

Plasma Cell Gingivitis Due to an Antitartar Toothpaste: An Unusual Case Report

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Abstract

Plasma cell gingivitis is a rare form of atypical gingival inflammation which is characterized by erythematous marginal gingival hypertrophy which may involve the adjacent attached gingiva. Plasma cell gingivitis (PCG) is an atypical form of gingivitis and is characterized by massive infiltration of plasma cells into the subepithelial connective tissue. It is a rare condition; the cause of which is still not fully understood. It is often attributed to an allergic reaction to the components of chewing gums, dentifrices or various diet components. A case of PCG is presented here in a 52-year old male who switched to an anti-tartar toothpaste on the advice of a dentist which manifested in a generalized form. This report is the first, as far as we know, that relates PCG to the use of Glister toothpaste manufactured by Amway. The histological examination revealed the infiltration of polyclonal plasma cells. The PCG disappeared within two weeks of stopping the use of the dentifrice.

Keywords: Plasma cell gingivitis; Anti-tartar toothpaste; Glister toothpaste.

Introduction

The increase in the size of gingiva is a common feature of gingival inflammation. Several causes for gingival hyperplasia have been incorporated in the dental literature like an inflammatory, drug-induced, neoplastic enlargements, systemic diseases like leukemia^{1,2} chronic kidney disease patients on immunosuppressants,³ to name a few. Among them, few are uncommon and come under the category of conditioned enlargement. Plasma Cell Gingivitis (PCG), also known as plasma cell gingivostomatitis is an abnormal type of gingival

inflammation which involves marginal as well as attached gingiva. The exact cause of its appearance is not known, but it is believed to be an allergic or exaggerated response to the ingredients of chewing gum, toothpaste/ or maybe to some dietary elements.

In the early 1940s and 1950s, many reports of mucosal hypersensitivity to edible gum had come out. The cinnamon compounds in them, as well as, toothpaste were identified as the etiological agents in causing PCG. The histopathology revealed a dense penetration of plasma cells separated by collagenous stroma seen predominantly in the keratinized gingiva. These findings can mimic some fatal diseases like multiple myeloma and solitary plasmacytoma, and hence a thorough case history to rule out these conditions is warranted.

Similarly, desquamative gingivitis has also been reported with the use of anti-tartar dentifrices. Pyrophosphates and flavouring agents have been identified as the offending agents. Oral reactions to cinnamon compounds (cinnamon oil, cinnamic acid, cinnamic aldehyde)⁴⁻⁶ used to mask the taste of

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pyrophosphate produce intense erythema of the gingiva characteristic of PCG as seen in this particular case.

Case Report: A 52-year-old man reported to the outpatient department of Periodontics and Oral Implantology with the chief complaint of bleeding and swollen gums for one month. The medical and dental history was non-contributory. There was a particular emphasis on diabetic status as diabetes significantly increases the chances of periodontal disease as compared to non-diabetic patients.⁷ The patient did not report of any deleterious habits like smoking, alcohol intake or any form of tobacco usage. He was well built and nourished and was cooperative. He had undergone scaling six months back. Extraoral examination showed no gross facial asymmetry with a straight profile and competent lips. Upon further asking, he told that he had started using Glister toothpaste around two months back and had developed swollen gums 2-3 weeks after that. Intraoral examination revealed strawberry red coloured enlargement of gingiva present throughout with rolled out marginal gingiva and blunt interdental papilla. Gingiva appeared friable, granular and bled easily, and desquamative gingivitis was also present (Figure 1). He was advised to change the toothpaste in his daily regimen for two weeks. There were minimum plaque and calculus, which was not consistent with the inflammation seen in the oral cavity. No cutaneous lesions were present elsewhere. Complete hemogram and blood sugar levels revealed no abnormalities and were well within normal limits.

