



# Aggressive Giant Cell Hard Palate Reparative Granuloma with nasal cavity invasion.

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## Abstract

While seldom seen, giant cell reparative granuloma (GCRG) is a benign lesion; it can affect the area of the head and neck, primarily in the mandible and less frequently in the maxilla. GCRG usually develops in the first three decades of life but can be seen at any age, and it is seen more frequently in women than in men.

It can be locally aggressive, despite its benign nature, with bone erosion and loss of teeth.

A 16-year-old female patient was admitted to our clinic with symptoms of FB sensation in the roof of her mouth with palatal swelling that she had noticed 3 months ago, along with nasal obstruction mostly on the right side. A mass of 2.5 cm x 3 cm in size was found in the midline of the hard palate during the physical examination.

Under general anesthesia, Surgical excision was done through a transoral approach with safety margins. Buccinator muscle flap was harvested and repair of the defect of the hard palate and nasal floor was done And the operation was completed.

Histopathological analysis reveals that in a stroma rich with fibroblasts and lesions of new bone structures, a high number of giant cells are present. After these results, GCRG was diagnosed.

Post-operative follow-up, The epithelization process was observed to be complete and the defect was healed with no recurrence.

**Keywords:** Giant cell, reparative granuloma, hard palate.

## 1. Introduction

While seldom seen, giant cell reparative granuloma (GCRG) is a benign lesion; it can affect the area of the head and neck, primarily in the mandible and less frequently in the maxilla.[1]. GCRG usually develops in the first three decades of life but can be seen at any age, and it is seen more frequently in women than in men.

[2]. With bone erosion and loss of teeth, it can be locally aggressive., although its benign nature.

[3]. A connection between previous trauma and the development of GCG is suspected, but it can not be proven. Despite the various treatment alternatives described in the literature, the most widely used treatment approach remains surgical excision or curettage. [4]. A case of hard palate GCG and nasal floor invasion, along with the lower part of the nasal septum and the lower half of the inferior turbinate, is presented here.

## 2. Case report

In our clinic, a 16-year-old female patient was admitted with symptoms of FB sensation in the roof of her mouth with palatal swelling that she had noticed 3 months ago, along with nasal obstruction mostly on the right side. There is no history of dysphagia or dysphonia or nasal regurgitation and no history of injuries or surgery or propensity to bleed or swelling of the neck or other swelling of the body.

A mass of 2.5 cm x 3 cm was found in the midline of the hard palate during the physical examination (Fig.1). The mass was smooth, covered on the left side by mucosa, stretched and dark in color, and found to be elastic in palpation and tender in deep palpation. The mass was identified with a rigid nasal endoscope, eroding the nasal floor and situated in the right nasal passage, infiltrating the lower part of the nasal septum and the right inferior turbinate. Other than these findings, no other pathology was found. There were normal ranges of routine hematological and biochemical test findings, although there was a mild elevation of PTH caused by vit. D deficiency and radiological assessment of the parathyroid gland. was also negative.

In the hard palate, computerized tomography (CT) scanning and MRI indicate that a solid mass has been detected (Fig. 2). The mass has been observed to erode the nasal floor and to damage the lower

part of the nasal septum and the right inferior turbinate, measuring 4 cm in width and having visible opaqueness.

There was a fine-needle aspiration biopsy (FNAB) performed. The biopsy is consistent with the GCG's central type. The findings were within usual limits for calcium, phosphate, alkaline phosphatase, and urinary analyses.

Under general anesthesia and with safety margins, the mass was excised by a transoral approach. During surgery, the mass separated from the healthy palate. It was accompanied by endoscopic excision of the lower portion of the nasal septum and the posterior part of the right inferior turbinate. The intraoperative biopsy was confirmed to be benign and the buccinator muscle flap was harvested and the defect of the hard palate and nasal floor was repaired and the procedure was completed (fig. 3).

