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Exploring a crucial aspect of geriatric surgery, this article delves into the pathogenesis of acute intestinal obstruction in elderly and senile patients

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Abstract: Pathomorphological changes in the intestine in the development of acute intestinal obstruction depend on the form of the lesion. So, in patients with strangulated intestinal obstruction, the main affected area is considered to be the strangulation area, that is, the place where the main vessels and nerves of the mesentery are compressed. Along with this, compression occurs in the intestine itself, leading to trophic disorders in this area. In contrast to the above, with obturation intestinal obstruction, the main pathological manifestations occur in the adductor part of the intestine. With the development of dynamic intestinal obstruction, the intestine does not have a specific lesion area, and all pathological processes diffuse. This review article is devoted to analysing literature on the mechanisms of development and manifestation of acute intestinal failure and the features of their manifestation in elderly and senile patients age.

Keywords: Acute intestinal obstruction, gerontology, pathogenesis, pathomorphology.

Introduction: Acute intestinal obstruction due to intestinal entrapment in the hernial sac has its characteristics in the pathogenesis of the development of intestinal necrosis. It is known that the time of development of necrosis of the strangulated loop of the

intestine with the cessation of arterial flow to it is longer than with impaired venous blood flow. Numerous experimental studies have shown that intestinal necrosis due to impaired arterial blood flow can be up to 6 hours, while with impaired venous outflow, the time of development of intestinal necrosis is 3-4 times shorter. There are also reports reducing this period to 1 hour [1].

In clinical practice, there are descriptions that with a surgical resolution of intestinal obstruction in cases of strangulated hernia, the external picture of changes in the strangulated intestinal loop may not always meet the criteria for viability. Discolouration of the serous membrane of the intestine, the appearance of pulsation and peristalsis of the strangulated intestinal loop are considered criteria for preserving it and not resorting to resection. At the same time, in elderly and senile patients, as described in the literature, there is a certain critical period when, even in restoring arterial blood supply to the intestinal loop, necrobiotic changes in the mucous membrane continue. As the authors of these publications point out, such an unaccounted process is the cause of the development of perforation of the strangulated intestinal loop in the postoperative period. Restoration of arterial blood flow through the intestinal vessels can occur bypassing the capillary network of the mucous membrane. The leading cause of such a pathological process in elderly and senile patients is the formation of many small thrombi. As a result, thrombogenic disorders in the microcirculation of the intestinal mucosa involve the venous circulatory systems in the pathological process. Thrombophlebitis develops, spreading to the portal vein. As a result, the mechanism of coagulopathy is triggered. In clinical practice, this phenomenon is called "no-reflow" [2].

In elderly and senile patients, the intestinal loop subjected to ischemia becomes a source of toxic products formed as a result of impaired metabolism in the intestinal cavity. This is also facilitated by the decay products of tissues occurring in the intestinal mucosa. Conditions are created for the rapid penetration of microorganisms through the affected intestinal wall, its translocation first into the local, and then into the systemic circulation [3].

An obstacle in the intestinal cavity due to its incarceration leads to stretching the adductor part of the organ. The movement of the contents through the intestine stops, and a reflex change occurs in peristalsis. It acquires a propulsive character. At the same time, the duration of irritation of the intestinal wall, namely receptors, leads to inhibition of the peristalsis processes of the entire gastrointestinal tract. There is a violation of the evacuation of the contents, which worsens over time, especially in elderly and senile patients. Such aggravation contributes to the progressive accumulation of gases and fluid in the intestinal cavity, stretching its walls more [4].

In their observations, clinicians noted that this phenomenon develops on a local scale in young and mature patients in the form of an incomplete volume of signs of the systemic inflammatory response syndrome, while in elderly and senile patients, it can become widespread and manifest itself as a general reaction of the current type, especially with a pronounced pain syndrome [5].

The compensatory reaction of the body is aimed at inhibiting intestinal peristalsis, which replaces hyperperistalsis. The final link in this pathological process is the development of intestinal paresis. As the results of studies of these pathological processes show, the mechanisms of the development of intestinal paresis are based on the body's reflex reactions, hypoxic states (primarily in the intestinal system itself), toxaemia, and water-electrolyte imbalance [6].

Studies have proven that irritation of nerve receptors of the intestinal mucosa is the leading cause of the development of inhibitory reflexes between different parts of the intestine (enteroenteral). Such reflexes are the basis for inhibiting the contractile activity of the intestinal muscles. As a result, paresis and intestinal paralysis develop [7].

