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Original Article

Voice Disorders and Reflux Disease – A Prospective Study

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Abstract

Introduction: Reflux laryngitis is a voice disorder that results from irritation and swelling of the vocal cords due to the backflow of stomach fluids into the throat. This backflow is called laryngopharyngeal reflux disease (LPRD) (acid that reaches the level of the throat). LPRD is one of the overlooked causes in patients with voice disturbance.

Aim: This study aims to study voice disturbances in 50 patients with suspected LPRD.

Materials and Methods: In this prospective study, 50 patients with suspected LPRD were included. The diagnosis was made based on the patient's history, video laryngoscopy, esophagoscopy, and the lower esophageal mucosa's biopsy. All the LPRD patients were treated with tab. rabeprazole 20 mg for 8 weeks. All the patients subjectively evaluated their voice problems using the Voice Handicap Index (VHI) questionnaire after treatment.

Results: The results of VHI showed the severity of the voice problems of the patients with LPRD. Video laryngoscopy and history proved LPRD in all 50 patients. Esophagoscopy, combined with the esophageal biopsy, detected signs of possible GERD in 40 patients (80%). Video laryngoscopy, combined with a subjective voice assessment, performed based on VHI before and after treatment with a proton-pump inhibitor showed a significant improvement.

Conclusion: Video laryngoscopy assessment of the laryngeal mucosa and esophagoscopy supplemented with a biopsy of the lower esophageal mucosa, showed to be a convenient diagnostic method when GERD and LPRD were suspected. Rabeprazole, a proton-pump inhibitor, proved to be very useful in the treatment of LPRD.

Key words: Laryngopharyngeal reflux, Proton-pump inhibitor, Voice disorders

INTRODUCTION

Gastroesophageal reflux disease (GERD) is defined as the reflux of stomach contents into the esophagus with pathohistological changes of the mucous membrane. When this reflux affects the laryngeal and pharyngeal mucosa, it is termed laryngopharyngeal reflux disease (LPRD).[1,2] Esophageal mucosa has a protective mechanism against aggressive factors of stomach contents, and it remains intact when physiological reflux happens in the night. The laryngeal lining above the upper esophageal sphincter is not as strong a protective lining and so when acidic contents of stomach reflux into the larynx they get irritated and inflamed.[3] Laryngeal and pharyngeal mucosa are very sensitive, and the acid peptic reaction of the stomach contents rapidly leads to mucosal injury. LPRD commonly occurs in daytime due to upper esophageal sphincter dysfunction.[4] The most common part of larynx affected is the posterior half including the arytenoids, interarytenoid junction, and post 1/3 of vocal cords. The esophageal reflux manifests as heartburn, belching, frequent clearing of throat, regurgitation, and bitter taste. The most typical extraesophageal manifestation includes hoarseness of voice. Others include persistent cough, choking episodes, and breathing difficulty.[5] In day-to-day practice, LPRD is mostly not recognized as it is a silent reflux and diagnostic and therapeutic protocols are insufficient. Due to the high prevalence of disease and varied clinical manifestations, most patients report to family physicians. Improper clinical evaluation and inadequate diagnostic options are the biggest challenges in treating reflux effectively. Proper understanding of the etiopathogenesis plays a significant

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role in treating GERD and LPRD. Untreated LPRD can lead to laryngeal cancer.[6] The development of the disease can be life threatening considerably affecting the quality of life. In our study, we have focused on the importance of diagnostic options, including proper clinical history, clinical evaluation, video laryngoscopy, esophagoscopy, and biopsy of the lower esophagus. The patients are treated with proton-pump inhibitors for 8 weeks along with weight reduction, lifestyle modification, and dietary alterations. The improvement of voice after treatment is assessed with Voice Handicap Index (VHI) questionnaire.

Aim

This study aims to study voice disturbances in patients with suspected laryngopharyngeal reflux disease.

