Position Paper

Oral Features of Mucocutaneous Disorders*

Part of periodontology involves the diagnosis and treatment of a variety of non–plaque-related diseases of the periodontium. The International Workshop for a Classification of Periodontal Diseases and Conditions noted that the periodontist may be called upon to manage non–plaque-related mucocutaneous disorders either alone, or as part of a treatment team consisting of physicians, dentists or other allied health care professionals. This informational paper will review the etiology, clinical manifestations, diagnosis, and treatment of the most common chronic mucocutaneous diseases, including those that may present as desquamative gingivitis or intraoral vesiculobullous lesions. This paper is intended for the use of periodontists and other members of the dental profession. *J Periodontol 2003;74:1545-1556*.

DESQUAMATIVE GINGIVITIS

Desquamative gingivitis is a clinical feature of a variety of diseases. It is characterized by epithelial desquamation, erythema, ulceration, and/or the presence of vesiculobullous lesions of gingiva and other oral tissues. This phenomenon can be a manifestation of a number of dermatoses, most commonly lichen planus, cicatricial pemphigoid (benign mucous membrane pemphigoid), and pemphigus vulgaris[1-3] (Tables 1 and 2). Biopsy specimens obtained from mucosal lesions may sometimes provide equivocal histopathologic findings and are often inadequate as a single examination to establish the correct diagnosis because several diseases can produce a subepithelial blister. Therefore, direct immunofluorescence examination is necessary to establish a definitive diagnosis. Oral lesions may occur first or very early in several mucocutaneous disorders.[4-6] Accurate diagnosis and effective treatment of these lesions may greatly diminish or reverse disease progression.

LICHEN PLANUS

Lichen planus is a relatively common dermatologic disease that affects the skin and mucous membranes, including the oral cavity. Although the etiology of lichen planus is unknown, its immunologic features suggest a cell-mediated immune response to intraepithelial antigens.[7,8] Lichen planus generally develops between the ages of 40 and 70, and it is more common in females than males.[9,10] Skin and oral lesions of lichen planus in children are rare but have been reported.[11,12] Oral manifestations occur in approximately 2.0% of the general population,[13] while cutaneous lesions occur in 0.4%.[14] Ten percent to 20% of patients with lichen planus demonstrate oral as well as cutaneous lesions.[15]

Intraoral features of lichen planus include reticular, papular, plaque-like, atrophic, ulcerative, and bullous lesions. The reticular pattern occurs most frequently[16] and is often seen as white lace-like lesions located bilaterally on the buccal mucosa. The reticular, plaque-like, and papular forms are generally asymptomatic and may require no treatment. Patients with these types of lesions may report a change in surface texture or roughness in the area that is affected. The atrophic, ulcerative, and bullous forms of the disease are referred to as erosive lichen planus. It is usually the onset of erosive lesions that motivates patients to seek treatment. Patients often present with a combination of painful erosive lesions in conjunction with white lesions. Patients with erosive lichen planus may exhibit desquamative gingivitis and a positive Nikolsky's sign, characterized by epithelial separation from the underlying connective tissue as a result of minor trauma. A small percentage of patients with lichen planus will experience transient small bullae or vesicles involving the mucosal surfaces.[17]

In addition to the oral cavity, lesions may also be seen on the skin, esophagus, genitalia, and rarely the eyes.[18,19] Skin lesions occur alone or in combination with intraoral lesions and present as recurrent violaceus, keratotic, pruritic patches. Vulvovaginal-gingival and peno-gingival syndromes refer to a variant of lichen planus that affects the gingiva as well as the genitourinary tract of either men or women.[20,21]

In lichen planus, as well as other dermatologic diseases affecting the oral mucosa, biopsy specimens are essential in establishing a diagnosis for erosive and plaque-like forms and very helpful for reticular forms.

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Table 1.

Mucocutaneous Diseases That May Present with Desquamative Gingivitis

Chronic ulcerative stomatitis	Linear IgA disease	Pemphigus
Dermatitis herpetiformis	Lupus erythematosus	-Vulgaris
Drug induced	Pemphigoid	- Vegetans
Epidermolysis bullosa aquisita	- Bullous	- Folliaceus
Erythema multiforme	- Cicatricial	- Erythematosus
Graft-versus-host disease	- Ocular	- Paraneoplastic
Lichen planus	- Anti-epiligren	- Benign famial
		Psoriasis

Table 2.

Medications for the Treatment of Diseases Associated with Desquamative Gingivitis

Topical	Intralesional	Systemic
Triamcinolone	Triamcinolone	Prednisone
Fluocinonide		Azathioprine
Clobetasol		Cyclophosphamide
Betamethasone		Methotrexate
Halobetasol		Gold
Retinoids		Dapsone
Tacrolimus		Cyclosporin

The histologic features of lichen planus include epithelial acanthosis and hyperkeratosis, liquifaction degeneration of the epithelial basal cells, saw-tooth rete ridges, and a dense, band-like, sub-basilar infiltrate of T lymphocytes.[22-24] These classic histologic features are more commonly seen in skin biopsies, while mucosal biopsy specimens are often less distinctive in character. Although immunofluorescence studies of lichen planus do not suggest pathognomonic features associated with the disease, direct immunofluorescence examination may be of value in supporting the diagnosis or ruling out other diseases.[6,25,26] A linear or a shaggy deposit of fibrin or fibrinogen at the basement membrane is often observed in biopsy specimens, which are examined using direct immunofluorescence techniques. In addition, cytoid bodies are commonly seen at the epithelial-connective tissue interface and are thought to represent necrotic keratinocytes.[27-29] Although the etiology remains elusive, these histologic and immunofluorescence features suggest that the condition represents a cell-mediated autoimmune response to basal keratinocytes that express a foreign or altered self-antigen.[30,31] This suggestion is supported by recent data which indicate that external substances such as mercury in dental amalgams may induce keratinocyte ICAM-1 expression, increased binding of T cells to normal keratinocytes, and increased production of TNF-α in vitro.[32]

Lichenoid lesions resembling lichen planus may occur in association with the use of medications, including antimalarial drugs, antihypertensives, and non-steroidal anti-inflammatory agents.[33] Lichenoid lesions demonstrate clinical, histologic, and immunofluorescence patterns similar to idiopathic lichen planus, and they often resolve without recurrence following discontinuation of the identified medication.[34]

Exposure to dental restorative materials and cinnamon flavoring agents has also been reported to induce lichenoid reactions.[35-50] Lichen planus may be associated with systemic diseases including hypertension and diabetes mellitus as well as hepatitis B and C.[51-64] Lesions identical to lichen planus are seen in patients with acute and chronic graft-versus-host disease and lupus erythematosus.[65-72]

Treatment of oral lichen planus requires elimination of potential factors associated with lichenoid reactions, elimination or control of local irritants, and the effective use of therapeutic agents that suppress excessive lymphocyte function. Patients with erosive lichen planus are often successfully treated with corticosteroids. Topically applied medications such as fluocinonide and clobetasol gel, beclomethasone dipropionate spray (inhaler), or dexamethasone mouthrinses are effective in inducing remission of lesions.[31,73-75] Short-term tapering doses of systemic corticosteroids such as prednisone or intralesional injections are useful in severe episodes as well as in recalcitrant cases.[76] Although expensive to use, systemic and topically administered cyclosporin has shown promising results.[77,78] Recently, topical tacrolimus has been shown to be an effective form of treatment for oral lichen planus.[79-83] Other medications such as griseofulvin, azathioprine, cyclophosphamide, dapsone, retinoids, metronidazole, lev-

amisole, thalidomide, and low molecular weight heparin have shown some treatment efficacy, but evidence based data are lacking. In addition, the potential for significant side effects may limit their use.[84-94] Periodontists who administer these drugs should be aware of reported side effects and be prepared to take appropriate action should any occur. A physician may need to be involved in diagnosis of associated systemic disease and in provision of systemic therapy. In these circumstances, coordinated follow-up involving both the dentist and physician is important. Although some patients experience complete remission following therapy, lichen planus is more often persistent/recurrent in nature and is likely to require periodic retreatment.

