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CARDIOVASCULAR DISORDERS CAUSED BY MALNUTRITION IN CHILDREN

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Abstract. *Congenital heart disease is the most common congenital anomaly, occurring in almost 1% of live births (1). Among birth defects, congenital heart disease is the leading cause of infant mortality. The most common congenital heart defects diagnosed in infancy are muscular and perimembranous ventricular septal defects, followed by secundum atrial septal defects, with an overall prevalence of 48.4 per 10,000 live births (2, 3, 4). The most common "blue" congenital heart defect is tetralogy of Fallot, which is twice as common as transposition of the great arteries (4.7 versus 2.3 per 10,000 births). Overall, bicuspid aortic valve is the most common congenital heart defect, with a prevalence of 0.5–2.0%.*

Keywords: *Pathophysiology, Clinical manifestations, Diagnostics, Neonatal screening.*

СЕРДЕЧНО-СОСУДИСТЫЕ ЗАБОЛЕВАНИЯ, ВЫЗВАННЫЕ НЕДОЕДАНИЕМ У ДЕТЕЙ

Аннотация. *Врожденный порок сердца является наиболее распространенной врожденной аномалией, встречающейся почти у 1% живорожденных (1). Среди врожденных пороков врожденный порок сердца является основной причиной детской смертности. Наиболее распространенными врожденными пороками сердца, диагностируемыми в младенчестве, являются мышечные и перимембранозные дефекты межжелудочковой перегородки, за которыми следуют вторичные дефекты межпредсердной перегородки, с общей распространенностью 48,4 на 10 000 живорожденных (2, 3, 4). Наиболее распространенным «синим» врожденным пороком сердца является тетрада Фалло, которая встречается в два раза чаще, чем транспозиция магистральных артерий (4,7 против 2,3 на 10 000 родов). В целом, двустворчатый аортальный клапан является наиболее распространенным врожденным пороком сердца с распространенностью 0,5–2,0%.*

Ключевые слова: *Патофизиология, Клинические проявления, Диагностика, Неонатальный скрининг.*

Introduction

Common environmental factors include maternal disease (e.g., diabetes mellitus, rubella, systemic lupus erythematosus) or maternal use of teratogenic agents (e.g., lithium, isotretinoin, anticonvulsants). Maternal age is a known risk factor for certain genetic disorders, particularly Down syndrome, in which the fetus may have heart defects. It is not clear whether maternal age is an independent risk factor for congenital heart defects. Paternal age may also be a risk factor (1).

Some chromosomal abnormalities (aneuploidies), such as trisomy 21 (Down syndrome), trisomy 18 (Trysomy 13), and monosomy X (Turner syndrome), are strongly associated with congenital heart disease. However, some of these abnormalities account for only 5-6% of patients with congenital heart disease. Many other conditions involve subchromosomal deletions (microdeletions), subchromosomal duplications, or single gene mutations. These mutations often cause congenital syndromes that affect multiple organs in addition to the heart. Examples include DiGeorge syndrome (microdeletion at 22q11.2) and Williams syndrome (sometimes called Williams-Buren syndrome) (microdeletion at 7p11.23). Single gene defects that cause syndromes associated with congenital heart disease include mutations in the fibrillin-1 gene (Marfan syndrome), TXB5 (Holt-Oram syndrome), and PTPN11 (Noonan syndrome). Defects in a single gene can also cause isolated (i.e., nonsyndromic) congenital heart defects. In approximately 72% of patients with congenital heart disease, no genetic etiology is identified (2 , 3 , 4). The risk of recurrence of congenital heart disease in a family depends on the cause of the disease. The risk for new mutations is insignificant, ranging from 2% to 5% for nonsyndromic multifactorial congenital heart disease and 50% if the cause is an autosomal dominant mutation. Family screening for bicuspid aortic valve in an individual requires screening in a family with a prevalence of 9% (5). Identification of genetic factors is important because most patients with congenital heart disease survive to adulthood and potentially start a family.

Normal fetal circulation

The fetal circulation is characterized by:

The passage of blood around the non-ventilated lung from right to left through the patent ductus arteriosus (connecting the pulmonary artery to the aorta) and the foramen ovale (connecting the right and left atria). Shunting is supported by the high resistance of the pulmonary arterioles and the low resistance to blood flow in the systemic circulation (including the placenta). Approximately 90–95% of the right ventricular output bypasses the lungs and enters the systemic circulation directly. The ductus arteriosus of the fetus is maintained open by the low systemic fetal

PaO2 (approximately 25 mm Hg) and locally produced prostaglandins. The foramen ovale is maintained open by pressure differences in the atria: the pressure in the left atrium is relatively low because of the small amount of blood returning from the lungs, and the pressure in the right atrium is relatively high because of the large volume of blood returning from the placenta.

