
Objective: To explore the features and clinical significance of serum pepsinogen (PG) assay in a follow-up study on a high-risk gastric cancer (GC) population.

Methods: A total of 444 participants from a high-risk area of GC in north China were enrolled in this follow-up study from April 1997 to December 1999. Serum PG was measured by enzyme-linked immunosorbent assay (ELISA), and the percentage changes in PG were calculated with 'PG follow-up/PG first test' thrice from the beginning to the end of these 30 months. Stomach diseases were diagnosed by a gastroscopy with biopsy examination. *Helicobacter pylori* (*H. pylori*) status was assessed by histopathological examination and serum *H. pylori*-immunoglobulin (Ig)G antibody assay with ELISA.

Results: In all groups except for the 51–60-year olds no significant differences of percentage changes in PGII and the PGI/II ratio were observed during 30-month follow-up period. In the superficial gastritis (SG) group the percentage change in PGI of group A (after 6 months' follow up) was significantly lower than that of group B (after 12 months' follow up) (0.69 vs 0.97, \( P = 0.002 \)) in SG→SG; while in SG→normal (NOR), it was significantly higher than that in SG→atrophic gastritis (AG) (0.94 vs 0.79, \( P = 0.022 \)). In the AG group the percentage change in the PGI/II ratio of group A was significantly higher than that of group C (after 30 months' follow up) (1.13 vs 0.75, \( P = 0.042 \)) in AG→AG; and the percentage changes in PGI and PGII in AG→NOR were significantly lower than those in AG→SG (0.43 vs 0.87, \( P = 0.000 \); 0.60 vs 1.11, \( P = 0.010 \), respectively). In the *H. pylori*− (Hp−) group, the percentage change in PG of Hp−→Hp+ was significantly higher than that of Hp−→Hp− (0.94 vs 0.81, \( P = 0.026 \)). Percentage changes in PGI and PGII of Hp+→Hp− were significantly lower than those of Hp−→Hp+ (0.74 vs 0.93, \( P = 0.000 \); 0.86 vs 1.15, \( P = 0.000 \), respectively), while the percentage change in the PGI/II ratio was higher than that the group of Hp+→Hp− (0.90 vs 0.70, \( P = 0.022 \)).

Conclusion: The serum PG levels were influenced by the physiopathologic status of gastric mucosa and *H. pylori* infection, but they altered during the period of follow up. Serum PG assay might be a feasible and appropriate procedure to use in following up on a high-risk GC population.