

Prosthetic upper jaw restoration with zygomatic and conventional implants in a patient with a giant cell lesion

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Abstract

The rehabilitation choice of patients undergoing resective surgery until some time ago was that of a removable prosthesis.

With the introduction of implants and digitized programming, it is possible to perform previsualization of the surgical treatment and program all the various rehabilitation prosthetic phases.

The authors present a case of a patient suffering from a giant cell lesion whose upper right jaw was surgically removed and then prosthetically rehabilitated.

So, the only possibility we have of reducing the invasiveness of surgical treatments is to make an early diagnosis.

Key words: prosthetic restoration, zygomatic implant, giant cell lesion.

Introduction

Giant cell lesions of the maxilla and paranasal sinuses represent a rare, locally aggressive disorder that presents as a soft tissue mass with distinct histologic and clinical features. Giant cell injury is a locally aggressive benign primary bone neoplasm. It was first described by Sir Ashley Cooper in 1818. It accounts for 5-15% of all benign bone tumors. It most often affects women between the 2nd and 3rd decades of life. 1 to 3% occur in children under 14 years of age.

They are located in the epiphyses and metaphyses of the long bones (knee, femur, tibia, radius) (1).

In the maxillofacial area, they are very rare and are found, in order of frequency, in the mandible, upper jaw, skull base, and mandibular condyle. In these cases, it is necessary to rule out a disease associated with Paget's (2-4). It represents the most common lesion associated with secondary aneurysmal bone cysts (39%). Inflammatory, angiogenic, and osteoclastic, although none of them is demonstrated. The role played



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by the p 53 suppressor gene in its genesis has recently been confirmed (5).

It usually manifests as a single, asymptomatic, or painful tumor with rapid expansion that generally settles in the vestibular sulcus. It can cause displacement and/or loss of teeth, oral opening limitations, pathological fractures (11-37%), or paresthesias.

Other times, it manifests as incidental radiolucency on a conventional radiological examination. Radiologically, it manifests as a multilocular radiolucent image in "soap bubbles," similar to that described for ameloblastoma (7).

Less commonly, it presents as a unilocular image. Sometimes, it causes an expansion and thinning of the cortical with sclerotic edging and root resorption. In half of the cases, the bone cortex is destroyed.⁶ Angiographic studies are beneficial, showing a hypervascular lesion in most cases (60-65%), although there are hypovascular (26-30%) or even avascular (10%) cases (8).

The lesion is not a granuloma in the strict histologic sense, and clinically, it is not reparative, often demonstrating neoplastic features (9).

Because there is evidence that the often-cited differences between giant cell granuloma and giant cell tumor are not as well-defined as commonly believed, we prefer to use the noncommittal term "giant cell lesion." (10)

Likely, giant cell "granuloma" of the jaws and giant cell tumors of other bones represent a continuum of a single disease process rather than being completely separate entities (11).

The tomograph showed a well-defined radiolucent area that extended from the right lateral incisor to the left second molar region in the maxillary (Figure 2).

