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INTERFACE DERMATITIS OR LICHENOID TISSUE REACTION: A REVIEW

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ABSTRACT

Interface dermatitis (also called lichenoid tissue reaction) is an inflammatory skin dermatosis in which the junction between the epidermis and papillary epidermis is obscured. The number of uncommon, clinically diverse, and poorly understood inflammatory skin manifestations are linked by the presence of a set of histopathological features that have traditionally been included as Interface dermatitis. All these inflammatory skin diseases are associated with a set of histological features, primarily involving the dermo-epidermal junction. Sustained interface reactions often result in loss of pigment from basal cells and their ingestion by melanophages. The lesions in interface dermatitis may vary from flat to raised, scaly to smooth, which depends upon epidermal reaction. Interface dermatitis is divided into the type of cells that is dominant (lymphocytic or neutrophilic or lymphohistiocytic) or by the severity of the inflammation. Treatment of this various group of cutaneous disorders is guided by the degree of symptomatology, disability, and associated systemic illness. This review tries to encapsulate the current knowledge about interface dermatitis.

KEYWORDS: Interface dermatitis, lichenoid tissue reaction, dermo-epidermal junction, vacuolar alteration**.**

INTRODUCTION

Dermatologic defects characterized by pathology at the dermo-epidermal junction are etiologically diverse and controversial groups seen in a somewhat nebulous anatomical location. Lichenoid tissue reaction, also known as interface dermatitis, includes conditions in which the primary pathology involves the "interface," which is the dermo-epidermal junction. It encompasses the stratum basal layer, the dermo-epidermal junction, papillary and adventitial dermis around the adnexal structures. They form a functional component, with pathology in anyone affecting all the individual constituents.[1]

Interface reactions are cell-mediated immunologic reactions that target basal keratinocytes that inhabit above the dermo-epidermal junction.[2]

Definitions

Several clinically divergent, poorly understood, and relatively not common inflammatory skin diseases are linked together by the presence of a pattern of not uncommon histopathological elements that traditionally has been referred to as the "lichenoid tissue reaction." These elements consist of a pattern of epidermal basal cell morphological change that has been variously described as being "liquefactive/hydropic/vacuolar".[3] More modern investigators have begun to refer to this histological pattern as "interface dermatitis" rather than as a lichenoid tissue reaction. They used "interface dermatitis" more broadly. It refers to the inflammatory infiltrate that obscures the dermal-epidermal junction, which is a finding in skin biopsy.[4] These investigators feel that the term "lichenoid tissue reaction" should be reserved for the cell-rich subset of interface dermatitis diseases such as lichen planus (that is, the designation "lichenoid"). However, the name "lichenoid tissue reaction" for this group of diseases remains in use. Thus, to be as inclusive as feasible, the terms lichenoid tissue reaction and interface dermatitis will be used synonymously in this review.

Epidemiology

The epidemiology of many interface dermatitis disarray has not been systematically investigated. Several such manifestations are pretty rare, making population-based epidemiological studies challenging to perform. While

one of the most common dermatosis lichen planus has a prevalence of up to 4%.[5]

Etiopathogenesis

The basal cell injury is a common denominator of these separate groups of disorders. The term "interface dermatitis," often adopted for lichenoid disorders, denotes that the inflammatory infiltrates and basal cell damage appear to hide the dermo-epidermal junction. The epidermal basal cell injury leads to cell death and vacuolar modification (liquefactive degeneration). The so-called Civatte bodies are injured epidermal cells with shrunken eosinophilic cytoplasm and pyknotic nuclear residues (apoptosis). However, some manifestations show frank necrosis of the epidermis instead of apoptosis. Filamentous degeneration is an added type of cell destruction, which may display none of the above changes. Melanin incontinence is seen more frequently in drug or solar damage induced dermatoses.[6,7]

Recent work has suggested that some distinctive interface dermatitis skin disorders participate in a general inflammatory signaling pathway, including plasmacytoid dendritic cell-derived interferon-alpha (IFN-α). This signaling pathway turns out to amplify cytotoxic T cell injury to the epidermal basal cell compartment. Autoimmune attack of T-cells is the main pathological phenomenon in the lichenoid tissue reaction. It has been felt that cytotoxic T-lymphocytic cells denote the major effector cell type for the basal cell layer injury pattern common to lichenoid tissue reaction disorders. These inferences are based primarily upon the histopathological findings of human lichenoid tissue reaction diseases and experimental studies. In few lichenoid tissue reaction disorders, the antigens are targeted by activated T cells are known (for example, alloantigens in graft-versus-host skin disease). In others, the targeted antigen is thought to be an autoantigen (for example, Ro/SSA and La/SSB in neonatal lupus erythematosus and subacute cutaneous lupus erythematosus). However, in most cases, the targeted antigen is unknown, with cross-reactivity between environmental antigens (for example, viral, drug, chemical) and self-antigens being suspected. Few chemicals, drugs, and infectious agents have been implicated as triggers for lichen planus and lichenoid drug eruptions.[8,9]

