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Extragenital Lichen Sclerosus and Vitiligo: A Rare Association

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Abstract

Case Report

Lichen sclerosus and vitiligo are two depigmenting disorders which may occur separately or rarely, in combination. Their association may seem logical because both these disorders are characterized by the suspicion of an autoimmune pathogenesis. We wanted to present a 58-year-old female patient, who was diagnosed with vitiligo and lichen sclerosus as a result of dermatological examination and histopathological examinations with no notable medical history.

INTRODUCTION

Vitiligo is an acquired, idiopathic disorder characterized by depigmented macules that result from damage to and destruction of melanocytes. Two of the major theories of the pathogenesis of vitiligo are the autoimmune theory and the autocytotoxicity theory[1,2]. Lichen sclerosus (LS) is a chronic destructive inflammatory disease that affects the epidermis and dermis, particularly the genital and perineal areas[3]. However, cases of coincidental LS and vitiligo are rare reported. Although the exact mechanism of the co-development of LS and vitiligo is unknown, both have been reported to be associated with autoimmune diseases or certain infections[4].

CASE REPORT

58-year old woman presenting with achromic macules and atrophic-looking plaques evolving over 1 year. Clinical examination showed two types of lesions: achromic macules measuring 10-15 cm at both axillary areas (Figure 1) and a large number of atrophic-looking plaques at the anterior and posterior side of the trunk (Figure 2). No mucosal lesions or nail involvement was detected. The patient had no history of trauma or sunburn; no drugs were taken in the previous months. And she has no other disease history. A clear contrast enhancement was seen in wood light examination in colorless areas at the axillary areas and vitiligo was diagnosed for these lesions. A skin biopsy taken from the atrophic plaque demonstrated orthokeratosis, focal parakeratosis, atrophy in the epidermis and subepidermal edema-homogenization, increased collagen fibers, perivascular lymphocytic infiltration was seen in the dermis (Figure 3). Based on both clinical and histological features, she was diagnosed as LS. The patient did not recall any history of familial autoimmune diseases. Laboratory tests on autoimmune panels (antinuclear antibody, anti-DNA screening, anti-Scl 70) were all negative and the other blood tests were normal. We started the topical corticosteroid for all lesions.

