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Desquamative Gingivitis

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Intended Audience: Dentists, Dental Hygienists, Dental Students, Dental Hygiene Students
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This course is designed to acquaint the clinician with the possible etiologies, treatments and recommendations for the patient who exhibits the characteristics of desquamative gingivitis (DG). Patient management is discussed in four separate phases: diagnosis, control, consolidation and maintenance. Relevant histological and direct immunofluorescence examinations are discussed and suggested biopsy techniques for optimal results are explained.

Conflict of Interest Disclosure Statement

- The authors report no conflicts of interest associated with this course.

Overview

Classifying mucosal disease states is difficult for the clinician since many mucosal diseases have similar oral appearances. The term "desquamative gingivitis" (DG) is often used as a descriptive term, because the etiology of the inflammation may originate from multiple sources and, ultimately, it may prove to be a clinical feature of one of the mucocutaneous diseases.

This course is designed to acquaint the clinician with the possible etiologies, treatments and recommendations for the patient who exhibits the characteristics of DG. Patient management is discussed in four separate phases: diagnosis, control, consolidation and maintenance. Relevant histological and direct immunofluorescence examinations are discussed and suggested biopsy techniques for optimal results are explained.

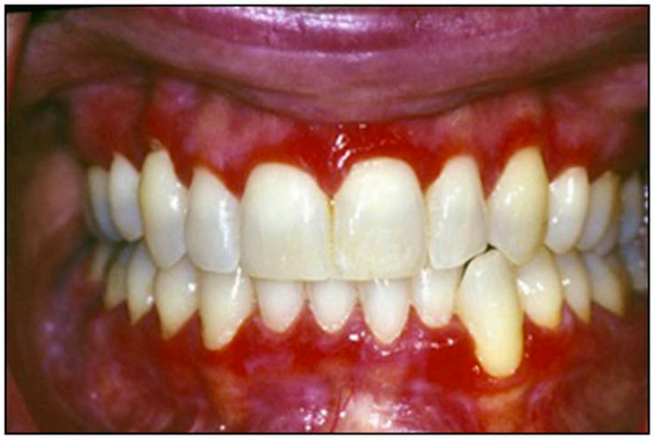


Figure 1. Desquamative Gingivitis.

Learning Objectives

Upon completion of this course, the dental professional should be able to:

- Define the clinical term desquamative gingivitis.
- Identify four oral mucosal diseases or disorders that are most commonly associated with desquamative gingivitis.
- Differentiate between plaque-related gingivitis and chronic gingival inflammation associated with mucogingival desquamation.
- Be familiar with diagnostic interventions necessary to confirm the etiology of conditions associated with desquamative gingivitis.
- Be familiar with treatment protocols useful in patient management of the diseases and disorders most often associated with desquamative gingivitis.

Course Contents

Glossary

desquamative gingivitis (DG) – A clinical term used to describe gingival tissues that demonstrate potentially painful erythema, hemorrhage, sloughing, erosion, and ulceration.

direct immunofluorescence – Fluorescence microscopy technique designed to identify specific autoantibodies in the biopsied tissue.

exfoliative cytology – The microscopic examination of cells that have been shed from a lesion or have been recovered from a tissue for the diagnosis of disease. Also called *cytopathology*.

indirect immunofluorescence – Fluorescence microscopy designed to detect the presence of autoantibodies in the patient's serum.

Nikolsky's sign – Epithelial desquamation that is induced by the application of a firm sliding or rubbing force. This phenomenon is commonly observed in several mucocutaneous disease states.

patch test – A type of skin test for hypersensitivity in which filter paper or gauze saturated with the substance in question is applied to the skin, often on the back; a positive reaction is characterized by reddening or swelling at the site.

reflective confocal microscopy – An *in vivo*, non-invasive, autofluorescence microscope that is able to penetrate body tissues and to show the cytoplasm of epithelial cells, connective tissue cells, blood vessels, etc. Studies are underway to identify specific confocal features of lesions that are consistent with those found in conventional histologic microscopy, thereby enabling the diagnosis of a lesion without requiring a biopsy.

vesiculobullous disease – A type of mucocutaneous disease that is characterized by vesicles and bullae (i.e., blisters). Both vesicles and bullae are fluid-filled lesions, and they are distinguished by size (vesicles being less than 5-10 mm and bulla being larger than 5-10 mm).

Introduction

Desquamative gingivitis (DG) is a clinical term used to describe gingival tissues that demonstrate potentially painful gingival erythema, hemorrhage, sloughing, erosion, and ulceration (Figure 1 and Box 1). Lesions may be generalized or localized and may extend into the alveolar mucosa. Often similar lesions are found elsewhere in the oral cavity. DG is most frequently caused by mucocutaneous diseases with the most common being oral lichen planus *mucous membrane pemphigoid* and *pemphigus vulgaris*.[30,49,56,65,72,77,90] Other potential causes include: *lupus erythematosus, graft versus host disease, erythema multiforme, epidermolysis bullosa, epidermolysis bullosa acquisita, chronic ulcerative stomatitis, lichen planus pemphigoides, plasmacytosis, plasma cell gingivitis, orofacial granulomatosis, foreign body granulomas,* and *linear IgA disease*.[49,59,75,92,97] Hypersensitivity to dental materials, dental hygiene products or food flavorings and preservatives may mimic DG, while several systemic disorders including *Crohn's disease, psoriasis, sarcoidosis,* and *adverse drug reactions* may possess some but usually not all of the clinical features of DG.[30] Unpublished data from the Stomatology Center at Texas A&M University-Baylor College of Dentistry (TAMUBCD) indicates over 90% of DG treated at that center were the result of one of four conditions: oral lichen planus, mucous

membrane pemphigoid, pemphigus vulgaris or hypersensitivity reactions to dental hygiene products, food flavorings or preservatives.[26]

The clinical features of what is now consistent with DG were described in the dental literature as early as 1856, but it is believed to have been first discussed in the English dental literature by Tomes and Tomes in 1894. In 1932 Prinz coined the term *desquamative gingivitis*,[66,94]

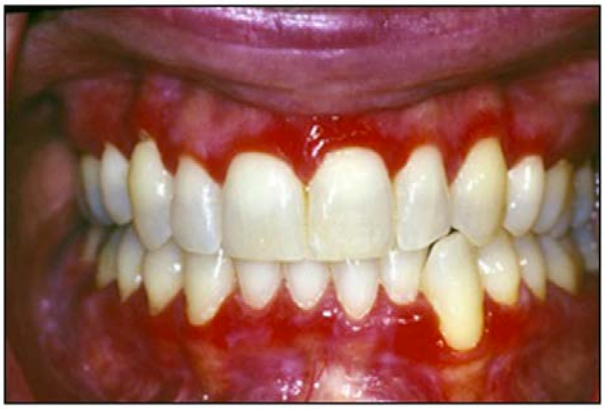


Figure 1. Desquamative Gingivitis.

and the consensus view at the time was that it represented a specific disease, possibly related to hormonal deficiencies in older women and occasionally men.[108] This thinking may have been triggered by the observation that DG was far more common in females than males. However, in 1964 Glickman and Smulow suggested DG may have multiple causes.[38] It was not until the advent of immunofluorescence diagnostic techniques that it became obvious DG was, in fact, a clinical manifestation of a variety of diseases and disorders capable of affecting either gender.[13]

DG is often found to be closely associated with epithelial desquamation after application of a sliding or rubbing force on normal-appearing gingiva (Nikolsky's sign – Figure 2). This phenomenon is very common in several mucocutaneous disorders.[33,75,77,94,108]

The ability to readily determine the correct etiology of DG may sometimes be difficult due the extremely friable nature of the affected tissues. This can lead to separation or loss of the epithelial layer of biopsied tissue, making it virtually impossible

- A clinical manifestation of several diseases and disorders featuring gingival *erythema, sloughing* of the gingival epithelial tissues and potentially painful *erosive* gingival lesions.
- Usually caused by mucocutaneous diseases with the most common being *lichen planus, mucous membrane pemphigoid* and *pemphigus vulgaris.* Other causes include *hypersensitivity reactions* to various oral hygiene products and dental materials.
- Determination of etiology usually requires histopathological examination and direct immunofluorescence testing.

Box 1. Desquamative Gingivitis.

- Gingival erythema not resulting from plaque
- Desquamation and erosion of gingival epithelium
- Blister formation
- Other intraoral and/or extraoral lesions
- Possible positive Nikolsky's sign (epithelial desquamation after application of a shearing force on normal-appearing gingival).

Box 2. Characteristic Features of Desquamative Gingivitis.

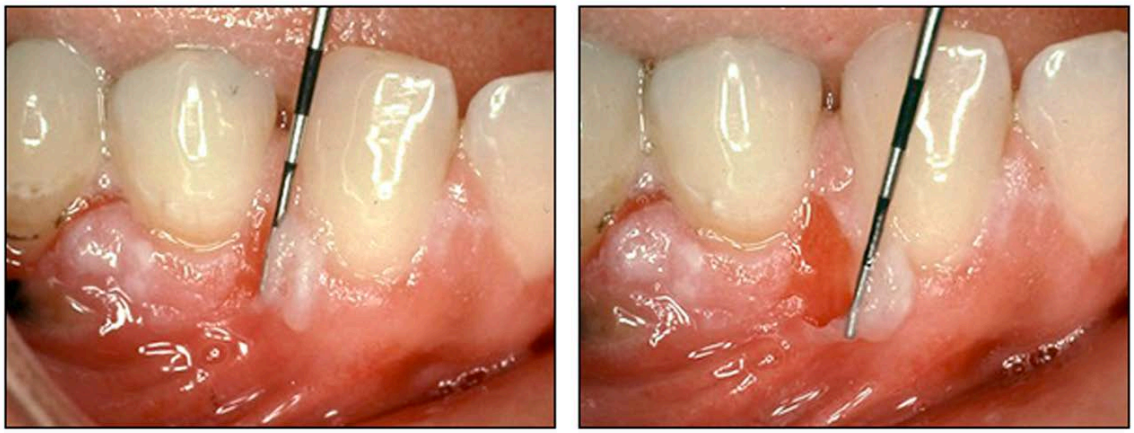


Figure 2. Nikolsky's sign.
The epithelium is dislodged by gentle pressure.

to determine the correct histopathologic and/or immunofluorescence diagnosis.

Phases of Diagnoses and Therapy

Diagnosis and management can be divided into four phases as described by Bystryn in 1988 and Sciubba in 1994.[15,84] These are:

- **Diagnostic Phase** – essential to successful management.
- **Control Phase** – therapy designed to reduce or eliminate signs and symptoms of disease.
- **Consolidation Phase** – reduction or elimination of therapy as the condition improves.
- **Maintenance Phase** – long-term control of the disease process maintaining the patient in a state of comfort and usually freedom from the disease.

Diagnostic Phase

Box 3 identifies the steps usually required to establish an accurate diagnosis of oral diseases and disorders.

Asking questions regarding the patient's past medical and dental history of the onset, duration, previous treatment and response to previous treatment can often be of value in diagnosis.

- *Do the lesions come and go?*
- *How long have you had the signs and symptoms?*
- *Are lesions becoming progressively more painful?*
- *Is there a family history of these types of lesions?*
- *What medications do you take? Why? And for how long?*
- *Does anything seem to trigger worsening of the lesions?*

- Past History
- Clinical appearance
- Biopsy
  - Histology
  - Direct immunofluorescence
- Indirect immunofluorescence
- *Candida* culture/smear
- Ancillary tools
  - Exfoliative cytology
  - "Brush" biopsy
  - Fluorescence lighting

Box 3. iagnostic Phase.[4]

- *What treatment has been rendered at this point?*
- *Was the treatment effective?*

As the term mucocutaneous implies, the patient should be carefully examined for both intraoral and extraoral lesions. For example, mucous membrane pemphigoid and pemphigus vulgaris may exhibit oral, cutaneous, genital and ocular lesions (Figure 3), while oral lichen planus may be accompanied by cutaneous lesions 12-14% of the time.[26]

Genital lesions are common in oral lichen planus, especially among women (the so-called vulvovaginal-gingival lichen planus syndrome – Figure 4).[24,26,61,62] Sites of involvement of mucocutaneous diseases may include the pharynx, esophagus, larynx, eye, anus and occasionally the internal organs.[30]

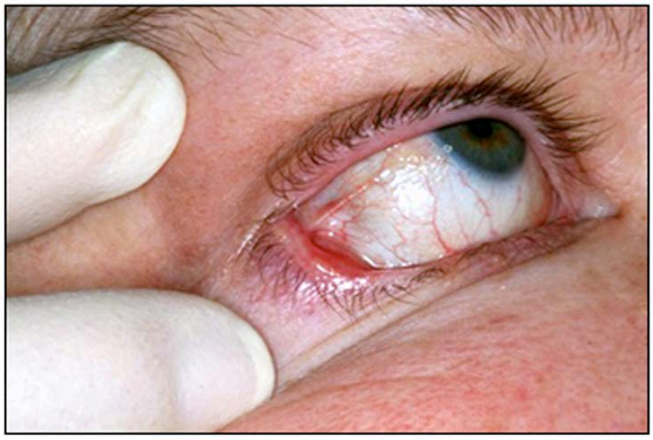


Figure 3. Early ocular lesion of mucous membrane pemphigoid.
Note the beginning scar attaching the eyelid to the cornea.

