Oral Manifestations of Cutaneous Disease

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The list of diseases which manifest both oral and cutaneous pathology is extensive and consideration of them all is beyond the scope of a single brief report. Nevertheless, it may be useful to summarize the findings of those diseases in which both oral and cutaneous lesions are, or can be, significant. Recognition of the one can be a clue to the identity of the other, and evaluation of both skin and mucosa may often provide the first evidence of systemic disease.

The discussion will be divided into two parts: 1) primarily dermatologic diseases with oral manifestations; and 2) other diseases in which oral and cutaneous lesions are prominent features. It will be limited to a consideration of noninfectious disease processes.

I. Primarily Dermatologic Diseases with Oral Manifestations.

*Erythema multiforme* is an acute inflammatory disease of the skin of obscure etiology, although infectious and/or allergic mechanisms are suspect. A wide variety of predisposing factors has been implicated, the most prominent of which are infections (herpes simplex, primary atypical pneumonia) and drugs (sulfonamides, phenylbutazone). X-ray and carcinoma have also been associated with the disease. As the name implies, the lesions are multiform and may consist of papules, bullae, and the characteristic target lesions. Oral manifestations are seen in the so-called “major” type of erythema multiforme and consist of vesicles on the lips, tongue, buccal and gingival mucosa which rupture to produce painful erosions (Fig 1). The disease tends to be self-limited, although the major form is occasionally fatal and may warrant the institution of corticosteroids as well as supportive local care.

*Lichen planus.* This chronic papulosquamous disease is of unknown etiology, although viral and neurologic etiologies have been proposed, and psychic factors have frequently been found to be associated with the disorder. The eruption of flat-topped, angulated, violaceous papules beginning on the extremities and favoring flexor surfaces is quite characteristic, though a similar eruption termed lichenoid drug eruption occurs with a variety of drugs (antimalarials, alpha-methyldopa, gold, para-aminosalicylic acid (PAS), thiazides, tetracycline, and others). Mucous membrane lesions occur in 50% of cases; they may occur in the absence of skin lesions, and they have been observed in drug-induced lichen planus due to quinacrine, PAS, gold, sodium thiosulfate and phenothiazines. Lacy hyperkeratotic striae on the buccal mucosa and tongue are characteristic, though hyperkeratotic papules and ulcerated lesions may also be seen (Fig 2). The differential diagnosis of oral lesions includes leukoplakia, candidiasis, lupus erythematosus, and secondary syphilis, though the presence of the typical Wickham’s striae and of cutaneous lesions help in confirming the diagnosis. Corticosteroids in Orabase* and topical anesthetics are useful in the management of this condition.

*Bullous diseases.* Certain bullous diseases are characterized by mucous membrane lesions. *Pemphigus vulgaris* and its variant, *pemphigus vegetans,* are notorious for producing oral lesions. They are present, in fact, in almost every case, and in pemphigus vulgaris, over 50% of patients develop their first lesions in the oral mucosa. Large, flaccid bullae which rupture to leave denuded areas are found on the lips, buccal mucosa, floor of the mouth, and

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PATTERSON: ORAL MANIFESTATIONS OF CUTANEOUS DISEASE

Fig 1—Blistering and erosions of lips, conjunctival involvement, and systemic toxicity in the major form of erythema multiforme.

undersurface of the tongue. On the skin, recurrent crops of flaccid bullae showing Nikolsky’s sign are found in pemphigus vulgaris. Biopsy is useful in making the diagnosis, and direct and indirect immunofluorescence are characteristic, showing intercellular binding of IgG and complement in stratified epithelium. Corticosteroids and cytotoxic agents are used in treating this disease. Oral lesions are also seen in bullous pemphigoid and a similar disorder, cicatricial pemphigoid, but they are not seen in another blistering eruption, dermatitis herpetiformis.

Psoriasis. Mucous membrane lesions in psoriasis are rarities, but a small number of cases have been reported. Rather rigid criteria must be met in order for a lesion to qualify as oral psoriasis. Lesions must be clearly located on mucosa and not contiguous with skin lesions; they should be found coincidentally with cutaneous lesions and their course should parallel that of skin lesions. Histologic features are suggestive, but only suggestive, of the disease.

II. Diseases in which Oral-Cutaneous Lesions are a Prominent Feature.

Connective Tissue Diseases.

Behçet’s syndrome is an uncommon condition of unknown etiology characterized by recurrent ulcerations of the oral cavity and genitalia and by iritis. Thrombophlebitis, arthralgia, neurologic lesions, and erythema nodosum may also occur. It is seen most often in males in the third decade and presents as discreet, punched-out ulcers with erythematous borders and gray-yellow bases in the oral mucosa. A helpful diagnostic test is the occurrence of a pustule with surrounding erythema at the site of needle prick injury. This occurs 24 hours after injury and is most prominent at the height of an attack. It may be difficult to distinguish oral lesions from those of aphthous stomatitis, pemphigus or pemphigoid, and erythema multiforme. Treatments have included antibiotics, gamma globulin, and corticosteroids with or without azathioprine, all with varying results. The course is usually long and benign, although recurrent uveitis can result in severe eye damage, and neurologic involvement is indicative of a poor prognosis.
Reiter's syndrome is the familiar triad of urethritis, arthritis, and conjunctivitis. Perhaps it should be considered a tetrad, since mucocutaneous lesions occur in 80% of patients. Skin findings include hyperkeratotic lesions of the palms and soles (keratoderma blennorrhagicum) and psoriasiform plaques on the skin and scalp. Oral involvement is seen in up to 40% of cases and presents as painless superficial erosions of the palate, buccal mucosa, tongue, or gingiva. Treatments have included anti-inflammatory agents, methotrexate, antimalarials, and indomethacin. The disease may abate after two to six months or persist as recurrent attacks at varying intervals.

