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The state of neonatal neurosonography in infants with perinatal nervous system damage

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Abstract: In this study, the authors conducted ultrasound examinations of the central nervous system in newborns with perinatal nervous system damage. It was observed that infants born to mothers with a complicated obstetric history and pathological course of delivery constitute a high-risk group for perinatal nervous system damage. To early detect structural brain changes, neurosonographic examinations of the central nervous system are recommended for all newborns in the high-risk group.

Keywords: Hypoxia, asphyxia, depth of the anterior horns of the lateral ventricles, intraventricular hemorrhage, brain edema.

Introduction: Perinatal hypoxia occupies a prominent place among perinatal factors that affect not only the fetus's condition but also the characteristics of the neonatal period, ultimately impacting the child's health and future development [5]. More than half of all cases of central nervous system (CNS) dysfunction in infants are attributed not to acute hypoxia during childbirth but to prolonged, chronic hypoxia in the fetus and newborn [1,3]. Among perinatal brain injuries, cerebrovascular pathology takes a leading role. One of the primary causes of hemorrhagic and ischemic brain injuries is cerebral hemodynamic disturbances [4]. Hypoxia is recognized as the primary etiological factor in perinatal nervous system pathology, cerebral vascular disorders, leading to the development of hemorrhagic and ischemic CNS injuries in newborns [2,4].

Objectives

The aim of this study was to investigate the clinical manifestations and neurosonography features in newborns with perinatal nervous system injuries.

METHODS

A total of 60 newborns with various gestational ages and perinatal nervous system injuries were observed in the Physiological and Neonatal Intensive Care

Department of the Samarkand Regional Perinatal Center.

The criteria for including children in the study groups were as follows: Group I consisted of 20 healthy newborns born to healthy mothers aged 21 to 33 years, with no complicated obstetric history, and a normal course of pregnancy and delivery. Among them, 12 were full-term infants, and 8 were "conditionally healthy" preterm infants. The group of "conditionally healthy" preterm infants included children born with a gestational age between 35 and 37 weeks and a body weight ranging from 1500 to 2500 grams.

The second group consisted of 20 children who experienced acute asphyxia during childbirth but were born to healthy mothers. The causes of acute hypoxia were as follows: cesarean section (5); umbilical cord entanglement around the neck (5); prolonged labor (7); foot and breech presentation (3). Group II comprised 20 newborns born to healthy mothers who experienced acute asphyxia during childbirth, with Apgar scores averaging below 6-7 points. The clinical picture manifested as a syndrome of increased neuro-reflex excitability characterized by regurgitation, sleep disturbances, chin tremors, restlessness, spontaneous Moro reflex (phase I), and a syndrome of depression characterized by muscle hypotonia, hypodynamia, weak suckling, horizontal nystagmus, and gastrointestinal dyskinesias.

The third group included 20 newborns who experienced chronic intrauterine hypoxia. The causes of chronic intrauterine hypoxia were severe anemia (5); exacerbation of chronic pyelonephritis with severe preeclampsia (5); elevated blood pressure and edema (4); threatened abortion and vomiting in pregnant women (4); prolonged gestosis (1); complete low fetal presentation (1). This group exhibited low Apgar scores of 1-3 points, a complicated obstetric-gynecological history, and more pronounced signs of immaturity. When studying neurological symptoms in these children, there were observations of no reaction to examination and painful stimuli, adynamia, areflexia, atony, a sluggish or absent pupillary reaction to light, sometimes localized ocular symptoms. The skin was cyanotic, pale with a "marble shade" (indicative of microcirculation disturbances). Spontaneous breathing was shallow, with intercostal retraction. Heart sounds were diminished, and moderate hepatomegaly was palpable.

The diagnosis of perinatal encephalopathies, depending on the nervous system lesions, was established according to the classification of perinatal nervous system injuries in newborns by Sarnat and Sarnat in 1976.

The structural ultrasound examination of the brain using B-mode (neurosonography) was performed on the GE Logic F 8 device (USA) with the use of multi-frequency convex probes of 5.5 MHz.

Statistical data analysis was carried out using specialized SPSS software (version 29, IDV Co., Armonk, NY, USA).

