B-ENT, 2008, **4**, 45-48

Giant cell granuloma of the temporal bone: a case report

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Key-words. Giant cell granuloma; temporal bone; giant cell tumour

Abstract. *Giant cell granuloma of the temporal bone: a case report. Problem*: Giant cell granuloma (GCG) is a rare non-neoplastic bone lesion that occurs mostly in the jawbones; few cases arise in the remainder of the skull, including the temporal bone. Previously, giant cell lesions of the temporal bone were regarded as giant cell tumours (GCT). The importance of distinguishing GCG from GCT lies in the presumed difference in prognosis; GCTs have higher rates of recurrence, metastasis, and malignant transformation.
Methodology: We describe the case of a 12-year-old child with temporal bone GCG extending to the middle cranial fossa. The patient underwent a subtotal petrosectomy via retroauricular approach, associated with resection of the zygomatic process.
Results: No evidence of recurrence was found 36 months later.
Conclusion: The diagnosis of GCG was based on clinical history, histology, imaging, and response to treatment. The patient was treated with the standard surgical approach, and has a good outcome at three years follow-up.

Introduction

Giant cell "reparative" granuloma (GCG) was first reported as giant cell lesion of the jaw in 1953 by Jaffe.[1] The term reparative referred to its supposed post-traumatic origin[2-5]; now, exclusion of the term "reparative" is widely accepted.[6] GCG is a rare benign lesion that occurs mostly in the jawbones; few cases arise in the remainder of the skull, including the temporal bone.[6-9] Although the etiology and pathogenesis of GCG remain unclear, its histological and clinical features and clinical behaviour are now well established, helping to differentiate GCG from other giant cell lesions. Histologically, the lesion is composed of mononuclear stromal cells and multinucleated giant cells.[10] Although GCG is believed to be non-neoplastic, the lesion grows quite rapidly.

We describe a case of a 12-year-old child affected by GCG of the temporal bone extending to the middle cranial fossa. He presented with retro-auricular swelling (otomastoiditis-like) and polypoid mass filling the auditory external ear canal.

Case report

A 12-year-old child was admitted to our department in April 2004 complaining of otalgia of the right ear, retroauricular "otomastoiditis-like" swelling, and otorrhoea. Two months prior, he suffered an episode of right-side acute otitis media that was treated with antibiotics, followed by persistent otorrhoea and progressive development of an external ear canal mass. On examination, a painful fluctuant retroauricular mass measuring approximately 4 cm in diameter, and a polypoid mass narrowing the external auditory canal were found. Pure-tone audiometry showed conductive right-side hearing loss. Laboratory examination showed normal levels of calcium, alkaline phosphates, and inorganic phosphorus. Other neurological tests and general examination were normal.

Computed tomography (CT) revealed an osteolytic expansive lesion involving the mastoid, the middle ear, and the external auditory canal; it reached the inferior parotid gland, the lateral part of the masseteric muscle, and enveloped the mandibular condyle (Figure 1). On magnetic resonance imaging (MRI), the mass was isointense to the brain on T1- and hypointense on T2-weighted images, and was strongly enhanced after contrast. The mass had eroded the roof of the anterior segment of the mastoid cavity, coming into contact and thickening the dura overlying the glenoid cavity (Figures 2-4).

The patient underwent a subtotal petrosectomy via a retroauricular approach, associated with resection of the zygomatic process. The lesion was completely excised with a "safe" margin of uninvolved bone, the roof of the