MATERIALS AND METHODS

In this prospective study, 50 patients between the age group of 18 and 45 years who presented to the Outpatient Department of ENT in Sree Balaji Medical College and Hospital, with laryngopharyngeal problems in whom GERD was suspected were included in the study. The diagnosis of LPRD was made based on the patient's history and video laryngoscopy using a 45° rigid endoscope. All the LPRD patients were treated with rabeprazole (20 mg) once a day for 8 weeks, combined with appropriate dietary and lifestyle changes. All the patients subjectively evaluated their voice problems using the VHI questionnaire before and after the treatment. Before the treatment, the esophagoscopy and the mucosa biopsy in the lower third of the esophagus were performed for all 50 LPR patients.

Typical esophagitis above the lower esophageal sphincter, hiatal hernia, or dysfunctional lower esophageal sphincter indicated GERDs possibility. Intraepithelial eosinophils, basal zone thickening, and papillary lengthening in the esophageal biopsy specimen were supposed to indicate the prolonged acid reflux. The biopsy was marked as positive when all three criteria were fulfilled. The histologic examination of the esophageal specimens was compared to the results of the esophagoscopy and video endolaryngoscopy. The results of the video endolaryngoscopy and VHI questionnaire were compared, before and after the treatment with rabeprazole.

RESULTS

The main symptoms of the LPRD patients were hoarseness (30 patients), throat clearing (10 patients), and globus pharyngeus sensation (10 patients), two of them being simultaneously present in all LPRD patients [Figure 1].

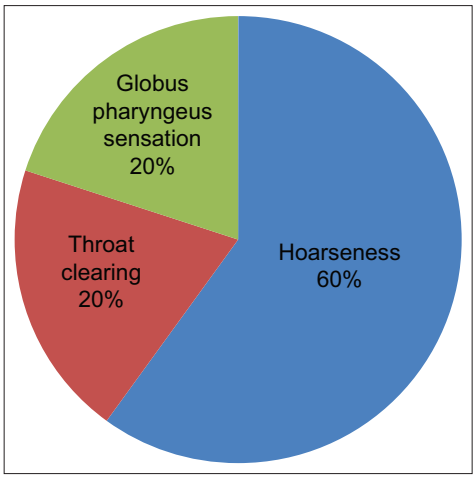

Figure 1: Distribution of symptoms

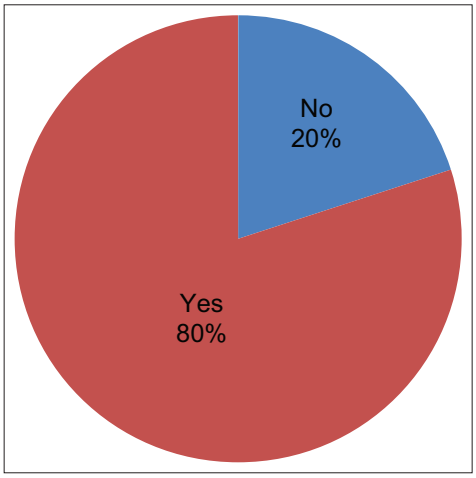

Figure 2: Distribution of GERD in study patients

After the treatment with rabeprazole, the LPRD patients felt relief and felt that their problems decreased by 20% as per the VHI. Esophagoscopy confirmed the possibility of GERD in 14 LPRD patients. The histopathological examination of the esophageal biopsy specimens indicated prolonged acid reflux in 20 patients in whom esophagoscopy did not detect any signs of reflux esophagitis, hiatal hernia, or dysfunctional lower esophageal sphincter. Both examinations, when combined, detected signs of possible GERD in 40 patients (80%) [Figure 2]. Video endolaryngoscopy and history proved extraesophageal or LPRD in all 50 patients.

DISCUSSION

Our study confirmed that LPRD could cause considerable voice problems. The appropriate treatment with a proton-pump inhibitor significantly reduces a patient's problems. In GERD and LPRDs diagnostics, the patient's history and video laryngoscopy are superior to the esophagoscopy.

