Serum Progastrin and Its Products, Gastric Acid Secretion and Serum Pepsinogen I in Gastric Cancer

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Abstract
Background: Numerous studies have shown an association between Helicobacter pylori (Hp) infection and gastric cancer (GC). Study: This study was designed to determine the role of cytotoxin-associated gene A (CagA)-positive Hp infection, serum amidated gastrins and their precursor, progastrin, gastric acidity and serum pepsinogen I (PG-I) levels in gastric cancerogenesis in 74 cancer patients and in 77 age- and gender-matched controls. Serum IgG antibodies to Hp and CagA and levels of IL-8 and PG-I were measured by ELISA, while progastrin and amidated gastrin by specific radioimmunoassay. Results: The overall Hp and CagA seropositivity in GC patients were significantly higher (82 and 60%, respectively). Progastrin and amidated gastrin levels over their cutoff points (122 and 32 pM, respectively) were found in a significantly larger number of GC (59.4 and 44.5%) than in controls (9.0 and 16.8%, respectively). Histologically, all these GCs with increased serum progastrin and amidated gastrins were of intestinal type and showed CagA and Hp seropositivity. Serum IL-8 and gastric pH, above their cutoff points (pH >4.5), and serum PG-I level below its cutoff point (44.2 µg/l) were observed in a significantly higher number of GC patients as compared to controls. Conclusions: (1) GC patients have higher Hp and CagA seroprevalence than matched controls, confirming that CagA-positive Hp infection is associated with higher risk of GC; (2) serum levels of amidated gastrins and their precursor, progastrin, as well as IL-8 are significantly higher, while serum PG-I levels are reduced in intestinal type GC compared to controls, and (3) determination of high serum progastrin, amidated gastrins and IL-8 combined with low serum PG-I may be useful biomarkers of GC.

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