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DESQUAMATIVE GINGIVITIS: A DIAGNOSTIC CLINICAL INDICATOR OF PEMPHIGOID BULLOSA

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ABSTRACT

Desquamative gingivitis is not a clinical pathology, but rather a clinical indication of several mucocutaneous illnesses. Desquamative gingivitis is characterized by the desquamation of the epithelial layer, severe erythematous erosions, and/or vesiculobullous lesions of the gingiva. These vesiculobullous lesions can be identified by histological and immunofluorescence investigations in conjunction with clinical observations. Chronic autoimmune illness pemphigoid bullosa only seldom affects mucous membranes and the skin. High morbidity and possibly fatal consequences can result from desquamative lesions with oral and extra-oral symptoms. Thus, knowledge and comprehension of the entity and related diseases and disorders are crucial for clinical treatment. The article is a case study of a 34-year-old female patient who had desquamative lesions of attached gingiva on her maxilla and mandible. After the histological analysis revealed a subepidermal bullous lesion with mononuclear infiltration, the illness was identified as pemphigoid bullosa.

KEYWORDS- Desquamative gingivitis, Corticosteroids, Vesiculobullous lesions

INTRODUCTION

Desquamative gingivitis (DG) is a general descriptive term that indicates the presence of diffuse desquamation, erythema, erosion, and blistering of the attached and marginal gingiva.[1] Prinz and Merrit introduced the term chronic diffuse desquamative gingivitis to further identify the illness after Tomes and Tomes' initial description in 1894.[2] Usually, the changes are confined to the labial gingival mucosa, which varies from bright to dark red and is edematous. The epithelium is quite friable and can be removed easily from the underlying connective tissue, leaving a red surface that bleeds readily after minimal trauma. Other gingival sites, such as palatal and lingual surfaces, are rarely involved.[3] Several mucocutaneous disorders, e.g., lichen planus, pemphigoid, pemphigus vulgaris (PV), erythema multiforme (EM), lupus erythematosus (LE), drug-induced lesions, and others, are listed in this subgroup together with allergic reactions to dental materials, foods, and other substances for topical application. Irritant and allergic contact dermatitis can also be potential initiating or exacerbating factors. The most common contactants are mouthwashes, toothpastes, dental materials, and medications.[4] Besides their heterogeneous nature, all of these

disorders share two features: an immunomediated pathogenesis and desquamative gingivitis (DG).[5] Though specific diseases are known to underlie DG, reduced oral hygiene caused by gingival discomfort and bleeding on flossing leads to dental plaque accumulation, which typically aggravates the clinical severity of the inflammation.[6] Accumulation of dental plaque leads to release of proinflammatory cytokines, such as interleukin-1 and tumor necrosis factor alfa, recruitment of inflammatory cells, and periodontal inflammation.[7] These cytokines also upregulate matrix metalloproteases, which can then breakdown collagen and lead to loss of periodontal attachment and bone destruction.[8] Diagnosis of the lesion should be made after biopsy examination & immunofluorescence test (direct and indirect) with correlated clinical findings. DG may persist for months to years, with exacerbations or remissions. Extensive involvement may result in widespread erosions and compromise cutaneous integrity; deaths may occur in elderly and/or debilitated patients. The mainstay of treatment is systemic glucocorticoids. Patients with local or minimal disease can sometimes be controlled with topical glucocorticoids alone; patients with more extensive lesions generally respond to systemic glucocorticoids either alone or in combination with immunosuppressive agents.[9]