A provisional diagnosis of Severe chronic generalized gingivitis was made. A conservative approach was planned initially with patient education, motivation followed by phase one therapy. The patient was also instructed to rinse with 0.2% chlorhexidine twice daily. Since the patient was using anti-tartar toothpaste, we raised our suspicion towards PCG and hence a biopsy was taken from the representative site. On the persistence of the symptoms after two weeks of phase one therapy, the patient was scheduled for a biopsy of the lesion. The procedure was carried out under strict asepsis, and all the protocols were followed. The patient was administered local anesthesia, and after subjective symptoms were positive, we used a Bard parker handle and a No 15 BP blade, gingival tissue was removed from the attached gingiva of the maxillary anterior region and was subsequently sent for histopathological examination. (Figure 2) The patient was advised routine post-operative instructions and necessary medications were prescribed. While the histopathological reports were awaited scaling and root planing was continued. In the subsequent visits, the patient reported improvement in the condition. Histopathology revealed an abundant congregation of plasma cells in the connective tissue without the presence of any atypical cells, and hence a final diagnosis of Plasma Cell Gingivitis was made. (Figure 3) Within one month of the stoppage of the anti-tartar toothpaste, the lesion completely subsided without any treatment. (Figure 4).



Figure 1. Initial diagnosis for PCG



Figure 2: Gingival tissue for histopathological examination

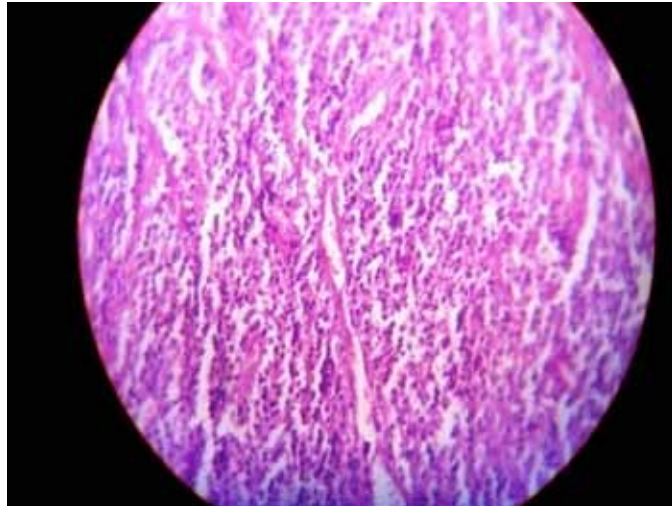


Figure 3: Histopathological examination



Figure 4

Discussion

Plasma cell gingivitis is an uncommon and rare condition that manifests clinically as a diffuse erythematous gingival lesion which may or may not extend to the mucogingival junction.⁸ The number and infiltration of plasma cells vary in various cases and are said to result from an allergic hypersensitivity reaction to various flavouring agents used in chewing gums and dentifrices. Lamey et al. 1990⁶ and Miller RL 2002⁹ reported that Cinnamonaldehyde, which is usually added to dentifrices to mask the unpleasant taste of pyrophosphate, has been associated with the development of PCG. The etiology is primarily unknown but is believed to be associated with the cinnamon compounds used to mask the taste of pyrophosphates in anti-tartar toothpaste. Macleod and Ellis in 1989 reported a case related to the use of herbal toothpaste. Cinnamon, when used as a flavoring agent in toothpaste, was found to be an etiologic factor in gingivitis.⁸ Miller et al. had reported 14 cases of cinnamon induced stomatitis.⁹ The present case also had desquamative gingivitis which further stresses the role of cinnamon compounds for the same as the patient was using anti-tartar toothpaste. The differential diagnosis of the condition is critical because of its similarity with other mucosal lesions. Most cutaneous disorders were eliminated for consideration by the absence of skin lesions, a negative Nikolsky sign and no abnormalities in the hemogram in this case. Differential diagnosis needs to be done to exclude it from other aggressive lesions such as pemphigus, and once the diagnosis of PCG is confirmed, we should scout for the antigenic substance. In this particular case, the patient had switched to anti-tartar toothpaste (Glister from Amway), and that was the only contributing history.

Conclusion

This case again stresses the role of history taking in arriving at a correct diagnosis. Irrational use of herbal or anti-tartar toothpaste should be avoided, and as the use of herbal products are on the rise. Early diagnosis of plasma cell gingivitis is essential because it mimics other aggressive diseases like leukaemia and multiple myeloma.

Conflict of Interests: None

Ethical Permission: Approved

Funding: Nil

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