CASE REPORT

Lichen striatus and pityriasis lichenoides chronica in an 11-year-old girl: An etiologic relationship?

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Abstract

Lichen striatusis a rare linear papulardermatosis that primarily occurs in children. The lesions have a linear distribution following Blaschko's lines. Pityriasis lichenoides is an uncommon benign skin disorder with two major variants: acute and chronic.Herein, we report the case of an 11-year-old girl with concurrent pityriasisli chenoides chronica and lichen striatus, a previously unreported association. Although it remains unclear whether there is an aetiological relationship between the two diseases or whether their coexistence was coincidental in our patient, but some common mechanisms may be involved in the two diseases.

Keywords: Lichen striatus, Pityriasislichenoides chronica, Infection.

Introduction

Lichen striatus (LS) is a benign, self-limited, linear, inflammatory dermatosis of unknown aetiology. It usually affects children between the ages of 5 and 15 years.[1] It characteristically appears as flat-topped scaly erythematous papules that cluster in a continuous or interrupted linear configuration. This pattern corresponds to the lines of Blaschko. The lesions are typically solitary and unilateral, occurring more frequently on the limbs. Onset is usually sudden, with the disease progressing over days or weeks and slowly decreasing spontaneously within 6-12 months. Post-inflammatory hypopigmentation may last months to years.[1-3]

The aetiology of LS is still unknown, but the most widely accepted hypothesis is that it involves a combination of genetic and environmental factors. It is believed that lines of Blaschko indicate somatic mosaicism reflecting the distribution of clones of abnormal keratinocytes during development.[1]

Pityriasis lichenoides (PL) is an uncommon benign skin disease consisting of two variants: acute PL (pityriasis lichenoides etvarioliformisacuta [PLEVA]) and chronic PL (PL chronic [PLC]). PLEVA and PLC are polar ends of a clinicopathological spectrum rather than two independent diseases. PLEVA tends to present more acutely with papulovesicles that may develop necrotic, ulcerative, or haemorrhagic changes. PLC is characterised by small, scaly papules with no evolution to necrotic lesions.[1,4]

Although the exact pathogenesis of PLC is not understood, but several possible theories exist, including the hypothesis that the disease may represent an inflammatory response to an infectious agent or an immune complex-mediated hypersensitivity vasculitis. In addition, monoclonal T cells have been found in some cases of PLC and many cases of PLEVA, suggesting that the disorders, though not true cutaneous T cell lymphomas, represent inflammatory reactions to an underlying T cell dyscrasia.[4,5]

Herein, we report the case of a young girl with concurrent PLC and LS, which is a previously unreported association.

Case Report

An 11-year-old girl presented with an eight-week history of pruritic, linear erythematosus papular eruption on the right arm associated with simultaneous erythematous papules with overlying scale on the trunk (Figures 1a, 1b).The lesions of the arm followed Blaschko's lines. About three weeks before the manifestation of dermatoses, the patient had suffered from a varicella infection. At the time of presentation, some disseminated hypopigmented scars were visible on the trunk .The patient had no history of atopy or other medical problems and was taking no medications.

On examination, she had linearly arranged erythematous papules on the right arm following the lines of Blaschko and numerous erythematous to reddish brown papules with overlying mika like scale on the trunk. No mucosal lesions or nail involvement was detected. A skin biopsy taken from the arm showed epidermal hyperkeratosis, focal parakeratosis, exocytosis, diffuse lysis of the basal layer, band-like lymphocytic infiltration of the dermis, lymphocytic infiltrate around the hair follicle, and scattered melanin incontinence (Figure-2a). Based on

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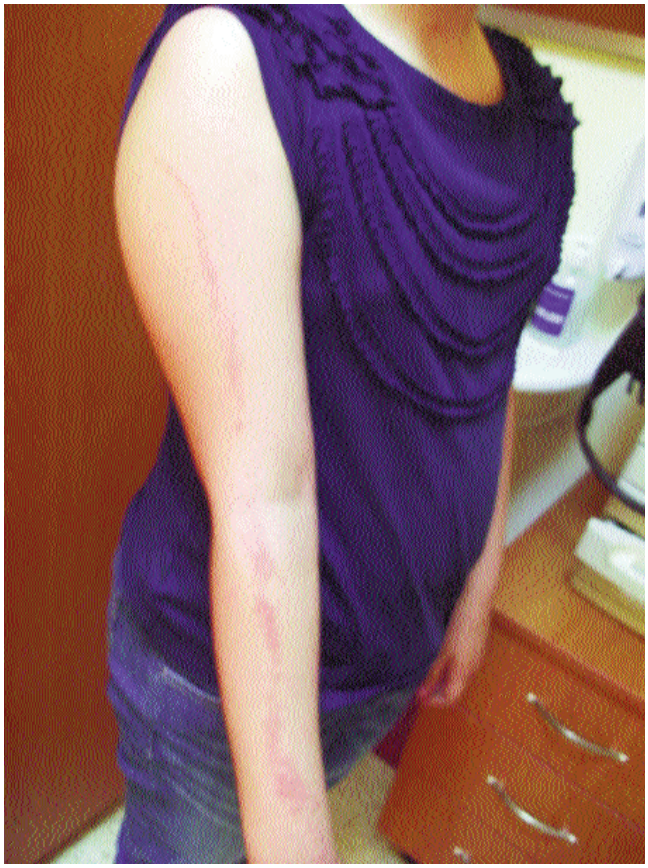

Figure-1a: Linearerythematous papulereruption along the Blaschko line on the right arm.