Scleroderma also features oral pathology. Fibrosis and atrophy of circumoral skin and widening of the periodontal space are relatively well-known findings. Less often appreciated are deformation of gingival papillae with the formation of granulation tissue in gingival pockets, atrophy of the mucosa with prominent venous pattern, and papillary atrophy of the tongue, producing the so-called "chicken tongue" appearance.

Occasionally (in 10% to 20% of cases) systemic lupus erythematosus is accompanied by oral lesions, and it should be noted that mucosal ulcers are one of the fourteen diagnostic criteria established by the American Rheumatism Association (ARA). Lesions may appear as pinpoint atrophic areas with keratotic margins and surrounding hyperemia. Petechiae on the palate, buccal mucosa, gingiva, or tongue develop into shallow, painful ulcers with gray, necrotic bases.

Gold toxicity can produce hemorrhagic, ulcerative, and exfoliative stomatitis. Skin manifestations include exfoliative dermatitis and lichenoid eruptions.

Methotrexate is a chemotherapeutic agent used in the treatment of lymphomas, leukemias, and (of importance to dermatologists) severe, recalcitrant psoriasis. Toxicity may develop as shallow whitish patches on the oral mucosa, surrounded by erythematous borders. Large areas of epithelium may then necrose and slough. Many chemotherapeutic protocols include the use of folic acid (citrovorum factor) to counteract methotrexate toxicity.

Genetic Disorders.

Of the many genetic disorders with oral-cutaneous manifestations, two have been selected. Peutz-Jeghers syndrome consists of mucocutaneous pigmentation and gastrointestinal polyposis. It is inherited as an autosomal dominant trait. Flat brown, black, or blue pigmented spots are seen on the vermilion borders of the lips, oral mucosa, perioral, nasal, and orbital skin, dorsa of fingers and toes (especially over joints), palms, and soles. Though skin pigmentation tends to fade after puberty, oral pigmentation remains for life. Melena and intussusception are the chief complications. It is important to note that, although malignant change of small bowel polyps is rare, there is an increase in incidence of cancer above the ligament of Treitz and in the colon.

Neurofibromatosis is also inherited as an autosomal dominant trait. The occurrence of multiple café au lait spots in neurofibromatosis is well known. Four to 7% of cases have oral involvement with single or multiple tumors, usually on the tongue. Malignant degeneration of neurofibromas is uncommon but does occur in 2% of patients and presents as a variant of fibrosarcoma.

Metabolic Disorders.

Cutaneous pathology in Addison's disease consists of diffuse pigmentation accentuated on exposed surfaces, sites of friction, palmar creases, and scars which have developed during adrenal insufficiency. The oral pigmentation is spotty in appearance and may occur in advance of other characteristics of the disease (Fig 3). The pigmentation results from increased output of beta-MSH from the pituitary, unchecked by the normal adrenal-pituitary feedback.
Addisonian buccal pigmentation may be difficult to distinguish from that of normal dark-skinned individuals and from Peutz-Jeghers syndrome, but other clinical features should aid in making this diagnosis.

**NUTRITIONAL DISORDERS.**

A discussion of oral-cutaneous disease would not be complete without a consideration of nutritional deficiency diseases.

*Ariboflavinosis* manifests as glossitis and cheilosis. Glossitis results from atrophy of the papillae of the tongue, along with dilatation and proliferation of the capillaries and concomitantly slowed circulation. The latter accounts for the tongue's characteristic magenta color. Cheilosis describes the denuded, reddened appearance of the lips at the line of closure and maceration at the angles of the mouth. Seborrheic accumulations around the nose are also seen.

The cutaneous features of *pellagra* are familiar, consisting of photosensitivity manifested by erythema of exposed areas. This burning, itching eruption desquamates to leave deep pigmentation and eventual atrophy. A necklace of dermatitis (Casal's necklace) and seborrhealike dermatitis of the nose (dyssebacea) are other characteristic findings. Oral disease includes an intense stomatitis, involving the tongue, gingiva, and palate, and reddening and ulceration of the lips. Pellagra today is often seen as part of a multiple nutritional deficiency state in alcoholics and chronically ill individuals.

In conclusion, this brief discussion includes only a few of many disorders in which oral and cutaneous manifestations play a prominent role. It emphasizes the importance of a thorough oral-mucosal examination in the evaluation of any perplexing cutaneous disease.

**REFERENCES**


