The Laparoscopic Nissen Fundoplication Eliminates Obstructive Sleep Apnea Syndrome Due to Gastroesophageal Reflux Disease

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Abstract Gastroesophageal reflux disease may produce esophageal syndromes, such as heartburn and regurgitation. It is a common clinical presentation with extraesophageal manifestations, such as asthma, arrhythmia, snoring, and sleep disturbance, which could make identifying it more difficult than the usual esophageal symptoms. The aim of this study is to characterize the extraesophageal manifestations in patients with gastroesophageal reflux disease and investigate the effect of laparoscopic Nissen fundoplication. We describe the case of a 38-year-old male patient with a history of sleep disturbance attributable to gastroesophageal reflux disease, which resolved on successful laparoscopic Nissen fundoplication treatment. The long-standing sleep apnea obviously improved after laparoscopic Nissen fundoplication treatment. To our knowledge, this is a rare case of successful laparoscopic Nissen fundoplication treatment of a patient with extraesophageal manifestations induced by gastroesophageal reflux disease. The results indicate an underlying mechanism for extraesophageal manifestations and the success of laparoscopic Nissen fundoplication treatment.

Keywords Laparoscopic Nissen fundoplication · Obstructive sleep apnea syndrome · Gastroesophageal reflux disease

Case Report

We report the case of a 38-year-old male patient who had been experiencing acid reflux and heartburn for 10 years, as well as attacks of shortness of breath, stridor, and laryngeal tightness for 6 years. He was otherwise healthy and had no history of smoking, drinking, allergies, or family history of any similar diseases. He received six emergency rescue treatments from a local hospital due to suffocation. We found edema of the throat and laryngospasm when we did incision of trachea at the updated emergency rescue.

Laboratory examinations, heart ultrasound, and chest X-ray yielded no abnormalities. Gastroscopy presented reflux esophagitis with Los Angeles grade C and Barrett esophagus. The *Helicobacter pylori* culture was positive. The Demesster score of 24-hour esophageal pH monitoring was 78.34. It showed severe pathological acid reflux in the distal esophagus. Esophageal manometry indicated that the pressure of lower esophageal sphincter was low. Polysomnography (PSG) found severe nocturnal anoxemia. The patient's apnea hypopnea index (AHI) was 45.7 and the min SpO₂ was 57 %.

The patient was diagnosed with gastroesophageal reflux disease (GERD), reflux esophagitis with LA-C, *H. pylori* (+), Barrett esophagus, and obstructive sleep apnea syndrome (OSAS). He was treated with drugs for GERD, including domperidone, famotidine, and omeprazole. After the chemotherapy, the symptoms, including acid reflux and heartburn, were relieved. However, there were no obvious changes in symptoms such as the attacks of shortness of breath, stridor, laryngeal tightness, and sleep apnea. The repeat PSG found that AHI was 40 and the min SpO₂ was 55 %.

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The patient agreed to undergo a laparoscopic Nissen fundoplication (LNF) on November 24, 2009, and the results were immediately obvious. After 2 weeks of LNF, the symptoms of acid reflux, heartburn, shortness of breath, stridor, and throat tightening disappeared. One year later, the patient's weight was not obviously changed. The 24-hour esophageal pH monitoring showed the Demesster score of 20.3. The laryngospasm has not appeared. The edema of the throat was obviously relieved. PSG found the patient's AHI to be 19 and the min SpO₂ to be 75 %.

Discussion

GERD is a common event primarily accompanied by clinical complaints of heartburn and regurgitation. However, a significant percentage of patients with GERD may experience extraesophageal manifestations such as asthma, laryngitis, excessive throat phlegm, and chronic cough [1].

Obstructive sleep apnea syndrome (OSAS) is a disorder characterized by repetitive obstruction of the upper airway, resulting in apneic and hypopneic episodes during sleep. It is an important risk factor for atherosclerosis and cardiovascular morbidity [2].

GERD has been noted to be a frequent occurrence in patients with obstructive sleep apnea (OSA) [3]. This report deals with a patient who had been diagnosed and treated for sleep apnea. The patient displayed marked improvement in his symptoms following the diagnosis of GERD and treatment with laparoscopic Nissen fundoplication.

Sleep-disordered breathing results from a combination of factors affecting upper airway patency and control of ventilation [4]. Most obstructive apnea patients have anatomically small upper airways. Most therapies aim to reduce apnea severity by increasing airway size and stability. The precise nature of the relationship between OSA and GERD—specifically, whether or not one is involved in the pathogenesis of the other—remains unclear.

Many pathophysiological mechanisms have been proposed to explain the association between GERD and respiratory tract diseases [5]. One mechanism involves spraying gastric contents into the bronchial system [6], which can result in an exudative mucosal reaction that partially obstructs the airway. A second mechanism is through the activation of a vagal reflex from the esophagus to the lung resulting in bronchospasms.

GERD may precipitate symptoms suggestive of OSA, including nocturnal choking and arousal by microaspiration of gastric acid. Acid reflux may also cause laryngeal inflammation and edema, which in turn may worsen upper airway obstruction, as well as cause tracheal contractions that could lead to OSA episodes. Obstructive episodes decrease intrathoracic pressure and increase transdiaphragmatic pressure.

This could trigger GER events, particularly if there is intercurrent transient lower esophageal sphincter (LES) relaxation as well. It has been suggested that increased arousal in patients with OSA, together with reduced sleep efficiency, may trigger transient LES relaxation and promote acid reflux.

Traditional therapeutic interventions include weight loss, avoidance of alcohol, nasal continuous positive airway pressure (nCPAP) [7], uvulopalatopharyngoplasty, maxillomandibular surgery, and tracheostomy [8]. Although the exact relationship between acid reflux and breathing-related sleep disturbances remains to be elucidated, in some instances, pharmacologic treatment for GER has shown a beneficial effect on PSG parameters [9]. Endoscopic Stretta technique has also been used to treat extraesophageal manifestations, such as snoring, sleep apnea, and arrhythmia.

LNF could inhibit reflux or reduce transpharyngeal spraying of gastric and duodenal contents, thereby preventing microaspiration and vagus nerve stimulation [6]. It eliminates heartburn and regurgitation in approximately 90 % of patients. However, the effect of fundoplication on OSA is less predictable. The case presented here shows that the symptoms of OSA due to GERD are relieved by LNF.

We believe there are two mechanisms that could explain the phenomenon of clear decrease in OSA. First, LNF reduced the esophageal acid exposure, improved recovery of the lower esophageal sphincter function, increased gastric emptying speed, and restored impaired esophageal peristalsis [10]. The laryngeal inflammation and edema regressed and relieved upper airways obstruction and apnea. Second, LNF could cut off and destroy the sensory nerve ending distribution on the surface of the esophagus; thus, it could decrease the reflex of the vagus nerve even if acid reflux persists.

In conclusion, although LNF can control heartburn and regurgitation in approximately 90 % of GERD patients [11], the current case is perhaps the first to demonstrate its use for treating symptoms induced by OSA. Extraesophageal manifestations of GERD can be misdiagnosed easily. When patients have recurrent refractoriness of asthma, laryngitis, atypical retrosternal pain, or even OSA, physicians should consider the possibility of GERD. LNF is an effective procedure that can relieve GERD symptoms and improve the quality of life of patients.

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