ASSESSMENT TECHNIQUES AND SPECIFIC CARE AT PATIENTS WITH PERITONITIS

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Abstract

Acute diffuse peritonitis is a major surgical emergency, which, in the absence of adequate treatment, evolves to death within 5-7 days in common forms or within 2-3 days in severe forms. The patient is urgently admitted to the intensive care unit of the general surgery department. The study group included 42 patients, of which 27 with acute peritonitis due to appendiceal perforation, 6 patients with acute peritonitis due to duodenal ulcer perforation and 9 patients with abscessed appendiceal plastron. Surgical treatment was associated with antibiotic treatment: pre-, intra- and postoperative, as well as with general supportive measures associated with specific appropriate nursing care, so that the evolution was favorable and with as few complications as possible. The interventions performed were: appendectomy with peritoneal lavage and multiple drainage in 27 cases; ulcer suture with epiploonoplasty, peritoneal lavage and multiple drainage in 6 cases; peritoneal cavity lavage and multiple drainage in 9 cases with abscessed appendicular plastron. The major objective of the study was to provide data on the contribution of nursing in the treatment of patients with acute peritonitis, as well as its role in the therapeutic scheme in order to achieve a favorable postoperative evolution and a superior quality of life.

Key words: peritonitis, sepsis, lavage, drainage, nursing interventions

Peritonitis is the inflammation of the peritoneal serosa, a membrane that covers the abdominal organs and the inner surface of the abdominal walls. The peritoneal space is a "virtual" space located between the visceral peritoneum (the one that covers the abdominal organs, constituting their serosa) and the parietal peritoneum (which lines the abdominal walls on the inside).

Normally, in the peritoneal space, there is a minimal amount of fluid, which allows the two peritoneal membranes to slide freely over each other. In pathological conditions, a larger amount of fluid accumulates in the peritoneal space or various fluids are accidentally discharged (bile, gastric juice, intestinal fluid, pus, etc.).

Depending on the extension of the inflammatory process, peritonitis is divided into localized peritonitis (the infection is limited to an area of the abdomen) and generalized peritonitis. Untreated, localized peritonitis leads to the formation of an abscess, and in the generalized form, microbes can reach the blood (septicemia), causing septic shock. If no intervention is made, the patient dies. This is how the particular seriousness of this disease is understood. The moment of surgery should not be postponed, because the prognosis is all the better, the more promptly the intervention is carried out. The condition is more severe in the elderly, with a more precarious defense of the body. In them, the diagnosis is more difficult, given the more blurred clinical picture.[3]

Surgical treatment must be framed by antibiotic treatment: pre-, intra- and postoperative, as well as general supportive measures. The intervention aims to evacuate the infected fluid from the peritoneal cavity, remove the source of contamination (if necessary, suture the perforation, remove the organ - appendix), lavage of the cavity with large amounts of antiseptic solution and multiple peritoneal drainage with drain tubes. The pathophysiological disorders in the evolution of acute diffuse peritonitis have been systematized in 3 phases or stages of peritoneal irritation, declared peritonitis and neglected peritonitis.

Peritenal irritation is the initial phase of diffuse acute peritonitis, which usually spreads during the first 6 hours after onset. It is considered reversible. In chemical peritonitis, as a result of irritation of the peritoneal interoreceptors by the intraperitoneal digestive effusion from a perforation of an irritating organ (gastroduodenal content, bile, pancreatic enzymes, colonic content), an abundant exudate rich in fibrin and antibodies appears, which tend to limit the inflammatory process. The brutal excitation of the peritoneal interoreceptors explains the intensity of the pain and the appearance of inflammation, simultaneously with the initiation of reflex mechanisms: contracture of the abdominal muscles and paralysis of the digestive muscles. The greater omentum, the true "gendarme" of the peritoneal cavity, is attracted by chemotaxis to the site of maximum irritation and adheres to the perforation. If the perforation is covered and completely sealed, the peritonitis remains in this phase and usually progresses to remission, a situation in which emergency surgery is no longer necessary. The classic example is that of a covered ulcerative perforation.[4] Manifest peritonitis or purulent peritonitis continues the peritoneal irritation phase. The presence and multiplication of pathogenic microbial agents in the peritoneal cavity profoundly disrupts the functions of the peritoneum through irritation of the peritoneal interoreceptors, progressive paralysis of the muscles of the entire digestive tract and alteration of intestinal digestion and absorption. Clinically manifest peritonitis is characterized by the appearance of purulent peritonitis with false membranes, concomitantly with the onset of hypovolemic and toxicoseptic shock.[3]