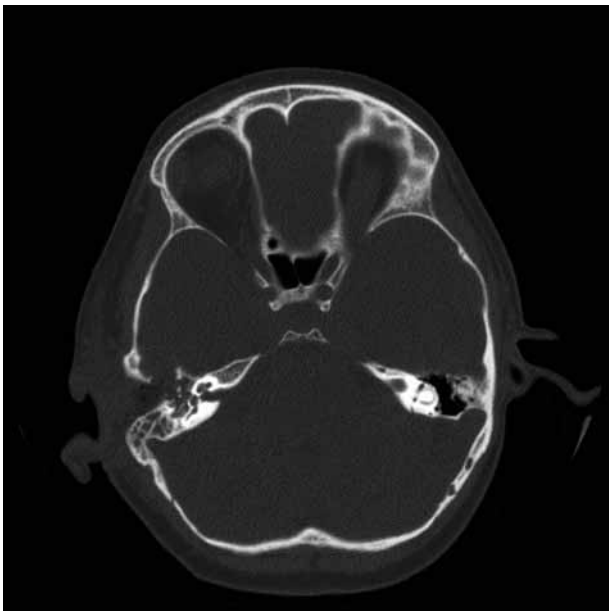

Figure 1
Axial CT. Erosion of the anterior wall of the tympanic cavity

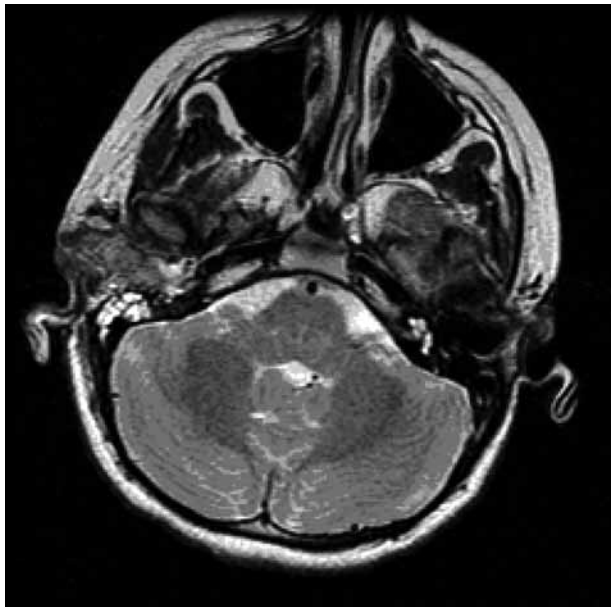

Figure 2
MR-axial T2. Hypo-intense lesion

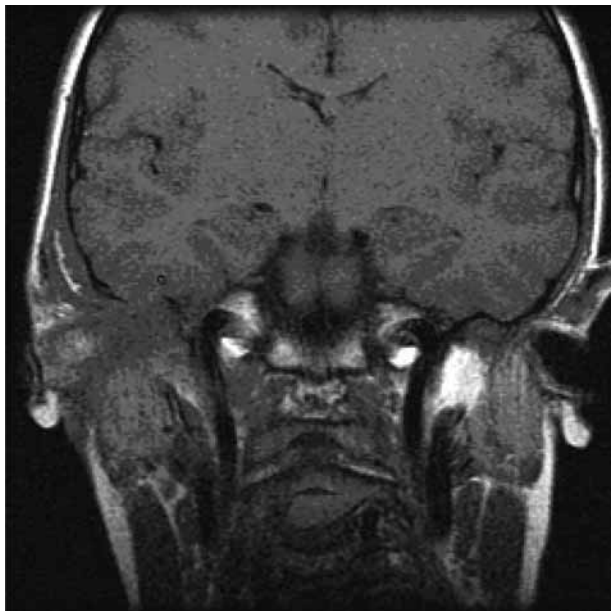

Figure 3
MR-coronal T1. Isontense erosive lesion

mastoid cavity was repaired with an organic plaque, and the external auditory canal was closed.

Histological examinations revealed reticular and fibrillar connective tissue stroma containing a large number of spindle-shaped giant cells, hemorrhage, and newly formed bone. The final diagnosis was GCG.

The postoperative period was uneventful. The patient was discharged, symptom-free, after of 10 days. His postoperative CT and MRI showed a radical excision of the lesion; no evidence of recurrence was found 36 months later.

Hearing was normal on the unaffected side, while conductive hearing loss of 60 dB was present on the operated side, with a normal bone threshold. The patient underwent a trial with a "BAHA soft band"® for 10 days, but refused definitive application of the device for aesthetic reasons.

