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Research Article

PECULIARITIES OF THE COURSE OF ASPIRATION SYNDROME IN NEWBORNS

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Dilfuza Narzikulovna Ibragimova
Trainee Assistant of the Department of Neonatology, Faculty of Postgraduate Education Samarkand State Medical Institute, Uzbekistan

Dilshoda Turdiqulovna Akramova
PhD, assistant to the department of neurology Tashkent Medical Academy, Uzbekistan

Olia Askarovna Sharipova
Doctor of Medical Sciences, Associate Professor of Pediatrics and Medical Genetics Department Samarkand State Medical Institute, Uzbekistan

ABSTRACT

Respiratory distress syndrome is a non-infectious pathological process in lungs developing in children in the first hours or days of life with acute respiratory failure caused by primary surfactant system failure, insufficiency of lung tissue, pathological processes taking place in lungs against the background of considerable suppression of vital functions of the body.

Respiratory distress syndrome is one of the main causes of morbidity and death of premature and premature babies.

KEYWORDS

Respiratory distress syndrome, preterm and premature infants.

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INTRODUCTION

Respiratory distress syndrome in newborns (RDS) is respiratory failure of varying severity, mainly in preterm infants in the first 2 days of life, due to immaturity of the lungs and primary surfactant deficiency. This pathology occurs in 1% of all live births and in 14% of newborns with a birth weight of less than 2500 g. Respiratory distress syndrome in newborns and its consequences cause 30-50% of neonatal deaths in the USA.

Natural surfactants have a faster clinical effect, but the final results are not different from those of artificial surfactants. It has been suggested that surfactant isolated from the lungs of calves and piglets may induce an immunological response to a foreign protein, but conclusive data have not been obtained. The most natural infant surfactant from amniotic fluid cannot be widely used, as treatment of one child requires the use of amniotic fluid from 10 caesarean sections. Immature lung tissue and surfactant deficiency play a major role in the development of respiratory distress syndrome in newborns.

Surfactant is a surfactant synthesised in the lungs. The synthesis of surfactant in the alveoli (the structural units of the lungs) begins at 20-24 weeks gestation through ethanolcholine methylation reactions. During this period the rate of synthesis is low. From week 34-36 the choline pathway begins to function and surfactant accumulates in large quantities. Symptoms of respiratory distress syndrome in preterm infants are detected from the first day of life, less often from the second. The Apgar score at birth can be anything. Intense dyspnea (up to 80-120 breaths per minute) with auxiliary muscles, sternum retraction, abdominal bulging on inhalation, and noisy, groaning, "grunting" exhalation are noted.

According to WHO, respiratory distress syndrome (RDS) is one of the leading causes of perinatal mortality. According to various authors, the mortality of children with BDM ranges from 35 to 75%. It occurs more often in premature babies, less often in preterm babies. K.A. Sotnikova points out that the term 'respiratory distress syndrome' is arbitrary. By her definition, "respiratory distress syndrome is a special clinical condition of the newborn, characterized by an early onset (in the first 2 days of life) and often a rapid increase against a background of

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significant suppression of vital body functions, symptoms of respiratory failure". One of the main causes of SIDS are pneumopathies (hyaline membranes, atelectasis, extensive pulmonary haemorrhage, oedema-haemorrhagic syndrome, congenital lung malformations, spontaneous pneumothorax) and intrauterine pneumonia. In the pathogenesis, irrespective of the cause of ADS, surfactant deficiency and obturation syndrome, hypoxia, metabolic acidosis, impaired metabolism, leading to changes in homeostasis and disorders of the central and autonomic nervous system, endocrine and cardiovascular systems, impaired ratio between ventilation and blood flow, immunosuppression. Of great importance is the disruption of the relationship between ventilation and blood flow in the lungs, and an increase in vascular permeability. Surfactant deficiency is now given leading importance in the pathogenesis. It is believed that the surfactant system matures fully by 35-36 weeks of intrauterine development. In a baby born before this term, the existing reserves of surfactant provide the beginning of breathing, its deficiency leads to alveolar collapse on exhalation, a sharp increase in the work of respiratory muscles. As a result of alveolar collapse, there is no continuous gas exchange in the lungs, leading to hypoxaemia and hypercapnia.

Diagnosis. The diagnosis of ADR is traditionally made on the basis of anamnesis, clinical and radiological symptoms. A triad of signs characteristic of SRS, appearing as early as the first hours of illness, is identified radiologically:

1. Diffuse decreased transparency of the pulmonary fields
2. Reticulogranular reticulation
3. Streaks of translucency in the lung root area (air bronchogram).

In severe cases, total darkening of the pulmonary fields is possible, and the borders of the heart may not be differentiated.

Recently, there have been methods in the medical arsenal to determine the degree of maturity of both the lung tissue itself and the surfactant system. The most common and informative test is the lecithin to sphingomyelin ratio in amniotic fluid, tracheal fluid or gastric aspirate at birth.

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CONCLUSIONS

Lung maturation and functional capacity are critical for survival. Based on the degree of prematurity, the lungs may be partially or completely immature and therefore unable to provide adequate respiratory function due to the absence or insufficient amount of surfactant produced. In such situations the newborn is indicated for surfactant replacement therapy.

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