CASE REPORT

Jessner's Lymphocytic Infiltration of the Skin – A Case Report and Discussion of Current Literature

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Summary
Jessner's lymphocytic infiltration of skin is an uncommon disorder characterised by benign accumulations of lymph cells in the skin. It is of unknown aetiology and presents as erythematous papules and plaques on the face, neck and/or back, with possible itching of the skin surrounding the lesions. The lesions may remain unchanged for many years and then spontaneously disappear, without residual scarring. Current literature on this topic are limited and numerous treatments have been tried with limited success. We report a recent case of Jessner's lymphocytic infiltration seen in our practice and discuss the histology and our approach to management.

Key words: Jessner's lymphocytic infiltration

Case Report

A 41-year-old woman presented to our clinic in December 2016 with three years of erythematous papules and plaques on her face. She denied any itching but was bothered by her cosmetic appearance. She also did not experience any photosensitivity. She had a past history of Sjogren's disease with minimal joint symptoms and was anti Ro positive. This was previously treated with oral steroids, doxycycline and at presentation was on a maintenance dose of hydroxychloroquine at 200mg per day for the previous 1 year.

Clinical examination showed multiple erythematous infiltrated papules and plaques with no epidermal changes, predominantly distributed on her chin bilateral cheeks and temporal regions (Figure 1). There was no other lesion on the body and the rest of her physical examination was normal.

Her ANA was negative but her ENA showed Anti Ro positive. All her other blood tests including further connective tissue disease screening (ESR, CRP, C3, C4, CK, Rh factor, anti-dsDNA, lupus anti-coagulant, anti-cardiolipin antibodies) blood film and urinalysis were unremarkable. Phototesting was not performed due to patient preference.

Histopathology of the skin biopsy showed mild superficial perivascular and peri-adnexal

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lymphoplasmacytic cells infiltration with absence of lichenoid reaction (Figure 2-3). Alcian Blue PAS stain showed superficial perivascular and perifollicular mucin deposit. Giemsa stain was negative for mast cells. Jessner's lymphocytic infiltration was the most likely diagnosis given the superficial changes. Immunofluorescence studies were not performed. The histological features of Jessner's lymphocytic infiltration is difficult to distinguish from tumid lupus erythematosus. However tumid lupus erythematosus shows abundant and denser mucin deposition which is not demonstrated in this case.

She was treated initially with an increased dose of hydroxychloroquine (400 mg daily) and Cutivate® cream (fluticasone propionate 0.05%) topically twice a day. Intralesional steroids were not used due to concern about possible atrophy. No new lesion developed but there was only mild improvement after 2 months. Hence oral Methotrexate was started at 15mg/week with folic acid supplementation. She remained stable with overall limited improvement at the time of this publication.

Figure 1. Clinical photo of patient's left cheek

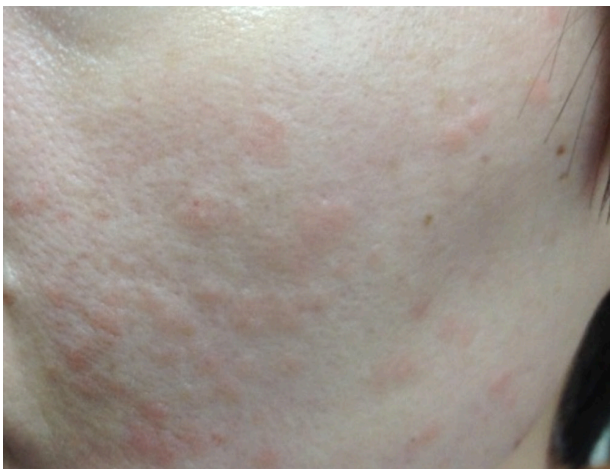


Figure 2. HEx400: Moderate perivascular and periadnexal lymphoplasmacytic cells infiltration in the superficial dermis. Lichenoid reaction at the epidermal basal layer was not seen. (Hematoxylin-eosin stain; original magnification: x400.)

