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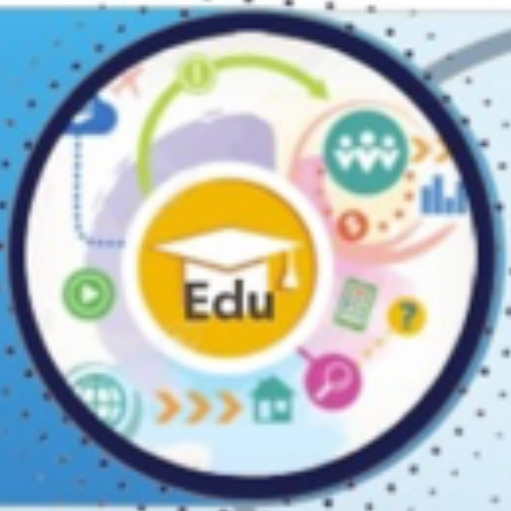

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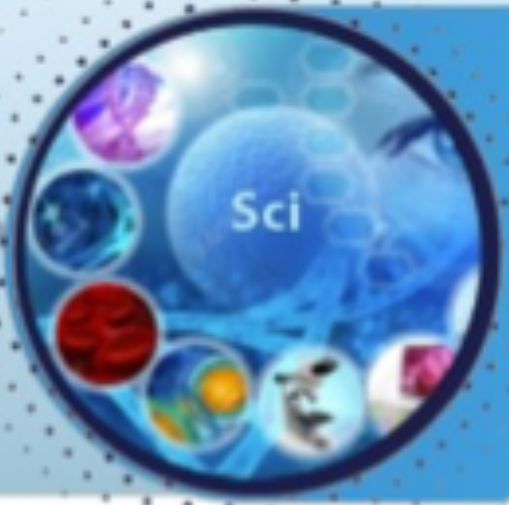

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Review Article

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Pathogenesis of Acute Intestinal Obstruction in Elderly and Senile Age Patients

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ABSTRACT

Pathomorphological changes in the intestine during the development of acute intestinal obstruction depend on the form of the lesion of this organ. Thus, in patients with strangulation intestinal obstruction, the main area of the lesion 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 and the area of the intestine itself, leading to a violation of trophism in this area. In contrast to the above, in case of obturational intestinal obstruction, the main pathological manifestations occur in the leading part of the intestine. With the development of dynamic intestinal obstruction, the intestine does not have a certain area of the lesion and all pathological processes are diffuse. A common, unifying content in the development of acute intestinal obstruction in elderly and senile patients is considered to be the presence of a number of pathological syndromes, such as enteral insufficiency, water-electrolyte balance disorders, protein imbalance, endotoxiscosis, sepsis, etc. To properly understand the physiology and pathology of acute intestinal obstruction, a deep understanding of the anatomy of the intestine is required. The basis of intestinal anatomy is rooted in intestinal embryology.

This review article presents up-to-date information on the anatomical prerequisites of pathogenesis and clinical and laboratory changes in acute intestinal obstruction in elderly and senile patients.

Keywords: acute intestinal obstruction, old and senile age, mechanism of disease development

Among the most common pathologies in urgent abdominal surgery, in elderly and senile patients, acute intestinal obstruction is still in the lead [1].

Back in 1954, O.H. Kment provided detailed information on the features of this nosological form of acute surgical pathology [2]. Based on the analysis of a sufficient amount of clinical material, he was offered a therapeutic and diagnostic complex algorithm, which made it possible to identify risk factors for the development of

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postoperative complications of acute intestinal obstruction in patients of elderly and senile age.

In elderly and senile patients, according to N.J. Menon and others. [3], J. Waisberg and others. [4], D.Y. Cheung et al. [5], recognized is the complexity of pathophysiological changes in the body, leading to severe forms of acute intestinal obstruction in the postoperative period, which in turn contribute to a high frequency of fatal outcomes. According to V.N. Bernakinas [6] the presence of a large number of concomitant diseases also play a large role in this.

Acute intestinal obstruction due to intestinal fringement in the hernial sac has its own characteristics in the pathogenesis of intestinal necrosis. It is known that the time of development of necrosis of the pinched intestinal loop at the cessation of arterial flow to it has a longer time than in case of 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 in case of impaired venous outflow, the development time of intestinal necrosis is 3-4 times shorter. There are also reports of reducing this period to 1 hour [7].