Histopathological analysis reveals that in a stroma rich with fibroblasts which showed hemorrhage, and a lesion consisting of new bone formation, a large number of giant cells such as osteoclast. After these findings, the case was diagnosed as GCRG.

During a physical examination that took place on the eighth week after surgery, the epithelization process was completed and the defect was healed (fig.4). In the 15th month of the operation, no recurrence was observed in the physical examination.

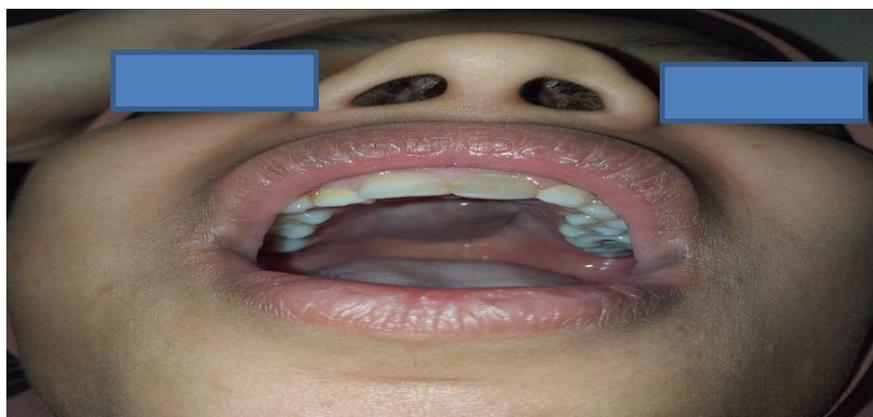


Fig.(1) The mass seen from the mouth



Fig. (2) pre-operative CT& MRI images axial ,coronal & sagittal cuts



Fig. (3) Operative procedure with palatal incision ,dissection of the lesion .reconstruction of the palatal defect with buccinator rotational flap.



Fig. (4) Post-operative follow-up after 2weeks and 8 weeks with no recurrence with successful buccinator rotational flap.

### 3. Discussion

Giant cell granuloma was first described by Jaffe in 1953 as a giant cell reparative granuloma of the jawbones. It is a rare and benign lesion, generally affecting the mandible and maxilla[5]. With a predilection for females that were not as strong as previously thought, (F/M= 2:1), an incidence of 1.1 per 100 was found. GCG occurred most commonly in the young population occurring respectively between the ages 10–14 and 15–19 years with peak incidence for males and females [6].

Caustic causes include inflammation, trauma, and intramedullary bleeding, but a definitive etiology for the disorder has not been discovered [7].

The mandible was more frequently involved than the maxilla, as described earlier. The most affected area was the anterior portion of the first mandibular molar. Less common locations are found in the front and back of the premolar and incisive teeth. The front and the right side are the most affected parts of the maxilla.

Other sites, such as the nasal cavity, temporal bone, and paranasal sinuses, may be involved, even though the mandible and maxilla are the most common sites in the head and neck regions[8].

Multinuclear giant cell aggression involving several hemorrhagic foci on cellular fibrous biological tissue provides the histological classification of giant cell reparative granuloma independent of the presence of reactive bone trabecula. [9].

There are two clinical types of GCRG: peripheral and central. Peripheral GCG is more prevalent, Occurring from the periosteum or connective tissue as part of the alveolar ridge and gingival mucosa. Although the central form is endosteal (bone-based) in nature, meaning they originate from inside the cortex, it can be located in the maxilla, the tem-

poral bone, and the paranasal sinuses, which are less common in the mandible. [10,11].

It is possible to distinguish the clinical behavior of giant cell reparative granuloma into aggressive and nonaggressive types. The most commonly seen form is the non-aggressive form with characteristic gradual growth and painless swelling. This form shows smooth limits in palpation and elastic consistency. However, the swelling is painful in an aggressive form and develops rapidly.

