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LICHENOID INTERFACE DERMATITIS AND ORAL LICHENOID REACTIONS: A REVIEW

Dermatology

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ABSTRACT

Lichenoid tissue reactions or Interface Dermatitis are some of the commonly encountered clinical and histological presentations in dermatology and pathology. The term Lichen refers to a group of skin diseases characterized by eruptive skin lesions which are grouped together as they resemble the algae lichen found growing on rocks. Interface dermatitis can be classified based upon the cell type that dominates the infiltrate or by the intensity of the interface inflammation. This review focusses in detail on lichenoid interface dermatitis and oral lichenoid reactions.

KEYWORDS

Lichenoid reactions, interface dermatitis, skin lesions, oral lesions

INTRODUCTION:

The term Lichen refers to a group of skin diseases characterized by eruptive skin lesions which are grouped together as they resemble the algae lichen found growing on rocks[1,2]. Lichenoid tissue reaction or interface dermatitis embrace several clinical conditions, the prototype of which is lichen planus and its variants, drug induced lichenoid dermatitis, special forms of lichenoid dermatitis, lichenoid dermatitis in lupus erythematosus, and miscellaneous disorders showing lichenoid dermatitis, the salient clinical and histological features of which are described to facilitate their diagnosis [3]. Lichenoid eruptions are uncommon skin rashes that can be induced by many environmental agents, medications or industrial by-products such as inhaled particles[4]. Lichenoid reaction is induced by external factors which could be either topical or systemic. A number of triggering factors such as restorative materials, graft versus host reaction and broad group of drugs are known to cause Lichenoid reaction [1,2].

TERMINOLOGY AND CLASSIFICATION:

The term ***"lichenoid"*** refers to papular lesion of certain skin disorders of which lichen planus is the prototype. However, this type of reaction can also be seen skin disorders associated with systemic illnesses like *lupus erythematosus* and the skin changes of potentially fatal disorders such as *graft versus host disease, Stevens Johnson syndrome* and *toxic epidermal necrolysis* [3]. Lichenoid reaction can be considered as a disease itself or as an exacerbation of an existing OLP. The term ***Interface Dermatitis*** refers to the finding in a skin biopsy of an inflammatory infiltrate that abuts or obscures the dermo-epidermal junction [3,5]. When the lesion has been induced by a medication it can be called more specifically a *lichenoid drug eruption*. Historically, interface dermatitis has been classified based upon the cell type that dominates the infiltrate (ie, neutrophilic, lymphocytic, or lymphohistiocytic). Interface dermatitis can also be classified by the intensity of the interface inflammation; regarding lymphocytic interface dermatitis, there are 2 broad categories in our classification scheme. These include cell-poor interface dermatitis (table 1) when only a sparse infiltrate of inflammatory cells is present along the DEJ, or cell rich. The infiltrate in lesions of cell-rich lymphocytic interface dermatitis (table 2) typically occurs as a heavy band like process that obscures the basal layers of the epidermis; this is often termed a *lichenoid* interface dermatitis [5,6]. The rash of a lichenoid drug eruption can sometimes be difficult to distinguish from idiopathic lichen planus because of similarities in the clinical appearance and the pathology seen on skinbiopsy [4]. Lichen planus and lupus erythematosus are the most common and best studied representatives of the lichenoid tissue reaction.

Table 1. Prototypes of Cell-Poor Interface Dermatitis

Erythema multiforme
Autoimmune connective tissue disease, particularly
Systemic lupus erythematosus
Dermatomyositis
Mixed connective tissue disease
Graft-versus-host disease
Morbiliform viral exanthem
Morbiliform drug reaction

Table 2. Prototypes of Cell-Rich Interface Dermatitis

Idiopathic lichenoid disorders
Lichen planus
Lichen nitidus
Lichen striatus
Lichenoid autoimmune connective tissue disease, particularly
Discoid lupus erythematosus
Anti-RO–positive systemic lupus erythematosus
Mixed connective tissue disease
Lichenoid and granulomatous dermatitis
Lichenoid purpura
Lichenoid and fixed drug reaction

Oral lichenoid reactions include the following disorders:[7]

- Oral lichen planus
- Lichenoid contact reactions
- Lichenoid drug eruptions
- Lichenoid reactions of graft-versus-host disease (GVHD)

Oral LCRs are included in the section below where allergic reactions are discussed since these lesions represent a delayed hypersensitivity reaction to constituents derived from dental materials or flavoring agents in for foods and other ingested substances.

