Leukoedema of the Oral Mucosa

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Leukoedema of the oral mucosa is a whitish or whitish-gray edematous lesion of the buccal and labial oral mucosa. The condition is seen most frequently among black people, and has not yet been reported in the Korean dermatologic literature. We report a 28-year-old Korean woman affected by leukoedema of the oral mucosa. She presented with a 3 year history of white plaques on both buccal mucosae. The diagnosis was clinically based on the presence of white plaques on both buccal mucosae which disappeared when the lesion was stretched, and histologically based on the acanthosis and intracellular edema.
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INTRODUCTION

Leukoedema of the oral mucosa was first described by Sandstead and Lowe[1] in 1953. This disorder usually affects the buccal and labial mucosal surface. Leukoedema is so called, due to the lesion's white clinical appearance 'leuko' and the histopathological finding of intracellular 'edema'[2]. Leukoedema is a relatively frequent condition in black people. But it has not previously been reported in the Korean dermatologic literature to our knowledge. We herein report a case of leukoedema of the oral mucosa in a 28-year-old Korean woman.

CASE REPORT

A 28-year-old Korean woman presented with a 3-year history of diffuse whitish plaques on both buccal mucosae, which seemed to vary in degree with time.

Physical examination revealed irregularly confluent, whitish plaques which could not be removed with a wooden tongue blade (Fig. 1A, B). But these lesions disappeared when the mucosa was stretched. Oral hygiene was not poor. She was otherwise healthy and had no family history of this condition. Routine laboratory analyses were within normal limits. An incisional biopsy specimen was obtained from one of the plaques of the buccal mucosa. Histopathologically, the epidermis was pale and showed acanthosis with intracellular edema. There was neither dyskeratotic cells in the epidermis nor significant inflammatory cells in the dermis (Fig. 2). PAS staining revealed no significant glycogen deposition in the keratinocytes. These histopathologic findings were consistent with a diagnosis of leukoedema.

Because she had no symptoms and was reluctant to take any medication, treatment was not given.

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DISCUSSION

Leukoedema is an asymptomatic, whitish or whitish-gray edematous lesion of the oral mucosa. It is an acquired disorder that may be present at any age, commonly has periods of exacerbation and remission, and involves only the buccal or labial mucosa. It presents as a bilateral, symmetrical,

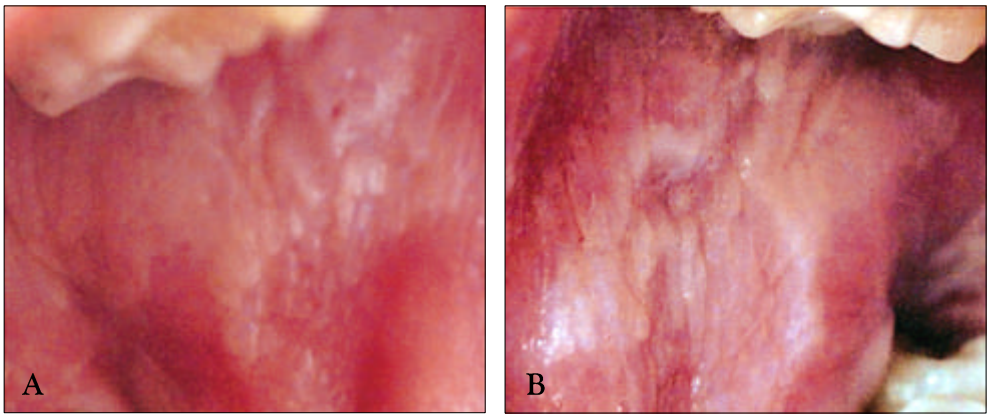

Fig. 1. (A) Left buccal mucosa. (B) Right buccal mucosa. Both buccal mucosae show irregularly confluent edematous white plaques.

