Section: ENT

Original Research Article

AN ANALYTICAL STUDY ON PHONATION AMONG MALES WITH VOICE-RELATED SYMPTOMS IN NON-SMOKERS VERSUS CHRONIC SMOKERS

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ABSTRACT

Background: Smoking is a well-known risk factor for voice disorders, contributing to chronic inflammation and structural changes in the larynx. These changes adversely affect vocal quality and can be effectively evaluated using a combination of videostroboscopy, acoustic analysis and perceptual assessment tools. **Objectives:** This study aimed to compare vocal fold vibratory characteristics, acoustic voice parameters, and perceptual voice quality in male chronic smokers and non-smokers with voice-related symptoms to detect smoking-induced voice abnormalities. **Materials and Methods:** A comparative cross-sectional study was conducted among 60 male patients (30 chronic smokers and 30 non-smokers), aged 18–60 years. All participants underwent videostroboscopic examination, acoustic voice analysis, and perceptual evaluation using the GRBAS scale. **Result:** Chronic smokers exhibited significantly higher rates of vocal fold asymmetry (43.3% vs. 13.3%; p = 0.02) and bilaterally reduced mucosal wave activity (20% vs. 0%; p = 0.033) than non-smokers. The fundamental frequency was significantly higher in smokers (203.13 ± 48.44 Hz) than in non-smokers (164.22 ± 44.36 Hz; p = 0.002). Shimmer values were higher in smokers and approached statistical significance (1.74 ± 2.46 dB vs. 0.79 ± 0.75 dB; p = 0.052). Although jitter and noise-to-harmonic ratio were elevated in smokers, the differences were not significant. Perceptual evaluation revealed significantly greater severity of hoarseness (p = 0.001) and roughness (p = 0.029) among smokers, while differences in breathiness, asthenicity, and strain were not significant. **Conclusion:** Chronic smoking is associated with significant impairment of vocal fold function and voice quality. The combined use of videostroboscopy, acoustic parameters, and the GRBAS scale enables the early detection and comprehensive evaluation of smoking-related voice changes.

INTRODUCTION

Healthy voice production depends on the integrity and proper functioning of laryngeal structures. Anatomical changes, particularly those resulting from harmful exposures such as smoking, can significantly impair voice quality. Chronic smoking is strongly associated with various vocal cord pathologies, including laryngitis, Reinke's oedema, and leucoplakia.[1] The term "smoker's larynx" describes the specific morphological and functional changes induced by smoking, characterised by chronic inflammation and alterations in voice quality. These changes often manifest as deviations in fundamental frequency and other acoustic features.[2]

Smoking remains a major global health concern, with over 1 billion smokers worldwide and millions of annual deaths, disproportionately affecting men. It is a well-established risk factor for both vocal disorders and chronic respiratory diseases, such as asthma. Smoking cessation improves voice health and reduces the risk of associated diseases. Studies indicate that smokers are at a significantly higher risk of vocal fatigue and voice impairment compared to non-smokers.[3]

Voice disorders, such as vocal lesions causing hoarseness, profoundly impact emotional well-being and work performance. Vocal polyps are the most common lesions observed, with symptoms including hoarseness, cough, foreign body sensation, and throat pain.[4,5] Early and accurate diagnosis is essential, and

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videostroboscopy plays a critical role in this process. It is widely used in clinical settings to evaluate glottal closure and mucosal pliability during phonation. Rigid telescopic laryngoscopy combined with a stroboscope is routinely employed by surgeons to diagnose vocal fold pathologies and assess the vibratory function of the glottis.[5]

Objective voice assessment is significantly strengthened by the use of quantitative acoustic measures, which are effective in distinguishing normal from pathological laryngeal function. These noninvasive tools play a crucial role in evaluating various voice disorders, including laryngitis and vocal cord paralysis. Specifically, acoustic parameters such as shimmer and jitter have proven useful in differentiating laryngeal pathologies and assessing the severity of dysphonia. The integration of multiple acoustic parameters has been shown to enhance the diagnostic accuracy of evaluating voice quality deviations.[6,7]

