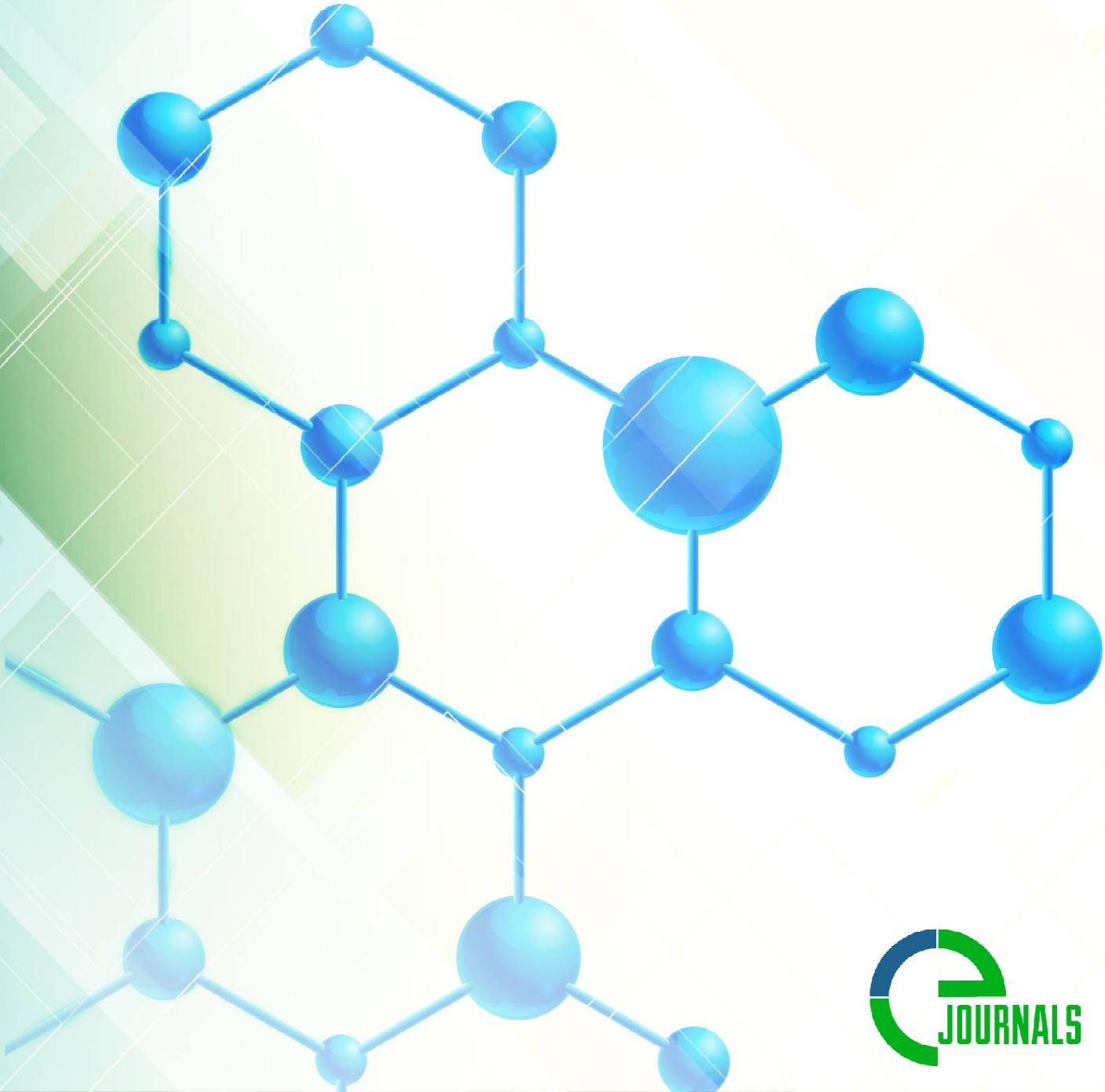
EUROPEAN JOURNAL OF

MOLECULAR MEDICINE



JOURNALS

European Journal of Molecular medicine

Volume 1, No.4, September 2021

Internet address: ttp://ejournals.id/index.php/EJMM/issue/archive
E-mail: info@ejournals.id
Published by ejournals PVT LTD
DOI prefix: 10.52325
Issued Bimonthly
Potsdamer Straße 170, 10784 Berlin, Germany

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INTRA-ABDOMINAL HYPERTENSION - CONDITIONS OF OCCURRENCE, PATHOGENETIC MECHANISMS OF DEVELOPMENT

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Abstract.Throughout the world, intra-abdominal hypertension (IAH) is recognized as a serious cause of death in critically ill patients. Despite the existing achievements in the field of surgery, anesthesiology and resuscitation, urgent medicine, optimization of organizational measures, the introduction of new technologies into medical practice, a high mortality rate remains.

