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Plasma cell gingivitis: A clinical and pathological case report

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Abstract
Aim: To describe the diagnosis and management of Plasma Cell Gingivitis with oral presentation.
Background: An uncommon benign condition known as plasma cell gingivitis (PCG) is typically observed on attached and marginal gingiva. Histopathologically, it is identified by the isnfiltration of plasma cells in connective tissue. Antigen-induced hypersensitivity reaction is regarded as the main etiological cause.
Case Description: Here the present case is of an 41-year-old female patient suffering from gingival enlargement along with high fever since last 15 days. Histopathological evaluation of tissue revealed lesion as plasma cell gingivitis. After giving medication the lesion heals gradually without any surgical intervention.
Conclusion: Early detection is crucial because plasma cell gingivitis shares pathologic features with leukaemia, multiple myeloma, discoid lupus erythematosus, atrophic lichen planus, desquamative gingivitis, and cicatricial pemphigoid that must be distinguished through hematologic testing.

Keywords: Gingival lesion, Hypersensitivity, Plasma cell gingivitis

Introduction

A very uncommon benign inflammatory disorder of the gingiva is called plasma cell gingivitis (PCG). Atypical gingivostomatitis, idiopathic gingivostomatitis, allergic gingivostomatitis, and plasma cell gingivostomatitis are a few synonyms for plasma cell gingivitis [1]. The lesion was initially described by Zoon in 1952, who used the phrase "plasma-cell infiltrate." Lips, tongue, vulva, conjunctiva, nasal mucosa, larynx, and epiglottis have also been documented to exhibit these lesions [2]. Allergens that produce hypersensitive reactions which will lead to PCG. Chewing gums, particular toothpaste ingredients, cinnamon, mint, red pepper, and khat leaves are a few of the allergies that have been identified [3]. However, depending on the aetiology, PCG has been divided into three categories: PCG caused by allergens, neoplastic origin and an unknown cause [4]. Clinically, PCG is distinguished by erythematous and edematous gingiva that are well defined and frequently extend to the mucogingival junction. Moreover, the gingiva appears red, friable, and bleeds easily on provocation [1]. An early identification in such circumstances is essential because PCG mimics lesions linked to discoid lupus, lichen planus, cicatricial pemphigoid, leukaemia, and myeloma [1]. This case involves a PCG in a 41 years old female patient with enlargement of gingiva.

Case Description

One female patient, age 41 years, reported to the department of Periodontics with chief complaint about severe swelling of gums since last one month with fever. (Fig 1). During the intraoral examination, it was discovered that the middle third of the clinical crowns were covered by generalised, significant gingival hypertrophy. Gingiva was fiery red, oedematous, and friable, with the absence of stippling, and easily bleeds on slight provocation. Probing depth ranged from 4-5 mm and an attachment loss approaching 5-6 mm was recorded. No mobility of teeth was present. There is not much evidence of involvement of local factors. The patient's personal, medical, and dental histories were not relevant. An investigative hematologic analysis produced no notable results. Nikolsky's sign was negative and there was no cutaneous lesion. Gingival biopsy has taken for provisional diagnosis.

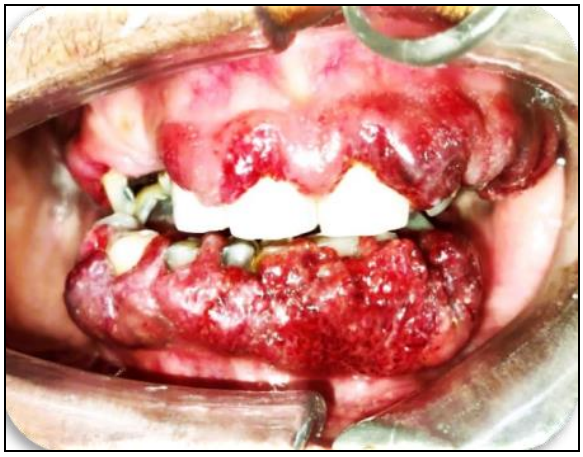

Fig 1: Pre-Operative buccal view

Fig 2: Pre operative view Lingualside

According to histopathological analysis, the squamous epithelium was parakeratinized, stratified, and of varied thickness, with areas of thinned-out epithelium and distinct rete ridges (Fig 3,4). Connective tissue was made up of a dense, chronic, inflammatory cell infiltrate that was mostly made up of plasma cells, lymphocytes, collagen fibres, and blood vessels with endothelial lining. The individual plasma cells had eccentric round nuclei with cartwheel chromatin patterns and abundant cytoplasm. The aforementioned characteristics pointed towards plasma cell gingivitis [PCG].

