A Comparison of the Endoscopic and Histopathological Findings of Upper Gastrointestinal Mucosa with Helicobacter pylori infection

Gontar A. Siregar, Sumondang Pardede ***

*Division of Gastroentero-Hepatology, Department of Internal Medicine,
Faculty of Medicine, University of North Sumatera/H. Adam Malik Medan Central General Hospital
**Department of Pathologic Anatomy, Department of Internal Medicine, Faculty of Medicine, University of North Sumatera/H. Adam Malik Medan Central General Hospital

ABSTRACT

Helicobacter pylori infects the gastric mucosa or the duodenal wall undergoing gastric meteplasia, and is found in nearly 100% of chronic gastritis and duodenal ulcers. Helicobacter pylori produces urease that converts urea into ammonia, which will protect the organism from the acidic environment and will cause further damage to the gastrointestinal mucosa.

Helicobacter pylori can be detected through histopathological evaluation, macroscopic endoscopy, serologic test, urea breath test, biopsy urease test, culture and stool analysis. Histiopathologically, Helicobacter pylori infection demonstrates neutrophil infiltration into the gastric mucosa, classified as focal infiltration.

We conducted a prospective study of 50 chronic dyspeptic patients. We took their history, performed physical examinations, gastroscopy with judgement for macroscopic endoscopic appearance, histopathology from biopsy specimens, and the CLO test.

There were 50 chronic dyspeptic patients in the study, with an age ranging from 23-81 years, and a mean age 49±12 year. Most of them were male (33 cases). There were 17 female cases.

From the CLO test, there were 30 cases with CLO (+) and 20 cases CLO (-). From the 30 cases with CLO (+), 22 were male and 8 female. Gastroscopy revealed 25 cases of gastric ulcer, 7 duodenal ulcer, 2 gastric cancer, 15 gastritis, and 30 gastropathy. A gastroscopic appearance of chronic dyspepsia with positive Helicobacter pylori were found mostly in gastric ulcer (18 cases), followed by duodenal ulcer (6 cases), gastritis (5 cases) and one case of gastric cancer. A gastroscopic appearance of chronic dyspepsia with negative Helicobacter pylori were found mostly in gastritis 10 cases, while the remaining in gastric ulcer (7 cases), gastric cancer (2 cases), and a case of duodenal ulcer. Gastroscopy revealed 15 cases of gastritis. From the 5 gastritis cases with CLO (+), 3 cases had lesions located at the antrum and 2 cases at the corpus, while from the remaining 10 cases of gastritis with CLO (-), 8 cases had lesions located at the antrum, and 2 at the corpus. The time to colonization was shortest in duodenal ulcer (grade IV), followed by gastric ulcer (grade II) and gastritis (grade I) in CLO (-) examination. From cases of gastritis with CLO (+), 4 were moderate cases and one case severe, while from cases of gastritis with CLO (-) there were 7 mild cases, and 3 moderate cases with no severe case found.

***Keywords**: Endoscopic findings, histopathology, Helicobacter pylori.*

INTRODUCTION

Helicobacter pylori are spiral-shaped, Gram-negative bacteria that colonize the surface epithelium of the stomach, under the mucosal membrane. The organism produces enzymes that degrade mucous, and possesses urease activity. *H. pylori* infects gastric mucosa or duodenal wall that has undergone gastric metaplasia. *H. pylori* is found in almost 100% cases of chronic gastritis and duodenal ulcer. Yet not all infections of *H. pylori* cause gastroduodenal diseases; some cases of positive *H. pylori* are asymptomatic.[1,2]

Since its finding in 1983, *Helicobacter pylori* has been classified as a new bacterial pathogen. It is part of the etio-pathogenesis of different kinds of upper digestive tract diseases, such as antric gastritis, chronic gastritis, peptic ulcer, and gastric cancer. References stated that an agreement was made to eradicate *H. pylori* as part of the management.[1, 3]

Atrophic gastritis and intestinal metaplasia have been accepted as pre-cancerous lesions of gastric cancer, excluding cancer of the gastroesophageal junction and lower esophagus. Various endoscopic reports showed that 80% gastric cancer was related to gastritis caused by *Helicobacter pylori*.[4]

The pathogenesis of *Helicobacter pylori* infection is the destruction of upper digestive tract mucosa. The organism produces large quantities of urease enzyme that converts urea to ammonia; this condition protects the organism from the acid environment and in turn causes destruction of mucosal cells of the digestive tract. The presence of *H. pylori* and its colonization rate correlates with the inflammatory cell infiltration rate, involving polymorphonuclear cells and mononuclear cells.[1,3]

