EXTRAGENITAL LICHEN SCLEROSUS POSSIBLY SECONDARY TO A KOEBNER PHENOMENON INDUCE BY ARTIFICIAL SOLARIUM

Ozturk P1,*, Ciralik H2, Dogan Y1
1.Kahramanmaras Sutcu Imam University School of Medicine, Department of Dermatology Kahramanmaras Turkey
2.Kahramanmaras Sutcu Imam University School of Medicine, Department of Pathology Kahramanmaras Turkey

Correspondence
Dr Perihan Ozturk. Kahramanmaras Sutcu Imam University, School of Medicine, Department of Dermatology Yorukselim mah. Hastane Cad. No. 32 46050 Kahramanmaras Turkey
Email: drperihanozturk@hotmail.com


ABSTRACT

Lichen sclerosis et atrophicus (LSA) is a chronic inflammatory mucocutaneous disorder that predominantly affects prepubertal girls and postmenopausal women. The Koebner phenomenon is recognized in lichen sclerosus. The etiology remains unknown. We report on the case of a 32 years-old woman with extragenital LSA following artificial solarium exposure. We presented this case because of not found previously in the literature.

Key words: Lichen, sclerosus, solarium, koebner phenomenon

INTRODUCTION

Lichen sclerosis et atrophicus (LSA), first reported by Hallopeau in 1889, is a chronic inflammatory skin disease that usually affects the anogenital area. Its typical histology was defined by Darier in 1892. Historically, numerous different terms have been applied to be condition in males, including LSA, balanitis xerotica obliterans, guttate morphea, krauroris penii and lichen albus. This confusion of nomenclature has hindered understanding of the condition. More recently, a consensus appears to have been reached that these entities are all variants of the same condition. LSA may also occur extragenitally and the typical locations are the thigh, neck, and trunk1,2. The etiology of LSA is obscure, but genetic susceptibility, autoimmune mechanisms, infective agents like human papilloma virus and spirochetes, and Koebner phenomenon has been postulated as causative factors1,3. We report the first case of extragenital LSA as a Koebner-phenomenon appearance following artificial solarium exposure.
CASE REPORT

We reported the case of a 32-year-old woman presented with a four-month history of a white patch on her back. Five months ago two times artificial solarium had been applied. The patient did not have a history of trauma. On physical examination, we found a macular white lesion on the back of the patient that were 5 × 6 cm in size and featured a creamy-white atrophic plaque with sclerosis (Figure 1). Laboratory findings regarding blood count, biochemical test, CRP, and erythrocyte sedimentation rate were normal. Borellia burgdorferi antibodies were negative. A punch biopsy was taken from the lesion for histopathological examination: there was a thinning of the epidermis, melanin pigment in the basal layer, homogenization of collagen tissues in the upper dermis, and a small number of perivascular lymphocytes (Figure 2). The case was diagnosed as LSA by physical and histopathology findings.

Figure 1. The lesions were 5 × 6 cm in size and featured a creamy-white atrophic plaque with sclerosis.
Figure 2. There was a thinning of the epidermis, melanin pigment in the basal layer, homogenization of collagen tissues in the upper dermis, and a small number of perivascular lymphocytes.

**DISCUSSION**

LSA is a chronic inflammatory disease more commonly found in women. It can occur at any age, but there are two populations in which it is more predominant: in postmenopausal women, and in prepubertal children. The lesions generally affect the anogenital area. 15–20% of patients also have lesions in extragenital sites, particularly at the upper region of the back, neck, periumbilical region, axillas, and wrists. Our case featured white sclerotic plaques 5 × 6 cm in diameter on the back, as a case of extragenital LSA.

In recent years, species *Borrelia* have been recognized as the aetiologic agents of several apparently diverse diseases, including Lyme disease and acrodermatitis chronica atrophicans, and possibly some types of lichen sclerosus. *Borrelia* burgdorferi antibodies were negative in our case.

The cause of LSA is unknown, but a series of factors are thought to be involved in its pathogenesis: a) hormonal factors, b) autoimmune mechanisms, given the association with autoimmune disorders such as vitiligo, thyroiditis, or pernicious anemia and the higher frequency of organ-specific antibodies; c) infectious factors, such as the human papilloma virus, hepatitis C, and *Borrelia burgdorferi*; d) genetic factors, given that cases
of familial LSA have been described and have been related to various histocompatibility antigen (HLA) subtypes; e) endocrine factors, as several authors have found a statistically significant prevalence of diabetes mellitus; and f) repeated trauma (Koebner phenomenon\textsuperscript{3,4}.

The isomorphic response, also known as the Koebner phenomenon, consists of the appearance of typical lesions of a certain dermatosis in areas of otherwise healthy skin that has previously been subjected to different kinds of trauma. They are divided into four groups:

1. Category I: a true Koebner phenomenon, only seen with psoriasis, vitiligo, and lichen planus.
2. Category II: a pseudo-Koebner, which includes warts, molluscum contagiosum, and pyoderma gangrenosum.
3. Category III: dermatoses with occasional lesions in areas of trauma, such as Kaposi sarcoma, Darier disease, and erythema multiforme.
4. Category IV: A doubtful isomorphic phenomenon, which appears in diseases such as pemphigus vulgaris, eczema, or lichen nitidus\textsuperscript{4-5}.

LSA is included in category III of the Koebner phenomenon\textsuperscript{4}. It has been associated with burns\textsuperscript{5}; venous hypertension\textsuperscript{6} (related to varicose veins); vaccines\textsuperscript{7}; vulvovaginitis and pellagra\textsuperscript{8}; repeated pressure, friction from clothing, trauma, and traumatic and surgical scars; UV radiation and ionizing radiation\textsuperscript{10}; and insulin injection sites\textsuperscript{4}. LSA developing on herpes zoster scars (isotopic response) could be included in this group\textsuperscript{11}. Some cases of reports in the literature are presented in Table 1. We think that the etiology of our case was solarium as a Koebner phenomenon.

Table 1. Some reported examples of Koebner response in lichen sclerosus.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Year of publication</th>
<th>Site/trigger for Koebner response</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>2010</td>
<td>Insulin injection site</td>
</tr>
<tr>
<td>5</td>
<td>1988</td>
<td>Sunburn</td>
</tr>
<tr>
<td>6</td>
<td>2004</td>
<td>Venous hypertension</td>
</tr>
<tr>
<td>7</td>
<td>1976</td>
<td>Vaccination site</td>
</tr>
<tr>
<td>8</td>
<td>2009</td>
<td>Pellegra</td>
</tr>
<tr>
<td>9</td>
<td>1984</td>
<td>Surgical scar</td>
</tr>
<tr>
<td>10</td>
<td>1985</td>
<td>Radiotherapy</td>
</tr>
<tr>
<td>11</td>
<td>2005</td>
<td>Herpes zoster scar</td>
</tr>
</tbody>
</table>

CONCLUSION

In conclusion, the reason for reporting this case was to describe a patient suffering from extragenital LSA, who had developed lesions at the site of solarium exposure as a result of a Koebner phenomenon; this association we found was first in the literature.

REFERENCES