

Characteristic of Neuronspecific Markers in Preterm Infants with Hypoxic-ischemic Encephalopathy

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Abstract

Problems of diagnosis of hypoxic-ischemic encephalopathy in preterm neonates remain relevant, serious and not fully studied and not fully refined directions.

The revealed regularities and peculiarities depending on the gestational age of newborns allows as to assert that applied immune-fermentative studies for the diagnosis of hypoxic-ischemic encephalopathy show that they need further improvement, and instrumental methods are not allowed to have an early and clear understanding about the prevalence of pathological process, predict outcome and to develop therapeutic tactics.

Keywords: Hypoxic-ischemic encephalopathy; Neuron specific markers; Periventricular leukomalacia; Central nervous system

Introduction

It is known that the newborn babies with low and very low body weight make up the most high-risk perinatal deaths and cases of disability group [1], among them the perinatal mortality rate is 16-20 times higher than the same indicator of babies with 2500 g birth weight [2].

The study of literature data [3] shows that for the mentioned contingent a significant and important role have the consequences of their perinatal brain lesions of central nervous system (CNS).

In the opinion of group of authors [4], it cannot be underestimated the role of CNS lesions in the children's pathology forming disabling system, the structure of disabilities make up about 50%, perinatal pathology makes in 70-80%.

According to the literature sources, on the one hand, the use of modern instrumental methods of perinatal intracranial lesions, such as neurosonography, magnetic resonance imaging, dopplerography and etc., are not always able to get the final information about verity assessment neonatal CNS diseases, on the other hand, there are not fully explored and understood a number other problems of their treatment by means of drugs and non-drugs.

Current State of Problem

It was indicated that clinical research of newborn babies with low and very low body weight in their first hours of life, even in the days and months, is not always allows to detect a clear and simple picture of neurological defect [5].

Has been detected, that in the mature brain of the newborn with severe cerebral ischemia neuron specific enolase (NSE) and glial fibrillary acidic protein levels in blood were increased [6].

According to the literature, in case of hypoxic ischemic lesions of central nervous system the activated leukocyte cell adhesion molecules (ALCAM), protein S100, and apoptosis marker DR5 the first weeks of newborn life can be used for outcome prediction of the structural changes in tissues of nerve [5].

Some authors have studied changes the brain-derived neurotrophic factor (BDNF), ciliary neurotrophic factor (CNTF), vascular endothelial growth factor (VEGF), activated leukocyte cell adhesion molecule (ALCAM) and apoptosis marker DR5 among 120 newborns diagnosed with perinatal hypoxic CNS lesions aged 25-42 weeks of gestation. Children are divided into 4 groups according to neurosonographic research. I group of 30 newborns, there are no changes, II group of 30 newborn with periventricular leukomalacia, III group of 30 newborn with periventricular hemorrhage, IV group of 30 newborn with periventricular leukomalacia and hemorrhage. The authors identified groups of newborn 2-4 significant increase in blood levels of DR5 and ALCAM, BDNF and VEGF lowering. CNTF is found only infants with conjunction intraventricular hemorrhage and periventricular leukomalacia [7].

About Treatment Problems

Several authors [8-10] propose to apply hypothermia. Other doctors [11,12] offers hypothermia for treatment in newborns, taking into account their severe pathological development, fairly sophisticated diagnostic tools, complex of measures to apply the method hypothermia with combining a nootrop medicines.

In recent years, a number of authors nominated for the position, according to which the oxidation stress has a direct role in brain cell damage caused by brain ischemia [13].

Oxidative stress can develop both against the background of an already existing disease, exacerbating the severity of its course, and act as a key factor in the development of the pathological process [14].

Literature data shows that opportunities of treatment CNS disorders and hypoxia oxidative stress, from available used drugs, only few are used in perinatal practice.

There were made the studies [15], which established that the 2.1% increase in hypoxia model of newborn respirable air, hydrogen can save brain morphology.

Conclusion

The works of authors proved [16] that endogenous hydrogen arises by anaerobic metabolism through the colon have shown that per oral admission of lactulose significantly increases the amount of endogenous hydrogen generation [17].

Lactulose is applicable remedy for the treatment of cerebral ischemic diseases. Mechanism was that, endogenous hydrogen, as a result of interaction with lactulose and bacteria in the gastrointestinal tract, improves the condition of the brain, reducing the damage to neurons and can prevent apoptosis [18].

The stroke model was produced in Sprague-Dawley rats through middle cerebral artery occlusion. Intra-gastric administration of lactulose substantially increased breath hydrogen concentration. Behavioral and histopathological verifications matched biochemical findings. Behaviorally, rats in the lactulose administration group won higher neurological scores and showed shorter escape latency time in the Morris test.

Zhai et al. demonstrated a novel therapeutic effect of lactulose on cerebral ischemia/reperfusion injury and the probable underlying mechanisms. Lactulose intra-gastrically administered possessed neuroprotective effects on cerebral I/R injury in rats, which could be attributed to hydrogen production by the fermentation of lactulose through intestinal bacteria and Nrf2 activation [19].

Concluded that lactulose facilitates endogenous hydrogen generation, reduces oxidation stress and possible to assign during ischemic brain lesions.

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