CASE REPORT

Lichen sclerosus et atrophicus of the oral mucosa

E. F. Mendonça, R. F. Ribeiro-Rotta, M. A. G. S. Silva, A. C. Batista

Department of Oral Medicine (Oral Pathology, Stomatology and Radiology), School of Dentistry, Federal University of Goiás, Brazil

Lichen sclerosus et atrophicus (LSA) is a chronic, benign, depigmenting disease of the skin and mucous membranes most frequently affecting the female genitalia. Involvement of the oral mucosa without concurrent genital or skin lesions has been reported only occasionally in the literature. In view of the rarity of reported cases, one lesion affecting only the labial mucocutaneous area is presented along with a description of the disease's clinical and histopathological findings.

| Oral Pathol Med (2004) 33: 637-40

Keywords: lichen planus; lichen sclerosus et atrophicus; oral pathology

A 20-year-old white female was referred to the Centro Goiano de Doenças da Boca -C.G.D.B. – School of Dentistry, Federal University of Goiás for examination of a white lesion on the lower lip. The lesion was totally asymptomatic and had been present for an unknown period of time. The patient was otherwise well, and she was taking no medication. She did not drink alcohol, smoke tobacco, or chew areca nut. No history of trauma or surgery in the area of the white lesion, and no familial history of similar disease were reported.

Clinical examination revealed a depigmented white macule on the right vermilion of the lower lip extending to the inside of the labial mucosa (Fig. 1A,B). The lesion was approximately 2×7 cm in size. Clinical diagnosis of vitiligo and lichen sclerosus et atrophicus (LSA) were established. The patient was referred to a consultant dermatologist and gynecologist for further investigation but no skin and genital similar lesions were identified.

An incisional biopsy of the lesion was obtained. The microscopic examination of sections stained with hematoxylin and eosin showed mucosal parakeratinized stratified squamous epithelium of reduced thickness, accompanied by hydropic degeneration of the basal cells at focal sites (Figs 2 and 3A,B). A subepithelial hyalinization, and diffuse mononuclear inflammatory infiltrate localized underlying were observed (Fig. 2). There was no evidence of epithelial dysplasia or neoplasia. A section stained by Verhoeff's method showed scarceness of elastic fibers in the superficial connective tissue (Fig. 4A,B). Some clear cells compatible with melanocytes showed immunoreactivity for S-100 antibody (Novocastra Laboratories Ltd, clone S1/61/69) (1:200





Figure 1 (A and B) Clinical presentation shows white macule on the right lower lip and labial mucosa.

Correspondence: Aline Carvalho Batista, Departamento de Ciências Estomatológicas, Disciplina de Patologia, Universidade Federal de Goiás, Faculdade de Odontologia, Rua: T-36, n. 3033, Edificio Dom Arthur, Apt.602, Setor Bueno – 74223-050, Goiânia – GO –Brazil. Tel./Fax: +55 62 521 1886. E-mail: ali.caba@uol.com.br Accepted for publication August 11, 2003

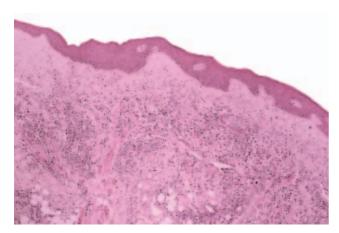


Figure 2 Low-powered photomicrograph showing epithelium atrophic and parakeratotic, subepithelial hyalinization, and underlying diffuse mononuclear inflammatory infiltrate (hematoxylin-eosin, original magnification $\cong 12.5 \times$).

in 1% PBS-BSA-immunoperoxidase method) (Fig. 5-A,B). Their presence ruled out vitiligo. It was concluded that the histopathologic features were consistent with the diagnosis of LSA. As there were no symptoms and few cosmetic concerns, further treatment was not deemed necessary. To date, after a follow-up period of 3 years (Fig. 6A,B), no recurrence nor development of new oral mucosal, genital or cutaneous lesions have been observed.

Comments

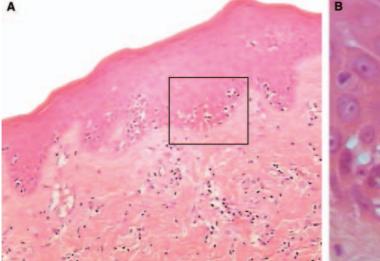
According to the reports in the literature, the prevalence of LSA affecting the oral mucosa alone with the diagnosis supported by microscopic evaluation is extremely low. The absence of suitable knowledge of the histopathologic features of oral LSA may have led to wrong diagnosis, and this may contribute to the

scarcity of reports of oral LSA. Only nine cases with microscopically verified oral LSA have been reported (1–7). Of these nine cases, five occurred in women (1, 2, 4, 6) and four in men (3, 6, 7). The range of age was from 43 years (2). In all cases, the patients were white (1-7). No skin or genital lesions developed after the follow-up period in any of the reported cases or in case. This suggests that oral LSA may appear without accompanying skin or genital lesions. The oral lesions of LSA are usually asymptomatic and may have been overlooked in the past. We purport that many lesions of LSA of the oral mucosas are clinically and histopathologically misdiagnosed.

In the present case, the clinical characteristics are very similar to the cases reported by Schulten et al. (6) and by Brown et al. (7). In all three cases, a depigmented porcelain-white macule on the lower lip was present and in these cases the histopathological confirmation was mandatory.