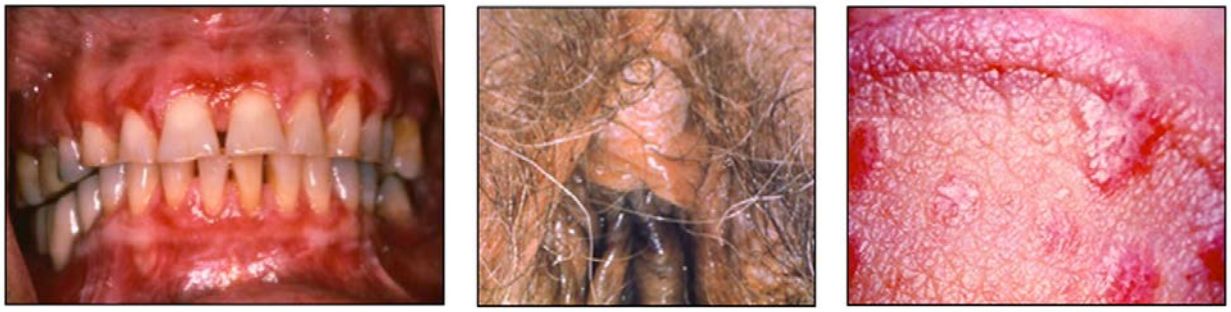


Figure 4. The vulvovaginal-gingival lichen planus syndrome.

On occasion the gingiva and/or other oral sites present with the classical appearance of specific mucocutaneous diseases that may support the tentative diagnosis. For example, the presence of reticular lesions in addition to the erosions associated with DG may strongly suggest a diagnosis of oral lichen planus. However, some other diseases may present with similar clinical appearance (*lupus erythematosus, chronic ulcerative stomatitis, graft versus host disease, lichen planus pemphigoides*) so further diagnostic steps are often indicated.[30,34,49,59,75,90,95,103] Histopathology examination of biopsied tissue remains the gold standard for accurate diagnosis of most oral diseases and disorders although direct (DIF) and indirect (IIF) immunofluorescence is often considered diagnostic for classic autoimmune diseases such as mucous membrane pemphigoid and pemphigus vulgaris.[13,30,77,95] Conversely, negative immunofluorescence findings should be anticipated in biopsies of hypersensitivity reactions, while DIF is only considered to be supportive but not diagnostic of oral lichen planus.[30,75,82]

Biopsy sites should be selected that appear to have an intact epithelial surface (Boxes 4 and 5). It is usually of little value to select an erosive lesion for biopsy since the epithelium is often missing.

Consequently, perilesional sites or even normal appearing tissue sites may be more appropriate for biopsy, especially if DIF evaluation is to be used. Autoimmune diseases such as MMP and PPV will often present with positive DIF findings even in normal appearing tissue.[56,93] Conversely, there is no benefit in obtaining a biopsy of normal tissue for histopathological evaluation in suspected non-autoimmune disorders. Figure 5 reveals an individual with DG for whom alternative biopsy sites are preferred.

Of all oral soft tissues, the gingiva is likely to be the site that is most often traumatized by normal functions such as chewing, toothbrushing,

- Choose an area of intact epithelium
- Include perilesional and possibly distant tissue
- Consider selecting normal appearing tissue for some DIF testing, when necessary
- *When possible, avoid gingival biopsies*

Box 4. Biopsy Site Selection.

- Formaldehyde for routine histological evaluation
- Ambient temperature transport media (Michel's solution) for DIF
- Obtain transport media from pathology facility and/or immunology lab, usually without charge

Box 5. Biopsy Shipment.

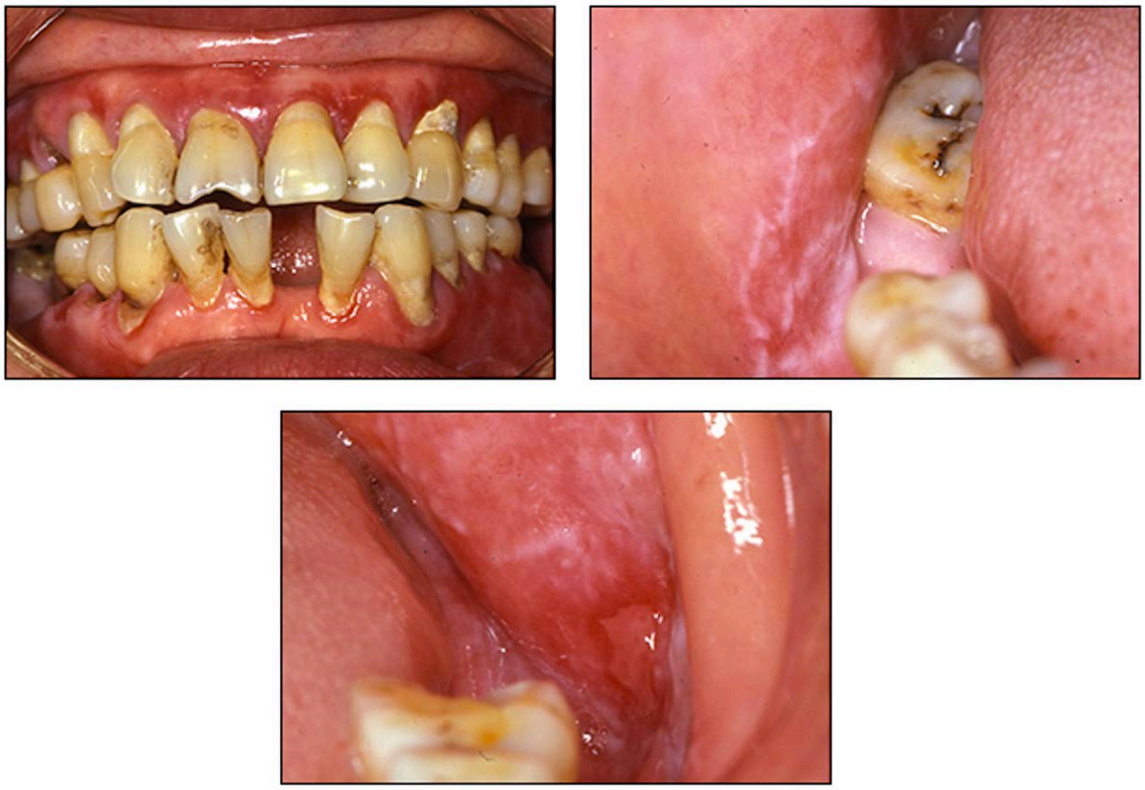


Figure 5. Biopsy site selection.
Due to risk of gingival desquamation, an alternate biopsy site should be selected when possible. Suitable alternative sites are present bilaterally in this patient.

flossing, use of toothpicks, gingival massage, or ingestion of hot or caustic foods or liquids. This is likely to cause the epithelium to slough when a mucocutaneous condition is present, thereby interfering with biopsy diagnosis. This sloughing is also especially common when a gingival biopsy is performed. Consequently, lesions in other oral sites should probably be selected for biopsy.[30,43,74] Data from the TAMUBCD Stomatology Center indicates 18.9% of *oral lichen planus* patients and 16.3% of *pemphigus vulgaris* patients had lesions confined only to the gingiva. This indicates accessible extragingival oral sites are usually available.[74,77] In contrast, over 67% of patients with oral *mucous membrane pemphigoid* had lesions that were confined exclusively to the gingiva, indicating the type of mucocutaneous disease present can markedly influence the necessity for a gingival biopsy.[74] Nonetheless, there is often no option to performing a gingival biopsy. In the past this factor has presented a major impediment to confirmation of the histopathological and immunofluorecence diagnosis.[80] However, some newer developments in biopsy techniques may aid in retaining intact epithelium in gingival biopsies.[31] In addition, studies are currently being conducted to evaluate the effectiveness of reflectance confocal microscopy in diagnosis of mucocutaneous diseases without requiring a biopsy.[2]

Special Gingival Biopsy Techniques

If a gingival biopsy is required, one should be careful to take the following steps into consideration (Figure 5):

1. The specimen should be taken apical to the free gingival margin, since most marginal areas are inflamed and the inflammatory process may mask the histopathologic features necessary to establishing the correct diagnosis.
2. Local anesthesia is achieved being careful to avoid direct injection into the biopsy site to prevent hemorrhage, cellular distortion and epithelial disruption in the biopsy area.
3. Often it is necessary to submit tissue samples for both histologic and direct immunofluorescence evaluation since the pathologic process may represent an autoimmune condition such as *mucous membrane pemphigoid* or *pemphigus vulgaris*.[54,56,75]
4. *Oral lichen planus* has autoimmune properties found within lesions but lacks the systemic immunologic features confirming autoimmunity. Nevertheless, immunofluorescence studies of *oral lichen planus* may be indicated because a linear pattern of anti-fibrinogen at the basement membrane zone and the presence of immunoglobulins in cytoid bodies are supportive of the diagnosis.[10,14,23,75]
5. Other mucocutaneous diseases such as *chronic ulcerative stomatitis, lichen planus pemphigoides, graft versus host disease,* and *discoid lupus erythematosus* may mimic the clinical and histological features of oral lichen planus so DIF results may be of great value in supporting the diagnosis of OLP.[90]
6. Traditional biopsy protocol calls for incorporating lesional and perilesional tissue in the biopsy procedure. However, in DG, lesional sites often have lost their surface epithelium and the perilesional area immediately adjacent to the lesion is extremely friable. This coupled with the trauma of the biopsy itself may result in epithelial sloughing and diagnostic failure. Sano, et al. defined perilesional tissue as the area within 1 cm of the lesion, while sites beyond 1 cm of the lesions are considered distant.[79] They found no significant difference in DIF findings between the perilesional (66.1%) and distant sites (64.7%). They also suggested separate H&E and DIF biopsies were preferable to the traditional practice of splitting a single biopsy for the two analyses.

Punch Biopsy

Do not rotate the cutting edge on a gingival biopsy.

Punch biopsy techniques have been suggested by Eisen and others as a useful technique for gingival biopsies, possibly because there is only a minimal lateral shearing force applied when using this method.[24,50,81,85,97] However, the described technique recommends a rotational force during the instrument insertion which may inadvertently exert a lateral shearing force. Using the punch technique for DG biopsies is probably suitable since it may be possible to avoid the rotational movement due to the thinnest of the gingiva and the ease of penetration to bone. To date, however, there are no studies that confirm the punch technique is superior to conventional scalpel techniques on gingiva.

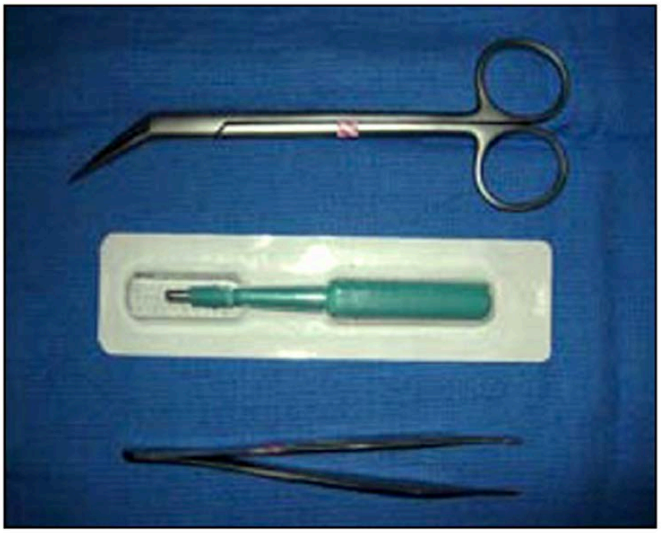


Figure 6. The Punch Biopsy.
Usually the selected instrument is 4-6 mm in diameter.

