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LETTER TO THE EDITOR

Postprandial gastrin-17 level is a useful dynamic marker for atrophic gastritis

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Abstract

Atrophic gastritis and intestinal metaplasia may progress to gastric malignancy. Non-invasive serum biomarkers have been extensively studied and proven to be useful as a screening tool to stratify risk and identify patients for endoscopy to detect early gastric cancer. These non-invasive biomarkers have been endorsed and recommended by many international consensus guidelines. In this letter, we reviewed the literature and evidence supporting the use of serum biomarkers as a dynamic test to monitor the status of atrophic gastritis.

Key Words: Atrophic gastritis; Pepsinogen; Gastrin-17; Gastric cancer; Biomarkers

Core Tip: Serological markers such as pepsinogen and gastrin-17 (G-17) have been proposed as useful surrogate biomarkers for atrophic gastritis. Previous studies have shown that patients with atrophic corpus gastritis have low pepsinogen-I and pepsinogen ratio values but high G-17 levels, whereas patients with atrophic antral gastritis have low G-17 levels. Low serum levels of pepsinogen and G-17 are predictive of extensive gastric atrophy and early gastric cancer. Kotelevets *et al* demonstrated the sensitivity of postprandial gastrin levels in gastric atrophy can be improved with a specific cut-off level. Postprandial G-17 levels may serve as dynamic markers for atrophic gastritis.

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TO THE EDITOR

Atrophic gastritis and intestinal metaplasia increase the risk of gastric cancer[1]. This was first proposed in 1975 by Correa *et al*[2,3] and is known as Correa's cascade, where a chronic infection by *Helicobacter pylori* causes non-atrophic gastritis, subsequently progressing to chronic atrophic gastritis, intestinal metaplasia, dysplasia, and eventually adenocarcinoma. This observation was proven in a Dutch study, where the annual incidence of gastric precancerous condition (atrophic gastritis, intestinal metaplasia, and dysplasia) progressing to gastric cancer was found to be 0.1% for chronic atrophic gastritis, 0.25% for intestinal metaplasia, 0.6% for mild-to-moderate dysplasia, and 6% for severe dysplasia[1]. Sui *et al*[4] provided further evidence through a meta-analysis of 13 studies with 655937 subjects, confirming that gastric atrophy is associated with an increased risk of gastric cancer.

There have been multiple international guidelines on the management of patients with atrophic gastritis. The European Societies of Gastrointestinal Endoscopy, European Helicobacter and Microbiota Study Group, European Society of Pathology, and Sociedad Portuguesa de Endoscopia Digestiva [Management of epithelial precancerous conditions and lesions in the stomach (MAPS) II guidelines][5], as well as the American Gastroenterological Association (AGA)[6], have provided guidance. Both guidelines recommend eradication of *Helicobacter pylori* in all patients with gastric atrophy. However, while the MAPS II guidelines recommend systematic endoscopic surveillance in all patients with severe gastric atrophy (with or without intestinal metaplasia), the AGA guidelines focus only on intestinal metaplasia and advised against systematic surveillance, recommending surveillance based on shared decision-making between clinicians and patients.

Currently, gastric cancer screening programs have been implemented in countries with a high incidence of gastric cancer, such as Japan, Korea, and China[7-12]. This aims to enable diagnosis at an earlier stage, thereby improving patient survival.

