Article published online: 2021-11-01

CASE REPORT

Desquamative gingivitis: A clinical sign in mucous membrane pemphigoid

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ABSTRACT

Gingival desquamation is a clinical sign in which the gingiva appears reddish, friable with desquamation of epithelium. It may be the result of various disease process such as, mucous membrane pemphigoid (MMP), oral lichen planus, and pemphigus vulgaris which accounts for a major cause of desquamation. MMP is a rare chronic autoimmune disorder characterized by subepithelial bullae clinically and suprabasilar split histologically most commonly affecting the oral cavity manifesting as a desquamative gingivitis. Here is a case of pemphigoid appearing as a desquamative gingivitis.

Key words
Desquamative gingivitis, immunofluorescence, pemphigoid, suprabasilar split

INTRODUCTION

Chronic desquamative gingivitis is a clinical entity characterized by erythematous and desquamative involvement of the free and attached gingiva. More than 100 years ago, this condition was described by Tomes.[1] Later Prinz[2] in 1932 first coined the term "chronic diffuse desquamative gingivitis" for cases characterized by severe epithelial desquamation. The management of chronic desquamative gingivitis has been a major problem, largely because the etiology of the disease has been elusive with early investigators believing that it was due to a single cause. Etiologic factors that have been considered includes estrogen deficiency, hypothyroidism and nutritional deficiencies.[3]

In 1953, Glickman speculated that chronic desquamative gingivitis may represent a manifestation of several disease processes. While he favored an endocrine etiology, he thought that the condition might be a variant of chronic mucous membrane pemphigoid (MMP) or bullous lichen planus. McCarthy *et al.*[4] proposed a classification of desquamative gingivitis based on etiologic considerations such as dermatoses, hormonal influences, and abnormal responses to irritation, chronic infections, and idiopathic causes.

A. Dermatological disease
   - Cicatrial pemphigoid
   - Lichen planus
   - Psoriasis
   - Bullous pemphigoid
   - Epidermolysis bullosa acquisita
   - Contact stomatitis
B. Endocrine disease
   - Estrogen deficiencies following oophorectomy and in postmenopausal stage
   - Testosterone imbalance
   - Hypothyroidism
C. Aging
D. Abnormal response to bacterial plaque
E. Idiopathic
F. Chronic infection
   - Tuberculosis
   - Chronic candidiasis
   - Histoplasmosis.

McCarthy *et al.* concluded that a classification based on etiology permits a rational therapeutic approach.

Identification of the underlying etiology of desquamative gingivitis originally depended solely on clinical and histologic criteria. Recent techniques are involving

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Quick Response Code:	Website: www.ejgd.org
	DOI: 10.4103/2278-9626.163338

immunofluorescence (IF) assay would be more beneficial in obtaining confirmatory diagnosis. Two types of IF are of prime value for lesions of desquamative gingivitis: Direct IF for detection of *in vivo* bound immunoglobulins and complement in biopsy specimens and indirect IF for detection of serum antibodies. Direct IF consists of examining cryostat-cut frozen sections on which fluorescein-labeled reagents to human immunoglobulin G (IgG), IgA, IgM and complement components have been incubated.[5] The practicality of direct IF tests on mucosal biopsy specimens have been greatly enhanced in recent years by Michel's[6] development of a special holding solution for transport of biopsies. For indirect IF, antibodies in the serum will bind to the specific tissue components. The bound immunoglobulins can be identified with a fluorescein-labeled antibody to the bound immunoglobulin.

The purpose of this case report is to present a case of desquamative gingivitis, with its clinical and microscopic features presented along with a brief review of the literature.

CASE REPORT

A 42-year-old male patient reported to outpatient Department of Periodontology, with a complaint of increase salivation and for routine check-up for cleaning his teeth. Oral examination revealed bright red lesion with diffused area of desquamation and erythema involving buccal aspect of marginal and attached gingiva in the region of 21, 22, 23 measuring 3 cm × 2 cm, resembling desquamative lesion [Figure 1]. The patient was unaware of this lesion since it was painless. There was no associated ocular, cutaneous and genital lesion. Personal history revealed that he was a tobacco chewer since 20 years for 8–10 times a day. His medical history had no relevant significance. Treatment included complete oral prophylaxis and oral hygiene instructions. Patient was re-evaluated after 15 days for follow-up. There was a significant improvement in periodontal condition but no improvement in the lesion. The lesion was still bright red, painless and showed signs of loss of stippling. Routine complete blood investigation was performed which resulted in hematologic parameters within confines of the normal range. After obtaining an informed consent from the patient, an excisional biopsy was performed, and the specimen was sent for histopathological examination. Based on clinical findings the case was considered to be desquamative gingivitis as a provisional diagnosis. However, differential diagnosis included pemphigus vulgaris and cicatricial pemphigoid.

