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Desquamative gingivitis: A review

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Abstract
Desquamative gingivitis (DG) is characterized by the erythematous gingiva, desquamation and erosion of the gingival epithelium, and blister formation. It is a common clinical manifestation in several diseases. Contact allergic reactions to various oral hygiene products and chemical agents have also been reported to represent as DG. The management of DG has been a major problem, largely because the etiology of the disease has been elusive. In this paper, my aim was to review the current literature on the pathogenesis, diagnosis, management and prognosis of DG.

Keywords: *Desquamative gingivitis, Mucocutaneous diseases, Contact allergic reactions.*

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I. Introduction

The term "desquamation" is derived from the Latin word 'Desquamare', which means scraping fish flakes. As a word, desquamation means 'loss of epithelial elements in small and large amounts, peeling of skin, and exfoliation'[1]. Chronic desquamation of the gingiva is referred to as desquamative gingivitis (DG)[2]. Chronic desquamative gingivitis was described for the first time by Tomes and Tomes in 1894[3]. In 1932, Prinz used the term 'chronic diffuse desquamate gingivitis' for chronic diffuse inflammation cases, which were characterized by severe epithelial desquamation in the marginal gingiva[4]. In actual use, the term 'Desquamative gingivitis' is used for a specific clinical symptom and it is not a diagnosis alone [2,3,5,6,7]. DG is a clinical finding, which progresses with vesicular formation, atrophy, erosion and desquamation, characterized with diffuse erythema of the marginal and keratinized gingiva [6,8,9,10]. Lesions start with diffuse erythema and minimal desquamation. The affected gingiva epithelium is very fragile and tends to exfoliate easily, even with the slightest trauma [3]. Large ulceration areas can be observed in some cases [11]. The desquamative gingivitis is seen after puberty, especially in individuals over 30 years of age [3]. It is more common in women than in men. It has been reported that it can rarely be observed in children [2,3,8,12].

Desquamation of epithelial tissue is generally seen in free and keratinized gingiva. Generally, the lesions that affect the buccal/labial surfaces of the gingiva, although not formed due to bacterial plaque, are exacerbated with plaque accumulation [13]. Although they are generally observed in the anterior region, they can be seen in any gingival area [6]. Similar lesions can be seen in the edentulous alveolar ridge. While only desquamations can be observed in the patients, there can also be associated vesicular-bullous lesions, in addition to ulcerative and lichenoid lesions. In severe cases, it can be seen generalized at the oral mucosa, and the alveolar mucosa can be affected together with the gingiva [13]. The patient can either have no complaints or there can be a burning sensation or severe pain. In general, there is chronic pain, which especially increases with the intake of acidic foods. Limitation of oral function and speech difficulties due to pain can also be observed [2,6]. Nisengard and Levine [14] have mentioned some characteristics in order to diagnose the clinical findings as DG, since gingival erythema is not associated with plaque and presence of gingival desquamation. They also emphasized that the Nikolsky phenomenon is generally positive in DG patients. Only a single etiologic factor was considered in the first desquamate gingivitis (DG) cases. Initially, it was suggested that gingival desquamations were related to hormonal changes due to menopause on the basis that gingival desquamations were more common in the middle-aged and in women [13]. In 1964, Glikman and Smulow[15] stated that DG could be the symptom of severe conditions, especially mucocutaneous diseases. Recently, it has been generally accepted that DG can be the initial symptom of vesicular bullous diseases and can emerge as a result of reactions against some chemicals and allergens, and that it is not related to hormones.

Differential diagnosis and prognosis

The differential diagnosis of desquamative gingivitis (DG) includes a wide spectrum, such as chemical and electrical burns, allergic reactions, hormonal disorders and mucocutaneous diseases. Furthermore, a similar clinical pattern can be observed in reactions developing against mouthwashes (figure 1) chewing gums, cosmetic products, drugs, cinnamon and dental materials[16]. It is suggested that the disease may be observed when there is lack of estrogen or progesterone[17]. Additionally, there are idiopathic gingival desquamative

lesions without any etiologic factors (figure 2,3). There are conflicting arguments on whether it is a symptom of oral lichen planus (figure 3), mucous membrane pemphigoid (Figure 4), or a clinical manifestation of these diseases [12,18]. In many articles, it has been reported that DG is related to lichen planus, mucous membrane pemphigoid and pemphigus vulgaris (88% - 98%).[6,9,13,16]

