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RESEARCH

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The role of hoarseness in the reflux symptom index: diagnostic overlap between laryngopharyngeal reflux and dysphonia

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Abstract

Background The Reflux Symptom Index (RSI) is widely used for assessing laryngopharyngeal reflux (LPR). However, hoarseness—a symptom included in the RSI—may arise from numerous non-reflux-related voice disorders, which can inflate scores and compromise diagnostic specificity.

Methods This retrospective study included 239 patients diagnosed with voice disorders. Voice Handicap Index (VHI) and RSI scores were obtained from clinical records. Gastroesophageal reflux disease (GERD) was confirmed by 24-h oesophageal pH monitoring. Univariate linear regression and multivariate generalised linear modelling were performed to examine the relationship between VHI, GERD, and RSI scores.

Results Univariate analysis revealed that each 1-unit increase in VHI score corresponded to a 0.348-unit increase in RSI score (B = 0.348, SE = 0.056, $p < 0.001$). Multivariate analysis confirmed that VHI remained an independent predictor of RSI, irrespective of GERD status ($p < 0.001$). Overall, 59% of patients had RSI scores ≥ 13, including 41.5% of those without GERD, suggesting substantial diagnostic overlap.

Conclusion Hoarseness-related voice handicap significantly elevates RSI scores independent of reflux status. Clinicians should exercise caution when interpreting elevated RSI values in patients with voice disorders and consider complementary diagnostic tools to prevent misdiagnosis and unnecessary antireflux therapy.

Keywords Gastroesophageal reflux disease, Hoarseness, Laryngopharyngeal reflux, Voice disorder

Background

Laryngopharyngeal reflux (LPR) is a clinical condition in which gastric contents reflux into the upper aerodigestive tract, causing a variety of laryngeal and pharyngeal symptoms [1]. In the United States, LPR has been reported to affect up to 50% of individuals with voice complaints [2] and has been implicated in the pathogenesis of several laryngeal disorders, including vocal fold nodules, granulomas, chronic laryngitis, and subglottic stenosis [3, 4].

The gold standard for diagnosing LPR is 24-h multichannel intraluminal impedance pH monitoring, which allows for objective detection of pharyngeal reflux episodes. However, its high cost, limited availability, and low patient tolerance have restricted its use in routine clinical practice [1]. To overcome these limitations, clinicians have increasingly relied on subjective assessment tools,

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such as the Reflux Symptom Index (RSI) and Reflux Finding Score (RFS), both developed by Belafsky et al. [5, 6]. The RSI is a nine-item self-administered questionnaire designed to evaluate the severity of LPR-related symptoms, with scores ≥ 13 considered indicative of LPR [5].

Notably, the first item in the RSI—"Hoarseness or a problem with your voice"—targets a symptom that is not unique to LPR and is commonly observed in numerous other laryngeal pathologies. Hoarseness can result from structural lesions (e.g., nodules, polyps), inflammatory or infectious processes, neurological conditions, or functional voice disorders [7]. Thus, patients with non-reflux-related voice disorders may score high on the RSI solely due to hoarseness, even in the absence of true reflux pathology.

This raises concern about the specificity of the RSI in accurately diagnosing LPR among patients with primary voice disorders. Despite its widespread use, only a limited number of studies have critically examined the potential confounding effect of hoarseness on RSI scoring.

Although gastroesophageal reflux disease (GERD) and LPR are conceptually distinct entities—differing in pathophysiology, symptomatology, and diagnostic criteria—their clinical manifestations often overlap [8]. Moreover, current patient-reported outcome tools such as the RSI do not differentiate between these conditions. Given this diagnostic ambiguity, we defined reflux status based on clinical GERD diagnosis established by the gastroenterology department using 24-h oesophageal pH manometry. This classification was used to stratify patients into reflux-positive and reflux-negative groups for the purpose of statistical analysis.

In this context, we hypothesised that hoarseness—regardless of underlying reflux status—may artificially inflate RSI scores, potentially leading to overdiagnosis of LPR. Therefore, the aim of this study was to evaluate the relationship between hoarseness (measured by the Voice Handicap Index [VHI]) and the RSI score, and to determine whether the RSI score remains elevated to a clinically significant extent in patients with voice disorders even in the absence of GERD.

