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TOPIC HIGHLIGHT

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Efficacy of *Helicobacter pylori* eradication for the prevention of metachronous gastric cancer after endoscopic resection for early gastric cancer

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Abstract

Helicobacter pylori (*H. pylori*) plays an important role in gastric carcinogenesis, as the majority of gastric cancers develop from *H. pylori*-infected gastric mucosa. The rate of early gastric cancer diagnosis has increased in Japan and Korea, where *H. pylori* infection and gastric cancer are highly prevalent. Early intestinal-type gastric cancer without concomitant lymph node metastasis is usually treated by endoscopic resection. Secondary metachronous gastric cancers often develop because atrophic mucosa left untreated after endoscopic treatment confers a high risk of gastric cancer. The efficacy of *H. pylori* eradication for the prevention of metachronous gastric cancer remains controversial. However, in patients who undergo endoscopic resection of early gastric cancer, *H. pylori* eradication is recommended to suppress or delay metachronous gastric cancer. Careful and regularly scheduled endoscopy should be performed to detect minute metachronous gastric cancer after endoscopic resection.

Key words: *Helicobacter pylori*; Gastric cancer; Endoscopic resection; Metachronous cancer

Core tip: In Japan and Korea, mucosal gastric cancer without concomitant lymph node metastasis is usually treated with endoscopic resection. However, gastric cancer recurrence following endoscopic resection is a significant problem. Secondary metachronous gastric cancers often develop due to atrophic mucosa left untreated after endoscopic treatment. Currently, all available evidence suggests that *Helicobacter pylori* (*H. pylori*) eradication represents a primary chemopreventive strategy. However, the efficacy of *H. pylori* eradication for the prevention of metachronous gastric cancer has been controversial. Therefore, endoscopists should inspect the entire stomach for minute or occult metachronous gastric cancer. In addition, regular surveillance endoscopy should be performed.

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INTRODUCTION

Gastric cancer ranks as the fourth most common cancer and the second most frequent cause of death from can-

cer, accounting for 10.4% of cancer deaths worldwide[1]. Although the incidence of gastric cancer has declined in recent years, it remains a major health concern. Gastric carcinogenesis is a multifactorial process influenced by an interaction of host factors, including *Helicobacter pylori* (*H. pylori*) infection and environmental factors, such as diets rich in salt and nitrates/nitrites[2]. *H. pylori* plays an important role in gastric carcinogenesis, as the majority of non-cardia gastric cancers develop from *H. pylori*-infected mucosa[3]. *H. pylori* colonizes the gastric mucosa and triggers a series of inflammatory reactions, which are considered an important cause of chronic atrophic gastritis[4,5], an early step in a series of mucosal changes in the stomach leading to cancer. The current model for stomach carcinogenesis begins with gastritis and proceeds to chronic atrophic gastritis, followed by intestinal metaplasia, dysplasia, and, finally, carcinoma[6]. This model is supported by a considerable number of clinicopathological and epidemiological studies conducted in countries with a high incidence of gastric cancer; indeed, the World Health Organization has categorized *H. pylori* as a group 1 carcinogen for gastric cancer on the basis of these epidemiological studies[7,8]. Furthermore, animal models have clearly shown a causal link between *H. pylori* and gastric cancers[9,10], and *H. pylori* eradication has been shown to be a primary preventive measure for gastric cancer in these models[11]. However, the primary prevention of gastric cancer through *H. pylori* eradication in humans remains controversial.

