To the Editor:
A 34-year-old woman presented for evaluation of dry mouth and painless peeling of the oral mucosa of 2 months' duration. She denied any other skin eruptions, dry eyes, vulvar or vaginal pain, or recent hair loss. A recent antinuclear antibodies test was negative. The patient's medical history was otherwise unremarkable and her current medications included multivitamins only.

Oral examination revealed peeling gray-white tissue on the buccal mucosa and mouth floor (Figure 1). After the tissue was manually removed with a tongue blade, the mucosal base was normal in color and texture. The patient denied bruxism, biting of the mucosa or other oral trauma, or use of tobacco or nonsteroidal anti-inflammatory drugs.

Biopsies from the buccal mucosa were performed to rule out erosive lichen planus and autoimmune blistering disorders. Microscopy revealed parakeratosis and intracellular edema of the mucosa. An intraepithelial cleft at the parakeratotic surface also was present (Figure 2). Minimal inflammation was noted. Fungal staining and direct immunofluorescence were negative.

The gray-white clinical appearance of the oral mucosa resembled leukoedema, but the peeling phenomenon was uncharacteristic. Histologically, leukoedema typically has a parakeratotic and acanthotic epithelium with marked intracellular edema of the spinous layer.1,2 Our patient demonstrated intracellular edema with the additional finding of a superficial intraepithelial cleft. These features were consistent with the observed mucosal sloughing and normal tissue base and led to our diagnosis of leukoedema with mucosal desquamation. This clinical and histologic picture was previously described in another report, but a causative agent could not be identified.2
Because leukoedema can be secondary to chemical or mechanical trauma, we hypothesized that the patient’s toothpaste may be the causative agent. After discontinuing use of her regular toothpaste and keeping the rest of her oral hygiene routine unchanged, the patient’s condition resolved within 2 days. The patient could not identify how long she had been using the toothpaste before symptoms began.

Our case as well as a report in the literature suggest that leukoedema with mucosal desquamation may be the result of contact mucositis to dental hygiene products. Reports in the dental literature suggest that a possible cause for oral mucosal desquamation is sensitivity to sodium lauryl sulfate (SLS), an ingredient used in some toothpastes, including the one used by our patient. The patient has since switched to a non-SLS-containing toothpaste and has remained asymptomatic. She was unwilling to reintroduce an SLS-containing product for further evaluation.

Sodium lauryl sulfate is a strong anionic detergent that is commonly used as a foaming agent in dentifrices. In products with higher concentrations of SLS, the incidence of oral epithelial desquamation increases. Triclosan has been shown to protect against this irritant phenomenon. Interestingly, the SLS-containing toothpaste used by our patient did not contain triclosan.

Although leukoedema and mucosal desquamation induced by oral care products are well-described in the dental literature, it is important for dermatologists to be aware of this phenomenon, as the differential diagnosis includes autoimmune blistering disorders and erosive lichen planus, for which dermatology referral may be requested. Further studies of SLS and other toothpaste ingredients are needed to establish if sloughing of the oral mucosa is primarily caused by SLS or another ingredient.

REFERENCES