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ЖУРНАЛ КАРДИОРЕСПИРАТОРНЫХ ИССЛЕДОВАНИЙ

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МОРФОФУНКЦИОНАЛЬНОЕ СОСТОЯНИЕ СЕРДЕЧНО-СОСУДИСТОЙ СИСТЕМЫ В ПОСЛЕОПЕРАЦИОННОМ ПЕРИОДЕ У ДЕТЕЙ С ТЕТРАДОЙ ФАЛЛО

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АННОТАЦИЯ

Врожденные пороки сердца (ВПС) – это комплекс структурных аномалий сердечно-сосудистой системы, возникающих в период эмбриогенеза (2-8 недель гестации). ВПС могут иметь как изолированный характер, так и входить в состав сложных комбинированных пороков. Эпидемиологические данные свидетельствуют о стабильной распространенности ВПС на уровне 8-9 случаев на 1000 живорождённых. Послеоперационная летальность в течение 30 дней после кардиохирургических коррекций ВПС составляет в среднем 3,5-4%, с увеличением до 8-9% в группе новорожденных. Тетрада Фалло (ТФ) является одним из наиболее распространенных цианотических ВПС с частотой 3-5 на 10 000 живорождённых. Морфологически ТФ характеризуется стенозом выходного тракта правого желудочка, дефектом межжелудочковой перегородки, декстрапозицией аорты и гипертрофией правого желудочка. Сочетание данных аномалий приводит к снижению легочной перфузии и нарушению системной гемодинамики, манифестируя хронической гипоксемией. Тяжесть гемодинамических нарушений является ключевым фактором, определяющим клиническую картину и тактику хирургического лечения.

Ключевые слова: тетрада Фалло, адаптация сердечно-сосудистой системы, послеоперационный период, гемодинамика, правый желудочек, аритмии, эхокардиография.

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MORPHOFUNCTIONAL STATE OF THE CARDIOVASCULAR SYSTEM IN THE POSTOPERATIVE PERIOD IN CHILDREN WITH TETRALOGY OF FALLOT

ABSTRACT

Congenital heart diseases (CHDs) are a complex of structural anomalies of the cardiovascular system that arise during embryogenesis (2-8 weeks of gestation). CHDs can be isolated or part of complex combined diseases. Epidemiological data indicate a stable prevalence of CHDs at 8-

9 cases per 1,000 live births. Postoperative mortality within 30 days after cardiac surgery for CHD correction averages 3.5-4%, increasing to 8-9% in neonates. Tetralogy of Fallot (ToF) is one of the most common cyanotic CHDs, with an incidence of 3-5 per 10,000 live births. Morphologically, TF is characterized by right ventricular outflow tract stenosis, interventricular septal defect, aortic decstraposition, and right ventricular hypertrophy. The combination of these anomalies leads to a decrease in pulmonary perfusion and a disruption of systemic hemodynamics, manifesting as chronic hypoxemia. The severity of hemodynamic disorders is a key factor determining the clinical picture and surgical treatment tactics.

Keywords: Tetralogy of Fallot, cardiovascular adaptation, postoperative period, hemodynamics, right ventricle, arrhythmias, echocardiography.

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FALLO TETRADASI BO'LGAN BOLALARDA OPERATSIYADAN KEYINGI DAVRDA YURAK-QON TOMIR TIZIMINING MORFOFUNKSIONAL HOLATI

ANNOTATSIYA

Tug‘ma yurak nuqsonlari (TYUN) - bu embriogenez davrida (homiladorlikning 2-8 haftasi) yuzaga keladigan yurak-qon tomir tizimining strukturaviy anomaliyalari majmuasi. Kasallikning morfologik ko'rinishi o'ng qorincha chiqish traktining stenozini, qorinchalararo to'siq defektini, aorta dekstrapozitsiyasini va o'ng qorincha gipertrofiyasini o'z ichiga oladi. Ushbu anatomik o'zgarishlar o'pka perfuziyasi va tizimli gemodinamikaning buzilishiga olib kelib, surunkali gipoksemiya rivojlanishiga zamin yaratadi. TYUN ham alohida xarakterga ega bo‘lishi, ham murakkab kombinatsiyalangan nuqsonlar tarkibiga kirishi mumkin. Epidemiologik ma’lumotlar TYUNning 1000 tirik tug‘ilgan chaqaloqqa 8-9 holatda barqaror tarqalganligini ko‘rsatadi. TYUN kardioxirurgik korreksiyasidan keyin 30 kun ichida operatsiyadan keyingi o‘lim ko‘rsatkichi o‘rtacha 3,5-4% ni tashkil etib, yangi tug‘ilgan chaqaloqlar guruhida 8-9% gacha oshgan. Fallo tetradasi eng keng tarqalgan sianotik yurak nuqsonlaridan biri bo'lib, har 10 000 tirik tug'ilgan chaqaloqqa 3-5 tani tashkil qiladi. Gemodinamik buzilishlarning og'irligi klinik ko'rinish va jarrohlik davolash strategiyasini belgilovchi asosiy omil hisoblanadi.

