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Review Article

Pediatrics for better child health

Congenital Heart Disease in New Borns and Children, Clinical Features and Management- A Review

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Abstract: Congenital heart disease / congenital heart defects are more common than anomalies of any other systems of the human body. These defects occur in-utero during the organogenesis period, particularly in 1st trimester and the congenital heart disease may occur subsequently during development of the baby due to various environmental, genetic and developmental growth of the heart upto 1 year of life and these defects or illnesses result in various clinical implications with clinical signs and symptoms for us to diagnose and give appropriate therapy. Most of the cyanotic congenital heart disease cyanosis develops at birth and some congenital heart disease particularly Fallot's develop cyanosis late in the neonatal period. Most of the congenital heart disease respond to prostaglandin therapy, particularly this therapy is contraindicated in total anomalous pulmonary venous connection. This review explains the timing of referral of these CHD babies and children to the pediatric cardiologist and if necessary to pediatric cardiac surgeons. It is essential to know congenital heart disease forms a bulk which needs more pediatric cardiologists and pediatric cardiac surgeons. Only 200 pediatric cardiologists and 180 pediatric cardiac surgeons are available in the whole of India. Hence to give adequate care, proper identification and need for referral at an appropriate time is essential to decrease the morbidity and mortality in pediatric cardiac patients.

Keywords: congenital heart disease, congenital heart disease repair, congenital heart disease mortality, congenital heart disease surgical care.

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1. INTRODUCTION

Congenital heart disease is considered to be a gross structural abnormality of the heart or intrathoracic blood vessels that are actually or potentially of functional significance as described by Mitchell et al.[1] All babies presenting beyond the age of 12 months were also excluded, based on epidemiological studies because most clinically significant heart disease would have represented by this age. Examination of the newborn baby before discharge from hospital and of infants at 6 weeks of age for signs of congenital heart disease is recommended in health for all children (1). A normal examination in a newborn does not exclude heart disease. To diagnose congenital heart disease two points need to be considered such as 1)signs of cyanosis, cardiac murmur and splitting of 2nd heart sound 2)the babies screened further by ECHO and Doppler to confirm congenital heart disease. Congenital heart diseases are the most common anomalies with prevalence of one third of birth defects [2-4]. These defects require surgical intervention or inserting a trans-catheter in the neonatal period. Epidemiology of CHD accounts for nearly one third of all major congenital anomalies. The prevalence of CHD in infancy is estimated at 6-8 per 1000 live birth, 25% are life threatening and require early intervention. The prenatal exposure which increases the risk of CHD are Gestational diabetes, rubella, lupus, vitamin deficiency and teratogens[5](classification is shown in fig 1)

1.1 ETIOLOGY OF CYANOSIS?

The causes of CHDare cardiac and non-cardiac. The cardiac causes include presence of cyanosis, murmur on cardiac examination and absence of pulse.The non-cardiac causes include Pulmonary –Intrapulmonary, Extra pulmonary - Pneumothorax, trachea esophageal fistula, congenital diaphragmatic hernia and Airway obstruction.[6]Hematological- Methemoglobinemia and Polycythemia. Other causes are - sepsis, acrocyanosis, and hypoglycemia.

CLASSIFICATION OF CONGENITAL HEART DISEASE.