Esophagoscopy with a biopsy of the esophageal mucosa is a convenient diagnostic method when GERD and LPRD are suspected. The diagnosis of LPRD is based on a patient's symptoms, laryngeal findings, and subjective results. Some GERD is physiologic, occurring mostly after meals.

Ambulatory 24 h double probe (simultaneous esophageal and pharyngeal probes) with pH monitoring is the most suitable diagnostic method. On the other hand, several studies proved that evident signs of LPRD could be detected, even in patients with negative 24 h pH monitoring.[7] It was also proved that pepsin is activated, even in values of pH higher than 4.[8]

LPRD is known to contribute to posterior acid laryngitis, laryngeal contact ulcers or granuloma formation, epithelial dysplasia and laryngeal cancer, chronic hoarseness, pharyngitis, sore throat, globus sensation, dysphagia, buccal burning, asthma, pneumonia, nocturnal choking, and dental diseases. These manifestations are believed to be caused by direct contact of the gastric content and injury to the pharyngeal or laryngeal mucosal surfaces. Acid reflux inside the distal esophagus itself also stimulates vagally mediated reflexes, leading to bronchospasm and coughing disorders.[9]

Extraesophageal reflux can cause damage to the laryngeal mucosa from coughing, voice abuse, intubation, or lower respiratory tract infection. LPRD has been implicated as being causative or contributory in laryngeal pathologic states such as vocal nodules, Reinke's edema, and scar formation as in idiopathic subglottic stenosis, functional laryngeal movement disorders such as muscular tension dysphonia, paradoxical vocal fold motion, and paroxysmal laryngospasm. LPRD also lowers the cough threshold.[10]

In GERD patients, the occurrence of extraesophageal symptoms is as high as 67%.[11] There are many patients with voice disorders who have LPRD as the main or one of the important reasons for their dysphonia. An endoscopic examination of the larynx usually reveals the signs of LPRD with arytenoids congestion, axed vocal cords gap, but the scenario requires a combination of more diagnostic procedures to confirm the clinical suspicion of GERD and LPRD.

In our study, the results of the video laryngoscopy correlated very well with the histological findings of the esophageal mucosa specimens. Video laryngoscopy is a very simple method, which can be easily repeated and is well tolerated by the patients.

LPRD patients can be treated with dietary and lifestyle modifications, alginates, and proton-pump inhibitors. The results of our study confirmed that the treatment with rabeprazole was successful. Our patients assessed that their problems (dysphonia, globus pharyngeus sensation, throat clearing, etc.) decreased after the 2-month therapy. Further improvement is expected with prolonged rabeprazole treatment.

The subjective assessment of voice problems and the acoustic analysis of voice samples confirmed the results of the video laryngoscopy. Following treatment with rabeprazole, the typical LPRD lesions on the laryngeal mucosa diminished to a large extent, and the vocal function of the larynx was much improved. Therefore, LPRD should not be overlooked in the treatment of dysphonic patients. Another objective assessment of the voice improvement is acoustic analysis of voice samples that will confirm the VHI subjective results.

CONCLUSION

LPRD causes severe voice disorders, globus pharyngeus sensation, and frequent coughing. Esophagoscopy supplemented with a biopsy of the esophageal mucosa can be a suitable method to prove GERDs occurrence. Video laryngoscopy is superior in the diagnosis of LPRD and correlates very well with the esophageal mucosa specimens' histological findings. Combining these procedures are supposed to be a very successful method in the diagnostics of GERD and especially LPRD. Rabeprazole proved to be very useful in the treatment of LPRD. Subjective and objective voice assessment methods can demonstrate an improvement by the end of the 2-month therapy. LPRD appears to have a substantial adverse influence on voice quality and can be an important overlooked cause in patients with voice disturbance.

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