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Tumoral Calcinosis of the Temporomandibular Joint: CT and MR Findings

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Summary: In this article, the CT, three-dimensional CT, and MR findings are reviewed in a 59-year-old woman with tumoral calcinosis involving the temporomandibular joint.

Index term: Temporomandibular joint, neoplasms

Tumoral calcinosis is an uncommon disease characterized by periarticular soft tissue hyperplasia and calcification. Large joints such as the hip, shoulder, and elbow most frequently are involved. This entity most commonly presents in the first 2 decades of life. A familial predisposition has been reported. The cause, however, remains unknown. We present an unusual case of tumoral calcinosis that involves the temporomandibular joint in a woman experiencing intermittent temporomandibular joint pain.

Case Report

A 59-year-old white woman initially presented to her oral surgeon for removal of a tooth. A panorex revealed a large left temporomandibular joint lesion. She was referred to the otolaryngology service for further evaluation of her lesion.

The patient had no complaints, but on questioning admitted to mild intermittent pain in the left temporomandibular joint. There was no history of trauma to the head and neck. Medical history was significant for hypertension and systemic lupus erythematosus, which were well controlled with hydroxychloroquine and prednisone. Her family history was unremarkable. Physical examination revealed minimal fullness of the left temporomandibular joint. Mobility and range of motion of the jaw were normal. All laboratory values, including a complete blood count, serum electrolytes, calcium, phosphorous, alkaline phosphatase, blood urea nitrogen, creatinine, and sedimentation rate were within normal limits.

Noncontrast computed tomography (CT) (Fig 1A and B), contrast CT, and three-dimensional CT (Fig 1C and D) demonstrated an approximately 2-cm round dense mass

involving the left temporomandibular joint space. The mass consisted of a circumferential cluster of densely calcified aggregates that surrounded the mandibular condyle and expanded the joint space. Remodeling of bone was noted around the lesion, although no bony destruction was evident. There was no evidence of abnormal enhancement following contrast administration. The right temporomandibular joint was normal in appearance.

Magnetic resonance (MR) imaging was performed with a 1.5-T superconductive magnet (GE Signa). Noncontrast T1-weighted (550/30/2 [repetition time/echo time/excitations]) axial sequences (Fig 1E) and contrast-enhanced T1-weighted (550/20/2) coronal sequences (Fig 1F) were obtained. These images demonstrated a nonenhancing low signal intensity lesion within the left temporomandibular joint consistent with a calcified mass. The lesion also demonstrated predominantly low signal intensity on T2-weighted (2200/80/1) axial images (Fig 1G). A joint effusion was not present. No intracranial extension was identified.

Surgical biopsy and debulking of the lesion were performed through a preauricular approach. Grossly, a large gritty mass with calcifications was identified in the temporomandibular joint space.

Histologic evaluation revealed a uniform pattern of dense, relatively avascular, collagenous stroma sprinkled irregularly throughout, with small and frequently coalescing calcified particles similar to those noted in dystrophic calcification. There was no true metaplasia into bone or cartilage, but the larger calcified masses appeared angular, albeit amorphous. Frequently the more structured calcified masses were encased within cystic spaces, presumably created by the artifactual pulling away of surrounding soft tissues or shrinkage of the calcified masses during processing (Fig 1H). Occasional chronic inflammatory cells were noted within the fibrous stroma. A few epithelioid and multinucleated giant cells were found at one site, a site also demonstrating increased vascularity (Fig 1I). Stromal cells were almost exclusively composed of mature fibrocytes. Only a few small foci of stromal edema and necrosis were present. Osteophytes were not present, nor was synovium. Although the tumor was well demarcated from surrounding soft tissues, a true capsule was lacking.

