Abstract
The term desquamative gingivitis (DG) describes a clinical condition in which the gingival tissues are erythematous, blistering, and eroding. It is not a diagnosis but is instead a term applied to the manifestation of a multitude of mucocutaneous, systemic, allergic, and immunologic diseases. The majority of cases are caused by oral lichen planus, pemphigus vulgaris, and mucous membrane pemphigoid, but many less common sources need to be considered in the differential diagnosis as well. These include erythema multiforme, lupus erythematosus, drug-induced lesions, graft versus host disease, chronic ulcerative stomatitis, plasma cell gingivitis, linear IgA disease, dermatitis herpetiformis, porphyria, epidermolysis bullosa acquisita, paranephragmatitis and neoplastic disorders, and allergic reactions. The dental clinician can play a crucial role in the diagnosis of these conditions, some of which can cause significant morbidity and even mortality. This course will comprehensively review the clinical, histologic, and serologic findings commonly associated with desquamative gingivitis. The dental clinician can play a crucial role in the diagnosis of these conditions, some of which can cause significant morbidity and even mortality. This course will comprehensively review the clinical, histologic, and serologic findings commonly associated with desquamative gingivitis.

Educational Objectives
At the conclusion of this educational activity participants will be able to:
1. Define the term desquamative gingivitis.
2. List common and rare disorders that encompass this term.
3. Review the clinical, histological, and serological findings commonly associated with desquamative gingivitis.
4. Identify treatments suggested for the disorders associated with desquamative gingivitis.

Author Profiles
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Dr. Naomi Marie Ramer is currently Associate Professor of Pathology and Dentistry and Director of Oral Pathology at Mount Sinai Hospital. She is the Program Director for the newly accredited Oral and Maxillofacial Pathology Residency Program at Mount Sinai. She is author and co-author of more than 40 publications and book chapters and has presented at numerous professional symposia. She was named Best Dentist in America (2004) and Best Dentist Oral Pathologist (2011). In 2005, she began a long standing research project on Adenoid Cystic Carcinoma.

Dr. Molly Cohen is a graduate of the University of Pennsylvania School of Dental Medicine. She practiced general dentistry in Philadelphia and is now a first year resident in Mount Sinai Hospital’s Oral and Maxillofacial Pathology program.

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Etiology, Diagnosis, and Treatment of Desquamative Gingivitis
A Peer-Reviewed Publication
Written by Scott Froum, DDS, Dr. Naomi Marie Ramer and Dr. Molly Cohen

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Abstract
The term desquamative gingivitis (DG) describes a clinical condition in which the gingival tissues are erythematous, blistering, and eroding. It is not a diagnosis but is instead a term applied to the manifestation of a multitude of mucocutaneous, systemic, allergic, and immunologic diseases. The majority of cases are caused by oral lichen planus, pemphigus vulgaris, and mucous membrane pemphigoid, but many less common sources need to be considered in the differential diagnosis as well. These include erythema multiforme, lupus erythematosus, drug-induced lesions, graft versus host disease, chronic ulcerative stomatitis, plasma cell gingivitis, linear IgA disease, dermatitis herpetiformis, psoriasis, epidermolysis bullosa acquisita, paraneoplastic and neoplastic disorders, and allergic reactions. The dental clinician can play a crucial role in the diagnosis of these conditions, some of which can cause significant morbidity and even mortality. This course will comprehensively review the clinical, histologic, and serologic findings commonly associated with DG and include other rare disorders that should be considered in the differential diagnosis of DG.

