ISSN: 1813-1638

The Medical Journal of Tikrit University

Available online at: www.mjotu.com

Demographic and Endoscopic Characteristics of Peptic Ulcer Disease in Mosul

Khaldoon T. Al-Abachi [(1)]

(1) Department of Medicine, College of Medicine, University of Ninevah, Iraq

***Keywords*:**

Demography,
Duodenal ulcer,
Endoscopy,
Gastric ulcer.

ARTICLE INFO

Article history:

Received 03 Apr 2022
Accepted 13 May 2022
Available online 01 Sep 2022

ABSTRACT

Background: Peptic ulcer disease (PUD) is a common gastrointestinal problem with significant morbidity and mortality despite introduction of effective therapy. Some aspects of this disease carry regional and ethnic variations.

The aim of the present study is to demonstrate demographic and endoscopic characteristics of gastroduodenal ulcers in Mosul, and display their frequencies and bleeding complications.

Patients & methods : This is a retrospective, cross-sectional study of 326 patients diagnosed endoscopically with peptic ulcers in Al-Salam General Hospital in Mosul during a period from January 2018 to December 2020. Details of patients records regarding demographic, clinical, and endoscopic findings were collected and analyzed.

Results: Total number of patients was 326. Duodenal ulcer (DU) comprised 286 patients (205 males, 81 females), and gastric ulcer (GU) 40 (18 males, 22 females). Mean age of DU patients was 34.1 ± 13.9 years, and GU 54.9±15.3 years. Male:female ratio was (223/103, 2.2:1). DU:GU ratio was (286:40, 7.2:1). The main site of DU was the duodenal bulb (283/286, 99%) and for GU the antrum and lesser curve (28/40, 70%). Around 90% of ulcers were single. Bleeding was a presenting symptom in 20% of patients, and in 32.3% were associated with the use of mainly non-steroidal anti-inflammatory drugs (NSAIDs).

Conclusions: In Mosul city DU affects a relatively younger ages compared to GU. DU was more predominant in males. DU:GU ratio was wide. Ulcers were mainly single and located in the duodenal bulb and antrum of the stomach. Bleeding was a common presentation of PUD. NSAIDs contributed largely to bleeding complications.

DOI: http://dx.doi.org/10.25130/mjotu.28.2022.01

**Corresponding author E mail* : khaldoon.Abdulrazzak@uoninevah.edu.iq

Introduction:

Peptic ulcer is a common disease of the digestive system. In a review of published studies, the incidence of uncomplicated PUD was approximately 0.1% per year, and the lifetime prevalence is 5%-10% in the general population [1, 2]. Globally, number of new cases of PUD in 2015 was 87.4 million causing 267,500 deaths [3]. Marshall and Warren discovered *helicobacter pylori (H.pylori)* as an etiologic agent of PUD in the early 1980s [4]. Frequent use of aspirin and NSAIDs are associated with the development and complications of PUD [5]. Smoking is an established risk factor for PUD while relation to alcohol consumption is inconsistent [6]. A Swedish report by Malaty HM, et al involving a large cohort of twins found that genetic impact is of moderate importance for predilection to PUD [7]. There is a recent trend of declining prevalence of PUD [8]. The epidemiology of PUD is changing in relation to time and place. The disease presents earlier and affects younger age groups in developing countries which may be attributed to *H.pylori* infection at younger age [9]. It has been observed that the mean age of both affected sexes increased over recent times [10]. In both developed and developing countries, the age groups which are affected by GU are elder than DU. Male: female ratio is widely variable across the world and male gender affection usually predominates. The ratio of DU:GU is changing and shows wider variations in Asians than Caucasians [9]. Classically peptic ulcers are usually single, located in the first part of the duodenum and antrum of the stomach. PUD represents the most common cause of upper gastrointestinal bleeding contributing to about 28% to 59% of all causes [11]. Drugs mainly NSAIDs, aspirin, and anticoagulants contribute largely to peptic ulcer bleeding [12].

Mosul city is the center of Nineveh province. Geographically it is located in north of Iraq; a subtropical, middle east country located in west Asia. The

province is inhabited by more than three million population both urban and rural [13]. Hospitals in Mosul receive patients from the city and also from villages and districts located within the province.

The aim of this study is to elucidate demographic features, endoscopic characteristics, and bleeding complications of PUD in Mosul.