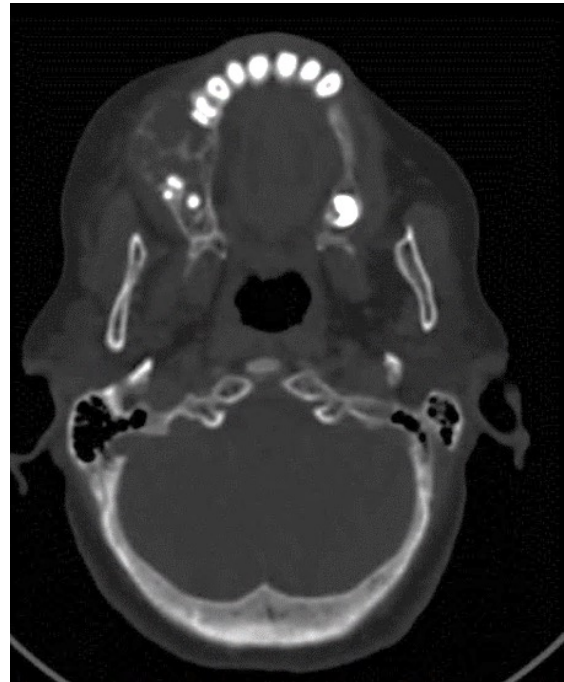


Figure 2

Case reports

A 52-year-old woman presented to the Department of Maxillofacial of the National Autonomous University of Mexico with a painless swelling on the right side of the face for six months. (Figure 1) The swelling had an insidious onset and progressed slowly. There was no history of any loosening of teeth. There was no history of trauma to the face five to six years ago. There were no facial paraesthesias, nasal discharge, epiphora, or systemic symptoms. Medical history and family history were non-contributory.



Figure 1.

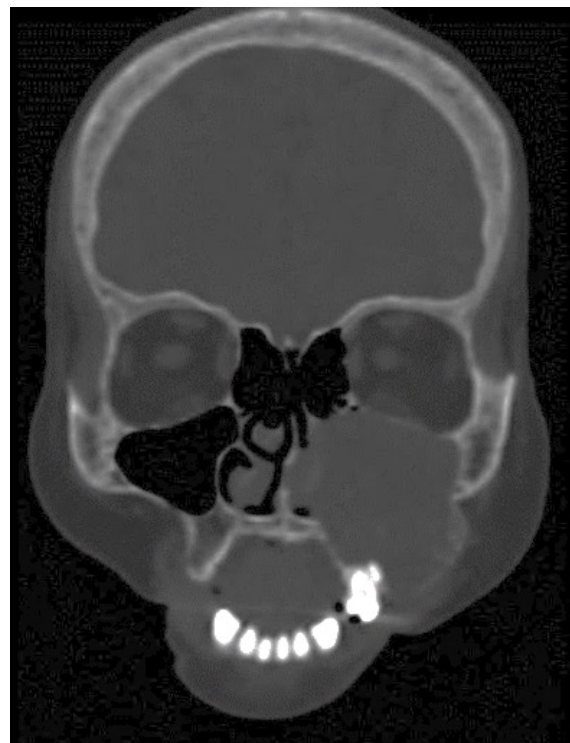


Figure 3



Figure 4.

A maxillary tomogram showed the absence of the bony hard palate on the right side with an associated soft tissue mass extending into the nasal area; this image infiltrates the medial wall of the nostrils, where it infiltrates the nasal septum, middle and inferior turbinates, respecting the floor of the orbit, in the axial section can be seen an image with mixed areas in the retromolar area of the maxilla with Hypodense predominance compared to bone tissue.

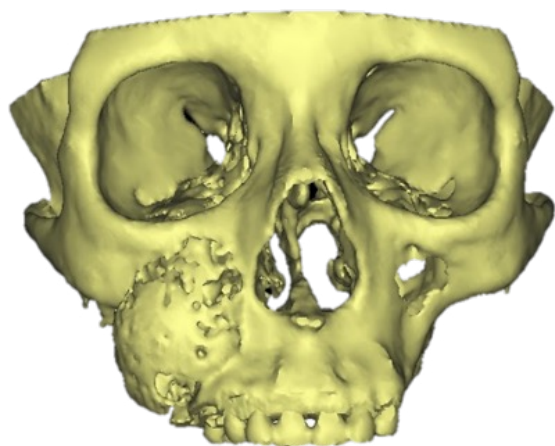


Figure 5

Based on the topography, it was decided to develop a 3D reconstruction to later elaborate stereolithography for planning the surgical phase, the conformation of the mesh for the orbit, and removing the part of the upper jaw (Figs. 5-8).

