

# Concomitant Presentation of Central Giant Cell Granulomas in the Anterior Mandible and Palate of a Male Patient: A Clinical Image

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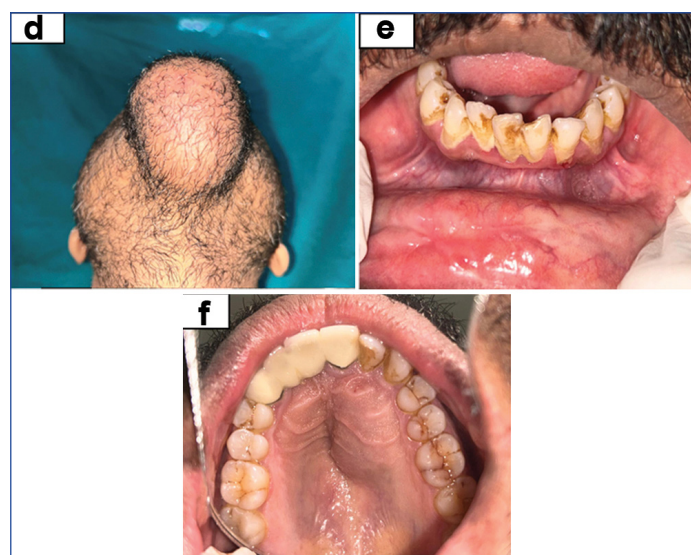
**Keywords:** Mandible, Maxilla, Maxillofacial trauma, Neoplasm

A 30-year-old male patient reported to the Outpatient Department (OPD) with the complaint of a progressive painless swelling in the lower front region of the jaw [Table/Fig-1a]. The swelling was persistent since eight months without any associated discharge, paraesthesia, or functional difficulty. He was hypertensive and was on antihypertensive therapy since nine years. He also reported a history of maxillofacial trauma three years back due to a road traffic accident, which had resulted in intrusion of the maxillary anterior teeth into the nasal cavity. The affected teeth were subsequently extracted, and a fixed dental prosthesis was placed approximately one year ago. There was no other significant family history of similar lesions.

General physical examination revealed a well-oriented, moderately built male with a facial disfigurement which was evident. Extraoral examination revealed a diffuse swelling in the lower one third of the face, involving the anterior mandible, approximately 5.1 cm in diameter which was firm to hard and non-tender on palpation [Table/Fig-1d,e]. Intraoral examination revealed a second, well-defined swelling on the mid-palatal region, approximately 3 cm×2 cm [Table/Fig-1f], with intact overlying mucosa, no signs of discharge, firm to hard and non-tender on palpation. The dentition was intact with no mobility or displacement of adjacent teeth, and no mucosal changes were noted in association with either lesion.

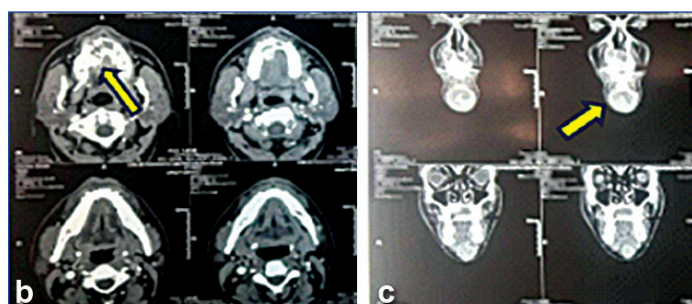


**[Table/Fig-1a]:** Extraoral facial profile of the patient.



**[Table/Fig-1d-f]:** d) Extraoral image showing the mandibular lesion; e) Intraoral image showing the labial vestibular region of the mandible; f) Intraoral image showing the incidental palatal swelling.

The patient had already undergone Computed Tomography (CT) [Table/Fig-1b,c] scans from a private imaging centre, which revealed a well-defined, multilocular expansile lesion involving the anterior region of the mandible. The imaging suggested a central lesion of possible giant cell origin, and the patient was referred for further clinical evaluation, radiographic correlation, and definitive management.



