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Lymphocytic infiltrations of face

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Abstract The immune system protects our organism and, of course, our skin from harmful factors. One of the key elements of the immune system is lymphocytes. Lymphocytes play a role in the pathogenesis of various skin diseases. Lymphocytic infiltrates are seen in many skin diseases. Some of the skin diseases characterized by lymphocytic infiltration show up in specific anatomic locations, whereas other entities can be placed in all areas of the body. The course of lymphocytic infiltrations of the face is variable and unpredictable, most often lasting from months to years. The most important diseases with lymphocytic infiltration of the face are pseudolymphomas. This review discusses various types of cutaneous pseudolymphomas and other diseases with lymphocytic infiltration mainly involving the face.

Introduction

The immune system protects our organism and, of course, our skin against harmful factors. The immune system is always on the alert to stay healthy. The lifelong, ongoing struggle for protection is often unnoticed, but it is remarkable in the case of sickness. One of the key elements of the immune system is lymphocytes. Lymphocytes play a role in the pathogenesis of various skin disorders.[1]

Lymphocytic infiltrates are encountered in the pathologic examination of many skin diseases. Certain histopathologic findings can be detected in the skin diseases characterized by lymphocytic infiltrations. These findings are types of cell (T, B), whether the cells are of benign or malignant character, the settlement of infiltration, infiltration features (diffuse, nodular, etc), and lack of other cells.[2-5]

Some of the skin diseases characterized by lymphocytic infiltrations show up in specific anatomic locations, whereas others can be placed in all areas of the body. In this article, we aimed to mention pseudolymphomas (PSLs) first and other skin diseases characterized by lymphocytic infiltrations involving the face.[2-5]

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Pseudolymphoma

PSL is a heterogeneous group of benign reactive T-cell or B-cell lymphoproliferative processes of diverse causes that simulate cutaneous lymphomas clinically, histologically, or both. PSL is an expansive term, including a number of unrelated disorders that may mimic lymphomas, mainly because of their histologic features. PSL has a worldwide distribution and affects all races and ethnic groups. It occurs in both adults and children. Women are more commonly affected than men. Lesions are usually seen on the face; the neck and back may also be involved (Figure 1).[4,5]

In most cases, PSL is idiopathic; however, some lesions are associated with exposure to foreign antigens from arthropods (bites, stings, infestations), infections (herpes zoster, *Borrelia burgdorferi*), tattoos, acupuncture, trauma, gold jewelry, vaccinations, hyposensitization injections, or medications. Medications that may cause PSL include phenytoin, carbamazepine, phenobarbital, β-blockers, calcium channel blockers, angiotensin-converting enzyme inhibitors, allopurinol, D-penicillamine, penicillin, mexiletine chloride, cyclosporine, and agents that inhibit the binding of histamine to H_1, H_2, or H_{1c} receptors. These drugs include conventional H_1 and H_2 antagonists, as well as tricyclic and nontricyclic antidepressants and phenothiazines (Tables 1 and 2).[6]

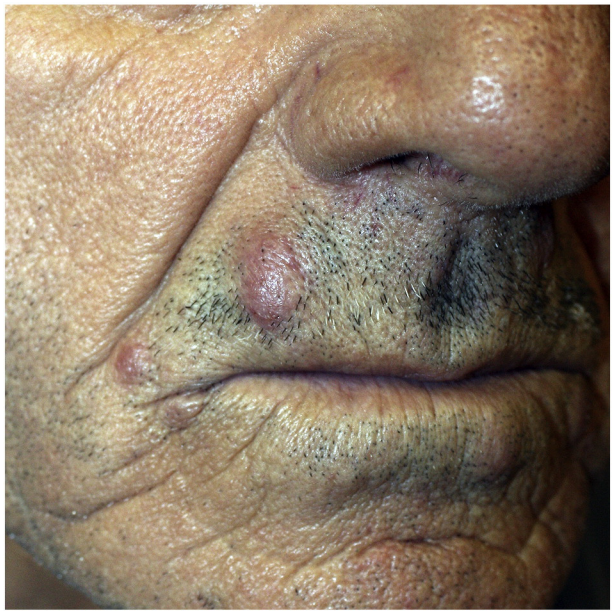

Fig. 1 Pseudolymphoma: nodular and papular lesions localized on the face.

