The Giant Cell Granuloma about a Case
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DOI: 10.21276/sjmcr.2019.7.8.27 | Received: 20.08.2019 | Accepted: 27.08.2019 | Published: 30.08.2019

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Abstract

Giant cell granuloma (GCG) is an infrequent benign lesion of the jawbone that sometimes exhibits aggressive behavior. The main locations are the maxillae, especially the mandible, with some extragathic bone sites, more frequently in women with very variable radiological aspects, however, the diagnosis will be made by histological examination. We report the case of a 78-year-old patient with giant-cell granuloma who was slow-moving and who underwent surgical excision + curettage. He currently available bibliographic data concerning the various possible GCG treatments show that curettage surgery, whether or not associated with larger excision, is the conventional treatment that causes the least recurrence.

Keywords: Giant cell granuloma, radiological aspects, bibliographic data.

INTRODUCTION

The giant cell granuloma (GCG) called gingival giant epulis is an infrequent benign lesion of the jaws, sometimes with aggressive behavior. It was originally described by Jaffe in 1953 as a local repair reaction.

The main locations are the maxillae, especially the maxilla, sometimes with bone sites, especially the small distal bones (metacarpus, phalanges, metatarsals), but all localizations are possible.

This lesion occurs more frequently in women. There are very variable radiological features, however, the diagnosis will be made only by histological examination [1].

The case reported relates to a 78-year-old patient with a giant-cell repair granuloma of slow progression who was treated with surgical resection.

OBSERVATION

78-year-old patient, with no significant pathological history who consulted for oral swelling, progressing for 6 months coinciding with multiple dental extractions. This swelling progressively increased in volume with episodes of oral bleeding, the examination revealed the presence of a retro molar mass facing tooth 17, fleshy rounded, purplish, 1.5 cm in diameter, and firm consistency without bleeding in contact with a paramedian granulomatous maxillary gingiva (Picture-1).

Nasofibroscopy did not indicate a suspicious lesion, and the cervical examination did not note palpable lymphadenopathy.

The CT scan (Picture-2) showed a rounded formation, measuring about 13 mm, located at the back of the tooth 17, with contrast enhancement. Its contours are clear, without bone lysis. Can evoke an arterial angioma.

The angio- MRI returned to a fleshy lesion or venous angioma rather than arterial angioma. A surgical biopsy under general anesthesia suspected a repair granuloma giant cells maxillary.

A biological assessment including an exploration of the blood count, an enzymological assay of LDH (Lactate Dehydrogenase) normal value, a calcium phosphate biological assessment made it possible to eliminate a brown tumor of the maxillae secondary to hyperparathyroidism.

The patient underwent resection of the tumor under general anesthesia, endo-buccal, which was haemorrhagic and friable, throughout the excision phase. With a complete curettage of the entire colonized bone remodeled, of consistency "wetsugar".
Because of its implantation in the tumor, the tooth 17 is extracted. The completeness of the excision is controlled by a 30° opticsinusoscopy. The sinus was uninjured, with reconstruction by a fammflap (Picture-3).

Anatomo-pathological examination confirmed the diagnosis of repair granuloma with giant cells of the maxillary, high light ingfasciculateranges of fusiform cells fairly regularly accompanied by collagenous dots strewn with numerous multinucleated giant cells.

On this background, there are some plaques of hemorrhagic suffusion with siderophages, without mitotic or nuclear abnormalities. The post operative course was simple with weaning of the pedicled flap at 2 months.

The follow-up of our patient did not note a recurrence on a follow-up of 6 months. With good viability and trophicity of the flap (Picture-4).
**DISCUSSION**

GCG is a benign tumor, which is part of giant cell tumors and pseudo tumors, still poorly understood, of unknown etiology and very variable evolution.

According to the WHO classification, tumors and pseudo tumors are part of a polymorphous tumor group of non odontogenic origin that includes all conjunctival maxillary tumors [4].

