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Muhammad Waqas

Aga Khan University, muhammad.waqas@aku.edu

Naeem Sultan Ali

Aga Khan University

Muhammad Zubair Tahir

Aga Khan University

Syed Ather Enam

Aga Khan University

Zeeshan-ud-din

Aga Khan University

See next page for additional authors

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Muhammad Waqas, ., Ali, N. S., Tahir, M. Z., Enam, S. A., Zeeshan-ud-din, ., Ikram, M. (2012). Giant cell reparative granuloma of temporal bone: Case report of a 62-year-old male. *Journal of Cranio-Maxillary Diseases*, 1(2), 137-140.

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Authors

Muhammad Waqas, Naeem Sultan Ali, Muhammad Zubair Tahir, Syed Ather Enam, Zeeshan-ud-din, and Mubasher Ikram

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Case Report

Giant cell reparative granuloma of temporal bone: Case report of a 62-year-old male

Muhammad Waqas, Naeem Sultan Ali¹, Muhammad Zubair Tahir, Syed Ather Enam, Zeeshan-ud-din², Mubasher Ikram¹

Departments of Neurosurgery, ¹Otorhinolaryngology, and ²Pathology, Aga Khan University Hospital, Karachi, Pakistan

ABSTRACT

Giant cell reparative granuloma (GCRG) is an uncommon non-neoplastic reactive tumor which occurs almost exclusively within the mandible and maxilla. GCRG of the temporal bone is a rare condition. It has been found to affect predominantly adolescents and adults (age: 10-25 years). We report a case of a 62-year-old male with GCRG of left temporal bone who presented to us with progressive left temporal swelling for 3 months. It was associated with hearing loss. There was no history of trauma. A non-contrast computed tomography scan brain showed a locally destructive lesion involving squamous temporal bone closely related to the left temporal lobe and infratemporal fossa. Magnetic resonance imaging brain with contrast showed a hypointense lesion on T1 and with peripheral contrast enhancement after gadolinium injection. Patient underwent left temporal craniotomy with atticotomy, mastoidectomy, duraplasty, and opening of middle ear and temporomandibular joint. A bone graft was then taken from right iliac crest and used to repair the resulting defect. Final histopathology report confirmed GCRG. We discuss radiological and histopathological features of lesion in this case report.

Keywords: Benign lesions of skull, giant cell reparative granuloma, temporal bone

INTRODUCTION

Giant cell reparative granuloma (GCRG) is an uncommon non-neoplastic reactive tumor which occurs almost exclusively within the mandible and maxilla. Cases involving the skull are few. GCRG of the temporal bone is exceedingly rare with so far less than 20 cases reported in the international medical literature. It has been found to affect predominantly adolescents and adults (age: 10-25 years) with a male to female ratio of 1:2. Although regarded as a benign process, it can behave aggressively, and surgical excision has been advised whenever

possible.^[1] We report a case of a 62-year-old male with GCRG of left temporal bone.

CASE REPORT

A 62-year-old male presented to us with a history of progressive left temporal swelling for 3 months. His comorbidities included hypertension and obstructive sleep apnea. The swelling was associated with decreased hearing in left ear. He could not recall any history of trauma. On examination, there was sagging of left external auditory canal, with ipsilateral conductive hearing loss. Swelling was non-fluctuant, non-tender, and 4 × 3 cm in dimension. There was no facial palsy.

A non-contrast computed tomography (CT) scan brain [Figure 1] showed a locally destructive lesion involving squamous temporal bone closely related to the left temporal lobe and infratemporal fossa. Magnetic resonance imaging (MRI) brain

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Correspondence to:

Dr. Syed Ather Enam, Department of Neurosurgery, Aga Khan University Hospital, P.O. Box 3500, Stadium Road, Karachi 74800, Pakistan.
E-mail: ather.enam@aku.edu

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with contrast showed a hypointense lesion on T1 and with peripheral contrast enhancement after gadolinium injection [Figure 2a and b]. It showed extension up to temporal lobe causing extra-axial compression.

Inconclusive fine needle aspiration cytology was followed by an incisional biopsy which showed high suspicion of chronic GCRG. Patient underwent left temporal craniotomy with atticotomy, mastoidectomy, duraplasty, and opening of middle ear and temporomandibular joint. A bone graft was then taken from right iliac crest and used to repair the resulting defect. Intraoperatively, lesion was found to be soft avascular infiltrating temporalis, middle skull fossa, with intradural extension and extension

into infratemporal fossa. However, there was no involvement of brain parenchyma.

