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MORPHOLOGICAL ALTERATIONS IN CONGENITAL HEART DISEASE IN INFANTS

Gulyamova G.D.
Tashkent State Medical University, Tashkent, Republic of Uzbekistan
gulbahor.gulyamova@bk.ru
https://orcid.org/0009-0000-0956-2954

Abstract: Congenital heart defects (CHD) represent structural abnormalities of the heart and great vessels that develop during embryogenesis (3–8 weeks of gestation). In infants, the morphological pattern depends on the type of defect, the severity of hemodynamic disturbances, the degree of hypoxia, and the progression of heart failure. The prevalence of congenital heart defects in the Republic of Uzbekistan demonstrates certain variability; approximately 8.7% of all cases are attributed to Tetralogy of Fallot.The conducted morphological study of Tetralogy of Fallot revealed pronounced structural alterations in the myocardium, predominantly affecting the right ventricle. Hypertrophic and dystrophic changes of right ventricular cardiomyocytes were observed, whereas the left ventricle showed mainly atrophic and sclerotic processes. In addition to alterations in the contractile myocardium, disturbances of the cardiac conduction system were identified. Both quantitative and qualitative transformations of fiber bundles originating from the sinoatrial node were noted, along with heterogeneous hypertrophic and atrophic changes in various branches of the intraventricular conduction system. In defects associated with valvular abnormalities and ventricular septal defects, a predominance of His bundle fibers directed toward the right ventricle was observed, with a relatively reduced distribution to the left ventricle.Alterations of the microcirculatory bed were also detected. Myocardial hypertrophy led to disruption of capillary histoarchitecture, deformation of vascular lumina, and disorganization of the capillary network. These structural changes were accompanied by local hemodynamic disturbances, reduced capillary perfusion efficiency, and increased vascular permeability, contributing to the development of interstitial edema and aggravation of hypoxic processes in the myocardium.

Keywords: congenital heart defects, Tetralogy of Fallot, morphology, cardiac conduction system

Relevance

Congenital heart defects (CHDs) are structural anomalies in the development of the heart and great vessels that originate during embryogenesis (weeks 3–8 of gestation). In infants, the morphological presentation is determined by the specific type of defect, the severity of hemodynamic disturbances, the degree of hypoxia, and the progression of heart failure [1, 2].

The incidence of CHDs is notably high, with various authors estimating it between 0.8% and 1.2% among all newborns. The spectrum of congenital heart defects ranges from relatively mild developmental vascular and cardiac impairments to severe forms of pathology incompatible with life.Modern data regarding the frequency, structure, and risk factors of CHD formation in children across different regions show a wide

range of fluctuations. In regions of the Russian Federation, official statistics report a rate of 3.17–8.0 per 1000 newborns[3,4].. According to the Ministry of Health of the Russian Federation, in 2006, the total number of registered children under 14 with CHD reached 213,816, with congenital defects occupying a leading position (34%) within the structure of cardiovascular pathology in infants[5,6,10].To date, domestic and foreign literature describes more than 90 anatomical variants of CHD and approximately 200 different combinations. In various regions, defects of the heart and great vessels rank 1st or 2nd in the structure of all congenital malformations, competing for the top position with musculoskeletal anomalies[7,8,9]. According to several authors, the most common defects include ventricular septal defect (VSD), patent ductus arteriosus (PDA), coarctation of the aorta, transposition of the great vessels, and Tetralogy of Fallot. In approximately one-third of cases, CHD is associated with congenital anomalies of the musculoskeletal system, central nervous system, gastrointestinal tract, and the urogenital system.

Goal and Objectives

To characterize the proliferative activity of cardiac mesenchymal cells in relation to the age of patients diagnosed with Tetralogy of Fallot.

Materials and Methods

A retrospective study was conducted on 34 autopsy materials from infants under one year of age who died from CHD at the Pathological Anatomy Center of the Republic of Uzbekistan. Heart tissue sections obtained via morphological methods were fixed in 10% buffered formalin for 72 hours. Following a 1-hour wash in running water, the samples were dehydrated in a graded alcohol series (70%, 80%, 90%, 100%). The samples were then embedded in paraffin blocks. Sections with a thickness of 5–7 μm were prepared using a microtome. After deparaffinization in xylene, the sections were stained with hematoxylin and eosin (H&E). The results were analyzed using light microscopy and documented via photography.