Patients and methods

This study was carried out in the endoscopy unit of Al-Salam General Hospital in Mosul during a period from January 2018 to December 2020. The data were collected from patients records. Registered patient informations included name, age, gender, date of examination, referring source, premedications, clinical indications of endoscopy, drug use, and details of endoscopic findings. The endoscopy unit is an open-access unit that receives patients referred from outpatient clinics, inpatients wards, emergency department, and private clinics. Japanese Olympus white light endoscopes with videoscope (GIF SP-20 and Q40) were used for examination. Only reports of patients attending first time endoscopy with a diagnosis of DU and GU were included. Patients whose biopsy results established a diagnosis of malignancy, patients with healed DU, and gastric outlet obstruction were excluded from the study. Cases with perforated ulcer were managed separately in the emergency surgical department and were not included in our records. Endoscopic diagnosis of peptic ulcers represents mucosal break greater than 3-5 mm with a depth reaching the submucosa [8]. Ulcer location in the stomach were recorded in the following sites (cardia, corpus, lesser curve, antrum, and prepyloric area). DU location included the four anatomical parts of the bulb (anterior, posterior, superior, inferior), and postbulbar area. Number of ulcers in each patient was registered. Size of DU more than 2 cm was recorded and regared as giant ulcer [14]. Endoscopic signs of bleeding were assessed according to Forrest classification as Forrest 1 lesions (a-

brisk bleeding, b-blood oozing), Forrest 2 (a- visible blood vessel at ulcer base, b- blood clot cover, c-hematin base), and Forrest 3 (clean base ulcer, absent signs of bleeding) [15].

Data were analyzed using statistical package for social sciences (SPSS, version 20, USA). Descriptive statistics were applied to calculate the mean± SD, range, percentage, and P value (Level of significance was set at < 0.05) wherever appropriate. Tables and figures were applied to clarify results of statistical analysis.

The study protocol was approved by the Medical Ethics Committee of Ninevah University and Mosul health directorate (license number 67 on 27 April 2018). All patients records included signed written agreement prior to endoscopic examination.

Results

A total of 326 patients (male 223, female 103) were included in the present study. Demographic features of DU and GU are depicted in (Table 1).

Table 1: Patients demography

Variables	Number (%)	P-value
Sample size (patients)	326	
DU	286 (87.7)	
Male	205 (71.7)	
Female	81 (28.3)	
Male/Female ratio: 2.5:1		<0.001
GU	40 (12.3)	
Male	18 (45)	
Female	22 (55)	
Male/Female ratio: 0.8:1		<0.15
Total Male/Female ratio: 2.2:1		<0.001
Mean age (years)		
DU 34.1 ±13.9		
GU 54.9 ±15.3		
Age range (years)		
DU 11-75		
GU 22-90		
Main age group affected (years)		
DU 20-29	99/286 (34.6%)	<0.001
GU 60-69	10/40 (25%)	<0.001

Age groups distribution are shown in (Figure 1). DU affected mainly age group (20-29) years, and for GU (60-69) years (p<0.001).

