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ABSTRACT

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GASTROINTESTINAL BLEEDING AGAINST THE BACKGROUND OF NON-CIRRHOTIC PORTAL HYPERTENSION IN A PATIENT WITH MYELOFIBROSIS (CASE REPORT)

Portal hypertension in the absence of cirrhotic transformation of the liver parenchyma is considered a rare pathology with a difficult diagnostics. Non-cirrhotic portal hypertension includes a heterogeneous group of conditions based on porto-sinusoidal vascular disease of the liver and chronic portal vein thrombosis. For a long time, the course of the disease can remain asymptomatic and is usually manifested by the development of one of the complications of portal hypertension. One of the most dangerous and often fatal complications is bleeding from varicose veins of the esophagus and stomach. The purpose of this article is to demonstrate, using the example of a clinical case, the peculiarities of diagnosis and methods of successful treatment of bleeding from esophageal phlebectasia in case of non-cirrhotic portal hypertension caused by splenic vein thrombosis.

The article describes the clinical case of a 57-year-old patient with a bleeding from esophageal varices with non-cirrhotic portal hypertension caused by splenic vein thrombosis due to myelofibrosis. The patient was urgently hospitalized with symptoms of hemorrhagic shock, which required intensive care, varix ligation and hemotransfusion therapy.

After stabilization of hemodynamic parameters, the patient underwent a porto-azygos disconnection operation, which included devascularization of the distal esophagus, proximal stomach, transection of the esophagus and splenectomy.

Conclusions. Myeloproliferative diseases are one of the many causes of non-cirrhotic portal hypertension, which often remain unrecognized until its decompensation. The diagnosis of non-cirrhotic portal hypertension is often difficult, which directly affects the prognosis. The authors' goal was to draw attention, raise awareness, and demonstrate the

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pathogenetic link between these conditions, the given clinical case of surgical treatment can be a variant of medical tactics aimed at controlling bleeding from esophageal and gastric phlebectasia.

The positive feature of the performed surgical intervention is the direct impact on the source of bleeding, namely, the disconnection of the portal and caval venous systems, which ensured the cessation of blood flow through the esophageal-gastric collaterals.

Taking into account the risks of anticoagulant therapy in patients with portal hypertension with Portal vein thrombosis and bleeding from varicose veins of the esophagus and stomach we recommend long-term (3–6 months) anticoagulant therapy in patients with prothrombotic conditions after porto-azygos disconnection and exclusion of “dangerous” esophageal-gastric collaterals, which makes it possible to effectively control hemorrhagic complications.

Key words: portal vein thrombosis, portal hypertension, bleeding, myelofibrosis.

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ШЛУНКОВО-КИШКОВА КРОВОТЕЧА НА ТЛІ НЕЦИРОТИЧНОЇ ПОРТАЛЬНОЇ ГІПЕРТЕНЗІЇ У ХВОРОГО НА МІЄЛОФІБРОЗ (КЛІНІЧНИЙ ВИПАДОК)

Портальна гіпертензія за відсутності циротичної трансформації печінкової паренхіми вважається рідкісною патологією, а діагностика її часто буває запізнілою. Нециротична портальна гіпертензія включає гетерогенну групу станів, основу яких складають портосинусоїдальні судинні захворювання печінки та хронічний тромбоз ворітної вени. Тривалий час перебіг захворювання може залишатися безсимптомним і проявляється, як правило, розвитком одного з ускладнень портальної гіпертензії. Одним з найнебезпечніших, а часто й фатальним ускладненням, є кровотеча з варикозно-розширених вен стравоходу та шлунку. Мета роботи полягає в тому щоб продемонструвати на прикладі клінічного випадку особливості діагностики та методику успішного лікування кровотечі з стравохідних флєбектазій на тлі нециротичної портальної гіпертензії, спричиненої тромбозом ворітної та селезінкової вен.

В статті описаний клінічний випадок 57 річного пацієнта з явищами варикозно-розширених вен стравоходу на тлі нециротичної портальної гіпертензії ускладненої кровотечею, що виникла внаслідок тромбозу ворітної та селезінкової вен, зумовленої мієлофіброзом. Хворий був госпіталізований в ургентному порядку з ознаками геморагічного шоку, що вимагало проведення інтенсивної терапії, лігування вариксів та гемотрансфузійної терапії. Після стабілізації гемодинамічних показників виконано операцію портоазигального роз'єднання, що передбачала деваскуляризацію дистального відділу стравоходу, проксимального відділу шлунка, апаратна транссекція стравоходу та спленектомія (етапи операцій Sugiura та Hassab).

