



LARYNGOPHARYNGEAL REFLUX AS A DETERMINANT OF POSTOPERATIVE HEALING AFTER ENDOLARYNGEAL MICROSURGERY: A CONTROLLED CLINICAL STUDY

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ABSTRACT

Background: Laryngopharyngeal reflux (LPR) is an important factor affecting laryngeal mucosal integrity and may impair postoperative healing. **Objective:** To evaluate the effect of postoperative LPR treatment on wound healing and acoustic voice outcomes following endolaryngeal microsurgery (EM). **Methods:** Forty-two patients undergoing EM were divided into LPR-treated and untreated groups. Serial videolaryngostroboscopy (VLS) and acoustic analyses were performed over a 6-month follow-up period. **Results:** The LPR-treated group demonstrated significantly faster recovery of vocal fold symmetry and mucosal wave propagation. Jitter and shimmer decreased, while harmonic-to-noise ratio (HNR) and signal-to-noise ratio (SNR) increased significantly ($p < 0.05$). **Conclusion:** Postoperative LPR management significantly enhances both structural and functional recovery following EM.

INTRODUCTION

Voice disorders affect approximately 3–9% of the population and represent a significant clinical burden.^[1]

Benign vocal fold lesions such as nodules, polyps, cysts, leukoplakia, Reinke's edema, and sulcus vocalis primarily arise from trauma affecting the lamina propria and are commonly treated with endolaryngeal microsurgery (EM).^[2]

Laryngopharyngeal reflux (LPR) has emerged as an important contributor to laryngeal mucosal injury. Unlike the esophagus, the larynx lacks effective protective mechanisms such as bicarbonate secretion, mucosal barrier function, and efficient acid clearance. Therefore, even limited exposure to gastric acid and pepsin may result in clinically significant injury.^[3-5]

Postoperative healing after EM is a dynamic biological process involving inflammation, extracellular matrix deposition, epithelialization, and remodeling. These phases may persist for several months, and disturbances

during this process may result in fibrosis, impaired vibratory function, and persistent dysphonia.^[6,7]

Experimental data suggest that gastric acid exposure negatively affects laryngeal wound healing and may promote scar formation, stenosis, and prolonged inflammation.^[8,9] Clinical studies have also demonstrated that reflux is highly prevalent among patients with laryngeal and voice disorders and can be detected using dedicated diagnostic methods.^[10,11]

Based on these observations, postoperative LPR treatment may improve both structural healing and functional voice recovery following EM. Therefore, the present study aimed to evaluate the impact of postoperative anti-reflux therapy on videolaryngostroboscopic and acoustic voice parameters in patients undergoing EM.

MATERIALS AND METHODS

This controlled study included 42 patients undergoing EM for benign vocal fold lesions. Patients were divided

into two groups: a treatment group receiving lansoprazole 30 mg twice daily combined with alginate acid therapy for 6 months, and a control group receiving no specific LPR treatment.

Follow-up evaluations were conducted at 1 week, 2 weeks, 1 month, 2 months, and 6 months postoperatively.

Outcome measures included videolaryngostroboscopic parameters (symmetry, mucosal wave, and amplitude) and acoustic parameters (fundamental frequency, jitter, shimmer, HNR, and SNR).

Statistical analyses were performed using the Mann–Whitney U test, Wilcoxon signed-rank test, and Spearman correlation analysis. A p-value of < 0.05 was considered statistically significant.

RESULTS

A total of 42 patients were included in the analysis, with 21 patients in the treatment group and 21 in the control group. Baseline demographic and clinical characteristics, including age and sex distribution, were comparable between groups ($p > 0.05$), indicating homogeneity of the study population (Table 1).

Table 1: Indicating homogeneity of the study population.

Time	Treatment Jitter	Control Jitter
Pre	1.98	2.02
1M	0.55	0.93
2M	0.22	1.05
6M	0.15	0.45

Videolaryngostroboscopic (VLS) evaluation demonstrated a significantly faster recovery pattern in the treatment group. Restoration of vocal fold symmetry was observed as early as the first postoperative month in the treatment group, whereas similar findings were delayed until the second month in the control group. Likewise, normalization of mucosal wave propagation occurred earlier and more consistently among patients receiving LPR treatment.

