

was strongly suspected because of the patient's typical mucocutaneous lesions. Therefore, targeted genetic testing for the *endoglin* and *ACVRL1* genes should be performed to establish an early diagnosis if there are characteristic cutaneous features and/or a family history of HHT.

The cutaneous stigmata, which are characterized by an accumulation of small-caliber and superficial vessels, can cause cosmetic concerns and a risk for bleeding^{1,2,5}. In our case, treatment with pulsed dye laser resulted in dramatic improvement of the skin lesions, similar to results reported by Halachmi et al.². Multiple treatments and follow-up visits can be necessary because of the lower response of HHT than non-HHT telangiectasia and the possible accumulation of new vascular lesions².

In conclusion, to initially rule out an HHT diagnosis, genetic study is an available option. Because of the superficial nature of vascular lesions, shorter wavelength vascular lasers such as the pulsed dye laser can be considered an effective and safe treatment option for HHT.

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Lymphoplasmacytic Plaque in Children

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Dear Editor:

Fried et al.¹ reported on a specific disease entity using the term "Pretibial lymphoplasmacytic plaque in children".

Herein, we describe a 3-year-old girl with a 6-month his-

tory of a solitary, asymptomatic, erythematous, scaly plaque with some papules over the right knee (Fig. 1A). She had a clean medical history with no history of travel abroad, injury, or insect bite. The biopsy specimen was

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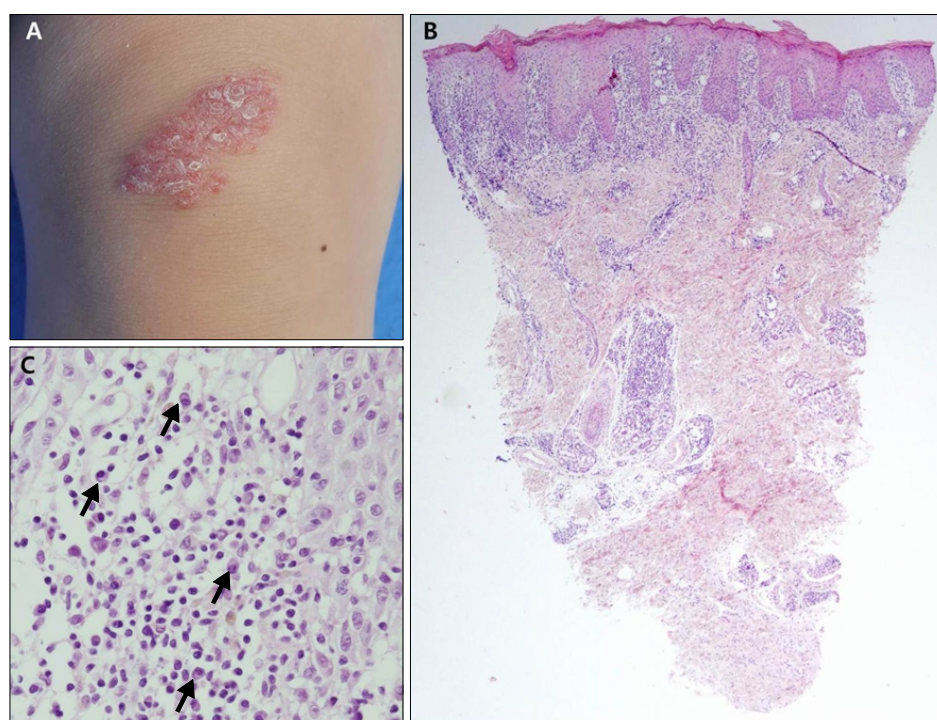


Fig. 1. (A) Solitary reddish scaly plaque on the right knee. (B) Punch biopsy shows dense inflammatory infiltrate in lichenoid arrangement with perivascular and periadnexal extension into the deep dermis (H&E, $\times 40$). (C) Higher magnification revealing the infiltrate is composed of numerous plasma cells admixed with lymphocytes (H&E, $\times 400$).

