Periodontology

Desquamative Gingivitis

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Is only a clinical term that describes a peculiar clinical picture. This term is not a diagnosis and a series of laboratory procedures should be used to arrive to final diagnosis.

It was used to describe a peculiar conditions characterized by intense erythema ,desquamation and ulceration of the free and attached gingiva.

Approximately 50% of cases are localized to the gingiva , although patients can have involvement of the gingiva plus other intraoral and extra oral sites.

Desquamative gingivitis is not a specific disease entity but a gingival response associated with a variety of conditions. Approximately 75% of cases have a dermatological genesis. Cicatricial pemphigoid , lichen planus ,bullous pemphigoid, pemphigus vulgaris and lupus erythromatosus can clinicaly manifest as desquamative gingivitis.

DIAGNOSIS OF DESQUAMATIVE GINGIVITIS:

A SYSTEMATIC APPROACH

The following represents a systematic approach to elucidate (explain) the disease triggering desquamative gingivitis.

- **History.**

Data regarding the symptomatology associated with this condition, as well as its historical aspects (i.e., when did the lesion start, has it gotten worse, is there a habit that exacerbates the condition, etc.), provide the foundation for a thorough examination.

Information regarding previous therapy that has been directed to alleviate the condition should also be documented.

Clinical Examination.

Recognition of the pattern of distribution of the lesions (i.e., focal or multifocal, with or without confinement to the gingival tissues.

In addition, a simple clinical maneuver such as Nikolsky's sign offers insight into the plausibility of the presence of a vesiculobullous disorder.

- Biopsy.

Given the extent and number of lesions that may be present in a given individual, an **incisional** biopsy is the best alternative to begin the microscopic and immunological evaluation.

Once the tissue is excised from the oral cavity, the specimen can be bisected and then submitted for **microscopic** examination.

In general, an incisional biopsy of uninvolved (normal) mucosa will show the same immunofluorescent findings as the biopsy of the perilesional tissue.

Immunofluorescence.

For direct immunofluorescence, unfixed frozen sections are incubated with a variety of fluorescein-labeled, anti-human serum (anti-IgG, anti-IgA, anti-IgM, anti-fibrin and anti-C3).

For indirect immunofluorescence, unfixed frozen sections of oral or esophageal mucosa from an animal such as a monkey are first incubated with the patients serum to allow the attachment of any serum antibodies to the mucosal tissue then the tissue incubated with fluorescence labelled antihuman serum.

Immunofluorescence tests are positive if a fluorescent signal is observed either in the epithelium, its associated basement membrane or in the underlying connective tissue.

Management.

Once the diagnosis is established, the dentist has to choose the optimum management for the patient.

This is accomplished according to the following three factors:-

1) practitioner's experience

2) systemic impact of the disease

3) systemic complications of the medications

In the first one, the dental practitioner takes **direct** and exclusive responsibility for the treatment of the patient. Such is the case in erosive lichen planus that is responsive to topical steroids.

In the second scenario, the dentist **collaborates** with another health care provider to concurrently evaluate and/or treat a patient.

The third and final scenario is when the patient is immediately **referred** to a dermatologist for further evaluation and treatment. This occurs in conditions where the systemic impact of the disease transcends the boundaries of the oral cavity and results in significant morbidity and even mortality.

When oral treatment is provided, periodic evaluation is needed to monitor the response of the patient to the selected therapy.

DISEASES CLINICALLY PRESENTING AS DESQUAMATIVE GINGIVITIS

A-Lichen Planus

Lichen planus is an immunologically mediated mucocutaneous disorder where host T lymphocytes play a central role .

Numerous epidemiologic studies have shown that oral lichen planus presents in 0.1% to 4% of the population."The majority of patients with oral lichen planus are middle-aged and older females with a 2:1 ratio of females to males.

Oral Lesions.

Although there are several clinical forms of oral lichen planus (reticular, patch, atrophic, erosive and bullous), the most common are the reticular and erosive subtypes.

