

Milia Developed on Lichen Striatus of the Face

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Secondary milia occur by underlying skin diseases, such as bullous diseases and various inflammatory skin diseases. Lichen striatus is a linear papulosquamous disorder mostly on the extremities and trunk, but facial lesions are rare. Herein we describe an 11-year-old girl with lichen striatus on the face, and secondary milia on the lesion later. We suggest that lichen striatus may be one of the causes of secondary milia. (Ann Dermatol 15(4) 160~162, 2003).

Key Words : Milia, Lichen striatus, Face

Milia are small white keratinous cysts, and divided into primary and secondary groups. Secondary milia occur by various underlying skin diseases. Lichen striatus (LS) is a papulosquamous disorder with a linear distribution, and common in children. The linear papules follow Blaschko's lines, mostly on the extremities and trunk, but facial lesions are unusual. They resolve spontaneously with residual hypopigmentation. We report a rare case of milia developed on facial LS, which suggests the association of milia with LS.

CASE REPORT

An 11-year-old Korean girl presented with asymptomatic erythematous grouped papuloplaques with curved band-like distribution on left cheek just lateral to the nose for one year. In addition, a few whitish tiny cysts developed on the surface 1 month before the visit (Fig. 1). They started with small papules and spread along Blaschko's lines on the cheek. She complained of intermittent pruritus, and didn't have any past history of trauma or aggravation after sun exposure. She had a history of

atopic dermatitis and allergic rhinitis. On laboratory findings, there were peripheral eosinophilia (13.2%), and elevated immunoglobulin E level (243 IU/mL). But urinalysis, liver function test, antinuclear antibody (ANA) and chest x-ray were normal or negative.

A skin biopsy specimen showed dense lymphoid cells infiltrates around hair follicles and eccrine glands. Small keratin-filled cysts were observed in upper dermis, and were surrounded by lymphocytic infiltrates (Fig. 2A). On high power view, there were epidermal thinning, few necrotic keratinocytes, vacuolar changes on basal layer, and nodular lymphocytic infiltrations in superficial and deep perivascular area (Fig. 2B). However, there was no dermoepidermal thickening, and direct immunofluorescence (DIF) tests revealed no immune deposits. We diagnosed the lesion as milia on LS and treated with milia extraction and topical corticosteroid cream. The lesion of lichen striatus still remained after follow-up of 2 months, but spontaneous remission was expected.

DISCUSSION

Secondary milia develop in various inflammatory skin diseases such as epidermolysis bullosa, pemphigus, bullous pemphigoid, porphyria cutanea tarda, herpes zoster, and contact dermatitis. Also they can occur after following situations such as trauma of dermabrasion, healed vesicular eruption, long-term corticosteroid use, and 5-FU therapy.

Contrast to primary milia that are derived from the

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Fig. 1. A few whitish tiny cysts on the erythematous grouped papuloplaques with curved band-like distribution on left cheek just lateral to nose.

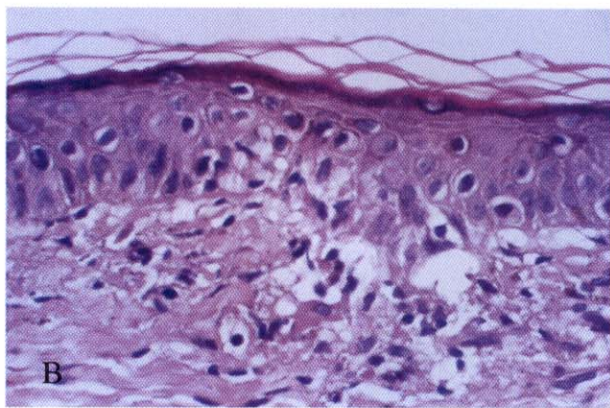


Fig. 2B. Epidermal thinning, few necrotic keratinocytes, vacuolar changes on basal layer, and lymphocytic infiltrates in dermoepidermal junction (haematoxylin and eosin, original magnification $\times 400$).

infundibulum of vellus hairs at the most inferior point, secondary milia are from eccrine sweat ducts, aberrant epidermis or hair follicles¹. So, secondary milia can develop after any skin diseases affecting skin appendages and epidermis. Milia in a plaque-like aggregation within the areas of facial lupus erythematosus (LE) have been reported². Milia formation

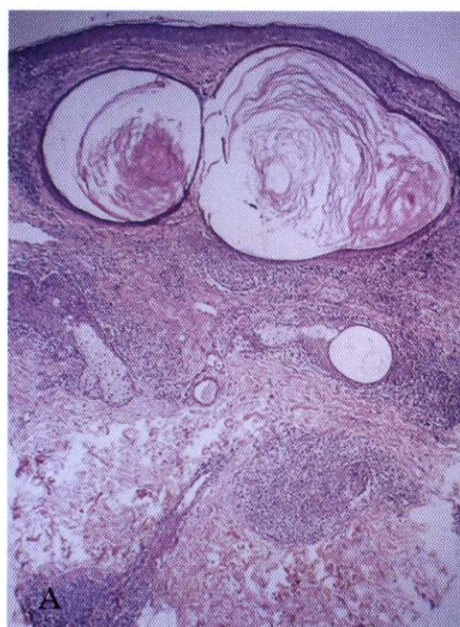


Fig. 2A. Small keratin-filled cysts in upper dermis, and nodular lymphocytic infiltrations in superficial and deep perivascular area. Also dense lymphoid cells around hair follicles and eccrine glands (haematoxylin and eosin, original magnification $\times 40$).

might be secondary to damage to the adnexal structures². Gianotti *et al.*³ reviewed forty-one cases of LS. One of the remarkable histopathologic findings in twenty two classical cases was the tendency of infiltrates in the reticular dermis to be aligned along epithelial structures of hair follicles and eccrine ducts. In our patient, lymphoid infiltrates are denser around hair follicles and eccrine glands. Therefore milia may arise secondarily in inflamed hair follicles.

Lee *et al.*⁴ reported four patients suggesting a peculiar type of LS of the face, which revealed overlapping findings of linear variant of discoid lupus erythematosus (DLE). Linear tiny papular eruptions and negative serological tests were suggestive of LS, but chronicity and improvement with hydroxychloroquine supported linear LE. In our patient, histologic features could be LS or LE, but linear papular lesions, one-year duration of disease, negative ANA and DIF tests, and no central atrophy or scales favor the diagnosis of LS. Linear LE and LS have some similar clinical and histologic features. Childhood DLE is very rare. In 1998, Abe *et al.*⁵ reported two Japanese girls with DLE following the lines of Blaschko of face. One patient had positive ANA (1:80) and DIF, but the other negative

findings. His cases differed from LS in those findings such as positive DIF in one patient, a history of chronic facial lesions, and good response to dapsone. He also reviewed five patients of linear DLE on the face, and ANA was negative in all patients but one. Green *et al.*⁶ reported linear childhood DLE following the lines of Blaschko of face. The patient had bilateral facial and truncal lesions, high ANA titers(1:80). He reviewed four cases of childhood linear LE, and three cases were developed on extremities and one case was on face, but the final diagnosis was LS⁷.

Herein we report an interesting case of secondary milia developing on the LS of face.

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