The paralysis of the muscles of the digestive tract, which occurred in the previous phase, paralytic dilatation of the intestinal loops produces a large amount of fluid and gases. The sequestration of water and electrolytes in the lumen and walls of the digestive tract and in the loops causes massive fluid retention in the peritoneal cavity (Randal space III), to which are added losses through sweating, vomiting, perspiration (hypovolemic shock), causing a decrease in circulating blood volume and severe hydroelectrolytic imbalances, with the appearance of extracellular dehydration associated with intracellular dehydration.

The modification of hydrostatic pressure and colloidosmotic pressure (protein loss in space III) profoundly affects local and systemic transcapillary exchanges. Electrolyte disorders are secondary to the loss of sodium, potassium, chlorine and magnesium ions. Hyposodemia, hypokalemia, hypochloremia (predominant loss through vomiting and sweating) and hypomagnesemia (responsible for nervous phenomena) are established. Cell membrane exchanges are affected, with the replacement of extracellular Na+ with intracellular K+ (ATPase-K+ dependent Na+ and K+ pump) and the development of metabolic acidosis. Non-protein nitrogen increases (hypovolemia but renal hypoirrigation, hypercatabolism) and hematocrit due to dehydration. [4] Transcapillary and transmembrane exchange disorders are exacerbated and aggravated by the over-addition of systemic toxemia (through the resorption of microbial exotoxins) which will initiate the cascade of the systemic inflammatory response, with the appearance of toxicoseptic shock.

Neglected peritonitis is the advanced evolutionary phase of diffuse peritonitis, which follows untreated declared peritonitis and usually appears after 36-48 hours from onset. It is marked by the installation of paralytic occlusion and on a systemic level, with the appearance of multiple organ failure (MOF). Practically all organs are affected to a variable degree and in various associations, but in the final stage irreversible multi-organ failure sets in. The most frequently encountered components of MOF are acute respiratory failure, cardio-respiratory failure, and acute renal failure.[3]

Respiratory failure is due to both diaphragmatic immobility through phrenic contracture (irritation of the diaphragmatic peritoneum) and abdominal distension (paralytic intestinal occlusion), as well as hydroelectrolytic disorders.

Cardio-circulatory failure is the consequence of hypovolemia, which causes hemodynamic changes, with a progressive reduction in venous return to the right heart, until the appearance of cardiorespiratory arrest. It is caused, at first, by tissue hypoperfusion.

Subsequently, tissue ischemia characteristic of shock sets in. Massive fluid losses with hemoconcentration and stasis in the microcirculation sector favor the appearance of microthromboses, an aggravating factor by

accentuating peripheral hypoxia. In addition, the opening of arteriovenous shunts is added, which further reduces the amount of blood reaching the tissues.

Rheological disorders in the microcirculation culminate in the appearance of disseminated intravascular coagulation (DIC syndrome).

Acute renal failure is the result of renal hypoperfusion and is manifested by the appearance of oliguria and then anuria, concomitantly with an increase in serum urea and creatinine. Bacterial toxins cause damage to the nephrons, with the possible appearance of organic renal failure.

Adrenal insufficiency is also the consequence of hypoperfusion of the adrenal medulla and adrenal cortex; thus, a massive discharge of adrenaline and noradrenaline into the circulation occurs, which causes strong vasoconstriction, with increased hypoxia and exacerbation of metabolic tissue changes through a "vicious circle" mechanism. The intensity and grouping of clinical manifestations may suggest the phase of evolution of the peritonitis process. In the peritoneal irritation phase, the patient presents with pain and abdominal contracture. The pain is more intense at the level of the lesion and may radiate to the shoulder, interscapulo-vertebral or dorsal region. In acute gastroduodenal perforations it may become shock-like, due to the brutal irritation of the peritoneal interoceptors (vagal shock). The abdominal contracture may be less marked at the onset, but becomes pronounced within a few hours. The patient also presents with reflex vomiting, initially alimentary, which may then be bilious. Thirst may occur as a result of fluid loss, hiccups and, after a few hours, paralytic ileus. The patient presents with positive Blumberg, Dieulafoy, Mandel and Grassman signs. He may also present with oliguria, fever, leukocytosis (over 12000-15000/mm3), with neutrophilia. The phase of declared peritonitis (peritoneal shock phase) usually sets in after 6 hours from the onset. In this phase, the pain decreases in intensity and meteorism, poraceous vomiting appear (due to paralytic ileus). The signs of Blumberg, Dieulafoy and Mandel decrease in intensity, and the Grassman sign is more intense.[2]

