# RISK FACTORS AND COMPLICATION RATES FOR PEPTIC ULCER DISEASE

**Panjiyev Jonibek Abdumajidovich**

Department of Fundamental Medical Sciences of the Asian International University, Bukhara, Uzbekistan.

***https://doi.org/10.5281/zenodo.14895151***

***Abstract.*** *Peptic ulcer disease (PUD) is characterized by discontinuation in the inner lining of the gastrointestinal (GI) tract because of gastric acid secretion or pepsin. It extends into the muscularis propria layer of the gastric epithelium. It usually occurs in the stomach and proximal duodenum. It may involve the lower esophagus, distal duodenum, or jejunum. Epigastric pain usually occurs within 15-30 minutes following a meal in patients with a gastric ulcer; on the other hand, the pain with a duodenal ulcer tends to occur 2-3 hours after a meal. Peptic ulcer disease (PUD) is a common digestive disorder that generally refers to an acid peptic injury in the stomach, duodenum, Meckel's diverticulum, or esophagus. Most studies on PUD focused on Helicobacter pylori (H. pylori) infection, which afects gastrointestinal diseases, such as PUD and gastritis.*

*However, peptic ulcers are related to various risk factors other than H. pylori infection, including socioeconomic, environmental, and psychological characteristics and other potential factors. Besides, Peptic ulcer disease (PUD) if not diagnosed and treated promptly can lead to serious complications.*

***Keywords:*** *peptic ulcer, risk factors, gastric cancer, penetration, perforation.*

# ФАКТОРЫ РИСКА И ЧАСТОТА ОСЛОЖНЕНИЙ ПРИ ЯЗВЕННОЙ БОЛЕЗНИ

***Аннотация.*** *Язвенная болезнь (ЯБ) характеризуется нарушением внутренней оболочки желудочно-кишечного тракта (ЖКТ) из-за секреции желудочной кислоты или пепсина. Она распространяется на мышечный слой желудочного эпителия. Обычно она возникает в желудке и проксимальном отделе двенадцатиперстной кишки. Она может затрагивать нижнюю часть пищевода, дистальный отдел двенадцатиперстной кишки или тощую кишку. Боль в эпигастрии обычно возникает в течение 15–30 минут после еды у пациентов с язвой желудка; с другой стороны, боль при язве двенадцатиперстной кишки, как правило, возникает через 2–3 часа после еды. Язвенная болезнь (ЯБ) — распространенное расстройство пищеварения, которое обычно относится к кислотному пептическому поражению желудка, двенадцатиперстной кишки, дивертикула Меккеля или пищевода. Большинство исследований ЯБД были сосредоточены на инфекции Helicobacter pylori (H. pylori), которая поражает желудочно-кишечные заболевания, такие как ЯБД и гастрит. Однако язвенная болезнь связана с различными факторами риска, помимо инфекции H. pylori, включая социально-экономические, экологические и психологические характеристики и другие потенциальные факторы.*

*Кроме того, язвенная болезнь (ЯБД), если ее не диагностировать и не лечить своевременно, может привести к серьезным осложнениям.*

***Ключевые слова:*** *язвенная болезнь, факторы риска, рак желудка, пенетрация, перфорация.*

Peptic ulceration is a major public health problem. It is estimated that each year, peptic ulcer disease (PUD) affects 4 million people around the world. Individuals with PUD are at risk of developing complications such as gastroduodenal haemorrhage, perforation, penetration, and obstruction, and mortality among patients with these complications is high. Peptic ulcer perforation (PUP) is a frequent emergency condition worldwide associated with high mortality if left untreated. It presents as an acute abdominal condition, with localised or generalised peritonitis and a high risk for developing sepsis and death. PUP is a surgical emergency and carries a mortality ranging from 1.3% to 20%. Thirty-day mortality rates reaching 20% and 90-day mortality rates of up to 30% have been reported. Being closely related to advanced age, increased burden of comorbidity may partially explain the higher mortality among elderly patients; however, several other factors affect this high mortality. While Helicobacter pylori and use of non-steroidal anti-inflammatory drugs (NSAID) are frequent causes of PUP, demographic differences in age, gender, perforation location, and aetiology exist between countries, as do mortality rates, with several risk factors potentially influencing the development of PUP and postoperative mortality.