Gastritis is classified into three groups according to macroscopic appearance (seen through endoscopy):

- Erosive and hemorrhagic gastritis, occurring frequently in severely ill patients (stress ulcer) and in patients that take NSAIDs.
- Non-erosive and non-hemorrhagic gastritis, the major cause being *H. pylori*. Also commonly called non-specific gastritis, since the clinical state and the etiology could not be determined through histopathologic evaluation.
- Specific gastritis, showing characteristic histopathologic and endoscopic findings similar to granulomatous gastritis.

According to its location, non-erosive and non-hemorrhagic gastritis can be classified into four groups, as follows:[4,6]

1. The antrum type, occurring more frequently in *H. pylori* infection.
2. The fundus type, infrequent in *H. pylori* infection.
3. The pan-gastritis type, involving fundus and antrum.
4. The multifocal atrophic type.

The histopathological characteristic of *H. pylori* is neutrophil infiltration of the gastric mucosa, known as the focal infiltrative type. The epithelial changes are usually complex, resembling most gastritis associated with *H. pylori* infection.[7]

To diagnose *H. pylori* infection, some methods of detection that are used are histopathological examination, macroscopic endoscopy, serologic test, urea breath test, urease biopsy test, culture, and fecal analysis.[4] Rapid urea test for *H. pylori* has a sensitivity rate of 80-90%. Histopathological examination of stomach biopsy specimens is recommended as a diagnostic support if the urea test is negative.[7, 8]

In this paper, we report endoscopic and histopathological findings from *H. pylori*-infected gastric mucosa.

MATERIALS AND METHOD

A prospective study was performed in fifty patients suffering from chronic dyspepsia; history taking and physical examination were performed on all patients. All of the patients had dyspepsia for more than two months, and one month prior to the research all had discontinued the use of antibiotics, proton-pump inhibitors, or H_2 blockers. Patients were excluded if they suffered from chronic liver disease, liver cirrhosis, and dyspepsia caused by other organic diseases.

Gastroscopy, histopathology from biopsy, and CLO tests was performed on all patients.

For the histopathological examinations, hematoxyllin and eosin stainings were used. Using the stainings, digestive tract abnormalities and pre-malignant conditions can be identified with a sensitivity rate of more than 90% and a specificity rate of more than 90%.

The grading of the colonization rate of *H. pylori* was performed using the CLO test, and was divided in four grades, i.e.:

Grade 4 : (+) 20 minutes
Grade 3 : (+) 1 hours
Grade 2 : (+) 2 hours
Grade 1 : (+) 24 hours
Grade 0 : (-)

All the participants were asked for their informed consent. A statistical analysis and a descriptive report were then made.

RESULTS

There were fifty patients with chronic dyspepsia, with an age ranging from 23-81 years old, with a mean age of 49±12 years old. Thirty-three cases were male and 17 cases were female. (Table 1)

Table 1. Demographic Data of Dyspeptic Patients

N	50
Age range (years)	23 - 81
Mean age	49 ± 12
Male	33
Female	17

CLO tests were performed on all fifty cases of chronic dyspepsia, with the results being 30 of CLO (+) and 20 cases of CLO (-). Out of the 30 cases of CLO (+), most were male (22 cases), and the remaining 8 cases were female. (Table 2)

Table 2. CLO Test Results

	CLO (-)	CLO (+)
N	20	30
Age range (years)	23 - 81	26 - 71
Mean age	48.6 ± 13	49.3 ± 11
Male	11	22
Female	9	18

Gastroscopy was performed on all cases, with the following results: 25 cases of gastric ulcer, 7 cases of duodenal ulcer, 2 cases of gastric cancer, 15 cases of gastritis, and 30 cases of gastropathy.

