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Treatment tactics for gastric erosive-ulcerative bleeding on the background of liver cirrhosis

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Analysing the polymorphism of etiological factors that contribute to the development of erosive-ulcerative gastric lesions in patients with liver cirrhosis (acid-peptic factors, transformation of venous blood flow, immune complexes, etc.), it should be noted that portal hypertension is the most important cause of these lesions. Bleeding from erosive-ulcerative gastric lesions is a direct cause of death, occupying the first place in the structure of mortality and far exceeding ulcer perforations in this regard. Mortality in the first massive acute gastric bleeding reaches 50 %, after the second — 70 %, in hepatic coma — 80 % or more.

Objective — to improve the treatment outcomes for patients with erosive and ulcerative bleeding in the stomach due to liver cirrhosis.

Materials and methods. The treatment outcomes of 192 patients with the hepatic form of portal hypertension (liver cirrhosis) who were hospitalised in the therapeutic and surgical departments, which are the clinical bases of the Department of Surgery No. 2 at Bogomolets National Medical University, were studied for the period from 2005 to 2023. During upper endoscopy, degenerative changes in the gastric mucosa were detected in 94 (48.9 %) patients. Specifically, gastric erosions were observed in 31 (33.0 %) patients, gastric ulcers in 34 (36.2 %), and portal gastropathy in 29 (30.8 %) patients. A total of 88 (46.6 %) patients with liver cirrhosis and acute gastric bleeding were admitted to the surgical department as an emergency. According to the Child-Pugh scale, the vast majority of patients were at the stage of sub- and decompensation (71 patients, or 89.1 %). In 41 (45.2 %) patients, gastric cancer was attributed to gastric erosive and ulcerative lesions: erosive gastritis — in 20 (48.8 %) patients and gastric ulcer — in 21 (51.2 %) patients.

Results. Enhancing the protective properties of the mucous-bicarbonate barrier of the gastric mucosa as part of pathogenetic conservative treatment schemes can reduce the influence of this mechanism on the development of hemorrhagic complications in 89.4 % of patients. The risk of bleeding in this group of patients with liver cirrhosis is 15.1 %, which requires the search for new methods of conservative treatment. In 79.6 % of cases, primary endoscopic hemostasis of bleeding erosive-ulcerative gastric lesions was effective when combined with drug therapy. For 53.1 % of patients, it proved to be the final treatment. This enabled a reduction in overall mortality to 22.4 % from 50 % ($p < 0.05$) and, if necessary, a delay in surgical intervention. Out of the total number of patients, 19 (38.8 %) patients required surgical intervention, with 73.7 % of them undergoing urgent surgery and 26.3 % undergoing delayed surgery. When considering operative methods for peptic ulcers complicated by hemorrhage, preference should be given to organ- and function-preserving interventions. They yield more favourable immediate outcomes for this extremely challenging group of patients (postoperative mortality was reduced to 26.3 % vs. 50.0 % ($p < 0.05$).

Conclusions. Acute gastric bleeding resulting from erosive-ulcerative gastric lesions, which are complications of liver cirrhosis, can significantly impair the health of patients. However, by implementing modern treatment technologies, such as surgical procedures, it is feasible to achieve a 50 % reduction in both overall and postoperative mortality.

Keywords
endoscopic hemostasis, hemorrhage, liver cirrhosis, stomach ulcer.

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Acute gastric bleeding (AGB) complicates the course of several diseases of the upper gastrointestinal tract (above the ligament of Treitz), and its frequency is 50—150 cases per 100,000 people per year [10]. Thus, in the United States, more than 300.000 people are hospitalised for AGB annually [3]. In the total structure of bleeding from the upper gastrointestinal tract, peptic ulcer disease accounts for 41.0 % (gastric ulcer accounts for 23.1 %), and erosive lesions account for 34.9 % [11]. Liver cirrhosis, as one of the most common forms of portal hypertension syndrome accompanied by the development of varicose veins of the oesophagus and cardiac part of the stomach, often causes their rupture and the development of profuse AGB (10.1 %) [1]. However, it has been found that varicose veins are a source of bleeding in only 42.0 % of patients with liver cirrhosis, and other sources of AGB (58 % of cases) are gastric erosion and ulcers [2].