For skin and genital lesions, the characteristic changes in the epithelium include marked hyperkeratosis with plugging of hair follicles, loss of rete ridges, atrophy of the epithelium and hydropic degeneration of the basal layer. Within the upper dermis there is usually marked edema with homogenization of the collagen bundles, whilst in the mild-dermis a band-like lymphocytic infiltrate predominantes. Ravitis in 1957 (3) first described microscopic features of these lesions confined to the oral mucosas only. The histological features observed for oral LSA are quite similar to those for skin, with the exception of hyperkeratosis.

We conclude that the most striking histopathological features of oral LSA at date, including our case, are focus of the hydropic degeneration of basal cells, subepithelial hyalinization of the connective tissue, a slightly diffuse, band-like mononuclear



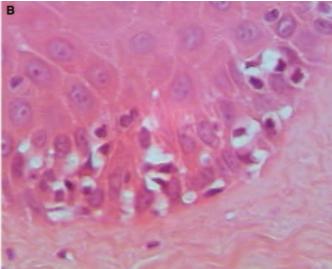


Figure 3 (A and B) Photomicrograph shows hydropic degeneration of the basal cells. B shows a close-up view of the detached area A [hematoxylin-eosin, original magnification $\cong 100 \times$ (A) and $\cong 250 \times$ (B)].

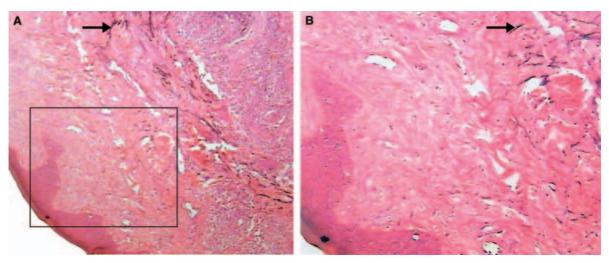


Figure 4 (A and B) Photomicrograph shows scantiness of elastic fibers in the superficial connective tissue (arrow). B shows a close-up view of the detached area A [Verhoeff's stain, original magnification $\cong 100 \times (A)$ and $\cong 250 \times (B)$].

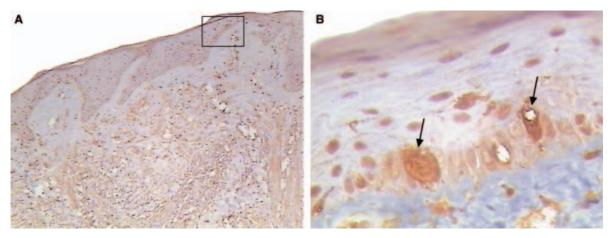


Figure 5 (A and B) Photomicrograph showing immunoreactivity for S-100 antibody in basal cell (arrow). B shows a close-up view of the detached area A [immunohistochemical staining, original magnification $\approx 100 \times (A)$ and $\approx 250 \times (B)$].



Figure 6 (A and B) Clinical appearance of the lesion with follow-up period of 3 years.

infiltration in the deeper connective tissue and atrophy of the epithelium. The scantiness or loss of elastic fibers of the connective tissue was commented in five cases of the nine described, and the intracellular edema of the spinous layer was present in only two cases.

It is sometimes difficult to differentiate oral LSA from oral lichen planus, oral submucous fibrosis, oral scleroderma and oral vitiligo by means of histological features. However, the band of lymphocytes at the epithelium connective tissue junction that is usually found in lichen planus is not evident in LSA, and

640

scantiness or loss of elastic fibers, usually present in LSA, is not found in scleroderma (2). The presence of the melanocytes in the basal epithelium in the LSA eliminates vitiligo and the absence of the obliteration or narrowing of blood vessels and epithelial atypia eliminate submucous fibrosis (2). Thus, because of the microscopic findings present in oral LSA, it is possible to establish a correct diagnosis for this lesion.

Treatment of oral LSA is usually unnecessary because of its asymptomatic nature, benign behavior, few cosmetic concerns and no evidence of recurrence. However, the efficacy of topical and intralesional application of corticosteroids to resolve these lesions has been demonstrated (2, 7). Although no cases of malignant change associated with oral LSA have been reported, regular long term follow-up is indicated.

References

 Jensen T, Worsaae N, Melgaard B. Oral lichen sclerosus et atrophicus: a case report. Oral Surg Oral Med Oral Pathol 2002; 94: 702-6.

- Buajeeb W, Kraivaphan P, Punyasingh J, Laohapand P. Oral lichen sclerosus et atrophicus. *Oral Surg Oral Med Ora Pathol* 1999: 88: 702–6.
- 3. Ravits HG, Welsh MD. Lichen sclerosus et atrophicus of the mouth. *Arch Dermatol* 1957; **76**: 56–8.
- Araújo VC, Orsini SC, Marcucci G, Araújo NS. Lichen sclerosus et atrophicus. Oral Surg Oral Med Oral Pathol 1985; 60: 655–7.
- Macleod RI, Soames JV. Lichen sclerosus et atrophicus of the oral mucosa. Brit J Oral Maxillofac Surg 1991; 29: 64-5
- Schulten EAJM, Starink THM, Van Der Waal I. Lichen sclerosus et atrophicus involving the oral mucosa: report of two cases. *J Oral Pathol Med* 1993; 22: 374–7.
- Brown AR, Dunlap CL, Bussard DA, Lask JT. Lichen sclerosus et atrophicus of the oral cavity. Report of two cases. Oral Surg Oral Med Ora Pathol 1997; 84: 165–70.

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.