HISTOPATHOLOGICAL PATTERN OF GASTRIC BIOPSIES OF HELICOBACTER PYLORI POSITIVE PATIENTS IN SARDJITO GENERAL HOSPITAL, YOGYAKARTA

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ABSTRACT

Objective: To determine the gastric histopathological types of H. pylori positive patients.

Materials and Methods: Study design was prospective study. Consecutive patients who were suffering chronic dyspepsia underwent endoscopic examination between August 1998 and December 1999. The biopsy specimens were taken from the gastric antrum and corpus and sent to the pathologist for histopathological typing and H. pylori examinations. H. pylori were also confirmed with CLO and IgG-Helicobacter pylori test.

Results: There were 92 patients (48 male (M) and 44 female (F)) who underwent endoscopical gastric biopsies between August 1998 and December 1999. Fifty six (60.87%) patients suffered from chronic superficial gastritis, 11 (11.96%) from chronic atropic gastritis, 2 (2.17%) from chronic gastritis with metaplasia, 3 (3.27) from gastric ulcer, and 2 (2.17%) from gastric signet-ring cell carcinoma. Twenty one (22.8%) patients was found to be H. pylori positive based on histopathologic examination with CLO and IgG-H. pylori tests. Those were 5 (8.90%) patients with chronic superficial gastritis, 7 (63.63%) chronic atrophic gastritis, 3 (100%) gastric ulcer, 2 (100%) chronic gastritis with metaplasia, 1 (50%) signet-ring carcinoma. The age range of the H. pylori positive patients were between 16 and 76 years old.

Conclusion: There were twenty one (22.8%) H. pylori positive patients out of 92 endoscopied patients and a high percentage tendency of H. pylori positively in chronic atrophic gastritis, gastric ulcer, and chronic gastritis with metaplasia, although most of the patients had chronic superficial gastritis. Further study with larger sample is needed to get clearer picture of H. pylori distribution based on gastric histopathological types.

Key words: histopathological type, gastric biopsy, H. pylori, gastritis

INTRODUCTION

Helicobacter pylori is recognized as an important cause of gastric tissue injury and there is extensive evidence of the role of *H.pylori* in causing gastroduodenal pathology.¹

The prevalence of *H.pylori* vary among Indonesia's provinces; 19.04-86.1% in chronic gastritis and 71.4-100% in peptic ulcers.²

This bacterium is also correlated with the risk of gastric cancer.³ Chronic atrophic gastritis and its associated abnormality, intestinal metaplasia, which are both usually correlated with *H. pylori* infection, are the lesions most closely linked to an increased risk of gastric can-

cer.3,4

The aim of this study was to determine the distribution of the gastric histopathological types of *H.pylori* positive patients who were detected histopathologically, and also with CLO test and IgG-*H.pylori*.

MATERIAL AND METHOD

Study subjects were patients who were suffering chronic dyspepsia underwent endoscopic examination prospectively between August 1998 and December 1999 in Gastrohepatology Division of Internal Medicine Department of Sardjito General Hospital, Yogyakarta, In-

Tabel 1. Sex distribution of gastric histopathological types of patients who

underwent endoscopic examination

Histopathological type	Male n(%)	Female n(%)	Subtotal n(%)
Chronic superficial gastritis	29(60.42)	27(61.29)	56(60.87)
Chronic atrophic gastritis	5(10.42)	6(13.62)	11(11.96)
Chronic gastritis*	10(20.08)	8(18.16)	18(19.56)
Chronic gastritis with metaplasia	1(2.08)	1(2.27)	2(2.17)
Gastric ulcer	1(2.08)	2(4.54)	3(3.27)
Signet ring cell carcinoma	2(4.16)	0(0)	2(2.17)
Total	48	44	92

^{*}These were not interpreted into their chronic gastritis sub-groups

Table 2. Distribution of gastric histopathological type based on

H. pylori positive

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Histopathological type	n	H.pylori- positive	%
		n	
Chronic superficial gastritis	56	5	8.90
Chronic atrophic gastritis	11	7	63.63
Chronic gastritis	18	3	16.67
Chronic gastritis with metaplasia	2	2	100
Gastric ulcer	3	3	100
Signet ring cell carcinoma	2	1	50
Total	92	21	22.8

Table 3. Age distribution of *H.pylori* positive

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Age	n(%)	Female/male		
16 – 25	2(8.52)	1/1		
26-35	3(14.28)	3/0		
36-45	7(33.32)	1/6		
46-55	3(14.28)	2/1		
56-65	3(14.28)	1 /2		
66-75	2(8.52)	2/0		
76-85	1(4.76)	1/0		

donesia. Patients were chosen as subjects in a consecutive manner. Two gastric biopsies samples from the antrum and two from the corpus were taken (also from suspected lesions), and examined with H & E or Giemsa staining for histopathological type and *H. pylori* coexistence. We also confirmed the existence of *H. pylori* with CLO-test and IgG-*H.pylori* test (EIA). Patients with positive *H. pylori* findings in two of the three examinations were included in the study. Patients with history of *H. pylori* eradication, recent antibiotics treatment, renal insufficiency, liver cirrhosis, pregnancy woman, and proton pump inhibitor usage within one month prior to endoscopy were excluded from the study.