**[Table/Fig-1b,c]:** b) Axial section showing the maxillary lesion marked by the arrow in CT; c) Coronal section showing the mandibular lesion marked by the arrow in CT.

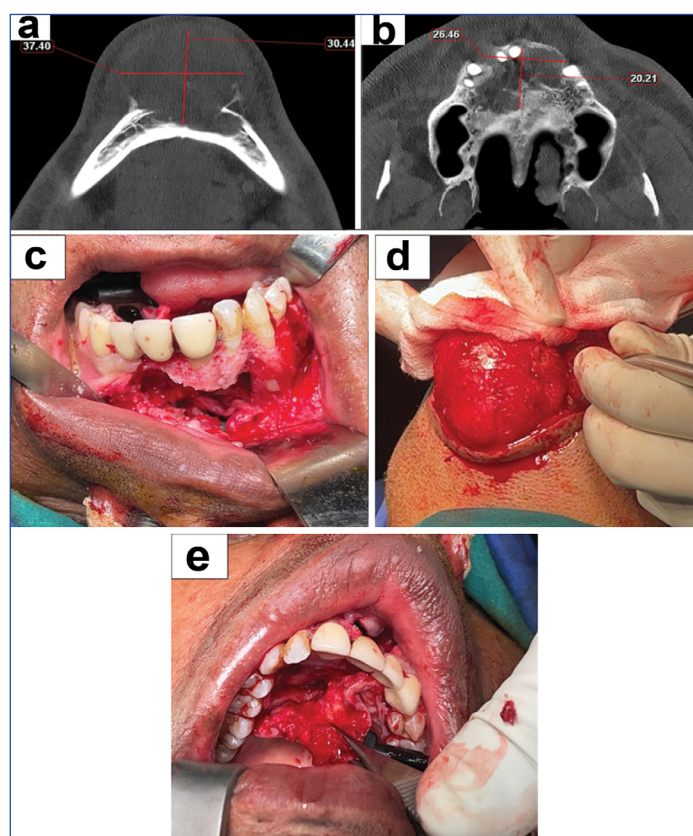
Given the history of maxillofacial trauma, the presence of two distinct intraosseous swellings, and the imaging findings suggestive of a multilocular lesion, a provisional diagnosis of Central Giant Cell Granuloma (CGCG) was considered. However, in view of the multilocular appearance and multifocal presentation, differential diagnoses such as Odontogenic Keratocyst (OKC), odontogenic myxoma, aneurysmal bone cyst, and brown tumour of hyperparathyroidism were also taken into consideration.

To supplement the existing CT findings, a Cone-Beam Computed Tomography (CBCT), scan was performed to evaluate the detailed cortical and internal architecture of the lesions [Table/Fig-2a,b]. On CBCT, a well-defined multilocular lesion in the anterior mandibular region crossing the midline with thinning and perforation of labial cortical plate was noted. In maxilla, a well-defined radiolucent lesion was observed in anterior region crossing the midline along

with thinning of the palatal cortical plate, thinning and perforation of buccal cortex. The presence of two lesions in separate jaw regions raised the differential diagnosis of multifocal CGCG, brown tumour and odontogenic keratocyst.

Laboratory investigations were carried out to rule out systemic conditions such as hyperparathyroidism, which may present with similar radiological and histopathological findings. These included a Complete Blood Count (CBC), haemoglobin (Hb), fasting blood glucose, glycated haemoglobin (HbA1c), and serum Parathyroid Hormone (PTH) levels- all of which were within normal limits. Based on the clinical, radiological, and haematological findings, an excisional biopsy was planned for both lesions to obtain a definitive diagnosis and initiate appropriate management.

The patient underwent surgical excision and enucleation of the mandibular lesion under general anaesthesia. The lesion was removed from the area of anterior mandible through a No. 10 blade excision. Subperiosteal dissection was done to allow visualisation of the margin of the lesion before excising it [Table/Fig-2c,d]. Once the defect was created, autograft was made by removing a bone fragment measuring 9 cm from the fibula bone on the left leg. Subsequent to the excision, a non-vascularised fibular autograft was positioned within the defect, followed by placement of reconstruction plates to re-establish mandibular continuity and provide structural stability. Suturing was performed using 2-0 Vicryl and 3-0 Ethilon.

