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AN AWARENESS /CHALLENGES ON CONGENITAL HEART DISEASE

Shrivastav, Sonika[1*]; Yadav, Dinesh[2]; Attri, Kavita[3];Chaturvedi, Sushma[4]

[1]SGT University, Department of Pharmacology, SGTCOP, Gurugram, Haryana- 122505, India
[2]SGT University, Department of Pharmacognosy, SGTCOP, Gurugram, Haryana- 122505, India.
[3]SGT University, Department of Pharmaceutics, SGTCOP, Gurugram, Haryana- 122505, India.
[4]SGT University, Department of Pharmacology, SGTCOP, Gurugram, Haryana- 122505, India

*Sonika.bphm@gmail.com

Abstract

Congenital heart disease is a birth defect due to improper development of embryonic heart. Globally 9 out of 1000 new-born children have this defect at the time of birth. There is no root cause identified yet for CHD however this defect can occur due to heredity, maternal defect and environmental factors. This defect can be identified at the stage of 18-22 week pregnancy through standard anomaly scan if CHD is suspected than Foetal echocardiography was performed with five heart transverse planes is performed for the final identification of the defect. CHD is classified on the basis of shunts, is the unusual blood flow from one side of heart to another side. There are 6 types of CHDs which includes Left to right shunts (Atrial level: ASD, TAPVC, Ventricular level: VSD and Truncus arteriosus), Right to left shunts (TOF and TGA physiology), Left heart obstructive lesions (Obstructed veins, Mitral stenosis, Aortic stenosis, Coarctation, Interrupted aortic arch and Hypoplastic left heart syndrome), Right heart obstructive lesions (Pulmonary stenosis / atresia, Tricuspid stenosis and Hypoplastic right heart), Single ventricles and others (Vascular rings, Venous anomalies and Arteriovenous fistulae). There is no medication available which can prevent CHD 100% however pregnant ladies should stick to a balanced diet and healthy lifestyle, daily exercise is recommended.

Key words: Congenital heart disease, Foetal echocardiography, Truncus arterios

Introduction

Congenital heart diseases (CHD) are the most commonly found congenital defects, and account for more deaths in the first year of life than any other medical condition.[1] In Latin language Congenital means Con = together, Genital = Born, so congenital disease means disease which is present at the time of the birth . A congenital heart defect (CHD) is an abnormal changes in the anatomy and physiology of the heart. CHD defects can happen in heart walls, valves, arteries and veins.[1] These defects have a negative impact on blood flow in the heart. When blood flow is not normal it can go into wrong places, sometimes it can be blocked. 28% of congenital birth defects are CHD. The birth prevalence of CHD is reported to be 8 to 12 per 1000 live births. Considering a rate of 9 per 1000 live births, If we take these statics around 1.35 million children are born with CHD each year world-wide. Around 20 to 30% of these heart defects are severe, defined as being life threatening and requiring surgery and intensive care within the first year of birth.

With rapid advances in diagnosis and treatment of CHD, vast majority of children born with CHD in developed countries reach adulthood. However, this is not the case for children born in developing countries as such advanced medicinal facilities are not available for all children.[2]

Etiology

In the process of heart development that initiates at 15th day of embryonic development. It consists of an organized series of morphologic development that involve five major steps: (1) In the first step pre-cardiac cells are migrated from the primitive streak and at the myocardial plate paired cardiac crescents are assembled, (2) In second step the primitive heart tube has been formed by coalescence of the cardiac crescents which subsequently establish the heart, (3) In the third step cardiac looping happens which assures the proper alignment of cardiac chambers, (4) In the fourth step septa and heart chamber formation occurs, (5) In fifth step cardiac conduction system and coronary vasculature development happens. This is a complex process, each part of heart has to be developed at the right time an according to the cascade of genes and gene products.

CHD is usually caused by improper development of embryonic structure, or a failure of the structure to develop at the time of early embryonic or foetal stage. Usually structural and functional development influences the anatomical defect. Although depictions of unusual heart development in foetuses and children have remained indistinctly defined, substantial knowledge about the etiology of CHD have been made during the last decade. Some deformity might be straight forwardly acquired through gene transfer, hereditary turmoil, or be related with the results of an environmental toxin or diet. Alternatively, arbitrary mistakes in cell movement prompting ill-advised heart advancement are possible. Together, the discoveries stress the unpredictable and multifactorial reasons for the CHD where extra research stay required.