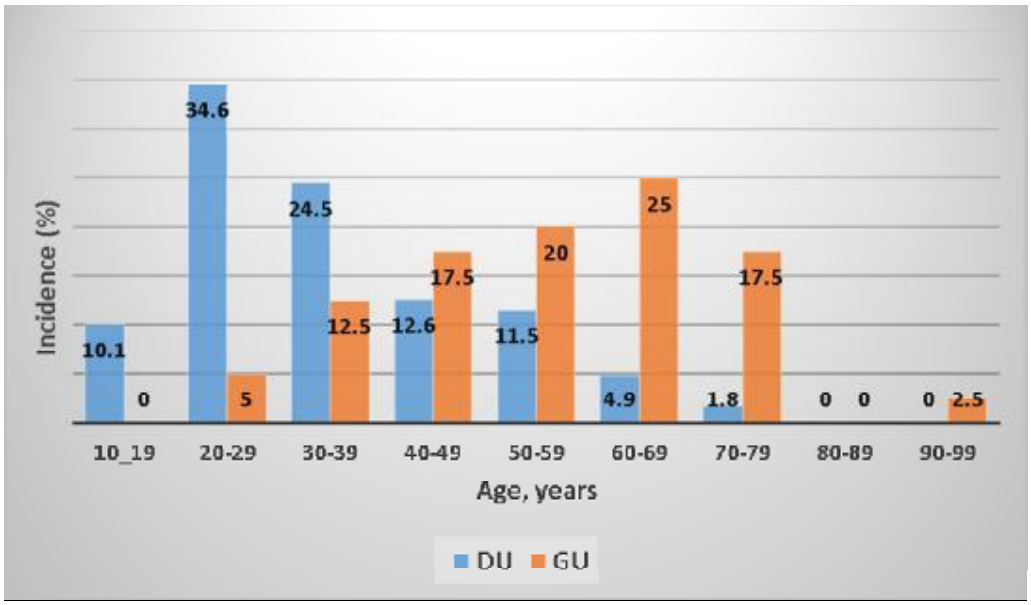

Figure 1: Age group distribution of DU and GU patients

DU main site was the duodenal bulb with predominance of anterior bulbar position; and for GU, the antrum. Ulcers were mainly single (Table 2). Four participants displayed giant DU (two males aged 33 years, 50 years, the former had bleeding ulcer; and two females, aged 50 years, 63 years, the latter was diabetic).

Table 2: Endoscopic findings of peptic ulcers

Variables	Number (%)	P.value
Site of ulcer		
DU	286	
Bulbar	283 (98.9)	
Anterior	133 (47)	<0.001
Posterior	82 (29)	
Superior	37 (13)	
Inferior	31 (11)	
Post bulbar	3 (1.1)	
GU	40	
Antrum, lesser curve	28 (70)	<0.001
Prepyloric	7 (17.5)	
Corpus	4 (10)	

Cardia	1	(2.5)	
Number of ulcers			
DU	286		
Single	257	(89.9)	<0.001
Two	27	(9.4)	
Three	1	(0.4)	
Four	1	(0.4)	
GU	40		
Single	37	(92.5)	<0.001
Two	2	(5)	
Three	1	(2.5)	
Giant DU	4/286 (1.4)		
DU:GU ratio 286/40 7.2:1			<0.001

Among the 326 patients, 65 presented with bleeding (65/326, 19.9%). Drug use (NSAIDs, aspirin, clopidogril) were responsible for (21/65, 32.3%) of bleeding incidents (Table 3).

Table 3: Bleeding status of peptic ulcers

Variables	**DU**		**GU**		**P.value**
	Number 44 (%)		**Number 21 (%)**		
Bleeding rate	44/286	(15.4)	21/40	(52.5)	0.001
Drug use	12/44	(27.3)	9/21	(42.9)	0.001
Endoscopic findings					
Brisk bleeding	4	(9.1)	1	(4.8)	0.2
Blood ooze	8	(18.2)	3	(14.3)	0.01
Visible vessel	3	(6.8)	1	(4.8)	0.35
Blood clot	10	(22.7)	9	(42.9)	0.001
Hematin base	5	(11.4)	2	(9.5)	0.08
Clean base	14	(31.8)	5	(23.8)	0.005

Among 40 GU patients, four were associated with concomitant DU (4/40 10%).

Discussion

PUD displays variable demographic features throughout the world. These variations are partly due to environmental and racial factors of which *H.pylori* prevalence and NSAIDs use play a major role. The current study showed that the mean age of DU patients and the main affected age groups lie in the third and fourth decade of life. A study from Saudia Arabia by El-Munshid found the mean age of DU patients, 35.3 years [16]. Another study from Pakistan by Rashid involving 425 patients, age group 20-30 years was mainly affected [17]. A report from epidemiological study in Sweden by Aro, Mean age of DU patients was 53.3 years [18]. An article by Lam SK reviewing reports of PUD in different regions of the world concluded that Asians present their ulcer symptoms a decade earlier than Caucasians, which may be ascribed to high prevalence of *H.pylori* infection at a younger age [9]. The mean age of GU patients was two decades elder than DU at presentation (55 year versus 34 year $p < 0.001$). Reports from eastern and western countries showed that the main age groups affected by GU were in the 6th decade of life and elder than ages of DU patients [19, 20, 21]. Duodenal mucosa is more vulnerable to the effect of stomach acid compared to gastric mucosa and hence, risk factors for PUD affect duodenal mucosa at younger ages. On the other hand, aging process impairs gastric mucosal defenses that are generated by mucus, bicarbonate, and rich blood supply [22]. Thus, GU occurs at elder ages, especially if NSAIDs are used. The gap between ages of DU and GU patients is narrower in the western developed countries than developing countries in Asia and Africa. Possible explanation is likely due to higher prevalence of *H.pylori* infection with its sequel of DU at younger ages in developing nations [23]. There are wide variations in male:female gender ratio of PUD across different countries and even in the same country. A review article by Tovey F, from India surveying 33 reports, found an average gender ratio of17:1 [24]. In

