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TO STUDY THE ASSOCIATION OF H.PYLORI AND LARYNGOPHARYNGEAL REFLUX (LPR) AND THEIR INFLUENCE ON LARYNGEAL PATHOLOGIES.

ENT

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ABSTRACT

Background: Laryngopharyngeal reflux occurs when gastric contents pass the upper oesophageal sphincter causing symptoms like sore throat, hoarseness, excess throat mucus, coughing etc. On comparison, LPR most often occurs during the daytime when the person is in upright position whereas gastroesophageal reflux disease occurs commonly in the supine position at night or during sleep. Materials & method: This prospective study was carried out in the Department of Otorhinolaryngology, ASCOMS. About 60 patients (46 males; 14 females) were selected. These patients were asked for presence or absence of the classic symptoms of LPR following which a reflux symptom index (RSI) was established and also endoscopic evaluation of the larynx was done and reflux finding score (RFS) was determined. Patients with an RSI of 14 or more and/or a RFS of 7 or more were considered to have LPR. After these procedures, patients underwent upper GI endoscopy to check for the presence or absence of H.Pylori by rapid urease test. Results: Out of 60 patients, 41 had laryngopharyngeal reflux alone, 1 patient was H.Pylori + alone, 1 had LPR + H.Pylori, 17 were normal. Out of the 41 LPR patients, 61.8% were males and 38.2% were females. Majority of the LPR patients were between the age group of 37– 50 years with primary complaint of hoarsness of voice with maximum patientsdiagnosed as chronic laryngitis and the age range distribution was almost equal among the H.Pylori + and LPR group. Conclusion: The prevalence of LPR in laryngeal pathologies was seen to be higher, therefore being statistically significant. The results demonstrated that there is no association between H.pylori infection and LPR in causing laryngeal pathologies.

KEYWORDS

Laryngopharyngeal reflux, H.Pylori, Laryngeal pathologies

INTRODUCTION:

Laryngopharyngeal reflux is the backflow of stomach contents into larynx and pharynx through the upper oesophageal spinchter which is one of the most common reasons of laryngeal inflammation. LPR can occur during day as well as at night. The most common symptoms associated with LPR are hoarseness of voice, dysphagia, globus pharyngis, cough and excessive throat clearing[1]. Gastroesophageal reflux disease (GERD) on the other hand refers to abnormal reflux of gastric acid into oesophagus resulting in injury to the mucosa, most common symptoms of which are heartburn and regurgitation. H pylori is a micro-aerophilic, gram-negative spiral organism[2], established as a major cause of chronic gastritis and peptic ulcer, therefore causing greatest risk for development of GERD. There are four barriers to reflux reaching the larynx which include lower oesophageal sphincter (LES), upper oesophageal sphincter (UES), epithelial resistance factors and oesophageal peristalsis. Dysfunction in any of these will lead to symptoms of LPR or GERD[3,4,5].

Risk factors for LPR and GERD are closely related including consuming a diet heavy in acidic or fatty foods and caffeine or alcohol, eating large meals before going to sleep, obesity and smoking[6]. Even though tobacco use is well recognised as the primary cause of Reinke's edema in most patients, this condition can arise solely from chronic acid reflux onto the vocal cords[7,8]. The primary differences between LPR and H.Pylori causing GERD are the symptoms manifested and the underlying anatomical defect which tends to be the lower oesophageal sphincter in GERD and the upper oesophageal sphincter in LPR[9].

MATERIALAND METHODS:

This prospective hospital based study was conducted in the Department of Otorhinolaryngology, ASCOMS Jammu during November 2022 to October 2023.

About 60 patients (46 males; 14 females) were selected on the basis of their presenting complaints. All patients were asked about the presence or absence of the classic symptoms of LPR following which a reflux symptom index (RSI) **(Table 1)** was established based on their answers to the RSI questionnaire. All the patients underwent endoscopic evaluation of the larynx through which their reflux finding score (RFS) **(Table 2)** was determined. Patients with an RSI of 14 or more and/or a RFS of 7 or more were considered to have LPR. After these procedures, patients underwent upper GI endoscopy to check for the presence or absence of H.Pylori by rapid urease test.

