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Original Research Paper

Dental science

# INVESTIGATION, DIAGNOSIS AND MANAGEMENT OF DESQUAMATIVE GINGIVITIS

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KEYWORDS :

## INTRODUCTION:

The term desquamative gingivitis describes the clinical appearance of gingiva that are red, glazed, often edematous, with loss of stippling, and that have areas of superficial epithelial desquamation and/or ulceration. Vesicles, white striae or flecks may also be seen. Desquamative gingivitis is not a disease entity but a clinical manifestation of several different disorders. Desquamative gingivitis may be an isolated finding or may be accompanied by other lesions of the oral mucosa that help to determine the clinical diagnosis. Causes of desquamative gingivitis include:

**Dermatoses, which include:**

- Lichen planus
- Mucous membrane pemphigoid
- Pemphigus
- Dermatitis herpetiformis
- Linear IgA disease
- Epidemolysis bullosa

Lichen planus and pemphigoid are the most common causes of desquamative gingivitis. Pemphigus is much less common than lichen planus and pemphigoid, and the other conditions mentioned are rare causes of desquamative gingivitis.

we are describing two cases of desquamative gingivitis (lichen planus and pemphigoid) that were treated topical corticosteroids and nystatin used. More than six month follow up showed periodic recurrence, however with longer disease-free periods and less severe symptoms.

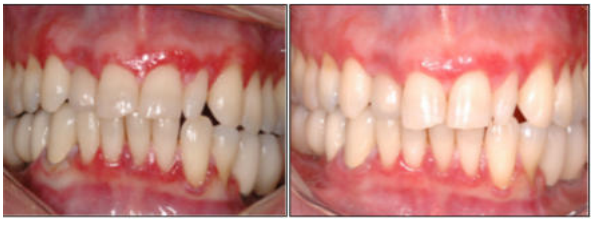
The oral lichen planus is a chronic inflammatory mucocutaneous disease with uncertain etiology and auto-immune pathogenesis [1–3]. The lesions are usually multiple and almost always have a bilateral symmetrical distribution, characterized by relapses and remissions periods [2–5]. OLP affects approximately 1% to 4% of the population, with a peak incidence in the 30–60 years age [5,7] and women are more commonly affected than man [2]. Most patients are usually asymptomatic, however burning sensation and pain interference with speaking and eating are common symptoms in the atrophic and erosive forms [7–9]. Spontaneous remission is rare and many lesions require treatment. The most widely used agent in treatment are corticosteroids, which can be used topically, intralesional or systemically [4–7]. Erythematous or ulcerated lesions that affect the entire width of the attached gingiva cause a condition called desquamative gingivitis [2]

Mucous membrane pemphigoid (MMP) is a heterogeneous group of autoimmune, chronic inflammatory, subepithelial blistering diseases, predominantly of mucous membranes, with lesions that can affect the oral, ocular, genital, nasopharyngeal, esophageal, and laryngeal mucosa and the skin [10]. Other names have been used for these conditions, including cicatricial pemphigoid, benign pemphigoid and oral pemphigoid. The disorders are characterized by linear deposition of IgG, IgA or C3 along the epithelial basement membrane zone, and a number of target antigens have been identified [11]. The major sequelae of these disorders are scarring and the associated loss of function, except in some patients whose disease is confined to the oral mucosa. Life-threatening airway obstruction and sight threatening ocular scarring can occur. Patients should be referred routinely to an ophthalmologist for assessment, and symptoms that suggest involvement of other mucosa such as the larynx or genitalia, or the presence of skin lesions, also warrant immediate referral to the appropriate specialist.

The gingival lesions may be the presenting symptom of a more generalized oral or systemic condition, and rigorous investigation is necessary to elucidate a diagnosis. The presence of dental plaque is an important exacerbating factor whatever the underlying cause.

## CASE 1

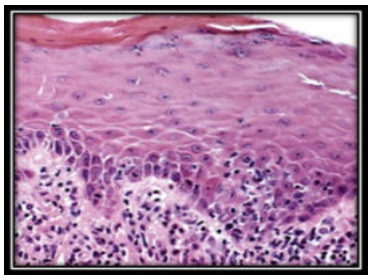
A 40-year-old healthy female was referred to the periodontist from civil dental clinic with a chief complain of pain and associated soreness of the gingiva for about six months, with periods of exacerbation and quiescence. Intraoral examination revealed erythematous and diffuse desquamative lesions of the gingiva, which were generalized for all maxillary region, bilaterally [fig 1a]. The associated teeth had no mobility and no bleeding on probing, but a variable amount of dental biofilm was observed. Based on the clinical features we carried out incisional biopsy. On histopathological examination confirmed the diagnosis of atrophic-erosive oral lichen planus.