Introduction
Desquamative gingivitis describes a painful, non-plaque induced, sloughing of the gingiva. The lesions of DG can occur at any gingival site (Figure 1) but are most common on the labial aspect of anterior teeth.1 It is considered to be a manifestation of a number of diseases, most peaking in the fourth to sixth decades of life 2 that not only affect oral health, but systemic health as well. DG may represent an early manifestation or the only clinical presentation of many of these diseases, and early detection can greatly improve the prognosis for the patient. Histopathologic and serologic findings identify the causative disorders as oral lichen planus (OLP), pemphigus vulgaris (PV), mucous membrane pemphigoid (MMP),1,6 erythema multiforme (EM),6,7 graft versus host disease (GVHD),1,2,8 lupus erythematosus (LE),9,10 chronic ulcerative stomatitis (CUS),11 plasma cell gingivitis (PCG),12,13 linear IgA disease (LAD),6,14 dermatitis herpetiformis (DH),17 epidermolysis bullosa (EB),15 epidermolysis bullosa acquisita (EBA),2,6,15 paraneoplastic pemphigus (PP),4,6 psoriasis (PS),16-19 foreign body gingivitis (FBG),20 drug-induced lesions (DI),4 and leukemias.1 A comprehensive review of the differential diagnoses, demographics, histopathology, necessary serology tests, and treatments is presented in this course to assist the clinician in the management of the wide array of these diseases.

Oral Lichen Planus
OLP is a mucocutaneous, immune-mediated disorder with an often sub-acute onset. It is caused by autocytotoxic T-lymphocytes that trigger apoptosis of epithelial cells usually causing white striations and plaques with occasional blisters and bullae of the gingiva.1 It is most common in middle-aged adults with a 3:2 predilection for women and an oral incidence of 0.1%-2.2%. It can affect the skin, nails, esophagus, glans penis, and vulva, as well.6 It has been classically estimated to be responsible for 24%-45%7 of cases of DG but may be as high as 75%.1 Diagnosis of DG caused by OLP is based on the histopathological findings of a biopsy. The characteristic histologic feature of lichen planus (Figure 2) is a subepithelial band-like infiltrate of T-lymphocytes with liquefaction of the basal cell layer that may result in a cleft. The epithelium is atrophic in erosive forms with “saw-toothed” rete ridges. Degenerating, eosinophilic keratinocytes called Civatte bodies may be seen at the junction of the epithelium and connective tissue. Immunofluorescent studies are nonspecific usually showing a shaggy deposition of fibrinogen along the basement membrane zone but may be useful to rule out diseases with a similar histologic presentation including systemic only clinical presentation of many of these diseases, and early detection can greatly improve the prognosis for the patient. Histopathologic and serologic findings identify the causative disorders as oral lichen planus (OLP), pemphigus vulgaris (PV), mucous membrane pemphigoid (MMP),1,6 erythema multiforme (EM),6,7 graft versus host disease (GVHD),1,2,8 lupus erythematosus (LE),9,10 chronic ulcerative stomatitis (CUS),11 plasma cell gingivitis (PCG),12,13 linear IgA disease (LAD),6,14 dermatitis herpetiformis (DH),17 epidermolysis bullosa (EB),15 epidermolysis bullosa acquisita (EBA),2,6,15 paraneoplastic pemphigus (PP),4,6 psoriasis (PS),16-19 foreign body gingivitis (FBG),20 drug-induced lesions (DI),4 and leukemias.1 A comprehensive review of the differential diagnoses, demographics, histopathology, necessary serology tests, and treatments is presented in this course to assist the clinician in the management of the wide array of these diseases.

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lupus erythematosus (SLE), chronic ulcerative stomatitis (CUS), and lichenoid reactions. The recommended treatment of OLP is topical corticosteroids such as fluocinonide, betamethasone, or clobetasol. These can be used with custom trays to improve contact with the affected area. Immunosuppressive agents have also been used, but are not recommended as a first line of treatment due to significant cost, severe side effects, and inconclusive literature. The condition is chronic and patients should be informed that there will be a recurrence and they will need long-term follow-up.

