Case 20.1

Patient: A 35-year-old Thai male from Buriram

Chief Complaint: Asymptomatic brownish patches on left upper extremity for 2 years

Present Illness: The patient presented with 2-year history of asymptomatic light brownish patches on left arm and forearm. He denied previous traumatic injury or any systemic symptoms.

Past History: He was previously healthy and not taking any medication**.**

Dermatological Examination (Figure 20.1-2)**:** Well-defined light brownish sclerotic and atrophic patches with wrinkle surface, size 4x1 cm. on left arm and 4x3 cm. on left forearm, respectively. No lesions on other sites of mucosa and skin included genitalia.

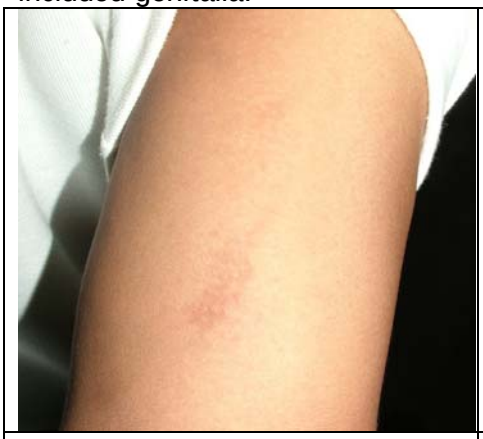

Figure 20.1

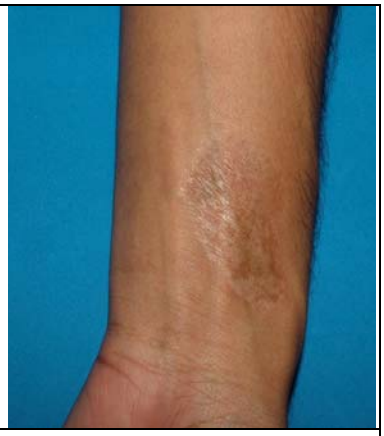

Figure 20.2

Physical Examination: unremarkable

Histopathology: (S09-13273) (Figure 20.3-4): Compact hyperkeratosis and mild epidermal atrophy. Homogenized collagen bundles with patchy lymphocytic infiltrate in the thicken papillary dermis

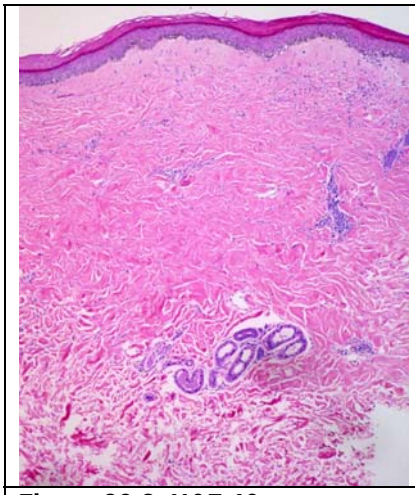

Figure 20.3, H&E 40x

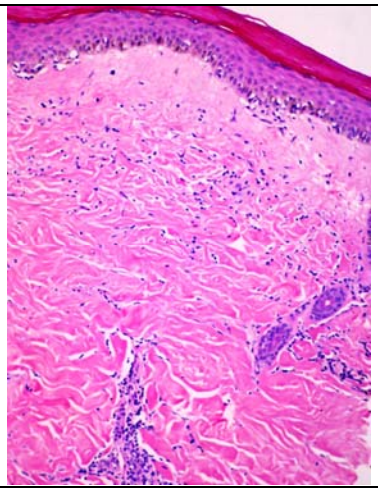

Figure 20.4, H&E 100x

Case 20.2

Patient: A 43-year-old Thai female from Bangkok

Chief Complaint: Linear hypopigmented atrophic patch on left axillary area for 3 months.

Present Illness: The patient presented with 3-month history of slow expanding, asymptomatic hypopigmented patch on left axillary area. The lesion had progressed in linear distribution to left side of chest wall and inner arm. She complained her skin was not smooth, however no musculoskeletal abnormalities was occurred.

Past Illness: HBV carrier, Iron deficiency anemia secondary to hypermenorrhea from myoma uteri

Current Medications: FBC 1x3, Folic 1x1

Dermatological Examination (Figure 20.5)**:** Well-defined linear atrophic hypopigmented patch with irregular border and mottle brownish macules on left side of chest, axillae, and inner arm. No lesions on other sites of mucosa and skin included genitalia.

