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

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Relevance. Acute respiratory distress syndrome (ARDS) is one of the main problems of intensive care, develops in patients with various diseases, injured and injured, is characterized by non-cardiogenic pulmonary edema, severe gas exchange disorders and high mortality, which justifies the need to find new ways to improve the diagnosis and treatment results of this disease.

In most European countries, an average of 13 to 30 cases of ARDS are registered per 100,000 population per year, in the USA from 45 to 75 cases, in Russia from 15,000 to 25,000 cases per year. In recent years, there has been an increase in the frequency of ARDS diagnosis, due to an increase in the number of man-made disasters, road injuries, terrorist activity, deterioration of the environmental situation, aging of the population, deterioration of the premorbid background, widespread and uncontrolled use of medicines, the use of aggressive and invasive diagnostic and treatment methods, and expansion of surgical activity.

Despite the development of medical technologies, over the past 20 years in the world there has been only a tendency to decrease mortality in ARDS, which remains very high - from 22% to 74%, while in 16% - 24% of patients it is not possible to correct critical hypoxemia in any way.

In the treatment of patients with ARDS, it is necessary to use the most modern.high-cost and expensive medical equipment, which causes high material costs (on average from 1.5 to 5 million rubles and from 80,000 to 3200,000 US dollars per patient, corresponding to 1sho).

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. In the experimental model of ARDS caused by a direct damaging factor - acidinpepsin aspiration, damage to the bronchial and alveolar epithelium prevailed, which led to accumulation of fluid in the interstitial, diapedetic
hemorrhages, edema of the interalveolar septa, rapid development of alveolar edema. The development of distelectases was noted. Thus, in the experimental model of ARDS caused by acidin-pepsin aspiration, changes were primarily localized in the epithelium of respiratory bronchioles and alveoli, followed by the development of interstitial and alveolar edema.

In the experimental model of ARDS caused by the influence of an indirect damaging factor - acute massive blood loss in combination with thrombosis of microcirculation vessels, morphological changes were characterized by the development of perivascular infiltration, endothelial damage and interstitial edema. In dilated capillaries, stasis and aggregation of erythrocytes were noted, impaired lymph drainage, with accumulation of fluid in the

interstitial and impaired patency of bronchioles. Edematous fluid and shaped blood elements were injected into the alveoli, distelectases and atelectases were detected. Thus, after acute massive blood loss in combination with thrombosis of microcirculation vessels, the primary morphological changes consisted in damage to the endothelium, aggregation of erythrocytes and stasis in the hemomicrocirculation of the lungs, the development of interstitial and alveolar edema, obstruction of bronchioles with widespread atelectasis.

Conclusions. 1. Morphological changes in the lungs of rats during acidin-pepsin aspiration are characterized by damage to the bronchial and alveolar epithelium, the development of atelectasis of dependent zones, pronounced interstitial and alveolar edema, and in acute massive blood loss in combination with thrombosis of the microcirculatory bed — damage to the endothelium of hemomicrocirculation vessels and pronounced diffuse atelectasis.
2. Acute respiratory distress syndrome caused by direct damaging factors, in contrast to acute respiratory distress syndrome caused by an indirect mechanism of lung damage, is characterized by: faster the development of acute respiratory failure, more pronounced pecardiogenic pulmonary edema with an extravascular lung fluid index of more than 15 ml/kg of body weight, less static thoracopulmonary compliance (less than 25 ml/em of water), a lower level of optimal positive pressure at the end of exhalation (on average 12 cm of water) and lower intraabdominal pressure (in an average of 15 cm of water), while the frontal X-ray and computed tomogram reveal asymmetric focal seals in the dorsal and basal zones of the lungs; while in acute respiratory distress syndrome caused by an indirect mechanism of lung damage, symmetrical, diffuse changes are revealed.