# Desquamative Gingivitis

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Desquamative gingivitis is a clinical condition with a chronic course, not specific to a particular disease, characterized by intense erythema, scaling, vesicles, and/or blisters that may involve both the marginal free gingiva (MG) and the neighboring adherent gingiva (AG).

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## 1. Introduction

The term “chronic desquamative gingivitis” was first mentioned in a text in 1894 by authors J. Tomes and G. Tomes [1], and it was not until several years later, in 1932, that the term “diffuse chronic desquamative gingivitis” was introduced by Prinz [2], which was used to describe a clinical manifestation characterized by intense erythema, desquamation, and vesicles and/or bullae, subsequently resulting in erosions and/or ulcers, that could involve both the marginal free gingiva (MG) and the neighboring adherent gingiva (AG). Subsequently, following McCarthy’s 1960 hypothesis, Glickman and Smulow illustrated in 1964 that desquamative gingivitis (DG) is a nonspecific clinical manifestation that can be observed in a large number of pathological conditions [3][4]. Most cases in which DG is found in the clinical setting occur when a mucocutaneous pathology is present, such as oral lichen planus (OLP), mucous membrane pemphigoid (MMP), and pemphigus vulgaris (PV), which are the diseases most frequently associated with DG [5][6].

Lichen planus (LP) is a chronic recurrent immune-mediated inflammatory dermatosis of unknown etiology that can affect skin, mucosa, and nails. It most frequently affects Caucasian women between 50 and 70 years of age, and the oral cavity’s sites experiencing the greatest impact, in descending order of frequency, are the posterior buccal mucosa, the gingiva, the dorsum and lingual margins, the palate, and the lips, almost always with a tendency to produce bilateral and symmetrical lesions [7][8][9]. OLP lesions are characterized by altered immune regulation mediated by CD8+ T lymphocytes, resulting in damage at the level of the keratinocytes, with a specific target of the pathology being basement membrane keratinocytes. At the histopathological level, different degrees of hyper-, ortho-, and parakeratosis, liquefaction of basal cells, basal membrane thickening, and atrophy of the epithelial ridges can be observed in such lesions; at the level of the lamina propria, however, a banded infiltrate formed predominantly by T lymphocytes is observed [10][11][12]. The worldwide prevalence of LP lesions at the level of the oral cavity is estimated to be between 1% and 2%: almost half of these lesions see gingival tissue involved in an obvious manner, and about 10% of patients with OLP have only gingival manifestations in the form of an erythematous lesion, which is clinically referred to as “desquamative gingivitis” [13][14][15]. OLP occurring at the gum level can be identified in clinical terms by the existence of redness (atrophic lichen planus), erosions, and/or

ulcerations (erosive lichen planus) of bullae/blisters (bullous lichen planus). In each of these gingival manifestations, OLP lesions vary in the clinical form, extent, and degree of gingival involvement [16][17][18].

Mucous membrane pemphigoid (MMP) and pemphigus vulgaris (PV) represent the main subtypes of autoimmune bullous diseases that most commonly and predominantly affect the oral mucosa; the former most frequently affects female subjects aged 50–70 years, electively in the oral mucosa (90% of cases) and conjunctival mucosa (65% of cases); the latter, on the other hand, predominantly affects skin and oral mucosa, having precisely the latter as the site of first appearance in 50–70% of cases, without, however, presenting a clear predilection for either sex [19]. MMP localizes electively at the gingival tissue level, causing erythema, edema, scaling, blistering, and ulceration; this frequently leads it to be mistaken for periodontal disease. Other less frequent localizations are the buccal mucosa, tongue, and palate. MMP lesions are characterized by the linear deposition of IgG, IgA, or C3 along the epithelial basement membrane zone and the presence of autoantibodies directed against an antigen present at the basement membrane level, BPAG-2. This leads to a clear separation of the epithelial lamina from the underlying lamina propria, with the formation of a subepithelial blister [20][21]. Instead, oral PV lesions are due to the presence of autoantibodies directed against a component of the desmosome–plasma membrane complex, namely a transmembrane protein called desmoglein-3 (Dsg-3), and this results in the creation of an intracellular blister due to acantholysis, with a process likely related in this formation to the release of a plasminogen activator and proteinase that is followed by damage to the desmosomes. Once more, it is feasible to visually detect the accumulation of IgG or C3 in proximity to the basement membrane using direct immunofluorescence [22][23][24].

