The Role of Gastroesophageal Reflux and Helicobacter pylori in the Pathogenesis of Benign Laryngeal Lesions

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Background and study aim: Benign laryngeal lesions are prevalent conditions that can significantly impact voice quality and vocal function. Although their etiology is multifactorial, gastroesophageal reflux disease (GERD) and Helicobacter pylori (H. pylori) infection have emerged as potential risk factors contributing to the development and progression of these lesions. **Patients and Methods:** This case-control, prospective study included 100 participants, with 50 patients diagnosed with benign laryngeal lesions (case group) and 50 age- and sex-matched controls. All patients underwent a comprehensive evaluation, including laryngoscopy, H. pylori stool antigen testing, and assessment of GERD symptoms using the Reflux Symptom Index (RSI). Laryngeal biopsies from cases were examined histopathologically to confirm H. pylori's presence. **Results:** GERD was significantly more prevalent in the case group (66%) compared to controls (20%) ($P < 0.001$), and H. pylori infection was detected in 34% of cases versus 10% of controls ($P = 0.002$). The coexistence of GERD and H. pylori was noted in 30% of cases compared to 8% of controls ($P = 0.005$). Laryngeal findings, such as vocal cord edema and arytenoid edema, were significantly more common in patients with both GERD and H. pylori (93% and 100%, respectively) compared to those with GERD or H. pylori alone. The presence of vocal nodules was not significantly associated with H. pylori infection alone ($P = 0.600$).**Conclusion:** The study demonstrates a strong association between GERD, H. pylori infection, and the development of benign laryngeal lesions. Combining these disorders appears to worsen laryngeal mucosal injury, worsening symptoms and lesions.

INTRODUCTION

The benign lesions of the larynx such as nodules and polyps of the vocal cords, Reinke's edema, and chronic laryngitis often impair voice production and airway function [1]. These lesions are a result of inflammation, or irritation, of the laryngeal mucosa by vocal abuse or other forms of abuse such as smoking and other irritants [2]. However, there is increasing evidence that suggests that GERD and infection with H pylori may play a role in their pathogenesis as well [3].

Gastro-esophageal reflux (GERD) is Characterized by the reflux of stomach acid and digestive enzymes, especially pepsin, into the upper neuro gastrointestinal tract. This reflux is associated with damage and inflammation of the mucosa [4]. One of the subtypes of GERD is laryngopharyngeal reflux (LPR), which is known to cause chronic irritation and edema of the larynx and voice disorders. Patients of LPR most often seek treatment for changes in their voice, cough, throat discomfort, difficulty swallowing, and in some cases even a cough that is chronic in nature [5].

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Have noticed that the laryngeal disease induced by GERD is associated with ailments such as vocal cord edema, hyperthyroid erythematous, and posterior hypertrophy of the larynx, which implies that acid exposure has a direct effect on the laryngeal tissues [6.[

Apart from GERD, H. pylori has been found in the tonsils, nasal mucosa, and even laryngeal tissue. The possible role of H pylori in laryngeal disease is largely unknown, although some suggest that it may be directly through mucosal colonization or indirectly through exacerbating GERD [3]. Several studies have reported the coexisting nature of H pylori with GERD, and it's suggested that H pylori-induced gastritis changes the pattern of gastric acid secretions, which may be more severe in reflux and its consequences on the larynx. However, there is controversy on the extent that H pylori infection may worsen GERD and whether it presents a protective effect through lowering acid output [7-9 .[

The goal of this study was to assess the frequency of diagnosis of GERD and H. pylori infection in patients with otherwise benign laryngeal lesions and their possible contribution to disease pathogenesis. The analysis sought to establish the impact of GERD compared to healthy controls through symptom assessment, GI manifestations, and laryngoscopic exam results while clarifying the role of H pylori in laryngeal pathology. These correlations could enhance the precision of diagnosis and treatment of patients with chronic laryngeal diseases.

PATIENTS AND METHODS

Study Design

This case-control, prospective study was conducted at Qena University Hospital, Assiut University Hospital, involving patients diagnosed with benign laryngeal lesions who were scheduled for endoscopic surgical excision and histopathological evaluation.

Inclusion Criteria

- Age: 18 to 70 years.
- Both male and female patients.
- Patients with benign laryngeal lesions confirmed by clinical evaluation and scheduled for surgical excision.
- All participants consented to undergo Helicobacter pylori (H. pylori) stool antigen testing and histopathological evaluation for H. pylori detection in excised laryngeal tissue.

