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Desquamative Gingivitis – A Review of Literature

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ABSTRACT:

A clinical condition known as gingival desquamation causes the gingiva to appear reddish, glossy, and friable due to epithelial damage. A kind of gingival keratinization disorder, desquamative gingivitis (DG) is characterized by erosions of the desquamative layer, edematous erythema of the free and connected gingiva, and chronic ulceration. A gingival disease process may be the cause of gingival desquamation. Skin involvement is infrequent; mucous membranes are typically affected. The majority of cases involve the oral cavity, with desquamative gingivitis being the most typical symptom. It has also been noted that contact allergies to several chemical agents and mouth hygiene products can manifest as DG. The aetiology of DG has proven to be elusive, which has made care of the condition extremely challenging. There are several etiologic variables associated with the development of said lesions. Even when aetiology is taken into account, systemic or topical corticosteroids are frequently used as a form of treatment. Since it's still unclear what causes this ailment.The pathophysiology, diagnosis, treatment, and prognosis of DG are covered in this review article.

INTRODUCTION

The term “chronic desquamative gingivitis,” first used by Prinz in 1932, refers to a group of gingival disorders characterised by extreme erythema, desquamation, and ulceration. (1). McCarthy (1960) described desquamative gingivitis as the gingival reaction to multiple related disorders. It was discovered that the gingiva was not specifically affected by a disease, as previously believed, but rather was a clinical presentation of other illnesses (2). Tomes & Tomes (3) originally documented chronic desquamative gingivitis in 1894. It is important to note that "desquamative gingivitis" is not a diagnosis in and of itself, but rather a phrase used to describe a specific clinical symptom (4, 3, 5, 6). Diffuse gingival erythema (DG) is a clinical disease characterized by the formation of vesicles, atrophy, erosion, and desquamation of the marginal and keratinized gingiva (5,7,8,9). Diffuse erythema and mild desquamation are the initial signs of a lesion. Even with the slightest trauma, the gingiva epithelium that is affected is extremely fragile and susceptible to exfoliation (3). In certain instances, large ulceration patches are seen(10). Desquamative gingivitis is common after puberty, especially among those older than 30 (3). Women are more likely to experience this than males. There are very few reports of it affecting youngsters (4, 3, 7, 11). In free and keratinized gingiva, degranulation of the epithelium is often seen. Even if lesions on the buccal/labial surfaces of the gingiva were not initially caused by bacterial plaque, their severity is often increased by plaque buildup (12). They can be seen in any gingival area (5), even though they are typically seen in the anterior region. Similar lesions are observed in the alveolar ridge of the edentulous teeth. Even though the patients only exhibit desquamations, ulcerative and lichenoid lesions may

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also be present, as well as vesicular-bullous lesions. In extreme situations, it may be observed to be widespread in the oral mucosa, affecting both the gingiva and the alveolar mucosa (12). The patient may have intense pain or a burning sensation, or they may have no symptoms at all. Chronic pain is prevalent in general and is particularly exacerbated by eating acidic foods. Pain can also lead to limitations on oral function and speech impairments are observed (4,5). Since gingival erythema does not relate to plaque or the presence of gingival desquamation, Nisengard and Levine (13) have listed a few signs to classify the clinical findings as DG. They further stressed that, in individuals with DG, positive Nikolsky phenomena is seen. In the initial cases of desquamate gingivitis (DG), only one etiologic component was taken into account. Since gingival desquamations were more prevalent in middle-aged women, it was first hypothesized that they were linked to the hormonal shifts that occur after menopause (12). Glikman and Smulow(14) noted in 1964 that DG may be a sign of serious illnesses, particularly those affecting the mucocutaneous tissues. It is now widely accepted that DG is unrelated to hormones and that it may be the first sign of vesicular bullous disorders as well as a reaction to certain chemicals and allergens. Features of Desquamative gingivitis given in [table 1]

Table 1 Features of Desquamative gingivitis

Gingiva has a friable, glossy, red, and fiery appearance.
The initial signs of a lesion includes diffuse erythema and mild desquamation.
Presents with painful erosions, ulcers, and blistering desquamation.
Oral manifestation include diffuse erythema of the marginal and keratinized gingiva.
Histopathologically connective tissue layer inflammation and edema, along with epithelium degradation.
The Nikolsky phenomenon is typically positive.
DIF, biopsies are necessary for diagnosis.
The basic principle of treatment is oral hygiene combined with topical steroids.