The typical reticular lesions are asymptomatic, bilateral, and consist of interlacing white lines on the posterior region of the buccal mucosa.

The lateral border and dorsum of the tongue, hard palate, alveolar ridge, and gingiva may also be affected.

In addition, it is not unusual for the reticular lesions to have an erythematous background, a feature that is associated with the coexistence of candidiasis.

The erosive subtype of lichen planus is often associated with pain and clinically manifests as atrophic, erythematous areas.

Fine white radiating striations are observed bordering the atrophic zones. These areas may be sensitive to heat, acid, and spicy foods .

Oral lichen planus lesions follow a chronic course and have alternating, unpredictable periods of quiescence and flares.

Gingival Lesions.

Up to 10% of patients with oral lichen planus have lesions restricted to the gingival tissue that may occur as one or more types of four distinctive patterns:

1. Keratotic lesions. These raised white lesions may present as groups of individual papules, linear or reticulate lesions, or plaque like configurations

2. *Erosive or ulcerative lesions.* These extensive erythematous areas with a patchy distribution may present as focal or diffuse hemorrhagic areas. These lesions are exacerbated by slight trauma (e.g., tooth brushing)

3. Vesicular or bullous lesions. These raised, fluid-filled lesions are uncommon and short lived on the gingiva, quickly rupturing and leaving an ulceration.

4. Atrophic lesions. Atrophy of the gingival tissues with ensuing epithelial thinning results in erythema confined to the gingiva.

Therapy.

The keratotic lesions of oral lichen planus are asymptomatic and do not require treatment once the microscopic diagnosis is established. However, follow-up of the patient every 6 to 12 months is warranted to monitor suspicious clinical changes and the emergence of an erosive component.

In contrast, the erosive, bullous, or ulcerative lesions of oral lichen planus are treated with high-potency topical steroid such as 0.05% fluocinonide ointment (Lidex, three times daily).

B-Pemphigoid

The term *pemphigoid* applies to a number of cutaneous, immune-mediated, subepithelial bullous diseases that are characterized by a separation of the basement membrane zone .Among these conditions, **bullous pemphigoid** which is nonscarring and mainly affects the skin, and **mucous membrane pemphigoid(cicatricial pemphigoid)**affects the oral cavity, conjunctiva and the mucosa of nose, vagina ,rectum ,esophagus and urethra , produce scarring.

Immunologically both types associated with IgG 3 and C3 immune deposites along the basement membrane detected by immunofluoresence tests.

ORAL LESIONS.

Oral lesions have been reported to occur secondarily in up to 40% of the cases.

There is an erosive or desquamative gingivitis presentation and occasional vesicular or bullous lesions in the attached gingiva,but may occur in other sites of oral cavity.

Bullae tend to have thick roof and rupture in 2 to 3 days leaving irregular area of ulceration in which healing may take up to 3 weeks.

THERAPY.

Because its etiologic factors are unknown, treatment of bullous pemphigoid is designed to control its signs and symptoms.

The primary treatment is a moderate dose of systemic prednisone. Steroid-sparing strategies (prednisone plus other immunomodulator drugs) are used when high doses of steroids are needed or the steroid alone fails to control the disease .

For localized lesions of bullous pemphigoid, high potency topical steroids or tetracycline with or without nicotinamide can be effective.

C-Pemphigus Vulgaris

The pemphigus diseases are a group of autoimmune bullous disorders that produce cutaneous and/or mucous membranes blisters .

Pemphigus vulgaris is the most common of the pemphigus diseases .It is a potentially lethal chronic.

A predilection in women usually after the fourth decade of life, but has also been reported in children and even newborn.

In approximately *60%* of patients with pemphigus vulgaris, the oral lesions are the first sign of the disease and may herald the dermatologic involvement by a year or more.

immunofluorescence tests revealed autoantibodies to the pemphigus vulgaris antigen which is a cell surface glycoprotein present in keratinocytes leading to damaged cell to cell adhesion structures and intraepithelium bullae.

