Volume 4, Issue 5, May 2026 ISSN (E): 2938-3765

# AGE-RELATED FEATURES OF GASTRIC AND DUODENAL PEPTIC ULCER DISEASE

Kasimova M. B.
Eshmurzayev A.T.
Ulmasbekov A. K.
Tashkent State Medicine University

**Abstract**
Peptic ulcer disease is widespread worldwide, accounting for 10-15% of all gastrointestinal diseases. Gastric and duodenal ulcers are among the most common diseases, affecting 7-10% of the adult population. This literature review discusses current views on the pathogenesis of gastric and duodenal ulcers, including the role of Helicobacter pylori infection, in patients of various age groups. The pathogenetic characteristics and clinical and morphological manifestations of peptic ulcer disease in young and elderly individuals are examined in detail.

**Keywords**: Duodenal ulcer, gastric ulcer, age, Helicobacter pylori, pathogenesis.

## Introduction

Peptic ulcer disease is a chronic, cyclic disorder characterized during exacerbation periods by the formation of ulcers in the gastroduodenal zone. Despite improvements in preventive, therapeutic, and diagnostic methods, peptic ulcer disease (PUD) remains one of the most common gastrointestinal diseases worldwide for which patients seek medical care.

The incidence of PUD increases beginning at 18–25 years of age and reaches its peak at 35–40 years (62,6%). By the age of 40, PUD develops in 74,6% of patients. With increasing age, especially among women, the number of patients suffering from gastric ulcers and the risk of complications increase.

In young patients, ulcers are usually located in the duodenal bulb and less frequently in the stomach; they are generally small in size and heal relatively quickly. In patients under 25 years old, the ratio of gastric to duodenal ulcer localization is 1:14, and during adolescence it is 1:18. In general, the characteristic features of PUD in young patients include: a short medical history (up to 1,5 years), rapid development of the disease with pronounced ulcerative symptom complexes; rapid response to treatment (within 5 days); small ulcerative defects (up to 0,4 cm) with shallow depth and limited inflammatory changes in the mucosa of the gastric antrum and pyloroduodenal region; and absence or slight deformity of the duodenal bulb after ulcer scarring. In some young patients, PUD may have a latent or atypical course with predominance of dyspeptic syndrome. The absence of clearly expressed clinical manifestations sometimes leads to PUD presenting with complications in adolescents (approximately 8,5% of patients).

Young patients with PUD often exhibit extra-gastrointestinal manifestations associated with H.pylori infection, including cheilitis, angular stomatitis, resistant iron-deficiency anemia unrelated to gastrointestinal bleeding, thrombocytopenic purpura, neurodermatitis, urticaria, and others.



Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

Researchers associate these changes with immunological disorders characteristic of H.pylori infection.

Peptic ulcer disease is sufficiently widespread among elderly and senile populations. According to L.N.Valenkevich, newly diagnosed gastric ulcers occur in 5% of patients older than 60 years and in 3% of patients older than 75 years. The ratio of gastric and duodenal ulcers changes with age, with gastric ulcers becoming more common. In elderly individuals, gastric ulcers occur 1.7 times more often than duodenal ulcers, and in senile age they occur 3 times more frequently. In elderly patients, two variants of disease development are usually distinguished. The first is long-standing PUD that first appeared during youth or middle age, with preserved alternation of exacerbation and remission periods; this variant accounts for 30–50% of all PUD cases in elderly individuals. The second variant is "late-onset" PUD developing after the age of 60.

Long-standing PUD differs from the disease in young and middle-aged patients mainly by a higher frequency of combined lesions (gastric and duodenal), more frequent and prolonged exacerbations, and slower healing of ulcerative defects. A change in ulcer localization is characteristic: in patients diagnosed with duodenal ulcer disease during youth or mature age, gastric ulcers may develop in old age. PUD first appearing in senile age is characterized by certain clinical peculiarities: in more than 30% of cases, "late" gastroduodenal ulcers are large or giant in size and are often located in the subcardial region and body of the stomach. In PUD developing after the age of 60, dyspeptic manifestations predominate and seasonality of the disease is almost absent. In this form, the proportion of long-term non-healing ulcers reaches 25%. Ulcers in the "late" form of gastric ulcer disease are equally localized in the stomach and duodenum; combined complications are observed in 11,5% of cases, which is 2.4 times more frequent than in younger patients.

In many elderly patients, gastroduodenal ulcers occur with latent clinical manifestations and often present with complications, the incidence of which increases from 31% at 60–65 years to 76% at 75–80 years.

