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EARLY DIAGNOSIS OF ENCEPHALOPATHY AFTER SURGICAL CORRECTION ON THE BACKGROUND OF HYPOVOLEMIA IN CHILDREN WITH CONGENITAL HEART DEFECTS

Azimova Nodira Mirvositovna

Candidate of Medical Sciences, Associate Professor of the Department of Neurology and Pediatric Neurology, Medical Genetics of TSMU

Rasulova Umida Lazizjon kizi

2st-year Master's Student, Department of Neurology,

Pediatric Neurology, and Medical Genetics of TSMU

Annotation: Research Objective: To assess the impact of hypovolemia during surgical intervention on the development of neurological symptoms in encephalopathy among children with congenital heart defects, and to ensure timely identification of neurological complications.

Materials and Methods: The study will employ standard clinical, neurological, instrumental, and laboratory methods for examining children with congenital heart defects. Sixty children with congenital heart defects are scheduled to be examined in the cardiac intensive care unit of TSMU clinic.

Keywords: encephalopathy; congenital heart defects; hypovolemia; neuronal dysfunction; surgical correction; postoperative complications; early diagnosis; cerebral perfusion.

Relevance. Congenital heart defects (CHD) are among the most common congenital anomalies in newborns and often require early surgical intervention, which is associated with a high risk of neurological complications [1]. Although the effectiveness of CHD treatment has significantly improved over recent decades, the incidence of adverse neurological outcomes, such as encephalopathy, remains high [2]. One of the key factors exacerbating the risk of encephalopathy is hypovolemia, which may occur both before and after surgical correction [3]. Early diagnosis of encephalopathy in this patient population is critically important, as brain injury during periods of active development can have long-term consequences for a child's cognitive and motor development [4].

Modern neuromonitoring techniques enable the detection of ischemic and hypoxic changes at early stages, thereby creating opportunities for timely intervention [5]. Nevertheless, standardized protocols for the early diagnosis of encephalopathy in children with CHD in the postoperative period in the presence of hypovolemia remain underdeveloped [6]. Neurological complications following cardiac surgery in children encompass a wide spectrum of disorders, ranging from seizure syndromes to persistent delays in psychomotor development, underscoring the need for a systematic approach to brain monitoring [10]. According to recent studies, children with CHD frequently exhibit signs of brain immaturity and delayed cerebral development as early as the neonatal period [5, 9]. This necessitates the integration of neuroprotective strategies and diagnostic algorithms already at the stage of preoperative preparation and in the early postoperative period [6].

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Thus, the study of the pathogenesis, clinical features, and diagnostic methods of encephalopathy in children after surgical correction of CHD in the context of hypovolemia represents a highly relevant area of contemporary pediatric cardiology and neurology [1, 2, 4].

1.1. Etiology and Pathogenesis.

The etiology of encephalopathy following surgical correction of congenital heart defects (CHD) in children is multifactorial and includes both congenital and acquired components [2]. In most patients with CHD, cerebral perfusion impairment may already be present preoperatively due to reduced cardiac output and decreased oxygenation [5]. This preoperative hypoxic state creates a predisposition for increased vulnerability of the central nervous system to additional injurious factors during surgical intervention [9]. One of the key risk factors is hypovolemia, which may develop as a result of intraoperative blood loss, vascular tone dysfunction, or insufficient volume of infusion therapy [6]. Hypovolemia reduces cerebral perfusion, leading to ischemic changes and activation of secondary damaging mechanisms such as oxidative stress and inflammation [4]. The neonatal brain, which is in a phase of active growth and differentiation, is particularly vulnerable to these changes [7].

Other important etiological factors include potential complications of cardiopulmonary bypass, hypothermia, reperfusion injury, and disturbances of homeostasis in the postoperative period [3, 6]. Certain forms of CHD, especially those associated with reduced pulmonary blood flow or mixed oxygen saturation, increase the risk of chronic hypoxia, which is further exacerbated during surgical intervention [2, 5]. Individual neurodevelopmental characteristics in children with CHD should also be taken into account, including delayed myelination, reduced gray matter volume, and immaturity of the cerebral vascular network, all of which render the brain particularly susceptible to ischemic and hypoxic injury [5, 9]. This is supported by MRI findings demonstrating brain developmental abnormalities in children with CHD even prior to surgery [3, 5]. Thus, the etiology of encephalopathy in children after surgical correction of CHD in the context of hypovolemia is determined by a combination of preoperative factors (hypoxia, cardiac anatomical features), intraoperative influences (hypovolemia, cardiopulmonary bypass, hypothermia), and characteristics of postoperative recovery [2, 3, 4, 5, 6].