Thus Giant Cell Tumors are divided into 3 majorentities:

- Cherubism or cystic disease multilocular family of maxillary,
- The GCG theaneurysmal cyst.

To these three entities we add 2 other types of lesions [4]:

- The true tumor with giant cell,
- The brown tumors of hyperparathyroidism.

The GCG occurs more frequently in women than in men with a location twice as common in the mandible as in the maxilla. In men, the peak incidence is between 10 and 14 years, while in women it is found between 15 and 19 years [5].

In our case it is an elderly male subject with a localization at the level of the maxillary upper. Rare cases are associated with genetic abnormalities such as Cherubism, Noonan Syndrome, or Neurofibromatosis Type 1.

The etiology of GCG is currently poorly specified but the hypothesis of post traumatic intraosseous hemorrhages is the most plausible. In most cases, the characteristic macroscopic appearance is a fleshy, reddish brown mass, resembling a splenic pulp [3].

Radiologically, appearance ranging from a small unilocular lesion to a large multilocular lesion associated with dental displacements, root resorptions or perforations of the cortical bone [6]. In our case the appearance was suggestive of a rather vascular lesion.

The peculiarity of this lesion lies in the difficulty of its clinical and radiographic diagnosis, soonly a biopsy will make it possible to establish the diagnosis and eliminate the suspicion of any other giant cell lesion (brown tumor of the maxilla secondary to hyperparathyroidism, a true tumor giant cells, cherubism).

Currently the conventional treatment of GCG is surgical and consists of curettage associated or not with an excision. The recidivism rate ranges from 11% to 35% [9].

Our observation confirms the superiority of surgery to limit recurrence although our decline is insufficient. A 2005 Lange et al., [9] study in Germany of 80 patients with GCG (16 aggressive cases and 64 non-aggressive cases) and all treated with curettage, reported a recurrence in 26.3% of patients, in a period of 0 to 10 years [10].

The Kruse-Losler study of 2006, involving 26 patients (10 aggressive cases and 16 non-aggressive cases) treated by curettage and surgical resection, reported a recurrence of 11.5% within 9 months to 12 years [10].

Such curettage associated or not with an excision, may require the avulsion of the teeth opposite or increase the risk of injury of the surrounding anatomical elements. As such, other less invasive therapies have been proposed in the treatment of GCG.

Some authors have reported favorable results with intra-lesional administration of corticosteroids [11-13]. However, currently, no controlled clinical study is published concerning this therapy. Other authors have also demonstrated the favorable role of calcitonin in the treatment of GRCG [10, 14, 15].

Interferon α, administered as monotherapy in the treatment of aggressive GCG, also seems capable of interrupting the growth of lesions, sometimes of reducing their size. It is often necessary to add a surgical procedure to eliminate the lesion [10].

In this case, it is difficult to attribute the share of each therapeutic in the overall success of the treatment. Recently, a cytokine called RANKL, strongly implicated in the activity of osteoclasts including giant cells, has been found in GCG [16]. A cytokine which is inhibited by osteoprotegerin, which could then limit the extension of the tumor. Finally, Imatinib (Gleevec, Novartis Pharma, Basel, Switzerland) a protein tyrosine kinase inhibitor of myeloid leukemia and gastro intestinal tumors [17]. Preliminary results of GCGG treatment with Imatinib are promising [18]. Radiotherapy is reserved for recurrent or unresectable cases [7].

**CONCLUSION**

GCG is an infrequent tumor in daily practice. Its rarity leads to a difficulty of diagnosis, which calls for a rigorous approach requiring not only careful interrogation but also careful clinical examination, adapted imaging, a targeted biological assessment and an efficient anatomopathological analysis.

The currently available bibliographic data concerning the various possible GCG treatments show that curettage surgery, whether or not associated with larger excision, is the conventional treatment that causes the least recurrence.
Finally, it is necessary to insist on the interest of the early detection and the knowledge of the diagnostic elements of this type of tumors, often benign, but whose destructive consequences are disabling.

RÉFÉRENCES