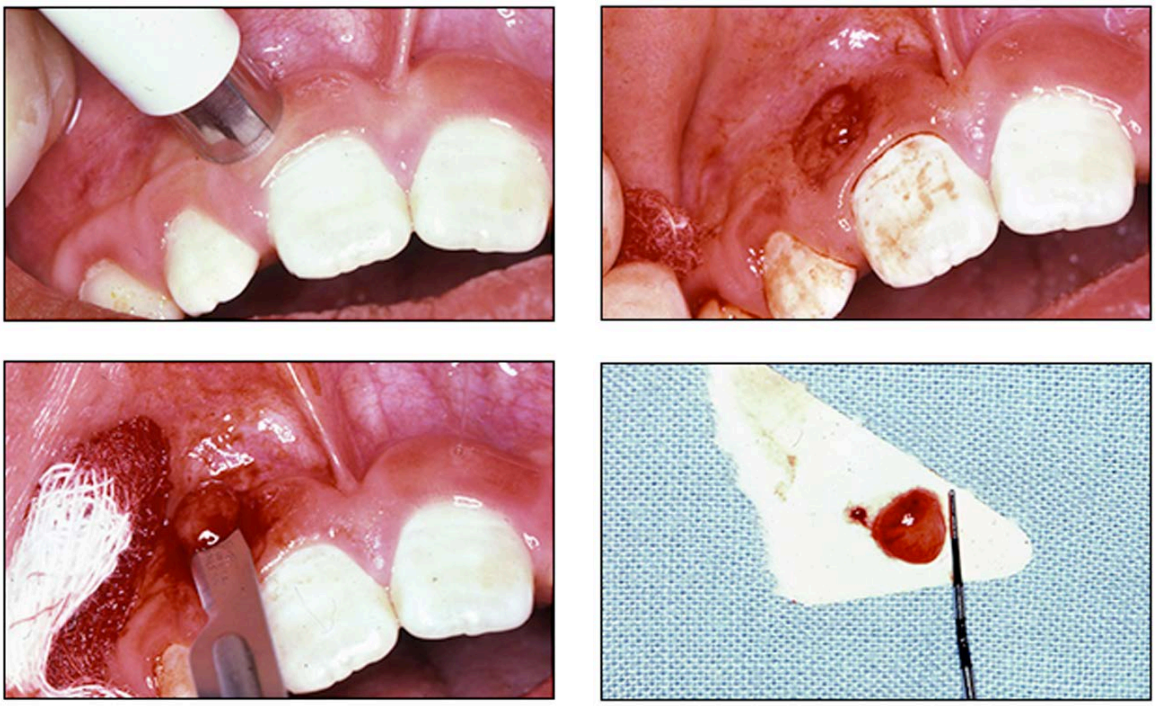


Figure 7. Steps in a gingival punch biopsy.
The instrument is placed and gently rotated until bone is reached. The separated tissue must then be undermined for removal.

Stab-and-roll Biopsy

In 2014, Endo, et al. reported successful retention of intact epithelium in 51 of 52 lesional, perilesional or distant DG biopsy sites (98.1%), and the pathologic diagnosis was confirmed in all 52 tissue samples.[31] They termed their method the *stab-and-roll technique* which is designed to avoid lateral shearing forces likely to disrupt epithelial integrity by uniformly directing the cutting forces internally to bone. In the *stab* motion, the tip of a #15 scalpel blade is gently inserted to bone while in the *roll* motion the blade is rolled from the tip along its full cutting edge, thereby applying only an internally directed force.

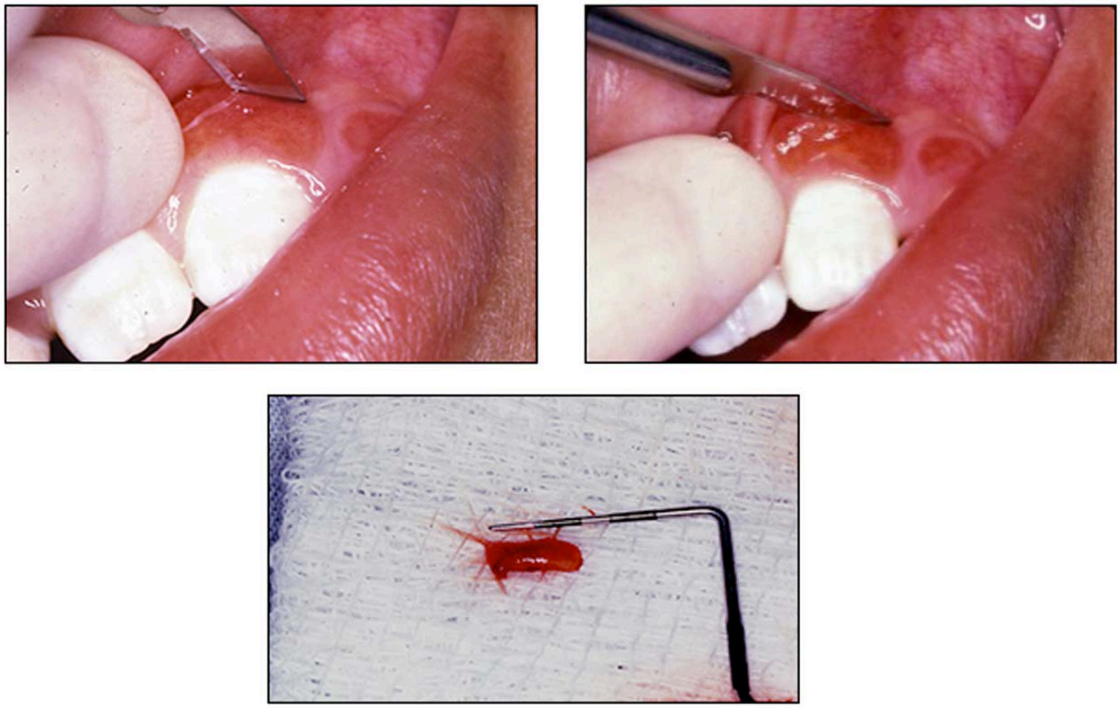


Figure 8. The *stab-and-roll* biopsy technique designed to avoid lateral shearing forces.

If more tissue is required, the tip is again inserted into the incision line and the rolling stroke is repeated. The soft tissue is gently undermined with a scalpel and removed with a non-serrated small tissue forceps. No controls were used in this study but comparison between the stab-and-roll successful results (98.1%) versus other reported techniques (58.8-60.0%) suggests a significant improvement.[85,87]

Whatever biopsy technique was chosen, it is extremely important the specimen be appropriately transported to the pathologic or immunofluorescence laboratory. Tissue for histopathology examination is transported in formalin. However, tissue placed in formalin cannot be evaluated by DIF. Instead the DIF specimen can be quick-frozen in liquid nitrogen and immediately transported in that fashion, but most often it is transported in a special transport medium (Michel's transport medium).[14,7] Most laboratories that perform immunofluorescence will provide the transport medium free of charge.

Evaluation for *Candida*

If possible, salivary cultures for candidiasis should be performed at baseline in patients with DG in order to determine whether or not *candida* is present in the oral cavity. If it is present, and if the diagnosis affirms a condition requiring the use of topical or systemic steroids or other immune suppressants, prophylactic antifungal medication may be indicated at the onset of treatment. Culturing is not available in many dental practices so the clinician should consider empirically prescribing an antifungal agent if immunosuppressants are to be used and be especially vigilant for evidence of candidiasis at all follow-up appointments.[47]

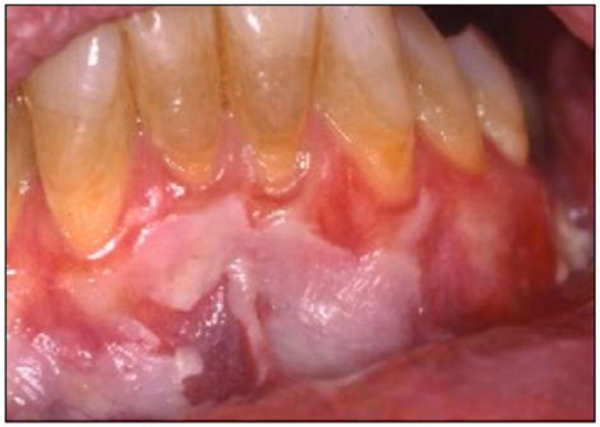


Figure 9. Atypical candidiasis, secondary to use of topical corticosteroids.

Ancillary Techniques or Devices

On occasion the use of ancillary techniques or tools may be appropriate. *Exfoliative cytology* coupled with DIF may provide a rapid, painless method for identifying PV although it would ***not*** be of major benefit in identifying other mucocutaneous diseases causing DG.[5,34,103] A cytological biopsy commonly referred to as a "*brush biopsy*" is the oral equivalent of the cervical Papanicolaou (Pap) test which normally identifies the presence of abnormal cells suggestive of premalignant or malignant changes. The brush biopsy is usually used in the oral cavity as a minimally invasive method for determining whether or not atypical cells are present and whether a biopsy is indicted. Consequently, it is rarely used in diagnosis of DG since abnormal but non-malignant cellular changes are already suspected. In most instances the need for a conventional biopsy is self-evident in patients with DG. On occasion, however, a brush biopsy may be indicated, i.e., to differentiate between white lesions caused by hyperkeratosis and those associated with the plaque form of oral lichen planus. The brush biopsy also may occasionally be appropriate for patients who refuse a traditional biopsy or for severely medically compromised individuals who may not be able to tolerate a conventional biopsy.[8]

The value of using *autofluorescence devices* (Velscope, Identifi 3000, Oral ID) as a component of general dental/oral examinations has not yet been determined. Although there is some evidence that the devices may be helpful if there is suspicion that dysplastic or malignant changes have occurred, the devices may be of little value in DG diagnosis because inflammatory lesions often result in false positive findings indistinguishable from premalignant or malignant changes.[27,109]

Therapy Phases

Control Phase of Therapy

In the past, patients with DG were often told there was no known treatment for their condition

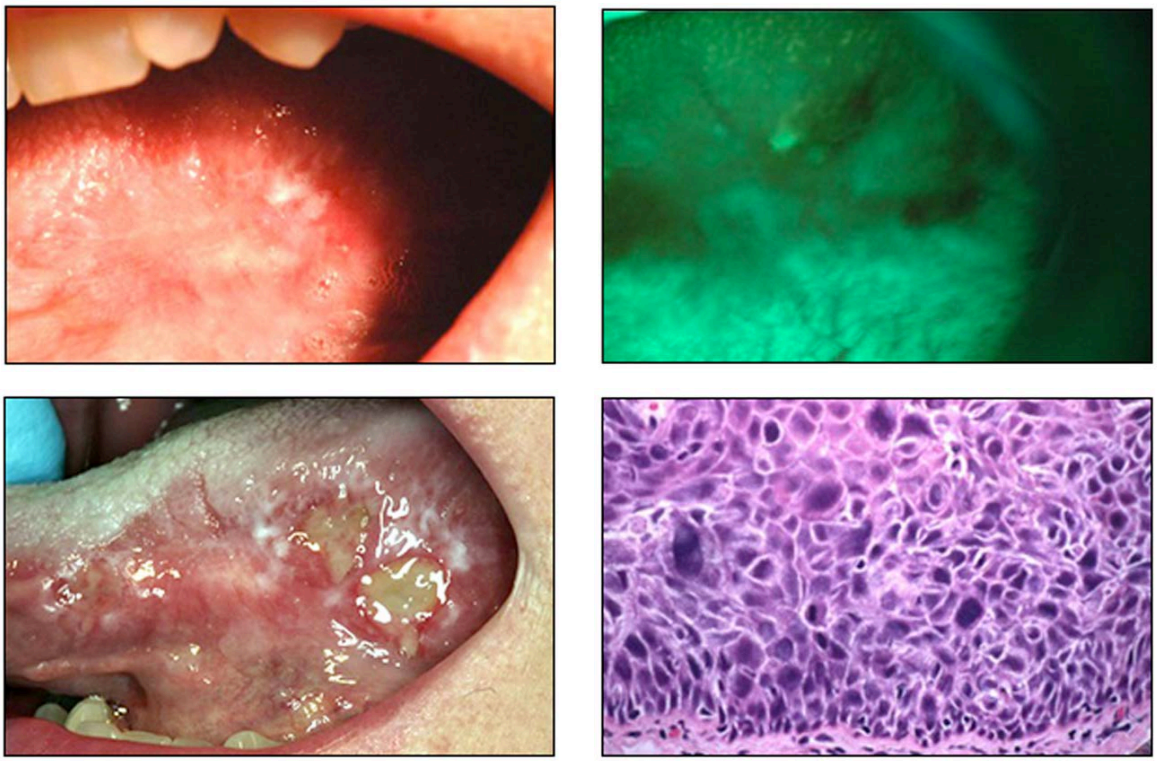


Figure 10. Benefits of fluorescence devices in a patient with a previous history of tongue cancer and recurrent lesions. Biopsies of dark areas showed severe dysplasia in all three sites.

and the patient would just have to learn to live with it. At this time, however, that kind of thinking is totally unacceptable and in most instances the patient's disease or disorder can be markedly improved and on some occasions, clinical features are eliminated.[48] In the foreseeable future, it may be possible to manage mucocutaneous diseases even more effectively with stem cell therapy.[91] Meanwhile, current treatment modalities rarely fail to offer patient relief if not complete remission. Following successful diagnosis, the control phase is directed toward applying intense therapy to suppress the disease or disorder in a matter of days or weeks. The clinician must carefully balance the efficacy of treatment methods versus patient safety and patient acceptance. For example, if topical corticosteroids are prescribed the patient may become alarmed over the fact the material is identified as being for external use only. To avoid undue concerns, the patient should be informed that *off label* use of topical corticosteroids in the oral cavity is largely accepted and has been documented by many scientific papers as a suitable alternative to systemic corticosteroid therapy.[17,30,57,64,75]

Topical corticosteroids (Box 6) or other topical anti-inflammatory medicaments (Box 7) often require multiple daily applications so they should be of sufficient potency to avoid patient disenchantment if improvement is minimal.

