Case 15.1

Patient: A 54-year-old Thai female from Trang

Chief complaint: Progressive itching rash at upper and lower extremities for 1 month

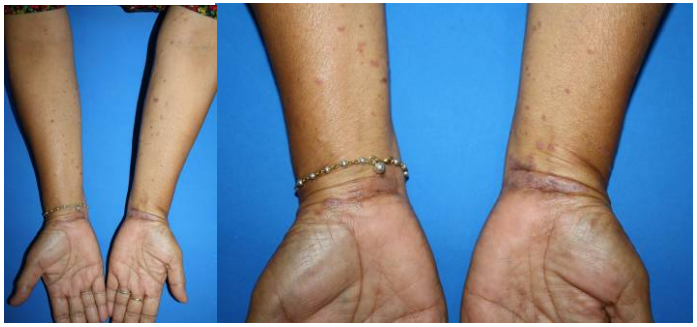
Present illness: The patient noticed progressive itching violaceous skin eruption at upper and lower extremities for 1 month. Precipitating factors are hot weather and gardening.

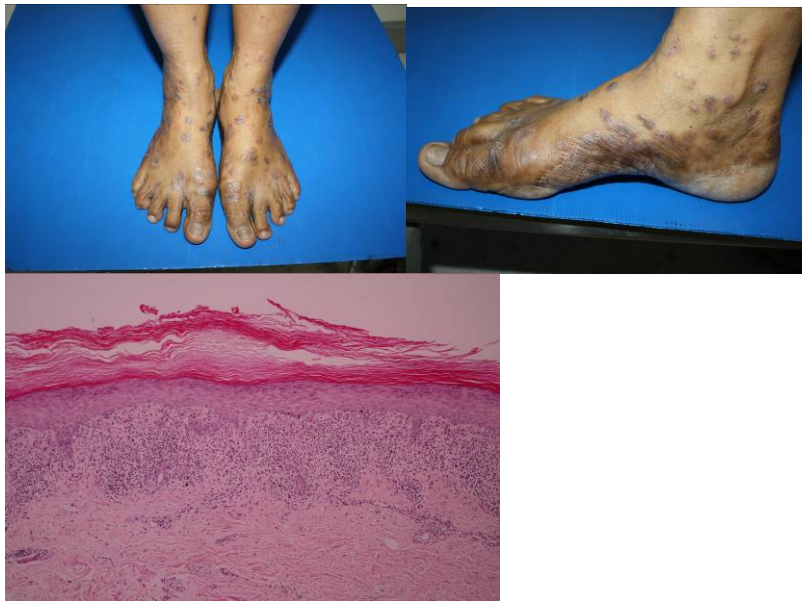
Past history: Unremarkable

Family history: Non contributory

Dermatological examination: Multiple discrete erythematous to violaceous flat topped polygonal papules and plaques, size 3-5 mm at flexor area of both forearms and 3-5 cm at both feet and wrists.

Histopathology (s11-09439): compact hyperkeratosis, hypergranulosis and saw-toothed epidermal hyperplasia
dense lichenoid inflammatory-cell infiltrate of lymphocytes obscuring the dermoepidermal junction





Diagnosis: Lichen planus
Treatment: Betamethasone cream twice daily
10% urea cream twice daily

Case 15.2

Patient: A 70-year-old Thai male from Nonthaburi

Chief complaint: Pruritic rash at upper and lower extremities for 2 months

Present illness: The patient presented with a 2-months history of pruritic hyperkeratotic plaques. It initially located at lower extremities and then gradually progress to upper extremities. His skin rash was very itchy.

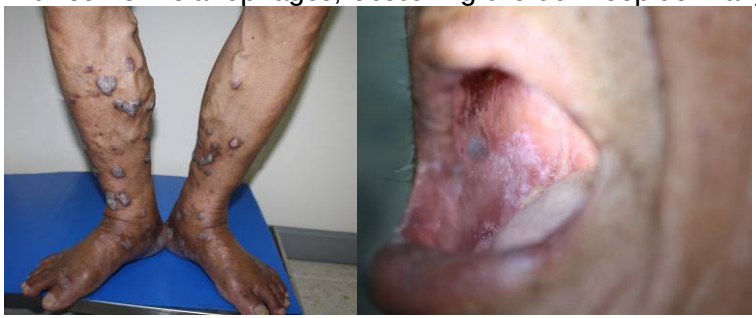
Past history: Unremarkable

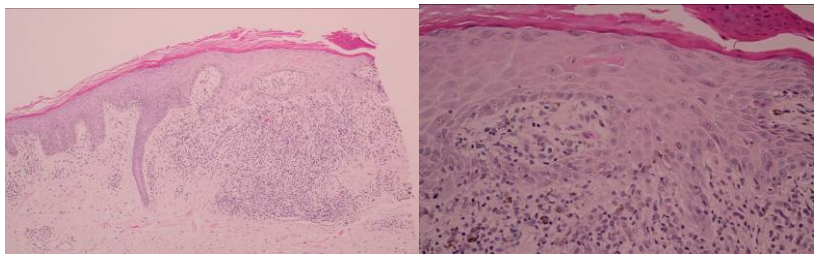
Family history: Non contributory

Dermatological examination: Multiple discrete linear brownish thick hyperkeratotic excoriated plaques at both lower extremities
Multiple discrete violaceous flat top polyglonal papules and plaques at both extensor surface of forearms
Multiple whitish reticulated patches at both side of buccal mucosa
Localized dystrophic of proximal nail plate at left ring finger

