

CHANGES IN THE MORPHOFUNCTIONAL ORGANIZATION OF THE INTRAORGAN HEMOCIRCULATORY BED OF THE LIVER WITH MECHANICAL ACUTE SMALL BOWEL OBSTRUCTION

Nguyen Cao Cuong¹, Milyukov Vladimir Yefimovich²,
Sharifova Kheyalya Murshud Kizi², Sharifov Elshan Rovshan Oglu³

TÓM TẮT

Title: Changes in the morphofunctional organization of the intraorgan hemocirculatory bed of the liver with mechanical acute small bowel obstruction

Từ khóa: phân tích yếu tố, phân tích yếu tố khám phá

Keywords: acute intestinal obstruction, intra-organ vascular bed of the liver, arteries of the liver, veins of the liver, microvasculature.

Lịch sử bài báo:

Ngày nhận bài: 5/5/2021;

Ngày nhận kết quả bình duyệt: 25/5/2021;

Ngày chấp nhận đăng bài: 20/6/2021.

Tác giả: ¹ Yersin university of Da Lat; ² Chair of Human Anatomy, Sechenov University, Russia; ³Head of the clinic "Today's Dental", Russia

Email: cuongnc@yersin.edu.vn

Nghiên cứu dựa trên phân tích mẫu vật sinh thiết thu được thông qua mô hình hóa tắc ruột non cấp tính cơ học trên động vật thí nghiệm. Một phương pháp được sử dụng để đánh giá những thay đổi về thể tích của lòng mạch trên cơ sở nghiên cứu hình thái của các mạch máu trên một đơn vị diện tích của nhu mô gan. Theo tiến triển của bệnh, chúng tôi đã phát hiện ra sự khác biệt và mô hình chung những thay đổi hình thái của hệ thống mạch máu của gan so với thắt nghẹt và tắc nghẽn ruột non cấp tính (ASBO).

ABSTRACT

The study is based on the analysis of biopsy material obtained by modeling mechanical acute small bowel obstruction in experimental animals. A method was used to assess changes in the volume of the vascular bed on the basis of a morphometric study of the area of blood vessels per unit area of the liver parenchyma. According to the development of the disease, we found differences and general patterns of regional changes in the morphofunctional organization of the intra-organ hemovascular channel of the liver with strangulation and obstructive acute small bowel obstruction (ASBO).

Introduction

In connection with the development of surgical technique and anesthesiology, the number of complex operations on the organs of the abdominal cavity is growing and, at the same time, the incidence of adhesive disease (AD) is steadily increasing (Ozturk E, Iersel M, Stommel M, Schoon Y, Broek R and Goor H., 2018, Meier RPH,

Saussure WO, Orci LA, Gutzwiller EM, Morel P, Ris F, Schwenter F., 2014, Thornblade LW, Truitt AR, Davidson GH, Flum DR, Lavalley DC., 2017(Thornblade LW, Truitt AR, Davidson GH, Flum DR, Lavalley DC). The overall mortality in AD is from 13% to 55% (Thornblade LW, Truitt AR, Davidson GH, Flum DR, Lavalley DC., 2017, Catena F, Saverio SD, Coccolini F, Ansaloni L, Simone