Controversy exists regarding the potential for malignant transformation in patients with lichen planus.[95-98] Some clinical investigations have demonstrated an increased incidence of oral cancer in lichen planus lesions ranging from 0.4% to 5.6%.[99-105] Others, however, have questioned the validity of histologic features used to establish the initial diagnosis. Some early precancerous (dysplastic) lesions may present with lichenoid features, and create the impression of malignant transformation from preexisting lichen planus lesions.[106] A recent systematic analysis, however, indicated that individuals with oral lichen planus may have a 10-fold increased risk of developing squamous cell carcinoma when compared to the general population.[107] Regardless of the dispute, it is clear that regular recalls are important to assess the character of recurrent lichen planus or lichenoid lesions, and periodic biopsies are often necessary for areas that do not respond to treatment.

MUCOUS MEMBRANE PEMPHIGOID

Mucous membrane pemphigoid (benign mucous membrane pemphigoid, cicatricial pemphigoid) is a humoral autoimmune disorder that predominantly affects the oral cavity. Other mucosal surfaces may also be involved, including the conjunctiva, nares, larynx, esophagus, upper respiratory tract, rectum, or genitalia.[108-113] The mean age of onset is 50 years or older. However, case reports of mucous membrane pemphigoid in children and young adults exist.[114] Females are affected more often than males, at a ratio of 2:1.[115,116]

The oral cavity usually represents the first and often the only site of disease involvement. Intraoral manifestations of mucous membrane pemphigoid include desquamative gingivitis, vesiculobullous lesions, and ulcerations. Patients often exhibit a positive Nikolsky's sign with epithelial sloughing and exposure of painful bleeding surfaces beneath. Periods of exacerbation and remission are common, although some lesions may remain unrelenting for years.[117,118] The gingiva is by far the most common intraoral site affected,[116,119] and the lesions tend to heal with insignificant scarring.

In contrast, ocular lesions often exhibit progressive scarring leading to fusion of ocular and eyelid conjunctiva (symblepharon formation).[120,121] Continued scar formation may ultimately result in blindness if untreated.[122,123] Ocular lesions have been reported to occur in 11% to 61% of patients with mucous membrane pemphigoid, while skin lesions occur in 0% to 11%.[124-126] Related conditions such as bullous pemphigoid primarily affects the skin, while antiepiligrin cicatricial pemphigoid characteristically involves the eyes as well as the oral mucosa and/or skin and may represent a paraneoplastic form of the disease.

In mucous membrane pemphigoid, one or more of several heterogeneous antigens (BP180, BP230, laminin 5, and others) found within the basement membrane adhesion complex may be targeted, resulting in an immune response.[31,118,127,128]

Histologically, biopsy specimens from patients with mucous membrane pemphigoid demonstrate a subbasilar separation of the epithelium from the underlying connective tissue. Subepithelial vesicle formation and vacuolation in the basal lamina occur below intact epithelium. In contrast to lichen planus, the inflammatory infiltrate is non-specific in nature, consisting of lymphocytes, plasma cells, and neutrophils.[129]

Direct immunofluorescence testing reveals a linear deposition of complement (usually C_3) and IgG or other immunoglobulins at the basement membrane zone.[130-133] Intact epithelium and connective tissue are critical in evaluating a specimen with direct immunofluorescence techniques. Because desquamation can often be induced by minor trauma, perilesional areas may be chosen as an appropriate site to biopsy, and repeat biopsies may be required if desquamation occurs. Serum indirect immunofluorescence testing has been believed to be of little diagnostic value in mucous membrane pemphigoid since circulating basement membrane antibodies are often not detected.[134] However, improved techniques have recently demonstrated small quantities of circulating serum antibodies in patients with the disease.[135]

Pemphigoid-like lesions have been identified in patients taking systemic medications such as captopril, carbamazepine, clonidine, furosemide, penicillamine, and practolol.[136-145] Elimination of the targeted medication by a physician should be considered when drug reactions are suspected. A form of paraneoplastic

pemphigoid associated with internal malignancy has also been described. These findings indicate that a diagnosis of mucous membrane pemphigoid warrants a medical referral and complete evaluation.[146,147]

Treatment of mucous membrane pemphigoid often includes potent topical corticosteroids alone or in combination with systemic corticosteroids.[124] Dapsone, an antimicrobial agent with immunosuppressive activity, has shown some promise.[129,148,149] Periodic blood studies are necessary, however, when administering dapsone due to its potential to induce hemolytic anemia. Other systemic medications, including immunosuppressive agents such as azathioprine, methotrexate, levamisole, cyclophosphamide, and mycophenolate mofetil may also be effective in the treatment of pemphigoid,[150-152] but the potential for side effects must be considered and managed should they occur. Successful treatment with a tetracycline derivative or a combination of tetracycline and niacinamide has been reported.[153-155] Control of dental plaque and local irritants is important in the management of patients with mucous membrane pemphigoid.[156] Coordinated effort between the dentist and physician is important in developing the most effective treatment regimen for patients requiring systemic therapy. In addition, it is important to refer pemphigoid patients to an ophthalmologist for evaluation.

PEMPHIGUS VULGARIS

Pemphigus vulgaris is a potentially life-threatening autoimmune disease that results in bullae formation involving the skin and/or mucous membranes. It occurs most frequently between the fourth and sixth decades of life and affects individuals of Jewish or Mediterranean descent more frequently than others. The overall incidence of pemphigus vulgaris has been estimated to be 0.5 to 3.2 per 100,000 persons,[157] affecting both genders equally.

Intraoral manifestations of pemphigus vulgaris include intraepithelial separation resulting in the formation of bullous lesions. The bullae soon rupture, leaving painful erosions with ragged borders. Gingival lesions can occur and, along with other oral lesions, may represent the first manifestations of the disease. Lip lesions are typical, in contrast to pemphigoid where they are rare.[158,159] Minor insults to any oral tissues, however, can result in desquamation (Nikolsky's sign).

Skin lesions feature the formation of bullae which quickly rupture, leaving multiple areas of ulceration. The ulcers may cover a significant portion of the body and result in death due to septicemia or fluid and electrolyte loss.[160] In approximately 70% of patients, the initial lesions of pemphigus vulgaris occur in the oral cavity, and oral involvement is evident in almost all patients with advanced disease.[160-162]

Histologically, pemphigus vulgaris is characterized by acantholysis and suprabasilar bullae formation. The basal cells lining the floor of the bullae are often arranged in a tombstone pattern, and acantholytic keratinocytes (Tzanck cells)[163] float freely within the blister fluid. The inflammatory infiltrate in pemphigus vulgaris is predominantly mononuclear.

Examination of specimens with direct immunofluorescence techniques reveals the deposition of complement and IgG, IgM, or IgA[164] within the intercellular spaces of the epithelium, resulting in a reticular pattern diagnostic of pemphigus vulgaris. The antigenic stimulus is desmoglein III, an intercellular desmosomal adhesion molecule.[31] Serum indirect immunofluorescence testing also typically shows epithelial cell surface antibody in substrate tissue. However, circulating antiepithelial antibodies may not be present in patients with early lesions involving the oral cavity.

Pemphigus vulgaris may be associated with systemic medications including captopril, penicillamine, rifampin, and interferon.[165] Paraneoplastic pemphigus is characterized by painful mucosal lesions similar to pemphigus vulgaris in patients suffering from an underlying neoplasia, most commonly lymphoma, leukemia, sarcoma, and squamous cell carcinoma.[117,166-168]

Pemphigus vulgaris is treated by moderate to high doses of systemic corticosteroids alone or in combination with topical corticosteroids.[160,169-171] Azathioprine and other corticosteriod sparing drugs may be introduced into the therapeutic regimen to help control recalcitrant cases.[135,172] Other systemic medications including dapsone[173] and cyclosporin A[174] have shown some efficacy. Since effective therapeutic outcomes may require long-term treatment, this disease is probably best managed by a team approach involving both the dentist and physician. It is important to refer pemphigus patients to an ophthalmologist for evaluation.