McCarthy et al.(2) developed a system for categorizing the etiology of desquamative gingivitis, including dermatoses, hormone impacts, and aberrant reactions to irritation, persistent infections, and idiopathic factors.

A. “Dermatological disease
- Cicatrial pemphigoid
- Lichen planus
- Psoriasis
- Bullous pemphigoid
- Epidermolysis bullosa acquisita
- Contact stomatitis

B. Endocrine disease
- Deficiencies of estrogen following oophorectomy in postmenopausal stage
- Testosterone imbalance
- Hypothyroidism

C. Aging

D. Abnormal response to bacterial plaque

E. Idiopathic

F. Chronic infection
- Tuberculosis
- Chronic candidiasis
- Histoplasmosis.

According to McCarthy et al., an etiology-based classification permits a rational therapeutic approach.

CLINICAL CHARACTERISTICS OF DESQUAMATIVE GINGIVITIS IN DERMATOLOGICAL DISORDERS

1. Lichen planus

Desquamative gingivitis is most often brought on by lichen planus. It's the root cause of around 45% of all instances of desquamative gingivitis.(15) The most prevalent sites of infection are the lateral edge of the tongue, the gingiva, and the posterior buccal mucosa.Desquamative gingivitis may be the sole symptom in 10% of those with oral LP (21).Its atrophic form reveals itself most often as gingiva, and it may affect the whole thickness of the connected gingiva. Plaque-induced gingivitis may complicate the clinical presentation.”Oral LP lesions and vulvovaginal LP lesions may be related (referred to as “vulvovaginal-gingival syndrome”) (17,16) The gingival and vulvovaginal lesions can develop simultaneously, as is frequently the case, or one develops after the other within two years of the other's commencement. When lichenoid oral lesions coexist with lichen sclerosus

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lesions in the mouth and vulva, a disease described as "lichen planus/lichen sclerosus overlap syndrome" can also develop (18). When gingiva is examined locally in these patients, erythematous gingiva with weak keratotic lines may be seen. It is important to thoroughly examine the skin and the remaining oral mucosa for characteristic lichenoid lesions.

2. Immune-mediated blistering conditions

Desquamative gingivitis (19) is a frequent first symptom in people with mucous membrane pemphigoid.Desquamative gingivitis has been associated to mucous membrane pemphigoid in roughly 35% of cases (2). It is common to see erythematous gingiva without stippling that spreads apically from the gingival margins to the alveolar mucosa. Patches of superficial erosions in the gingiva may be followed with tags of detached epithelium (20). A positive Nikolsky's sign and vesiculobullous lesions are possible.

Several illnesses within the pemphigus group, especially pemphigus vulgaris, have oral lesions as their primary symptom. Bullae rupture leads to significant gingival desquamation, erosions, and ulcers, all of which distinguish this condition(21). The Nikolsky symbol is noticeable and encouraging. Desquamative gingivitis is also linked to intraepidermal IgA pustulosis and paraneoplastic pemphigus (15). Oral lesions may be the presenting symptom, occur simultaneously with or after cutaneous manifestations, or be the sole symptom present.Desquamative gingivitis is a rare complication of dermatitis herpetiformis, although it has been linked to a history of gluten intolerance (21). This condition is characterized by erythema, white lesions, and erosions in the buccal and lingual gingiva.Desquamative gingivitis (22) may present themselves as oral vesicles, ulcers, and gingival lesions in adults with linear IgA disease.