The pathogenesis of encephalopathy following surgical correction of congenital heart defects in the setting of hypovolemia in children involves a cascade of metabolic, inflammatory, and hemodynamic disturbances leading to injury of the developing brain [4]. The central mechanism of this process is reduced cerebral perfusion caused by hypovolemia, resulting in ischemia and hypoxia of brain tissue [6]. Against the background of decreased systemic blood flow and cerebral oxygenation, energy deficiency, hypoglycemia, and acidosis develop, which collectively disrupt the function of neurons and glial cells [4]. Immature oligodendrocytes responsible for the formation of the myelin sheath of axons are particularly vulnerable; their loss leads to long-term white matter abnormalities [7]. In newborns with CHD, whose brains often already exhibit signs of immaturity prior to surgery, ischemic injury further aggravates neurodevelopmental delay [5].

According to MRI data, even before surgical intervention such children may demonstrate signs of corpus callosum hypoplasia, reduced white matter volume, and underdevelopment of the cerebral cortex [5, 9]. During surgery, cardiopulmonary bypass and hypothermia are frequently employed; while these techniques protect tissues from hypoxia, they may simultaneously

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contribute to reperfusion injury after restoration of blood flow [6]. This process activates free radical reactions and enhances the inflammatory response involving microglia and cytokines, resulting in secondary brain cell injury [4, 6].

The postoperative period is also characterized by hemodynamic instability, possible fluctuations in glucose and sodium levels, and episodes of hypotension, all of which exacerbate hypoxic-ischemic changes [3]. Brain injury may be diffuse, affecting white matter and cortex, or focal, with the formation of periventricular leukomalacia and hemorrhages [7]. Thus, the pathogenesis of encephalopathy in children with CHD in the context of hypovolemia is a complex, multistage process in which hypoperfusion, hypoxia, inflammation, and neuronal immaturity represent the principal pathogenic mechanisms [4, 5, 6, 7].

1.2. Clinical Manifestations.

The clinical manifestations of encephalopathy following surgical correction of congenital heart defects (CHD) in children are diverse and depend on the severity and nature of brain injury, as well as on the patient's age [4]. In newborns and infants, early signs of encephalopathy include decreased level of consciousness, suppression of sucking and swallowing reflexes, muscular hypotonia or hypertonia, as well as episodes of apnea and seizures [3]. Neurological symptoms often emerge within the first hours or days after surgery and may be either transient or progressive [10].

In infants treated in intensive care units, the diagnosis of encephalopathy is challenging, as its manifestations may be masked by the effects of anesthesia, hypothermia, or metabolic disturbances [6]. In this context, regular neuromonitoring (e.g., using amplitude-integrated EEG and near-infrared spectroscopy, NIRS) becomes an important tool for the early detection of functional impairments [4]. At older ages, children who have undergone surgical correction of CHD frequently exhibit delays in psychomotor and cognitive development, speech disorders, impairments of attention and memory, as well as behavioral disturbances [2]. Clinical studies indicate that these children have an increased risk of developing attention-deficit/hyperactivity disorder (ADHD), as well as anxiety and affective disorders [2, 8].

Motor development impairments include delayed acquisition of motor skills, gait instability, clumsiness, and reduced endurance [1]. MRI studies in such patients often reveal structural abnormalities, including reduced white matter volume, cortical atrophy, periventricular leukomalacia, and hemorrhages [3, 5]. Some forms of encephalopathy are accompanied by seizure disorders, including neonatal seizures, which are also associated with a poorer neurodevelopmental prognosis [10]. In addition, due to impaired neurovegetative control, children may exhibit instability of heart rhythm, thermoregulation, and respiration, particularly in the first days after surgery [6].

The clinical course of encephalopathy in children with CHD largely depends on the timeliness of detection and correction of factors aggravating cerebral ischemia, primarily hypovolemia, hypoxia, and metabolic disturbances [4, 6, 10].