Bleeding from gastric erosive and ulcerative lesions (GEUL), as a direct cause of death, ranks first in the structure of mortality and far exceeds ulcerative perforations. Despite modern advances in medical and endoscopic hemostasis, mortality rates in AGB remain consistently high at 8—10 % [8]. It is well known that primary profuse AGB causes the death of every fourth patient, while only about 50 % of patients survive after rebleeding. It is in portal hypertension that AGB is profuse 5 times more often compared to peptic ulcer disease due to serious coagulation disorders that occur in chronic liver disease. Mortality in the first massive AGB reaches 50 %, after the second — 70 %, in hepatic coma — 80 % or more [9].

The introduction of potent antisecretory drugs into clinical practice has expanded the possibilities for conservative treatment of AGB. The increased understanding of the pathogenesis of GEUL in individuals with liver cirrhosis has facilitated the broader implementation of medications that help restore the mucosal-bicarbonate barrier of the gastric mucosa [4, 6]. Advancements in endoscopic technique have long allowed the widespread use of fibroesophagogastroscopy (FEGS) for both diagnostic and therapeutic purposes. This includes the use of various types of vascular coagulation, local injection therapy with vasoconstrictors or sclerosing drugs, clipping, etc. [7]. Today, there is a clear doctrine that uncomplicated gastroduodenal ulcers require only conservative treatment, provided that histological confirmation of benign gastric lesions is available. However, the number of emergency interventions for AGB does not tend to decrease sharply, especially in hospitals that lack appropriate endoscopic equipment and trained personnel.

Indications for emergency surgery in case of AGB are profuse bleeding when it is impossible to stop it endoscopically and the combination of bleeding with ulcer perforation (6.1—10 %) [12]. The choice of surgical treatment for AGB remains unclear. Up to now, the classic Billroth — I gastric resection remains the operation of choice, and in some cases, Billroth — II gastric resection. However, this issue, especially with concomitant liver cirrhosis, is quite controversial, requiring the search for new technical solutions.

Objective — to improve the treatment outcomes for patients with erosive and ulcerative bleeding in the stomach due to liver cirrhosis.

Materials and methods

The treatment outcomes of 192 patients with hepatic portal hypertension (liver cirrhosis) hospitalised in the therapeutic and surgical departments, which are the clinical bases of the Department of Surgery No. 2 at Bogomolets National Medical University, in the period from 2005 to 2023, were studied. Of the 192 patients, 108 (56.3 %) were men and 84 (43.7 %) were women. The age of the patients ranged from 28 to 62 years, with an average age of 43.2 ± 2.3 years. The cause of liver cirrhosis in 81 (42.2 %) patients was viral hepatitis, in 65 (33.9 %) — alcoholism, and in 46 (23.9 %) — toxic (including medications) factors.

The examination of patients included, in addition to physical methods, laboratory blood tests, ultrasound examination of the abdominal cavity and FEGS. The functional reserve of the liver was assessed by the Child-Pugh scale according to changes in bilirubin, albumin, prothrombin index, ascites and encephalopathy. Patients were divided into three groups depending on the stage of the disease: compensation (61 patients, or 31.8 %), subcompensation (81 patients, or 42.2 %) and decompensation (50 patients, or 26.0 %). Oesophageal varices were diagnosed in 163 (84.9 %) patients, splenomegaly — in 84 (43.8 %). Ascites was detected in 119 (62.0 %) patients. In the vast majority of patients (64 patients, or 53.8 %), ascites was detected at a stable stage, and in 42 (35.3 %) patients — at a progressive stage, and only in 13 (10.9 %) patients, it was transient. Hypersplenism occurred in 69 (35.9 %) patients, and jaundice in 44 (22.9 %).

Degenerative changes in the gastric mucosa during FEGS were detected in 94 (48.9 %) patients: gastric erosion in 31 (33.0 %) patients, gastric ulcers in 34 (36.2 %), and portal gastropathy in 29 (30.8 %) patients. Among the gastric ulcerative lesions, Johnson's type 1 ulcers predominated (26 patients, or 76.5 %), 2 (11.7 %) patients had type 2 ulcers, and 4 (11.8 %) patients had type 3 ulcers.

Chronic gastric lesions accounted for 64.7 % (22 patients) and acute lesions for 35.3 % (12 patients). In 21 (61.8 %) patients, the ulcerative defect was large (2—3 cm), and in 6 (17.6 %) patients, a giant ulcer (over 3 cm) was found.