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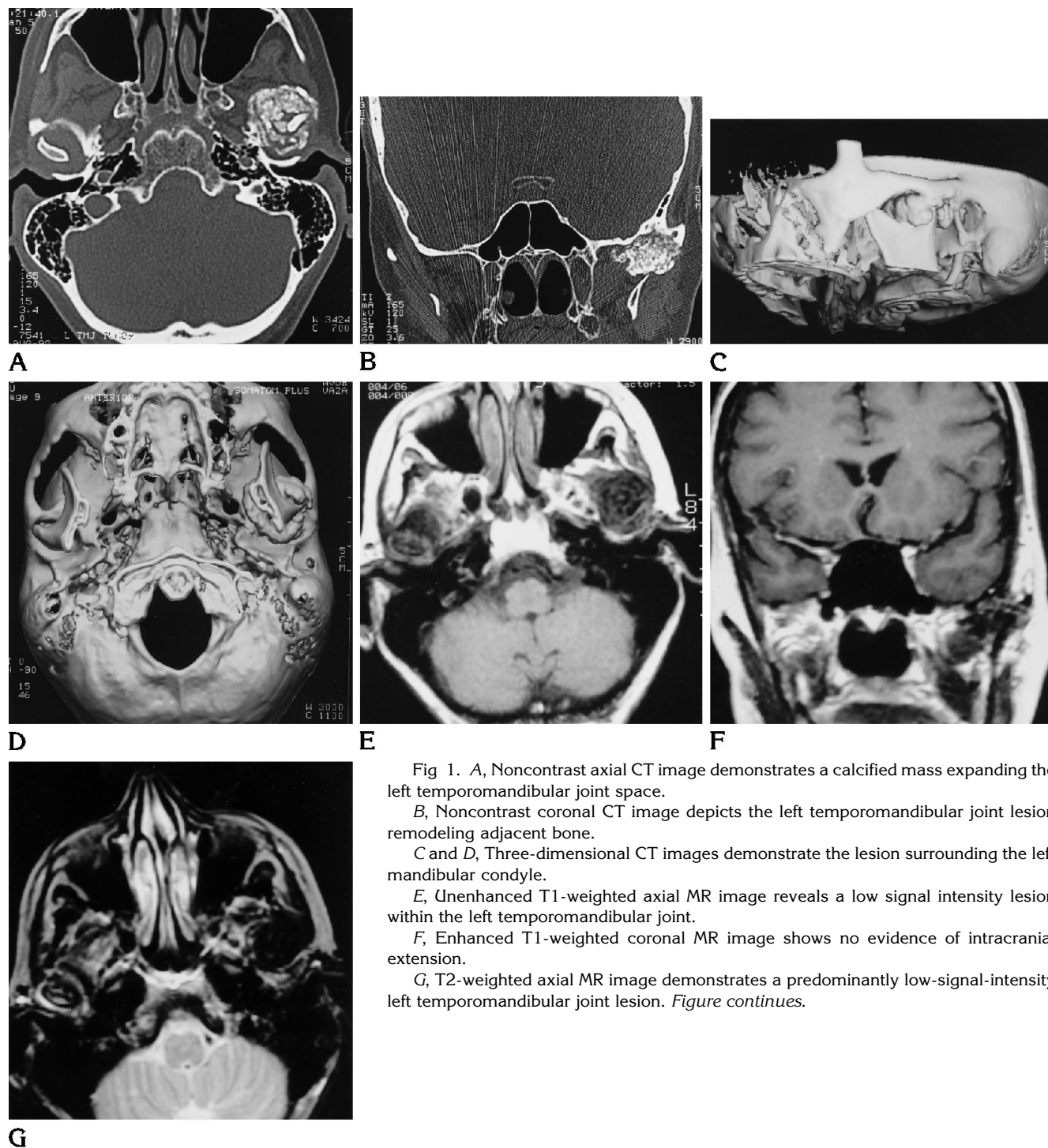


Fig 1. A, Noncontrast axial CT image demonstrates a calcified mass expanding the left temporomandibular joint space.

B, Noncontrast coronal CT image depicts the left temporomandibular joint lesion remodeling adjacent bone.

C and D, Three-dimensional CT images demonstrate the lesion surrounding the left mandibular condyle.

E, Unenhanced T1-weighted axial MR image reveals a low signal intensity lesion within the left temporomandibular joint.