Patients should also be informed about possible adverse side effects of their proposed therapy so they may be prepared should side effects occur (Box 8). If they do develop side effects, they should be instructed to inform the clinician immediately. As mentioned, the most common side effect of the use of topical immunosuppressants is secondary candidiasis. The presence of this condition may cause the patient to believe they are not successfully responding to therapy since their mouth continues to be uncomfortable. These factors, disenchantment and adverse side effects can usually be avoided by discussions with the patient early in the treatment process; by evaluating the patient every 2-3 weeks; and changing treatment if results are minimal or adverse side effects occur.[74,75]

To date, no study has confirmed an adverse systemic reaction to judicious application of oral topical or intralesional cortcosteroids.

High Potency Topical Corticosterdroids

- 0.25% Desoximetasone – spray, ointment or ***gel***
- 0.20% Fluocinolone – cream or ointment
- 0.05% Fluocinonide – cream, ointment or ***gel***
- 0.50% Triamcinolone Acetonide – cream or ointment
- Monitor quantity used and do not exceed 15 grams within two weeks

Very High Potency Topical Corticosteroids

- Betamethasone dipropionate – 0.05% *gel,* cream, ointment
- Clobetasol 0.05% – *gel,* cream, ointment
- Halobetasol – 0.05% cream, ointment

Intralesional Corticosteroids

- Triamcinolone acetonide – 5 ml vial containing 10 mg/m
- Use 1 ml tuberculin syringe
- Inject 0.1 mg per square cm of lesion
- May repeat if necessary 2-3 times at 3-4 week interval
- ***Do not use on gingiva or hard palate!***

Box 6. Topical and Intralesional Corticosteroids.

- Topical tacrolimus (Protopic) – FDA warning
- Topical pimecrolimus (Elidel) – FDA warning
- Tacrolimus rinse – 1 mg/1000 ml. Rinse 4x daily
- Effective plaque control
- Topical cyclosporine A – expense
- Soft "plumper" mouthguards – prevent cheek, lip and tongue irritation
- Replace faulty restorations or restorations causing a contact lichenoid reaction

Box 7. Other Intraoral Treatments.

Potential Oral Adverse Effects:

- Xerostomia
- *Candidiasis*
- Epithelial atrophy

Potential Systemic Adverse Effects:

- Adrenal suppression
- Hypertension
- Blurred vision
- Elevated blood glucose
- GI hemorrhage

Box 8. Potential Adverse Effects of Topical or Intralesional Corticosteroids.

Control Phase Alternatives

The clinician has several alternatives to choose from in managing DG. *Aggressive* therapy with very high potency topical or short-term systemic corticosteroids may bring about improvement of lesions more rapidly but are more likely to initiate adverse side effects, therefore requiring more frequent recall intervals. *Moderate* therapy with high or very high potency topical steroids (Box 9) and/or intralesional steroid injections is often a suitable alternative in severe lesions of the tongue, soft palate and buccal or labial mucosa. Mild therapy with medium or low potency topical corticosteroids such as triamcinolone mixed into a denture adhesive, another adherent paste or under an adhesive patch, may cause fewer side effects but also may prolong therapy and may not be effective for advanced DG. Topical cream, ointment or gel medicaments may be used with a carrier tray to achieve more prolonged soft tissue contact.[34,51]

Although immunosuppressants applied in carrier trays are often very effective in controlling DG, the clinician should take care to avoid potential adverse effects if trays are used (Box 10).

The authors prefer moderate to aggressive therapy depending on the nature of the desquamative condition. Topical corticosteroids are often available as a cream, ointment or gel. Some evidence suggests the gel form is more retentive in the oral cavity. Typically, a very high or high potency topical steroid such as clobetasol proprionate 0.05% gel is prescribed initially to be used 2-3 times daily until significant clinical improvement has taken place. Concerns are sometimes raised over the possibility of creating immune suppression with the use of intraoral corticosteroids or other immunosuppressant topical agents (Box 11). However, to date no studies have confirmed this possibly because the

quantity of medicament used in the oral cavity is quite small compared to skin applications. However, it is probably wise to closely monitor steroid use. Empirically, we advise patients they should use no more than 15 mg of corticosteroid in a two-week period.

After achieving satisfactory DG improvement or resolution, the patient transitions into the *consolidation phase* of therapy. However, if the patient is only minimally responsive to topical therapy, one might consider the use of an alternative therapeutic approach or referral to a physician for more potent immunosuppressant therapy. Insufficient response may also alert the clinician to the possibility the DG may represent a lichenoid drug reaction that may require close medical/dental coordination to address.

Consolidation Phase of Therapy

After an acceptable level of control of the disease or disorder has been achieved, gradual tapering of medication, frequency of use and/or potency should be initiated while the patient is carefully

- **Aggressive therapy** with very high potency topical or systemic corticosteroids
- **Moderate therapy** with high potency topical corticosteroids combined with intralesional injections when indicated
- **Mild therapy** with medium or low potency topical corticosteroids and carrier trays (triamcinonlone in a paste, denture adhesives, adhesive patches etc.)

Box 9. Control Phase Alternatives.

- Insertion and removal may initiate gingival desquamation – relieve sites as necessary
- Increased risk for secondary candidiasis – monitor
- Risk of increased systemic uptake – discontinue as soon as possible
- Risk of gingival epithelial thinning – discontinue as soon as possible

Box 10. Disadvantages to Carrier Trays.

- Oral application of fluocinonide resulted in no detectable systemic uptake.[106]
- Application of large quantity of topical clobetasol to skin resulted in significant systemic uptake. Effect was dose related.[108]
- Oral use of topical clobetasol 1.5 gm daily for 2 weeks resulted in a small but detectable systemic uptake.[107,109]
- To date, no study has confirmed an adverse systemic immunosuppression related to judicious use of oral topical corticosteroids.

Box 11. Immune Suppression.

monitored at 2-3 week intervals. The goal is to achieve complete remission or minimize symptoms and, if possible, to discontinue therapy. If the lesions are slow to heal, the intensity of therapy may need to be increased or an alternative medication selected.[84]

Maintenance Phase of Therapy

Once the therapeutic goal has been achieved and the patient is comfortable and able to perform effective oral hygiene, the goal is to maintain this state of partial or complete remission on a long-term basis. The mucocutaneous diseases associated with DG are usually of unknown or autoimmune etiology. Consequently, if remission is achieved, it can be anticipated the lesions may recur at some future point. The patient should be alerted to recognize early signs and symptoms of recurrence and to reinitiate therapy for a few days in order to eliminate the symptoms before they become severe.[19,36,40,51,70] In order to confirm long-term stability, an appropriate recall appointment interval should be established and every effort made to sustain dental and periodontal health. The patient should be instructed to be alert to developing lesions in other parts of the body and to confer with their physician should this happen. It is also of importance to bear in mind there is some evidence to indicate several mucocutaneous diseases may be associated with a slightly increased incidence of malignant transformation, making maintenance recall visits to all treating health care providers an important component of their therapy.[14]

The Most Frequent Diseases and Disorders

Oral Lichen Planus (OLP)

Lichen planus is an idiopathic t-cell mediated inflammatory condition. Although its etiology is unknown OLP is sometimes associated with other medical conditions such as hypothyroidism, diabetes mellitus, graft vs host disease, HIV infection, Hepatitis C infection, and psychological stress.[4,54,77,98] Although the issue is controversial there continue to be reports suggesting a slightly increased incidence of oral cancer among individuals with longstanding OLP or lichenoid mucositis.[1,53,54,55,89]

OLP can affect individuals of all ages, although it usually involves individuals ranging in age from 40-70 years with a female to male ratio of at least 2:1.[16,69,99] Data from the TAMUBCD Stomatology Center indicates a female to male ratio of 3:1 among more than 1000 OLP patients with an average age of 56.0 years.[74] In 1968, Andreason described OLP as occurring in 6 different forms: papular, reticular, plaque-like, atrophic, ulcerative and bullous.[6] However, more recently it is common to simplify the terminology designating a keratotic (reticulated) form consisting of papular, reticular or plaque-like lesions, an erythematous (atrophic) form and an erosive form (ulcerative or bullous lesions).[23] Any of these forms of OLP can affect the gingiva. The buccal mucosa is the most frequent clinical site (62%); 55% of patients in the Stomatology Center have gingival lesions, while nearly 20% have lesions confined entirely to the gingiva. DG primarily represents the erythematous and erosive forms of OLP, and it has been reported approximately 30% of OLP patients will have DG.[77]

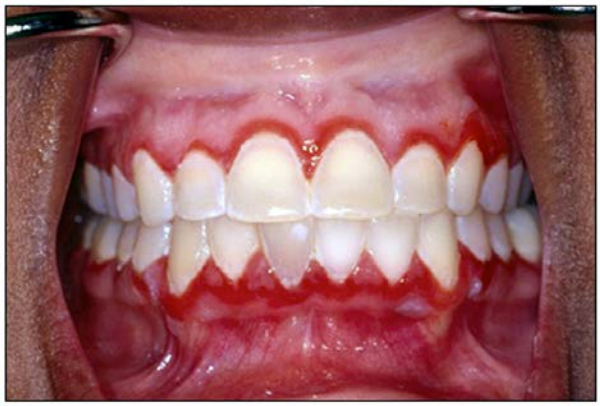


Figure 11. DG in a patient with OLP.

Histopathologic features of OLP include a band-like infiltration of lymphocytes in the sub-basal connective tissue and liquefaction degeneration of the basal cells of the epithelium.[49,75] The basal cell liquefaction may cause the epithelium to detach from the connective tissue, especially if traumatic forces are present as in the gingiva.

Some authorities feel it is not necessary to perform DIF on patients with OLP, although most agree that histopathologic evaluation is desirable. Their rationale is if DIF is found to be necessary it can be accomplished with another biopsy. This is certainly a valid concept, especially since DIF is often relatively expensive, but, in our opinion, DIF is important in DG to assist in ruling out other

diseases. Although the DIF findings are non-specific, they are supportive of diagnosis of OLP if there is a linear deposition of fibrin or fibrinogen along the basement membrane zone and possibly immunoglobulin cytoid bodies in the underlying connective tissue.[39,54,75]

OLP-related DG may be particularly resistant to treatment compared to other mucosal sites. Some authorities recommend the use of a dexamethasone oral rinse but DG may be resistant to that approach. Therefore, treatment is often accomplished in a stair-step format beginning with a high potency topical corticosteroid such as fluocinonide or a very high potency steroid such as clobetasol.[91] The gel form of these products seems to be more retentive on oral mucosa than the cream or ointment form. Other topical treatments may include topical tacrolimus) or topical pimecrolimus).[19] Management may require the fabrication of a soft, flexible, 1-mm thick vacu-form plastic removable appliance to serve as a carrier for the topical medication. The appliance should be constructed so it covers involved gingiva but with spacing to allow pain free insertion and removal.[51] If trays are used, the topical medication should first be manually applied to the gingiva, then the carrier tray is gently placed to cover the gingiva and increase

