

Reply to: “Comment on ‘Desquamative gingivitis: Clinical findings and diseases’”



To the Editor: We would like to thank Chessa et al for their interest in our article¹ and for presenting an unusual and interesting case of oral mucosal peeling syndrome.² Although their case highlights a rare presentation of leukoedema, it should be emphasized that, overall, leukoedema is a benign, asymptomatic condition. It can be seen in all age groups, though severity increases with age, and leukoedema has no sex predilection.³ It can occur as a result of chemical or mechanical trauma, though many patients do not report any obvious insults. This condition typically affects the buccal and labial mucosal surfaces and, as such, can be a mimic of lichen planus. A helpful differentiating feature is the disappearance of more mild lesions upon stretching of the oral mucosa. Also, tooth impressions can sometimes be seen and help point toward this diagnosis. Diagnosis can be further established on histopathologic studies, which show the lichenoid infiltrates of lichen planus lacking and marked intracellular edema of the spinous layer within an acanthotic epidermis with overlying parakeratosis.³ Unlike other mimics of leukoedema, such as lichen planus or leukoplakia, this condition has no tendency to become malignant.

MacDonald et al⁴ previously reported a similar case of mucosal desquamation occurring within an area of leukoedema that resolved upon discontinuation of sodium lauryl sulfate-containing toothpaste. Similar biopsy findings were noted to this case, as histopathology demonstrated an intraepithelial cleft within an edematous epidermis with overlying parakeratosis. Their case was also attributed to be due to a desquamating form of leukoedema incited by sodium lauryl sulfate. Sodium lauryl sulfate is a strong anionic detergent that alone in healthy patients with no gingival disease has been demonstrated to result in desquamation.⁴ This effect increases with higher concentrations of sodium lauryl sulfate, with 60% of healthy persons developing desquamation at a concentration of 1.5%.⁴ Of note, this irritant response can be partially mitigated by the presence of triclosan in the toothpaste.⁵

In both of these cases, it is unclear if leukoedema provides additional susceptibility to the detergent effects of sodium lauryl sulfate or if leukoedema develops in response to the chronic irritation from the product. In addition, in neither case was patch testing to sodium lauryl sulfate

specifically performed. Despite the response to sodium lauryl sulfate being an irritant dermatitis, positive patch-test reactions to sodium lauryl sulfate have been shown to correlate to skin irritant reactions, and it would be interesting to know the severity and results of patch testing for sodium lauryl sulfate in these patients.⁶ Nonetheless, in the correct clinical context and when other more common etiologies of desquamative gingivitis have been excluded with histopathology, oral mucosal peeling syndrome can be considered; the case presented by Chessa et al² highlights an interesting additional differential diagnosis for desquamative gingivitis.

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