HELICOBACTER PYLORI IN DYSPEPTIC PATIENTS IN DEPARTMENT OF INTERNAL MEDICINE M. DJAMIL HOSPITAL PADANG

Nasrul Zubir, Dona Elfira, Julius

Department of Internal Medicine, M. Djamil Hospital, Padang

ABSTRACT

Objective : *Helicobacter pylori infection is probably the most common infection caused by pathogenic bacteria and affects more than half of the world population. These bacteria caused many gastroduodenal pathology such as chronic active gastritis, peptic ulcer and gastric malignancy. This study investigated Helicobacter pylori infections in dyspeptic patients.*

Materials & Methods: *Forty dyspeptic patients were examined using upper gastrointestinal endoscopy and biopsy for histopathologic examination.*

Results*: The symptom of the dyspeptic patients were epigastric tenderness (100%). Endoscopic abnormalities were found in 39 (97.5%) cases. The gastric abnormalities were chronic antral gastritis in 24 (60%), esophagitis and gastritis in 7 (17.5%), and gastric ulcer in 3 (7.5%). Twenty four out of 40 patients had anti-Helicobacter pylori positive (60%) and histopathologic examination were positive 18 (45%). The histopathologic examination found atrophy in 18 (45%), MN in 18 (45%), PMN in 18 (45%), and metaplastic in 4 (10%).*

Conclusion *: The prevalence of Helicobacter pylori in dyspeptic patients were 60% (serology) and 45% (histopathology).*

Key words*: Helicobacter pylori, dyspeptic, serology, histopathology*

INTRODUCTION

Dyspepsia is a term frequently used by patients to describe a variety of symptoms such as a feeling of excessive gas, epigastric discomfort, and nausea, even up to vomiting.

The pathogenesis of dyspepsia is still controversial, whether it is caused by gastric acid, inflammation of gastric mucosa, or the *Helicobacter pylori* infection.

Camilesi (1995), assumed that a disturbance in the central nervous systems, autonomic systems and local factors in dyspeptic patients cause dysmotility of the stomach.

Dyspepsia could be classified clinically and pathophysiologically into functional dyspepsia and organic dyspepsia.

Chronic gastritis, stomach ulcer, duodenal ulcer, gastro esophageal reflux and others also could cause dyspepsia.

Dyspepsia in *Helicobacter pylori* is caused by the enzyme and several toxins secreted by the bacteria and cause inflammation of the gastric mucosa (Dunn BE,1993).

The presence of *Helicobacter pylori* can be detected with many methods such as the CLO test, *Helicobacter pylori* bacteria culture, histopathologic examination, urea breath test, and anti-HP serology marker examination.

MATERIAL AND METHOD

This prospective-cross sectional study was conducted on approximately 40 patients with dyspepsia as a major complaint in the Department of Internal Medicine M.Djamil Hospital Padang and based on the upper gastrointestinal endoscopic examination, histopathologic examination and anti-HP serology marker examination.

RESULTS

There were 40 patients with dyspepsia as a major complaint consisted of 18 males (45%) and 22 females (55%) with ratio of 1:1.2

The average age is 44.6 years old with age range from 21 years old up to 70 years old.

From table 1, we can find that the most common major

Table 1. The distribution of dyspeptic patients based on major complaints

No	Major complaints	Major complaints	Major complaints percentage
1.	Epigastric discomfort	40	100.0
2.	Heart burn	17	42.5
3.	Regurgitation	13	32.5
4.	Abdominal distention	39	97.5
5.	Stomach fullness	34	85.0
6.	Bloating	23	57.5
7.	Eructation	31	77.5
8.	Anorexia	18	45.0
9.	Nausea	34	85.0
10.	Vomit	4	10.0

Table 2. Endoscopic findings from 40 dyspeptic patients and Anti-HP (+)

