BRIEF REPORT

ACUTE TEMPOROMANDIBULAR ARTHRITIS IN A PATIENT WITH BRUXISM AND CALCIUM PYROPHOSPHATE DEPOSITION DISEASE

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The temporomandibular joint has been regarded as "gout-proof" (1). Calcium pyrophosphate deposition disease (CPDD) is also unusual at this site. There has been a report of a painless destructive lesion tantamount to a calcium pyrophosphate (CaPPi) tophus involving this joint; it occurred without history of acute attacks in the area (2). Involvement of the temporomandibular joint is not mentioned in standard textbook discussions of CPDD. Several authors who reported large series of cases of CPDD, listing involved joints, failed to note attacks involving this joint (3–7). Similarly, in the unpublished experience of our clinic, which has had 76 cases of CPDD, involvement of this joint was not apparent except for the 1 patient reported here, a man with bruxism.

Case Report. A 56-year-old man was referred to our arthritis clinic because of pain and swelling at the left temporomandibular joint of 3 weeks duration and at the right wrist of 1 week duration. Two years previously, an attack of pain and swelling occurred in 1 foot, preventing walking for a week before it resolved completely. The patient's wife of 30 years reported that he had had loud attacks of nocturnal bruxism all of their married life. In spite of this, he had been free from headache or pain in the temporomandibular joints until the current attack.

Physical examination showed tender, circumscribed swelling (Figure 1) and redness and increased heat at the left temporomandibular joint. The patient had marked pain when he attempted to open his jaw. Because of these symptoms, his mandibular functional movements were limited. The right wrist was tender, swollen, and warm (Figure 2). One-half milliliter of turbid fluid aspirated from a fluctuant area near the right radial styloid was sterile to culture. Compensated polarized light microscopy revealed numerous leukocytes as well as intracellular and extracellular weakly positive birefringent crystals, rhomboid and rod shaped. Levels of serum calcium, phosphorus, alkaline phosphatase, uric acid, and iron were normal, and results of assays for total iron binding capacity and rheumatoid factor were negative. Chondrocalcinosis was noted in x-ray films of the knees and wrists. Roentgenograms of the temporomandibular joints, including laminograms, showed severe degenerative changes bilaterally without soft tissue calcification (Figures 3 and 4).

After the crystals were identified in the wrist aspirate, a tentative diagnosis of CPDD was made for the arthritis of the temporomandibular joint as well as the inflamed wrist. Indomethacin, 25 mg 4 times daily, was started and resulted in relief of pain within a few hours. After 2 days, the patient was free from objective changes, motion pain, and tenderness at both previously involved joints. Aspiration of the joint had been planned to exclude septic arthritis, but it was cancelled when the inflammation rapidly resolved.

A month later, a dental consultant noted normal, painless range of motion at both temporoman-

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Submitted for publication May 22, 1981; accepted in revised form September 10, 1981.

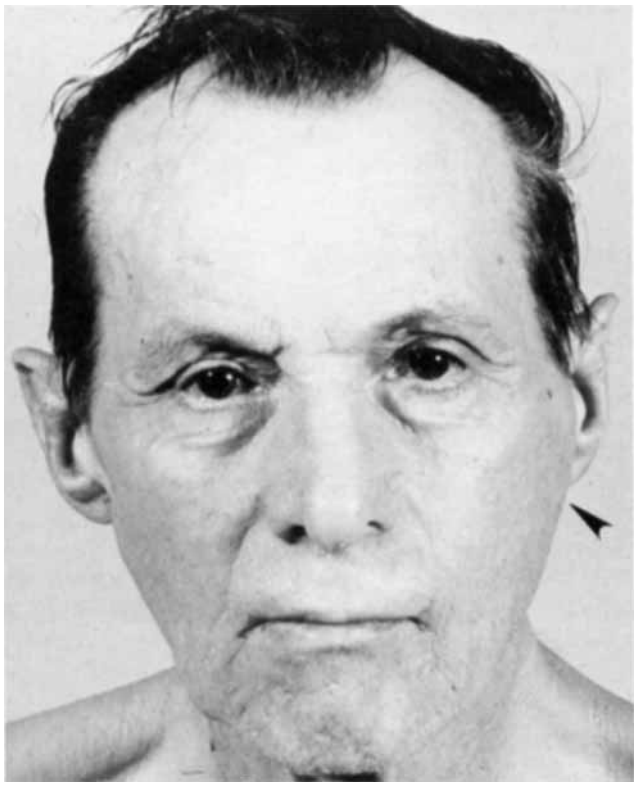

Figure 1. Swelling overlying the left temporomandibular joint, seen as loss of normal concavity below the zygoma (**arrowhead**).

dibular joints despite the severe bilateral degenerative changes noted by radiology.