PSORIASIS

Psoriasis is a chronic inflammatory mucocutaneous disorder that affects from 1% to 3% of the world population.[175] Skin lesions usually involve the elbows, knees, sacrum, and scalp. They present as localized or generalized erythematous plaques or papules covered with white hyperkeratotic scales. A pustular form of the disease also exists. Intraoral psoriatic lesions are relatively uncommon. They may occur in the presence or absence of cutaneous lesions, but they are most often found in association with skin manifestations.

Intraoral manfestations range from the presence of irregular erythematous lesions with raised yellow to white borders to frank ulcerations as well as desquamative gingivitis.[176-178] Although most patients are asymptomatic, others may complain of tenderness, pain, burning, or roughness in the affected areas.[179]

Lesions involving the gingiva may affect the periodontal status of patients with intraoral manifestations of psoriasis.[180-182] Psoriasiform lesions also include benign migratory glossitis, stomatitis areata migrans, and Reiter's syndrome.[175,183-185] Histologically, these lesions demonstrate epithelial thickening with elongated rete ridges and a chronic lymphocytic inflammatory infiltrate. Intrapapillary microabscesses are a common presentation within the epithelium along with migrating polymorphonuclear leukocytes.[186] Direct immunofluorescence testing may reveal immunoreactants in the stratum corneum of the epithelium.

No definitive treatment for psoriasis has been established. Cutaneous lesions may be managed using a variety of topical and systemic agents. Systemic immunosuppressant drugs such as corticosteroids, cyclosporin, methotrexate, acitretin, and mycophenolate mofetil may be useful in recalcitrant cases, although their effectiveness may be limited due to adverse side effects or toxicity.[187,188] Oral lesions may undergo spontaneous remission or remission in response to systemic therapy. Symptoms of persistent oral psoriasiform lesions may respond to topical corticosteroid therapy or palliative mouthrinses. Meticulous oral hygiene and control of any source of inflammation may be helpful, especially for gingival lesions.

GRAFT-VERSUS-HOST DISEASE

Oral complications occur in almost all patients receiving bone marrow transplantation.[189] Graft-versus-host disease (GVHD) is an immunologic reaction that occurs in 70% to 80% of bone marrow transplant patients and is an important cause of morbidity and mortality. Lesions may occur in various sites including the lungs, liver, gastrointestinal tract, skin, and mucous membranes. Intraoral manifestations of GVHD include lichenoid lesions that may become ulcerative, resulting in significant discomfort.[72,190-194] These lesions are clinically and histologically similar to those associated with lichen planus, lichenoid drug eruptions, and lupus erythematosus.[195] Although primary care of GVHD is managed by the patient's medical team, treatment of oral complications of GVHD often includes the elimination of local irritants[196] and the use of topical medications such as corticosteroids, azathioprine, and cyclosporin.[197-199]

CHRONIC ULCERATIVE STOMATITIS

Chronic ulcerative stomatitis is a rare mucocutaneous disorder that was first described in the early 1990s. It primarily affects elderly females.[200-203] Patients present with desquamative gingivitis that is refractory to treatment with corticosteroids. Direct immunofluorescence examination reveals deposition of IgG in the basal one-third of the epithelium, while indirect immunofluorescence demonstrates the presence of stratified epithelium-specific antinuclear antigen that is pathognomonic for chronic ulcerative stomatitis. Treatment appears to be most effective with systemic hydoxychloroquine.[204-206] However, successful results have been reported using high-potency topical corticosteroids.[207]

LUPUS ERYTHEMATOSUS

Lupus erythematosus is an autoimmune disorder that may involve the oral cavity along with the skin and internal organs. Historically described as discoid or systemic forms, lupus erythematosus is now classified into the systemic form, a bullous form of systemic lupus erythematosus, a neonatal form, a chronic cutaneous form, and a subacute cutaneous form.[208-210] Lupus erythematosus is more common in women and blacks, and a genetic predisposition for the disease is apparent.[211]

The classic description of systemic lupus erythematosus includes chronic fever, weight loss, symptoms of arthritis, a malar or butterfly rash, effusion, and glomerulonephritis. Oral lesions are present in up to 40% of patients. Other skin conditions may be present, including discoid plaques on the face and scalp, alopecia, and vesiculobullous lesions.

Oral lesions are characterized by the presence of a central erythematous erosion or ulceration surrounded by a white rim with radiating keratotic striae. The most frequent sites of involvement are the hard and soft palate, buccal mucosa, and the vermillion border of the lips. The gingiva may take on a desquamative appearance, and patients may complain of burning or soreness. Other mucosal surfaces may also be affected including the oropharyngeal mucosa, nares, larynx, and epiglottis.[212-216]

Histologic findings suggestive of lupus erythematosus include keratinocyte vacuolization, subepithelial PAS-positive deposits, lamina propria edema, PAS-positive thickening of vascular basement membranes, and a severe or perivascular lymphocytic infiltrate.[217] Similar histopathologic features may be associated with lichen planus, and a lupus/lichen planus overlap syndrome has been described.[67-70]

Direct immunofluorescence testing reveals immunoreactants at the basement membrane zone with granular deposits of IgM, IgG, IgA, C3, and fibrinogen as well as the occasional presence of cytoid bodies.[6,218]

Oral and skin lesions respond to topical and intralesional corticosteroids with variable results. Systemic corticosteroids alone or in combination with other immunosuppressive agents such as cyclophosphamide may be useful in severe cases. Antimalarial drugs may produce satisfactory control, and topical or systemic retinoids may be beneficial. Gold salts and cyclosporin have also been used successfully in the treatment of lupus erythematosus.[219]

SUMMARY

The oral mucosa may be affected by a variety of mucocutaneous diseases. The erosive gingival lesions associated with vesiculobullous diseases such as lichen planus, mucous membrane pemphigoid, and pemphigus vulgaris have been collectively referred to as desquamative gingivitis. It must be remembered that other less common mucocutaneous conditions also affect the oral mucosa, including lupus erythematosus, bullous pemphigoid, epidermolysis bullosa acquisita, and linear IgA disease.

Adequate treatment is predicated on establishing the correct diagnosis and eliminating potential etiologic factors. While biopsy specimens and histologic examination including immunofluoresence tests are essential in arriving at a definitive diagnosis, the clinical appearance and history of the lesions provide very significant information. This paper has reviewed the features of common mucocutaneous diseases that have the ability to induce intraoral lesions.