One of the most important problems in geriatrics is polymorbidity, meaning the presence of multiple pathologies in patients. Thus, the morbidity of elderly patients often resembles an iceberg, with most of it "hidden underwater". Peptic ulcers frequently coexist with ischemic heart disease (67%), arterial hypertension (55%), osteoarthritis (16,6%), type 2 diabetes mellitus (27,5%), chronic nonspecific pulmonary diseases (22,2%), liver diseases (18%), gallbladder diseases (24,4%), and pancreatic diseases (16,5%). At the same time, peptic ulcer disease remains an independent disorder, although its course and clinical manifestations may undergo significant changes. Age-related changes in the digestive organs and numerous concomitant diseases reduce the protective factors of the gastric and duodenal mucosa, decrease treatment effectiveness, contribute to frequent relapses and prolonged persistent disease course, and increase the risk of complications.

According to modern concepts, ulcer formation in the stomach and duodenum results from an imbalance between "aggressive" and "protective" factors. The most important roles are attributed to acid-peptic and infectious (H.pylori) factors, antibacterial resistance, and the protective factors of the gastroduodenal mucosa determining reparative processes. Aggressive factors include the effects of acid-peptic activity (excessive vagal nerve activity, hyperproduction of gastrin, increased mass of parietal cells in the gastric mucosa), H.pylori contamination, and disturbances in gastroduodenal motility (accelerated or delayed evacuation of acidic gastric contents and duodenogastric reflux).



Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

Protective mechanisms of the mucosa include adequate mucus production and high-quality elastic-viscous properties, bicarbonate secretion, regeneration of epithelial cells in the gastroduodenal region, sufficient mucosal blood supply, and immune defense. Most of the aforementioned aggressive and protective factors are genetically determined, and the balance between them is maintained through coordinated neuroendocrine interactions.

The pathogenetic significance of H.pylori in the development of PUD has been proven by numerous studies; the microorganism is detected in 90–95% of duodenal ulcer cases and in 70-85% of gastric ulcer cases. In the Russian Federation, the average prevalence of H.pylori infection among adults exceeds 80%, and its frequency increases with age. According to other authors, the prevalence of H.pylori infection among elderly patients is high, reaching approximately 40-60% in individuals without gastrointestinal diseases and more than 70% in cases of gastroduodenal pathology. Progression of chronic atrophic gastritis in elderly individuals is accompanied by the disappearance of H.pylori: only 45% of patients older than 80 years have H.pylori-associated ulcers.

Ulcer formation is not always associated with H.pylori. There may also be H.pylori-negative peptic ulcer disease. In 1995, G.N.Tytgat proposed another hypothesis replacing in 1989 D.Grem's hypothesis "No H.pylori – no peptic ulcer disease" with "No H.pylori – no H.pylori-associated peptic ulcer disease." This hypothesis emphasizes that PUD may develop and recur even in the absence of H.pylori.

Hypersecretion of hydrochloric acid and increased proteolytic activity of gastric juice play an important role in the development of gastric ulcer disease. Hypersecretion of hydrochloric acid may result from hypergastrinemia, vagal stimulation, and may also be genetically determined. However, the peptic theory of ulcerogenesis cannot explain the many cases of duodenal ulcers developing in individuals with normal or even low acidity levels, nor the absence of gastric ulcers throughout life in individuals with high acid production. From this perspective, it becomes even more difficult to explain the role of acid-peptic aggression in elderly patients, in whom gastric ulcers often develop under normal or hypoacidic conditions.

In elderly patients, peptic ulcer disease differs not only clinically and morphologically, but also etiopathogenetically. Existing data regarding the role of H.pylori in the development of gastroduodenal pathology in elderly individuals are limited; however, they indicate that this etiological factor remains significant in elderly patients. In elderly and senile individuals, the role of factors contributing to weakening of the protective properties of the gastroduodenal mucosa increases in the pathogenesis of "late-onset" peptic ulcer disease. Patients with PUD demonstrate significant reductions in secretion of fucoglycoproteins, as well as slowed secretion of mucin, prostaglandin E, and epidermal growth factor.

As humans age, angiopathies develop in the gastroduodenal mucosa and microcirculation changes due to hypoxia resulting from impaired blood rheology. Consequently, the resistance of the mucosa to aggressive factors decreases, trophic processes are disturbed, and regeneration slows. PUD is characterized by disturbances in local blood flow, blood rheology, and biogenic amine metabolism, leading to the development of hypoxia in the gastroduodenal mucosa. Increased retrodiffusion of H+ ions damages microvascular walls and causes microthrombosis, resulting in local ischemia and even microinfarctions in the gastric and duodenal mucosa. These processes are most frequently observed



Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

in the lesser curvature of the stomach and the duodenal bulb, where blood supply is poorer than in other regions of the gastroduodenal mucosa.