In this context, it is impossible not to note the influence of intra-abdominal hypertension syndrome, in fact, on the abdominal organs. It is based on the direct effect of high pressure on the organs of the gastrointestinal tract, the blood vessels that feed them and the portocaval system as a whole. Researchers have found that the blood supply to the abdominal cavity and retroperitoneal space is disrupted when intra-abdominal pressure rises to 15 mm Hg. Organ blood flow does not decrease proportionally to cardiac output. However, it depends on the so-called perfusion pressure of the abdominal cavity, which is the difference between the average arterial and intra-abdominal pressure. The value of perfusion pressure is a criterion for assessing the degree of ischemia of the abdominal cavity organs; when examining the microcirculation of the intestinal wall, its violation is noted at the level of intra-abdominal pressure above 10 mm hg. Art. [8].

Also, with intra-abdominal hypertension, oxygenation of the stomach wall decreases, which is manifested by a decrease in the pH of its mucous membrane. Considering the decrease in cardiac output and impaired urinary function, sequestration of fluid in the third space and edema of the intestinal walls are aggravated, which leads to the progression of intra-

abdominal hypertension, thereby closing the vicious circle [9].

Impaired blood supply to the intestine also significantly affects intestinal motor activity. This can be due to both local and general disorders in the circulatory system. Among them, a drop in systemic blood pressure, an increase in the resistance of the vascular bed, and a violation of microcirculation as a result of increased thrombosis were proven.

When the vasoconstrictor nerves are irritated, sympathetic mediators are released, which, against the background of developing hypoxia, impaired blood flow, and hemodynamics, form the inhibitory effect of intestinal peristalsis.

Intraenteral hypertension is considered to be one of the leading factors in the development of blood supply disorders to the intestinal mucosa and subsequently to all its walls. This creates the effect of mechanical compression of blood vessels, particularly capillaries.

The blood supply system of the small intestine has a number of peculiar features. The blood vessels are more isolated by their location in its muscular layer. Blood circulation in this layer of the intestine creates conditions for capillaries, which, in the form of a drainage system, supply its mucous membrane. This mechanism is histologically manifested by vascular drainage systems that provide blood flow in the submucosa and mucous membranes.

The vessels of the muscular layer of the intestine form a double capillary blood supply network. The first network has an external location that runs parallel to the smooth muscle bundles of the longitudinal layer of the muscular membrane. The second network has an internal location that runs in the circular direction of the muscular membrane.

Thus, from the point of view of pathogenesis, acute intestinal obstruction is a stage-based pathological process, at the first stage of which reflex processes predominate, associated mainly with overstretching of the adductor part of the intestine. Further, increasing enteral insufficiency leads to the progression of water-electrolyte disorders and the penetration into the systemic circulation (as a result of malabsorption of various substances from the lumen of the intestine), which serves as the beginning of the development of endotoxicosis. It should be noted that impaired microcirculation of the intestinal wall plays one of the leading roles in the pathogenesis of acute intestinal obstruction.

A thorough understanding of the physiology and pathology of acute intestinal obstruction requires a thorough understanding of the intestine's anatomy. The basis of intestinal anatomy is rooted in intestinal embryology. Starting from the fourth week of pregnancy, the primitive intestine forms when the head, tail, and lateral folds include the dorsal portion of the yolk sac [10].

The three germinal layers of the primitive intestine differentiate into specific elements of the mature intestine [11]. The endoderm forms the intestinal mucosa, liver, and pancreas, while the splanchnopleuric mesoderm forms connective tissue and muscle components. Ectodermal components contribute to the functioning of the intestinal nervous system [12].

The primitive intestine may be in development and anatomically divided into anterior, intermediate and posterior. The previous stage of intestinal development passes into the formation of the pharynx, oesophagus, stomach, duodenum, pancreas, liver, biliary system and lower respiratory tract. The middle intestine forms the small intestine, the appendicular process, the ascending colon, and the proximal transverse colon. The posterior sheet of the intestinal tube forms the distal part of the transverse colon, the sigmoid colon, the rectum, and the proximal part of the anus.

The intestinal vascular system and the nervous system develop in tandem, and the macrovascular elements follow a similar anatomical distribution.