MISCELLANEOUS CONDITIONS

1. Chronic ulcerative stomatitis

It's an uncommon, persistent autoimmune condition with painful, long-lasting mouth lesions resembling erosive lipomatosis. The results show erosions and ulcers in the lingual, buccal, and gingival mucosae. Rarely, it could be connected to mucosal lesions of the conjunctiva and genitalia. IgG nuclear deposits are seen in the basal third of the epithelium, according to DIF investigations (23)

2. Erythema multiforme major

The oral mucosa is nearly always involved in erythema multiforme major. The oral lesions may appear alone, in conjunction with, or before to the development of the cutaneous lesions. Lip edoema, labial erosions, intraoral blisters, and serosanguinous discharge are among its symptoms (21).Despite reports that gingival involvement is rare, the widespread oral ulcers and sloughing associated with this condition may make it easy for neglect when it occurs (24).

3. Systemic Lupus Erythematous

Oral lesions are one of the main signs and symptoms of lupus erythematosus. This case of gingivitis is more likely to be localised than generalised. The presence and severity of gingival lesions are predictive of disease progression. Discoid lupus erythematosus causes plaques on the palate and oral mucosa that resemble lichen planus. Ulcerative, hyperkeratotic plaques on the palate and buccal mucosa are characteristic of systemic lupus erythematosus (25). Desquamative gingivitis may progress to discoid lupus if it's followed by erosions of the labial mucosa and a bloody discharge. The blue red color of edematous gingival margins is a telltale indicator of capillary involvement in mixed connective tissue diseases and dermatomyositis (25).

4. Epidermolysis bullosa

The features of this condition include microstomia, scarring, recurrent blistering, oral mucosal fragility, and abnormalities in the enamel. Furthermore, gingival involvement (such as in epidermolysis bullosa simplex, Dowling – Meara type) may manifest as gingival hyperkeratosis and desquamative gingivitis (26).

5. Plasma cell gingivitis

Although uncommon, desquamative gingivitis might be misdiagnosed as medicamentosa stomatitis, an allergic response to products like mouthwashes, flavored mints, chewing gum, etc. Stomatitis venenata is a comparable reaction that may develop as a contact allergic response to medicines taken orally or administered intravenously (such as penicillin or aspirin burns) (27).

6. Toxic epidermal necrolysis

Gingival desquamation may be a prominent symptom of this condition. HIV/AIDS patients are more likely to experience this, which is accompanied by systemic symptoms as fever, coughing, malaise, etc. (21).

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CONDITIONS SIMILAR TO DESQUAMATIVE GINGIVITIS

1. Foreign body gingivitis

It is a persistent inflammatory disease that is not triggered by plaque and rarely affects the gingiva (38). It occurs when foreign particles, such as dental prophylactic paint or restorative material, are introduced. It could appear as granulomatous or lichenoid reaction disguising itself as desquamative gingivitis (28).

2. Candidiasis

Oral candidiasis can affect the gingiva, especially in people with compromised immune systems. Because of this, it appears erythematous and granular and is sometimes confused with desquamative gingivitis (39).

3. Wegener's granulomatosis

These oral diseases manifest as painful, erythematous mouth ulcers and gingival enlargements (sometimes known as "strawberry gingivitis"). this may be confused with desquamative gingivitis (40).

4. Graft-versus-host disease

Allogeneic hematopoietic stem cell transplantation may result in oral graft-versus-host disease that can impair gingival tissue. In the buccal, glossal, and labial gingiva, this results in erythema, erosions, and epithelial desquamation, producing a clinical appearance similar to "desquamative gingivitis (41).

5. Kindler's syndrome

The gingiva appears erythematous and erosive. Acral atrophy, poikiloderma, photosensitivity, and newborn bullae are among the prominent cutaneous features (42).

6. Factitious gingivitis

Self-inflicted oral injuries might resemble lesions of desquamative gingivitis, although being unusual in their location and mode of occurrence (43).