**Pemphigus Vulgaris**

PV is a rare, chronic, autoimmune disorder that results in blistering of the skin and mucosa. It has an incidence of one to five cases per one million people per year and is usually seen in middle-aged adults especially of Mediterranean, South Asian, and Jewish descent, with rare cases in children. Though very uncommon, if untreated it can lead to death from fluid loss, electrolyte imbalance, and septicemia. PV accounts for 3% to 15% of cases of DG (Figure 3a) and involvement of the oral mucosa in the early stages of PV can be observed in 70% of cases. Lesions of this disease present as fluid-filled blisters that rupture, leaving behind irregularly-shaped, erythematous, painful ulcerations. A positive Nikolsky sign where slight rubbing of the skin or mucosa elicits bulla formation in affected areas is a characteristic feature. The bullae of this disease are caused by an autoantibody against the epidermal cell surface glycoproteins desmoglein 3 and desmoglein 1. These autoantibodies inhibit the ability of molecules to adhere to one another causing an intraepithelial cleft above the basal cell layer. Biopsies should be taken from perilesional, not ulcerated tissue. Specimens show intraepithelial separation above the basal cell layer leaving behind a characteristic “tombstone” pattern. The cells of the epithelium break apart individually as diagnostic rounded cells called “Tzanck cells” in cytologic smears. Diagnosis is confirmed with direct immunofluorescence (Figure 3d) where antibody and complement are noted in the intercellular spaces of the epithelium and indirect immunofluorescence, which demonstrates circulating epithelial autoantibodies in patient serum. ELISA (enzyme-linked immunosorbant assay) can be used to detect circulating autoantibodies as well.

Treatment includes systemic corticosteroids, usually prednisone, with immunosuppressive agents, such as azathioprine or cyclosporin. Topical corticosteroids have been used with good results in the oral cavity as well, but treatment must include systemic therapy. Referral to a dermatologist who has experience with immunosuppressives is recommended. PV can undergo complete remission with therapy; however, it has a 5%-10% mortality rate usually due to long-term need for systemic corticosteroids.

**Mucous Membrane Pemphigoid**

MMP is a chronic, blistering, mucocutaneous autoimmune disease. It may represent a group of diseases all of which involve tissue-bound autoantibodies that are directed against components of the basement membrane. The lesions of MMP
are commonly found on the gingiva (Figure 4a) and may be painful. The average age of onset is between 50 and 60 years old, and women are affected twice as often as men. MMP can affect extraoral sites with conjunctival involvement leading to blindness and laryngeal involvement causing airway obstruction. It has classically been estimated to be the cause of 35%-48% (the majority) of cases of DG, but some recent studies suggest that it may be closer to 8%-14%. MMP appears clinically similar to PV; however, the clinician is more likely to be able to identify the vesicles or bullae of MMP at presentation due to subepithelial clefting of MMP versus intraepithelial clefting of PV. Positive Nikolsky sign is also observed.

Biopsy should be taken of perilesional tissue (Figure 4b) which shows a split between the surface epithelium and underlying connective tissue below the basement membrane. Microscopic diagnosis should be confirmed with direct immunofluorescence (Figure 4c), which highlights a continuous linear band of immunoreactants at the basement membrane zone. The immunoreactants are usually C3 and IgG; but IgA and IgM may also be seen.

**Erythema Multiforme**

EM is a mucocutaneous blistering and ulcerative condition that has an acute onset and is self-limiting but may be chronic and episodic as well. Patients are usually in their third or fourth decades of life and men are affected more often than women. Its cause is uncertain but may be an immunologic response to medications such as NSAIDs, barbiturates, sulfonamides, and certain antibiotics, or infectious agents, especially the herpes simplex virus. The severity of EM varies and includes; EM minor, EM major, Stevens-Johnson Syndrome (SJS) and toxic epidermal necrolysis (TEN). Orally, hemorrhagic crusting of the lips and cutaneously, target lesions are characteristic symptoms. A positive Nikolsky sign is noted. EM minor is usually self-limiting but EM major and TEN can be progressive and life-threatening. A recent study suggests that EM (Figure 5) may be responsible for 2% of cases of DG.

Perilesional biopsy shows subepithelial or intraepithelial vesiculation with mixed inflammatory infiltrate and sometimes basal cell and keratinocyte necrosis. There may be perivascular inflammation as well. Necrosis of the whole dermis or mucosa is seen in severe cases. Immunofluorescence is nonspecific but is helpful in ruling out other vesiculobullous diseases.