Methods

Study design and participants

This retrospective study included 239 patients who presented to the otolaryngology outpatient clinic with complaints of hoarseness and were diagnosed with various voice disorders. The study was conducted in accordance with the principles of the Declaration of Helsinki and approved by the Ankara University Human Research Ethics Committee (Approval No. I8-539–20; 28 September 2020). The requirement for written informed consent was waived due to the retrospective design.

Voice disorder diagnoses were established through comprehensive otolaryngological examination. All patients underwent videolaryngostroboscopy (VLS) to assess vocal fold structure, mucosal wave characteristics, and glottic closure patterns. Perceptual voice evaluation was performed during standard clinical assessment, and diagnostic confirmation was made by at least two experienced laryngologists. Patients with neurological conditions or systemic disorders known to affect phonation were excluded.

Demographic characteristics, RSI scores, and VHI scores were collected from patient records. GERD was diagnosed by the gastroenterology department based on 24-h oesophageal pH monitoring. To evaluate the presence of LPR, RSI scoring was used. According to the original study by Belafsky et al., scores ≥ 13 are considered indicative of LPR [5]. Additionally, the Turkish validity study by Akbulut et al. supports a similar diagnostic threshold [9].

Statistical Analysis

All statistical analyses were performed using SPSS version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) or median (range), and categorical variables as counts and percentages. The Shapiro–Wilk test was applied to assess the normality of distribution.

Comparisons between groups were performed using the independent-samples t-test or Mann–Whitney U test for continuous variables, and the chi-square test or Fisher's exact test for categorical variables. Spearman's correlation coefficient was calculated to evaluate the association between RSI and VHI scores. Univariate linear regression was used to assess the effect of VHI on RSI scores. A multivariate generalised linear model was subsequently employed to determine whether VHI independently predicted RSI scores after adjusting for GERD status. A p-value < 0.05 was considered statistically significant.

Results

The study included 239 patients with a mean age of 43.4 ± 12.6 years. Of the total cohort, 54.0% were females, 54.8% had a history of smoking, and 49.4% reported vocal abuse or misuse. Comorbidities other than GERD were present in 25.1% of patients, with hypertension being the most common. The distribution of voice disorders was as follows: vocal fold polyps (30.5%), sulcus vocalis (21.8%), Reinke's oedema (20.9%), vocal fold nodules (10.5%), intracordal cysts (7.5%), muscle tension dysphonia (3.8%), chronic laryngitis (1.7%), mutational falsetto (1.7%), spasmodic dysphonia (1.3%), and vocal tremor (0.4%).

A confirmed diagnosis of GERD based on 24-h pH oesophageal monitoring was present in 104 patients (43.5%). No statistically significant differences were observed between GERD-positive and GERD-negative patients with in terms of age, sex, smoking status, history of vocal abuse, comorbidities, or distribution of voice disorders (Table 1).

The median VHI score for the entire population was 23 (range, 0–44), and the median RSI score was 15 (range, 0–49). Patients with GERD had significantly higher VHI and RSI scores compared with those without GERD ($p=0.003$ and $p<0.001$, respectively). Overall, 141 patients (59%) had RSI scores ≥ 13. RSI ≥ 13 was observed in 85/104 (81.7%) GERD-positive patients compared with 56/135 (41.5%) GERD-negative patients (χ^2, $p<0.001$; Fig. 1). Whilst elevated RSI was frequently encountered in the absence of documented GERD, this pattern is consistent with limited specificity and potential diagnostic overlap rather than definitive false positivity.

When stratified by voice disorder type, elevated RSI scores (≥ 13) were observed across nearly all diagnostic categories (Fig. 2). The highest frequencies were found in patients with vocal fold polyps (42/73, 57.5%), sulcus vocalis (32/52, 61.5%), and Reinke's oedema (34/50, 68.0%). Even among functional conditions such as muscle tension dysphonia (6/9, 66.7%), the majority exceeded the RSI cut-off, indicating that hoarseness and related complaints may inflate RSI irrespective of the underlying aetiology.