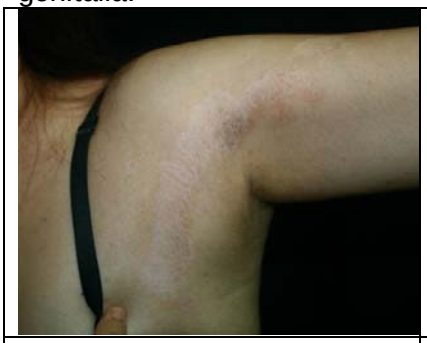

Figure 20.5

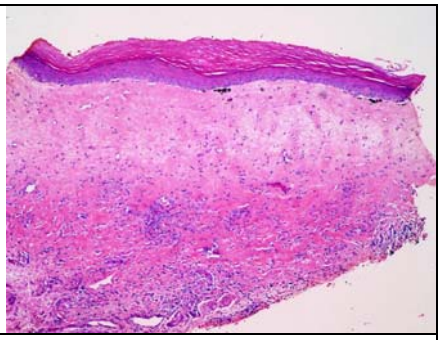

Figure 20.6, H&E 40x

Physical examination: unremarkable

Histopathology (S10-9968) (Figure 20.6)**:**

- Mild epidermal atrophy
- Superficial and deep perivascular infiltrate of lymphocytes
- Homogenized collagen bundle with telangiectasia in thicken papillary dermis

Diagnosis: Lichen sclerosus et atrophicus

Presenter: Lalida Paisanwattanakul

Consultant: Suthinee Rutnin

Discussion:

Lichen sclerosus et atrophicus(LS), usually appearing in the dermatologic literature under the names of lichen sclerosus, balanitis xerotica obliterans, and kraurosis vulvae, is an inflammatory disease with a multifactorial origin.[1]

The pathogenesis of lichen sclerosus is unknown. Several infective agents such as *Borrelia burgdorferi* [2], HPV[3], and hepatitis C[4], have not conclusively linked to LS. Other autoimmune disorders eg. Thyroid disease, vitiligo, and alopecia also have some evidence that could be related to this condition.[5] Recently, low-titer autoantibodies against the extracellular matrix protein-1(ECM-1) was identified and seem to play a role in 67% of LS.[6]

LS is relatively uncommon, more prevalent in females in the fifth or sixth decade than males.[5] Children under the age of 10 years are also affected. It is a disease that can affect both the extragenital skin and the anogenital region. Cutaneous findings are characterized by polygonal papules and porcelain-white plaques with atrophic fragile skin, fissure, telangiectasia, purpura, erythema and the classical figure of eight pattern of vulva and anus are observed on physical examination. Anogenital manifestations affect quality of life due to the severe itching, soreness, dyspareunia, dysuria, discomfort with defecation, or genital bleeding and, with time, may lead to destructive scarring. Vulvar disease seems to have an increased risk of squamous cell carcinoma, but the role of additional co-factors (e.g., human papillomavirus infection or prior radiotherapy) has not been defined. Extragenital manifestations are generally non-pruritic and affect the site of local trauma, thigh,neck, trunk, shoulder and wrist.

Histopathologic finding is specific. In the early lesion demonstrates superficial dermal edema, associated with a band-like lymphocytic infiltrates beneath that zone. The epidermis is thinned, with orthohyperkeratosis is especially pronounced at follicular openings and may lead to plugging. The most important changes are found in the superficial dermis, where the pale staining reflects homogenized dermal collagen and extreme edema in the early stages. Clefting and

hemorrhage within homogenized papillary dermis is often seen. Mature lesion is replaced by fibrosis.

The goal for treatment of LS is to relieve the symptoms and prevent cutaneous complication. Topical potent and ultrapotent corticosteroids remain the mainly used agent on both genital and extragenital lesions. Clobetasol propionate 0.05%cream applied for 12-24 week[(7)], 6-8 week course in pedriatric patients[8] can cause clinical improvement which confirmed by histopathology and prevent scarring.[9]Mometasone furoate cream may be an alternative to clobetasol propionate for treatment of vulvar LS, with similar efficacy but higher levels of safety and tolerability.[10]

Calcineurin inhibitor,both topical tacrolimus and pimecrolimus have been used as topical treatments for vulvar lichen sclerosus.[11] In patients with refractory generalized extragenital LS, the combination of pulsed high-dose corticosteroids and low-dose methotrexate therapy should be considered, especially in refractory to conventional treatment.[12]

Less clinical response were reported for the treatment of genital LS is topical testosterone and progesterone. Systemic retinoid plays a role for nonresponsive cases or intolerance to high potency topical corticosteroids and surgical modalities should be spared for only the complicated LS.[13]

In our patients were treated with 0.05%clobetasol cream twice daily and the skin lesions are minimally improved

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