CASE REPORT

Giant Cell Tumor (Osteoclastoma) of the Petrous Bone: Case Report

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***ABSTRACT*—A case of a basal middle fossa giant cell tumor occurring in a 46-year-old man is described. The lesion appeared at the computed tomography (CT) scan examination as an hypodense mass with a peripheral "ring-like" enhancement, and no evident erosion of the skull base. The tumor, which infiltrated the basal temporal parenchyma, was removed via a temporal transzygomatic craniotomy, and extensive drilling of the petrous bone. Despite the occurrence of significant postoperative complications, the patient ultimately showed a good clinical outcome, with no signs of recurrence at the 1-year follow-up CT scanning. The clinical and diagnostic aspects and the management policy of this rare lesion are discussed.**

Giant cell tumors, or osteoclastoma, are uncommon bone neoplasms, rarely involving the craniofacial skeleton.[1–3] Actually no more than 60 cases of cranial giant cell tumors have been described in the literature.[3–6]

We encountered a case of basal middle fossa osteoclastoma occurring in a young adult. The rarity of the lesion, its atypical computed tomography (CT) appearance, and the problems related to its management, prompted the present report.

CASE REPORT

A 46-year-old Caucasian, right-handed man was admitted in May, 1996, with a chief complaint of progressively decreasing hearing on the right of 8 months duration. At admission the neurological examination was within normal limits except for a 90% right conductive hearing loss. Plane skull X rays were normal. CT scan examination showed a large (5 cm diameter), hypodense lesion, which showed a high enhancement along the peripheral portion following injection of contrast medium (Fig. 1A and 1B). The mass occupied the right middle fossa and showed intimate relationships with the petrous bone, which, however, did not appear to be eroded (Fig. 1C).

The mass was approached using a basal subtemporal transzygomatic approach. The upper branch of the facial nerve was spared by performing a subgaleal-subfascial dissection of the soft tissues, as described in a previous paper.[7] The mass was mainly extradural, but invaded the basal temporal dura and infiltrated the brain parenchyma. The intradural portion of the lesion was removed by ultrasonic aspirator until the surface of the petrous pyramid was exposed. This appeared to be quite abnormal, and was carefully drilled in the attempt to achieve gross total removal of the lesion. However, due to the lack of reliable landmark for safely conducting bone dissection in the direction of the carotid artery and the facial nerve, drilling was stopped as soon as the bone appeared to be grossly normal. Moreover, we

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could not identify the greater superficial petrosal nerve (GSPN) to cut it for avoiding tension to the facial nerve because it was embedded in the tumor mass itself. Drilling was complicated by troublesome, continuous bleeding, which was eventually controlled with Oxycel and bone wax. Histologic examination of the mass showed a highly cellular tumor, with ovoid stromal cells containing multinucleated giant cells (Fig. 2). Histologic diagnosis was giant cell tumor.

The patient awoke with complete right facial paralysis. Otherwise, his neurological examination was normal. Twenty-four hours later, his consciousness slightly decreased, and postoperative CT scan showed a 3-cm blood clot in the surgical cavity (Fig. 3), which was eventually removed.

The patient progressively improved thereafter, and was discharged on the 21st postoperative day. A later postoperative CT scan showed no evidence of residual tumor (Fig. 4). At the last follow-up, 10 months after surgery, the patient showed his preoperative conductive hearing loss and a slight right facial paresis (House grade IV). The remaining neurological examination was normal.

DISCUSSION

Giant cell tumors are locally aggressive bone lesions characterized by vascularized tissue that contains numerous multinucleated giant cells dispersed through plump, spindly and/or ovoid cells. Giant cell tumors, otherwise named "osteoclastomas" (usually in British literature), arise predominantly along the epiphyses of the long bones, occur with equal frequency in both sexes, and, as a rule, present in the third and fourth decades of life. The skull is a rare location for these tumors: approximately 1% in large casuistics.[5]

