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FEATURES OF EARLY ENTERAL NUTRITION IN PATIENTS WITH INTRAABDOMINAL HYPERTENSION SYNDROME IN SEVERE ACUTE PANCREATITIS

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ABSTRACT

The problem of treating severe acute pancreatitis is currently not solved. It was suggested that a large number of treatments. But unfortunately, that it seems to be working in experimental and initial clinical trials have not proved their effectiveness in the major randomized trials and cannot be recommended for widespread use in terms of evidence-based medicine. This statement relates to antimicrobial prophylaxis in the enzymatic step using antiproteaznyh, 5-fluorouracil, somatostatin drugs, anti-inflammatory drugs. Volumetric fluid therapy, sufficient early nutritional support is considered effective and recommended for use in severe acute pancreatitis. This article describes the importance of using early enteral nutrition in patients with this pathology.

KEYWORDS: *Enteral Nutrition, Pancreatitis, Intraabdominal Hypertension Syndrome, Intra-Abdominal Pressure.*

INTRODUCTION

The starting point of the development of intraabdominal hypertension in acute pancreatitis is considered to be the production of proinflammatory cytokines, an increase in capillary permeability and sequestration of fluid into the intra- and retroperitoneal space. Progressive reduction of the free volume of the abdominal cavity and an increase in intra-abdominal pressure aggravates pancreatic hypoperfusion and accelerates necrobiotic processes [1].

Of great importance in the pathogenesis of intraabdominal hypertension in acute pancreatitis is paralytic intestinal obstruction, which, on the one hand, increases intra—abdominal pressure, and on the other hand, progresses under the influence of intraabdominal hypertension -associated mesenteric ischemia, increases the permeability of the gastrointestinal mucosa, translocation of enteral microflora, infection of necrotic pancreatic tissues and retroperitoneal space [2].

Iatrogenic causes may also contribute to the development of intraabdominal hypertension in patients with acute pancreatitis: rigidity of the anterior abdominal wall against the background of insufficient analgesia, as well as massive infusion of crystalloids at an early stage of the disease [3].

The elevated position of the diaphragm in intraabdominal hypertension leads to a decrease in the elasticity of the chest, an increase in intra-thoracic pressure and a decrease in respiratory volume. Ventilation-perfusion discrepancy is manifested by a violation of oxygenation and the development of respiratory failure requiring mechanical ventilation [4].

An increase in pressure on the inferior vena cava, as well as mechanical compression of the heart and the main vessels of the chest, create hypertension in the small circle of blood circulation, making it difficult for venous return to the right parts of the heart. All this contributes to a decrease in cardiac output, a decrease in blood pressure, a violation of organ perfusion, the occurrence of arrhythmias, especially in patients initially suffering from coronary heart disease [5].

Changes in central hemodynamics, as well as direct compression of the renal parenchyma with reduction of renal blood flow, reduce perfusion pressure and filtration gradient, contributing to the development of oliguria. It is proved that an increase in intraabdominal pressure of more than 20 mm Hg is associated with oliguria and reduction of cardiac output. Violation of arterial hepatic and portal blood flow leads to ischemic damage to hepatocytes, mitochondrial dysfunction, impaired glucose metabolism, decreased lactate clearance [6].

Thus, intraabdominal hypertension makes a significant contribution to the development of systemic (organ dysfunction) and local (purulent-necrotic) complications of acute pancreatitis, which allows many researchers in recent years to consider it as one of the markers of severe disease.

Diagnostics. According to the recommendations of the World Society for the Study of Abdominal Compartment Syndrome, in critically severe patients with risk factors for intraabdominal hypertension, intra-abdominal pressure should be measured every 4-6 hours. The most common measurement method remains intravesicular, since the pressure in the lumen of the bladder exactly corresponds to intraabdominal, as well as due to technical simplicity and non-invasiveness. The resolution of the World Society for the Study of Abdominal Compartment Syndrome requires expressing the level of intra-abdominal pressure in mmHg and strictly adhere to the measurement algorithm to exclude false high results. In particular, the assessment of intra-abdominal pressure should be carried out in a horizontal position of the patient in the absence of tension of the muscles of the anterior abdominal wall for 30-60 seconds after the introduction of 25 ml of sterile isotonic sodium chloride solution into the bladder. To date, there are no recommendations for routine monitoring of intra-abdominal pressure in acute pancreatitis, however, the results of a recent prospective study indicate in favor of monitoring intra-abdominal pressure in patients with severe acute pancreatitis with persistent organ failure and (or) systemic inflammatory reaction syndrome [7].

Predictors of abdominal compartment syndrome are signs of compression of the inferior vena cava and kidneys according to the results of multispiral computed tomography; an increase in the ratio of anteroposterior to transverse abdominal size, the formation of bilateral inguinal hernias. There is also information about the correlation of the serum level of procalcitonin with the value

of intra-abdominal pressure in acute pancreatitis, which, according to experts, can be used in determining therapeutic tactics [8].

Prevention and conservative treatment. Priority in the prevention and correction of intraabdominal hypertension in acute pancreatitis is given to non-surgical techniques. Despite the importance of replenishing the volume of circulating blood and optimizing pancreatic perfusion, it was found that hyperhydration lasting more than 48 hours increases the risk of abdominal compartment syndrome and increases mortality. An accurate marker of visceral perfusion and a possible endpoint of infusion rehydration is the index of abdominal perfusion pressure, the achievement of which at the level of 60 mm Hg correlates with greater survival in intraabdominal hypertension and compartment syndrome. As a laboratory guideline for the adequacy of infusion therapy in recent years, it is recommended to use the lactate level [9].