7. Squamous cell carcinoma

Gingival enlargement is a manifestation of gingival squamous cell carcinoma, which arises from the keratinized mucosa. It is possible to misdiagnose this condition as desquamative gingivitis (43).

PATHOGENESIS

In slices of gingival tissue, the ground substance of the basement membrane and the subjacent connective tissue may be seen using the periodic acid-leucofuchsin technique (34). This process has been particularly helpful in assessing these alterations because the connective tissue is the source of many gingiva problems (35). Extreme gingival alterations are seen in desquamative gingivitis, which may indicate alterations in the connective tissue. Clinical findings demonstrate that, with the touch of a finger or with a blast of air, the epithelial layer neatly separates from the underlying connective tissue. Several areas of the gingiva are ulcerated and bleeding (35). Histopathological examination of this disturbance by Ziskin and Zegarelli revealed changes.Third, they noted swelling and inflammation in the connective tissue layer, as well as damage to the epithelium (36). "Epithelial thickening or thinning, inflammation, damage to the basement membrane zone (BMZ), disruption of the cell-cell and cell-basement membrane adhesion systems of epithelial cells, and the formation of vesicles and/or bullae (37), all result from immune-mediated processes that alter the biology of epithelial and subepithelial areas. Either the basement membrane was nonexistent, uneven, or enlarged. These modifications were also seen in the basement membranes of the connective tissue's many enlarged blood vessels. Disintegration of connective tissue fibers was seen at a variety of sites (35). Desquamative gingivitis is characterized by a disruption in the ground substance of the gingival connective tissue."The interstitial components' changing features are an indication of this. a) The basement membrane has either been completely dissolved or is only present in a limited area. (b) Glycoprotein residues in the soil are more abundant and may be found in both water and alcohol at greater amounts. Partially responsible for these changes is the erroneous production of depolymerizing enzymes, which modify the ground material and cementing substance of the epithelial cells(35).

INVESTIGATIONS

Sample collection is required for exfoliative cytology and/or biopsy. During the surgical procedure, special attention should be given to the selection of the biopsy site and the handling of tissue (especially in cases of bullous lesions). It is necessary to submit a sample of the formalin-fixed biopsy material for histopathologic analysis and to use a portion that has been rapidly frozen in liquid nitrogen for DIF staining. Typically, IIF—which makes use of the patient's serum—usually completes the diagnostic procedure (45)

Histopathological examination

A biopsy of the perilesional mucosa is superior to a biopsy of the ulcerated mucosa.Tissue is collected,

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fixed in formalin, embedded in paraffin, and stained with H&E according to industry standards(2,21) for light microscopy investigation.Table 2 summarizes the histological hallmarks of the most prevalent mucocutaneous diseases manifesting as desquamative gingivitis.

Immunofluorescence investigation

In order to do direct immunofluorescence, unfixed frozen sections are incubated with a variety of fluorescein-labeled antihuman sera (anti-IgG, anti-IgA, anti-IgM, antifibrin, and anti-C3). In indirect immunofluorescence, the patient's serum is used to treat unfixed frozen slices of monkey oral or esophageal mucosa, allowing any antibodies to bind to the mucosal tissue. Desmosome-shaped proteins Dsg 1 and Dsg 3 are targeted by IgG autoantibodies in response to stimuli, leading to blister formation on the epithelium. The skin is affected by the generation of Dsg 1 autoantibodies in Pemphigus foliaceus. Although both Dsg 1 and Dsg 3 are expressed in the skin, the expression of Dsg 1 is more prevalent in the superficial layer, whilst that of Dsg 3 is more frequent in the basal and suprabasal layers. Dsg 3 is mostly expressed in oral epithelium. Desmosomal cadherin, made up of Dsg 1 and Dsg 3, is what keeps epithelial cells together. Bullae occur in the suprabasal area of PV as soon as the spinous cells become less sticky as a result of anti-Dsg 3 anti-bodies. Antigen Dsg 4 is one of a kind when it comes to pemphigus. In addition to Dsg antigens, other molecules that bind acetylcholine and molecules that mimic keratinocyte annexins, such as pemphaxin (52), may have a role in the etiopathogenesis of the illness.

