

ORIGINAL ARTICLE

Association of *Helicobacter pylori* Infection with Atrophic Gastritis in patients with Dyspepsia

Mohammad Nosrati¹, Mohsen Sadeghi^{2*}, Farnaz Mohseni³

¹Internal medicine resident, Imam Hossein Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran

²Internal medicine resident, Imam Hossein Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran

³Medical doctor, Tehran University of Medical sciences, Tehran, Iran

*Corresponding author: Mohsen Sadeghi (MD)

ABSTRACT

Chronic gastritis is a common cause of dyspepsia. In the setting of chronic gastritis, glandular and mucosal epithelium atrophy can lead to atrophic gastritis. One of the important risk factors of this disease is *Helicobacter pylori* infection. Atrophic gastritis can lead to intestinal metaplasia that if remains untreated can progress to malignancy. So the aim of our study is evaluation of association between *Helicobacter pylori* and atrophic gastritis. All patients with dyspepsia after consideration of inclusion and exclusion criteria were undergone necessary laboratory tests and upper GI endoscopy. After that, pathology report of gastric biopsy investigated for gastritis and *H.pylori* infection. Of the 54 patients enrolled in the study, 23 (6/42 %) were males and 31 (4/57 %) were female. The mean age of the participants in the study was 57.7 ± 10.5 years. 29 (53.7 %) were positive for *H. pylori* infection. 6 patients (11/ 1%) had atrophic gastritis. All patients with atrophic gastritis in this study were positive for *H. pylori* ($p = 0.01$) and all patients with atrophic gastritis had intestinal metaplasia. ($P < 0.001$). According to our study, effective treatment of *H.pylori* infection and modification of life style against this infection would cut the circle of chronic (atrophic) gastritis, intestinal metaplasia and malignancy.

Key words: Atrophic gastritis, Chronic gastritis, *Helicobacter pylori*, Intestinal metaplasia

Received 10/03/2016 Accepted 11/07/2016

©2016 Society of Education, India

How to cite this article:

M Nosrati, M Sadeghi, F Mohseni. Association of *Helicobacter pylori* Infection with Atrophic Gastritis in patients with Dyspepsia. Adv. Biores., Vol 7 [6] November 2016: 20-24. DOI: 10.15515/abr.0976-4585.7.6.2024

INTRODUCTION

Chronic gastritis is the most common cause of dyspepsia. If the inflammation is severe and so that leads to atrophy of the glands and mucosal epithelium are called atrophic gastritis. Atrophic gastritis divided into two main groups: Autoimmune (type 1), frequently in fundus, and non-autoimmune (type 2), frequently in antrum, atrophic gastritis. Two groups regardless of different Pathogenesis and place of involvement are similar in histopathological changes [1]. Despite complex etiology, many factors are playing role as etiology of non-immune atrophic gastritis. Factors have been proposed in this field are alcohol consumption, smoking, biliary reflux, allergies to food, drugs such as NSAIDs. But the most important factor is *Helicobacter pylori* infection is. Complication of atrophic gastritis is very important. Intestinal metaplasia is an important one which, if remains untreated, lead to gastric malignancy. Occurrence of intestinal lymphoma (Maltoma) in presence of *Helicobacter pylori* infection is other serious complications of the disease. Duodenal and gastric ulcers are other complications that could lead to situations such as perforation, obstruction [2]. With regard to the items listed above, it is important to separate patients with chronic gastritis from other patients who have similar symptoms and prescribe specific treatment and monitoring of to prevent mentioned complications. So the aim of the current study was to determine the prevalence of atrophic gastritis and its association with *Helicobacter pylori*.

MATERIAL AND METHODS

Patients with dyspepsia referred to Gastroenterology Clinic enrolled to study, after informed consent, complete physical examination was performed for all of them, then necessary blood tests was taken. Inclusion criteria: patients with symptoms of dyspepsia, patients older than 30 years. Exclusion criteria: patients older than 80 years, patients with acute abdomen, heart disease, COPD patients, Patients with coagulation disorders and anemia, patients with acute gastrointestinal bleeding, history of alcohol use, cigarette smoking, the risk of biliary secretion of gastric reflux, food allergies, history of NSAID consumption. Patients who matched with criteria entered to study. An experienced endoscopist performed upper endoscopy for patient under acceptable sedation, during procedure biopsies were taken from the antrum and fundus, but because of most atrophic gastritis associated *H.pylori* infection are in antrum, analysis was performed on antral biopsy.