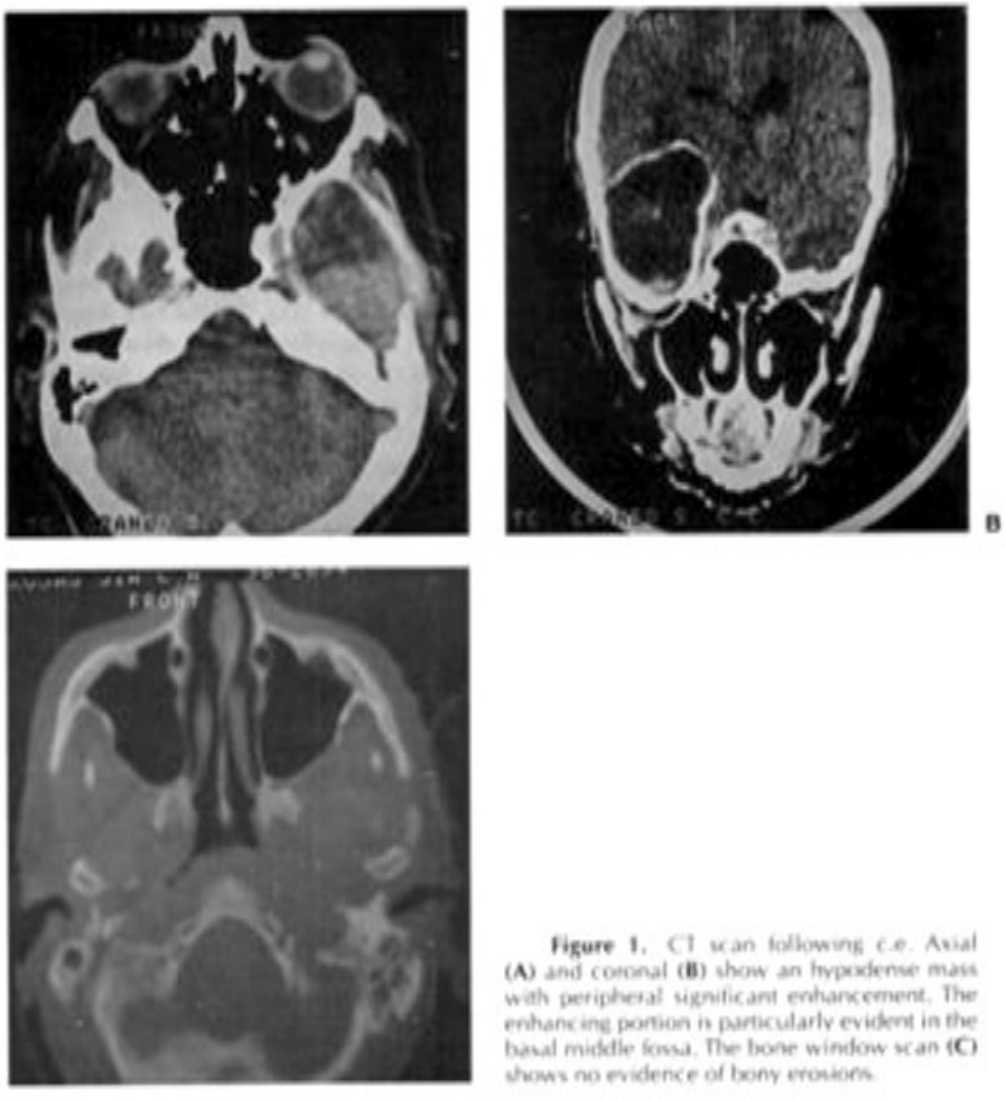

Figure 1. CT scan following c.e. Axial **(A)** and coronal **(B)** show an hypodense mass with peripheral significant enhancement. The enhancing portion is particularly evident in the basal middle fossa. The bone window scan **(C)** shows no evidence of bony erosions.

