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# CLINICAL AND LABORATORY DYNAMICS OF HYPOXIC-ISCHEMIC ENCEPHALOPATHY IN THE ACUTE PERIOD IN NEWBORNS

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**Abstract:** Intrauterine fetal hypoxia occupies one of the first places in the structure of the causes of perinatal diseases and mortality. This pathology, as a rule, is a consequence of placental insufficiency, accompanying almost all complications of pregnancy - miscarriage, gestosis, developmental delay or death of the fetus, premature birth, acute or chronic infection.

Keywords: pathomorphology, pathophysiology, biochemistry, hyperammonemia

#### Introduction

The urgency of the problem. Intrauterine fetal hypoxia occupies one of the first places in the structure of causes of perinatal diseases and mortality. This pathology, as a rule, is a consequence of placental insufficiency, which accompanies almost all complications of pregnancy - miscarriage, gestosis, developmental delay or fetal death, premature birth, acute or chronic infection [11,16,48].

Recent epidemiological studies indicate the leading role of brain lesions that occurred in period perinatal in the dysadaptation, and in some cases, disability of children. Thus, in the structure of children's disability, lesions of the nervous system account for about 50% [27,56,67,99,102]. Thus, 35-40% of children with disabilities are disabled due to perinatal lesions of the nervous system [67,68,92]. Therefore, the issues of hypoxic-ischemic encephalopathy relevant.

The neonatal period and the first year of a child's life are characterized by the most active period of brain maturation, and the action of such an aggressive factor as hypoxia on the developing brain of a child dictates the need for further study of this pathology [6,10,59].

In the scientific community, the study of the diagnosis and clinical consequences of perinatal lesions of the central nervous system (CNS) is actively continuing, and significant progress has been made in studying the

mechanisms of development of certain forms of hypoxic - ischemic lesions of the central nervous system in newborns. Previous studies revealed that the central links in the pathogenesis of hypoxic lesions of the central nervous system are both cerebrovascular disorders and metabolic disorders [69,76,83,97].

Revealing the dynamics of metabolic disorders in CNS lesions in newborns with CHD remains an urgent task and opens up fundamentally new opportunities both for understanding the pathogenesis and for early diagnosis and correction of identified disorders [4, 6, 32].

Since the gestational age of the newborn at the time of birth plays a special role in the structure of disability from the consequences of HIE, the study of the clinical, instrumental and metabolic characteristics of hypoxic lesions of the central nervous system in premature infants is of particular interest.

The issues of the features of the clinical, instrumental and laboratory course of hypoxic lesions of the central nervous system in newborns depending on the gestational age both in the acute period and in dynamics, as well as the further neuropsychic development of children, remain poorly studied and require additional scientific substantiation.

It is important to develop additional criteria for the severity of hypoxic lesions of the central nervous system in newborns with HIE for



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timely, accurate diagnosis of pathological conditions and more correct dynamic monitoring of children with this pathology.

**Purpose of the study**: to establish the nature of clinical and instrumental, psychomotor and metabolic changes in hypoxic lesions of the central nervous system in newborns with different gestational periods in the acute period and in dynamics.

Material and research methods. We observed 60 newborns with a gestational age of 28 to 41 weeks with hypoxic lesions of the central nervous system of the early period. Children were divided into 3 groups: group 1 of 20 newborns with HIE with gestational age 28-31 weeks, group 2 of 20 newborns with HIE with gestational age 32-37 weeks, and group 3 of 20 newborns with HIE and gestational age 38-41 weeks. The control group consisted of 20 healthy full-term newborns.

At all stages of the study and observation of newborns, a gynecological and obstetric anamnesis was collected, the features of the course of pregnancy and childbirth were studied. The early neonatal period was assessed taking into account data on gestational age, birth weight and length, head and chest circumference, physiological loss of body weight, the state of the child at birth on the Angar scale, the presence of resuscitation measures, and the type of feeding were analyzed. The neuropsychic status of the examined children was assessed in dynamics.

