CASE REPORT

Desquamative Gingivitis: A Diagnostic Challenge and Therapeutic Approach – A Case Report

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Abstract

Context / Background: Desquamative gingivitis is a gingival keratinization disorder characterized by chronic ulceration, epithelial desquamation, and erythematous changes involving the free and attached gingiva. The condition is often refractory to treatment and may be associated with a range of autoimmune or mucocutaneous disorders. Owing to its unclear etiology, management is primarily symptomatic.

Case Presentation: We report a case of a 55-year-old female presenting with chronic desquamative gingivitis associated with localized periodontitis stage II, grade B. Clinical examination revealed diffuse erythema, epithelial peeling, and a positive Nikolsky's sign. Histopathological evaluation confirmed suprabasal clefting with acantholytic cells and a tombstone appearance, suggestive of pemphigus vulgaris.

Management and Prognosis: The patient underwent thorough oral prophylaxis and was prescribed topical corticosteroids (0.1% triamcinolone acetonide), vitamin E, multivitamins, and omega-3 supplementation. Symptomatic improvement was noted within one week, and the patient was advised regular follow-up for long-term monitoring due to the chronic nature of the condition.

Clinical Implications / Conclusion: This case highlights the importance of early recognition of desquamative gingivitis as a potential manifestation of underlying autoimmune disease. Prompt diagnosis and appropriate management are essential to prevent progression and improve patient quality of life.

Keywords: Corticosteroids, Desquamation, Erythema, Periodontitis

Introduction

Desquamative gingivitis (DG) refers to the presence of epithelial desquamation, erythema, and erosions on the gingival tissues. Several autoimmune and mucocutaneous disorders can present with DG, among which the erosive, ulcerative, and atrophic variants of oral lichen planus (OLP) are most commonly implicated.[1-3] The condition was first described by Tomes and Tomes in 1894, and the term "Chronic desquamative gingivitis" was later introduced by Prinz in 1932.[4] The word "desquamation," derived from the Latin Desquamare meaning "to scrape fish scales," refers to the shedding or peeling of epithelial layers.[5]

Clinical manifestations range from mild localised erythema to extensive involvement with spontaneous bleeding. Mild cases may cause sensitivity to spicy or acidic foods or irritation from dentifrices, while severe forms significantly affect oral comfort and quality of life. Painful lesions often hinder effective oral hygiene, increasing the risk of plaque accumulation and periodontal breakdown.[6]

Histopathologically, the epithelium and underlying connective tissue exhibit characteristic changes depending on the underlying disease. DG may mimic several dermatoses, including OLP, mucous membrane pemphigoid (MMP), and pemphigus vulgaris (PV). Although hormonal, microbial, and allergic factors have been suggested, the exact etiology of DG remains unclear. [7] It predominantly affects adults particularly women although occasional cases in children have been reported.[4]

Pemphigus, first described by Wichman in 1791, comprises a group of chronic blistering disorders. Advances in

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diagnostic criteria have established pemphigus as an autoimmune condition characterized by intraepidermal blistering. Histologically, it shows epithelial clefting and immunologically is associated with IgG autoantibodies against keratinocyte surface antigens. The major subtypes include pemphigus vulgaris, pemphigus foliaceus, and paraneoplastic pemphigus, with pemphigus vulgaris constituting approximately 70% of cases.[8]

Case Report

A 55-year-old female patient reported to the Department of Periodontology, Annoor Dental College & Hospital, Muvattupuzha, Kerala having a chief complaint of burning sensation in the upper and lower region of gingiva for 3 months which aggravates on having spicy & hot food. On examination, there is a diffuse distribution with patchy areas of bright red and gray involving both the marginal and attached gingiva. The surface texture presented as smooth and shiny and on running the surface of probe on the gingiva resulted in peeling of the epithelium and exposure of bleeding connective tissue. (Fig 1) The patient also reported limitation of oral function and speech difficulties due to pain.

The patient reported no significant dental history and exhibited no associated skin lesions. A comprehensive extraoral examination confirmed the absence of dermatological manifestations. On intraoral evaluation, Nikolsky's sign was elicited and found to be positive over the affected gingival areas. The patient reported having dyslipidemia and stress for 2 years. The exact etiology for the appearance of desquamated lesions in this patient was not identified. After obtaining written consent, an incisional gingival biopsy was taken from the mandibular posterior region for the histopathological examination. The histopathological examination was done in Department of Oral and Maxillofacial pathology and oral microbiology in Annoor dental college and hospital, Muvattupuzha. The hematoxylin & eosin (H & E) stained tissue showed para-keratinized stratified squamous surface epithelium associated with a fibrovascular connective tissue. The epithelium exhibited a suprabasal cleft at the epithelial–mesenchymal junction, with numerous acan-