Acoustic analysis revealed statistically significant improvements in all measured parameters in the treatment group compared with controls (Table 1). Jitter values decreased from 1.98 ± 0.6 to 0.15 ± 0.05 in the treatment group, whereas the control group showed a more limited reduction from 2.02 ± 0.7 to 0.45 ± 0.1 ($p < 0.05$). Similarly, shimmer values demonstrated a greater reduction in the treatment group.

Harmonic-to-noise ratio (HNR) and signal-to-noise ratio (SNR) increased significantly in the treatment group, reflecting improved voice quality and stability. These improvements were less pronounced in the control group ($p < 0.05$).

Correlation analysis demonstrated a strong association between structural recovery (VLS findings) and functional acoustic improvements, suggesting that enhanced mucosal healing directly translates into improved phonatory performance. Figure 1 illustrates the temporal recovery trajectory of key acoustic parameters in both groups.

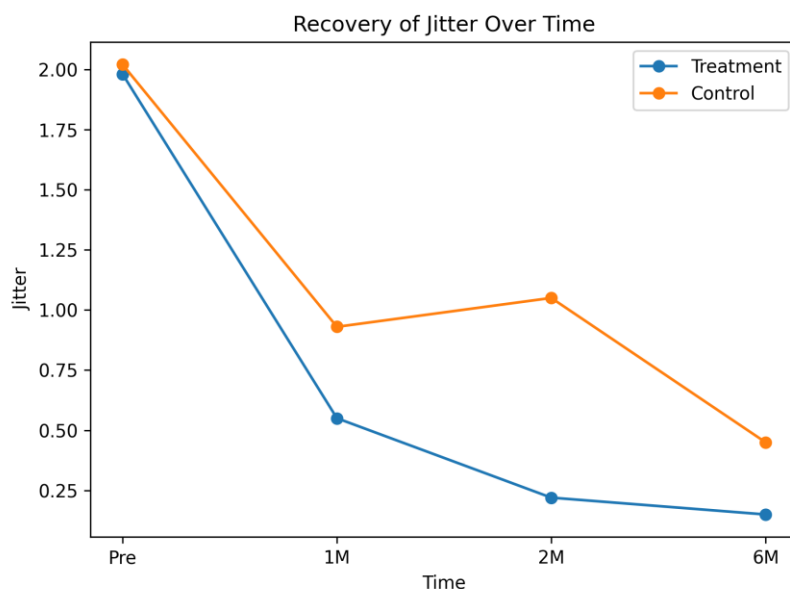


Figure 1: Recovery of jitter over time.

DISCUSSION

This study demonstrates that LPR is a significant determinant of postoperative healing following EM. The biological plausibility of this finding is well supported,

as refluxed gastric contents—particularly acid and pepsin—can prolong inflammation, delay epithelial regeneration, and disrupt extracellular matrix remodeling in laryngeal tissue.^[3,8,9]

The viscoelastic properties of the vocal folds depend on the structural integrity of the lamina propria. Therefore, any disruption in the wound healing process may directly impair phonatory function. Experimental studies have shown that abnormal healing is associated with disorganized collagen deposition, reduced elastin and hyaluronic acid content, and impaired tissue biomechanics.^[6,7]

The acoustic findings observed in this study are clinically meaningful. Reductions in jitter and shimmer indicate improved cycle-to-cycle vibratory stability, while increases in HNR and SNR reflect enhanced harmonic structure and reduced noise within the voice signal. These findings support the concept that improved mucosal healing leads directly to improved voice quality.^[10,11]

From a therapeutic perspective, the results support the use of postoperative anti-reflux therapy in selected patients undergoing EM. Previous studies have shown that reflux symptoms may improve earlier than objective laryngeal findings, suggesting that treatment duration should be sufficient to influence tissue-level healing rather than symptom control alone.^[12]

These findings are also consistent with the broader principles of phonosurgery, which emphasize not only precise lesion removal but also preservation of healthy tissue and optimization of the postoperative environment.^[13,14] Emerging regenerative approaches, including growth factor therapy and mesenchymal stem cell applications, may further enhance healing in the future. However, current clinical practice relies primarily on controlling modifiable factors such as reflux.^[15]

The main limitations of this study include its retrospective design and the lack of routine objective reflux monitoring. Nevertheless, the findings reflect real-world clinical practice and provide clinically relevant evidence that postoperative LPR treatment may accelerate healing and improve voice outcomes following EM.

