Endoscopic Gastritis: What Does It Mean?

Sun-Young Lee

Published online: 19 April 2011
© Springer Science+Business Media, LLC 2011

One can confidently assume that there is no evidence of endoscopic gastritis when the gastric mucosa has a regular pinkish color without discoloration or structural alteration, the gastric rugae are not thicker than 5 mm, and if the gastric mucosa expands well upon inflation with air. However, inflammatory changes induce subsequent apoptosis and gland damage, which can lead to regeneration, fibrosis, or metaplasia [1]. Inflammatory changes such as color and/or structural changes are noted in endoscopic findings, and are thus labeled endoscopic gastritis. Endoscopic gastritis can be classified into seven types: (a) superficial, (b) hemorrhagic, (c) erosive, (d) verrucous, (e) atrophic, (f) metaplastic, and (g) hypertrophic [2]. Erosive gastritis is diagnosed when endoscopy reveals small elevations with central umbilication, mimicking octopus' suckers, which are usually observed in the antrum. It can also be defined as a flat or minimally depressed white spot surrounded by a reddish area that is sometimes accompanied by small superficial bleeding [3]. Since the distinction between erosion and tiny shallow ulcer is arbitrary, the latter might be included under the term "erosion." When the erosion is elevated, it is generally defined as verrucous gastritis or gastritis varioliformis rather than as erosive gastritis.

In this issue of *Digestive Diseases and Sciences*, Yamamoto et al. [3] provide an interesting finding that lower serum levels of adiponectin are associated with an

increased risk of endoscopic erosive gastritis, independently of body mass index (BMI). BMI is known to be associated with abnormal upper endoscopic findings, such as erosive gastritis, gastric ulcer, duodenal ulcer, and reflux esophagitis [4]. Interestingly, when Yamamoto et al. analyzed factors including age, sex, alcohol habits, smoking habits, BMI, blood pressure, cholesterol, triglyceride, glucose, and insulin in 2,400 participants, BMI was significantly higher and the serum adiponectin level was significantly lower in gastritis-positive participants than in gastritis-negative participants. That study indicated a possible role of hypoadiponectinemia in erosive gastritis, in addition to a previous study that showed a link between low plasma adiponectin levels and gastric cancer [5].

Erosive gastritis has been considered relatively less significant than *Helicobacter pylori*-related endoscopic gastritis, since *H. pylori* infection involves a multistep progression from gastritis to glandular atrophy, intestinal metaplasia (IM), dysplasia, and ultimately to intestinal-type gastric cancer [6]. We have recently shown that the endoscopic diagnosis of open-type atrophic gastritis is related to the histological diagnosis of IM and Cdx2 expression [7]. Higher levels of Cdx2 expression are related to open-type atrophic gastritis and metaplastic gastritis, but not to closed-type atrophic gastritis and nonatrophic/nonmetaplastic cases. The location of the atrophic gastric border was found to be strongly related to histological IM and Cdx2 expression. Our findings suggest that Cdx2 expression is an early change that is correlated with the presence of histological IM, and which occurs before endoscopic metaplastic gastritis, and that the risk of development of intestinal-type gastric cancer will continue to remain high during the process of gastric carcinogenesis in open-type atrophic gastritis and metaplastic gastritis, but not in closed-type atrophic gastritis and nonatrophic/

S.-Y. Lee (✉)
Department of Internal Medicine,
Konkuk University School of Medicine, Seoul 143-729, Korea
e-mail: sunyoung@kuh.ac.kr

S.-Y. Lee
Research Institute of Medical Sciences, Konkuk University,
Seoul, Korea

Fig. 1 Open-type atrophic gastritis combined with metaplastic gastritis. Whitish patches, whitish plaques, and homogeneous whitish discoloration on the endoscopic findings suggesting metaplastic gastritis are more prominent in chromoendoscopic findings using 1.5% acetic acid mixed with 0.2% indigo carmine (**b**) than in those findings before applying chromoendoscopy (**a**)

Table 1 Gastritis that happens after *Helicobacter pylori* infection

Parameter	Recent <i>H. pylori</i> infection	Long-term <i>H. pylori</i> infection
Main subjects	Children and adolescents	Middle- or old-aged adults
Endoscopic finding	Hypertrophic gastritis Hemorrhagic gastritis Nodular gastritis	Atrophic gastritis Metaplastic gastritis
Major microscopic finding	Acute polymorphonuclear cell infiltrates Chronic mononuclear infiltrates	Gastric atrophy Intestinal metaplasia
After successful <i>H. pylori</i> eradication	Decreased intensities of acute polymorphonuclear cell infiltrates and chronic mononuclear infiltrates	Rare changes on gastric atrophy and intestinal metaplasia
Gastric acid	Usually increased	Usually decreased
Pepsinogen I	No change	Decreased

Fig. 2 Nodular gastritis diagnosed by endoscopy. Micronodules measuring 2–5 mm in diameter (**a**) are observed from the antrum to the low corpus of the stomach, indicating diffuse-type nodular gastritis. Nodular elevations are more prominent on chromoendoscopic findings using 0.2% indigo carmine spray. Three months after successful *H. pylori* eradication, nodular elevations have diminished (**b**)

nonmetaplastic cases. The endoscopic assessment of open-type atrophic gastritis indicates that patients with histological IM are at high-risk of gastric cancer. Interestingly, metaplastic gastritis is usually combined with atrophic gastritis, and can be highlighted upon chromoendoscopy using indigo carmine and acetic acid during an endoscopic examination (Fig. 1).

