Adenoid Movement Contributing to Acquired Velopharyngeal Insufficiency

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An 18 years old male presented to our clinic with complaints of chronic tonsillitis. On examination, his tonsils were 3+ in size and no submucus cleft was appreciated. A decision to perform tonsillectomy was made. Tonsillectomy was performed without any intraoperative complications. No adenoidectomy was performed. Immediately after surgery, the patient noted hypernasal speech and nasal regurgitation on *per os* fluid intake. Video fiberoptic nasopharyngoscopy was performed while the patient vocalized sibilant sounds (/s/ and /sh/) as well as words beginning with plosive consonants (/p/ and /b/). Figure 1 represents time-elapsed still captures taken from the video recording while the patient verbalized the /b/ in the word “baby.” A central velopharyngeal insufficiency was seen and also consistently found on other sibilant and plosive enunciations. Interestingly, it was also noted that the lateral aspects of the adenoid bed completely medialized and contributed to the velopharyngeal closure, though incompletely with the soft palate resulting in insufficiency. Figure 2 shows the adenoid contribution more clearly when the nasopharyngoscope tip was positioned midway in the nasal cavity facing posteriorly.

This is the first reported case in the literature that we are aware of where the adenoid tissue itself moved to the midline contributing to velopharyneal closure. This unique “circular” closure pattern does not easily fit into the current system of valving patterns as described initially by Skolnick, et al in 1973.¹ The classic circular pattern of velopharyngeal closure entails medialization of the lateral pharyngeal walls and soft palate elevation. The other 3 classic patterns can be classified as: coronal, circular with Passavant’s ridge, and sagittal. Variations on these patterns have been described influenced by adenoid shape and size, soft palate physical as
well as functional abnormalities, and uvular movement. One can now add adenoid movement as another possible variable.

Given we did not obtain a nasopharyngoscopy exam prior to tonsillectomy, it is hard to say whether the current closure pattern existed prior to surgery or whether this closure pattern developed after tonsillectomy to compensate for the lack of tonsillar contribution (true circular pattern). Regardless, given it was apparent that the adenoid cleft was a factor contributing to the velopharyngeal insufficiency, the following options for correction were presented to the patient:

1) Adenoidectomy based on the idea that the lateral adenoid bulk itself was contributing to the velopharyngeal insufficiency
2) Posterior pharyngeal flap
3) Cymetra injection nasopharyngoplasty (into the adenoid cleft)
4) Do nothing and give it time for spontaneous resolution.

In the end, the patient elected for monitoring. On follow-up 2 months later, all symptoms of velopharyngeal insufficiency resolved without any specific intervention. Patient declined repeat nasopharyngoscopy.

References

Figure 1: Time-elapsed still captures from a video recording during a video fiberoptic nasopharyngoscopy examination. The nasopharyngoscope tip is positioned right at the nasopharyngeal inlet looking down into the oropharynx. The patient is enunciating the plosive /b/ in “baby” in this figure. Note the medialization of the lateral aspects of the adenoid bed contributing to velopharyngeal closure. In spite of the adenoid contribution to closure, a central velopharyngeal insufficiency with the soft palate exists.
Figure 2: Time-elapsed still captures from a video recording during a video fiberoptic nasopharyngoscopy examination. The nasopharyngoscope tip is positioned midway in the nasal cavity facing the nasopharynx. One can see with phonation that the lateral aspects of the adenoid bed medializes and contributes to velopharyngeal closure.