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ORIGINAL ARTICLE

Lack of oral involvement in a large cohort of women with vulvar lichen sclerosus – a multicenter prospective study

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Summary

Background and Objectives: We assessed the prevalence of oral lichen sclerosus in a cohort of women affected with vulvar lichen sclerosus (VLS).

Patients and Methods: This prospective, observational study included adult female patients with a histologically proven VLS who attended the Vulva Clinics of three Dermatology Units from January 2020 to July 2023. During this period, all VLS patients were asked to be examined in their oral cavities in order to detect any possible sign of oral diseases, which were then biopsied.

Results: Three hundred women (mean age 64.5 ± 13.0 years) were included, of whom 21 (7%) had a concurrent extragenital LS. In six (2%) patients, white, non-removable lesions were found at oral inspection and biopsied. All cases were histologically lichen planus. No other clinically relevant oral lesions were found. The six women with histologically proven oral lichen planus had more frequent periodontal and autoimmune diseases than the other included subjects.

Conclusions: Our findings confirm the absolute rarity of oral involvement in patients with VLS. They suggest that the oral mucosa, unlike the genitals, is a setting which weakly leads to development of LS, also in predisposed subjects.

KEYWORDS
anatomical area, chronic inflammatory diseases, lichen planus, oral lichen sclerosus, pathogenesis, Vulvar lichen sclerosus

INTRODUCTION

Lichen sclerosus is a chronic inflammatory, progressive skin disease with a proclivity for anogenital skin.[1] It is more common in females than males and the prevalence of vulvar lichen sclerosus (VLS) is estimated between 0.1% and 3% in the prepubertal and peri-/postmenopausal periods, respectively.[2] Primary lesions of vulvar lichen sclerosus present as smooth, ivory or porcelain-white spots that tend to coalesce into thin, crinkly patches and plaques. Ecchymosis, itching-related excoriations, hyperkeratosis and erythema may occur.

Post-inflammatory progressive scarring and atrophy[3,4] may cause irreversible changes in anogenital architecture, which may lead to varying degrees of fusion or loss of labia minora, narrowing of the vaginal introitus and burying of the clitoris.

An increased risk of developing squamous cell carcinomas in the context of VLS is also recognized.[5,6] Most patients with VLS complain of distressing symptoms, such as itching, burning, stinging and dyspareunia.[7,8] Growing evidence has shown that VLS has a huge impact on well-being and quality of life due to its troubling symptoms, chronic course, sexual dysfunction, disfiguring anatomical

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changes, partial and temporary response to treatment and risk of cancer progression.[9–12]

With reference to VLS physiopathology, it is conceivable that environmental factors, acting as generic damage-associated molecular patterns on a genetic background, ignite an immune response.[13–15] More specifically, in VLS the elicited immune cascade which leads to chronically raised levels of pro-inflammatory cytokines predominantly belongs to the Th1 type.[3] This inflammatory state causes tissue and microvascular injury as well as activation of signaling pathways involved in fibroblast and collagen metabolism.[16,17] Dysregulation of pro- and anti-fibrotic mechanisms determines the degree of dermal fibrosis, which represents the histopathological culmination of this process.

Extragenital lesions are described in 15%–20% of patients with genital LS,[18] but only 6% of cases are represented by isolated extragenital lesions.[19] Extragenital involvement can occur on any skin area but is most common on the neck, upper back, breasts, shoulders, axillae, flexor sides of the wrists, thighs and abdomen. Skin LS is more often asymptomatic than in genital localization.

Oral involvement is even rarer and thus far only 41 cases of histopathologically proven oral LS (OLS) have been reported.[20] Oral LS appears more frequently in women, without a bimodal distribution in terms of age, unlike anogenital forms.[21] It can occur in different sites of the mouth and at the vermillion border and presents with thin, atrophic, shiny, whitish, ivory- or porcelain-white macules or plaques with well-demarcated borders.[20,22,23] These clinical features may mimic other chronic, white, flat, plaque-type lesions, such as lichen planus, leukoplakia, chronic hyperplastic candidiasis, oral submucous fibrosis, and morphea. In addition, some OLS cases may show characteristics of autoimmune bullous diseases.

