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Case Report

PLASMA CELL GINGIVITIS- A CASE REPORT AND LITERATURE REVIEW

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ABSTRACT

Plasma cell gingivitis is an uncommon inflammatory condition of the gingiva, characterized by plasma cell infiltration in the gingiva. The etiology is largely unknown, and it is thought to be due to a hypersensitivity reaction to an allergen. The diagnosis is based on comprehensive history taking, clinical examination and appropriate investigations. Hereby, presenting a case of plasma cell gingivitis, its clinical and histopathology findings and treatment by gingivectomy and long term follow-up

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INTRODUCTION

Plasma cell gingivitis is an uncommon and benign condition characterized by well-demarcated gingivitis, most commonly extending up to the mucogingival junction[1].It was first reported in the early 1970s as plasmocytosis of the gingiva, idiopathic gingivostomatitis, plasma cell gingivostomatitis and allergic gingivostomatitis.[2] The etiology is largely unknown,but thought to be due to an allergen hypersensitivity.Some common allergens attributed are chewing gums, certain components of toothpastes, cinnamon, mint, red pepper, *khat*leaves etc..,[1]According to the etiology, this condition has been categorized into three groups:

1. PCG due to allergens.
2. PCG due to neoplastic origin.
3. PCG due to unknown cause

Case Report

An 18-year-old female patient came to the Department of Oral and maxillofacial surgery with a chief complaint of a growth in her gums in the upper and lower front teeth region for past two months associated with pain and bleeding. The patient initially noticed a small swelling of the gingiva which gradually increased to the current size. The past medical history was not significant. On clinical examination, edematous, incompetent and fissured lips were noted. Intraorally, the patient had poor oral hygiene and generalized plaque and calculus. Gingiva had a reddish pink appearance and bleeding on probing [Fig 1]. There were no other associated mucosal lesions.

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Orthopantamogram revealed no alveolar bone pathologies. Provisional diagnosis of wegeners granulomatosis (autoimmune disease) was made. Differential diagnosis included pyogenic granuloma, generalized chronic inflammatory gingival enlargement, desquamative gingival lesions, acute leukemia, multiple myeloma. A thorough hematological screening was performed that included Complete blood count, peripheral blood smear, viral markers and Antigen-antibody reaction. All were found to be within normal limits except for a mild elevation of leukocytes. Antigen-antibody reaction reveals negative.

Incisional biopsy of the lesion was done under local anesthetic coverage and the tissue was subjected to histopathology examination. Histopathological examination reported plasma cell gingivitis. [Fig. 2].The patient was instructed to avoid possible allergens like chewing gums, cosmetics and food additives. Topical application of 2% fusidic acid and systemic antihistamines was administered for four weeks to rule out if the lesion regressed. When there was no improvement, Gingivectomy was planned under local anesthesia after oral prophylaxis. Gingivectomy was done [Fig. 3] and the specimens were sent again for histopathology examination. Surgical site was packed with Coe-pak with antibiotics and analgesics prescribed for 5days. The final histopathological report confirmed the diagnosis [Fig. 4]. After 8weeks the patient reported with an uneventful healing [Fig.5].

DISCUSSION

Plasma cell gingivitis is a benign and uncommon condition of the gingiva. It is characterized by well-demarcated, erythematous and edematous gingiva most commonly

extending into the mucogingival junction. This condition was first reported by Kerr *et al* in 1981.[3]Histologically, it is marked by a dense,diffuse and massive infiltration of normal plasma cells separated into small clusters by strands of collagen into the subepithelial gingival tissue..Timms *et al* has classified plasma cell gingivitis into three types :

