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Complex Single Ventricule Anomaly, Its Structural Variants, Treatment

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Abstract:

A single ventricle or univentricular heart is a broad term covering various cardiac structural abnormalities in which one ventricle is severely underdeveloped, or a ventricular septal wall did not form. Through various mechanisms, the anomalous structure typically results in the mixing of oxygenated and deoxygenated blood. Occurrences are generally caused by genetic factors, though environmental factors are known to promote malformation.

Keywords: oxygen-poor, embryogenesis, endocardial cushion, DiGeorge syndrome, Hypoplastic left heart syndrome (HLHS).

Introduction

Single ventricle heart defects can cause children to become cyanotic (turn a blue color), since a mixture of oxygen-poor (blue) and oxygen-rich (red) blood vessels leaves the heart and goes to the body. Just how much oxygen or how little oxygen depends on the type, location, and severity of the defect. Some children will only be mildly cyanotic, while others won't have enough oxygen in the blood to meet the body's needs and will need early treatment. Single ventricle defects are rare, affecting only about five out of 100,000 newborns. They are also one of the most complex heart problems, usually requiring at least one surgery. The origin of each condition varies by type and is influenced by multiple factors. The development process for each variation is not entirely understood.

Malformation occurs in embryogenesis, during days 30 to 56 of gestation. Cardiac structural abnormalities are commonly associated with lateralization disorders, such as situs inversus totalis and heterotaxy. In patients with primary ciliary dyskinesia (PCD), 12% of patients had evidence of heterotaxy. Other genetic causes have also been identified, including Tbx5 and GATA4; the inactivation of both genes has a direct influence on the formation of the ventricular septum. Genetic malformations without explicitly known causes have also been postulated, suggestively induced by

defects in the formation of endocardial cushions and developmental influence by dynamic blood flow. These other causes are associated with extracardiac structural anomalies, as seen in DiGeorge syndrome.

Environmental factors also influence cardiac structural formation. Risk factors include:

- Increasing parental age
- Phenylketonuria, pregestational diabetes, febrile illnesses, influenza, maternal rubella, anticonvulsants, ibuprofen, sulfasalazine, thalidomide, trimethoprim-sulfonamide, retinoids, marijuana, and organic solvents. Lithium is associated with an increased risk of cardiac malformations, specifically at higher doses and with use during the first trimester

Key univentricular variations and typical features:

- Hypoplastic left heart syndrome (HLHS): The left ventricle, mitral valve, aortic valve, and aorta are underdeveloped.
- Tricuspid atresia: The tricuspid valve fails to develop, leading to an underdeveloped right ventricle.
- Ebstein anomaly: Abnormal development of tricuspid valve leaflets causes right ventricular atrialization. The anomaly is associated with various cardiac structural abnormalities, including pulmonary valve pathologies, septal defects, and electrical conduction lesions.
- Double outlet right ventricle: The aorta and the pulmonary artery exit from the right ventricle, leaving the left ventricle underdeveloped.
- Double inlet left ventricle: Both atria connect to the left ventricle, resulting in an underdeveloped right ventricle.
- Atrioventricular canal defect: An atrial or ventricular septal defect forms large enough to make a functionally single ventricle.

With a single ventricle, mixed oxygenated blood circulates throughout the body. Depending on the structural anomaly, a patent ductus arteriosus (PDA), atrial septal defect (ASD), ventricular septal defect (VSD), or communication in the great arteries may be required to maintain pulmonary and systemic circulations. The presentation of a single ventricle may be identified as early as the 18th week of gestation. Other structural variations may also be seen near this time, including mispositioning of the great arteries and reversal of blood flow through portions of the fetal cardiac system. Ultrasound may also reveal extracardiac structural manifestations, assisting in diagnosis. Post-natal presentations vary depending on underlying structural variations. Typically, a heart murmur, tachypnea, respiratory distress, cyanosis, or hypotension may be present when circulation and oxygenation are not satisfactory. Other physical exam findings, such as hepatomegaly or dysmorphic features, may be clues to more underlying abnormalities. A neonate may not present symptoms at birth or before discharge if circulation is adequate at the time of examination. Closure of the patent ductus arteriosus (PDA) or changes in flow to end organs may precipitate these symptoms after discharge.

Treatment options of the univentricular variants depend on the time of discovery, prognosis, and goals of care. Prognosis must be discussed in depth before proceeding with treatment, as an intervention may be futile in some instances. Univentricular heart syndrome medical management is targeted to the underlying pathology. Supplemental oxygen will help alleviate hypoxemia, and acid-base or metabolic disturbances should have correctable factors mended. Inhaled nitric oxide is beneficial to reduce resistance in the pulmonary vasculature, allowing more blood to be oxygenated by the lungs. In strained hearts, inotropes may assist contraction force, but catecholamines should be avoided due to arrhythmogenesis When a patent ductus arteriosus (PDA) is needed to maintain

collateral flow, prostaglandin E1 prolongs the opening of the ductus arteriosus, providing a bridge to permanent interventions Non-steroidal anti-inflammatory medications should be avoided to maintain a patent ductus arteriosus. Catheter-based management is also based on underlying etiology, time of discovery, and prognosis. When discovered in utero, catheter-based structural interventions and valvuloplasty can mitigate sequala by correcting anomalies during the developmental process. Many interventions can also be performed after delivery, though further development is less influenced. In Ebstein anomaly, associated pulmonary arteriovenous malformations can be occluded using a transcatheter approach. Surgical intervention can also correct any of the abovementioned anatomical variations, though technique varies by condition. The Fontan procedure, a popular choice for intervention, works by delivering blood to the lungs utilizing central venous pressure, and reduced intrathoracic pressure. Optimal pressure dynamics with low pulmonary flow resistance allows for anterograde circulation. Though reliably successful, the Fontan procedure should not be the only option considered. In patients with severe disease, palliative surgery may be the best option and is typically preferred over choosing strict comfort measures. Data is unclear if the Fontan procedure is superior to palliative options Patients should be referred to tertiary care centers for specialized evaluation and treatment. Cardiac transplantation may be considered, albeit associated with suboptimal outcomes due to comorbidities. Cardiac transplantation may also be required despite previous alternative procedures.

All in all, complex single ventricle is a serious problem and without surgery, most children would not be able to survive the first year of life. Surgery involves a staged approach done in either two or three steps, depending on the degree of pulmonary blood flow.

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