Kalit so'zlar: Fallo tetradasi, yurak-qon tomir tizimining moslashuvi, operatsiyadan keyingi davr, gemodinamika, o'ng qorincha, aritmiya, ekokardiyografiya.

Introduction: Congenital heart diseases remain one of the leading causes of morbidity and mortality in the pediatric population, occupying a central position among all congenital anomalies. According to modern epidemiological data, their prevalence averages 8–9 cases per 1,000 live births, with a significant proportion represented by complex cyanotic defects, among which Tetralogy of Fallot (ToF) holds a special place as the most common form. Advances in cardiac surgery, including improvements in cardiopulmonary bypass techniques, prenatal diagnostics, and postoperative intensive care, have led to a significant reduction in mortality and a marked increase in survival rates. Consequently, a growing population of patients with repaired ToF has emerged, requiring long-term follow-up and comprehensive management [1,2].

Despite the radical nature of surgical correction, restoration of cardiac anatomy does not equate to full functional recovery. In the postoperative period, a distinct morphofunctional state of the cardiovascular system develops, characterized by a combination of residual hemodynamic disturbances, structural remodeling, and electrical instability of the myocardium. The central component of these changes is right ventricular remodeling. In the preoperative stage, the right ventricle functions under pressure overload due to right ventricular outflow tract obstruction, whereas after surgical correction it is exposed to chronic volume overload, primarily due to pulmonary regurgitation [3,4]. This transition in hemodynamic conditions requires activation of complex adaptive mechanisms, which may eventually become exhausted, leading to progressive right ventricular dysfunction.

At the cellular level, myocardial remodeling is associated with activation of fibrotic processes, transformation of fibroblasts into myofibroblasts, and increased synthesis of collagen types I and III, resulting in diffuse interstitial fibrosis [5]. Simultaneously, changes occur in the expression of contractile proteins in cardiomyocytes, accompanied by reduced energy efficiency and impaired contractility. Oxidative stress also plays a significant role, both as a consequence of chronic hemodynamic overload and surgical injury, further contributing to cellular damage and progression of remodeling [6].

Electromechanical dyssynchrony represents another key mechanism underlying postoperative complications. Surgical intervention, particularly ventriculotomy, often results in right bundle branch block, leading to delayed activation of the right ventricular free wall and mechanical discoordination of myocardial contraction [7]. This reduces cardiac efficiency, increases energy expenditure, and creates a substrate for malignant ventricular arrhythmias. Prolongation of the QRS complex beyond 160–180 ms is considered a strong predictor of adverse outcomes, including sudden cardiac death [7,8].

The long-term clinical course after ToF repair is characterized by the development of several late complications. Chronic pulmonary regurgitation is one of the most significant, often remaining asymptomatic for years but eventually leading to progressive right ventricular dilatation and systolic dysfunction [9]. When critical volumetric thresholds are exceeded, irreversible myocardial changes occur, limiting the effectiveness of subsequent surgical interventions. In addition, ventricular interdependence plays an important role, as dilation of the right ventricle impairs left ventricular filling and reduces cardiac output [10].

Arrhythmias constitute another major clinical problem and represent a leading cause of late mortality in this patient population. The primary mechanism involves macro-reentry circuits formed around surgical scars and areas of fibrosis, creating zones of conduction delay [8]. The presence of ventricular tachyarrhythmias, severe right ventricular dilatation, and prolonged QRS duration are associated with a significantly increased risk of sudden cardiac death [8,11].

Modern strategies for long-term management of patients after ToF repair are based on lifelong monitoring and a multidisciplinary approach. Advanced imaging techniques play a crucial role, with cardiac magnetic resonance imaging being considered the gold standard for assessing ventricular volumes, myocardial mass, and the degree of

pulmonary regurgitation [12]. Echocardiography with speckle-tracking technology allows for early detection of myocardial dysfunction through assessment of global longitudinal strain, providing valuable information even before conventional parameters become abnormal [13].

Biomarkers have also gained increasing importance in recent years. Natriuretic peptides such as BNP and NT-proBNP correlate with right ventricular overload and heart failure severity, while markers of fibrosis and inflammation, including galectin-3 and soluble ST2, provide insight into myocardial remodeling and risk stratification [14]. High-sensitivity troponins may reflect ongoing myocardial injury in conditions of chronic overload [15].