The surgical department admitted 88 (46.6 %) patients with cirrhosis with AGB as an emergency, and according to the Child–Pugh scale, the vast majority of patients were at the stage of subcompensation and decompensation (71 patients, or 89.1 %). GEUL was the cause of AGB in 41 (45.2 %) patients: erosive gastritis — in 20 (48.8 %) patients, gastric ulcer — in 21 (51.2 %) patients. In 23 (56.1 %) patients, the first clinical sign of AGB was dizziness, and in 38 (92.7 %) — it was vomiting of blood or «coffee grounds». Melena was detected in all the patients studied. Symptoms of hemorrhagic shock (acute pallor of the skin, heart rate acceleration, cold clammy sweat, hypotension, and short-term loss of consciousness) were recorded in 26.0 % of cases (19 patients). The degree of bleeding severity was determined by changes in the shock index, hematocrit, hemoglobin, erythrocytes, heart rate, and arterial and central venous pressure. Mild AGB (circulating blood volume (CBV) deficit below 20 %) was detected in 21 (23.9 %) patients, moderate AGB (CBV deficit 20—30 %) in 40 (45.5 %) patients, and severe AGB (CBV deficit over 30 %) in 27 (30.6 %) patients.

An additional 8 patients were transferred from the therapeutic department who developed AGB from GEUL while undergoing conservative treatment of liver cirrhosis: in 5 patients, the cause of bleeding was ulceration, and in 3 patients, erosion. Thus, the total number of patients with AGB caused by GEUL in the setting of liver cirrhosis was 49 out of 94 (52.1 %).

The choice of treatment method for bloody GEUL depended on the intensity of bleeding (according to the Forrest endoscopic classification of GI ulcers), the degree of blood loss, the patient's age, and the general condition of the body. We widely used local endoscopic hemostasis with the help of hemostatic solutions, argon-plasma coagulation, and the clipping of bleeding vessels. Conservative methods included restoration of CBV deficiency, hemostatic therapy, and anti-ulcer treatment. Given the severe coagulation system disorders brought on by liver failure, it is important to note the high efficacy of somatostatin analogues among the hemostatic medications. Anti-ulcer treatment included the use of modern anti-secretory drugs (especially proton pump blockers, as well as cytoprotectors and repair agents). Using a direct urease test during FEGS, we found that about 60 % of patients with gastric ulcers had concomitant helicobacteriosis. This finding necessitated supplementary anti-helicobacter therapy, according to the existing Maastricht consensus 1—6. The efficacy of eradicating the microorganism was checked using a direct respiratory (^{13}C-urea) test one month after the end of the anti-relapse course.

A total of 19 (38.8 %) patients underwent surgery for AGB caused by a gastric ulcer. Of these, 14 (73.7 %) patients underwent surgery (emergency surgery was performed in 6 patients and urgent surgery in 8 patients). Early-delayed surgical interventions were performed in 5 (26.3 %) patients. The vital indication for surgical treatment of patients was profuse bleeding with unsuccessful endoscopic and medical hemostasis, and the absolute indication was recurrence of bleeding in the hospital. The tactics of surgical treatment of patients with a bleeding gastric ulcer depended on its localization (type of ulcer according to Johnson), course (acute or chronic), severity of bleeding, presence of other complications of peptic ulcer disease (penetration or perforation), and general condition of the body (age, severity of concomitant disease). Among the radical surgical interventions, 15 (83.3 %) patients preferred economical gastric resection, which involves the removal of the ulcers with an inflammatory periulcer infiltrate and immediate histological examination of the extracted material. This organ- and function-preserving approach is based on our own morphological analysis of the intraoperative material as well as gastrobiopsy data regarding the actual incidence of gastric ulcer cancer, which does not exceed 1 % [5]. In 3 (16.7 %) patients with AGB, due to the extremely severe condition, palliative intervention was performed, which consisted of stopping the bleeding by stitching the bleeding vessel in the ulcer.

All the operated patients had a type 1 bloody gastric ulcer that exceeded 3 cm in diameter and was accompanied by a large inflammatory infiltrate. We performed an economical (plastic) gastric resection with our own modification, which consisted of partial mobilisation of the stomach along the small curvature with preservation of the Latarjet nerve and radical excision of the ulcer along with the surrounding inflammatory infiltrate (Figure). Through the gastrotomy opening, we ensured the proper patency of the oesophagus and passed a nasogastric tube through the pyloric sphincter. If the tissue defect was substantial, it was sutured in a sequential manner. First, the upper and lower edges of the defect were sutured longitudinally to ensure more accurate and uniform suturing of the main defect. This was done using a double-row suture. Then, the suturing was continued in a transverse direction. This modification was performed on 10 (66.7 %) patients.