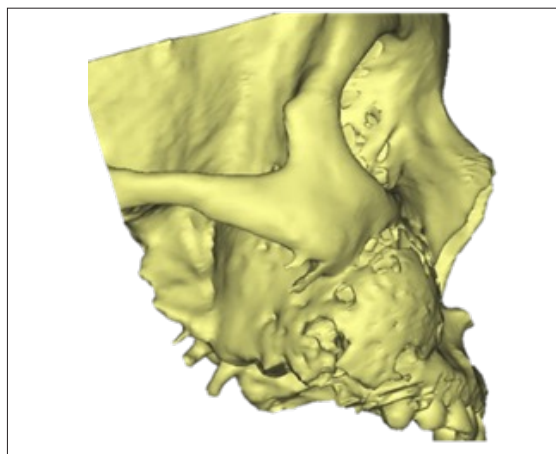


Figure 6



Figure 7

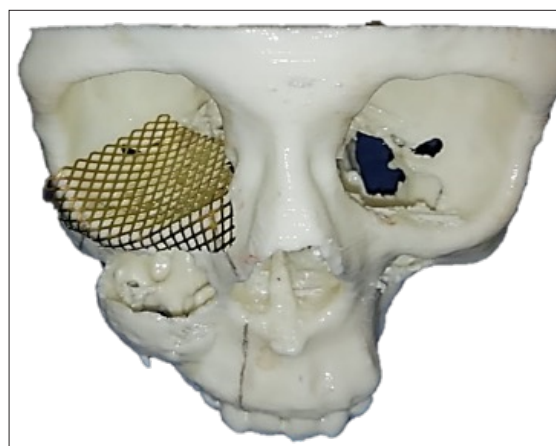


Figure 8

The eye was approached through a transcaruncular incision as a way of approach, which was carried out in combination with a pure transconjunctival incision, respecting the tear canaliculi (Figs. 9,10).

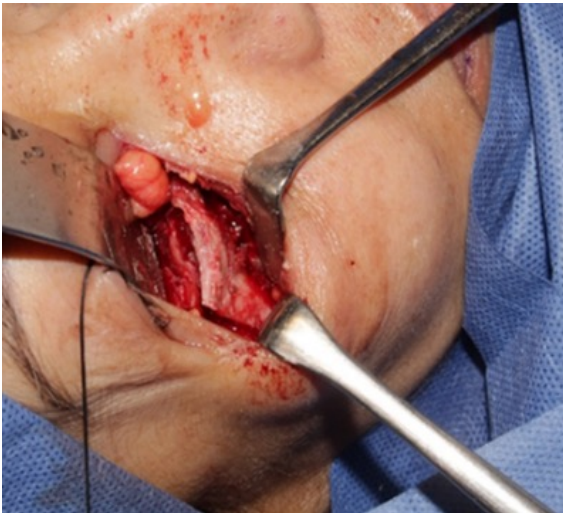


Figure 9



Figure 12

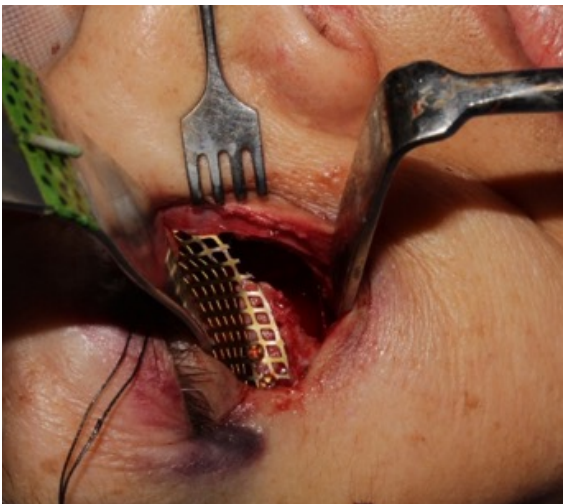


Figure 10

For the upper jaw portion, an intraoral approach was performed to make the cut previously planned in the stereolithography (Figure 11).

