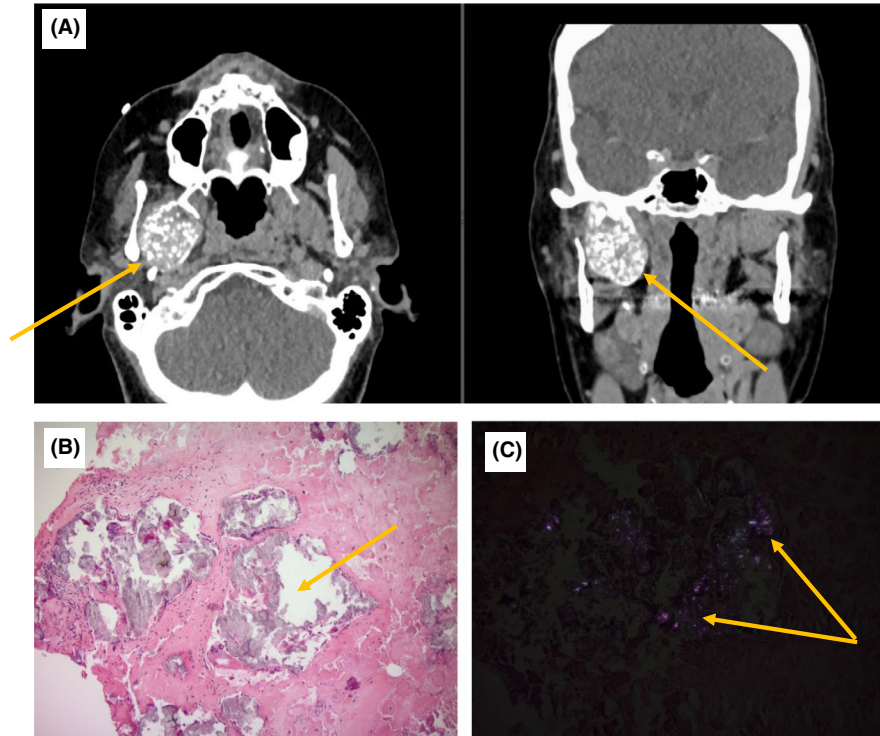


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Clinical Image: Pseudogout of the temporomandibular joint



Three years prior to the rheumatology consultation, the patient, a 58-year-old woman, developed jaw pain. She was referred to an otolaryngologist, who noted pain with opening of the jaw at the right temporomandibular joint (TMJ) with decreased oral aperture. A subsequent computed tomography scan demonstrated a 3×5×5 cm mass (denoted by arrows), with epicenter in the right masticator space extending to the TMJ and medially displacing the parapharyngeal space (A). Biopsy of the right masticator space mass demonstrated positively birefringent rhomboid-shaped crystals (arrows) consistent with a diagnosis of calcium pyrophosphate dihydrate deposition disease (CPPD) (B and C). She underwent a partial resection of the mass, but full resection was not possible because of its proximity to the internal carotid artery.

She was referred to rheumatology for further evaluation and management. Inflammatory markers were normal. Laboratory evaluation for secondary causes of CPPD was negative. Radiographs of the bilateral hands and knees demonstrated no evidence of chondrocalcinosis. She was treated with oral corticosteroids, colchicine, and intralesional corticosteroid injections without response; ultimately, nonsteroidal anti-inflammatory drugs (NSAIDs) were used for pain. After 2 years of follow-up, she continues to have no articular manifestations of CPPD beyond the right TMJ, and repeat imaging confirmed the stability of the mass.

The TMJ is an unusual location to develop pseudogout or CPPD, but cases have been reported in the literature (1–3), and thus awareness of this entity is important for timely and accurate assessment. Benign and malignant lesions arising from the TMJ can have overlapping clinical features, and therefore imaging and pathologic evaluation often play a critical role in diagnosis (4). Although imaging can be suggestive of non-neoplastic etiologies, in some cases, features are not specific, and tissue diagnosis is needed to exclude malignancy. Radiographically, tophaceous pseudogout can appear similarly to many neoplasms, including osteochondroma, chondroblastoma, and chondrosarcoma, and can demonstrate a bony mass with or without osseous destruction, making differentiation from more ominous lesions such as chondrosarcoma difficult (4). Benign conditions such as synovial chondromatosis, tumoral calcinosis, and pigmented villonodular synovitis can also affect the TMJ but usually demonstrate more specific imaging findings such as joint space narrowing, erosions, and intraarticular loose bodies (3–5). Intraoperatively, tophaceous pseudogout shows noncohesive, “grit-like” chalky material deposited within the synovium of the joint capsule; in contrast, neoplastic lesions typically show hard, cohesive masses that originate from or adhere to joint-articulating surfaces or the meniscus (4). Ultimately, the identification of characteristic birefringence of CPPD crystals is critical to differentiate the lesion from other potential mimickers. The etiology of CPPD is often idiopathic but can be hereditary or associated with

trauma, surgery, or metabolic or endocrine disorders (6). Surgical excision and intraarticular glucocorticoids are the primary therapy; however, systemic agents, including systemic steroids, colchicine, and NSAIDs, have been used (2,3,5). By recognizing this rare manifestation of CPPD, appropriate evaluation and management can be promptly pursued.

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