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 consensuses 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 gastroendoscopy 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.
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)

DISCUSSION

In this study, we found the decreased incidence of H pylori incidence 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.

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)

In this study, it showed that the incidence of H pylori infection and intestinal metaplasia and gastric cancer can be seen in figure 1.
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. 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.

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.

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-DQB1*0401 and it was concluded that this strain was an important marker for the development of gastric cancer.

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.

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.

ACKNOWLEDMENT

The authors express great thanks to Miss Resmin, Tiur and Soka, Lila, Walu for their excellent assistance.

REFERENCES