Spearman's correlation analysis revealed a moderate positive correlation between VHI and RSI scores ($r=0.399$, $p<0.001$; Fig. 3). In univariate linear regression, each 1-unit increase in VHI score resulted in a 0.348-unit increase in RSI score (B = 0.348, SE = 0.056, $p<0.001$). Multivariate generalised linear modelling further confirmed that the VHI score remained an independent predictor of RSI score after adjustment for GERD status (Table 2).

Discussion

LPR is recognised as the most common extraesophageal manifestation of GERD [3]. Its typical symptoms include postnasal drip, dysphagia, hoarseness, chronic cough, and frequent throat clearing, many of which overlap with other benign laryngeal conditions [3, 6, 10–12].

In clinical practice, the RSI, introduced by Belafsky et al., is widely used for the assessment of LPR symptoms due to its simplicity and non-invasive nature [5]. However, emerging literature has raised concerns

Table 1 Comparison of baseline characteristics of patients with and without GERD

Characteristics	Total, *n* = 239	GERD		*p* value
		Present, *n* = 104	Absent, *n* = 135	
Age, mean ± SD, years	43.4 ± 12.6	43.1 ± 12.7	43.6 ± 12.5	0.788
Female sex, *n* (%)	129 (54.0)	58 (55.8)	71 (52.6)	0.625
Smoking, *n* (%)	131 (54.8)	59 (56.7)	72 (53.3)	0.601
Vocal abuse/misuse, *n* (%)	118 (49.4)	53 (51.0)	65 (48.1)	0.666
Comorbidities, *n* (%)	60 (25.1)	21 (20.2)	39 (28.9)	0.124
Hypertension	22 (9.2)	9 (8.7)	13 (9.6)	0.796
Hypothyroidism	17 (7.1)	5 (4.8)	12 (8.9)	0.224
Diabetes mellitus	11 (4.6)	5 (4.8)	6 (4.4)	0.894
Asthma	5 (2.1)	0 (0.0)	5 (3.7)	0.070
Others	23 (9.6)	7 (6.7)	16 (11.9)	0.183
Voice disorder, *n* (%)				
Vocal fold polyps	73 (30.5)	35 (33.7)	38 (28.1)	0.948
Sulcus vocalis	52 (21.8)	21 (20.2)	31 (23.0)	
Reinke's oedema	50 (20.9)	22 (21.2)	28 (20.7)	
Vocal fold nodules	25 (10.5)	10 (9.6)	15 (11.1)	
Intracordal cysts	18 (7.5)	8 (7.7)	10 (7.4)	
MTD	9 (3.8)	3 (2.9)	6 (4.4)	
Chronic laryngitis	4 (1.7)	1 (1.0)	3 (2.2)	
Mutational falsetto	4 (1.7)	2 (1.9)	2 (1.5)	
Spasmodic dysphonia	3 (1.3)	1 (1.0)	2 (1.5)	
Vocal tremor	1 (0.4)	1 (1.0)	0 (0.0)	
VHI, median (min–max)	23 (0–44)	25 (0–43)	21 (0–44)	**0.003**
RSI, median (min–max)	15 (0–49)	22 (1–49)	10 (0–40)	**< 0.001**

Abbreviations: *GERD* Gastroesophageal reflux disease, *MTD* Muscle tension dysphonia, *RSI* Reflux symptom index, *SD* Standard deviation, *VHI* Voice handicap index