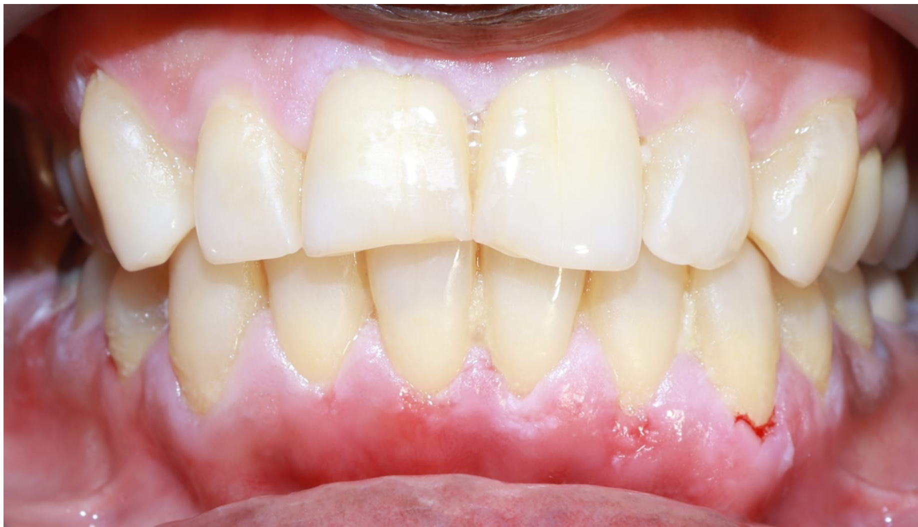


Fig 1: Preoperative view of Desquamation noted in maxillary and mandibular region

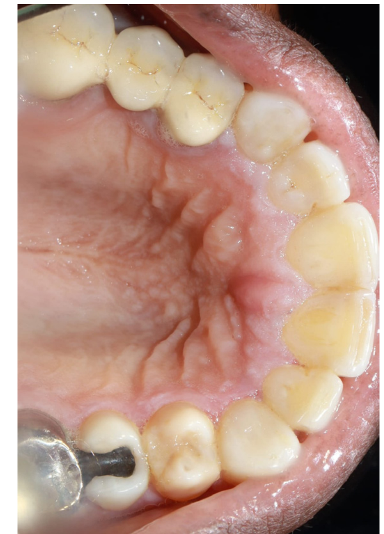
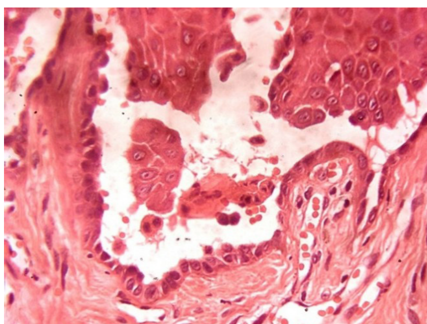


Fig 2: Preoperative view of Desquamative gingivitis presented on the marginal gingiva



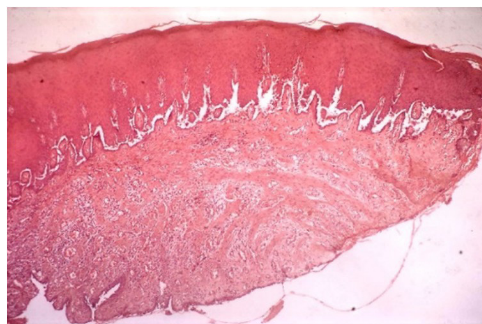


Fig 3: Histologic section showing suprabasilar split

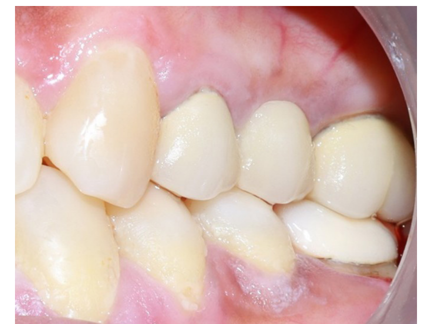


Fig 4: Appearance of desquamative gingival lesions in the mandibular region after 1week of treatment

tholytic (Tzanck) cells displaying hyperchromatic nuclei within the split area. The connective tissue papillae showed basal keratinocytes arranged in a characteristic tombstone pattern. The underlying connective tissue demonstrated a moderately dense chronic inflammatory infiltrate, numerous capillary vessels, and extravasated red blood cells. Bacterial colonies were also noted.