The definitive diagnosis of desquamative gingivitis is very difficult and complicated. Determination of the etiologic factors that cause the lesions or making the diagnosis of the underlying systemic disease can take a long time. Detailed history of the patient, systemic symptoms, presence of similar lesions at other sites of the body, medications used, contact with chemical materials and the family history should be questioned[3]. If there is suspicion of allergy, a patch test against dental materials can be performed on the patient[16]. The definitive diagnosis can be made by histopathological, direct (DIF) and indirect immune fluorescent (IIF) examinations of the tissues obtained from the lesions, in addition to examination of autoantibodies in the circulation. Several mucocutaneous diseases in which clinical desquamative gingivitis is observed have been reported in the literature i.e Lichen planus, Mucous membrane pemphigoid, Pemphigus vulgaris, Bullous pemphigoid, Paraneoplastic pemphigus, Dermatitis herpetiformis, Chronic ulcerative stomatitis, Psoriasis, Linear Ig A disease, Pyostomatitis vegetans, Erythema multiforme, Discoid lupus erythematosus, Dyskeratosis congenita, Epidermolysis bullosa, Graft-versus-Host disease, Plasma cell gingivitis, Foreign body gingivitis, Kindler syndrome, Ulcerative colitis, Acute myeloid leukemia (AML), Dermatomitosis, mixed connective tissue disorders, Crohn disease, Sarcoidosis, Drugs or chemicals implicated include various oral oral health care products, Sodium lauryl sulphate, Magnesium monoperoxyphthalate. The clinical condition generally exacerbates with plaque accumulation, trauma or improper brushing. The clinical picture worsens with the disruption of oral hygiene practices due to pain and bleeding[3,16]. The disease continues chronically with periods of remission and exacerbation. Recovery of the gingiva may take months[11]. Although the intraoral presence of desquamative gingival lesions differ, various durations from 2 months to 25 years have been reported[13].

Treatment

If there are previously determined etiologic factors (allergen materials, etc.) that cause DG, those should be eliminated and oral hygiene practices should be improved. Subgingival and supragingival plaques should be removed and proper teeth brushing with a soft brush in addition to flossing should be recommended[16]. Besides, patients should be warned about mechanical and chemical trauma. Intraoral restorations or prosthesis should be removed [8]. Systemic and topical corticosteroids are used for the medical treatment of DG. Topical corticosteroids are commonly used to treat DG. However, their effects are limited due to the saliva volume and the tongue movements which decreases the effectiveness of the treatment. Direct application of chlobetasole propionate to the affected site is recommended [3,11]. Custom built silicone or acrylic carriers which provide long term contact of the drug with the gingival lesion can be prepared to increase the effectiveness of the topical treatment.

As for local lesions, beclomethasone dipropionate inhaler (50-100 microgram/spray), fluticasone propionate (50 microgram/spray) nasal spray can be directly applied onto the lesions four times a day. Furthermore, 0.1% triamcinolone orabase can also be used. For generalized lesions, prednisolone (5-10 mg), betamethasone (0.5-1 g) or fluticasone tablets dissolved in water can be used as a mouthwash for at least two minutes 2-3 times a day [16]. Use of 0.15% benzydamine hydrochloride mouthwash is also recommended for its analgesic and anti-inflammatory effects. Topical use of sicatrizing drugs as supportive treatment accelerates regression of lesions [16]. There are cases that have been reported concerning the successful use of topical tacrolimus (0.0%3, 0.1%, 0.3%). However, its use is not preferred, due to the necessity of controlling serum tacrolimus levels at certain intervals and because of the side effects in some patients [11,16]. Use of drugs such as cyclosporine, azathioprine, and dapsone has also been mentioned in the literature[16]. Estrogen support for the treatment of DG has been recommended based on the presence of estrogen-sensitive receptors in the human gingiva and estrogen destruction [17]. The idea of estrogen therapy has been rejected since the estrogen receptors expressions in the gingival tissues are not related to the presence or absence of estrogen as well as the side effects of estrogen.[18,19]

II. Conclusion

Desquamative gingivitis can be the clinical symptom of some dermatitis and mucocutaneous diseases and the underlying primary cause should be evaluated meticulously. Taking detailed patient history, performing a careful intraoral examination and determining the presence or absence of similar lesions at other sites of the body are the most important steps in clinical practice. Definitive diagnosis of DG should be made by incisional biopsy, histopathological examination and DIF. Gingival lesions are controlled by improving oral hygiene and the use of topical corticosteroids. If there is an underlying systemic disease, the case should be consulted with the physician.

