J Oral Maxillofac Surg
60:237-238, 2002

THE ETIOLOGY OF SUPERFICIAL ORAL MUCOCELES

To the Editor:—For those of us with a long-term interest in benign salivary gland disease, Dr Mandel has presented an unusual and very interesting case report, "Multiple Superficial Oral Mucoceles," in the August issue of the *Journal* (J Oral Maxillofac Surg 59:928, 2001). He states that "the etiology of the multiple superficial mucocele is not known." I would like to propose a cause for these lesions.

Dr Mandel clearly describes the development of the vast majority of mucoceles of the minor of accessory salivary glands as an extravasation or leakage of mucus into the surrounding submucosal tissue after trauma to the salivary duct. However, there is a cause other than physical trauma for mucocele development that is confined, in most cases, to the midportion of the junction of the hard and soft palate and almost the entire soft palate. This is the so-called retention (cyst) phenomenon, which arises as a result of narrowing of the duct openings after chronic, low-grade surface irritation. This produces ductal dilation and visible surface swelling. These lesions are significantly smaller than the extravasation mucoceles, developing from the other accessory salivary glands.

One needs only examine the excisonal biopsy specimen presented to note that the cavity is entirely intraepithelial rather than submucosal and there is a dilated duct immediately inferior to the lesion. This is exactly what you might expect to find with the retention-type mucocele.

We see this effect at times in patients with nicotine stomatitis in whom the heat or noxious tobacco products result in local surface irritation. Are there any other possible causes for mucosal irritation? The author made reference to a case report suggesting tartar control toothpaste as a possible cause. We are also aware of patients who indulge in frequent mouthwashing with hydrogen peroxide, deodorant mouthwashes, antiplaque solutions, and, the most popular, Listerine, which are clearly irritative. All of these contain significant concentrations of alcohol which, over a period of time, can affect the mucosal surface. I wonder whether the author specifically inquired about such habits or any other habits that might have a similar irritating affect when taking the medical history?

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In Reply:—I am responding to Dr Baurmash's comments regarding my publication "Multiple Superficial Oral Mucoceles: Case Report." It would seem that his critique presents 2 issues. Is the case an example of a retention cyst and is it a consequence of some form of low-grade surface inflammation with a resultant duct blockage that creates an intraepithelial retention of secretions?

Retention mucus cysts are well known, but they are not the norm when it comes to mucoceles. Definitive diagnosis of a retentive entity requires the demonstration of an intact epithelial lining around the mucus pool. No such lining was seen in my case. Dr Baurmash suggests that examination of the illustrated histology shows that the vesicle is entirely intraepithelial. I disagree. Admittedly, there is a remnant of epithelium forming what appears to be a very small lining segment at the base of the mucus pool. This appearance can be interpreted in 2 ways that would argue against an intraepithelial localization. First, it could be a result of a 3-dimensional effect, with mucus simultaneously dissecting its way through the surface epithelium at a superficial level and extending beneath the basement membrane at a deeper level and isolating the surface epithelium in the process. Another explanation rests in the observations of Eveson[1] and Jensen,[2] who state that partial epithelial regeneration occurs across the vesicle floor. In fact, Eveson[1] asserts that this regeneration gives the lesion a pseudointraepithelial appearance.

It is also important to recognize the inflammatory response infiltrating the underlying lamina propria at the base of the mucocele where the epithelium is absent. Here the mucus is in direct contact with the inflamed subepithelial tissue. Where is the limiting epithelial wall? Furthermore, close examination of the inferior peripheral margins of the mucocele shows fluid cleaving its way between the basement membrane and the lamina propria. Neither the responding inflammation at the vesicle's floor, nor the peripheral mucus extension, are something that I would expect to see in a totally confined intraepithelial lesion or in a walled off epithelial-lined retention cyst.

True, a dilated duct is visible below the mucocele, but does this prove the presence of a retention cyst? I think not. Rather, the dilation probably resulted from the back pressure created by the confined mucocele, which has failed to find an outlet for its fluid content.

Mucosal irritation was considered as a possible etiology. However, the surrounding palatal mucosa clinically showed no signs of inflammation, as per the published illustration. I too want answers; otherwise, frustration sets in. Therefore, the patient was questioned regarding mouthwashes, smoking, spices, the use of sanguinaria, exerting negative oral pressure, chewing gum, and so on. I came up with a blank.

I agree that duct orifice irritation and narrowing, with duct dilation, does occur in nicotine stomatitis. However, I have not seen mucus pools. The local swellings that are present are due to piling up of keratin around the inflamed

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duct orifice, as well as the submucosal inflammatory infiltrate.

Although minor trauma with duct laceration, is the accepted explanation for the standard mucocele, the soft palate would be the unusual recipient of such an event, particularly if there are no clinical signs or history of trauma. In my reported case, I do believe that superficially placed ducts have ruptured, with a consequent escape of mucus. It would seem that the explanation for the clinical and histologic manifestations remains an enigma, at least to me.

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References

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doi:10.1053/joms.2002.30877

MORE HARVARD REMINISCENCE

To the Editor:—I was delighted to read the reminiscences of Drs Aziz and Goldberg at Harvard. To their adventures I would like to add mine.

While serving in the United States Army during the Korean War, I wrote to the Deans of Admission of all the Dental Schools in the United States. I outlined my very average undergraduate record and requested application for admission. There were only a few replies, most suggesting additional work at the undergraduate level before again making application. The most memorable reply came from the dean at Harvard; he suggested that I consider another vocation!

Fortunately I did not take his advice. I received my DDS and MS degrees at Loyola University in Chicago and completed the residency in Oral Surgery at Cook County Hospital. Among my teachers and mentors were Sicher, Schour, Orban, Toto, Stuteville, Kostrubala, Laskin, Gans, and Sorensen. There were none better!

But for the onset of macular degeneration I would be in the operating room today!

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doi:10.1053/joms.2002.31337