* Bold text indicates statistical significance at $p<0.05$ level

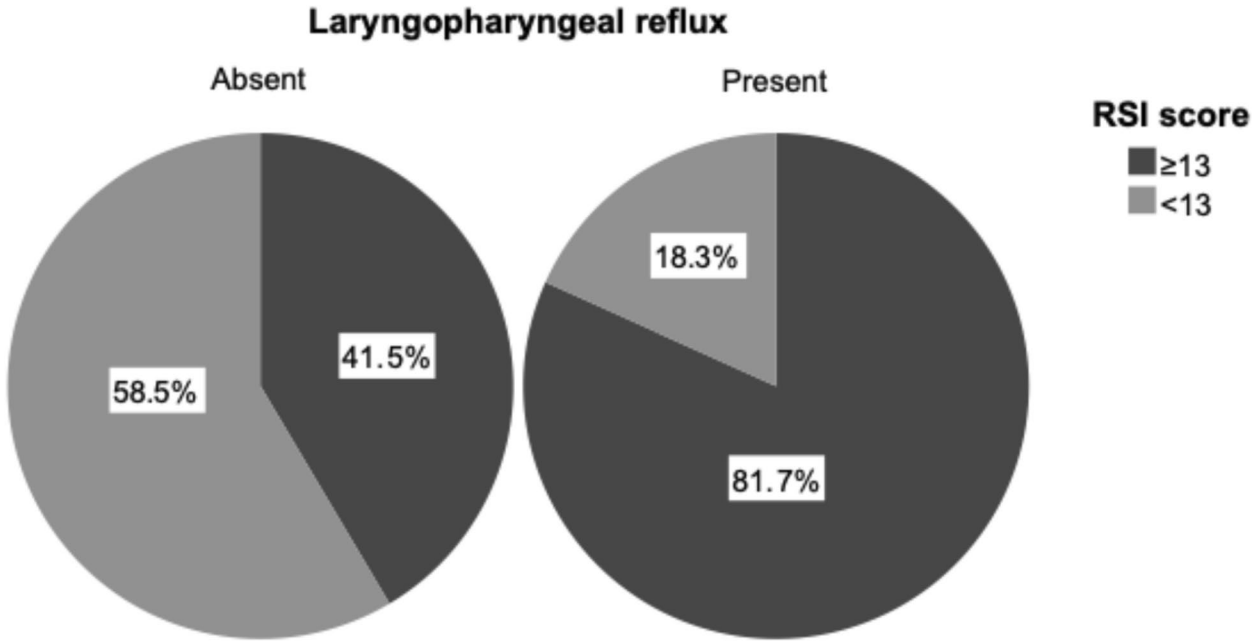


Fig. 1 Distribution of patients with and without gastroesophageal reflux disease according to RSI scores ($p < 0.001$)

