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Giant Cell Reparative Granuloma of the Petrous Temporal Bone: A Case Report and Literature Review

ABSTRACT—Giant cell reparative granuloma (GCRG) is an unusual, benign bone lesion that most commonly affects the maxilla and mandible; skull involvement is rare. The etiology is uncertain but may be related to trauma. GCRG is difficult to distinguish from giant cell tumor of the bone and has a lower recurrence rate. Thirteen reports of temporal bone GCRG in 11 patients have been reported. One report of a petrous GCRG in a 3-year-old girl has been identified. A 38-year-old male presented with a 2-year history of fullness in his left ear, ipsilateral hearing loss, and intermittent cacostmia. Computed tomography and magnetic resonance imaging revealed a large left-sided anterior temporal extradural mass. The patient underwent a left frontotemporal craniotomy and resection of a left temporal fossa tumor that involved the petrous and squamous parts of the temporal bone. The patient's post-operative course was uneventful, except for increased hearing loss secondary to opening of the epi-tympanum. Follow-up at one month revealed no other problems. Histopathology of the specimen was consistent with a giant cell reparative granuloma.

In 1953, Jaffe¹ first described the uncommon bone lesion giant cell reparative granuloma (GCRG) of the jawbone. This lesion occurs most commonly in maxilla and mandible^{2,3}; rare cases of GCRG have been reported in the bones of the skull, including the orbit,^{4,5} paranasal sinuses,⁶ cranial vault,^{7,8} temporal,^{9–19} sphenoid,²⁰ and ethmoid^{21,22} bones, and in the small bones of the hands and feet.²³ Considered a benign lesion of bone, GCRG is locally aggressive and requires surgical excision. It is important to distinguish GCRG from true giant cell tumor of bone, which has a higher incidence

of recurrence. The following discussion reviews the literature and presents a case of a giant cell reparative granuloma in the petrous temporal bone of a 38-year-old man.

CASE REPORT

A 38-year-old otherwise healthy man presented with a 2-year history of fullness in his left ear and left-sided hearing loss. In addition, the patient perceived un-

Figure 1. (A) Computed tomography (CT) of skull base bone windows demonstrating erosion of bone from floor of middle fossa. (B) CT of head with contrast with hypodense mass in left anterior middle cranial fossa with rim of bone and slight contrast enhancement.

usual odors intermittently. Previous evaluation suggested these symptoms were related to sinus disease, but a septoplasty performed to relieve the symptoms had been ineffective. Physical examination was normal except for left-sided maximum conductive hearing loss confirmed with audiometry. Laboratory data were normal. Skull radiographs were interpreted as normal. Computed tomography revealed a large hypodense left anterior temporal mass with a rim of calcification and

erosion of the floor of the middle fossa, the superior semicircular canal, and into the mastoid air cells (Fig. 1, A and B). Magnetic resonance imaging demonstrated a $5.4 \times 4 \times 4$ -cm anterior temporal fossa extradural mass that was encapsulated, multilobular, and septated, with enhancement of the capsule and some of the septa (Fig. 2, A and B). The lesion had no associated edema but had marked mass effect on the adjacent temporal lobe. The patient underwent a left frontotemporal cran-

Figure 2. Axial (A) and coronal (B) plane magnetic resonance imaging with gadolinium demonstrating multilobular anterior temporal mass with significant mass effect and rim contrast enhancement.

Figure 3. (A) Giant cell-rich area of the tumor within a background of mononuclear stromal cells and spindle-shaped fibroblasts, indistinguishable from true giant cell tumor of bone. Hematoxylin and eosin; original magnification 280 $\times$. (B) Adjacent area of the tumor, dominated by mononuclear stromal cells, hemorrhage, fibrosis, and foam cells, with only rare giant cells. Hematoxylin and eosin; original magnification 280 $\times$. (C) Section of tumor showing erosion and destruction of bone. Hematoxylin and eosin; original magnification 280 $\times$.

iotomy and resection of a left temporal fossa tumor that involved the epidural space, left temporal fossa, and petrous and squamous parts of the temporal bone.