Great importance is given to the choice of the nature of the infusion medium: it is assumed that the total use of crystalloids contributes to the development of intraabdominal hypertension. Colloidal solutions, in particular, hydroxyethyl starch, stay longer in the vascular bed and allow faster replenishment of the volume of circulating blood compared to crystalloids. Randomized studies in patients with severe acute pancreatitis demonstrate the advantages of combined administration of isotonic salt solutions with hydroxyethyl starches in the prevention of abdominal compartment syndrome, as well as in reducing the need for ventilation compared with the total use of crystalloids.

Pathogenetic correction of intraabdominal hypertension in acute pancreatitis includes 3 main directions: reducing the "volume of hollow organs" associated with the development of paralytic intestinal obstruction; reducing the amount of pathological contents of the abdominal cavity and retroperitoneal space (necrosis and exudate); reducing the rigidity of the anterior abdominal wall. Adequate sedation and analgesia, including neuromuscular blockade, are necessary to exclude excitation and non-synchronous respiratory movements that increase the tone of the anterior abdominal wall. Enteral decompression, as well as the appointment of prokinetic drugs, contribute to the elimination of paralytic intestinal obstruction and gastrostasis. The possibilities of early enteral nutrition in intraabdominal hypertension are associated with the restoration of perfusion and intestinal motility. The results of a prospective clinical study demonstrate that in patients with severe acute pancreatitis, early enteral nutrition prevents the development of intraabdominal hypertension, improves clinical outcomes of the disease, but has no significant effect on mortality. A number of randomized studies have shown the possibilities of prolonged veno-venous hemofiltration in the prevention and elimination of intraabdominal hypertension against the background of severe acute pancreatitis through the elimination of cytokines (IL-6 and TNF-α) and negative hydrobalance[10].

Surgical treatment. In the case of an increase in intraabdominal hypertension against the background of conservative measures, percutaneous drainage of acute fluid accumulations is justified. Z.X. Sun et al. in a randomized study, the effectiveness of abdominal drainage was compared with conservative measures. It was found that the volume of fluid evacuated through drainage correlated with the level of intra-abdominal pressure, the duration of hospitalization and the dynamics of scores on the APACHE-II scale.Despite the achieved reduction in the intensity of pain syndrome, the duration of inpatient treatment, significant differences in the level of postoperative mortality in clinical groups have not been established. In the presence of extensive fluid accumulations, percutaneous drainage helps to reduce intraabdominal hypertension, but the

possibility of further development of organ dysfunction and compartment syndrome remains unpredictable [7].

To date, there is no consensus on the optimal duration of surgical decompression. It has been shown that in patients with acute pancreatitis with intra-abdominal pressure above 25 mm Hg. during the first 14 days of intensive therapy, the mortality rate is more than 50%. With the development of abdominal compartment syndrome on the background of severe acute pancreatitis, surgical decompression improves the functional state of the kidneys, lungs. According to P. Mentula et al., early decompression contributes to a significant reduction in mortality (from 46 to 18%), as well as a decrease in respiratory and renal insufficiency in 54% of patients. The authors recommend surgical decompression in patients with intra-abdominal pressure above 25 mmHg during the first 4 days after the onset of the disease. Similar results were obtained in an experimental model of severe acute pancreatitis in laboratory pigs: decompression upon reaching the intra-abdominal pressure level of 25 mm Hg was associated with an improvement in systemic hemodynamics and the morphological state of the pancreas, which, in general, contributed to a decrease in mortality [11].

The most common method of surgical decompression in abdominal compartment syndrome is median laparotomy. At the same time, all layers of tissues (skin, aponeurosis, peritoneum) from the xiphoid process of the sternum to the pubic articulation are dissected in layers by vertical incision. Such access makes it possible to monitor the state of the intestine and diagnose possible ischemic injuries in a timely manner. Despite a significant reduction in the frequency of complications such as intestinal fistulas, the preservation of the defect of the anterior abdominal wall in the future often requires reconstructive surgery and even skin grafting.

As an alternative, a transverse approach can be used below the costal arch on the right and left, which facilitates necrectomy of meso- and paracolic tissue in case of infection of necrosis. Among other things, this access has advantages in the technique of primary wound closure.

Another option for surgical correction of intraabdominal hypertension is subcutaneous fasciotomy, performed from small horizontal skin incisions. The remaining intact peritoneum provides a lower probability of developing purulent-necrotic complications, including the formation of intestinal fistulas. According to A. Leppäniemi et al., the effectiveness of subcutaneous fasciotomy of the white line of the abdomen was comparable to decompression laparotomy and was accompanied by mortality at the level of 40% in a group of 10 patients [11]. The achieved economic effect was due to a decrease in the need for follow-up interventions and patient care. Closed monitoring of intra-abdominal pressure must be provided without fail in order to confirm the adequacy of decompression after the intervention. This is associated with the risk of recurrence of intraabdominal hypertension, and some experts do not recommend fasciotomy when the intra-abdominal pressure is more than 35 mm Hg [12].

To date, there is no convincing evidence of the advantages of one or another method of surgical treatment of intraabdominal hypertension in patients with severe acute pancreatitis. It is not clear, in particular, how effectively minimally invasive methods (percutaneous drainage) provide decompression and prevention of the subsequent need for laparotomy.

CONCLUSIONS

Thus, the problem of the development of intraabdominal hypertension in acute pancreatitis has acquired exceptional importance in recent years, which, apparently, is associated with a change

in tactical approaches to the treatment of the disease and, in particular, with the rejection of routine open laparotomy interventions in the pre-infectious phase of the disease. Despite the existence of a consensus on the pathophysiological significance of increased intra-abdominal pressure and the need for its monitoring, the issues of treatment and prevention of intraabdominal hypertension in acute pancreatitis remain controversial.

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