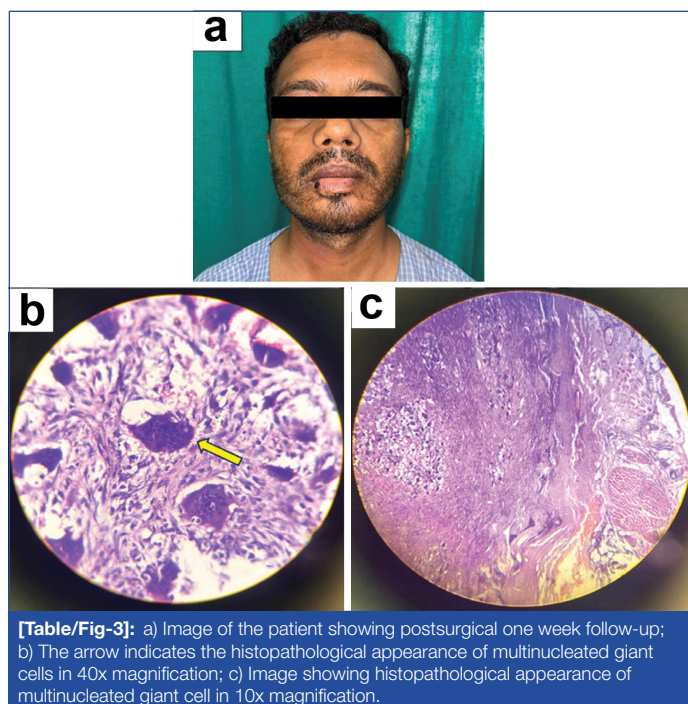


**[Table/Fig-2]:** a) Axial section showing the extent of the mandibular lesion in CBCT; b) Axial section showing the extent of the maxillary lesion in CBCT; c-e) Image showing surgical excision of the lesion.

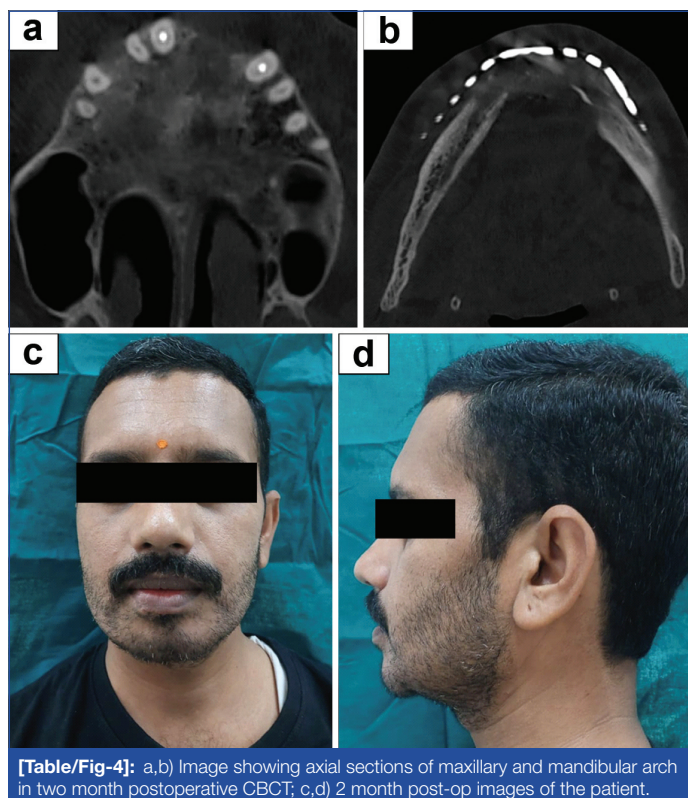
The lesions on the palate were excised through a No. 10 blade, and full-thickness mucoperiosteal flap was raised to aid excision [Table/Fig-2e]. After excision, smoothing of the bony surfaces was carried out using a vulcanite bur and the excised specimens were sent for histopathological examination. Patient visited after one week of the surgery [Table/Fig-3a].