CASE REPORT

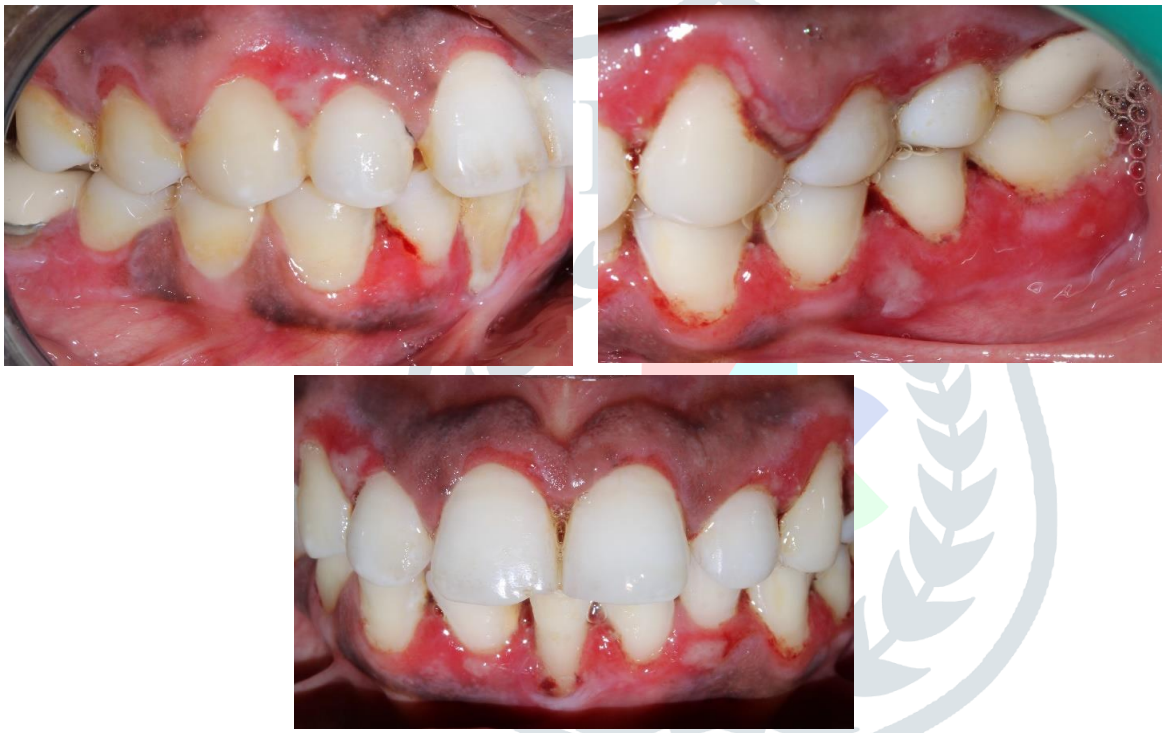

Figure 1 : Intraoral desquamative lesion

A 34 years old female patient who reported to the department of periodontics, Darshan Dental College & Hospital, Udaipur with a complaint of burning sensation, tenderness & bleeding in the gums, which worsened on consumption of hot & spicy food over the period of 1 years. The patient noticed blisters on the gums occasionally, which would heal eventually without any medical attention. The medical history was non-contributory. There was no associated ocular, cutaneous or genital lesions.

Intraoral examination revealed an erythematous and inflamed facial gingiva with areas of normal papillary gingiva irt 12,11,21,22,44,43. Involved areas had red gingiva with smooth and shiny surface and rolled borders. Faint white striae were visible bordering the areas showing desquamation. (Fig 1) Complete oral prophylaxis was done using hand instruments and topical application of Kenacort 0.1% (Triamcinolone) (Fig 2) and Patanjali Dant Kanti was replaced by Colgate white which is softer on gums. Added instructions to stop consuming hot & spicy food were given. Over a period of 1 month reduced inflammation was seen which further deteriorated in follow-up examinations. An incisional biopsy was taken from maxillary attached gingiva irt 13.

Histopathology showed focal desquamatous epidermal layer. Underneath fibrovascular tissue shows marked mononuclear infiltrate. No basal layer dyskeratosis was observed. Findings were suggestive of subepidermal bullosa lesion – pemphigoid bullosa. (Fig 3) Thereafter the patient was prescribed with prednisolone 40mg, candid gel (clotrimazole), Tab cetzine (cetirizine hydrochloride tablets) 10mg. Improvement in the lesion was observed and patient was kept on maintenance. (Fig 4)