Post-operatively, patient remained well. There was no post-operative deficit and his left sided conductive loss improved. Histopathology report showed tissue containing sheets and aggregates of plump to oval cells having abundant eosinophilic cytoplasm and rounded nuclei with scattered multinucleated giant cells with hemosiderin-laden macrophages [Figure 3]. On immunohistochemistry, CD68 marker was also found positive [Figure 4]. Mitotic figures were occasional. Slides were reviewed with several histopathologists and diagnosis of central chronic GCRG was made.

At three months follow-up, MRI with contrast did not show any recurrence [Figure 5]. He complains of mild discomfort on opening up of jaw for which jaw exercises are advised.



Figure 1: A non-contrast computed tomography scan axial image showing a locally destructive temporal bone lesion

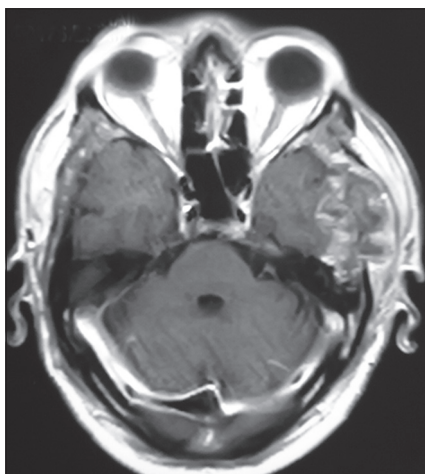


Figure 3: Post-contrast pre-operative Magnetic resonance axial image

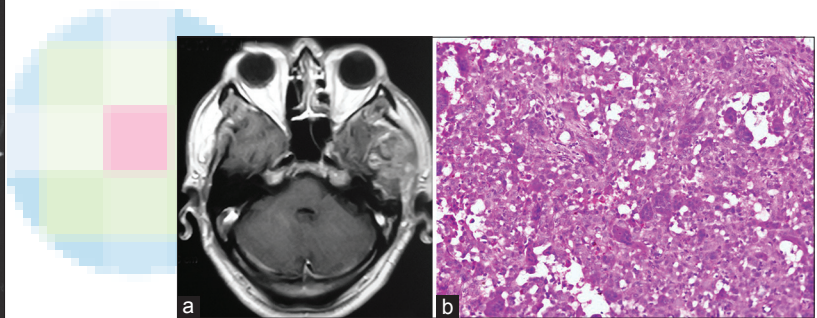


Figure 2: (a) Magnetic resonance imaging (MRI) brain with contrast showed a hypointense lesion on T1 and with peripheral contrast enhancement after gadolinium injection (b) H and E staining of tissue sample