Other predicted effects can be observed, such as facial asymmetry, Loss or alteration of the location of the teeth, expansion of the bone, and cortical perforation. Cortical perforation is more frequently seen in the maxilla relative to the mandible because of the thinness of the cortical bone in the former, with a high recurrence rate in the aggressive type.

These findings are considered prognostic factors in terms of local invasion and recurrence following adequate therapy. The histological difference between the two categories is not evident. [12, 13].

There have been no clear radiological findings described where unilocular or multilocular radiolucent lesions with or without teeth distortion, root resorption, and cortical degradation can be observed. When the size of the lesion increases, the propensity to be aggressive and multilocular increases. The CT scan offers a more thorough GCRG study than traditional radiology.

In the differential diagnosis, palatal swelling has several lesions, where the lesions can arise from salivary, fibrous, nervous, bone, and lymphoid tissue[15]. In the present case, we mention a case of a 16- year old girl with intact mucosal covering with palatal mass, which made us think about minor salivary gland neoplasm of the first.

Histopathological review, on the other hand, shows numerous giant cells that should be diagnosed differently: hyperparathyroidism brown tumor, giant cell tumor, GCG, fibrous dysplasia, type II neurofibromatosis, cherubism, and aneurysmal bone cysts. [16]

Aneurysmal cyst bone was excluded due to the absence of cystic spaces and amorphous calcification with chondroid aura, despite the presence of prominent red blood cell extravasation. The same histologic characteristics share the remaining possible diagnoses. However, normal values of calcium, phosphorus, and PTH exclude hyperparathyroidism from a brown tumor; negative staining of p63 excludes giant cell tumor; Due to the absence of characteristic clinical manifestations, type I cherubism and neurofibromatosis were omitted.

Therefore, the central giant cell granuloma of the hard palate was the final diagnosis. The standard treatment is typically surgical. Depending on the degree of soft tissue penetration and the mass size, the surgical choices range from simple curettage, an excisional biopsy to blocking resection. The most commonly prescribed is curettage or resection of the lesion.

Chuong et al. suggest block resection to be used in cases of aggressive lesions displaying painful, cortical perforations[12]. In the literature, The rate of recurrence has been described between 11% and 35% where surgical resection is associated with 5.6%-11.5% recurrence rates when compared to 12.5%-46% simple curettage. [17].

Other alternative methods of treatment have been established to avoid surgery during childhood and to treat the recurrence of aggressive lesions[4]. Among these alternative treatment approaches, weekly intralesional injections of steroids. [18].

Another treatment choice is the application of subcutaneous interferon[19]. Also, calcitonin is used in therapy for its

antagonizing osteoclastic bone resorption properties. [20]. However, there is not enough evidence to justify their use with or without surgical resection.

#### 4. Conclusion

Giant cell reparative granuloma is a rare lesion that can affect the head and neck regions most commonly found in the mandible. For diagnosis Histopathological analysis is a must. In the present case, aggressive giant cell reparative granuloma of the hard palate is an uncommon site for GCRG along with the invasion of the nasal floor together with the inferior part of the nasal septum and posterior half of the inferior turbinate.

Therefore, when examining masses located in the hard palate, GCRG diagnosis should be an option. Surgical excision was conducted with safety margins using a transoral approach. The Buccinator muscle flap was harvested and the defect of the hard palate and nasal floor was repaired and the procedure was completed.

Post-operative follow-up, the process of epithelization was found to be complete and the defect was healed without recurrence.

#### References

1. de Lange, J. and H.P. van den Akker, *Clinical and radiological features of central giant-cell lesions of the jaw.* Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology, 2005. **99**(4): p. 464-470.
2. Kaffe, I., et al., *Radiologic features of central giant cell granuloma of the jaws.* Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology, 1996. **81**(6): p. 720-726.
3. Sezer, B., et al., *Intralesional corticosteroid injection for central giant cell granuloma: a case report and review of the literature.* Turk J Pediatr, 2005. **47**(1): p. 75-81.

