

UDK: 616.34-002.44-005.1-07-08: 616.3-092

PATHOPHYSIOLOGICAL POINT OF VIEW IMPAIRED HEMOSTASIS IN GASTRODUODENAL BLEEDING OF ULCER ETIOLOGY



Alimov Sukhrob Usmonovich
Tashkent Medical Academy, Republic of Uzbekistan, Tashkent

ЯРА ЭТИОЛОГИЯЛИ ГАСТРОДУОДЕНАЛ ҚОН КЕТИШДА ГЕМОСТАЗ БУЗИЛИШИННИНГ ПАТОФИЗИОЛОГИК ҚАРАШЛАРИ

Алимов Сухроб Усмонович
Тошкент тиббиёт академияси, Ўзбекистон Республикаси, Тошкент ш.

НАРУШЕНИЯ ГЕМОСТАЗА ПРИ ГАСТРОДУОДЕНАЛЬНЫХ КРОВОТЕЧЕНИЯХ ЯЗВЕННОЙ ЭТИОЛОГИИ С ПАТОФИЗИОЛОГИЧЕСКОЙ ТОЧКИ ЗРЕНИЯ

Алимов Сухроб Усмонович
Ташкентская медицинская академия, Республика Узбекистан, г. Ташкент

e-mail: alimov.suxrob@tma.uz

Резюме. Қон кетиши билан асоратланган меъда ва ўн икки бармоқ ичак яраси мавжуд 119 нафар беморлар текиширилди. I даражали қон кетиши билан клиникага тушган беморларда тромбоцитлар миқдори $185 \pm 8,0 \times 10^9/\text{л}$ ни ташиқил қилди. II гуруҳдаги беморларда қон кетишининг биринчи суткасида веноз қон таркибидаги тромбоцитлар миқдори $268,13 \pm 6,13 \times 10^9/\text{л}$ ни ташиқил этди, 3 кун да эса $157 \pm 4,5 \times 10^9/\text{л}$ гача камайган. 7 суткага келиб бу кўрсаткич $245,5 \pm 5,7 \times 10^9/\text{л}$ га ошган. 3 гуруҳ беморларда кўрсаткичлар қўйидагича ўзгарди: тромбоцитлар миқдори $130 \pm 3,5 \times 10^9/\text{л}$, 3 суткада эса уларнинг камайиши кузатилди, лекин фарқ сезиларли бўлмади. Биринчи суткада қисман фаол тромбопластин вақти (ҚФТВ) $25,4 \pm 1,5$ сек. гача ўзгарди кейинчалик 7 суткага келиб назорат гуруҳи билан солиштирганда $30,0 \pm 2,0$ сек. бўлганини кўришимиз мумкин.

Калит сўзлар: меъда яра касаллиги, ўн икки бармоқ ичак яра касаллиги, қон кетиши, гемостаз, тромбоцитлар агрегацияси.

Abstract: 119 patients with gastric and duodenal ulcer with complicated bleeding. At the time of admission to the clinic in patients with the first degree of blood loss, the level of platelets was $185 \pm 8,0 \times 10^9/\text{l}$. In patients of group II, on the first day of bleeding, the content of platelets in the venous blood was $268,13 \pm 6,13 \times 10^9/\text{l}$ with a decrease on the 3rd day to $157 \pm 4,5 \times 10^9/\text{l}$. By the 7th day, this indicator increased to $245,5 \pm 5,7 \times 10^9/\text{l}$. In patients of group III, it changed in the following way: the number of platelets was $130 \pm 3,5 \times 10^9/\text{l}$, and on the 3rd day their decrease was noted, but the difference with the initial parameters. On the first day of bleeding, activated partial thromboplastin time was up to $25,4 \pm 1,5$ sec. with its subsequent increase on the 7th day up to $30,0 \pm 2,0$ seconds in comparison with the control group.

Key words: gastric ulcer, bleeding, hemostasis, duodenal ulcer, platelet aggregation.