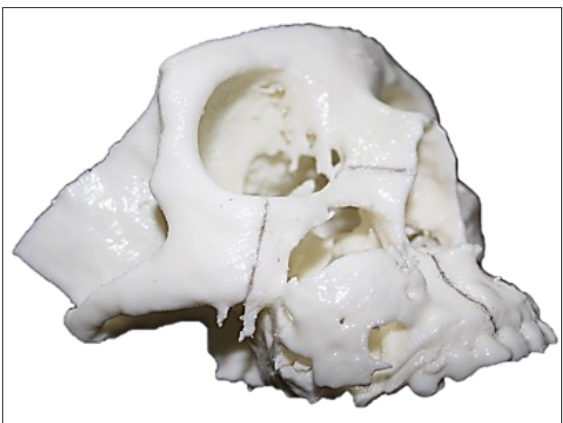


Figure 11

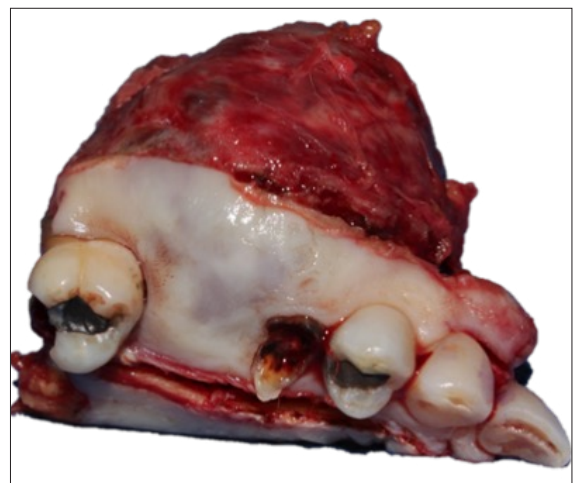


Figure 13

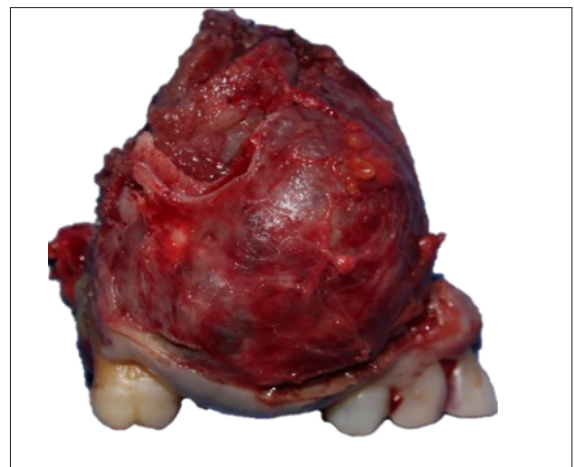


Figure 14

After removing the upper jaw, a maxillofacial prosthesis was placed to maintain the contours of the tissues and the facial appearance of the patient and avoid further problems. Figure 15,16



Figure 15



Figure 16

Pathology findings

A potentially aggressive osteolytic lesion is observed locally, composed of numerous multinucleated giant cells immersed in a spindle cell stroma with a fibrous appearance. At higher magnification, bone spicules surrounded by multinucleated giant cells (arrows) are observed, which are related to osteoclasts due to their osteolytic effect. H&E; 40x.

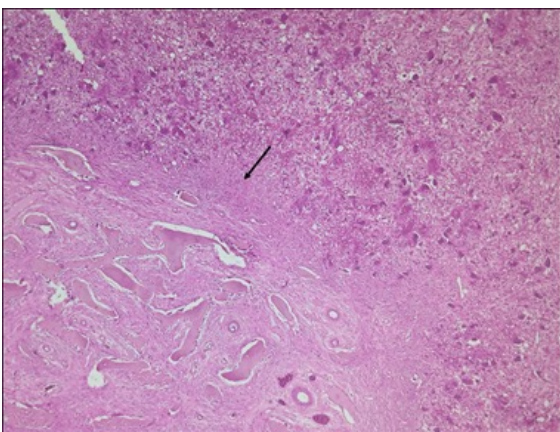


Figure 17

Multinucleated giant cells have variability in size and shape, can have up to 20 nuclei, and mitosis, but no atypia. Note its association with a spindle cell stroma and blood vessels with extravasated erythrocytes (arrows). H&E; 40x (Figs. 17,18).

