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Precancerous gastric lesions: pathophysiology and symptomatology

*¹Adriana Botezatu, ¹Nicolae Bodrug, ²Viorel Istrate

¹Department of Internal Medicine, Discipline of Geriatrics and Occupational Diseases
Nicolae Testemitanu State University of Medicine and Pharmacy, Chisinau, the Republic of Moldova

²Medical Center "Excellence", Chisinau, the Republic of Moldova

*Corresponding author: adriana.botezatu@usmf.md

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Abstract

Background: Independent risk factors for chronic atrophic gastritis, gastric intestinal metaplasia and gastric cancer are: *Helicobacter pylori* infection (especially virulent CagA strains), genetic factors (advanced age, reflecting the duration of *Helicobacter pylori* infection, male gender, family history of gastric cancer in first-degree relatives), gastric ulcer, biliary reflux, acute or chronic gastric inflammation, smoking, alcohol consumption, prolonged use of proton pump inhibitors or non-steroidal anti-inflammatory drugs, diet low in fruits, vegetables and vitamin C, excessive salt intake and consumption of canned foods with salt). *Helicobacter pylori* infection and inflammatory cells induce the expression of inducible nitric oxide synthase in the gastroduodenal mucosa, which causes various clinical lesions (duodenal ulcer, gastric ulcer and chronic gastritis without ulcer). Another important condition associated with *Helicobacter pylori* infection is gastric cancer. Overproduction of nitric oxide, through inducible nitric oxide synthase and oxidative stress, is a genotoxic and mutagenic metabolism which plays a crucial role in the process of gastric carcinogenesis.

Conclusions: Chronic atrophic gastritis is considered a multifaceted condition because it can manifest itself through a variable spectrum of nonspecific gastric and extra-gastric symptoms, with an overlap of the clinical features of the two entities of chronic atrophic gastritis – autoimmune and associated with *Helicobacter pylori* infection. Thus, in contrast to the classic perception of a silent condition, patients with chronic atrophic gastritis report a wide range of gastrointestinal symptoms, ranging from dyspeptic symptoms to those of gastroesophageal reflux.

Key words: gastric intestinal metaplasia, epithelial dysplasia, gastric cancer, *Helicobacter pylori*.

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