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Congenital Heart Disease in the Newborn and Importance of Early Intervention

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Abstract

Although prenatal diagnostic techniques have improved significantly, 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 findings are nonspecific and equivocal, making diagnosis difficult. 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 open, but can become severe as the PDA contracts hours to days later. It can lead to acidosis/shock/cyanosis or even death. Conduit-dependent congenital heart lesions, which can be classified as conduit-dependent systemic or pulmonary diseases, are physiologically very different from each other and therapeutic strategies should be tailored to the clinical condition and cardiac malformation. Unavoidable early onset is often considered a medical emergency. Differential diagnoses include congenital metabolic disorders, neonatal sepsis, neonatal persistent pulmonary hypertension (PPHN), and other pulmonary diseases. Urgent identification of these at-risk neonates requires timely referral to a pediatric cardiologist, and timely intervention is key to reducing mortality and morbidity. The overview below describes the clinical manifestations, assessment, and treatment of congenital heart defects that manifest early in life.

Keywords: Congenital heart disease; Early intervention

Introduction

Although efforts have been made to detect significant CAD during the prenatal period or shortly after birth, the majority of neonates with cardiac defects remain undetected until severe symptoms develop.

Because infants with these life-threatening heart defects may be asymptomatic at first or have unclear clinical signs, most of the time, routine physical examination will detect a serious condition. Birth is a major event from the fetus to the postpartum cycle. The most important changes are from the underwater amniotic fluid environment and placental gas exchange to respiration and pulmonary ventilation. Air breathing implies a sharp drop in pulmonary vascular resistance and a marked increase in pulmonary blood flow. Fetal structures critical to fetal blood flow, such as the foramen ovale, ductus venous, and ductus arteriosus [1-2], are no longer required for survival and begin to close. Neonates with CHD associated with duct-dependent pulmonary or systemic perfusion or with mixed physiology such as TGA, do not make a proper transition and are at increased risk of disability and collapse.

Incidence

The incidence of CHD is estimated at 6-8 per 1,000 live births in the general population [3]. Infant mortality from CHD is about 3%4). Not all her CHD is detectable before or at the time of death. Critical cardiac defects with high premature mortality if not treated immediately after birth include HLHS, aortic stenosis (COA)/interrupted aortic 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 [4-6]. However, other relevant important factors such as a combination of congenital anomalies, low birth weight, prematurity, pulmonary problems, persistent pulmonary hypertension and sepsis also influence the overall neonatal outcome of CHD.

Classification of CHD

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

1. Life-threatening CHD

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

2. Clinically relevant CAD

Structural cardiac defects that affect cardiac function but are unlikely to collapse should be treated early. The 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. Clinically insignificant CHD

An anatomically defined cardiac malformation but not functionally or clinically significant. These include small VSD, atrial septal defect (ASD), and mild pulmonary stenosis (PS) detectable only by echocardiography and requiring no treatment. There are two types of conduit-dependent cardiac lesions. As shown in Table 1, duct-dependent body circulation (also called left-sided obstructive lesion)

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includes HLHS and its variants, severe AS, severe COA, and IAA and its variants [7]. They require ductal patency to maintain blood flow throughout the body or even just the underside of the body. As a result, blood flow 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 significant TOF, PA and its variants, significant 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). The versatility of CHD is immense due to the large number of combinations of defects that can affect different 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 significant TOF, PA and its variants, significant 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). The versatility of CHD is immense due to the large number of combinations of defects that can affect different levels of the heart (atrial, ventricular, septal, venous, or aortic).

Cyanotic CHD categories can be divided into reduced pulmonary blood flow with right-to-left shunt lesions (PA, TA with shunts at the atrial or ventricular level). Poorly mixed lesions (transposition physiology); and right-to-left 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 flow. Therefore, fetuses with mild left-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 difficult to differentiate from the cyanotic heart disease mentioned above.

Physiological change of the heart after birth

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 right atrium through the umbilical vein and fetal venous tract. The lungs are incapable of supplying oxygen and receive only about 7% of the fetal cardiac output. The rest of the right ventricular output is diverted to the descending aorta via the ductus arteriosus. That is, it is a one-way "left-right shunt". In addition, blood flows "right to left" through the foramen ovale on the atrial plane. These two primary communications allow the two sides of the heart to be related and maintain a wide variety of cardiac abnormalities that have been found to be critical to postnatal life. But the biggest change after birth is that the placenta is no longer responsible for circulation and both lungs are responsible for oxygenation during the first few breaths.

When umbilical vein flow is abruptly interrupted and regurgitation from the lungs into the left atrium is markedly increased, the valve-like fossa ovalis virtually closes, and the prostaglandin-dependent ductus arteriosus also closes to the ligaments of the arteries, leading to left and right venous flow. The heart remains functional. Independence. If the infant has significant persistent cyanosis and acidosis, ductal closure may be delayed.

Clinical manifestations

Clinical signs of significant neonatal heart disease can be ambiguous. A heart murmur usually does not help in this situation. For pediatricians to identify 'doing bad' neonates, confirm high indicators of suspicion, recognize the need for prompt cardiac evaluation, and rule out serious congenital heart problems that require early intervention it is very important to These include persistent central cyanosis, acidosis of unknown origin, and tachypnea without pulmonary problems. Initial evaluation includes saturation monitoring, perfusion status (blood gas analysis), and assessment of blood pressure in all extremities. [10,11] Various vascular-dependent congenital heart malformations requiring urgent prostaglandin infusion. Some neonates with significant congenital heart disease may present clinically with epidural obstruction hours to days after birth. At this point the baby showed severe acidosis/cyanosis/shock and even sudden death.

1. Difficulty breathing

Persistent tachypnea or dyspnea may indicate lung or heart problems. Large shunt lesions manifest as dyspnea, tachypnea, feeding difficulties, irritability, and stress. Weaning from a ventilator can be difficult in preterm infants with large left-to-right shunts. Cyanosis with severely reduced pulmonary blood flow usually causes 'silent tachypnea' without noticeable shortness of breath.

2. Signs of Poor Blood Circulation

As described above, neonates with duct-dependent systemic circulatory impairment have progressive dyspnea, coldness, poor perfusion and sticky mottled skin suggestive of acidosis, shock, oliguria due to end organ damage, and terminal dyspnea. Eventually, it develops progressive cardiovascular collapse upon duct closure 2,5,9). A significant murmur on chest radiograph and S3 canter with or without cardiac hypertrophy may support these findings.

3. Cyanosis

Desaturated oxygen in newborns is more often due to lung problems than heart problems. Persistent hypoxia intolerable to 100% oxygenation suggests cyanotic CAD rather than pulmonary problems. Easily perform hyperoxia testing with arterial blood gas analysis while delivering 100% oxygen at the bedside. An increase in PO2 above 220 mmHg indicates respiratory disease. 100-220 mm Hg requires examination for cyanosis CAD. Less than 100 mm Hg indicates cyanotic CHD, and marked cyanosis less than 40-50 mm Hg likely indicates a bad mixed disease such as TGA 5). A positive or borderline hyperoxia test warrants further evaluation for cyanotic CAD by a pediatric cardiologist. Hyperoxia testing is not always helpful in distinguishing between coexisting cardiac and pulmonary problems or PPHN with right-to-left shunting at both the atrial and ductus arteriosus levels.

Conclusion

In this era of vastly improved prenatal and postnatal diagnostics, it remains challenging for pediatricians to accurately identify symptomatic neonates suspected of CAD. Pediatricians should always

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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