In clinical practice, there are descriptions that with the surgical resolution of intestinal obstruction in cases of a pinched hernia, the external picture of the change in the pinched intestinal loop may not always meet the criteria of viability. The change in the color of the intestinal serous membrane, the appearance of pulsation and peristalsis of the pinched intestinal loop are considered to be the criteria for its preservation and not to resort to resection. At the same time, in elderly and senile patients, as described in literary sources, there is a certain critical period, when even in the case of restoration of arterial blood supply to the intestinal loop, necrobiotic changes in the mucous membrane still continue. As the authors of these publications point out, this unaccounted-infored process is the reason for the development of perforation of the pinched 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 main reasons for this pathological process in elderly and senile patients are the formation of a lot of small blood clots. 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 gate vein. As a result, the coagulopathy mechanism is triggered. Such a phenomenon in clinical practice is called "no-reflow" [8].

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. The decay products of tissues occurring in the intestinal mucosa also contribute to this. Conditions are created for the rapid penetration of microorganisms through the affected intestinal wall, its translocation at the beginning into the local and then into the systemic bloodstream [9].

An obstacle that occurs in the intestinal cavity, due to its infringement, leads to the stretching of the leading part of the organ. The movement of the contents through the intestine stops and a reflex change in peristalsis occurs. It acquires a propulsive character. At the same time, the duration of irritation of the intestinal wall, namely the receptors, leads to the inhibition of the peristaltic processes of the entire gastrointestinal tract. There is a violation of the evacuation of contents, which is aggravated over time and in elderly and senile patients. Such aggravation contributes to the progressive accumulation of gases and fluid in the intestinal cavity by stretching its walls more and more [10].

In their observations, clinicians noted that this phenomenon in young and mature patients develops on a local scale, in the form of an incomplete volume of signs of systemic inflammatory response syndrome, while in elderly and senile patients they can become common and manifest as a general reaction by type of current, especially with severe pain syndrome [11].

The compensatory reaction of the body is aimed at inhibiting intestinal peristalsis, which replaces hyperperistalsis. The closing link of this pathological process is the development of intestinal paresis.

As the results of studies of these pathological processes show, the mechanisms of intestinal paresis development are based on reflex reactions of the body, hypoxic states (primarily in the machine of the intestine itself), toxemia of water-electrolyte balance disorders [12].

Studies have proven that irritation of the nerve receptors of the intestinal mucosa is the main reason for the development of inhibitory reflexes between different parts of the intestine (entero-enteral). Such reflexes are the basis for the suppression of the contractile activity of the intestinal muscles. As a result, paresis and intestinal paralysis develop [13].

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, blood vessels that feed them and the Portocaval

system as a whole. The researchers have found that the blood supply to the abdominal cavity and retroperitoneal space is disturbed when the intra-abdominal pressure is raised to 15 mm Hg. Organ blood flow is not reduced in proportion to the cardiac output, but depends on the so-called perfusion pressure of the abdominal cavity, which is the difference between average arterial and intra-abdominal pressure. The value of perfusion pressure is a criterion for assessing the degree of ischemia of the abdominal organs, in the study of microcirculation of the intestinal wall, its violation is noted at the level of intra-abdominal pressure above 10 mm Hg. [14].

Also, in intra-abdominal hypertension, the oxygenation of the stomach wall decreases, which is manifested by a decrease in the pH of its mucous membrane. Taking into account also the decrease in cardiac output and violation of urinary function, the sequestration of fluid in the third space, swelling of the intestinal walls are aggravated, which leads to the progression of intra-abdominal hypertension, thereby closing the vicious circle [15].

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

In case of irritation of the vasoconstrictor nerves, sympathetic mediators are released, which, against the background of developing hypoxia, blood flow disorders and hemodynamics, form the inhibitory effect of intestinal peristalsis.

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

Se sabe que el sistema de suministro de sangre del intestino delgado tiene una serie de peculiaridades. Los vasos sanguíneos del intestino delgado están más separados por la ubicación en su capa muscular. La circulación sanguínea en esta capa del intestino crea condiciones para los capilares, que en forma de sistema de drenaje suministran su membrana mucosa. Tal mecanismo se manifiesta histológicamente por los sistemas de drenaje vascular que proporcionan flujo sanguíneo en las membranas submucosa y mucosa.

