EDELWEISS PUBLICATIONS
OPEN ACCESS

https://doi.org/10.33805/2572-6978.149
Volume 4 Issue 2 | PDF 149 | Pages 4

Dental Research and Management

Case Report ISSN: 2572-6978

Plasma Cell Gingivitis as a Predisposing Factor for Plaque-Induced Periodontitis: A Case Report

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Citation: Bonilla VAU, Arambu C, Romero H and Guifarro JJ. Plasma cell gingivitis as a predisposing factor for plaque-induced periodontitis: A case report (2021) Dental Res Manag 4: 71-74.

Received: Nov 24, 2020
Accepted: Jan 13, 2021
Published: Jan 18, 2021

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Abstract

Plasma cell gingivitis is a benign lesion of unknown etiology characterized by massive infiltration of plasma cells into the connective tissue of the gingiva. Clinically it presents as a gingival enlargement with erythema and some areas with the presence of desquamation, it is usually asymptomatic, but on some occasions the patient may present pain and gingival bleeding. Diagnosis requires clinical-pathological correlation. Based on the foregoing, we present a case report of a 25-year-old female patient diagnosed with plasma cell gingivitis with idiopathic etiology based on the clinical and histopathological study.

Keywords: Plasma cell gingivitis, Periodontal ligament, Periodontitis, Oral pathology

Introduction

Plasma cell gingivitis is also known as atypical gingivostomatitis, idiopathic gingivitis and allergic gingivitis. It is a rare benign condition of the gingiva of unknown etiopathogenesis, characterized by the proliferation of plasma cells in the connective tissue. Identified for the first time as a mucosal hypersensitivity reaction associated with secondary cheilitis in the 1940s and 1950s, it had its peak between 1966 and 1971, with more than 50 cases reported [1-5].

Its relationship with an unknown allergen present in different chewing gums flavored with cinnamon and mint was established. The pathology is mentioned as a transitory syndrome that is attributed to purification in the formula of different products such as chewing gums, sweets and toothpastes that until now appear as the main etiological suspects. Some reports also suggest plaque as an allergenic factor [6-8]. Clinically it presents as an erythematous enlargement that affects marginal and adherent gingiva, generally accompanied by epithelial desquamation, and the patient generally complains of pain, tenderness, and bleeding when brushing.

The diagnosis requires a histopathological study characterized by epithelial hyperplasia with an underlying stroma with the presence of a dense chronic inflammatory infiltrate in which plasma cells of normal morphology predominate [9]. The clinical pathologic correlation is essential to differentiate from clinically similar lesions such as pemphigus, membrane pemphigoid, allergic or lichenoid reaction, leukemia. Histologically, it can mimic multiple myeloma and solitary plasmacytoma [10].The objective of this report is to present the clinical case of idiopathic plasma cell gingivitis and its clinical characteristics, histopathological study with immunohistochemistry.

Case Report

A 25-year-old female patient who attended the private practice for the main reason for the consultation who presented desquamative erythematous gingival hyperplasia with generalized bone loss with an evolution time of 8 years refers to undergoing multiple nonsurgical and surgical periodontal treatments without responding to this therapy (**Figure 1, Figure 2 and Figure 3**).

Figure 1: Initial photo facial view.

Figure 2: Initial photo right side view.

Figure 3: Initial photo left side view.

The patient has no relevant medical history and complementary laboratory tests showed normal levels, so leukemia or other hematological alterations are ruled out. The extra oral clinical examination showed no changes in skin color, facial symmetry and no lymphadenopathy were palpated, the intraoral clinical examination showed generalized inflammation and a complete periodontal probing was performed in which there is an interproximal clinical insertion loss. Detectable in 2 or more teeth and there is clinical attachment loss >3 mm or with a pocket >3 mm in 2 or more teeth. The periodontal diagnosis is stage III periodontitis because it presents a radiographic interproximal insertion loss >5 mm, a generalized horizontal bone loss can be observed that extends to the middle of the roots >30% of the teeth, there is furcation of the teeth 16, 14, 24, 26, 36 and 46 (**Figure 4**).

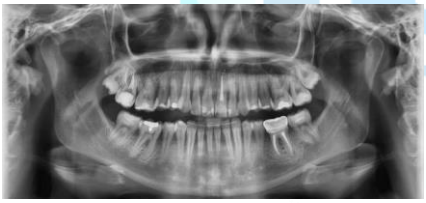

Figure 4: Panoramic radiography.

There is the possibility of losing teeth, the chewing function is preserved and the treatment of periodontitis does not require complex rehabilitation of the function. Regarding the degree of progression, it is classified as grade C, there is indirect evidence of bone destruction is not consistent with the amounts of plaque and calculus, some destruction patterns suggest periods of rapid progression, and it has not responded to periodontal therapeutic controls. No other lesions were observed in the oral cavity. The presence of a negative Nikolsky sign and the absence of cutaneous lesions ruled out mucocutaneous diseases.

Due to the clinical aspect, time of evolution, not responding to periodontal therapies and that a definitive etiological factor was not known, an incisional biopsy was performed, under a local anesthesia was given with 4% articaine with 1: 100.00 epinephrine. The biopsy was chosen between pieces 21 and 22, taking a representative lesion with tissue from the lesion and healthy tissue at the apical edge of these (**Figure 5**). Exodontia was carried out on tooth 16 that showed tooth mobility; it was observed at the time of extraction that the tooth was surrounded by granulation tissue (**Figure 6**). Both were subjected to histopathological study.