the UK and Netherlands, the ratio is < 2:1 [19]. There is also temporal variations in the same population, and this ratio in USA has narrowed over the last decades to become even 1:1 [25]. In the present study male:female ratio of DU patients was 2.5:1, which could be explained on higher prevalence of *H.pylori* infection in male gender [26]. In GU, females were more affected with a non-significant difference (p=0.15). Larger study sample of GU will display Gender ratio more precisely. Gender ratio variation is generally higher in the east compared to the west [9]. In recent times the process of civilization which affected most of the populations in the globe, made women share equally men the work load of living and hence, both sexes are exposed to similar environmental risk factors for PUD like *H.Pylori* infection, NSAIDs, smoking, and even social and job stresses, all these factors resulted in narrower gender ratio [8]. There is diverse DU/GU ratio across different geographical locations. A study from China by Li Z, et al reported a ratio of 2.2/1 [27]. Other studies from Senegal documented a ratio of10.7:1 [28], Colombia 1.7:1 [29], and USA 4:1 [30]. In Japan GU is more prevalent than DU, possibly explained on ethnic and environmental risk factors. An epidemiological study by Kawai, et al reported a mean ratio of GU:DU 1.7:1 of nine districts [31]. Over the last decades, there is a trend of declining incidence of DU, partly due to improved sanitation and eradication of H.pylori infection [8]. Wider use of NSAIDs and aspirin contributes largely to rising incidence of GU particularly in the aged populations [32]. DU is mainly located in the duodenal bulb and only less than 5% are located in the postbulbar area [33, 34]. The anterior bulbar wall is more commonly involved by DU relative to other bulbar sites. A comprehensive article by Rau W, et al (2019) explained the predilection sites of PUD, its singular nature and complications, to be due to topographic and geometric distribution of submucosal blood vessels of the stomach and duodenum that results in

relative functional paucity of blood supply in areas with increased metabolic demands [35]. It was reported that postbulbar ulcers are liable for more complications [33]. An Iraqi study by Al-Bahrani, et al involving1320 patients with DU reported single ulcer in 68.3% of cases, and in 45.8%, the ulcers were located on the anterior wall, and other sites of the bulb were less frequently involved [34]. In our series four cases of giant DU (diameter >2 cm) were encountered. These ulcers are liable for complications and carry higher morbidity and mortality [14]. The majority of GUs in the present report were single and located mainly in the antrum, lesser curve, and incisura, which are the classical sites of benign gastric ulcers. Bakir T, et al from Turkey in their series of187 patients with GU reported 89% of ulcers were located in the antrum, 79% were single ulcer, and in 18% associated with DU [36].

Bleeding is a major complication of PUD occurring in15%-20% of patients [37]. The use of drugs mainly NSAIDs contributes to increased frequency of ulcer bleeding in a magnitude of four- to six fold [38]. In the present study 15.4% of DU patients and 52.5% of GU patients presented with bleeding, with a significant difference ($P<0.001$). This result can be explained on the basis of higher frequency use of NSAIDs drugs by GU patients (42.9% of GU patients used drugs versus 27.3% in DU patients $p<0.001$). Our findings confirmed that presentation with bleeding was commoner in GU than DU, and drugs mainly NSAIDs were incriminated as a causal factor of ulceration with its bleeding complication in a rate higher in GU than DU [39]. Florid endoscopic signs of bleeding ulcers are likely to be encountered if endoscopy is performed during the first 24 hour of patient presentation. Any delay of examination, for a variety of reasons, is likely to result in higher frequency of finding ulcers with absent signs of active bleeding [40]. In our series, frequency of finding endoscopic signs of actively bleeding ulcers is less than other signs

(16/65, 24.6% versus 49/65, 75.4%) with significant difference (P <0.001) which is likely due to delayed patient presentation.

Our study is limited by being a single hospital-based study with limited number of patients. Larger multicenter studies across the country are needed for more accurate results.

Conclusions

The present study in Mosul city in Iraq showed that DU affected mainly younger age patients (less than 40 years age), while GU affected older ages (above 50 years age). Males were twicely affected compared to females. DU is much commoner than GU. Ulcers were mainly single. The main site of DU was the bulb and for GU the antrum and lesser curve. Bleeding is a common presentation of PUD and drugs play a major causal role

Acknowledgement

The author would like to express his thanks and gratitudes to the endoscopy medical staff in Al-Salam General Hospital in Mosul for their support, collaboration and care of patients.

