Journal of the Turkish Academy of Dermatology
eISSN 1307-394X

Case Report

DOI: 10.6003/jtad.1372c2

Pityriasis Lichenoides Chronica Following *Cesarean* Delivery

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Published:
J Turk Acad Dermatol 2013; **7 (2)**: 1372c2
This article is available from: http://www.jtad.org/2013/2/jtad1372c2.pdf
Key Words: pityriasis lichenoides chronica, delivery

Abstract

Observations: Pityriasis lichenoides is a papulosquamous disorder with remissions and exacerbations. The aetiology of pityriasis lichenoides is unclear. There are isolated reports of the development of pityriasis lichenoides et varioliformis acuta during pregnancy. But pityriasis lichenoides chronica following cesarean delivery was never reported so far. We present a case of pityriasis lichenoides chronica that occurred within15 days after giving birth to her first child by cesarean section. Pregnancy is a condition with profound endocrine and metabolic alterations that are generally well tolerated by the body. However, it is known to generate a state of humoral and cellular immunosuppression. We have thought that is pityriasis lichenoides chronica following cesarean delivery may be associated with decreased immunosuppression after delivery.

Introduction

Pityriasis lichenoides (PL) is a papulosquamous disorder with remissions and exacerbations. The aetiology of PL is unclear. However, PL is thought by some researchers to represent a hypersensitivity reaction to an infectious agent [**1, 2**]. There are isolated reports of the development of pityriasis lichenoides et varioliformis acuta (PLEVA) during pregnancy [**3, 4, 5**]. However, pityriasis lichenoides chronica (PLC) following cesarean delivery was never reported so far. We presented a case of PLC that occurred following cesarean delivery.

Case Report

A 26–year–old woman presented with multiple, pruritic papules on her trunk, lower and upper extremities. The patient had no history of drug use or infections and was otherwise healthy. She reported that pruritic papules appeared within 15 days after giving birth to her first child by cesarean section, six months ago. While papular lesions

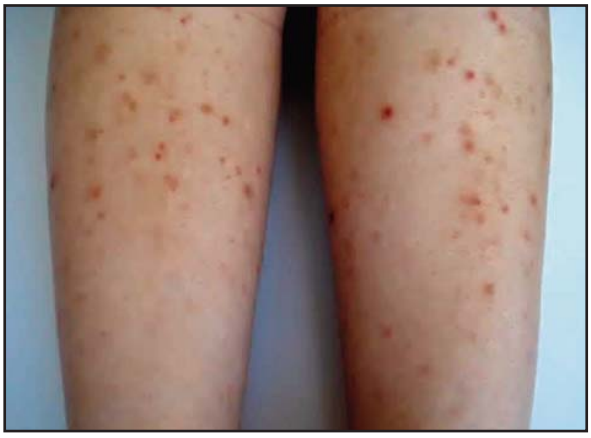

Figure 1. Erythematous papules and hyperpigmented macules on her legs

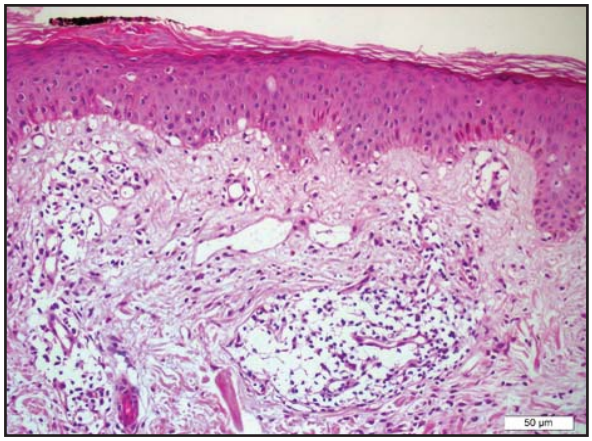

Figure 2. Focal parakeratosis and extravasation of lymphocytes with epidermal invasion; lymphohistiocytic perivascular infiltrate of the dermis (H&E, X200)

heal with letting hyperpigmented macules within several weeks, new papular lesions consisted of another region (**Figure 1**). There was no mucosal involvement. Histopathological findings confirmed the diagnosis of PLC, showing: parakeratosis, acanthosis, and extravasation of lymphocytes with epidermal invasion; edema and lymphohistiocytic perivascular infiltrate of the dermis (**Figure 2**). No laboratory abnormalities were detected; antibodies against the most common etiologic infectious agents were absent. Topical tacrolimus 0.1% ointment and antihistaminic drugs were administered. One month later, a partial resolution of the lesions was observed.

Discussion

PL is an inflammatory skin disorder of unknown aetiology, characterized by a self-limited skin eruption seen in either acute or chronic form. The acuta form of PL is known as PLEVA. The descriptive terms acute and chronic refer to the characteristics of the individual lesions and not the course of the disease [**1, 2**]. PLEVA is frequently characterized by eruptions of pink, orange or purpuric papules that undergo central vesiculation, may ulcerate and resolve with haemorrhagic crusts [**3**]. Symptoms include burning and pruritus. The lesions usually occur on the trunk and flexural areas of the extremities, but generalized eruptions may ocur [**1, 2**]. It is usually self-limiting, disappearing in a few weeks or months [**5**]. Varioliform scars and postinflammatory hyper- and hypopigmentation may result. The primary lesion of PLC is an erythematous papule that acquires a reddish-brown hue and becomes covered by a central adherent micaceous scale that can be easily removed to reveal a shiny, pinkish-brown surface. The papules regress over a period of weeks, often with residual hyper- or hypopigmentation. PLC, a process that may persist for years with frequent recurrences [**1, 2**].

PLC histopathology reveals focal parakeratosis, minimal amounts of necrotic keratinocytes, minimal vacuolar degeneration of the basal layer, edema, mild superficial perivascular lymphohistiocytic infiltrate that only focally obscures the dermoepidermal junction, occasional extravasated erythrocytes [**1, 2**].

The differential diagnosis for PL includes lymphomatoid papulosis (especially), arthropod bite reactions, varicella, *Gianotti–Crosti* syndrome, erythema multiforme, pityriasis rosea, guttate psoriasis, vasculitis, and secondary syphilis. As opposed to PL, lymphomatoid papulosis is characterized by large, atypical, nonlymphoid cells that may resemble *Reed–Sternberg* cells, many neutrophils, few lymphocytes, few or no necrotic keratinocytes, and little or no vacuolar degeneration of the basal layer. Clinically, the papules of lymphomatoid papulosis may develop into nodules, tumors, and large plaques (unlike PL). In contrast to PL, lymphomatoid papulosis is known as a CD30+ lymphoproliferative disorder [**2**].

Pregnancy is a condition with profound endocrine and metabolic alterations that are generally well tolerated by the body [**3**]. However, pregnancy is known to generate a state of humoral and cellular immunosuppression, with serum inhibition of interleukin (IL) 2 formation and IL-1 activation and a reduction in polymorphonuclear cell chemotaxis and adhesion [**6**]. Inflammatory diseases can be triggered by reduction of immunosupression after birth. We have thought that is PLC following cesarean delivery may be associated with decreased immunosupression after birth.

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