Results and Discussion

The analysis of morphological changes in the right ventricle (RV) in Tetralogy of Fallot established several key findings. Group hypertrophy of the RV myocardium cardiomyocytes was predominantly identified in the anterior ventricular wall. This included branched hypertrophy; under 200x magnification, large, hyperchromic, well-defined cells in transverse orientation were observed. The count of large cardiomyocytes ranged from 220–255 per field of view (200x), which is 2.25 times higher than the control group (100–125), with cell size increasing 2.5 times.

The density of vessels between cardiomyocytes-specifically capillaries and small-caliber vessels-was 1.75 times lower than in the control group. These changes indicate that compensatory mechanisms in the right heart chambers are well-developed. Additionally, a significant amount of pale pink inclusions (glycogen) was detected in the cytoplasm of atypical cardiomyocytes (pacemaker cells) located along the perimeter of the right ventricle. Cavernous-type lymphatic vessels of varying widths were also identified interspersed among the cardiomyocytes (see Fig. 1).

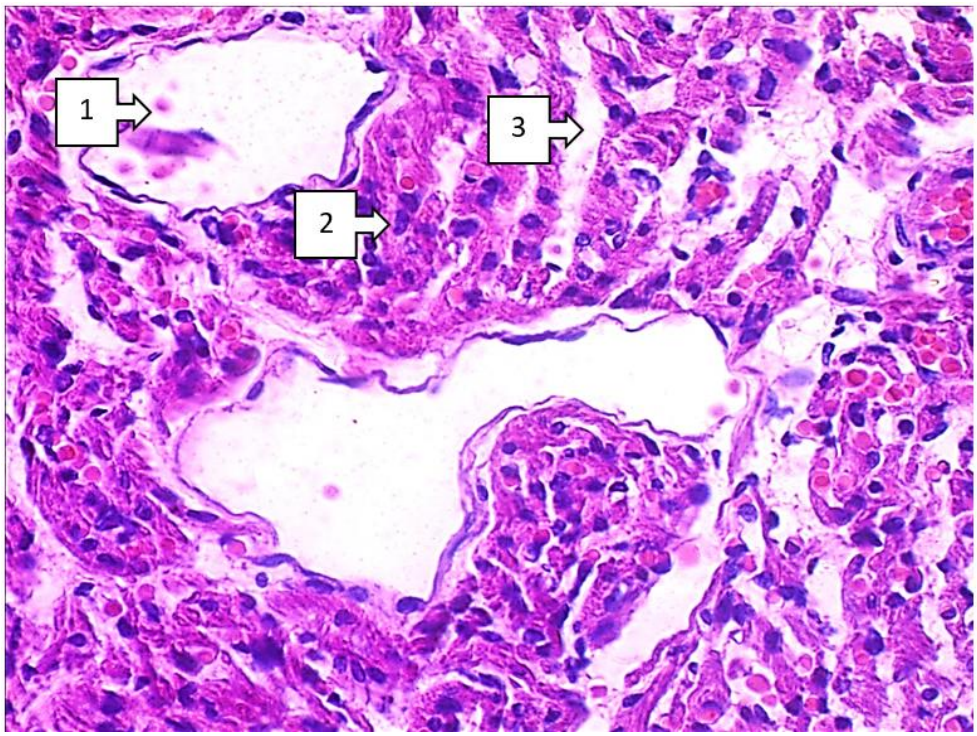

Figure No. 1. Tetralogy of Fallot. Myocardium of the anterolateral branch of the right ventricle. (1) Cavernously dilated lymphatic vessels; (2) large, hyperchromically stained cardiomyocytes; (3) irregular interstitial edema of the connective tissue. Staining: H&E, Magnification: 20x10.