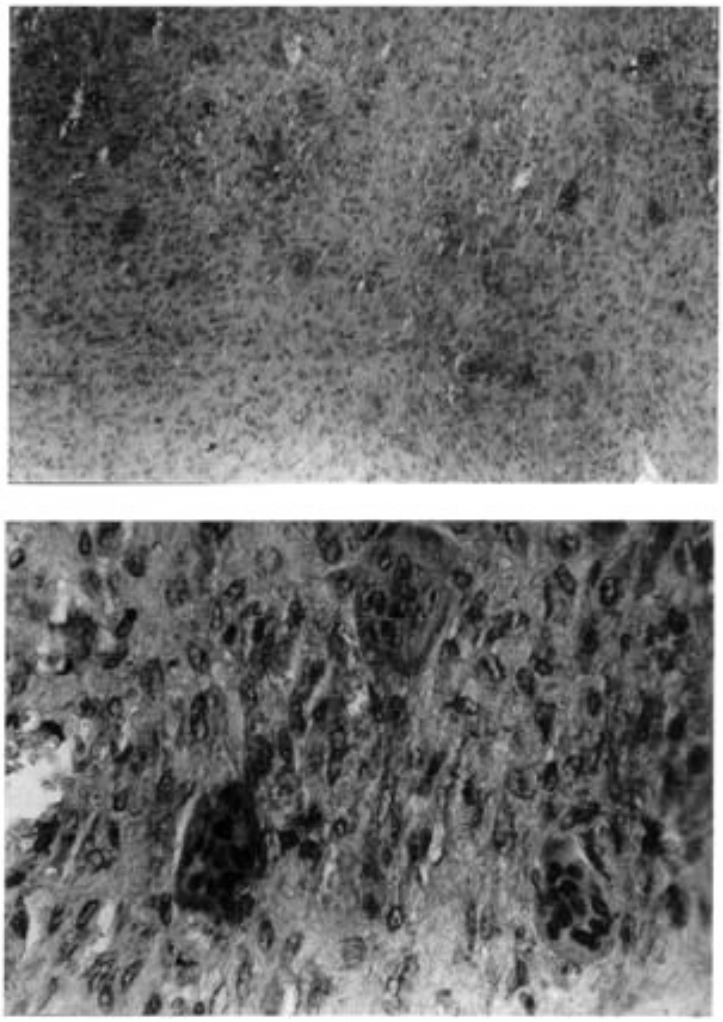

Figure 2. **(A)** Photomicrograph of paraffin section showing a highly cellular tumor and multiple multinucleated giant cells. (H/E ×400.) **(B)** Photomicrograph of paraffin section showing ovoid stromal cells and multinucleated giant cells. (H/E ×1000.)

As far as the rare cranial osteoclastomas are concerned, the sphenoid and the temporal bones appear to be the most common locations.[2,4,8,9] Occasional instances of orbital,[10] calvarial,[11] and occipital[12] giant cell tumors have been described. A giant cell tumor presenting as a mass occupying the middle fossa are extremely rare, and only two instances have been described up to date.[13]

These tumors are as a rule extradural and do not invade the intradural compartment. However, two of the cases reported in the literature,[13,14] one of which was located in the middle fossa as the present case,[13] showed a pattern of dural invasion and a tendency to infiltrate the brain parenchyma, a finding that was observed also in our patient.

Cranial osteoclastomas can give rise to variable symptoms obviously in accordance with their actual location. Temporal giant cell tumors may present with facial pain, hearing loss (as a rule of conductive type), and if extensive, facial paresis and symptoms related to its intracranial extension such as epilepsy and increased intracranial pressure (ICP).[6,12,15] Clinical presentation with conductive hearing loss may forviate the clinician and ultimately delay the diagnosis, as it occurred in previously described cases[13,16–19] and in our patient also.

As to ancillary diagnostic tests, skull X rays and angiography have represented the traditional investigations for cranial osteoclastomas.[4,6,9,10,13,14,20] The advent of modern neuroimaging technology has changed the traditional scenario in this as well as generally in all cranial lesions, and CT and magnetic resonance imaging (MRI) have maximally restricted the indications for diagnostic arteriography. The usual appearance of osteoclastoma on cranial CT scanning is that of an homogeneous hyperdense mass highly enhancing with contrast medium.[3,5,11,13,16,21–27]

Bony erosions are also as a rule definitely demonstrated by CT scan examination,[3,10,13] although sometimes the bone adjacent the lesion can appear to be hyperplastic.[3]

Our case appeared to be quite different, for two reasons: significant bony erosions were absent at the CT

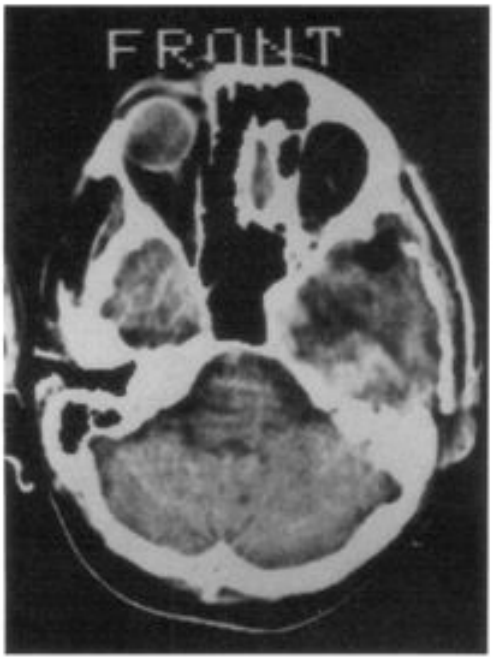

Figure 3. Postoperative CT scan shows a 3-cm blood clot in the surgical cavity. The surface of the petrous bone appears to be irregular as a result of surgical drilling.

