37. FIGHTING THE MULTI ORGAN FAILURE

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Introduction: Multiple organ failure is the commonest cause of death in the intensive care unit setting. There are numerous precipitating factors including sepsis, trauma and pancreatitis. The resulting tissue hypoxia, exaggerated inflammatory response and generation of free oxygen radicals leads to tissue damage and organ dysfunction. No definitive treatment exists despite considerable efforts to find a 'magic bullet'. Management still revolves around support of organ function and prevention of iatrogenic complications until recovery occurs. An increasing emphasis is being placed on prevention of organ dysfunction, including maintenance of tissue oxygenation, nutrition and infection control. Multiple organ failure (MOF) is the commonest cause of death in the intensive care unit (ICU). A clinical assessment of a high likelihood of irreversible organ failure, particularly when multiple organs are involved, is the usual factor prompting a decision to withdraw treatment or not to add further therapy. Sepsis is one precipitating factor for MOF; numerous other causes of tissue damage are well recognized, e.g. trauma, burns and pancreatitis. No definitive treatment exists and controversy surrounds many aspects of the management of MOF. Problems include (i) a shortage of major multi-center, controlled studies in a well-defined patient population (other than immunotherapy trials which are often of flaweddesign), (ii) an inclination to use unproved interventions,(iii) over-extrapolation of data from laboratory studies,(iv) an often uncritical acceptance of simplified, schematic representations of inflammatory mechanisms, (v) variable disease syndrome definitions and (vi) diagnostic imprecision. The above contribute to the current lack of hardened-fast rules regarding patient management; instead, there are a number of generally accepted guidelines which still provide considerable scope for treatment variability. Examples of current grey areas include selective gut decontamination, extracorporeal respiratory support and prophylaxis against stress-ulcer-related bleeding. There is also the widespread, though as yet unproven and unlicensed, use of nitric oxide inhalation in acute lung injury, and the quest for a single 'magic bullet' to ameliorate the generalized, exaggerated inflammatory response associated with severe sepsis. In the case of the strongly promoted concept of 'supranormalizing' hemodynamic parameters in the critically ill patient, whereby elevated values of cardiac output, oxygen delivery and oxygen consumption were striven for, it was several years before this approach was shown to be ineffective. Nevertheless, and despite the above caveats, there has been progress in several areas. A better, though still incomplete, insight is being gained into the pathophysiological mechanisms underlying the exaggerated inflammatory response that frequently underlies MOF. There is a greater appreciation of the need to prevent organ dysfunction by optimizing the circulation and avoidance or rapid correction of tissue hypoxia in high-risk patients. There is are cognition of the importance of standard definitions, for example sepsis, the systemic inflammatory response syndrome (SIRS), the multiple organ dysfunction syndrome (MODS) , the acute respiratory distress syndrome (ARDS) and acute lung injury. There is also a recognized need to improve the description of organ dysfunction. In addition, general advances and the increasing availability of intensive care, superior 'whole body' organsupport, appropriate infection control, nutrition and pressure area care, and avoidance of iatrogenic pulmonary barotrauma, have all contributed to improvements in outcome.

Objective: To determine whether translocation of bacteria or endotoxin occurred into the thoracic duct in patients with multiple organ failure (MOF) is take active role in MOF.

Methods: 1. Meta-analysis of 156 patients from retrospective – preview date base of patients in MOF. 2. The thoracic duct was drained for 5 days in patients with MOF caused either by generalized fecal peritonitis (n = 4) or by an event without clinical and microbiologic evidence of infection (n = 4). Patients without MOF who were undergoing a transthoracic esophageal resection served as controls. In lymph and blood, concentrations of endotoxin, proinflammatory cytokines, and anti-inflammatory cytokines were measured.

Experimental data: Description – in heart of Mousses, was examined the function of heart (in vitro), in 3 state: 1. Normal without intervention. (Control group). 2. With lymph of MOF state. 3. With MOF state + thoracic duct ligation.

Conclusion: This meta-analysis study provides evidence that translocation (especially of endotoxin) occurs into the thoracic duct. These data do support the concept that the thoracic duct is a major route of bacterial translocation in patients with MOF

38. NEGATIVE PRESSURE THERAPY IN THE TREATEMENT OF SUPPURATED EVENTRATED LAPAROTOMIC WOUND

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Introduction: The laparotomic suppurated eventrated wound is a postoperative complication caused by contamination, suppuration and necrosis of the abdominal wall anatomical layers (subcutaneous fat, ventral aponeurosis, peritoneum) with eventration of abdominal organs. The use of negative pressure therapy in the treatment of laparotomic suppurated eventrated wound is described worldwide in the specialty literature, but its efficiency depending on the etiology of the intraabdominal infection remains insufficiently studied.

Purpose and Objectives: Reporting the results of the treatement with negative pressure of the patients with laparotomic suppurated eventrated wounds.

Materials and Methods: From October 2012 until March 2014, negative pressure therapy was used in the treatement of laparotomic suppurated eventrated wounds in 22 patients with the mean age of 64.2 and sex ratio M:F being 15:7. The study included patients with laparotomic suppurated eventrated wounds due to diffuse peritonitis in the following nosologies: inguinal hernia with small bowel necrosis (1), postoperative ventral hernia (1), gastric adenocarcinoma with perforation (1), gangrenous cholecystic perforation (1), closed abdominal trauma with bowel (1) and small intestine injury (1), fistular Crohn's disease (1), gangrenous perforative appendicitis (3), colon ischemia and necrosis with perforation (2), nonspecific ulcerative colitis with intestinal obstruction (1), colon diverticulum perforation (2), benign tumor of the colon with mechanic obstruction (1), colon adenocarcinoma with mechanic obstruction (3), colon adenocarcinoma with perforation (3).

Results: The negative pressure therapy set at 75-105 mmHg was applied after necrectomy, with a mean duration of the sessions of 48-72 hours. The number of sessions was determined by the type of intraabdominal infection. Wound closure criteria were: the presence of mature granulation tissue, the type of inflammatory-regeneratory cytological imprints and the decrease of the amounts of wound microflora from 10^{6-7} to 10^{2-3}. The treatment was carried out in two stages: the first stage - negative prèssure therapy with suturing of the ventral aponeurosis, the second stage - continuing negative pressure therapy with complete closure of the laparotomic wound. Definitive closure of the abdominal wall was possible in 19 patients. 3 patients died, the mortality rate constituting 13.6%.

Conclusions: The use of negative pressure therapy in the treatment of the laparotomic suppurated eventrated wounds allows to: eliminate the septic source, decrease the frequency of dressings changes- one at 48-72 hours with a fascial closure rate of 86.4%.

Keywords: negative pressure therapy, laparotomic suppurated eventrated wounds, fascial closure

39. HAEMOSTASIS FOR VARICEAL BLEEDING IN PATIENTS WITH LIVER CIRRHOSIS

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Introduction: Variceal bleeding is a severe complication of cirrhosis leading to significant morbidity and mortality. Ruptured esophageal varices cause approximately 70% of all upper