Treatment should begin with identification and removal of the cause, if one can be found. Minor forms will regress spontaneously and emphasis should be on hydration and topical analgesics. Use of corticosteroids for all forms of the disease may be detrimental and is not recommended. The best treatment for SJS and TEN is yet to be determined, but patients usually require in-patient treatment.
The following entities comprise a very small minority of the cases of desquamative gingivitis.

**Paraneoplastic Pemphigus**

PP is a vesiculobullous disorder related to benign and malignant hematologic and nonhematologic neoplasms. There have been 150 reported cases with clinical presentations ranging from PV-like to EM-like to OLP-like, and 100% of documented cases have oral involvement. PP usually has a sudden onset with vesiculobullous lesions of the skin and mucosa. Histologic presentation resembles PV with intraepithelial clefting and MMP with subepithelial clefting. Direct immunofluorescence is not diagnostic but indirect immunofluorescence with rat bladder mucosa shows a specific pattern of localization to intercellular areas of the epithelium. Diagnosis is confirmed with immunoprecipitation of desmoplakin I and II, major bullous pemphigoid antigen, envoplakin, and periplakin. Treatment of tumor-source may improve symptoms along with corticosteroids and immunosuppressants, but PP has high morbidity and mortality.

**Lupus Erythematosus**

LE is an autoimmune connective tissue disease. Women are affected six to ten times more than men with average age at diagnosis ranging from 15 to 40 years. Patients present with fever, weight loss, arthritis, fatigue, and a classic “butterfly” rash across the nose and malar area. Kidney failure is the most significant aspect of the disease. Up to 75% of patients will have an oral complication with LE ranging from xerostomia to ulceration. Histologically, oral lesions of LE demonstrate a pattern very similar to OLP, best distinguished using immunofluorescence. In LE, direct immunofluorescence will demonstrate a positive lupus band test showing subepithelial immunoglobulin and complement deposition at the basement membrane zone. For additional confirmation, 95% of LE patients produce antinuclear antibodies on evaluation of serum. Most patients will respond to antimalarial therapy and NSAID usage. Systemic corticosteroids are needed in more severe cases, and oral lesions will usually respond to systemic therapy.

**Psoriasis**

Psoriasis is a chronic disease characterized by an increased proliferation of keratinocytes presenting as cutaneous erythematous papules and plaques with white scales. Dermal psoriasis presents in the second or third decade of life with equal distribution among men and women. Intraoral psoriasis is rare and may present as geographic tongue or less commonly as DG. It is unclear if intraoral psoriasis can occur without the cutaneous form. Diagnosis requires a biopsy that should show parakeratotic, acanthotic epithelium with anastomosing rete ridges and focal areas of thin epithelium. Dilatation of superficial capillaries and neutrophils should be seen in the superficial layers. A fungal origin should be ruled out. Oral lesions follow the clinical course of the skin lesions and specific treatment of oral lesions is only necessary if symptomatic. This treatment should include a topical anesthetic and topical corticosteroid.

**Linear IgA Disease**

LAD is an acquired, autoimmune vesiculobullous disorder of unknown cause. Its peak incidence is in patients between 60 and 65 years old and it affects women more than men (2:1). It predominantly affects the skin, but can also present as oral ulcerations and DG. A biopsy specimen will show a subepithelial split. However, direct immunofluorescence distinctly demonstrates a homogeneous, linear deposition of IgA only at the basement membrane zone. This can be seen in lesional and nonlesional tissue. LAD will usually not respond to topical corticosteroids. Treatment should include low-dose systemic corticosteroids with dapsone or sulfapyridine, but many cases resolve spontaneously.

**Chronic Ulcerative Stomatitis**

CUS is an immune-mediated disease in which patients develop an autoantibody to a protein involved in epithelial growth and differentiation. CUS is most commonly a disease of women in their sixth decade of life. Clinically and histologically, the lesions are very similar to OLP but with a more varied inflammatory infiltrate. CUS is often treated as OLP, and when topical corticosteroids are ineffective, direct and indirect immunofluorescence studies are performed. Direct immunofluorescence studies show IgG autoantibodies directed against the nuclei of stratified squamous cells of the basal one-third of the epithelium. Stratified epithelial specific antinuclear antibodies are also detected by indirect immunofluorescence. Hydroxychloroquine, an antimalarial drug, has been shown to be an effective treatment, but caution should be exercised with its use as side effects include retinopathy and aplastic anemia.