Better understanding for the etiology and hazard variables of CHD is significant, and will help prepare for appropriate safeguard measures and treatment rules by doctors just as general wellbeing officials. The following represents all revealed potential reasons for CHD to date.[3-5]

1. Genetic disorder

The human genome, which contains around 20,000 to 25,000 genes, is involved coding and non-coding regions that are basic for appropriate protein structure and expression. The coding DNA sequence determines the amino acid sequence and in this way the protein structure, and structure determines function. The non-coding sequences may contain promoters and regulation of transcription. Usually, DNA sequences remain unchanged during vertical

genetic transfer to the offspring. Abnormal protein folding may cause an inappropriate development of many organs, including the heart. So genetics is one of the major cause of cardiovascular malformation, without a doubt the hereditary issue speak to the most widely recognized reason for CHD.[3,6] Deformities in chromosomes related with CHD are differing; a few examples are aneuploidy or polyploidy, inappropriate rearrangement during mitosis and meiosis, translocation, inversion or deletions. Significantly, certain chromosomes were accounted for to have a more noteworthy level of criticalness and of rates to heart improvement, and subsequently similar imperfections in various chromosomes may not result in similar defects.[7]

2. Maternal factors

Different teratogenic agents considered as the etiologic agents of CHD. For example, pregnant ladies who have insulin-dependent diabetes mellitus, and the individuals who take certain medicine for example, skin inflammation and epilepsy medicine, have a higher hazard for having babies with CHD. Ladies with drugs or alcohol addicted additionally have inclining danger. A few investigations propose that drinking liquor or utilizing cocaine, particularly during the pregnancy, can expand the danger of congenital heart defects. The basic biological principle mechanism of teratogens action that causes CHD include susceptible stage of organogenesis development, genetic differences in susceptibility, dose response relationships, and specific actions of the teratogenic agent. The most elevated level of embryonic and foetal affectability or vulnerability to unfavourable impacts of exposure to teratogens happens during the first trimester.

Intense maternal worry during the periconceptional period was related with expanded danger of conveying babies with certain inherent irregularities especially with conotruncal heart imperfections and neural tube defects.[8-11]

3 Environment or lifestyle factors

Proof of teratogenic contamination in specific conditions and work environments is sporadic, but environmental factors are an increasingly more common reason for multifactorial inheritance CHD.

Accessible proof recommends the finding of the higher frequency of CHD babies from ladies who dwell in zone with drinking water contaminated by trichloroethylene, dichloroethylene and chromium. While maternal exposure to paint, enamel, rural synthetic compounds, agriculture solvents, dyes and lead have occasionally found factually connected with CHD. Ingestion of heavy metals and lifetime aggregation of a lot of heavy metals through eating regimen likewise influences CHD advancement in infants[12,13].Exposure of herbicides and rodenticides was related with an expanded danger of TGA(transposition of great arteries), while potential introduction to pesticides was related with TAPVR (total/partial anomalous pulmonary venous return) and VSD (ventricular septal defect) Ambient air contamination such carbon monoxide (CO), nitric oxide (NO),ozone (O_3), and sulfur dioxide (SO_2) may cause CHD subject to poison levels. [14,15]

Diagnosis of CHD during pregnancy

Pre-birth finding of CHD is significant for both foetal prognosis and diminishing financial weight of family and society. In the Netherlands in January 2007 an across the nationwide screening programme, with the intend to distinguish congenital anomalies, was presented. Before 2007 foetal ultrasound was only performed for the assessment of obstetric complication during pregnancy or in pregnancies with an expanded danger of foetal abnormalities from the norm (first- or second-degree relative with CHD or other congenital anomalies).

The present screening methodology in most western nations is a standard anomaly. The standard anomaly scan is performed somewhere in the range of 18 to 22 weeks gestational age as indicated by a national convention. The assessment of the foetal heart abnormality scan involved the evaluation of the four-chamber view (size of the heart and position in thorax, symmetry of the atria and ventricles, identification of atrioventricular valves and crux) and the right and left outflow tract views. [16,17]

The three-vessel view portrays the spatial relationship of the aorta and pulmonary artery and is helpful in the location of outflow abnormalities. On the off chance that a congenital (heart) imperfection is suspected, the lady is sent to one of the tertiary centres. In the tertiary centres focuses foetal echocardiography is performed by a specific perinatologist in a joint effort with a paediatric cardiologist.[16,18]