The most common gastroscopic finding of chronic dyspepsia with *H. pylori* (+) was gastric ulcer (18 cases), followed by six cases of duodenal ulcer, five cases of gastritis, and one case of gastric cancer. Meanwhile, the most common gastroscopic finding of chronic dyspepsia with *H. pylori* (-) were gastritis (10 cases), followed by 7 cases of gastric ulcer, 2 cases of gastric cancer, and 1 case of duodenal ulcer. (Table 3)

The gastroscopy results showed fifteen cases of gastritis. In five cases of CLO (+) gastritis, the location of the lesions were on the antrum (3 cases) and on the fundus (2 cases), while in the 10 cases of CLO (-) gastritis, 8 cases were on the antrum and 2 cases were on the fundus. (Table 4)

Table 3. Gastroscopy and CLO Test Results

	CLO (-)	CLO (+)
Gastric ulcer	7	18
Duodenal ulcer	1	6
Gastritis	10	5
Gastic cancer	2	1
Gastropathy	13	17

Table 4. Distribution of gastritis in gastroscopy and CLO test

	CLO (+)	CLO (-)
Gastritis	5	10
Antrum	3	8
Fundus	2	2

The correlation of *H. pylori* colonization time and the presence of gastritis, duodenal ulcer, or gastric ulcer was as follows: the shortest colonization time was found in duodenal ulcer (grade IV), and then gastric ulcer (grade II), and gastritis (grade I). (Figure 1)

The correlation between gastritis and the presence of *H. pylori* infection was as follows: in CLO (+) gastritis, there were 4 cases with a moderate rate of infection and 1 case with a severe rate of infection, where as in CLO (-) gastritis, there were 7 cases with a mild level of infection and 3 cases with a moderate level, and no case with a severe level of infection. (Figure 2)

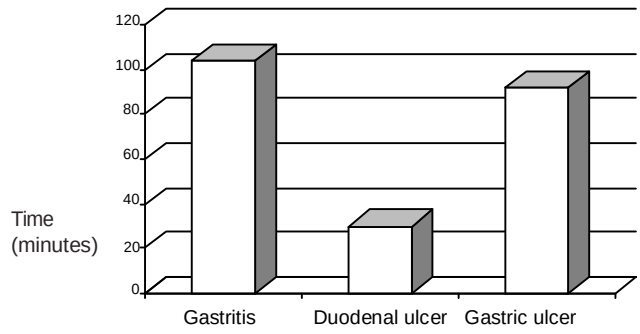

Figure 1. The correlation between *H. pylori* colonization time and gastritis, duodenal ulcer, or gastric ulcer

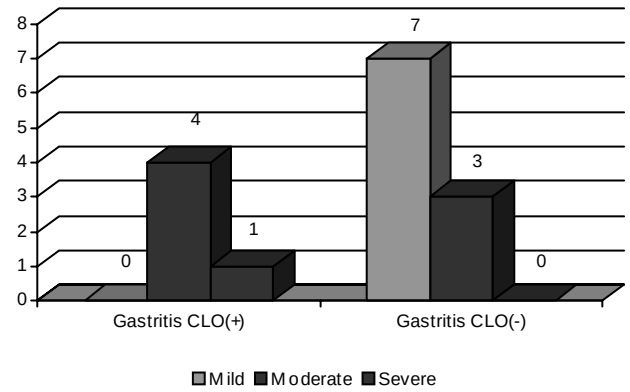

Figure 2. The correlation between the severity of gastritis and the presence of *H. pylori* infection

DISCUSSION

Fifty patients with chronic dyspepsia were included in the research, with an age ranging from 23 to 81 years old, mean of age 49±12 years old. Most were male (33 cases). There were 17 female cases. CLO test results were as follows: thirty cases with CLO (+) and twenty cases with CLO (-). Out of the thirty cases with CLO (+), 22 cases were male and 8 cases were female.

The data showed that 60% of the chronic dyspepsia patients were Helicobacter (+). Other research showed that *H. pylori* infections were found in more than 90% of duodenal ulcer patients, 60-70% of gastric ulcer, and 60-90% of those with chronic gastritis. [8]

Out of the 30 cases with CLO (+), 5 (16.7%) suffered from gastritis; whereas out of the 20 cases with CLO (-), 10 (50%) were found with gastritis. Literature mentions that *H. pylori* is the cause of gastritis in more than 80% of cases.[8] A research by Duo Kim et al showed that 85% gastritis were *H. pylori* (+). [9]

The main symptoms of CLO (+) dyspepsia were epigastric pain, feeling of fullness, and nausea, in that order, while in CLO(-) dyspepsia, patients mostly had nausea, some had epigastric pain and feeling of fullness. Hence, there are no considerable difference in the main symptoms of those with *H. pylori* (+) chronic dyspepsia and *H. pylori* (-) chronic dyspepsia.