#### RESULTS AND ITS DISCUSSION:

Achievements of the fundamental sciences pathomorphology, pathophysiology, biochemistry, methods of instrumental diagnostics and technologies for assisting newborn children - formed the basis for a serious change in the understanding of the pathogenetic mechanisms of perinatal diagnostic search algorithms, pathology, therapy tactics and follow-up of newborns and young children. The most significant changes in the statistical indicators of mortality and morbidity were noted among premature

babies. The high frequency of severe concomitant perinatal pathology in this category of children led to an increase in childhood disability, in the structure of which the leading positions (21.2%) belong to the pathology of the nervous system and sensory organs (Barashnev Yu.I., 2006). Among the etiological factors,

In this regard, under our supervision there were 60 newborns with different gestational age and with HIE of varying severity of the early period.

According to the results of clinical and instrumental examination, all children were diagnosed with hypoxic damage to the central nervous system of varying severity: 14 newborns (in 23.3% of cases) had mild central nervous system damage, 26 (43.3%) had moderate, and 20 (33.3%) severe. (Figure 3.1.1).

The diagnosis of hypoxic lesions of the central nervous system in newborns was made in accordance with the Classification of perinatal lesions of the nervous system in newborns, 2000 [49].

The severity of hypoxic encephalopathy was established on the basis of clinical syndromes and examination results according to the following criteria: - for mild degree, cerebral ischemia of the 1st degree (mild), intrapartum hypoxia, mild asphyxia at birth; excitation of the central nervous system is more common in full-term, depression - in premature, lasting no more than 5-7 days; moderate hypoxemia, acidosis; NSG - no pathological abnormalities; for moderate severity: cerebral ischemia of the II stage, the syndrome of suppression of excitation, hypertensive-hydrocephalic syndrome, moderate periventricular edema or IVH of the 1st stage. on NSG;

- for severe: cerebral ischemia grade III, severe depression / excitement syndrome, convulsive syndrome, dense periventricular edema or IVH grade II. on the NSG.



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**Figure 3.1.1.** 

Distribution of newborns with HIE according to the severity of CNS lesions.



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Taking into account that the degree of gestational maturity of the fetus determines both the morphological features of cerebral damage and the spectrum of somatic pathology of the neonatal period, gestational age was used as the main grouping feature in the analysis and presentation of the results obtained. In accordance with the tasks, the observed newborns were divided into the following groups: group 1 - 20 newborns with HIE with gestational age 28-31 weeks, group 2 - 20 newborns with HIE with gestational age 32-37 weeks and group 3 - 20 newborns with HIE and with a gestation period of 38-41 weeks.

The clinical severity of perinatal CNS pathology was analyzed both as an independent factor and in combination with gestational age.

There were no significant intergroup differences in sex composition, although in the first comparison group there was a slight predominance of girls. 11 (55%) newborns in group I, 2 (10%) children in group II and 1 child (5%) with intrauterine growth retardation (IUGR) II-III degree in group III had very low birth weight (Figure 3.1. 2.). It should be noted that children with extremely low body weight were not observed. In general, the general characteristics of the comparison groups corresponded to the literature data on gender composition, body weight and condition at birth, as well as the frequency of occurrence of lesions of varying severity in children born at

different stages of gestation (Table 3.1.1).

Figure 3.1.2.The frequency of observation of cases of very low body weight in the compared groups.



Thus, the sample we formed representatively reflected the characteristics of the population of newborns with perinatal pathology of the central nervous system (Perlman J., 2009).

Table 3.1.1. Characteristics of children of the compared groups by sex and body weight

| weight          |           |  |        |   |        |                                  |        |
|-----------------|-----------|--|--------|---|--------|----------------------------------|--------|
| Groups          |           | I group,<br>GV 28-<br>31<br>weeks.<br>n = 20 |        | II group,<br>GV 32-<br>37 weeks<br>n = 20 |        | III group, GV 38-41 weeks n = 20 |        |
|                 |           | Ab<br>s.                                     | %      | Abs.                                      | %      | Ab<br>s.                         | %      |
| Floor           | Boy<br>s  | 7  | 3<br>5 | nine                                      | 4<br>5 | eig<br>ht                        | 4<br>0 |
|                 | Girls     | 13   | 6<br>5 | elev<br>en                                | 5<br>5 | 12                               | 6<br>0 |
| Weig<br>ht, gr. | Ran<br>ge | 1090.0<br>-<br>2610.0                        |        | 1167.0-<br>3560.0                         |        | 1500.0-<br>4800.0                |        |
|                 | M ± m     | 1580.1<br>1 ±<br>245.7                       |        | 2231.07<br>± 424.3                        |        | 3225.5<br>± 549.9                |        |