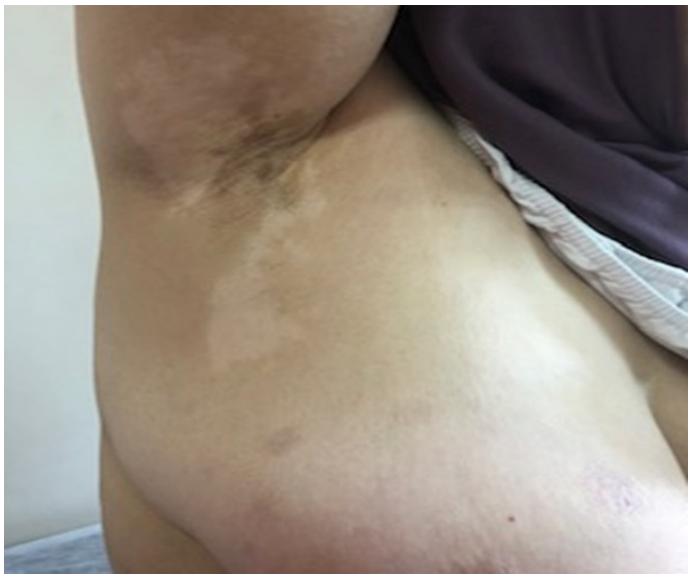

Figure 1:Achromic macules measuring 10-15 cm at both axillary areas

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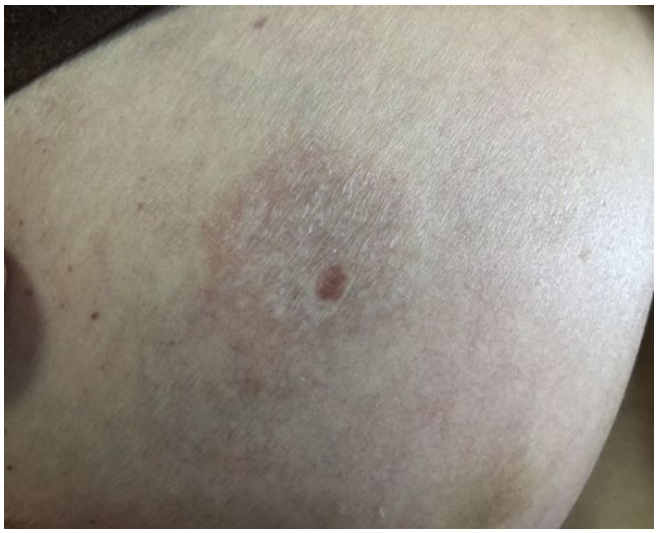

Figure 2: Atrophic-looking plaques at the anterior and posterior side of the trunk.

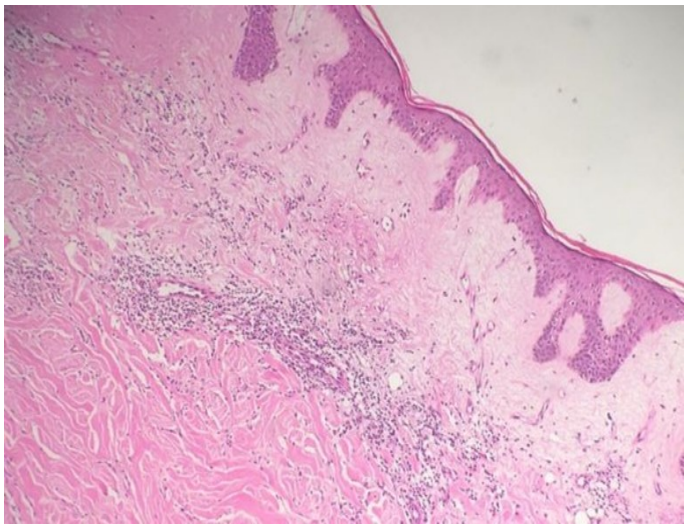

Figure 3: Epidermis:orthokeratosis, focal parakeratosis, atrophy in the epidermis. Dermis:subepidermal edema-homogenization, increased collagen fibers, perivascular lymphocytic infiltration

DISCUSSION

Vitiligo is an acquired, idiopathic disorder characterized by depigmented macules that result from damage to and destruction of melanocytes[1]. Two of the major theories of the pathogenesis of vitiligo are the autoimmune theory and the autocytotoxicity theory[1,2]. The autoimmune theory speculates that patients with vitiligo form autoantibodies against melanocytes. Vitiligo has been associated with antibody-mediated autoimmune diseases such as thyroid disease, pernicious anemia, diabetes mellitus, Addison disease, alopecia areata, and myasthenia gravis[5]. LS is a chronic relapsing skin condition characterized by early inflammation followed by chronic scarring and skin atrophy. Etiology of LS remains unclear, but the literature suggests a likely autoimmune phenomenon in a genetically predisposed individual. Previous infections, trauma, and occlusive moist environments are also contributing factors. Although classically seen in the anogenital area (80%-98%), it can be seen in extragenital sites in 15% to 20% of patients[6]. Extragenital sites, although far less common. Only 6% of LS are isolated to extragenital lesions, as seen in our case[7]. Colocalization of vitiligo and LS is rare in the dermatologic literature. In a survey of patients with vitiligo, some were also noted to have genital LS, and Wallace suggested that this association might be significant[7]. Indeed, both vitiligo and LS have been reported to exhibit the Koebner phenomenon; hence, mechanical stimulation may be symptom triggers in genetically predisposed individuals[4]. LS and vitiligo are two depigmenting disorders which may occur separately or, rarely, in combination. Their association may seem logical because both these disorders are characterized by the suspicion of an autoimmune pathogenesis. Therefore, this pathological association between LS and vitiligo in our patient, highlights the key role of epidermal lichenoid inflammatory process in the disappearance of melanocytes and, possibly, in the induction of a auto-immune process.

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