Frozen section performed at the time of surgery was initially interpreted as giant cell tumor of bone. Additional sampling by permanent sections displayed a lesion composed of multinucleated giant cells with mononuclear stromal cells and associated hemorrhage, fibrosis, cholesterol clefts, foam cells, and hemosiderin-laden macrophages with erosion and focal destruction of bone (Fig. 3, A–C). The final histopathologic diagnosis was GCRG. The patient's postoperative course was uneventful, except for complete sensorineuronal hearing loss. Follow-up revealed no other problems.

DISCUSSION

GCRG of the maxilla and mandible is a benign lesion of bone that most commonly presents in the first two decades of life^{9,13} and is twice as common in women as men.⁷ Histologically, GCRG is composed of multinucleated giant cells that cluster in a patchy distribution within a stroma of oval or spindle-shaped fibroblastic cells.^{9,19} GCRG also frequently contains foci of osteoid substance, hemorrhage, and hemosiderin deposits.^{9,13,14}

The pathogenesis of GCRG is controversial; Jaffe¹ originally proposed GCRG is a local reparative process in response to a trauma-induced intraosseous hemorrhage. However, many patients with GCRG have no history of trauma. Infectious and developmental etiologies have been suggested.^{2,9} Using immunohistochemistry studies, Maruno et al.¹⁹ demonstrated proliferative activity in the stromal component with none in the giant cells. This suggested stromal proliferation may be the mechanism by which GCRG expands.

The first case of GCRG in the temporal bone was reported by Hirschl and Katz in 1974.^{9,10} One year previously in 1973, Wolfowitz and Schmanan²⁴ reported a case of a giant cell lesion of the temporal bone in which a probable GCRG could not be accurately distinguished from a true giant cell tumor of bone. Hirschl and Katz⁹ reviewed the previously published literature on true giant cell tumor of the temporal bone and determined 18 of 23 cases previously reported as giant cell tumor were actually GCRG. Since Hirschl's report, GCRG has been recorded in the orbit,^{4,5} paranasal sinuses,⁶ sphenoid,²⁰ ethmoid,^{21,22} and cranial vault.^{7,8} Thirteen reports of GCRG of the temporal bone in 11 patients have been published in the literature.^{9–19} Four of these cases occurred in males and seven in females, with an age range of 4 months to 56 years. Most patients were less than 35 years old at the time of diagnosis. The most common presenting signs

Table 1. Cases of Giant Cell Reparative Granuloma of the Temporal Bone

Case	Author	Year	Age	Sex	Symptoms	Trauma	Therapy	Recurrence
1	Hirschl	1974	34	F	Ear/neck pain, hearing loss, tinnitus	Yes	R	No
	Katz*	1974	36	F	Ear/neck pain, hearing loss, tinnitus	Yes	R	No
2	Colclasure	1981	10	M	Hearing loss, mass	NR	S	No
3	Colclasure	1981	22	M	Hearing loss, mass	NR	S, R	No
4	Lin	1989	41	M	NA	NA	S	Yes; 3 yr
5	Tesluk	1989	56	M	Hearing loss, mass, facial weakness, vertigo, weight loss	No	S	No
6	Ciappetta	1990	25	M	Severe headache, diplopia, eyeball protrusion, nausea/vomiting, urinary incontinence	Yes	S	No
7	Cohen	1993	4 m	F	Mass	NR	S	NR
8	Lewis	1994	32	F	Ear fullness, mass	Yes	S	No
	Ung†	1998	36	F	Ear fullness, mass	NR	S	Yes; 4 yr
9	Nemoto	1995	36	M	Mass, pain, hearing loss	No	S	NR
10	Nemoto	1995	28	M	Hearing loss, mass, tinnitus	NR	S	NR
11	Maruno	1997	3	F	Facial palsy, hearing loss	No	S	Yes; 1 yr

*Same as case 1 reported by Hirschl.

