

## Localized Pityriasis Lichenoides Chronica: A Brief Report

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### Abstract

**Observation:** Pityriasis lichenoides (PL) is a benign lymphocytic infiltrative skin disease that presents as papulonecrotic, vesicular or papulosquamous lesions. Etiology of the disease is still unclear. It usually occurs in children and lesions are usually located on the trunk and extremities. A few cases have been reported about localised eruption. We present a case, 12 years old girl who has localised eruption on the right thigh for 2 years .

### Introduction

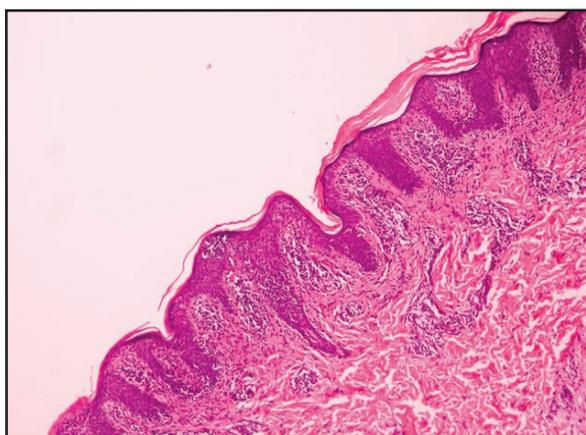
Pityriasis lichenoides is papulosquamous disorder with unknown etiology.

Pityriasis lichenoides can be seen either acute or chronic form. Classification of the disease is determined by eruption morphology and duration [1]. Acute form named pityriasis lichenoides et varioliformis acuta (PLEVA) shows recurrent papular eruptions evolves to vesicular hemorrhagic and necrotic lesions. Other form is pityriasis lichenoides chronica (PLC) is characterized persistent scaling papular eruption [2]. PLC can be de novo or evolve from PLEVA. Two form of the disease can cause hypopigmentation or hyperpigmentation. Scar is rarely seen after chronic form healing. Acute lesions lead deeper destruction in dermis so scar can be seen more than chronic form. Systemic signs such as fever and lymphadenopathy can be present in PLEVA [3]. PLEVA can rarely be lethal form that is mentioned febrile ulceronecrotic

*Mucha-Hebermann* disease differentiated from PLEVA by a rapid progression of necrotic papules to large coalescent ulcers with necrotic crusts, hemorrhagic vesicles and pustules [4, 5]. PLC be more seen tree to six fold than PLEVA [6]. Pityriasis lichenoides le-



**Figure 1.** Pityriasis lichenoides chronica erythematous macules and papules, hypopigmented macules on the right thigh



**Figure 2.** Hematoxylin-eosin, x 10, hyperkeratosis, lymphocytic infiltration with lichenoid pattern and extravasated

sions usually located on the trunk and proximal extremities but anywhere on the skin or even oral mucosa can be included [6].

### Case Report

A 12-years-old girl presented with history of skin rash had been present for two years. Patient's parents referred to different clinics and she was used topical corticosteroids, oral antihistaminics and emolient without any benefit. Systemic organ examination was normal. In dermatological examination there was 3- 5 mm diameter erythematous macules and papules that surfaces minimal scaly. All these papules were monomorf and located on the medial side of right thigh. There were hypopigmentary macules without any atrophy (**Figure 1**). She has no complain only slight pruritus. Results of Hematologic and blood biochemical analyses were normal.

One of the papule was taken for histopatological investigation with 4 mm punch device. Histologic findings in lesion, hyperkeratosis, lymphocytic infiltration with lichenoid pattern and extravasated erythrocytes in upper layer of epidermis, irregular acanthosis, and spongiosis (**Figure 2**).

With all these finding and clinical presentation suggested pityriasis lichenoides chronica. We started to patient systemic erythromycine 500 mg twice daily and topically mild corticosteroid over one month. Lesions persisted during this therapy and erythromycine treatment was changed to tetracycline 250 mg twice daily PO. The lesions remained localized to inner thigh during treatment. The patient was out of follow up after 6 months.

### Discussion

Even first case of about the pityriasis lichenoides came from 19<sup>th</sup> century, nature of the disease has not been known well. PL may be part of clonal T-cell cutaneous lymphoproliferative disorders. Rare case reports exhibited PL evolve into T-cell lymphoma supported this theory [7, 8]. Direct immunofluorescence (DIF) examination in PL lesions can show vascular of IgG, C3 fibrin deposition around blood vessels and dermoepidermal junction that caused PL is mentioned in vasculit chapter in some textbooks. Inflammatory respond from to infectious agent or a T-cell dyscrasia and immun complex-mediated hypersensitivity are other probably mechanism of disease [7, 8]. There is clonal proliferation of cytotoxic memory T cells as a response for antigens stimulus. Now PL is considered as benign lymphoproliferative disease by most of authors [2, 7, 8].

Skin lesions of the PL have predilection to trunk and flexural regions of extremities. Most patients are presented with generalized eruption. Sometimes skin lesions can affect only truncal area or only extremities [9]. *Gelmetti* et al classified PL as central (face, trunk and inguinal involvement), peripheral (extremities, palm and sole involvement) and diffuse (both truncal and extremities involvement) [9]. *Weinber* et al investigated 27 PL cases, found out that 16 patient truncal distribution, 16 patient extremities, 7 patient generalized, one patient only upper trunk and one patient only buttocks and thighs [2]. *Wahie* et al noted that most frequently affected area is upper limbs over % 90 in all age group and face and acral regions is less effected area between %5-%10 [10].

In the literature, there are few cases that have been reported with localized PL. First localized PL was 50 years old men with lesions on the left lateral trunk reported by *Cliff* et al in 1996 [11]. A women 62 years old was reported by *Child* as different localization that 4 years history of lesions on right breast. Repeated biopsy revealed for this women that lesions evolved to cutaneous T-cell lymphoma [12]. *Martin* et al reported 9 years-old boy who had lesions in the lower abdominal region and groin [9]. *Kossard* reported two patients one of them was 66 years old man had right foot localization and the other was 58 years old

women had foot localization [12]. Halbesleben reported 71 years old man with left dorsal foot localization [11]. Totaly three patient was reported as acral localization and three patient central involvement. Histopathological changes in PLC is very similar to PLEVA but lesser degree [7]. Epidermal changes in PLC are focal parakeratosis, acanthosis, focal spongiosis, minimal amounts of necrotic keratinocytes, minimal vacuolar degeneration of the basal layer, invasion of erythrocytes and lymphocytes. Edema, mild superficial perivascular lymphohistiocytic infiltration and scarce extravasated erythrocytes dilatation of superficial vessels can be seen in dermis [3, 5].

The treatment modalities for PL are phototherapy, systemic antibacterials, topical and systemic corticosteroids. In addition immunosuppressants and immunomodulating agents are recommended for severe case [13].

PL can be confused with other diseases as guttate psoriasis, pityriasis rosea, arthropod bite reactions, lichen planus, and secondary syphilis [11]. In our research, this case is seventh patient describe as localised PL in the literature. Biopsy is necessary to differentiate to other papulosquamous disease. In case of localized papulosquamous eruptions, we must add to PL to list of differential diagnosis.

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