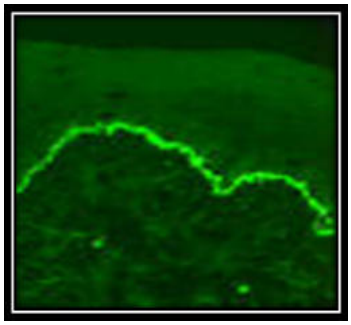
(1a) (1b)

Clinical aspect of the lichen planus on the upper and lower gums, pre (1a) & post (1b) photograph Histopathological examination shows stratified squamous epithelium appears acanthotic with wedge shaped projection of rete pegs into subepithelial and focal vacuolar degeneration of basal layer and occasional degenerated keratinocyte (civatte bodies) also seen. Subepithelial shows dense band like inflammatory

infiltrate abutting the epithelium comprising of lymphocyte and plasma cells.



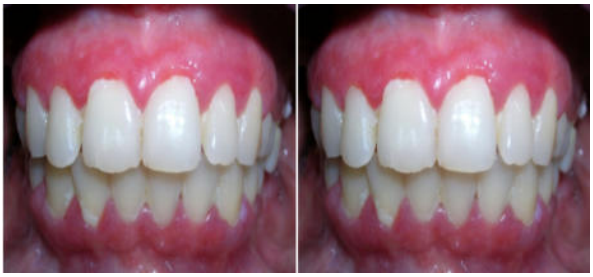
Direct immunofluorescence (DIF) to identify the presence of autoantibodies to epidermal antigens. Immunofluorescence shows linear IgG deposit seen in the basal layer of epidermis



A prescription of a ointment containing 0.1% triamcinolone, 100000 units/ml oral suspension nystatin and multivitamin tablets was given. Following completion of 2-weeks treatment, substantial improvement was observed in the gingival lesions (Fig. 1b). The patient reported reduction of pain.

**CASE 2**

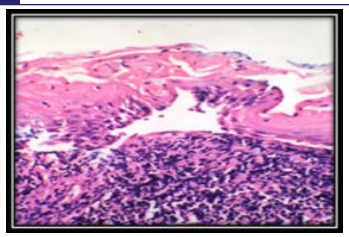
A 36-year-old healthy female was referred to periodontist from Dental centre with a chief complain of pain and burning sensation in gums of upper front teeth region in the past 2 months, with periods of exacerbation and quiescence. Patient was relatively asymptomatic around 2 months back. Developed burning sensation and pain which gradually increased with consumption of spicy food. which were generalized for all maxillary region, bilaterally [fig 2a]. The associated teeth had no mobility and no bleeding on probing, but a variable amount of dental biofilm was observed. Based on the clinical features we carried out incisional biopsy. On histopathological examination confirmed the diagnosis of Mucous Membrane Pemphigoid.



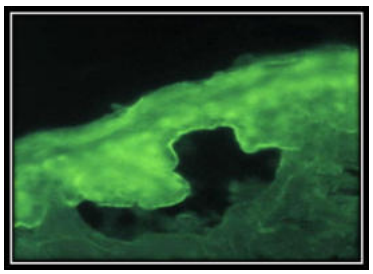
(2a) (2b)

Clinical aspect of the pemphigoid on the upper and lower gums, pre (2a) & post (2b) photograph

Histopathological examination shows stratified squamous epithelium. There is focal evidence of separation of epidermis from underlying dermis forming a vesicle. Few acantholytic cells are seen in this space. Underlying superficial subepithelial shows marked broad band like chronic mononuclear inflammatory infiltrate, No typical cells/ granulomas evidence of malignancy seen.



Definitive diagnosis often requires biopsy for histopathological examination and, where an autoimmune vesiculobullous disorder such as pemphigoid is suspected, for direct immunofluorescence (DIF) to identify the presence of autoantibodies to epidermal antigens. Immunofluorescence shows linear IgG deposit seen in the basal layer of epidermis.