Both cutaneous and oral LS are prone to koebnerization and physical trauma, continuous pressure, and scarring may be the most relevant eliciting factors for these localizations.[24]

The majority of patients with OLS are asymptomatic. Pain, soreness, itching and tightness when opening the mouth have been rarely reported. Gingival involvement may lead to gingival recession and loss of periodontal attachment.[20,21,23] About a quarter of the patients with OLS have concurrent anogenital involvement.[25]

Malignant transformation of OLS has not been reported, suggesting that OLS has a better prognosis when compared with genital LS and with oral lichen planus.[25]

The main aim of the present study was to assess the prevalence of OLS in a cohort of women affected with VLS. A secondary objective was to better characterize any patients with dual oral and vulvar involvement.

MATERIAL AND METHODS

Study design and setting

The present study was set up as a multicenter, prospective, observational study, which involved the Vulva Clinics of the Dermatology Units of the University Hospitals of Ferrara, Ancona, Bologna and Firenze, Italy. From January 2020 to July 2023, all the subjects affected with VLS who attended the aforesaid Vulva Clinics were asked to have their oral cavities and lips examined in depth for detecting any possible sign of disease, above all OLS, in this anatomical areas. In the presence of either white or erosive lesions or in any case suggestive of an oral or lip disorder, a biopsy and histological examination were carried out. Demographic, history and clinical data, as well as the histological diagnosis, at both vulvar and oral level, were collected in a personal medical record.

This study aimed to assess the prevalence of OLS and of any other oral disorder in VLS patients.

Study patients

All adult (≥ 18 years), female patients with a clinical and histological diagnosis of VLS who attended the participating Vulva Clinics for either first or control visits during the study period were eligible. The content and purposes of the study were explained in advance. Specifically, these women were asked to consent to being carefully observed in their oral cavity, including the lips, in order to assess the presence of LS in these anatomical sites. It was further explained that if suspicious lesions were found, a biopsy for histological examination would be offered to them. Written informed consent was required from patients before study enrollment. The following inclusion criteria were applied: *(1)* over 18 years old, *(2)* histological confirmation of VLS, *(3)* consent to participate. Exclusion criteria were as follows: *(1)* concomitant inflammatory skin diseases, especially those with potential oral involvement, such as lichen planus and pemphigus vulgaris, *(2)* concomitant active vulvar or oral infections, proven by microbiological assessment, *(3)* refusal or inability to undergo the study assessments and procedures. Concurrent extragenital LS as well as ongoing treatments for VLS were not exclusion criteria.

This study was approved by the local Ethics Committee.

Study assessments

The following data were recorded from all included patients: *(1)* age (at the visit; in the case of multiple visits across the study period, the age at the last visit was considered; in the case of an oral biopsy, the age at the time

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of the biopsy was considered), *(2)* VLS duration, recorded as the time between the patient-reported onset of symptoms and the visit or oral biopsy, *(3)* detection of white or erosive or, more broadly, suspicious oral lesions, including lips, *(4)* topography of the suspicious oral lesions, *(5)* any other disorder of the oral mucosa, including paraphysiological conditions, such as fissured tongue or *lingua plicata* and geographic tongue or benign migratory glossitis, and periodontal diseases, *(6)* histological diagnosis of the oral biopsy, *(7)* relevant comorbidities, especially concurrent autoimmune diseases.

Statistical analysis

A database was created containing all the study data. The Shapiro-Wilk test was used to assess the normality of distribution of the continuous variables. In the presence of symmetry of the distributions, the variables were represented with mean and standard deviation (SD) or, in the case of non-normal distribution, with the median value and interquartile range [1Q 3Q]; categorical data were expressed as total numbers and percentages.

Comparisons between groups of values were performed with Student's t-test for independent samples or Mann-Whitney's U test, as appropriate in the case of quantitative variables. To compare groups of categorical variables, contingency tables were made and analyzed by Chi-square test or, in the case of values of five or less, by Fisher's exact test.

Microsoft Excel (Microsoft Corporation, Redmond, USA) with the Real Statistics Resource Pack software add-in (http://www.real-statistics.com/) was used for computation.