Intestinal vasculogenesis begins as a response to the rapid growth of intestinal parenchymal growth. Mesodermal cells form blood islands embedded in the mesodermal elements surrounding the sac wall. These blood islands differentiate into hemangioblasts under the control of fibroblast growth factor-2.

Hemangioblasts can be divided into two distinct groups. Peripheral hemangioblasts differentiate into angioblasts under the control of vascular endothelial growth factor, which later forms endothelial cells and primitive blood vessels [13].

Once this primary vascular bed is established, an additional vascular bed is added through angiogenesis, which is controlled by vascular endothelial growth factor, platelet-forming growth factor, and transforming growth factor-β [14].

Central hemangioblasts differentiate into hematopoietic stem cells, which further differentiate into their myeloid (monocytes, macrophages, neutrophils, basophils, eosinophils, erythrocytes, megakaryocytes, dendrites) and lymphoid (T cells, B cells, NK cells).

The three main arterial branches from the dorsal aorta are preserved and mature to provide mature derivatives of the primitive intestine. The celiac artery supplies derivatives to the anterior, the superior mesenteric

artery of the intestine, and the inferior mesenteric artery supplies derivatives to the hindgut. These major arterial trunks sequentially branch into smaller vessels until they eventually pierce the intestine's longitudinal and round muscle layers to enter the submucosa.

Arterioles branch into smaller arterioles. These small arterioles form vascular shunts with other larger arterioles. These arterioles remain in the submucosa of the intestine and represent a bridge between the micro- and macrocirculation of the intestine. Arteriole plexuses are the main sites of vascular resistance and, thus, the primary regulators of intestinal blood flow [15].

Smaller arterioles arise from the previous ones and enter the intestinal mucosa. Each level 3 arteriole enters a single villi, forming a terminal capillary network. Before entering the mucous membrane, these arterioles branch into capillary networks that enter the muscular layers of the intestine. Collecting venules from each villi flow into the mucous membrane. They do not run near their arterial counterparts until they reach the level of the submucosa [16].

Regulation of intestinal blood flow can be divided into external and internal elements [17].

Extrinsic regulation refers to control from a location other than the gut, often through the autonomic nervous system and cardiovascular reflexes. It usually functions to maintain systemic cardiovascular homeostasis and may work through local intestinal circulation.

Internal control of blood circulation in the intestine refers to the regulation produced by mediators formed and released locally in the intestine and its vessels. Internal regulation functions to preserve intestinal microcirculatory homeostasis and ensure the delivery of oxygen and nutrients to the intestine. There is a balance between vasoconstrictive and vasodilative effects in the intestine of newborns at the internal level [18].

Intestinal microcirculatory blood flow is primarily based on resting vascular resistance. It is the resistance for flow through the regional circulation under steady-state hemodynamic conditions. Blood flow is inversely proportional to resistance, so increased resistance leads to a decrease in blood flow, which is also poor. Vascular resistance is inversely proportional to the radius of the fourth power vessel. This means that small vasoconstrictive or vasodilative changes cause significantly more significant changes in vascular resistance and blood flow. The intestinal microcirculation of a newborn child is characterized by lower vascular resistance at rest compared to the elderly. This leads to a higher blood flow rate and increased delivery of nutrients and oxygen [19].

For more than forty years, scientists have investigated the potential link between intestinal circulation and intestinal obstruction. Initial observations noted a correlation between perinatal asphyxia and subsequent perforation of the gastrointestinal tract [20].

This was called the diving reflex because it was physiologically similar to the known redistribution of cardiac output to the brain observed in diving mammals [21].

It was assumed that the external neurogenic redistribution of blood flow from the splanchnic organs to the brain led to intestinal ischemia. Initial studies in newborn piglets confirmed this hypothesis, demonstrating mucosal damage after acute asphyxia [22]. This hypothesis fell out of favour because later studies noted that patients with acute intestinal obstruction rarely suffered from such disorders in the circulatory system in the elderly and old age [23]. Sustained adrenergic stimulation, a central aspect of the reflex, causes a sustained decrease in intestinal blood flow and may cause intestinal tissue hypoxia [24].

Some evidence suggests that abnormal parameters of superior mesenteric artery blood flow with high vascular resistance are related to intestinal tone [25] and possibly a later stage of acute intestinal obstruction [26]. However, other attempts to link acute intestinal obstruction to macrocirculatory disorders of intestinal blood flow have yielded mixed results.