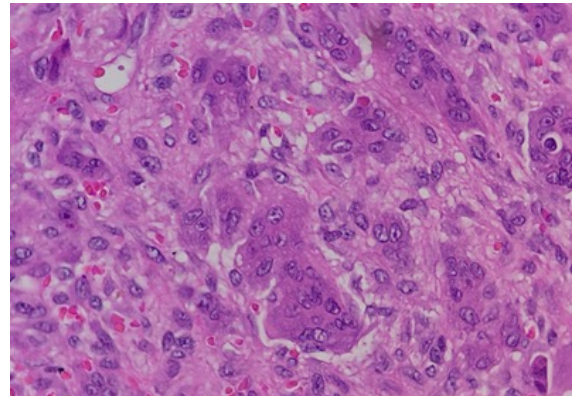


Figure 18

After surgery, the patient received secondary treatment of chemotherapy. The second CT scan showed the maxillectomy and bone remnant performed to plan rehabilitation using zygomatic, pterygoid, and conventional implants (Figs. 19,20).

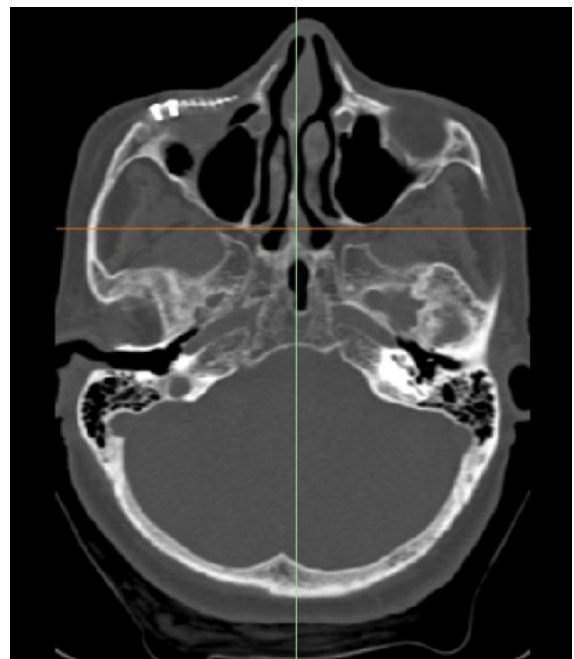


Figure 19

A diagnostic wax-up was developed to analyze the occlusion and the prosthetic plane and the location for the placement of zygomatic, pterygoid, and conventional implants; a third tomography was taken for a 3D reconstruction with the markers corresponding to the wax-up (Figs. 21-23).