Quantitative tools, such as acoustic analysis and electroglottography, are indispensable for objective voice evaluation. Acoustic analysis offers precise and measurable insights into vocal function, thus improving diagnostic precision. When used alongside endoscopic techniques, such as videostroboscopy, these methods provide a comprehensive overview of vocal health.[8] In addition, the GRBAS scale serves as a reliable, valid, and noninvasive perceptual tool for assessing voice disorders. Its visual analogue format allows for greater sensitivity to subtle vocal changes when compared to traditional perceptual rating scales.[9] Furthermore, video stroboscope enables detailed visualization of vocal fold motion and can detect abnormalities that may be overlooked during standard rigid video laryngoscopy or fibre-optic laryngo-pharyngoscopy, making it a valuable adjunct in voice disorder assessment.[10]

The significant impact of smoking on voice health, the attributable risks involved, and the importance of early diagnosis and voice evaluation for effective management are highlighted. This study emphasises the role of vocal hygiene and employs perceptual, acoustic, and endoscopic methods to comprehensively assess voice production.

Objectives

To compare vocal fold movements (using a video stroboscope, acoustic voice parameters) and perceptual voice quality (using the GRBAS scale) between male smokers and non-smokers with voice-related symptoms and to evaluate the diagnostic utility of these measures in identifying smoking-induced voice changes.

MATERIALS AND METHODS

This prospective comparative cross-sectional study was conducted among 60 male patients with voice-related symptoms in the Department of ENT and Head and Neck Surgery at a tertiary care hospital in South India, over nine months from January 2024 to September 2024. Informed consent was obtained from all patients, and the institutional ethics committee approved the study before its commencement.

Inclusion and exclusion criteria

The study included male patients aged 18–60 years who presented with voice-related symptoms. Patients were excluded if they were <18 or >60 years old, had a diagnosis of chronic laryngitis, suffered from significant medical illnesses such as chronic obstructive pulmonary disease or coronary artery disease.

Methods

Male patients were assigned to two groups (n = 30): chronic smokers and non-smokers (n = 30). Smokers were defined as individuals who smoked at least two cigarettes per day for a minimum duration of one year. All patients underwent voice evaluation using a video stroboscope, acoustic voice analysis, and the GRBAS scale.

Videostroboscopy was performed to assess the vibratory patterns of the vocal folds. After topical anaesthesia with 10% lignocaine spray, the larynx was visualised using a 4 mm rigid Hopkins rod endoscope for initial video laryngoscopy. This was followed by video stroboscopic examination using an 8 mm, 70° rigid Hopkins rod telescope. During the procedure, the patients were asked to phonate sustained vowels (e.g. long “eee”) while the stroboscopic light was activated in sync with the patient's fundamental frequency using a Karl Storz stroboscope. Images were captured using a video capture card and analysed. The stroboscopic parameters evaluated included symmetry, mucosal wave, glottis closure, and periodicity of the vocal folds.

Acoustic voice analysis was conducted using the PHONOLAB software version 03.02.08 (ECLERIS). Voice samples were recorded in a soundproof room using a low-impedance commercial microphone positioned at a standardised distance of 30 cm from the patient’s mouth. The patients were instructed to produce sustained vowels (/a/ and /i/) and read a phonetically balanced passage at a comfortable pitch and loudness. Each vowel was sustained for 15–20 s, and high-quality continuous segments were selected for analysis. The parameters analysed included the fundamental frequency, jitter, shimmer, and noise-to-harmonics ratio.

The GRBAS scale was used for the perceptual auditory evaluation of voice quality and dysphonia. This scale includes five parameters: grade (G), roughness (R), breathiness (B), asthenia (A), and strain (S), each rated on a 4-point scale (0 = normal, 1 = slight, 2 = moderate, 3 = severe). GRBAS scoring was performed by a trained evaluator, and the results were documented in the format GxRxBxAxSx (e.g. G2R1B2A2S1).

Statistical Analysis

The collected data were analysed using IBM SPSS Statistics software, version 23.0. Descriptive

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statistics, including frequency and percentage, were used to summarise categorical variables, whereas continuous variables were expressed as mean and standard deviation (SD). An unpaired t-test was used to compare continuous variables between the two independent groups. The chi-square test was used to assess associations between categorical variables; however, Fisher's exact test was employed when the expected frequency in any cell of a 2×2 table was <5, and $p < 0.05$ was considered significant for all analyses.

RESULTS

Among the 60 patients included in the study, 80% were between 21 and 50 years of age, 5% were below 20 years of age, and the remaining were above 50 years of age. The age of the patients ranged from 19 to 58 years. The mean age of chronic smokers was 40.07 ± 10.32 years, while that of non-smokers was 37.81 ± 13.78 years; the difference was not significant ($p = 0.480$).