References

1. Lin KJ, García Rodríguez LA, Hernández-Díaz S. Systematic review of peptic ulcer disease incidence rates: do studies without validation provide reliable estimates? Pharmacoepidemiol Drug Saf 2011;20(7):718-728. doi:10.1002/pds.2153.
2. Lanas A, Chan FKL. Peptic ulcer disease. Lancet 2017;390:613-624. doi:10.1016/S0140-6736(16)32404-7.
3. GBD 2015 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 310 diseases and injuries, 1990-2015: a systematic analysis for the Global Burden of Disease Study 2015. Lancet 2016;388:1545–1602. doi: 10.1016/S0140-6736(16)31678-6.
4. Marshall BJ, Warren JR.

Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. Lancet 1984;323:1311-15. doi: 10.1016/s0140-6736(84)91816-6.

5. Rafaniello C, Ferrajolo C, Sullo MG, et al. Risk of gastrointestinal complications associated to NSAIDs, low-dose aspirin and their combinations: results of a pharmacovigilance reporting system. Pharmacol. Res. 2016;104:108–114. doi:10.1016/j.phrs.2015.12.026.
6. Rosenstock S, Jørgensen T, Bonnevie O, et al. Risk factors for peptic ulcer disease: a population based prospective cohort study comprising 2416 Danish adults. Gut 2003;52(2):186–193. doi: 10.1136/*gut*.52.2.186.
7. Malaty HM, Graham DY, Isaksson I, et al. Are genetic influences on peptic ulcer dependent or independent of genetic influences for Helicobacter pylori infection. Arch Intern Med.2000;160(1):105-109. doi:10.1001/archinte.160.1.105.
8. Xie X , Ren K, Zhou Z et al. The global, regional and national burden of peptic ulcer disease from 1990 to 2019:a population-based study. BMC Gastroenterology 2022; 22 (58): 1-13. doi.org/10.1186/s12876-022-02130-2
9. Lam SK. Differences in peptic ulcer between East and West. Baillieres Best Practice and Research. Clinical Gastroenterology 2000;14(1):41-52. doi:10.1053/bega.1999.0058.
10. Bardhan KD, Royston C. Time, change and peptic ulcer disease in Rotherham, UK. Dig Liver Dis. 2008;40(7):540-6. doi: 10.1016/j.dld.2008.02.024.
11. Van Leerdam ME. Epidemiology of acute upper gastrointestinal bleeding. Best Pract Res Clin Gastroenterol

2008;22(2):209–24. doi: 10.1016/j.bpg.2007.10.011.

12. Joo MK, Park CH, Kim JS et al. Clinical Guidelines for Drug-Related Peptic Ulcer, 2020 Revised Edition. Gut and Liver 2020;14 (6): 707-726. doi.org/10.5009/gnl20246.
13. Iraq Governorate. http://www.irfad.org/iraq-governorate-ninewa/. Online cited 24 April 2022.
14. Newton EB, Versland MR, Sepe TE. Giant duodenal ulcers. World J Gastroenterol 2008;14 (32):4995-4999. doi: 10.3748/wjg.14.4995.
15. Forrest JA, Finlayson ND, Shearman DJ. Endoscopy in gastrointestinal bleeding. Lancet 1974;2:394-7. doi: 10.1016/s0140-6736(74)91770-x.
16. El-Munshid HA, Dissanayake AD, Al-Breiki H, et al. Duodenal ulcers in the eastern region of saudi Arabia. Annals of Saudi Medicine 1990;10(3),1990: 258-267.
17. Rashid MN, Soomro AM, Channa NA. Prevalence of different types of peptic ulcer disease and treatment modalities used by patients in hyderabad, Sindh. Pak J Physiol 2016;12(1):6-9.
18. Aro P , Storskrubb T, Ronkainen J, et al. Peptic Ulcer Disease in a General Adult Population The Kalixanda Study: A Random Population-based Study. Am J Epidemiol 2006;163 (11): 1025–1034. doi: 10.1093/aje/kwj129.
19. Groenen MJM, Kuipers EJ, Hansen BE et al. Incidence of duodenal ulcers and gastric ulcers in a Western populations: Back to where it started. Can J Gastroenterol.2009;23(9):604-608. doi: 10.1155/2009/181059.
20. Ahmed W, Qureshi H, Alam SE, et al. Gastric Ulcer in Karachi. J Pak Med Assoc. 1992;42(9):207-10.
21. Ramírez-Ramos A, Watanabe-Yamamoto J, et al. Decrease in

prevalence of peptic ulcer and gastric adenocarcinoma at the Policlínico Peruano Japonés, Lima, Peru, between the years 1985 and 2002. Analysis of 31,446 patients. Acta Gastroenterol Latinoam 2006;36(3):139-146.