Exclusion Criteria

- Patients with a negative H. pylori stool antigen test.
- Patients previously treated for H. pylori infection or those who had received chemotherapy.
- Patients with systemic diseases contraindicating surgery or with other conditions affecting vocal cord function (e.g., neurological disorders.*(*
- Patients with malignancy or inflammatory lesions of the larynx.
- Patients refusing participation or deemed uncooperative.

Patient Assessment

All participants of the study had a complete ENT and gastroenterology workup done. The history taken as a part of the ENT assessment was directed toward upper airway symptoms with particular reference to changes in voice quality, swallowing issues, and other associated symptoms of the upper airway. The physical examination included inspection of the nose and the paranasal sinuses, ear examination, indirect laryngoscopy, and then either flexible or rigid laryngoscopy to examine the laryngeal mucosa and the vocal folds. Features of the lesions such as size, site, and laryngeal edema or erythema were noted .

A gastroenterology consultation was done to rule out gastrointestinal components of GERD. All participants filled in the Reflux Symptom Index (RSI) questionnaire to assess their symptoms including but not limited to hoarseness, chronic cough, throat clearing, heartburn, and the globus sensation. Patients who scored above or equal to 13 on the RSI scale were regarded as symptomatic for LPR-associated GERD.

Diagnosis of GERD

GERD was diagnosed based on clinical presentation, and the RSI score, and further confirmed through esophagogastroduodenoscopy (EGD) in selected cases, as per the American College of Gastroenterology guidelines [10]. Additional diagnostic tests, including 24-hour pH

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monitoring, were performed when indicated to evaluate reflux severity.

H. pylori Detection

H. pylori infection was initially screened using the H. pylori stool antigen test, a non-invasive and reliable method for detecting active infection [11].

Statistical Analysis

Data were analyzed using SPSS version 20.0 for Windows (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as mean ± standard deviation and categorical variables as numbers and percentages. The chi-square test or Fisher's exact test was used to compare categorical data, while independent sample t-tests were applied for continuous variables. A P-value < 0.05 was considered statistically significant.

RESULTS

This case-control study was conducted at the Otorhinolaryngology Department, Qena University Hospital, and Paediatrics Department, Assiut University Hospital from November 2021 to October 2023 to assess the role of h-pylori in the pathogenesis of the benign laryngeal lesions. It included 50 cases with benign laryngeal lesions and 50 age and sex-matched control .s

As shown in Table 1 both groups were matched for age (p = 0.895) and sex (p = 0.688). For the laryngeal symptoms, cases had significantly (p = 0.016 and < 0.001) higher rates of hoarseness of voice and habitual throat clearance (56% and 64%) than control (32% and 24%). On the other hand, cases had significantly (p < 0.001) lower rates of hemoptysis (8%) in comparison with control (44%). All other symptoms showed insignificant differences (p > 0.05). Regarding the laryngeal findings by laryngoscopy, cases had significantly (p < 0.001) higher rates of vocal cord edema, arytenoid edema, and red uvula (72%, 90%, and 78%) compared with control (28%, 42% and 56%). Contrarily, cases had insignificantly (p = 0.142) higher rates of nodule (72%) than control (58%.(

As illustrated in Table 2, respecting the GIT symptoms, cases had significantly (p < 0.001, 0.002, 0.005, and <0.001) higher rates of epigastric pain, weight loss, dyspepsia, and hiccough (86%, 36%, 68%, and 54%) compared with control (50%, 10%, 40% and 8%). In contrast, cases had insignificantly (p = 0.114) higher rates of bloating (52%) than control (38%). Also, cases had insignificantly (p=0.412) lower rates of loss of appetite (26%) in comparison with control (30%). As well, both groups were matched for both Melena and haematemesis (6%, p=1.000.(

The prevalence of GERD was significantly higher in the case group (66%) compared to the control group (20%) (P < 0.001). Similarly, H. pylori infection was more common among patients with laryngeal lesions (34%) than in controls (10%) (P = 0.002). The coexistence of GERD and H. pylori was observed in 30% of cases versus 8% of controls (P = 0.005). GERD without H. pylori was found in 36% of cases compared to 12% of controls (P = 0.004), while H. pylori infection without GERD showed no significant difference between groups (P = 0.600) as shown in (Figure 1.(

