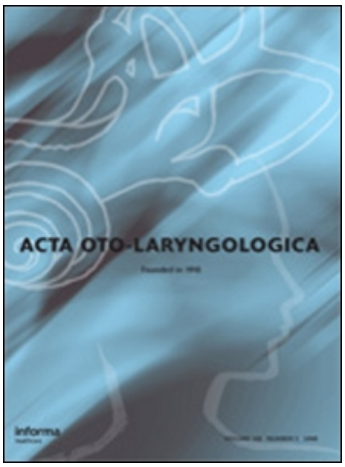

Acta Oto-Laryngologica
Publication details, including instructions for authors and subscription information:
http://www.informaworld.com/smpp/title~content=t713690940

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Online Publication Date: 01 July 2006

To cite this Article Marioni, Gino, Marchese-Ragona, Rosario, Guarda-Nardini, Luca, Stramare, Roberto, Tognazza, Elia, Marino, Filippo and Staffieri, Alberto(2006)'Giant cell tumour (central giant cell lesion) of the maxilla',Acta Oto-Laryngologica,126:7,779 — 781
To link to this Article: DOI: 10.1080/00016480500504200
URL: http://dx.doi.org/10.1080/00016480500504200

Acta Oto-Laryngologica, 2006; 126: 779–781

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CASE REPORT

Giant cell tumour (central giant cell lesion) of the maxilla

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Abstract
The giant cell tumour (GCT) is a benign, locally invasive lesion that accounts for about 20% of benign bone tumours. Approximately 2% of all GCTs arise in the head and neck region. Giant cell lesions in the craniofacial skeleton other than the jaws are uncommon; the majority of them occur in the sphenoid, ethmoid and temporal bones. GCT of the maxilla has seldom been described. We present the case of an 83-year-old patient with an advanced GCT of the left maxilla who underwent en bloc resection through maxillectomy. Reconstruction of the orbitary frame and maxilla was performed with autologous calvaria and a temporalis muscle pedicled flap. Our successful maxillary reconstruction based on the association between autologous calvarial bone sticks bent with titanium miniplates and a temporalis muscle pedicled flap allowed the involvement of only one donor area for both hard and soft tissues. At 1-year follow-up, our patient showed no evidence of recurrent GCT, with satisfactory aesthetic results.

Keywords: *Giant cell tumour, central giant cell lesion, maxilla, reconstruction, autologous*

Introduction

The giant cell tumour (GCT) is a benign, locally invasive lesion. GCTs account for about 5% of all primary bone tumours and about 20% of benign bone tumours. The epiphyses of long bones, especially the distant femur, proximal tibia and distant radius, are the most common sites. The sacrum is the most common site for a GCT involving flat bones.

Approximately 2% of all GCTs arise in the head and neck region, $<1\%$ in the skull [1,2]. Giant cell lesions in the craniofacial skeleton other than the jaws are uncommon; the majority of them occur in the sphenoid, ethmoid and temporal bones [1]. A review of the literature showed that GCT of the maxilla has been seldom encountered [3].

We present a case of GCT of the maxilla and briefly discuss the associated diagnostic, surgical and reconstructive problems.

Case report

In January 2003, an 83-year-old caucasian male patient came to the Department of Otolaryngology-Head and Neck Surgery of Padova University because of a 3-year history of a slowly enlarging left maxillary swelling. There was no history of maxillary trauma; the patient worked in the past as a varnisher. The patient reported nasal obstruction and a feeling of left orbital compression without nasal discharge, epistaxis, pain, diplopia or loosening of maxillary teeth.

Physical examination revealed a left maxillary swelling with displacement of the ipsilateral orbit. The rhinoscopic examination showed a right deflexion of nasal septum; the left nasal fossa was occupied by an esophitic lesion and serous secretions. The oral examination showed a significant hard palate swelling. The objective examination of nasopharynx, larynx and ears was normal. No cervical lymph node enlargement was seen. Biopsies were performed

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(Received 28 October 2005; accepted 30 November 2005)

ISSN 0001-6489 print/ISSN 1651-2551 online
DOI: 10.1080/00016480500504200

under video-rhinoscopic control (0° rigid endoscope) and through a hard palate mucosa incision. Histopathological evaluation of biopsy specimens revealed a giant cell lesion.

Computed tomography (CT) showed an expansive lesion of the anterior left maxilla, completely filling the maxillary sinus and extending superiorly beyond the floor of the ipsilateral orbit and inferiorly to the hard palate (Figure 1A, B). Haematological investigations showed normal serum calcium levels (2.25 mmol/L; normal values 2.10–2.55 mmol/L). A parathyroid 99mTc-MIBI scintigraphy showed a significant capitation of the maxillary lesion without evidence of parathyroid adenoma or multiple gland hyperplasia.

The patient underwent left maxillectomy through a para-lateral nasal access (Weber-Fergusson modified incision). After an en bloc radical lesion resection, the reconstruction of the orbitary frame and the maxillary sinus anterior wall was carried out with autologous calvaria remodelled sticks bent with titanium miniplates (Figure 1D, E). The reconstruction of hard palate dehiscence was carried out with a temporalis muscle pedicled flap.

The final pathological evaluation on permanent sections revealed both large multinuclear giant cells and mononuclear spindle-shaped stromal cells (Figure 1C). Thin-walled vessels and foci of haemorrhage were also evident. The pathologist reached a diagnosis of GCT (central giant cell lesion).

