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Mucocutaneous Conditions may have possible role in the manifestation of Desquamative Gingivitis

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Abstract

Mucocutaneous Conditions are a group of disorders confined to the epithelium, involving the skin, oral mucosa and also other various mucosal sites like nasal mucosa, conjunctiva and genital mucosa. The common mucocutaneous conditions include lichen planus, pemphigus vulgaris and pemphigoid. Other less common could be systemic lupus erythematous is and erythema multiforme, chronic ulcerative stomatitis, plasma cell gingivitis, linear IgA disease, These diseases often present with common and similar clinical manifestations in oral cavity and diagnosis based on his to logical changes of the tissues after biopsy and immunofluorescence is done. The Most prevalent and earliest oral sign of these conditions could be Desquamative Gingivitis (DG), mostly identified by the dental practitioner, therefore in the present review we aimed to observe the clinical, histologic and immunofluorescent features of these conditions which will enable the clinician to more effectively recognize and diagnose such conditions.

Keywords: Mucocutaneous conditions, Desquamative gingivitis, Immunofluorescence

Introduction

Lichen planus, Pemphigus vulgaris and Pemphigoid are the most common Mucocutaneous conditions mainly observed in the dermatology practice, a multidisciplinary approach is necessary for the effective care. Based on the clinical presentation, the dental practitioner may be the first to identify oral lesions and hence plays an

important role in the early diagnosis and management of disease.[1]

Desquamative Gingivitis (DG) the chronic desquamation of the gingiva, is one such sign presented early in oral cavity. This could be due to gingival response to under lying such conditions. Desquamative gingivitis could be misdiagnosed as plaque induced gingivitis leading to delayed diagnosis and improper treatment of the under lying serious systemic diseases.[2] Their exact diagnosis is better established by his to pathological and immunofluorescence evaluation. Word desquamation derives from the Latin word 'desquamare' which means to scrape the scales off a fish.[3] Prinz coined the term Chronic Desquamative gingivitis in 1932.[3-4]

Lichen planus (LP) is an auto immune inflammatory mucocutaneous disorder common in middle-aged to elderly females than males where host T cells play a major role involving the mucosal surfaces and the skin. Desquamative gingivitis is an intra oral finding, presenting in Popular, bullous, reticular, plaque-like, atrophic, and erosive forms, most commonly erosive and reticular types. In LP an immune reaction does not result in clinical attachment loss and periodontitis. Histologically it is characterized by hyper keratosis, saw tooth rete pegs, hydropicdegeneration of the basal layer leads to epithelial thinning – detachment of the epithelium from the under lying connective tissue leads to sub-epithelial vesicle formation, infiltration of T-lymphocytes in lamina propria. Direct immunofluorescence of both lesional & perilesional biopsy specimen reveal fibrin deposits along the basement membrane and Scattered clusters of Civatte bodies (IgM) in the upper areas of lamina propria.[4-6]

Pemphigus vulgaris is another auto immune disorder, potentially lethal chronic condition. Greater predilection in women, usually after 40 years. The epidermal & mucous membrane blisters occurs when the cell-to-cell adhesion structures are damaged by the action of circulating autoantibodies to the pemphigus vulgaris antigens, which are cell surface glycoproteins (desmoglein, DSG) present in keratinocytes. Oral Lesions are presented with small vesicle to large bullae which rupture leading to area of ulceration. Any region of the oral cavity can be involved, soft palate is more often involved. Lesions are less often confined to gingiva, may present as erosive or desquamative gingivitis. Nikolsky's sign is path gnomic for pemphigus which signifies the difference of intra epidermal blisters from sub epidermal blisters. This is demon strated by applying lateral pressure on the border of an intact blister, which results in the dislodgment of the normal epidermis and extension of the blister.[6] His to pathology reveals an Intra-epithelial split, occurs above the basal cell layer, acantholysis, connective tissue consists of chronic in flammatory cell infiltrate. Immunofluorescence shows IgG in the inter cellular spaces[5,7]

Pemphigoid [5] is a group of cutaneous immune-mediated sub epithelial bullous diseases mainly bullous pemphigoid and mucous membrane pemphigoid (MMP). Bullous Pemphigoid is a chronic, auto immune subepidermal disease with tense bullae that rupture & become flaccid in the skin and oral lesions include erosive, bullous, vesicular type of desquamative gingivitis, there is no evidence of acantholysis in histopathology, vesicles are subepithelial than intraepithelial, the epithelium separates from the connective tissue at the basement membrane zone. Direct Immunofluorescence is positive in 100% of these patients, IgG and C3 deposits along the epithelial basement membrane.

