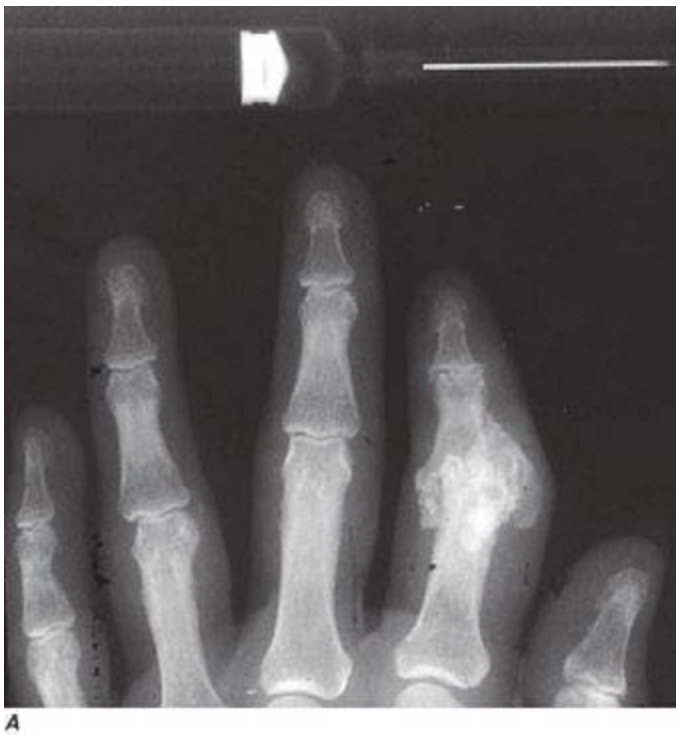
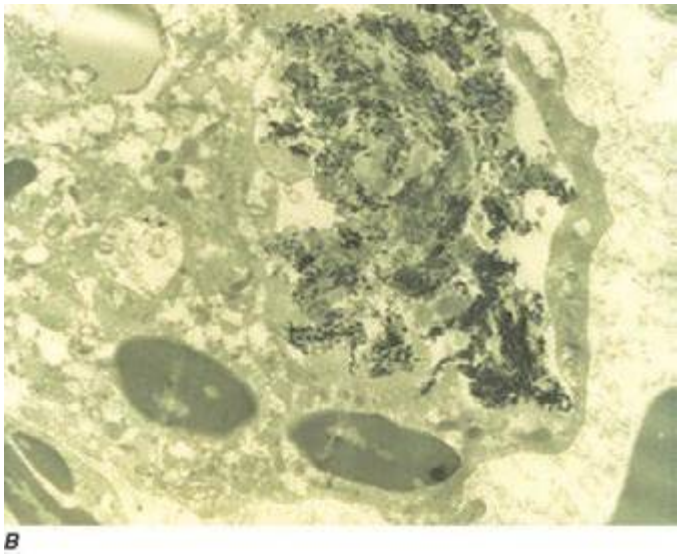


A



B

FIGURE 395-3 ***A.*** Radiograph showing calcification due to apatite crystals surrounding an eroded joint. ***B.*** An electron micrograph demonstrates dark needle-shaped apatite crystals within a vacuole of a synovial fluid mononuclear cell (30,000×).

intensity of symptoms. Local injection of disodium ethylenediaminetetraacetic acid (EDTA) and SC anakinra have been suggested as effective in single studies of acute calcific tendinitis at the shoulder. Other reports have described that IV gamma globulin, rituximab, calcium channel blockers, or bisphosphonates may help diffuse calcinosis. Periarticular apatite deposits may be resorbed with resolution of attacks. Agents to lower serum phosphate levels may lead to resorption of deposits in renal failure patients receiving hemodialysis. In patients with underlying severe destructive articular changes, response to medical therapy is usually less rewarding.

CAOX DEPOSITION DISEASE

PATHOGENESIS

Primary oxalosis is a rare hereditary metabolic disorder (Chap. 434e). Enhanced production of oxalic acid may result from at least two different enzyme defects, leading to hyperoxalemia and deposition of CaOx crystals in tissues. Nephrocalcinosis and renal failure are typical results. Acute and/or chronic CaOx arthritis, periarthritis, and bone disease may complicate primary oxalosis during later years of illness.

Secondary oxalosis is more common than the primary disorder. In chronic renal disease, calcium oxalate deposits have long been recognized in visceral organs, blood vessels, bones, and cartilage and are now known to be one of the causes of arthritis in chronic renal failure. Thus far, reported patients have been dependent on long-term hemodialysis or peritoneal dialysis (Chap. 336), and many had received ascorbic acid supplements. Ascorbic acid is metabolized to oxalate, which is inadequately cleared in uremia and by dialysis. Such supplements and foods high in oxalate content usually are avoided in dialysis programs because of the risk of enhancing hyperoxalosis and its sequelae.

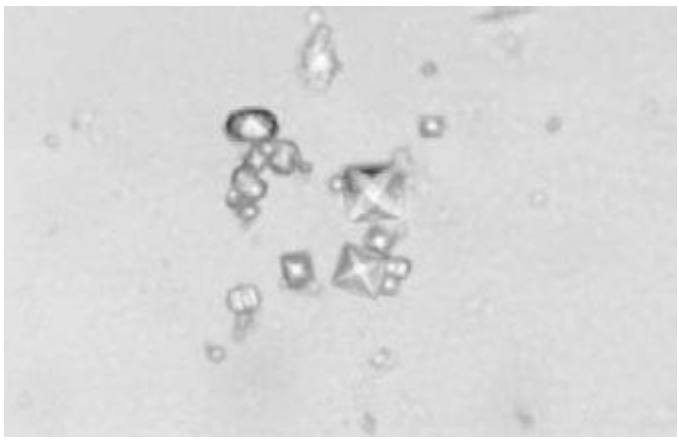


FIGURE 395-4 **Bipyramidal and small polymorphic calcium oxalate crystals** from synovial fluid are a classic finding in calcium oxalate arthropathy (ordinary light microscopy; 400×).

CLINICAL MANIFESTATIONS AND DIAGNOSIS

CaOx aggregates can be found in bone, articular cartilage, synovium, and periarticular tissues. From these sites, crystals may be shed, causing acute synovitis. Persistent aggregates of CaOx can, like apatite and CPP, stimulate synovial cell proliferation and enzyme release, resulting in progressive articular destruction. Deposits have been documented in fingers, wrists, elbows, knees, ankles, and feet.

Clinical features of acute CaOx arthritis may not be distinguishable from those due to urate, CPP, or apatite. Radiographs may reveal chondrocalcinosis or soft tissue calcifications. CaOx-induced synovial effusions are usually noninflammatory, with <2000 leukocytes/μL, or mildly inflammatory. Neutrophils or mononuclear cells can predominate. CaOx crystals have a variable shape and variable birefringence to polarized light. The most easily recognized forms are bipyramidal, have strong birefringence (Fig. 395-4), and stain with alizarin red S.

TREATMENT CALCIUM OXALATE DEPOSITION DISEASE

Treatment of CaOx arthropathy with NSAIDs, colchicine, intra-articular glucocorticoids, and/or an increased frequency of dialysis has produced only slight improvement. In primary oxalosis, liver transplantation has induced a significant reduction in crystal deposits (Chap. 434e).

Acknowledgment

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