The immunofluorescence results in a few dermatological disorders that manifest as desquamative gingivitis are summarised in Table 2.

Table 2 Histological features and Immunofluorescence of Disorders Associated With DG

Disease	Histopathology	Immunofluorescence	
		Direct	Indirect
Lichen planus	Saw toothed rete pegs, hydropic degeneration of the basal layer, and hyperkeratosis; Colloid bodies are visible in the lamina propria, along with a thick, band-like infiltration mainly composed of lymphocytes. (28)	Fibrin, fibrinogen at “BMZ* (linear,shaggy pattern);cytoid bodies (47)	Negative (47)
Mucous membrane pemphigoid	subepithelialclefting with intact basal layer and epithelial separation from the underlying lamina propria (28)	C3, IgG at BMZ (linear pattern) (47)	Circulating basementmembraneantibodies often non detectable (47)
Pemphigus	There is intraepithelial clefting above the basal cell layer; acantholysis is evident; and basal cells have a characteristic “row of tombstone” appearance (28).	“IgG, IgA, IgM, complement within the epithelialintercellular spaces(reticular pattern) (47)	Antiepithelialantibodies(anti-Dsg3 anti-Dsg1) (47)
Chronic ulcerative stomatitis	liquefaction of the basal cell layer, subepithelialclefting, hyperkeratosis, acanthosis, and persistent, band-like lymphohistiocytic infiltration (28)	Speckled, finelygranular pattern of IgG depositionin the nuclei ofKeratinocytes(confined to thebasal cells andlower thirdof the spinouslayers)	Antibodies tostratifiedepithelia (47)

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		(47)	
Dermatitis herpetiformis	Neutrophils, eosinophils, and fibrin accumulate in the connective tissue papillae (28)	IgA in the upperpapillary dermisof perilesional skin(granular pattern) (47)	IgA endomysial antibodies in 70% of cases; antigliadin antibodies in 30% of cases (28)
Bullous pemphigoid	subepithelialclefting that leaves the basal layer intact and causes epithelial separation from the underlying lamina propria (28).	C3 Linear deposits with or without IgG at the basement membrane zone in almost of the cases (28)	IgG antibodies against basement membrane zone 40%-70% of cases (28)
Systemic Lupus Erythematosus	perivascular inflammation, basal cell degeneration, hyperkeratosis, and epithelial atrophy (28).	IgM, IgG, and C3 at BMZ(shaggy,granular pattern)and perivascular (47)	>95% of patients had ANA; >50% had DNA and ENA antibodies (28)
Epidermolysis bullosa acquisita	subepithelialclefting that leaves the basal layer intact and causes epithelial separation from the underlying lamina propria (28).	C3 and IgG at BMZ (thick linear pattern) (47)	Positive on saltsplit skin:antibodies toskin BMZ(dermal part);antibodiesto type VIIcollagen (47)

ANA: Anti-nuclear antibodies, ENA: Extractable nuclear antigen, BMZ: basement membrane zone

DIAGNOSIS

The process of diagnosing a condition associated with DG involves taking a thorough medical history, review of past and present medical history, and a complete clinical examination (45).