Reinke's oedema was more prevalent in chronic smokers (17%) than in non-smokers (7%), and vocal fold keratosis (13% vs. 7%), vocal fold cysts (7% vs. 3%), vocal fold paralysis (7% vs. 0%), and inter arytenoid granuloma (3% vs. 0%) were also more common among smokers. In contrast, non-smokers exhibited a higher incidence of laryngitis (27% vs. 10%) and vocal fold nodules (17% vs. 13%). Conditions such as sulcus vocalis were equally observed in both groups (10% each), while phonatory gap (10% vs. 7%) and spasmodic dysphonia (10% vs. 7%) were slightly more common in non-smokers than in smokers. Additionally, vocal fold papilloma (3%) and puberphonia (3%) were observed exclusively in non-smokers. [Table 1]

Table 1: Comparison of laryngeal diagnoses

Laryngeal diagnosis	Chronic smokers, N(%)	Non-smokers, N(%)
Reinke's oedema	5(17%)	2(7%)
Vocal nodules	4(13%)	5(17%)
Vocal fold keratosis	4(13%)	2(7%)
Sulcus vocalis	3(10%)	3(10%)
Laryngitis	3(10%)	8(27%)
Vocal fold cyst	2(7%)	1(3%)
Vocal fold paralysis	2(7%)	0
Phonatory gap	2(7%)	3(10%)
Spasmodic dysphonia	2(7%)	3(10%)
Vocal fold polyp	2(7%)	1(3%)
Interarytenoid granuloma	1(3%)	0
Vocal fold papilloma	0	1(3%)
Puberphonia	0	1(3%)

Footnotes: Percentages may not sum to exactly 100% because of rounding.

Chronic smokers demonstrated a significantly higher incidence of absent vocal fold symmetry (43.3%) than non-smokers (13.3%) ($p = 0.02$). Periodicity was preserved in both groups, with 96.7% of smokers and 100% of non-smokers exhibiting normal periodicity ($p = 1.00$). Although the overall glottic closure patterns did not differ significantly between the groups ($p = 0.338$), abnormal closure patterns, such as incomplete (6.7%) and irregular closure (3.3%), were observed exclusively among smokers. Complete glottic closure was more frequent in non-smokers (80%) than in smokers (56.7%). Regarding the mucosal wave, bilaterally small wave motion was identified in 20% of smokers and was absent among non-smokers ($p = 0.033$), whereas normal mucosal wave motion was significantly more common in non-smokers (93.3%) than in smokers (60%) ($p < 0.05$) (Table 2).

Table 2: Comparison of laryngeal stroboscopic parameters

		Chronic smokers	Non-smokers	P-value
Symmetry	Absent	13 (43.3%)	4 (13.3%)	0.02
	Present	17 (56.7%)	26 (86.7%)	
Periodicity	Absent	1 (3.3%)	0	1
	Present	29 (96.7%)	30 (100%)	
Glottis Closure	Anterior gap	1 (3.3%)	1 (3.3%)	0.338
	Hourglass	1 (3.3%)	2 (6.7%)	
	Incomplete	2 (6.7%)	0	
	Irregular	1 (3.3%)	0	
	Posterior gap	3 (10%)	1 (3.3%)	
	Spindle gap	5 (16.7%)	2 (6.7%)	
	Complete	17 (56.7%)	24 (80%)	
Mucosal Wave	Bilaterally small	6 (20%)	0	0.033
	Left small wave	4 (13.3%)	1 (3.3%)	
	Right small wave	2 (6.7%)	1 (3.3%)	
	Normal	18 (60%)	28 (93.3%)	

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Chronic smokers exhibited a significantly higher mean fundamental frequency (203.13 ± 48.44 Hz) than non-smokers (164.22 ± 44.36 Hz; p = 0.002). The standard deviation of the fundamental frequency was marginally higher in smokers (5.59 ± 2.28) than in non-smokers (4.49 ± 2.59) (p = 0.084).

Jitter, an indicator of frequency instability, was lower among smokers (102.81 ± 33.75) than among non-smokers (154.30 ± 208.19) (p = 0.191), likely due to the high variability observed in the non-smoking group. Shimmer, reflecting amplitude perturbation, was higher in smokers (1.74 ± 2.46 dB) than in non-smokers (0.79 ± 0.75 dB) (p = 0.052). The noise-to-harmonic ratio (NHR), a measure of voice signal degradation, was also elevated in smokers (0.17 ± 0.15) relative to non-smokers (0.13 ± 0.08), although the difference was not significant (p = 0.178) (Table 3).

