

Dermoscopic findings in extragenital lichen sclerosis

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ABSTRACT

Lichen sclerosis (LS), also known as lichen sclerosis et atrophicus is a chronic inflammatory dermatosis of unknown aetiology. It has both genital and extragenital presentations, nevertheless genital forms significantly outnumber extragenital LS. Dermoscopy is noninvasive diagnostic tool traditionally employed in pigmented lesions, however its usefulness in inflammatory skin conditions is becoming continuously more meaningful. Although the clinical diagnosis of fully developed LS rarely causes difficulties, unusual presentations require differentiation from the diseases such as lichen planus, morphea, extramammary Paget disease, SCC and others. In these cases histopathology contributes to the diagnosis. Studies on the use of dermoscopy in LS are sparse, nevertheless some dermoscopic features of LS has been described.

Key words: Extragenital lichen sclerosis; Dermoscopy; Lichen sclerosis

INTRODUCTION

Lichen sclerosis (LS) is a chronic, usually asymptomatic, inflammatory dermatosis that results in epidermal atrophy and scarring. It is prevalent in females with bimodal onset in prepubertal and postmenopausal age group [1,2]. It most often affects genital and perianal areas of postmenopausal women, however it can affect men and pre-pubertal children. LS is ten times more common in women than in men. Extragenital LS affects 10% of women with vulval disease [3]. Penile LS is the leading cause of the phimosis in adult men [1]. Genital LS has been associated with a certain risk of squamous cell carcinoma (SCC), whereas extragenital LS does not appear to predispose to cancer. Cancer is estimated to affect up to 5% of patients with vulval, penile or anal LS [3].

The exact aetiology of LS has not been ascertained yet, however, evidence points to an increased likelihood of autoimmune and genetic component. The most common autoimmune diseases associated with LS are autoimmune thyroiditis, alopecia areata, vitiligo and pernicious anemia [3].

CASE REPORT

A 32-year-old woman with no previous medical history of autoimmune diseases, with multiple

itchy, hypopigmented, atrophic, well-demarcated, brightening round and oval papules and plaques on the upper back since 1,5 year (Fig. 1). Hair, nails, oral mucosa and anogenital region were unaffected. Blood investigations did not show any abnormalities. The patient has not been treated yet.

The dermoscopic examination revealed white-yellowish structureless areas with comedo-like openings and sparse linear vessels in the centre of each lesion and an erythematous halo, which is a marker of activity in LS (DermLite DL4) (Fig. 2).

The histopathology of the lesion showed epidermal atrophy, hyperkeratosis, follicular plugging and basal vacuolization. The dermis showed oedema, initial homogenization of collagen, interstitial and perivascular lymphocytic infiltration (Figs. 3 and 4).

DISCUSSION

The clinical differentiation of LS and morphea, especially in extragenital regions, is a diagnostic challenge. White structureless areas and comedo-like openings are typically seen in LS [4], whereas fibrotic bands are characteristic of morphea. Nevertheless, comma shaped vessels, hairpin like vessels and dotted

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Figure 1: Multiple brightening hypopigmented round and oval papules and plaques on the upper back.



Figure 2: White-yellowish structureless area with comedo-like openings and sparse linear vessels in the centre and an erythematous halo.

vessels are usually absent in morphea and are seen only in LS [5].

Comedo-like openings in LS are predominant in early lesions, whereas white chrysalis like structures suggest homogenization of collagen in the dermis and can be seen only in late lesions. Long persisting lesions appear atrophic [2,6].

Dermoscopic structures in LS correlate with histopathology: white structureless areas are representing epidermal atrophy, whereas comedo-like openings are representing follicular plugging in histopathology [5].

Histopathology of morphea shows the continuity of the basal membrane zone (BMZ), whereas in LS numerous invaginations are present in BMZ [7].

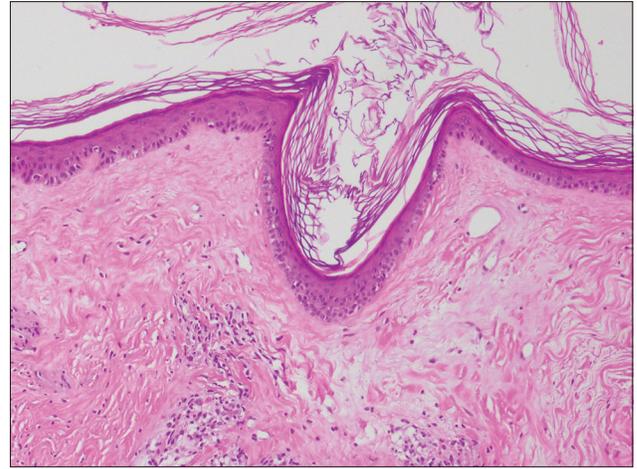


Figure 3: Lichen Sclerosus- epidermal atrophy, follicular plugging and basal vacuolization, initial homogenization of collagen in the dermis [HE x 20].

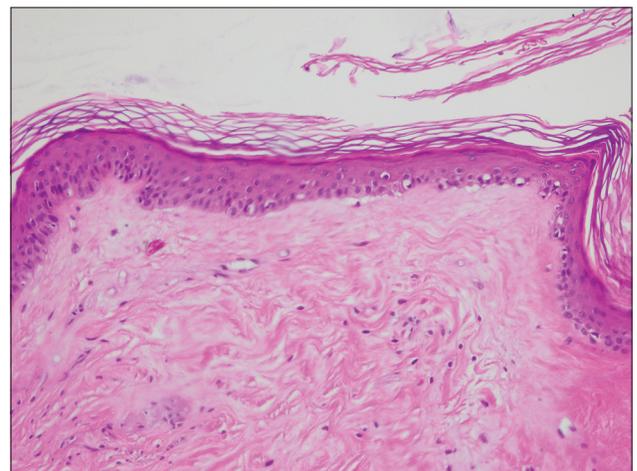


Figure 4: Lichen Sclerosus - epidermal atrophy, follicular plugging and basal vacuolization [HE x 20].

CONCLUSION

Although the clinical diagnosis of fully developed LS rarely causes difficulties and the disease is usually recognized by its appearance, early forms or unusual presentations require differentiation from other diseases including morphea, lichen planus, SCC and others. In these cases histopathology contributes to the diagnosis and is mandatory for any clinical situation in which co-existing SCC cannot be ruled out. Early diagnosis and treatment play a substantial role in patient's prognosis and result in decreased risk of malignancy and scarring.

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