BD, Sartelli M, Goor HV., 2016). Every year, more than 1% of previously operated patients are treated for AD in surgical hospitals, and 50-75% of patients with AD develop acute commissural intestinal obstruction (ACIO), which combines signs of both strangulation and obstructive obstruction. Adhesive intestinal obstruction among all types of intestinal obstruction is 60-90%, while in 90-94% of cases it is acute small bowel obstruction (Ekin Ozturk, Marianne van Iersel, Martijn MWJ Stommel, Yvonne Schoon, Richard RPG ten Broek, Harry van Goor., 2018, Lee M. J., Sayers A. E., Drake T. M., Marriott P. J., Anderson I. D., Bach S. P., Bradburn M, Hind D, Verjee A., Fearnhead N. S., 2019). With complication of acute commissural small bowel obstruction with peritonitis, postoperative mortality is 25-70%, and in the terminal stage of peritonitis with the development of toxic-septic shock (TSS) and multiple organ failure syndrome (MOFS), mortality reaches 85-100% (Ekin Ozturk, Marianne van Iersel, Martijn MWJ Stommel, Yvonne Schoon, Richard RPG ten Broek, Harry van Goor., 2018, Thornblade LW, Truitt AR, Davidson GH, Flum DR, Lavalley DC., 2017, Lee M. J., Sayers A. E., Drake T. M., Marriott P. J., Anderson I. D., Bach S. P., Bradburn M, Hind D, Verjee A., Fearnhead N. S., 2019). From the foregoing, it obviously follows that acute small bowel obstruction is an unresolved problem of modern emergency surgery (Ekin Ozturk, Marianne van Iersel, Martijn MWJ Stommel, Yvonne Schoon, Richard RPG ten Broek, Harry van Goor., 2018).

One of the early manifestations of MOFS is the development of liver failure, which, however, is diagnosed only in the late stages. Blood containing toxic

substances resulting from ischemic lesion and/or tissue necrosis of a pathologically altered small intestine section enters the liver during ASBO through the portal vein. Consequently, it is the liver with small bowel obstruction that is the first organ that takes the brunt of toxinemia, and with the development of MOFS, one of the first manifestations of this formidable complication is the development of liver failure, which is diagnosed only in the late stages. The occurrence and development of acute liver failure (ALF) is the cause of death of 43-71% of patients with widespread purulent peritonitis (Cerniy V., Turek Z., Parizkova R., 2009, Maronne G., Shah VH., Gracia-Sancho J., 2016).

The portal vein and its own hepatic artery enter the liver through a recess called the portal of the liver, which is located on the lower surface of the right lobe closer to its posterior edge (Couinaud C., 1999, Wohlleber D., Knolle PA., 2016).

The main function of the hemocirculatory system in the liver is transcapillary exchange, carried out by the endothelium of sinusoidal capillaries, which receive blood from the interlobular branches of the portal vein and its own hepatic artery, providing the supply of hepatocytes with nutrients, oxygen and removal of metabolic products (Wohlleber D., Knolle PA., 2016, Laurie D. DeLeve, Ana C. Maretta-Mira., 2017). Sinusoid walls normally do not have a basal membrane and are formed by flat endothelial cells with large intercellular spaces, which facilitates the transfer of protein-bound substances from sinusoids, first to the Disse space located between endothelial cells and hepatocytes, and then to hepatocytes

themselves, and also accelerates the excretion of lipoproteins and other substances from hepatocytes to sinusoids (Laurie D. DeLeve, Ana C. Maretta-Mira., 2017, Yao Ni, Juan-Mei Li, Ming-Kun Liu, Ting-Ting Zhang, Dong-Ping Wang, Wen-Hui Zhou, Ling-Zi Hu, Wen-Liang Lv., 2017). Blood from sinusoids enters the central lobular vein, and then into the collective and hepatic veins, which flow into the inferior vena cava (Laurie D. DeLeve, Ana C. Maretta-Mira., 2017, Yao Ni, Juan-Mei Li, Ming-Kun Liu, Ting-Ting Zhang, Dong-Ping Wang, Wen-Hui Zhou, Ling-Zi Hu, Wen-Liang Lv., 2017, Poisson J, Lemoine S, Boulanger C, Durand F, Moreau R, Valla D, Rautou PE., 2017). The outflow of blood from the hepatic lobules is provided by the central veins, which, when exiting the hepatic lobules, flow into the sublobular veins (Cerniy V., Turek Z., Parizkova R., 2009, Maronne G., Shah VH., Gracia-Sancho J., 2016, Wohlleber D., Knolle PA., 2016). When merging, the sub-lobular veins form the hepatic veins, and they, in turn, are the efferent vascular system of the liver. The hepatic veins in the abdominal cavity are the last branches that the inferior vena cava takes (Wohlleber D., Knolle PA., 2016). The hepatic veins in the groove area of the inferior vena cava exit and immediately flow into the inferior vena cava (Cerniy V., Turek Z., Parizkova R., 2009). In the classical case, 3 veins (right, middle and left) leave the liver, but according to the literature, the number of liver veins flowing into the inferior vena cava can reach up to 25 vessels (Wohlleber D., Knolle PA., 2016).

