

폐경 여성에게 발생한 경화성 태선 1예

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Lichen Sclerosis in a Post-Menopausal Woman: A Case Report

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Lichen sclerosis (LS) refers to a benign, chronic, progressive dermatologic condition that primarily causes anogenital lesions in middle aged women. But LS has been identified in patients of all ages including those in the first years of life. The risk of squamous cell carcinoma of the vulva is 4% to 5% in women with LS, and has been reported to be as high as 7% in some reviews. We recently encountered a case of LS in a post-menopausal woman without hormone therapy. To remind clinicians of the neglected issue of LS, we report a case of LS with literature review.

Key Words: Lichen sclerosis, Postmenopause

Introduction

Lichen sclerosis (LS) refers to a benign, chronic, progressive dermatologic condition characterized by marked inflammation, epithelial thinning, and distinctive dermal changes. The previous designation was LS et atrophicus. The “et atrophicus” was dropped because areas of thickening and hyperplasia often occur. LS usually occurs in the anogenital region (85 to 98 percent of cases), but can develop on any skin surface. Extragenital lesions are present in 15 to 20 percent of patients.¹ In post-menopausal women, the disorder is most common when endogenous estrogen production is low. Sideri et al.² showed no difference in sexual habits, smoking history, education, or dietary habits.

Besides age and post-menopause state, no other risk factors for LS have yet been identified. The anogenital lesion can cause severe discomfort and manifest with erosion, porcelain-white plaques, papules and wide degrees of sclerosis. Potent topical corticosteroids associated with skin care are thought to be the most successful therapy.

Case Report

A 56-year-old woman was referred to Soonchunhyang University Hospital from a local clinic for whitish vulvar lesion (Fig. 1A). Pap smear and vulvar punch biopsy was performed. Pap smear test was negative for intraepithelial lesion, Human papillomavirus (HPV) deoxyribonucleic

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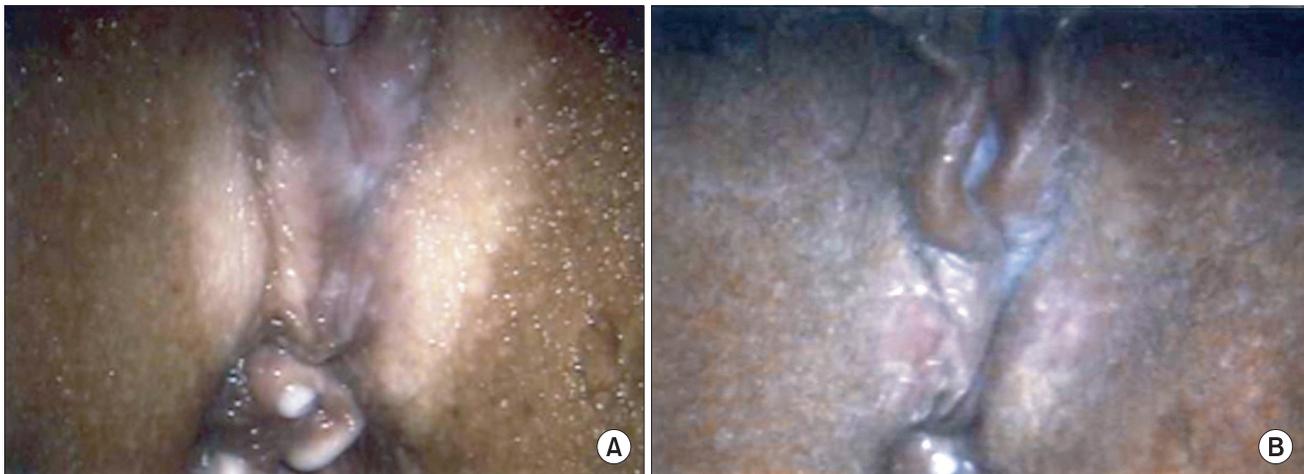


Fig. 1. Vulva of woman with lichen sclerosus. (A) The vulva area shows pallor, fusion and resorption of labia, pale polygonal plaques, erythema and erosive lesion. (B) Punch biopsy was performed, and after 6 days, the lesion was spontaneously healed.

acid (DNA) chip test was positive. Pathologic findings indicate that there was no malignant lesion, and diagnosed subepithelial sclerosis. After punch biopsy, the lesion was spontaneously healed (Fig. 1B). We counseled the patient and recommended annual examination.

Discussion

LS, also known as balanitis xerotica obliterans, is a chronic inflammatory skin disease which may cause substantial discomfort and morbidity, most commonly in adult women.