In Japan and Korea, mucosal gastric cancer without concomitant lymph node metastasis is usually treated with endoscopic resection (*i.e.*, endoscopic mucosal resection and endoscopic submucosal dissection). Secondary metachronous gastric cancers often develop due to atrophic mucosa left untreated after endoscopic treatment, which confers a high risk of gastric cancer. The Japanese Society for *Helicobacter* Research has recommended that patients undergo *H. pylori* eradication therapy following endoscopic treatment of early gastric cancer for the prevention of metachronous cancers[12]. However, few studies have evaluated the efficacy of *H. pylori* eradication for metachronous gastric cancer prevention. The aim of this review is to evaluate existing evidence on the efficacy of *H. pylori* eradication for the prevention of metachronous gastric cancer after endoscopic resection of early gastric cancer. We reviewed large-scale epidemiological studies, meta-analyses, and animal model-based investigations linking gastric cancer to *H. pylori* infection. Literature searches using the Medline and PubMed databases were performed as part of the preparation for the implementation of this guideline. Our search was performed using index words related to *H. pylori* ("*Helicobacter pylori*" OR "*Helicobacter*" OR "pylori" OR "eradication"), gastric cancer ("gastric cancer" OR "stomach cancer" OR "metachronous"), and treatment ("endoscopic resection" OR "endoscopic submucosal dissection").

EFFECT OF *H. PYLORI* ERADICATION ON PRIMARY PREVENTION OF GASTRIC CANCER

Intervention studies completed in Columbia[13], China[14], and Japan[15] have suggested that *H. pylori* eradication is the most effective approach to gastric cancer prevention, but that it is more effective in patients with no basal atrophic gastritis or intestinal metaplasia. A pooled analysis of six studies with a total of 6695, largely Asian, participants followed for 4-10 years showed that the relative risk of gastric cancer after *H. pylori* eradication was 0.65 (95%CI: 0.43-0.98)[16]. However, a large-scale, double-blind randomized study in China showed that gastric cancer was still diagnosed after successful eradication of *H. pylori* and that eradication did not lead to a significant decrease in the incidence of gastric cancer[17]. A meta-analysis of four randomized intervention studies with gastric cancer incidence serving as a secondary outcome showed a non-significant OR of 0.67 (95%CI: 0.42-1.07)[18]. Currently, all available evidence suggests that *H. pylori* eradication represents a primary chemopreventive strategy in a subset of subjects. However, *H. pylori* eradication in patients who have already developed advanced pre-neoplastic lesions does not prevent gastric cancer development.

RISK OF METACHRONOUS CANCER IN PATIENTS WHO HAVE UNDERGONE ENDOSCOPIC RESECTION AND *H. PYLORI* ERADICATION FOR EARLY GASTRIC CANCER

The rate of early gastric cancer diagnosis has increased due to improved diagnostic procedures and use of endoscopy as a screening tool, particularly in asymptomatic individuals in Japan and Korea. In Japan, 60% of gastric cancers are early-stage tumors[19]. Early intestinal-type gastric cancer without concomitant lymph node metastasis is usually treated with endoscopic resection. Guidelines for the treatment of gastric cancer state that intestinal-type mucosal cancer < 20 mm in diameter with no evidence of ulcer or ulcer scar is an indication for endoscopic resection[20]. However, gastric cancer recurrence following endoscopic resection of early gastric cancer is a significant problem, as gastric cancer patients may have a higher *H. pylori* infection rate (71%-95%)[21] and a more abnormal mucosa with atrophic gastritis or intestinal metaplasia than the general population. Therefore, field carcinogenesis can result in gastric cancer, as the gastric environment is likely to promote the occurrence of secondary cancer.

Gastric cancer can recur in the stomach as a local recurrence or metachronous cancers. Metachronous gastric cancer is defined as new carcinoma that develops in areas other than the primary site at least 1 year after

