Large Giant Cell Reparative Granuloma of the Petrous Bone
—Case Report—

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Abstract

A 41-year-old man presented with a large mass bulging over the suprazygomatic temporal region. Neuroradiological examination showed that the huge extra-axial mass with osteolytic character originated from the upper surface of the petrous bone. Preoperative obliteration of the feeding arteries with superselective intravascular embolization was helpful for the total removal of the tumor. Histological examination revealed that the tumor consisted of massive fibrohistiocytic proliferation with numerous heavily hemosiderin-laden macrophages and numerous multinucleated giant cells. The most probable diagnosis was giant cell reparative granuloma. Therefore, no postoperative irradiation or other adjuvant therapy was given.

Key words: giant cell reparative granuloma, skull base surgery, petrous bone, intracranial mass

Introduction

Giant cell reparative granuloma (GCRG) is basically considered to be identical to central giant cell granuloma, but this remains controversial because of the absence of clear-cut definitions.6,18) GCRG has a benign course and intracranial occurrence is extremely rare.14) Inadequate information is available to correctly manage this tumor.8,11,12) All giant cell lesions of the temporal bone were regarded as giant cell tumors (GCTs).7,12) However, the differential diagnosis of GCRG from GCT is important because the biologic behaviors are quite different.3,12) GCRG occurs predominantly in the mandible and GCT in the epiphyses of the long bones,6) but both lesions are rare in the skull. GCRG is frequent in the first 2 decades of life, whereas GCT occurs in older patients.12) GCT of the bone rarely appears in patients below the age of 18 years and practically never in those under the age of 10 years.12)

The first case of GCRG was located in the mandible and maxilla.14) Subsequently, GCRG has occurred in many sites including the orbit,19) cranial vault,11) temporal bones,1,2,6,12,15,17) sphenoid bone, ethmoid bone,24) facial bones,4,20) and short and long bones of the axial skeleton.5,8) GCRG has recurred in the intracranial region or cranial vault.3,8,12,15)

GCRG may not be a neoplasm but a reactive and reparative mass to past episodes such as trauma and unknown events.5,8,10–12,23) Extensive surgical removal is recommended, since the tumor is benign,21) and leads to a complete cure. However, a presumptive diagnosis of GCRG is extremely difficult to make before surgery. Therefore, surgical resection is the best choice of treatment for this lesion. We describe a case of GCRG with destruction of the temporal squama, zygomatic arch, temporal base, and the structures forming the auditory canal and pathway. The possible histological diagnosis was GCRG.

Case Report

A 41-year-old man presented to our institute on January 26, 2001 with a large mass bulging over the suprazygomatic temporal region and a feeling of fullness in his right ear. He had never experienced any type of head trauma. Physical examination showed this mass had formed a 4 cm external bulge and the arch was dislocated inferiorly and partially lytic. Radiography demonstrated a lytic lesion in the temporal bone including the basal region, the glenoid fossa of the temporomandibular junction, and
the zygomatic arch. The arch was dislocated inferiorly due to compression of the bulging mass. Computed tomography showed a large mass with soft-tissue density and enhancement with contrast medium. The mass probably originated from the temporal bone, especially the anterior rim of the petrous portion around foramina ovale, lacerum, and rotundum, because the temporal squama was circumscribed by a thin rim of displaced cortical bone and the basal structure was remarkably lytic (Fig. 1). Right external carotid angiography showed tumor staining supplied by the temporal branches of the internal maxillary artery and branches of the middle meningeal artery. Magnetic resonance (MR) imaging showed a large and multifocal isointense mass located mainly in the temporal fossa (Fig. 2). The mass included several nodules and the cyst wall which was enhanced by gadolinium. No preoperative biopsy was performed. The presumptive diagnosis was chondroblastoma, chondrosarcoma, cystic meningioma, and other tumors.

Before surgery, some branches of the middle meningeal and internal maxillary arteries were occluded by superselective intravascular embolization using polyvinyl alcohol and Guglielmi detachable coils. The whole aspect of the mass, temporal base, zygomatic arch, and parotid gland were exposed through a large Falconer’s skin incision. The temporal fascia was incised in curved-linear fashion to include the frontal branch of the facial nerve and the zygomatic arch was exposed as far as the lateral wall of the orbital fossa. The upper and posterior parts of the parotid gland were dissected from the fascia of the masseter muscle. The divided zygomatic arch was reflected inferiorly and the entire temporal muscle was elevated from the lateral skull but left attached to the coronoid process of the mandible. Part of the mass was exposed. The soft tumor easily bled. Frozen section examination identified only the features abundant in giant cells and no histological diagnosis could be made.