Key words: intra-abdominal hypertension - (IAH), abdominal cavity syndrome - (ACS), intra-abdominal pressure - (IAP), abdominal compression syndrome - (ACS)

ИНТРААБДОМИНАЛЬНАЯ ГИПЕРТЕНЗИЯ- УСЛОВИЯ ВОЗНИКНОВЕНИЯ, ПАТОГЕНЕТИЧЕСКИЕ МЕХАНИЗМЫ РАЗВИТИЯ

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Аннотация. Во всем мире внутрибрюшная гипертензия (ВБГ) признана как серьезная причина смертности у пациентов, находящихся в критическом состоянии. Несмотря на существующие достижения в области хирургии, анестезиологии и реаниматологии, ургентной медицине оптимизацию организационных мероприятий, внедрение новых технологий в медицинскую практику, сохраняется высокий уровень летальности.

Ключевые слова: интраабдоминальную гипертензию - (ИАГ), синдром брюшной полости - (СБП), внутрибрюшного давления - (ВБД), синдрома абдоминальной компрессии - (САК)

According to world statistics, the incidence of intra-abdominal hypertension syndrome - (IAHS) in patients with trauma to the abdominal cavity and after abdominal surgery reaches 30% with the occurrence of abdominal syndrome - (AS) in 5.5% of cases [1]. In therapeutic intensive care units, the incidence of intra-abdominal pressure - (IAP) can be up to 24%. The mortality rate from AS is high - 42-68%, but if the syndrome is left untreated, it rises to 100% [2].

Normal abdominal pressure in the abdomen is about zero. Its increase is not immediately accompanied by the development of AS. Of great importance is the rate of increase in (IAP) [3].

A variety of diseases of the abdominal cavity and retroperitoneal space can lead to an increase in IAP. The most common among them are: acute intestinal obstruction, excessive tension when suturing the anterior abdominal wall with compression of internal organs, laparoscopic methods of performing operations, intra-abdominal bleeding, hemorrhage into the retroperitoneal space, as well as intestinal paresis with a closed abdominal trauma, fractures of the pelvic bones, acute pancreatitis, the transferred surgical intervention [4].

Currently, the following list of diseases and conditions leading to the development of abdominal compression syndrome (ACS) is generally recognized [5,8]: pathological conditions after surgery:

post-traumatic, postoperative and complicated by bleeding into the abdominal cavity and retroperitoneal space; laparotomy surgery and hernia repair with contraction of the abdominal wall during suturing; widespread peritonitis; infiltration or edema of internal organs after surgery; pneumoperitoneum during laparoscopy; postoperative intestinal obstruction; (acute expansion of the stomach) postoperative paresis of the gastrointestinal tract. Post-traumatic conditions (trauma-related):

external compression; burns and polytrauma; post-traumatic intra-abdominal or retroperitoneal bleeding; edema and infiltration of internal organs after massive infusion therapy. As complications of somatic diseases:

peritonitis; massive hydration therapy; decompensated (tense) ascites with liver cirrhosis or tumors; acute pancreatitis; acute intestinal obstruction; hemorrhages from a ruptured aneurysm of the abdominal aorta. The predisposing factors of ACS include: hypothermia at temperatures less than 33 ° C .; shift of the acid-base balance spectrum towards acidosis (blood pH less than 7.2); volumetric blood transfusion (more than 10-20 doses / day); pathological changes in the coagulogram of various origins; septic conditions regardless of etiology.

The growth of IAP is more often observed during operations of large ventral hernias, on large vessels of the peritoneum, with pancreatic necrosis, intestinal obstruction, or widespread peritonitis [2].

