Congenital Heart Disease in the Newborn and Importance of Early Intervention

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Abstract

Although prenatal diagnostic techniques have improved signi fcantly, the accurate detection and appropriate treatment of newborns with congenital heart disease (CAD) has always been of great interest to pediatricians. Congenital heart malformations range from benign to serious conditions such as: Prompt diagnosis and treatment are necessary if the baby is to survive. Unfortunately, these life-threatening heart conditions may not be apparent early in life and most clinical and physical fndings are nonspeci fc and equivocal, making diagnosis di f cult. Decisions require a high suspicion index and keen insight. Many serious malformations can go unnoticed early in life when the patent ductus arteriosus (PDA) is wide open05.5@0tut can becom osevere05.5@0as he pDA)55(contracts hours05.5@0to days05.5@(I arch (IAA), TGA, TAPVR, severe aortic stenosis (AS), pulmonary artery atresia (PA), and tricuspid atresia (TA). Although individually rare, CHD is a major cause of neonatal mortality. Early intervention may reduce cardiac-related neonatal mortality from 2-3/1000 to 0.6-0.8/1000 [1-6] However, other relevant important factors such as a combination of congenital anomalies, low birth weight, prematurity, pulmonary problems, persistent pulmonary hypertension and sepsis also inuence the overall neonatal outcome of CHD.

C . . . CHD

From a clinical perspective, CHD can be divided into three main categories.

1. L . . . CHD

Structural heart malformation with high likelihood of cardiovascular collapse and debilitating if not treated early. ese include TGA, COA/IAA, AS and HLHS/mitral atresia, PA, and obstructive TAPVR.

2. C . . . CAD

Structural cardiac defects that a ect cardiac function but are unlikely to collapse should be treated early. e most common defects in this group are ventricular septal defect (VSD), complete atrioventricular septal defect (AVSD), atrial septal defect (ASD), and tetralogy of Fallot with good pulmonary artery anatomy disease (TOF).

3. C . . . CHD

An anatomically de ned cardiac malformation but not

includes HLHS and its variants, severe AS, severe COA, and IAA and its variants [7]. ey require ductal patency to maintain blood ow throughout the body or even just the underside of the body. As a result, blood ow is reduced, leg pulses become weak and undistinguishable, and oliguria due to renal dysfunction develops over time.

Another type is duct-dependent pulmonary circulation (also known as right-sided obstructive lesion) with signi cant TOF, PA and its variants, signi cant PS, TA with PS/PA (with/without VSD). , including single-ventricular hearts with PS/PA, and severe forms of Ebstein's anomaly). TGA with an intact interventricular septum (TGA/IVS) acts as a conduit-dependent lesion, whereas large ASD is more important for circulatory admixture). Most of these CHDs show progressive cyanosis that does not respond to adequate oxygenation. Because fetal physiology is chronically adapted to intrauterine hypoxia, newborns can tolerate a degree of cyanosis better than older infants and children). e versatility of CHD is immense due to the large number of combinations of defects that can a ect di erent levels of the heart (atrial, ventricular, septal, venous, or aortic). Another type is duct-dependent pulmonary circulation (also known as right-sided obstructive lesion) with signi cant TOF, PA and its variants, signi cant PS, TA with PS/PA (with/without VSD). , including single-ventricular hearts with PS/PA, and severe forms of Ebstein's anomaly). TGA with an intact interventricular septum (TGA/IVS) acts as a conduit-dependent lesion, whereas large ASD is more important for circulatory admixture) [8-9]. Most of these CHDs show progressive cyanosis that does not respond to adequate oxygenation. Because fetal physiology is chronically adapted to intrauterine hypoxia, newborns can tolerate a degree of cyanosis better than older infants and children). e versatility of CHD is immense due to the large number of combinations of defects that can a ect di erent levels of the heart (atrial, ventricular, septal, venous, or aortic).

Cyanotic CHD categories can be divided into reduced pulmonary blood ow with right-to-le shunt lesions (PA, TA with shunts at the atrial or ventricular level). Poorly mixed lesions (transposition physiology); and right-to-le shunts with mixed intracardiac lesions (TAPVR, single-ventricular physiology, truncus arterioles Some coronary artery disease develops during the fetal period because the growth of heart structures depends on blood ow. erefore, fetuses with mild le -sided obstructive lesions may progress to stenosis/HLHS over time. Similarly, pulmonary atresia with an intact interventricular septum is considered a late phenomenon that begins as severe pulmonary artery stenosis. PPHN is another serious condition associated with other high-risk neonatal factors5) that is di cult to di erentiate from the cyanotic heart disease mentioned above.

During the fetal period, oxygen and nutrient transport occurs through the placenta, which receives approximately 40% of the total fetal cardiac output). It returns to the rightery

suspect congenital heart malformations for which early intervention is important. CAD should be suspected if the patient is in shock or shows cyanosis despite adequate treatment. Early detection and immediate intervention for these critically ill patients is the only way to save lives. Many of these babies have to be transferred to tertiary centers. Appropriate course of action for suspected infants should be discussed early with the pediatric cardiology center to avoid pursuing other less likely diagnoses. Treating such conditions may worsen the patient's clinical condition before and during transport. If the fetus has the above CHD on prenatal echocardiography or if serious postnatal sequelae are expected, planned delivery at a tertiary center where treatment can be provided is recommended.

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