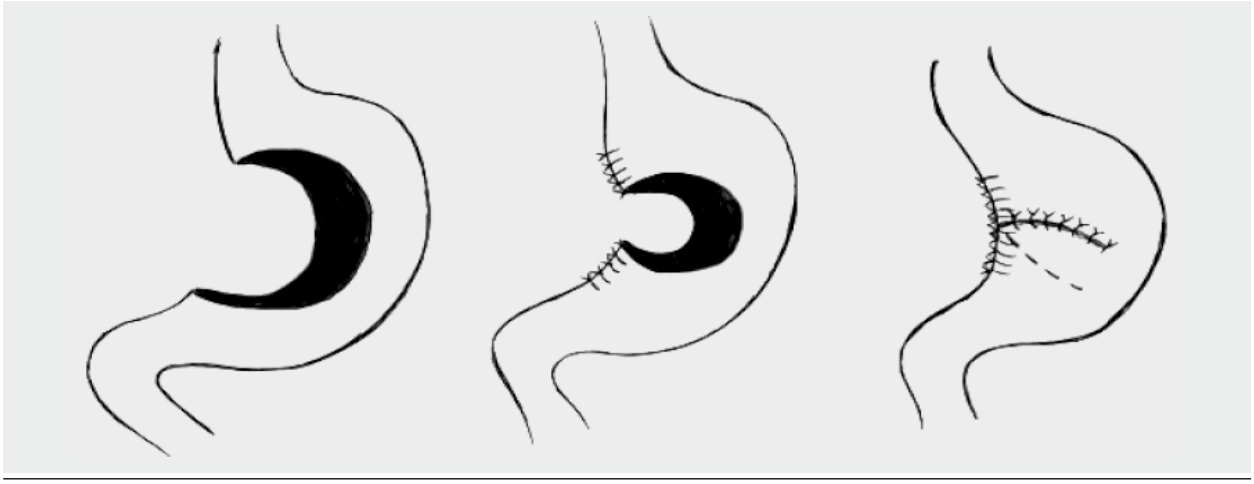

Figure. **Stages of plastic gastric resection**

In 5 (33.3 %) patients, the operation revealed a combination of AGB with penetration of a giant gastric ulcer into the small omentum and involvement of the Latarjet nerve in the inflammatory infiltrate. During the radical removal of the ulcer, this nerve was usually transected. To ensure the normal evacuation function of the pyloroduodenal junction, this operation was supplemented with segmental pyloroplasty according to Deaver — Burden (without violating the integrity of the gastric mucosa).

Results

Conservative pathogenetic treatment of degenerative changes in the gastric mucosa without AGB was effective in 45 of 53 patients (84.9 %), including 29 (100 %) patients with portal gastropathy, 8 (72.7 %) patients with gastric erosions, and 8 (61.5 %) patients with gastric ulcers.

In 8 (15.1 %) patients, despite treatment, there was an AGB (ulcers — 5, erosions — 3), which was stopped endoscopically in 4 (50 %) patients. Another 4 patients underwent surgery after unsuccessful attempts to stop the bleeding of gastric ulcers.

A total of 41 (45.2 %) patients with liver cirrhosis were admitted to the surgical department on an emergency basis with GEUL-induced AGB. Emergency surgery after diagnosis verification by FEGS was performed in 6 patients with profuse bleeding, and emergency intervention after attempts at endoscopic and medical hemostasis was performed in 4 more patients. Endoscopic hemostasis of AGB, along with medical therapy, effectively stopped bleeding in 31 (75.6 %) patients, including 20 (100 %) patients with erosive gastritis and 11 (52.4 %) with gastric ulcer. In 22 (53.6 %) patients, in parallel with stopping AGB, healing of the morphological substrate of the disease (ulcers and/or erosion) was achieved within 4—8 weeks of conservative treatment. However, during the first week after the bleeding was stopped, its recurrence occurred in the remaining 9 patients, and in 4 (44.4 %) patients, it was stopped endoscopically. However, in 5 patients with gastric ulcers (55.6 %), repeated attempts to stop the bleeding were ineffective, and therefore these patients underwent surgery (early-delayed operations).