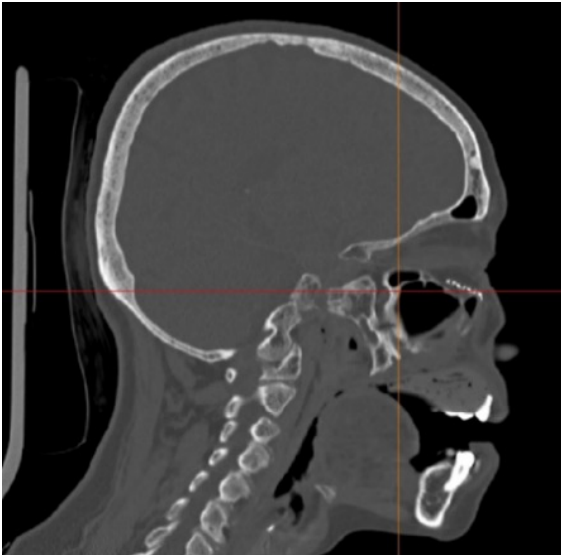


Figure 20

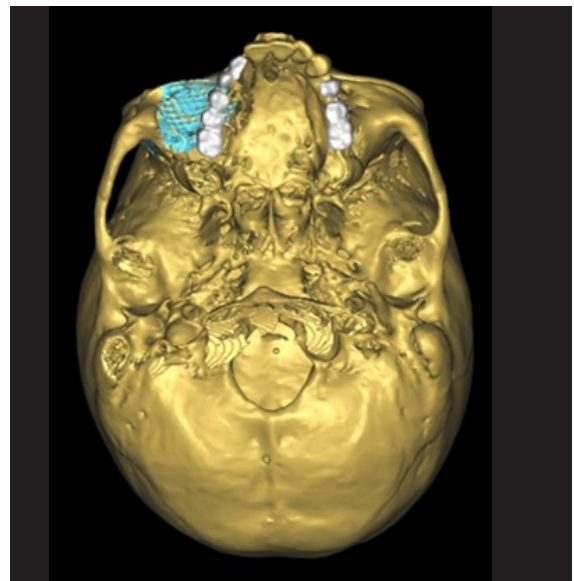


Figure 23



Figure 21

The upper right side presented a fairly large vertical and horizontal discrepancy during the initial examination. The upper right central side did not have bone support due to the resective surgery of the tumor, so the tumor was extracted (Figure 24).



Figure 24



Figure 22

Extractions of the three remaining anterior teeth were performed to achieve a better prosthetic and aesthetic plane; the process was regularized for better aesthetics and the emergence profile of the anterior implants and the prosthesis (Figure 25).

The 3D reconstruction based on the tomography was analyzed, and it was determined to place an 11.5 mm Nobel Biocare right pterygoid implant, a 38 mm zygomatic implant in the right zygomatic implant, extraction of the remaining anterior teeth, and placement of two 13 mm Nobel Biocare implants in the left upper central and left canine areas. A Nobel Biocare 40 mm zygomatic implant was placed on the upper left side (Figs. 27-29).



Figure 25

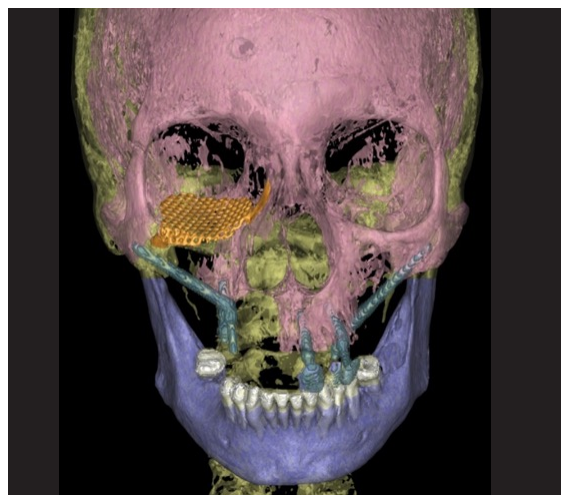


Figure 28

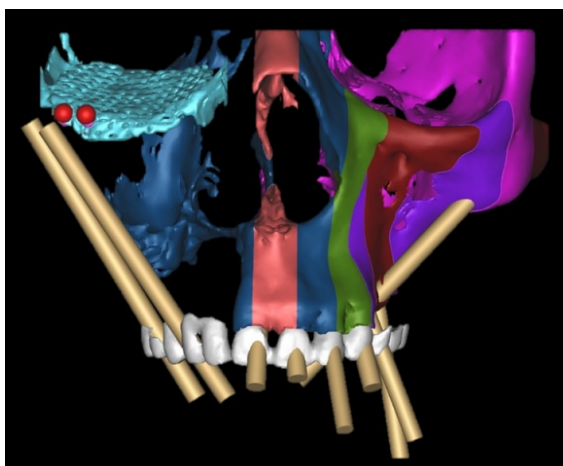


Figure 26

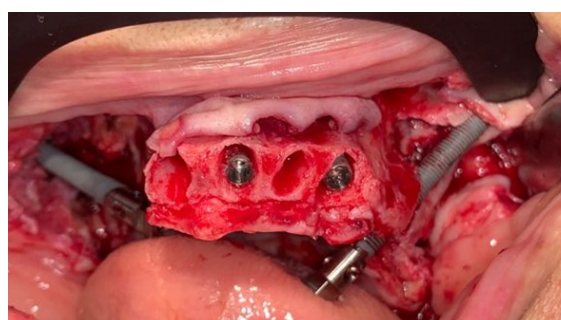


Figure 29

In the upper part, an immediate loading protocol was made with provisionalization (Figure 30).