Висновки. Мієлопроліферативні захворювання є однією з багатьох причин розвитку нециротичної портальної гіпертензії, які

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часто залишаються нерозпізнаними до моменту її декомпенсації. Діагностика НЦПГ часто буває складною, що безпосередньо впливає на результати лікування пацієнтів. Метою авторів було звернути увагу та підвищити настороженість, показати патогенетичний зв'язок між цими станами, а наведений клінічний випадок оперативного лікування може бути варіантом лікувальної тактики, спрямованої на контроль кровотечі з стравохідних та шлункових фіблектазій.

Позитивними особливостями проведеного оперативного втручання є безпосередній вплив на осередок кровотечі, а саме роз'єднання портальної та кавальної венозних систем, що забезпечило припинення кровотоку по стравохідно-шлункових колатераліях.

Враховуючи ризики антикоагулянтної терапії у пацієнтів з портальною гіпертензією на тлі тромбозу v.portae її гілок та кровотечею з ВРВ стравоходу та шлунку ми рекомендуємо призначати довгострокову (3–6 місяців) антикоагулянтну терапію хворим з протромботичними станами після оперативного втручання спрямованого на виключення «небезпечних» колатералей езофаго-кардіального шляху одним з варіантів порто-азиального роз'єднання, що дає можливість ефективно контролювати геморагічні ускладнення.

Ключові слова: тромбоз ворітної вени, портальна гіпертензія, кровотеча, мієлофіброз.

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ABBREVIATIONS

PH – portal hypertension

NCPH – non-cirrhotic portal hypertension

PSVD – porto-sinusoidal vascular diseases of the liver

VV – varicose veins

EGD – esophagogastroduodenoscopy

INTRODUCTION / ВСТУП

Portal hypertension (PH) in the absence of cirrhotic transformation of the liver parenchyma is considered a rare pathology with difficult diagnosis. Noncirrhotic portal hypertension (NCPH) includes a heterogeneous group of conditions based on portal-sinusoidal vascular disease of the liver (PSVD) and chronic portal vein thrombosis [1]. The course of the disease can remain asymptomatic for a long time and is usually manifested by complications of portal hypertension. One of the most dangerous and often fatal complications is bleeding from varicose veins of the esophagus and stomach. Therefore, PSVD cannot be considered a disease with a favorable course, because portal vein thrombosis, liver failure, and mortality are common phenomena in patients with this pathology [2]. We present a clinical case of surgical treatment of a patient with bleeding from esophageal varices in case of NCPH

that developed as a result of presinusoidal block caused by myelofibrosis.

Case report. A 57-year-old patient was admitted to the intensive care unit of the "Kyiv City Clinical Hospital of Emergency Medical Care" with the clinic of acute bleeding from the upper gastrointestinal tract. During hospitalization, vomiting of fresh blood with clots was observed, blood pressure 80/50 mmHg, heart rate 100 bpm.

After the stabilization of the hemodynamic parameters, an EGD was performed, the examination revealed the presence of varicose veins (VV) of the III degree (12 mm) with red spots that bled on contact in the middle and lower thirds of the esophagus. Also, in the cardia, along the greater and lesser curvature, VVs up to 10 mm were visualized; multiple erosions were found in the area of the body of the stomach.

Laboratory indicators on admission: Hb – 40 g/l, Er

– $2,8 \times 10^{12}$, Plt – 851×10^9 , L – $32,8 \times 10^9$; total protein – 41 g/l, albumin 21 g/l, total bilirubin – 19 $\mu\text{mol/l}$, AlAT – 0.92 $\mu\text{mol/l}$, AsAT – 0.98 $\mu\text{mol/l}$; urea – 9.0 $\mu\text{mol/l}$, creatinine – 115 $\mu\text{mol/l}$; PI – 52%, fibrinogen 1.3 g/l, INR – 1.5. Serological markers of viral hepatitis HCV, HBV were negative, it is also known from the anamnesis that the patient did not abuse alcohol. According to the data of computed tomography with

contrast enhancement, it was found (see Fig. 1 and 2): thrombosis of v.portae, v.lienalis, signs of portal hypertension: RVV of the esophagus in the areas of the middle and lower third, the presence of gastric varices, multiple collaterals in the area of the portal of the spleen. Splenomegaly. Right-sided large hydrothorax. Ascites.