**Plasma Cell Gingivitis**

PCG is a hypersensitivity reaction first recognized as an allergy to an ingredient used in chewing gum in the 1960s and 70s. Classically, it presented as a triad of symptoms: gingivitis, glossitis, and angular cheilitis. Since the removal of this ingredient from gum, the number of cases has dropped dramatically and was once considered to no longer exist. However, allergies to certain herbal toothpastes, mints, and peppers may still present as plasma cell gingivitis, which is currently usually limited to the gingiva. Histologically, psoriasiform hyperplasia with underlying plasma cell infiltrate is seen. These plasma cells should be tested for polyclonality to rule out malignancy. A comprehensive diary of everything eaten and taken orally should be made, and PCG will resolve when the offending agent is identified and eliminated.

**Foreign Body Gingivitis**

Finding small, localized areas of DG after restorative or hygiene procedures may be due to FBG. It is caused by foreign bodies in the connective tissue thought to enter by damage to
sulcular epithelium during dental procedures. It can occur at any age but is most often seen in adults. Histologically, there is either a granulomatous or lichenoid inflammation, but identification of foreign particles is needed to confirm the diagnosis. It may be difficult to identify the foreign bodies that are usually anywhere from 1-5μm. Initial diagnosis of OLP is often made, but lichenoid inflammation with significant numbers of non-lymphocyte inflammatory cells, localized and small areas of DG confined to the gingiva, history of dental treatment in the area, and unresponsiveness to topical corticosteroids should prompt a search for foreign materials. The affected tissue should be surgically excised and may require grafting if particularly eroded.

Dermatitis Herpetiformis
DH is an autoimmune disease associated with gluten sensitivity, specifically celiac disease. It most commonly affects individuals of Northern European descent between the ages of 20 and 40. There is also a slight female predilection though oral involvement is more likely seen in males. Extremely pruritic, erythematous vesicles are the common cutaneous signs with rapidly rupturing vesicles orally. Its course is chronic with periods of remission and recurrence exacerbated by gluten. Histologically, accumulations of neutrophils in the papillae forming microabscesses are noted, causing vacuolization and blister formation between the tips of the papillae and the epithelium. Direct immunofluorescence will show granular IgA deposits in perilesional samples. Around 85% of patients will produce antigliadin, antismooth muscle endomysium, and antitransglutaminase antibodies. A gluten-free diet and dapsone are the treatment of choice for DH. Complications include scarring of DH lesions and increased risk of developing lymphoma.

Graft Versus Host Disease
GVHD is a complication of allogeneic bone marrow transplantation, a therapy for diseases of the blood and bone marrow, where grafted cells attack the host. Oral involvement is common and may be the only presenting symptom of the disease. GVHD clinically resembles OLP as described above often with accompanying pain and xerostomia. It has both acute and chronic courses. GVHD resembles OLP histopathologically as well. Thus, history of transplant is the key to diagnosis. Treatment by the dental practitioner is limited to topical corticosteroids and topical analgesics for oral lesions but includes medical management with systemic immunosuppressants. Topical tacrolimus has also been shown to be successful if other treatments are ineffective.

Epidermolysis Bullosa and Epidermolysis Bullosa Acquista
EB refers to a spectrum of genetic diseases that are characterized by formation of blisters with only minor trauma to both skin and mucosa. It is caused by defects in the attachment of epithelial cells to each other or the underlying submucosa with the histology reflecting the location of the defect. EBA is a non-hereditary, acquired, autoimmune disease that is not related to EB but presents similarly. It is caused by autoantibodies directed against type VII collagen and histologically exhibits subepithelial clefting. EBA will have a positive salt split skin test forming an artificially induced bulla with deposition of IgG at the connective tissue side of the lesion, the location of type VII collagen. Stressing the importance of homecare with fluoride use and a non-cariogenic diet in order to prevent the necessity for dental treatment is the best way to manage oral EB and EBA. Treatment of EBA may require topical steroids and immunosuppressants as well.