Introduction. In recent years, the frequency of gastro duodenal ulcerative bleeding (UGHB) has been increasing among the population. At the same time, the severity of a patient's condition with UHD is determined by the intensity and volume of blood loss, the age of the patients, and the presence of concomitant diseases in them [7]. Despite the successes in conservative, endoscopic and surgical treatment (the use of H₂-blockers, proton pump inhibitors, anti-Helicobacterial drugs, laser therapy, etc.), the number of patients with complicated peptic ulcer disease has

not decreased over the past decades. Healed ulcers recur in 60-90% of patients, and 50-60% develop complications such as bleeding, perforation and stenosis [3,4]. The successes achieved in the conservative treatment of peptic ulcer disease have led to a reduction in the number of surgical interventions for this pathology. along with that, such a formidable complication as gastrointestinal bleeding accounts for 15-25% of all complications of peptic ulcer disease and requires surgeons to develop new tactical approaches. [1.6]. In spite of the significant heterogene-

ity of results among different trials, all authors assessed the frequency of rebleeding and the effectiveness of hemostasis. [2]. Bleeding causes profound changes in the hemostatic system.

The aim of the study. To study the violation of the hemostasis system depending on the degree of blood loss in patients with gastric ulcer and duodenal ulcer complicated by bleeding.

Materials and methods. Under surveillance were 119 patients with gastric ulcer and duodenal ulcer with complicated bleeding.

All patients were hospitalized at the clinic of surgical diseases of the Tashkent Medical Academy (based on the city clinical hospital No. 1) for the period 2019-2021.

The patients were divided into 3 groups:

I - the main group - 119 patients;

II - Control group (practically healthy persons) with 20 people.

Patients of the main group were divided into three subgroups:

The first group includes patients with the first degree of blood loss;

In the second - patients with the second degree of blood loss;

And, the last group includes patients with the third degree of blood loss.

At the same time, the duration of bleeding in patients before hospitalization was taken into account. The study of the hemostasis system was carried out upon admission to the clinic. The study of the vascular - platelet link of hemostasis included counting the number and induced platelet aggregation. The parameters of coagulation hemostasis included the determination of activated partial thromboplastin time (phase I of thromboplastin formation) (APTT), phase II of thrombin formation (prothrombin index), phase III of fibrin formation (plasma fibrinogen content, thrombin time). The data obtained were processed statistically using the « Statistica 10.0» software package.

Results. Studies have shown that at the time of admission to the clinic of patients with the first degree of blood loss, the level of platelets was $185 \pm 8.0 \times 10^9/l$ compared with the control group, and on the 3rd day their increase was noted, but the difference with the initial parameters was significant ($P \leq 0.05$). On the first day, insignificant primary hyperaggregatory thrombocytopeny was observed during the induction of ADP with a normal radius of the formed aggregates. The study of the coagulation link of hemostasis revealed its activation on the first day of bleeding in the form of a shortening of the activated partial thromboplastin time to 29.6 ± 2.5 sec. ($P \leq 0.05$) with its subsequent increase on the 7th day to 35.2 ± 2.0 sec ($P \leq 0.05$) compared with the control group. The prothrombin index during the observation process ranged from $97.6 \pm 1.5\%$ to $85.0 \pm 2.0\%$. The

fibrinogen content in plasma on the 1st day of bleeding was 2.22 ± 0.2 g/l, by the 3rd day - 2.4 ± 0.2 g/l ($P \leq 0.05$), and by the 7th day increased to 2.8 ± 0.2 g/l ($P \leq 0.05$).