Foetal heart examination was performed with the lady in supine position. Foetal echocardiography was performed with five heart transverse planes.[19,20] The improved and streamlined five transverse planes were as per the following: the first and most caudal plane is a transverse perspective on the upper abdomen area: moving cephalad. The following is the traditionally four-chamber view. The third is the plane regularly named the five-chamber view, in which the aortic root is pictured. The fourth transverse view uncovers the bifurcation of the pulmonary arteries. The fifth is the three vessels and trachea plane to uncover the principle pulmonary trunk in direct communication with the ductus arteriosus.[19,21]
Colour Doppler is another method to identify normal anatomy defects, septal deformities, and irregular flow of blood stream, related and other associated heart defect, valvular stenosis, coarctation, and hemodynamic compromise such as regurgitation and poor contractility.[22]

Types of congenital heart disease

In a normal cardiac anatomy, heart having a complete septation of oxygenated and deoxygenated blood. Both the circulations run parallelly, each feeding the other, and maintain a 1-to-1 volume relationship on the pulmonary and systemic sides of the circulation. The deoxygenated blood returns to the right atrium (RA) and then pumped to the lungs as the pulmonary blood flow. After that it becomes oxygenated, and then returns via the pulmonary veins to the left atrium (LA) and is pumped to the aorta as the systemic blood flow or cardiac output (Qs).[23]
Shunt is a important term before understanding the different type of congenital heart disease. "shunt" alludes to an unusual association allowing blood to stream from one side of the cardiovascular circulation to the next. In left-to-right shunt a oxygenated, pulmonary venous blood directly return to the lungs rather than being pumped to the body. A right-to left shunt is permit the deoxygenated, systemic venous return to bypass the lungs and return to the body without getting to be oxygenated.[23]

LEFT TO RIGHT SHUNTS

left to right shunts is more commonly seen in the patients of congenital heart disease. In a physiological left to right shunt oxygenated blood returns back to the lungs to get re-oxygenated. This creates a overabundance in the circulation. It will increased venous return from the lungs via the pulmonary veins to the left atrium and the left ventricle (LV). This creates a volume overload on the LV and pulmonary circulation which ultimately decreased systemic cardiac output.

Atrial septal defect

An atrial septal defect is one of the type of congenital heart defect. It is is a birth defect of the heart in which there is a hole in the septum that divides both the atria of the heart. Size of hole can vary. Sometimes it may close on its own or may require surgery.

During the gestational period when baby's heart develops, several openings in the wall are present which further dividing the upper chambers of the heart (atria). These opening usually close during pregnancy or shortly after birth.

Sometime one of these openings does not close and a hole is left out, which is called an atrial septal defect. This hole increases amount of blood flow through the lungs. Over the time increase amount of blood flow may cause damage to the blood vessels in the lungs. Which results several problems in adulthood, such as high blood pressure and heart failure. Some other problems also may arrhythmias, and increased risk of stroke.

In an ASD, there is a left to right shunt in the atrium. This results in dilatation of right atrium and right ventricle with increased pulmonary venous return to the left atrium. In an ASD bi-atrial and right ventricle (RV) volume overload occurs.[24]

Both direction and magnitude of blood flow determines through the size of the defects in the atria septum and by the relative pressures, which relate to the compliances of the left and right ventricles. In large atrial septal defects, both atrial pressures are equalized, and the shunt only depends on the ratio of the ventricular compliances.[25]

Ventricular septal defect (VSD)

Same as ASD, VSDs are openings in the septum of ventricle and are classified according to their location. According to morphology ventricular septum can be divided into 2 segments, the membranous septum and the muscular septum.

The membranous septum is small and is situated at the base of the heart between the inlet and outlet segments of the muscular septum and below the right and noncoronary cusps of the aortic valve. The septal leaflet of the tricuspid valve separates the membranous septum into 2 parts, the pars atrioventricularis and the pars inter ventricularis. Tricuspid, aortic, and mitral continuity is through this central fibrous body.

