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Serum Pepsinogen II as A Marker of Both *Helicobacter pylori* and Acetyl Salicylic Acid-Related Gastritis

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Background. Both *Helicobacter pylori* and acetyl salicylic acid (ASA) are risk factors for peptic ulcer. Serum pepsinogen II (PGII) levels increase in the presence of *H. pylori*-related gastritis. A biomarker of severity of ASA gastritis should be useful to adopt considering the lack of symptoms in ASA induced mucosal injury.
Aim. To evaluate the effect of both ASA and *H. pylori* on PG II levels and gastric histology.
Methods. One hundred twenty-two consecutive dyspeptic patients (80 women, mean age: 64.1 years ± 12) of whom 52 with chronic assumption of low dose of ASA for prophylaxis of cardiovascular events (ASA positive) and 70 without assumption of ASA (ASA negative) were enrolled. Gastrointestinal endoscopy with biopsies and a blood sample for serological levels of PGII and antibodies IgG anti-*H. pylori* were performed.
Results. ASA-positive patients had significantly higher mean PGII levels (13.47 ± 4.5 SD µg/L) than ASA negative (8.35 ± 3.1 µg/L, $p < .001$), as well as *H. pylori* positive (13.41 ± 4.0 SD µg/L) in comparison to *H. pylori*-negative patients (8.30 ± 4.0 µg/L, $p < .001$). The significant highest mean value of PGII levels was found in ASA-positive and *H. pylori*-positive patients (15.96 µg/L). ASA-positive patients had significantly higher mean of severity chronic gastric inflammation both in corpus and antrum than ASA negative as well as considering *H. pylori* positive in comparison to *H. pylori*-negative patients.
Conclusion. Both ASA and *H. pylori* infection induce increased levels of sPGII. These factors seems to have an additional effects on serum PGII levels. High PGII levels could single out patients with high ASA induced gastropathy and mainly candidates for PPI gastroprotection.