- **Medical history and history of complaint**

History: The beginning, progression, aggravating and alleviating variables, and previous treatments received should all be considered during the history-taking process. Patients may present with no symptoms at all or, more frequently, report chronic gingival discomfort, a little burning sensation, or excruciating pain at the afflicted location.(2).The patient's past medical history of systemic diseases and current medications should be taken into account in a proper medical history. This will be crucial in figuring out possible conditions that could be associated with DG. It is particularly crucial to record any history of extraoral skin lesions or eye issues. Any treatment the patient is receiving from additional relevant fields, such as dermatologists, ophthalmologists, gynaecologists, and ear, nose, and throat specialists (45). When OLP lesions first appear, they may not cause any symptoms, and the patient may not even be aware that they exist. Patients who appear with full desquamative lesions may experience a range of symptoms, from minor discomfort while brushing their teeth or eating certain meals (particularly spicy or acidic foods) to more severe pain that negatively affects their quality of life. Noting a history of blistering or ulceration is crucial because it could point to vesiculobullous disorders. It is important to look into how lesions start and develop (45). It may be likely to determine possible triggering factors, such as medications or infections, by looking at the events of the day or weeks before the lesions appeared. Noting the products used for oral hygiene regimen is also crucial. A common detergent that is used in a lot of dental hygiene products is sodium lauryl sulphate. When used by certain patients, it has been linked to mucosal irritation (46). Eating acidic foods, like citrus fruits, may make the symptoms worse. Additionally, search for deliberate or inadvertent local aggravating causes such as deposition of plaque, masticatory pressures, poor dental restorations, etc. It is recognised that there are periods of remission and aggravation for symptoms.(28).

- **Clinical examination**

The most often affected part of the gingival epithelium is the buccal/labial surface (free attached gingiva). Clinically, desquamative gingivitis presents as burning red, glazed, atrophic, or eroded gingiva.(17, 21) Less severe cases contain erythema and edema. In more

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extreme situations, mucosal desquamation with blister formation, erosions, ulcerations, and paraesthesia may occur. Nikolsky's sign, which is indicative of a variety of vesiculobullous disorders and entails the creation or spread of a blister under lateral pressure applied to the mucosa that seems normal, requires a thorough local examination.

Finding oral lesions with a distinct appearance that can help diagnose various DG-associated conditions is the aim of the clinical evaluation.

OLP lesions often present with highly distinctive characteristics. They typically have a keratotic papular or reticular shape and affect several oral areas. It may be challenging to distinguish a desquamative type lesion linked to OLP from other types of DG when it is present in the attached gingivae; however, keratotic lesions are frequently present at the gingival margin or scattered throughout the affected area, which might support a provisional diagnosis (45).

Additionally, the location of dental restorative materials and their association with the lesions should be carefully noted. It has been noted that certain restorative materials, such as amalgam, composites, nickel, and cobalt, can cause reactions similar to lichenoid type. Lesions resemble OLP lesions in appearance, however they might resolve when different dental material is used in instead of the original one. In order to detect hypersensitive reaction, some of these patients may benefit from a referral for patch testing (46).

Mucous membrane pemphigoid and Pemphigus vulgaris lesions might initially appear as blister formation and broad erythema, then break down to cause ulcerations in specific areas (45).

- **Definitive diagnosis − Referral to an expert**

For additional testing and a conclusive diagnosis, a specialist referral to an oral medicine or periodontology facility is usually necessary. This would typically involve direct immunofluorescence and H&E staining analysis of a tissue sample in addition to indirect immunofluorescence on a blood sample (45).

Diseases linked to DG typically have histological alterations that affect both the underlying connective tissue and the epithelium. OLP changes often manifest as variations in epithelium thickness, which can range from reduction (erosive types of OLP) to increases (hyperkeratosis of OLP). Blister production is a characteristic of antibody-mediated diseases including Mucous membrane pemphigoid and Pemphigus vulgaris, which are subepithelial and intraepithelial, respectively (45).

Immunofluorescence investigations can be used to examine pathologic immunoreactants found in DG-associated conditions in addition to histological evaluation. Immune complexes, complement cascade components, fibrinogen and fibrin, and different immunoglobulin classes (IgG, IgM, and IgA) are examples of immunoreactant types (47). When typical characteristics are absent from histopathological investigation, immunofluorescence tests are extremely helpful in accurately diagnosing disease associated with DG.