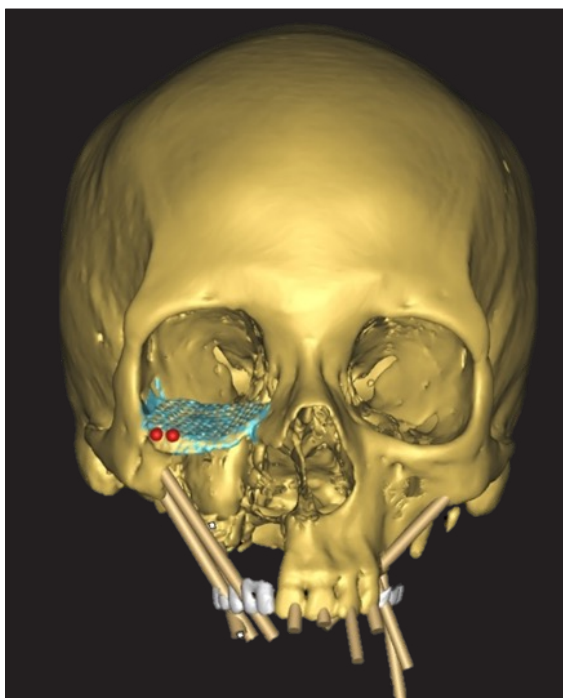


Figure 27



Figure 30

In the lower part, orthodontics was performed to improve anterior crowding and expand the arch to achieve better arch conformation and occlusion. Once healed after 3 months, an impression was taken to make two micro-milled structures that could receive two types of prosthesis (Figure 31).



Figure 31

The vertical and horizontal defect caused by the maxillectomy was compensated in both removable and fixed prostheses.

Because these types of injuries recur in 2 to 3 years, it was decided to make a removable prosthesis to be checked at intervals when necessary.

The milled microstructure was designed to receive housing for a removable partial prosthesis for two attachments, OT Cap de Rehin 83 on the left side and two on the right side (12) (Figs. 32,33).



Figure 32

In this way, the entire structure obtains passive rigidity by being wholly attached to the structure bilaterally. For the second fixed prosthesis in porcelain metal, only one of the Rehin 83 OT cap attachments is removed from the drill microstructure and screwed in using four screws (Figs. 34,35).

It was possible to obtain complete patient satisfaction regarding occlusal, functional, and aesthetic aspects by combating the collapse of respective surgery through elaborate prostheses (Figs. 36-38)



Figure 33



Figure 34



Figure 35



Figure 36



Figure 37



Figure 38

Discussion

Before 1953, giant cell lesions of the jaws were usually diagnosed as “giant cell tumors” and were generally considered similar to the giant cell tumor of long bone. In 1953, Jaffe proposed the term “giant cell reparative granuloma” for lesions found in the jaws. He believed these jaw lesions only mimicked the actual giant cell tumor of bone and had several clinical and histologic differences from the actual giant cell tumor of bone. Jaffe stated that the jaw lesions were not true neoplasms but represented a local reparative reaction (13).

Subsequently, this concept was widely accepted, and most publications after 1953 have referred to these lesions as giant cell reparative granuloma or giant cell granuloma (13,14).

In a recent study of an extensive series of giant cell lesions of the jaws, Whitaker and Waldron correlated the clinical behavior with the histologic findings (15).

