Immuno-Pathologic diseases: Dr. Rehab Faisal

Oral lichen planus: (OLP)

The etiology of OLP is not known, but in the recent studies evident the immune system has a primary role in the development of this disease .OLP is a chronic inflammatory disease of skin and mucous membrane; it mainly affects patients of middle age or over especially women, it is common and if untreated can persist for many years.

Clinically:

OLP may contain both red and white elements and provide together with the different textures, types of OLP

$ Reticulum

$ Papules

$ Plaque-like

$ Bullous

$ Erythematous (atrophic)

$ Ulcerative.

The distribution of lesion is mostly the buccal mucosa particularly posteriorly; the next most common site is the tongue, lip, palate and gingiva. The lesions are very often symmetrical, but in some cases are more prominent on one side than another.

-**Reticular:** - is characterized by fine white lines or striae that may form a network but can also show annular (circular) patterns. The striae often display a peripheral erythematous zone, which reflects the subepithelial inflammation, most frequently this form is observed bilaterally in the buccal mucosa and rarely on the mucosal side of the lips .

-**Papular type:-** It is clinically characterized by small white dots, sometimes the papular elements merge with striae as part of the natural course.

-**Plaque-type:-** shows a homogeneous well-demarcated white plaque often, but not always, surrounded by striae. Plaque-type lesions may clinically be very similar to homogeneous oral leukoplakia.

Note: Typically, the reticular, papular, and plaque-like forms of OLP are asymptomatic, although the patient may experience a feeling of roughness.

-Erythematous (atrophic) OLP:- is characterized by a homogeneous red area. It is present in the buccal mucosa or in the palate and striae are frequently seen in the periphery. Some patients may display erythematous OLP affecting attached gingiva.

-Ulcerative type:- Clinically, the fibrin-coated ulcers are surrounded by an erythematous zone frequently displaying radiating white striae. As for the erythematous form of OLP, the affected patient complains of a smarting sensation in conjunction with food intake.

Histopathology:

-Hyperkeratosis.

- A subepithelial band–formed infiltrate dominated by T lymphocytes and macrophages

-Degeneration of basal cells known as liquefaction degeneration. These features refer to the immune system being involved in the pathogenesis of OLP.

Diagnosis: - A biopsy for histopathology examination.

Treatment -:

-Topical corticosteroid.

-Kenalog in ora-base ointment (1% Triamicnolone in oral past) 2-4 times daily.

-In severe cases, the treatment become with systemic corticosteroid.

How can differentiate clinically between LP and OLP?

- In LP the demarcation is usually very distinct, while in OLP the white components have a more diffuse transition to the normal oral mucosa.
- The lack of a peripheral erythematous zone in LP.

Drug-Induced Lichenoid Reactions :-(DILRs)

Drugs or their metabolites act as haptens trigger a lichenoid reaction, like Penicillin, gold, and sulfonamides. The other drugs like antihypertensive agents (methyldopa), thiazide (diuretic) & anti-malaria.

Clinically: Lichenoid drug eruptions appear similar to lichen planus and may be severely pruritic .

Management: Discontinuance or change the drug and symptomatic treatment with topical steroids are often sufficient.

Lichenoid reaction of graft-versus-host disease: (GVHD)

The oral lesion of GVHD has the same clinical appearance of OLP but the lesion is usually more generalized, also skin involvement (pruritic maculopapular primarily affecting the palms and soles), however the oral cavity may be the primary or even the only site of chronic GVHD.

Lupus Erythematosus: (SLE, DLE)

There are two types which are chronic discoid L.E. (localized type) and systemic L.E. (disseminated type). Etiology not known but genetic factors appears to be important. Autoimmune disease involves immune complexes. Environmental factors as sun exposure, drugs, chemical substances, and hormones which all have been reported to aggravate the disease. Females are affected much more frequently than males.

Clinically:

The oral lesions observed in SLE and DLE are similar in their characteristics. The typical clinical lesion comprises white striae with a radiating orientation, and these may sharply terminate toward the center of the lesions, which has a more erythematous appearance. The most affected sites are the gingiva, buccal mucosa, tongue, and palate.

DLE is restricted to the skin and usually occur on the face. These lesions may form butterfly-like rashes over the cheeks and nose known as malar rash. While the SLE characterized by skin rash (maculopapular), lymphadenopathy, kidney, liver, lung & nervous system are also frequently involved.

Diagnosis: SLE diagnosis with 4 or more of 11 criteria present at any time.

-Malar rash.

-Discoid lesion.

-Photosensitivity.

-Presence of oral ulcers.

-Non erosive arthritis of two joints or more.

-Serositis.

-Renal disorder.

-Neurological disorder.

-Hematological disorder (leukopenia, lymphopenia, thrompocytopenia and hemolytic anemia)

-Immunologic disorder (anti-DNA, anti-SM, or antiphospholipid antibodies).

-Direct immunohistochemistry is conducted to reveal granular deposition of IgM, IgG, IgA, and C3 (lupus band test) anti-nRNA (antinuclear ribonucleo-protein)

Laboratory Findings :

-Antinuclear antibodies are frequently found in patients with SLE and can be used to indicate a systemic involvement, but patients with other rheumatologic diseases, such as Sjögren's syndrome and rheumatoid arthritis, may be positive .