endoscopic resection. As endoscopic resection spares a larger area of the gastric mucosa, multiple metachronous cancers may occur more frequently after endoscopic resection than after partial gastrectomy. The estimated recurrence rate of early gastric cancer in the gastric stump is 1.13%-1.90%[22,23]. Hosokawa *et al*[24] reported that the cumulative 5-year prevalence of residual gastric cancer post-partial gastrectomy for early gastric cancer was 2.4%. An increased risk of metachronous gastric cancers is expected in patients treated by endoscopic resection, in proportion to the larger area of gastric mucosa remaining in these patients. The length of follow-up has varied in previous studies, but the annual incidence rate of metachronous gastric cancer after endoscopic resection ranged from 3.3%-3.5%[19,25,26]. *H. pylori* eradication from the residual gastric mucosa after endoscopic treatment of early gastric cancer has an inhibitory effect on the occurrence of metachronous gastric cancer[27,28], and has been recommended in various guidelines[12,29]. Uemura *et al*[27] assigned patients who had undergone endoscopic therapy for early gastric cancer to an *H. pylori* eradication group or a non-eradication group and performed long-term follow-up. During a follow-up period of approximately 5 years, secondary gastric cancer was detected in 10 of 67 (15%) patients in the non-eradication group and 0 of 65 patients in the eradication group[27]. This study was not evaluated highly because the sample size was small and the patients were not strictly randomized. However, a multicenter retrospective study by Nakagawa *et al*[28] yielded a similar result. This study included 2835 patients with a median follow-up period of 2 years, during which *H. pylori* was eradicated in 356 (13%) patients. Metachronous gastric cancers developed in 8 (2%) patients successfully treated for *H. pylori*, compared with 129 (5%) patients with persistent *H. pylori* infection (OR = 0.42, 95%CI: 0.20-0.86, $P = 0.021$). A prospective randomized trial by Fukase *et al*[30] showed a significant decrease in the incidence of metachronous gastric cancer after endoscopic resection of early gastric cancer during a 3-year follow-up period. Secondary cancers were observed in significantly fewer (9 of 255) patients in the *H. pylori* eradication group compared with the control group (24 of 250 patients; HR = 0.339, 95%CI: 0.157-0.729, $P = 0.003$). In both groups, intestinal metaplasia was observed in 45%-65% of patients and gastric mucosal atrophy was moderate or severe in 80%-90% of patients. The authors concluded that *H. pylori* eradication might also be effective in patients who have early gastric cancer with mucosal atrophy and intestinal metaplasia. They suspected that eradication would inhibit the occurrence of new gastric cancer and reduce the growth rate of cancers that do occur. Because the 3-year follow-up period of this study was too brief to evaluate whether eradication prevents new occurrence of gastric cancer, this study likely evaluated clinical cancers that developed from occult cancer; *i.e.*, cancer that existed but was not detectable at the time of endoscopic treatment. If the detection time of residual cancer in the eradication group was delayed compared with the control group, these findings may thus indicate that *H. pylori* eradication decreases the rate of gastric cancer growth.

In contrast, a recently reported retrospective multicenter study showed that *H. pylori* eradication after endoscopic resection of early gastric cancer does not reduce the incidence of metachronous gastric cancer[31]. During overall follow-up periods of 1.1-11.1 (median, 3.0) years, metachronous gastric cancer developed in 13 of 177 (14.3%) patients in the persistent group and 15 of 91 (8.5%) patients in the eradicated group (log-rank test, $P = 0.262$). This finding is in agreement with a report from China that *H. pylori* eradication did not significantly reduce the incidence of gastric cancer[17]. Based on a multivariate logistic regression analysis, severe mucosal atrophy at baseline and during > 5 years of follow-up were found to be independent risk factors for the development of metachronous gastric cancer. The authors concluded that *H. pylori* should be eradicated before the progression of gastric mucosal atrophy. Haruma *et al*[32] also reported that severe mucosal atrophy was more frequent in patients with metachronous gastric cancer after *H. pylori* eradication than in those without, and that severe corpus atrophy was the only independent risk factor for cancer after *H. pylori* eradication. According to one recent multicenter retrospective study[26], the mean annual incidence rate of metachronous cancers was 3.5% in 1258 patients with early gastric cancer who were followed after endoscopic resection. The incidence rate did not differ between patients with or without *H. pylori* eradication[26].

Until now, the efficacy of *H. pylori* eradication for the prevention of metachronous gastric cancer has been controversial. Despite incongruities, as suggested by the existence of several guidelines, *H. pylori* eradication has been recommended to suppress or delay metachronous gastric cancer in patients who have undergone endoscopic resection of early gastric cancer. At the very least, we suspect that such benefits will be greater in patients without atrophy or intestinal metaplasia. However, eradication does not completely eliminate the risk of developing gastric cancer. Therefore, endoscopists using newly developed imaging techniques, such as magnifying endoscopy and narrow band imaging, should inspect the entire stomach for minute or occult metachronous gastric cancer. In addition, regular surveillance endoscopy should be performed every 6-12 mo for 5 years after initial endoscopic resection.