Atrophic gastritis and metaplastic gastritis indicate chronic *H. pylori* infection, whereas nodular gastritis is an endoscopic finding of recent *H. pylori* infection (Table 1).

Nodular gastritis is often known as lymphofollicular gastritis or chicken-skin-appearing gastritis (Fig. 2). This special type of antral gastritis is characterized by an unusual military pattern with prominent lymphoid follicles in a biopsy specimen. In East Asian countries wherein the prevalence of *H. pylori* infection is higher than in the West, the incidence of nodular gastritis among adults is reported to be up to 2.9% [8, 9]. Notably, a possible association between nodular gastritis and gastric cancer has been suggested with the characteristics of female predominance,

dominant, poorly differentiated or signet ring cell type gastric cancer, and pangastritis leading to an advanced stage scirrhous cancer (so called Borrmann type IV cancer) that usually invades the greater curvature side of the corpus [10]. We have recently reported cases of nodular gastritis in young women with atypical findings who required specific treatment for their gastric lesions [11]. Our findings suggest that, in contrast to cases in children, novel *H. pylori* infection may lead to nodular gastritis in some adults, and some may progress to diffuse-type nodular gastritis that invades both the antrum and body of the stomach. A few cases of diffuse-type nodular gastritis may progress further to gastric neoplasm, especially in young women, due to the occurrence of fewer atrophic changes.

Hypertrophic gastritis, congestive gastropathy, and hemorrhagic gastritis are major endoscopic findings that can be seen immediately after *H. pylori* infection. *H. pylori* infection is known to be a cause of several gastric diseases such as peptic ulcer disease, mucosa associated lymphoid tissue lymphoma, and gastric cancer with accumulating evidences that chronic inflammation can induce gene mutations, gene amplifications, and DNA dislocations in the affected cells [12]. When *H. pylori* is concentrated only on the antrum, it usually leads to duodenal lesions such as duodenal ulcer. However, when *H. pylori* spreads throughout the stomach (i.e., both the corpus and the antrum), it leads to gastric lesions such as gastric ulcer or gastric cancer [13]. It seems that the risk of gastric cancer increases with the degree of pangastritis, IM, and severe atrophy [14].

In summary, there are several important features of endoscopic gastritis that have significant meaning. For example, erosive gastritis is linked to obesity and hypoadiponectinemia [3], whereas open-type atrophic gastritis, metaplastic gastritis, and nodular gastritis are linked to gastric cancer [6–14]. Therefore, one must pay attention to the diagnostic features of gastritis during the endoscopic examination.

Conflict of interest The preparation of this manuscript was not supported by external funding. The author declares no conflict of interest relevant to this manuscript.

References

1. Genta RM. Review article: gastric atrophy and atrophic gastritis—nebulous concepts in search of a definition. *Aliment Pharmacol Ther.* 1998;12:17–23.
2. Dixon MF, Genta RM, Yardley JH, et al. Classification and grading of gastritis. The updated Sydney System. International workshop on the histopathology of gastritis, Houston 1994. *Am J Surg Pathol.* 1996;20:1161–1181.
3. Yamamoto S, Watabe K, Tsutsui S, et al. Lower serum level of adiponectin is associated with increased risk of endoscopic erosive gastritis. *Dig Dis Sci.* (Epub ahead of print). doi:[10.1007/s10620-011-1681-3](https://doi.org/10.1007/s10620-011-1681-3).
4. Kim HJ, Yoo TW, Park DI, et al. Influence of overweight and obesity on upper endoscopic findings. *J Gastroenterol Hepatol.* 2007;22:477–481.
5. Ishikawa M, Kitayama J, Kazama S, Hiramatsu T, Hatano K, Nagawa H. Plasma adiponectin and gastric cancer. *Clin Cancer Res.* 2005;11:466–472.
6. Correa P. A human model of gastric carcinogenesis. *Cancer Res.* 1988;48:3554–3560.
7. Ahn SY, Lee SY, Hong SN, et al. Endoscopic diagnosis of open-type atrophic gastritis is related to the histological diagnosis of intestinal metaplasia and Cdx2 expression. *Dig Dis Sci.* 2011;56:1119–1126.
8. Shiotani A, Kamada T, Kumamoto M, et al. Nodular gastritis in Japanese young adults: Endoscopic and histological observations. *J Gastroenterol.* 2007;42:610–615.
9. Chen MJ, Wang TE, Chang WH, Liao TC, Lin CC, Shih SC. Nodular gastritis: an endoscopic indicator of *Helicobacter pylori* infection. *Dig Dis Sci.* 2007;52:2662–2666.
10. Kamada T, Tanaka A, Haruma K. Nodular gastritis and gastric cancer. *Nippon Rinsho.* 2005;63:557–559.
11. Choi HJ, Lee SY, Lee JH, et al. Two atypical cases of nodular gastritis: A poorly differentiated gastric adenocarcinoma and a pseudo-low grade gastric MALT lymphoma. *Gastroenterol Res.* 2010;3:41–45.
12. Lee SY, Shin YW, Hahm KB. Phytoceutical: mighty but ignored weapon against *Helicobacter pylori* infection. *J Dig Dis.* 2008;9:129–139.
13. Takahashi S, Tokunaga K, Ito M. Quantity and location of intragastric *H. pylori* in relation to frequency and location of peptic ulcer. *Helicobacter Res.* 2006;10:87–89.
14. Leodolter A, Ebert MP, Peitz U, et al. Prevalence of *H. pylori* associated ‘high risk gastritis’ for development of gastric cancer in patients with normal endoscopic findings. *World J Gastroenterol.* 2006;12:5509–5512.