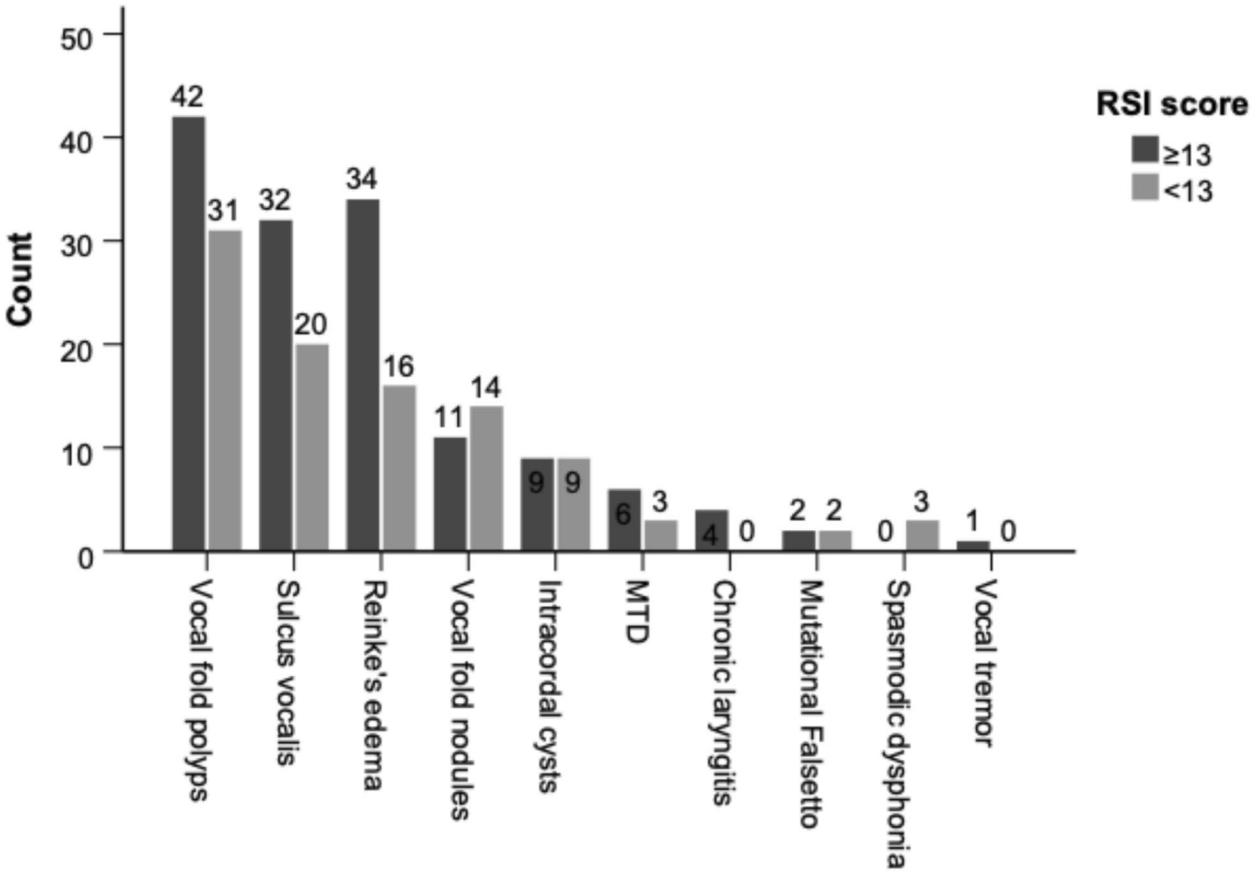


Fig. 2 Distribution of RSI scores (≥ 13 and < 13) among patients with different voice disorders