In relation to the liver, 2 groups of venous anastomoses are distinguished: hepatofugal (portocaval) and hepatopetal (portopal). Hepatofugal anastomoses connect the portal vein to the vena cava

system, excluding blood flow through the liver, and hepatopetal anastomoses carry blood directly to the liver. Hepatopetal anastomoses include numerous venous trunks embedded in the ligaments of the liver, the largest of which are known as accessory portal veins (Sappey veins) (Mak KM, Png CYM., 2019, Michael C. Lowe and Michael I. D'Angelica., 2016).

The purpose of our work: is to identify patterns of regional changes in the morphofunctional organization of the liver hemocirculatory bed at different periods of development of strangulation and obstructive acute small bowel obstruction.

Materials and methods:

Since the collection of biopsy material from the liver during acute strangulation and obstructive small bowel obstruction in the dynamics of the development of the disease in patients in the clinic is not possible by ethical criteria, the only available method of collecting material for research and objective assessment of regional changes in the morphofunctional organization of the intra-organic vascular bed of the liver at different stages of development of strangulation and obstructive ASBO is experimental simulation of the disease.

The objects of experimental research in our work were dogs. This is due to the fact that the anatomy, physiology and morphological structure of the liver of a dog and a person are closest to each other. The anatomical structure, physiology and functions of the digestive system, the main sources of blood circulation and the path of blood outflow, the spatial architectonics of the arterial and venous channels, and, consequently, the hemocirculatory system

in the digestive system in dogs and humans are similar. In addition, a fairly equal position in the phylogenetic series of dogs and humans determines the uniformity of reactions. All this together allows us to extrapolate the results of the experiment into clinical research and practice.

The study was performed on 33 adult mongrel dogs of both sexes weighing 17-20 kg. The experimental part was carried out in accordance with guidance documents, such as the “Guide for the Care and Use of Laboratory Animals of the National Institute of Health (National Institute of Health - NIH, Bethesda, USA)” and “Rules for Working with Experimental Animals,” approved by the Ethics Committee for Federal State Treasury Institution “The Main Military Clinical Hospital named after Academician NN Burdenko” of the Ministry of Defense of the Russian Federation and the local ethics committee of the First MG MU named after I.M. Sechenov.

For 2 animals, the study of the morphofunctional organization of the intra-organ hemovascular bed of the liver was normal. In animals of the second group (12 animals), a strangulation of small bowel obstruction was simulated for a follow-up period of 3, 6, 12, and 24 hours, followed by an analysis of the patterns of regional changes in the morphofunctional organization of the intra-organ liver blood vessel. In 12 animals of the third group, a simulation of low obstructive small bowel obstruction was performed for a follow-up period of 1, 2, 3, and 6 days and a study was held of the patterns of regional morphofunctional organization of the intra-organ liver hemovascular bed. The fourth, control group consisted of 7 animals, in

which the morphofunctional organization of the intra-organ hemovascular channel of the liver was studied after laparotomy without intervention on the vessels and organs of the abdominal cavity after 3, 6, 12 and 24 hours, as well as after 2, 3 and 6 days. Comparison of the results of the study in the second and third groups with the results in the fourth group reveals changes in the morphofunctional organization of the intra-organ hemovascular channel of the liver, caused by acute small bowel obstruction, but not the consequences of laparotomy in modeling the disease.

Modeling of strangulated acute small bowel obstruction was performed under anesthesia, ligature application with nylon thread No. 5 at a distance of 100 cm from the ileocecal transition to a loop of the small intestine 15 cm long and its mesentery.

