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Evaluation of the Relationship Between Reflux Esophagitis and Helicobacter Pylori Stomach Infection in Patients Referring to Zahedan Hospitals, Sistan and Baluchistan Province, Iran

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Abstract

Gastro-oesophageal reflux disease is a common disorder of the digestive tract. Exposure of the oesophageal mucosa to gastric acid can lead to mucosal damage as in oesophagitis. The aim of this study was to determine the relationship between Helicobacter pylori infection in the stomach and reflux oesophagitis in patients, referring to the endoscopy units of hospitals in Zahedan between 2015 and 2019. In this cross-sectional study, all patients (under 15, 15-25, 25-35, 35-45 and 45 years and older) who referred to the Endoscopy units of two randomly selected hospitals in Zahedan during 2015-2019, were studied. H. pylori infection was diagnosed from pathological specimens. Information on all patients, including age, sex, presence of oesophagitis and its grade, and H. pylori infection was recorded on questionnaires and their relationships with oesophagitis and its grade as well as H. pylori infection were analyzed statistically. Tables and charts were used to illustrate the data. T-tests and Chi-squared tests were used to investigate the relationships between variables. The results showed that the frequency of oesophagitis in our patients was 52.8%, with the highest frequency being obtained for grade B oesophagitis (48%). In this study, the prevalence of H. pylori infection was 57.5%. This rate was 55.4% in patients with oesophagitis, with a statistically significant difference from the group without oesophagitis. There was a significant relationship between grade A oesophagitis and H. pylori infection. In conclusion, we were able to demonstrate a significant relationship between H. pylori infection and oesophagitis in affected patients.

Keywords: *H. pylori* bacteria, Reflux oesophagitis, Endoscopy unit, Hospital, Zahedan

Introduction

Helicobacter pylori (*H. pylori*) is a gram-negative, spiral, microaerophilic bacillus that can be isolated only from humans and other primates (Aydogdu *et al.*, 2009). It has the ability to reach the protective mucus layer at the surface of gastric mucosa and to survive the extreme acid content of the stomach thanks to its 4-6 flagella (Aziz et *al.*, 2015), and by avoiding low pH areas using chemiotaxis it first colonizes the antrum, where there are no acid-producers cells (Bore *et al.*, 2017). Approximately, half of the world's population is infected with the bacterium and the prevalence of its infection varies in different countries (Camilo *et al.*, 2017), with about 25 per cent in developed countries, and around 80 per cent in developing countries, including Iran (Queiroz, 2014). It is colonized in the gastric mucosal epithelium and, through the formation of oxidative stress, causes damage and apoptosis, thereby causing changes in the gastric epithelial tissue (Ciclitira, 2001, Feighery, 1999). Symptoms of the infection include decreased appetite, abdominal pain during the night or while eating, weight loss, pallor and other abdominal symptoms (Camilo, 2017; Diamanti, 1999). The H. pylori infection also leads to gastritis, peptic ulcers and chronic pathological changes in the gastric mucosa, such as atrophy, metaplasia and adenocarcinoma (Ghoshal & Chourasia, 2010). Gastro oesophageal reflux disease is a common disorder caused by an increase in the frequency or duration of oesophageal contact with the contents of the stomach. Oesophageal mucosa contact with gastric acid can lead to visible mucosal lesions in the endoscopy, including erosive oesophagitis, oesophageal stricture, and Barrett's oesophagus (Haruma, 2004; Jozefczuk *et al.*, 2005). Since the prevalence of H. pylori infection and gastro-oesophageal reflux disease is high (Kandulski & Malfertheiner, 2014), some correlation is expected to exist between them. The results of studies show that H. pylori may be directly correlated with oesophagitis and gastro-oesophageal reflux disease through various mechanisms. These mechanisms include increased acid secretion, direct infection of the columnar epithelium in the distal oesophagus with H. pylori, and indirect damage due to release of harmful substances into the gastric juice (Konturek *et al.*, 2000). Colonization of the gastric mucosa with H. pylori can lead to diffuse and atrophic gastritis, which reduces gastric acid and therefore oesophagitis (Konturek *et al.*, 2000). The association between H. pylori infection and oesophagitis has been controversial over recent decades. H. pylori infection, despite increasing the risk of peptic ulcer and gastric cancer, has been associated with a substantial reduction in the risk of oesophagitis, Barrett’s oesophagus and oesophageal adenocarcinoma (Diamanti *et al.*, 1999; Luzza *et al.*, 1999). Referring to several population studies, there is a noticeably inverse relationship between H. pylori and gastro-oesophageal reflux, but when considering the single patient this relationship is difficult to explain, since gastro-oesophageal reflux is a disease determined by several

concomitant factors. Some studies have examined the association between H. pylori infection, Barrett's oesophagus and adenocarcinoma, with contradictory results (Memeo *et al.,* 2005). The results of some studies have indicated that eradication of H. pylori may cause oesophagitis reflux and exacerbate the symptoms of oesophagitis (Konturek *et al.,* 2000; Nejad *et al.,* 2011). Studies have shown a low prevalence of H. pylori in patients with gastro-oesophageal reflux disease and oesophagitis (Villanacci *et al.,* 2006). Considering the high prevalence of oesophagitis and H. pylori in endoscopic patients and the importance of H. pylori infection treatment and eradication, the aim of the current study was to determine the relationship between H. pylori stomach infection and reflux oesophagitis in patients referring to the Endoscopy Units of two hospitals in Zahedan from 2015-2019.