The exact prevalence of LS is unknown. The exact prevalence of LS is difficult to estimate because affected patients may have no symptoms and patients referred to hospital may be seen by specialists in dermatology, gynecology, pediatrics, geriatrics, or urology. However, Wallace estimated the prevalence of LS to range from 1 of 300 to 1 of 1,000 of all patients referred to a community based dermatology department.² LS is more prevalent in women than men, with ratios varying from 6 : 1 in some reviews to 10 : 1 in others. Most cases of LS in females occur in postmenopausal women with a mean age of onset in the fifth to sixth decade of life, with only 7% to 15% of cases occurring in prepubertal girls.³

Familial aggregations of LS among fathers and daughters, mothers and daughters, sisters, and twins (identical and fraternal) have been reported. Trauma, injury, and sexual

abuse may trigger symptoms in genetically predisposed individuals, which suggests presence of the Koebner phenomenon (ie, development of isomorphic pathologic lesions in the traumatized uninvolved skin of patients who have a cutaneous disease).⁴

Disorders of the immune system are more common among patients with LS, suggesting autoimmune mechanisms and immune dysregulation may be involved in the etiology of the disease.⁴⁻⁶

The highest incidence of LS in women is observed during low estrogen physiological states, such as the premenarchal child and the postmenopausal women. While a hormonal hypothesis for the etiology of LS has been postulated, no association between LS and pregnancy, hysterectomy, contraceptive use, or postmenopausal hormone therapy has been found.²

The diagnosis of LS is based upon the presence of characteristic clinical manifestations, ideally with histological confirmation.⁷ The classic features of uncomplicated LS are a thinned epidermis with hyperkeratosis, a wide band of homogenized collagen beneath the dermo-epidermal junction, and a lymphocytic infiltrate beneath the homogenized area.⁸

In the early stages of LS, not only is the clinical appearance sometimes hard to interpret but so is the histologic picture.⁹ The features may be similar to psoriasis or lichen planus with irregular acanthosis, mild hyperkeratosis, hypergranulosis, mild lichenoid lymphocytic

infiltrate with lymphocyte tagging and dilated blood vessels immediately under the basement membrane, focal basement membrane thickening, luminal hyperkeratosis of adnexal structures, submucosal edema and homogenization, lichenoid lymphocytic infiltrate, and lymphocyte exocytosis.⁹ The most important management point here is that the diagnosis of LS is usually clinical. A biopsy may be helpful but should not be interpreted in isolation. In early disease, the clinicopathological correlation may be difficult and such individuals may need follow-up and a repeat biopsy if the clinical findings significantly change and diagnostic uncertainty persists. A nonspecific biopsy does not rule out lichen sclerosus, although classic histologic findings confirm the diagnosis.⁸

The risk of squamous cell carcinoma (SCC) of the vulva is 4% to 5% in women with LS, and has been reported to be as high as 7% in some reviews. And LS can be found in adjacent areas in more than 60% of cases of vulvar SCC. As mentioned, analysis of women with vulvar LS revealed systemic cellular immune dysregulations, which may theoretically create a permissive environment for the development of SCC. In contrast extragenital LS does not seem to carry a risk of malignant change.¹⁰ From a clinical management point of view, it would be helpful to identify which individuals with LS are at particular risk of developing malignancy.⁸

There is no universally accepted management plan for women with LS. Management of LS involves education and support, behavioral modification to ensure good vulvar hygiene, medication, and, in a small subset of cases, surgery. The goals for treatment of LS are to alleviate symptoms and discomfort, prevent anatomical changes such as stricture, and prevent malignant transformation. Biopsy of the initial lesion before initiation of medical or surgical management is necessary to confirm the existence of LS and to rule out SCC. Controversy exists regarding whether everyone must be treated, even those who are asymptomatic, but many advocate that asymptomatic patients should be treated to prevent progression of the disease and possibly development of malignancy.⁸

Patients should be followed up according to need. Initially a follow-up is helpful to ensure that there is adequate disease response and appropriate compliance with therapy.

This initial follow-up is suggested at 3 months following the initiation of high-potency topical steroids, with a second follow-up at 6 months to check that the disease remains well controlled. If the disease is uncomplicated and well controlled, then patients can be discharged with an annual review in primary care.⁸

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국문초록

경화성 태선 뚜렷한 염증, 얇아진 상피, 간지러움과 통증을 동반하는 분명한 진피의 변화를 특징으로 하는 양성, 만성, 진행성 피부학적 상태이다. 경화성 태선은 만 1세의 유아를 포함한 생애 전 연령대에서 발생할 수 있다. 여러 문헌에 따르면, 회음부에 경화성 태선이 발생한 여성의 4-7%에서 편평 세포암이 발생할 수 있으므로 쉽게 간과할 수 있는 질환이 아니다. 본 저자는 호르몬 치료를 받지 않는, 폐경후 여성에게서 발생한 경화성 태선을 간단한 문헌 고찰과 함께 보고하여, 임상 의사들이 쉽게 무시하고 넘어갈 수 있는 질환인 경화성 태선을 재조명 해보고자 한다.

중심단어: 경화성 태선, 폐경