The clinical and radiologic criteria Choung et al. and Ficcaro et al. suggested for separating giant cell lesions into aggressive and nonaggressive types were used. Aggressive lesions were characterized by pain, rapid growth, cortical perforation, and root resorption of teeth involved by the tumor (17).

Nonaggressive lesions showed few or no symptoms, lack of cortical perforation, root resorption, and slow growth. The study of Whitaker and Waldron showed significant differences in the distribution of giant cells and the frequency of osteoid within lesions that recurred as opposed to those that did not (15).

Campanacci (1987) divides tumors into three stages:

- Stage I: intra-osseous lesion with histology and indolent radiology.
- Stage II: Intra-osseous lesion with cortical expansion and thinning but with intact periosteum and benign histology. Stage III: extra-bony lesion of an aggressive nature but with benign histology. The vast majority (70-80%) are stage II (18).

Some authors classify them as aggressive and non-aggressive. The most common aggressive form is large tumors with rapid growth, pain, bleeding, and tooth mobility. There may be root resorption and perforation of the bone cortex. They have a high recurrence rate, especially in the first 3-4 years after treatment, and if simple curettage is performed (40-60%), en bloc resection reduces it to 7-10% (19-21).

The non-aggressive form corresponds to asymptomatic tumors, which are smaller and have much less recurrence after treatment. Diagnosis is complicated and must be based together on clinical findings, radiology, and histology. It is often necessary to study serum calcium, phosphorus, and PTH to differentiate it from brown tumors from hyperparathyroidism. In extensive lesions or aggressive behavior, adjuvant therapies can be used to reduce the size and risk of tumor bleeding during surgery. These include preoperative embolization, intralesional corticosteroid injection, and systemic administration of calcitonin. It should be noted that, with daily subcutaneous injection of interferon alfa-2a, complete tumor regressions and bone filling of residual cavities have been achieved (22).

Isolated radiotherapy is not recommended due to its potential for malignancy, except in patients who refuse surgery. However, some authors describe its successful use in primary tumors and recurrences.

The treatment of choice is surgical, recommending an en-bloc resection with wide safety margins given the high risk of recurrence and the potential for malignant transformation that it exhibits. In cases where the histological diagnosis is made after surgical excision, with free margins, follow-up with periodic controls is recommended (23,24).

Diagnosis is complicated and must be based together on clinical findings, radiology, and histology. It is often necessary to study serum calcium, phosphorus, and PTH to differentiate it from the brown tumor of hyperparathyroidism. The tumor tissue is friable and highly vascular, with small cystic areas, whitish-gray necrotic foci, hemorrhage, fibrosis, and xanthomatous regions (25).

Two factors have been attributed to the etiopathology: 1.-Inflammatory, angiogenic, and osteoclastic, although none is clearly demonstrated. The role played by the p53 suppressor gene in its genesis has recently been confirmed (5).

Other research methods have recently been employed to discern differences between giant-cell granulomas and giant-cell tumors (26).

Regezi et al. found that HLA-DR antigen detection was not helpful in differentiation between congenital, reactive, and neoplastic giant cell lesions (27).

Eckardt et al. found that nuclear DNA analysis using image cytometry was of no assistance in separating aggressive from nonaggressive giant cell lesions (28). However, in view of the overlapping clinical and histologic features, we favor the concept previously proposed by researchers: that giant cell lesions of the jaws and giant cell tumors of the extragnathic skeleton are not distinct and separate entities. The authors prefer to consider them to represent a continuum of a single disease process modified by the anatomic location and possibly other factors not yet clearly understood. Until future research delineates some method for separating giant cell tumors from giant cell "granulomas," we believe it is more logical to use the more noncommittal designation of giant cell lesion for the jaw lesions. Certainly, the designation of "granuloma" for an aggressive, recurring lesion, such as case 1 in the present report or cases reported by others, is not appropriate (6, 30, 31, 51, 52, 53, 54).

The authors advocate surgical resection for all giant cell lesions of the maxilla and paranasal sinuses.

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