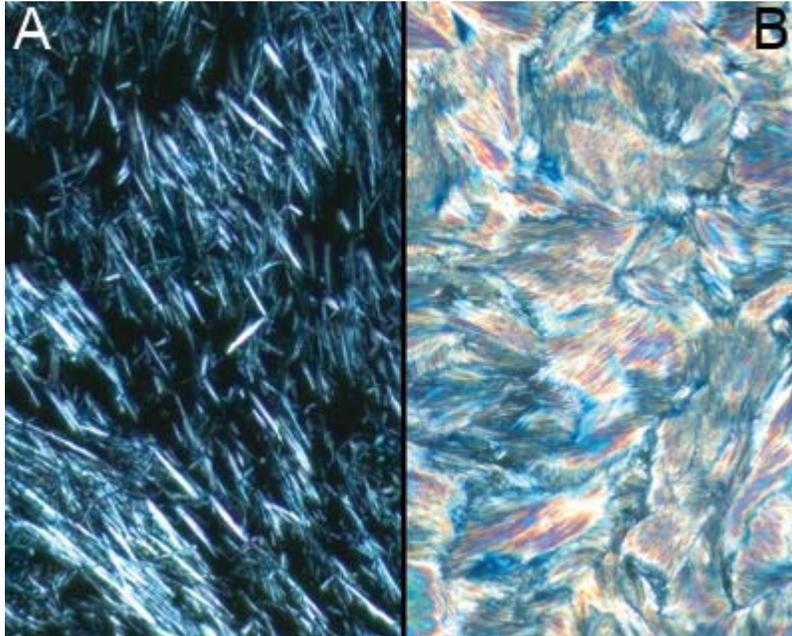


Figure 1. A: Urate is seen as strongly negative, birefringent, needle-shaped crystals under polarized light. B: Sheaves of elongated monosodium urate crystals are visible in alcohol fixed and stained with methylene blue.

Gout is caused by disordered purine metabolism resulting in hyperuricemia. Symptoms are related to the precipitation of monosodium urate (uric acid) crystals, typically in joint spaces or soft tissue. Primary gout is caused by an increase in uric acid production, while secondary gout is caused by either a decrease in urinary uric acid excretion or an overproduction of purine secondary to increased cell turnover (e.g., tumor lysis). Predisposing clinical factors include older age (fifth and older decade), male sex, obesity, heavy alcohol ingestion, a purine-rich diet, certain medications (e.g., thiazide diuretics), and genetic factors.

Patients with gout may present with episodes of acute arthritis that are initiated by the crystallization of urate within acutely inflamed synovial tissue. The first toe is the most commonly involved joint (gouty pain in the great toe is called *podagra*). Chronic gout leads to long-term crystal deposition, usually in cooler body sites around joints and cartilage; the accumulation of these crystals results in pathognomonic tophi, or chalky deposits.

In the head and neck region, gout may involve the auricle, larynx and, infrequently, the temporomandibular joint. Gouty tophi involving the external ear may occur in the helix and antihelix, presenting as firm nodules that may ulcerate. When the cricoarytenoid joint, vocal fold, or infraglottis is involved, gouty tophi can present as an exophytic papillary lesion and mimic carcinoma. They can also present as small, grainy mucosal lesions. Cricoarytenoid joint involvement can lead to hoarseness, pain, dysphagia, and vocal fold fixation. When the larynx is involved, patients generally have severe multifocal disease.

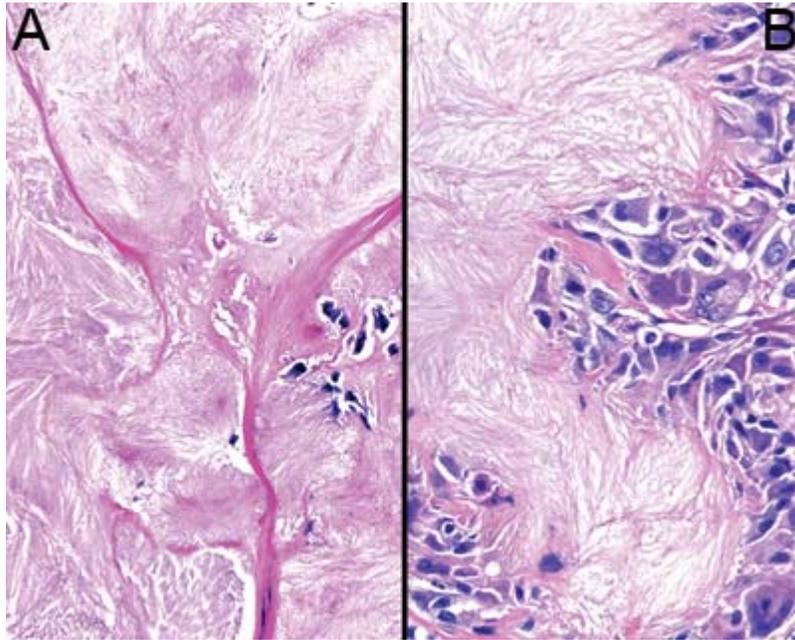


Figure 2: A: Amorphous fibrillary crystalline tissue deposits of tophaceous gout in formalin-fixed tissue represent the proteinaceous matrix that surrounds dissolved crystals (H&E). B: A granulomatous reaction palisades around a gouty tophus (H&E).

On gross pathologic examination, tophaceous gout deposits appear as yellow-white chalky material. Urate within aspirated or scraped material is seen as strongly negative, birefringent, needle-shaped crystals under polarized light (figure 1, A) or alcohol-fixed, methylene-blue-stained (figure 1, B). In pseudogout (deposition of calcium pyrophosphate), the crystals are rhomboid. Histologically, gouty tophi appear as amorphous amphophilic material similar to amyloid deposits (figure 2, A). The crystals within these tissue deposits are difficult to visualize with routine fixation, tissue processing, and staining because they are dissolved by aqueous solutions during formalin processing. Therefore, tissue submitted to the pathology laboratory for the identification of uric acid crystals should include a scrape (smear) onto an unfixed slide and/or alcohol-fixed material (figure 1) in addition to a formalin-fixed specimen. Often the urate crystals are surrounded by granulomatous inflammation with foreign-body giant cells (figure 2, B) and a lymphoplasmacytic infiltrate. Such a reaction can mimic a rheumatoid nodule. Large tophi may undergo ossification.

Renal failure is responsible for death in up to 20% of patients with gout. Treatment for acute gout includes colchicine and nonsteroidal anti-inflammatory drugs. Avoidance of alcohol and purine-rich foods, combined with allopurinol or probenecid pharmacotherapy, can help in managing chronic hyperuricemia.

Suggested reading

Adair C. Non-neoplastic lesions of the ear and temporal bone. In: Thompson LDR, ed. Head and

Neck Pathology. Philadelphia: Elsevier Health Sciences; 2006:371-96.

Guttenplan MD, Hendrix RA, Townsend MJ, Balsara G. Laryngeal manifestations of gout. Ann Otol Rhinol Laryngol 1991;100(11): 899-902.

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