|| Print ISSN: 2589-7837 || Online ISSN: 2581-3935 ||
International Journal of Medical Science and Diagnosis Research (IJMSDR)
Available Online at www.ijmsdr.com
PubMed (National Library of Medicine ID: 101738824)
Case Report Volume 4, Issue 3; March: 2020; Page No. 39-42

UNUSUAL PRESENTATION OF PERIPHERAL GIANT CELL GRANULOMA IN A PATIENT WITH RAPIDLY PROGRESSING PERIODONTITIS - A CASE REPORT

Dr Vaneet Kaur Sandhu[1], Dr Yashmeet Kaur[2], Dr Sarfaraz Padda[3]

[1]MD., Pathology, Associate Professor, Department of Pathology, Guru Gobind Singh Medical College and Hospital Faridkot, Punjab, India

[2]MDS, Oral Surgery Associate Professor, Department of Oral Surgery, IBN Sina National College for Medical Studies, Jeddah, Kingdom of Saudi Arabia

[3]MDS., Oral Medicine and Radiology Associate Professor, Department of Oral Medicine and Radiology, IBN Sina National College for Medical Studies, Jeddah, Kingdom of Saudi Arabia

Conflicts of Interest: Nil

Corresponding author: Dr Vaneet Kaur Sandhu

ABSTRACT

Peripheral giant cell granuloma (PGCG) is a localised, benign, sometimes aggressive hyperplastic reactive lesion of oral cavity with an unclear etiology occurring in the relation to gingival and alveolar mucosa. Rapidly Progressing Periodontitis (RPP) usually presents at second to fourth decade of age with a female predilection. Local irritation as plaque and calculus play a vital role. We present here a case and management of a young female suffering from RPP with complete destruction of the alveolar bone and a huge PGCG in left mandibular body region.

Key words: Morphology of peripheral giant cell granuloma, periodontitis

Introduction

Peripheral Giant Cell Granuloma (PGCG) is a localized, benign, sometimes aggressive hyperplastic reactive lesion of oral cavity. Jaffe in 1953 proposed the term “giant-cell reparative granuloma” for such lesions occurring in the jaws which was later renamed as “peripheral giant cell reparative granuloma”.[1]

PGCG is thought to originate from the interdental tissue (periosteum or periodontal ligament) in presence of local irritant factors as the sub-gingival plaque and calculus[2-3] Rapidly Progressing Periodontitis(RPP) is a multifactorial disease, usually seen in young adults with a female predilection.[4-5]

We present here a case and management of a young female suffering from RPP with complete destruction of the alveolar bone and a huge PGCG in left mandibular body region.

Case Report

A 22 year female reported to Oral and Maxillofacial Department with the chief complaint of swelling in her lower jaw for the past three years with spontaneous exfoliation of few teeth and generalized mobility in the teeth. Intraoral examination revealed very poor oral hygiene with plaque, calculus and debris, mobility in all teeth with more than six mm deep pockets, generalized gingival hyperplasia and bleeding on probing. An irregular, bluish-red, erythematous, firm with a sessile base lesion in left body region of mandible extended from midline to distal of left second molar with an approximate antero-posterior dimension of four cm and a maximum bucco-lingual dimension of three and half cm which slowly increased to its present size (fig 1). Submental and bilateral submandibular lymph nodes were tender. The Orthopantomogram revealed excessive alveolar bone resorption with scooped out appearance in bilateral lower posterior region with only basal bone remaining (fig 2). CECT of facial bones revealed ill-defined enhanced lesion in relation to alveolar process of left mandible causing bone destruction (fig 3).

Patient was referred to Department of Periodontology where the plaque and unstimulated saliva sample was taken and sent for the microbiological assessment. Collected sample was immediately spread over blood agar plate and divided into three parts and each part spread over blood agar plate, tripticase soya agar supplemented with 5% blood and thioglycolate broth for aerobic, anerobic and gram staining respectively.

Complete blood, urine and hormonal analysis was found to be within normal limits. The incisional biopsy for the lesion in the mandible under local anaesthesia was taken and was sent to department of Pathology in 10% formalin.The tissue was processed in automated tissue processor,paraffin blocks were made and sections were cut with microtome and sections were stained with Hematoxylin and Eosin for examination under light microscope. Histopathology showed lobular mass of fibroblasts with numerous osteoclast like multinucleated giant cells associated with haemorrhage and hemosiderin in subepithelium. Overlying epithelium was ulcerated (fig 4,5,6). Based on periodontal, radiological and

microbiological examination patient was diagnosed with RPP and was advised for total extraction. Under general anaesthesia, the lesion in mandible was excised completely from its base and all teeth in both jaws were extracted. The sockets were curetted for the removal of all the granulation tissue, debris and calculus. Primary closure was attempted and healing was uneventful (fig 7). Patient is under follow up for the last three years with no recurrence.