F, Enhanced T1-weighted coronal MR image shows no evidence of intracranial extension.

G, T2-weighted axial MR image demonstrates a predominantly low-signal-intensity left temporomandibular joint lesion. *Figure continues.*

No part of the mass was seen to make direct contact with the underlying bone. Although the specimen lacked bone and cartilagenous metaplasia and contained few giant cells, the constellation of histologic findings are consistent with tumoral calcinosis.

Follow-up CT and MR examination 8 months after diagnosis demonstrated recurrence of the temporomandibular joint lesion.

Discussion

Tumoral calcinosis is a rare disease entity of unknown cause. Approximately 200 cases have been reported since Duret's first description in 1899 (1, 2). Diagnostic criteria for tumoral calcinosis includes the presence of large calcified

Fig 1.—continued. *H*, The lesion displays various calcified bodies, including semilunar, globular and stellate, many with pseudocystic spaces surrounding them. The stroma is extremely dense and avascular.

I, Multinucleated giant cells are present in a relatively vascular site.



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juxtaarticular masses. Classically, large joints are involved. The most commonly affected sites (in order of decreasing frequency) are the hip, elbow, shoulder, scapula, foot, leg, knee, and hand (3). Four head and neck cases have been reported, and one of these was associated with the temporomandibular joint (4–7). The latter case and our patient are among the few cases reported in elderly persons. This disease occurs predominantly in persons under 20 years of age and primarily in black patients (2, 6). There is no gender predilection. Autosomal recessive inheritance occasionally has been associated with tumoral calcinosis, but most cases are spontaneous and sporadic, as in the present example (2). Although the lesions usually are painless, they can produce symptoms resulting from their bulky size and location. The treatment of choice is wide surgical excision, although recurrence is not uncommon (7, 8).

This case of tumoral calcinosis is unusual because of its location and the age and race of the patient. We could find only one other documented case of tumoral calcinosis around the mandibular condyle, which occurred in a 66-year-old Japanese man (7). Based on the CT and MR findings, the differential diagnosis also includes joint space disease related to synovial chondromatosis, neuropathic joint disease, chronic renal failure, calcium pyrophosphate deposition disease, and primary tumors of bone or cartilage.

Synovial chondromatosis, like tumoral calcinosis, more commonly affects larger joints such as the knee, elbow, or hip (9). Approximately 40 cases have been described in the temporomandibular joint, with more frequent involvement of the right temporomandibular joint (10). The disease is monoarticular and of unknown cause. It usually affects young to middle-aged patients, with a slightly increased incidence in female subjects. The clinical presentation of

synovial chondromatosis differs from tumoral calcinosis in that patients with synovial chondromatosis complain of progressive preauricular swelling or pain (9, 10). Typically in synovial chondromatosis, a large joint effusion is present, whereas joint fluid is unusual in tumoral calcinosis (9).

In synovial chondromatosis, synovial metaplasia develops with foci of highly cellular cartilage. These foci can detach from the synovium to become intraarticular loose bodies that can calcify or ossify (9, 11). CT examination may demonstrate multiple intraarticular densities if the particles have sufficiently calcified, although the absence of loose bodies radiographically does not exclude the diagnosis. In a review of the literature, none of the CT images of synovial chondromatosis of the temporomandibular joint demonstrated the striking large calcified mass evident in our case and in the case reported by Shirasuna et al (7).

A neuropathic joint presents a histology similar to the present case. However, more inflammatory cells and more destruction of the temporomandibular joint itself would be expected with the extensive involvement noted in our patient.