Table (3) shows the prevalence of laryngeal lesions varied significantly based on GERD and H. pylori status. Vocal cord edema was most common in the GERD-positive, H. pylori-positive group (93%), followed by GERD-positive patients without H. pylori (78%) and those with H. pylori alone (50%). Arytenoid edema was observed in 100% of patients with both GERD and H. pylori, while it was slightly lower in GERD-positive, H. pylori-negative patients (89%). The red uvula was prevalent in 87% of those with both GERD and H. pylori, with a similar pattern observed in GERD positive, and H. pylori negative (83%). Nodule formation was higher in patients with GERD and H. pylori (73%) compared to other groups, indicating a potential synergistic effect.

Table (1). Sociodemographic Characteristics, Laryngeal Symptom Comparisons, and Laryngeal Findings by Laryngoscopy of the Studied Groups

Characteristics	Case (n = 50)	Control (n = 50)	P-value
Age/years (mean ± SD)	34.24 ± 17.1	34.84 ± 16.7	0.859
Sex			
• Female	24 (48%)	22 (44%)	0.448
• Male	26 (52%)	28 (56%)	
Laryngeal Symptoms			
• Hoarseness of Voice	28 (56%)	16 (32%)	0.016
• Dysphagia	20 (40%)	12 (24%)	0.086
• Stridor	13 (26%)	12 (24%)	0.817
• Choking	10 (20%)	10 (20%)	1.000
• Pain (Local/Referred)	14 (28%)	7 (14%)	0.070
• Habitual Throat Clearance	32 (64%)	12 (24%)	**<0.001***
• Aspiration	5 (10%)	3 (6%)	0.461
Laryngeal Findings by Laryngoscopy			
• Vocal Cord Oedema	36 (72%)	14 (28%)	**<0.001***
• Arytenoid Oedema	45 (90%)	21 (42%)	**<0.001***
• Red Uvula	39 (78%)	28 (56%)	**0.019***
• Nodule	36 (72%)	29 (58%)	0.142

Data are represented as mean ± stander deviation, or number (percentage).
∗ $p \leq 0.05$ is significant.

Table (2). GIT Manifestation Comparisons of the Studied Groups

GIT Manifestation	Case (n = 50)	Control (n = 50)	P-value
Epigastric Pain	43 (86%)	25 (50%)	**<0.001***
Weight Loss	18 (36%)	5 (10%)	**0.002***
Bloating	26 (52%)	19 (38%)	0.118
Dyspepsia	34 (68%)	20 (40%)	**0.005***

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GIT Manifestation	Case (n = 50)	Control (n = 50)	P-value
Loss of Appetite	13 (26%)	15 (30%)	0.412
Melena	6 (12%)	6 (12%)	1.000
Hematemesis	6 (12%)	6 (12%)	1.000
Hiccough	27 (54%)	4 (8%)	**<0.001***

Data are represented as mean ± stander deviation, or number (percentage).
∗ $p \leq 0.05$ is significant.

Table (3). Laryngeal Lesions Based on GERD and *H. pylori* Status

Laryngeal Lesions	GERD Positive (n = 33)	H. pylori Presence (n=17)	GERD Positive, H. pylori Positive (n = 15)	GERD Positive, H. pylori Negative (n = 18)	GERD Negative, H. pylori Positive (n = 2)	GERD Negative, H. pylori Negative (n = 15)
Vocal Cord Oedema	28 (85%)	13 (76%)	14 (93%)	14 (78%)	1 (50%)	7 (47%)
Arytenoid Oedema	31 (94%)	14 (82%)	15 (100%)	16 (89%)	1 (50%)	13 (87%)
Red Uvula	27 (82%)	12 (71%)	13 (87%)	15 (83%)	1 (50%)	10 (67%)
Nodule	23 (70%)	11 (65%)	11 (73%)	12 (67%)	0 (0%)	13 (87%)

Data is represented as a number (percentage).

