

Incidence of Helicobacter pylori Infection and Gastric Cancer : an 8-year Hospital Based Study

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ABSTRACT

Aim: to observe the tendency of decreased prevalence of *H pylori* infection in a 14 year-period and observe the prevalence of intestinal metaplasia and gastric cancer

Methods: all patients who were diagnosed with dyspepsia and underwent esophagogastroduodenoscopy in Cikini hospital Jakarta from January 1998 until December 2005 were evaluated.

We evaluated the histopathologic result of *H pylori*, the presence of intestinal metaplasia and gastric cancer. Data was grouped for 1 year period of time and was presented descriptively.

Results: decreased prevalence of *H. Pylori* infection was found, from 12.8% in 1998, 12.4% in 1999, 14.7% in 2000, 9.6% in 2001, 11.9 % in 2002, 3.8% in 2003, 2.3% in 2004, 2.9% in 2005.

Intestinal metaplasia was 4.7% in 1998, 3.2% in 1999, 3.1% in 2000, 2.3 % in 2001, 7.6% in 2002, 8.3% in year 2003, 6.5% in 2004, 7.1% in 2005.

Prevalence of gastric cancer was 2.2% in 1998, 0.25% in 1999, 1.1% in 2000, 1.1% in 2001, 1.1% in 2002, 1.8% in 2003, 1.7% in 2004, 3.9% in 2005.

Conclusion: there was decreased prevalence of *H pylori* infection in 8 year-period but there was no decreased prevalence of intestinal metaplasia and gastric cancer found.

Key words: *Helicobacter pylori* infection, intestinal metaplasia, pre-cancer lesion, gastric cancer.

INTRODUCTION

Since the last few years, it has already been widely known that chronic *H pylori* infection may cause inflammatory infection of gastric mucosa that soon will develop peptic ulcer or gastric cancer and MALT lymphoma.¹⁻⁵ It is also known that gastric cancer is arising from chronic gastritis-mucosal atrophy-intestinal metaplasia.⁶

In 1994, a IARC (International Agency for Research in Cancer) working group had proved that *H pylori* infection was associated with the development of gastric cancer. That is the reason why *H pylori* is classified as a class I carcinogenic agent.⁷ Since then, experts around the world have recommended the importance of *H pylori* eradication.^{1,3,4,8} Various consensus were made including in Indonesia.⁸

Several arguments regarding the importance of *H pylori* eradication had been expressed although available data does not always indicate high prevalence of *H pylori* in gastric cancer.

To date, data regarding the *H pylori* infection and gastric cancer in Indonesia are still limited. This study is aimed at investigating the trends of prevalence of *H pylori* infection and intestinal metaplasia and gastric cancer in 14 year-period.

METHODS

This is a retrospective study. All patients with dyspepsia underwent gastroduodenoscopy and biopsy in Cikini hospital in 1998-2005. We collect histopathologic result from medical records. Biopsy was done as routine procedure. The samples were taken by endoscopy from pre-pyloric antrum and gastric corpus/angulus. They were put in bottle containing formalin 10% and then stained with H/E. If necessary, Giemsa staining in PA laboratory of FK-UKI/PGI Cikini hospital. Prevalence of *H pylori* infection, intestinal metaplasia and gastric cancer were classified into several groups.

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Data from 1 year period of time were presented descriptively.

RESULTS

In a period of 8 years in Cikini hospital 2903 patients underwent endoscopy. During this period there were 262 patients who had H pylori infection. Evidence of H pylori infection was based on histopathologic examination. It showed that prevalence of H pylori infection had decreased gradually. In 1998 there were 52 cases of 407 patients (12.8%) with H pylori infection, in 2001 there only 34 patients of 353 (9.6%) and 2005 there were only 2.9 patients. (Table 1)

Table 1. Prevalence Rate of Hp Infection

Year	Number of Patients	Number of Hp+	Prev. rate (%)
1998	407	52	12.8
1999	403	50	12.4
2000	359	53	14.7
2001	353	34	9.6
2002	352	42	11.9
2003	371	14	3.8
2004	351	8	2.3
2005	307	9	2.9