Materials and Methods

All patients who underwent endoscopy for various reasons, such as gastrointestinal bleeding, dyspepsia, abdominal pain, anaemia, and other signs of risk and indications, were included in the study if their information was complete. Reports with incomplete data were excluded. In this cross-sectional study, all patients who referred to the Endoscopy Units of two hospitals in Zahedan for endoscopy during 2015-2019 were studied. Prior to pathological sampling and before endoscopy, informed consent for the use of patient information in relevant research was obtained from all patients. All patient information was also kept confidential, and disclosure of information was avoided. Identification codes were used to prevent registration of the patient's name and surname. The data were recorded on SPSS software as numerical codes. The severity of oesophagitis was determined by Los Angeles classification, consisting of 4 grades, such as A, B, C and D. The endoscopy was performed by two gastrointestinologists in the endoscopy units of the hospitals mentioned. Additionally, endoscopic reports were drawn from the hospital system and information including age, sex, cause of endoscopy and current lesions in different parts of the digestive system were recorded on the checklist. Further, pathologic reports on these patients were obtained from pathology laboratories. Diagnosis of H. pylori infection was based on pathological specimens. All patients' information, including age, sex, oesophagitis and its grade, and H. pylori infection, was recorded on the questionnaire. After data collection, the data were entered into the SPSS version 15 and analyzed for association between oesophagitis and its grade, and H. pylori infection.

Data analysis

T-tests and chi-squared tests were used to investigate the relationship between H. pylori stomach infection and reflux oesophagitis in patients referring to the Endoscopy Units of two hospitals in Zahedan during 2015-2019 and the level of significance in this study was considered to be ($p \leq 0.05$).

Results and Discussion

The demographic characteristics of the patients are shown in Table 1. This shows the variables, cumulative frequency and percentages. Lower and the higher frequency corresponded to the under 15s and 35-45-year-old patients respectively.

Table 1. Demographic characteristics of patients

Variables	Frequency	Percentage
Under 15	9.5	9.6
15-25	35.5	26.2
25-35	56.2	20.2
35-45	76.2	20.1
More than 45 years	100	23.5
Male	50	50
Female	90	50
Fischer (Negative)	81.5	81.5
d2 positive	95	18.2
Negative H. pylori	54.5	54.5
Positive H. pylori	100	45.2
Negative scalping	81.5	81.5
d2 positive	96	18.6
Negative atrophy	52.5	52.5
Positive	92	47.2
Marsh 1 Negative	87.5	87.2
d2 positive	100	12.2
Marsh 2 negative	96.6	96.5
Positive (d2)	94	3.5
Marsh 3 Negative	90.2	90.2
Positive (d2)	98	9.2
Negative d. mild. atrophy	62.5	62.5
Positive (d2)	96.4	33.5

Positive (Bulb)	98.1	1.5
d2 and bulb	99	2.1
Negative d. mod. atrophy	95	95.5
Positive (d2)	92	4.5
Negative d. severe. atrophy	94.2	93
Positive (d2)	97	5.4

The results of relationships between oesophagitis and H. pylori infection are shown in Table 2. The data showed a statistically significant difference in the group without oesophagitis.

Table 2. Relationships between oesophagitis and Helicobacter pylori infection

Variables	SEM	Total	H. pylori	
			Positive (%) No	Negative (%) No
Age 15 or under	0.52	(100) 14	(50) 7	(50) 7
15-25		(100) 39	(35.9) 14	(64.1) 25
25-35		(100) 30	(56.7) 17	(43.3) 13
35-45		(100) 30	(46.7) 14	(53.3) 16
More than 45 years old		(100) 35	(42.9) 15	(57.1) 20
Male	0.01	(100) 74	(55.4) 41	(44.6) 33
female		(100) 74	(35.1) 26	(64.9) 48
Fischer Negative	0.54	(100) 121	(45.5) 55	(54.5) 66
Positive (d2)		(100) 27	(44.4) 12	(55.6) 15
Negative scalping	0.29	(100) 121	(43.8) 53	(56.2) 68
Positive (d2)		(100) 27	(51.9) 14	(.48.1) 13
Negative atrophy	0.08	(100) 78	(51.3) 40	(48.7) 38
Goodness		(100) 70	(38.6) 27	(61.4) 43
Marsh 1 Negative	0.03	(100) 130	(40.8) 53	(59.2) 77
Positive (d2)		(100) 18	(77.8) 14	(22.2) 4
Marsh 2 negative	0.41	(99) 143	(44.8) 64	(55.2) 79
Positive (d2)		(100) 5	(60) 3	(40) 2
Marsh 3 Negative	0.53	(100) 134	(45.5) 61	(54.5) 73
Positive (d2)		(100) 14	(42.9) 6	(57.1) 8
Negative d. mild. atrophy		(100) 93	(51.6) 48	(48.4) 45
Positive (d2)	0.05	(100) 50	(32) 16	(68) 34
Positive (Bulb)		(100) 2	(100) 2	(0) 0

