

Letter to the Editor

In Reference to A 10-Year Study of the Etiopathogenesis of Cysts With a Study of Seromucinous Glands in Vocal Folds

Dear Editor:

The origin of vocal fold (VF) retention cysts (VFRCs) remains poorly understood. In their study, Nerurkar et al. concluded that decreased laryngeal hydration and vocal abuse may be predisposing factors for VFRC formation.¹

First, the daily consumption of ≤ 4 glasses of water/beverages was used to define VF dehydration, without consideration of many factors influencing the VF hydration assessment including medication, daily activity, environmental humidity, and the reflux that leads to pathophysiological mechanisms of mucus dehydration.^{2,3} The authors used the reflux finding score (RFS) for the reflux diagnosis, but the RFS-reported findings are non-specific and encountered in healthy subjects⁴ or in patients with vocal abuse.⁵

The vocal abuse origin hypothesis was based on the high clinical proportion of VFRCs in the striking zone, which had a low proportion of seromucinous glands in a cadaveric study. Precisely, the glands were detected in only 32.50% of VF samples (N = 13). This proportion is still low as reported in previous studies,⁶ raising a question about the sensitivity of the detection approach.

Second, there is no description of the materials and methods used for laryngeal observation and no definition of the striking zone. The materials description makes sense because the VF lesion visualization with a rigid endoscope may provide a false idea of the position of the lesion along the VF free edge; many lesions are located more anteriorly than they appears on the videolaryngostroboscopic exams. Consequently, the striking zone definition seems extremely difficult without a description of the materials.

Third, the authors did not provide information about the experience and skills of the laryngologists who detected the VFRCs over the 10-year study. This point is crucial, because the detection of cysts is more difficult than the detection of polyps and requires training.⁷ If the detections were made by the same laryngologists, their experience and skill would be improved throughout the 10-year period, leading to a biased incidence evolution.

Fourth, the authors supported their hypothesis of the vocal abuse origin on studies reporting similar findings.^{7,8} Neither Nerurkar et al. nor any of the above-mentioned

authors have differentiated VFRCs and epidermoid cysts (ECs); one study being characterized by a higher proportion of ECs.⁸ This point undoubtedly leads to biased conclusions, because the pathophysiology of these two types of cysts differs. VFRCs are related to a blockage of the ductal system of the seromucinous glands, whereas ECs would be broadly associated with vocal abuse, phonotrauma, and epidermoid cell invagination into Reinke's space.⁹ The female/male ratio, the VFRCs low recurrence rate, and the poor inflammatory reaction around the excised VFRCs are other clinical differences between VFRCs and phonotrauma lesions such as ECs.^{3,9,10}

The article by Nerurkar et al. highlights the importance of studying the predisposing factors of VFRCs and ECs. However, it is premature to state that vocal abuse may be a predisposing factor for VFRCs. Future prospective studies identifying the predisposing factors should consider VFRCs and ECs separately, with special attention for clinical presentation (materials/methods), postoperative evolution, and the association between reflux, VF dehydration, reflux-induced hoarseness, and compensatory voice behaviors.

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