Leukemia
Leukemias are a group of malignancies derived from hematopoietic stem cells. Leukemias can have oral manifestations, especially myelomonocytic types, including diffuse gingival enlargement with or without bleeding and ulceration of the gingival mucosa. In a recent study, one patient out of a group of 125 was found to have DG (Figure 6) caused by acute myeloid leukemia (AML). Biopsy will show sheets of poorly differentiated myelomonocytic or lymphoid cells. Diagnosis is made with biopsy and peripheral blood studies. Oral hygiene should be stressed, and patients should be followed closely as severe oral infections may occur.

Drug-Induced Forms
Lichenoid, MMP-like, PV-like, and lupus-like eruptions can be caused by a wide variety of drugs. Listed here are just a few of the many. Lichenoid lesions may be caused by antimalarials, beta-blockers, and NSAIDs. Antirheumatics and antibiotics can cause MMP-like reactions. ACE inhibitors, antibiotics, and ibuprofen are among the drugs that will cause a PV-like eruption. Finally, a lupus-like reaction can occur with hydantoins, carbamazepine, lithium and many others.
References

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Scott Froum, DDS, is a periodontist and co-editor of Surgical-Restorative Resource e-newsletter, as well as a contributing author for DentistryIQ and Dental Economics. He is a clinical associate professor at the New York University Dental School in the Department of Periodontology and Implantology. Dr. Froum is in private practice in New York City. You may contact him through his website at www.drscottfroum.com.

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Notes
1. The first line of treatment for oral lichen planus is:  
a. Immunosuppressive agents taken systemically  
b. Topical corticosteroids  
c. Antiviral agents  
d. Antibiotics

2. Slight rubbing of the skin eliciting bulla formation is known as:  
a. Linear erythema  
b. Epithelial denudation  
c. Nikolsky sign  
d. Acanthosis

3. Rounded cells typically seen in cytologic smears of pemphigus vulgaris are called:  
a. Rete ridges  
b. Tzanck cells  
c. Parakeratotic cells  
d. Basement membrane

4. Mortality rates associated with pemphigus vulgaris are in the range of:  
a. 5-10%  
b. 20-30%  
c. 40-50%  
d. Above 50%

5. The average age of onset for mucous membrane pemphigoid (MMP) is:  
a. 20-30 years old  
b. 30-40 years old  
c. 40-50 years old  
d. 50-60 years old

6. Common sequelae associated with MMP include:  
a. Blindness  
b. Airway obstruction  
c. Hair loss  
d. Both a and b

7. A patient diagnosed with MMP should also be referred to a:  
a. Cardiologist  
b. Ophthalmologist  
c. Endocrinologist  
d. None of the above

8. Common symptoms of erythema multiforme (EM) include:  
a. Hemorrhagic crusting of the lips  
b. Target lesions  
c. Butterfly lesions  
d. Both a and b

9. EM can be caused by:  
a. NSAIDs  
b. Antibiotics  
c. Herpes simplex virus  
d. All of the above

10. Typical treatment for EM includes the use of:  
a. Corticosteroids  
b. Antiviral agents  
c. Identification and removal of the cause  
d. Antibiotics

11. What percentage of paraneoplastic pemphigus cases show oral involvement?  
a. 10%  
b. 30%  
c. 60%  
d. 100%

12. Women are affected by lupus erythematous (LE) how many times more than men?  
a. 1-2 times  
b. 3-4 times  
c. 4-5 times  
d. 6-10 times

13. Which of the following is a classic symptom of LE?  
a. Target lesion  
b. Oral lesions  
c. Butterfly rash  
d. Hirsutism

14. Treatment of LE can include:  
a. NSAIDs  
b. Antimalarial therapy  
c. Corticosteroids  
d. All of the above

15. Intraoral psoriasis may present as:  
a. Geographic tongue  
b. Gingivitis  
c. Wickham striae  
d. Both a and b

16. Direct immunofluorescence of chronic ulcerative stomatitis patients show which antibodies directed against the epithelium?  
a. IgA  
b. IgE  
c. IgG  
d. IgD