RESULTS

Study patients

Based on inclusion and exclusion criteria, 300 women affected with histologically proven VLS were included (mean age 64.5 ± 13.0 years). No eligible patient denied consent to observation of their oral cavity and possible biopsy. The included patients were affected with VLS for an average of 86 months (± 97.9). A considerable proportion of them (36%) were affected with a concurrent autoimmune disease, mainly thyroiditis. Table 1 summarizes the main features of the study patients.

Twenty-one (7%) women presented an extragenital cutaneous LS.

Various pathological or paraphysiological conditions were found with the systematical clinical inspection of the oral cavity and are reported in detail in Table 2. Periodontal disorders were found in a quarter of the study women (25.3%) while fissured tongue was the most common (12.7% of the subjects) among the paraphysiological conditions.

TABLE 1 Characteristics of the study patients.

	Total (n = 300)
Age (yrs.), mean ± SD	64.5 ± 13.0
VLS duration (months), mean ± SD	86.1 ± 97.9
Concurrent autoimmune diseases, n (%)	109 (36.0)
Thyroiditis	56 (18.7)
Alopecia areata	4 (1.3)
Celiac disease	6 (2.0)
Connective diseases	30 (10)
Vitiligo	8 (2.7)
Morphea	3 (1.0)
Diabetes mellitus, n (%)	46 (15.3)
Smoker, n (%)	42 (14.0)
Denture wearer, n (%)	83 (27.7)
Previous systemic immunosuppressive treatments, n (%)	36 (12.0)
Extragenital LS, n (%)	21 (7.0)
Disease duration (months), mean ± SD	48.8 ± 28.8

Abbr.: VLS, vulvar lichen sclerosus; SD, standard deviation; yrs., years; LS, lichen sclerosus; VLS, vulvar lichen sclerosus

TABLE 2 Oral features of the study population.

	Total (n = 300)
Black hairy tongue, n (%)	1 (0.3)
Geographic tongue, n (%)	11 (3.7)
Median rhomboid glossitis, n (%)	6 (2.0)
Atrophic glossitis, n (%)	6 (2.0)
Fissured tongue, n (%)	38 (12.7)
Periodontal disease, n (%)	76 (25.3)
Erythema	25 (8.3)
Bleeding	24 (8.0)
Gum recession	56 (18.7)

Histology of white or suspicious oral lesions

In six (2%) patients, white, non-removable lesions were found at oral inspection and biopsied. All cases were histologically lichen planus (Figures 1 and 2). No alternative histological diagnoses, including lichen sclerosus, were detected. No other clinically suspicious oral or lip lesions, such as erosive lesions, were found. The main features of the six women who underwent oral biopsies are detailed in Table 3. Table 4 reports the anatomical locations of the biopsied oral white lesions.

Comparing the six women with histologically proven oral lichen planus with the others, no differences were found in terms of age or VLS duration. Five of them had concurrent

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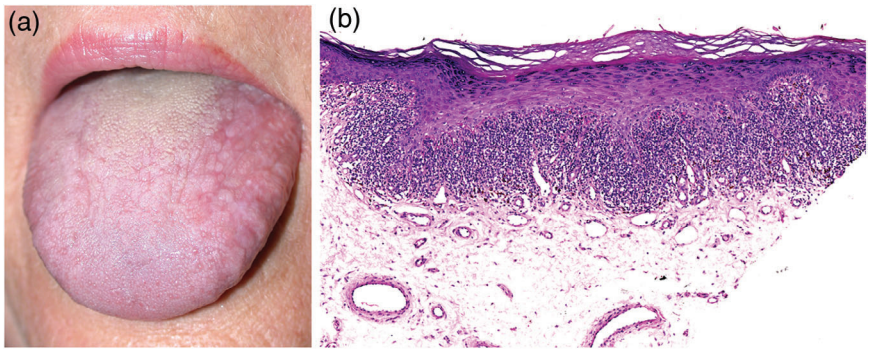

FIGURE 1 (a) Numerous whitish, flattened papules, some tending towards confluence, on the tongue dorsal surface. (b) Histologic examination showed edema, dense band-like lymphocytic infiltrate in the papillary and mid chorion, basket-woven orthokeratosis, hypergranulosis in the epithelium and vacuolar alteration at dermoepidermal junction (hematoxylin-eosin stain, original magnification x 11).