Classifications

The histopathological classification is most important as the word interface dermatitis refers to a skin biopsy finding of an inflammatory infiltrate that obscures the dermo-epidermal junction. (Table 1) Historically, interface dermatitis has been classified based upon the cell type that dominates the infiltrate (i.e., neutrophilic, lymphocytic, or lymphohistiocytic). The intensity of the interface inflammation can also classify interface dermatitis; there are two broad categories in our classification scheme regarding lymphocytic interface dermatitis. These include cell-poor interface dermatitis, when only a sparse infiltrate of inflammatory cells is present along the dermo-epidermal junction or cell-rich. The infiltrate in cell-rich lymphocytic interface dermatitis lesions typically occurs as a heavy band-like process that hides the basal layers of the epidermis; this is often termed a lichenoid interface dermatitis.[4]

Table 1: Types of Interface Dermatitis

Cell-Poor Interface Dermatitis	Cell-Rich Interface Dermatitis
Erythema multiforme	Idiopathic lichenoid disorders
Autoimmune connective tissue disease, particularly	Lichen planus
Systemic lupus erythematosus	Lichen nitidus
Dermatomyositis	Lichen striatus
Mixed connective tissue disease	Lichenoid autoimmune connective tissue disease,
Graft-versus-host disease	particularly
Morbiliform viral exanthem	Discoid lupus erythematosus
Morbiliform drug reaction	Anti-RO–positive systemic lupus erythematosus
	Mixed connective tissue disease
	Lichenoid and granulomatous dermatitis
	Lichenoid purpura
	Lichenoid and fixed drug reaction

Another way these conditions are characterized by sparse infiltrates and vacuolar change at the dermo-epidermal junction (vacuolar interface dermatitis) and those that, in addition to vacuolar change, also have denser, band-like infiltrates (lichenoid interface dermatitis). In vacuolar-type, basal cell vacuolization is the most prominent pathological finding and is accompanied by variably dense perivascular and interstitial infiltrate consisting of lymphocytes. Examples of this pattern are early cutaneous LE, erythema multiforme (EM), viral exanthems, and acute graft versus host reaction. In lichenoid type, the classical histopathological finding is a thick band-like infiltrate in the papillary dermis, which often obscures basal cell vacuolization, which may be inconspicuous or even absent. Lichen planus is the best example of this type of interface dermatitis.[1]

Clinically lichen planus is considered the prototype interface dermatitis disorder. The papules of the lichen planus are shiny, polygonal, and plane-topped of varying sizes and occurs in groups creating a pattern that resembles lichen growing on a rock. (Table 2) The

reaction can be deduced from the essential feature of epidermal basal cell damage, whether primary or secondary.[10]

Table 2: Interface dermatitis: Clinical variants	
Prototype	Lichen planus Erythema dyschromicum perstans Keratosis lichenoides chronica Lupus erythematosus – Lichen planus overlap syndrome Lichen nitidus
Other variants	Lichen striatus Lichen planus-like keratosis Lichenoid drug eruptions
Drug Induced	Fixed drug eruptions Erythema multiforme Toxic epidermal necrolysis Lupus erythematosus AIDS interface dermatitis Graft versus host disease Paraneoplastic pemphigus
Miscellaneous	Poikilodermas Pityriasis lichenoides Lichenoid purpura Lupus erythematosus

Examples of interface dermatitis

So as to get a general understanding of interface dermatitis, two prototype disorders, each belonging to cell-poor and cell-rich interface dermatitis, are described. Providing detailed information about all the diseases which have interface dermatitis is beyond the scope of this review.

