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Central Giant Cell Granuloma of the Temporal Bone

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Summary: We present two cases of central giant cell granuloma in the temporal bone. CT showed an enhancing tumor causing a smoothly margined temporal squamosa and floor of the middle fossa. External carotid angiograms showed tumor stain mainly supplied by the temporal branches of the internal maxillary artery. In one case, MR images showed a hypointense mass on both T1- and T2-weighted images that was thought to reflect the pathologic character of this lesion.

Index terms: Granuloma; Temporal bone, neoplasms

Central giant cell granuloma is a synonym of giant cell reparative granuloma. Reparative is not currently accepted as a suitable descriptive term because of the actual destructive nature of the giant cell granuloma (1). A central giant cell granuloma is a rare nonneoplastic lesion, usually seen in the jaw bones. Although this lesion had been considered peculiar to the jaw bones, a few cases of lesions outside the jaw bones have been reported (2–6). We found reports of four cases of such a lesion in the skull, three in the temporal bone and one in the cranial vault (2–5). We present two cases of central giant cell granuloma in the temporal bone with special reference to neuroradiologic findings.

Case Reports

Case 1

A 36-year-old man noticed a right temporal mass that gradually enlarged. As the mass increased in size he began to have pain. He had no history of trauma to the area. On admission, a smooth, elastic, firm mass was palpable in the right temporal area. Neurologic examination was normal except for a right conductive hearing loss. Laboratory data, including serum calcium, phosphorus, and alkaline phosphatase levels, were unremarkable.

Skull radiography showed a defect of the right temporal squamosa and the floor of the middle fossa. Computed tomography (CT) (Fig 1A and B) showed a hyperdense mass in the right middle fossa that enhanced after administration of intravenous contrast material. At bone window setting, the mass was noted to expand the temporal squamosa and displace a thin rim of cortex in a smooth fashion (Fig 1C and D). The mass appeared to originate from the temporal bone and extended to the region of the eustachian tube. Involvement of the eustachian tube with secondary otitis media was thought to be responsible for a conductive hearing loss. Most of the mass was relatively hypointense on T1-weighted magnetic resonance (MR) images (Fig 1E) and hypointense on T2-weighted images (Fig 1F). A part of the mass showed prolonged T1 and T2 relaxation times. On T1-weighted images (Fig 1G) after intravenous administration of gadopentetate dimeglumine, most of the mass did not enhance, although areas of long T1 and T2 on precontrast MR images showed intense enhancement. A right external carotid angiogram (Fig 1H) showed tumor stain fed by the temporal branches of the internal maxillary artery.

At surgery, a friable dark brown mass with a tendency to bleed was found and completely excised. Microscopic examination (Fig 1I) showed multinucleated giant cells scattered unevenly around foci of hemorrhage, spindle-shaped fibroblasts, dense collagenous fibers with abundant hemosiderin pigment, and new bone formation. The pathologic diagnosis was central giant cell granuloma.

Case 2

A 28-year-old man with no history of trauma had tinnitus and hearing loss that gradually worsened over several months. A right external auditory canal mass was found, and an incisional biopsy was performed. Microscopic examination suggested a giant cell tumor. He was referred to our institution for complete excision of the tumor. Neurologic examination at the time of admission revealed conductive hearing loss on the right side. Laboratory data were normal.

Skull radiography showed a well-demarcated, multicystic, radiolucent area in the right temporal squamosa and a bony defect in the floor of the middle fossa. CT (Fig 2A and B) showed a large mass with soft-tissue density that enhanced after intravenous administration of contrast.
Case 1: central giant cell granuloma of the temporal bone.

A, Precontrast CT scan shows a hyperdense mass in the right temporal bone.
B, Postcontrast CT scan shows an enhanced tumor.
C, Axial and D, coronal CT scans at bone window setting. Thin, displaced bony rim is well appreciated, indicating a bone origin.
E, Coronal T1-weighted inversion-recovery MR image (2000/30/1 [repetition time/echo time/excitations], 550-millisecond inversion time) shows a relatively hypointense mass containing more hypointense areas.
F, Coronal T2-weighted image (2000/120/2) shows a hypointense mass. Hypointense areas seen within the mass on the T1-weighted image show hyperintensity.
G, Coronal T1-weighted image (500/30/2) after intravenous injection of gadopentetate dimeglumine shows lack of enhancement of most of the mass. Areas of prolonged T1 and T2 are well enhanced.
H, Lateral subtraction angiogram of the right external carotid artery shows a hypervascular mass supplied by temporal branches of the internal maxillary artery.
I, Microscopic photograph (×20) shows multinucleated giant cells (small arrows) scattered unevenly around foci of hemorrhage, spindle-shaped fibroblasts, and dense collagen fibers with abundant hemosiderin pigment (large arrows).
The mass was thought to originate from the temporal bone because it was circumscribed by a thin rim of displaced cortical bone. A right external carotid angiogram (Fig 2C) showed tumor stain supplied by the temporal branches of the internal maxillary artery and petrosquamosal branch of the middle meningeal artery.