List of causative/ exacerbation factors for OLR[8]

1) Drugs

- *Anti-malarials*
- *Non-steroidal anti-inflammatory drugs*
- *Angiotensin- converting enzyme inhibitors*
- *Diuretics*
- *ß-blockers*
- *Oral hypoglycemics*
- *Gold salts*
- *Penicillamine*
- *Anti-retrovirals*

2) Dental materials
- *Dental amalgam*
- *Composite and resin-based materials*
- *Metals* (eg: *nickel*)

3) Chronic liver disease and Hepatitis C virus
4) Stress
5) Genetics
6) Tobacco chewing
7) Graft-versus-Host disease

Medications commonly reported to trigger a lichenoid drug eruption include:
- Antihypertensives – ACE inhibitors, beta-blockers, nifedipine, methyldopa
- Diuretics – hydrochlorothiazide, frusemide, spironolactone
- Non-steroidal anti-inflammatory drugs (NSAIDs)
- Phenothiazine derivatives
- Anti-convulsants – carbamazapine, phenytoin
- Drugs to treat tuberculosis
- Antifungal drug – ketoconazole
- Chemotherapeutic agents – hydroxyurea, 5-fluorouracil, imatinib
- Antimalarial agents such as hydroxychloroquine Sulfa drugs
- including sulfonylurea hypoglycaemic agents, dapsone, mesalazine, sulfasalazine
- Metals – gold salts
- Others – allopurinol, iodides and radiocontrast media, interferon-α, omeprazole, penicillamine, tetracycline

Some new medications that have been reported in association with lichenoid drug eruptions include:
- Tumour necrosis factor antagonists such as infliximab, etanercept and adalimumab
- Imatinib mesylate (tyrosine kinase inhibitor)
- Misoprostol (prostaglandin E1 agonist)
- Sildenafil citratus (Viagra™)

If a lichenoid eruption has developed to a drug, then it is quite possible for the same reaction to appear more quickly after exposure to another medication in the same family. Examples reported have included the proton pump inhibitors (for dyspepsia) and the HMGCoA reductase inhibitors (for high cholesterol)[4].

EPIDEMIOLOGY:
The precise epidemiological characteristics of these lesions are difficult to establish, due to the diverse and imprecise terminology used in establishing a final diagnosis of lichen planus (LP) as "probable", "possible" or "suggestive", in those situations where the histopathological and clinical findings are less clear than in classical lichen planus[6]. It has been reported that the prevalence of OLP is 1.9%; however, the figure depends on the population studied [9]. Extensive epidemiological and clinical studies of OLRs have been undertaken in the United States, UK, Japan, and Scandinavia, while studies on possible disease associations have been reported on Japanese, British, and other European patients[10].

PATHOGENESIS:
There is a lot of advances going on in understanding the pathophysiology of interface dermatitis. The most prominent feature is the individual cell necrosis of keratinocytes. T cell mediatedcytokine damage is the most likely mechanism resulting in these diseases[15]. The lichenoid or lichen planus like actinic keratosis, is another example that basal cell damage, though by a neoplastic process, can lead to a typical lichenoid reaction[3,14].

Components of this interface are: 1) Basal layer of epidermis: tall columnar cells, perpendicular to basement membrane ,2) Dermoepidermal junction, 3) The papillary dermis in contact with basement membrane, 4) The adventitial dermis around the adnexal structures [15]. Usually, there is some lymphocyte tagging along the DEJ pointing to the immunopathogenetic basis, namely, cellular cytotoxicity, either in the context of a type 4 immune reaction or of antibody-dependent cellular cytotoxicity, or a type 2 immune reaction, when autoantibodies target components of the basement membrane zone to trigger an antibody-dependent cellular cytotoxicity reaction [5]. The possible mechanism involved in the variability of expression of lichenoid tissue reaction depends on the degree and pattern of expression of the intercellular adhesion molecule-1 (ICAM-1)[13].