Numerous studies of sociodemographic characteristics and peptic ulcers identifed various risk factors, such as age, low education, low socioeconomic status or low salary, household member crowding, unemployment, marital strain, a blue-collar household, meal intake regularity, breakfast skipping, smoking, heavy alcohol intake, high body mass index (BMI), nonsteroidal anti-infammatory drugs (NSAIDs), musculoskeletal pain, headache, psychological and physical stress, and previous peptic ulcers. For example, an important risk factor for PUD is cigarette smoking.

Smoking is a risk factor for chronic active ulcers or asymptomatic PUD in the United States, Israel, Taiwan, Denmark, and Norway and in American men of Japanese ancestry. Alcohol intake was also associated with PUD. However, several studies disagreed with the association of PUD with alcohol intake and smoking. Low education level is a risk factor for PUD because education level was related to living conditions, such as lifestyle, diet, and social stress, and these conditions are part of the multifactorial etiology of PUD. Similar to education level, low socioeconomic class or status is associated with PUD. Populations in low socioeconomic class or status are linked to heavy alcohol intake, smoking, hard physical work, hygiene, concerns about dismissal, inadequate nutrition, use of painkillers, and psychological stress.

Psychological stress and physical stress afect the development of ulcers because stress aggravates gastroduodenal blood fow, reduces acid bufering in the duodenum, and diminishes gastric hypersecretion. Stress tends to be uncontrolled and unpredictable, promotes the onset of disease, and is one of the most common risk factors for PUD.

In addition, peptic ulcer disease (PUD) can lead to serious complications if left untreated.

The major complications include:

1. Gastrointestinal Bleeding (Most Common)

Due to erosion of blood vessels in the ulcer bed.

2. Perforation (Life-Threatening)

Full-thickness ulceration leads to perforation into the peritoneal cavity.

3. Penetration

Ulcer erodes into adjacent organs (e.g., pancreas, liver).

4. Gastric Outlet Obstruction

Due to chronic inflammation, edema, or scarring in the pyloric region.

5. Malignant Transformation (Rare)

Chronic gastric ulcers may undergo malignant changes, especially if H. pylori is present.

Gastric ulcer bleeding is a serious complication of peptic ulcer disease (PUD) and is a major cause of upper gastrointestinal bleeding (UGIB). It occurs when an ulcer erodes into a blood vessel, leading to acute or chronic hemorrhage.

Clinical Presentation:

- Hematemesis – Vomiting fresh red blood or "coffee-ground" material.
- Melena – Black, tarry stools (digested blood).
- Hematochezia – Bright red blood per rectum (if severe, fast bleeding).
- Epigastric pain – Often worsens with eating (gastric ulcers).
- Signs of shock (in massive bleeding) – Hypotension, tachycardia, pallor, dizziness, syncope.

Perforation is a life-threatening complication of peptic ulcer disease (PUD), occurring when an ulcer erodes through the full thickness of the stomach wall, leading to peritonitis and potential sepsis. It is a surgical emergency.

Clinical Presentation:

- Sudden, severe epigastric pain – "Knife-like" pain, often radiating to the shoulders (due to diaphragmatic irritation).
- Rigid, board-like abdomen – Classic sign of peritonitis.
- Absent bowel sounds – Due to paralytic ileus.

- Tachycardia, hypotension – Signs of shock if sepsis develops.
- Fever, leukocytosis – In late stages.

Penetration occurs when a gastric ulcer extends beyond the stomach wall into adjacent structures, such as the pancreas, liver, biliary tree, or colon. Unlike perforation, there is no free air in the peritoneal cavity because the ulcer erodes into another organ rather than creating a hole into the peritoneal space.