Low obstructive small bowel obstruction was modeled by applying a nylon ligature, squeezing the lumen of the small intestine without involving its mesentery, 100 cm proximal to the ileocecal transition.

The choice of the observation time was based on the features of the clinical manifestations of ASBO. The manifestation of clinical symptoms of acute strangulation small bowel obstruction occurs almost immediately from the moment of strangulation formation and is accompanied by intense pain, as the mesentery of the small intestine is involved in the pathological process, which contains both blood vessels and a large number of nerve endings. Patients in this category are soon seeking medical help and quickly delivered to medical institutions.

Obstruction of the lumen of the small intestine does not occur instantly: for some time after reducing the diameter of the lumen of the small intestine, passage of intestinal contents through the intestine is possible (in clinical practice, this condition is often called “partial small bowel obstruction”), and the mesentery during obstructive small bowel obstruction is not involved in the pathological process. When the decrease in the lumen of the intestine reaches a certain critical level, there is a spastic reduction of its walls, followed by a complete cessation of passage and the manifestation of clinical symptoms of acute intestinal obstruction. Due to the lack of severe pain, patients in this category, as a rule, do not seek medical help for a long time.

Research methods included:

1 - experimental design techniques - modeling of acute strangulation and obstructive small bowel obstruction;

2 - methods for the study of liver morphology;

3 - morphometric methods for assessing the organization of the intra-organ hemocirculatory channel of the liver;

4 - statistical analysis of the results of the study.

The methods for studying liver morphology under normal conditions and after modeling small bowel obstruction were the same.

We used a method for assessing changes in the volume of the vascular bed on the basis of a morphometric study of the vascular area per unit area of the liver parenchyma on a histological preparation when studying the area of the interlobular arteries, interlobular veins and central veins of the liver lobules on the square of

the organ parenchyma with an area of $600 \times 103 \mu\text{m}^2$, the preparations were stained with hematoxylin and eosin.

Morphometry was performed using the program "Adobe Photoshop CS3 Extended RUS", "Image Pro", "Image 3.0". Statistical processing of the results was carried out by methods of variation statistics in the programs “Statistica 6.0. (Stat Soft Inc., USA)”, “Atte Stat”. Results were considered statistically significant at $p < 0.05$ (95% accuracy). To assess the relationship between changes in the HCC indicators, the Spearman correlation coefficient (r) was determined. The dependence of all signs was considered statistically significant at $p < 0.05$.

The ratio of the volume of the vascular bed of the portal system, the system of own hepatic artery and the system of hepatic veins (central veins of the liver lobules) was calculated by comparing the calculated value of the total radius of the vascular bed using the formula $S = \pi r^2$, where S is the area of the vascular bed in the selected standard area of tissue. The estimated total diameter of the vascular component in the assessment of the results is used for greater clarity and simplification of numerical indicators (ratio of numbers), since this indicator is characterized by a strict mathematical dependence on the area of the vascular bed.

The results of the study:

Normally, the area occupied in the preparations of the liver by the interlobular veins is 33.6 times larger than the area occupied by the interlobular arteries, and the area occupied by the central veins of the liver lobules (system of the inferior vena cava) is 37.2 times larger than the area occupied by the interlobular arteries. The

ratio of the estimated total diameter of the vascular component of the portal vein system and the system of own hepatic artery is 5.8 : 1, the system of the inferior vena cava and the system of own hepatic artery 6 : 1 (Fig. 7). From this it follows that the main role in ensuring blood supply to the liver belongs to the vessels of the portal system, and the volume of the central veins of the lobules of the liver - vessels of the inferior vena cava basin, is the total volume of the vascular bed of the portal vein and vessels of the own hepatic artery basin.

3 hours after the creation of a model of strangulated ASBO, the area of interlobular veins compared with the norm statistically significantly increases 1.62 times and interlobular arteries 2.82 times (Fig. 1, Fig. 2), which corresponds to the calculated ratio of the total diameter the vascular component of the portal vein system and the system of the own hepatic artery 4.39: 1.