The excised section underwent histopathologic examination in which numerous multinucleated giant cells were seen dispersed within a background of ovoid to spindle shaped mesenchymal cells. Areas of haemorrhage and haemosiderin pigmentation were noted

consistent with the diagnosis of CGCG [Table/Fig-3b,c]. A two month postoperative follow-up showed satisfactory healing [Table/Fig-4].



**[Table/Fig-3]:** a) Image of the patient showing postsurgical one week follow-up; b) The arrow indicates the histopathological appearance of multinucleated giant cells in 40x magnification; c) Image showing histopathological appearance of multinucleated giant cell in 10x magnification.



**[Table/Fig-4]:** a,b) Image showing axial sections of maxillary and mandibular arch in two month postoperative CBCT; c,d) 2 month post-op images of the patient.

CGCG is an uncommon, benign lesion but sometimes locally aggressive intraosseous lesion of the jaws, first described by Jaffe HL in 1953 [1,2]. It is characterised histologically by the presence of numerous multinucleated giant cells in a fibrous connective tissue stroma [3,4]. CGCGs constitute about 7% of all benign jaw lesions and are more commonly found in the mandible, particularly in the anterior region, often crossing the midline [5].

The lesion is most frequently reported in females under 30 years of age. Multifocal CGCGs are relatively rare with only a few cases reported [6,7]. Multifocal presence of CGCG is even rarer. Mohammadi F et al., has reported multifocal occurrence in younger age group [7]. In comparison, the present case demonstrated two concomitant lesions in the anterior mandible and maxilla in a non-syndromic male patient, adding to the limited pool of such

reported cases. The exact aetiology of CGCG remains unclear, though trauma, vascular malformations, and genetic mutations have been suggested [8]. Notably, patient had a history of significant orofacial trauma three years prior due to a road traffic accident. This trauma may have contributed to the development of the lesion as it is hypothesised that trauma may induce intraosseous haemorrhage and trigger a reparative response, leading to proliferation of fibroblasts and recruitment of osteoclast-like giant cells [9].

The pathogenesis of CGCG is closely linked to osteoclastic activity. The multinucleated giant cells are osteoclast-like and contribute to bone resorption [10]. They are thought to arise from mononuclear stromal cells under the influence of cytokines, particularly via the RANK-RANKL pathway, leading to increased osteoclastic differentiation and osteolysis [10].

This case is of particular clinical interest due to the rarity of multiple synchronous CGCGs in a non-syndromic, systemically healthy male patient, especially with simultaneous involvement of the anterior mandible and the hard palate. While multifocal CGCG is sometimes associated with syndromic conditions such as Noonan syndrome, neurofibromatosis type 1, no systemic signs or family history suggestive of syndromic association were identified in our patient [10]. Therefore, this case emphasises the importance of thorough systemic evaluation and a high index of suspicion for multifocal presentation, even in non-syndromic patients.

In conclusion, although CGCG is a well-recognised entity, its multifocal occurrence in unusual sites such as the palate, especially in

adult male patients, is exceedingly rare. This case contributes to the growing body of evidence that challenges the traditional demographic and anatomical predilections of CGCG and underscores the need for individualised diagnostic and therapeutic strategies.

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### PLAGIARISM CHECKING METHODS: [Jain H et al.]

- Plagiarism X-checker: Apr 04, 2026
- Manual Googling: May 05, 2026
- iThenticate Software: May 07, 2026 (6%)

### ETYMOLOGY: Author Origin

EMENDATIONS: 6

### AUTHOR DECLARATION:

- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Mar 18, 2026**

Date of Peer Review: **Apr 16, 2026**

Date of Acceptance: **May 09, 2026**

Date of Publishing: **Sep 01, 2026**