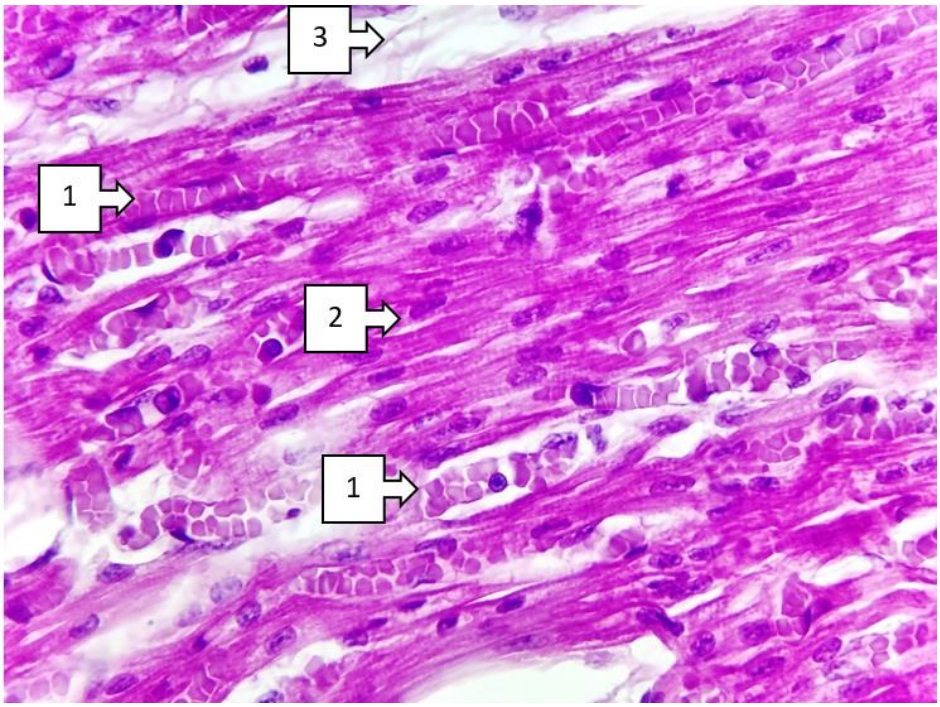

Fig. No. 2. Tetralogy of FallotLongitudinal section of the myocardium. (1) Sludge phenomenon in the majority of capillaries; (2) large hyperchromic cardiomyocytes; (3) interstitial edema. Staining: H&E, Magnification: 40x10.

In Tetralogy of Fallot, specific alterations in the histioarchitectonics of the majority of capillaries are

observed as a result of hypertrophic changes in the ventricular myocardial cardiomyocytes. This, in turn, disrupts hemodynamic laws, leading to localized circulatory impairment in the capillaries and increased permeability of the capillary walls. Simultaneously, this creates the "sludge phenomenon" within dilated capillaries. Depending on the duration of the process, these conditions trigger the activation of fibroblasts in these areas and an increase in sparse fibrous structures. Consequently, this leads to atrophic changes in bundles of cardiomyocytes that have not undergone hypertrophy and possess low functional activity. In terms of cardiac contractions, these clinical and morphological characteristics create the conditions for the development of arrhythmic contractions. Furthermore, necrobiotic processes develop in focal cardiomyocytes due to the formation of microthrombi from sludged erythrocytes within the capillaries (see Fig. 2).

Conclusion

The morphological study of Tetralogy of Fallot in infants under one year of age allows for the following conclusions The right ventricular myocardium undergoes significant structural remodeling characterized by branched and group hypertrophy of cardiomyocytes. The increase in the number of hyperchromic cardiomyocytes (up to 2.25 times compared to the control) and their size (2.5 times) serves as a key compensatory mechanism to overcome hemodynamic overload.A critical disproportion was identified between the hypertrophied muscle mass and the vascular supply, with capillary density being 1.75 times lower than normal. The presence of cavernously dilated lymphatic vessels and the "sludge phenomenon" in the capillaries indicates severe impairment of both blood and lymph microcirculation, contributing to chronic tissue hypoxia.The accumulation of glycogen in pacemaker cells and the development of interstitial edema signal a metabolic shift under hypoxic conditions. The progression of these changes leads to the activation of fibroblasts and the replacement of functional myocardium with fibrous structures, particularly affecting non-hypertrophied cardiomyocyte bundles.The identified necrobiotic processes and the formation of microthrombi within sludged capillaries provide a structural basis for the development of cardiac arrhythmias and progressive heart failure in infants with Tetralogy of Fallot.Focal endocardial thickening, subendocardial atrophy, and fatty degeneration of the right ventricular cardiomyocytes near the valves constitute a specific morphological complex that can be used for the post-mortem differential diagnosis of Tetralogy of Fallot and the assessment of its severity.

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