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REFERENCES

1. Holmstrup P. Non-plaque induced gingival lesions. *Ann Periodontol* 1999;4:20-31.
2. Nisengard RJ. Periodontal implications: Mucocutaneous disorders. *Ann Periodontol* 1996;1:401-438.
3. Nisengard RJ, Rogers III RS. The treatment of desquamative gingival lesions. *J Periodontol* 1987;58:167-172.
4. Rogers RS, Sheridan PJ, Jordon RE. Desquamative gingivitis. Clinical, histopathologic and immunopathologic investigations. *Oral Surg Oral Med Oral Pathol* 1976;42:316-327.
5. Rogers RS, Sheridan PJ, Nightingale SH. Desquamative gingivitis: Clinical, histopathologic, immunopathologic, and therapeutic observations. *J Am Acad Dermatol* 1982; 7:729-735.
6. Helander SD, Rogers RS. The sensitivity and specificity and direct immunofluoresence testing in disorders of mucous membranes. *J Am Acad Dermatol* 1994;30: 65-75.
7. Chaiyarit P, Kafrawy AH, Miles DA, Zunt SL, Van Dis ML, Gregory RL. Oral lichen planus: An immunohistochemical study of heat shock proteins (HSPs) and cytokeratins (CKs) and a unifying hypothesis of pathogenesis. *J Oral Pathol Med* 1999;28:210-215.
8. Hakkinen L, Kainulainen T, Salo T, Grenman R, Larjava H. Expression of integrin alpha9 subunit and tenascin in oral leukoplakia, lichen planus, and squamous cell carcinoma. *Oral Dis* 1999;5:210-217.
9. Simpson HE. The age and sex incidence and anatomical distribution of oral leukoplakia and lichen planus. *Br J Dermatol* 1957;69:178-180.
10. Mollaoglu N. Oral lichen planus: A review. *Br J Oral Maxillofac Surg* 2000;38:370-377.
11. Nanda A, Al-Ajmi HS, Al-Sabah H, Al-Hasawi F, Alsaleh QA. Childhood lichen planus: A report of 23 cases. *Pediatr Dermatol* 2001;18:1-4.
12. Alam F, Hamburger J. Oral mucosal lichen planus in children. *Int J Paediatr Dent* 2001;11:209-214.
13. Axell T, Rundquist L. Oral lichen planus–A demographic study. *Community Dent Oral Epidemiol* 1987;15:52-56.
14. Arndt KA. Lichen planus. In: Fitzpatrick TB, ed. *Dermatology in General Medicine*. New York: McGraw Hill Book Co; 1979.
15. Conklin RJ, Blasberg B. Oral lichen planus. *Dermatol Clin* 1987;5:663-673.
16. Thorn JJ, Holstrup P, Rindum H, Pindborg JJ. Course of various clinical forms of oral lichen planus. A prospective follow-up study of 611 patients. *J Oral Pathol Med* 1988;17:213-318.
17. Silverman S Jr., Bahl S. Oral lichen planus update: Clinical characteristics, treatment responses and malignant transformation. *Am J Dent* 1997;10:259-263.
18. Eisen D. The evaluation of cutaneous, genital, scalp, nail, esophageal, and ocular involvement in patients with oral lichen planus. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1999;88:431-436.
19. Abraham SC, Ravich WJ, Anhalt GJ, Yardley JH, Wu TT. Esophageal lichen planus: Case report and review of the literature. *Am J Surg Pathol* 2000;24:1678-1682.
20. Eisen D. The vulvovaginal-gingival syndrome of lichen planus. The clinical characteristics of 22 patients. *Arch Dermatol* 1994;130:1379-1382.
21. Cribier B, Ndiaye I, Grosshans E. Peno-gingival syndrome. A male equivalent of vulvo-vaginal-gingival syndrome? *Rev Stomatol Chir Maxillofac* 1993;94: 48-51.
22. Walker DM. The inflammatory infiltrate in lichen planus lesions. An autoradiographic and ultrastructural study. *J Oral Pathol Med* 1976;5:277-286.

23. Dockrell HM, Greenspan JS. Histochemical identification of T cells in oral lichen planus. *Oral Surg Oral Med Oral Pathol* 1979;48:42-46.
24. Regezi JA, Deegan MJ, Hayward JR. Lichen planus: Immunologic and morphologic identification of the submucosal infiltrate. *Oral Surg Oral Med Oral Pathol* 1978;46:44-52.
25. Yih WY, Maier T, Kratochvil FJ, Zieper MB. Analysis of desquamative gingivitis using direct immunofluorescence in conjunction with histology. *J Periodontol* 1998;69:678-685.
26. Raghu AR, Rao NN. Immunofluorescence in oral lichen planus and oral lichenoid reaction. A review. *Indian J Dent Res* 2001;12:29-34.
27. Nieboer C. The reliability of immunofluorescence and histopathology in the diagnosis of discoid lupus erythematosus and lichen planus. *Br J Dermatol* 1987;116: 189-198.
28. Kilpi AM, Rich AM, Radden BG, Reade PC. Direct immunofluorescence in the diagnosis of oral mucosal disease. *Int J Oral Maxillofac Surg* 1988;17:6-10.
29. Schiodt M, Holmstrup P, Dabelsteen E, Ullman S. Deposits of immunoglobulins, complement, and fibrinogen in oral lupus erythematosus, lichen planus, and leukoplakia. *Oral Surg Oral Med Oral Pathol* 1981;51: 603-608.
30. Thornhill MH. Immune mechanisms in oral lichen planus. *Acta Odontol Scand* 2001;59:174-177.
31. Silverman S Jr., Eversole LR. Immunopathologic mucosal lesions. In: Silverman S Jr., Eversole LR, Truelove EI, eds. *Essentials of Oral Medicine*. Hamilton, ON: B.C. Decker Inc.; 2001.
32. Little MC, Griffiths CEM, Watson REB, Pemberton M, Thornhill MH. Activation or oral keratinocytes by mercuric chloride: Relevance to dental amalgam-induced oral lichenoid reactions. *Br J Dermatol* 2001;144:1024-1032.
33. Wright JM. Oral manifestations of drug reactions. *Dent Clin North Am* 1984;28:529-543.
34. Kilpi AM, Rich AM, Radden BG, Reade PC. Direct immunofluorescence in the diagnosis of oral mucosal diseases. *Int J Oral Maxillofac Surg* 1988;17:6-10.
35. Eversole LR, Ringer M. The role of dental restorative metals in the pathogenesis of oral lichen planus. *Oral Surg Oral Med Oral Pathol* 1984;57:383-387.
36. Bolewska J, Hansen HJ, Holmstrup P, Pindborg JJ, Stangerup M. Oral mucosal lesions related to silver amalgam restorations. *Oral Surg Oral Med Oral Pathol* 1990;70:55-58.
37. Bolewska J, Holmstrup P, Moller-Madsen B, Kenrad B, Danscher G. Amalgam associated mercury accumulations in normal oral mucosa, oral mucosal lesions of lichen planus and contact lesions associated with amalgam. *J Oral Pathol Med* 1990;15:39-42.
38. Bratel J, Hakelberg M, Jontell M. Effect of replacement of dental amalgam on oral lichenoid reactions. *J Dent* 1996;24:41-45.
39. Henricksson E, Mattsson U, Hakamsson J. Healing of lichenoid reactions following removal of amalgam. A clinical follow-up. *J Clin Periodontol* 1995;22:287-294.
40. Holmstrup P. Oral mucosa and skin reactions related to amalgam. *Adv Dent Res* 1992;6:120-124.
41. James J, Ferguson MM, Forsyth A, Tulloch N, Lamey PJ. Oral lichenoid reactions related to mercury sensitivity. *Br J Oral Maxillofac Surg* 1987;25:474-480.
42. Jameson MW, Kardos TB, Kirk EE, Ferguson MM. Mucosal reactions to amalgam restorations. *J Oral Rehab* 1990;17:293-301.
43. Lind PO. Oral lichenoid reactions related to composite restorations. Preliminary report. *Acta Odontol Scand* 1988;45:53-55.
44. Lundstrum IM. Allergy and corrosion of dental materials in patients with oral lichen planus. *Int J Oral Surg* 1984;13:16-24.
45. Zhu YX. Clinical and histologic analysis of mucous membrane lesions associated with dental restorations. *Chin J Stomatol* 1989;24:49-53.
46. Allen CM, Blozis GG. Oral mucosal reactions to cinnamin-flavored chewing gum. *J Am Dent Assoc* 1988; 116:664-667.
47. Maibach HI. Cheilitis: Occult allergy to cinnamic aldehyde. *Contact Dermatitis* 1986;15:106-107.
48. Miller RL, Gould AR, Bernstein ML. Cinnamon-induced stomatitis venenata: Clinical and characteristic histopathologic features. *Oral Surg Oral Med Oral Pathol* 1992;73:708-716.
49. Katta R. Lichen planus. *Am Fam Physician* 2000;61: 3319-3324,3327-3328.
50. Scalf LA, Fowler JF Jr., Morgan KW, Looney SW. Dental metal allergy in patients with oral, cutaneous, and genital lichenoid reactions. *Am J Contact Dermat* 2001; 12:146-150.
51. Kirtak N, Inaloz HS, Ozgoztasi, EZ. The prevalence of hepatitis C virus infection in patients with lichen planus in Gaziantep region of Turkey. *Eur J Epidemiol* 2000;16: 1159-1161.
52. Ayala F, Balato N, Tranfaglia A, Guadagnino V, Orlando R. Oral erosive lichen planus and chronic liver disease. *J Am Acad Dermatol* 1986;14:139-140.
53. Carrozzo M, Gandolfo S, Carbone M, et al. Hepatitis C virus infection in Italian patients with oral lichen planus: A prospective case-control study. *J Oral Pathol Med* 1996; 25:527-533.
54. Gordon SC. Extrahepatic manifestations of hepatitis C. *Digest Dis* 1996;14:157-168.
55. Halevy S, Ingber A, Sandbank M. The role of abnormal glucose tolerance, human lymphocyte antigen (HLA) typing, and urolithiasis in lichen planus. *J Am Acad Dermatol* 1985;13:134.
56. Katz M, Pisanti S. Oral erosive lichen planus and chronic active hepatitis. *J Am Acad Dermatol* 1985;12:719.
57. Lozada-Nur F, Luangjarmekorn L, Silverman S, Karam J. Assessment of plasma glucose in 99 patients with oral lichen planus. *J Oral Med* 1985;40:60-61.
58. Mobacken H, Nilsson LA, Olsson R, Sloberg K. Incidence of liver disease in chronic lichen planus of the mouth. *Acta Dermatol Venereol* 1984;64:70-73.
59. Pawlotsky JM, Dhumeaux D, Bagot M. Hepatitis C virus in dermatology. A review. *Arch Dermatol* 1995;131: 1185-1193.
60. Scully C, Potts AJC, Hamburger J, Wiesenfuld D, McKee JI, El Kom M. Lichen planus and liver disease: How strong is the association? *J Oral Pathol* 1985;14:224-226.