Statistical analysis

Data are expressed as the mean \pm SD. Data analysis was performed using SPSS 22 software. To investigate the association between quantitative variables using student t - test and χ^2 test was used to assess qualitative variables. P value less than 0.05 is considered significant.

RESULTS

Of the 54 patients enrolled in the study, 23 (42.6%) were males and 31 (57.4%) were female. The mean age of men was 63.1 ± 8.5 years old and women 53.6 ± 10 years old. The mean age of the participants in the study was 57.7 ± 10.5 years old. According to the pathology of patients, 29 patients (53.7%) were positive for *H. pylori* infection and 25 (46/3%) were negative. Pathological findings in 51 patients (94.4%) were suggestive for chronic gastritis. 6 patients (11/1%) had atrophic gastritis. 17 patients (31.5%) had intestinal metaplasia, and 37 patients were negative for this change.

Relationship between sexual groups and Helicobacter pylori infection, chronic gastritis, atrophic gastritis and intestinal metaplasia Pearson was assessed with chi square. Only chronic gastritis was significantly higher in women, ($P = 0.03$) of the 51 patients chronic gastritis, 31 females and 20 males. Other findings related to gender were not significant (table 1).

Table 1. frequency of helicobacter pylori infection, chronic and atrophic gastritis and intestinal metaplasia in males and females

	Frequency (%)	Helicobacter pylori infection*		Chronic gastritis**		Atrophic gastritis***		Intestinal metaplasia****	
		+	-	+	-	+	-	+	-
females	31(42.6%)	16(55.1%)	15(60%)	31(60.7%)	0(0%)	3(50%)	28(58.3%)	9(52.9%)	22(59.4%)
males	23(57.4%)	13(44.9%)	10(40%)	20(39.3%)	3(100%)	3(50%)	20(41.7%)	8(47.1%)	15(40.6%)
All patients	54(100%)	29(100%)	25(100%)	51(100%)	3(100%)	6(100%)	48(100%)	17(100%)	37(100%)

* $P=0.72$, ** $P=0.03$, *** $P=0.69$, **** $P=0.65$

Relationship between age and Helicobacter pylori infection, chronic gastritis, atrophic gastritis and intestinal metaplasia analyzed with Independent T-test method, no significant association was seen except chronic gastritis that in the lower age rate of gastritis was higher (Table 2).

Table 2. mean age of patients with helicobacter pylori infection, chronic and atrophic gastritis and intestinal metaplasia

	Helicobacter pylori infection*		Chronic gastritis**		Atrophic gastritis***		Intestinal metaplasia****	
	+	-	+	-	+	-	+	-
Mean age of patients	56.2 \pm 9.7	59.4 \pm 11.2	56.9 \pm 10.1	70.3 \pm 10.2	54.6 \pm 9.9	58.1 \pm 10.6	56.1 \pm 11.2	58.4 \pm 10.2

* $P=0.47$, ** $P=0.03$, *** $P=0.47$, **** $P=0.79$

Among patients who had no gastritis, Helicobacter pylori infection also was absent. But among those who had chronic gastritis, Helicobacter pylori infection were negative in 22 patients, while positive in 29 patients ($P=0.05$) among those who had chronic gastritis (51 patients), 34 patients were negative metaplasia and 17 were positive. In contrast all patients who had not gastritis were negative for intestinal metaplasia. Based on the analysis performed, the amount of metaplasia in case of chronic gastritis compared with those who had not, there was no significant difference. ($P=0.22$)

The association of *Helicobacter pylori* infection and the metaplasia, results of the patients without infection; 20 patients had no metaplasia and 5 were metaplasia. In case of patients with *H. pylori* infection, 17 patients had metaplasia and 12 were without metaplasia. (p=0.09) In patients with atrophic gastritis, *Helicobacter pylori* infection was significantly higher, meaning that all patients with atrophic gastritis in this study were positive for *H. pylori* (P = 0.01).