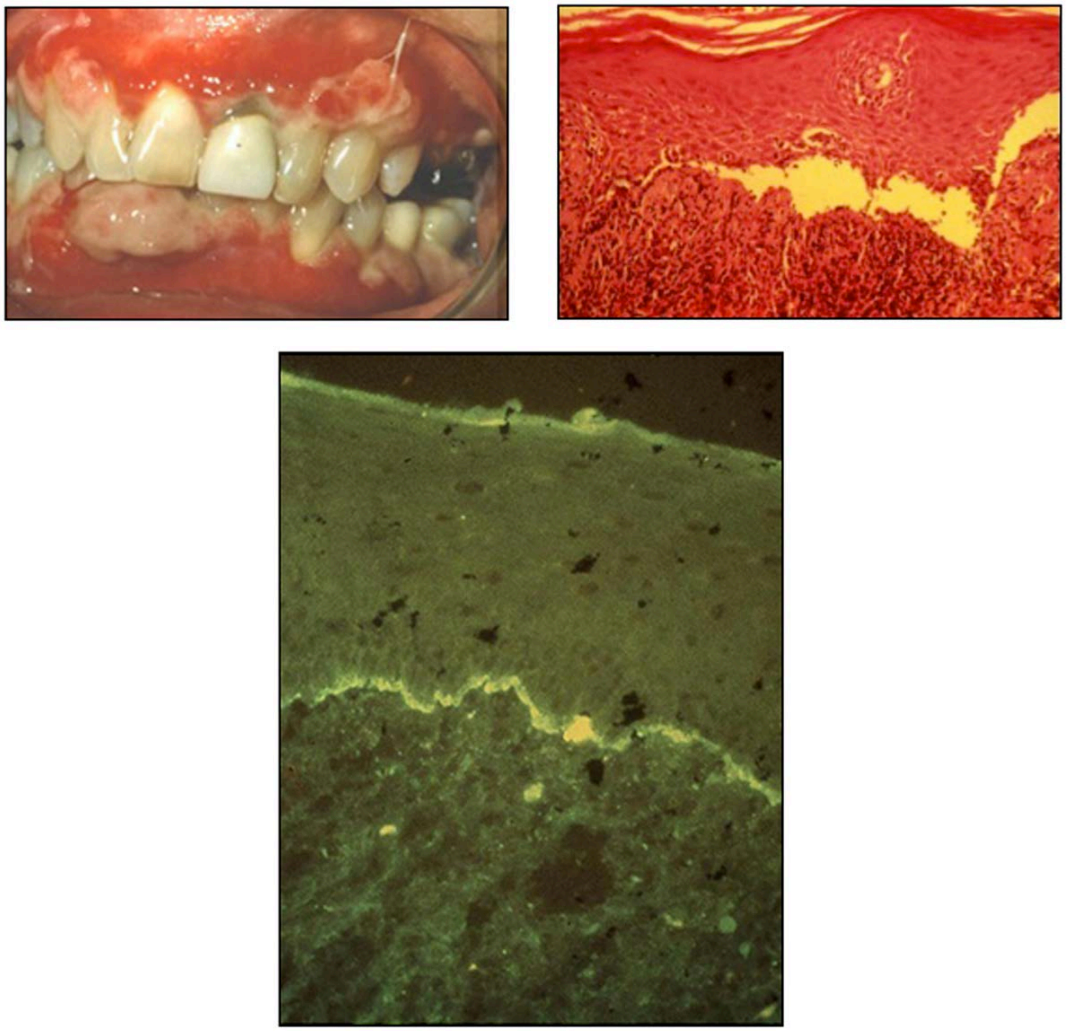


Figure 12. Severe oral lichen planus.
DG with histologic confirmation and DIF support showing a linear pattern of fibrinogen at the basement membrane zone.

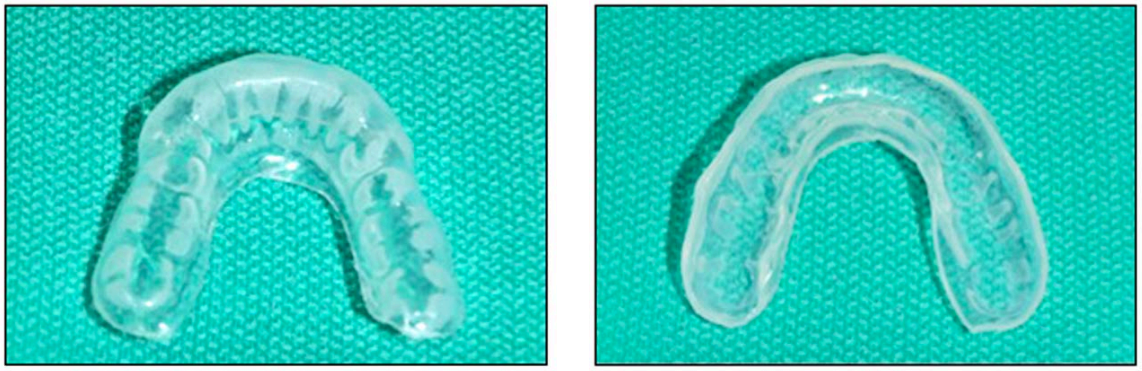


Figure 13. Corticosteroid omnivac carrier tray.

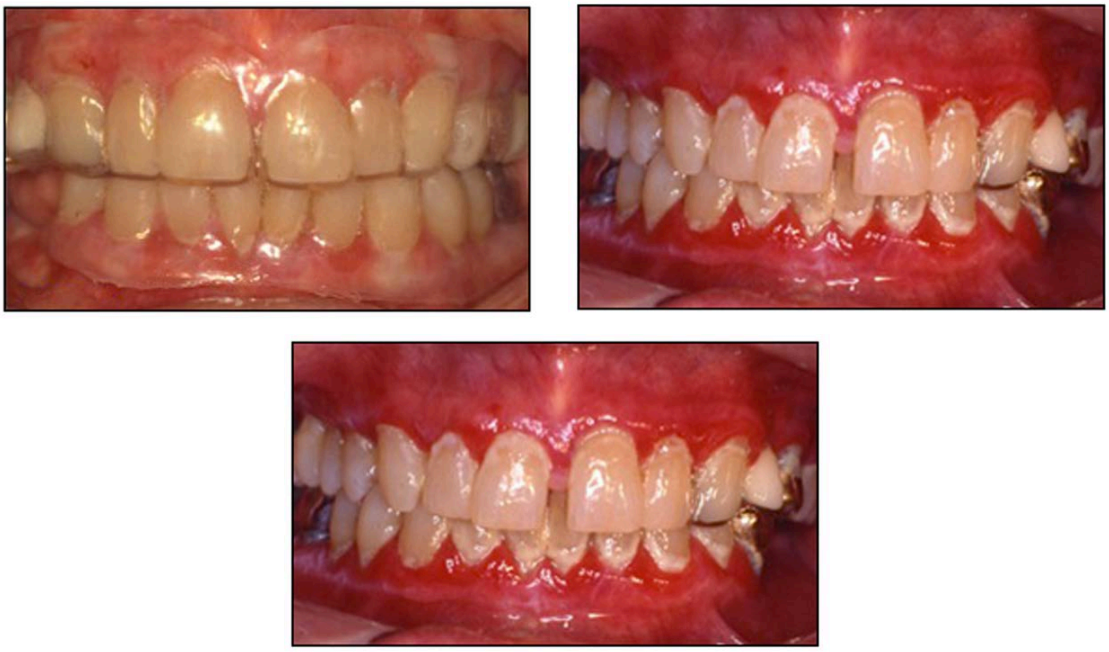


Figure 14. Effective carrier tray treatment.
DG with histologic confirmation and DIF support showing a linear pattern of fibrinogen at the basement membrane zone.

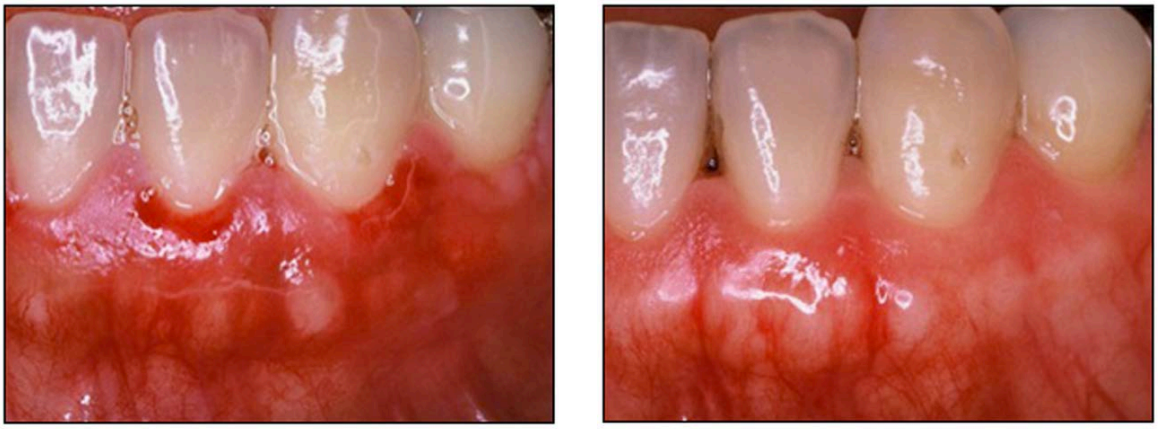


Figure 15. The “key” for oral lichen planus - early diagnosis and treatment.

the time of contact of the immune suppressant with the gingival lesions. One suggested protocol is to use the trays twice daily for 20 minutes during each application.

Although effective oral hygiene has been reported as important to DG resolution, it is often difficult to achieve during the acute phase of gingival lichen planus.[9,78] However, it is most important to improve plaque control even in the early stages of the disease.[30,40,75]

- Instruct patients to gently perform oral hygiene measures using a super-soft toothbrush and a bland toothpaste.
- Use a non-alcohol containing antimicrobial mouthrinse twice daily.
- Patients may be able to gently floss without tissue damage. If not, they should forgo the use of floss pending improvement in their gingival health.
- Maintain a 2-3 month recall interval to provide professional assistance in plaque control.
  - Gentle debridement of plaque and calculus is indicated but vigorous scaling and root planning should be avoided.
  - Hand instruments may be best for debridement since sonic or ultrasonic devices may tend to dislodge friable epithelial and cause sloughing.

It is important to remember DG with clinical and histologic features of lichen planus or a lichenoid mucositis may be caused by a lichenoid drug reaction or a contact lichenoid reaction to dental materials or dental hygiene products.[42] Patients who are resistant to conventional lichen planus therapy should possibly be investigated for such etiologic possibilities.[30,95] A variety of treatment options have been described although use of long-term (> 2-3 weeks) systemic immunosuppressive medicaments may be best performed by the patient's physician.[4,9,14,19]

Mucous Membrane Pemphigoid

Pemphigoid is a group of rare, debilitating autoimmune blistering diseases that can primarily affect the skin (bullous pemphigoid) or mucous membranes (mucous membrane pemphigoid). It may affect children and young adults, but it is most often found in older individuals of either sex.[18] On some occasions blistering begins during or shortly after pregnancy (pemphigoid gestationis).[10] Although the disease may begin on skin or mucous membranes, if untreated, it may spread to other areas of the body. On occasion pemphigoid is named according to the site of involvement (i.e., ocular pemphigoid and oral pemphigoid), and sometimes it is named according to the DIF location of immunoglobulin (i.e., linear IgA disease). Often oral mucous membrane pemphigoid (MMP) occurs as the first or only manifestation of the disorder with DG as the only noted clinical feature. However, the mucosa of the eyes, nose, genitalia and throat may be involved.[3] Ocular MMP is one of the most devastating forms of the disease, and upon diagnosis of oral MMP it is very important to refer the patient to an ophthalmologist to rule out any ocular involvement. Older medical literature indicates that prior to development of relatively effective treatment modalities, many individuals with eye lesions would lose vision in one or both eyes due to progressive scarring.[17,83] Scarring can also result in upper airway obstruction although scarring is very rare on oral mucosa.[36] Occasionally, various medications (captopril, clonidine, furosemide, penicillamine, practolol and others) may trigger a pemphigoid-like process, but these lesions are far more common on skin.[94]

Oral MMP has a mean age of onset of 50 years, and it affects women slightly more often than men. Although lesions may be present on any oral mucosal tissue, the gingiva is usually involved (92.5%) and often DG is the only oral feature of the disease (62.3%).[36] When possible, it is very important to perform H&E and DIF biopsy evaluation. Histologically MMP involves the basement membrane zone between the epithelium and connective tissue, and it is characterized by separation between the basal cells of the epithelium and the underlying connective tissue. A mild to moderate mixed inflammatory infiltrate is usually present in the lamina propria. DIF reveals a linear pattern of complement (C3) and immunoglobulin G (IgG) along the basement membrane zone. Occasionally IgM and/or IgA may be present in the linear pattern.

Therapy for oral MMP usually consists initially of topical or systemic corticosteroids as described previously. If topical steroids are used,

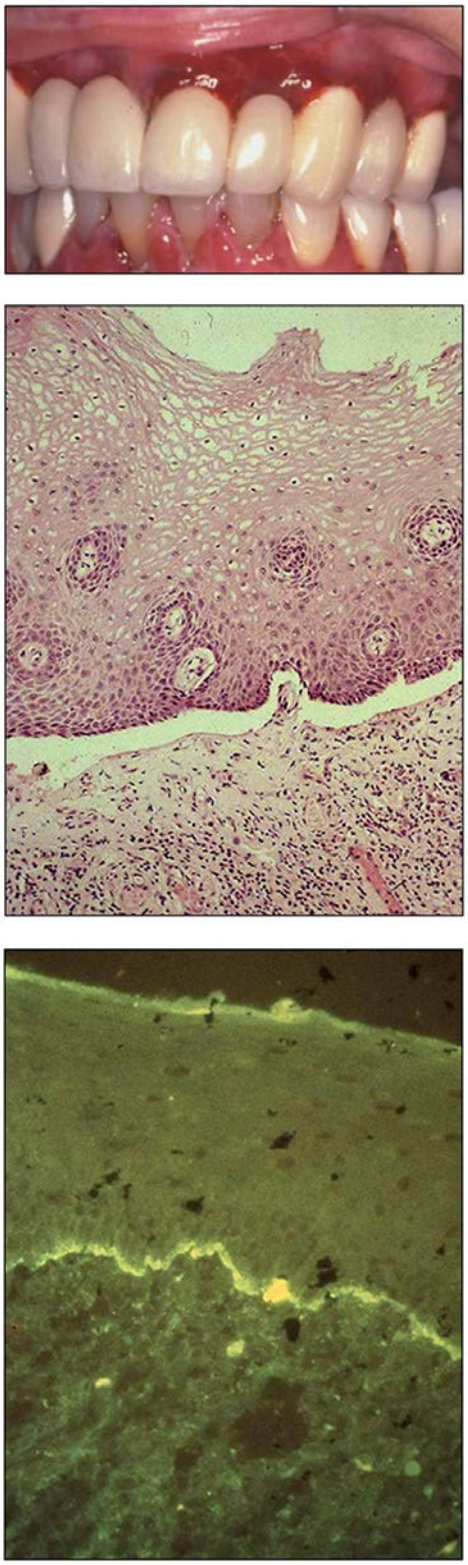


Figure 16. Mucous membrane pemphigoid and biopsy confirmation. DIF shows a linear deposit of IgG in the basement membrane zone.

