

LETTER

Detailed studying of Langerhans cells in female genital lichen sclerosis

Dear Editor,

Genital lichen sclerosis (GLS) is an inflammatory skin disease which most commonly occurs in postmenopausal women, and is characterized by white macules and plaques. There is limited data regarding its pathogenesis; a hormonal factor, chronic inflammation/infection, and an autoimmunity basis have been suggested as causative factors¹. Langerhans cells (LCs) are dendritic cells that constitute 3-5 % of all epithelial cells and may regulate immunity¹⁻². Data regarding the number of LCs in GLS is still controversial¹⁻².

We conducted a single-center, prospective study that aimed to assess the number of LCs in skin biopsies of postmenopausal women with GLS and compare it with healthy controls. Inclusion criteria were postmenopausal females with pathology proven GLS, who presented with active, late lesions of GLS. Control group consisted of healthy, postmenopausal females who underwent a plastic surgical operation of the vulva region. Exclusion criteria were skin conditions that would interfere with GLS evaluation. Skin tissue specimens were obtained from both groups. Ethics board approval was obtained, and written consent was provided.

Three-micrometer paraffin unstained sections were taken, and immunohistochemical staining was performed using CD1a and CD207 antibodies. The number of LCs in each case was estimated per 10 consecutive high-power fields at 400x magnification.

Statistical analysis was performed using IBM SPSS Statistics for Windows, Version 22.0 (IBM SPSS, IBM Corp., Armonk, NY, USA). Kolmogorov-Smirnov test was used to test the normality of continuous variables. Mann-Whitney test was used to compare continuous variables.

The final sample consisted of 42 patients with GLS and 11 controls. The number of LCs in GLS, using both CD1a and CD207 stain, was significantly lower compared with the controls [CD1a: 8.35 (0.70-31.60) vs 18.30 (14.0-24.50), $p < 0.001$; CD207: 4.60 (0.00-21.80) vs 9.00 (7.10-16.80), $p < 0.001$].

LCs were mainly recognized by CD1a expression of LCs in human epidermis. As nowadays, the state-of-the-art marker for LCs is anti-langerin/CD207, the number of LCs using both CD1a and CD207 were examined and found lower in GLS patients.

Initially, Carli et al showed that the number of epidermal CD1a+ LCs was increased in cases of GLS compared to controls, suggesting that the skin immune system is involved in GLS pathogenesis²⁻³. Later, Rotsztejn et al reported that the dysregulation of the skin immune system might lead to suppression of LCs in the vulvar epithelium¹. It was shown that, in late GLS, the mean number of LCs was lower as compared to controls¹. Moreover, a large decrease in the LCs in vulvar SCC indicated that the extremely low level or absence of LCs might be connected with carcinogenesis¹.

Use of both stains reinforces the validity of our results and attests the evidence that LCs are lower in GLS compared to controls. The ascertainment that LCs are altered in GLS may trigger the study of novel therapeutic agents and furthermore, may contribute to a better understanding of SCC development in GLS lesions.

Keywords: Lichen sclerosis, Langerhans cells, CD1a, CD207, dendritic cells

Conflict of interest

None.

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