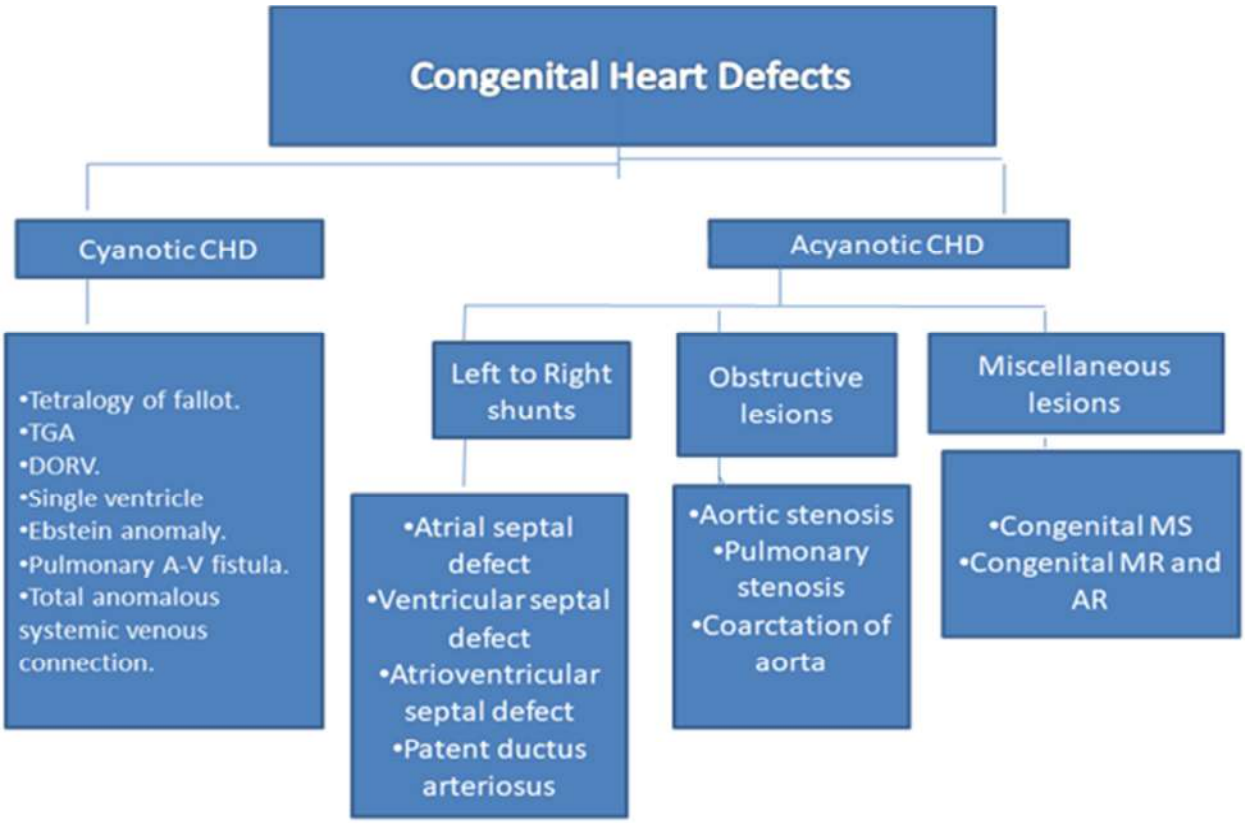

Fig.1 Showing Cyanotic AndAcyanotic Heart Disease. Is MadeaccordingTo Indian Status

1.2 CYANOTIC CONGENITAL HEART DISEASES

A child with cyanosis, is always a concern for both parents and treating doctor. Central cyanosis affects 3-4 % of all newborns and usually points to a serious underlying disorder, which would need thorough investigations and may need emergency treatment. Persistence of cyanosis beyond 2 hours is unusual. The etiology of cyanosis can be varied and warrants a thorough understanding of the basic pathophysiology to arrive at an early diagnosis. Most common form of cyanotic heart disease is Tetralogy of Fallot. All cyanotic congenital heart diseases require intervention, mostly by surgical repair. As a general rule, patients with cyanotic congenital heart disease with increased pulmonary blood flow and pulmonary venous hypertension present early, have poor prognosis without interventions as compared to those with pulmonary stenosis. Trans-catheter intervention has resulted in better outcomes for most congenital heart disease. The time of interventions dealt subsequently. The classifications of congenital heart disease of developed countries are not appropriate for developing countries, like India.

1.3 TETRALOGY OF FALLOT

It is the commonest cyanotic congenital heart disease, with a

prevalence of 5-10% [7].The cyanosis in newborn occurs mostly 2nd to 3rd week of life. The severity of cyanosis depends on severity of right ventricular outflow tract obstruction with increasing obstruction to pulmonary blood flow, the right to left shunt increases across the large ventricular septal defect, thereby increasing cyanosis. Obstruction to right ventricular outflow, juxtaposition of aorta, right ventricular hypertrophy. Clinical features include symptoms of heart failure, cyanosis not at birth, cyanosis occurs later in the first 4 months of life. Severe cyanosis occur as the ductus closes. Cyanotic spell, dyspnea on squatting position, gasping respiration and syncope after vigorous crying, metabolic acidosis, growth and developmental delay, pulse usually normal ,x-ray showed narrow bulge normal ovular in size, ECG will show right axis deviation. Complications include cerebral thrombosis, brain abscess. Associated anomalies are congenital absence of pulmonary wall, abscess of pulmonary artery, also known as catch 22. treatment depends on the severity of right ventricular outflow obstruction, IV fluids, correct dehydration and pain. In less severe cases primary repair 4-6 months of life.

