Synovial chondromatosis of the temporomandibular joint with intracranial extension

Case report

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The authors report the unusual presentation of an intracranial extension of synovial chondromatosis of the temporomandibular joint. The patient presented with a peripheral facial nerve paralysis and anacusis. Computerized tomography revealed the lesion, but fine-needle biopsy was inconclusive. Craniotomy with removal of the tumor was performed, and pathological studies confirmed the diagnosis. The facial nerve dysfunction was thought to be secondary to direct neural compression.

KEY WORDS • synovial chondromatosis • temporomandibular joint • skull • neoplasm

Synovial chondromatosis is a benign process characterized by the development of metaplastic cartilaginous foci within the synovial membranes of articulating joints. First described in 1813, it was thought to be a neoplastic process; however, after suitable study by pathologists, it was classified as a metaplastic process involving synovial membrane. Trauma to the involved joint is thought to play a role in the development of this condition. It is rarely seen in the large joints of the body. Involvement of the temporomandibular joint has been reported in a few instances, but to our knowledge, intracranial spread has never before been reported.

Case Report

This 59-year-old woman noted the onset in early 1986 of right-sided facial pain, most marked in the temporomandibular joint area. The pain intensified, causing her to seek medical attention.

Examination. She was evaluated by various physicians and attempts were made to obtain a biopsy of an infratemporal mass using a fine needle. She noted the rapid onset of a right facial nerve paralysis on May 1, 1986, and was told that she had a Bell’s palsy. An initial computerized tomography (CT) scan in late May failed to show a lesion. During this time, the patient also noted hearing loss on the right. A magnetic resonance image showed an abnormality both extra- and intracranially. An arteriogram was performed which was not diagnostic. A specimen obtained by needle biopsy was reported as being benign synovial tissue, assumed to be from the temporomandibular joint. A high-resolution CT scan of the temporal bones revealed definite erosion of the labyrinthine segment of the facial nerve and erosion into the apex of the cochlea. A malignancy was highly suspected at that time. The patient was then referred for surgery.

Operation. On December 11, 1986, a middle fossa craniotomy was performed with removal of a cystic mass containing cartilage fragments of various sizes. The facial nerve was not explored because it was believed that its function would partially recover once the compression was relieved. The hearing loss was considered permanent. The mass was in direct continuity with the temporomandibular joint through a small defect in the floor of the middle fossa.

Postoperative Course. The patient’s recovery was uneventful. Her hearing is still absent 13 months postoperatively, but her facial nerve function is returning. By House’s facial nerve grading system, she is classified in Grade IV/VI and is expected to experience further return of facial nerve function. The final pathological diagnosis was benign synovial chondromatosis. A fol-
low-up CT scan revealed no recurrence; however, since
the resection did not include the entire synovial lining
of the temporomandibular joint, recurrence is a possi-
bility. If this occurs, a primary temporomandibular
joint procedure will be required.

Discussion

Aggressive surgical treatment of temporomandibular
joint disorders is increasing in frequency. The ability to
diagnose surgically correctable lesions of the temporo-
mandibular joint partially reflects the availability of
better diagnostic modalities, including high-resolution
CT scanning and joint arthroscopy. Lesions once
thought to be extremely rare are now being recognized
more frequently.

Synovial chondromatosis, most common in the knee
and less so in the shoulder and hip, is a joint disorder
in which metaplasia in the synovial connective tissue
of the joint causes small foci of hyaline cartilage ("joint
mice") to form. Involvement of the temporomandibular
joint is rare, with only five cases reported before
1969. Since then, approximately 30 cases have been
added to the literature. Intracranial extension, how-
ever, has not previously been reported.

Pathologically, the synovial lining of diarthrodial
joints is damaged by the metaplastic process. The
formed cartilaginous nodules become calcified and
grow. They may become detached from the synovium
and float free in the synovial fluid. Other disease
processes, such as degenerative joint disease or traum-
atic injuries, can cause similar loose bodies in the
joint space (Fig. 1 left).

Extracapsular extension to the parotid has been re-
ported to mimic a parotid gland neoplasm. Our case
was very unusual since no significant temporomandi-
bular joint symptoms were present prior to surgery. The
primary presenting signs and symptoms pointed to
cranial nerve involvement by a very aggressive disease
process. The amount of bone erosion visible on CT was
impressive (Fig. 1). The possibility of a benign neo-
plasm such as a menigioma or neurinoma was also
considered.

Our patient was not seen by an oral surgeon before
surgery since her signs and symptoms pointed to a
malignant tumor at the base of the skull that had eroded
intracranially. One biopsy specimen obtained with a
fine needle suggested the problem but was inconclusive.
If we had been aware of the diagnosis before surgery, a
combined approach to the temporomandibular joint
and tumor extension would have been performed. As
it was, only the intracranial contents and part of the
synovium of the temporomandibular joint could be
removed. Pathological diagnosis is relatively easy when
the appropriate tissue is obtained.

References

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Fig. 1. Computerized tomography scans. Left: Axial view showing calcified "joint mice." Right: Coronal
view of the intracranial mass extension.
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