

EC GASTROENTEROLOGY AND DIGESTIVE SYSTEM Research Article

The Effect of *Helicobacter pylori* on the Recurring Complaints of Patients with Previous Anti-Reflux Surgery

Fatin R Polat*

Associate Professor, Department of Surgery, Dr. Namık Kemal University Medical Faculty, Tekirdag, Turkey

*Corresponding Author: Fatin R Polat, Associate Professor, Department of Surgery, Dr. Namık Kemal University Medical Faculty, Tekirdag, Turkey.

Received: January 16, 2018; Published: February 20, 2018

Abstract

Purposes: Gastroesophageal reflux disease (GERD) is a multifactorial disease. When GERD symptoms become refractory in despite of adequate medical therapy, surgical interventions may be indicated. Before proceeding with an anti-reflux operation, several factors should be evaluated. The aim of this study is to determine the role of Helicobacter pylori (HP) infection in patients who had undergone anti-reflux surgery previously because of symptomatic reflux disease.

Method: Between 2000 and 2014, thirty six patients who previously had anti-reflux surgery and received endoscopy due to recurring complaints were accepted for this retrospective clinical study. During the endoscopic procedure; antral biopsies were performed and evaluated.

Results: HP was detected to be positive in 34 of 36 patients. HP infection was found to be significantly related with increased incidence of recurring complaints related with GERD in patients who previously had anti-reflux surgery.

Conclusions: Before proceeding with an anti-reflux operation, precipitating factors such as presence of gastric outlet obstruction (because of HP) and the condition of oral hygiene shall be evaluated carefully. HP eradication before anti-reflux surgery, in both of the oral and the gastric cavity, may prevent the recurrence of the patients' GERD associated complaints after surgery.

Keywords: Anti-Reflux Surgery; Helicobacter pylori; GERD

Abbreviations

HP: Helicobacter pylori; HH: Hiatal Hernia; GERD: Gastroesophageal Reflux Disease; PPI: Proton Pump Inhibitor

Introduction

Gastroesophageal reflux disease (GERD) is a multifactorial disease. The roles of *Helicobacter pylori* (HP), hiatal hernia (HH), environmental, dietary, oral hygiene, periodontal disease and host physiological factors are well established [1-6]. HP infection represents one of the most common and medically prominent infections worldwide. When GERD symptoms are troublesome despite adequately dosed medical therapy and life-style modifications therapy, surgical correction may then be indicated [7].

In most of the patients who had anti-reflux surgery, many of the symptoms may recur in a short time after surgery [4,8]. This is thought to be the result of insufficient preoperative evaluation. Such as HP should be investigated, pH meter should be done and the anatomical structure of the esophagus (abdominal part) should be known. We suspect that lack of an extensive investigation for the presence of HP infection may be one of the main underlying reasons for failing anti-reflux surgery. This study was carried out to determine the role of HP infection in the recurrence of the disease in patients who previously had anti-reflux surgery for symptomatic reflux disease.

Material and Methods

After the approval of the local institution's ethics committee, a retrospective analysis was performed for 8,540 patients who had upper gastrointestinal endoscopy over a 14-year period (from January 2000 to December 2014) at Van State Hospital and Sakarya Yenikent State Hospital in Turkey. Among them, thirty six patients had previously anti-reflux surgery, admitted to our endoscopy unit due to recurring complaints. The patients were accepted for the study after acquisition of their written informed consent. The presence of HP infection in the patients who had previously undergone anti-reflux surgery was analysed. The cases were divided into two groups according to the presence of HP infection; HP positive and negative. Demographic data (age, sex), and severity of HP infection were comparatively analysed.

Technique for HP identification; The severity of HP infection was classified as follows: HP -(negative); no HP bacterium is present in the evaluated area, HP +(positive); 10 to up HP bacteria per area in microscope. Statistical analysis was processed with SPSS® ver. 21.0 (Chicago IL). P < 0.05 was accepted to be statistically significant.