1.4 TOTAL ANOMALOUS PULMONARY VENOUS RETURN (TAPVC)

It is an admixture type of lesion characterized by drainage of all pulmonary veins into the systemic venous side. Based on the site of drainage the defect can be classified into 4 types – supracardiac, cardiac, infracardiac and mixed type[8].

1.5 ACYANOTIC CONGENITAL HEART DISEASE

Acyanotic heart diseases can be broadly classified into left to right intracardiac shunts, valvular heart diseases and others. Clinical cyanosis is seen only if the systemic oxygen saturation is less than 85%.There are many cardiac structural defects that are grouped as admixture lesions which have mild systemic desaturation in the range of 85-94%, Clinically they may remain acyanotic[8]. Most common type of congenital Acyanotic heart disease is ventricular septal defect. [9]

1.6 VENTRICULAR SEPTAL DEFECT (VSD)

Ventricular septal defect is the most common. 3 to 5per 1000 live births.[10-11] ventricular septal defects are classified into perimembranous, outlet or subpulmonary, inlet and muscular types. Spontaneous closure is common with small to moderate muscular ventricular septal defects, whereas large muscular defects, inlet and outlet type of ventricular septal defects almost never close. Flow related pulmonary artery hypertension is invariably present in large, non-restrictive ventricular septal defects and pulmonary vascular obstructive changes may begin in the first two years of life in up to 80% of these children.[12]Small ventricular septal defects need follow up, and should only be closed if the patient has had an episode of infective endocarditis or develops Aortic regurgitation, most often due to aortic cusp prolapse. Moderate ventricular septal defects can be closed by 2-5year of age, if asymptomatic and have no evidence of pulmonary artery hypertension. However, early closure is recommended in symptomatic patients. Large ventricular septal defects need to be closed early, preferably by 6months of age. Patients who present late with advanced pulmonary veno-occlusive disease i.e., high Peripheral vascular resistance, need evaluation at a tertiary centre before deciding for surgical closure. Standard surgical approach is closure of ventricular septal defect with a patch. In specific (e.g., in very low birth weight neonates, sepsis difficult to close ventricular septal defects), temporary palliation with pulmonary artery banding (PAB) may be an option.

1.7 LABORATORY DIAGNOSIS

To prove heart disease clinically we need ECG, X-RAY, Echo for diagnosing various disorders. In suspected congenital heart disease, babies should not be sent directly to echo, but before sending a suspected congenital heart disease patients, do pulse oximetry of all four limbs. In pulse oximetry readings less then 95 % can be sent for echo for diagnosis. Various timings of interventions for particular disease over the intervention of any heart disease if its symptomatic you have to do intervention.

1.8 TIMING FOR INTERVENTION- CYANOTIC HEART DISEASE

1.8.1 TETRALOGY OF FALLOT

Propanaltherapy(1-4mg/kg/d in 2-3 divided doses), stable patients should undergo total repair between 6 to 12 months age. The definitive surgery is best delayed till 3-4year of age.

1.8.2 SINGLE VENTRICLE

Intervention is performed in staged fashion: initially at 6-12 months of age , the Superior vena cava is anatomized to pulmonary artery. Then at 4-5 years of age inferior vena cava is also diverted to the pulmonary artery.(as shown in fig 2)

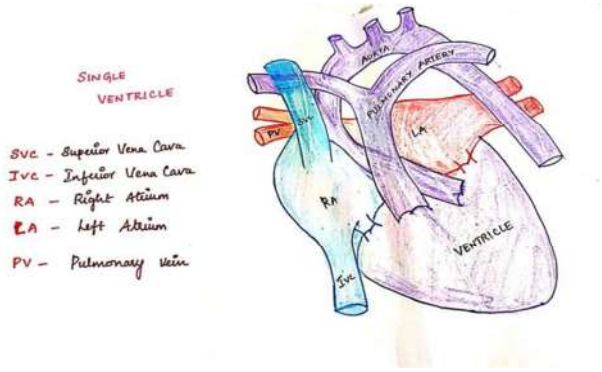

Fig.2. Single Ventricle Is One Of The Cause For Cyanotic Heart Disease

1.9 TOTAL ANAMOLOUS PULMONARY VENOUS RETURN

Obstructive total anomalous pulmonary venous return requires immediate surgery. It is the only congenital heart disease were prostaglandin E1 infusion can worsen the clinical condition and is contraindicated. It should be corrected as early as possible even if asymptomatic.