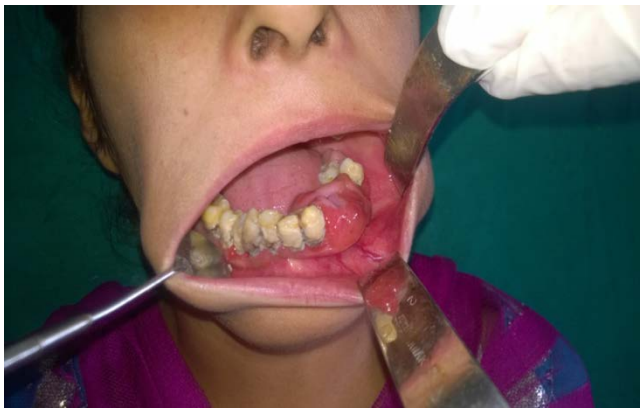

Figure 1: Preoperative clinical picture

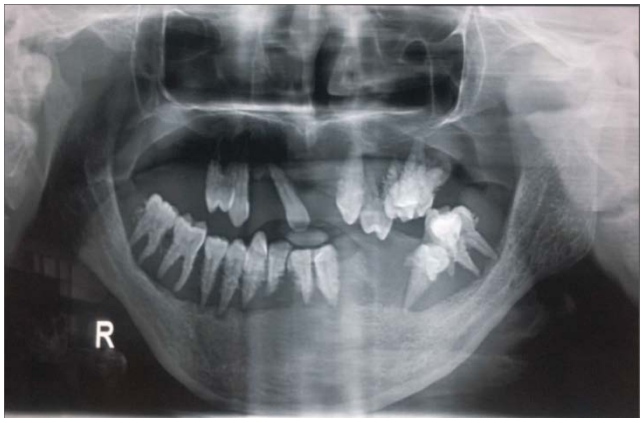

Figure 2: Orthopantomogram depicting the alveolar bone destruction

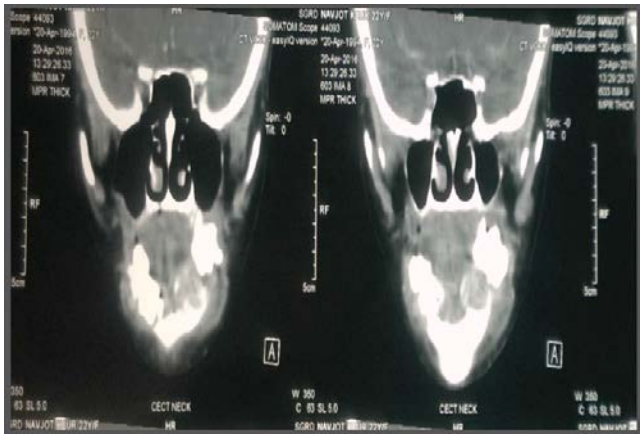

Figure 3: CECT facial bones with ill defined enhanced lesion in the left mandibular body region

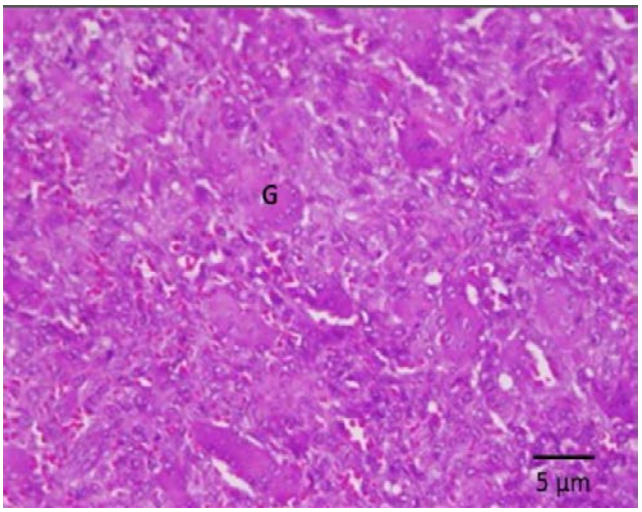

Figure 4: H & E section of the lesion showing Giant cell and proliferating fibroblasts x 40