61. Lunel F, Cacoub P. Treatment of autoimmune and extrahepatic manifestations of HCV infection. *Ann Med Interne* (Paris) 2000;151:58-64.
62. Varela P, Areias J, Mota F, Canelhas A, Sanches M. Oral lichen planus induced by interferon-alpha-N1 in a patient with hepatitis C. *Int J Dermatol* 2000;39:239-240.
63. Lodi G, Porter SR, Scully C. Hepatitis C virus infection. Review and implications for the dentist. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1998;86:8-22.
64. Bagan JV, Ramon C, Gonzalez L, et al. Preliminary investigation of the association of oral lichen planus and hepatitis C. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1998;85:532-536.
65. Mattsson, T, Sundqvist, KG, Heimdahl, A, Dahllof G, Ljungman P, Ringden O. A comparative immunological analysis of oral mucosa in chronic graft-versus-host disease and oral lichen planus. *Arch Oral Biol* 1992;37:539-547.
66. Schubert MM, Sullivan KM. Recognition, incidence, and management of oral graft-versus-host disease. *NCI Monogr* 1990;9:135-143.
67. Camisa C. Lupus erythematosus/lichen planus overlap syndrome. *J Am Acad Dermatol* 1984;12:297.
68. Plotnick H, Burnham TK. Lichen planus and coexisting lupus erythematosus versus lichen planus-like lupus erythematosus. *J Am Acad Dermatol* 1986;14:931-938.
69. Schiodt M. Oral discoid lupus erythematosus. III. A histopathologic study of sixty-six patients. *Oral Surg Oral Med Oral Pathol* 1984;57:281-293.
70. Schiodt M, Pindborg JJ. Oral discoid lupus erythematosus. I. The validity of previous histopathologic diagnostic criteria. *Oral Surg Oral Med Oral Pathol* 1984;57:46-51.
71. Pagliaro JA, White S, Strutton G, Guerin D. Lichen planus-like eruption following autologous bone marrow transplantation for chronic myeloid leukaemia. *Australas J Dermatol* 2001;42:188-191.
72. Nicolatou-Galitis O, Kitra V, Van Vliet-Constantinidou C, et al. The oral manifestations of chronic graft-versus-host disease (cGVHD) in paediatric allogenic bone marrow transplant. *J Oral Pathol Med* 2001;30:148-153.
73. Cawson RA. Management of oral lichen planus. In: McDonald RE, Hurt WC, Gilmore HW, Middleton RA, eds. *Current Therapy in Dentistry*, vol. 7. St. Louis: The C.V. Mosby Company; 1980:63-68.
74. Balciunas BA, Overholser CD. Diagnosis and treatment of common oral lesions. *Am Fam Physician* 1987;35:206-220.
75. Pedersen A, Klausen B. Glucocorticosteroids and oral medicine. *J Oral Pathol* 1984;13:1-15.
76. Hurt WC. Pharmacologic management of stomatologic problems. *Dent Clin North Am* 1984;28:545-554.
77. Lamey PJ, Boyle MA, Simpson NB, Ferguson MM. A pilot study of griseofulvin therapy in erosive oral lichen planus. *J Oral Med* 1987;42:233-235.
78. Lozada F. Prednisone and azathioprine in the treatment of oral inflammatory mucocutaneous diseases. *Oral Surg Oral Med Oral Pathol* 1981;52:257-260
79. Rozycki TW, Rogers RS, Pittelkow MR, et al. Topical tacrolimus in the treatment of symptomatic oral lichen planus: A series of 13 patients. *J Am Acad Dermatol* 2002;46:27-34.
80. Vente C, Reich K, Rupprecht R, Neumann C. Erosive mucosal lichen planus: Response to topical treatment with tacrolimus. *Br J Dermatol* 1999;140:338-342.
81. Kalrakatsou F, Hodgson TA, Lewsey, JD, et al. Management of recalcitrant ulcerative oral lichen planus with topical tacrolimus. *J Am Acad Dermatol* 2002;46:35-41.
82. Lener EV, Brieva J, Schachter M, West LE, West DP, el-Azhary RA. Successful treatment of erosive lichen planus with topical tacrolimus. *Arch Dermatol* 2001;137:419-422.
83. Skaehill PA. Tacrolimus in dermatologic disorders. *Ann Pharmacother* 2001;35:582-588.
84. Aufdemorte TB, de Villez RL, Gieseker DR. Griseofulvin in the treatment of three cases of oral erosive lichen planus. *Oral Surg Oral Med Oral Pathol* 1983;55:459-462.
85. Ho VC, Gupta AK, Ellis BJ, Nickoloff BJ, Voorhees JJ. Treatment of severe lichen planus with cyclosporine. *J Am Acad Dermatol* 1990;22:64-68.
86. Frances C, Boisnic S, Etienne S, Szpirglas J. Effect of the local application of cyclosporin A on chronic erosive lichen planus of the oral cavity. *Dermatologica* 1988;177:194-195.
87. Ferguson MM, Simpson NB, Hammersley N. The treatment of erosive lichen planus with a retinoid–etretinate. *Oral Surg Oral Med Oral Pathol* 1984;58:283-287.
88. Chan ES, Thornhill M, Azkrzewska J. Interventions for treating oral lichen planus. *Cochrane Database Syst Rev* 2000;(2):CD001168.
89. Lu SY, Chen WJ, Eng HL. Response to levamisole and low dose prednisolone in 41 patients with chronic oral ulcers: A 3 year open clinical trial and follow-up study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1998;86:438-445.
90. Buyuk AY, Kavala M. Oral metronidazole treatment of lichen planus. *J Am Acad Dermatol* 2000;43:260-262.
91. Hodak E, Yosipovitch G, David M, et al. Low-dose low-molecular weight heparin (enoxaparin) is beneficial in lichen planus: A preliminary report. *J Am Acad Dermatol* 1998;38:564-568.
92. Stefanidou MP, Ioannidou DF, Panayiotides JG, Tosca AD. Low molecular weight heparin: A novel alternative therapeutic approach for lichen planus. *Br J Dermatol* 1999;141:1040-1045.
93. Nasr IS. Topical tacrolimus in dermatology. *Clin Exp Dermatol* 2000;25:250-254.
94. Camisa C, Popovsky JL. Effective treatment of oral erosive lichen planus with thalidomide. *Arch Dermatol* 2000;136:1442-1443.
95. van der Meij EH, Schepman KP, Smeele LE, van der Wal JE, Bezemer PD, van der Waal I. A review of the recent literature regarding malignant transformation of oral lichen planus. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1999;88:307-310.
96. Lozada-Nur F. Oral lichen planus and oral cancer: Is there enough epidemiologic evidence? *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2000;89:265-266.
97. Silverman S Jr. Oral lichen planus: A potentially premalignant lesion. *J Oral Maxillofac Surg* 2000;58:1286-1288.
98. Eisen D. The clinical features, malignant potential, and systemic associations of oral lichen planus: A study of 723 patients. *J Am Acad Dermatol* 2002;46:207-214.