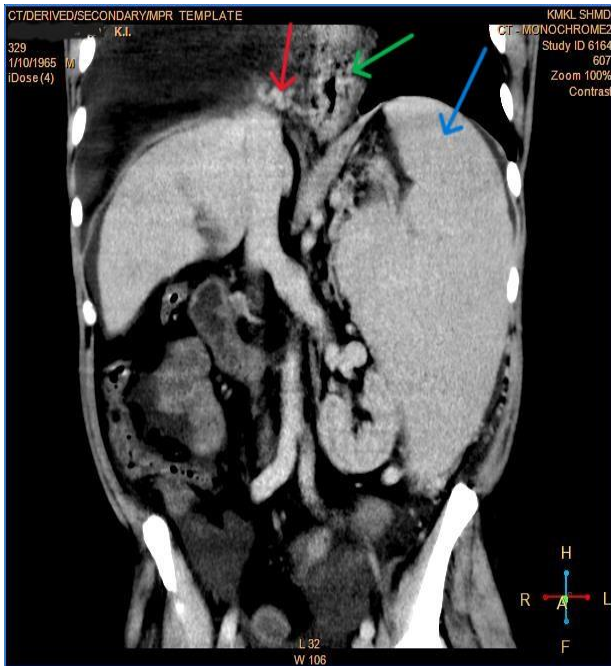


Figure 1 – Axial computed tomography with contrast in the portal vein demonstrates: the presence of esophageal varices, which are visualized in the form of tortuous tubular structures with an uneven contour, protruding into the lumen of the esophagus (green arrow), v.diaphragmatica inferior (red arrow, draining blood from the esophageal varices into the v.cava inferior), splenomegaly (blue arrow)



Figure 2 – Axial computed tomography with contrast enhancement of the portal vein demonstrates: a hypodense clot (green arrows) that completely occludes the v.portae, dilated v.gastrica sinister (yellow arrow), the presence of veins of Retzius (blue arrows), which are visualized as multiple tortuous dilated collateral vessels in the retroperitoneal space, draining into the v.cava inferior

Taking into account the unstable hemodynamics and the lack of a complete examination of the patient, including CT with contrast of the visceral vessels, which is crucial for defining of the treatment tactics, watch & wait tactics was chosen.

12 hours after the patient was admitted to the hospital, a relapse of bleeding occurred, the Blackmore-Sengstaken tube was installed. After removal of the tube, in 48 hours, bleeding from the esophageal gland relapsed again, bleeding was stopped, varix ligation was stopped (5 ligature rings were applied). In order to correct hematological parameters, blood transfusion therapy was carried out in the amount of 3 doses (550 ml) of erythrocyte mass to the target level of Hb – 70 g/l, FFP 2 doses (350 ml), 20%

albumin solution of 100 ml for 5 days, hemostatics, octreotide, cephalosporins and proton pump blockers were administrated.

The absence of the cause of chronic liver disease, along with the presence of severe portal hypertension, the combination of splenomegaly (210 x 88 x 79 mm) and thrombocytosis (851×10^9) suggested that the patient had myeloproliferative disease. Based on the examination, the diagnosis was established: Idiopathic non-cirrhotic portal hypertension. Thrombosis of the portal and splenic veins. Varices of the esophagus and stomach of the III degree, complicated by bleeding. Splenomegaly. Ascites. Right-sided hydrothorax. Acute severe post-hemorrhagic anemia.

Taking into account of the high risk of recurrent

bleeding, after stabilization of vital indicators, the patient was operated. A modified laparoscopic-assisted porto-azigal disconnection operation was performed, which included gastro-esophageal devascularization, instrumental transection of the esophagus and splenectomy (stages of Sugiura and Hassab operations). Intraoperatively, the surface of the liver is smooth, visually without cirrhotic changes. In the postoperative period, the patient received antibacterial (third-generation cephalosporins), anticoagulant therapy, which included the introduction of low molecular weight heparin from the 2nd day of the postoperative period, the infusion of albumin, early enteral nutrition with high protein. The patient was discharged from the hospital in satisfactory condition on the 9th day of the postoperative period. The patient is prescribed warfarin. The necessity of this prescription is explained by the need to prevent the possible developments of thrombosis to the superior mesenteric vein or the occurrence of thrombosis of any location of the caval system. Subsequently, the patient was consulted by a hematologist, additional examinations were carried out, which revealed *JAK2 V617T* mutations, and bone marrow punctate revealed an increase in the number of myeloid cells and atypical megakaryocytes, indicating primary myelofibrosis. 4 weeks after discharge from the hospital, the patient underwent a control EGD, in which varicose veins decreased, on a control ultrasound investigation clot in the portal vein is unchanged.