A prescription of a ointment containing 0.1% triamcinolone, 100000 units/ml oral suspension nystatin and multivitamin tablets was given. Following completion of 4-weeks treatment, substantial improvement was observed in the gingival lesions (Fig. 2b). The patient reported reduction of pain.

**DISCUSSION**

The gingiva is a target of autoimmune diseases and about 10% of patients with oral lichen planus have the disease confined to the gingiva, clinically named desquamative gingivitis [2]. Desquamative gingivitis can present as reticular, erosive or atrophic subtypes however, the clinical appearance of desquamative gingivitis is not pathognomonic and may represent the gingival manifestation of many others autoimmune diseases [12]. Exacerbations of desquamative gingivitis have been associated to periods of psychological stress, anxiety and mechanical trauma [4]. Chronic low-grade irritation from dental biofilm, calculus, may also cause exacerbation of desquamative gingivitis [13]. The presence of gingivitis and periodontitis can complicate the symptoms of desquamative gingivitis.

Certain local factors including plaque and calculus deposits are associated with a significantly higher incidence of erythematous and erosive lesions, whereas good oral hygiene is essential and can enhance healing. However, the periodontal treatment fails to completely resolve the lesions, may be due to the immune nature of lichen planus. On the other hand, other authors [13], suggested in global terms that periodontal status in lichen planus is no worse than that observed among healthy controls, therefore, periodontal attachment loss was found to be very similar in lichen planus patients both with and without gingival involvement. They failed to demonstrate that lichen planus patients have increase long -term risk of periodontal disease.

There is a large number of available treatments due to recurrence, high prevalence and risk for malignant transformation of oral lichen planus [4]. Anti-inflammatory agents mainly topical corticosteroids are the most widely used in the treatment of oral lichen planus. Other therapeutic agents that have been investigated are acitretin, retinoids, immunosuppressant's such as cyclosporin, azathioprine, mycophenolate mofetil, tacrolimus and pimecrolimus, thalidomide, interferon alpha, levamisole and phototherapy are used.

Mucous membrane pemphigoid consists of a group of subepithelial blistering diseases primarily involving the mucosal surfaces. A consensus group of researchers in 2002[14] determined that mucous membrane pemphigoid was the best designation for this group of disorders. The term "benign" mucous membrane pemphigoid was deemed inappropriate because of the potential for serious complications in some cases specially the lesions were very severe. The term "cicatricial" pemphigoid excluded affected individuals who do not develop scarring. Site-specific terms such as "ocular" cicatricial pemphigoid excluded individuals with multiple site involvement. Since in this patient the characteristic lesions were restricted only to the mucosa it was diagnosed as Mucous membrane pemphigoid [15, 16, 17].

The disease is autoimmune in nature. Pathogenesis of Mucous membrane pemphigoid 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 but there may also be complement-mediated cell lysis. Mucous membrane pemphigoid is characterized by junctional separation at the level of the basement membrane, which gives rise to a sub-basilar split [18], which was observed in the present histopathologic section. Pemphigus is similar in its clinical presentation to Mucous membrane pemphigoid but it produces acantholysis with cleavage of the spinous cell layer. In pemphigus the vesicle is intraepithelial whereas in mucous membrane pemphigoid due to the separation of connective tissue from the epithelium the vesicle is subepithelial [14,19]. In contrast to lichen planus the inflammatory infiltrate is non-specific in nature comprising of lymphocytes, plasma cells, neutrophils with few eosinophils. Patients with Mucous membrane pemphigoid rarely have circulating autoantibodies to BMZ components hence indirect immunofluorescence is usually not indicated as a diagnostic procedure [20]

For treatment of both the disease we are used topical corticosteroids. Function of corticosteroids suppress cell mediated immune activity, but therapeutic responsiveness may differ between patients. This aside, they can be effectively applied to the lesions with minimal potential for systemic damage. Reports have already shown the effectiveness of 0.1% triamcinolone in the management of oral lichen planus and pemphigoid. It is the most potent of all topical steroids, exerts anti-inflammatory, immunosuppressive and antimitotic effects influencing the growth, differentiation and function of various cells and inhibiting cytokines [21]. Triamcinolone inhibits the production of phospholipase A2 and subproducts as lipoxygenase and cyclooxygenase [21], this would explain its greater effectiveness.

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