Histopathology: (s11-012562A)

Mild hyperkeratosis, saw-tooth deep epidermal hyperplasia, patch lichenoid inflammatory-cell infiltrate of lymphocytes admixed with some melanophages, obscuring the dermoepidermal junction





Diagnosis: Hypertrophic lichen planus

Treatment: 0.05% clobetasol propionate ointment twice daily
10% salicylic acid cream (thick lesion) once daily
10% Urea cream twice daily
Oral antihistamine

Presenters: Ruedee Phasukthaworn
Chayada Kokpol

Consultant: Somsak Tanrattanakorn

Discussion:

Lichen planus (LP) is an unique common inflammatory disease which affects the skin, mucous membrane, hair and nail. The estimated prevalence of LP is less than 1 percent of general population without significant differences in race and gender[1]. More than two thirds of patients are 30-60 years old.

The pathogenesis of LP is not entirely understood. The activated T lymphocytes are recruited to the dermal–epidermal junction and induce apoptosis in basal keratinocytes. Both CD4+and CD8+ T lymphocytes are found in the lesional skin, with a predominance of the CD8+ cells[2].

The classic cutaneous presentation of Lichen planus is symmetrical, grouped, erythematous to violaceous, flat-topped, polygonal papule. Superimposed,reticulated white scale, termed "Wickham's striae" may be present over the surface of well-developed papule. The eruption often fully develops within 1-4 months from the onset.

Lichen planus usually distributes symmetrically and bilaterally; predominance sites are the flexor surfaces of the wrists and forearms, the dorsal surface of the hands, anterior aspect of the lower legs, neck and presacral. Oral mucosal and genitalia is additional common site of LP. Whereas the face and palmoplantar are usually spared.

LP is usually quite pruritic especially in generalized form except for hypertrophic LP, which is localized but extremely pruritic. The Koebner phenomenon is commonly seen in LP. Many clinical variants of cutaneous LP have been described in Table 1.

Table 1: Clinical variants of Lichen planus.

LP variants	Clinical presentation
▪ Mucosal LP	▪ Whitish, reticulated patches or plaques with or without erosion or ulceration
▪ Nail LP	▪ Classic pterygium formation
▪ Inverse LP	▪ Lesions of LP confined to intertriginous regions
▪ Linear LP	▪ LP along blaschkoid distribution
▪ Lichen planopilaris	▪ Follicular involvement of the scalp, resulting in scarring alopecia
▪ Hypertrophic LP	▪ Hypertrophic, pruritic nodules, typically present on extremities
▪ Bullous LP	▪ Vesicles or bullae present within lesions of LP
▪ Actinic LP	▪ Atrophic, hyperpigmented lesions; present in photodistributed area
▪ Annular LP	▪ Violaceous, annular plaques
▪ Erosive LP	▪ Painful, eroded, or ulcerated lesions, often involving mucosal sites
▪ LP pigmentosus	▪ Hyperpigmented lichenoid plaques on sun-exposed or intertriginous sites
▪ Perforating LP	▪ Lesions of LP from which hyaline bodies are extruded through the epidermis
▪ LP pemphigoides	▪ Overlap syndrome of LP and bullous pemphigoid
▪ LP erythematosus	▪ Overlap syndrome of LP and lupus erythematosus

The role of infection in development of LP have been reported for many years including; HCV[3, 4], syphilis, HSV type 2, HIV, amebiasis and chronic bladder infection. Cutaneous eruptions similar to LP (both clinically and histologically) have been related to a variety of drugs such as angiotensin-converting enzyme (ACE) inhibitors, thiazide diuretics, antimalarials, quinidine and gold. Patch testing to metals, flavorings, and plastics may be indicated in patients with oral LP.

Differentail diagnoses of LP include lupus erythematosus (LE), lichen nitidus, lichen striatus, lichen sclerosus, pityriasis rosea, erythema dyschromicum perstans (ashy dermatosis), psoriasis and secondary syphilis.

The clinical findings of typical mucocutaneous LP are reasonably specific. No specific laboratory tests for LP have been developed. Major histopathology finding of lichen planus are basal epidermal keratinocyte damage and lichenoid-interface lymphocytic reaction. Epidermal hyperkeratosis, wedge-shaped area of hypergranulosis and elongation of rete ridge (saw tooth appearance) are found. Direct immunofluorescence (DIF) testing demonstrates the deposition of several immunoglobulins at cytoid bodies with IgM and shaggy fibrinogen deposition at the basement membrane.

Various medical therapies are used for the treatment of cutaneous LP; including medium- to high-potency topical corticosteroids[5, 6], systemic corticosteroids (oral or intramuscular injection), topical calcineurin inhibitors[7], systemic retinoid, phototherapy (NBVUB)[8, 9], photochemotharepy (PUVA)[9] and immunosuppressive drug (cyclosporine[10] and azathiopine).

Our patient no. 1 presented with classic LP. She was treated with betamethasone and 10% urea cream twice daily. Her symptom has moderate improvement.

Patient no. 2 (hypertrophic LP) was treated with superpotent topical corticosteroid, keratolytic and oral antihistamine, which can alleviate his symptom.

Reference

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