DISCUSSION

NCPH is often associated with systemic hematological diseases, prothrombotic conditions, genetic disorders, immunological disorders, exposure to toxins, hepatotoxic drugs, and chronic infections, which may play a direct role in pathophysiological changes in the liver [3, 4, 5]. These diseases includes a large number of clinical-pathological variants and various histological models [6, 7]. In addition, NCPHs are have risks of splanchnic vein thrombosis, with 90% of all cases occurring in the portal vein [8].

So, the presence of a mutation *JAK2* at myeloproliferative neoplasm (MPN), is a prothrombotic predictor of thrombosis of the main venous vessels [9].

As a rule, in patients suffering from NCPH, the evolution of PH and the progression of gastroesophageal phlebectasias is faster, and bleeding occurs more often

than in patients with cirrhosis of the liver [1].

Thus, portal and splenic vein thrombosis with the development of PH caused by myelofibrosis was asymptomatic until decompensation, which was manifested by massive bleeding, which requires urgent hospitalization.

The optimal treatment of bleeding from VV of the esophagus and stomach as a manifestation of NCPH remains a debatable issue due to the lack of a sufficient number of randomized controlled trials [10]. Therefore, the modern standard for NCPH is the treatment of diseases that directly caused the development of portal hypertension. Since the diagnosis of myelofibrosis is almost always delayed due to the peculiarities of clinical manifestations at the onset of the disease, therefore, today, the tactics and treatment of complications remain the same as in cirrhosis of the liver [11,12,13]. Demonstrating this clinical case, we would like to draw attention to the probability of developing bleeding from VV, in case of presinusoidal decompensation, which was the first manifestation of PH and a consequence of thrombosis of the splanchnic venous vessels in case of myeloproliferative disease.

CONCLUSIONS

Myeloproliferative diseases are one of the many causes of non-cirrhotic portal hypertension, which often are not unrecognized until it is decompensated. Diagnosis of NCPH is often complex, which directly affects the results of patient treatment. The authors' goal was to pay attention and increase alertness, to show a pathogenetic relationship between these conditions, the given clinical case of surgical treatment may be a variant of therapeutic tactics aimed to controlling of bleeding from esophageal and gastric phlebectasias.

The positive impact of the surgical intervention is the direct effect on the bleeding, particularly, the separation of the portal and caval venous systems, which ensured the cessation of blood flow through the esophageal-gastric collaterals.

After surgery aimed at excluding "dangerous" venous collaterals of the esophago-cardiac pathway, which makes it possible to effectively control hemorrhagic complications, we recommend prescribing long-term (3–6 months) anticoagulant therapy under constant monitoring of the general condition of patients and coagulogram indicators.

PROSPECTS FOR FUTURE RESEARCH / ПЕРСПЕКТИВИ ПОДАЛЬШИХ ДОСЛІДЖЕНЬ

Further studies with an increase in the sample of patients and the duration of follow-up should aim to identify the long-term consequences of treatment of PH complicated by bleeding from VV of the esophagus and stomach, as well as to establish the advantages or disadvantages of the proposed treatment tactics.

AUTHOR CONTRIBUTIONS / ВКЛАД АВТОРІВ

All authors substantively contributed to the drafting of the initial and revised versions of this paper. They take full responsibility for the integrity of all aspects of the work.

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CONFLICT OF INTEREST / КОНФЛІКТ ІНТЕРЕСІВ

The authors declare no conflict of interest.

COMPLIANCE WITH ETHICAL REQUIREMENTS / ВІДПОВІДНІСТЬ ЕТИЧНИМ СТАНДАРТАМ

Ethical approval was obtained by the Bioethics Committee of the O.O. Bogomolets National Medical University. The patient gave written consent to participate in the study in accordance with the recommendations of the Committees on the Ethics of Biomedical Research, the legislation of Ukraine in the field of health care and the Helsinki Declaration of 2000, Directive of the European Community 86/609 "On Human Participation in Biomedical Research".

ARTIFICIAL INTELLIGENCE DISCLOSURE / ВИКОРИСТАННЯ ШТУЧНОГО ІНТЕЛЕКТУ

The authors declared that they did not use artificial intelligence-based technologies when writing and editing the manuscript.

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