

Case Report

Contact hypersensitivity: local reaction on gums

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ABSTRACT:

Contact hypersensitivity is a rare form of atypical gingival inflammation which is characterized by mild erythematous gingival involving the attached gingiva to atypical erythematous enlargement 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. Two documented case of contact hypersensitivity who switched to an anti-tartar toothpaste on the advice of a dentist manifested from mild gingival erythema to gingival enlargement. The histological examination revealed infiltration of polyclonal plasma cells. The symptoms disappeared within two weeks of stopping the use of the dentifrice.

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INTRODUCTION

Allergic contact stomatitis might be caused by a wide range of substances, including the aromatic compounds like carvone, cinnamonaldehyde and cinnamon essential oil. Formaldehyde, the acrylates used in making dentures and several metals including nickel, palladium, gold and mercury used in dental amalgam might also causes Contact Stomatitis. Allergic contact sensitivity was a hypersensitivity reaction (type IV) that affects only individuals who had previously been sensitized to the allergen.¹ Some practitioners used patch tests to confirmed the diagnosis of allergic contact stomatitis.¹

Contact Stomatitis might easily develop on keratinized as well as on non-keratinized mucosa mainly affecting the lateral edges of the tongue, the attached gingiva and buccal mucosa. The clinical features of allergic Contact stomatitis included oedema, erythema, cracking, ulcerative areas, hyperkeratosis in the form of plaques or striations, desquamation and vesicles¹. The histopathological pictures reveals hyperorthokeratosis, acanthosis or atrophy, all of which might be accompanied by liquefactive degeneration of the basal layer. Ulcerated areas were characterized by fibrinopurulent exudates. A

perivascular lymphocytic infiltrate might also be present.¹

Plasma cell gingivitis (PCG) is an atypical and rare form of gingival enlargement and is characterized by massive infiltration of plasma cells into the subepithelial connective tissue. Gingival ulceration is rare. Several causes for gingival hyperplasia have been incorporated in the dental literature like an inflammatory, drug-induced, neoplastic enlargements, systemic diseases like leukaemia,^{2,3} chronic kidney disease patients on immunosuppressants.⁴ The aetiology was difficult to elicit as it might be related to specific allergens like flavouring agents such as cinnamonaldehyde and cinnamon present in chewing gum, toothpastes etc.⁵⁻⁸ Pyrophosphates and flavouring agents have been identified as the offending agents. Oral reactions to cinnamon compounds (cinnamon oil, cinnamic acid, cinnamic aldehyde)⁹⁻¹¹ used to mask the taste of pyrophosphate produce intense erythema of the gingiva characteristic of PCG as seen in this particular case. It might be of unknown origin also.^{5, 8} Kerr et al. reported a case of PCG or plasmacytosis of the gingiva in 1971. There had been reports of occurrence of PCG due to the chewing of khat leaves, which was popular in many African countries⁷ as seen in this particular case.

The classic presentation of PCG was an asymptomatic diffuse, erythematous and oedematous appearance of gingiva in the anterior maxilla with bleeding on minimal manipulation.⁷

The histopathology revealed a dense penetration of plasma cells separated by collagenous stroma seen predominantly in the keratinized gingiva.⁷ 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.

CASE REPORT 1

A 45-year-old man reported to the outpatient department of Periodontics and Oral Implantology with the chief complaint of bleeding and reddish gum 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.¹² 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. Upon further asking, he told that he had started using anti tartar (PYX-G) toothpaste around five months back and had developed reddish discolouration 2-3 weeks after that. Intraoral examination revealed darkish red discoloured marginal and attached gingiva. (Fig.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.

CASE REPORT 2

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. The patient did not report of any deleterious habits like smoking, alcohol intake or any form of tobacco usage but gave a history of 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. (Fig.2) 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 conservative approach was planned for both the cases 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 patients was using anti-tartar toothpaste and the symptoms persisted 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 lesional and perilesional tissue was removed from the attached gingiva of the maxillary and mandibular anterior region respectively and was subsequently sent for histopathological examination. 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 manifested hyperorthokeratosis, acanthosis accompanied by liquefactive degeneration of the basal layer (fig 3).¹

Histopathology revealed 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. (Fig.4) Within one month of stoppage of the anti-tartar toothpaste, the lesion completely subsided without any treatment.



Figure 1 shows contact sensitivity reaction manifested as desquamative gingivitis



Figure 2 shows plasma cell gingivitis (gingival enlargement) manifested as desquamative gingivitis

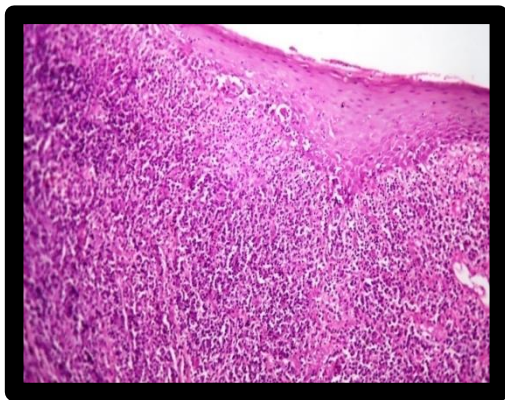


Figure 3 shows vacuolar degeneration with acanthosis

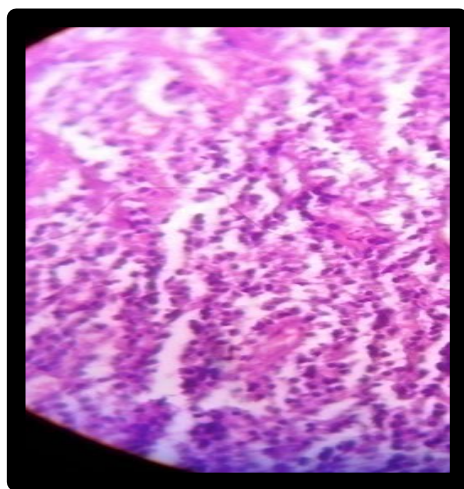


Figure 4 shows presence of plasma cells in H&E stained slide of plasma cell gingivitis (100X magnification)

DISCUSSION

Gingival hypersensitivity is an uncommon and rare condition which manifests clinically as a diffuse erythematous gingival lesion or may manifest as gingival enlargement which may or may not extend to the mucogingival junction.¹³ The present cases had desquamative gingivitis which stresses the role of cinnamon compounds for the same as the patient was using anti-tartar toothpaste. Lamey et al. 1990¹¹ and Miller RL 2002¹⁴ 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 etiological factor in gingivitis.¹³ Miller et al. had reported 14 cases of cinnamon induced stomatitis.¹⁴ In cases of hypersensitivity enlargement 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. 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 toothpaste¹⁵(Glister and PYX - G), 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 contact stomatitis, plasma cell gingival enlargement is essential because it mimics other aggressive diseases like PEMPHIGUS, leukaemia and multiple myeloma.

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