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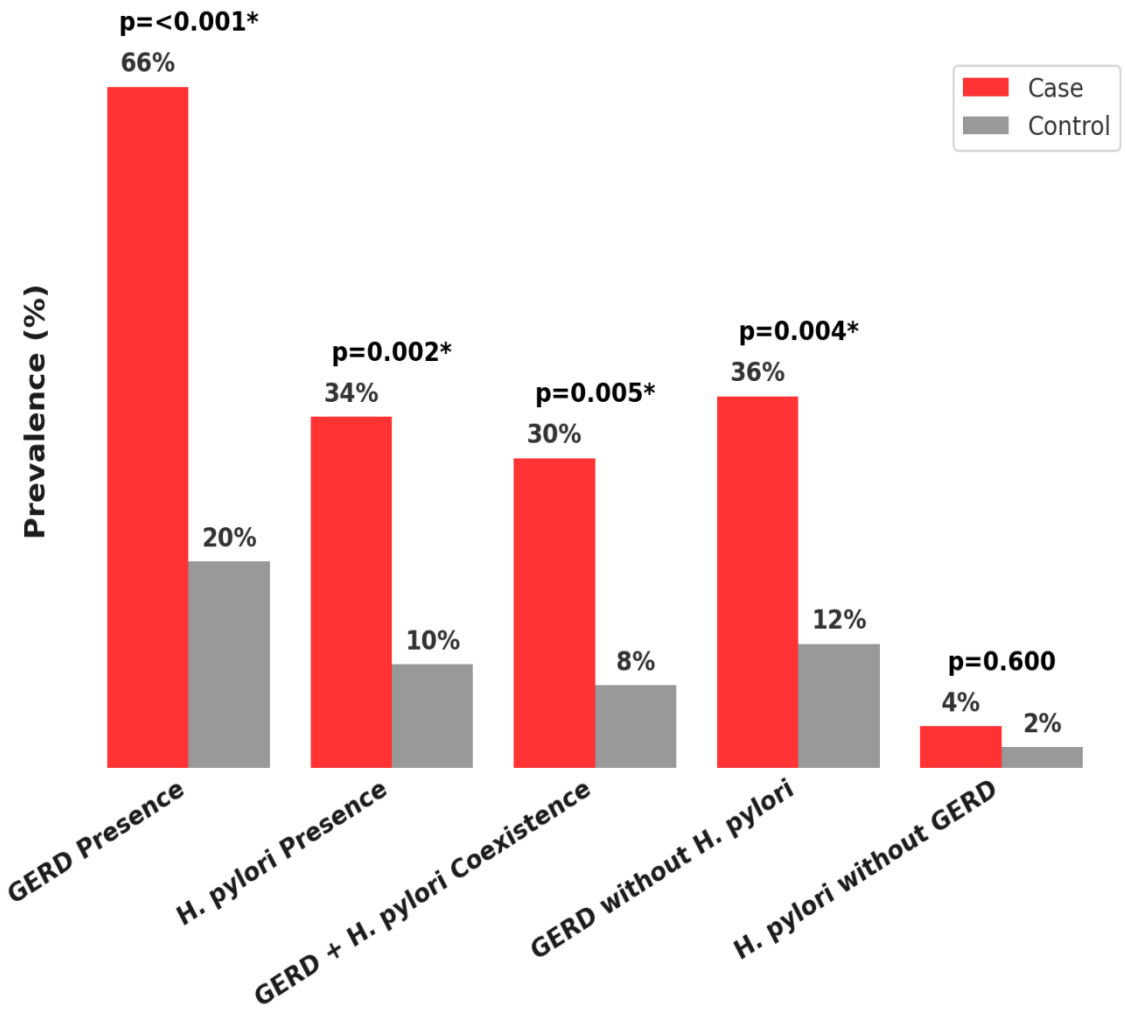

Figure (1). Prevalence of GERD and H. pylori Infection in the Studied Groups

DISCUSSION

GERD and H. pylori have been implicated in the pathogenesis of benign laryngeal lesions, affecting vocal fold integrity and mucosal inflammation. This study evaluates their correlation with laryngeal pathologies, analyzing data from affected patients and controls.

The mean age of the case group (34.24 ± 17.1 years) and control group (34.84 ± 16.7 years) showed no significant difference (p = 0.859), suggesting that age is not a major determinant in laryngeal lesion development. In addition, sex distribution was comparable between groups (p = 0.448). These findings are consistent with a study by Liu et al. (2024) [3], which found no age or sex predisposition for GERD-associated laryngeal symptoms .