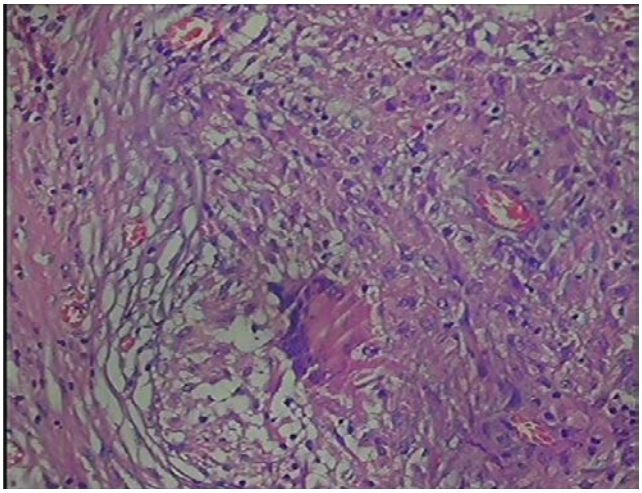

Figure 5: high power shows multinucleate cells and fibroblast and areas of haemorrhage x 40

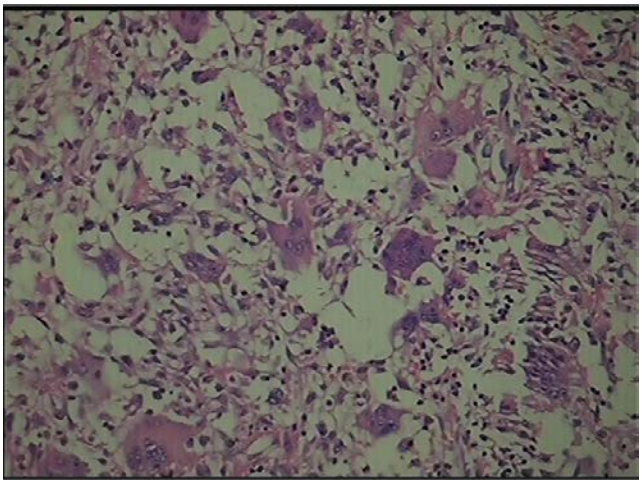

Figure 6: Light microscopic H& E sections shows fibroblast and giant cells x 40

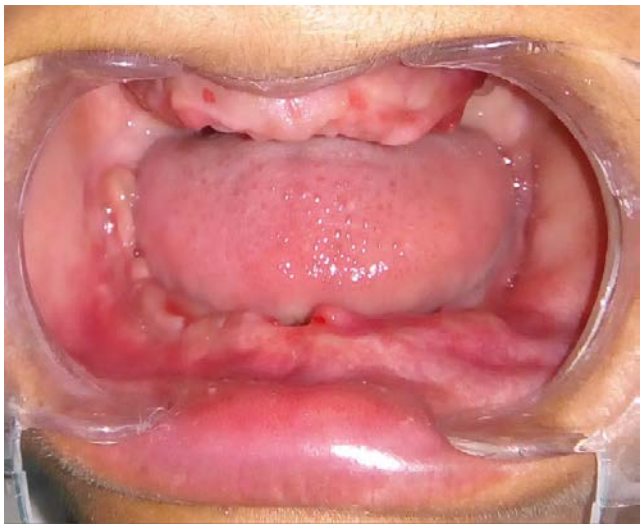

Figure 7: Uneventful healing one week postoperative picture.

Discussion

PGCG is a relatively common, non-neoplastic, inflammatory reactive lesion of oral cavity developing in relation to gingiva and alveolar mucosa both in dentate and non-dentate region in response to chronic irritation. It effects an age range of six to 88 years but most commonly seen in fourth to sixth decade of life with a slight female predilection.[2-3,6] Authors have stated that lesions which are anterior to the permanent molars are noticed early, have more incidence with a relative smaller size which could be due to the osteolytic activity present during the transition of deciduous to permanent dentition.[6]

The reported size of PGCG ranges from as small as half cm to large fungating growths of seven cm with superficial ulcerations.[2,6-7] Lack of awareness about oral hygiene with continuous irritation from plaque and calculus might have led to such a huge size in our case.

Lesions are mostly smooth, pedunculated sometimes sessile proliferations with a soft to firm consistency. They usually appear as red to purplish though pigmented lesions too are reported. It usually presents as painless swelling except if secondarily infected or develop ulcerations due to repeated trauma.[2-3,7] As it is a soft tissue lesion it rarely affects the underlying bone though long standing and large lesions can lead to superficial radiographically evident erosion.[2] The differential diagnosis for the present case included pyogenic granuloma, peripheral ossifying fibroma, peripheral ameloblastoma and Langerhans histiocystosis which were histopathologically ruled out.