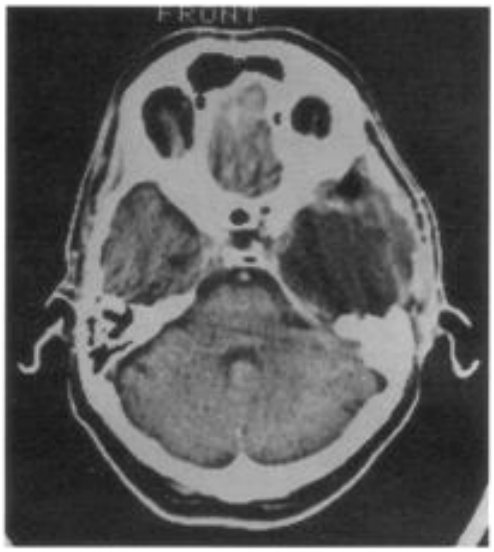

Figure 4. Fig. 4: Late postoperative CT scan. This examination shows a basal porencephalic area, which resulted from reoperation due to postoperative blood clot accumulation. No residual tumor is demonstrated.

scan, a fact that was confirmed at surgery; the lesion was hypodense except for its basal portion, and after injection of contrast it showed only a peripheral rim enhancement.

This previously undescribed CT appearance led to include another diagnostic possibility, that is, osteoclastoma, in the presence of an hypodense intracranial lesion showing peripheral "ring-like" enhancement.

Histologic diagnosis of giant cell tumor necessitates thoughtful evaluation. Differential diagnosis should include other bone-invading tumors such as chondrosarcoma and chordomas, and mostly nontumor diseases such as aneurysmal bone cyst, giant cell reparative granuloma, "Brown tumor" of hyperparathyroidism, and fibrous dysplasia.[3,6]

In the present case the lesion appeared to be highly cellular, with ovoid stroma cells surrounding multiple multinucleated giant cells. Hemosiderin was detected, however, clear blood-filled cavernous spaces were absent, a fact that excludes the possibility of an aneurysmal bone cyst. "Brown tumor" of hyperparathyroidism was easily excluded on an clinical basis. The absence of foci of osteoid and/or bone formation, as well as of osseous metaplasia, were considered reasonable arguments for considering the present lesion neither fibrous dysplasia, nor a giant cell reparative granuloma. Moreover, the absence of mitotic figures excluded the possibility of other, more aggressive bone-invading tumors. The final diagnosis was osteoclastoma (giant cell tumor) of the basal temporal bone.

The goal of surgical treatment of giant cell tumors is radical removal of the lesion. However, this is not always feasible when the lesion invades the base of the skull,[3,6,13] as it happened in the present case. The technique of skull base surgery, with section of the zygoma[7] and temporal bone drilling, can definitely help in facilitating the removal of a tumor located in the basal middle fossa.[13] However, surgical radicality is influenced also by other factors and extensive invasion of the basal bone has been considered a factor that would prevent radical lesion removal also when using an appropriate skull base approach.[28,29] In the present case we performed extensive drilling of the involved bone. However, because the surface of the petrous pyramid appeared to be quite abnormal, and reliable landmarks for identifying the carotid and facial canals could not be observed, bone drilling in the direction of the petrous carotid artery and the facial nerve was unavoidably limited.

Accordingly, we could not be certain of having eradicated the lesion. The patient awoke with facial paralysis, in spite of the fact that the facial canal was respected.

Not unexpectedly, the facial nerve significantly recovered its lost function in the following months. We believe that this temporary complication can be ascribed to the unavoidable tractions exerted on the greater superficial petrosal nerve during removal of the basal portion of the tumor.

Tumor vascularity represented a problem during surgery in our patient, and was a likely facilitating factor for the occurrence of the postoperative bleeding that prompted reoperation.