22. Guslandi M, Pellegrini A, Sorghi M. Gastric mucosal defences in the elderly. Gerontology1999; 45(4):206-8. doi: 10.1159/000022088.

23. Khedmat H, Afshar RK, Agah S, et al. Helicobacter pylori infection in the general population: a middle Eastern perspective.Caspian J Intern Med 2013;4:745-53.

24. Tovey F. Peptic ulcer in India and Bangladesh. Gut 1979;20 (4):329-347. doi: 10.1136/gut.20.4.329.

25. Kurata JH, Haile BM, Elashoff JD. Sex differences in peptic ulcer disease. Gastroenterology 1985;88(1 pt 1):96-100. doi: 10.1016/s0016-5085(85)80139.

26. Ibrahim A, Morais S, Ferro A, et al. Sex-differences in the prevalence of Helicobacter pylori infection in pediatric and adult populations: Systematic review and meta-analysis of 244 studies. Dig Liver Dis 2017; 49(7):742-749. doi: 10.1016/j.dld.2017.03.019.

27. Li Z, Zou D, Ma X, et al. Epidemiology of peptic ulcer: endoscopic results of the systemic investigation of gastrointestinal disease in China. Am J Gastroenterol. 2010;105 (12):2570-7. doi: 10.1038/ajg.2010.324.

28. Mbengue M, Seck A, Dia D, et al. Gastroduodenal peptic ulcer: descriptive study. Dakar Med.2003;48(3):176-80.

29. Arango LAA, Boada DG, Cuadrado MPV. Epidemiological evolution of acid peptic disease in an endoscopic diagnostic centre in Bogotâ from 1993 and 2007. Rev Col Gastroenterol

2010;25(2):135-148.

30. Kurata JH, Haile BM. Epidemiology of peptic ulcer disease. Clinics in Gastroenterology1984;13(2):289-307.
31. Kawai K, Shirakawa K, Misaki F, et al. Natural History and Epidemiologic Studies of Peptic Ulcer Disease in Japan. Gastroenterology 1989;96(2):581-5. doi: 10.1016/s0016-5085(89)80053-8.
32. Lee MW, MD, Katz PO. Nonsteroidal Antiinflammatory Drugs, Anticoagulation, and Upper Gastrointestinal Bleeding. Clin Geriatr Med 2021;37: 31–42 doi.org/10.1016/j.cger.2020.08.004.
33. Yamane T, Umeda A, Shimao H. Analysis of Recent Cases of Postbulbar Duodenal Ulcer in Japan. Open Journal of Gastroenterology 2017;7(10**)**:271-278. doi: 10.4236/ojgas.2017.710028.
34. Al-Bahrani ZR, Kassir ZA, Al-Doree W. The location and multiplicity of chronic duodenal ulcer (a study of 1320 patients in Iraq). Gastroenterol Jpn 1980;15(6):539–542. doi: 10.1007/bf02773756.
35. Rau W, Hohaus C, Jessen E. A Differential Approach to Form and Site of Peptic Ulcer. Scientific Reports 2019; 9(8683):1-21. doi: 10.1038/s41598-019-44893-x.
36. Bakir T, Kazancioglu S, Özoran Y. Evaluation of the Gastric Ulcer Patients in Black sea Region of Turkey. Journal of Islamic Academy of Sciences 1988;1(1):67-69.
37. Ramakrishnan K, Salinas RC. Peptic Ulcer Disease. Am Fam Physician 2007;76(7):1005-1012.
38. Arlt GD, Leyh M. Incidence and pathophysiology of peptic ulcer bleeding. Langenbecks Arch Surg 2001;386(2):75-81. doi:

10.1007/s004230000193.

39. Russell R I. Non-steroidal anti-inflammatory drugs and gastrointestinal damage — problems and solutions. Postgrad Med J 2001;77:82–88. doi: 10.1136/pmj.77.904.82 .

40. Lau JYW, Yu Y, Tang RSY et al. Timing of Endoscopy for Acute Upper Gastrointestinal Bleeding. N Engl J Med 2020;382 (14):1299-308. doi:10.1056/NEJMoa1912484.