d2 and bulb		(100) 3	(33.3) 1	(66.7) 2
Negative d. mod. atrophy	0.39	(100) 141	(44.7) 63	(55.3) 78
Positive (d2)		(100) 7	(57.1) 4	(42.9) 3
Negative d. severe. atrophy	0.53	(100) 140	(45) 63	(55) 77
		(100) 8	(50) 4	(50) 4

The results of some reliable studies have been conducted on the association between oesophagitis and H. pylori infection for example, different studies in Asia have shown an inverse correlation between H. pylori infection and reflux oesophagitis (Haruma, 2004; Kandulski & Malfertheiner, 2014; Villanacci *et al.*, 2006; Rostami-Nejad *et al.*, 2016). In a Diamanti study, the prevalence of erosive oesophagitis and Barrett's oesophagus were reported to be 4.3% and 1%, respectively (Diamanti *et al.*, 1999), suggesting that H. pylori infection is inversely correlated with erosive oesophagitis (Luzza *et al.*, 1999). The result of another study showed that in patients undergoing upper endoscopy, the group with erosive oesophagitis had a higher prevalence of H. pylori infection than the control group, and the prevalence of the infection was inversely correlated with oesophagitis to different degrees (Villanacci *et al.*, 2006). A study that was performed on endoscopies showed that 49.5% had oesophagitis, 67.8% of whom were confirmed pathologically, and 77.7% of patients diagnosed with oesophagitis by endoscopy, had H. pylori (Rostami-Nejad *et al.*, 2006). In some studies, the prevalence of erosive oesophagitis and Barrett's oesophagus with a high prevalence of H. pylori infection was low (Kandulski & Malfertheiner, 2014; Memeo *et al.*, 2005; Nejad *et al.*, 2011; Shavalipour *et al.*, 2017). Studies in western countries have also shown that there is an inverse correlation between H. pylori infection and reflux oesophagitis (Shavalipour *et al.*, 2017). And this relationship in our study was significant. A study by Villanacci showed that there was an inverse correlation between oesophagitis and H. pylori infection, and that the infection had a protective role, countering the incidence of oesophagitis (Villanacci *et al.*, 2006). However, in some studies, a direct correlation between H. pylori infection and oesophagitis has been observed. For example, Fatin et al., in a study of patients with upper endoscopy, divided the patients into two groups: H. pylori-infected and non-H. Pylori infected. It was observed that H. pylori infection and oesophagitis and its grade were directly correlated (Jozefczuk *et al.*, 2015). In addition, in another study, the prevalence of H. pylori infection in gastro-oesophageal reflux disease patients and was compared with a control group; this showed that the prevalence of the infection was significantly higher in the gastro-oesophageal reflux disease patients group than in the control group (Rostami-Nejad *et al.*, 2006; Vakil, 2006). The results of these studies are consistent with our study (Villanacci

et al., 2006; Rostami-Nejad *et al.,* 2006). Meanwhile, a study of Konturek's patients with reflux symptoms used manometry, oesophageal pH measurements and biopsy. In that study, no association was observed between H. pylori infection and lower oesophageal sphincter pressure, oesophageal manometric waves and oesophagitis in pathological examinations (Konturek *et al.,* 2000). A study conducted of patients with gastro-oesophageal reflux disease showed that H. pylori infection had no significant association with gastro-oesophageal reflux disease and erosive oesophagitis (Yaghoobi *et al.,* 2010). In a study by Queiroz of gastro-oesophageal reflux disease patients, the eradication of H. pylori infection did not affect the clinical, endoscopic and manometric characteristics of the patients (Queiroz, 2004). Additionally, other investigations showed that there was no difference between H. pylori prevalence in gastro-oesophageal reflux disease patients and the controls conducted, but the prevalence of the cag A gene of H. pylori and the co-existence of cag A and cag E were significantly higher in the control group (Rostami-Nejad *et al.,* 2006; Rostami-Nejad *et al.,* Queiroz, 2004).

Conclusion

In conclusion, we were able to demonstrate that there was a significant relationship between H. pylori infection and oesophagitis in patients with oesophagitis, and the highest frequency of H. pylori infection was observed in grade B oesophagitis.

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