Hp +: Helicobacter pylori was positive based on histopathologic examination

In the same period, it also showed the prevalence of intestinal metaplasia. In 1998, there were 19 of 407 cases of intestinal metaplasia, 2001 there were 8 of 353 patients (2.3%), in 2005 there were 23 of 351 patients (6.5%). (Table 2)

Table 2. Incidence of Intestinal Metaplasia

Year	Number of Gastroscopy	Number of IM+	Incidence rate (%)
1998	407	19	4.7
1999	403	13	3.2
2000	359	11	3.1
2001	353	8	2.3
2002	352	27	7.6
2003	371	31	8.3
2004	351	23	6.5
2005	307	22	7.1

In this study, it showed that the incidence of gastric cancer in 1998 was 9 out of 407 patients (2.2%), in 2001 there were 4 patients out of 353 (1.1%) while in 2005, 12 out of 307 patients (3.9%). (Table 3)

The incidence of H pylori infection and intestinal metaplasia and gastric cancer can be seen in figure 1.

Table 3. Incidence of Stomach Cancer

Year	Number of Gastroscopy	Number of Hystopath +	Incidence rate (%)
1998	407	9	2.2
1999	403	1	0.25
2000	359	4	1.1
2001	353	4	1.1
2002	352	4	1.1
2003	371	7	1.8
2004	351	6	1.8
2005	307	12	3.9

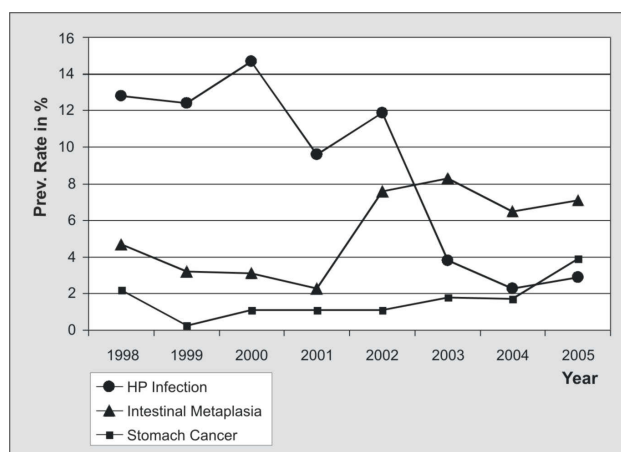


Figure 1. Prevalence rate of H pylori infection and incidence rate of intestinal metaplasia and gastric cancer

DISCUSSION

In this study, we found the decreased incidence of H pylori infection from 12.8% (1998) to 2.9% (2005). On the other hand, the incidence of intestinal metaplasia varies from year to year but tends to increase from 4.7% (1998) to 7.4% (2005). The incidence of gastric cancer was very low, only 1.2% per year. However, in 2005 there were more cases of gastric cancer (3.9%) compared to the previous years.

In this study, we did not find any correlation between the number of H pylori infected patients and incidence of gastric cancer because the decreased H pylori infection was not followed by the decreased incidence of gastric cancer. Limitation of this study was the small number of patients who were included.

Several foreign studies involving greater number of patients reported significant relation between the incidence of gastric cancer and H pylori infection.

"The Euro Gast" study covered 17 population from 13 countries and found significant correlation between H pylori infection and incidence of gastric cancer and also mortality due to it.⁹

The study by Sakiki et al, found more than 50% of Japanese population had H pylori infection, but only a

small number of patients found to have gastric cancer. Discrepancy of these study results might be associated with a different strain of *H pylori*, host or environment factor and infection period.¹⁰

A study by Rene W van der Hulst et al was aimed at investigating correlation between *H pylori* eradication and decreased intestinal metaplasia and atrophy. This study also observed microorganism factors such as *cagA* in *H pylori* at early infection and the course of disease after eradication. They found that successful eradication therapy in fact, did not change the grade of intestinal metaplasia and atrophy in 1 year follow up.¹¹