Several screening strategies for gastric cancer have been proposed. Screening endoscopy is the most widely accepted method, but it is very operator dependent, and accuracy varies between centers[13]. High performance endoscopy, high resolution magnifying endoscopy, and virtual chromoendoscopy have been recommended by some guidelines to improve the detection rate of early gastric cancer[14]. Serology markers, such as pepsinogen and gastrin-17 (G-17), have been proposed as useful biomarkers to assess the gastric mucosa[15-17]. G-17 is secreted in antral G cells, and its level may be an indicator of antral atrophy[17]. Gastrin production increases after food intake; therefore, evaluating G-17 following a protein-rich meal is more accurate than fasting gastrin[18]. Serum pepsinogen is well established as a non-invasive indicator of atrophic gastritis and gastric cancer[19,20]. This was clearly demonstrated by Cao *et al*[15] who showed that pepsinogen, pepsinogen ratio (PGR), and G-17 values were significantly related to the grades and sites of atrophic gastritis. Patients with atrophic corpus gastritis had low pepsinogen-I (PGI) and PGR values, but high G-17 levels, whereas patients with atrophic antral gastritis had low G-17 levels. The authors concluded that atrophic corpus gastritis may be screened by low serum PGI and PGR, and high G-17 values, whereas gastric cancer may be screened by increased serum G-17 and significantly low serum PGI and PGR values. Similarly, Kikuchi *et al*[16] demonstrated that low serum levels of pepsinogen and G-17 are predictive of extensive gastric atrophy with a high risk of early gastric cancer. The serum pepsinogen-I/II ratio showed an inverse relationship with the extent of atrophy with the cut-off levels of 3.2 for endoscopic atrophic gastritis and 3 for histological atrophic gastritis, with sensitivity and specificity of 65.9% and 58.0% for the antrum, and 71.3% and 53.7% for the corpus, respectively[21]. A meta-analysis of 20 studies involving 4241 subjects evaluating the performance of serum biomarkers for the diagnosis of atrophic gastritis appeared to be reliable, with a sensitivity of 74.7% and specificity of 95.6%[22].

A few international consensus guidelines have recommended pepsinogen serology in atrophic gastritis: MAPS I[23] and II[4] consensus, the Maastricht VI/Florence Consensus[24], and the Kyoto consensus[25]. MAPS 1 and II recommend using low PGI and/or a low PGI/II ratio to identify patients with advance atrophic gastritis for endoscopy. According to the MAPS II guidelines, only individuals with stages III and IV of the operative link on gastritis assessment and operative link for gastritis intestinal metaplasia assessment should undergo surveillance. The Maastricht VI/Florence Consensus advocates using PG testing in countries with an intermediate risk of gastric cancer. The guidelines also state that PG and G-17 levels, along with a few other biomarkers, are useful to predict gastric mucosal atrophy. The Kyoto consensus confirmed that serological biomarkers are useful in identifying patients at higher risk of gastric cancer[25].

A commercially available screening test, GastroPanel, has been shown to be useful with high sensitivity[1]. However, there is wide heterogeneity in the results obtained in studies included in this meta-analysis, with sensitivity ranging from 32% to 98%. Other studies by Chapelle *et al*[26] and McNicholl *et al*[27] have shown that the diagnostic performance of GastroPanel was not significantly better than PGI alone.

In a study by Kotelevets *et al*[28] assessing the effectiveness of serological markers of gastric mucosal atrophy, the sensitivity of postprandial G-17 was 62.2% for serology cut-off levels of 0-4 pmol/L and 100% for a cut-off level of 0-10 pmol/L for the detection of atrophic gastritis. The study concluded that serological screening of multifocal atrophic gastritis by assessing serological levels of postprandial G-17 is a cost-effective method with a high level of sensitivity. Postprandial G-17 is an earlier marker of regression of atrophic gastritis than morphological examination of a gastric

biopsy in accordance with the Sydney system[27]. Therefore, postprandial G-17 is recommended for dynamic monitoring of atrophic gastritis after treatment.

In conclusion, serology testing may be useful as a mass screening tool and an important step before referral for endoscopy. This approach may improve the detection rate of precancerous condition or early cancer. Combining pepsinogen and G-17 levels could be helpful in identifying intestinal metaplasia and dysplasia among individuals with atrophic gastritis. The level of serum biomarkers may provide a valuable dynamic status of the progression or regression of atrophic gastritis.

FOOTNOTES

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