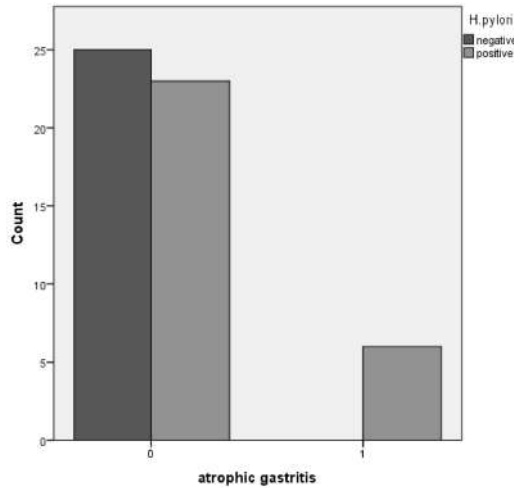


Figure 1. Frequency of helicobacter pylori infection in patients with and without atrophic gastritis

The relationship between atrophic gastritis and intestinal metaplasia in a way that in each 6 patients with atrophic gastritis were intestinal metaplasia, but in 48 other patients who had not atrophic gastritis, there were only 11 cases of intestinal metaplasia (P <0.001).

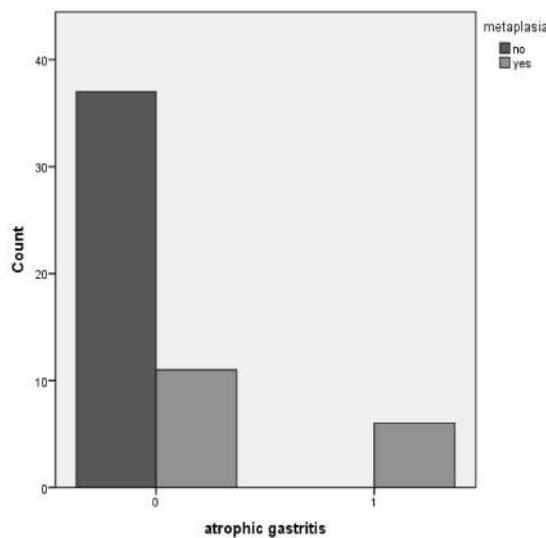


Figure 2. Frequency of intestinal metaplasia in patients with and without atrophic gastritis

DISCUSSION

According to various studies, two-thirds of people worldwide are suffering from *H. pylori* infection induced chronic gastritis [3]. Although the epithelium of the gastrointestinal tract, particularly the stomach epithelial cells act firmly against micro-organisms by several mechanisms such as integrity, high turnover, autophagia and so on. *Helicobacter pylori* overcome this barrier with different ways and colonized in stomach epithelium. This chronic infection can process a chronic inflammatory response that can lead to ulcer or neoplasia [4].

The end result of *H. pylori* induced gastritis is atrophic gastritis which is a precursor of malignancy. If atrophic changes of gastric mucosa is associated with replacement of connective tissue and structure of glands naturally preserved, non-metaplastic atrophic gastritis occurred. But if mucous glands structure

changes, metaplastic atrophic gastritis has been created. It should be noted that the treatment and eradication of *H. pylori* infection can cause reversible effect on these changes [5]. Other studies have also shown that atrophic gastritis in setting of *H. pylori* infection, increases the risk of gastric cancer and elevated gastrin level due to H.P infection can cause carcinoid tumor [6,7].

In this study, we also found that atrophic gastritis was significantly associated with *H. pylori* infection ($p=0.01$) and in all patients with atrophic gastritis, intestinal metaplasia was found ($P < 0.001$).

Although presence of relationship between *Helicobacter pylori* infection and pre-malignancy and malignancy changes, is controversial. *Helicobacter pylori* infection is considered as a risk factor in the development of atrophic gastritis, gastric metaplasia and cancer; But there is still doubt that chronic infection with *Helicobacter pylori* and induced gastritis, lonely, is capable to lead to metaplasia in the setting of atrophic gastritis. For example, in one study in Africa, the prevalence of intestinal metaplasia and gastric cancer was significantly low; In contrast the prevalence of *H. pylori* in the same area was high. In another study that was conducted in Yemen, despite a 97% prevalence of *H. pylori* infection, the prevalence of intestinal metaplasia was about 4% [8]. Our study also showed that association of *H. pylori* infection and intestinal meaplasia was not considerable ($P = 0.09$).

Mere association between *H. pylori* infection and gastric cancer is studied in many surveys. For example, in one study, *H. pylori* were observed significantly in the gastric mucosa of patients with malignancy and premalignant changes. Eurogast study showed that *H. pylori* infection increases 6 fold the risk of malignancy [9]. Two Cohort and Case-Control meta-analysis also showed that *H. pylori* infection increases 2 fold the risk of gastric cancer. In a large prospective study was conducted on 1,526 Japanese patients, 1246 patients were positive for *H. pylori* infection, in follow-up, after about 7.8 years(average), 36 patients with *H. pylori* infection progressed to gastric cancer, whereas none of the patients without infection, had cancer [9]. In this study we found that the positive association between *Helicobacter pylori* infection, gastritis, chronic atrophic gastritis and intestinal metaplasia is a considerable cycle.