99. Silverman S, Gorsky M, Lozada-Nur F. A prospective follow-up study of 570 patients with oral lichen planus: Resistance, remission, and malignant association. *Oral Surg Oral Med Oral Pathol* 1985;60:30-34.
100. Murti PR, Daftary DK, Bhonsle RB, Gupta PC, Mehta FS, Pindborg JJ. Malignant potential of oral lichen planus: Observations in 722 patients from India. *J Oral Pathol Med* 1986;15:71-77.
101. Holmstrup P, Thorn JJ, Rindum J, Pindborg JJ. Malignant development of lichen planus-affected oral mucosa. *J Oral Pathol* 1988;17:219-225.
102. Salem G. Oral lichen planus among 4277 patients from Gizan, Saudi Arabia. *Community Dent Oral Epidemiol* 1989;17:322-324.
103. Sigurgeirsson B, Lindelof B. Lichen planus and malignancy: An epidemiologic study of 2071 patients and a review of the literature. *Arch Dermatol* 1991;127: 1684-1688.
104. Mignogna MD, Lo Muzio L, Lo Russo L, Fedele S, Ruoppo E, Bucci E. Clinical guidelines in early detection of oral squamous cell carcinoma arising in oral lichen planus: A 5-year experience. *Oral Oncol* 2001; 37:262-267.
105. Kim J, Yook JI, Lee EH, et al. Evaluation of premalignant potential in oral lichen planus using interphase cytogenetics. *J Oral Pathol Med* 2001;30:65-72.
106. Eisenberg E, Krutchkoff DJ. Lichenoid lesions of oral mucosa: Diagnostic criteria and their importance in the alleged relationship to oral cancer. *Oral Surg Oral Med Oral Pathol* 1992;73:699-704.
107. Drangsholt M, Truelove EL, Morton TH Jr, Epstein JB. A man with a 30 year history of oral lesions. *J Evid Base Dent Pract* 2001;2:123-135.
108. Cole WC, Leicht S, Byrd RP Jr, Roy TM. Cicatricial pemphigoid with an upper airway lesion. *Tenn Med* 2000;93:99-101.
109. Stallmach A, Weg-Remers S, Moser C, Bonkoff H, Feifel G, Zeitz M. Esophageal involvement in cicatricial pemphigoid. *Endoscopy* 1998;30:657-661.
110. Farrell AM, Kirtschig G, Dalziel KL, et al. Childhood vulval pemphigoid: A clinical and immunopathological study of five patients. *Br J Dermatol* 1999;140: 308-312.
111. Foster CS, Ahmed AR. Intravenous immunoglobulin therapy for ocular cicatricial pemphigoid: A preliminary study. *Ophthalmology* 1999;106:2136-2143.
112. Sallout H, Anhalt GJ, Al-Kawas FH. Mucous membrane pemphigoid presenting with isolated esophageal involvement: A case report. *Gastrointest Endosc* 2000;52:429-433.
113. Ramlogan D, Coulsom IH, McGeorge A. Cicatricial pemphigoid: A diagnostic problem for the urologist. *J Royal Coll Surg Edinb* 2000;45:62-63.
114. Cheng YS, Rees TD, Wright JM, Plemons JM. Childhood oral pemphigoid: A case report and review of the literature. *J Oral Pathol Med* 2001;30:372-377.
115. Hardy KM, Perry HO, Pingree GC, Kirby TJ. Benign mucous membrane pemphigoid. *Arch Dermatol* 1971; 104:467-475.
116. Shklar G, McCarthy PL. Oral lesions of mucous membrane pemphigoid. A study of 85 cases. *Arch Otolaryngol* 1971;3:354-364.
117. Scott JE, Ahmed AR. The blistering diseases. *Med Clin North Am* 1998;82:1239-1283.
118. Scully C, Carrozzo M, Gandolfo S, Puiatti P, Monteil R. Update on mucous membrane pemphigoid. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1999;88:56-68.
119. Silverman S Jr., Gorsky M, Lozada-Nur F, Liu A. Oral mucous membrane pemphigoid. A study of sixty-five patients. *Oral Surg Oral Med Oral Pathol* 1986;61: 233-237.
120. Holsclaw DS. Ocular cicatricial pemphigoid. *Int Ophthalmol Clin* 1998;38:89-106.
121. Messner EM, Hintschich CR, Partscht K, Messer G, Kampik A. Ocular cicatricial pemphigoid. Retrospective analysis of risk factors and complications. *Ophthalmologe* 2000;97:113-120.
122. Leonard JN, Wright P, Haffenden GP, Williams DM, Griffiths CEM, Fry L. Skin diseases and the dry eye. *Trans Ophthalmol Soc U K* 1985;104:467-476.
123. Baum J. Clinical manifestations of dry eye states. *Trans Ophthalmol Soc U K* 1985;104:415-423.
124. Lamey PJ, Rees TD, Binnie WH, Rankin KV. Mucous membrane pemphigoid–Treatment experience at two institutions. *Oral Surg Oral Med Oral Pathol* 1992;74: 50-53.
125. Gallagher G, Shklar G. Oral involvement in mucous membrane pemphigoid. *Clin Dermatol* 1987;5:18-27.
126. Shklar G, McCarthy PL. Oral lesions of mucous membrane pemphigoid: A study of 85 cases. *Arch Otolaryngol* 1971;93:354-364.
127. Terezhalmy GT, Bergfeld WF. Cicatricial pemphigoid (benign mucous membrane pemphigoid). *Quintessence Int* 1998;29:429-437.
128. Setterfield J, Theron J, Vaughan RW, et al. Mucous membrane pemphigoid: HLA-DQB1*0301 is associated with all clinical sites of involvement and may be linked to antibasement membrane IgG production. *Br J Dermatol* 2001;145:406-414.
129. Rogers RS III, Seehafer JR, Perry HO. Treatment of cicatricial (benign mucous membrane) pemphigoid with dapsone. *J Am Acad Dermatol* 1982;6:215-223.
130. Bean SF. Cicatricial pemphigoid. Immunofluorescent studies. *Arch Dermatol* 1974;110:552-555.
131. Brody HA, Wuepper KA. Mucous membrane pemphigoid–Immunofluorescent studies. *J Dent Res* 1969; 48:1248-1250.
132. Laskaris G, Demetrios N, Angelopoulos A. Immunofluorescent studies in desquamative gingivitis. *J Oral Pathol* 1981;10:398-407.
133. Laskaris G, Angelopoulos A. Cicatricial pemphigoid: Direct and indirect immunofluorescent studies. *Oral Surg Oral Med Oral Pathol* 1981;51:48-52.
134. Laskaris G, Demetrios N, Angelopoulos A. Immunofluorescent studies in desquamative gingivitis. *J Oral Pathol* 1981;10:398-407.
135. Korman NJ. New immunomodulating drugs in autoimmune blistering diseases. *Dermatol Clin* 2001;19: 637-648. viii
136. Mallet L, Cooper JW, Thomas J. Bullous pemphigoid associated with captopril. *DICP* 1989;23:63.
137. Ingber A, Grunwald MH, Feuerman EJ. Carbamazepine-induced bullous eruption or bullous pemphigoid? *Postgrad Med J* 1984;60:307.