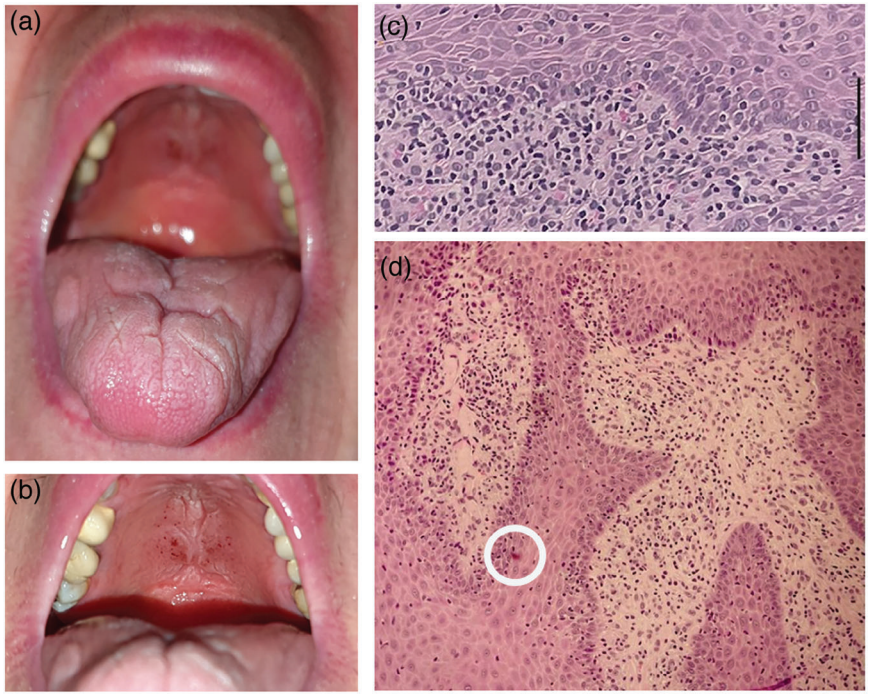

FIGURE 2 (a) Whitish plaque with linear fissures on the dorsal surface of the tongue and (b) punctate elements with a petechial appearance on the soft palate. (c) Histologic examination from a tongue biopsy revealed a band-like lymphohistiocytic infiltrate with acanthosis, lymphocytic peribasal exocytosis (hematoxylin-eosin stain [HE], original magnification x 11) and (d) apoptotic basal keratinocytes (Civatte bodies, in the circle; HE, x 10).

periodontal disorders and concomitance of autoimmune diseases.

DISCUSSION

The present study aimed to evaluate concomitant oral diseases in a large group of women affected with histologically confirmed VLS. In particular, we were interested in quantifying the co-occurrence of vulvar and oral LS. The patients included had characteristics rather in line with what was expected. They were about 60 years old on average, with a long-lasting VLS and with a frequent co-occurrence of other autoimmune diseases (Table 1). In 7% of them, an extra-genital involvement was observed.

Overall, the overview offered by the inspection of the oral cavity in these patients appears to reflect the typical situation of women in this age group. Indeed, no conditions of particular clinical relevance appear to emerge.

Regarding the main study objective, two key findings should be emphasized: no woman had histologically proven OLS, while six patients (2%) had white lesions that were histologically consistent with lichen planus.

Considering the first point, the absence of OLS confirms the extreme rarity of the oral localization of LS,[20] even in women with vulvar involvement. The reason for the macroscopic discrepancy between the prevalence of LS at vulvar level, and anogenital level more in general, compared to its oral localization can only be hypothesized. Considering that the genetic predisposition to LS is intrinsic to each patient, regardless of LS location, its different topographical incidence should probably be sought in the difference in exogenous triggers present in the different anatomical locations. It is conceivable that triggering stimuli present in the genital area, and specific to this location, whether microbial, chemical (such as urine), mechanical or irritant in nature, are particularly effective in activating the pathogenetic cascade underlying the development of LS.[14,26–30]

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TABLE 3 Characteristics of the patients with oral involvement.