Tissue was transported in 10% formalin for histopathological examination which revealed parakeratinized stratified squamous epithelium with subepithelial clefting. There was also the presence of scattered mild, mixed chronic inflammatory cells in the underlying connective tissue along with a few eosinophils. The basal cells were attached to the overlying epithelium and not to the basement membrane, suggestive of MMP [Figures 2 and 3]. Direct immunofluorescence examination revealed the presence of IgG immunoglobulin in the basement membrane [Figure 4]. Thus, the diagnosis of oral pemphigoid was established based on clinical, histological and IF features.

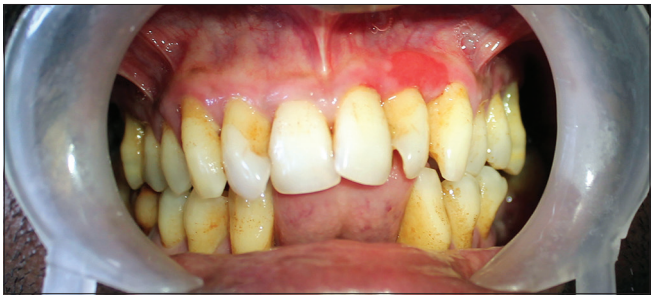

Figure 1: Preoperative photograph

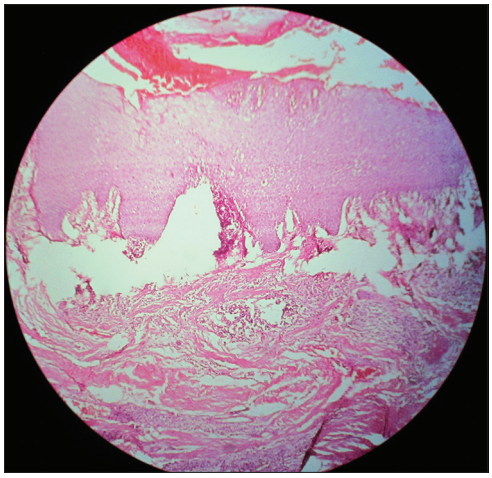

Figure 2: ×10 magnification showing subbasilar separation of the epithelium from the underlying connective tissue

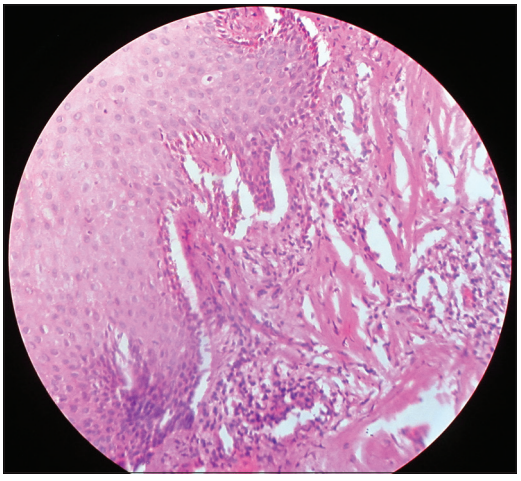

Figure 3: ×40 magnification showing subbasilar separation of the epithelium from the underlying connective tissue