Table 3: Comparison of acoustic voice parameters

	Chronic smokers	Non-smokers	P-value
Fundamental frequency	203.13 ± 48.44	164.22 ± 44.36	0.002
S.D of the fundamental frequency	5.59 ± 2.28	4.49 ± 2.59	0.084
Jitter (ms)	102.81 ± 33.75	154.3 ± 208.19	0.191
Shimmer (dB)	1.74 ± 2.46	0.79 ± 0.75	0.052
Noise to the harmonic ratio	0.17 ± 0.15	0.13 ± 0.08	0.178

Only 16.7% of chronic smokers had grade 0, compared to 60% of non-smokers. In contrast, 73.3% of smokers had grade 1 hoarseness versus 40% of non-smokers, and 10% of smokers had grade 2 hoarseness, while none of the non-smokers did. Overall, chronic smokers demonstrated significantly higher grades of hoarseness than non-smokers (p = 0.001). Regarding vocal roughness, grade 0 (absence of roughness) was observed in 40% of smokers and 73.3% of non-smokers.

Moderate roughness (Grade 2) was observed in 13.3% of smokers and 3.3% of non-smokers, indicating a significantly higher occurrence of roughness among smokers (p = 0.029). Breathiness did not differ significantly between the groups (p = 0.838), with the absence of breathiness noted in 83.3% of smokers and 80% of non-smokers. Asthenicity was more common among smokers (43.3% with grade 1 or 2) than among non-smokers (20%), although this difference was not significant (p = 0.123). Similarly, moderate strain (grade 2) was observed in 10% of smokers and none of the non-smokers (p = 0.065) (Table 4).

Table 4: Comparison of GRBAS parameters

Grade		Chronic smokers	Non-smokers	P-value
Grade	0	5(16.7%)	18(60%)	0.001
	1	22(73.3%)	12(40%)	
	2	3(10%)	0	
Roughness	0	12((40%)	22(73.3%)	0.029
	1	14(46.7%)	7(23.3%)	
	2	4(13.3%)	1(3.3)	
Breathiness	0	25(83.3%)	24(80%)	0.838
	1	4(13.3%)	4(13.3%)	
	2	1(3.3%)	2(6.7%)	
Asthenicity	0	17(56.7%)	24(80%)	0.123
	1	12(40%)	6(20%)	
	2	1(3.3%)	0	
Strain	0	9(30%)	16(53.3)	0.065
	1	18(60%)	14(46.7%)	
	2	3(10%)	0	

DISCUSSION

In our study, Reinke's oedema was notably more prevalent in chronic smokers (17%) than in non-smokers (7%), along with higher incidences of vocal fold keratosis (13% vs. 7%), vocal fold cysts (7% vs. 3%), and vocal fold paralysis (7% vs. 0%). Vocal fold polyps were observed in 7% (2 cases) of smokers and 3% (1 case) of non-smokers. Similarly, Banjara et al. reported oedema in 84% of smokers versus 54% of non-smokers, abnormal vocal fold edges in 93.9% vs. 68.1%, abnormal vocal fold texture in 98% vs. 62%, and erythema in 76% vs. 24%, all significant ($p<0.05$).2 Effat and Milad found non-smokers had more vocal fold polyps, including bilateral cases, while smokers had significantly larger polyps ($p<0.01$).[11]

Pinar et al. observed increased vocal fold erythema, asymmetry, amplitude, and periodicity abnormalities in smokers using a video stroboscope ($p<0.05$).[12] Similarly, Awan et al. reported significantly higher rates of oedema ($\chi^2 = 4.46$, $p<0.05$) and abnormal phase symmetry ($\chi^2 = 5.51$, $p<0.05$) in smokers, with erythema more prevalent among smokers ($\chi^2 = 2.10$, $p<0.10$), suggesting vascular or inflammatory changes.[13] These studies show that chronic smoking significantly increases structural and inflammatory vocal fold abnormalities.