Thorough oral prophylaxis (scaling & polishing) was done. The patient was advised to rinse her mouth with 0.2% chlorhexidine mouthwash, enforced to brush her teeth & also asked to maintain good oral hygiene. The patient was prescribed multivitamins, Omega 3 capsules, and topical application of vitamin E and steroid (0.1% triamcinolone acetonide). Based on the clinical presentation and histopathological findings, a final diagnosis of Pemphigus Vulgaris presenting as Desquamative Gingivitis was established.

Discussion

Most cases of desquamative gingivitis are of severe mucocutaneous diseases. Lichen planus, mucous membranous pemphigoid, and pemphigus vulgaris account for more than 80% of cases. Although oral lesions of Pemphigus vulgaris (PV) can occur anywhere in the oral cavity, the oral lesion noted to be limited to gingiva is between 3% and 30% (Endo et al., 2018).[9] In this case, the patient had lesions in several sites of the oral cavity. However, she came to us with concern about painful and bleeding gingiva with a burning sensation, which is the characteristic of desquamative gingivitis, despite having other oral lesions. For the definitive diagnosis of Pemphigus, the presence of the following features is a must which is: The presence of appropriate clinical lesions, biopsy specimens showing acantholysis, and the presence of autoantibodies in either tissue or serum or both.[10] Diagnosis as pemphigus vulgaris of this case was made keeping in mind the following findings:[1] Intraepithelial split and Tzanck cell in histological examination of gingival biopsy specimens,[2] tombstone appearance,[3] burning sensation in gingiva on eating spicy food,[4] Nikolsky's sign,[5] positive response to the use of topical corticosteroid.

Direct immunofluorescence (DIF), which is considered the gold standard for confirming autoimmune blistering diseases, was not performed in this case due to financial constraints. Although DIF greatly aids in differentiating conditions such as pemphigus vulgaris, mucous membrane pemphigoid, and lichen planus by demonstrating specific patterns of immunoglobulin and complement deposition, the clinical features and histopathological findings in this patient were sufficiently characteristic to support the diagnosis. The mucosal lesions in the gingiva generally do not scar, but scarring is a critical and common finding in ocular and other mucosal lesions. This may result in further complications such as blindness, laryngeal or pharyngeal stenosis, and even death. In severe cases, adhesions may develop between the buccal mucosa and alveolar mucosa and the floor of the mouth and tongue resulting in ankyloglossia.[9]

The probable pathogenesis of Pemphigus includes an autoantibody- induced complement-mediated sequestration of leukocytes with resultant release of cytokines and leukocytes causing detachment of basal cells from the basement membrane zone. This results in subepithelial slit formation which is the classic histopathological finding in PV. Studies have shown that the antigens associated with PV are more frequently seen in the lamina lucida portion of the basement membrane.[11]

The oral cavity usually represents the first or only site of disease involvement. The oral cavity presentation is seen in almost all cases and the initial presentation is oral in 48% and ocular in 30%. Gingival involvement is seen in 100% of cases in the oral cavity.[11] The disease usually appears in the fourth and fifth decade of life as in our case.[12] It is more common in females with a female-to-male ratio of 2:1; however, in our case, it is presented in a female patient. Intraoral sites such as buccal mucosa, tongue, palate, and floor of the mouth may also be involved. Conjunctiva is the second most involved site with an occurrence rate of 3–48%. Progressive scarring and corneal damage may lead to blindness in 15% of cases.[9] A multidisciplinary approach is necessary to manage MMP to reduce disease-related complications. Recently, low-level laser therapy has also been used to improve tissue healing after local corticosteroid application.[10] In a study by De Carvalho et al., it was concluded that Photo biomodulation led to a significant reduction of pain and clinical scores of the lesions, not showing significant differences when compared to topical corticosteroids.[13] Progression of pemphigus vulgaris to extraoral involvement may require systemic corticosteroid treatment. However, long-term use of corticosteroids may lead to herpes virus and fungal infections (Schmidt et al., 2019). As recurrence of pemphigus vulgaris is possible, the patient should be monitored for a long period, perhaps lifelong.

Conclusion

Pemphigus Vulgaris presenting as desquamative gingivitis can pose a diagnostic challenge due to its similarity to other mucocutaneous disorders. Early recognition and appropriate management are essential to prevent disease progression and associated complications. As the oral cavity may be the initial or only site of involvement, dentists play a crucial role in timely identification. This case highlights the importance of comprehensive clinical evaluation and histopathological assessment in establishing an accurate diagnosis and guiding effective treatment.

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