Results

The study includes 10 male (28%) and 26 female (72%) patients. The median value for the age of the patients is 40.5 (33 - 48) years. The recurring complaints of patients were similar regardless of gender. Antral biopsies were performed in all the patients during the procedure. The histological evaluation for HP infection in the specimens showed that HP infection existed in 34 (%94,5) of 36 the patients. The mean time period before admission to our clinic was 6 - 10 months after previous anti-reflux surgery. No mortality or complication occurred during the procedure. None of the patients had an antral biopsy performed prior to surgery. There is a statistically significant (p < 0.05) relationship between HP and the recurring complaints of patients who had previous anti-reflux surgery.

Helicobacter pylori	The patients with anti-reflux surgery	GERD (Grade I-IV)
Negative (0)	2 (%5,5)	0
Positive (+)	34 (%94,5)	30
Total	36 (%100)	30/36 (%83,3)

Table 1: Incidence of HP In Patients With Previous Anti-reflux Surgery.

The recurring complaints of patients	Frequency of symptoms	
Substernal or epigastric burning pain	29 (%80,5)	
Regurgitation	20 (%55)	
Excessive flatulence	32 (%88,8)	
Dysphagia	32 (%88,8)	
Atypical symptoms	15 (%41,6)	
No any complaints	4 (%11,1) (2 case HP -)	

Table 2: The recurring complaints of patients.

Discussion and Conclusion

HP is a micro-aerobic, gram-negative bacterium. HP infection represents one of the most common and medically prominent infections worldwide. HP is accumulated in the antrum. Infection produces chronic antral gastritis, increased acid and gastrin secretion, and decreased mucosal resistance to acid. Also HP causes gastric outlet obstruction as a causal factor in the development of (GERD) [1,2].

GERD is a common disease that accounts for approximately 75% of esophageal pathology [9-11]. GERD is a multifactorial disease; the roles of HP, hiatal hernia, environmental, dietary, oral hygiene, periodontal disease and host physiological factors are well established

[4,5]. The typical manifestations of GERD include dental caries, dry mouth, feeling of oral acid/burning sensation, halitosis, erythema of the palatal mucosa and uvula. For diagnosis, it is mandatory exclude other causes, like dietary factors, drugs, poor oral hygiene, eating behavior disorders, genetic and racial factors [6]. The esophageal pH monitoring and/or endoscopy are usually necessary just to comfirm the diagnosis of GERD [6]. All of the case undergo endoscopy for comfirm the diagnosis of GERD. % 83 in patients who had undergone anti-reflux surgery previously because of symptomatic reflux disease were identified symptom of GERD (Table 1, 2).

GERD is the most common disease of the gastrointestinal tract for which patients seek medical therapy. Various endoscopic anti-reflux interventions, although innovative, have not been successful consistently in controlling gastroesophageal reflux [11]. Pathophysiology in GERD is related to abnormal exposure of the distal esophagus to refluxed stomach contents [11]. When GERD symptoms(heartburn, Excessive flatulence, regurgitation, chest pain, and/or supraesophageal symptoms) become refractory in despite of adequate proton pump inhibitor (PPI) therapy and life-style modifications therapy, surgical correction may be indicated. Alternatively, surgical therapy should be considered in symptomatic patients who have achieved relief with medical therapy but to whom the prospect of a lifetime of medicine is undesirable [10].

There is a linear and violent relationship between GERD and HP [2,3]. According this retrospective study; there is a linear and violent relationship between HP and the recurrence of complaints in patients with previous anti-reflux surgery (p < 0.05). On the other hand, there is a high degree of correlation between reflux threshold and the degree of hiatal hernia [7].