17. Plasma cell gingivitis (PCG) was first recognized as an allergy to an ingredient in:  
a. Candy  
b. Gum  
c. Mouthwash  
d. None of the above

18. PCG symptoms include:  
a. Gingivitis  
b. Glossitis  
c. Angular cheilitis  
d. All of the above

19. PCG is usually resolved by:  
a. Antibiotics  
b. Corticosteroids  
c. Removing the offending agent  
d. None of the above

20. Foreign body gingivitis can often be caused by:  
a. Restorative procedures  
b. Hygiene procedures  
c. Medical procedures  
d. Both a and b

21. Treatment of foreign body gingivitis should include:  
a. Antibiotics  
b. Corticosteroids  
c. Removal of the affected tissues  
d. None of the above

22. Dermatitis herpetiformis (DH) is an autoimmune disease associated with:  
a. Antibiotic sensitivity  
b. Cinnamon sensitivity  
c. Gluten sensitivity  
d. None of the above

23. Treatment of DH includes:  
a. Topical dapsone  
b. Gluten free diet  
c. Antibiotics  
d. Both a and b

24. Complications of DH include:  
a. Rapidly rupturing vesicles  
b. Lymphoma  
c. Scarring  
d. All the above

25. Which of the following is correct regarding graft versus host disease?  
a. A complication of bone marrow transplantation  
b. Oral involvement may be the only symptom  
c. Resembles oral lichen planus  
d. All the above

26. Which of the following is correct regarding epidermolysis bullosa (EB)?  
a. Sensitivity to medications  
b. Environmentally induced  
c. Genetics  
d. None of the above

27. The best way to manage EB from a dental standpoint is to:  
a. Administer antibiotics  
b. Use topical steroids  
c. Stress the importance of homecare and give fluoride  
d. None of the above

28. Leukemias are malignancies derived from:  
a. Liver cells  
b. Hematopoietic cells  
c. Cardiac cells  
d. Smooth muscle cells

29. Oral involvement of leukemias include:  
a. Gingival enlargement  
b. Ulcerations  
c. Bleeding  
d. All of the above

30. Lichenoid reactions can be caused by:  
a. Medications  
b. Dental materials  
c. Oral hygiene agents  
d. All of the above
Etiology, Diagnosis, and Treatment of Desquamative Gingivitis

Educational Objectives

1. Define the term desquamative gingivitis.
2. List common and rare disorders that encompass this term.
3. Review the clinical, histological, and serological findings commonly associated with desquamative gingivitis.
4. Identify treatments suggested for the disorders associated with desquamative gingivitis.

Course Evaluation

1. Were the individual course objectives met?
   - Objective #1: Yes No
   - Objective #2: Yes No
   - Objective #3: Yes No
   - Objective #4: Yes No

Please evaluate this course by responding to the following statements, using a scale of Excellent = 5 to Poor = 0.

2. To what extent were the course objectives accomplished overall? 5 4 3 2 1 0
3. Please rate your personal mastery of the course objectives. 5 4 3 2 1 0
4. How would you rate the objectives and educational methods? 5 4 3 2 1 0
5. How do you rate the author's grasp of the topic? 5 4 3 2 1 0
6. Please rate the instructor's effectiveness. 5 4 3 2 1 0
7. Was the overall administration of the course effective? 5 4 3 2 1 0
8. Please rate the usefulness and clinical applicability of this course. 5 4 3 2 1 0
9. Please rate the usefulness of the supplemental webinography. 5 4 3 2 1 0
10. Do you feel that the references were adequate? Yes No
11. Would you participate in a similar program on a different topic? Yes No
12. If any of the continuing education questions were unclear or ambiguous, please list them.
13. Was there any subject matter you found confusing? Please describe.
14. How long did it take you to complete this course?
15. What additional continuing dental education topics would you like to see?

For Questions Call 216.398.7822

PennWell maintains records of your successful completion of any exam for a minimum of six years. Please contact PennWell for a copy of your continuing education credits earned. This report, which will list all credits earned to date, will be generated and mailed to you within five business days after the exam.

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