	Total (n = 6)
Age (yrs.), mean ± SD	64.5 ±13.7
VLS duration (months), mean ± SD	73.7 ±69.2
Concurrent autoimmune diseases, n (%)	5 (83.3)
Thyroiditis	1 (16.7)
Alopecia areata	2 (33.3)
Celiac disease	2 (33.3)
Connective diseases	0 (0)
Vitiligo	1 (16.7)
Morphea	0 (0)
Diabetes mellitus, n (%)	1 (16.7)
Smoker, n (%)	0 (0)
Denture wearer, n (%)	1 (16.7)
Previous systemic immunosuppressive treatments, n (%)	1 (16.7)
Extragenital LS, n (%)	0 (0)
Oral features	
Black hairy tongue, n (%)	0 (0)
Geographic tongue, n (%)	0 (0)
Median rhomboid glossitis, n (%)	0 (0)
Atrophic glossitis, n (%)	2 (33.3)
Fissured tongue, n (%)	0 (0)
Periodontal disease, n (%)	5 (83.3)
Erythema	3 (50.0)
Bleeding	2 (33.3)
Gum recession	2 (33.3)

Abbr.: VLS, vulvar lichen sclerosus; SD, standard deviation; yrs., years; LS, lichen sclerosus; VLS, vulvar lichen sclerosus

TABLE 4 Features of oral involvement.

	Total (n = 6)
Oral disease duration (months), mean ± SD	23 ±19.9
Anatomical site, n (%)	
Tongue	2 (33.3)
Buccal mucosa	5 (83.3)
Hard palate	0 (2.0)
Soft palate	2 (33.3)
Gum	3 (50.0)

Abbr.: SD, standard deviation

On the other hand, the absence of these triggers at extragenital level, and especially in the oral cavity, voids an essential factor for the clinical expression of LS, even in predisposed subjects such as women with VLS. Thus, in the oral cavity, essential players of the multifactorial process implicated in the pathogenesis of LS may be missing. In particular, the exogenous triggers capable of igniting the abnormal immune reactivity and inflammation that lead to the histological and clinical expression of the disease at genital level may be absent in the oral cavity. The lack of OLS cases did not allow us to delve deeper into the secondary objective of this study, which was to characterize patients with coexisting vulvar and oral involvement.

The prevalence of oral lichen planus in the study population does not seem to significantly differ from that found in the general population, although it is located at the upper limits of the reported ranges.[30] An interesting finding is that five out of the six women with oral lichen planus had concomitant autoimmune diseases. The association between genital LS and oral lichen planus has already been addressed.[31] The findings from this study could indicate that patients with VLS, and particularly those with a more marked tendency towards autoimmune diathesis, may tend to develop oral lichen planus. The reason why they may develop lichen planus and not LS remains to be clarified. Consistently with the hypothesis formulated above, it could be assumed that triggers present in the oral cavity, particularly in subjects predisposed to autoimmune reactivity, are more favorable to the development of an inflammatory pathway which leads to histological changes consistent with lichen planus rather than LS.[32,33] For example, the triggering role of periodontitis has been supposed in the onset of OLP, although there is no unequivocal evidence.[34] In this regard, 5 of the 6 women with OLP had signs of periodontal disease.

When commenting on the results of this study, some limitations must be taken into consideration. Although the histological examination of the oral lesions was very thorough and the diagnosis of lichen planus was made after a careful differential diagnosis process, it must be said that OLS, especially early lesions, may histologically have overlapping features with lichen planus or psoriasis. In our opinion, the histological diagnoses are reasonably well founded but a margin, albeit minimal, of misinterpretation cannot be excluded with absolute certainty. Our conclusions regarding oral LS are limited by the low prevalence of this condition and could be strengthened in future by larger sample sizes. The study was not aimed at investigating possible etiological or triggering agents. The considerations made in the interpretation of the results are therefore to be considered speculative. The women included were recruited from highly specialized clinics and may not be fully representative of the population.

Despite these limitations, this study found the absence of OLS in a population of women affected by VLS. This finding confirms the absolute rarity of oral involvement of LS, even in patients with genital forms. The concomitance of oral lichen planus in a rate of our study population suggests that the oral cavity is a setting favoring this disorder instead of LS.

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CONFLICT OF INTEREST STATEMENT

None.

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