A study by Cheli R, Crespi M, Testino G, and Citarda F found that *H pylori* is a cofactor and contributed in the development of gastric malignancy but this was individually based.¹²

A study by Watanabe et al found that *H pylori* strain, host factor and infection period correlated to the development of gastric cancer. In this study, the strain of *H pylori* was HLA-DQBI*0401 and it was concluded that this strain was an important marker for the development of gastric cancer.¹³

Aside from microorganism factor and individual fashion, several studies tried to find the relation between diet and development gastric cancer. Nozaki et al found that high salt intake would increase host response of *H pylori* infection and it was an important role in the development of gastric cancer.¹⁴

Many studies, showed that there are various factors which may cause gastric cancer and gastric mucosal changes such as intestinal metaplasia and gastric mucosal atrophy in addition to *H pylori* infection. A multicenter study is needed to investigate the correlation between *H pylori* infection and gastric cancer and the important related factors including host, agent and environment.

CONCLUSION

In this study, the decreased prevalence of *H pylori* infection was not found in to be association with the incidence of intestinal metaplasia and gastric cancer.

Multi-center and prospective studies are necessary to investigate the incidence of gastric cancer in patients with *H pylori* infection.

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REFERENCES

1. Fennerty MB. *Helicobacter pylori*. Arch Intern. Med 1994;154: 721-7.
2. O'Connor HJ. *Helicobacter pylori* and gastric cancer: a review and hypothesis. Eur J Gastroentero Hepatol. 1992;4(2):103-9.
3. Marshall BJ. *Helicobacter pylori*. Am J Gastroenterol. 1994;89:116-28.
4. Simpponen PH. *Pylori-induced gastritis is risk factor for peptic ulcer disease and gastric cancer*. Report of an International Symposium held in Dublin, Ireland, July 1992: The role of acid inhibition in the management of *H Pylori* infection.
5. Lam SK, Talley NJ. *Helicobacter pylori* consensus: report from the 1997 Asia Pasific consensus. J Gastroenterol Hepatol. 1998;13:1-12.
6. Pentti Sipponen. Gastric cancer: pathogenesis, risks, and prevention. J Gastroenterol. 2002;37(Suppl XIII):39-44.
7. Drahislava Pantoflickova, Andre L. Blum. In: Achtman M and Suerbaum S, editors. *Helicobacter pylori: molecular and cellular biology*. England: Horizon Scientific Press; 2001. p. 3-8.
8. KSHPI, Konsensus Nasional Penanggulangan Infeksi *Helicobacter pylori*, Jakarta: 2001.
9. The Eurogast Study Group. An International Association Between *H pylori* Infection and Gastric Cancer. Lancet. 1993;341:1359-62.
10. Sakaki N, Momma K, Yamada Y. *Helicobacter pylori* infection and the development of atrophic gastritis assessed by endoscopy. Eur J Gastroenterol Hepatol. 1992;4(Suppl):S85-7.
11. Van der Hulst, RWM, Arie vander Ende, et al. Effect of *H pylori* eradication on gastritis in relation to *cag A*: a prospective 1-year follow-up study. Gastroenterol. 1997;113:25-30.
12. Cheli R, Crespi M, and Tertino G. Gastric cancer and *H pylori*: biologic and epidemiology inconsistencies. J Clin Gastroeterol. 1998;(1):3-6.
13. Watanabe Y, Aoyama N, Sakai T, Shirasaka D, Maekawa S, Kuroda K, et al. HLA-DQB1 locus and gastric cancer in *H pylori* infection. J Gastroenterol Hepatol. 2006;21:420-4.
14. Nobuyuki Shimizu, Masae Tatematsu, Michio Kaminishi. In: M. Kaminishi, K. Takubo, K. Majune, editors. The diversity of gastric carcinoma. Tokyo: Springer-Verlag; 2005. p. 5-7; 75-82.