The period of early postnatal adaptation was complicated in all children. The condition at



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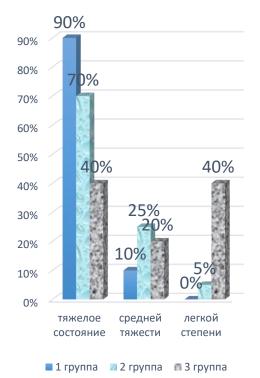
birth was assessed as severe in 20 (100%) children of group I, 18 (90%) children of group II, in 12 (60%) patients of group III, respectively (Figure 3.1.3).

The main scale used to determine the assessment of the state of the newborn and the degree of asphyxia in the child is the Apgar scale (Table 3.1.20

In this regard, during the study, it was found that low scores on the Apgar scale at 1 minute of life were detected in newborns with HIE in all compared groups, but with a greater frequency in newborns of groups 1 and 2 and significantly in relation to both healthy newborns (p <0.001) and to the group of children with HIE but born with a normal gestational age.

The vital activity indices of newborns at the 5th minute of life in the 1st group of the study remained at an assessment of 1-3 points in 50% of newborns, in the 2nd group this indicator was only 20%, while in all children

Figure 3.1.3. Distribution of newborns in the compared groups according to the severity of the general condition.



with normal gestational age with a score of 1-3

points, an improvement was observed.

Apgar score at 1 min. had a significant positive relationship with the gestational age of the child and the severity of subsequently diagnosed perinatal CNS pathology (Table 3.1.3). however, significant differences between the assessments of children with perinatal CNS pathology of varying severity occurred only in group III. So in children of this group with normal weight and gestational age, severe damage to the central nervous system was observed.

The severity of hypoxic damage to the central nervous system was distributed accordingly in the compared groups (table 3.1.4.). So, full-term newborns of the 3rd group of the study, hypoxic lesions of the central nervous system of severe severity were detected in only 5 (25%) children. The average degree of CNS damage was diagnosed in 3 (15%) newborns, and in the main



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Table 3.1.2. Assessment of newborns of the compared groups on the Apgar scale at 1 and 5 minutes.

