INFLUENCE OF GESTATION DATE ON THE SEVERITY OF HYPOXIC-ISCHEMIC ENCEPHALOPATHY IN NEWBORN

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Annotation: Identification of the leading cause of CNS dysfunction in a newborn, even with a thorough examination, is often a difficult task. The nature and depth of the pathological process in the brain and spinal cord remain unclear, neurological disorders are dynamic, and an accurate prediction of further development is difficult. In this regard, the use of the terms "encephalopathy" is justified, to refer to "transient and unclassified pathological conditions of the central nervous system." Despite the progress of perinatal medicine, hypoxic-ischemic encephalopathy remains an important cause of infant mortality and damage to the central nervous system, leading to disability in children.

Key words: hypoxic-ischemic encephalopathy, newborns, gestational age, severity, clinical symptoms.

ВЛИЯНИЕ СРОКА ГЕСТАЦИИ НА СТЕПЕНЬ ТЯЖЕСТИ ГИПОКСИЧЕСКИ-ИШЕМИЧЕСКОЙ ЭНЦЕФАЛОПАТИИ У НОВОРОЖДЁННЫХ

Аннотация: Выделение ведущей причины нарушения деятельности ЦНС у новорожденного даже при тщательном его обследовании является нередко сложной задачей. Характер и глубина патологического процесса в головном, спинном мозге остаются неясными, неврологические нарушения динамичны, точный прогноз дальнейшего развития затруднителен. В связи с этим оправдано использование терминов «энцефалопатия», для обозначения «преходящих и неклассифицированных патологических состояний ЦНС». Несмотря на прогресс перинатальной медицины, гипоксически-ишемическая энцефалопатия остаётся важной причиной детской смертности и повреждения центральной нервной системы, приводящей к инвалидности детей.

Ключевые слова: гипоксически-ишемическая энцефалопатия, новорождённые, сроки гестации, степени тяжести, клиниченская симптоматика.

INTRODUCTION

Perinatal hypoxic-ischemic encephalopathy (HIE) is a consequence of insufficient blood supply to the brain of a child during pregnancy, childbirth or during the first month of his life. Hypoxia-ischemia of the brain is the main cause of neurological damage in newborns. One of the main parameters characterizing the severity of hypoxic-ischemic brain damage during childbirth and during pregnancy is the Apgar score and the presence of meconium in the amniotic fluid. [1, 3, 5, 7, 10]

The consequences of HIE can be different: from a slight decrease in attention and restlessness of the child to severe forms of cerebral palsy. [6, 16, 19, 21]

There are 3 degrees of severity of encephalopathy. In most cases, hypoxic-ischemic encephalopathy appears at birth or within a few hours after birth. Children with moderate brain damage may appear healthy during the first days and even months of life. Pathology in them is detected during an ultrasound of the brain in the first month of life, when examined by a neurologist and other specialists. [12, 14, 15, 18] Babies with severe HIE and birth asphyxia usually require intensive care and are treated step by step in the maternity hospital and neonatal pathology department. [4, 11, 13, 20]

Perinatal hypoxic-ischemic encephalopathy of moderate and severe degree is one of the main risk factors for the development of cerebral palsy. The course of HIE is very individual, but, as a rule, it is accompanied by the death of some brain cells and requires timely and correct treatment. Compliance with this principle allows you to achieve significant improvement even with severe brain damage. [2, 8, 9, 17, 22]

MATERIALS AND METHODS

24 newborns born with signs of cerebral ischemia were examined and clinically observed. According to the gestational age of 38-39 weeks - 12 newborns, 40 -41 weeks - 7 newborns, 42 - 43 weeks - 5 newborns. From the anamnesis, the leading risk factors are tight entanglement of the umbilical cord around the neck, premature complete or incomplete placental abruption, diseases of the cardiovascular and respiratory system in the mother, insufficient respiratory efforts of the newborn.

More than half of the observations have a combination of 2-3 high risk factors for the development of posthypoxic complications.

Table 1. HIE severity and gestational age.

n	I degree	II degree	III degree
38-39 weeks - 12 newborns	9 (75%)	1 (8,3%)	2 (16,7%)
40-41 weeks - 7 newborns	-	5 (71,4%)	2 (28,6%)
42-43 weeks - 5 newborns	-	2 (40%)	3 (60%)
	9	8	7

RESULTS AND DISCUSSION

The development of the clinic and the course of the disease depended very much on the severity of hypoxic-ischemic encephalopathy. A mild degree of hypoxic-ischemic encephalopathy with signs of moderate muscle tone, revitalization of tendon reflexes during the first few days, a transient behavioral disorder in the form of weak sucking, irritability, restlessness or drowsiness was observed in 9 newborns from a gestational age of 38-39 weeks. In these newborns, after 3-4 days, the neurological status returned to normal. Hypoxic-ischemic encephalopathy of moderate severity with symptoms of muscular hypotension and a significant decrease in tendon reflexes, with sharply reduced sucking, grasping reflexes, lethargy, drowsiness, with periods of short-term apnea, with convulsions in the first day of life was observed in 8 newborns, of which 1 gestation 38-39 weeks, 5 from gestation 40-41 weeks, 2 from gestation 42-43 weeks. Complete neurological recovery in these newborns occurred within 1-2 weeks.

Severe hypoxic-ischemic encephalopathy with manifestations of stupor or coma, with no reaction to physical stimuli, with irregular breathing, diffuse hypotension of muscles and a deep

decrease in tendon reflexes, lack of sucking, swallowing, grasping reflexes, oculomotor disorders, early and frequent generalized seizures, instability of heart rhythm and blood pressure, renal insufficiency, expressed by signs of oliguria, and in the recovery period-polyuric syndrome, leading to a significant water-electrolyte imbalance was observed in 7 newborns, of which 2-from the gestation period of 38-39 weeks, 2-from the gestation period of 40-41 weeks, 3-from the gestation period of 42-43 weeks. In newborns with severe hypoxic-ischemic encephalopathy, the reaction to the environment gradually recovered by the 4th-5th day of life. Hypotension and feeding difficulties persisted, requiring tube feeding for a week and up to a month.

CONCLUSION

As can be seen from the information presented, when comparing the features of clinical symptoms depending on the gestational age, a severe degree of hypoxic-ischemic encephalopathy was mainly observed in the group of newborns with signs of postmaturity, that is, with a gestational age of 42-43 weeks. There is no clear boundary between the severity of hypoxic-ischemic encephalopathy. After an initial period of well-being, a sudden deterioration may follow, usually indicative of reperfusion disorders. Features of the staging of clinical symptoms of hypoxic-ischemic encephalopathy require the provision of clinical and laboratory monitoring for a long period of time.

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