Comment on “Desquamative gingivitis: Clinical findings and diseases”

To the Editor: We read with great interest the paper by Maderal et al on desquamative gingivitis. We would like to comment on an irritant dermatitis involving a nondescribed cause of desquamative gingivitis.

A 51-year-old man referred to us with a 10-year history of relapsing asymptomatic desquamative gingivitis. The patient's medical history was unremarkable, and he did not report any chemical oral trauma, apart from the consumption of >9 espresso coffee cups daily and the use of dental hygiene products. Moreover, he admitted that he had begun to bite the mucous desquamating areas without bleeding.

At inspection of the oral cavity, the patient showed a linear desquamation of the oral epithelium of the buccal mucosae near the dental arches and some areas of peeling in the mucosa near the labial commissures (Fig 1, A). A white epithelial layer of tissue was manually removed with a tongue blade, leaving a normal tissue base with no bleeding or erosions. Nikolsky sign was negative and genital mucosae were not involved.

Dermascopy of the white areas showed whitish scales without white and gray lines or dots correlated with Wickham striae (Fig 1, B). Cutaneous scrapings and swabs were also negative for bacteria and fungi. Patch testing with a European standard series integrated with resins, dental prostheses, and a fragrance series were used to investigate contact hypersensitivity. All series results were negative at 48 hours and 72 hours.

Our patient was treated successively with a topical solution of diflucortolone valerate plus josamycin once a day in association with topical tocopherol once a day for 3 months with no improvement.

A punch biopsy was eventually performed, and histopathology revealed an unspecific acanthosis with intracellular edema and superficial intraepithelial cleft of the epithelium lining the oral mucosa. Histopathology ruled out lichenoid diseases and autoimmune blistering disorders. A diagnosis of oral mucosal peeling syndrome was suspected. Discontinuation of toothpastes used by the patient was suggested and dental hygiene products without sodium lauryl sulfate were prescribed. The patient's clinical condition gradually improved, with a reduction of symptomatic episodes over time, confirming the suspected diagnosis.

Oral mucosal peeling, also known as oral epitheliolysis, is rarely reported in the literature and, to the best of our knowledge, has been never reported in dermatology journals. This entity was first described by Archard et al in 1968 and was also called leukoedema of oral mucosae. An important clinical clue is the presence of soft white tissue on oral mucosae that can easily be removed. Other histopathologic findings consistent with oral mucosal peeling syndrome that have been reported in the literature were detected in our patient, including increased thickness of the epithelium and intracellular edema of the Malpighian layer.

Oral mucosal peeling could be secondary to mechanical or chemical trauma, and the patient's toothpaste might be the causative agent. Sodium lauryl sulfate is used as a foaming agent in dentifrices, and at higher concentrations, the incidence of oral epithelial desquamation increases.

Although oral epitheliolysis has rarely been reported in the literature, we believe it should be considered in the differential diagnosis for desquamation of oral mucosae and gingiva, so dental hygiene products can be promptly replaced.

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