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A Rare Case of Peripheral Giant Cell Granuloma associated with Odontome

Dr. Neha Hissariya- Senior Lecturer, Oral Medicine and Radiology, Sri Aurobindo College of Dentistry (Corresponding author)

Dr. Tushar Phulambrikar- Professor and HOD Oral Medicine and Radiology, Sri Aurobindo College of Dentistry

Dr. Tanvi Dosi- Professor Oral Medicine and Radiology, Sri Aurobindo College of Dentistry

Dr. Sunaina Verma- Post Graduate Student, Oral Medicine and Radiology, Sri Aurobindo College of Dentistry

Detail of institution where work is carried out- Sri Aurobindo Dental College and Hospital, near Indore-Ujjain Highway Bhawarsala, Indore-452001 (Madhya Pradesh)

Address of corresponding Author-

Neha Hissariya

A5-825 Milan Heights, Pipliyana Indore- 452016

Email id- nehavyas1509@gmail.com

Abstract:

Peripheral giant cell granuloma (PGCG) is a benign tumor of the oral mucosa. Although its etiology is not clear, it may be a consequence of local chronic irritation or persistent trauma. A complex odontoma is a hamartomatous lesion in which all the dental tissues are well formed but occurring in a more or less disorderly pattern. The complex type of an odontome is slow growing and expanding lesions that is usually detected in the second decade of life. A 34 years-old man presented a exophytic growth which was violet-colored with pedunculated base in the buccal aspect of the maxillary left posterior region. Radiographically, the internal structure appeared as a mixed hypodense and hyperdense. Its effect on the adjacent structures included displacement of 28 distally, loss of buccal cortical plate i.r.t 27, 28. Expansion with thinning of the palatal cortical bone i.r.t 27, 28. The objective of this case report was to

document the main clinical and histopathological characteristics of a patient with a PGCG associated with an odontoma

Key words: Peripheral giant cell granuloma, Odontoma, Complex odontome, Hamartomas, Benign tumour.

INTRODUCTION:

Peripheral giant cell granuloma (PGCG) is a benign tumour of the oral mucosa that originates from the periosteal cells or the periodontal ligament. Its etiology is not clear, but is related to a persistent trauma. The lesion is also known as giant cell epulis, osteoclastoma, reparative giant cell granuloma, or giant cell hyperplasia. [1]

Clinically, PGCGs may present as a nodular lesion, predominantly bluish-red with a smooth shiny or protruded surface with variable size, rarely exceeding 2 cm in diameter, and are generally soft or rubbery. They are often asymptomatic, but occlusal interferences they may ulcerate and become infected. They show site predilection for premolar and molar regions. Treatment is surgical excision followed by elimination of possible irritant factors. [1,2]

Odontomas are hamartomas that account for 22% of the odontogenic tumors. They are the most common benign odontogenic tumors of epithelial and mesenchymal origin. [3] Odontomas are are classified into complex odontoma and compound odontoma based on gross, radiographic, and microscopic features, odontomas. According to WHO complex odontoma are malformation in which all of the dental tissues mainly are well formed but occur in disorderly pattern. [4]

CASE REPORT:

A 34-year-old male patient presented to the department of Oral Medicine and Radiology with a localized swelling on the upper left jaw of his face since 2 months. The swelling, as per the

patient, was insidious in onset, gradually progressive and not associated with loosening of the teeth on the concerned region. There was no history of any discharge or difficulty in chewing or swallowing but patient experienced bleeding from the concerned region while brushing of his teeth. Other medical history was non-contributory. There was also no history of trauma in the area of chief complaint.

Extra-oral examination revealed a diffuse swelling on the left side of his face extending from commissure of his lip on the left side till mid face region antero-posteriorly and from the malar prominence till commissure of his lip on the left side superior-inferiorly causing mild facial asymmetry. The overlying skin appeared to be normal and was non tender, and there was no associated lymphadenopathy.

The intraoral examination revealed an intact permanent dentition in place, a pedunculated reddish violet growth present in left upper alveolus associated with molars extending mesiodistally from mesial aspect of 26 to the left maxillary tuberosity and superoinferiorly from the occlusal plane to the depth of the buccal vestibule. A faint bulge on the left side of hard palate was also seen. The last molar appeared to be drifting toward the distal aspect. On palpation, consistency was soft to firm and faint bony crepitus was felt on the palatal aspects.

Radiographic findings:

The intra-oral periapical radiograph and orthopantomograph revealed no extra-ordinary findings apart from the distal drifting i.r.t 28.

Later on, a volumetric Cone Beam Computed Tomography (CBCT) was made with Kodak CS9300, CBCT machine: covering left posterior Maxilla.

Axial and sagittal view demonstrate hypodense lesion extending medio-laterally from the distal aspect of 26 till maxillary tuberosity with well-defined hyperdense borders. The

internal structure as a mixed hypodense and hyperdense. Its effect on the adjacent structures included displacement of 28 distally, loss of buccal cortical plate i.r.t 27, 28. Expansion with thinning of the palatal cortical bone i.r.t 27, 28. Maxillary sinus demonstrated isodensity in the left maxillary sinus along with breach in the floor of the maxillary sinus in the left maxillary sinus, which is suggestive of maxillary sinus pathology and bone erosion, respectively.