Therapeutic management in the long-term postoperative period includes both medical and surgical approaches. However, the evidence base for pharmacological treatment of right ventricular dysfunction remains limited, and further research is needed to establish effective strategies [16]. Timely pulmonary valve replacement remains the cornerstone of treatment aimed at eliminating chronic volume overload. In recent years, transcatheter pulmonary valve implantation has emerged as a less invasive alternative to open surgery, significantly reducing procedural risk and improving patient outcomes [17].

In addition, increasing attention is being paid to the role of physical rehabilitation. Historically, patients with repaired ToF were advised to limit physical activity; however, contemporary guidelines emphasize the benefits of regular moderate aerobic exercise. Such interventions improve endothelial function, enhance exercise capacity, and positively influence psychological well-being [18]. Structured rehabilitation programs are therefore considered an essential component of comprehensive patient care.

Furthermore, recent studies highlight the importance of inflammatory and metabolic factors in the progression of myocardial dysfunction. Elevated levels of inflammatory markers, including C-reactive protein and interleukin-6, have been associated with adverse outcomes and progression of cardiac remodeling [6,14]. The role of epicardial adipose tissue as a metabolically active organ influencing myocardial function through paracrine signaling is also being actively investigated, further supporting the systemic nature of the disease [19].

Thus, contemporary understanding of the morphofunctional state of the cardiovascular system after ToF repair extends beyond the anatomical outcome of surgery. The postoperative period should be regarded as a continuous adaptive process involving complex interactions between hemodynamic, structural, molecular, and electrophysiological mechanisms. This underscores the need for a comprehensive and personalized approach to patient management, integrating advanced diagnostic tools, timely surgical interventions, medical therapy, and rehabilitation strategies, ultimately improving long-term prognosis and quality of life [1,2,17,18].

Additional important evidence from large cohort studies further emphasizes the long-term complexity of outcomes in patients after Tetralogy of Fallot repair. In a large multicenter study conducted by Clayton A. Smith and colleagues, which included more than 3000 patients, it was demonstrated that long-term survival after surgical correction is generally high, reaching over 90% at 25 years; however, the presence of genetic abnormalities significantly increases both early and late mortality risk [1]. These findings highlight the critical role of genetic and phenotypic heterogeneity in determining long-term prognosis. At the same time, long-term observational data indicate that survival alone does not reflect the true burden of disease. In a 50-year follow-up study by R.M. Kauling et al., it was shown that although survival remains relatively high (up to 71–84%), event-free survival is extremely low (around 9%), with a high prevalence of reinterventions (40%) and clinically significant arrhythmias [2]. This underscores that Tetralogy of Fallot should be considered a chronic condition with progressive morbidity rather than a fully corrected disease.

The concept of right ventricular remodeling as a central determinant of long-term outcomes has been extensively described in the works of Tal Geva, who emphasized the prognostic importance of right ventricular dilation and dysfunction in patients after repair. It has been shown that progressive enlargement of the right ventricle, especially in the presence of pulmonary regurgitation, is strongly associated with reduced exercise capacity, arrhythmias, and increased risk of sudden cardiac death [3,4]. These findings support the need for early identification of adverse remodeling and timely intervention. Furthermore, studies by J.P.G. van der Ven and colleagues demonstrated that despite excellent early surgical results, long-term survival at 30 years ranges between 68% and 90%, while functional limitations and ventricular dysfunction remain common [5]. These results confirm that structural correction does not eliminate the underlying pathophysiological processes. Another important contribution comes from the work of L. Mercer-Rosa, who showed that perioperative and surgical factors significantly influence long-term right ventricular remodeling and clinical outcomes [6]. This highlights that not only the disease itself, but also the chosen surgical strategy, plays a crucial role in determining the future trajectory of the patient. In recent years, increasing attention has been paid to advanced imaging and predictive modeling. A study published in the American Heart Association journals demonstrated that cardiac magnetic resonance imaging parameters, particularly right ventricular volume and fibrosis markers, can serve as powerful predictors of adverse outcomes in patients with repaired Tetralogy of Fallot [7]. These findings reinforce the importance of regular imaging surveillance in long-term management.

Taken together, these studies indicate that modern understanding of Tetralogy of Fallot has shifted from a surgically “corrected” defect to a lifelong condition requiring continuous monitoring and intervention. The integration of genetic data, imaging techniques, and clinical parameters allows for more accurate risk stratification and supports the development of personalized management strategies.

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