Recently, optical coherence tomography (OCT) has been proposed as a first non-invasive diagnostic method for most immune-mediated diseases that may produce desquamative gingivitis (DG) (54). This diagnostic tool has been shown to show distinct patterns in DG-mediated pemphigus vulgaris (e.g., decreased epithelial thickness, intraepithelial unilocular blister, and acantholytic cells present in the blister) and mucous membrane pemphigoid (e.g., presence of inflammatory infiltrate and multilocular subepithelial blister). Additionally, OCT enables the storing and reviewing of images whenever necessary, which facilitates the tracking of treatment results and the early detection of illness recurrence before symptoms manifest (54, 55). Though research in this area is lacking, OCT may provide a new non-invasive approach for clinical preliminary evaluation and surveillance of such complicated disease (56).

DIFFERENTIAL DIAGNOSIS AND PROGNOSIS

Desquamative gingivitis (DG) can be differentiated from a wide range of conditions, including mucocutaneous illnesses, allergic reactions, chemical and electrical burns, and hormone imbalances.

Moreover, responses emerging against mouthwashes , chewing gum, cosmetics, medications, cinnamon, and dental materials show a similar clinical pattern (29). It has been proposed that the condition may manifest in the absence of progesterone or oestrogen (30). Furthermore, idiopathic gingival desquamative lesions devoid of any known aetiology might be found. There is controversy about whether this is a clinical manifestation of mucous membrane pemphigoid illness or a sign of oral lichen planus. It has been stated in numerous publications that DG and lichen planus are related. According to numerous reports, DG is

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associated with pemphigus vulgaris, mucous membrane pemphigoid, and lichen planus (88%–98%) (5, 8, 12, 29).

Desquamative gingivitis has an incredibly complicated diagnostic process. It may take some time to identify the underlying systemic condition or the cause of the lesions. Patient drug use, chemical exposure, and family history should also be investigated, as should the occurrence of additional areas on the body where comparable lesions have appeared (3). A patch test for dental materials may be performed (29) if there is concern that the patient may be allergic. Histopathological examination of tissue samples taken from lesions, as well as direct (DIF) and indirect (IIF) immunofluorescence examinations, and blood autoantibody testing, may provide a conclusive diagnosis (31).

Trauma, plaque buildup, or improper brushing usually make the clinical situation worse. When oral hygiene practises are disrupted, the clinical picture deteriorates because of pain and bleeding (3, 29). The condition worsens and remits periodically, but it never goes away. It could take months for the gingiva to recover (32). There have been reports of desquamative gingival lesions over a range of durations, from two months to twenty-five years, despite differences in their intraoral appearance (12).

TREATMENT

Collecting medical history information thoroughly is necessary for the management of desquamative gingivitis. Clinical and laboratory confirmation come next. Effective management of the illness and a favourable prognosis are made possible by the differentiation of the disorders (15)

➢ **General Treatment Considerations**

- Management of the underlying condition: Resolving the lesions associated with desquamative gingivitis requires prompt identification and adequate management of the underlying dermatological condition(33).
- Oral Hygiene: It is important to teach patients how to take care of their teeth without injuring themselves chemically or mechanically. This needs to be emphasised because using the wrong brushing or flossing method will make the lesions worse (28).
- If a skin patch test reveals the existence of allergen sensitivities, topical treatments such as antiseptic mouthwashes, toothpaste containing sodium lauryl sulphate, cosmetics, flavours, and food additives should be avoided (28).
- Drugs that have been discovered to be the cause of the disease should be discontinued (28).
- When using air abrasive polishing, care must be made to avoid applying it to gingiva since it might penetrate them and exacerbate the problem(28).