Discussion. The diagnosis of CaPPi arthropathy in the temporomandibular joint in this case is presumptive, though highly probable, since involvement cleared promptly in that and another joint from which CaPPi crystals had been recovered. In 2 years of followup, the patient has remained free of symptoms in the temporomandibular joints even though the bruxism continues. His severe bilateral osseous changes in those joints are typical for degenerative joint disease of the temporomandibular joints and are not uncommon with bruxism alone. Degenerative radiologic changes include reduction of joint space, erosions, flattening of the condylar head, and sclerosis. Over 40% of individuals with these findings are asymptomatic (8).

Pritzker and coworkers reviewed the apparent exclusion of the temporomandibular joint in CaPPi athropathy, pointing out the absence of hyaline cartilage in the adult joints, unique amoung diarthrodial joints of the body; they postulated either CaPPi crystal

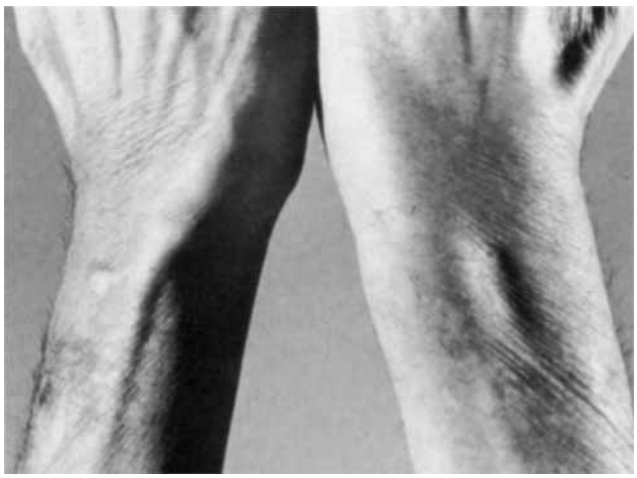

Figure 2. On the right, the swollen wrist contrasts with normal left wrist. (The apparent tendon sheath swelling on the left is a dilated vein.)

deposition de novo in synovium or origin of pyrophosphate (PPi) in contiguous bone in their patient with such joint involvement (2). Pritzker's patient was endentulous and no history of bruxism was noted. It is possible that bruxism served as a cofactor in our patient, however, because severe degenerative bony changes might augment local production of crystal. Indeed, Howell and coworkers have suggested that synovial PPi may be derived from the subchondral bone plate, perhaps entering through subchondral vessels, providing a reservoir for PPi at rest, during remodeling, or during mechanical abrasion from bone

Figure 3. Flattening of condylar head with anterior lipping, increased sclerosis of articular surfaces, and reduction of articular space are demonstrated in laminogram of the right temporomandibular joint.

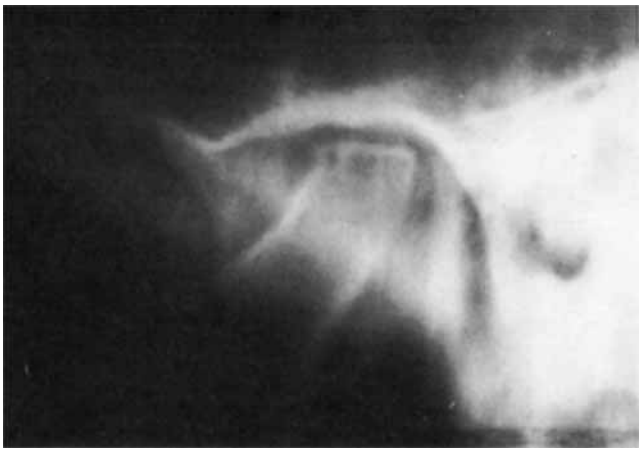

Figure 4. Flattened or "nailhead" condyle with erosion of anterior superior aspect, and some demineralization of the articular eminence is noted in the patient's left temporomandibular joint.

on bone contact (9). Noting that laminograms of the temporomandibular joints in our case did not demonstrate chondrocalcinosis, we doubt that the purported phlogistic CaPPi crystals originated from the relatively acellular fibrocartilaginous disc of the temporomandibular joint.

Pain and headache, but not swelling, associated with these joints may be seen in many conditions, such as chronic bruxism, osteoarthritis, meniscus perforation or displacement, and myofascial pain dysfunction syndrome. Rheumatologists commonly encounter involvement of the temporomandibular joint in ankylosing spondylitis and rheumatoid arthritis, but swelling of the joint is not seen (10,11). Discrete signs of inflammation have been virtually limited to infective arthritis. An older report of several patients with purported gout in this joint is doubtful, since none of the patients had a history of swelling of this or any other joint (12). Recent textbooks, however, include 2 patients with swelling of the joint and aspiration that yielded urate crystals (13,14).

The case presented is another illustration that CPDD, termed the great imitator (15), can closely simulate septic arthritis. Thus it appears that, confronted with redness or swelling at the temporomandibular joints, the clinician should consider unusual presentations of gout or CPDD as well as infective disease.

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