The area of the central veins of the liver lobules at this period of the experiment statistically significantly increases 2.68 times compared with the norm (Fig. 3), and the ratio of the estimated total diameter of the vessels of the system of the inferior vena cava and the system of the own hepatic artery becomes 5.95 : 1.

6 hours after the creation of a model of strangulated ASBO, dilatation of the vessels of the arterial link of the HCS is observed, a statistically significant increase in the area of the interlobular arteries by 4 times is observed when compared with the norm (Fig. 1). is 2.82: 1 (Fig. 2). The area of the central veins of the liver lobules statistically significantly decreased 2.73 times compared with the norm (Fig. 3), and the

ratio of the estimated total diameter of the vascular component of the system of the inferior vena cava and the system of the own hepatic artery becomes 1.85: 1

12 hours after the formation of acute strangulation small bowel obstruction, the area of interlobular arteries statistically significantly increases by 1.55 times when compared with the norm (Fig. 1) The area of interlobular veins at this period of the experiment when compared with the norm decreases by 1.09 times, which corresponds to the ratio of the estimated total diameter of the vascular component of the portal vein system and the system of the own hepatic artery is 4.45: 1 (Fig. 2)

At this time of the experiment, the area of the central veins when compared with the norm increases statistically significantly 1.54 times (Fig. 3), and the ratio of the estimated total diameter of the vascular component of the system of the inferior vena cava and the system of the own hepatic artery is preserved, as with the norm of 6.08 :1,

The area of interlobular arteries when compared with the norm statistically significantly increases by 3.63 times (Fig. 1). The area of interlobular veins at this period of the experiment compared with the norm is statistically significantly increased 1.92 times (Fig. 2), which corresponds to the ratio of the estimated total diameter of the vascular component of the volume of the portal vein system and the hepatic artery system 4.22: 1.

The area of the central veins in the lobules of the liver compared with the norm is statistically significantly increased 2.59 times (Fig. 3), and the ratio of the estimated total diameter of the

vascular component of the system of the inferior vena cava and the system of the own hepatic artery becomes 5.15 : 1.

1 day after the simulation of obstructive ASBO, when compared with the norm, the area of interlobular veins did not statistically significantly increase 1.19 times, the interlobular arteries 1.18 times (Fig. 6.7), which corresponds to the ratio of the estimated total diameter of the vascular component of the portal vein system and the hepatic artery system 5.82: 1.

The area of the central veins at this period of the experiment, when compared with the norm, decreased 1.17 times (Fig. 8), and the ratio of the estimated total diameter of the vascular component of the inferior vena cava system and the hepatic artery system becomes 5.19: 1.

2 days after modeling of acute obstructive small bowel obstruction, the area of interlobular arteries decreases statistically significantly compared with the norm by 1.38 times. (fig. 4). The area of interlobular veins compared with the norm is statistically significantly reduced by 1.48 times, which corresponds to a ratio of the estimated total diameter of the vascular component of the portal vein system and the system of the own hepatic artery 5.61: 1 (Fig. 5).

At this period of the experiment, the area of the central veins of the liver lobules increases 1.15 times compared to the norm (Fig. 6), and the ratio of the estimated total diameter of the vascular component of the system of the inferior vena cava and the system of the own hepatic artery becomes 7.71: 1.

When obstructive acute small bowel obstruction is formed 3 days after the start

of the experiment, when compared with normal values, the area of interlobular arteries statistically significantly increases by 1.55 times (Fig. 4). The area of interlobular veins at this period of the experiment compared with the norm is statistically significantly increased 1.49 times, which corresponds to a ratio of the estimated total diameter of the vascular component of the portal vein system and the own hepatic artery system of 5.68: 1 (Fig. 5).