➢ **The Symptomatic Approach**

- For circumscribed lesions, topical corticosteroids are the primary line of treatment (21,53).
- Fluticasone propionate nasal spray (50 microgram/spray) and beclomethasone dipropionate inhaler (50-100 microgram/spray) can be used directly to local lesions four times a day (28).
- For moderate localised lesions, fluorinated medications like fluocinonide (0.05%) are effective; for more severe lesions, topical tacrolimus (0.03%, 0.1%, 0.3%), clobetasol propionate (0.05%), and triamcinolone orabase (0.1%) are effective. Gel-based formulations are the recommended choice since they easily penetrate into the oral mucosa. It is further enhanced by the use of custom-made splints and silicone or acrylic carriers (28).
- If topical therapy is ineffective, 40–80 mg of oral prednisolone can be given daily for a week or 2-4 times a week for severe exacerbations of desquamative gingivitis, or 0.5–1 ml of a 1 mg/ml suspension of triamcinolone (28).
- In children with severe, refractory cases of desquamative gingivitis, dapsone is preferable due to their age and the possible adverse effects of oral steroids (28).
- Analgesic mouth rinse with 0.15% benzophenone hydrochloride can be used (28).
- Steroid-sparing drugs such as azathioprine, cyclosporine, and dapsone may also be utilized (28).
- Antifungal medications can be used to treat superinfection with candida (28).

SPECIFIC THERAPIES

- **Oral lichen planus (OLP)**

The preferred medication for symptomatic lesions is a topical corticosteroid. Topical corticosteroids have an anti-inflammatory and immunosuppressive impact and come in a variety of strengths and formulations. For the

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treatment of minor, isolated lesions that are easily accessible, like those on the tongue, palate, gingiva, or buccal mucosa, ointments like fluticasone combined with Orabase® are appropriate. Apply for 3−4 times daily.

When many lesions are present, corticosteroid mouthrinses, such as those containing fluticasone 400 mcg nasules or betamethasone 500 mcg soluble tablets, can be effective. It has also been suggested that medication persistence to oral lesions might be increased by using vacuum-formed steroid carrier trays (45). It's crucial that these personalized trays don't cause any harm to the gingiva while being used.Patients should be warned that secondary candidiasis is a frequent adverse reaction to topical corticosteroid formulations. Miconazole gel or chlorhexidine mouthwash are two examples of antifungals that may help stop candidiasis from returning (45).

The duration of corticosteroid therapy may be reduced with the use of other medications that "dampen down" the immune system. Among them are mycophenolate mofetil and azathioprine. Patients who are allergic to steroids may get relief from using topical calcineurin inhibitors such tacrolimus or pimecrolimus (49). Temporary burning or stinging at the application site is one of the side effects that has been documented.

- **Mucous membrane pemphigoid (MMP)**

When treating patients with severe, progressive mucosal lesions due to immune-mediated diseases, systemic corticosteroids are often the first line of defense (45).

Systemic corticosteroids given for a short period of time (2-3 weeks) may help improve DG lesions; topical medications may be used in combination with or after systemic corticosteroids. It is important to think about the risks associated with systemic steroid therapy, such as adrenal suppression, high blood pressure, impaired vision, high blood sugar, and gastrointestinal bleeding. Once again, the severity and persistence of the lesions will determine whether or not systemic immune suppressants such azathioprine and mycophenolate mofetil will be required. Patients with MMP who had both oral and ocular lesions have responded well to dapsone treatment. Considerable adverse effects include fatigue, anaemia, and shortness of breath (50).

- **Pemphigus vulgaris (PV)**

Systemic corticosteroids at moderate to high doses are the cornerstone of treatment for PV due to its mortality risk. Again, extra immunosuppressive medication may be needed. Steroid-resistant PV has previously been successfully treated with intravenous immunoglobulins (51).

CONCLUSION

In order to properly diagnose desquamative gingivitis, a complete history, an intra- and extraoral examination, a gingival biopsy for histology, and immunofluorescence tests are all required. Gingivitis may also be a clinical indicator of dermatitis and mucocutaneous diseases. Any lesions in the skin that coexist or develop later on, as well as any mucosae in the genitalia and eyes, must be recognised and treated. In order to effectively treat the underlying condition, a regular, comprehensive oral examination is required for these individuals. Desquamative gingivitis may be treated with occlusive topical corticosteroids and better oral hygiene.

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