Clinical Presentation:

Persistent, intense epigastric pain – more severe than typical ulcer pain and radiates to the back.

- Pain unaffected by food or antacids – Unlike simple PUD, pain does not improve after meals.
- Weight loss & anorexia – Due to chronic discomfort.
- Nausea & vomiting – If the ulcer affects gastric motility.
- GI bleeding (less common) – Occult blood loss may lead to anemia.

Gastric outlet obstruction (GOO) is a complication of chronic peptic ulcer disease (PUD), occurring due to edema, inflammation, or scarring (fibrosis) in the pyloric region. It leads to gastric stasis, causing persistent vomiting and nutritional deficiencies.

Clinical Presentation:

- Postprandial nausea & vomiting – Non-bilious, containing undigested food.
- Early satiety & bloating – Due to gastric distension.
- Epigastric pain – May improve after vomiting.
- Weight loss & dehydration – Due to chronic vomiting and poor intake.
- Visible peristalsis – "Succussion splash" on auscultation (due to retained gastric contents).

While most gastric ulcers are benign, some may undergo malignant transformation, particularly in the presence of chronic inflammation, persistent ulceration, or underlying H. pylori infection. Unlike duodenal ulcers, which are almost always benign, gastric ulcers have a higher risk of being associated with malignancy, especially if they fail to heal despite treatment.

Clinical Presentation:

- Persistent epigastric pain – Often dull and unresponsive to acid suppression therapy.
- Unintentional weight loss – A red flag for malignancy.
- Early satiety & bloating – Suggests gastric outlet involvement.
- Occult GI bleeding (iron deficiency anemia, melena, or hematemesis).
- Anorexia, nausea, and fatigue.

**Conclusions**: This study showed that decreased BMI, decreased Hb, increased age, and smoking were independent risk factors for development of PUP. Thus, this group of patients needs particular attention paid to suggestive symptoms with early diagnosis and optimal management of peptic ulcer disease.

**REFERENCES**

1. Lanas, A. & Chan, F. K. Peptic ulcer disease. Lancet 390, 613–624 (2017).
2. Torpy, J. M., Lynm, C. & Golub, R. M. Peptic ulcer disease. JAMA 307, 1329–1329 (2012).
3. Pütsep, K., Brändén, C.-I., Boman, H. G. & Normark, S. Antibacterial peptide from H. pylori. Nature 398, 671–672 (1999).
4. Sostres, C. et al. Peptic ulcer bleeding risk. Te role of Helicobacter pylori infection in NSAID/low-dose aspirin users. Am. J. Gastroenterol. 110, 684–689 (2015).
5. Suerbaum, S. & Michetti, P. Helicobacter pylori infection. N. Engl. J. Med. 347, 1175–1186 (2002).
6. Levenstein, S. Stress and peptic ulcer: life beyond helicobacter. BMJ 316, 538 (1998).
7. Jones, M. P. Te role of psychosocial factors in peptic ulcer disease: beyond Helicobacter pylori and NSAIDs. J. Psychosom. Res. 60, 407–412. https://doi.org/10.1016/j.jpsychores.2005.08.009 (2006).
8. Freston, J. W. Review article: role of proton pump inhibitors in non-H. pylori-related ulcers. Aliment Pharmacol. Ter. 15(Suppl 2), 2–5. https://doi.org/10.1046/j.1365-2036.2001.00114.x (2001).
9. Marshall, B. J. Helicobacter pylori in peptic ulcer: have Koch's postulates been fulflled?. Ann. Med. 27, 565–568 (1995).
10. Konturek, S. J. et al. Helicobacter pylori, non-steroidal anti-infammatory drugs and smoking in risk pattern of gastroduodenal ulcers. Scand. J. Gastroenterol. 38, 923–930. https://doi.org/10.1080/00365520310004696 (2003).
11. Narayanan M, Reddy KM, Marsicano E. Peptic Ulcer Disease and *Helicobacter pylori* infection. Mo Med. 2018 May-Jun;115(3):219-224. [PMC free article] [PubMed]
12. Lanas Á, Carrera-Lasfuentes P, Arguedas Y, García S, Bujanda L, Calvet X, Ponce J, Perez-Aísa Á, Castro M, Muñoz M, Sostres C, García-Rodríguez LA. Risk of upper and lower gastrointestinal bleeding in patients taking nonsteroidal anti-inflammatory drugs, antiplatelet agents, or anticoagulants. Clin Gastroenterol Hepatol. 2015 May;13(5):906-12.e2. [PubMed]