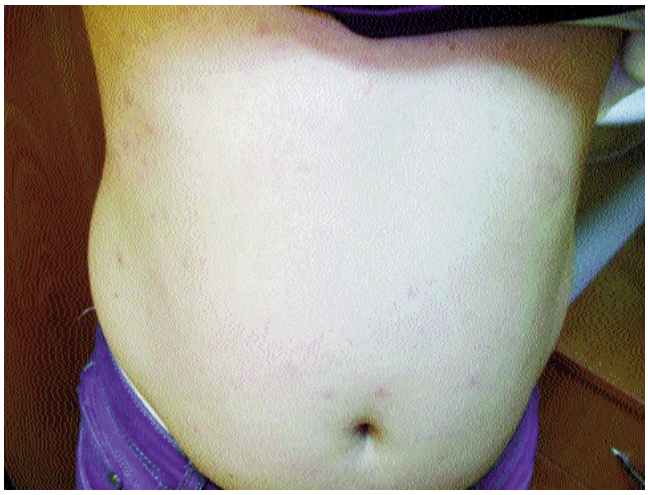

Figure-1b: Papulosquamous eruption on the trunk.

both clinical and histological features, she was diagnosed as LS.

A second skin biopsy from the trunk showed focal

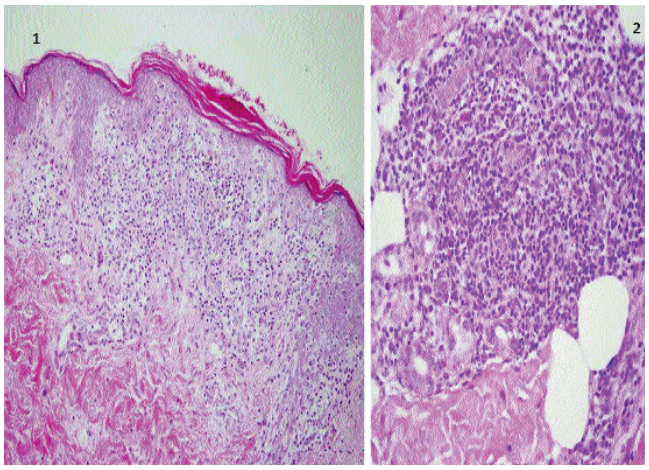

Figure-2a: 1)Hyperkeratosis, focalparakeratosis, perivascular lymphocytes infiltrate in the dermis (Hematoxylin and eosin stain X200). 2) Lymphosytes infiltrate around the hair follicle (Hematoxylin and eosin stain X 400).

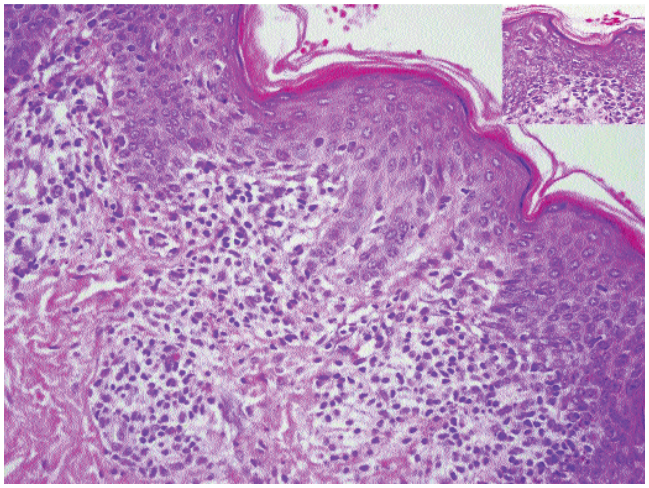

Figure-2b: Focalparakeratosis and perivascular lymphocytic infiltration in the upper dermis (Hematoxylin and eosin stain X 400). Small Image: Basal vacuolar degeneration (Hematoxylin and eosin stain X1000).

hyperkeratosis, parakeratosis, erythrocyte extravasation, basal vacuolar degeneration, and perivascular lymphocytic infiltration in the upper dermis (Figure-2b). This condition was compatible with PLC. The patient was started on treatment with topical corticosteroids. After two months of follow-up, it was seen that the lesions had disappeared, leaving mild hypopigmentation on the arm. However, the lesions on the trunk failed to regress completely.

Discussion

To the best of our knowledge, our patient is the first reported case of concurrent PLC and LS. The aetiology of

LS remains unknown. It has been observed in siblings and in atopic patients, or following immunisation or infection.[6-8] A seasonal preponderance of LS onset has been reported in several studies, suggesting a possible role of infective agents such as viruses in the aetiology. Peaks of LS are observed in spring and summer.[9] Occurrence of LS 10 days after flu-like fever in a pair of siblings and 10 days after tonsillitis has been reported.[6] Descriptions of the simultaneous occurrence of LS in pairs of siblings suggest a role for a common environmental stimulus (e.g. viral infection).[6,8] One study[10] reported a case of LS following varicella infection.

PL is a disease of unknown origin. It is most likely due to induction of a benign reactive proliferation of lymphocytes caused by infectious agents; associations with Toxoplasma gondii, herpes viruses (Epstein-Barr virus, varicella zoster virus [VZV], cytomegalovirus [CMV]), human immunodeficiency virus [HIV], parvovirus B19, and bacterial infections have been reported. The outbreak of the epidemic especially during winter or fall and the onset or flaring of disease with upper respiratory tract infections, streptoccocal pharyngitis and T.gondii infections support the hypothesis of an infective trigger of the disease.[4] However, one study[11] found a history of infection, mostly viral, in 30% patients with PL, although PL appears mostly in the winter or fall.

Our report contributes to the hypothesis of an infective aetiology in both diseases.

Conclusion

Both PL and LS diseases occurred in our patient following varicella infection, which might be a trigger of these diseases. Further research and case reports are needed.

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