†Recurrence of case 8 reported by Lewis.

R, radiotherapy; S, surgery; NR, not reported; NA, not available in translation.

and symptoms were hearing loss, a mass, pain, facial paresis, and tinnitus (Table 1).

The differential diagnosis of GCRG includes other giant cell lesions, such as true giant cell tumor of bone, aneurysmal bone cyst, brown tumor of hyperparathyroidism, chondroblastoma, and fibrous dysplasia. Brown tumors of hyperparathyroidism may occur at any age and are usually multiple.¹⁵ These tumors may be distinguished from GCRG by normal laboratory data, including normal levels of serum and urinary calcium, phosphates, and alkaline phosphatase.⁹ Aneurysmal bone cyst may be differential from GCRG by its microscopic appearance that reveals a honeycomb network of thin-walled cavities filled with blood.¹⁹ Chondroblastoma and fibrous dysplasia also have unique histologic presentations.²⁵

Because of its more aggressive behavior, the most important lesion to distinguish from giant cell reparative granuloma is true giant cell tumor of bone. True giant cell tumor of bone usually occurs in the epiphyses of long bones and is rare in the skull.^{11,13} Giant cell tumor occurs in an older population than GCRG; the age of onset for giant cell tumor ranges from 25 to 40 years, occurring rarely below the age of 20.¹⁴ Both GCRG and giant cell tumor appear as lytic defects on radiographic examination^{13,17} and must be distinguished by histology.

On histologic examination, giant cell tumor has larger

giant cells with a greater number of nuclei, and the giant cells are more uniformly dispersed than in GCRG.¹⁴ Giant cell tumor is less likely to have foci of osteoid, hemorrhage, hemosiderin, or fibrosis than GCRG and has an increased incidence of mitotic figures and foci of necrosis.^{17,19} Thorough sampling of the lesion is necessary because some microscopic fields of GCRG can be indistinguishable from true giant cell tumor. Differentiation between GCRG and giant cell tumor has important therapeutic implications. True giant cell tumor of bone is generally considered to have a worse prognosis than GCRG and requires adjunctive radiotherapy in addition to surgical excision.²⁵ A recent report suggests giant cell tumor of the head may be less aggressive than giant cell tumor of the long bones, with fewer reported cases of recurrence or metastasis.¹⁷ Giant cell tumor of bone has recurrence rates of up to 45%¹³ and in the long bones 1–6% of giant cell tumors metastasize to the lung within 5 years.³

In contrast, treatment of GCRG is complete surgical excision alone.^{11,17} For cases in which complete excision may be impossible, partial resection followed by radiotherapy may be acceptable.^{14,17} In general, GCRG has a good prognosis, with a low 12–16% incidence of recurrence;¹⁴ unlike true giant cell tumor, no cases of metastases or malignant transformation have been reported.²⁶ In the 11 reported cases of GCRG of the tem-

poral bone, 10 patients underwent surgical curettage or excision. Two patients additionally received radiotherapy and one patient received radiation alone as primary treatment. Of these 11 patients, 3 had recurrences, which occurred at 1, 3, and 4 years, postoperatively. Two patients proceeded to en bloc resection; one of these patients had no recurrence reported at 6-month follow-up. No information regarding recurrence was available for the second patient. The third patient received radiation and had no recurrence after 2 years (Table 1).

CONCLUSION

Giant cell reparative granuloma is a rare lesion in the temporal bone. This case, in which the initial pathology on frozen section was interpreted as giant cell tumor of bone, illustrates the importance of careful distinction between GCRG and true giant cell tumor of bone. It is necessary to differentiate between the two lesions because the outcome may influence therapeutic planning. True giant cell tumor requires adjunctive radiotherapy and surgical excision, whereas giant cell reparative granuloma should be treated with complete surgical excision alone when possible. Experience in previously published cases demonstrates most patients with GCRG of the temporal bone who are treated with complete surgical excision will have no recurrences.

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