Figure 5: Preoperative view.

Figure 6: Exodontia of tooth 16 shows granulation tissue at the apical level.

The histopathological study in hematoxylin and eosin showed the presence of hyperplastic parakeratinized stratified squamous epithelium on a stroma that presents a dense inflammatory infiltrate with a predominance of plasma cells (**Figure 7a and Figure 7b**). The histopathological result confirms the diagnosis of plasma cell gingivitis. Immunohistochemical markers for kappa and lambda light chains were performed to confirm the presence of plasma cells and reaffirm the diagnosis (**Figure 8a and Figure 8b**).

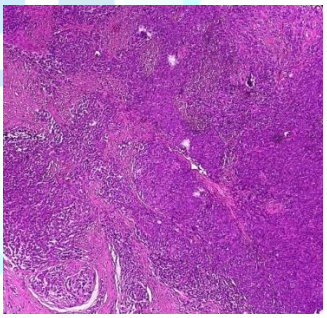

Figure 7a: Histopathological examination at 10x magnification shows subepithelial inflammation.

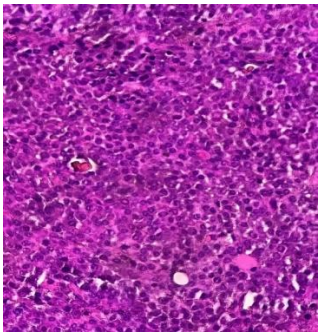

Figure 7b: Histopathological examination at 40x magnification shows a dense inflammatory infiltrate in which plasma cells are mostly observed.

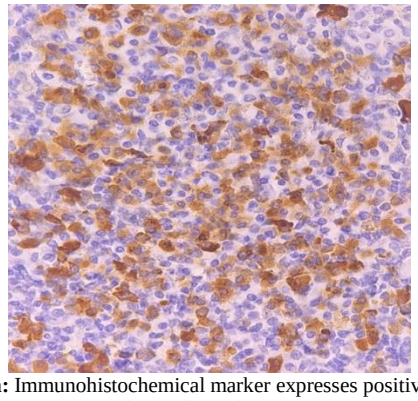

Figure 8a: Immunohistochemical marker expresses positivity for the kappa light chain ƙ.

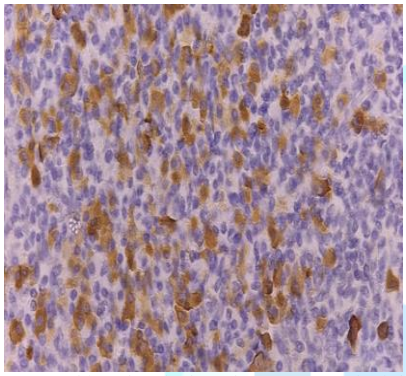

Figure 8b: Immunohistochemical marker expresses for λ lambda light chain.

Fifteen days postoperatively after taking the biopsy, the patient attended a follow-up appointment for assessment, showing in the extraction area of tooth 16 a gingiva with a decrease in size and volume, as well as in the intensity of staining (**Figure 9**), the lesions In the anterior region where the incisional biopsy was taken which showed no signs of any recurrence of the enlargement.

Figure 9: Postoperative follow-up.

Outcome

The management of plasma cell gingivitis is directed to the symptoms that the patient have, corticosteroid treatment is the most frequently used. Plasma cell gingivitis is a rare pathology that can be similar to multiple benign and neoplastic conditions, so a correct anamnesis and multidisciplinary investigation are necessary for its diagnosis.

Discussion

Plasma cell gingivitis is a rare benign condition of unknown etiology [11]. It is described as a benign, painful mucosite of plasma cells in the gingiva [1]. Clinically, it presents as an edematous and erythematous gingival enlargement in the maxillary and mandibular segments [12]. The etiology of this pathology is unknown, due to the intense presence of inflammatory cells it is believed that its origin is due to an allergic reaction as possible allergens have been associated with gum flavorings, toothpastes and menthol mouthwashes [6]. The differential diagnosis of these lesions is important due to their similarity to other pathologies; other lesions such as mucocutaneous vesicles in the absence of Nikolsky's sign and other malignant lesions were ruled out by histopathological, immunohistochemical and complementary hematological examinations [13,14].

Histopathological results showed a connective tissue with a dense inflammatory infiltrate, mostly plasma cells, confirming the diagnosis of plasma cell gingivitis [15]. Confirmation of plasma cell gingivitis requires immunohistochemistry showing polyclonal expression of the Kappa and Lambda chains which are free light chains of immunoglobulins that are considered markers of plasma cell activation. The results of the immunohistochemistry determined the polyclonal expression of the kappa and lambda light chains in a ratio of 2:1 suggestive of an inflammatory etiology [16]. Monoclonal expressions of plasma cells are observed in neoplasms such as multiple myeloma and extramedullary plasmacytoma in a ratio of 10:1 [17].

Three types of plasma cell gingivitis have been described: caused by an allergen (flavored gums, toothpaste, and mint-flavored mouthwashes), neoplastic and of unknown etiology [18-20]. The management of patients with plasma cell gingivitis is based on their symptoms, possible allergens and plaque control, which, as in our case, led to periodontitis and should be eliminated with the intention of observing possible causes.

Conclusion

Plasma cell gingivitis is an unknown pathology and is occasionally reported in the literature, it is important as dental personnel to recognize this pathology in which its diagnosis is based on the clinical-pathological correlation, as well as interdisciplinary management between the different diagnostic specialties for the correct treatment plan.

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