Desquamative gingivitis (DG) is a clinical condition that occurs more frequently in female subjects than in male subjects—a ratio of about 4:1—and in most cases, it appears after the age of 30, although it is observable at any age, starting from puberty and forward. It generally presents a chronic type of manifestation, with periods of remission and exacerbation: gingivitis may heal after a few months or, in some cases, DG may persist for several years [25][26]. Oral manifestations of desquamative gingivitis can vary in both size and seriousness, spanning from minor, restricted lesions to larger affected regions and occasionally accompanied by episodes of spontaneous bleeding [27]. Generally, when the first clinical signs of DG appear, erythema and minimal desquamation of the mucosa initially predominate; only later does the appearance of vesiculobullous lesions occur. There have been several observed cases of extensive areas of ulceration [28]. Less severe cases, characterized by limited symptoms, can result in heightened sensitivity to spicy and/or acidic foods and discomfort when using specific toothpaste. In contrast, more extreme cases with widespread symptoms typically cause significant and adverse effects on the patient's overall quality of life [18][29]. Painful gum and mouth lesions can deter patients from carrying out thorough and efficient tooth surface brushing, potentially raising the risk of long-term periodontal tissue damage due to the buildup of plaque at particular locations. Additionally, they may intensify gum inflammation in regions adjacent to the lesions caused by DG [30][31]. Other indirect consequences of plaque accumulation on the dental elements may be the occurrence of dental caries and halitosis [32][33][34][35]. The direct effects, on the other hand, that DG might have on periodontitis seem to be plausible, as there is some sharing of both pathogenetic mechanisms and mediators of inflammation at the base [36]. Certainly, the development of periodontal disease is indeed connected to a localized inflammatory response and the activation of the immune system triggered by bacterial components [37]. This process heavily relies on the stimulation of pro-inflammatory molecules and the

activation of cytokine networks, particularly interleukin-1 (IL-1) and tumor necrosis factor alpha (TNF-α) [38]. Conversely, autoimmune–inflammatory mechanisms are also pivotal in the development of most diseases associated with DG, often involving shared cytokine molecules/networks (for example, TNF-α in the case of OLP) [18][39]. Consequently, one could speculate that the simultaneous presence of bacterial-induced inflammation and autoimmune-related inflammation might exacerbate the harm inflicted on the tissues supporting the teeth.

## 2. Desquamative Gingivitis, Oral Hygiene, and Autoimmune Oral Diseases

Desquamative gingivitis (DG) is a clinical nonspecific condition distinguished by intense erythema, scaling, blistering, and/or vesicles and may involve both the marginal free gingiva (MG) and the nearby adherent gingiva (AG) [2][3][4]. This peculiar form of gingivitis, with a chronic course, has a higher frequency of onset in the female sex—M:F ratio of about 1:4—and in most cases tends to appear after the age of 30 years, peaking between 50 and 70 years of age, with local clinical manifestations commonly linked with oral lichen planus (OLP), mucosal pemphigoid (MMP), and pemphigus vulgaris (PV) [5][6][25][26]. Oral manifestations of DG can vary in both extent and severity: one can have mild, localized lesions, which can lead to sensitivity to certain types of particularly acidic and/or spicy foods and to some particular toothpastes; or one can have more extensive, sometimes bleeding and painful lesions, which can worsen both their lifestyle and their overall oral health [18][27][29][30][31].

While painful gum and oral lesions can impede effective oral hygiene routines and raise the likelihood of dental plaque buildup, there is currently limited research available on at-home oral care for individuals with DG and its potential impact on the development or continuation of periodontal disease in this population.

In 1985 and 1990, Erpenstein and Holmstrup et al. were the first to emphasize the significance of periodontal treatment and plaque management in individuals with ulcerative or atrophic lesions associated with OLP. Specifically, Holmstrup et al. gave their patients as atraumatic an oral hygiene protocol as possible, modifying their normal habits and including—when necessary—rinses with chlorhexidine 0.12% to further improve plaque control. Patients were observed over a span of 12 months, with follow-ups approximately every 3 months; this enabled us to showcase that post-treatment monitoring plays a crucial role in treatment plan formulation, as it can both facilitate enhancements in the patient's oral hygiene and alleviate painful symptoms concurrently. It was immediately apparent from these early studies that dental plaque control led to an elimination of the disorder's underlying cause; nonetheless, under certain circumstances, there was a positive change in the symptoms and seriousness of gingival lesions [40].