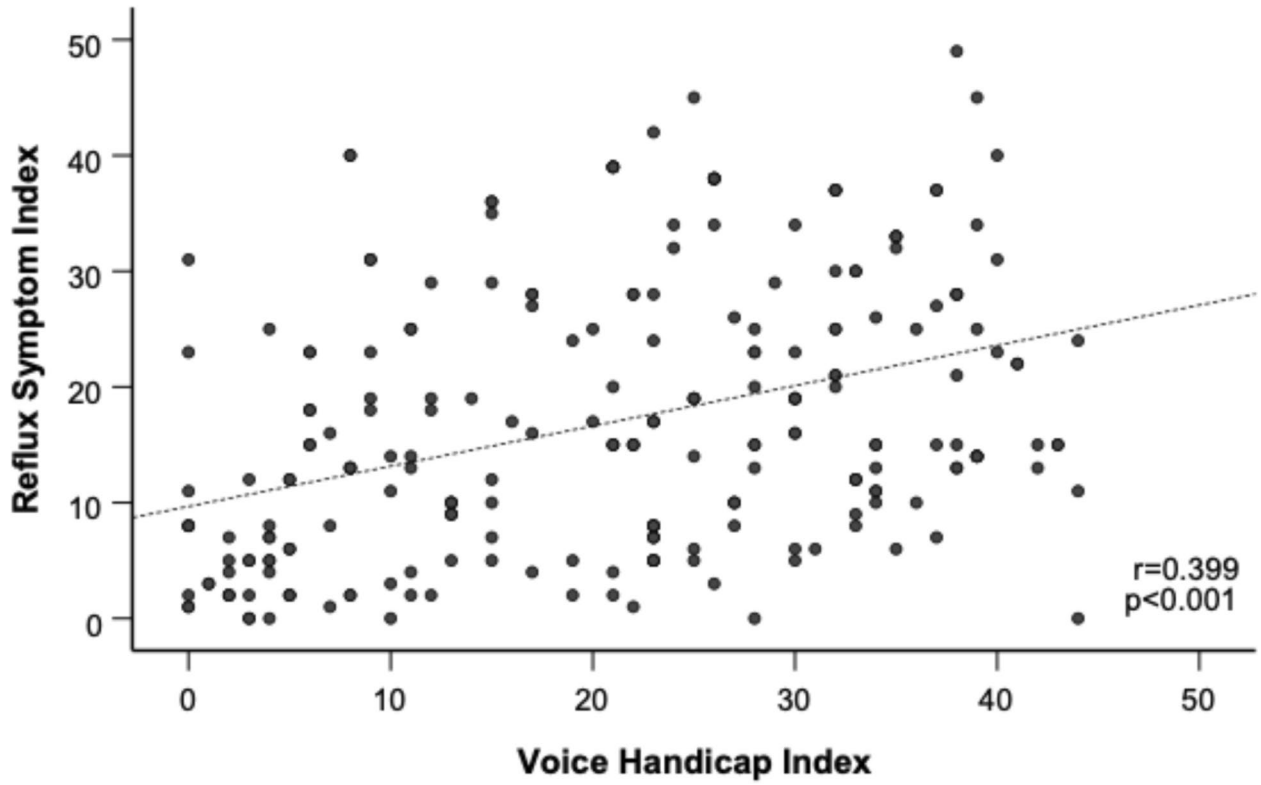


Fig. 3 Spearman correlation analysis between VHI and RSI scores

Table 2 Multivariate generalized linear model assessing the effects of GERD and VHI on reflux symptom index

Parameters	B	SE	Wald chi-square	*p* value
GERD	8.563	1.336	41.11	**< 0.001**
VHI	0.283	0.053	28.45	**< 0.001**

Abbreviations: *B* Beta coefficient, *GERD* Gastroesophageal reflux disease, *SE* Standard error, *VHI* Voice handicap index

* Bold text indicates statistical significance at $p < 0.05$ level

regarding the diagnostic limitations of the RSI, particularly when used in isolation [13–16].

Multiple studies have demonstrated that both the RSI and the RFS have limited sensitivity and specificity for confirming LPR. Their diagnostic accuracy is particularly low when not supported by objective tools such as 24-h impedance pH monitoring [13–17]. In particular, Nacci et al. highlighted the risk of false positives associated with relying solely on RSI scores, and advocated for refinement of the RSI to improve its diagnostic accuracy [17].

One key factor contributing to the potential overestimation of reflux symptoms on the RSI is hoarseness—a symptom included as the first item in the scale. Hoarseness is a non-specific complaint that may arise from a wide range of aetiologies, including vocal fold lesions, inflammatory disorders, neurogenic conditions, and functional dysphonia [7, 18–21]. While LPR may indeed contribute to hoarseness, it is seldom the sole cause in patients with established voice disorders.

The present study addresses this gap by demonstrating a significant association between VHI and RSI scores, independent of reflux status. Our univariate and multivariate analyses confirmed that hoarseness-related voice handicap can independently elevate RSI scores, even in the absence of GERD. This was further evidenced by the finding that 41.5% of patients without GERD exhibited RSI scores ≥ 13—the conventional cut-off for suspected LPR.

These results suggest that the RSI may overestimate reflux symptoms in patients whose hoarseness originates from non-reflux-related causes, potentially leading to misdiagnosis and unnecessary treatment. Clinicians should be cautious when interpreting elevated RSI scores in patients with known voice disorders, and consider complementary assessment tools such as the VHI or laryngeal examination findings before initiating empirical antireflux therapy.

The distribution of RSI ≥ 13 across structural, functional, and neurogenic dysphonias emphasises the non-specificity of hoarseness in the RSI. Notably, more than half of patients with vocal fold polyps, sulcus vocalis, and Reinke's oedema surpassed the RSI threshold, despite the absence of GERD in many cases. This supports the notion that RSI elevation reflects voice handicap burden as much as reflux, and strengthens the argument for complementary assessment tools in this population [3, 5, 17, 22].

It should also be acknowledged that LPR is not merely a mimic of dysphonia but a well-established etiological factor in structural vocal fold pathology. LPR-related laryngeal oedema, vocal fold swelling, and pseudosulcus formation may coexist with other benign laryngeal disorders. In such cases, elevated RSI scores in patients with non-reflux-related voice disorders may still represent genuine reflux involvement rather than false positives. This diagnostic overlap complicates interpretation and underscores the need for multimodal assessment, including both symptom and sign-based tools, to distinguish primary dysphonia from concomitant LPR [3, 6, 22].