4. Bataineh, A.B., T. Al-Khateeb, and A.R. Ma'amon, *The surgical treatment of central giant cell granuloma of the mandible*. Journal of oral and maxillofacial surgery, 2002. **60**(7): p. 756-761.
5. Jaffe, H.L., *Giant-cell reparative granuloma, traumatic bone cyst, and fibrous (fibro-osseous) dysplasia of the jawbones*. Oral Surgery, Oral Medicine, Oral Pathology, 1953. **6**(1): p. 159-175.
6. Edwards, P.C., et al., *Bilateral central giant cell granulomas of the mandible in an 8-year-old girl with Noonan syndrome (Noonan-like/multiple giant cell lesion syndrome)*. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology, 2005. **99**(3): p. 334-340.
7. Sidhu, M., H. Parkash, and S. Sidhu, *Central giant cell granuloma of jaws—review of 19 cases*. British Journal of Oral and Maxillofacial Surgery, 1995. **33**(1): p. 43-46.
8. Tian, X.-F., T.-J. Li, and S.-F. Yu, *Giant cell granuloma of the temporal bone: a case report with immunohistochemical, enzyme histochemical, and in vitro studies*. Archives of pathology & laboratory medicine, 2003. **127**(9): p. 1217-1220.
9. Van Der Waal, I., *Diseases of the jaws: diagnosis and treatment; textbook & [and] atlas*. 1991: Munksgaard.
10. Arda, H.N., et al., *Giant cell reparative granuloma originating from the ethmoid sinus*. International journal of pediatric otorhinolaryngology, 2003. **67**(1): p. 83-87.
11. Chaparro-Avendano, A., L. Berini-Aytes, and C. Gay-Escoda, *Peripheral giant cell granuloma. A report of five cases and review of the literature*. Medicina oral, patologia oral y cirugia bucal, 2005. **10**(1): p. 53-7; 48-52.
12. Chuong, R., et al., *Central giant cell lesions of the jaws: a clinicopathologic study*. Journal of oral and maxillofacial surgery, 1986. **44**(9): p. 708-713.
13. Üstündağ, E., et al., *Central giant cell granuloma*. International journal of pediatric otorhinolaryngology, 2002. **65**(2): p. 143-146.
14. Cohen, M.A., and Y. Hertzanu, *Radiologic features, including those seen with computed tomography, of central giant cell granuloma of the jaws*. Oral surgery, oral medicine, oral pathology, 1988. **65**(2): p. 255-261.
15. Moss, S., et al., *Slowly expanding palatal mass*. Journal of oral and maxillofacial surgery, 2001. **59**(6): p. 655-659.
16. Urade, M., et al., *Mandibular giant cell granuloma*. Oral Surgery, Oral Medicine, Oral Pathology, 1988. **66**(1): p. 121-122.
17. Kruse-Lösler, B., et al., *Central giant cell granuloma of the jaws: a clinical, radiologic, and histopathologic study of 26 cases*. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology, 2006. **101**(3): p. 346-354.
18. Abdo, E.N., et al., *Treatment of a central giant cell granuloma with an intralesional corticosteroid*. British Journal of Oral and Maxillofacial Surgery, 2005. **43**(1): p. 74-76.
19. Kaban, L.B., et al., *Antiangiogenic therapy of a recurrent giant cell tumor of the mandible with interferon alfa-2a*. Pediatrics, 1999. **103**(6): p. 1145-1149.
20. Pogrel, M., *Calcitonin therapy for central giant cell granuloma*. Journal of oral and maxillofacial surgery, 2003. **61**(6): p. 649-653.
21. Cannistra, C., et al., *Central giant cell granuloma of the palate; unusual localisation in a five year old child*. Bulletin du Groupement international pour la recherche scientifique en stomatologie & odontologie, 1999. **41**(4): p. 112-114.