Endoscopic results	N	Percentage (%)	Anti-HP(+)
Antral chronic gastritis (ACG)	24	60.0	14 (58.3%)
Esophagitis + BRG +ACG	1	0.3	-
BRG + ACG	1	0.3	-
Pan gastritis	2	0.5	2 (100%)
Stomach ulcer + ACG	3	7.5	2 (66.7%)
Esophagitis and gastritis	7	17.5	5 (71.4%)
Duodenum ulcer + BRG	1	0.3	1 (100%)
Normal	1	0.3	-
Total	40		24

Note: ACG = chronic antral gastritis, BRG= bile reflux gastritis

complaint of dyspeptic patients was epigastric discomfort in 40 patients (100%), followed with abdominal distention in 39 patients (97.5%) and stomach fullness and nausea each in 34 patients (85%).

From table 2, we can see that chronic antral gastritis in 24 patients (60.0%) was the most common cause followed by esophagitis and gastritis in 7 patients (17.5%).

The anti-HP was positive in 14 of 24 patients with chronic antral gastritis (58.3%) and positive in 5 of the 7 patients with esophagitis and gastritis (71.4%).

As from the 40 dyspeptic patients we could found 24 patients with anti-HP(+) (60%).

We also found atrophy of the gastric mucosa in 18 patients (45%), a spread of mononuclear cells found in 17 people (42.5%), polynuclear cells in 18 (45%), metaplasia in 4 (20%), carcinoid in 1(2.5%).

DISCUSSION

In this study from 40 patients with dyspepsia as a major complaint, was first stated with epigastric discomfort (100%), abdominal distention (97.5%) and that similar to the report of Pajeres and Santander (1996) and Velanovich (1996).

The average age of the dyspeptic patients in this study was 44.6 years old, and this was much older compared to the study by Purbayu H (1996) that concluded the average age for dyspeptic patients was approximately 36.9 years old. Though Vyas and colleagues (1994) reported older age, of 57 years old, which was similar to Pajares and Santander (1996) finding of 51.4 years and Cutler and colleagues (1995) of approximately 53.7 years old.

From the 40 dyspeptic patients who underwent endoscopic examination, we found abnormalities that most probably caused by chronic antral gastritis in 24 patient (60%), followed by esophagitis and gastritis in 7 patients (17.5%) and gastric ulcer in 3 patients (7.5%).

Malfertheiner P (1994) stated that *Helicobacter pylori* infection would increase the risk of chronic gastritis becoming ulcer 15 times. It could probably also be a predisposing factor for the development of gastric cancer.

Nasrul Zubir and Julius (1993) found *Helicobacter pylori* in 84.1% of chronic gastritis patients.

From the serologic examination of the dyspeptic patients, it was found that from the total 40 patients 24 (60%) of them had anti-HP (+).

From the 24 patients with chronic antral gastritis, there were 14 patients (58.3%) with anti-HP (+); from 7 patients with esophagitis and gastritis there were 5 patients (71.4%) had anti-HP(+).

The serologic examination is more important for

Table 3. The ages group classification of dyspeptic patients distribution with HP(+) based on serologic marker examination

Age group	Males			Females			Total		
	N	Ig G(+)	%	N	Ig G(+)	%	N	Ig G(+)	%
21-30	4	-	-	4	2	50.0	8	2	25.0
31-40	3	1	33.3	3	2	66.7	6	3	50.0
41-50	8	8	100	6	5	83.3	14	13	92.9
51-60	2	1	50.0	4	1	25.0	6	2	33.3
> 60	1	-	-	5	4	80.0	6	4	66.7
Total	18	10		22	14		40	24	

Table 4. Endoscopic and histopathologic appearance from 40 dyspeptic patients with *Helicobacter pylori* positive.