The muscular septum can be divided into inlet, trabecular, and infundibular components. The inlet portion is inferio posterior to the membranous septum. It starts at the level of the atrioventricular valves and finishes at their chordal attachments apically. An inlet VSD has no muscular rim between the defect and the atrioventricular valve annulus. Defects in the inlet muscular septum are called inlet VSDs. [26]

The defects can exist in isolation, can be complicated by additional intracardiac lesions, or can be part of more complex combinations, such as tetralogy of Fallot, double outlet right ventricle, transposition, or functionally univentricular hearts.[27]

Truncus arteriosus

The TAC is an embryonic conotruncal cardiac defect which consists in the persistence of the physiologic common arterial trunk. The two great arteries arising from the base of the heart (ascending aorta and pulmonary arterial trunk) do not differentiate, so there is occurrence of a single large vessel (with single arterial valve or truncal valve) that overhangs either a single ventricle or astride the two ventricles. The malformation is highly lethal, depending on the presence or absence of a pulmonary artery and associated extracardiac anomalies.

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RIGHT TO LEFT SHUNTS

A physiological right to left shunt is when the deoxygenated blood that returns from the tissues return back to the body without getting reoxygenated.[24]

Tetralogy of Fallot (TOF)

Because of right ventricle outflow tract obstruction, right to left shunt is present around the large non-restricted ventricular septa defect. Due to insufficient quantity of pulmonary blood flow patient becomes cyanotic. As additional blood flow sources are not present, RV is larger than LV because there is a decrease in pulmonary venous return. [24]

There are 4 abnormalities that happens in TOF defect and these are related with each other.

1. Outflow tract obstruction of RV, connection of lung and RV of heart is reduced and lungs get decreased blood flow. Pulmonary valve area is mainly effected due to narrowing, as this area has thick muscle. In this condition pulmonary artery can be small which is called hypoplastic and valve may also narrowed which is called stenotic.
2. VSD (Ventricular septal defect) – VSD is a hole between right and left ventricles, these ventricles are pumping chambers, this hole occurs in the wall.
3. Overriding of major blood vessel (aorta) – Aorta is the largest blood vessel from the heart to the body, in this case aorta is situated more rightward compare to normal. Aorta's position is above the VSD (ventricular septal defect).
4. Right ventricle Hypertrophy – In this abnormality right ventricle is more muscular because of tract obstruction in right ventricle.[31]

TGA (Transposition of great arteries)

This occurs when left ventricle is connected to pulmonary artery and right ventricle to aorta.

A parallel circulation is created compare to normal circulation in heart. The oxygenated blood returns to lungs from the pulmonary venous while deoxygenated blood returns back to systematic circulation instead of right heart. Result is a complete shift of blood flow compares to the normal flow.

Patient can survive if there is a shunt present from right part of heart to left part of heart and the other way around. If this happens deoxygenated blood will flow in pulmonary circulation and oxygenated blood will flow in systemic circulation. There is no deficiency of pulmonary blood flow in transposition physiology however there is a poor mixing of blood.An effective way to treat cyanosis is to create

a septostomy so that atrial level mixing can be increased.[32]

Hypoplastic left heart syndrome (HLHS)

HLHS is also a birth defect, during pregnancy when child is growing, the left heart doesn't develop correctly. As this is congenital heart defect(CHD) it requires surgery after birth. It is considered as critical CHD.

Hypoplastic left heart syndrome affects multiple structures present in left side of heart –

- Left ventricle is not developed and small in size
- Aorta is not developed correctly or it's size is small specially the ascending portion.
- Aortic valve is not developed or little in size.
- Children with HLHS usually have atrial septal defect, In this condition there is a hole present between right and left upper chambers of the heart

Hypoplastic left heart is associated with extra cardiac defects also, The most common abnormalities associated with HLHS are genitourinary, gastrointestinal, craniofacial and two vessel umbilical cord.[32]

Aortic stenosis

In this defect heart's aortic valve is small in size. In the normal case aortic valve opens in each contraction of the heart so that blood can pass from the heart to the whole of body. This valve is critical so narrowing of the valve blocks the blood flow and heart needs to work harder so that it can pump enough blood through the body. This has to be treated otherwise this condition will be life threatening and complications like heart failure, cardiac arrest and abnormal heart rhythms.

Calcium accumulation in the aortic valve is the main cause of aortic stenosis. This deposition will increase with age and due to the deposition valve becomes stiff and narrow over the period. This initiates the inflammatory process which associates with the coronary artery disease, such as high cholesterol, diabetes etc. Congenital abnormality of the aortic valve also may cause aortic stenosis. As compared to a normal aortic valve which comprises three cusps a valve with two cusps, also known as a bicuspid valve becomes narrow easily due to the increased risk of calcium build-up. [33,34]

Hypoplastic right heart syndrome (HRHS)

Hypoplastic right heart syndrome (HRHS), in this syndrome pulmonary valves and tricuspid valve are under-developed and affect right ventricle. Right to left shunting is present and happens through inter atrial communication. Tricuspid and pulmonary atresia are connected with extreme shapes. Congenital heart defects can be present in multiple associations however there are examples of patients surviving adulthood with under-developed tracts that too without any repair of these defects.[35]

Arteriovenous fistula

If there is a direct connection between a vein and an artery, this is called as arteriovenous fistula. It can be present as a result of pathology or created surgically.