Right frontotemporal craniotomy was performed to expose the upper and anterior aspects of the tumor involving the dura mater. The tumor was strongly adhered to the dura mater and was sharply dissected. Resection of the tumor which could not be dissected from the dura mater resulted in exposure of the surface of the petrous bone, so the tumor possibly originated from this area. Tumor extension was posteriorly from the lateral margin of the foramina lacerum, ovale, spinosum, and rotundum anteriorly to the superior orbital fissure. Almost the whole of the temporal bone was damaged but the maxillary condyle was spared. Only the antrum mastoidea of the petrous bone was opened but the auditory function was preserved. After complete resection of the tumor (Fig. 3), excised fascia of temporal muscle was placed to protect the middle and internal auditory apparatus.

The postoperative course was uneventful. Within a month, the contours of the face except the forehead were almost completely normalized from a cosmetic viewpoint (Fig. 4).

The tumor removed from the right temporal bone...
was a large bulging osteolytic mass measuring approximately $4 \times 4 \times 1.8$ cm in size, dark reddish in tint, and rust colored after fixation in 10% formalin. Histological examination found massive fibrohistiocytic proliferation with numerous heavily hemosiderin-laden macrophages and numerous multinucleated giant cells (Fig. 5). There were some cystic spaces, chondro-osseous metaplasia, and reactive new bone formation. Many tumors or conditions present with numerous multinucleated giant cells and the differential diagnosis is often not easy, but GCRG was the most probable diagnosis in this case.

**Discussion**

The present case illustrates two problems with the preoperative histological diagnosis of GCRG and the clinical management. GCRG consists of small, elongated, or oval-shaped stroma cells mixed with multinucleated giant cells. GCT also consists of two similar types of cells, but the giant cells are larger and more numerous. Giant cells in GCRG tend to have a patchy distribution, whereas those in GCT are uniformly dispersed. Mitotic figures and foci of necrosis are present in GCT but not in GCRG. The differential diagnosis of GCRG from benign giant cell lesions such as aneurysmal bone cyst is also important. In the present case, our histological diagnosis was GCRG. GCRG is similar to brown tumor associated with hyperparathyroidism and mesenchymal tumor caused by osteomalacia. However, these possibilities were excluded by clinical analysis. The differential diagnosis of GCT is a very difficult problem because the definition is not clear, and reported cases of GCTs of the bone include many variants of GCRG.

Recently, genuine giant cell tumor of the bone has been considered as a true neoplastic change, but we thought the present case was a reactive and reparative process rather than a true neoplasm because each component of this lesion was thought to be normal tissue and devoid of apparent atypism. Eosinophilic granuloma was ruled out because the mononuclear cells were histiocytes and not Langerhans’ cells. This histological evidence indicated GCRG as the most likely diagnosis. The pathogenesis of GCRG remains unclear. The lesion may represent a local reparative process related to intraosseous hemorrhage or periosteal reaction induced by trauma. However, many cases including the present case had no history of trauma. Thus, the term of “reparative” may be incorrect, but GCRG may still be a cellular reaction with non-neoplastic proliferation. The 5–6% of stromal cells, but none of multinucleated giant cells, in a case of recurrent GCRG

**Fig. 3** Magnetic resonance images 6 months after the surgery indicating total removal of the mass.

**Fig. 4** Pre- (left) and postoperative (right) photographs of the face showing no major cosmetic impairment after surgery.

**Fig. 5** Photomicrograph clearly demonstrating fibrohistiocytic proliferation mixed with multinucleated giant cells. Hematoxylin and eosin stain, $\times 150$. 
had a proliferative activity representative of a positive immunoreactivity for MIB-1 antibody. This suggests that the lesion expands by proliferation of the stromal component, with a growth rate roughly between those of typical benign and malignant brain tumors. Our immunohistological study similarly showed a proliferative activity associated with the positive response to MIB-1 antibody, suggesting the possibility of recurrence by proliferation of the stromal component (data not shown). Therefore, subsequent MR imaging was required to monitor recurrence of the present lesion.

Surgical design is quite important to totally remove the mass and preserve facial nerve function. Although impossible if the mass involves the facial nerve, the encapsulated mass was separated from parotid-facial nerve complex in the present case. The upper and posterior parts of parotid gland were dissected from the fascia of the masseter muscle. The mobilized zygomatic arch was reflected inferiorly, and the attachments to the masseter muscle were preserved. The above preparation enabled almost complete preservation of the facial function and exposure of the anterior configuration of the tumor. The facial nerve distributed to the forehead is likely to be impaired when the mass is dissected from the parotid gland, because the mass overlies the entry of the facial nerve into the parotid gland. The hinge of the temporal muscle exposed the inferior aspect and provided easy access to the skull base such as the foramina rotundum, lacerum, and ovale, and the superior orbital fissure. Recently, a case of temporal GCRG was reported, but the superior orbital fissure excluded the need for postoperative irradiation or other adjuvant therapy. After the present surgery, we plan MR imaging of this patient once a year to monitor recurrence.

References

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