Our study also revealed that chronic smokers had significantly higher rates of absent vocal fold symmetry (43.3%) than non-smokers (13.3%)

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(p=0.02). Bilaterally small mucosal waves were present in 20% of smokers but were absent in non-smokers (p=0.033). Normal mucosal wave motion was more frequent in non-smokers than in smokers (93.3% vs. 60%) (p<0.05). Similarly, Banjara et al. found that abnormal mucosal cover (38.3% vs. 15.2%), phase symmetry (14.9% vs. 2%), and pliability/stiffness (41.7% vs. 18.8%) were significantly higher in smokers (p<0.05). These findings indicate that smoking impairs vocal fold symmetry and mucosal wave function.[2]

In our study, glottis closure did not show any significant difference. However, incomplete (6.7%) and irregular closures (3.3%) were only observed in smokers, whereas complete closure was more common in non-smokers (80% vs. 56.7%). Banjara et al. similarly found no significant differences between smokers and non-smokers regarding vocal fold mobility, glottal gap size, closure pattern, ventricular fold compression, or organized lesions (p>0.05).[2]

In our study, regarding acoustic measures, smokers had lower jitter (102.81 ± 33.75) than non-smokers (154.30 ± 208.19, p=0.191) but higher shimmer (1.74 ± 2.46 dB vs. 0.79 ± 0.75 dB, p=0.052). This variability may have diluted potential group differences, highlighting the need for larger sample sizes or stratified analyses to detect subtle changes in acoustic parameters. In contrast, Banjara et al. and Chai et al. reported significantly elevated jitter and shimmer in smokers (p<0.05), reflecting vocal instability.[2,14]

In our study, chronic smokers exhibited a significantly higher fundamental frequency (203.13 ± 48.44 Hz) than non-smokers (164.22 ± 44.36 Hz, p=0.002). The NHR was also higher in smokers (0.17 ± 0.15) than in non-smokers (0.13 ± 0.08). In contrast, Banjara et al. found smokers had a lower fundamental frequency (131.39 ± 14.54 Hz vs. 139.33 ± 17.05 Hz, p<0.05) and no significant HNR difference.2 Similarly, Pinto et al. observed lower fundamental frequency in male smokers with higher jitter, shimmer, and NHR (p<0.05).[15]

Supporting our findings, Mohammadzadeh and Mousavi reported higher fundamental frequency and NHR in smokers of both genders (p<0.008), with reduced fundamental frequency variation indicating decreased vocal variability.[16] In contrast, Verma et al. noted decreased fundamental frequency and improved harmonics-to-noise ratio after smoking cessation, alongside significant reductions in jitter and shimmer.[17] The inconsistency in fundamental frequency findings warrants further research to clarify this issue.

Our study found significantly higher grades of hoarseness (p=0.001) and roughness (p=0.029) in smokers. Grade 0 hoarseness was observed in only 16.7% of smokers versus 60% of non-smokers, with moderate roughness (grade 2) more prevalent among smokers (13.3% vs. 3.3%). No significant differences were observed in breathiness, asthenicity, or strain. These findings align with Moya et al., who reported greater vocal dysfunction measured by the GRBAS scale (p<0.0001 to 0.015) in patients with genetic mutations.[18] Karnell et al. validated the GRBAS scale's reliability for dysphonia severity (r>0.80), showing strong concordance with CAPE-V, despite the weaker patient agreement.[19] Fujiki et al. found GRBAS scores correlated significantly with acoustic and aerodynamic parameters, reinforcing its utility in comprehensive voice evaluation.[20]

Our study highlighted that chronic smoking is strongly associated with structural, functional, and perceptual vocal fold impairments, including increased oedema, asymmetry, hoarseness, and acoustic instability. While the fundamental frequency results may vary, the GRBAS scale remains a reliable tool for assessing smoking-related dysphonia and supporting thorough voice assessment.

Limitations

The study was limited by its small sample size and male-only population, which may restrict its generalisability to the broader population, including females. The cross-sectional design precluded causal inference between smoking and voice changes. Additionally, we did not employ multivariate analysis to assess the predictive value of individual parameters. Longitudinal follow-up studies with larger and more diverse samples are recommended for future research.

CONCLUSION

Videostroboscopy combined with acoustic voice analysis offers a clinically feasible and effective method for the comprehensive evaluation of vocal fold function. This approach allows for a detailed assessment of anatomical and phonatory features, providing objective data on the impact of chronic smoking. The GRBAS scale adds subjective perceptual evaluation, thereby enhancing the overall assessment. Significant differences between smokers and non-smokers were noted for various stroboscopic and acoustic parameters. The unexpectedly higher fundamental frequency in smokers warrants further investigation in larger population-based studies. These findings highlight the utility of the video stroboscope as a valuable tool for assessing smoking-related vocal fold changes.

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