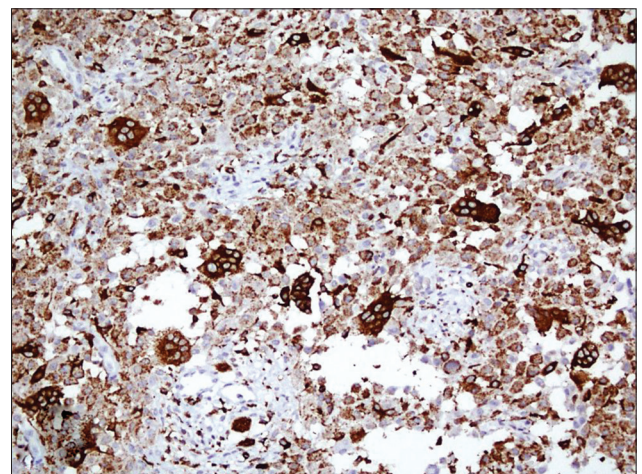


Figure 4: Immunohistochemistry for CD68 markers

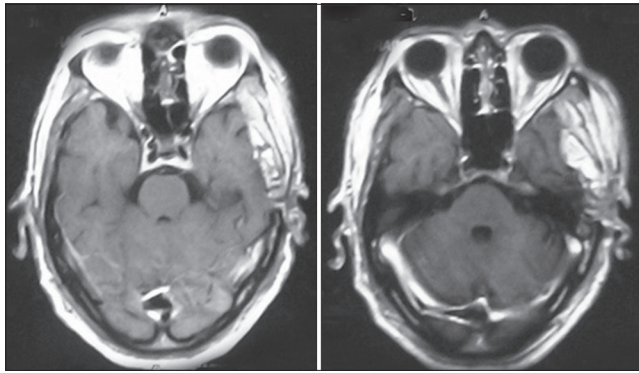


Figure 5: Post-operative magnetic resonance imaging axial images with contrast

DISCUSSION

GCRG is a rare benign granulomatous lesion, which is postulated to be a result of an exacerbated reparative process related to previous trauma and intraosseous hemorrhage. It is more frequently seen in mandibular or maxillary regions, ethmoid bone, sphenoid bone, and petrous temporal bone.^[1] As diagnosis of GCRG is challenging, the actual frequency of this lesion in the skull base is hard to determine. Less than 20 well-documented cases have been reported in the last 35 years.^[2] Adolescents and young adults (age: 10-25 years) are more prone with a male to female ratio of 1:2. In a few cases, a definitive history of trauma prior to the development of GCRG is noted but many authors consider a traumatic etiology unlikely.^[3] Clinical features depend on the site of the bony involvement and compromise of the nearby neurovascular structures. Initially localized pain, initially a localized pain, and then diffuse swelling followed 1-2 weeks later by a rapid progressive well localized swelling with or without the features of raised intracranial pressure and cerebral compression. Involvement of the ethmoid bone results in nasal symptoms, whereas sphenoid bone involvement produces orbital symptoms (mainly diplopia) and the petrous temporal bone involvement causes hearing deficits, vertigo, tinnitus, giddiness, and facial paresis.

Plain radiographs often show expansion of the bone with thinning of the cortex by a well-demarcated multiloculated radiolucent lesion. On CT, an iso-attenuation or a mixed attenuation osteolytic bone lesion with compression of the brain or orbital contents is usually visualized. The MRI scan shows an isointensity or a mixed intensity mass lesion on

the T1-weighted images with mild enhancement on intravenous administration of gadolinium, whereas the T2-weighted images show a non-homogeneous hypointense or isointense lesion.^[4]

Histological examination in GCRG shows the stroma mainly formed by the abundant spindle-shaped fibroblastic cells and small giant cells with a few nuclei around the hemorrhagic foci. There are no features suggestive of malignancy. In the differential diagnosis of GCRG, the other giant cell containing lesions, i.e., giant cell tumor of the bone, an aneurysmal bone cyst, and a brown tumor of the hyperparathyroidism should be considered. GCRGs have a very similar radiographic appearance to giant cell tumor (GCT). Differentiation between the two largely depends upon the histological location and shape of giant cells. The presence of osseous metaplasia favors a diagnosis of GCRG. The presence of hemosiderin pigment is thought to be one of the most important findings that differentiate GCRG from GCT because perivascular hemorrhage and hemosiderin deposition are believed to occur to a lesser extent in GCT than in GCRG.

Management of GCRG is mainly a microsurgical excision as with curettage alone, recurrence occurs in about 15% of cases. Malignancy and metastasis have not been reported in GCRG.^[3] In cases of incomplete surgical removal, post-operative radiation should be considered.^[1] Due to the complexity of vital structures at the skull base, complete surgical excision may be sometimes impossible in this anatomic region. Therefore, primary radiotherapy has been recommended when the GCRG is not amenable to surgery. However, radiotherapy is reported to be less effective and to carry the risk of sarcomatous induction.^[1] Because of potential recurrence and aggressive growth behavior, treatment strategies can be challenging and regular clinical and radiological follow-up investigations are indispensable.^[5]

There was no history of trauma in our patient. GCRG at this age has not been reported before. We also had a pre-operative incisional biopsy of the lesion which provided a very important hint as to the nature of lesion and management plan. To remove the disease process completely, temporomandibular joint (TMJ) had to be opened and this has led to mid-post-operative discomfort. Graft used from iliac crest helped to repair the defect created by excision

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of lesion. We could not find a lot of examples using bone graft in cases of excision of GCRG lesions.

CONCLUSION

GCRG is a rare entity thought to be related to trauma relatively more common in adolescents and young adults especially females. We describe its incidence in an elderly male with no history of trauma and involving more of squamous than petrous temporal bone.

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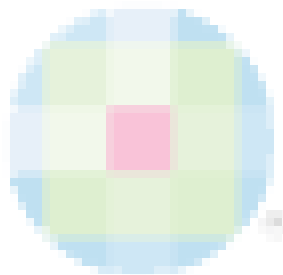
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How to cite this article: Waqas M, Ali NS, Tahir MZ, Enam SA, Z, Ikram M. Giant cell reparative granuloma of temporal bone: Case report of a 62-year-old male. *J Cranio Max Dis* 2012;1:137-40.

Source of Support: Nil. **Conflict of Interest:** None declared.

Submission: July 10, 2012, **Acceptance:** September 30, 2012



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