DEVELOPMENT AND EVALUATION OF AN ALGORITHM FOR DIFFERENTIAL DIAGNOSIS OF VARIANTS OF ACUTE RESPIRATORY DISTRESS SYNDROME

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Relevance. The main mechanism of ARDS pathogenesis is damage to the vascular endothelium of the pulmonary hemomicrocirculation and alveolar epithelium by various endogenous and/or exogenous damaging factors, which leads to the development of non-cardiogenic pulmonary edema, severe violations of gas exchange and biomechanics, followed by a cascade of subcellular, cellular and organ hypoxic damage.
The role of various biologically active substances, toxic products, medicines, microbes, viruses and their waste products in the development and outcomes of ARDS is discussed.
Timely and correct diagnosis of ARDS is a complex clinical problem. Traditional diagnostic criteria to a greater extent characterize the late stages of ARDS development, whereas its early stage - acute lung injury (OPL) is not always diagnosed, which is the reason for the late onset and often inadequate treatment.
There are many etiological factors leading to the development of ARDS: various diseases, wounds, injuries, poisoning, surgical and diagnostic measures, etc. All these factors can be divided into direct (aspiration, pneumonia, blunt chest injury with lung contusion, inhalation of toxic substances, drowning, etc.) and indirect (sepsis, shock, severe combined trauma, hemotransfusion, some medications, artificial blood circulation, etc.).
The purpose of the study. To improve the results of treatment of patients with acute respiratory distress syndrome by substantiating, developing and implementing methods of differential diagnosis and differentiated use of respiratory, non-respiratory and pharmacological treatment methods.
Materials and methods of research. 60 patients, injured and injured (28 men, 32 women, aged 18 to 69 years) with ARDS, the causes of which were direct and indirect damaging factors, were prospectively examined.
Research results. The results of the study showed differences in clinical signs and dynamics of their development in patients, injured and injured with ARDS caused by direct and indirect damaging factors.
ARDS caused by direct damaging factors is characterized by the absence of a "light" gap, the rapid development and progression of acute respiratory failure, encephalopathy, wet wheezing of various sizes, and low effectiveness of mask ventilation.
ARDS caused by indirect damaging factors is characterized by the presence of a "light" interval from the moment of exposure to aggression factors to the manifestation of ODN (from several hours to 3 days), a "poor" auscultative picture, the development of abdominal hypertension, multiple organ failure, the effectiveness of mask ventilation at an early stage.
The results of the study revealed that in patients, injured and injured of group A, with the manifestation of ARDS caused by direct damaging factors, thoracopulmonary compliance was lower than $p<0.05$ (on average by 19.6%), compared with ARDS caused by indirect damaging factors (Table 4). With ARDS caused by direct damaging factors, the content of intravascular fluid in the lungs was higher $p<0.05$ (on average by 35.1%) than in ARDS caused by indirect damaging factors.

In patients, injured and injured of group A, at the stage of cessation of respiratory support, thoracopulmonary compliance was lower than $p<0.05$ (on average by 14.8%) in ARDS caused by direct damaging factors, compared with ARDS caused by indirect damaging factors. At this stage of the study, all patients in group A the content of extravascular fluid in the lungs was within the normal range.

It can be assumed that with the manifestation of ARDS caused by direct damaging factors, the violation of lung biomechanics to a greater extent.

It is caused by hyperhydration, and in the later stages by fibrosis. In the manifestation of ARDS caused by indirect damaging factors, the violation of lung biomechanics is largely due to the widespread collapse of the alveoli.

Conclusions. In acute respiratory distress syndrome caused by direct damaging factors, compared with the indirect mechanism of damage lung, a longer (2-4 times) corrective effect and an oxygenation index is provided by an escalating method of optimizing positive pressure at the end of exhalation and the use of surfactant BL, and the reception of "opening" of the lungs and ventilation in "pronpositions" cause a less lasting effect (2-3 times). A more significant increase in the oxygenation index in acute respiratory distress syndrome caused by direct damaging factors is provided by ventilation in "pronposition", endobronchial administration of perfluorane, surfactant BL (by 1.9-2.9 times), with an indirect mechanism of lung damage, a greater clinical effect is caused by a de-escalation method of optimizing positive pressure at the end of exhalation and the "opening" method lungs.