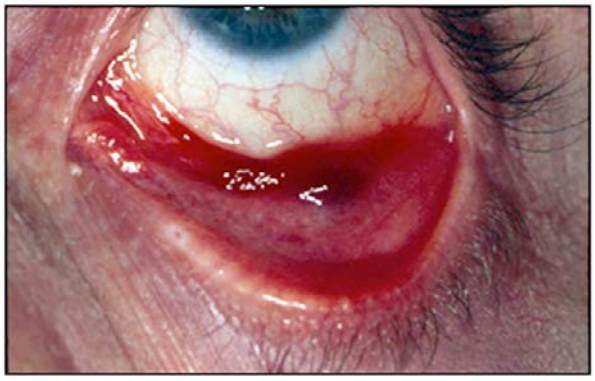


Figure 17. Severe MMP ocular lesion - danger of loss of vision.

a carrier tray is often beneficial.[101] Systemic corticosteroids may be useful for short-term (2-3 week) treatment designed to achieve improvement in DG lesions, but this therapy is usually supplemented with or followed by topical agents. While complete remission is rarely achieved in managing MMP, it is often possible to improve patient comfort. Individuals with MMP that is resistant to a conservative but aggressive therapy should be referred to their dermatologist or internist for management with more potent therapeutic agents.

Maintenance of periodontal and dental health is important and can be achieved using the methods previously described. It may be of interest to note two studies have compared the periodontal health of individuals with treated MMP associated DG and age and sex matched control patients without MMP. In the first study, there were no significant differences in the incidence of periodontitis between the two groups.[96] In a follow-up study, 5 years later involving half of the same MMP patients and their original age and sex matched controls, identical findings were evident. This appears to offer moderately strong evidence the improved oral health of individuals with treated oral MMP can result in the effective maintenance of periodontal and dental health.[81]

Pemphigus Vulgaris

Pemphigus is a rare, heterogeneous group of idiopathic, autoimmune vesiculo-bullous diseases of skin and mucous membranes that are characterized by development of antibodies to desmoglein (dg) 1 or 3, the intrarcellular transmembraneous glycoproteins involved in

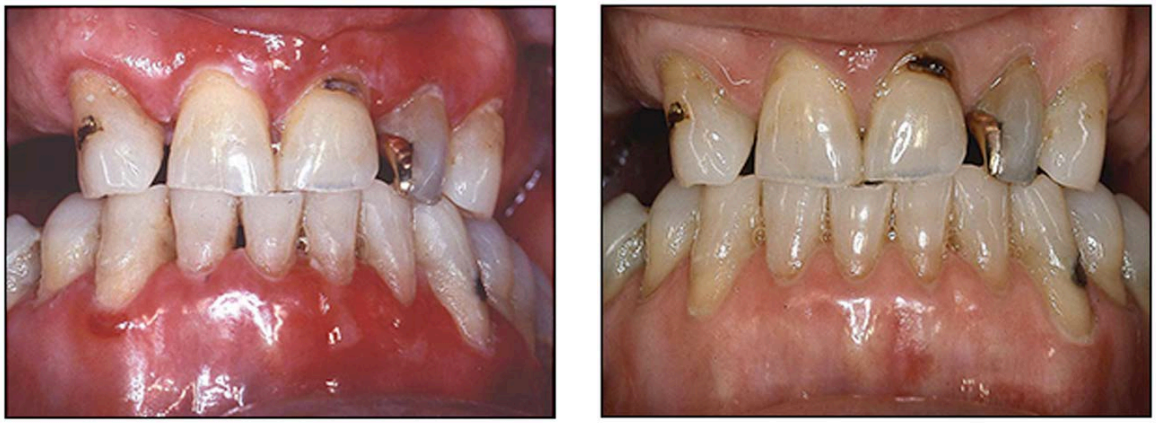


Figure 18. MMP before and MMP after successful treatment.

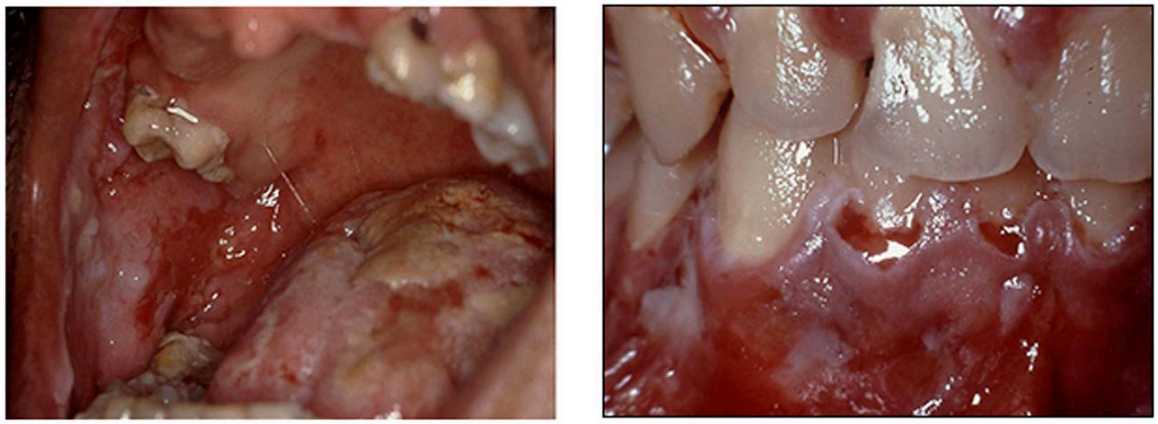


Figure 19. Pemphigus vulgaris – clinical.

maintenance of adhesion between epithelial cells of the skin and mucous membranes. Dg3 is the predominant antigen found in oral PV, while dg 1 and 3 are both elevated when the skin is affected. An interesting case series described successful topical treatment of mild oral PV associated only with dg3 but subsequently the condition recurred involving both skin and oral mucosa and lesions were accompanied by a transition to both dg1 and dg3 antigen.[32,35] Pemphigus vulgaris (PV) is the most common and severe form of the pemphigus disease group. It is potentially life-threatening and it causes various degrees of acantholysis in the epithelium, creating blistering lesions on mucosa and skin. PV can occur at any age and it affects males and females equally.[10,15,31,102] On skin, PV can involve multiple tissue surfaces leading to potential septicemia and loss of tissue fluid and electrolytes. Untreated PV was once considered to be invariably fatal. Fortunately several highly effective therapeutic techniques have been developed that have markedly reduced the incidence of fatality.[30,102]

PV often affects mucous membranes and can have devastating effects there as well and oral lesions are very common. Indeed, oral lesions are the first site of PV involvement in 50%-80% of patients, and early diagnosis and treatment can often minimize disease progression.[32,35]

Unfortunately, a recent survey sponsored by the International Pemphigus and Pemphigoid Foundation (IPPF) determined the average time from onset to diagnosis of PV and MMP is approximately 6 months or longer.[86] Consequently, the condition may be in an advanced stage before therapy is initiated. This fact may be of special importance for the dental provider since the first PV lesion typically occurs in the mouth and the symptomatic patient will usually seek care from their general dentist or periodontist. Dental health care providers are normally not responsible for the treatment of PV, rather they serve to facilitate diagnosis with biopsy evaluation of early lesions or referral for biopsy. Disease therapy is commonly provided

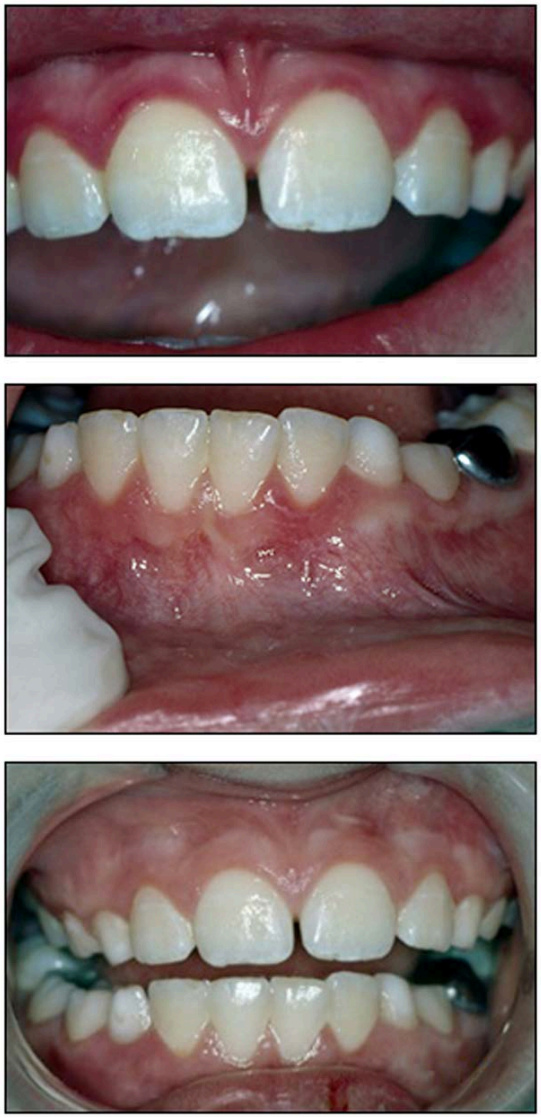


Figure 20. Early diagnosis and treatment of PV.

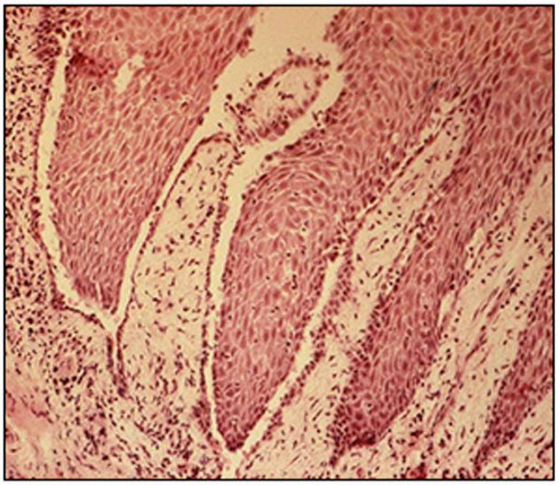


Figure 21. PV histology and DI.

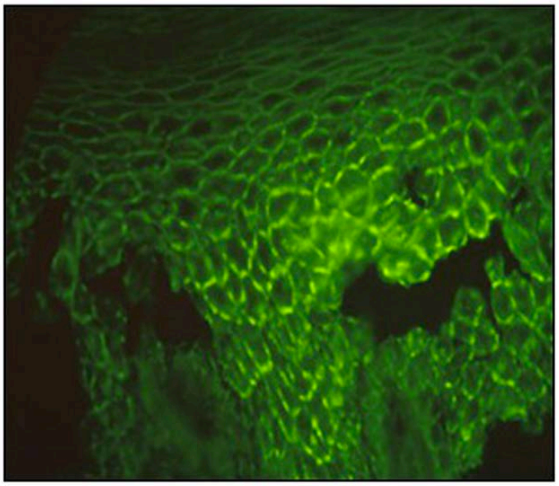


Figure 22. Immunofluorescence of Pemphigus Vulgaris showing IgG in the intracellular spaces of the epithelium and cellular separation.

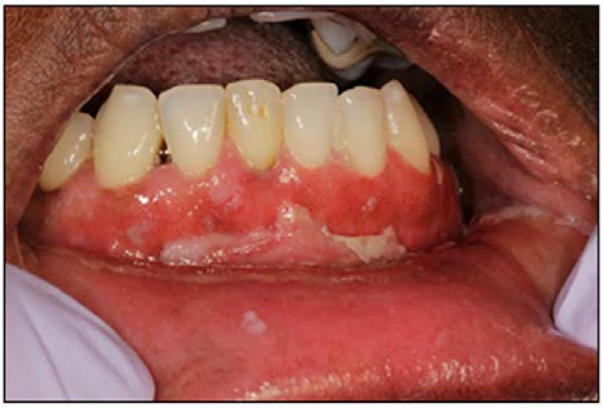


Figure 23. PV with severe epithelial slough.