1.10 PERSISTENT TRUNCUS ARTERIOSUS

Early surgical repair should be undertaken preferably by 6 weeks of age irrespective of symptoms.(as shown in fig 3)

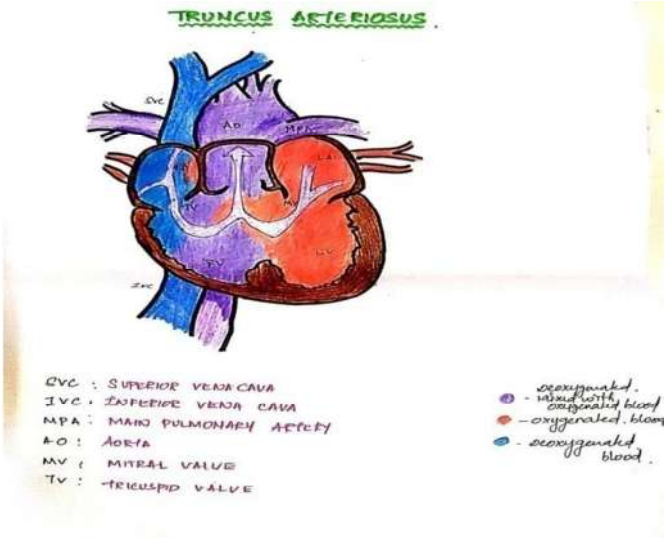

Fig.3.Truncus Arteriosus Is Also a Cause for Cyanotic Heart Disease

1.11 EBSTEIN'S ANOMALY

Neonates present with cyanosis and heart failure and may need stabilization with prostaglandin E1 and diuretics. Most neonates improve as pulmonary vascular resistance falls, surgery is considered only in those who do not improve. Older children are usually stable and indications for surgery include presence of symptoms, cyanosis, progressive cardiomegaly, worsening right ventricular function. (as shown in fig 4)

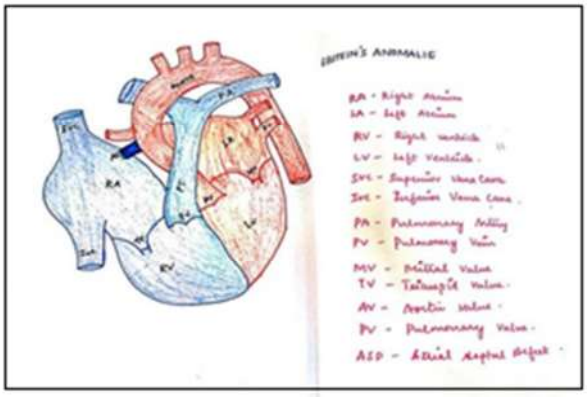

Fig.4.Ebstein's Anomaly Is Also a Cause of Cyanotic Heart Disease

1.12 DOUBLE OUTLET RIGHT VENTRICLE

Without PULMONARY STENOSIS- VSD closure by 6 months of age with PULMONARY STENOSIS –correction beyond 4-6 months of age.

1.13 TRICUSPID ATRESIA

Surgical intervention is to improve pulmonary blood flow. Shunt operations increase longevity. Group A are unlikely to survive beyond infancy without surgical intervention. Group B underwent surgery survived beyond 10 years

1.14 ACYANOTIC HEART DISEASE

1) ATRIAL SEPTAL DEFECT

Asymptomatic between2-4 years of age are better delayed to 4-5years of age. Early closure may be contemplated in symptomatic infants who may show heart failure and/or pulmonary arterial hypertension (PAH). Pulmonary arterial hypertension is seen in <10% of cases. Currently a number of secundum atrial septal defects are amenable to transcatheter closure by a device, thereby avoiding the need for open heart surgery.(as shown in fig 5)

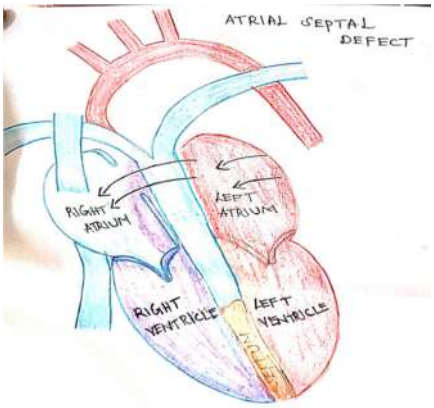

Fig.5. Atrial Septal Defect Is A Cause Of Acyanotic Heart Disease

TIMING OF INTERVENTION

Surgical repairs are preferably done before 3 months of age. Early intervention may be required if they are associated with more than moderate atrio-ventricular valve regurgitation. Patients with Down syndrome are known to develop irreversible pulmonary veno-occlusive disease as early as 6months of age and need to be intervened early.