Among laryngeal symptoms in our study, hoarseness was significantly more frequent in GERD-positive patients (56%) than in controls (32%, p = 0.016). This aligns with findings from Yılmaz et al. (2014) [12], who demonstrated that GERD contributes to vocal fold inflammation and subsequent dysphonia. The meta-analysis by Liu et al. (2024) [3], supported this association, reporting a higher risk of hoarseness in H. pylori-positive individuals with GERD (RR = 1.35; 95% CI, 1.12–1.63). However, Ercan et al. (2007) [13] and Islam et al. (2013) [14] did not find a significant correlation, indicating that factors such as smoking and vocal strain may also contribute.

In our study, habitual throat clearing was significantly more common in GERD cases (64%) versus controls (24%, $p < 0.001$). These results are in agreement with Schwizer & Fox (2004) [15] which identified excessive mucus production and persistent throat irritation as GERD-associated symptoms. In agreement with Joseph et al. (2022), [16] and Rouev et al. (2005) [17] reporting similar findings. However, Rubin et al. (2002) [18] found no significant relationship between GERD and throat clearing, suggesting that postnasal drip may be an alternative cause.

In the current study, laryngoscopic examination revealed a significantly higher prevalence of vocal cord edema in GERD patients (72%) compared to controls (28%, $p < 0.001$). These findings correspond with those of Kim et al. (2022) [19], who reported a 70% prevalence of vocal cord edema in individuals with positive GERD symptoms. This relationship was also confirmed by Zhang et al. (2022) [2] and Asyari et al. (2022) [7] observed higher frequencies of laryngeal edema in H. pylori-positive patients. On the other hand, Cekin et al. (2012) [20] and Oridate et al. (2006) [21] did not find a difference thus suggesting heterogeneity in the criteria employed for the diagnosis of laryngeal edema.

In our analysis, posterior arytenoid edema was noted to be present significantly more in controls with GERD (90%) than in patients without GERD (42%, $p < 0.001$), which confirms the previously established connection between the inflammation of the posterior larynx and exposure to acid. These findings reflect those observed by Mahmoud et al. (2020) [8]. As well as, Liu et al. (2024) [3] found a higher proportion of posterior laryngeal edema was found in patients with H. pylori and GERD as compared to those without the infection (RR = 1.19; 95% CI, 1.07–1.31). On the contrary, Kumral et al. (2019) [22] and Saruç et al. (2012) [23] did not observe any significant correlation which indicates that other contributing factors, such as nutrition and surrounding factors, might be involved.

The presence of a red uvula was more common in our patients with GERD as seen in 78% of the subjects in comparison to only 56% observed in the control group ($p= 0.019$). This indicates chronic inflammation due to acid reflux. Such findings are in accordance with Neenu et al. (2015) [24] who pointed out the role of H. pylori infection in uvula inflammation. This was also supported by Liu et al. (2024) [3] which pointed towards greater chances of having laryngeal erythema among individuals positive for H. pylori .

Compared to the control group, H. pylori infection was found in 34% of patients suffering from GERD and in 10% of those who did not have GERD ($p=0.002$) suggesting the positive contribution of H. pylori in intensifying the symptom of reflux. This supports Chen et al (2020) [9] who claimed the presence of H. pylori in 40% of chronic sufferers of laryngitis. The meta-analysis assessed and reported the pooled prevalence of the infection with H. pylori as 49% (95 % CI, 36 – 61) in patients with GERD which was in line with the result of this study [3 .[

Our results revealed the coexistence of GERD alongside H. pylori was observed in 30% of cases as opposed to only 8% in the control group ($p = 0.005$), indicating possible potentiating interaction between the two diseases. This result was also verified in the meta-analysis with H. pylori-positive patients having a relative risk (RR) of 1.35 (95% CI, 1.12–1.63) of developing symptoms of GERD [3]. Still other studies by Kumral et al. (2019) [22] and Saruç et al. (2012) [23] found no significant association while Pirzadeh et al. (2011) [25] suggested H. pylori could play a protective role through gastric acid secretion modulation.

In our result, laryngeal lesion distribution based on GERD and H. pylori status showed that vocal cord edema was more common in GERD-positive, H. pylori-positive cases (93%) compared to GERD-negative, H. pylori-positive cases (50%). Arytenoid edema was significantly more prevalent in GERD-positive, H. pylori-positive patients (100%) compared to GERD-negative, H. pylori-positive patients (50%). These findings align with Liu et al. (2024) [3], which demonstrated a higher risk of laryngeal inflammation in GERD-H. Pylori co-positive individuals. However, the variability in findings across studies suggests that while GERD and H.