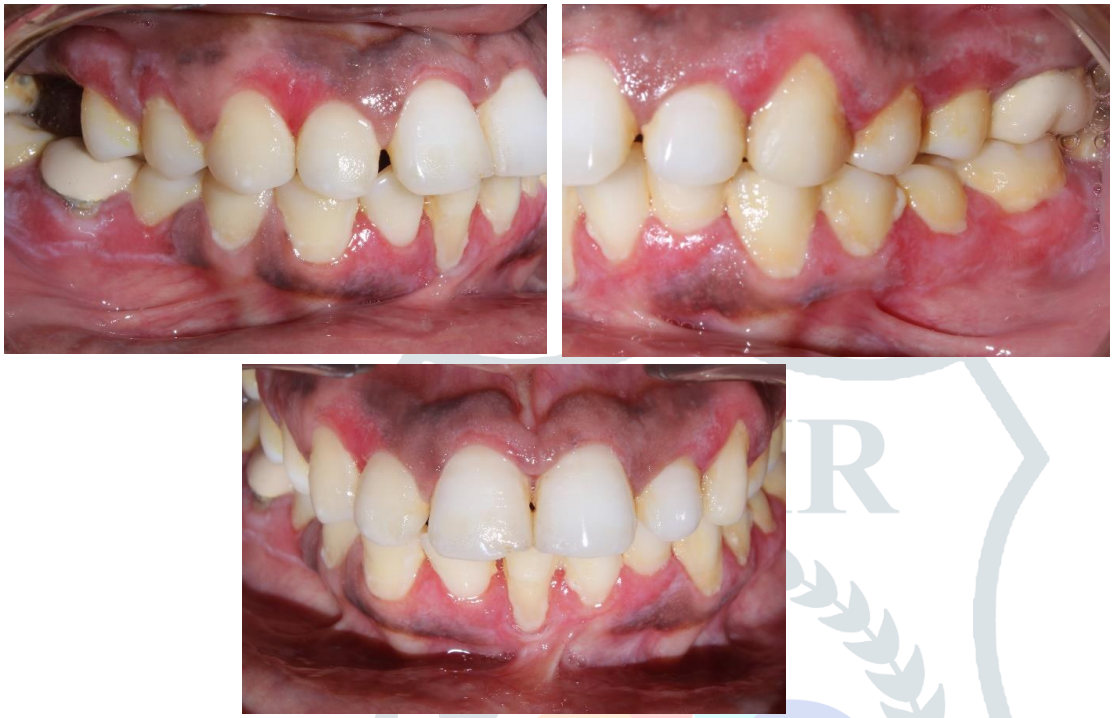

Figure 2 : After the use of tropical steroid

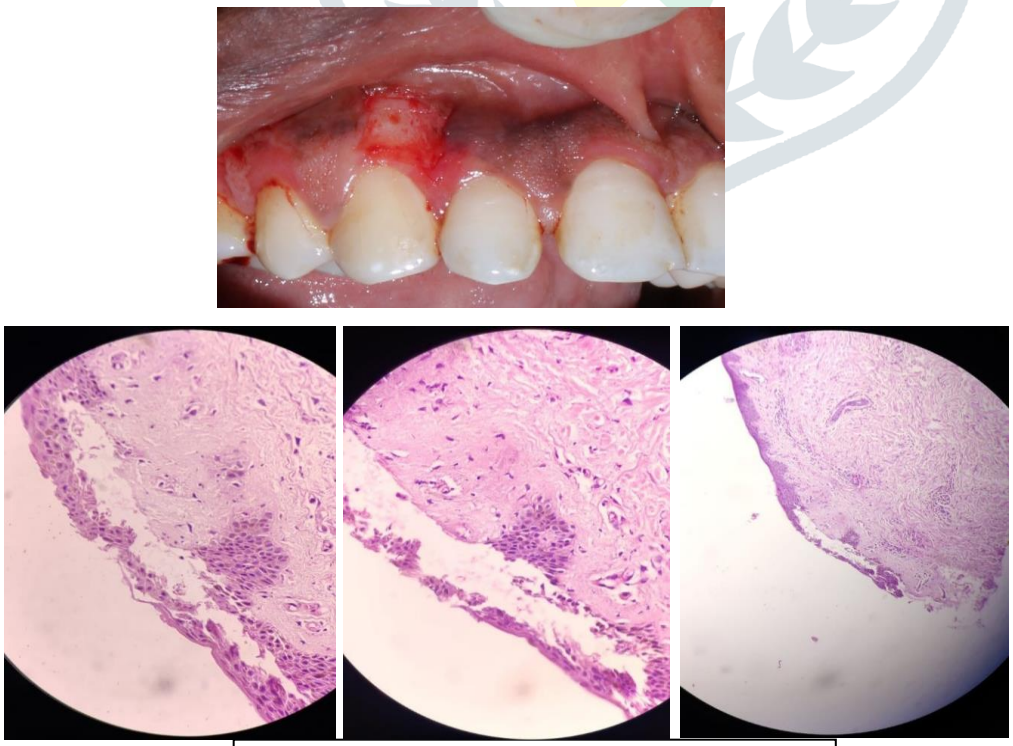

Figure 3 : Histopathology test

DISCUSSION

The term desquamative gingivitis (DG) describes a peculiar clinical presentation of the free and attached gingivae, characterized by intense erythema, desquamation, and ulceration. Approximately 50% of patients with DG have additional intraoral sites affected. DG is not a specific disease entity; rather, it is a gingival response associated with a number of conditions, most of them with a dermatologic genesis.[10] In our case patient had erosive lesions with burning sensations and treatment initiated with tropical corticosteroids and then systemic steroids were given due to poor response to the local treatment