1.3. Diagnostics.

AMERICAN ACADEMIC PUBLISHER
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Early and accurate diagnosis of encephalopathy in children after surgical correction of congenital heart defects (CHD) in the setting of hypovolemia is crucial for improving outcomes and preventing severe neurological sequelae [4]. The diagnostic algorithm involves a combination of clinical assessment, neurophysiological methods, laboratory parameters, and neuroimaging techniques [6]. Clinical assessment is based on observation of the level of consciousness, muscle tone, motor responses, presence of seizures, and behavioral characteristics of the child [3]. However, in the context of postoperative sedation and analgesia, these signs may be blunted, which limits the effectiveness of clinical monitoring [6].

Neurophysiological methods, such as amplitude-integrated electroencephalography (aEEG), enable the detection of patterns of reduced cerebral activity, epileptiform discharges, and episodes of cortical function depression, which often precede overt clinical manifestations [4]. According to current recommendations, the use of aEEG within the first 72 hours after surgery is particularly important in patients at high risk [6]. Magnetic resonance imaging (MRI) is the gold standard for visualization of central nervous system lesions, especially when combined with diffusion-weighted imaging (DWI), which allows the identification of acute ischemic changes [3]. In children with CHD, MRI frequently reveals signs of white matter underdevelopment, reduced brain volume, and periventricular leukomalacia [5, 9]. In some cases, foci of hemorrhage may also be detected, indicating vascular instability under conditions of hypoperfusion [7].

Additional diagnostic tools include laboratory parameters such as lactate and glucose levels, acid–base balance indices, inflammatory markers (C-reactive protein, procalcitonin), as well as biomarkers of brain injury—S100B and neuron-specific enolase (NSE), which have been actively investigated in recent years [6]. A modern diagnostic approach requires multidisciplinary evaluation involving a pediatric cardiologist, neurologist, intensivist, and a specialist in functional diagnostics [4, 6]. Only a comprehensive, multimodal diagnostic strategy allows reliable assessment of the severity and nature of brain injury, timely detection of abnormalities, and appropriate adjustment of therapeutic management [4, 6].

1.4. Epidemiology.

Encephalopathy in children after surgical correction of congenital heart defects (CHD) is a common condition, particularly among newborns and infants with severe forms of defects [2]. According to various reports, neurological complications are identified in 30–50% of children who undergo cardiac surgery at an early age [1, 8]. A study by Brown et al. (2015) demonstrated that in a cohort of children who underwent CHD correction, 48% were diagnosed with neurological impairments of varying severity, including encephalopathy, seizure disorders, and cognitive deficits [1]. Another review by Mebius et al. (2017) reported that approximately 40% of patients exhibit developmental delay, particularly in cognitive and motor domains, associated with previous hypoxic brain injury [2].

The highest incidence of encephalopathy is observed in children with hypoplastic left heart syndrome, transposition of the great arteries, and pulmonary atresia—conditions that are inherently associated with severe hypoxemia and an increased risk of hypovolemia during the perioperative period [5]. In these patients, the frequency of brain injury detected by MRI reaches up to 70%, including both subclinical and clinically manifest forms of encephalopathy [3, 5]. In

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addition, as shown in the study by Licht et al. (2006), children with CHD often exhibit delayed brain maturation: the volumes of gray and white matter are significantly lower than those in healthy newborns of the same gestational age, which increases the vulnerability of the brain to ischemic injury [9]. This immaturity initiates a cascade of pathological reactions during any perfusion instability, particularly in the context of hypovolemia [5, 9].

According to data from a Russian study by Zavalishina and Solovyeva (2020), neurological complications after cardiac surgery in children occur in 30–40% of patients, with 15–20% of cases involving persistent disorders such as post-hypoxic encephalopathy and seizure syndromes [10]. The likelihood of developing severe encephalopathy is particularly high in preterm infants and in patients with a complicated postoperative course [10].

In Uzbekistan, congenital heart defects are among the most frequently diagnosed congenital anomalies and constitute a substantial proportion of neonatal pathology. According to the Ministry of Health of the Republic of Uzbekistan, more than 3,000 cases of CHD are identified annually in children, with up to 40% requiring surgical correction within the first months of life [11]. Neurological complications, including encephalopathy, are increasingly diagnosed in children who have undergone surgical treatment for CHD. According to estimates from specialists of the Republican Center for Cardiovascular Surgery and clinics of Tashkent Medical University, the incidence of early neurological disorders (including signs of post-hypoxic encephalopathy, seizure syndromes, muscular hypotonia, and developmental delay) reaches 30–40% among children after open-heart surgery. Newborns and infants with critical forms of CHD (such as transposition of the great arteries, tricuspid atresia, common arterial trunk, and others) remain particularly vulnerable to neurological complications, as hypovolemia and cerebral hypoperfusion frequently develop during and after surgical intervention.