The area of the central veins of the liver lobules after 3 days from the start of the experiment is statistically significantly reduced compared to the norm by 3.15 times (Fig. 6), and the ratio of the estimated total diameter of the vascular component of the system of the inferior vena cava and the system of its own hepatic artery becomes 2.76: 1.

6 days after the formation of obstructive ASBO, there is a statistically significant increase in the area of interlobular arteries by 1.27 times when compared with normal values (Fig. 4) At this period of the experiment, the maximum statistically significant decrease in the area of interlobular veins compared with the norm is 4.11 times, which corresponds to the ratio of the estimated total diameter of the intra-organ vascular component of the portal vein system and the system of the own hepatic artery 2.54: 1 (Fig. 5). The area of the central veins after 6 days from the start of the experiment is statistically significantly reduced by 1.37 times compared with the norm (Fig. 6), and the ratio of the estimated total diameter of the vascular component of the system of the inferior vena cava and the system of the own hepatic artery becomes 4.62 : 1.

Conclusion:

When modeling strangulated ASBO, the mesentery of the small intestine is involved in the pathological process, which is a powerful stress effect.

After 3 hours after the formation of strangulated ASBO, a decrease in blood flow to the liver flowing from the organs of the abdominal cavity, including from a pathologically altered portion of the small intestine, is observed. This can be a generalized stress mechanism of shock ischemia of the gastrointestinal tract, justified by severe pain when the injured loop of the small intestine is involved in the pathological process of the mesentery, and a compensatory regulatory mechanism aimed at limiting the flow of toxins into the liver. The area of central veins naturally increases due to an increase in blood flow through the interlobular arteries.

6 hours after the creation of a model of strangulated ASBO, we observe an increase in arterial blood supply to the liver and a decrease in blood flow through the portal vein system, which occurs due to reflex spasm of arterial vessels of the shock organ (small intestine). Therefore, with an increase in the significance of the arterial component of blood circulation, an even more significant decrease in the flow of blood to the liver flowing from the abdominal organs, including from the pathologically altered portion of the small intestine and from the zones of reactive changes in the organs of the abdominal cavity and peritoneum, characterizing the initial stage, is observed. development of peritonitis. The area of the central veins naturally decreases due to a decrease in blood flow through the portal vein system.

12 hours after the formation of strangulated ASBO, the area of interlobular arteries decreases, which indicates a generalized spasm of the vessels of the large circle of blood circulation with spread to the vessels of the celiac trunk. The decrease in blood supply to the liver through the portal vein system at this time of the experiment provides a decrease in the flow of toxins into the liver from the necrotic small intestine.

24 hours after the formation of strangulated ASBO, vasodilation of the arterial collector is observed, most likely due to toxic damage to the central neuro-regulatory vascular mechanism, and an increase in venous inflow through the portal vein system, which increases the venous outflow through the central veins of the liver lobules, which are the initial link in the lower pool vena cava (Fig. 8). Consequently, there is a relative increase in intra-organ blood circulation both due to arterial inflow and due to an increase in blood flow to the liver, flowing from the abdominal organs, including from a pathologically altered portion of the small intestine. An increase in intra-organ blood circulation both due to arterial inflow, and due to an increase in portal inflow, leads to an increase in the area of central veins.

During the formation of obstructive ASBO after 1 day, statistically significant morphofunctional changes in the organization of the hemocirculatory bed in the liver are not observed.

2 days after the simulation of obstructive ASBO a generalized spasm of arterial vessels of the large circle of blood circulation occurs, which is accompanied by a decrease in blood flow to the liver both

through the portal vein system and along the interlobular arteries from the system of its own hepatic artery, which indicates organ ischemia, which may be the basic basis for the violation of the functional activity of hepatocytes and, consequently, the development of functional liver failure in general. An increase in the area of central veins, taking into account a decrease in blood flow through the arterial link of the HCS and portal vein, may indicate an increase in hypertension in the inferior vena cava due to the attachment of the compartment syndrome - an increase in intra-abdominal pressure, which is accompanied by difficulty in the outflow of blood from the hepatic veins to the inferior vena cava vein.