Surgery revealed a friable dark brown mass with a tendency to bleed. The tumor was completely excised. Histologic examination (Fig 2D and E) showed findings consistent with a central giant cell granuloma.

**Discussion**

Central giant cell granuloma is a rare nonneoplastic lesion commonly located in the mandible and maxilla. Jaffe (7) considered it to be a local reparative reactive process related to traumatic intraosseous hemorrhage or a periosteal reaction. However, a history of trauma often is not obtained from the patient (8). The cause of this lesion is controversial. After reviewing the clinical, histologic, and histomorphometric features of central giant cell granulomas and giant cell tumors, some pathologists suggested that these two lesions represent a spectrum of a single disease process modified by the age of the patient and the site of occurrence (1). In general, central giant cell granuloma is histologically characterized by a loose, slightly vascular stroma composed of both oval and spindle-shaped fibroblastic cells. Multiple areas of hemorrhage, abundant hemosiderin pigment, and marked fibrosis are always present in a central giant cell granuloma. The giant cells are multinucleated, but, unlike those in a giant cell tumor, they are relatively small and unevenly distributed. They are usually clumped around the hemorrhagic areas. The number of nuclei is smaller than in the giant cells associated with the giant cell tumor of bone. Delicate trabeculae of newly formed bone are frequently present within the tissue of a central giant cell granuloma (2).

On the other hand, in a giant cell tumor the stroma is predominantly composed of plump round and oval cells. Fresh hemorrhage is slight to moderate, and hemosiderin deposits are rare and small. New bone formation is conspicuously absent (2).

Central giant cell granuloma of the temporal bone is extremely rare. To our knowledge, only
three such cases have been reported in the English language literature (2–4). However, Hirschl and Katz (2) stated that 18 of 23 reported cases of giant cell tumor arising in the temporal bone actually proved to be giant cell reparative granuloma (central giant cell granuloma). Previously published case reports have discussed findings of plain skull radiography and CT but not MR. In our cases, CT showed a soft-tissue density or hyperdense mass extending from the temporal squamosa to the middle fossa. The lesion enhanced after intravenous administration of contrast material. The bone was not destroyed but rather expanded and eroded smoothly by the tumor. These CT findings are nonspecific and cannot differentiate this entity from other lesions such as giant cell tumors or aneurysmal bone cysts.

In case 1, most of the tumor was hypointense on T1- and T2-weighted images. Unfortunately, we could not obtain MR images in case 2. The signal intensity may reflect the histologic character of a central giant cell granuloma. Hemosiderin is known to shorten T2 relaxation times (9). Fibrous tissue decreases signal intensity on T1- and T2-weighted images because the mobile spin density of predominantly fibrous tissue is low, providing little MR signal (10). Abundant hemosiderin pigment and marked fibrosis in the stroma were thought to be responsible for the hypointensity of most of the mass on T1- and T2-weighted images.

The differential diagnosis on imaging includes giant cell tumor, osteitis fibrosa cystica (brown tumor), and aneurysmal bone cyst. Compared with unaffected bone, giant cell tumors show hypointensity on T1-weighted images and isointensity to hyperintensity on T2-weighted images (11). Osteitis fibrosa cystica may be indistinguishable histologically from central giant cell granuloma of bone (2). The MR appearance of osteitis fibrosa cystica has not been described. Although it may appear similar to central giant cell granuloma, osteitis fibrosa cystica can be excluded by measuring levels of serum calcium, phosphorous, and alkaline phosphatase. Aneurysmal bone cysts are expansile, lytic bone lesions that on pathologic examination consist of thin-walled, blood-filled cavities lacking normal endothelium and elastic lamina (12). The MR appearance of an aneurysmal bone cyst has been reported as an expansile lesion bordered by a thin low-signal rim, increasing signal with augmented T2 weighting, and possibly a lobulated contour and/or fluid levels within (12). On the basis of pathologic findings, MR imaging might be of help in making an accurate diagnosis of central giant cell granuloma, although further reports are necessary to determine its efficacy.

References

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