The muscular contraction gradually attenuates being replaced by the progressive abdominal distension of the paralytic ileus. Intestinal transit is completely suppressed, both for feces and for gases. Important hydroelectrolytic disorders are established with a decrease in serum Na^+, K^+, Mg^{2+} and Cl^-. Hemoconcentration occurs, with an increase in hematocrit, metabolic acidosis and extrarenal azotemia. The patient presents with fever and hyperleukocytosis. In the neglected peritonitis phase, the patient presents a Hippocratic facies and is restless, agitated, but conscious. The abdomen is relaxed, without muscle contracture and without significant pain. The biological state is profoundly affected by multiple organ insufficiency. The patient presents with fecal vomiting (a sign of severe prognosis), scleral subicterus due to liver insufficiency, anuria, tachypnea, cyanosis (due to bloating and diaphragm immobility), dry tongue (a sign of intense dehydration).

To evaluate the prognosis, the APACHE II severity prediction score is calculated, based on the comparative evaluation of biochemical parameters determined upon admission and during evolution, and the Glasgow score.[1] Paraclinical explorations relevant in peritonitis are radiological and ultrasound.

Plain abdominal radiography is extremely useful in the diagnosis of perforations of the cavitary viscera. In peritonitis due to gastroduodenal or colic perforations, pneumoperitoneum is evident, which, in orthostatism, appears as a thin air layer that appears between the liver and diaphragm, and if it is more abundant, also between the spleen and diaphragm.

Abdominal ultrasound is indicated for differential diagnosis; in peritonitis, it can reveal the presence of exudate in the abdominal cavity and/or immobility of intestinal loops. CT and MRI reveal the existence of an abdominal pathology.[5]

Acute diffuse peritonitis is a major surgical emergency, which, in the absence of treatment, evolves to death in 5-7 days (common forms) or in 2-3 days in severe forms. The patient is urgently admitted to the intensive care unit of the surgery department. Treatment must be early (from the onset) and complex, aiming at the following

therapeutic objectives: suppressing the source of peritoneal contamination, combating infection and toxicoseptic resorption through antibiotic therapy, toileting and draining the peritoneal cavity, correcting volemic, hydroelectrolytic and acid-base disorders through perioperative intensive therapy, preventing or reducing possible complications. The major therapeutic objectives (elimination of the source of contamination and peritoneal drainage) are met by surgical treatment, which is indicated urgently and as early as possible from the onset of diffuse peritonitis. In the postoperative period, sustained treatment of the patient is imperative with the use of vasopressors and cardiac stimulants as needed, in parallel with a sufficient fluid intake to maintain diuresis at a level of at least 2000 ml/ 24 hours and with the resumption of intestinal transit as soon as possible postoperatively.[5]

Severe sepsis cannot always be controlled only through a single intervention, requiring continuous peritoneal lavage and in certain situations scheduled interventions to prevent the onset of multiple organ failure syndrome (MSOD – MSOF). The study group consists of patients with peritonitis hospitalized between January 2022 and January 2025 in the Surgery Department of Turceni Hospital.

The study is retrospective and was conducted based on the analysis of observation sheets, diagnostic and treatment protocols, admission or transfer registers, but also on clinical observation during internships carried out in this department.

Patients who presented to the emergency room with peritonitis and life-threatening cardiovascular pathology or with advanced hepato-renal pathology who were transferred to hospitals with a higher level of competence and where there was the possibility of permanent pre- and postoperative monitoring in intensive care units and the possibility of performing appropriate treatment in order to prevent multiple organ failure syndrome were not included in the clinical study.