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

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

Three germ layers of the primitive intestine are differentiated into specific elements of the mature intestine [16]. The endoderm forms the intestinal mucosa, liver and pancreas, while the splanchnopleuric mesoderm forms connective tissue and muscle components, and ectodermal components contribute to the work of the intestinal nervous system [17].

The primitive intestine may be in development and anatomically divided into anterior, intermediate and posterior. The previous stage of intestinal development turns into the formation of the pharynx, esophagus, 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 leaf of the intestinal tube forms the distal part of the transverse colon, sigmoid colon, rectum and proximal part of the anus.

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

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

Hemangioblasts can be divided into two separate groups. Peripheral hemangioblasts differentiate into angioblasts under the control of the vascular endothelial growth factor, which later form endothelial cells and primitive blood vessels [18].

After the creation of this primary vascular bed, an additional vascular bed is added through angiogenesis

under the control of vascular endothelium growth factor, platelet-forming growth factor and transforming growth factor-β [19].

Central hemangioblasts are differentiated into hematopoietic stem cells, which are further differentiated 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. Celiac artery supplies anterior derivatives, the upper mesenteric artery of the intestine, and the lower mesenteric artery supplies derivatives of the posterior intestine. These main arterial trunks are successively branched into smaller vessels until they eventually pierce the longitudinal and round muscle layers of the intestine to enter the submucosal membrane.

Arterioles branch into smaller arterioles. These small arterioles form vascular shunts with other larger arterioles. These arterioles remain in the submucus membrane of the intestine and are a bridge between intestinal micro- and macrocirculation. Arteriole plexuses are the main places of vascular resistance and thus the main regulators of intestinal blood flow [20].

Smaller arterioles arise from the previous ones and enter the intestinal mucosa. Each level 3 arteriole enters one villi, forming a terminal capillary network. Before entering the mucous membrane, these arterioles branch into capillary networks, which enter the muscular layers of the intestine. Collecting venules from each villi flow into the mucous membrane. They do not run in close proximity to their arterial counterparts until they reach the level of the submucosal membrane [21].

Regulation of intestinal blood flow can be divided into external and internal elements [22].

External regulation refers to control from a place other than the intestines, often through the autonomic nervous system and cardiovascular reflexes. It usually functions to preserve systemic cardiovascular homeostasis and can work at the expense of local intestinal circulation.

Internal control of blood circulation in the intestine refers to the regulation produced by mediators, which are formed and released locally in the intestine and its vessels. Internal regulation functions for local preservation of intestinal microcirculatory homeostasis to ensure the delivery of oxygen and nutrients to the intestines. There is a balance between vasoconstrictive and vasodilative effects in the intestines of newborns at the internal level [23].

Intestinal microcirculating blood flow is largely based on vascular resistance to rest. This is resistance to the flow through regional circulation under stationary hemodynamic conditions. Blood flow is inversely proportional to resistance, so increased resistance leads to a decrease in blood flow, with the class of which it is also scarce. The vascular resistance is inversely proportional to the radius of the 4th power vessel. This means that small vasoconstrictive or vasodilative changes cause significantly larger 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 an increase in the delivery of nutrients and oxygen [24].

Scientists have been investigating the potential relationship between intestinal circulation and intestinal obstruction for more than forty years. Initial observations noted a correlation between perinatal asphyxia and subsequent perforation of the gastrointestinal tract [25].

This was called a diving reflex, as it was physiologically similar to the known redistribution of cardiac output into the brain observed in diving mammals [26].

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 damage to the mucous membrane after acute asphyxia [27]. This hypothesis fell out of favor, as later studies noted that patients with acute intestinal obstruction not in the elderly and in old age rarely suffered from such disorders in the circulatory system [28]. Steady adrenergic stimulation, the central aspect of the reflex, causes a steady decrease in intestinal blood flow and can cause hypoxia of intestinal tissue [29].