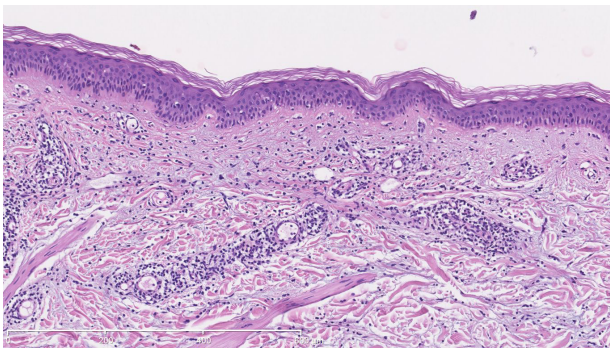
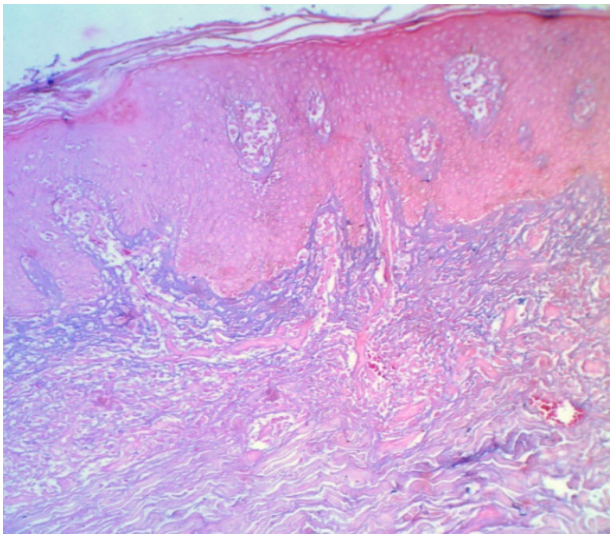


Figure 3. ABPAS stain highlighted the mucin deposit.



Discussion

Jessner's lymphocytic infiltration of the skin is an uncommon disorder. It is a benign chronic T-cell infiltrative disease, first described in 1953 by Jessner and Kanof[1]. The condition may remain unchanged for several years and then disappear spontaneously without residual scarring.

This condition often presents as rosy-red papules, plaques and less commonly nodules mostly on the upper face, cheeks, neck and back. The lesions are non-scaly in nature and may last for several months. They often expand from the perimeter to form well defined, smooth, red plaques. Central clearing may occur. The skin surrounding the lesions may be reddened or itchy. Sensitivity to sunlight may occur but is unusual. Generally the symptoms disappear after several years but they may recur later.[1, 2]

The exact cause of Jessner's lymphocytic infiltration is not known. It has been found that abnormal numbers of lymphocytes accumulate in the skin.[2] There is doubt whether this is a disorder distinct from other benign lymphocytic infiltration of the skin.[3] Some believe that Jessner's lymphocytic infiltration may be a type of lupus erythematosus tumidus (LET) or discoid lupus erythematous (DLE)[3-6]. Symptoms of other disorders can be similar to Jessner's lymphocytic infiltration of the skin and these include lupus erythematosus tumidus, polymorphous light eruption, certain pseudolymphomas, lymphocytoma cutis, mycosis fungoides, leprosy.[2, 3] Evolution to cutaneous or extracutaneous lymphoma is not common although some can progress to discoid lupus erythematosus or are related to lupus erythematosus.[4]

Skin biopsy of a recently formed lesion is very helpful for excluding these other possible conditions. Histopathologically, Jessner's lymphocytic infiltration is characterised by a superficial and deep primarily perivascular, sleeve-like lymphocytic dermal infiltrate with a predominance of small mature polyclonal lymphocytes without epidermal or basal membrane involvement.[4] Jessner's lymphocytic infiltration is sometimes difficult to distinguish histologically from polymorphic light eruption or chronic cutaneous lupus erythematosus. Chronic cutaneous lupus erythematosus can be differentiated by the presence of lichenoid reaction and its involvement of the dermoepidermal junction. Epidermal atrophy and focal thickening of the dermoepidermal junction were found more common in chronic cutaneous lupus erythematosus[3]. Deposits of mucin in the reticular dermis have been described.[3]

Prognosis is good because lymphocytic infiltration of the skin may resolve spontaneously.[2] A period of observation may be reasonable as the lesions may clear spontaneously. However, many patients elect to start treatment early because the lesions are cosmetically unacceptable.

Numerous treatments have been tried with variable success. These include cosmetic camouflage, topical or intralesional steroids, oral hydrochloroquine, systemic steroids, cryotherapy, methotrexate, dapsone, thalidomide, auranofin, phototherapy and pulsed dye laser.[1, 7, 8, 9, 10, 11]. Treatment with anti-malarials is usually effective especially in cases with photo-sensitivity[7].

Conflict of Interest Declaration

The authors have no conflict of interest to declare.

Acknowledgement

The authors would like to thank the Director General of Health, Malaysia for permission to publish this paper.

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