138. Van Joost T, Faber WR, Manuel HR. Drug-induced anogenital cicatricial pemphigoid. *Br J Dermatol* 1980; 102:715-718.
139. Panayiotou BN, Prasad MV, Zaman MN. Furosemide-induced bullous pemphigoid. *Br J Clin Pract* 1997;51: 49-50.
140. Koch CA, Mazzaferri EL, Larry JA, Fanning TS. Bullous pemphigoid after treatment with furosemide. *Cutis* 1996; 58:340-344.
141. Weller R, White MI. Penicillamine in the etiology of bullous pemphigoid. *Ann Pharmacother* 1998;32:1368.
142. Bialy-Golan A, Brenner S. Penicillamine-induced bullous dermatoses. *J Am Acad Dermatol* 1996;35: 732-742.
143. Weller R, White MI. Bullous pemphigoid and penicillamine. *Clin Exp Dermatol* 1996;21:121-122.
144. Joost TV, Crone RA, Overjijk AD. Ocular cicatricial pemphigoid associated with practolol therapy. *Br J Dermatol* 1976;94:447-450.
145. Miralles J, Barnadas MA, Baselga E, Gelpi C, Rodriquez JL, de Moragas JM. Bullous pemphigoid-like lesions induced by amoxicillin. *Int J Dermatol* 1997;36:42-47.
146. Fujimoto W, Ishida-Yamamoto A, Hsu R, et al. Anti-epiligrin cicatricial pemphigoid: A case associated with gastric carcinoma and features resembling epidermolysis bullosa acquisita. *Br J Dermatol* 1998;139:682-687.
147. Setterfield J, Shirlaw PJ, Lazarova Z, et al. Paraneoplastic cicatricial pemphigoid. *Br J Dermatol* 1999;141: 127-131.
148. Ciarrocca KN, Greenberg MS. A retrospective study of the management of oral mucous membrane pemphigoid with dapsone. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1999;88:159-163.
149. Rogers RS, Mehregan DA. Dapsone therapy of cicatricial pemphigoid. *Semin Dermatol* 1988;7:201-205.
150. Lever WF. Pemphigus and pemphigoid. A review of the advances made since 1964. *Am J Dermatopathol* 1979;1:2-32.
151. Gorlin RJ. Vesiculo-bullous lesions. *Gerodontics* 1985; 1:105-107.
152. Lu SY, Chen WJ, Eng HL. Response to levamisole and low-dose prednisolone in 41 patients with chronic oral ulcers: A 3-year open clinical trial and follow-up study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1998;86:438-445.
153. Thornfeldt CR, Menkes AW. Bullous pemphigoid controlled by tetracycline. *J Am Acad Dermatol* 1987;16: 305-310.
154. Kreyden OP, Borradori L, Trueb RM, Burg G, Nestle FO. Successful therapy with tetracycline and nicotinamide in cicatricial pemphigoid. *Hautarzt* 2001;52: 247-250.
155. Reiche L, Wojnarowska F, Mallon E. Combination therapy with nicotinamide and tetracyclines for cicatricial pemphigoid: Further support for its efficacy. *Clin Exp Dermatol* 1998;23:254-257.
156. Damoulis PD, Gagari E. Combined treatment of periodontal disease and benign mucous membrane pemphigoid. Case report with 8 years maintenance. *J Periodontol* 2000;71:1620-1629.
157. Korman N. Pemphigus. *J Am Dermatopathol* 1988;18: 1219-1238.
158. Orlowski WA, Bressman E, Doyle JL, Chasens AI. Chronic pemphigus vulgaris of the gingiva. A case report with a 6-year follow-up. *J Periodontol* 1983;54: 685-689.
159. Markitziu A, Pisanty S. Gingival pemphigus vulgaris. Report of a case. *Oral Surg Oral Med Oral Pathol* 1983;55:250-253.
160. Mashkilleyson N, Mashkilleyson AL. Mucous membrane manifestations of pemphigus vulgaris. *Acta Derm Venereol* 1988;68:413-421.
161. Sirois D, Leigh JE, Sollecito TP. Oral pemphigus vulgaris preceding cutaneous lesions: Recognition and diagnosis. *J Am Dent Assoc* 2000;131:1156-1160.
162. Mignogna MD, Lo Muzio L, Bucci E. Clinical features of gingival pemphigus vulgaris. *J Clin Periodontol* 2001;28:489-493.
163. Coscia-Porrazzi L, Maiello FM, Ruocco V, Pisani M. Cytodiagnosis of oral pemphigus vulgaris. *J Acta Cytol* 1985;29:746-749.
164. Stanley JR, Yaar M, Hawley-Nelson P, Katz SI. Pemphigus antibodies identify a cell surface glycoprotein synthesized by human and mouse keratinocytes. *J Clin Invest* 1982;70:281-288.
165. Marinho RT, Johnson NW, Fatela NM, et al. Oropharyngeal pemphigus in a patient with chronic hepatitis C during interferon alpha-2a therapy. *Eur J Gastroenterol Hepatol* 2001;13:869-872.
166. Hsiao CJ, Hsu MM, Lee JY, Chen WC, Hsieh WC. Paraneoplastic pemphigus in association with a retroperitoneal Castleman's disease presenting with a lichen planus pemphigoides-like eruption. A care report and review of the literature. *Br J Dermatol* 2001;144: 372-376.
167. Allen CM, Camisa C. Paraneoplastic pemphigus: A review of the literature. *Oral Dis* 2000;6:208-214.
168. van der Waal RI, Pas HH, Nousari HC, et al. Paraneoplastic pemphigus caused by an epitheloid leiomyosarcoma and associated with fatal respiratory failure. *Oral Oncol* 2000;36:390-393.
169. Correll AW, Schott TR. Multiple, painful vesiculoulcerative lesions in the oral mucosa. *J Am Dent Assoc* 1985;110:765-766.
170. Ferguson CD, Taybos GM. Diagnosis and treatment of pemphigus. *Quintessence Int* 1985;7:473-476.
171. Lamey PJ, Rees TD, Wright JM, Simpson NB. Oral presentation of pemphigus vulgaris and its response to systemic steroid therapy. *Oral Surg Oral Med Oral Pathol* 1992;74:54-57.
172. Mignogna MD, Lo Muzio L, Mignogna RE, Carbone R, Ruoppo E, Bucci E. Oral pemphigus: Long-term behavior and clinical response to treatment with deflazacort in sixteen cases. *J Oral Pathol Med* 2000;29: 145-152.
173. Basset N, Guillot B, Michel B, Meynadier J, Guilhou JJ. Dapsone as initial treatment in superficial pemphigus. Report of nine cases. *Arch Dermatol* 1987;123: 783-785.
174. Page EH, Wexler DM, Guenther LC. Cyclosporin A. *J Am Dermatopathol* 1986;14:785-791.
175. Zhu JF, Kaminski MJ, Pulitzer DR, Hu J, Thomas HF. Psoriasis: Pathophysiology and oral manifestations. *Oral Dis* 1996;2:135-144.