| and 5 minutes. |     |        |                  |        |                  |     |              |     |
|----------------|-----|--------|------------------|--------|------------------|-----|--------------|-----|
| Apg            | 1st |        | 2nd              |        | Group            |     | Control      |     |
| ar             | gro | up     | group 3          |        | group            |     |              |     |
| scor           |     | •      |                  | 1      |                  |     |              | •   |
| e              |     |        |                  |        |                  |     |              |     |
| (Poi           |     |        |                  |        |                  |     |              |     |
| nts)           |     |        |                  |        |                  |     |              |     |
| 1113)          | 1   | 5      | 1                | 5      | 1                | 5   | 1            | 5   |
|                |     |        |                  |        |                  |     |              |     |
|                | m   | m<br>i | m<br>:           | m<br>i | m<br>i           | mi  | mi           | mi  |
|                | in  |        | i                |        |                  | nut | n            | nut |
|                |     | n      | n                | n      | n                | es  |              | es  |
|                |     | u      |                  | u      |                  |     |              |     |
|                |     | t      |                  | te     |                  |     |              |     |
|                |     | e      |                  | S      |                  |     |              |     |
| 4 -            |     | S      | _                |        | _                |     |              |     |
| 1-3            | 1   | 1      | 9                | 4      | 3                | -   | -            | -   |
| poin           | 4   | 0      | 4                | (      | (                |     |              |     |
| ts             | (7  | (      | 4                | 2 0    | (<br>1<br>5      |     |              |     |
|                | 0   | 5      | 5                |        |                  |     |              |     |
|                | %   | 0      | %                | %      | %                |     |              |     |
|                | )   | %      | )                | )      | )                |     |              |     |
|                | *,  | )      |                  |        |                  |     |              |     |
|                | *   |        |                  |        |                  |     |              |     |
|                | *   |        |                  |        |                  |     |              |     |
| 4-6            | 6   | 1      | 8                | 4      | 1                | 6   | -            | -   |
| poin           | (3  | 0      | (                | (      | 0                | (30 |              |     |
| ts             | 0   | (      | 4                | 2 0    | (                | %)  |              |     |
|                | %   | 5      | 0                |        | 5                |     |              |     |
|                | )   | 0      | %                | %      | 0                |     |              |     |
|                |     | %      | )                | )      | %                |     |              |     |
|                |     | )      |                  |        | )                |     |              |     |
| 7-8            | -   | -      | 3                | 1      | 7                | 14  | 3            | -   |
| poin           |     |        | (                | 2      | (                | (70 | (1<br>5<br>% |     |
| ts             |     |        | 1                | (      | 3                | %)  | 5            |     |
|                |     |        | 5                | 6      | 5                |     | %            |     |
|                |     |        | (<br>1<br>5<br>% | 0      | (<br>3<br>5<br>% |     | )            |     |
|                |     |        | )                | %      | )                |     |              |     |
|                |     |        |                  | )      |                  |     |              |     |
| 9-             | -   | -      |                  |        |                  |     | 17           | 20  |
| 10             |     |        |                  |        |                  |     | (8<br>5      | (10 |
| poin           |     |        |                  |        |                  |     | 5            | 0%  |
| ts             |     |        |                  |        |                  |     | %            | )   |
|                |     |        |                  |        |                  |     | )            |     |
|                |     |        |                  |        |                  | •   |              |     |

Table 3.1.3.

The relationship between gestational age, the severity of hypoxic damage to the central nervous system and the Apgar score at 1 minute.

| at I milute. |             |             |  |  |  |  |
|--------------|-------------|-------------|--|--|--|--|
|              | Severity of | Apgar score |  |  |  |  |
|              | CNS         | at 1 minute |  |  |  |  |
|              | Damage      |             |  |  |  |  |
| 1 group      | r = +0.705  | r = +0.688  |  |  |  |  |
| (28-31       |             |             |  |  |  |  |
| weeks)       |             |             |  |  |  |  |
| Group 2      | r = +0.612  | r = +0.525  |  |  |  |  |
| (32-37       |             |             |  |  |  |  |
| weeks)       |             |             |  |  |  |  |
| 3 group      | r = +0.407  | r = +0.500  |  |  |  |  |
| (38-41       |             |             |  |  |  |  |
| weeks)       |             |             |  |  |  |  |

Thus, it can be concluded that premature infants with severe HIE have the greatest severe metabolic disorders, which tend to normalize over time, but still significantly differ from the norm. Also, premature infants have more severe shifts in blood gas composition compared to full-term infants with DIE. These facts may contribute to the worsening of neurological symptoms in HIE in the acute period, as well as in the later period of the development of hypoxic - ischemic encephalopathy.

#### **CONCLUSIONS**

- 1. The hypoxic-ischemic nature of the lesion of the central nervous system in newborns depends on the gestational age and is manifested by oppression syndromes in premature infants, agitation syndromes and hypertensive-hydrocephalic phenomena in full-term infants.
- 2. An increase in the activity of the enzyme alkaline in newborns with HIE, is especially pronounced in premature newborns, throughout the entire neonatal period and plays a significant role in the mechanisms of hypoxic damage to the central nervous system.
- 3. In HIE, neonates with different gestational



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ages have hyperammonemia, which contributes to the severity of the HIE condition.

4.HIE in newborns is accompanied by disturbances in the gas composition of the blood, which requires dynamic monitoring and timely oxygen therapy

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