-Moderate to high titers of anti-DNA and anti-Smith antibodies are almost pathognomonic of SLE.

-Antibodies associated with Sjögren's syndrome, SLE [anti-SS-A(RO) and anti-SS-B(La.)

Management:

-Topical steroids to relief of oral symptoms such as clobetasol propionate gel 0.05%, betamethasone dipropionate 0.05%

-Immunosuppressive drugs used to treat LE.

▼Allergic Reactions:

Lichenoid Contact Reactions (LCRs): are considered as a delayed hypersensitivity reaction to constituents derived from dental materials. Hg is usually considered the primary etiologic factor; other amalgam constituents may initiate LCR.

Clinically :

LCRs display the same reaction patterns as seen in OLP. The most clinical difference between OLP and LCR is the extension of the lesions. LCRs are confined to sites that are in contact with dental materials, such as the buccal mucosa and the border of the tongue, non-symptomatic, but when erythematous or ulcerative the patient may has discomfort from spicy and warm food constituents. Lichenoid reactions in contact with composites have been observed on the mucosal side of both the upper and lower lips.

Management :

Replacement of dental materials in direct contact with LCR

Reactions to Dentifrice and Chlorhexidine:

Delayed hypersensitivity reactions to toothpastes and mouth washes have been reported, but these reactions are rare.

Clinically: fiery red edematous gingiva, which may include both ulcerations and white lesions.

▼Toxic Reactions:

Reactions to smokeless tobacco: Smokeless tobacco can be divided into three different groups: chewing tobacco, moist snuff, and dry snuff. The lesion may be noted as wrinkles at the site of application or may display a white and leathery lesion which sometimes contains ulceration.

Smoker's palate: The most common effects of smoking are presented clinically as dark brown pigmentations of the oral mucosa (smoker's melanosis) and as white leathered lesions of the palate, usually referred to as nicotine stomatitis or smoker's palate. As part of this lesion, red dots can be observed representing orifices of accessory salivary glands, which can be enlarged.

▼Reactions to trauma-:

Mechanical: like

-linea alba due to chronic chewing & sucking of the cheek produce a thin band on buccal mucosa bilaterally at the level of the occlusal plane.

-Frictional hyperkeratosis clinically characterized by a white lesion without any red elements and observed in areas of the oral mucosa subjected to increased friction caused by food intake (edentulous alveolar ridge or any part of oral mucosa exposed to trauma), non-symptomatic.

Chemical :

-Aspirin burn: in the buccal sulcus adjacent to painful tooth lead to white sloughy epithelium.

-Uremic stomatitis: extensive pseudomembranous white lesion in patients with renal failure due to increase blood urea nitrogen level (above 50 mg/dl(

Thermal: Smokers of cigarettes, cigars and pipes.

▼Other Red and White lesions :

Benign migratory glossitis (geographic tongue) :

It is an annular lesion affecting the dorsum and margin of the tongue. The typical clinical presentation comprises a white, yellow, or gray slightly elevated peripheral zone, reflecting atrophy of the filiform papillae. Non symptomatic

Etiology = genetic factors.

Management: no treatment but topical anesthesia when symptom is reported.

Leukodema: etiology unknown.

Clinically: is a white alteration of the oral mucosa. The condition is found bilaterally in the buccal mucosa and sometimes at the borders of the tongue .

Diagnosis: gentle stretching results in a temporary disappearance, by this way can differentiate from other oral keratosis like LP.

Management: There is no demand for treatment as the condition is no symptomatic.

White sponge nevus: is an autosomal dominant disorder.

Clinically: It is a white lesion with an elevated and irregular surface. The most affected sites are the buccal mucosa, but the lesion may also be in other areas of the oral cavity covered by keratinized epithelium.

Management: no treatment.

Hairy tongue:

The etiology of hairy tongue is unknown in most cases.

Number of predisposing factors that have been related to this disorder:

-Neglected oral hygiene ،

-a shift in the microflora ،

-antibiotics and immunosuppressive drugs ،

-Oral candidiasis.

-Excessive alcohol consumption.

-Therapeutic radiation.

-Smoking habits.

Clinically :

Hairy tongue is characterized by an impaired desquamation of the filiform papilla, which leads to the hairy-like clinical appearance.The elongated papillae have to reach lengths in excess of 3 mm.The lesion is commonly found in the posterior one-third of the tongue but may involve the entire dorsum. Hairy tongue may adopt colors from white to black depending on food constituents and the composition of the oral micro-flora.

Diagnosis: The diagnosis is based on the clinical appearance.

Management :

The treatment of hairy tongue is reduction or elimination of predisposing factors and removal of the elongated filiform papillae. The patients should be instructed on how to use devices developed to scrape the tongue.

Oral Medicine

References :

- Burket’s Oral Medicine 12th edition 2015.

- Burket’s Oral Medicine 11th edition 2008.

- CAWSON'S ESSENTIALS OF ORAL PATHOLOGY AND ORAL MEDICINE seven edition 2002.

Thank you