CLINICAL FEATURES:
LICHEN PLANUS
OLP usually presents as a symmetrical bilateral or multiple lesion. It can occur as 6 types of clinical variants namely reticular, papular, plaque like, erosive, atropic and bullous [8, 16, 17]. In many patients, the onset of OLP is insidious, and patients are unaware of their oral condition. Some patients report a roughness of the lining of the mouth, sensitivity of the oral mucosa to hot or spicy foods, painful oral mucosa, red or white patches on the oral mucosa, or oral ulcerations [8,18].

DRUG INDUCED LICHENOID REACTIONS
Lichenoid drug eruptions are caused by a variety of medications ranging from thiazide diuretics to antihistaminics and antihypertensive agents with _-blocking and ACE inhibitor properties [5,20]

FIXED DRUG ERUPTIONS
They may probably be induced by drug acting as hapten, binding to keratino/melanocytes producing an immunological reaction, an antibody-dependent cellular cytoid response. The clinical lesions may mimic lichenoid interface dermatoses. [3]

GRAFT VERSUS HOST DISEASE
Chronic graft versus host disease occurring after 3 months of transplantation or later may resemble lichenoid reactions, affecting the palms, soles, trunk buttocks, and thighs 3. In lesions of chronic GVHD, a lichenoid morphology supervenes. Ultimately, chronic inflammation in this setting may result in a sclerodermoid tissue reaction [5,23,24,25,26.]

LUPUS ERYTHEMATOSUS
Lupus erythematosus (LE) is an autoimmune disorder affecting skin, hematopoietic, and lymphoreticular organs, joints, kidney, lung, serosa, and cardiovascular structures in concert or in isolation. Lupus erythematosus is subdivided clinically into systemic (SLE), subacute cutaneous (SCLE), and discoid (DLE) forms, each with its own characteristic skin findings [5,21,22]

ERYTHEMA MULTIFORME
Erythema multiforme is a distinctive clinical pathologic entity with a wide variety of underlying causes. The classic lesion has a targetoid morphology with a peripheral rim of erythema and a central zone of pallor. Some lesions manifest a dusky or violaceous appearance with no true central clearing[5]

LICHENOID DERMATITIS
In Late Secondary dermatitis, lichenoid reaction is mainly formed by plasma cells.

In HSV, atypical mucocutaneous manifestations are seen in immunosuppressed population.

In AIDS related lichenoid dermatitis, there is severe cutaneous photosensitivity.

In Pityriasis lichenoides et varioliformis acuta (PLEVA) , there is epidermal necrosis due to focal epidermal cell death and lymphocytic infiltration in dermo epidermal junction.

In Dermatomyositis the skin lesions, there is prominent basal vacuolar changes and severe cases resemble acute lupus erythematosus. Dermatomyositis combines an inflammatory myopathy with characteristic skin lesions: the often-subtle heliotrope rash, the Gottron papule, a violaceous or hypopigmented papule over the joints of the fingers, erythema of the upper back (the "shawl sign"), extensive erythema of the extensor surfaces of the arms, scaly alopecia, and cuticular overgrowth with periungual telangiectasias [5,19]

In Lichen Sclerosus there is a chronic inflammatory dermatosis that results in white plaques with epidermal atrophy.

In Lichenoid Contact Dermatitis, the lesions usually results from contact with rubber and chemical used in clothing dyes and wine industries.

In Lichen spinulosus, It is characterized by round-to-oval patches of grouped follicular papules with rough keratotic centers.

CONCLUSION:

A bandlike inflammatory infiltrate that obscures the DEJ, that is, a lichenoid inflammatory process, calls to mind a set of differential diagnostic considerations that is distinct from those that flow from a cell-poor interface injury pattern. When there is evidence of changes in clinical appearance, the follow-up period should be shortened and additional biopsy should be performed.

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