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Cutaneous Pseudolymphomas

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Abstract

Background: Cutaneous pseudolymphoma is a term used to describe skin lesions that bear a clinic and/or histopathologic resemblance to lymphoma. Cutaneous pseudolymphoma is mainly classified as two major categories, including mixed B and T cell pseudolymphomas, and T cell pseudolymphomas. Cutaneous pseudolymphomas may be divided into various subtypes according to the clinical and histopathological features. Although there is not a common consensus about the classification of clinicopathological subtypes, cutaneous pseudolymphomas in this article are mentioned as cutaneous lymphoid hyperplasia, Kimura's disease, angiolymphoid hyperplasia with eosinophilia, Castleman disease, pseudo mycosis fungoides, lymphomatoid contact dermatitis, and Jessner's lymphocytic infiltration of the skin. The distinction of cutaneous pseudolymphomas from cutaneous lymphomas is very important. Because there are remarkable differences in the aspect of clinical course, prognosis and therapy modalities.

Introduction

Cutaneous pseudolymphoma refers to a heterogeneous group of benign reactive T or B cell lymphoproliferative processes that simulate cutaneous lymphomas clinically and/or histologically [**1**]. The inflammatory infiltrate is bandlike, nodular, or diffuse. It is composed predominantly of lymphocytes with or without other inflammatory cells [**2**]. Depending on the predominant cell type in the infiltrate, cutaneous pseudolymphomas are divided into two major categories.

1.Mixed B and T cell pseudolymphoma.

2. T cell pseudolymphoma [**1**]

The clinicopathologic subtypes of mixed B, and T cell type consist of cutaneous lymphoid hyperplasia, *Kimura*'s disease, angiolymphoid hyperplasia with eosinophilia, *Castleman* disease.

On the other hand, T cell pseudolymphomas consist of pseudo mycosis fungoides, lymphomatoid contact dermatitis, *Jessner*'s lymphocytic infiltration of the skin (**Table 1**).

CLINICOPATHOLOGIC SUBTYPES

Cutaneous Lymphoid Hyperplasia

Cutaneous lymphoid hyperplasia has a world-wide distribution and affects all races and ethnic groups. It occurs in both adults and children. Females are more commonly affected than males.

Cutaneous lymphoid hyperplasia is characterized by a relatively dense lymphoid infiltrate, centered in the reticular dermis, that is usually B-cell rich and may resemble lymphoma clinically and/or histopathologically.

Although many terms such as lymphocytoma cutis, cutaneous lymphoplasia are used to refer to this type of pseudolymphoma, cutaneous lymphoid hyperplasia is the preferred term because it accurately describes the underlying pathophysiology of the lesion.

In most cases, cutaneous lymphoma is idiopathic; however, some lesions are associated with exposure to foreign antigens from arthropods (bites, stings, infestation) or infections (*Herpes zoster*, *Borrelia burgdorferi*) or tattoos, acupuncture, gold jewellery, trauma, vaccinations, and medications [**1, 3**].

Medications that may induce cutaneous lymphoid hyperplasia could be drugs such as phenytoin, carbamazepine, phenobarbital, β-blockers, calcium channel blockers, angiotensin converting enzyme inhibitors, allopurinol, D-penicillamine, penicillin, cyclosporine, antidepressants and antihistaminic drugs.

It has been proposed that, rather than being the target of an immune response themselves, these latter agents may alter lymphocytes in a way that promotes a cutaneous lymphoid hyperplasia response to other antigens in some individuals [**4, 5**].

When approaching to the patient with cutaneous pseudolymphoma and/or cutaneous lymphoma, the clinical history should elicit information about the duration and symptomatology of lesions, the nature and pace of clinical progression, past treatment, local and systemic exposure to foreign antigens, including medications and personal or family history about other lymphoproliferative disorders. It should also include review of systems focusing on so called lymphoma B symptoms, such as fever of unknown origin, unexplained weight loss, night sweating, fatique and malaise. A general physical examination is important with special attention to the type and distribution of skin lesions and to the status of peripheral lymph nodes, liver and spleen. However, the scanning tests such as a complete blood count, routine biochemistry screening and chest radiography help to exclude extra cutaneous involvement. There can still be uncertainty as to whether the lymphoid infiltrate represents a pseudolymphoma or a lymphoma, additional workup could be helpful. This includes computed tomography of the chest, abdomen and pelvis, as well as bone marrow aspiration and biopsy. Biopsy can be performed from abnormally enlarged lymph nodes [**1, 6**].