The postoperative course was regular without the occurrence of diplopia. Serial clinical and radiological follow-up controls were planned (Figure 1F). At the last follow-up, 1 year after the intervention, the patient showed no evidence of recurrent disease. The aesthetic results were also satisfactory (Figure 1G).

Discussion

The stroma of most GCTs is vascular and contains numerous thin-walled capillaries, often with small areas of haemorrhage. These lesions may be associated with secondary aneurysmal bone cyst formation but also contain solid areas with the typical histological appearance of GCT. The pathologic differential diagnosis of GCT is extensive, including giant cell reparative granuloma, brown tumour of hyperparathyroidism, osteoblastoma, chondroblastoma, aneurysmal bone cyst, non-ossifying fibroma, foreign body reaction and osteosarcoma with abundant giant cells. These lesions can be difficult to distinguish from one another, particularly at fine-needle aspiration or with frozen section specimens, emphasizing the need for careful and thorough clinical, pathological and radiological correlation. This is particularly true of brown tumour of hyperparathyroidism, which can be indistinguishable from GCT at pathological analysis. Laboratory analysis should be performed to exclude this possibility in all cases. In our case calcium level determination and a parathyroid 99mTc-MIBI scintigraphy ruled out the presence of a parathyroid adenoma or multiple gland hyperplasia.

CT scan can provide a detailed assessment of maxillary CGT, showing the soft tissue mass of the lesion, cortical perforation, amount of bony destruction and extension toward important adjacent anatomic structures, such as orbit and cranial base, that may not be clearly shown by conventional radiography. CT scanning is invaluable to surgical planning and management. Magnetic resonance

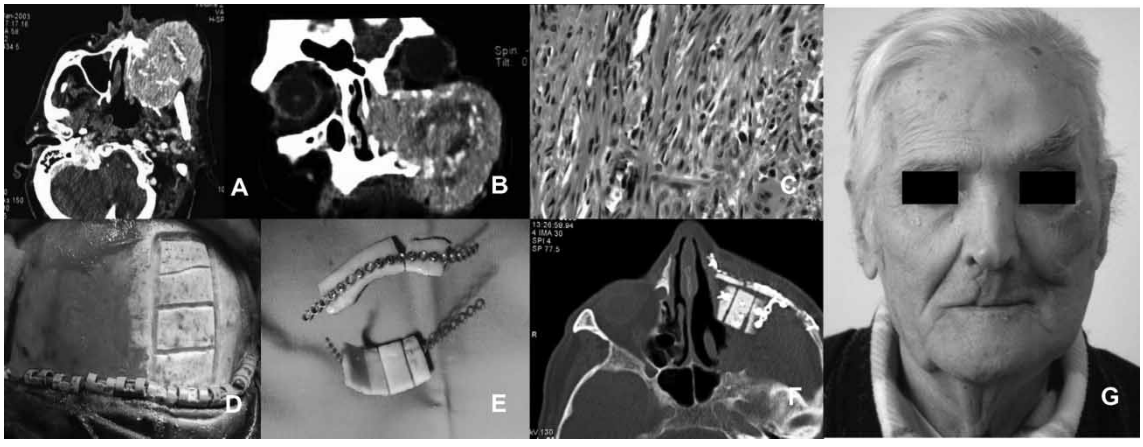

Figure 1. (A and B) Preoperative CT scan: axial and coronal views showing an expansive lesion of the anterior left maxilla involving the maxillary sinus and extending superiorly beyond the floor of the orbit and inferiorly to the hard palate. (C) Maxillary giant cell tumour (H&E, ×20). (D) Intraoperative preparation of calvaria with a micro-drill. (E) Calvaria remodelled sticks bent with titanium mini-plates. (F) Four-month postoperative CT scan control, axial view. (G) Postoperative aesthetic results (1-year follow up).

imaging (MRI) is superior to CT in delineating soft-tissue tumour extent because of its improved contrast resolution. On the other hand, the solid components of GCT demonstrate low to intermediate signal intensity on T2-weighted MRI. Bone scintigraphy shows increased radionuclide uptake in the majority of GCTs.

Although the type of primary surgical removal of GCT is the most significant factor in disease recurrence, a relation also exists between recurrence rates and interruption of the cortex and soft tissue extension. In the maxilla, the cortical plates are thin and may be invaded by the GCT at an early stage.

From a review of the available literature, surgical excision appears to be the treatment of choice for maxillary GCT. Regardless of the site of presentation, marginal resection or curettage is associated with a high GCT recurrence rate (40–60%). Wide radical resection shows a reduced recurrence rate (7%) [4]. After extended GCT resection, reconstruction with allografts has been described. The advantage of using autologous tissue is relatively rapid incorporation with a lower risk of infection (especially with vascularized grafts). We describe a successful maxillary reconstruction based on the association between autologous calvarial bone sticks bent with titanium miniplates and a temporalis muscle pedicled flap. With this reconstructive approach there was only one donor area for both hard and soft tissues. Because of the risk of sarcomatous transformation, radiation therapy is generally avoided or reserved for GCTs that are considered inoperable.

According to the literature, GCT recurrences usually occur within the first 3 years after primary treatment (80–90% of the cases). Consequently, patients should be evaluated at 4-month intervals for the first 2 years and at 6-month intervals thereafter up to 5 years. Long-term follow-up of GCT is mandatory because late distant metastases have been also reported.

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