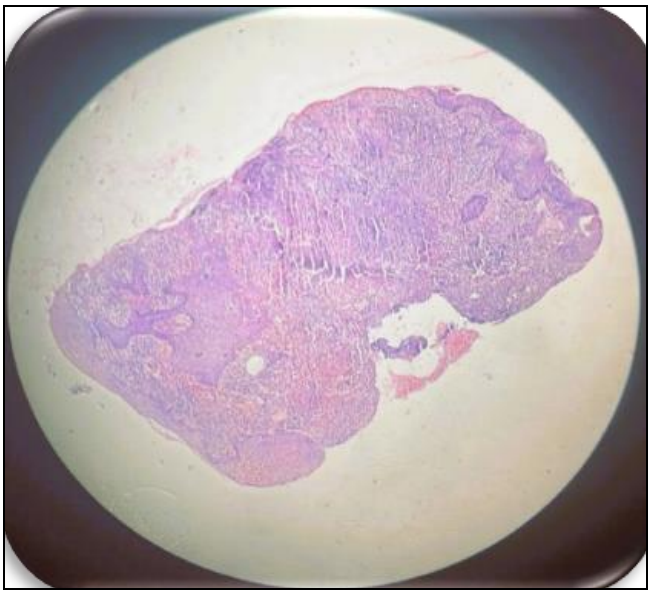

Fig 3: Histopathological View Low Magnification

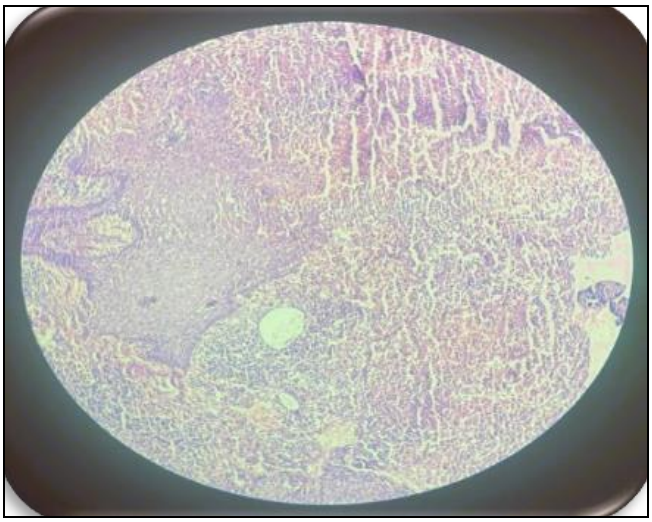

Fig 4: Histopathological View High Magnification

Scaling and root surface debridement were performed as part of phase I therapy, and the patient was instructed to continue brushing twice daily with a soft-bristled toothbrush and rinsing with 0.2% chlorhexidine for two weeks. Antihistaminic drug and non steroidal anti inflammatory drug has given as a part of palliative treatment. Patient has recalled after 14 days and the lesions started to regress. After one month follow up the lesion was healed completely [Fig-5].

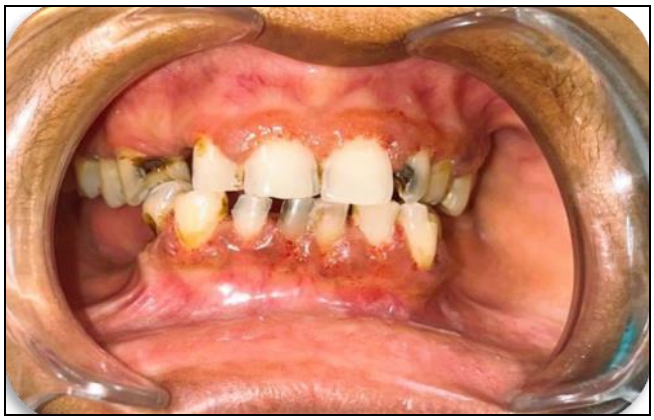

Fig 5: Post operative view after 3 Month

Discussion

A rare inflammatory disorder called PCG is characterised by widespread and intense infiltration of plasma cells into connective tissue [5]. In 1981, Kerr and Kenneth found gingival hypertrophy in gum chewers, which went away after the habit was broken [6]. The lesion typically develops as a result of a local irritant; if the patient is aware of the irritation early, the lesion may spontaneously disappear with nonsurgical periodontal therapy [7]. Candida albicans may play a role in the progression of PCG, according to Silverman *et al*. [8]. The herpes virus was also regarded by Jayaraman *et al*. as a causal factor [9]. Last but not least, the pathogenesis of PCG may involve prolonged mechanical irritation [10]. Numerous occurrences of PCG, particularly in locations where plaque was abundant, were linked to chronic generalised periodontitis [11]. Typically, plaque-rich areas have a plasma cell infiltration, which may make the lesion to easily differentiate [12].

Numerous treatment approaches, including corticosteroids, antibiotics, laser destruction, electrocoagulation by cautery, excision of the tissue with a standard surgical knife, and even radiation therapy, were suggested to control PCG.

Corticosteroids are widely used with unfavourable outcomes. Using topical or intralesional steroids did not alleviate symptoms at all, according to a systematic assessment of the literature published in 1986 [10]. In other studies, corticosteroids were effective in shrinking the lesion, however this shrinkage was accompanied by scarring [13]. With chemotherapy and steroids (prednisolone), Fogarty *et al*. handled a case of PCG involving the larynx and achieved a brief decline; nevertheless, symptoms returned after ceasing the therapy [14].

Based on the theory that the tumour developed as a result of a Candida infection, topical antifungals were employed. Nystatin used topically, however, was ineffective [10].

Low dosage radiation therapy was employed to treat the PCG, and this led to a relative improvement. However, surgical excision, regardless of the method—conventional surgical blades, electrocoagulation, CO2 laser, or cryotherapy—remains the gold standard in the long run [11].

In the current report, phase 1 periodontal therapy has performed to remove the associated plaque in order to decrease any chance of recurrence or plaque induced exacerbation. The patient was instructed to change feeding habits and avoid to use new kind of tooth abrasive to avoid the possibility of exacerbation by unknown allergen.

Conclusion

PCG mimics a number of other deadly diseases, including leukaemia and multiple myeloma, thus early detection and effective treatment of the lesion are essential. To rule out other lesions and make an accurate diagnosis, a thorough case history must be performed in addition to haematological, histological, and immunohistochemical testing.

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