Considering the pathophysiology of this pathological syndrome, it should be remembered that with an increase in pressure in the abdominal cavity, a large role belongs to the elastic properties of its walls and the volume of its contents [2,9]. At the same time, it is difficult to establish a linear dependence of IAP on the volume of the abdominal cavity. In this case, intra-abdominal pressure increases disproportionately sharply in response to the same increase in abdominal volume [9].

An increase in intra-abdominal pressure is not always accompanied by the occurrence of ACS, which is why there are no exact figures in the literature, at which this pathological syndrome develops. However, there is a pattern: the higher the intra-abdominal pressure and the more factors leading to an increase in intra-abdominal pressure, the more likely the development of ACS is. Moreover, the faster the intra-

abdominal pressure builds up, the higher the likelihood of ACS.

Increased intra-abdominal pressure shifts the diaphragm high up, increases the pressure in the pleural areas [5,6] and thereby significantly increases the mean intrathoracic pressure, which is reflected in the tone of large vessels and heart function [6]. Increased pressure in the chest cavity reduces ventilation of the lungs, significantly reduces the severity of filling the ventricles of the heart, changes the pressure gradient on the myocardium, increases the pressure in the pulmonary capillaries, reduces the stroke volume of the heart, and reduces venous return [6].

C. Toenes et al. [8] proved that the increase in intra-abdominal pressure up to 40 mmHg causes the appearance of atelectasis in the lower lobes of both lungs. Increasing tachycardia significantly reduces cardiac output [8].

The total peripheral vascular resistance will increase with increasing intra-abdominal hypertension. Blood pressure in patients with ACS can be different with a pronounced hypothesis in the terminal stage of the process [5,9]. The actual pressure indicators in the large vessels of the chest cavity during the development of SAH cannot reflect the true value of the circulating blood volume, nor the objective state of cardiac activity [11]. Disorders of lymphatic drainage along the thoracic duct develop in proportion to the change in intra-abdominal pressure, and lymphatic drainage along it stops when the pressure in the abdominal cavity rises to 30 centimeter of water column [11].

An increase in pressure in the abdominal cavity significantly slows down blood flow through the inferior vena cava and significantly reduces the return of venous blood [12]. The volumetric blood flow along the superior mesenteric artery sharply decreases [5] and the blood supply to the mucous membrane of the gastrointestinal tract is disrupted [2] with the possible development of necrosis of all layers of the intestinal wall and peritonitis [2]. Heart failure and decreased renal function, as well as intensive infusion therapy aggravate the sequestration of fluid into the so-called "third space", intestinal edema and paresis, further increases intra-abdominal hypertension (IAH) and closes the "vicious circle". Loss of the barrier function of the ischemic intestinal mucosa aggravates the phenomenon of bacterial translocation, leading to their breakthrough into the systemic circulation and abdominal cavity and initiates the development and progression of the abdominal septic process [12].

It has been established that the existence of intra-abdominal hypertension over 25 mmHg more than one hour leads to a change in homeostasis and disrupts the barrier function of the mucous membrane of the digestive tract. Bacterial translocation occurs to the lymph nodes [1], spleen and portal vein. With intra-abdominal hypertension 10 mm. rt. Art., arterial hepatic blood flow decreases, and portal begins to suffer at a level of 20 mmHg [3] with the formation of necrosis of 12% of hepatocytes. Liver failure is increasing. Intra-abdominal hypertension impairs blood circulation in the abdominal wall and negatively affects the healing of laparotomic wounds [3,12] and can cause ischemia and necrosis of the peritoneum [12].

The causes of renal failure in ACS in response to an increase in IAP are: compression of the renal veins and renal parenchyma [5], increased production of the hormone aldosterone and renin, and a decrease in the glomerular filtration rate [5]. A 2-fold decrease in urine output is already noted at a level of intra-abdominal hypertension of more than 10-15 mmHg within 24 hours, complete anuria develops at a level of intra-abdominal pressure exceeding 30 mmHg [5]. It is indicated that after decompression of the abdominal cavity, renal function, as a rule, is restored after a certain period of time [5].

Thus, the problem of intra-abdominal hypertension is of interest to surgeons, who have identified a relationship between the tension of the anterior peritoneal wall and the degree of respiratory failure. There have been many studies devoted to the negative effect of IAH on central and intracardiac hemodynamics. It was obvious that an increase in IAH leads to an increase in mortality in patients.

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