176. Richardson LJ, Kratochvil FJ, Zieper MB. Unusual palatal presentation of oral psoriasis. *J Can Dent Assoc* 2000;66:80-82.
177. Matarasso S, Vaia E, Fusco A, Riccitiello F, Nicolo M. Desquamative gingivitis: Etiopathogenetic and clinical assessment. *Minerva Stomatol* 1989;38:359-368.
178. Younai FS, Phelan JA. Oral mucositis with features of psoriasis: Report of a case and review of the literature. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1997;84:61-67.
179. Zunt SL, Tomich CE. Eythema migrans–A psoriaform lesion of the oral mucosa. *J Dermatol Surg Oncol* 1989; 15:1067-1070.
180. Brice DM, Danesh-Meyer MJ. Oral lesions in patients with psoriasis: Clinical presentation and mangement. *J Periodontol* 2000;71:1896-1903.
181. Yamada J, Amar S, Petrungaro P. Psoriasis-associated periodontitis: A case report. *J Periodontol* 1992;63: 854-857.
182. Pogrel MA, Cram D. Intraoral findings in patients with psoriasis with a special reference to ectopic geographic tongue (erythema circinata). *Oral Surg Oral Med Oral Pathol* 1989;66:184-189.
183. Femiano F. Geographic tongue (migrant glossitis) and psoriasis. *Minerva Stomatol* 2001;50:213-217.
184. Morris LF, Phillips CM, Binnie WH, Sander HM, Silverman AK, Mento MA. Oral lesions in patients with psoriasis: A controlled study. *Cutis* 1992;49:339-344.
185. Espelid M, Bang G, Johannessen AC, Leira JI, Christensen O. Geographic stomatitis: Report of 6 cases. *J Oral Pathol Med* 1991;20:425-428.
186. Ulmansky M, Michelle R, Azaz B. Oral psoriasis: Report of six new cases. *J Oral Pathol Med* 1995;24:42-45.
187. Koo JY. Current consensus and update on psoriasis therapy: A perspective from the U.S. *J Dermatol* 1999;26:723-733.
188. Geilen CC, Arnold M, Orfanos CE. Mycophenolate mofetil as a systemic antipsoriatic agent: Positive experience in 11 patients. *Br J Dermatol* 2001;144: 583-586.
189. Eisen D, Essell J, Broun ER. Oral cavity complications of bone marrow transplantation. *Semin Cutan Med Surg* 1997;16:265-272.
190. Hiroki A, Nakamura S, Shinohara M, Oka M. Significance of oral examination in chronic graft-versus-host disease. *J Oral Pathol Med* 1994;23:209-215.
191. Nakamura S, Hiroki A, Shinohara M, et al. Oral involvement in chronic graft-versus-host disease after allogeneic bone marrow transplantation. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996;82:556-563.
192. Eggleston TI, Ziccardi VB, Lumerman H. Graft-versus-host disease. Case report and discussion. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1998; 86:692-696.
193. Holmstrup P. Non-plaque-induced gingival lesions. *Ann Periodontol* 1999;4:20-31.
194. Majorana A, Schubert MM, Porta F, Ugazio AG, Sapelli PL. Oral complications of pediatric hematopoietic cell transplantation: Diagnosis and management. *Support Care Cancer* 2000;8:353-365.
195. McCartan BE, McCreary CE. Oral lichenoid drug eruptions. *Oral Dis* 1997;3:58-63.
196. Curtis JW Jr., Caughman GB. An apparent unusual relationship between rampant caries and oral mucosal manifestations of chronic graft-versus-host disease. *Oral Surg Oral Med Oral Pathol* 1994;78: 267-272.
197. Epstein JB, Reece DE. Topical cyclosporin A for treatment of oral chronic graft-versus-host disease. *Bone Marrow Transplant* 1994;13:81-86.
198. Epstein JB, Nantel S, Sheoltch SM. Topical azathioprine in the combined treatment of chronic oral graft-versus-host disease. *Bone Marrow Transplant* 2000; 25:683-687.
199. Epstein JB, Gorsky M, Epstein MS, Nantel S. Topical azathioprine in the treatment of immune-mediated chronic oral inflammatory conditions: A series of cases. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2001;91:56-61.
200. Jaremko WM, Beutner EH, Kumar V, et al. Chronic ulcerative stomatitis associated with a specific immunologic marker. *J Am Acad Dermatol* 1990;22: 215-220.
201. Chlorzelski TP. Chronic ulcerative stomatits (CUS): A new disease entity with a specific immunological marker (SES-ANA) (in Polish). *Przegl Dermatol* 1990; 77:229-232.
202. Beutner EH, Chorzelski TP, Parodi A, et al. Ten cases of chronic ulcerative stomatitis with stratified epithelium-specific antinuclear antibody. *J Am Acad Dermatol* 1991;24:781-782.
203. Church LF Jr., Schosser RH. Chronic ulcerative stomatitis associated with stratified epithelium specific antinuclear antibody. A case report of a newly described disease entity. *Oral Surg Oral Med Oral Pathol* 1992; 73:579-582.
204. Lewis JE, Beutner EH, Rostami R, Chorzelski TP. Chronic ulcerative stomatitis with stratified epithelium-specific antinuclear antibodies. *Int J Dermatol* 1996;35: 272-275.
205. Worle B, Wollenberg A, Schaller M, Kunzelmann KH, Plewig G, Meurer M. Chronic ulcerative stomatitis. *Br J Dermatol* 1997;137:262-265.
206. Chorzelski TP, Olszewska M, Jarzabek-Chorzelski M, Jablonska S. Is chronic ulcerative stomatitis an entity? Clinical and immunological findings in 18 cases. *Eur J Dermatol* 1998;8:261-265.
207. Lorenzana ER, Rees TD, Glass M, Detweiler JG. Chronic ulcerative stomatitis: A case report. *J Periodontol* 2000;71:104-111.
208. Gammon WR, Briggaman RA. Bullous SLE: A phenotypically distinctive but immunologically heterogeneous bullous disorder. *J Invest Dermatol* 1993;100: 28-34.
209. Ginzler EM, Antoniadis I. Clinical manifestations of systemic lupus erythematosus, measures of disease activity, and long-term complications. *Curr Opin Rheumatol* 1992;4:672-680.
210. Lee LA. Neonatal lupus erythematosus. *J Invest Dermatol* 1993;100:9-13.
211. Condemi JJ. The autoimmune diseases. *JAMA* 1987; 258:2920-2929.
212. McCauliffe DP. Cutaneous lupus erythematosus. *Semin Cutan Med Surg* 2001;20:14-26.

213. Meyer U, Kleinheinz J, Handschel J, Kruse-Losler B, Weingart D, Joos U. Oral findings in three different groups of immunocompromised patients. *J Oral Pathol Med* 2000;29:153-158.
214. Meyer U, Kleinheinz J, Gaubitz M, Schulz M, Weingart D, Joos U. Oral manifestations in patients with systemic lupus erythematosus. *Mund Kiefer Gesichtschir* 1997;1:90-94.
215. Jorizzo JL, Salisbury PL, Rogers RS, et al. Oral lesions in systemic lupus erythematosus. Do ulcerative lesions represent a necrotizing vasculitis? *J Am Acad Dermatol* 1992;27:389-394.
216. Burge SM, Frith PA, Juniper RP, Wojnarowska F. Mucosal involvement in systemic and chronic cutaneous lupus erythematosus. *Br J Dermatol* 1989;121: 727-741.
217. Bonaccorso A, Tripi TR. Oral lesions in systemic lupus erythematosus. I. The etiopathogenic aspects of lupus erythematosus. *Minerva Stomatol* 1998;47: 27-31.
218. Reibel J, Schiodt M. Immunohistochemical studies on colloid bodies (Civatte bodies) in oral lesions of discoid lupus erythematosus. *Scand J Dent Res* 1986;94: 536-544.
219. Heule F, van Joost T, Beukers R. Cyclosporine in the treatment of lupus erythematosus. *Arch Dermatol* 1986;122:973-974.