An important distinction that must be emphasised is that GERD and LPR involve different pathophysiological mechanisms. GERD is primarily related to lower oesophageal sphincter (LES) dysfunction, whereas LPR results from upper oesophageal sphincter (UES) incompetence. Therefore, the presence of GERD does not necessarily imply the presence of LPR, and vice versa. In the present study, GERD status was confirmed by 24-h distal oesophageal pH monitoring performed by the gastroenterology team. This technique evaluates acid exposure at the level of the LES but does not directly capture proximal or hypopharyngeal reflux events. Consequently, using GERD as a surrogate for laryngeal reflux may lead to misclassification, which should be considered when interpreting our findings [22, 23].

Smoking and sex differences have also been proposed as potential modulators of reflux-related symptoms. However, in our cohort, neither smoking status nor sex showed a significant association with GERD diagnosis. This may reflect the retrospective design, incomplete capture of lifestyle factors, or the multifactorial nature of GERD pathogenesis. Future prospective studies with systematic assessment of lifestyle and demographic covariates are warranted to clarify these relationships [22–24]. This highlights the importance of considering demographic and lifestyle factors in future research to refine risk stratification in reflux-related voice disorders.

The relationship between GERD and LPR remains complex. While 24-h oesophageal pH monitoring is considered the gold standard for diagnosing GERD, there is currently no universally accepted diagnostic gold standard for LPR. Symptom- and sign-based instruments such as the RSI and RFS, although widely used, lack sufficient specificity to distinguish reflux-related dysphonia from other causes. Importantly, dysphonia is not required for LPR, and GERD is not required for LPR either; these conditions may overlap but also frequently occur independently. This underscores the need to interpret RSI scores cautiously and in conjunction with laryngeal findings, clinical history, and, where feasible, objective proximal pH or impedance monitoring [22, 23].

Moreover, these findings raise important questions regarding the design of the RSI itself. Given that hoarseness is a frequent but non-specific symptom, future iterations of the RSI may benefit from item reweighting or stratified interpretation when applied to populations with pre-existing voice disorders.

Limitations

This study has several limitations. First, because of its retrospective design, the RFS was not consistently documented, limiting our ability to determine whether elevated RSI scores reflected true reflux-related pathology or coincidental findings in dysphonia. Second, GERD status was determined by distal oesophageal pH monitoring, which cannot adequately capture proximal or hypopharyngeal reflux events and may have led to misclassification. Third, although subgroup analysis revealed a higher prevalence of RSI ≥ 13 in GERD-positive patients, the absence of standardised laryngeal sign assessment limits definitive attribution of these elevations to LPR. Fourth, dietary data—an important contributor to reflux—were not available for analysis. Finally, although smoking status and sex were recorded, no significant associations with GERD were observed, which may reflect incomplete capture of lifestyle variables and the multifactorial nature of reflux pathogenesis. These limitations should be considered when interpreting the findings of this study.

Conclusion

This study demonstrated that hoarseness—quantified by the VHI—is an independent contributor to elevated RSI scores, independent of gastroesophageal reflux status. A substantial proportion of patients with voice disorders but without confirmed GERD exhibited RSI scores above the diagnostic threshold, indicating that RSI may overestimate reflux symptoms in this population.

These findings suggest that the presence of hoarseness from non-reflux-related etiologies can lead to apparent false-positive RSI elevations, potentially resulting in misdiagnosis and overtreatment of LPR. Clinicians should interpret RSI scores with caution in patients presenting with primary voice disorders and consider adjunctive

tools such as the VHI or objective pH monitoring when making treatment decisions.

Future research should focus on refining the RSI by adjusting symptom weighting or developing modified scoring systems that better account for overlapping laryngeal pathologies to improve diagnostic specificity in patients with voice disorders.

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Authors' contributions
E: Collecting the data of the study, conceiving and designing the study, obtaining ethics approval, writing the manuscript. C: Conceiving and designing the study. A: Collecting the data of the study. S: Collecting the data of the study. G: Conceiving the study.

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Data availability
The data that support the findings of this study are not publicly available due to privacy concerns, but may be obtained from the corresponding author upon reasonable request.

Declarations

Ethics and consent to participate
The study was approved by the Ankara University Human Research Ethics Committee (Approval No. I8-539–20; 28 September 2020), and the requirement for written informed consent was waived due to the retrospective nature of the study.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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