Medical treatment aims to reduce the duration and amount of esophageal exposure to gastric contents and to minimize the effects on the esophageal mucosa [7,10,11]. Approximately 25% to 50% of the patients with GERD have persistent or progressive disease, and it is this patient population that is best suited to surgical therapy [7]. Surgical treatment should be considered in patients who have symptomatic reflux, have manometric evidence of a defective lower esophageal sphincter (LES), and fail to achieve relief with maximal medical management [11,12]. Preoperatively, the patients should be evaluated sufficiently. In most of the patients who have anti-reflux surgery; a short time after surgery many of symptoms such as heartburn, regurgitation, chest pain, and/or supraesophageal symptoms.seem to be recurrening [7]. This condition may not be related with inaccurate surgery, but it may be a result of insufficient preoperative evaluation [7]. GERD is believed to have its origins within the stomach [7]. Before proceeding with an anti-reflux operation, several factors such as enough gastric outlet(one cause is HP) and oral hygiene should be evaluated [7]. Before surgery, biopsies on the antrum must be taken for HP and oral cavity shall be observed absolutely [5]. This condition is the cause of gastric outlet obstruction (because of HP) and recurrence of GERD symptoms. On the other side; dental plaque, poor oral hygiene, and periodontal diseases are risk factors for HP [6,7]. Because of that, the oral cavity has been proposed as a reservoir for HP [7].

As a result the patients who are candidate for anti-reflux surgery must be preoperatively researched for HP and additionally pH meter should be done and the anatomical structure of the esophagus (abdominal part) should be known. HP should be eradicated in the oral and gastric cavity to prevent the recurrence of complaints after anti-reflux surgery.

Conflict of Interest

The author has no conflict of interest to declare.

Bibliography

- 1. Safavi M., et al. "Treatment of helicobacter pylori infection: Current and future insights". World Journal of Clinical Cases 4.1 (2016): 5-19.
- 2. Polat FR and Polat S. "The Effect Of Helicobacter Pylori On Gastroesophageal Reflux Disease". *Journal of the Society of Laparoendo-scopic Surgeons* 16.2 (2012): 260-263.

- 3. Polat FR and Polat S. "The relationship between grade's of the Gastroesophageal Reflux Disease and Hiatal hernias". *HealthMed Journal* 6.7 (2012): 2268-2270.
- 4. Rasmussen LT., et al. "Helicobacter pylori detection in gastric biopsies, saliva and dental plaque of Brazilian dyspeptic patients". Memorias do Instituto Oswaldo Cruz 105.3 (2010): 326-330.
- 5. Polat FR. "Letter to editor: Helicobacter Pylori and Nissen Funduplication". *Gastrointestinal Cancer: Research and Therapy* 2.1 (2017): 1013.
- 6. Carini Francesco., et al. "Association Between Helicobacter Pylori Infection and Pathological Oral Manifestations". *Journal of Translational Medicine* 4.1 (2016): 9-15.
- 7. Blair A Jobe., *et al.* "Esophagus and Diaphragmatic Hernia". In: Schwartz SI, ed. Principles of Surgery. Seventh edition. New York: McGraw-Hill International Inc. 1 (2010): 941-1024.
- 8. Kahrilas PJ. "The role of hiatus hernia in GERD". Yale Journal of Biology and Medicine 72.2-3 (1999): 101-111.
- 9. Payão SL and Rasmussen LT. "Helicobacter pylori and its reservoirs: A correlation with the gastric infection". World Journal of Gastro-intestinal Pharmacology and Therapeutics 7.1 (2016): 126-132.
- 10. Rishindra M Reddy and Bryan F Meyers. "Esophagus". Klingensmith, Mary E. Chen(Editor), Washington Manual of Surgery, 5th Edition, Washington Lippincott Williams & Wilkins (2008): 154-169.
- 11. Peters JH and Demeeste TR. "Gastroesophageal reflux and hiatal hernia". Michael J. Zinner MJ, ed. Abdominal Operations. Tenth edition. London: Prentice Hall International Inc. (2012): 787-842.
- 12. Koike T., *et al.* "Helicobacter pylori infection prevents erosive reflux esophagitis by decreasing gastric acid secretion". *Gut* 49.3 (2001): 330-334.

Volume 5 Issue 3 March 2018 ©All rights reserved by Fatin R Polat.