Screening of Atrophic Gastritis and Gastric Cancer Using Serum Pepsinogen, Gastrin-17 and *Helicobacter pylori* IgG Antibodies *CAO Qin, RAN Zhihua, XIAO Shudong. Renji Hospital, Shanghai Jiaotong University School of Medicine Shanghai Institute of Digestive Disease, Shanghai (200001)*

Background: Currently the diagnosis of atrophic gastritis and gastric cancer are mainly made by endoscopy and histopathology. In recent years, determination of serum pepsinogen (PG) Ⅰ, PGⅡ, gastrin-17 (G-17) and *Helicobacter pylori* (*H. pylori*) IgG antibodies is regarded as new tests for screening chronic atrophic gastritis and gastric cancer. **Aims:** To evaluate the use of serum tests: serum PG Ⅰ, PG Ⅰ/PG Ⅱ ratio (PGR), G-17 and *H. pylori*-IgG antibodies to screen atrophic gastritis, so as to increase the early diagnosis rate of gastric cancer. **Methods:** A total of 458 patients with gastroduodenal diseases diagnosed by gastroscopy in Shanghai, high incidence area of gastric cancer, were recruited, and each of them underwent endoscopy with biopsies before serum tests were performed. These patients were divided into 5 groups based on endoscopic and histopathological findings: 92 patients in atrophic gastritis group, 141 in gastric cancer group, 58 in gastric ulcer group, 90 in duodenal ulcer group, and 77 (including mild non-atrophic gastritis) served as control group. Fasting serum samples for PG Ⅰ and PGⅡ, G-17, and *H. pylori*-IgG antibodies determination were analyzed by enzyme-linked immunosorbent assay (ELISA). **Results:** PG Ⅰ and PGR values decreased significantly in both atrophic gastritis and gastric cancer groups ($P<0.01$). For the best discrimination of atrophic gastritis, the cut-off values of PG Ⅰ and PGR calculated by receiver operating characteristic (ROC) curve were 82.30 μg/L (sensitivity 85.9%, specificity 75.1%) and 6.05 (sensitivity 78.3%, specificity 71.6%), respectively. PG Ⅰ, PGR and G-17 values were related significantly with grades and/or sites of atrophic gastritis ($P<0.01$). Patients with atrophic corpus gastritis had low PG Ⅰ and PGR values and high G-17 level, and patients with atrophic antral gastritis had low G-17 level. G-17 level increased significantly in the gastric cancer group ($P<0.01$). PG Ⅰ and PGR values were significantly lower in patients with advanced gastric cancer than those in patients with early gastric cancer ($P<0.01$), while there was no difference in G-17 level between them. The positivity rate of *H. pylori*-IgG antibodies was 54.5% in the control group. PG Ⅰ level was higher in *H. pylori*-positive patients than that in the *H. pylori*-negative ones ($P<0.01$), whereas there was no difference in G-17 level between them. The positivity rates of *H. pylori*-IgG antibodies were over 85% in all other four groups. **Conclusions:** Low serum PG Ⅰ, PGR and G-17 values are biomarkers of atrophic corpus and antral gastritis, respectively. Atrophic gastritis can be screened by serum PG Ⅰ and PGR values. Screening of gastric cancer can be based on increased serum G-17 level and decreased serum PG Ⅰ and PGR values. *H. pylori* infection is related to the change of PG level.

Key words Gastritis, Atrophic; Stomach Neoplasms; Pepsinogens; Gastrins; *Helicobacter pylori* IgG Antibodies