During the first day, APTT bleeding showed 32.1 ± 1.5 seconds. ($P \leq 0.05$) and on the 7th day, with a slight shortening, up to 35.0 ± 2.0 sec. ($P \leq 0.05$) compared to the control group. Several other patterns were found in patients of group II, where gastro duodenal bleeding was of the second degree of blood loss. On the first day of bleeding, the content of platelets in the venous blood was $268.13 \pm 6.13 \times 10^9/l$, followed by a statistically significant ($P \leq 0.05$) decrease on the 3rd day to $157 \pm 4.5 \times 10^9/l$ ($P \leq 0.05$). By the 7th day, this indicator increased to $245.5 \pm 5.7 \times 10^9/l$.

In the course of the study, the indicators of prothrombin indicator and thrombin time were established as follows:

The prothrombin index on the first day was $79.6 \pm 1.9\%$. By the third and seventh days it was $83.6 \pm 1.12\%$ and $81.0 \pm 1.5\%$. Thrombin time was 17.1 ± 1.2 sec. On the 3rd day, it increased to 17.9 ± 1.3 seconds, which indicates the onset of hypo coagulation.

The fibrinogen content in plasma on the 1st day of bleeding was 1.99 ± 0.2 g / l, by the 3rd day it increased to 2.22 ± 0.2 g / l ($P \leq 0.05$) with a further increase to 7- th day up to 2.4 ± 0.2 g / l ($P \leq 0.05$).

A change in the activated partial thromboplastin time to 27.4 ± 1.5 sec ($P \leq 0.05$) with a slight decrease to 31.5 ± 1.5 sec ($P \leq 0.05$) indicates the activation of the coagulation link of hemostasis, depending on the duration of bleeding in patients with gastro duodenal bleeding.

In patients of group III, who had gastro duodenal bleeding of the third degree of blood loss, the following indicators of the hemostasis system were determined: the number of platelets in patients with daily bleeding was $130 \pm 3.5 \times 10^9 / L$, and on the 3rd day their decrease was noted, but the difference with the initial parameters was unreliable ($P > 0.05$). On the first day, APTT bleeding was up to 25.4 ± 1.5 sec. ($P \leq 0.05$) with its subsequent increase on the 7th day to 30.0 ± 2.0 sec. ($P \leq 0.05$) compared to the control group.

Prothrombin and thrombin tests were $74.6 \pm 2.1\%$ and 16.8 ± 1.3 seconds on day 1, $72.6 \pm 1.3\%$ and 17.7 ± 1.7 seconds on day 2, and Day 3 $68.0 \pm 1.0\%$ and 17.0 ± 1.03 sec, the concentration of fibrinogen in plasma on the 1st day of bleeding was 2.22 ± 0.2 g/l, by the 3rd day it decreased to 1.77 ± 0.2 g/l ($P \leq 0.05$), and by the 7th day 1.99 ± 0.2 g/l ($P \leq 0.05$).

In patients with gastroduodenal ulcer complicated by bleeding in cardiovascular diseases, there is a convincing ($P \leq 0.05$) decrease in all studied parameters by almost 2 times, namely, lengthening of thrombin time. Such a change in the parameters of

hemostasis can be explained by the regular intake of antiplatelet agents. It was revealed that the indicators of the hemostasis system in patients concomitant with essential hypertension were marked by significant changes. In particular, there is a significant difference in the number of platelets ($174.0 \pm 2.0 \times 10^9/l$).

In the analysis of hemostasis indices in patients with diseases of the liver, biliary tract and chronic diseases of the pancreas, the platelet level was $165.0 \pm 3.5 \times 10^9/l$, the concentration of fibrinogen in the plasma was 2.17 ± 0.2 g/l, platelet aggregation was compared with the control group decreased by 35% ($51.3 \pm 2.1\%$), the prothrombin index - to $82.6 \pm 2.3\%$, respectively. The development of hepatic hypoxia leads to a decrease in the synthesis of protein, carbohydrates, pigments, antitoxic functions and urea metabolism in the liver. In patients with gastroduodenal bleeding of ulcerative etiology, metabolic changes in the liver are assessed as the effect of hemostasis on vascular resistance, in particular, on the vascular endothelial mechanism of hemostasis, on the other hand, a deficiency of enzymes involved in protein synthesis.