Figures

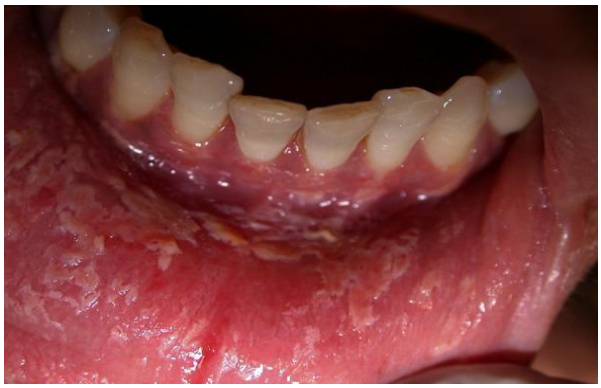

Fig.1 Mucosal and gingival desquamation as a result of an allergic reaction to toothpaste.

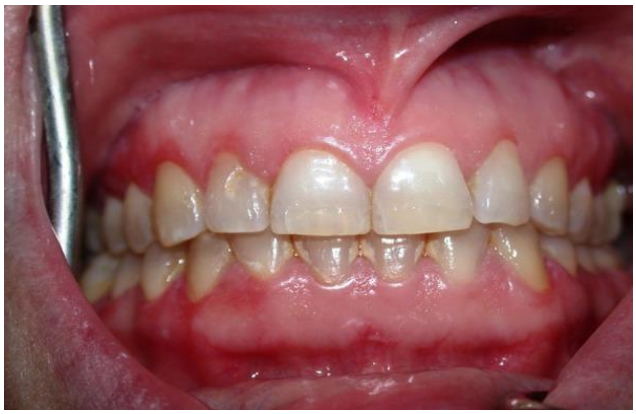

Fig.2 Desquamative gingivitis not related to disease or allergic reaction.

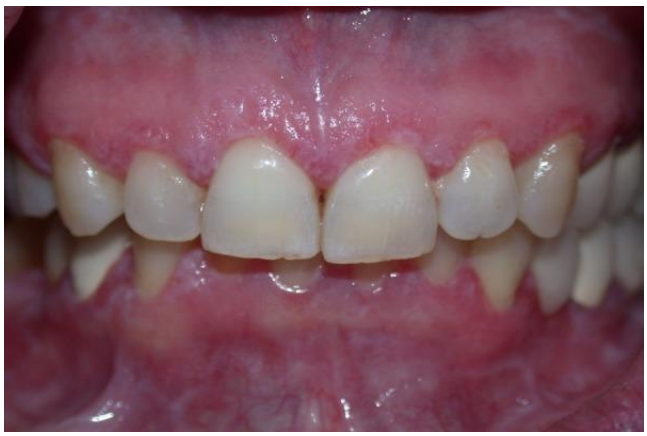

Fig.3 Atrophic form of Lichen Planus creates a typical desquamative gingivitis.

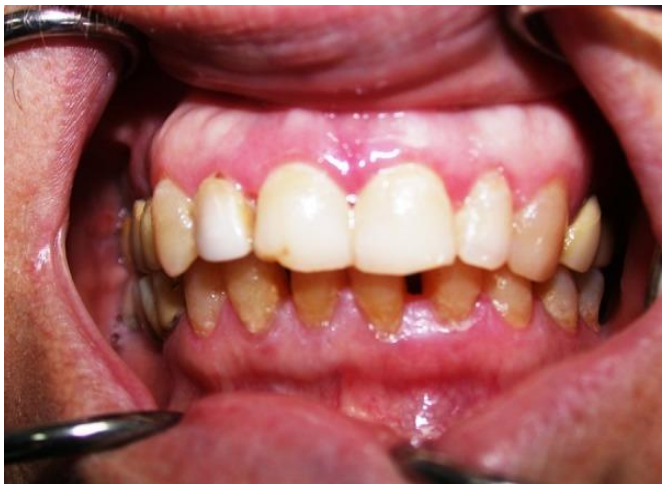

Fig.4 Intraoral appearance of mucous membrane pemphigoid is generally similar to desquamative gingivitis.

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