Endoscopic results	N	HP (+)	Atrophy	MN	PMN	Meta	Edema	Ca	Hyper
1. ACG	24	11(45.8)	12(50)	10(41.7)	9(45)	2(8.3)		1(4.2)	
2. Esophagitis + BRG + ACG	1	-	-	-	-	-	-	-	-
3. BRG + ACG	1	-	1(100)	-	-	-	-	-	-
4. Pan gastritis	2	2(100)	-	2(100)	2(100)	-	-	-	1(50)
5. Stomach ulcer + ACG	3	2(67)	1(33)	2(67)	2(67)	-	-	-	-
6. Esofagitis + gastritis	7	2(29)	3(42)	2(29)	4(57)	1(14)	-	-	-
7. Duodenum ulcer + BRG	1	-	-	-	-	-	-	-	-
8. Normal	1	1(100)	1(100)	1(100)	1(100)	1(100)	-	-	-
Total	40	18	18	17	18	4	1	1	1

screening for dyspeptic patients, because it is more important than screening for dyspeptic patients for *Helicobacter pylori* infections and it is useful to evaluating the progress and results of the therapy.

The histopathologic examination of the antrum showed gastric atrophy in 12 patients (50 %), which would be a premalignant mucosa.

In general, we found mucous atrophy in 18 patient (45%), the spread of mononuclear inflammation cells in 17 patients (42.5%), polimorphonuclear in approximately 18 (45%), metaplasia in 4 (20%) and carcinoid in 1 patient (2.5%). This finding is similar with others report that had been performed by other previous study (Murtono and Suripto,1998).

From the histopathologic results we could conclude that if the *Helicobacter pylori* enters the gastric mucosa therefore inflammation cells such as monocytes, neutrophyls, and macrophages will try to overcome the infection. The antigen of these bacteria will enter to the surface epithel and will cause inflammation that will be seen histopathologically as chronic superficial gastritis

The antigen of the bacteria that has reached the epithelial gland cells would cause inflammation of the gland cells, degeneration and even atrophy. That's why it is called chronic atrophic gastritis (Wyatt,1989).

CONCLUSIONS

1. The prevalence of *Helicobacter pylori* in dyspeptic patient was 60% from the serologic examination and 45% by histopathology.
2. The most prominent complaint of the dyspeptic patient was epigastric discomfort and abdominal distension.
3. From the endoscopic examination, we found the image of chronic antral gastritis in 60%, esophagitis and gastritis in 17.5% and stomach ulcer in 3%.
4. There needs to be further research with larger samples.

REFERENCES

1. Dunn BE. Pathogenic mechanisms of *Helicobacter pylori*. Gastroenterol Clin Nourth America 1993; 22 (1): 43-57
2. Pajares JM, Santander C. Prevalence of *Helicobacter pylori* infection associated histologyc chronic gastritis in Spanish population. J Gastroenterol 1996; 31:1-5
3. Purbayu H. Infeksi *Helicobacter pylori* pada pasien dispepsia di Unit Endoskopi Gastoenterologi RSUD dr.Sutomo Surabaya. Acta Medica Indonesiana 1996; (2): 359-69.
4. Vyas SK. Preendoscopy screening using sero diagnosis of *Helicobacter pylori* infection. Euro J Gastroenterol Hepatol 1994; 96: 783-5.
5. Cutler AF. Accuracy of invasive and noninvasive test diagnose *Helicobacter pylori*. Infection J Gastroenterol 1995; 109: 136-41.
6. Malfertheiner P,Pieramico O.*Helicobacter pylori*. In: Gustavson ed.The stomach. Tokyo: Churcil Living Stone, 1992: 297-311.
7. Zubir N, Julius. *Helicobacter pylori* on chronic gastritis. KOPAPDI 1993, Xxi, 2: 324-329.
8. Murtono, Suripto. Histopathology images on gastritis with or without a *Helicobacter pylori*. Medika 1998; XXIV:7: 448-451.
9. Wyatt JI, Gray SF. Detection of *Campylobacter pylori* by histology. In: *Campylobacter pylori* and gastroduodenal disease. BJ. Rathbone,1989: 63-68.