Primary endoscopic hemostasis of bleeding gastric erosive and ulcerative lesions, supplemented with drug therapy, was effective in 31 of 41 patients admitted to the surgical department with AGB (75.6 %) and in all 8 patients (100 %) transferred from the therapeutic department, for a total of 79.6 %. Additionally, it was final in 26 patients (22 and 4, respectively), resulting in 53.1 % efficiency.

A total of 19 (38.8 %) patients underwent surgery for AGB caused by a gastric ulcer. Among these, 14 (73.7 %) patients underwent surgery urgently (emergency surgery was performed in 6 patients and urgent surgery in 8 patients). Early-delayed surgical interventions were performed in 5 (26.3 %) patients. Good results of surgical treatment were achieved in 14 (73.7 %) patients, including after gastric resection in 87.5 % of cases.

The overall mortality rate in liver cirrhosis complicated by AGB from GEUL was 22.4 % (11 patients) vs. 50 % [9] ($p < 0.05$). Among the 6 patients treated conservatively, death occurred due to a combination of severe aggravating factors: hemorrhagic shock and severe liver failure resulting from the decompensated stage of liver cirrhosis.

The postoperative mortality rate was 26.3 % (5 patients died) vs. 60 % [9] ($p < 0.05$). Three patients underwent simple stitching of the bleeding vessel due to their extremely severe initial condition. Despite the short duration of the operation and surgical control of AGB, the patients died immediately

after the operation from decompensation caused by liver cirrhosis. The cause of death of radically operated patients in the early postoperative period was progressive hepatic encephalopathy.

Discussion

Analysing the polymorphism of etiologic factors that cause the development of GEUL in patients with liver cirrhosis (acid-peptic factors, venous blood flow transformation, immune complexes, etc.), it should be noted that portal hypertension is the most important cause of the latter. The incidence of erosions and ulcers is 48.9 % [4]. In this regard, enhancing the protective properties of the mucous-bicarbonate barrier of the gastric mucosa as part of pathogenetic conservative treatment schemes can reduce the influence of this mechanism on the development of hemorrhagic complications (84.9 %). The risk of bleeding in this group of patients with liver cirrhosis is 15.1 %, which requires the search for new methods of conservative treatment.

According to literature data, GEUL is the cause of bleeding in liver cirrhosis in 58 % of cases [2]. Mortality in the first massive AGB reaches 50 %, after the second — 70 %, in hepatic coma — 80 % or more [9].

The use of various endoscopic methods for hemostasis is quite effective (79.6 %) and enables a reduction in overall mortality to 22.4 % from 50 % ($p < 0.05$) and, if necessary, a delay in surgical intervention.

When considering operative methods for peptic ulcers complicated by hemorrhage, preference should be given to organ- and function-preserving interventions. They yield more favourable immediate outcomes for this extremely challenging group of patients (postoperative mortality was reduced to 26.3 % vs. 50.0 % ($p < 0.05$).

Conclusions

Gastric erosive and ulcerative lesions account for 48.9 % of patients with liver cirrhosis, and in 52.1 % of cases, they are complicated by bleeding.

An increase in transcapillary filtration during the oedematous ascitic stage of liver cirrhosis is the primary cause of gastric erosive and ulcerative lesions. Due to venous stasis in the gastric mucosa, this increase negatively affects the state of its mucosal bicarbonate barrier, resulting in a significant number of erosions and ulcers.

The use of complex conservative treatment in 89.4 % of cases in patients with gastric erosive and ulcerative lesions on the background of cirrhosis allowed the elimination of the morphological substrate of the disease (healing the ulcer, erosion) within 4—8 weeks and prevented the development of hemorrhagic complications.

In 79.6 % of cases, primary endoscopic hemostasis of bleeding erosive-ulcerative gastric lesions was effective when combined with drug therapy. For 53.1 % of patients, it proved to be the final treatment.

Acute gastric bleeding resulting from erosive-ulcerative gastric lesions, which are complications of liver cirrhosis, can significantly impair the health of patients. However, by implementing modern treatment technologies, such as surgical procedures, it is feasible to achieve a 50 % reduction in both overall and postoperative mortality.

Declaration of interests

The authors declare no conflict of interest.

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Ethics approval and written informed consents statements

The assessment and usage of all clinical data was approved and permitted before the study by the ethics committee of Bogomolets National Medical University. The study protocol conformed to the ethical guidelines of the «World Medical Association Declaration of Helsinki — Ethical Principles for Medical Research Involving Human Subjects» adopted by the 18th WMA General Assembly, Helsinki, Finland, June 1964 and amended by the 59th WMA General Assembly, Seoul, South Korea, October 2008.