Table 1. Previously reported cases showing the same findings with lymphoplasmacytic plaque in children

Study	Gender/age (yr)	Ethnicity	Location	Duration	Clinical appearance	Treatment
Gilliam et al. ^{5*}	F/15	Caucasian	Pretibial	11 years	4.5 cm reddish brown violaceous plaque	Pulse-dye laser with partial improvement of the color
	M/7	Caucasian	Pretibial	2 years	Cluster of dark reddish brown papulonodules	
Fried et al. ¹	F/11	Caucasian	Pretibial	5 years	4.0 cm reddish brown plaque	Intralesional steroid injections with partial remission
Ahn et al. ^{2*}	F/7	Asian	Buttock	7 years	3.0 cm erythematous scaly plaque	Topical application of methylprednisolone acetate and tacrolimus hydrate and intralesional steroid injections with partial remission
						Eventually treated with excision
Moulonguet et al. ³	F/5	Caucasian	Pretibial	10 months	3.5 cm reddish scaly plaque	Topicalsteroids (Clobetasol under occlusion) with slight improvement
	M/11	North African	Pretibial	1 year	3.0 cm reddish scaly plaque	Topical steroids with slight improvement
Porto et al. ⁴	F/17	Caucasian	Thigh	>10 years	2.0 cm pink serpiginousplaque	Excision and recurrence free
	F/2	Caucasian	3rd finger	2 years	8 mm erythematous scalyplaque	Observe the lesion while weighing treatment options
Present case	F/3	Asian	Knee	6 months	2.5 cm reddish scaly plaque	Topical steroids (0.3% Diflucortolone valerate ointment) with unsuccessful response and no change in size for a year

F: female, M: male. *Published with another diagnosis, isolated benign primary cutaneous plasmacytosis.

characterized by focal parakeratosis, irregular acanthosis, and dense dermal lymphoid infiltrates, admixed with numerous plasma cells. The plasma cells extended into the deep dermis with a perivascular and periadnexal pattern, as previously reported by Ahn et al.². The specimen also revealed a lichenoid reaction with basal vacuolization and lymphoid exocytosis, similar to those reported by Moulonguet et al.³ and Porto et al.⁴ (Fig. 1B, C).

The immunohistochemical studies showed mixed inflammation of the B-cell and T-cell lymphocytes. Additionally, a polyclonal pattern was confirmed by the co-existence of kappa and lambda light chain-positive cells. No organisms were identified on the acid fast bacilli, Giemsa, and periodic acid-Schiff staining. A serologic test for syphilis and the QuantiFERON-TB Gold (Cellestis Limited, Carnegie, Australia) test both yielded negative results.

Lymphoplasmacytic plaque (LPP) was originally termed "isolated benign primary cutaneous plasmacytosis in children," as it was thought to be a part of the spectrum of primary cutaneous plasmacytosis (PCP)⁵. Several similar cases, which have been reported as LPP or under a different diagnosis, shared common clinical features including a solitary plaque with a predilection for the lower legs and prevalence in the pediatric age group⁴. These features were distinct from those of PCP, which is characterized by multiple red-brown plaques located on the trunk, mainly in adult Asian patients¹. These previously reported cases, which we believe represent LPP, are described in Table 1. Histopathologically, LPP exhibited an irregular acanthosis with overlying parakeratosis and dermal lymphoplasmacytic infiltrates, with or without epithelioid granulomas. The localized inflammation and epidermal hyperplasia may contribute to the papulosquamous morphology of LPP. Clinically, the plaque seemed to be a benign chronic condition, although there was no effective treatment other than complete excision (Table 1).

Moulonguet et al.³ differentiated LPP from acral pseudolymphomatous angiokeratoma of children, suggesting that

these disease entities may reside on the spectrum of pseudolymphomas. Pseudolymphomas can develop after exposure to certain antigens. Interestingly, the histological pattern of a lichenoid inflammation with periadnexal infiltrate was very similar to those of lichen striatus, except for the heavy composition of plasma cells that is important for the diagnosis of LPP. Therefore, given the similarities in the age of onset and the histologic findings, we believe both conditions could be caused by a local reaction to an unknown, acquired stimulus in a specific age group.

In conclusion, although the pathophysiology is still unknown, LPP is a unique disease entity with typical clinical features. When a child presents with this type of plaque on the lower extremities, several disease entities including primary lymphoproliferative disorders, infectious lesions, other reactive infiltrates, or pseudolymphomas need to be considered. A final punch biopsy is necessary to confirm the diagnosis.

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