REFERENCES

1 **Parkin DM**. International variation. *Oncogene* 2004; **23**: 6329-6340 [PMID: 15322508 DOI: 10.1038/sj.onc.1207726]

2 **Houghton J**, Wang TC. Helicobacter pylori and gastric cancer: a new paradigm for inflammation-associated epithelial cancers. *Gastroenterology* 2005; **128**: 1567-1578 [PMID: 15887152 DOI: 10.1053/j.gastro.2005.03.037]

3 **Uemura N**, Okamoto S, Yamamoto S, Matsumura N, Yamaguchi S, Yamakido M, Taniyama K, Sasaki N, Schlemper RJ. Helicobacter pylori infection and the development of gastric cancer. *N Engl J Med* 2001; **345**: 784-789 [PMID: 11556297 DOI: 10.1056/NEJMoa001999]

4 **Kuipers EJ**, Uyterlinde AM, Peña AS, Roosendaal R, Pals G, Nelis GF, Festen HP, Meuwissen SG. Long-term sequelae of Helicobacter pylori gastritis. *Lancet* 1995; **345**: 1525-1528 [PMID: 7791437 DOI: 10.1016/S0140-6736(95)91084-0]

5 **Correa P**, Miller MJ. Helicobacter pylori and gastric atrophy--cancer paradoxes. *J Natl Cancer Inst* 1995; **87**: 1731-1732 [PMID: 7473824 DOI: 10.1093/jnci/87.23.1731]

6 **Correa P**. The epidemiology of gastric cancer. *World J Surg* 1991; **15**: 228-234 [PMID: 2031359 DOI: 10.1007/BF01659057]

7 **Forman D**, Newell DG, Fullerton F, Yarnell JW, Stacey AR, Wald N, Sitas F. Association between infection with Helicobacter pylori and risk of gastric cancer: evidence from a prospective investigation. *BMJ* 1991; **302**: 1302-1305 [PMID: 2059685 DOI: 10.1136/bmj.302.6788.1302]

8 **Parsonnet J**, Friedman GD, Vandersteen DP, Chang Y, Vogelman JH, Orentreich N, Sibley RK. Helicobacter pylori infection and the risk of gastric carcinoma. *N Engl J Med* 1991; **325**: 1127-1131 [PMID: 1891020 DOI: 10.1056/NEJM199110173251603]

9 **Watanabe T**, Tada M, Nagai H, Sasaki S, Nakao M. Helicobacter pylori infection induces gastric cancer in mongolian gerbils. *Gastroenterology* 1998; **115**: 642-648 [PMID: 9721161 DOI: 10.1016/S0016-5085(98)70143-X]

10 **Honda S**, Fujioka T, Tokieda M, Satoh R, Nishizono A, Nasu M. Development of Helicobacter pylori-induced gastric carcinoma in Mongolian gerbils. *Cancer Res* 1998; **58**: 4255-4259 [PMID: 9766647]

11 **Shimizu N**, Ikehara Y, Inada K, Nakanishi H, Tsukamoto T, Nozaki K, Kaminishi M, Kuramoto S, Sugiyama A, Katsuyama T, Tatematsu M. Eradication diminishes enhancing effects of Helicobacter pylori infection on glandular stomach carcinogenesis in Mongolian gerbils. *Cancer Res* 2000; **60**: 1512-1514 [PMID: 10749114]

12 **Asaka M**, Kato M, Takahashi S, Fukuda Y, Sugiyama T, Ota H, Uemura N, Murakami K, Satoh K, Sugano K. Guidelines for the management of Helicobacter pylori infection in Japan: 2009 revised edition. *Helicobacter* 2010; **15**: 1-20 [PMID: 20302585 DOI: 10.1111/j.1523-5378.2009.00738.x]