Inclusion criteria:

- Patients diagnosed with laryngeal pathologies - benign or malignant
- Age above 18 years

Exclusion criteria:

- Patients on treatment with proton pump inhibitors or H2 antagonists or antibiotics within a period of 4 weeks.
- Patients with recent history of gastrointestinal bleeding
- Patients who have received radiation therapy.

Table 1: The Reflux Symptom Index (RSI)

S.NO.	Symptoms	Within the last month, how did the following problems affect you? Circle the appropriate response 0 = No problem 5 = Severe problem
1	Hoarseness or a problem with your voice	0 1 2 3 4 5
2	Clearing your throat	0 1 2 3 4 5
3	Excess throat mucus or postnasal drip	0 1 2 3 4 5
4	Difficulty swallowing food, liquids, or pills	0 1 2 3 4 5
5	Coughing after you ate or after lying down	0 1 2 3 4 5
6	Breathing difficulties or choking episodes	0 1 2 3 4 5
7	Troublesome or annoying cough	0 1 2 3 4 5
8	Sensations of something sticking in your throat or a lump in your throat	0 1 2 3 4 5
9	Heartburn, chest pain, indigestion, or stomach acid coming up	0 1 2 3 4 5

To obtain their RSI score, patients assign a numerical value to each sign/symptom and then calculate the total. The maximum score is 45.

Table 2: Reflux Finding Score (RFS)

S.NO.	Finding	Interpretation
1	Subglottic edema	0 = absent 2 = present
2	Ventricular obliteration	0 =absent 2 = partial 4 = complete
3	Erythema/hyperaemia	0 =absent 2 = only in aretynoids 4 = diffuse
4	Vocal fold edema	0 =absent 1 = mild 2 = moderate 3 = severe 4 = polypoidal
5	Diffuse laryngeal edema	0 =absent 1 = mild 2 = moderate 3 = severe 4 = obstructing
6	Posterior commissure hypertrophy	0 =absent 1 = mild 2 = moderate 3 = severe 4 = obstructing
7	Granuloma/granulation	= absent 2 = present
8	Thick endolaryngeal mucus	0 = absent 2 = present

RESULTS:

It was a prospective study which was performed in 60 patients for a duration of 1 year from November 2022 to October 2023. The age of the patient ranges from 20-60 years with mean age 33.4 years. The male to female ratio was 3.2:1.

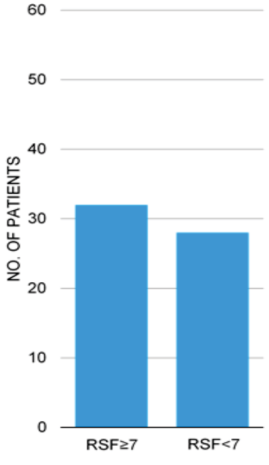

FIGURE: 1

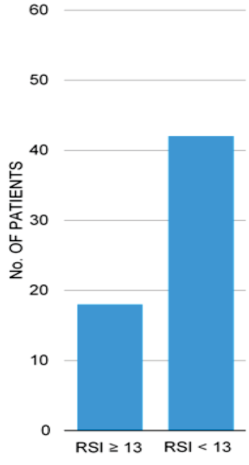

FIGURE: 2

Table 3: Primary complaint at presentation in ENT OPD

S.NO.	PRIMARY COMPLAINT	No. Of patients	PERCENTAGE (%)
1	Hoarseness of voice	22	36.6
2	Throat clearing	6	10
3	Foreign body sensation	18	30
4	Belching	7	11.7
5	Others	7	11.7