Oral Lesions.

Oral lesions of pemphigus range from small vesicles to large bullae. When the bullae rupture, they leave extensive areas of ulceration .

Virtually, any region of the oral cavity can be involved, but multiple lesions often develop at sites of irritation or trauma.

The soft palate is more often involved (80%), followed by the buccal mucosa *(46%),* ventral aspect or dorsum of tongue *(20%),* and lower labial mucosa (10%).

Oral lesions of pemphigus vulgaris are confined less often to the gingival tissues.

In these cases a clinical diagnosis of erosive gingivitis or desquamative gingivitis is seen as the sole manifestation of oral pemphigus.

Therapy.

The main therapy for pemphigus vulgaris is systemic corticosteroid therapy with or without the addition of other immunosuppressive agents.

Initially, when only steroids were employed, high initial and maintenance doses of steroids were necessary to control the disease

D-Chronic Ulcerative Stomatitis

Clinically, this condition presents with chronic oral ulcerations and has a predilection for women in their fourth decade of life.

The erosions and ulcerations present predominantly in the oral cavity, with only few cases exhibiting cutaneous lesions.

Immunofluorescence tests of normal and perilesional tissues revealed stratified epithelium specific antinuclear antibodies (SES-ANA). nuclear deposites of IgG mainly in the basal cell layer of normal epithelium.

Oral Lesions.

Painful, solitary small blisters and erosions with surrounding erythema are present mainly on the gingiva and the lateral border of the tongue.

Because of the magnitude and clinic features of the gingival lesions, a diagnosis of desquamative gingivitis is considered .

The hard palate may also present similar lesions.

Treatment.

For mild cases, topical steroids and topical tetracycline may produce clinical improvement; however, recurrences are common.

For severe cases, a high dose of systemic corticosteroids is needed to achieve remission.

Unfortunately, reduction of the corticosteroids doses results in relapse of the lesions.

Hydroxychloroquinine sulfate at a dosage of 200 to 400 mg per day seems to be the treatment of choice to produce complete, long-lasting remission

E-Erythema Multiforme

Erythema multiforme is an acute bullous and/or macular inflammatory mucocutaneous disease where a series of immunopathologic mechanisms occur.The genesis of ulcerative lesions affecting the skin and the mucousa is believed to reside in the development of immune complex vasculitis followed by complement fixation leading to leukocytoclastic destruction of vascular wall and small vessels occlusion ,the culmination of these events produces ischemic necrosis of the eoithelium and underlying connective tissue.

target or iris lesion with central clearing is the hallmark of erythema multiforme.

Erythema multiform minor: mild condition lasts for 4 weeks ,exhibits a moderate cutaneous and mucosal involvment.

Steven -Johnson syndrome: may lasts for a month or longer and involves the skin, conjunctiva , oral mucosa and genitalia.

Oral Lesions.

The oral lesions consist of multiple, large, shallow, painful ulcers with an erythematous border.

They may affect the entire oral mucosa in approximately 20% of erythema multiforme patients.

The lesions are so painful that chewing and swallowing are impaired .

The buccal mucosa and tongue are the most commonly affected sites, followed by the labial mucosa. Less commonly affected are the floor of the mouth, hard and soft palate, and the gingiva.

There are rare instances in which erythema multiforme may be confined exclusively to the gingival tissues, resulting in a clinical diagnosis of desquamative gingivitis.

The presence of hemorrhagic crusting of the lips is helpful in arriving at a clinical diagnosis of erythema multiforme.

Treatment.

There is no specific treatment for erythema multiforme. Some cases may even resolve spontaneously, and erythematous lesions may require no treatment. In contrast, patients exhibiting bullous or ulcerative lesions require intervention.

For mild symptoms, systemic and local antihistamines coupled to topical anesthetics and debridement of lesions with an oxygenating agent is adequate.

In patients with severe symptoms, corticosteroids are considered the drug of choice although, its use is controversial and not completely accepted.