After 3 days from the start of the experiment, in our opinion, a significant decrease in the area of the central veins of the liver lobules against the background of vasodilation of the interlobular arteries and interlobular veins indicates the opening of non-normal extraorganic (hepatofugal) porto-caval anastomoses and a change in the direction of blood outflow from the pathological focus in the thin the intestine bypassing the portal vein system (Fig. 9). This mechanism of redistribution of hemocirculation may also be the result of the development of a compartment during ASBO syndrome, with hypertension in the system of the inferior vena cava. Due to the fact that this mechanism of redistribution of blood circulation ensures the generalization of toxic substances to other organs and systems bypassing the intra-organ hepatic blood flow and, accordingly, eliminating the detoxifying function of the liver, conditions are created for the development and

progression of MOFS, and vasodilation of the arterial and venous collectors is more likely, due to toxic damage to the central regulatory vascular mechanism.

6 days after the formation of obstructive ASBO, vasodilation of the arterial collector is observed, which is most likely due to toxic damage to the central regulatory vascular mechanism. The area of the central veins also decreases due to a sharp decrease in blood flow through the portal vein system. At this period of the experiment, the maximum statistically significant decrease in the area of interlobular veins arises in comparison with the norm. Considering that the portal vein system is the main intra-organ blood circulation in the liver, a significant decrease in blood flow through this system indicates increasing liver ischemia with subsequent progression of impaired function and, consequently, blockade of the detoxifying function of the liver, generalization of toxinemia, which, in turn, leads to the development of functional insufficiency of other organs and systems.

Consequently, with the development of strangulated ASBO, due to severe pain, even with the rapid development of clinical symptoms of peritonitis, during the first days of the development of the disease, there are no significant changes in the morphofunctional organization of the intra-organ vascular bed in the liver. In the formation of obstructive ASBO, despite the absence of manifesting clinical manifestations of acute intestinal obstruction, the appearance of a hemocirculatory basis for the formation of liver failure and the development of MOFS is noted already from 2 days, which requires intensive preoperative preparation and corrective therapy in the postoperative period.

Fig. 1. Strangulation intestinal obstruction. Area of interlobular arteries per unit area of $600 \times 10^3 \mu\text{m}^2$

Fig. 2. Strangulation intestinal obstruction. Area of interlobular veins per unit area of $600 \times 10^3 \mu\text{m}^2$

Fig. 3. Strangulation intestinal obstruction. Area of the central veins per unit area of $600 \times 10^3 \mu\text{m}^2$

Fig. 4. Obturating intestinal obstruction. Area of the central veins per unit area of $600 \times 10^3 \mu\text{m}^2$

Fig. 5. Obturating intestinal obstruction. Area of interlobular veins per unit area of $600 \times 10^3 \mu\text{m}^2$

Fig. 6. Obturating intestinal obstruction. Area of the central veins per unit area of $600 \times 10^3 \mu\text{m}^2$

Fig. 7. Histological preparation of the dog's liver. Norm. Hematoxylin and eosin stain. Magnification x100.

Fig. 8. 24 hours of experimental strangulation small bowel obstruction. Pronounced plethora of the central and interlobular veins. Swelling of the stroma. Hematoxylin and eosin stain. X100 magnification

Fig. 9. 3 days of experimental low obstructive small bowel obstruction. Pronounced plethora of interlobular veins. Swelling of the stroma. Hematoxylin and eosin stain. Magnification x100.