Normal fetal circulation

In the fetus, the blood entering the right side of the heart is already saturated with oxygen through the placenta. Since the lungs are not ventilated, only a small amount of blood passes through the pulmonary artery. Most of the blood from the right side of the heart bypasses the lungs.

a. Oval mirror
b. Ductus arteriosus
c. Typically, these two structures close naturally soon after birth.
d. Normal fetal circulation
e. Changes in the perinatal period
f. Extensive changes in this system occur after the first breath, resulting in
g. Increased pulmonary blood flow
h. Functional closure of the foramen ovale

Pulmonary arteriolar resistance decreases dramatically due to vasodilation caused by lung expansion, increased PaO2, and decreased PaCO2. Elastic forces of the ribs and chest wall decrease pulmonary interstitial pressure, further increasing blood flow through the pulmonary capillaries. Increased venous return from the lungs increases left atrial pressure, which reduces the pressure difference between the left and right atria; this effect contributes to functional closure of the foramen ovale. Once pulmonary blood flow is established, venous return from the lungs increases, and left atrial pressure increases. Inhaled air increases PaO2, which causes the umbilical arteries to constrict. Placental blood flow is reduced or stopped, reducing arterial return to the right atrium. Thus, the pressure in the right atrium decreases, while the pressure in the left atrium increases. As a result, these 2 embryonic components of the interatrial septum (septum primum and septum secundum) are simultaneously compressed, which prevents flow through the foramen ovale. In most individuals, these 2 membranes eventually fuse, and the foramen ovale disappears.

However, in 25% of adults, the foramen ovale may remain patent with minimal or no residual shunting (1). Shortly after birth, systemic resistance becomes higher than pulmonary resistance, completely changing from the fetal state. Thus, the direction of blood flow through the ductus arteriosus is reversed, forming a left-to-right shunt of blood (the so-called transitional

circulation). This state lasts from birth (when blood flow in the lungs increases and functional closure of the foramen ovale occurs) until about 24-72 hours later, when the ductus arteriosus narrows. The blood entering the ductus from the aorta and its veins has a high PO2, which, together with changes in prostaglandin metabolism, leads to narrowing and closure of the ductus arteriosus. As soon as the ductus closes, the adult type of circulation is formed. At the same time, the two ventricles are constantly contracting, and there is no significant connection between the pulmonary and systemic circulation.

If there is stress immediately after birth, the newborn may revert to fetal circulation. Asphyxia with hypoxia and hypercapnia leads to constriction of the pulmonary arterioles and dilation of the ductus arteriosus, which leads to the reversal of the processes described above and the restoration of right-to-left shunting of blood through the newly opened ductus arteriosus and/or the foramen magnum. Consequently, the newborn is severely hypoxemic, a condition called persistent pulmonary hypertension or persistent fetal circulation (although umbilical circulation is not present). The goal of treatment is to eliminate the conditions that cause pulmonary vasoconstriction.

Pathophysiology of congenital heart defects

Congenital heart defects are classified as follows (see table: Classification of Congenital Heart Defects):

1. Cyanotic
2. Asiatic (left-to-right shunts or obstructive lesions)

The physiological consequences of congenital heart anomalies are very diverse, ranging from a heart murmur or pulse discrepancy in an asymptomatic child to severe cyanosis, heart failure, or vascular insufficiency.

Cyanotic heart anomalies

Various amounts of deoxygenated venous blood are shunted to the left side of the heart (right to left), which reduces systemic arterial oxygen saturation.

Cyanosis develops if > 5 g/dL (>50 g/L) of deoxygenated hemoglobin is present.

Complications of chronic cyanosis include polycythemia, thromboembolism (including stroke), bleeding disorders, brain abscess, and hyperuricemia. Hypercyanotic seizures may occur in infants with untreated tetralogy of Fallot or other complex congenital malformations involving dynamic stenosis of the subpulmonary valve and ventricular defects.

Depending on the abnormality, pulmonary blood flow may be reduced, normal, or increased (often resulting in heart failure in addition to cyanosis), resulting in varying degrees of cyanosis. Heart murmurs with variable sound are nonspecific.

Left to right shunts

Oxygenated blood from the left side of the heart (left atrium or left ventricle), or the aorta, is sent to the right side of the heart (right atrium or right ventricle), or the pulmonary artery, through a hole or junction between the 2 parts.

Immediately after birth, pulmonary vascular resistance is high, and flow through this connection may be minimal or bidirectional. During the first 24 to 48 hours of life, pulmonary vascular resistance gradually decreases, with increased left-to-right blood flow. The additional blood flow to the right side increases pulmonary blood flow and pulmonary artery pressure to varying degrees. The greater the increase, the more severe the symptoms; a small right-to-left shunt usually causes no symptoms or signs.