The inflammatory infiltrate is bandlike, nodular, or diffuse and is composed predominantly of lymphocytes with or without other inflammatory cells. Depending on the predominant cell type in the infiltrate, just as with lymphomas, PSLs are usually divided into B-cell and T-cell PSLs. These disorders often show broad patches and plaques, and often mimic cutaneous T-cell lymphomas. Patients with a B-cell pattern PSL present with a nodule or a group of discrete nodules and occasionally complain of pruritus. This distinction is not always possible, however. Lymphomatoid infiltration of both types can be observed in some diseases, such as lymphomatoid drug eruptions.[5]

Although there is no internationally accepted classification, cutaneous T-cell PSLs include idiopathic cutaneous T-cell PSL, lymphomatoid drug reactions, lymphomatoid contact dermatitis (LCD), persistent nodular arthropod-bite reactions, nodular scabies, actinic reticuloid (AR), and lymphomatoid papulosis. Cutaneous B-cell PSLs include idiopathic lymphocytoma cutis, borrelial lymphocytoma cutis (BLC), tattoo-induced lymphocytoma cutis, postzoster scar lymphocytoma cutis, and some persistent nodular arthropod-bite reactions.[5]

Table 1 Causes of pseudolymphomas[5]

Drugs	See Table 2
Foreign agents	Tattoo dyes, insect bites, scabies, injections of arthropod venom, vaccinations, hyposensitization injections, contactants, trauma, acupuncture, gold pierced earrings
Infections	*Borrelia burgdorferi*, varicella-zoster infection, HIV infection, *Leishmania panamensis*
Photosensitivity	Idiopathic (immune-mediated of unknown cause)

Table 2 Drugs implicated in pseudolymphoma[5]

Class	Drugs
Anticonvulsants	Phenytoin, carbamazepine, mephenytoin, trimethadione, phenobarbital, primidone, butabarbital, methsuximide, phensuximide
Antipsychotics	Chlorpromazine, thioridazine, promethazine
Angiotensin-converting enzyme inhibitors	Captopril, enalapril, benazepril
β-Blockers	Atenolol, labetalol
Calcium channel blockers	Verapamil, diltiazem
Diuretics	Moduretic, hydrochlorothiazide
Cytotoxics	Cyclosporine, methotrexate
Antirheumatics	Gold, salicylates, phenacetin, D-penicillamine, allopurinol, nonsteroidal anti-inflammatory drugs
Antibiotics	Penicillin, dapsone, nitrofurantoin
Antidepressants	Fluoxetine, doxepin, desipramine, amitriptyline, hydrochloride, lithium
Anxiolytics	Benzodiazepines (clonazepam, lorazepam)
Antihistamines	Diphenhydramine
H_2 antagonists	Cimetidine, ranitidine
Antiarrhythmics	Mexiletine chloride, procainamide
Topical agents	Menthol, etheric plant oil
Sex steroids	Estrogen, progesterone
Lipid-lowering agents	Lovastatin

Lymphomatoid contact dermatitis

LCD is a chronic and persistent allergic contact dermatitis histologically similar to mycosis fungoides (MF). It was initially described in a study[6] reported in 1976 in four patients with persistent allergic contact dermatitis proven by patch testing. The patients were urged to avoid potential allergens and all later reported complete resolution of their lesions.[5,6]

Clinically, there are pruritic generalized, discrete and confluent, erythematous, scaly, papules and plaques. An exfoliative erythroderma may occur. Results of patch tests for a variety of allergens may be positive. At least 15 cases of LCD currently have been reported, and responsible allergens could be metals, ethylenediamine dihydrochloride, paraphenylenediamine, and isopropyl diphenylenediamine.[5-10]

The histopathology of T-cell LCD resembles MF and shows a superficial bandlike T-cell infiltrate with epidermotropism. Epidermal spongiosis or spongiotic microvesiculation may be present, and this helps to differentiate it from MF, where there may be spongiosis, but not microvesiculation.