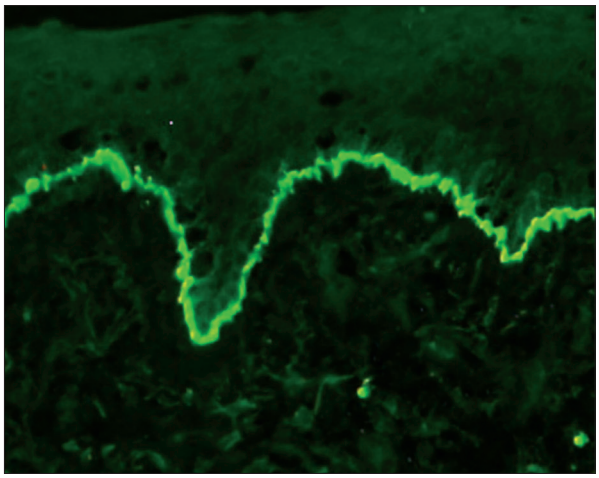

Figure 4: Direct immunofluorescence

The treatment included topical steroids Kenacort (0.1% triamnicolone acetonoide) which resulted in no improvement in the lesion persisting as before. Later excisional biopsy was performed that resulted in healing of the area without recurrence for post 6 months [Figure 5]. Further follow-up are required to rule out for recurrence of the lesion.

DISCUSSION

In the present case, 42-year-old male patient who presented with a bright red desquamative lesion which exclusively showed involvement of the oral mucosa without any mucosal or cutaneous involvement. There were a localized erythema and inflammation of maxillary anterior labial gingiva. However, palatal gingiva was unaffected. MMP consists of a group of subepithelial blistering diseases primarily involving the mucosal surfaces. Since in this patient the characteristic lesions were restricted only to the mucosa it was diagnosed as MMP.[2,3] Similar results were observed in the study done by Rogers *et al.*, Laskaris *et al.*, Gallagher and Shklar.[7-9] However reports by Agbo *et al.* are not in accordance with the present study since both gingival and conjunctival lesion was observed.[10] Pathogenesis of MMP probably includes an autoantibody-induced, complement mediated sequestration of leukocytes with resultant cytokine and leukocyte enzyme release and detachment of the basal cells from the basement membrane zone (BMZ) but there may also be complement-mediated cell lysis. MMP is characterized by junctional separation at the level of the basement membrane, which gives rise to a sub-basilar split,[4] which was observed in the present histopathologic section. Patients with MMP rarely have circulating autoantibodies to BMZ components hence indirect immunofluorescence is usually not indicated as a diagnostic procedure.

Treatment of MMP is based on the severity of symptoms and the site involved.[1,2] Patients with mild, localized lesions may often benefit from topical steroids such as beclomethasone dipropionate, betamethasone, clobetasol propionate, fluocinomide. Patients with more extensive lesions can be prescribed systemic steroids like prednisolone.[11] Other treatment regimens, which were effective in certain resistant cases, were immunosuppressive agents such as azathioprine, cyclophosphamide, cyclosporine, and dapsone. Sulphonamides and tetracyclines can also be implemented.

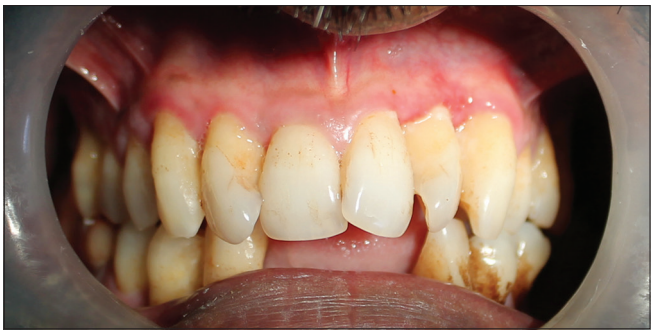

Figure 5: Postoperative photograph

Treatment of MMP is considered to be a compromised condition, due to its auto-immune nature. The lesions may occur intermittently and affect different parts of the oral mucosa at different times. Since this disease has an irregular pattern of recurrence, the patient should be followed carefully by an ophthalmologist whether or not conjunctival involvement is seen at the time of oral lesion diagnosis. Dentists should be in close contact with an ophthalmologist and dermatologist in cases with possible conjunctival or skin involvement.[2,12,13] Thus, a multispeciality treatment approach could be a valuable treatment modality.

ACKNOWLEDGMENT

The authors acknowledge the staff and students of the School of Dental Sciences, Karad, Satara, Maharashtra, India.

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How to cite this article: Abbayya K, Kadashetti V, Baad R, Lohana M. Desquamative gingivitis: A clinical sign in mucous membrane pemphigoid. Eur J Gen Dent 2015;4:136-9.

Source of Support: Nil, **Conflict of Interest:** None declared.