Table 4: Correlation of H.Pylori & LPR

S.NO.	ASSOCIATION	NO. OF PATIENTS	PERCENTAGE
1	LPR + H.Pylori +	1	1.7
2	NO LPR but H.Pylori	1	1.7
3	LPR + but NO H.Pylori	41	68.3
4	NO LPR and NOH.Pylori	17	28.3

Based on primary symptom at presentation, the predominant symptoms were hoarseness of voice (36.6%), globus sensation (30%) , throat clearing in (10%) while others like heartburn, difficulty swallowing accounting for about 11.7%**.** Among the H.Pylori and LPR positive patients, predominant symptoms was heartburn. The RSI was **≥ 13** in 18 patients (30%) and **<13** in 42 patients (70%) **(Figure 2)**. Based on laryngoscopic findings, most of the patients showed inter-arytenoid erythema (91.3%), vocal cord edema (78.3%), ventricular obliteration (73.9%), diffuse laryngeal edema (73.9%). The RFS was ≥ 7 in 32 patients (53.4%) and <7 in 28 patients (46.6%) **(Figure 1)**. Out of 60 patients, 41 had laryngopharyngeal reflux alone, 1 patient was H.Pylori + alone, 1 had LPR + H.Pylori , 17 were normal (Table 4). Out of the 41 LPR patients, 61.8% were males and 38.2% were females. Majority of the LPR patients were between the age group of 37–50 years with primary complaint of hoarsness of voice with maximum patients diagnosed as chronic laryngitis.

DISCUSSION:

LPR is usually a multifactorial disease. It may result from direct injury or by a secondary mechanism. Two hypotheses exist about how gastric acid precipitates extraesophageal pathologic response. The first says direct acid-pepsin injury to the larynx and surrounding tissues via esophago-pharyngeal reflux (microaspiration theory). The second hypothesis suggests that acid in the distal oesophagus stimulates vagal mediated reflexes (esophagobronchial reflex theory), which results in bronchoconstriction and chronic throat clearing and coughing, eventually leading to mucosal lesions[10]. These two mechanisms may act in combination to produce the pathologic changes seen in LPR. On the other hand, *H. pylori* infection causes to gastritis with reduced parietal cell activity and reduced acid secretion. Thus, causing risk for laryngeal carcinoma formation[11].

In this study, we have studied the association of H.pylori and Laryngopharyngeal reflux (LPR) and their influence on laryngeal pathologies. It was found that RSI and RFS are highly efficacious clinical scoring systems for assessment of LPR status. There was no relationship between LPR and H.Pylori in causing various benign laryngeal pathologies. Though, a significant relationship was found in LPR causing the vocal polyps, vocal nodules, chronic laryngitis etc.

In a study conducted by Ecru[12] to investigate the role of H.Pylori infection in LPR by upper gastrointestinal system endoscopy and double probe pH monitoring. It was concluded that there is no statistically significant relationship between gastric H.pylori infection and LPR. While in another study concluded by Tessera[13] for the expression of *H. pylori* positivity and degree of GERD correlated with LPR. It concluded that *H. pylori* positivity and degree of GERD were more adverse in patients with an RFS of ≥ 7.

Islam[14] investigated the existence of H.Pylori in patients with benign and malignant vocal fold pathologies and concluded that H. pylori was not found in the histological specimens of the vocal fold and interarytenoid region.

CONCLUSION:

We conclude from our study that a detailed history taking and endoscopic examination of larynx constitutes the basis for diagnosis of LPR. Endoscopic examination of larynx most commonly demonstrates finding like inter-arytenoid erythema, vocal cord edema, ventricular obliteration and diffuse laryngeal edema.

Laryngeal acid and pepsin sensitivity is greater in oropharyngeal mucosa than oesophageal mucosa and this constitutes the main difference of LPR and GERD pathophysiology. The prevalence of LPR in laryngeal pathologies was seen to behigher, therefore being statistically significant. The results demonstrated that there is no association between H.pylori infection and LPR in causing laryngeal pathologies.

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