The study group included 42 patients, of whom 27 (64.28%) had acute peritonitis due to appendiceal perforation, 6 (14.28%) had acute peritonitis due to duodenal ulcer perforation and 9 patients (21.42%) had abscessed appendiceal plastron. 29 patients were male (69.04%) and 13 patients were female (30.95%). The major objective of the study was to provide data on the contribution of surgical nursing in the treatment of patients with acute peritonitis, as well as its role in the therapeutic scheme in order to achieve a favorable postoperative evolution and a superior quality of life.

The specific short-term objectives were: the patient to benefit from emergency care to favor the evolution and minimize the risk of complications; the patient to be volemic and hydroelectrolytic balanced; to have physiological eliminations; to have normal and efficient breathing and circulation; the patient to have physiological and restful sleep.

The specific medium-term objectives were: the patient to mobilize and adopt comfortable positions; to have a satisfactory evolution, without complications; the patient to acquire the knowledge necessary to regain health.

Specific long-term objectives: the patient to present a state of physical well-being; the patient to reintegrate socially as quickly as possible. The data source was: observation sheets of patients with Following the evaluation of the nursing process in the study group, a favorable evolution was found without occlusive phenomena in the postoperative period, without cardiovascular or metabolic decompensations and alterations in the postoperative biological status.

The objectives of the preoperative treatment were: restoration of volemia, antibiotic therapy with cephalosporins in association with Metronidazole, nasogastric suction tube, monitoring of vital functions and anti-pain medication.

The interventions performed were: appendectomy with peritoneal lavage and multiple drainage in 27 patients (64.28%); ulcer suture with epiploonoplasty, peritoneal lavage and multiple drainage in 6 patients (14.28%); lavage of the peritoneal cavity and multiple drainage in 9 patients (21.42%) with abscessed appendicular plastron.

The intra-abdominal access method was represented by median laparotomy in 31 cases (73.80%) and by Jalaguier-type incision in 11 cases (26.19%). The resumption of intestinal transit was achieved in the first 48 hours from the moment of surgery in 25 cases (50.52%) and in 17 cases (40.47%) at 72 hours postoperatively by stimulating transit with Myostin and evacuating enemas.

Postoperative complications were represented by the presence of parietal suppurations in 15 patients (35.71%) with a simple evolution that did not require the extension of the hospitalization period by more than 4 - 5 days under targeted antibiotic therapy according to the antibiograms performed. The hospitalization interval of the patients was between 8 and 10 days in 19 cases (45.23%) and between 6 and 7 days in 23 cases (54.76%). No postoperative eventrations or other postoperative incisional defects were recorded after the surgical interventions performed.peritonitis; diagnostic and treatment protocols; registers of admissions and interhospital transfers.

CONCLUSIONS

Acute peritonitis represents the inflammatory reaction of the peritoneal serosa, diffuse or localized, of infectious or not origin, produced by varied and complex mechanisms. The diagnosis in acute peritonitis is a complex diagnosis, carried out according to an algorithm, which involves the establishment in successive stages of a positive, differential and etiological diagnosis.
The positive diagnosis is based on the anamnesis, the objective examination and the paraclinical investigations within which the abdominal radiography on empty is extremely useful in the diagnosis of perforations of the cavitary viscera and the abdominal ultrasound is indicated for the differential diagnosis, by highlighting the exudate in the abdominal cavity and/or the immobility of the intestinal loops.
Treatment must be early from the onset and complex, aiming at the following therapeutic objectives: suppression of the source of peritoneal contamination, combating infection and toxicoseptic resorption through antibiotic therapy, toileting and drainage of the peritoneal cavity, correction of volemic, hydroelectrolytic and acid-base disorders through perioperative intensive therapy, prevention or reduction of possible complications.
The surgical intervention is performed urgently, with general anesthesia, after several hours of preoperative preparation, the duration of which depends on the interval from the onset and, especially, on the biological terrain, but without exceeding 6 hours.
Surgical treatment must be strictly individualized for each case, depending on the nature of the peritonitis, the time elapsed since the onset, the patient's general condition, age and the existence of concomitant diseases. It must be doubled by pre-, intra- and postoperative intensive therapy, which corrects the serious imbalances of peritonitis.
The evolution and prognosis of the patient with peritonitis are determined by early surgical treatment and sustained hemodynamic rebalancing.

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