Conclusions. The main pathophysiological mechanisms of impaired hemostasis in patients with gastroduodenal bleeding are manifested by a decrease in platelet aggregation, an increase in the duration of bleeding, an increase in the activated partial thromboplastic time.

Increased platelet aggregation, as well as the presence of a relationship between the aggregation potential of blood, indicate the pathophysiological role of these changes in the development of patients with gastric ulcer and duodenal ulcer complicated by bleeding.

Literature:

1. Analysis of the results of treatment of gastric ulcer and duodenal ulcer complicated by bleeding /A. S. Yermolov [et al.] // Emergency surgery and infection in surgery: V All-Russian Scientific Conference of General Surgeons, united with the Plenum of the problem commission of the Russian Academy of Medical Sciences. – Rostov, 2008. – p.46-48
2. Gulov M.K., Abdulaeva S.I. - "The current state of the problem of treatment of ulcerative bleeding of the stomach and duodenum" // Bulletin of Avetsinna - № 4.- 2015. p. -108-115.

3. Zherlov G.K., Koshel A.P., Gibadulin N.V. et al. Choice of surgical tactics for gastroduodenal ulcerative bleeding // Vestn. surgeon.-2001.-№4.- p. -41-44.
4. Saveliev V.S., Kirienko A.I. Clinical Surgery. National leadership/M.: GEOTAR - media, 2013. - p.- 317-40.
5. Saidov D.S. - Dynamics of hemostasis indices and their correction by optimizing infusion-transfusion therapy for ulcerative gastroduodenal bleeding // Bulletin of the Academy of Medical Sciences of Tajikistan 2019.Vol. IX- №1. - p.- 67-75.
6. Tactics for gastroduodenal bleeding in elderly and senile persons / M.D. Diribov [et al.]//Emergency surgery and infection in surgery: V All-Russian Scientific Conference of General Surgeons, united. with the Plenum of the problem commission of the Russian Academy of Medical Sciences. – Rostov, 2008 – p.40-42.
7. Khadjibaev A.M., Rakhimov R.I., Nabiev A.A. Results of surgical treatment of ulcerative bleeding in patients with coronary heart disease // Inpatient replacement technologies: Ambulator surgery. 2020 (1-2) - p.-110-116.

НАРУШЕНИЯ ГЕМОСТАЗА ПРИ ГАСТРОДУОДЕНАЛЬНЫХ КРОВОТЕЧЕНИЯХ ЯЗВЕННОЙ ЭТИОЛОГИИ С ПАТОФИЗИОЛОГИЧЕСКОЙ ТОЧКИ ЗРЕНИЯ

Алимов С.У.

Резюме. Обследованы 119 пациентов с язвенной болезнью желудка и двенадцатиперстной кишки, осложненной кровотечением. В момент поступления в клинику у больных с первой степенью кровопотери уровень тромбоцитов составляло $185 \pm 8,0 \times 10^9/l$. У больных II группы, при первой сутки кровотечения содержание тромбоцитов в венозной крови составило $268,13 \pm 6,13 \times 10^9/l$ с снижением на 3-и сутки до $157 \pm 4,5 \times 10^9/l$. К 7-ым суткам этот показатель увеличился до $245,5 \pm 5,7 \times 10^9/l$. У больных III группы изменилось следующим образом: количество тромбоцитов было $130 \pm 3,5 \times 10^9/l$, а на 3-и сутки отмечено их снижение, но разница с исходными параметрами. В первые сутки кровотечения Активированное частичное тромбопластиновое время (АЧТВ) было до $25,4 \pm 1,5$ сек. с последующим его повышением на 7-и сутки до $30,0 \pm 2,0$ сек по сравнению с контрольной группой.

Ключевые слова: язвенная болезнь желудка, кровотечения, гемостаз, язвенная болезнь двенадцатиперстной кишки, агрегация тромбоцитов.