Some evidence suggests that abnormal parameters of blood flow of the upper mesenteric artery with high vascular resistance are associated with intestinal tone [30] and possibly a later stage of acute intestinal obstruction [31]. Nevertheless, other attempts to associate acute intestinal obstruction with macrocirculatory disorders of intestinal blood flow have yielded mixed results.

Because of these events, much attention was paid to the study of intestinal microcirculation. There is evidence 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 led to intestinal damage.

In the crypts of the intestinal mucosa, a very rich network of microvessels is formed. The capillary network that forms around the crypts has a basket-like shape. Capillaries along the crypts rise to the intestinal lumen and thus form vascular rings around the entrance.

Some capillaries that are located along and across the bottom of the crypts can carry blood into the venules. They immediately go into the venous plexus of the submucosal membrane.

From the other pericryptal capillaries, blood flows into the microvessels of the villi. Not far from the base of the intestinal villi, the subepithelial capillary plexus continues in a series of more or less straight capillaries connected by each other by capillary rings, which are located around the openings of the crypts adjacent to the villi. The arteriole, which supplies blood to the microvessels of the villi, is localized centrally. At the top of the villi, it turns into a capillary plexus consisting of subepithelial exchange microvessels of cylindrical shape, often having a twisted configuration. The larger ones go along the crest of the villi. All these capillaries move towards the base of the villi, where they turn into venules. Thus, a fountain type of distribution is formed.

When studying resectioned fragments of the small intestine in the strangulation zone on histological preparations, the elements of the microcirculatory bed are practically not differentiated. There are destructive changes in most microvessels - most of the field of view are hemorrhages. In the immediate vicinity of the demarcation line, both proximally and distally, there are significant changes in the design of blood microvessels, characterized by rupture of loops and networks of capillaries, violation of the integrity of arterioles and veins, abundance of extravasates. At a distance of nine centimeters from the strangulation zone, there is a pronounced expansion of the vein, their walls are corrugated. Most arterioles are spasmous, most of the capillary networks are torn, small hemorrhages 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 blood circulation intensity in the submucosal and external plexus changes, if they are normally 2:1, then in intestinal obstruction they are defined as 1:4, i.e. the mucous membrane has a greater blood deficiency.

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

Stretching 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 absorption disorders prevent the reabsorption of water, it is sequested in the intestinal lumen.

The swelling of the mucous membrane and submucosal layer of the intestine increases, the transudation of the liquid part of the blood into the intestinal lumen 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, as well as protein and electrolyte disorders progress in acute intestinal obstruction. The predominance of catabolic processes on anabolic, the loss of protein with transudate in the intestinal lumen and abdominal cavity explain the hypoproteinemia described by some authors.

Electrolyte disorders are diverse, they 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, as well as 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 developing in acute intestinal obstruction, which, however, does not always occur.

In the absence of propulsive peristalsis, the intestinal contents become a good environment for the development of microorganisms. It is well known that in case of intestinal obstruction, the number of colonies of aerobes and anaerobes increases, their growth is observed in both the skinny and ilium.

Changes occurring in the wall of the leading intestine significantly reduce its protective capabilities, which leads to the penetration of microorganisms through the intestinal barrier. Thus, the leading loop is a source of infection of the abdominal cavity. The experiment proved that with acute intestinal obstruction, it is possible to absorb large molecules, in particular albumin. Therefore, the products of vital activity of microorganisms, rotting and fermentation of food masses, absorbed from the intestinal lumen, become factors of primary aggression in the development of endotoxicosis. Endogenous intoxication is the leading pathogenetic link of

acute intestinal obstruction and is a complex, staged, multifactorial atocatalytic process, which over time becomes universal regardless of the trigger mechanisms.

Currently, computed tomography is widely used in clinical practice to predict and assess complications associated with acute intestinal obstruction. However, this diagnostic method has a number of drawbacks. For example, the use of computed tomography exposes patients to ionizing radiation, which can increase the risk of radiation-associated cancer. In addition, it is still difficult to differentiate stragulatory intestinal obstruction from obturation intestinal obstruction. Therefore, there is an urgent need to develop a more advanced diagnostic method to distinguish acute strangulation intestinal obstruction from obturation intestinal obstruction, the latter of which does not always require emergency surgery.

The literature reports that blood clotting indicators, in particular fibrinogen, are useful markers in the diagnosis of blood clotting disorders that may occur as a result of ischemia and hypoxia [32].