13 **Mera R**, Fontham ET, Bravo LE, Bravo JC, Piazuelo MB, Camargo MC, Correa P. Long term follow up of patients treated for Helicobacter pylori infection. *Gut* 2005; **54**: 1536-1540 [PMID: 15985559 DOI: 10.1136/gut.2005.072009]

14 **You WC**, Brown LM, Zhang L, Li JY, Jin ML, Chang YS, Ma JL, Pan KF, Liu WD, Hu Y, Crystal-Mansour S, Pee D, Blot WJ, Fraumeni JF, Xu GW, Gail MH. Randomized double-blind factorial trial of three treatments to reduce the prevalence of precancerous gastric lesions. *J Natl Cancer Inst* 2006; **98**: 974-983 [PMID: 16849680 DOI: 10.1093/jnci/djj264]

15 **Take S**, Mizuno M, Ishiki K, Nagahara Y, Yoshida T, Yokota K, Oguma K, Okada H, Shiratori Y. The effect of eradicating helicobacter pylori on the development of gastric cancer in patients with peptic ulcer disease. *Am J Gastroenterol* 2005; **100**: 1037-1042 [PMID: 15842576 DOI: 10.1111/j.1572-0241.2005.41384.x]

16 **Fuccio L**, Zagari RM, Eusebi LH, Laterza L, Cennamo V, Ceroni L, Grilli D, Bazzoli F. Meta-analysis: can Helicobacter pylori eradication treatment reduce the risk for gastric cancer? *Ann Intern Med* 2009; **151**: 121-128 [PMID: 19620164 DOI: 10.7326/0003-4819-151-2-200907210-00009]

17 **Wong BC**, Lam SK, Wong WM, Chen JS, Zheng TT, Feng RE, Lai KC, Hu WH, Yuen ST, Leung SY, Fong DY, Ho J, Ching CK, Chen JS. Helicobacter pylori eradication to prevent gastric cancer in a high-risk region of China: a randomized controlled trial. *JAMA* 2004; **291**: 187-194 [PMID: 14722144 DOI: 10.1001/jama.291.2.187]

18 **Fuccio L**, Zagari RM, Minardi ME, Bazzoli F. Systematic review: Helicobacter pylori eradication for the prevention of gastric cancer. *Aliment Pharmacol Ther* 2007; **25**: 133-141 [PMID: 17229238 DOI: 10.1111/j.1365-2036.2006.03183.x]

19 **Kato M**, Asaka M, Ono S, Nakagawa M, Nakagawa S, Shimizu Y, Chuma M, Kawakami H, Komatsu Y, Hige S, Takeda H. Eradication of Helicobacter pylori for primary gastric cancer and secondary gastric cancer after endoscopic mucosal resection. *J Gastroenterol* 2007; **42** Suppl 17: 16-20 [PMID: 17238020 DOI: 10.1007/s00535-006-1928-5]

20 **Nakajima T**. Gastric cancer treatment guidelines in Japan. *Gastric Cancer* 2002; **5**: 1-5 [PMID: 12021853 DOI: 10.1007/s101200200000]

21 **Ekström AM**, Held M, Hansson LE, Engstrand L, Nyrén O. Helicobacter pylori in gastric cancer established by CagA immunoblot as a marker of past infection. *Gastroenterology* 2001; **121**: 784-791 [PMID: 11606491 DOI: 10.1053/gast.2001.27999]

22 **Sowa M**, Kato Y, Onoda N, Kubo T, Maekawa H, Nishimura M, Nakanishi K, Maeda K, Chung YS, Umeyama K. Early cancer of the gastric remnant--with special emphasis on the importance of follow-up of gastrectomized patients. *Hepatogastroenterology* 1992; **39**: 400-404 [PMID: 1459518]

23 **Takeda J**, Toyonaga A, Koufuji K, Kodama I, Aoyagi K, Yano S, Ohta J, Shirozu K. Early gastric cancer in the remnant stomach. *Hepatogastroenterology* **1998; 45**: 1907-1911 [PMID: 9840174]

24 **Hosokawa O**, Kaizaki Y, Watanabe K, Hattori M, Douden K, Hayashi H, Maeda S. Endoscopic surveillance for gastric remnant cancer after early cancer surgery. *Endoscopy* 2002; **34**: 469-473 [PMID: 12048630 DOI: 10.1055/s-2002-32007]