The exact prevalence of asymptomatic atrophic gastritis in patients with this disorder in the course of creation, is not clear, however, it would be equal with its etiology (*H. pylori* infection and autoimmune gastritis) [10]. In our study, 11.1% patients had atrophic gastritis which this can be due to differences in the various stages of inflammation. Atrophic gastritis is manifested after many years, because the process of creation after *H. pylori* infection is long. So, diagnosed in older ages, usually at the age of 50 years old. (10) In our study, the mean age of patients with atrophic gastritis was more than 50 years (54.6 ± 9.9).

The prevalence of *H. pylori* infection in America is, 20% of patients younger than 40 years and 50% of patients older than 60 years [10] we revealed, patients with *H. pylori* infection is approximately 60 years old (56.2 ± 9.7). Although, this infection is more in Asians, Hispanic and African-Americans. As mentioned above, *Helicobacter pylori* infection affects approximately 50% of people in the world so chronic gastritis would have high prevalence. In our study 90% of patients had chronic gastritis. Also 53.7% of the patients were positive for *H. pylori* infection. This result is equal with international findings.

EI-zimaity and colleagues in a study that was conducted in 2006 showed that *H. pylori* infection is the main cause of chronic gastritis [11]. In our study, there was considerable difference between the positivity of *Helicobacter pylori* infection and chronic gastritis ($p=0.05$). Atrophic gastritis and *Helicobacter pylori* infection, affects both sexes equally (10). In this study, the association between sexes and development of atrophic gastritis and *Helicobacter pylori* infection was not significant.

CONCLUSION

The results showed that *H. pylori* infection has significant relationship with chronic gastritis and atrophic gastritis which are premalignant changes. Atrophic gastritis and intestinal metaplasia have also significant relationship. The eradication of *Helicobacter pylori* infection and lifestyle modification can prevent the progression to malignancy.

REFERENCES

1. Dixon MF, Genta RM, Yardley JH, Correa P. (1994). Classification and grading of gastritis. The updated Sydney System. International Workshop on the Histopathology of Gastritis, Houston. *Am J Surg Pathol* 1996; 20:1161.
2. Strickland RG, Mackay IR. (1973). A reappraisal of the nature and significance of chronic atrophic gastritis. *Am J Dig Dis*; 18:426.
3. Pamela J Jensen, Mark Feldman, (2011). J Thomas LaMont, Shilpa Grover, MD. Acute and chronic gastritis due to *Helicobacter pylori*. Up to date 21.2. Oct 6.
4. Shatha Alzahrani, Taslima T Lina, Jazmin Gonzalez, Irina V Pinchuk, Ellen J Beswick, Victor E Reyes. (2014). Effect of *Helicobacter pylori* on gastric epithelial cells. *World J Gastroenterol* September 28; 20(36): 12767-12780
5. Li ju choi. (2014). Endoscopic gastric cancer screening and surveillance in high risk groups. *Clin Endoscopic*. Nov;47(6):497-503.

6. Yanaoka K, Oka M, Ohata H, et al. (2009). Eradication of *Helicobacter pylori* prevents cancer development in subjects with mild gastric atrophy identified by serum pepsinogen levels. *IntJ Cancer*. 125(11):2697-703
7. Vannella L, Lahner E, Annibale B. Risk for gastric neoplasias in patients with chronic atrophic gastritis: a critical reappraisal. *World J Gastroenterol*. Mar 28 2012;18(12):1279-85
8. Pamela J Jensen, Mark Feldman, J Thomas LaMont, Shilpa Grover, MD. Metaplastic (chronic) atrophic gastritis .Up to date 21.2. Aug 9, 2012.
9. Sheila E crowe, Mark Feldman, Shilpa Grover. Association between helicobacter pylori infection and gastrointestinal malignancy. Up to date 21.2. Feb 13, 2013
10. Weck MN, Gao L, Brenner H. (2009). *Helicobacter pylori* infection and chronic atrophic gastritis: associations according to severity of disease. *Epidemiology*. 20(4):569-74.
11. El-Zimaity HMT. (2006). Gastric atrophy, diagnosing and staging. *World J Gastroenterol*; 12(36): 5757-5762

Copyright: © 2016 Society of Education. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.