1. Due to allergens.
2. Due to neoplastic origin.
3. Due to unknown cause

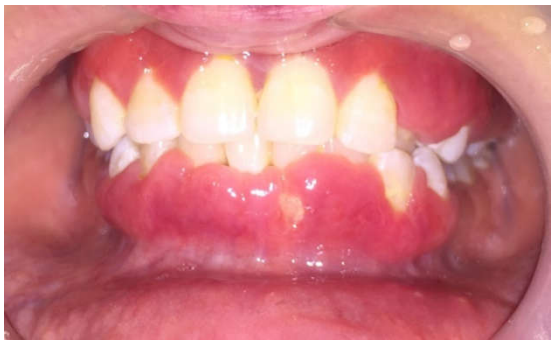

Figure 1 Preoperative clinical picture

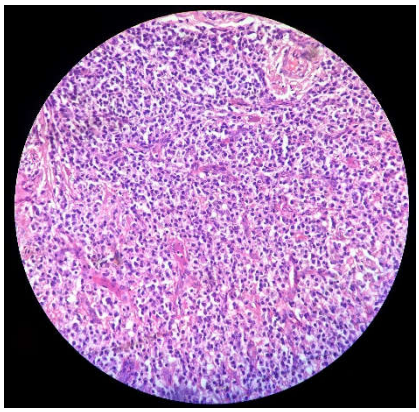

Figure 2 10x view- Incisional biopsy

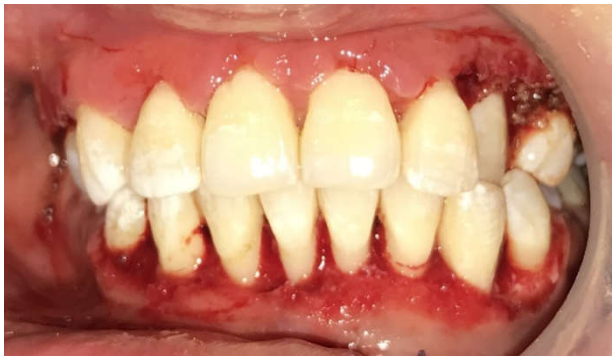

Figure 3 Intraoperative view

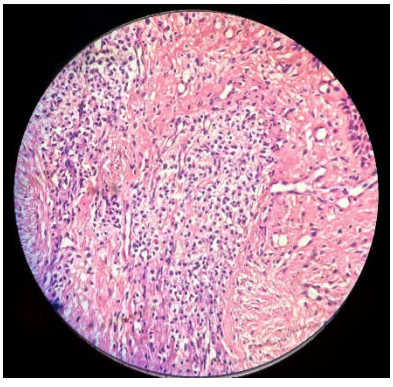

Figure 4 40X view-Excisional biopsy

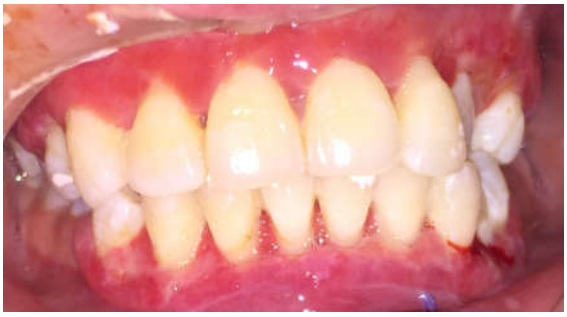

Figure 5 1 month Postoperative clinical picture

In our case, the patient reported with a diffuse, edematous and red swelling of maxillary and mandibular gingiva.Plaque induced gingivitis does not involve the entire width of the gingiva and involves only the marginal gingiva.The differential diagnosis with desquamative lesions was eliminated because of negative NIKOLSKY SIGN[4,5]. Hematological screeningis mandatory because such lesions may mimic that of acute leukemia so routine hematological examination along with antigen-antibody reaction was done thus excluding leukemia, lupus erythematosis and wegeners granulomatosis. Histologically this condition may imitate multiple myeloma, and extramedullary plasmacytoma..Plasma cell gingivitis should be diagnosed based on the histopathological features.Hence, the preoperative workup required a complete clinical, hematological and histopathological examinations [4,5].

Literature states that the plasma cell gingivitis is more common in anterior gingiva of the maxilla alone whereas in our case both the maxillary and mandibular gingival is affected [6].

Corticosteroids either topical or systemic have been useful in other mucosal lesion such as genital plasma cellular lesions, while oral plasma cellular involvement seems to be indifferent to this treatment [7,8].

Fusidic acid is a tetracyclic triterpenoic acid which prevents protein synthesis in both prokaryotic and eukaryotic cells. Also it lessens Interlekin-2 and interferon gamma production and reduces T-cell proliferation [10]. Chlorpheniramine is a first generation H1 antagonist which suppresses the H1 receptor. CPM is thought to be functional in plasma cell gingivitis since gingival fibroblast also express H1 receptors [9]. 2%fusidic acid cream as a topical agent along with H1 antagonist was prescribed for 4weeks but the spontaneous resolution of the lesion did not take place. The failure of response by the patient correctly to nonsurgical therapy necessitated a Gingivectomy.

Gingivectomy was done and the required specimens were sent for histopathological examination. Surgical site was packed with Coe-pak and antibiotics, analgesics were prescribed for 5days.

The final histopathological examination showed hyperplastic parakeratinized stratified squamous epithelium proliferating into the connective tissue in an arcading pattern. The connective tissue was denselycollagenous with focal dense collections of chronic inflammatory cells, chiefly plasma cells and also the cells were packed with plenty of microorganisms.

CONCLUSION

This case report emphasis on the necessity for a complete history taking, hematological examination along with clinicopathological correlation to achieve the final diagnosis. Gingivectomy followed by good oral hygiene measures is considered to be the definitive treatment in cases which does not respond to conventional therapy.

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