Cell-poor vacuolopathic interface dermatitis is defined by basilar keratinocyte and subepithelial vacuolopathy unaccompanied by a significant inflammatory infiltrate. The prototype is erythema multiforme. 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. Blisters may be observed. Common to cases of drug- or infectious-based etiology are focal areas of basilar vacuolopathy accompanied by lymphocyte tagging along the dermo-epidermal junction; suprabasilar lymphocytosis around degenerating keratinocytes also may be seen. In those cases mediated by infection, one typically observes a fairly brisk angiocentric superficial and deep lymphocytic infiltrate, along with a cell-poor interface dermatitis with minimal epidermal injury.[11]

We define Cell-rich interface dermatitis as inflammatory infiltrates along the dermo-epidermal junction that is of sufficient intensity that obscures, at least focally, the basilar keratinocytes. When this process is band-like, it may be termed lichenoid. The prototype example is lichen planus. It is considered an idiopathic dermatosis. Lichen planus manifests as violaceous, itchy, flat-topped, polygonal papules covered by a reticulated surface scale termed *Wickham striae*. Lesions typically manifest on the volar aspect of the forearms and other flexural surfaces of acral parts; genitalia is often involved. Lesions may be widespread. Oral changes accompany the cutaneous eruption in roughly one-half of cases, and these manifest as linear or reticulate whitish plaques and as lacy white patches along the occlusal lines of the buccal mucosa and elsewhere. Nail changes are frequently seen and manifest as dystrophies with ridging and splitting of the distal aspect of the nail plate. Variants of lichen planus include the atrophic form (*lichen planus actinicus*), hypertrophic lichen planus, bullous lichen planus, and linear lichen planus. Compact orthokeratosis overlying an epidermis that shows wedge-shaped thickening of the granular cell layer and a "sawtooth" pattern of acanthosis is prototypic for lichen planus. A dense, band-like lymphocytic infiltrate obscures the dermo-epidermal junction.[12]

As the prevalence of most of the interface dermatitis disorders is quite rare, the diagnosis is most of the time is based on the exclusion of other commonly present dermatitis or other systemic disorders.

Treatment

Many of the time, the treatment of interface dermatitis can be challenging. Treatment of these disorders is guided by the degree of symptomatology, disability, and associated systemic illness. Self-limited disorders such as lichen nitidus and lichen striatus are treated with topical immunomodulators (for example, corticosteroids, calcineurin inhibitors) until they spontaneously remit. Protection from sunlight or avoidance and broad-spectrum sunscreen use can be of benefit to ultraviolet light-induced interface dermatitis disorder such as cutaneous lupus and cutaneous dermatomyositis. Severely symptomatic, potentially disfiguring/disabling interface dermatitis is very hard to treat. Uncontrollable

lichen planus rubra and erosive lichen planus of the genitalia need systemic immunosuppressive or immunomodulatory therapy (for example, corticosteroids, mycophenolate mofetil, cyclosporine), with serious side effects. Similarly, systemic treatment approaches are required for fulminant, life-threatening interface dermatitis disorders such as erythema multiforme major (Stevens-Johnson syndrome) and toxic epidermal necrolysis. Interface dermatitis associated with potentially life-threatening systemic diseases is treated empirically in a graded fashion depending upon the severity of the cutaneous and systemic manifestations. Limited forms of cutaneous lupus, cutaneous dermatomyositis, and graft-versus-host skin disease can be treated with topical immunomodulatory therapy and systemic nonimmunosupressive anti-inflammatory agents such as the aminoquinoline antimalarials (chloroquine, quinacrine, hydroxychloroquine) and dapsone. When the cutaneous manifestations of these disorders are severe or are associated with significant systemic disease injury, systemic immunosuppressive/ immunomodulatory therapy is required. Because of the rarity of many of the interface dermatitis skin disorders, virtually all of the above-noted treatment modalities are carried out on an ‘‘off-label’’ non-FDA indicated basis.[13]

CONCLUSION

Interface dermatitis can be broadly broken down into cell-poor and cell-rich inflammatory processes and addressed as well by the character of the cellular infiltrate. A band-like inflammatory infiltrate that obscures the dermal-epidermal junction, 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. The T lymphocytes mediate damage to the basement membrane zone and the keratinocytes above them, the microscopic hallmarks being vacuolar alteration and single keratinocyte necrosis, respectively. Vacuolar alteration is characterized by small empty circular spaces of varying diameter along the dermal-epidermal junction. A comprehension of the pathobiology of the different entities that fall into these broad categories of injury will point the astute pathologist toward recognition of distinctive histomorphologic features which can, with clinic-pathologic correlation, can enable a precise etiopathologic diagnosis. Else it is mostly a diagnosis of exclusion. For most of these diseases, the treatment is immunosuppressant and anti-inflammatory drugs, and most of them are used off-label.

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