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pylori may interact to exacerbate symptoms, additional risk factors must be considered.

CONCLUSION

The findings support the significant role of GERD in benign laryngeal lesion development, with H. pylori acting as a potential cofactor. The presence of GERD-associated symptoms, laryngoscopic findings, and H. pylori prevalence in affected individuals highlights the need for early diagnosis and targeted intervention. However, inconsistencies in H. pylori detection rates across studies suggest the necessity for standardized diagnostic protocols. Future research should focus on controlled longitudinal studies with molecular profiling of H. pylori strains to clarify their precise contribution to laryngeal pathology and guide treatment strategies.

Data availability statement

The datasets that were assessed in the present study are available from the corresponding author upon request.

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Ethical approval:

The study was conducted by the Declaration of Helsinki (7th revision, 2013) and was approved by the Medical Ethics Committee of the Faculty of Medicine, Assiut University. Written informed consent was obtained from all participants before inclusion. Declaration of Competing Interest

The authors declare that they have no conflict of interest.

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HIGHLIGHTS

Significant Association Between GERD and H. pylori in Benign Laryngeal Lesions

- The study found a 66% prevalence of GERD in patients with benign laryngeal lesions compared to only 20% in controls [P < 0.001].
- *H. pylori* infection was significantly more frequent in cases (34%) than in controls (10%) [P = 0.002].
- The coexistence of GERD and *H. pylori* was observed in 30% of cases vs. 8% of controls [P = 0.005], indicating a potential synergistic effect**.**

Laryngeal Mucosal Injury is More Severe in GERD and *H. pylori* Coexistence.

- Patients with both GERD and *H. pylori* exhibited severe laryngeal inflammation, with vocal cord edema (93%) and arytenoid edema (100%) occurring at significantly higher rates compared to those with either condition alone**.**
- Vocal nodules *were not significantly linked to H*. pylori infection alone [P = 0.600], suggesting that GERD may play a more central role in lesion formation**.**

Implications for Diagnosis and Treatment

- The study underscores the need for early diagnosis and combined management of GERD and *H. pylori* in patients with chronic laryngeal conditions**.**
- Standardized diagnostic protocols and targeted interventions could improve patient outcomes by reducing inflammation and symptom severity**.**

REFERENCES

1. Smiianov VA, Smiianov YV, Plakhtiienko IO. Clinical anatomy, physiology research methods of the larynx. Diagnosis and treatment of the main types of its pathology. 2023.
2. Zhang Q-Q, Xie M, Guo R-X, Liu X-H, Ma S-J, Li N, et al. The effects of Helicobacter pylori eradication therapy on salivary pepsin concentration in patients with laryngopharyngeal reflux. European *Archives of Oto-Rhino-Laryngology*. 2022;279(11):5289-97.
3. Liu S, Li C, Xu S, Guo Y, Luo H. Association between Helicobacter pylori and laryngopharyngeal reflux disease: a systematic review and meta-analysis. *Journal of Voice*. 2024.
4. Herdiana Y. Functional food about gastroesophageal reflux disease (GERD). *Nutrients*. 2023;15(16):3583.