DISCUSSION:

Peripheric giant cell granuloma is an exophytic common lesion occurring in the oral cavity which is a perivascular lesion and is mainly consecutive to local irritants such as tartar, plaque, incompatible restoration, traumatic tooth extraction, and chronic inflammation. PGCGs can be seen in any age groups, but are more common in the 40-60year age group. The most common site for PGCGs is the mandible, they rarely occur maxilla. [5]. Peripheral giant cell granuloma can also be associated with dental implants, pregnancy and primary hyperparathyroidism. [6,7]

Odontomes are usually asymptomatic, discovered during routine radiographic examination when there is delayed eruption of permanent teeth.[9] Compound odontomas seldom cause bony expansion but complex odontomes often cause slight or even marked bony expansion The complex odontoma appears as an irregular mass of calcified material surrounded by a thin radiolucent area with smooth periphery, and the compound type shows calcified structures resembling teeth in the center of a well-defined radiolucent lesion. [9]

Trauma to the dental tissue have been implicated as one of the etiological factor for both PGCG and odontome. However the patient in the present case did not gave any history of trauma to the dental structures. Hence the etiology remains unclear.

Histologically, PGCG shows atrophic or hyperplastic stratified squamous epithelium having moderate inflammatory infiltrate and vessels in the superficial lamina propria. Connective tissue, has proliferation of multinucleated giant cells within a background of plump ovoid and spindle-shaped inflammatory cells, frequently with deposits of hemosiderin pigment throughout the tissue. [10] In this case, histopatholgical examination revealed proliferative parakeratinized stratified squamous surface epithelium and fibrocellular connective tissue stroma showing membranous multinucleated giant cells within background of plump ovoid and spindle-shaped mesenchymal cells. Chronic inflammatory cell infiltrate chiefly composed of lymphocytes along with single endothelial lined blood vessels are also seen along with osteoid tissue.

CONCLUSION:

PGCG and Odontoma are not very uncommon lesions of oral cavity but their coexistence is a rare entity. The precise diagnosis of the lesions is based on a thorough clinical, radiological, and histological finding, which may further help in successful treatment outcomes.

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Figures

Figure.1 Front Facial Profile of patient

Figure. 2 Intraoral Image showing the growth reddish violet growth

Figure.3 Intraoral image showing pedunculated base of the growth

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Figure 4 Intraoral photograph showing expansion of palatal cortical plate in left molar region

Figure 5 Excised gross specimen

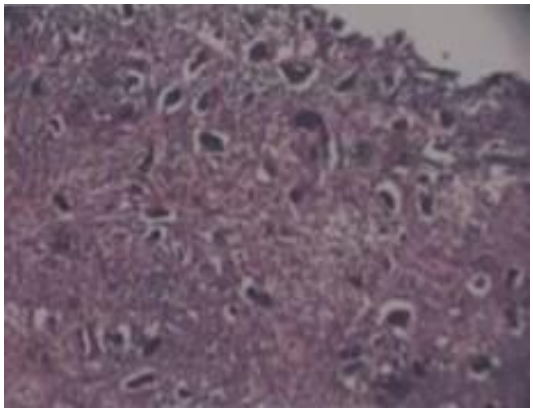

Figure 6 Histopathological view of the specimen

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Figure 7 IOPA showing distal drifting of 28

Figure 8 Orthopantomograph showing distal drift of 28

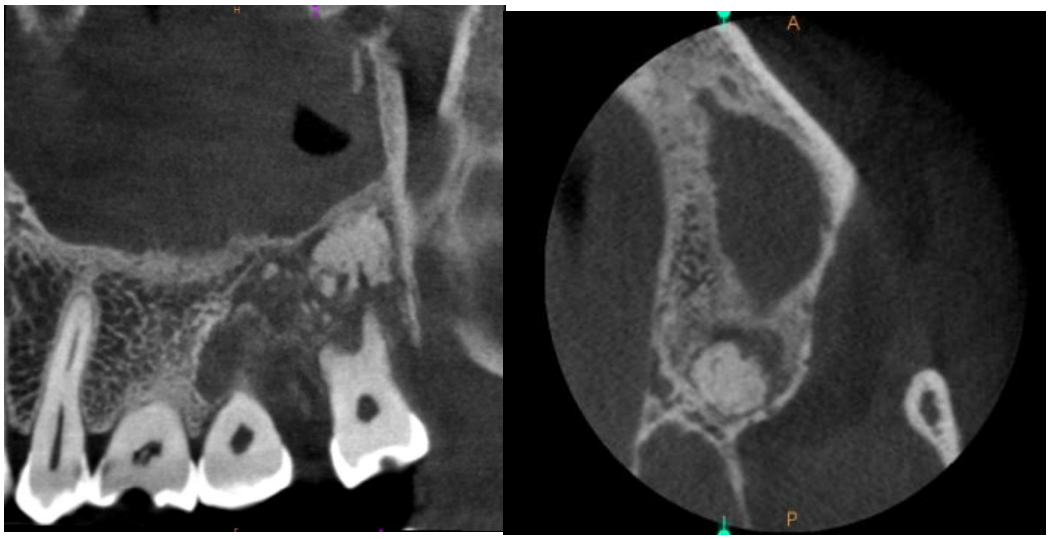

Figure 9 (a)&(b) Sagittal and axial view showing mixed hypodense and hyperdense region i.r.t 27, 28 along with the isodensity in the left maxillary sinus

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Figure 10 Axial view demonstrates expansion of palatal plate along with the breach in the continuity of buccal cortical plate