RESULTS AND DISCUSSION

During the analysis of neurosonography parameters, including the depth of the anterior horns (right and left) and the depth of the lateral ventricles (right and left), in healthy newborns and those with acute and chronic hypoxia, significant alterations were identified, demonstrating statistically significant differences. Conversely, data for the parameters of the third and fourth ventricles in healthy infants and those with acute or chronic hypoxia did not exhibit statistical variance.

Specifically, the depth of the right anterior horn of the lateral ventricles in healthy subjects was 0.3 cm, while in cases of acute asphyxia, it averaged 0.388 ± 0.100 cm, with statistical significance ($p \leq 0.05$), and in instances of chronic hypoxia, it measured 0.418 ± 0.124 cm ($p \leq 0.05$). On the left side, the depth of the anterior horns of the lateral ventricles was 0.3 cm in healthy subjects, 0.388 ± 0.103 cm in cases of acute asphyxia ($p \leq 0.05$), and 0.437 ± 0.133 cm in cases of chronic hypoxia, all showing statistical differences ($p \leq 0.05$).

The depth of the lateral ventricles' bodies on the right side in healthy newborns was 0.3 cm, whereas in cases of acute asphyxia, it averaged 0.426 ± 0.134 cm with statistical significance ($p \leq 0.05$), and in instances of chronic intrauterine hypoxia, it measured 0.448 ± 0.172 cm ($p \leq 0.05$). On the left side, the depth of the lateral ventricles' bodies was 0.3 cm in healthy subjects, 0.417 ± 0.147 cm in cases of acute asphyxia ($p \leq 0.05$), and 0.425 ± 0.185 cm on average in cases of chronic intrauterine hypoxia ($p \leq 0.05$). Conversely, the parameters of the third and fourth ventricles in healthy newborns and in children with acute asphyxia and chronic hypoxia did not exhibit statistically significant differences (Table 1)

Comparative characteristics of neurosonography parameters in newborns (M±m).

Table 1.

No	Groups of examined patients / Variables		Healthy newborns n=20	Acute hypoxia n=20	Chronic hypoxia n=20
1	The depth of the anterior horns of the lateral ventricles	Right (0,2-0,3 sm)	0,3±0,000	0,388±0,100 p≤0,05	0,418±0,124 p1≤0,05, p2>0.5
		Left (0,2-0,3 sm)	0,3±0,000	0,388±0,103 p≤0,05.	0,437±0,133 p1≤0,05, p2>0.5
2	The depths of lateral ventricles	Right (0,2-0,3 sm)	0,300±0,000	0,426±0,134 p≤0,05	0,448±0,172 P1≤0,05, p2>0.5
		Left (0,2-0,3 sm)	0,300±0,000	0,417±0,147 p≤0,05	0,425±0,185 p1≤0,05, p2>0.5
3	III ventricle (0,3-0,5 см)		0,450±0,000	0,515±0,124 p-p>0.5	0,492±0,173 p1>0.5; p2>0.5
4	IV ventricle (0,3-0,5 см)		0,460±0,000	0,461±0,101 p>0.5	0,462±0,111 p1>0.5 p2>0.5;

"P" stands for the significance of the differences between healthy and acute hypoxia groups.

"P1" stands for the significance of the differences between healthy and chronic hypoxia groups.

"P2" stands for the significance of the differences between the acute and chronic hypoxia groups.

During neurosonography in Group II (Figure 1) newborns, the following findings were observed: 1st-degree lateral ventricular dilatation (LVH) in 4 newborns (20%); ventriculomegaly in 6 newborns (30%); hypoxic changes in the basal ganglia and periventricular area in 9 newborns (45%), and one newborn without pathology (5%).

The ultrasonographic picture in Group III was characterized by immaturity of brain structures in 2 newborns (10%), hypoxic changes in the basal ganglia and periventricular area in 4 newborns (20%), ventriculomegaly in 6 newborns (30%), 1st to 2nd-degree lateral ventricular dilatation (LVH) in 6 newborns (30%), brain edema in one newborn (5%), and a pseudocyst of the cerebral ventricles in one newborn (5%).

CONCLUSIONS

Therefore, infants born with chronic intrauterine hypoxia and acute birth asphyxia are at risk of perinatal nervous system damage. To facilitate early detection and timely staged treatment of infants with hypoxic nervous system damage, it is recommended to perform neurosonography of the brain structures for all newborns in the risk group.

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