Written informed consent was obtained from all individual participants included in the study.

Authors contributions

I. V. Kolosovych: conception and design of the work, drafting the manuscript, critical revision of the manuscript; H. Uzun: data collection, data analysis and interpretation, drafting the manuscript.

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Лікувальна тактика при шлункових ерозивно-виразкових кровотечах на тлі цирозу печінки

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Аналіз поліморфності етіологічних чинників, які спричиняють розвиток ерозивно-виразкових уражень шлунка у хворих на цироз печінки (кислотно-пептичний чинник, трансформація венозного кровотоку, імунні комплекси тощо), виявив, що саме портальна гіпертензія є найважливішою причиною виникнення останніх. Кровотеча з ерозивно-виразкових уражень шлунка, як безпосередня причина смерті, посідає перше місце в структурі летальності. При першій масивній гострій шлунковій кровотечі летальність становить 26—50 %, після другої — 70 %, при печінковій комі ≥ 80 %.

Мета — поліпшити результати лікування хворих з ерозивно-виразковими кровотечами шлунка на тлі цирозу печінки.

Матеріали та методи. Проаналізовано результати лікування 192 пацієнтів із печінковою формою портальної гіпертензії (цироз печінки), госпіталізованих у терапевтичне та хірургічне відділення, які є клінічними базами кафедри хірургії № 2 Національного медичного університету імені О. О. Богомольця, в період з 2005 до 2023 р. Серед пацієнтів було 108 (56.3 %) чоловіків та 84 (43.7 %) жінки. Вік пацієнтів — від 28 до 62 років, середній вік — (43,2 ± 2,3) року. Дегенеративні зміни з боку слизової оболонки шлунка під час фіброезофагогастродуоденоскопії виявлено у 94 (48,9 %) хворих: у 31 (33,0 %) — ерозії шлунка, у 34 (36,2 %) — виразки шлунка, у 29 (30,8 %) — портальну гастропатію. У хірургічне відділення в порядку швидкої допомоги госпіталізовано 88 (46,6 %) пацієнтів із цирозом печінки та гострою шлунковою кровотечею. Більшість хворих (89,1 %) перебували в стадії субкомпенсації та декомпенсації за шкалою Child-Pugh. Ерозивно-виразкові ураження шлунка були причиною гострої шлункової кровотечі в 41 (45,2 %) хворого: ерозивний гастрит — у 20 (48,8 %), виразка шлунка — у 21 (51,2 %).

Результати. Посилення захисних властивостей слизово-бікарбонатного бар'єра слизової оболонки шлунка при патогенетичному консервативному лікуванні дало змогу зменшити вплив цього механізму на розвиток геморагічних ускладнень у 89,4 % випадків. Ризик розвитку кровотечі в цій групі хворих становить 15,1 %, що потребує пошуку нових методів консервативного лікування. Первинний ендоскопічний гемостаз кровоточивих ерозивно-виразкових уражень шлунка, було доповнено медикаментозною терапією в 79,6 % випадків, у 53,1 % хворих він був остаточним, що дало змогу знизити загальну летальність з 50,0 до 22,4 % ($p < 0,05$) і за потреби відтермінувати проведення оперативного втручання. Загалом прооперовано 19 (38,8 %) пацієнтів із гострою шлунковою кровотечею, спричиненою виразкою шлунка: в ургентному порядку — 73,7 % пацієнтів, ранні відстрочені оперативні втручання виконано 26,3 % хворих. Із оперативних методів при виразковій хворобі, ускладненій гострими шлунковими кровотечами, перевагу слід віддавати органощадним операціям та втручанням, що зберігають функції. Це поліпшить безпосередні результати лікування цієї надзвичайно складної категорії пацієнтів (післяопераційну летальність було знижено з 50,0 до 26,3 % ($p < 0,05$).

Висновки. Попри тяжкий стан хворих із гострими шлунковими кровотечами ерозивно-виразкового генезу, спричинений ускладненнями цирозу печінки, застосування сучасних технологій лікування, зокрема хірургічних, дало змогу зменшити загальну та післяопераційну летальність удвічі.

Ключові слова: ендоскопічний гемостаз, кровотеча, цироз печінки, шлункова виразка.

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