Lesional skin biopsy is an essential part of the diagnostic evaluation. The use of topical and systemic corticosteroids should be discontinued approximately four weeks before biopsy if possible, because these agents can attenuate to lymphoid infiltrate and thereby confuse their interpretation. Skin specimens should be large enough and deep enough to routine histopathologic, immunologic studies. If there is only a solitary lesion, it is generally best to obtain all biopsy material at one time, because inflammatory changes induced by the initial biopsy may interfere with the interpretation of subsequent specimens from the same localization.

The cutaneous lesions of cutaneous lymphoid hyperplasia present most commonly as a solitary nodule. However, it can appear as a localized array of nodules, papules and plaques.

Generalized forms occur rarely. The head, neck, extremities, breast and genitalia are common predilection sites.

Lesions have a doughy to firm consistency and range from red- brown to violaceous in color. Lesions may be pruritic or asymptomatic [**7, 8**].

Hydantoin associated pseudolymphoma syndrome is caused by anticonvulsant drugs like phenytoin. This syndrome is characterized by fever, lymphadenopathy, hepato-splenomegaly, arthralgia, eosinophilia and generalized cutaneous macules and papules or rarely nodules.

The macular and papular lesions of this syndrome share histopathologic features with those of hypersensitivity reactions to other drugs.

Acral pseudolymphomatous angiokeratoma of children presents as an unilateral eruption angiomatous papules on the extremities. This disorder is probably a variant of cutaneous lymphoid hyperplasia secondary to arthropod bites.

Large cell lymphocytoma, originally reported as a cutaneous pseudolymphoma, most likely

Table 1. Classification of Cutaneous Pseudolyphomas

Clinicopathologic Subtype	Predominant Lymphoid Subset	Predominant Localization	Major Associated Findings
Cutaneous lymphoid hyperplasia	B and T cell	Reticular dermis	-
Kimura's disease	B and T cell	Subcutis	Lympadenopathy
Angiolymphoid hyperplasia with eosinophilia	B and T cell	Reticular dermis	Eosinophilia
Castleman disease	B and T cell	Subcutis	Lympadenopathy, POEMS syndrome
Pseudo mycosis fungoides	T cell	Papillary dermis and epidermis	-
Lymphomatoid contact dermatitis	T cell	Papillary dermis and epidermis	Contact allergen
Lymphocytic infiltration of the skin (*Jessner*'s disease)	T cell	Perivascular and periadnexial dermis	-

represents a mixed cell and large cell infiltration form primary cutaneous B cell lymphoma.

Histopathologic examination of the cutaneous lymphoid hyperplasia lesions reveals dense, nodular and diffuse lymphoid infiltrate that is concentrated in the reticular dermis. In most cases, the epidermis is normal and separated from the underlying infiltrate by a narrow grenz zone of uninvolved papillary dermis. Histopathologically, the lesions of cutaneous lymphoid hyperplasia include generally a mixed B and T cell lymphocytes. However, various types of histiocytes, including macrophages, dermal dentritic cells, Langerhans cells are scattered throughout the infiltrate. Other cells are sometimes admixed, including plasma cells, eosinophils, mast cells, neutrophils and histiocytic giant cells. Plasma cells and eosinophils are particularly common in arthropod induced reactions. B cells may be organized into primary or secondary lymphoid follicules. Whereas, there are most commonly T cell lymphocytes in T cell cutaneous lymphoid hyperplasia.

Cutaneous pseudolymphomas having an increased risk of developing various forms of overt lymphomas have been reported. Therefore, a principal challenge in the differential diagnosis of cutaneous lymphoid hyperplasia is its distinction from cutaneous B cell lymphoma. As well, leukemia cutis should be ruled out. Differentiation between T cell cutaneous lymphoid hyperplasia and T cell lymphomas relies on the fact that most cases of mycosis fungoides exhibit marked epidermotropism.

Chronic cutaneous lupus erythematosus, *Jessner*'s lymphocytic infiltration of the skin, granuloma faciale are the other diseases that should be considered in the differential diagnosis. Furthermore, infrequently, polymorphous light eruptions, metastatic carcinoma, *Merkel* cell carcinoma, histiocytomas should be in your mind.

Cutaneous lymphoid hyperplasia lesions may resolve spontaneously or persist indefinitely. Nodular scabies is a well example for persistent cutaneous lymphoid hyperplasia.

In the treatment of cutaneous lymphoid hyperplasia related to infection with *B. Burgdorferi*, antibiotic therapy with cephalosporins could be used, as do some cases of idiopathic cutaneous lymphoid hyperplasia. Excision, glucocorticoids (topical, intralesional, and systemic), cryotherapy, antimalarials, minocycline and radiation therapy have all been used with various successes. Laser therapy and photodynamic therapy have also been beneficial in some cases [**1, 8, 9, 10, 11**].