I.G. Schoots et al. [33] reported intraluminal coagulation and fibrin deposition in the mouse model of intestinal ischemia-reperfusion.

In another study using a model of a disadvantaged hernia in rats, N. Zeybek and his colleagues [34] showed that D-dimer levels increased as ischemia worsened, and this change was significantly correlated with intestinal necrosis. In addition, in the same study, it was found that the number of leukocytes was significantly higher in the experimental group with intestinal necrosis compared to the control group.

Strangulation intestinal obstruction in elderly and senile patients is pathologically accompanied by intestinal microcirculation disorders, microthrombosis, blood clotting, hypoxia, tissue ischemia and even intestinal necrosis.

Thus, it can be assumed that the main pathological changes can lead to changes in blood clotting levels.

To date, the use of these coagulation indicators for differential diagnosis of acute strangulation intestinal obstruction compared to obturation intestinal obstruction has not been deeply studied.

In this retrospective study of patients with intestinal obstruction, many scientists sought to determine whether there is a link between blood clotting rates, as well as markers of inflammation and two types of intestinal obstruction. In addition, an assessment of their diagnostic value in the differentiation of acute strangulatory and obturation intestinal obstruction is required.

In case of intestinal obstruction, the following pathological changes can occur: dehydration, electrolyte disorders, secondary infection, intestinal microcirculation, blood clotting, microthrombosis, blood diseases, tissue ischemia, hypoxia and, ultimately, necrosis. Some of these effects (for example, ischemia and tissue hypoxia) can cause blood clotting disorders.

Of a number of clotting indicators, including prothrombin time, APTT, fibrinogen, thrombin time and D-dimer, it is fibrinogen that can be used as a screening or diagnostic indicator of hypercoagulation, and is also one of the molecular markers for monitoring coagulation, fibrinolysis and thrombosis.

During coagulation, the first indicators are eventually split to the D-dimer using thrombin and plasmin. However, the D-dimer index in plasma is associated with the degree of intestinal damage and intestinal obstruction necrosis when intestinal obstruction occurred.

In the presented results of foreign researchers, the indicator of thrombin time differed not only between patients with acute obturation and strangulation intestinal obstruction, but also between subgroups, including such types of intestinal lesions.

Compared to the prothrombin time, only the blood clotting index between stragulatory and obturation intestinal obstruction differed significantly. This result implied that the increased Fibonacci level was caused not only by acute phase protein, but also by blood clotting factors. Thus, the Fibonacci level can be used to indirectly reflect the degree of intestinal obstruction and intestinal necrosis, as well as intestinal blood flow in case of intestinal obstruction.

For a long time, leukocytes have been used as an indicator for the diagnosis of strangulatory intestinal obstruction, but modern literature data have shown that leukocytes do not differ significantly between the types of intestinal obstruction.

A study was conducted that scientists specifically compared the dynamics of changes in the level of leukocytes, C-reactive protein and Fibonacci using the ROC curve analysis and found that the areas under the ROC curve for C-reactive protein (0.78) and fibrinogen (0.80) were larger than for leukocytes (0.58). Therefore, C-reactive protein and fibrinogen are more important in the diagnosis of acute strangulation intestinal obstruction.

Since an infectious complication often occurs in acute intestinal obstruction in elderly and senile patients, the data provided by the researchers have shown that inflammation markers, such as C-reactive protein and neutrophils, also increase. In particular, the level of C-reac-

tive protein significantly differed between the group with obturational intestinal obstruction and the group with strangulation intestinal obstruction, as well as the ischemic subgroup and the subgroup of necrosis of patients with strangulation intestinal obstruction.

To date, there is evidence that the level of C-reactive protein can reflect the inflammatory state of stragulatory intestinal obstruction with necrosis, which is consistent with other studies.

In addition, the Fibonacci level was positively correlated with the level of C-reactive protein, but negatively correlated with thrombin time. Our results showed that coagulation rates, in addition to inflammation, may also reflect strangulation intestinal obstruction. Meanwhile, it was demonstrated that increased Fibonacci levels in acute strangulation intestinal obstruction were caused by blood clotting factors.

Moreover, results were obtained that showed that the combination of blood clotting markers and inflammation markers may be better for diagnosing acute strangulation intestinal obstruction.