RESULTS

There were 92 patients (48 male and 44 female) who underwent endoscopic gastric biopsies between August 1998 and December 1999 (table 1).

Twenty one (22.8%) out of 92 patients were *H.pylori*-positive. Twelve patients were *H.pylori*-positive by histopathologic, CLO, and IgG-*H.pylori* examinations. Seven patients were *H. pylori*-positive with histopathologic and CLO-test examinations and only two patients were *H. pylori*-positive with CLO-test and serologic examinations. The distribution of the gastric histopathological types of these *H. pylori*-positive patients is shown in table 2.

The age range of those patients was between 16 and 76 years old, and the youngest patient was a 16 years old girl with gastric ulcer. Table 3 shows the age distribution of *H. pylori* positive where the positivity accumulated mostly in mid age.

DISCUSSION

There were 22.8% *H.pylori* positive patients from 92 endoscopied patients. The results of the study show that the gastric histopathological pattern of *H. pylori* infected patients were similar with other studies.^{5,6} Chronic atrophic gastritis had higher frequency of positive *H. pylori* finding compared to chronic superficial gastritis. *H. pylori* was found in all cases of gastric ulcer and patients with intestinal metaplastic changes.

Chronic atrophic gastritis, intestinal metaplasia, and dysplasia are recognized as the premalignant lesions of gastric cancer. ^{4,5,7} In our study, we did not find dysplastic lesions and only two cases with signet ring cell carcinomas. *H.pylori* was detected in one of these cancers. This case was Bormann type I cancer located in the greater curvature of the gastric corpus. The frequency of gastric cancer was very low in our hospital. There were only 14 new cases between 1993-1997 among 82 395 patients. This is parallel with the low frequency of *H. pylori* positive in our hospital.

H. pylori is usually correlated with the incidence of the intestinal type of gastric cancer. The correlation between *H. pylori* and diffuse type gastric cancer is still inconclusive. *H. pylori* chronic gastritis is characterized by the accumulation of oxidative DNA damage with mutagenic and carcinogenic potential and this process is mediated by free radicals. ⁹

One study showed that chronic *H.pylori* infection seems to be responsible for genomic instability in a subset of *H. pylori* positive chronic atrophic gastritis cases, the eradication of *H.pylori* infection can reverse inflammation and related atrophy, metaplasia, and genomic instability.¹⁰

In conclusion, most of the patients suffered from non ulcer dyspepsia and the frequency of *H. pylori* positivity was low in our hospital, and there was also high percentage tendency of *H. pylori* positivity in chronic atrophic gastritis, gastric ulcer, and chronic gastritis with metaplasia, although most of the patients had chronic superficial gastritis. This gastric histopathological pattern was similar to other studies but the frequency of gastric cancer was very low in our hospital.

REFERENCES

- Dunn, BE, Cohen H., and Blaser, M. Helicobacter pylori. Clin Microbiol Rev 1997;10:720-41.
- Simadibrata M, HardjodisastroD, Djoyoningrat D, Rani A, Manan C, Arlina IA. *Helicobacter pylori* dan penyakit gastroduodenal usulan penatalaksanaan di Indonesia. Maj Kedok Indon 1996; 46: 179-88.
- Fuchs CS, Mayer RJ. Gastric Carcinoma. N. Engl. J. Med. 1995; 333: 32-41
- Sozzi M, Valentini M, Figura N, De Paoli P, Tedeschi RM, Gloghini A,Serraino D, Poletti M, and Carbone A. Atrophic Gastritis and Intestinal Metaplasia in *Helicobacter pylori* Infection: The role of CagA status. *Am J Gastroenterol* 1998; 93: 375-9.
- Mihara M, Haruma K, Kamada T, Komoto K, Yoshihara M, Sumii K, KajiyamaG. The role of endoscopic findings for the diagnosis of *Helicobacter pylori* infection: Evaluation in a country with high prevalence of atrophic gastritis. Helicobacter 1999; 4: 40-8.
- Annibale B, Marignani, Azzoni C, D'Ambra G, Caruana, D'Adda T, Fave GD, Bordi C. Helicobacter 1997;2:57-64.
- Williams MP, Pounder RE. Helicobacter pylori: from the benign to the malignant. Am. J. Gastroenterol 1999; 94(Suppl):S11-6.
- 8. Eslick GD, Lim LLY, Byles JE, Xia HHX, Talley NJ. Association of *Helicobacter pylori* infection with gastric carcinoma: a meta-analysis.Am.J.Gastroenterol.1999; 94: 2373-9.
- Farinati F, Cardin R, Degan P, Rugge M, Di Mario F, Bonvicini P, Naccarato R. Oxidative DNA damage accumulation in gastric carcinogenesis. Gut 1998; 42:351-6.
- Nardone G, Staibano S, Rocco A, Mezza E, Armiento FPD, Insabato L, Coppola A, Salvatore G, Lucariello A, Figura N, De Rosa G, Budillon G. Effect of *Helicobacter pylori* infection and its eradication on cell proliferation, DNA status, and oncogene expression in patients with chronic gastritis. Gut 1999;44:789-99