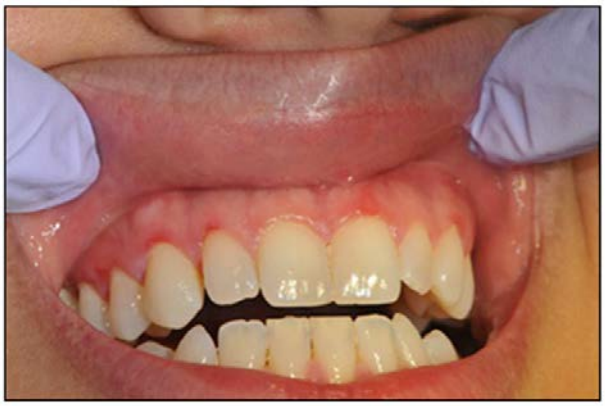


Figure 24. The "key" for PV-early diagnosis and treatment.

by dermatologists, otolaryngologists or internists after referral from the dental practice.

PV lesions can occur on any oral mucosal surface. The buccal mucosa is involved most often followed by the gingiva and tongue. Gingival lesions often present as DG and the gingiva may be the only site of lesions early in the disease process.[58]

Oral PV rarely responds to topical corticosteroid therapy, although intralesional steroid injections are sometimes of benefit for oral refractory sites other than the gingiva.[32,35] Systemic therapy can usually be best managed by a dermatologist, otolaryngolgist, or internist. Short- or long-term systemic corticosteroids are often prescribed, while steroid sparing therapy may be employed using aziathioprine, dapsone, mycofenolate mofetil, or others, and plasmapheresis or IV immune globulin has been utilized. At present, the monoclonal antibody rituximab is increasingly being used with reports of favorable results.[44,101]

Several reports of DG related PV lesions have described increased frequency and severity of dental and periodontal diseases in PV patients with gingival or other oral lesions.[92] However, the use of the conservative minimally invasive oral hygiene measures previously described may help maintain oral health during acute PV phases while gentle oral hygiene measures may be of significant benefit once the oral lesions have improved or reached remission. Although some have suggested the use of a powered toothbrush, our findings indicate sonic and powered toothbrushes may occasionally worsen DG in patients with PV because the brushes disrupt the epithelial mucosal layer rather easily.[92] As with other idiopathic autoimmune diseases, the lesions may occasionally recur after remission and the patient must be instructed to seek care immediately should that happen. A maintenance recall interval of approximately 3 months is often recommended for the first 1-2 years following disease resolution.

Oral Hypersensitivity Reactions

Delayed (Type 4) allergic reactions are surprisingly common in the oral cavity. They may represent a lichenoid drug reaction to systemic medications or, occasionally, drug induced erythema multiforme, but the reactions associated with DG are usually of contact allergy nature.[22,30,57,74,96] This means the soft tissue changes indicative of a hypersensitivity reaction usually occur exclusively in tissue that directly contacts the allergen. Such reactions may be seen in individuals who have received dental restorative materials to which they are sensitive (silver amalgam, cast metal-containing crowns, non- metallic restorative materials, cements, stainless steel orthodontic appliances, etc).[75,76] Normally hypersensitivity reactions to dental materials create a *localized DG*.[21] *Generalized DG* occurs when the individual is allergic to an ingredient of oral hygiene products such as toothpastes or mouthrinses, or in other oral products such as breath mints, dry mouth mints, chewing gum, colas, etc.[29,30,37,75,77] These oral reactions are consistent with those found in *contact dermatitis* reactions except that oral reactions usually require a longer period of contact time before they become evident. We postulate this may be because saliva dilutes or removes the antigen or serves as a buffer or neutralizer to it. The superficial vascularity of oral mucosa also may induce rapid absorption and dispersion of the antigen. Since allergens in dental hygiene products have broad generalized gingival contact, lesions mimicking DG may be created. Saliva definitely plays a role in host defense against such allergens and there is an increased incidence of oral reactions in individuals with salivary hypofunction.[20]

For reasons that are not clear, our data and that of others suggests women are far more susceptible to these types of reactions than men (4:1 females versus males).[60,74] DIF is not indicated because it is routinely negative.[75] Toothpaste may be the most common oral contact allergen. Signs and symptoms of toothpaste hypersensitivity may include generalized or localized DG often occurring in the anterior maxillary gingiva. The lesions may be accompanied by mucositis, glossitis or cheilitis. Lips may be edematous and perioral dermatitis is an occasional finding.[29,30,37] Dentifrice ingredients usually include flavoring agents, coloring agents, abrasives, detergents and preservatives.[20,60] Although one may be sensitive to any of these components, the flavoring agents, especially cinnamic aldehyde and preservatives such as sodium benzoate or methylparaben, are the most common sensitization components.[28,29,37] As mentioned, many patients are allergic to

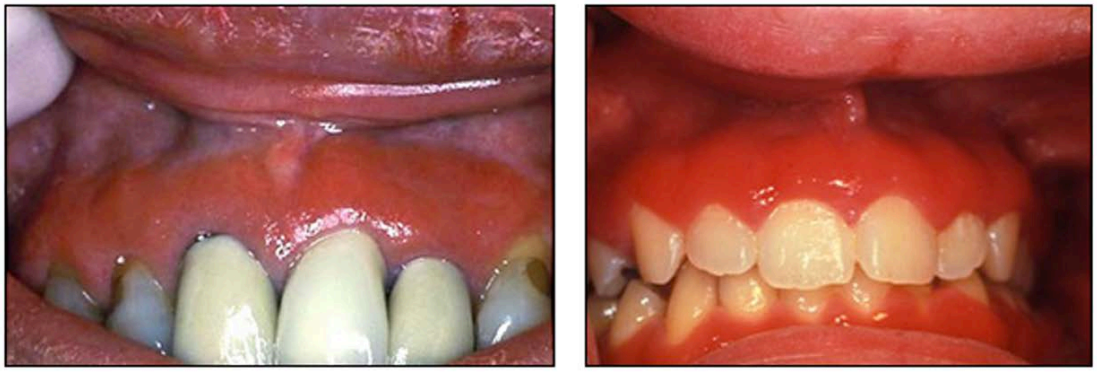


Figure 25. Two patients with contact hypersensitivity to toothpaste.

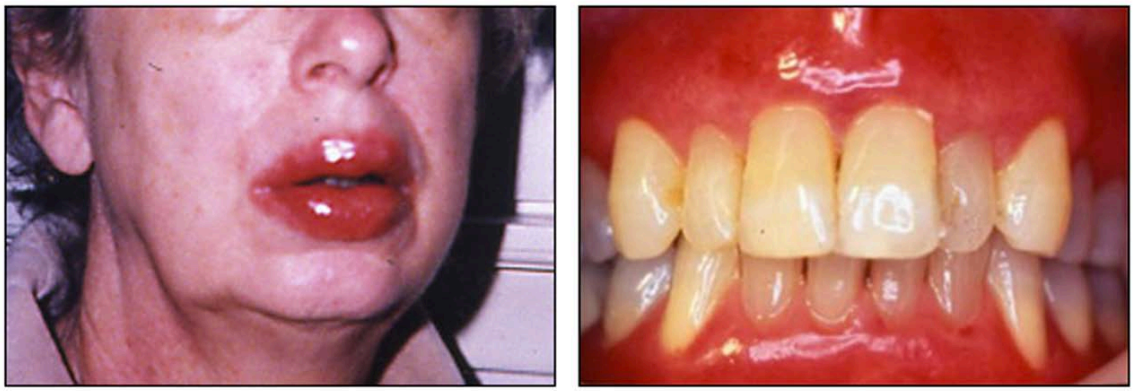


Figure 26. Toothpaste - Patient sought treatment from 8 HCSs for 4 months.

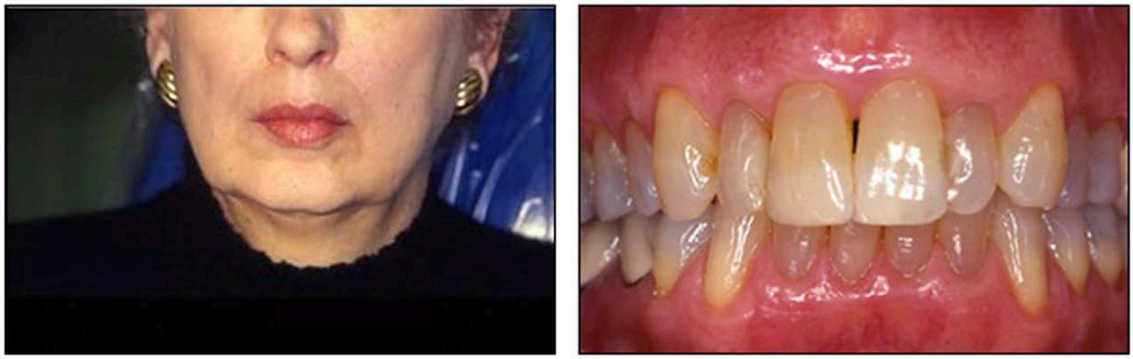


Figure 27. Toothpaste- 3 weeks later.

cinnamic aldehyde but other flavoring agents such as cinnamon oil, menthol, mint/spearmint/peppermint or oil of wintergreen have been increasingly found to cause such reactions.[42] The reason for this is presently undetermined, but one theory suggests the increase in therapeutic agents in toothpastes (tarter control, whitening, anti-dental hypersensitivity products, antimicrobials, etc.) may require a significant increase in the flavoring agents used in order to sustain a pleasant taste.[20] Allergens may also be found in chewing gum, candies, mints, colas and mouthrinses, again resulting in a generalized or localized DG, with or without involvement of other oral mucosa.[30,75] Allergy to dental restorative materials usually causes localized desquamation in gingival tissues directly contacting the restoration.[52,67,68,76,93]

On occasion, patch testing may be required to identify the specific antigen inducing a contact reaction and biopsy may provide histologic evidence *supporting* the diagnosis.[71,93] DIF is not indicated because it is routinely negative.[75] It should be noted,

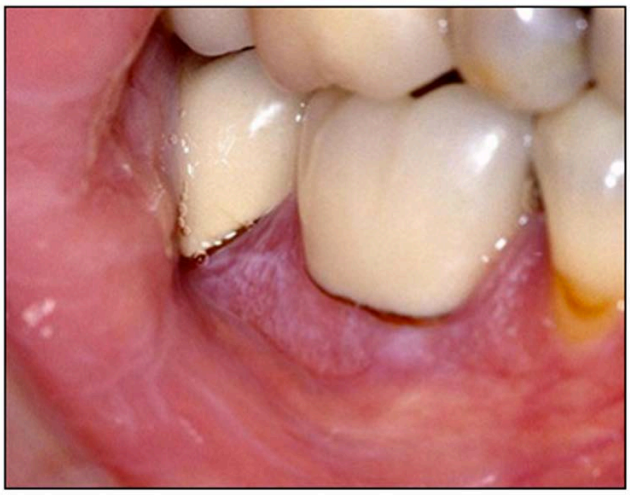


Figure 28. Contact lichenoid reaction to nickel in crowns.

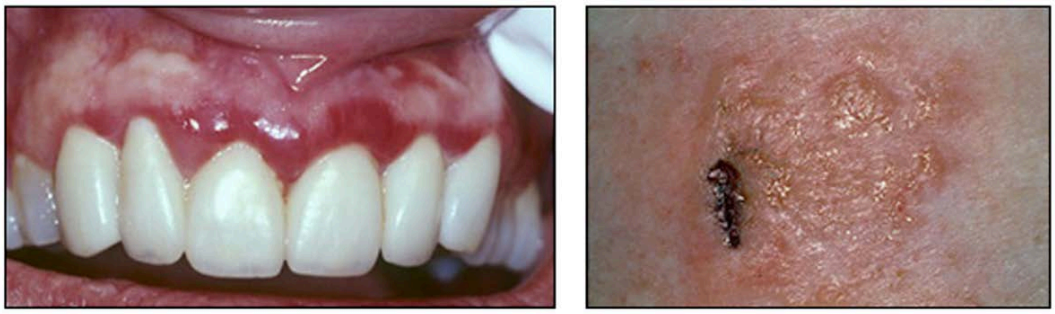


Figure 29. Contact reaction to gold in crowns, patch test positive.