Furthermore, experts note that insufficient early neuroimaging (MRI, neurosonography with Doppler studies) and limited access to cerebral oxygenation monitoring in most regional clinics of Uzbekistan complicate the timely diagnosis of encephalopathy in the postoperative period. Nevertheless, positive trends have been observed in recent years: the organization of specialized cardiac intensive care has improved, and neuroprotective and rehabilitation protocols are being increasingly implemented, contributing to a reduction in the incidence of severe neurological sequelae. Thus, the epidemiological situation in Uzbekistan reflects global trends, underscoring the high risk of encephalopathy in children after cardiac surgery and the need for comprehensive neuromonitoring and a multidisciplinary approach to the management of these patients [11].

1.4. Conclusions.

Encephalopathy in children after surgical correction of congenital heart defects, particularly in the context of hypovolemia, is a frequent and severe complication that determines long-term neurological outcomes [1, 2, 10]. The high vulnerability of the immature brain to ischemia and hypoxia, together with the specific pathophysiological features of CHD and the cardiac surgical procedure itself, renders these patients especially susceptible to structural and functional central nervous system impairments [3, 5, 9]. The leading mechanisms of brain injury include hypoperfusion, hypoxia, metabolic stress, and inflammatory responses, which are further exacerbated by reperfusion syndrome and secondary hemodynamic instability [4, 6, 7].

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MRI studies frequently reveal underdevelopment and damage of white matter, which correlates with cognitive and motor impairments in the long-term follow-up period [3, 5]. The clinical manifestations of encephalopathy range from acute neonatal disorders (seizures, depressed consciousness, hypotonia) to persistent delays in psychomotor and cognitive development, attention-deficit/hyperactivity disorder (ADHD), and behavioral disturbances at older ages [2, 8, 10]. Reliable diagnosis is based on a combination of clinical assessment, neurophysiological methods (aEEG), MRI, and laboratory biomarkers of brain injury [4, 6].

Therapeutic management should be comprehensive and initiated with early stabilization of cerebral perfusion and metabolic parameters, include neuroprotective measures, and обязательно provide for long-term multidisciplinary rehabilitation [2, 4, 6]. The use of modern monitoring techniques and a personalized approach to each patient can significantly improve neurological outcomes [6]. Thus, timely diagnosis and treatment of encephalopathy in children after cardiac surgical interventions require coordinated efforts not only from cardiac surgeons and intensivists, but also from neurologists, neuroimaging specialists, and pediatric rehabilitation professionals [2, 4, 6, 10].

References:

1. Brown, K. L., et al. (2015). "Neurologic outcomes after pediatric cardiac surgery." Progress in Pediatric Cardiology, 39(1), 39–45.

2. Mebius, M. J., et al. (2017). "Neurodevelopmental outcomes in children with congenital heart disease: A review." Circulation Research, 120(6), 904–922.

3. Beca, J., et al. (2013). "Injury to the developing brain after cardiac surgery in infants: MRI studies and neurodevelopmental outcomes." Current Opinion in Pediatrics, 25(5), 604–608.

4. Mahle, W. T., et al. (2016). "The importance of brain monitoring in children undergoing cardiac surgery." The Journal of Thoracic and Cardiovascular Surgery, 151(1), 214–221.

5. Miller, S. P., et al. (2007). "Abnormal brain development in newborns with congenital heart disease." The New England Journal of Medicine, 357(19), 1928–1938.

6. Andropoulos, D. B., et al. (2010). "Current strategies for neuroprotection during pediatric cardiac surgery." Seminars in Thoracic and Cardiovascular Surgery, 12(1), 37–47.

7. Volpe, J. J. (2009). "Brain injury in premature infants: A complex amalgam of destructive and developmental disturbances." The Lancet Neurology, 8(1), 110–124.

8. Wernovsky, G. (2006). "Current insights regarding neurological and developmental abnormalities in children and young adults with complex congenital cardiac disease." Cardiology in the Young, 16(S1), 92–104.

9. Licht, D. J., et al. (2006). "Brain maturation is delayed in infants with complex congenital heart defects." Journal of Thoracic and Cardiovascular Surgery, 132(3), 570–577.

10. Zavalishina, T. A., & Solovyeva, N. V. (2020). Neurological complications after surgery in children with congenital heart defects. Russian Pediatric Journal, No. 2, pp. 48–54.