The etiology of PGCG is not yet clear, but chronic irritation due to ill-fitting dentures, prosthesis, sharp restorations, sub-gingival and supra-gingival plaque and calculus play a major role as observed in our case.[2-3,7-8]

RPP usually presents at puberty to 35 years of age with a female predilection with 83% of patients with functional defects in neutrophils or monocytes. The gingival lesions are inflamed with a generalized pattern of distribution effecting most of the teeth causing mild crestal to severe extensive alveolar bone destruction. Local plaque irritation produces unusual gingival enlargements deepening the gingival pockets leading the sub-gingival bacteria to colonize and over grow which result in rapid alveolar bone loss leading to spontaneous teeth exfoliation. It presents as acute and remission phase after appropriate antibiotic coverage, oral prophylaxis and root planning procedures.4,5 Gram staining in the microbiological study performed in our case showed the presence of gram positive cocci and gram negative bacilli. In the aerobic culture, Streptococcus viridans was predominantly obtained after 24 hours of incubation at 37ºc. On anerobic culture, Porphyromonas gingivalis was followed by Provettala, Peptostreptococci and Capnocytophaga after 72 hours of incubation at 37ºc. Based on the culture report patient was prescribed appropriate antibiotic coverage prior to surgical intervention. Significant levels of antibodies to P.gingivalis, F.nucleatum, Streptococcus intermedius, Genellamorbillorum, Actinobacillus acetinomycetencomitans have been reported in literature in serum of patients suffering from RPP eliciting an aggressive aggravated host response depending upon the genetic, immunologic and environmental profile of the patient.[5]

Treatment modalities for RPP include root planning with complete oral prophylaxis and appropriate antibiotic coverage to decrease the subsequent acute bone resorption to lead into the quiescent phase and extraction of hopeless teeth, which was followed in our case.[4-5]

Complete surgical excision of lesion from its base along with the removal of local irritants is the treatment of choice to minimize the recurrence in case of PGCG.[2-3,7-8] Sclerotherapy using ethanolamine oleate when injected intralesionally produces fibrosis of endothelium of the blood vessels thus gives speedy and effective results in pendunculated lesions with a low recurrence rate.[7] Usage of lasers is not indicated for the curettage of deep lesions close to the bone.[9]

To conclude, general oral health awareness, timely referral, early diagnosis and intervention can decrease the destruction process in young patients suffering from PGCG and RPP.

References

1. Jaffe HL. Giant-cell reparative granuloma, traumatic bone cyst, and fibrous (fibro- osseous) dysplasia of the jaw bones. Oral Surg Oral Med Oral Pathol 1953; 6: 159-175.

2. Lester SR ,Cordell KG, Rosebush MS, Palaiologou AA, ManeyP. Peripheral giant cell granulomas: a series of 279 cases. Oral Surg Oral Med Oral Pathol Oral Radiol 2014; 118: 475-482.

1. Motamedi MH, Eshghyar N, Jafari SM, Lassemi E, Navi F, Abbas FM etal. Peripheral and giant cell granulomas of the jaws: A demographic study. Oral Surg Oral Med Oral Pathol Oral Radiol Endol 2007; 103:e39-43.

3. Page RC, Altman LC, Ebersole JL, Vandesteen GE, Dahlberg WH, Williams BL etal. Rapidly Progressive Periodontitis. A distinct clinical condition. J Periodontal 1983;54: 197-209.

4. Kamma JJ, Nakou M, Manti FA. Microbiota of Rapidly Progressive Periodontitis Lesions in association with clinical parameters. J Periodontol 1994;65:1073-8.

5. Gharbyah AZ, Assaf M. Management of a Peripheral Giant cell granuloma in the esthetic area of upper jaw: A case report. Int J Surg Case Rep 2014; 5: 779-82.

6. Ahmed WS. Efficacy of ethanolamine oleate sclerotherapy in treatment of peripheral giant cell granuloma. J Oral Maxillofac Surg 2016;74 : 2200-2206

7. Katsikeris N, Angelopoulou EK, Angelopoulos AP. Peripheral giant cell granuloma. Clinicopathologic study of 224 new cases and review of 956 reported cases. Int J. Oral Maxillofac Surg.1988; 17:94-99

8. Chaparro AV, Berini AL, Escoda CG: Peripheral giant cell granuloma: A report of five cases and review of literature. Med Oral Patol Oral Cir Bucal 2005;10: 53-7;48-52