5. Liu D, Qian T, Sun S, Jiang JJ. Laryngopharyngeal reflux and inflammatory responses in mucosal barrier dysfunction of the upper aerodigestive tract. *Journal of Inflammation Research*. 2021:1291-304.
6. Hránková V, Balner T, Gubová P, Staníková L, Zeleník K, Komínek P. Narrative review of the relationship between chronic cough and laryngopharyngeal reflux. *Frontiers in Medicine*. 2024;11:1348985.
7. Asyari A, Yerizel E, Putra AE, Firdawati F, Utami RA. Analysis of Helicobacter pylori in saliva of patients with laryngopharyngeal reflux and non-laryngopharyngeal reflux. *Open Access Macedonian Journal of Medical Sciences.* 2022;10(B):2433-6.
8. Mahmoud H, Kamel A, Hamed A, Shehata M, Askoura A, Omran A, et al. H. pylori and Pepsin as a Putative Risk Factors in Laryngeal Cancer Patients. Egyptian *Journal of Ear, Nose, Throat and Allied Sciences*. 2021;22(22):1-9.
9. Chen M, Fang Y, Cheng L, Wu H. Helicobacter pylori are associated with poor prognosis of laryngeal precancerous lesion. *Auris Nasus Larynx*. 2020;47(2):268-75.
10. Katz PO, Gerson LB, Vela MF. Guidelines for the diagnosis and management of gastroesophageal reflux disease. *Official journal of the American College of Gastroenterology| ACG*. 2013;108(3):308-28.
11. Chey WD, Leontiadis GI, Howden CW, Moss SF. ACG clinical guideline: treatment of Helicobacter pylori infection. *Official journal of the American College of Gastroenterology| ACG*. 2017;112(2):212-39.
12. Yılmaz T, Bajin MD, Günaydın RÖ, Özer S, Sözen T. Laryngopharyngeal reflux and Helicobacter pylori. *World Journal of Gastroenterology: WJG*. 2014;20(27):8964.
13. Ercan İ, Çakir BÖ, Uzel TÇ, Sakiz D, Karaca Ç, Turgut S. The role of gastric Helicobacter pylori infection in laryngopharyngeal reflux disease. *Otolaryngology—Head and Neck Surgery*. 2006;135(1):52-5.
14. Islam A, Oguz H, Yucel M, Koca G, Gonultas MA, Arslan N, et al. Does Helicobacter pylori exist in vocal fold pathologies and the interarytenoid region? Dysphagia. 2013;28:382-7.
15. Curcic J, Fox M, Kaufman E, Forras-Kaufman Z, Hebbard GS, Roy S, et al. Gastroesophageal junction: structure and function as assessed by using MR imaging. *Radiology*. 2010;257(1):115-24.
16. Joseph NA, Mathews SS, Anandan S, Veeraraghavan B, Bakthavatchalam YD, Pulimood AB. Lack of association of Helicobacter pylori in laryngeal pathologies. *Indian Journal of Otolaryngology and Head & Neck Surgery*. 2022:1-7.
17. Rouev P, Chakarski I, Doskov D, Dimov G, Staykova E. Laryngopharyngeal symptoms and gastroesophageal reflux disease. *Journal of Voice*. 2005;19(3):476-80.
18. Rubin J, Benjamin E, Prior A, Lavy J, Ratcliffe P. The prevalence of Helicobacter pylori infection in benign laryngeal disorders. *Journal of Voice*. 2002;16(1):87-91.
19. Kim YD, Shin CM, Jeong W-J, Kim YJ, Yoon H, Park YS, et al. Clinical implications of the gastroesophageal reflux disease questionnaire and reflux symptom index in patients with suspected laryngopharyngeal reflux symptoms. *Journal of Neurogastroenterology and Motility*. 2022;28(4):599.
20. Çekin E, Ozyurt M, Erkul E, Ergunay K, Cincik H, Kapucu B, et al. The association between Helicobacter pylori and laryngopharyngeal reflux in laryngeal pathologies. *Ear, Nose & Throat Journal*. 2012;91(3):E6-E9.
21. Oridate N, Takeda H, Yamamoto J, Asaka M, Mesuda Y, Nishizawa N, et al. Helicobacter pylori seropositivity predicts outcomes of acid suppression therapy for laryngopharyngeal reflux symptoms. *The Laryngoscope*. 2006;116(4):547-53.
22. Kumral TL, Gökden Y, Saltürk Z, Berkiten G, Yıldırım G, Ataç E, et al. The effect of gastric helicobacter pylori colonization on nasal functions. *Ear, Nose & Throat Journal*. 2019;98(6):346-50.
23. Saruç M, Aksoy EA, Vardereli E, Karaaslan M, Çiçek B, İnce Ü, et al. Risk factors for laryngopharyngeal reflux. European Archives of Oto-Rhino-Laryngology. 2012;269:1189-94.
24. Neenu AJ. Prevalence of Helicobacter pylori infection and influence of laryngopharyngeal reflux in patients with laryngeal pathologies: Christian Medical College, Vellore; 2015.
25. Pirzadeh A, Doustmohammadian N, Khoshbaten M, Doustmohammadion S. Is

there any association between Helicobacter Pylori infection and laryngeal carcinoma? *Asian Pacific Journal of Cancer Prevention*. 2011;12(4):897-900.

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