13. Huang JQ, Sridhar S, Hunt RH. Role of Helicobacter pylori infection and non-steroidal anti-inflammatory drugs in peptic-ulcer disease: a meta-analysis. Lancet. 2002 Jan 05;359(9300):14-22. [PubMed]
14. Snowden FM. Emerging and reemerging diseases: a historical perspective. Immunol Rev. 2008 Oct;225(1):9-26. [PMC free article] [PubMed]
15. Lanas A, Chan FKL. Peptic ulcer disease. Lancet. 2017 Aug 05;390(10094):613-624. [PubMed]
16. ASGE Standards of Practice Committee. Banerjee S, Cash BD, Dominitz JA, Baron TH, Anderson MA, Ben-Menachem T, Fisher L, Fukami N, Harrison ME, Ikenberry SO, Khan K, Krinsky ML, Maple J, Fanelli RD, Strohmeyer L. The role of endoscopy in the management of patients with peptic ulcer disease. Gastrointest Endosc. 2010 Apr;71(4):663-8. [PubMed]
17. Malfertheiner P, Megraud F, O'Morain CA, Gisbert JP, Kuipers EJ, Axon AT, Bazzoli F, Gasbarrini A, Atherton J, Graham DY, Hunt R, Moayyedi P, Rokkas T, Rugge M, Selgrad M, Suerbaum S, Sugano K, El-Omar EM., European Helicobacter and Microbiota Study Group and Consensus panel. Management of Helicobacter pylori infection-the Maastricht V/Florence Consensus Report. Gut. 2017 Jan;66(1):6-30. [PubMed]
18. Strand DS, Kim D, Peura DA. 25 Years of Proton Pump Inhibitors: A Comprehensive Review. Gut Liver. 2017 Jan 15;11(1):27-37. [PMC free article] [PubMed]
19. Zelickson MS, Bronder CM, Johnson BL, et al. Helicobacter pylori is not the predominant etiology for peptic ulcers requiring operation. Am Surg 2011; 77: 1054-60.
20. Lau JY, Sung J, Hill C, et al. Systematic review of the epidemiology of complicated peptic ulcer: incidence, recurrence, risk factors and mortality. Digestion 2011; 84: 102-13.
21. Soreide K, Thorsen K, Harrison EM, et al. Perforated peptic ulcer. Lancet 2015; 386: 1288-98.
22. Daniel VT, Wiseman JT, Flahive J, et al. Predictors of mortality in the elderly after open repair for perforated peptic ulcer disease. J Surg Res 2017; 215: 108-13.
23. Soreide K, Thorsen K, Soreide JA. Strategies to improve the outcome of emergency surgery for perforated peptic ulcer. Br J Surg 2014; 101: e51-64.
24. Unver M, Fırat O, Unalp OV, et al. Prognostic factors in peptic ulcer perforations: a retrospective 14-year study. Int Surg 2015; 100: 942-8.
25. Sung JJ, Kuipers EJ, El-Serag HB. Systematic review: the global incidence and prevalence of peptic ulcer disease. Aliment Pharmacol Ther 2009; 29: 938-46.
26. Wang YR, Richter JE, Dempsey DT. Trends and outcomes of hospitalizations for peptic ulcer disease in the United States, 1993 to 2006. Ann Surg 2010; 251: 51-8.