Fibonacci and C-reactive protein have high specificity and positive prognostic value, indicating that both of them are of some importance for intestinal suffocation in patients with intestinal obstruction (but other factors affecting clotting should be excluded).

The importance of lactic acid in acute strangulatory intestinal obstruction is rarely reported.

Thus, fibrinogen and C-reactive protein show a good ability to distinguish between acute strangulation and obturation intestinal obstruction. This indicates that blood clotting indicators and inflammation markers can be used as markers to predict unsatisfactory results in the treatment of acute intestinal obstruction.

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KEKSA YOSHDAGI BEMORLARDA O'TKIR ICHAK TO'SIQLARI PATOGENEZI

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ABSTRAKT

O'tkir ichak to'siqlari rivojlanishi davrida ichakdagi patomorfologik o'zgarishlar bu organning zararlanish shakliga bog'liq. Shunday qilib, strangulyatsiya ichak to'siqlari bilan og'rigan bemorlarda zararlanishning asosiy sohasi strangulyatsiya maydoni, ya'ni mesenteriyaning asosiy tomirlari va nervlarining siqilishi sodir bo'ladigan joy sanaladi. Bu bilan birga ichak sohasining o'zida siqilish ham kuzatiladi va bu sohada trofik buzilishlarga olib keladi. Yuqoridagilardan farqli o'laroq, ichak to'siqlari holatida asosiy patologik ko'rinishlar ichakning adduktor qismida paydo bo'ladi. Dinamik ichak to'siqlarining rivojlanishi bilan ichakning aniq zararlanish sohasi yo'q va barcha patologik jarayonlar diffuz bo'ladi. Enterik yetishmovchilik, suv-elektrolit balansi buzilishi, oqsil muvozanati, endotoksikoz, sepsis va h.k. kabi bir qator patologik sindromlarning mavjudligi keksa va senil bemorlarda o'tkir ichak to'siqlarining rivojlanishida keng tarqalgan, birlashtiruvchi tarkib sanaladi. Ichak anatomiyasining asosi ichak embriologiyasida ildiz otgan.

Ushbu ko'rib chiqish maqolasida keksa va qari bemorlarda patogenezning anatomik prekrizitlari va o'tkir ichak to'siqlarining klinik va laboratoriya o'zgarishlari bo'yicha zamonaviy ma'lumotlar keltirilgan.

Tayanch iboralar: o'tkir ichak to'siqlari, qariyalar va qarilik yoshi, kasallik rivojlanish mexanizmi

ПАТОГЕНЕЗ ОСТРОЙ КИШЕЧНОЙ НЕПРОХОДИМОСТИ У БОЛЬНЫХ ПОЖИЛОГО И СТАРЧЕСКОГО ВОЗРАСТА

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АБСТРАКТ

Патоморфологические изменения в кишечнике при развитии острой кишечной непроходимости зависит от формы поражения данного органа. Так, у больных со странгуляционной кишечной непроходимостью основной зоной поражения считается странгуляционный участок, то есть место, где происходит сдавление основных сосудов и нервов брыжейки. Наравне с этим сдавление происходит и самого участка кишечника, приводящих к нарушению трофики в данной зоне. В отличие от вышесказанного, при обтурационной кишечной непроходимости основные патологические проявления происходят в приводящем отделе кишечника. При развитии динамической кишечной непроходимости кишечник не имеет определенный участок поражения и все патологические процессы носят диффузный характер. Общим, объединяющим контентом в развитии острой кишечной непроходимости у больных пожилого и старческого возраста считается наличие ряда патологических синдромов, таких как энтеральная недостаточность, расстройства водно-электролитного баланса, белковый дисбаланс, эндотокискоз, сепсис и др. Чтобы правильно понять физиологию и патологию острой кишечной непроходимости требуется глубокое понимание анатомии кишечника. Основа кишечной анатомии уходит корнями в кишечную эмбриологию.

В данной обзорной статье представляются современные сведения анатомических предпосылок патогенеза и клинико-лабораторных изменений при острой кишечной непроходимости у больных пожилого и старческого возраста.

Ключевые слова: острая кишечная непроходимость, пожилой и старческий возраст, механизм развития заболевания