After these two articles, few other uncontrolled studies have demonstrated similar results in a small sample of patients. Guiglia et al., in a 2007 article, reported positive clinical effects by assigning, to a group of 30 patients with OLP and suffering from DG, both a professional and autonomous oral hygiene protocol and combining it with the application of topical steroids. The protocol included oral hygiene instructions, supra- and subgingival scaling, and the administration of chlorhexidine mouthwash at a concentration of 0.12% for a duration of 7 days alongside the

topical application of steroids. Patients were evaluated for about 3 months with follow-ups every month, in which the importance of proper home oral hygiene was reinforced to them [41].

In 2010, López-Jornet and Camacho-Alonso released a study with the objective of assessing the efficiency of a protocol designed to motivate oral hygiene, which included scaling, supragingival polishing of dental elements, and administration of a topical corticosteroid, in a group of 40 individuals with gingival lichen planus. The protocol included follow-ups after 4 and 8 weeks in which home oral hygiene instructions were reinforced; only when necessary were scaling and polishing of the teeth repeated. There was both a reduction in bacterial plaque and a reduction in gingival bleeding [42].

Salgado et al., in a 2013 publication, demonstrated that the reduction of dental plaque had the strongest correlation with enhancements in both the clinical presentation and symptom relief in a cohort of 20 patients suffering from OLP, specifically within the gingival tissues affected by the lesions [43].

Unlike prior research, indeed, despite the occurrence of an immune–inflammatory response associated with a chronic disease, they showed that the key to maintaining gingival health and improving painful symptomatology and gingival lesion severity is imperative to achieve adequate plaque control through the setting of proper supportive periodontal therapy. In 2015, Stone et al. were the first with their study to offer compelling proof of the significance of maintaining plaque control when treating patients with OLP gingival lesions. The randomized controlled trial involved a total of 82 patients, divided into two groups: the first received home oral hygiene instruction with sonic-type electric toothbrushes and interdental brushes; the second, on the other hand, was instructed to continue with their usual oral hygiene maneuvers. Both groups underwent follow-up visits at 4 and 20 weeks, and it was shown that the intervention group, compared with the control group, had statistically significant ($p < 0.05$) reductions in perceived pain, functional limitation, psychological discomfort, and plaque indices [44].

Subsequently, in 2018, Bianco et al. published a randomized controlled trial of a total sample of 32 patients diagnosed with desquamative gingivitis, in which the efficacy of intensive plaque control programs with a sonic toothbrush and with a manual toothbrush were compared: patients were divided into two separate groups, with the first receiving oral hygiene instruction with a sonic toothbrush and the second, on the other hand, receiving instruction with a manual one, and both clinical outcomes and matrix metalloproteinase (MMP) levels within the gingival crevicular fluid (GCF) were assessed. While there were no statistically significant variances observed between the utilization of a sonic toothbrush and a manual toothbrush, the group that employed the sonic toothbrush exhibited a statistically notable decrease in MMP-1 and MMP-9 levels [45].

Mergoni et al. in 2019 conducted a study of 60 patients—divided into an intervention group and a control group—with OLP with gingival lesions. This study was very similar in setting and results to the study by Stone et al. but with the difference that the intervention group was provided with manual soft brushes. The rationale for this choice was that this type of toothbrush appears to be more widespread in Italy to date [46].

Although there are—albeit few—findings in the literature inherent in the relationship between desquamative gingivitis and plaque control, to date there are still few published studies that have directed their focus on the role that periodontal pathogenic bacteria, and thus overt periodontal disease, have in influencing the course of gingival lesions ascribable to the term desquamative gingivitis. In 2017, a study conducted by Arduino et al. examined 11 bacterial species found in lesions associated with desquamative gingivitis related to oral lichen planus (OLP) and in cases of plaque-induced gingivitis. The research revealed a higher occurrence of bacterial species like Fusobacterium nucleatum, Aggregatibacter actinomycetemcomitans, and Eikenella corrodens in specimens from OLP patients compared to those without OLP [47]. Further insights into the bacterial role in the pathogenesis of OLP were provided by Choi et al. who, in patients with OLP lesions, found that resident oral cavity bacteria can (1) compromise the epithelial physical barrier, (2) be internalized into epithelial cells or T cells, and (3) promote T-cell chemokine release. Furthermore, they have demonstrated that the existence of pathogens in both the lamina propria and epithelium is closely linked to elevated T-cell levels in OLP tissues, implying that intracellular bacteria can have a substantial impact on the influx of immune cells [48].

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