Kimura's Disease and Angiolymphoid Hyperplasia with Eosinophilia

Kimura's disease and angiolymphoid hyperplasia with eosinophilia most commonly affect young to middle aged adults. *Kimura*'s disease is more common in Asian men, whereas angiolymphoid hyperplasia with eosinophilia is more common in women.

It is possible that *Kimura*'s disease represents a florid, subcutaneously deep located form of the same basic pathogenetic process that gives rise to classic dermal cutaneous lymphoid hyperplasia. There is controversy regarding whether *Kimura*'s disease and angiolymphoid hyperplasia with eosinophilia are variants of

the same disorder, although most favor the concept that they are distinct clinicopathologic entities despite some clinicopathologic overlap. Some regard angiolymphoid hyperplasia with eosinophilia essentially as a malformation of blood vessels caused by an underlying arteriovenous shunt. They consider the cutaneous lymphoid hyperplasia like an aspect of lesional infiltrate to be a secondary feature [**12, 13**].

Kimura's disease presents as solitary or multiple nodules up to 10 cm in diameter centered in the subcutis, most commonly involving the head and neck. Peripheral eosinophilia and regional lymphadenopathy are characteristic features. Angiolymphoid hyperplasia with eosinophilia tends to present with smaller, more superficial intradermal papulo-nodules that are typically unilateral. Salivary glands, lymph nodes and other cutaneous sites can also be affected in either disorder, although such localizations are more typically of *Kimura*'s disease.

In general, lesions are more superficial and the vascular features are more prominent in angiolymphoid hyperplasia with eosinophilia, whereas lesions are deeper and lymphoid features are more prominent in *Kimura*'s disease [**1, 14**].

Kimura's disease, which generally forms deeper, larger lesions, needs to be distinguished from cutaneous B cell lymphomas, sinus histiocytosis with massive lymphadenopathy, soft tissue tumors, and cutaneous metastatic carcinomas. Angiolymphoid hyperplasia with eosinophilia, which has smaller, more superficial lesions, should be differentiated from cutaneous lymphoid hyperplasia, cutaneous B cell lymphomas, hemangioma, angiosarcoma, pyogenic granuloma, nodular *Kaposi*'s sarcoma, bacillary angiomatosis and bartonellosis.

Kimura's disease has been associated with lichen amyloidosis and renal disorders like nephrotic syndrome.

Excision, topical corticosteroids (high potency), intralesional corticosteroids (5-40 mg/ml, monthly) have been used in the treatment as first line.

In second line, topical tacrolimus ointment, systemic corticosteroids (60/ 40/ 20 mg PO tapers, 5 days each), cyclosporine (2.5- 4 mg/kg/day), local radiation, vinblastine (15 mg/week IV), intravenous immunoglobulins can be used [**15, 16, 17**].

Castleman Disease

Castleman disease is a polyclonal lymphoproliferative disorder of unknown etiology. There are two variants of this disease. The hyaline vascular variant is more common in younger patients, whereas the plasma cell variant tends to occur in older individuals. Studies of multicentric *Castleman* disease demonstrate increased levels of lesional and circulating interleukins 1β and 6, which suggest that cytokine abnormalities may mediate the systemic manifestation of POEMS syndrome (polyneuropathy, organomegaly, endocrinopathy, M protein and skin changes such as hyperpigmentation and hypertrichosis). The plasma cell variant can be associated with this syndrome [**18**].

Castleman disease most often presents as an isolated mediastinal mass, although a multicentric form of the disease also exists. Lesions can be nodal and/or extra nodal. Rarely, it may present as solitary or multiple, subcutaneous or cutaneous tumors in various locations.

Histopathologically, the more common hyaline vascular type exhibits small lymphoid follicles surrounded by small lymphocytes arranged in a concentric, onion skin pattern. An extensive proliferation of capillaries is present between follicles. The rarer plasma cell type exhibits large hyperplastic secondary lymphoid follicles associated with highly vascular interfollicular zone rich in plasma cells [**1, 19**].

The differential diagnosis of *Castleman* disease includes angiolymphoid hyperplasia, Kimura's disease, plasmacytoma, myeloma, sinus histiocytosis, cutaneous lymphoid hyperplasia, cutaneous B cell lymphoma, non-mycosis fungoides T cell lymphomas.

Castleman disease has been associated with paraneoplastic pemphigus, vasculitis and peliosis hepatitis. Lymphomas and dentritic cell sarcomas can develop in the course of *Castleman* disease.

In the treatment of *Castleman* disease, surgical excision is effective therapy for localized lesions. Radiation therapy and chemotherapy have been used to treat multicentric variants [**20, 21**].