REFERENCES

- Ekin Ozturk, Marianne van Iersel, Martijn MWJ Stommel, Yvonne Schoon, Richard RPG ten Broek, Harry van Goor. Small bowel obstruction in the elderly: a plea for comprehensive acute geriatric care. *World Journal of Emergency Surgery*. 2018,13:48 <https://doi.org/10.1186/s13017-018-0208-z>
- Ozturk E, Iersel M, Stommel M, Schoon Y, Broek R and Goor H. Small bowel obstruction in the elderly: a plea for comprehensive acute geriatric care. *World Journal of Emergency Surgery*. 2018;13:48. doi.org/10.1186/s13017-018-0208-z
- Meier RPH, Saussure WO, Orci LA, Gutzwiller EM, Morel P, Ris F, Schwenter F. Clinical Outcome in Acute Small Bowel Obstruction after Surgical or Conservative Management *World J Surg*. 2014; 38(12): 3082–3088. doi: 10.1007/s00268-014-2733-6
- Thornblade LW, Truitt AR, Davidson GH, Flum DR, Lavalley DC. Surgeon Attitudes and Practice Patterns in Managing Small Bowel Obstruction: A Qualitative Analysis. *J Surg Res*. 2017; 219: 347–353. doi: 10.1016/j.jss.2017.06.052
- Thornblade LW, Truitt AR, Davidson GH, Flum DR, Lavalley DC. Surgeon Attitudes and Practice Patterns in Managing Small Bowel Obstruction: A Qualitative Analysis. *J Surg Res*. 2017; 219: 347–353. doi: 10.1016/j.jss.2017.06.052

- Catena F, Saverio SD, Coccolini F, Ansaloni L, Simone BD, Sartelli M, Goor HV. Adhesive small bowel adhesions obstruction: Evolutions in diagnosis, management and prevention. *World J Gastrointest Surg.* 2016 Mar 27; 8(3): 222–231. doi: 10.4240/wjgs.v8.i3.222
- Lee M. J., Sayers A. E., Drake T. M., Marriott P. J., Anderson I. D., Bach S. P., Bradburn M, Hind D, Verjee A., Fearnhead N. S. National prospective cohort study of the burden of acute small bowel obstruction. *BJS Open.* 2019; 3(3): 354–366. doi: 10.1002/bjs5.50136
- Cerniy V., Turek Z., Parizkova R. In situ assessment of the liver microcirculation in mechanically ventilated rats using sidestream dark-field imaging// *Physiol Res.* 2009.Nº58. P.49-55.
- Maronne G., Shah VH., Gracia-Sancho J. Sinusoidal communication in liver fibrosis and regeneration// *J of Hepat.* 2016. Vol.65.Nº3. P.608-617
- Couinaud C. Liver anatomy: portal(and suprahepatic) or biliary segmentation// *Digestive Surgery.* 1999. V.16. P.459–467.
- Wohlleber D., Knolle PA. The role of liver sinusoidal cells in local hepatic immune surveillance// *Clin Transl Immunology.* 2016. Vol.5. Nº12. P. e117
- Laurie D. DeLeve, Ana C. Maretta-Mira. Liver Sinusoidal Endothelial Cell: An Update. *Semin Liver Dis.* 2017; 37(4): 377–387. doi: 10.1055/s-0037-1617455
- Yao Ni, Juan-Mei Li, Ming-Kun Liu, Ting-Ting Zhang, Dong-Ping Wang, Wen-Hui Zhou, Ling-Zi Hu, Wen-Liang Lv. Pathological process of liver sinusoidal endothelial cells in liver diseases. *World J Gastroenterol.* 2017; 23(43): 7666–7677. doi: 10.3748/wjg.v23.i43.7666
- Poisson J, Lemoine S, Boulanger C, Durand F, Moreau R, Valla D, Rautou PE. Liver sinusoidal endothelial cells: Physiology and role in liver diseases. *J Hepatol.* 2017;66(1):212-227. doi: 10.1016/j.jhep.2016.07.009.
- Mak KM, Png CYM. The Hepatic Central Vein: Structure, Fibrosis, and Role in Liver Biology. *Anat Rec (Hoboken).* 2019 Oct 3. doi: 10.1002/ar.24273.
- Michael C. Lowe and Michael I. D’Angelica. Anatomy of Hepatic Resectional Surgery. *Surg Clin North Am.* 2016 Apr; 96(2): 183–195. doi: 10.1016/j.suc.2015.11.003