25 **Han JS**, Jang JS, Choi SR, Kwon HC, Kim MC, Jeong JS, Kim SJ, Sohn YJ, Lee EJ. A study of metachronous cancer after endoscopic resection of early gastric cancer. *Scand J Gastroenterol* 2011; **46**: 1099-1104 [PMID: 21668406 DOI: 10.3109/00365521.2011.591427]

26 **Kato M**, Nishida T, Yamamoto K, Hayashi S, Kitamura S, Yabuta T, Yoshio T, Nakamura T, Komori M, Kawai N, Nishihara A, Nakanishi F, Nakahara M, Ogiyama H, Kinoshita K, Yamada T, Iijima H, Tsujii M, Takehara T. Scheduled endoscopic surveillance controls secondary cancer after curative endoscopic resection for early gastric cancer: a multicentre retrospective cohort study by Osaka University ESD study group. *Gut* 2013; **62**: 1425-1432 [PMID: 22914298 DOI: 10.1136/gutjnl-2011-301647]

27 **Uemura N**, Mukai T, Okamoto S, Yamaguchi S, Mashiba H, Taniyama K, Sasaki N, Haruma K, Sumii K, Kajiyama G. Effect of Helicobacter pylori eradication on subsequent development of cancer after endoscopic resection of early gastric cancer. *Cancer Epidemiol Biomarkers Prev* 1997; **6**: 639-642 [PMID: 9264278]

28 **Nakagawa S**, Asaka M, Kato M, Nakamura T, Kato C, Fujioka T, Tatsuta M, Keida K, Terao S, Takahashi S, Uemura N, Kato T, Aoyama N, Saito D, Suzuki M, Imamura A, Sato K, Miwa H, Nomura H, Kaise M, Oohara S, Kawai T, Urabe K,. Sakaki N, Ito S, Noda Y, Yanaka A, Kusugami K, Goto H, Furuta T, Fujino M, Kinjyou F, Ookusa T. Helicobacter pylori eradication and metachronous gastric cancer after endoscopic mucosal resection of early gastric cancer. *Aliment Pharmacol Ther* 2006; **24**: 214-218 [DOI: 10.1111/j.1746-6342.2006.00048.x]

29 **Malfertheiner P**, Megraud F, O'Morain CA, Atherton J, Axon AT, Bazzoli F, Gensini GF, Gisbert JP, Graham DY, Rokkas T, El-Omar EM, Kuipers EJ. Management of Helicobacter pylori infection--the Maastricht IV/ Florence Consensus Report. *Gut* 2012; **61**: 646-664 [PMID: 22491499 DOI: 10.1136/gutjnl-2012-302084]

30 **Fukase K**, Kato M, Kikuchi S, Inoue K, Uemura N, Okamoto S, Terao S, Amagai K, Hayashi S, Asaka M. Effect of eradication of Helicobacter pylori on incidence of metachronous gastric carcinoma after endoscopic resection of early gastric cancer: an open-label, randomised controlled trial. *Lancet* 2008; **372**: 392-397 [PMID: 18675689 DOI: 10.1016/S0140-6736(08)61159-9]

31 **Maehata Y**, Nakamura S, Fujisawa K, Esaki M, Moriyama

T, Asano K, Fuyuno Y, Yamaguchi K, Egashira I, Kim H, Kanda M, Hirahashi M, Matsumoto T. Long-term effect of Helicobacter pylori eradication on the development of metachronous gastric cancer after endoscopic resection of early gastric cancer. *Gastrointest Endosc* 2012; **75**: 39-46 [PMID: 22018552 DOI: 10.1016/j.gie.2011.08.030]

32 **Haruma K**, Kusunoki H, Manabe N, Kamada T, Sato M, Ishii M, Shiotani A, Hata J. Real-time assessment of gastroduodenal motility by ultrasonography. *Digestion* 2008; **77** Suppl 1: 48-51 [PMID: 18204262 DOI: 10.1159/000111488]

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