Because of these events, much attention has been paid to studying intestinal microcirculation. Emerging evidence suggests that dysregulation at this level is associated with the development of acute intestinal obstruction. In experimental models of rats with acute intestinal obstruction, intestinal microvascular blood flow leads to intestinal damage.

In the crypts of the intestinal mucosa, a rich network of microvessels is formed. The capillary network around the crypts has a basket-like shape. The capillaries along the crypts rise to the intestine's lumen, forming vascular rings around the entrance.

Some capillaries, which are located along and across the bottom of crypts, can carry blood to the venules. They immediately go to the venous plexus of the submucosa.

From the rest of the pericryptal capillaries, blood flows into the villi microvessels. Near the base of the intestinal villi, the subepithelial capillary plexus continues into a series of more or less straight capillaries, interconnected by capillary rings located around the crypt openings adjacent to the villi. The arteriole, which supplies blood to the villi microvessels, is localized centrally. At the

apex of the villi, it passes into the capillary plexus, consisting of cylindrical subepithelial exchange microvessels, often convoluted. More significant of them go along the ridge of the villi. All these capillaries move towards the base of the villi, where they pass into venules. Thus, a fountain type of distribution is formed.

When studying resected fragments of the small intestine in the strangulation zone on histological specimens, the elements of the microcirculatory bed are practically not differentiated. Destructive changes in most microvessels are noted - most of the visual field are haemorrhages. Near the demarcation line, both proximal and distal, there are significant changes in the structure of blood vessels, characterized by rupture of loops and capillary networks, violation of the integrity of arterioles and venules, and an abundance of extravasates. At a distance of nine centimetres from the strangulation zone, there is a pronounced expansion of the venules; their walls are corrugated. Most arterioles are spasmodic, most capillary networks are ruptured, and small haemorrhages are noted behind the contour of the vascular walls.

A decrease in pressure in arterioles and capillaries, venous stasis, increased permeability of capillary walls and extravasation of formed blood elements have been proven. The ratio of the intensity of blood circulation in the submucosa and external plexuses changes; if they usually are 2:1, then in intestinal obstruction, they are determined as 1:4, i.e. the mucous membrane experiences a greater blood deficit.

Inhibition of motor activity determines the next pathophysiological stage of acute intestinal obstruction, in which electrolyte disorders and endotoxicosis become the leading ones.

Distension of the intestine stimulates the secretory activity of the intestinal wall, which leads to the filling of the intestine with liquid contents. At the same time, increasing malabsorption prevents the reabsorption of water; it is sequestered in the intestinal lumen.

Swelling of the mucous membrane and submucosa of the intestine increases, and transudation of the liquid part of the blood into the lumen of the intestine appears. Together, these processes entail the progression of hypovolemia, deterioration of the rheological properties of the blood, conditions arise for the development of circulatory hypoxia, which increases paresis, absorption disorders in the intestine, and protein and electrolyte disorders also progress with acute intestinal obstruction. The predominance of catabolic processes over anabolic ones and the loss of protein with transudate into the lumen of the intestine and abdominal cavity explain the hypoproteinemia described by some authors.

Electrolyte disorders are diverse and must be taken into account when planning infusion therapy. Many studies draw attention to the possibility of hyperkalemia due to the appearance of intracellular potassium from destroyed structures and a decrease in the excretion of potassium by the kidneys. The concentration of sodium in the blood practically does not change. Some authors describe hypochloremia that develops with acute intestinal obstruction, which, however, does not always occur.

In the absence of propulsive peristalsis, the intestinal contents become a suitable environment for the development of microorganisms. It is reliably known that with intestinal obstruction, the number of colonies of aerobes and anaerobes increases, and their growth is noted in both the jejunum and ileum.

Changes occurring in the wall of the adductor colon significantly reduce its protective capabilities, which leads to the penetration of microorganisms through the intestinal barrier. Thus, the adductor loop is a source of abdominal cavity infection. Experiments have shown that acute intestinal obstruction makes it possible to absorb large molecules, mainly albumin. Therefore, the waste products of microorganisms, putrefaction and fermentation of food masses, absorbed from the intestinal lumen, become factors of primary aggression in developing endotoxicosis. Endogenous intoxication is the leading pathogenetic link of acute intestinal obstruction. It is a complex, staged, multifactorial autocatalytic process that acquires a universal character over time, regardless of the triggering mechanisms.

Conflict of interest – none

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