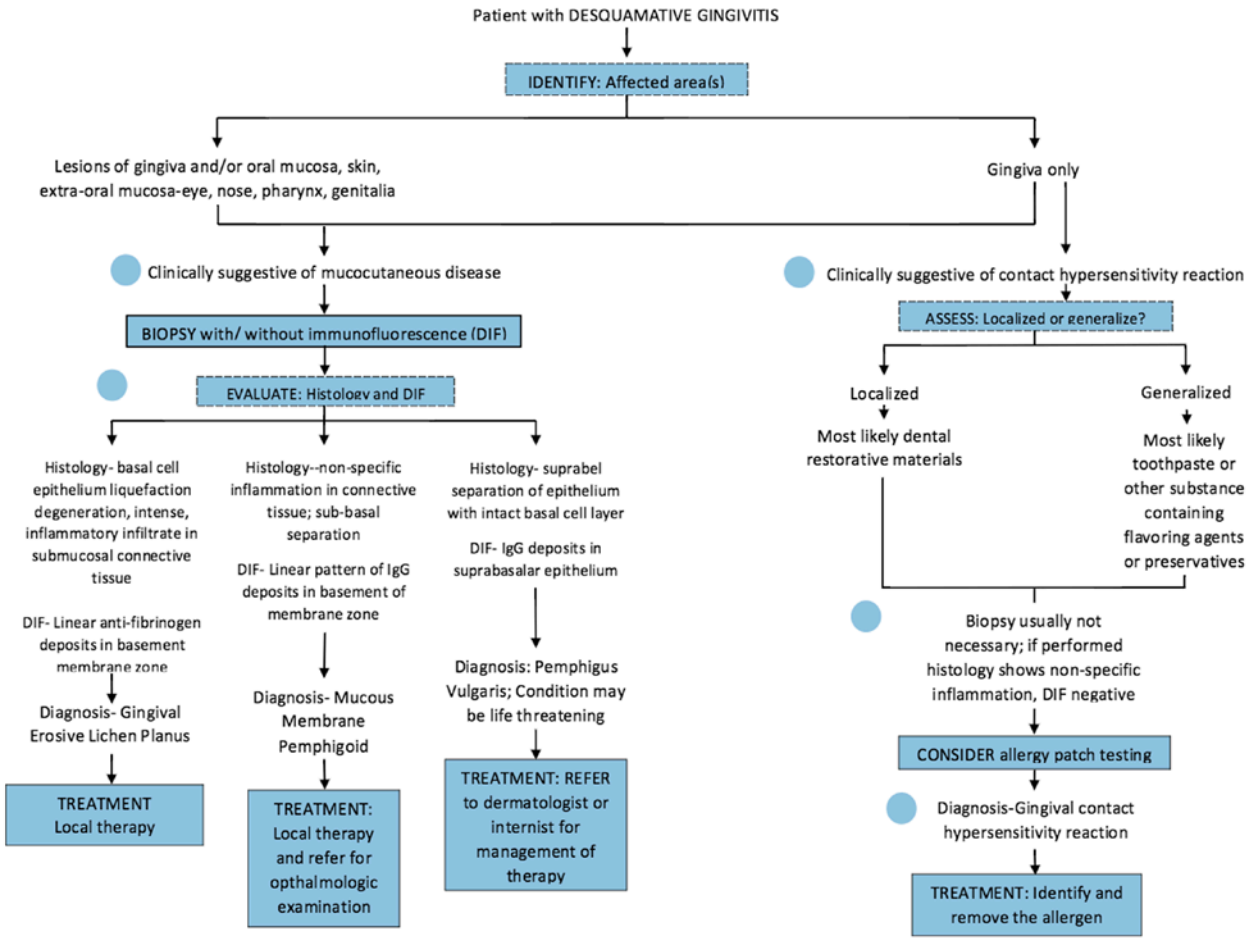
however, diseased tissue such as found in DG may be especially susceptible to contact allergic reactions and one should remain alert to the possibility of two simultaneous etiologic factors contributing to the severity of the lesion.

Treatment of hypersensitivity reactions involves identification and discontinuance of the causative allergen. During the diagnostic phase, the dental practitioner is often asked to provide advice concerning a bland alternative to a toothpaste suspected causing hypersensitivity. Children's or even infant's toothpaste or dry mouth toothpastes are often acceptable alternatives. Patients should be requested to discontinue use of chewing gum, mints, dark colas, and mouthrinses during the diagnostic phase. Once the causative agent is determined, other products can be gradually added back into the patient's customary routine one at a time in order to quickly determine whether or not a contact allergen is present in the product being added.

Conclusion

DG is a painful localized or generalized gingival lesion characterized by epithelial desquamation, erythema, edema and vesiculobullous lesions. It may often be accompanied by similar lesions involving other oral mucosal tissues but on numerous occasions it represents the only clinical manifestation of an oral disease or disorder. Although DG can be caused by a number of rare or uncommon mucocutaneous diseases, it most often represents clinical features of one of four specific disorders: oral lichen planus, mucous membrane pemphigoid, pemphigus vulgaris or oral contact allergic reactions. At present, many patients are not diagnosed until lesions have become severe although onset of DG is usually gradual and mild manifestations precede more severe lesions. Every effort should be made to accelerate the diagnostic process since dental health care providers have a major responsibility for early detection, diagnosis and treatment or appropriate referral after identifying suspicious lesions.

Appendix A. Algorithm



Appendix A. Algorithm.[75]

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Course Test Preview

1. Desquamative gingivitis may be present in various areas of the mouth and also may have varying appearances. However, they are commonly found _____.

a. in the alveolar mucosa
b. in the gingival tissue
c. in the gingival tissue and the alveolar mucosa
d. generalized affecting both the alveolar and gingival tissue
e. localized and generalized in both the alveolar and gingival tissue

2. All of the following may be associated with desquamative gingivitis EXCEPT _____.

a. Lichen planus
b. Erythema multiforme
c. Mucous membrane pemphigoid
d. Pemphigus vulgaris
e. Behcet's Syndrome

3. Baylor College of Dentistry data indicates that over 90% of desquamative gingivitis could be categorized into one of four conditions _____.

a. oral lichen planus, mucous membrane pemphigoid, pemphigus vulgaris or hypersensitivity
b. oral lichen planus, mucous membrane pemphigoid, pemphigus vulgaris or erythema multiforme
c. coral lichen planus, foreign body granulomas, pemphigus vulgaris or hypersensitivity
d. Reiter's syndrome, mucous membrane pemphigoid, pemphigus vulgaris or hypersensitivity
e. oral lichen planus, mucous membrane pemphigoid, pemphigus vulgaris or orofacial granulomatosis

4. Desquamative gingivitis was originally believed in 1932 to be _____.

a. contagious
b. developed by a virus
c. hormonal in women patients
d. predominately in men
e. affecting either sex but bacterial in nature

5. A key feature in desquamative gingivitis is _____.

a. Nikolsy's sign
b. Dupuytren's sign
c. heavy plaque
d. deeper pocket formation
e. severe pain

6. Biopsy specimens are difficult to obtain because of _____.

a. the location of the tissue
b. essential fibrous nature of the tissue
c. the heavy plaque accumulation
d. the inability to obtain the epithelial tissue layer
e. patient reluctance to consent to a tissue biopsy

7. In a survey by the International Pemphigus and Pemphigoid Foundation, a clinical diagnosis of oral lesions occurred after _____ months of time.

a. 2 months
b. 4 months
c. 6 months
d. 8 months
e. 10 months

8. The *consolidation phase*, cited by Brystyn and Scuibba, consists of _____.

a. diagnosis of the disorder
b. therapy designed to reduce or eliminate the symptoms of the disease
c. reduction or elimination of continual therapy
d. long-term control of the disease process
e. retreatment of the existing disease state

9. Which of the following is most likely to exhibit oral lesions, cutaneous lesions and ocular involvement?

a. Pemphigus vulgaris
b. Hypersensitivity
c. Lichen planus
d. Orofacial granulomatosis
e. Linear IGA disease

10. Negative immunofluorescence findings should be anticipated in biopsies of _____.

a. Mucous Membrane Pemphigoid
b. Pemphigus vulgaris
c. Hypersensitivity reactions
d. Oral lichen planus
e. Erythema multiforme

11. When taking a biopsy specimen, the clinician should select tissue that is _____.

a. in direct contact with the most severely ulcerated area
b. located only on desquamative gingiva
c. at the most apical area in the mouth
d. it makes no difference where the tissue sample is taken
e. selected because of an intact epithelial surface

12. An important point to remember when taking a biopsy is _____.

a. take a minimal amount of tissue
b. do not inject anesthesia directly into the biopsy site
c. submit either a histological tissue sample or a sample for immunofluorescence
d. always take a sample of tissue from an uninvolved site
e. remove tissue from sites where no evident epithelium is present

13. A biopsy technique that works well in cases involving desquamative gingivitis by retention of intact epithelium is _____.

a. the scalpel technique
b. the stab-and-roll technique
c. punch biopsy
d. non-serrated tissue forceps
e. any of the above techniques are acceptable

14. The special medium that is used to transport a DIF tissue specimen is named _____.

a. Formalin solution-10% Neutral Buffered
b. Potassium hydroxide transport medium
c. Michel's transport media
d. Roxycin sodium bicarbonate transport media
e. Caldwell's solution

15. The use of fluorescence devices are sometimes indicated for desquamative gingivitis. The results may be skewed because _____.

a. Desquamative gingivitis is more vascular than most other mucocutaneous disorders
b. Desquamative gingivitis usually produces a negative result
c. There are usually more keratotic cells present
d. Fluorescence results suggestive of malignancy may be seen in highly inflamed areas
e. Negative results occur because of the parakeratosis of the epithelium

16. Patients who have desquamative gingivitis have in the past been told that _____.

a. desquamative gingivitis is a disorder that has been linked to Parkinson's disease
b. using certain spices and flavoring agents have caused the disorder
c. they need to learn to live with it
d. there is an association with heart disease
e. the disorder will resolve totally with time

17. The age range of individuals most often affected by oral lichen planus is _____.

a. 20-30 years old
b. 30-60 years old
c. 40-70 years old
d. 50-80 years old
e. 20 and beyond

18. Direct Immunofluorescence may support the diagnosis of oral lichen planus because _____.

a. there will be a separation of epithelial cells
b. the DIF confirms the type of lichen planus
c. DIF may confirm other "skin" disease states
d. a linear deposition of fibrin is noted at the basement membrane zone
e. cytoid bodies are never seen in DIF analysis

19. Oral lichen planus desquamative gingivitis is usually treated with a _____ corticosteroid.

a. low potency cream
b. low potency mouthrinse
c. low potency ointment
d. high potency mouthrinse
e. high potency gel

20. Clobetasol 0.05% (cream) is considered a low dose topical corticosteroid and is recommended because the formulation is superior to the gel form.

a. The information is correct and the rationale is correct.
b. The information is wrong but the rationale is correct.
c. The information is wrong and the rationale is wrong.
d. The rationale is wrong but the information is correct.
e. The information is correct but the dosage of Clobetasol is wrong.

21. Dental hygiene patients with oral lichen planus often adhere to the following protocol _____.

a. scaling procedures are needed yearly
b. use of ultrasonic scaling is recommended
c. use of stronger mouthrinses limit bacteria and candida
d. increased frequency of recall appointments (2-3 months) to maintain periodontal health
e. flossing is contraindicated when there is inter-proximal bleeding

22. Often the only clinical feature that is noted in mucous membrane pemphigoid may be _____.

a. Nikolsky's sign
b. desquamative gingivitis
c. skin lesions
d. bullous oral lesions
e. genital lesions

23. A histological term that is used in the diagnosis of pemphigus vulgaris is _____.

a. hyper-chromatism
b. acantholysis
c. plemorphism
d. dense fibrous connective tissue
e. hyaline (amyloid)

24. Delayed oral hypersensitivity reactions are classified as _____.

a. Type 1
b. Type 2
c. Type 3
d. Type 4
e. May be all of the above.

25. The most frequent cause of oral hypersensitivity reaction is _____.

a. mints
b. gum
c. foods
d. toothpaste
e. environmental agents

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Dr. Burkhart received a Bachelor of Science degree in dental hygiene from Fairleigh Dickinson University School of Dentistry preceded by an Associate degree from Bergen Community College, Paramus, New Jersey, a Master of Education degree from North Carolina State University in Occupational Health Education, a Doctor of Education degree From North Carolina State University in Adult Education/Interdisciplinary studies (graduate level courses in oral pathology). She conducted a one-year postdoctoral fellowship in the section of Oral Pathology at the University of North Carolina School of Dentistry in Chapel Hill. Her dissertation topic was “Oral Lichen Planus Commonalities: Educational and Psychological Implications.” Dr. Burkhart is an Adjunct Associate Professor in the Department of Periodontics/Stomatology at The Baylor College of Dentistry where she has been a faculty member since 1997. She was previously a faculty member at UNC-Chapel Hill School of Dentistry, New Jersey College of Medicine and Dentistry and Florence Darlington Technical College in South Carolina. She is the founder and a faculty Co-Host of the International Oral Lichen Planus Support Group. Dr. Burkhart has presented papers and seminars both nationally and internationally on Oral Lichen Planus/ Mucosal Diseases and has published articles in national dental journals. She is co-author of “General and Oral Pathology for the Dental Hygienist” published in 2007 through Lippincott, Williams & Wilkins and the book is now in its 2013 second edition. She was a 2006 recipient of the ADHA Crest Award through Procter & Gamble and a 2012 recipient of the Mentor of Distinction award through Philips and PennWell publishing. As a columnist for RDH since 2007, she writes a monthly column titled, “Oral Exams” for the PennWell publication. Her website for seminars is www.nancywburkhart.com.

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