These were created initially so that it can provide access in haemodialysis. In haemodialysis waste products like creatinine, urea and excess water are removed from patient's blood. Kidney does take care of these wastes if functions normally however in case of kidney failure, haemodialysis is used to remove these. [36-37]

Prevention

As we know that accurate reason for CHD is obscure yet it might occur because of hereditary, maternal, or exposure of radiation and we can not choose

genetic mapping of a neonate but rather have pursue a basic guidelines to pregnant ladies for prevention of CHD in their infants having good diet, physical movement, lifestyle, enviorment and occupation that the parents should discuss with their primary care provider or obstetrician. Ladies of childbearing age likewise ought to acquire pre-birth care, including testing for diabetes and past rubella vaccination, they should also discuss any medication use with their obstetrician; and ought to keep away from contact with sick individuals, particularly those with rubella or influenza.

Ladies of childbearing age should take 400 micrograms of folic acid consistently beginning before pregnancy, which can lessen intrinsic heart and neural cylinder surrenders, and ought to stay away from particular sorts of practices, such as exposure to organic solvents, smoking and heavy alcohol use. In the event that a lady has no resistance to rubella, she ought to get immunized preceding pregnancy.

Recognition and taking preventive measures of diabetes previously and during pregnancy ought to be a significant advance for diminishing danger of CHD in neonates. Ladies should avoid the medication that has suspected to cause congenital disease ,have mentioned warnings for pregnant ladies and has to informed to their obstetrician before taking any kind of medicine. doctors has to educate the patient. If a patient having a medical history of CHD than it is suggested by the gynaecologist for screening for possible cardiac defects using foetal echocardiography during pregnancy.

Challenges

Although Advances in medical field has made possible the survival of child born with a complex medical problem reach to adulthood yet a serious defect with CHD can be overwhelming and stressful, emotionally, physically, and financially to caregivers. Due to increased risk of cardiovascular complications, they often require specialized assistance, follow-ups, re-do interventions, and healthcare needs. [38,39,40] Despite health insurance, parents face hard to meet medical and surgical expenses. Due to weaker immune system, parents may be required to take extra precautions like annual flu shot and a Tdap shot to safeguard their child's health. Parents face continuous concern keeping a fine line between healthy limits and over-protectiveness. Parents may even juggle to keep a balance with their other children to avoid their anxiety and fear. Educating CHD child in preschool or school can be an uphill task and pose challenges for school staff and even parents. Their child's limitations and awareness about child medical condition among school staff may be hard at times.

Conclusion

Congenital heart defects represent some of the more prevalent birth defects, that result in significant lifelong morbidity, and are an important cause of mortality attributed to birth defects. Mothers should take all the preventive measures and diagnosis during pregnancy. All the harmful measure like cigarette, alcohol and contraindicated medication should be avoided during pregnancy. Recent advancements in medical Sciences along with learning specialized parenting skills can bring silver lining to parents of CHD living with persistent uncertainty and fear.

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Table 1: Classification of Congenital Heart Diseases:[24]

1	Left to right shunts	Atrial level: ASD, TAPVC Ventricular level: VSD Great artery level: PDA, AP window, Truncus arteriosus Coronary level: ALCAPA, coronary fistula
2	Right to left shunts	TOF physiology TGA physiology
3	Left heart obstructive lesions	Obstructed veins Mitral stenosis Aortic stenosis Coarctation Interrupted aortic arch Hypoplastic left heart syndrome
4	Right heart obstructive lesion	Pulmonary stenosis / atresia Tricuspid stenosis Hypoplastic right heart
5	Single ventricle	
6	Others	Vascular rings Venous anomalies Arteriovenous fistulae

Abbreviations: ASD: atrial septal defect,TAPVC: total anomalous pulmonary venous connection,VSD: ventricular septal defect,PDA: patent ductus arteriosus, AP: aortopulmonary, ALCAPA: anomalous left coronary artery from pulmonary artery,TOF: tetralogy of Fallot,TGA: transposition of great arteries