Case Report

A new case of Giant Cell ‘Reparative’ Granuloma of the temporal bone related to trauma

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ABSTRACT

Giant Cell Reparative Granuloma (GCRG) is an uncommon benign non-neoplastic lesion that most commonly affects the mandible and maxilla. Only sporadic cases involving the skull base have been reported. The etiology of GCRG is uncertain but may be related to trauma. The origin of ‘reparative’ in the name comes from these lesions appearing after bone trauma. We present a new case of GCRG in the temporal bone of possible posttraumatic origin. To distinguish this type of lesions from true giant cell tumours of bone, from brown tumours, from inflammatory lesions or metastatic lesions, histologic examination is required. The true giant cell tumours of bone are the most important to diagnose as these are generally considered to have a prognosis that is worse than GCRG and require adjunctive radiotherapy in addition to surgical excision.

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1. Introduction

Jaffe described Giant Cell Reparative Granuloma (GCRG) first in 1953 [1].

The first case of GCRG in the temporal bone was reported in 1974 by Hirschl and Katz [2]. Although GCRG is considered a benign lesion of bone, it is locally aggressive and requires surgical excision. If resection of GCRG is incomplete, post-operative radiotherapy can be given.

Our case represents a new finding of a GCRG of posttraumatic origin.

2. Case report

In 2010, a 26-year-old man was hospitalised with a craniocerebral trauma due to impact on the right side of his skull. This resulted in a commotio cerebri and a fracture of the zygomatic arch (Fig. 1).

No therapy for the zygoma fracture was needed.

In 2013, he returned because of a painless swelling in the right preauricular region and fullness in that ear.

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Fig. 1. 2010: zygomatic arch fracture.
Visual inspection of the right eardrum was impossible because the swelling blocked his right meatus.

2.1. Radiologic findings (Fig. 2)

CT showed an enhancing mass in the right temporal fossa, with lytic aspect of the temporal bone, intracranial extensions and erosion of the anterior part of the os petrosum.

Magnetic Resonance Imaging (MRI) demonstrated an anterior temporal fossa extradural mass that was encapsulated and multilobular with hypo-intense signal on T2-weighted imaging. No cystic changes were present. On T1-weighted imaging the lesion is hypo-intense, with homogenous enhancement, and expansion in the adjacent soft tissue.

The mass was completely resected with preserved hearing. No adjuvant treatment was given.

Follow-up MRI in 2016 showed no signs of relapse.

2.2. Pathological findings (Fig. 3)

Histology revealed a nodular cellular lesion composed of numerous giant cells mixed up with non-atypical spindle shaped mononuclear
cells and numerous iron-loaded macrophages (siderophages). There were no mitotic figures and no nuclear atypia.

The cells revealed diffuse and strong immunoreactivity for CD68 and p16, confirming monocytic–histiocytic origin differentiation.

The differential diagnosis with a true giant cell tumour was retained. The absence of any tendency to regular distribution of the giant cells of uniformity among stromal cells are arguments that favour pro a giant cell reparative granuloma. In giant cell reparative granulomas, the stromal-cell nuclei also tend to be more elongated with tapered ends than in true giant cell tumours [3].

The complete morphologic and immunohistochemical findings and the clinical presentation favoured pro the histologic diagnosis of a reactive, reparative process than pro a true osseous neoplasm as a giant cell tumour is.

3. Discussion

The GCRGs were described by Jaffe [1] as benign processes limited to the mandible of maxilla and may be related to trauma and intraosseous haemorrhage.

The GCRGs have since been reported to occur in other bones [4,5].

The pathogenesis of GCRG is controversial.

Since the first GCRG of the temporal bone was published in 1974, several others have been mentioned.

No other cases of GCRG in the temporal bone have been reported in Belgium since.

In most cases, the patients are less than 35 years old. The most common presenting symptoms are hearing loss, a mass, pain, facial paralysis and tinnitus. These symptoms were also present in our patient.

Differential diagnosis of GCRGs includes other giant cell lesions, such as true giant cell tumours of bone, brown tumours of hyperparathyroidism, enchondromas or chronic inflammatory processes.

Because of their more aggressive behaviour, the most important lesions to distinguish from GCRGs are true giant cell tumours of bone. Compared to giant cell tumours GCRGs more often have foci of osteoid, haemorrhage, hemosiderin or fibrosis.

The trauma in 2010, with zygomatic arch fracture and histologic examination with numerous iron-overloaded macrophages support the reparative character of the lesion.

The absence of relapse after resection only supports the benign origin of this tumour.

4. Conclusion

This case illustrates again that GCRGs can have a posttraumatic origin.

GCRG is an important diagnosis to consider in patients with a posttraumatic bone lesion especially in unusual areas.

Conflicts of interest

There are no conflicts of interest.

References


Fig. 3. (A) H&E staining, ×16 shows the lesion on the right side, well delineated from fibrous stroma and striated muscle on the left. The tumour contains abundant brown pigment. (B) H&E staining, ×50 shows a cellular lesion. Scattered multinucleated giant cells can be appreciated. (C) H&E staining, ×200: numerous giant cell with variable size are seen. (D) H&E staining, ×400 demonstrates the cellular detail and presence of iron pigment in the tumour cells. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)