In this research, the gastroscopy results were as follows: 25 cases of gastric ulcer, 7 cases of duodenal ulcer, 2 cases of gastric cancer, 15 cases of gastritis, and 30 cases of gastropathy. The most common gastroscopic finding in chronic dyspepsia with *H. pylori* (+) were gastric ulcer (18 cases), followed by s6 cases of duodenal ulcer, 5 cases of gastritis, and 1 case of gastric cancer. On the other hand, most gastroscopic findings in patients with chronic dyspepsia with *H. pylori* (-) were gastritis (10 cases), followed by 7 cases of gastric ulcer, 2 cases of gastric cancer, and 1 case of duodenal ulcer.

Fifty percent of the dyspepsia cases were classified as non-ulcer dyspepsia.[10] Tsuji et al reported there was no difference in the characteristics of *H. pylori* (+) gastric ulcer and *H. pylori* (-) gastric ulcer.[11]

There are four types of gastritis, the antrum type, most frequently caused by *H. pylori* infection, the fundus type, the pan-gastritis type, and the multifocal atrophic type.[6]

In this research, gastroscopy revealed 15 cases of gastritis. Out of the five cases of gastritis with CLO (+), in 3 cases the lesions were found on the antrum, and in 2 cases the lesions were located on the fundus. Meanwhile, out of 10 cases of gastritis with CLO (-), in 8 cases the lesions were found on the antrum and in 2 cases the lesions were located on the fundus.

The correlation between the colonization time of *H. pylori* and gastritis, duodenal ulcer, and gastric ulcer were as follows: the shortest colonization time was found in duodenal ulcer (grade IV), and then gastric ulcer (grade II), and gastritis (grade I).

The correlation between gastritis and the presence of *H. pylori* infection was as follows: in CLO (+) gastritis, there were 4 cases with a moderate level of infection and 1 case with a severe level of infection, whereas in CLO(-) gastritis there were 7 cases with a mild level of infection, 3 cases with a moderate level, and no case with a severe level of infection.

CONCLUSION

There was more gastritis located in the antrum than in the corpus. Most cases of gastric ulcer were accompanied by *H. pylori* infection. Epigastric pain, nausea, and the feeling of fullness were the most dominant symptoms. Gastritis was not related to the density of *H. pylori*. The colonization of *H. pylori* was higher in gastric ulcer than in duodenal ulcer or gastritis.

REFERENCES

1. Dharmika Djojoningrat. Terapi Mutakhir Helicobacter pylori dalam Naskah Lengkap Penyakit Dalam PIT 2000, Bagian Ilmu penyakit Dalam FK-UI, Jakarta: pusatinformasi dan penerbitan;2000.p. 91-5
2. Graham DY. Helicobacter pylori infection in the pathogenesis of duodenal ulcer and gastric cancer : a Model.Gastroenterology 1997 ;113:1983-91.
3. Chiba N, Wikinson JM, Hunt RH. Clarithromycin or amoxicillin dual and triple therapies. In : Hp eradication: a meta analysis. Gut 3 1995;(12):31

4. Sipponen P, Marshall BJ. Gastritis dan Gastric Cancer dalam Jurnal Gastroenterology Clinic, 29. (3) Sep. 2000: 1-12
5. Robert E.M., Weinstein W.M : Helicobacter pylori-associated pathology Gastroenterol Clin NA,1993; 22: 59-72
6. Soemohardjo S. Helicobacter pilori dan Penyakit gastroduodenal, edisi II. Unit Peny Dalam RSU. Mataram.1997.p 1-38.
7. Warren JR. Gastric Pathology Associated with Helicobacter Pylori dalam Jurnal Gastroenterology Clinic,29.(3) 2000.p. 1-23
8. Kaniawati M. Helicobacter pylori. Forum Diagnosticum. Lab Prodia No.2/1995. 1-12.
9. Kim DJ, Baek JY. The Comparison of Histologic Gastritis in Patients with Duodenal Ulcer, Chronic Gastritic, Gastric Ulcer and Gastric Cancer. Yonsei Med J Vol. 40. 1999; 14-9.
10. Talley NJ, Vakil N,Ballard ED, Fennerty MB. Absence of Benefit of Eradicating Helicobacter pylori in patients with non ulcer dyspepsia. New Eng. J. 1999 ; 1106-11.
11. Tsuji H. Kohli Y. Fukumitsu S, Morita K, Kaneko H. et all. Helicobacter pylori-negative gastric and duodenal ulcers. J Gatroenterologi 1999;34:455-60.