Mucous membrane pemphigoid (Cicatricial pemphigoid) is a chronic vesiculo-bullous auto immune disorder, mainly affects women in 5th decade of life. This can involve skin, oral cavity, nasal mucosa, eosophagus, conjunctiva, urethra and vagina. Antigen anti body complexing occur at the basement membrane zone followed by compliment activation, leukocyte recruit Ment, next proteolytic enzymes are released and cleave the base Ment membrane. Immune responses directed against the BP-2 a major antigenic determinant, less often against epiligrin (laminin 5). Oral lesions include Desquamative gingivitis with areas of erythema, ulceration, vesiculation of attached gingival. Bullae have thick roof & rupture in 2-3 days leaving a irregular area of ulceration, healing of the lesion may take up to 3 weeks or longer. Direct Immunofluorescence, the main immuno-reactant are IgG and C3 which are confined to basement membrane[8-9]

Linear IgA disease (LAD) [5] clinically present as a pruritic vesiculobullous rash, painful ulcers, erosive gingivitis. Immunofluorescence reveals Linear deposits of IgA at the epithelial connective tissue interface. LAD mimics lichen planus both clinically & his to logically. Immunofluorescence & microscopic studies are necessary to establish the correct diagnosis.[10]

Systemic lupus erythematosus commonly seen in females, affecting vital organs like kidneys, heart, skin and mucosa. Classic cutaneous lesions characterized by presence of rash in the malar area with butterfly distribution. Orally ulcerative and lichen planus-like. Direct Immunofluorescence of the perilesional and normal tissue reveals immunoglobulins and C3 deposits at the dermal-epidermal interface and also Antinuclear antibodies are present in more than 95% of cases.[4]

Erythema multiforme is an acute bullous or macular in flammatory mucocutaneous life-threatening disease with series of immunopathologic mechanisms, which always involve oral mucosa presents as swollen lips, labial erosions, intra-oral blisters. Ischemic necrosis of epithelium and underlying connective tissue and Target or IRIS lesions with central clearing are hallmark of the disease. Sulfonamides, penicillins, phenylbutazone, phenytoin drug reactions, herpes simplex infection are being the known etiologic factors of this disease.[4]

Chronic ulcerative stomatitis mimics oral lichen planus both clinically and his to logically. The findings include ulcers and erosions in the gingival, buccal and lingual mucosae. It may rarely be associated with genital and conjunctival mucosal lesions. Direct Immunofluorescence studies of lesional and clinically normal mucosa reveals nuclear deposits of IgG in the basal one-third of the epithelium. Circulating antinuclear antibodies are also detectable.[4,5,11]

Plasma cell gingivitis is known to be the hypersensitivity reaction to mouth rinses, toothpastes chewing gum and many other flavorings products may present as des Qu amative gingivitis. It is referred to as stomatitis Medi camentosa. A similar reaction may occur as a contact allergic response to drugs taken orally or parenterally (e.g.: penicillin, aspirin burns) and is referred to as stomatitis venenata. Histological features of this lesion include spongiform pustules in the epithelium and a dense infiltrate of normal polyclonal plasma cells in the underlying lamina propria.[4,5]

Conclusion

To recognize DG as a clinical sign of mucocutaneous conditions, a thorough examination of the oral cavity, with the proper diagnosis involving a detailed history, with extra oral examination and incisional biopsy from a perilesional site with intact epithelium for his to pathology and Immunofluorescence studies is must for effective treatment of the under lying disease.

Cutaneous, nasal, ocular and genital lesions diagnosed patients are referred to a particular specialist is advised. The gingival lesions are usually treated by improved oral hygiene measures and topical corticosteroid therapy.

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