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Literature Review

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# Desquamative gingivitis as a clinical sign of oral lichen planus: review of literature

## Abstract

The term desquamative Gingivitis (GD) is used in the area of oral medicine to explain a periodontal clinical condition not associated with biofilm, which is usually observed in a group of patients presenting with autoimmune diseases such as: lichen Oral plane, Pemphigus vulgaris, and mucous membrane pemphigoid, among others. Desquamative gingivitis is most commonly mistaken for other plaque-related types of gingivitis, preventing a correct diagnosis of the lesion and consequently underlying systemic processes, some of which present an important Morbidity and even mortality.

**Keywords:** desquamative gingivitis, lichen planus, erythematous lesions, gingivitis

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## Introduction

Desquamative Gingivitis is a term that indicates diffuses desquamation, erythema and erosion of marginal and inserted gum, introduced by Pritz in 1932.[1] It's a clinical sign of chronic evolution with periods of remission and exacerbation. It's a gingival alteration not associated with biofilm and is framed within the gingival manifestations of systemic conditions. The lesions affect the gingiva free and adhered mainly, and according to the clinical affectation can be distinguished two types:[2]

a. **Mild forms:** Its course is acute, characterized by the presence of erythema and mild desquamation without ulceration. They are localized lesions caused by allergic mechanism or hypersensitivity to oral hygiene products, or trauma from parafunctional or aggressive brushing habits.[2]
b. **Severe forms:** They are a clinical manifestation of various mucocutaneous diseases. They have erythema, desquamation, and painful ulcerations that affect free and adhered gums.[2]

Oral Lichen planus (LPO) is an inflammatory mucocutaneous disease of unknown origin, but with an indication of being related to defense cells due to the prevalence of T-lymphocytes and immunoglobulins. In most cases it occurs between 30 and 60years,[3] is classified in: Reticulate, erosive and plaque. Desquamative Gingivitis can occur in all three types without clinical differences. LPO occurs more frequently with a reticular pattern and may even be asymptomatic, however in its erosive variant the lesions may occur with multiple ulcers in the oral cavity, which usually take pain and even present lesions Blisters.

## Literature review

Since 1856 it had already been reported in several books, as well as in medical and dental journals a type of chronic gingivitis described as unusually painful and with localized and generalized hemorrhage. In 1894, take and take described for the first time these lesions in the English-speaking literature and in, 1932, Prinz named this condition as Desquamative chronic gingivitis. For manyyears, Desquamative Gingivitis was thought to be a pathological entity of unknown etiology, which was possibly triggered by a deficiency of sex hormones in older women and rarely manifested in men. Today, however, there is little evidence to support this concept.[4]

The term desquamative gingivitis is a clinical description, not a diagnosis.[5] Erythematous lesions, located in the free and adhered gum, characterize it. Most of the time the changes are confined to the vestibular region and range from light red to intense. The epithelium is easily detached, as a consequence of the vesiculo-erosive lesions, leaving the connective exposed and bleeding to the slightest stimulus. Rarely the lingual or palatal gum is compromised[6] and the most common causes are lichen planus, mucous pemphigoid or Pemphigus. However, if this definition is used, the most frequent cause is lichen planus. This can affect the gum around even variable number of teeth. When the cause is a lichen planus, white scales may be identified on a red background. Desquamation or formation of blisters or small epithelium polyps may be identified on the margins of epithelial separation zones.[5]

### General clinical features

The clinical characteristics of this disease vary according to the severity of the lesion: the gingival mucosa acquires a bright red color, with small greyish opaque plaques, which take both the free and adhered gums. The superficial epithelium, when rubbed, is detached and reveals the connective tissue that, to clinical exploration, is very painful and bleeding. There is a dry burning sensation in the mouth and sensitivity to thermal changes. Highly seasoned foods are not tolerated and tooth brushing is very annoying, so plaque control is difficult, and patients often develop secondary, marginal gingivitis. The severity of desquamative gingivitis varies; Glickman and Smulow, quoted by Lemus and collaborators,[7] have described the forms mild, moderate and severe.

### Diagnosis

To begin the evaluation of desquamative gingivitis, it is essential to obtain a thorough anamnesis. The facts about the symptoms related to this condition, as well as the antecedents (onset of the lesion, aggravation, habits that exacerbate it, among others) represent the basis for a precise examination.

### Clinical examination

Recognition of the pattern of distribution of lesions (focal or multifocal, confined to the gum or not) provides the information needed to formulate the differential diagnosis. In addition, the positive Nikolsky sign, obtained through a simple maneuver, makes evident the presence of a vesiculo-blistering disorder.

**BX:** By virtue of the extent and number of lesions found in a given patient, incisional biopsy is the best alternative for starting the microscopic and immunologic evaluation.

