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SYSTEMS ANALYSIS OF CLINICAL, BIOCHEMICAL, AND NEUROIMAGING PREDICTORS OF NEONATAL HYPOXIC-ISCHEMIC ENCEPHALOPATHY

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Abstract: Perinatal hypoxic ischemia is caused by impaired cerebral blood supply and oxygenation during childbirth or in the first hours of life, which triggers a cascade of secondary energy failure, oxidative stress, and neuroinflammation. In recent years, significant attention has been devoted to improving methods for the early diagnosis and prognosis of hypoxic-ischemic injuries using clinical scales, neuromonitoring, biomarkers, and neuroimaging.

Keywords: hypoxic-ischemic encephalopathy, newborns, prognosis, biomarkers, neuroimaging, electroencephalography, neuroprotection, perinatal hypoxia, neonatal neurology, integrative diagnostics

Introduction

Despite advances in neonatology and pediatric neurology, the incidence of hypoxic-ischemic encephalopathy (HIE) in full-term infants is 1–5 per 1,000 live births, with the risk of disability increasing in direct proportion, including cerebral palsy, epilepsy, and psychomotor developmental delay [1]. Perinatal hypoxic ischemia is caused by impaired cerebral blood supply and oxygenation during childbirth or in the first hours of life, which triggers a cascade of secondary energy failure, oxidative stress, and neuroinflammation [2].

In recent years, significant attention has been paid to improving methods for early diagnosis and

predicting the outcomes of hypoxic-ischemic lesions using clinical scales, neuromonitoring, biomarkers, and neuroimaging [3]. The pathogenesis of hypoxic-ischemic encephalopathy is a complex cascade of metabolic and structural disorders that develop as a result of insufficient oxygen and nutrient supply to the brain during the perinatal period [4].

Neonatal hypoxic-ischemic encephalopathy (HIE) remains one of the leading causes of neurological morbidity and disability in the neonatal period, determining the high medical and social significance of this problem. The incidence of GIE is 1-6 cases per 1000 full-term newborns and increases significantly in premature infants, while severe forms of the disease are associated with high mortality and the formation of persistent neurological disorders [5,6,7,8]. The pathogenesis of hypoxic-ischemic brain damage in newborns represents a complex cascade of interconnected processes, including disorders of energy metabolism, excitotoxicity, oxidative stress, inflammatory reactions, and programmed cell death. The multifactorial nature of pathogenetic mechanisms and the variability of clinical manifestations necessitate a comprehensive approach to diagnosing and predicting the course of the disease.

Traditional clinical criteria for assessing GIE severity, based on neurological status and instrumental research data, do not always allow for the timely identification of patients with a high risk of adverse outcomes. In this regard, the development of integrated prognostic models, including the analysis of a wide range of clinical, laboratory, and neuroimaging parameters, is a pressing task.

Modern biochemical markers of neural damage, including neurospecific enolase, S-100β protein, glial fibrillar acid protein, and other neuroproteins, provide an opportunity to quantitatively assess the degree of nerve tissue damage and monitor the dynamics of the pathological process. Neuroimaging methods, including magnetic resonance imaging with diffusion-weighted images, MR spectroscopy, and functional MRI, allow for the detection of structural and metabolic changes in the early stages of the disease[9,10,11].

Systematic analysis of a set of prognostic factors using modern methods of mathematical modeling and machine learning opens up new perspectives for creating high-precision predictive models that contribute to personalized therapeutic approaches and improved disease outcomes.

Purpose of the study

To evaluate the diagnostic and prognostic significance of integrating clinical-neurological, laboratory, and instrumental indicators in newborns with hypoxic-ischemic encephalopathy

Research Material and Methods.

he study included 67 newborns (main group) who were born and subsequently underwent examination and treatment in the obstetric department and the neonatology and neonatal pathology department of the Multidisciplinary Clinic of Samarkand State Medical University. All children in the main group exhibited signs of hypoxic-ischemic damage to the central nervous system formed during the perinatal period. Depending on the severity of the condition and the severity of clinical and instrumental manifestations of hypoxic-ischemic brain damage, the main group was divided into subgroups. Subgroups were formed based on the pathogenetic mechanisms of hypoxic-ischemic damage, the severity of clinical symptoms, and the results of comprehensive clinical and instrumental examination. The control group consisted of 27 clinically healthy newborns without signs of perinatal damage to the central nervous system, comparable in gestational age and gender to the main group.

All newborns underwent comprehensive examination using a multi-domain approach. Clinical assessment included a detailed clinical-neurological examination with analysis of consciousness level, muscle tone, reflex sphere, motor activity, and the presence of pathological neurological symptoms. To objectify the severity of hypoxic-ischemic damage to the central nervous system, standardized scales were used, including the Apgar scale (at the 1st and 5th minutes of life), the Sarnat-Sarnat scale for

determining the degree of hypoxic-ischemic encephalopathy, and modified neonatal neurological scales for assessing the level of depression or excitation of the central nervous system. The use of scale methods has increased the reproducibility and comparability of clinical data.

Research Result and Discussion

In the first stage of the study, a clinical and neurological assessment of 67 newborns with hypoxic-ischemic damage to the central nervous system was conducted using the standardized Apgar and Sarnat-Sarnat scales. The control group consisted of 27 clinically healthy newborns. Analysis of the Apgar scale indicators revealed significant intergroup differences between the three subgroups of the main group and the control group[12,13]. Thus, the average score on the Apgar scale in the 1st minute of life in newborns with mild hypoxic-ischemic damage was 6.9±0.6, with moderate severity - 5.2±0.7, and with severe severity - 3.3±0.8, while in the control group, this indicator was significantly higher and amounted to 8.2±0.4 (ANOVA, $p<0.001$). A similar trend was observed in the 5th minute of life: 7.8±0.5, 6.1±0.6, 4.1±0.7, and 9.0±0.3 respectively ($p<0.001$). The distribution of hypoxic-ischemic encephalopathy degrees on the Sarnat-Sarnat scale clearly corresponded to the clinical stratification: in the first subgroup, 100% of newborns were diagnosed with grade I HIE, in the second subgroup, 100% were diagnosed with grade II, and in the third subgroup, all children were diagnosed with grade III hypoxic-ischemic encephalopathy ($\chi 2$, $p < 0.001$).

Thus, the integration of clinical, laboratory, and instrumental indicators allows for the formation of a prognostically significant model for assessing the severity and unfavorable course of hypoxic-ischemic encephalopathy in newborns. The proposed model can be considered a basis for personalized monitoring, early identification of high-risk groups, and optimization of therapeutic and diagnostic tactics in neonatal practice[14,15].

Conclusions

The severity of hypoxic-ischemic damage to the central nervous system in newborns is significantly correlated with clinical and neurological manifestations, laboratory markers of metabolic stress, and the severity of structural and functional changes in the brain according to instrumental research methods. The multi-domain approach ensures more accurate early risk stratification and predicts the unfavorable course of hypoxic-ischemic encephalopathy.

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