CONCLUSION

Postoperative LPR treatment significantly accelerates wound healing and improves acoustic voice outcomes following endolaryngeal microsurgery. Given the sensitivity of laryngeal tissues to reflux-related injury and the importance of optimal healing for successful phonatory recovery, anti-reflux therapy should be considered an essential component of postoperative management in appropriately selected patients.

REFERENCES

1. Berry DA, Verdolini K, Montequin DW, Hess MM, Chan RW, Titze IR. A quantitative output-cost ratio in voice production. *J Speech Lang Hear Res.*, 2001; 44: 29-37.

2. Johns MM. Update on the etiology, diagnosis, and treatment of vocal fold nodules, polyps, and cysts. *Curr Opin Otolaryngol Head Neck Surg*, 2003; 11: 456-461.
3. Koufman JA. The otolaryngologic manifestations of gastroesophageal reflux disease (GERD): a clinical investigation of 225 patients using ambulatory 24-hour pH monitoring and an experimental investigation of the role of acid and pepsin in the development of laryngeal injury. *Laryngoscope*, 1991; 101: 1-78.
4. Postma GN, Belafsky PC, Aviv JE, Koufman JA. Laryngopharyngeal reflux testing. *Ear Nose Throat J.*, 2002; 81(9,2): 14-18.
5. Helm JF, Dodds WJ, Hogan WJ, Soergel KH, Egide MS, Wood CM. Acid neutralizing capacity of human saliva. *Gastroenterology*, 1982; 83: 69-74.
6. Branski RC, Rosen CA, Verdolini K, Hebda PA. Acute vocal fold wound healing in a rabbit model. *Ann Otol Rhinol Laryngol*, 2005; 114: 19-24.
7. Thibeault SL, Gray SD, Bless DM, Chan RW, Ford CN. Histologic and rheologic characterization of vocal fold scarring. *J Voice*, 2002; 16: 96-104.
8. Little FB, Koufman JA, Kohut RI, Marshall RB. Effect of gastric acid on the pathogenesis of subglottic stenosis. *Ann Otol Rhinol Laryngol*, 1985; 94: 516-519.
9. Johnston N, Knight J, Dettmar PW, Lively MO, Koufman JA. Pepsin and carbonic anhydrase isoenzyme III as diagnostic markers for laryngopharyngeal reflux disease. *Laryngoscope*, 2004; 114: 2129-2134.
10. Koufman JA, Amin MR, Panetti M. Prevalence of reflux in 113 consecutive patients with laryngeal and voice disorders. *Otolaryngol Head Neck Surg*, 2000; 123: 385-388.
11. Rūta P, Virgilijus U, Viktoras S. Multidimensional voice analysis of reflux laryngitis patients. *Eur Arch Otorhinolaryngol*, 2005; 262: 35-40.
12. Belafsky PC, Postma GN, Koufman JA. Laryngopharyngeal reflux symptoms improve before changes in physical findings. *Laryngoscope*, 2001; 111: 979-981.
13. Hirano M. Chevalier Jackson Lecture 1995: Phonosurgery: past, present, and future. *Trans Am Bronchoesophagol Assoc*, 1995; 22-30.
14. Benninger MS, Alessi D, Archer S, et al. Vocal fold scarring: current concepts and management. *Otolaryngol Head Neck Surg*, 1996; 115: 474-482.
15. Kanemaru S, Nakamura T, Omori K, et al. Regeneration of the vocal fold using autologous mesenchymal stem cells. *Ann Otol Rhinol Laryngol*, 2003; 112: 915-920.