Pseudo Mycosis Fungoides

Eruptions mimicking mycosis fungoides occur typically in adults. Both genders can be affected. Pseudo T cell lymphomas may arise spontaneously or may occur in association with B cell chronic lymphocytic leukemia, as a rare variant of the eruption of the lymphocyte recovery or in association with ingestions of various drugs, including hydantoins, carbamazepine and antihistamines.

The lesions present clinically as one or a few plaques on the trunk or extremities. Occasionally, several plaques or a *Sezary*-like syndrome develop [**1**].

Histopathologically, there is a papillary dermal, band-like infiltrate containing mostly atypical lymphocytes with clefted and cerebriform nuclei. Compared with mycosis fungoides, epidermotropism is typically far less prominent and *Pautrier*'s microabscess-like aggregates are rare. Most cases contain polyclonal T cells, although occasional cases exhibit dominant T cell clonality. In the differential diagnosis of pseudo mycosis fungoides, lichenoid drug eruptions, lymphomatoid contact dermatitis, actinic reticuloid, follicular mucinosis, chronic radiodermatitis, seconder syphilis should be considered.

Severe pseudo mycosis fungoides eruptions such as erythroderma may be associated with serious complications, including disrupted skin barrier function, sepsis, cardiovascular imbalance.

In the first line of the therapy; drug discontinuation when a drug is blaimed, treatment of underlying disorder when an associated specific disease is detected, excision for isolated lesions, topical and systemic corticosteroids, ultraviolet B phototherapy and PUVA therapies are suggested [**22, 23**].

Lymphomatoid Contact Dermatitis

Lymphomatoid contact dermatitis is a chronic and persistent allergic contact dermatitis histologically similar to mycosis fungoides.

Lymphomatoid contact dermatitis has been observed in adults of both genders. Lymphomatoid contact dermatitis was described originally in four patients with persistent allergic contact dermatitis proven by patch testing. Responsible agents include gold, nickel and para-phenylenediamine. Skin lesions are generally pruritic [**24, 25**].

Lymphomatoid contact dermatitis is characterized by generalized red, scaly papules and plaques and this situation may become confluent with resultant exfoliative erythroderma.

Histopathologically, lymphomatoid contact dermatitis exhibits superficial lymphocytic dermatitis that contains foci of spongiosis simulating the appearance of the cutaneous T cell lymphomas. It must be differentiated from mycosis fungoides, usually on the basis of changes within the epidermis; specifically, in mycosis fungoides more atypical lymphocytes have a tendency to form *Pautrier*'s microabscesses. Frequently there is edema in the papillary dermis in lymphomatoid contact dermatitis, this finding is usually absent in mycosis fungoides [**1, 26**].

In differential diagnosis of the lymphomatoid contact dermatitis; mycosis fungoides and *Sezary* syndrome should be ruled out. However, conventional allergic contact dermatitis, irritant contact dermatitis, drug eruptions, and psoriasis should also be considered.

Avoidance of the responsible allergic agents leads to eventual resolution of the condition.

A search for the offending agent via patch testing is necessary when lymphomatoid contact dermatitis is considered.

In the therapy firstly, elimination of a suspected allergic agent, later topical corticosteroids, pimecrolimus and tacrolimus ointments are suggested [**27, 28, 29**].

Lymphocytic Infiltration of the Skin

Lymphocytic infiltration of the skin was first described by *Jessner*. Thereby, has been also named. Most of the cases occur in middle aged adults. Both genders almost are affected in the equal ratio. However, there is a rare familial variant. The etiology of *Jessner*'s lymphocytic infiltration is occult.

Clinically, the lesions present one or more erythematous plaques or nodules. It is generally localized to only one or a few sites on the face, neck, upper trunk or arms.

Histopathologic examination reveals superficial and deep, perivascular and periadnexal infiltrate of small mature lymphocytes, often with minor admixture of histiocytes, plasmacytoid monocytes and plasma cells. Lymphoid follicles and eosinophils are absent. Immunuhistologic studies have demonstrated a predominantly, HLA-DR-Leu-8+ T cell infiltration with some B cells and histiocytes admixed. In addition, immunohistologic studies have shown a predominance of CD8 T cells within lesional infiltrates [**30, 31**].

The differential diagnosis of *Jessner*'s lymphocytic infiltration includes chronic cutaneous lupus erythematosus, polymorphous light eruption, cutaneous lymphoid hyperplasia, lymphocytic lymphoma, leukemia cutis, granuloma faciale, erythema chronicum migrans. *Jessner*'s lymphocytic infiltration has a chronic course and may show periods of spontaneous remission and eventual resolution.

In treatment of *Jessner*'s lymphocytic infiltration; topical corticosteroids, topical tacrolimus and pimecrolimus, intralesional steroids, photo protection (if historically relevant), systemic corticosteroids, antimalarials, thalidomide, and acitretin may be effectively used [**32, 33**].

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