In elderly and senile individuals, the main indicators of gastric secretory function change: secretory volume decreases, gastric acidity decreases, and the production of bicarbonates and gastromucoproteins declines. These morphofunctional changes in the gastric mucosa during aging occur against the background of progressively increasing metabolic disturbances: reduced tissue and cellular respiration, insufficient formation of macroergic compounds and energy resources in cells and tissues, and decreased protein synthesis. These metabolic changes may be reflected in reduced levels of cyclic adenosine monophosphate and cyclic guanosine monophosphate in secretory cells of the gastric mucosa in elderly individuals.

In addition, aging disrupts neurohumoral regulation, general and local immunity, the balance between prooxidant and antioxidant systems, surface hydrophobicity of the mucosa, and biosynthesis of prostaglandins, which are essential components of the gastroduodenal mucosal defense system. During aging, the relationship between components of the autonomic nervous system changes, with the sympathetic system predominating over the parasympathetic system. In elderly and senile individuals, increased overall activity of the adrenergic system is accompanied by predominance of α-adrenergic inhibitory effects, manifested by decreased trophism of the gastric mucosa.

Several researchers believe that ulcers and erosions in elderly individuals are associated with impaired microcirculation in the mucosa, and that the main aggressive factors are hypoxia and related atrophic and metabolic disturbances. The significance of these factors – primarily atherosclerotic vascular changes reducing trophism of the gastric mucosa – is especially evident in cases where the disease develops without H.pylori involvement. In such patients, ulcers and erosions often develop against the background of hypertension, ischemic heart disease, diabetes mellitus, chronic nonspecific pulmonary diseases, and other conditions leading to impaired microcirculation in the gastric mucosa.

## Conclusion

Gastric and duodenal peptic ulcer disease in patients of different ages is a multifactorial disorder whose pathogenesis involves the central and autonomic nervous systems, biogenic amines, peptide hormones of the digestive tract, and expansion of H.pylori microorganisms, which necessitates a differentiated therapeutic approach.

## References

1. Alsinnari, Y.M., Alqarni, M.S., Attar, M. et al. Risk factors for recurrence of peptic ulcer disease: a retrospectivestudy in tertiary care referral center / Y.M. Alsinnari, M.S. Alqarni, M. Attar [et al.] (2022) // Cureus. – Vol. 14. – № 2. – P. e22001.

2. Chen, T.H. (2021) Epidemiology changes in peptic ulcer diseases 18 years apart explored from the genetic aspects of Helicobacter pylori / T.H. Chen, H.T. Cheng, C.T. Yeh // Transl. Res. – Vol. 232. – P. 115–120.

3. Holzer, K.J., Vaughn, M.G., Loux, T.M. et al. (2022) Prevalence and correlates of antisocial personality disorder in older adults / K.J. Holzer, M.G. Vaughn, T.M. Loux[et al.] // Aging Ment. Health. – Vol. 26. – № 1. – P. 169–178.



Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

4. Iwama, I., Yoshida, M., Hara, T., Nambu, R. Causes of melena and effective examination strategies in children (2021) // Front. Pediatr. – № 9. – P. 780356.
5. Kamada, T., Satoh, K., Itoh, T. et al. (2021) Evidence–based clinical practice guidelines for peptic ulcer disease 2020 // J. Gastroenterol. – Vol. 56. – № 4. – P. 303–322.
6. Lin, N., Smiley, A., Goud, M. et al. (2022) Risk factors of mortality in patients hospitalized with chronic duodenal ulcers / N. Lin, A. Smiley, M. Goud [etal.] // Am. Surg. – Vol. 88. – № 4. – P. 764–769.
7. Noguchi, H., Kumamoto, K., Harada, Y. et al. (2020) Prevalence of Helicobacter pylori infection rate in heterotopic gastric mucosa in histological analysis of duodenal specimens from patients with duodenal ulcer / H. Noguchi, K. Kumamoto, Y. Harada [et al.] // Histol. Histopathol. – Vol. 35. – № 2. – P. 169–176.
8. Zhao, J., Li, X., Liu, Q. et al. (2020) Clinical comparative analysis of various duodenal diseases in different age groups / // Turk. J. Gastroenterol. – Vol. 31. – № 7. – P. 489–496.
9. Yucel, O. (2019) Interactions between Helicobacter pylori and gastroesophageal reflux disease / O. Yucel // Esophagus. – Vol. 16. – № 1. – P. 52–62.
10. Бондарева, Д.В., Камышникова, Л.А., Ефремова, О.А., Халаимова, О.А. (2021) Оценка качества жизни пациентов с язвенной болезнью двенадцатиперстной кишки. *Тихоокеанский медицинский журнал*;3:79–82.
11. Вязников, В.А., Прокопьев, Е.С. (2022) Язвенная болезнь желудка и двенадцатиперстной кишки /– Киров: ФГБОУ ВО Кировский ГМУ Минздрава России, – 98 с.
12. Габбасова, Л.В., Волевач, Л.В., Палтусов, А.И., Крюкова, А.Я., Хисматуллина, Г.Я., Камалова А.А. и др. (2017) *Язвенная болезнь двенадцатиперстной кишки у лиц молодого возраста.* Тамбов: Консалтинговая компания Юком; 48 с.
13. Гудовских, Н.В. (2019) Особенности развития язвенной болезни двенадцатиперстной кишки у лиц мужского пола в молодом возрасте / Н.В. Гудовских, М.С. Чупина, Д.А. Толмачев // Дневник науки. – № 11 (35). – С. 9.
14. Ивашкин, В.Т., Маев, И.В., Царьков, П.В., Королев, М.П., Андреев, Д.Н., Баранская, Е.К., Бурков, С.Г., Деринов А.А., Ефетов, С.К., Лапина, Т.Л., Павлов, П.В., Пирогов, С.С., Ткачев, А.В., Трухманов, А.С., Федоров, Е.Д., Шептулин, А.А. (2020) Диагностика и лечение язвенной болезни у взрослых (Клинические рекомендации Российской гастроэнтерологической ассоциации, Российского общества колоректальных хирургов и Российского эндоскопического общества). *Российский журнал гастроэнтерологии, гепатологии, колопроктологии*. 30(1):49-70.
15. Канан, М.И.Т. (2021) Язвенная болезнь / М.И.Т. Канан // Наука и образование сегодня. – № 3 (62). – С. 69–75.
16. Касимова, М.Б., Юлдашева, У.Х., Эшмурзаев, А.Т. (2026) Оценка качества жизни пациентов с язвенной болезнью желудка и двенадцатиперстной кишки //Всероссийский терапевтический конгресс с международным участием «Боткинские чтения». Сборник тезисов. Санкт-Петербург, 16-17 апреля. С. 162-163.
17. Костоломова, Е.Г., Мамедов, С.Ф.О., Караисаев, А.Н.О. (2022) Современные данные о Helicobacter pylori / Е.Г. Костоломова, С.Ф.О. Мамедов, А.Н.О. Караисаев [и др.] // Наукосфера– № 1–1. – С. 26–37.



Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

18. Полозова, Э.И., Скворцов, В.В., Трохина, И.Е., Нефедов, Н.С. (2021) Особенности течения язвенной болезни желудка в разных возрастных группах. *Медицинский совет*. (5):38–44.
19. Пугачева, М.Г., Щапкова, М.М., Кондрашов, В.А. (2019) Современные представления об этиологии язвенной болезни желудка и двенадцатиперстной кишки. *Science Time*. 8(68):35–37.
20. Рашина, О. В., Чурносов, М. И. (2021) Гены-кандидаты язвенной болезни. Экспериментальная и клиническая гастроэнтерология. 186(2): 52–57.
21. Рашина, О. В., Чурносов, М. И. (2021) Многофакторный этиопатогенез язвенной болезни желудка и двенадцатиперстной кишки. Экспериментальная и клиническая гастроэнтерология. 192(8): 154–159.
22. Сергеева, А.В. (2022) Роль Helicobacter pylori в комплексной коморбидности человека / А.В. Сергеева, В.В. Шкарин, О.В. Ковалишена // Инфекция и иммунитет.– Т. 12. – № 1. – С. 21–32.
23. Эседов, Э.М., Маммаев, С.Н., Абасова, А.С., Акбиева, Д.С. (2020) Язвенная болезнь. Хронический гастрит (современный взгляд на патогенез, клинику, лечение) / Э.М. Эседов, С.Н. Маммаев, А.С. Абасова, Д.С. Акбиева. – Махачкала, – 305 с.



Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5