Discussion

Before their description, almost all giant cell lesions of the temporal bone were regarded as giant cell tumours (GCT). The importance of distinguishing GCG from GCT lies in the presumed difference in prognosis; GCT is associated with a higher rate of recurrence, metastasis, and malignant transformation.[11] GCG occurs predominantly in the jaw and GCT in the epiphyses of the long bones. However, both of these lesions are rare in the bones of the

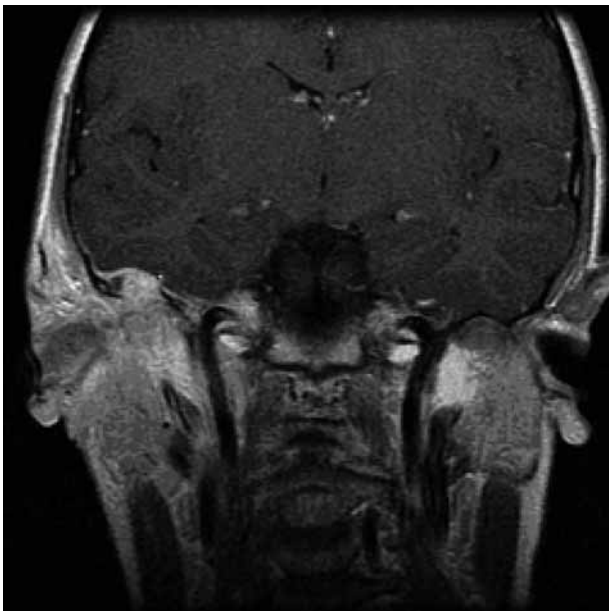

Figure 4
MR-coronal T1 after contrast. Strong lesional enhancement and dural thickening.

skull. Temporal bone GCG can occur at any age between 4 months and 72 years.[12] Some authors believe that GCG occurs most frequently in children or young adults 10-30 years of age,[7] with a female predilection.[1,13] However, other authors have not found any gender predilection.[14]

The etiopathogenesis of GCG has been debated since its first description by Jaffe[1], who considered the lesion to represent a local reparative process following traumatic intraosseus hemorrhage or periosteal reaction. In later descriptions, the presence of a traumatic event was inconsistent and the hypothesis of traumatic origin was rejected. More recently, it has been demonstrated that cell cycle-associated proteins such as MDM2, Ki-67, and PCNA are expressed in both of these lesions, although the percentage of Ki-67- and PCNA-positive cells is higher in GCG. Thus, GCGs seem to have higher proliferative activity than GCTs do.[15]

The diagnosis of GCG is made by the association of clinical history, histological features, radiological imaging, and response to treatment.[2,9,16] The clinical features vary according to the site of origin of the lesion; in the temporal bone, GCG generally causes sensory-neural or conductive hearing loss, vertigo, tinnitus, headache, facial nerve impairment, a palpable mass, and otitis-like symptoms.[4]

GCG often appears as an osteolytic lesion on CT images. On MRI, it is most frequently hypointense on both T1- and T2-weighted images and enhances after contrast injection.[17-19] Macroscopically, the lesion appears yellow-brown in color, soft and friable on a cut section.[1,8] Differential diagnosis of GCG, apart from GCT, can include brown tumour in hyperparathyroidism, aneurismal bone cyst, benign chondroblastoma, cherubism, fibrous dysplasia, osteogenic fibroma, and osteosarcoma.[1,2,8,16,20,21] GCG and GCT are histologically very similar. Nevertheless, in GCT, the giant cells have a greater number of nuclei and the giant cells resemble the stromal cells, whereas in GCG, the osteoclasts and the stromal cells of the fibroblastic type are distinctly different. Moreover, in GCG the giant cells cluster around areas of hemorrhage and necrosis while the stroma is relatively acellular with extensive osseous metaplasia.[11]

Treatment of GCG involves surgical removal, radiotherapy, and calcitonin. Complete surgical excision is considered the most effective treatment for GCG. If radical removal is difficult, as it is when the lesion is localized in the skull base, the mass could be targeted with multimodal therapy including partial resection and low total-dose high voltage radiotherapy.[8,16,22]

Treating GCG and GCT with calcitonin and osteoprotegrin is the topic of some interest: positive results are probably due to control of osteoclastogenesis via inhibition of the calcitonin receptor.[23] Because GCG tends to recur, even at a low rate, long-term follow-up is essential.

Our patient is free of disease after 36 months of follow-up, as demonstrated by contrast enhanced MRI. His bone conduction threshold on the operated side was normal, but he refused BAHA® application. He is a teenager who is concerned about his appearance, and considering that he reports no significant difficulties in the classroom, we decided not to press this issue.

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