modality. Histological examination was also done to confirm the diagnosis. Improvement in lesions was observed after systemic therapy.

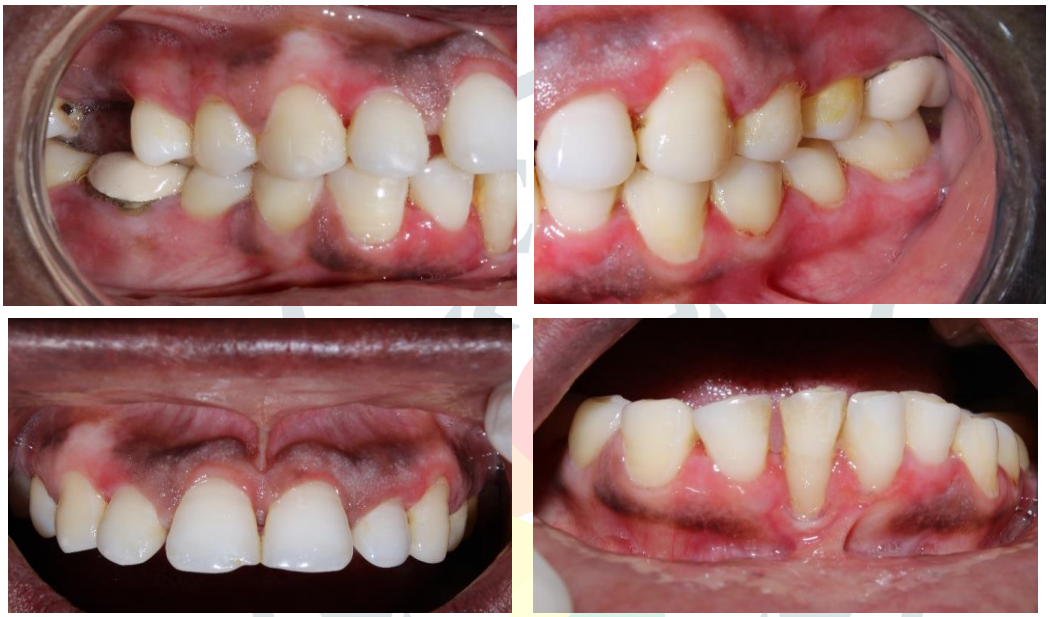

Figure 4 : Before & after the use of systemic steroids

CONCLUSION

It is necessary to form appropriate diagnosis to successfully treat desquamative lesions. Due to painful nature of the disease patients may find difficulty to maintain a good oral hygiene practices but they should be motivated and proper supportive care should be given to the patient. The reason of desquamative gingivitis in many people cannot be determined even after a rigorous diagnostic effort. So, the management is the key for providing comfort and symptomatic relief for the patient.

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