High-pressure anastomoses (in the ventricles or large arteries) become apparent within days to weeks; low-pressure shunts (atrial septal defects) become apparent much later. If left untreated, increased pulmonary blood flow and pulmonary artery pressure can lead to pulmonary vascular disease and ultimately Eisenmenger syndrome. Large left-to-right shunts (eg, large ventricular septal defect [VSD], patent ductus arteriosus [PDA]) cause excessive pulmonary blood flow and left ventricular volume overload, which can lead to the development of signs of heart failure and often results in heart failure in infancy. Large left-to-right shunts also reduce lung compliance and increase airway resistance. Together, these factors increase the likelihood of infants being hospitalized with respiratory syncytial virus or other upper or lower respiratory tract infections.

Obstructive lesions

Blood flow is obstructed, causing a pressure gradient to develop across the obstruction.

Ventricular hypertrophy and heart failure may develop as a result of excessive pressure load proximal to the obstruction. The most obvious manifestation is a heart murmur caused by turbulent flow through the obstruction (stenosis). For example, congenital aortic stenosis, which accounts for 3–6% of congenital heart defects, and congenital pulmonary stenosis, which accounts for 8–12% (1 , 2).

Heart failure

Some congenital heart defects (e.g., bicuspid aortic valve, mild aortic stenosis) do not significantly alter hemodynamics. Other defects cause pressure or volume overload, sometimes leading to heart failure. Heart failure develops when cardiac output is insufficient to meet the metabolic needs of the body or when the heart is unable to adequately maintain venous return, with pulmonary edema (in left ventricular failure), edema primarily in the tissues and organs of the abdominal cavity (in right ventricular failure), or both. There are many causes of heart failure in infants and children other than congenital heart defects (see table: " Common Causes of Heart Failure in Children "). Abstract: Congenital heart disease is the most common congenital anomaly, occurring in almost 1% of live births (1). Among birth defects, congenital heart disease is the leading cause of infant mortality.

The most common congenital heart defects diagnosed in infancy are muscular and perimembranous ventricular septal defects, followed by secundum atrial septal defects, with an overall prevalence of 48.4 per 10,000 live births (2, 3, 4). The most common “blue” congenital heart defect is tetralogy of Fallot, which is twice as common as transposition of the great arteries (4.7 versus 2.3 per 10,000 births). Overall, bicuspid aortic valve is the most common congenital heart defect, with a prevalence of 0.5–2.0%.

Common causes of heart failure in children

Congenital heart defect related to the ductus arteriosus

Patent ductus arteriosus is the normal connection between the pulmonary artery and the aorta; it is essential for proper fetal circulation. At birth, an increase in PaO2 and a decrease in prostaglandin concentrations lead to closure of the ductus arteriosus, which usually begins in the first 10-15 hours of life. Some congenital heart defects depend on the ductus arteriosus to remain open to maintain systemic blood flow (e.g., hypoplastic left heart syndrome, critical aortic stenosis, coarctation of the aorta) or pulmonary blood flow (cyanotic lesions such as pulmonary atresia or severe tetralogy of Fallot). Thus, with these defects, it is essential to maintain the ductus arteriosus in a patent state until definitive repair (usually by surgery) is achieved using intravenous administration of exogenous prostaglandins. Other abnormalities on physical examination include circulatory shock, circulatory collapse, abnormal 2nd heart sound (single or sharply divided S2), systolic click, galloping rhythm, or an abnormally slow, fast, or irregular rhythm.

Noises

Left-to-right shunts and obstructive lesions often produce a systolic murmur. The systolic murmur and chest wall vibration are close to their origin on the body surface, making this location

diagnostically useful. Increased flow through the pulmonary or aortic valve produces a mesosystolic murmur (systolic ejection). Regurgitant flow through the ventricular valve or through a ventricular septal defect produces a holosystolic (pansystolic) murmur that, as its intensity increases, obscures the first heart sound (S1). Patent ductus arteriosus usually produces a continuous murmur that is not interrupted by S2 as blood flows through the ductus during systole and diastole. This murmur is 2-tone and has a sharper sound during systole (induced by high pressure) than during diastole.

Cyanosis

Thickening of the terminal phalanges of the fingers

Central cyanosis is characterized by a bluish tinge to the lips and tongue and/or nail beds; it occurs when the deoxygenated hemoglobin level is increased (at least 5 g/dL [50 g/dL]) and indicates low oxygen levels (usually oxygen saturation <85%). Perioral cyanosis and acrocyanosis (bluish discoloration of the hands and feet), without cyanosis of the lips or nail beds, are caused by peripheral vasoconstriction rather than hypoxemia and are common, normal findings in newborns. Older children with prolonged cyanosis often develop the drumstick sign.

Heart failure

1. Signs and symptoms of heart failure in infants include:
2. Tachycardia
3. Tachypnea
4. Shortness of breath during feeding
5. Diaphoresis, especially while eating
6. Anxiety, nervousness
7. Hepatomegaly
8. Poor weight gain

Feeding dyspnea causes malnutrition and poor growth, which may be exacerbated by the increased metabolic demands associated with HF and frequent respiratory tract infections. Unlike adults and older children, most infants do not have dilated jugular veins and associated edema, but they sometimes have significant periorbital edema.

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