**Immunofluorescence:** Immunofluorescence tests are positive, if an immunofluorescent signal is observed in the epithelium, basal membrane or connective tissue underlying a specific variety of antigens in human serum (anti-IgG, anti-IgA, anti-IgM, Antifibrin and anti-C3) marked with Fluorescein.[8] By correct diagnosis, the different alterations that lead to the emergence of chronic desquamative gingivitis can be established, which has been considered as a sign of a wide variety of vesiculo-blistering diseases.[9] The AAP, in its exhaustive classification for the superficial chronic inflammatory processes (version of 1999), includes all those gingival manifestations of systemic conditions, and groups them in: mucocutaneous disorders (Lichen planus, pemphigoid, Pemphigus vulgaris, erythema multiforme, lupus erythematous, drug-induced and others), allergic reactions (mercury, nickel, acrylic, others), reactions attributed to toothpastes, mouthwashes, chewing gum additives, food additives) and other Unspecified conditions.[10,11] Due to the importance of these systemic conditions for the establishment of the accurate diagnosis, it was considered necessary to particularize in the alterations that they study with gingival manifestations of desquamative type.

## Histopathology

There are two histological varieties: the type bullosa and the lichenoid type. The blistering lesions produce the total separation of the epithelium and leave the connective discovered. This is a very important finding to rule out the presence of Pemphigus vulgaris. In this last disease, which is fatal in the absence of treatment, the blisters are produced by partial separation of the epithelium but the connective remains covered by the basal epithelial layers. This process is called acantholysis. The lichenoid variety presents some typical characteristics of lichen: hipropica degeneration of the basal layers of the epithelium, subepithelial lymphocyte infiltration, there may be areas of hyperkeratosis, hiperparaqueratosis or also epithelial atrophy.[12]

## Lichen planus

Oral lichen planus is the most frequent mucocutaneous disease in the oral cavity; however it is difficult to give an accurate prevalence data, since the numbers vary depending on the geographic region to be studied. In some patients it may affect the skin, oral mucosa and also other mucous membranes while in others it compromises the skin and oral mucosa only.[13] It is type of oral lichen planus (LPO) a thin, whitish reticulate, called Wickham striae, slightly elevated, which tend to adopt a reticular arrangement, arborifore or in the form of Lace.[10,14] In the LPO, they most frequently affect the oral vestibular mucosa, and the palate and sublingual region are rare. Gingival involvement is common, and in about 10% of cases LPO is observed only in the gums. The typical presence is that of chronic desquamative gingivitis.[15,16]

Gingival lesions may present the following patterns:

A. **Keratotic lesions:** white and elevated defects, with the appearance of groups of individual papules, such as linear lesions, reticular or plaques.
B. **Erosive or ulcerative lesions:** extensive erythematous areas of irregular distribution, which may occur as focal or diffuse hemorrhagic regions. Vesicular or blistering lesions: elevated, liquid-occupied abnormalities, rare and short-lived in the gum, as they soon rupture to leave an ulcer.
C. **Atrophic lesions:** atrophy of the gingival tissues, with the consequent thinning of the epithelium, causes Erythema limited to the gingiva.[8]

## Histopathology

From the microscopic point of view, there are three main characteristics of oral lichen planus: hyperkeratosis or parakeratosis, hydropic degeneration of the basal layer and dense infiltrated band with predominance of lymphocytes T in the lamina itself. It is typical that interpapillary projections have an aspect of "saw tooth".[8,17]

## Pathogenesis

Their cause and pathogenesis are unknown, but there is evidence suggesting that it is an immune disorder, in which T-lymphocytes are attracted to an antigen within the epithelium. Subgrouping both CD4 and CD8 of the T-cell are densely dispersed in the interface of the connective-epithelium tissue of the diseased tissue. This chronic inflammatory state proclaims epithelial alterations, the deposit of excessive amounts of fribrinogeno in the basal membrane and final destruction of the basal cell layer of the eputelio affects. Nervous people, highly tense, and those who are infected with the hepatitis C virus, are predisposed to lichen planus. Mostly lichen planus affects women over 40years of age. The disease is long-term, with periods of remission and exacerbation.[18]

## Diagnosis

Although there are still no diagnostic criteria for universally accepted LPO, we have so far followed the Who clinicopathological criteria,[19] although some authors question them. The clinical criteria would result in a presumptive clinical judgement that would have to be corroborated by histopathology.

## Clinical criteria

i. Presence of bilateral and mostly symmetrical lesions.
ii. Presence of striated white endoplasmic-papular.
iii. Erosive, Atrophic, blistering and plaque lesions (always in the presence of the oral mucosa of white striations).

## Histopathological criteria

a) Presence of yuxtaepitelial inflammatory infiltrate in band.
b) Signs of hydropic degeneration of the basal stratum.
c) Absence of epithelial dysplasia.

The differential diagnosis of lichen planus lesions should be performed, in the form of white predominance, with other white lesions[20] among which we emphasize:

i. Injury by mechanical, physical or chemical agents; Self-induced factitial injury, radiation, other physical or chemical agents.
ii. Microbial lesions: syphilis, fungi.
iii. Immunologic disorders: cicatricial pemphigoid, lupus, Morphea.
iv. Anormogénesis, hyperplasia and benign tumors: white nevus, dyskeratosis, psoriasis.
v. Precancer and cancer: Leukoplakias

When we are faced with a form of lichen planus red predominance with erythema-desquamative lesions, the differential diagnosis should be performed mainly with other Vesiculo-erosive lesions[21] such as Erythema multiform, Pemphigus vulgaris or Pemphigoid.

## Treatment

There is No drug that solves the lichen planus alone and

definitively. The most effective medications are corticosteroids, retinoid, cyclosporine and PUVA.[22]

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## Conflict of interest

Author declares that there is no conflict of interest.

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