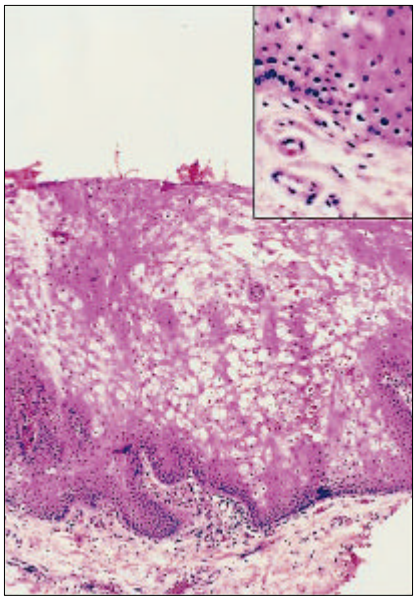

Fig. 2. Pale, edematous epithelial cells, and acanthosis of buccal mucosa (H & E, × 100): inset; there are no dyskeratotic epithelial cells (H & E, × 200).

smooth-surfaced area with a classic grayish-opalescent tinge. It can be incidentally diminished by stretching the mucosa, which is thought to be caused by hydrated epithelial cells[2].

The histopathologic examination of leukoedema reveals irregular acanthosis and intracellular edema in the epidermis. There is neither dyskeratosis in the epidermis nor infiltration of inflammatory cells in the dermis. PAS staining fails to identify any glycogen deposition[3].

Although the etiology is unknown, it has an increased in incidence in patients with poor oral hygiene, but has not been related to syphilis, nutritional factors, smoking habits, the number of dental fillings, or oral habits such as thumb sucking, tooth grinding, or cheek biting[4].

Leukoedema is a relatively frequent condition in black people, and has no sex predilection[5]. Durocher et al.[6] found leukoedema in 97% of black people, and they proposed that the condition be considered a variation of normal.

A differential diagnosis for leukoedema would encompass leukoplakia, Darier's disease, oral white sponge nevus, pachyonychia congenita, candidial infection, or cheek biting. Of these, the most frequent misdiagnosis are leukoplakia, oral white sponge nevus, and cheek biting.

Because of a strong clinical resemblance, leukoedema was once thought to be a probable precursor to leukoplakia. But leukoedema is a benign condition with no tendency to become malignant[7]. In addition, leukoplakia clinically shows less edematous whitish plaques and histopathologically dyskeratotic epithelial cells in the epidermis and inflammatory cells in the dermis[2].

Oral white sponge nevus is an autosomal dominant inherited disorder, with mutation in the mucosal keratin pair K4 and K13. This disorder usually appears in children and may progress until adolescence, then remain unchanged with time. It may involve the rectal, vagina, nasal mucosa, and sometimes esophagus[8,9].

Cheek biting is an intermittent, compulsive habit that produces a painful sensation. Although these lesions may resemble leukoedema in their clinical

and histologic appearance, the histologic features of the cheek biting surface are covered by a layer of bacteria and an inflammatory reaction can be observed in the papillary dermis[10].

The treatment of leukoedema is unnecessary. But, if patients have pain or a burning sensation, topical tretinoin may be tried. The lesions respond rapidly, but temporarily, to therapy with tretinoin[10].

REFERENCES

1. Sandstead HR, Lowe JW. Leukoedema and keratosis in relation to leukoplakia of the buccal mucosa in man. J Natl Cancer Inst 1953;14:423-437.
2. Duncan SC, Su WP. Leukoedema of the oral mucosa. Possibly an acquired white sponge nevus. Arch Dermatol 1980;116:906-908.
3. van Wyk CW, Ambrosio SC. Leukoedema: ultrastructural and histochemical observations. J Oral Pathol 1983;12:319-329.
4. Martin JL, Crump EP. Leukoedema of the buccal mucosa in Negro children and youths. Oral Surg Oral Med Oral Pathol 1972;34:49-58.
5. Martin JL. Leukoedema: an epidemiological study in white and African Americans. J Tenn Dent Assoc 1997;77:18-21.
6. Durocher RT, Thalman R, Fiore-Donno G. Leukoedema of the oral mucosa. J Am Dent Assoc 1972;85:1105-1109. Cited from reference 2.
7. Archard HO, Carlson KP, Stanley HR. Leukoedema of the human oral mucosa. Oral Surg Oral Med Oral Pathol 1968;25:717-728.
8. Corden LD, McLean WH. Human keratin diseases: hereditary fragility of specific epithelial tissues. Exp Dermatol 1996;5:297-307.
9. Witkop CJ Jr, Gorlin RJ. Four hereditary mucosal syndromes: comparative histology and exfoliative cytology of Darier-White's disease, hereditary benign intraepithelial dyskeratosis, white sponge nevus, and pachyonychia congenita. Arch Dermatol 1961; 84:762-771.
10. Waitzer S, Fisher BK. Oral leukoedema. Arch Dermatol 1984;120:264-266.