Periarticular soft tissue calcification is a common complication of chronic renal failure and may exhibit similar radiographic findings to idiopathic tumoral calcinosis. Differentiation between the two entities should be part of the diagnostic work-up and involves investigation of laboratory values, including calcium and phosphorus. Hyperphosphatemia only occasionally has been associated with idiopathic tumoral calcinosis but is invariably present in cases of tumoral calcification in chronic renal failure (12, 13). The patient in chronic renal failure often is hypercalcemic, whereas the calcium levels in tumoral calcinosis are normal (14). In addition to juxtaarticular sites,

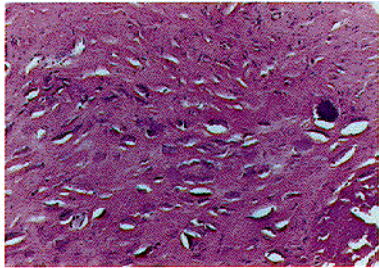
calcified deposits in chronic renal failure have been described in a variety of visceral organs, including the heart and lung. The lesions may or may not produce pain. The incidence of calcifications tends to increase with dialysis. With control of serum phosphorus, the nonvisceral calcifications often regress (15). In the current case, the patient had no evidence of renal disease or other pertinent underlying metabolic disturbances.

Pathologically, the calcified masses were not consistent with the well-defined, active cartilage "mice" of synovial chondromatosis, nor were they congruous with the histologic appearance of calcium pyrophosphate deposition disease. Malignant cells were not identified to suggest a neoplastic process.

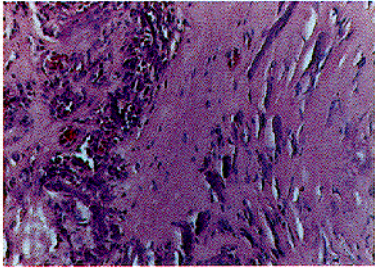
In conclusion, tumoral calcinosis rarely affects the temporomandibular joint. In the proper clinical setting, however, this entity should be considered in the differential diagnosis of a peri-articular calcified temporomandibular joint mass.

References

1. Duret M. Tumeurs multiples et singulieres des bourses sereuses. *Bull Mem Soc Anat Paris* 1899;74:725-731
2. Metzker A, Eisenstein B, Oren J, Samuel R. Tumoral calcinosis revisited: common and uncommon features. *Eur J Pediatr* 1988; 147:128-132
3. Viegas S, Evans E, Calhoun J, Goodwiller S. Tumoral calcinosis: a case report and review of the literature. *J Hand Surg* 1985; 10A(5):744-748
4. Chamyal P, Ahluwalia K. Tumoral calcinosis of the nose. *J Laryngol Otol* 1984;98:827-829
5. McKee P, Liomba N, Hutt M. Tumoral calcinosis: a pathological study of fifty-six cases. *Br J Dermatol* 1982;107:669-674
6. Calhoun K, Clark W, Sternberg C, et al. Tumoral calcinosis in the neck. *Otolaryngol Head Neck Surg* 1987;77:69-71
7. Shirasuna K, Sugiyama M, Yasui Y. Tumoral calcinosis around the mandibular condyle. *Int J Oral Maxillofac Surg* 1991;20:36-37
8. Harkess J, Peters H. Tumoral calcinosis. *J Bone Joint Surg* 1967; 49A:721-731
9. Herzog S, Mafee M. Synovial chondromatosis of the TMJ: MR and CT findings. *AJNR Am J Neuroradiol* 1990;11:742-745
10. Blankestijn J, Panders A, Vermey A, Scherpbier A. Synovial chondromatosis of the temporo-mandibular joint. *Cancer* 1985;55: 479-485
11. Casselman L, Demeulemeester L, Bossuyt M, et al. CT findings in synovial chondromatosis of the temporomandibular joint. *J Comput Assist Tomogr* 1987;11(5):898-900
12. Chew F, Crenshaw W. Idiopathic tumoral calcinosis. *AJR Am J Roentgenol* 1992;158:330
13. Prince M, Schaefer P, Goldsmith R, Chausmer A. Hyperphosphatemic tumoral calcinosis. *Ann Intern Med* 1982;96:586-591
14. Lakhar BB, Lakhar BN. Tumoral calcinosis. *Acta Paediatr Scand* 1991;80(4):474-476
15. Nguyen V, Nguyen K, Kamath V. Unusual feature of soft-tissue calcification in chronic renal failure: tumoral calcification. *Comput Med Imaging Graph* 1991;15(6):397-402



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