2) VENTRICULAR SEPTAL DEFECT

Timing of intervention

1. Small ventricular septal defects need follow up and should only be closed if the patient has had an episode of infective endocarditis or develops Aortic regurgitation, most often due to aortic cusp prolapse.
2. Moderate ventricular septal defects can be closed by 2-5 years of age, if asymptomatic and have no evidence of Pulmonary artery hypertension. However, early closure is recommended in symptomatic patients.
3. Large ventricular septal defects need to be closed early, preferred by 6 months of age. Patients who present late with advanced veno-occlusive disease i.e., high Peripheral vascular resistance, need evaluation at a tertiary centre before deciding for surgical closure.
4. Standard surgical approach is closure of ventricular septal defect with a patch.

3) ATRIO VENTRICULAR SEPTAL DEFECT

It accounts for 4% to 5% of all congenital heart disease.[23] It is the most common congenital heart disease seen in down's syndrome; accounting for almost half of the congenital heart disease in this population[24]. It can be considered as a spectrum of defects, ranging from complete atrio ventricular septal defect to partial atrio ventricular septal defect. Complete atrio ventricular septal defect is characterized by the presence of primum atrial septal defect, large ventricular septal defect and common atrio ventricular valves, which may be regurgitant. Pulmonary artery hypertension is invariably present. Complete atrio ventricular septal defect may be associated with hypoplasia of one of the ventricles (unbalanced atrio ventricular septal defect).

Timing of intervention

Complete atrio-ventricular septal defect requires early surgical repair, before 3 months of age. atrio ventricular septal defect which have absent or small ventricular septal defect (partial and intermediate atrio ventricular septal defect) and no or mild pulmonary artery hypertension may be selectively operated at 2 to 3 years of age. Early intervention may be required if they are associated with more than moderate atrio ventricular valve regurgitation. Patient's with down's syndrome are known to develop irreversible pulmonary veno-occlusive disease as early as 6 months of age and need to be intervened early.[24] unbalanced atrio ventricular septal defect are not amenable for total two ventricular repair and require early pulmonary artery banding in order to protect the pulmonary vasculature for future univentricular palliation.

4) PATENT DUCTUS ARTERIOSUS

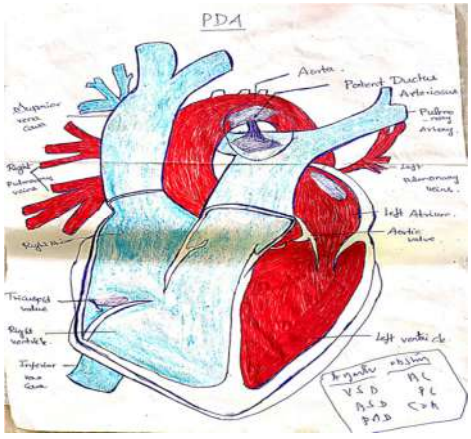

Fig.6. Patent Ductus Arteriosus Is Also A Cause Of Acyanotic Heart Disease

Early closure by 3months of age is recommended for symptomatic children with large patent ductusarteriosus. children with moderate patent ductusarteriosus and without heart failure can be intervened electively before one year of age and those with small patent ductusarteriosus can undergo closure between 12 and 18months of age. Closure is not recommended in silent patent ductusarteriosus. Device closure is preferable to surgical ligation. All symptomatic preterm neonates need treatment. Medical therapy with drugs such as Indomethacin and Paracetamol should be tried initially. Surgical ligation is to be contemplated in preterm children with hemodynamically significant patent ductusarteriosus who have failed medical therapy.(as shown in fig 6)

5) AORTIC STENOSIS

It can occur at valve(most common), subvalvular or supravalvar lesions. Valvular Aortic Stenosis is usually associated with abnormal aortic valve morphology, such as bicuspid or unicuspid valves. Bicuspid Aortic valve is the commonest cardiac malformation, seen in 1% of the general population, however significant stenosis is seen only in 0.2 to 0.4/1000 live births. Neonates with critical Aortic Stenosis, Prostaglandin E1 may be used for initial medical stabilization.(as shown in fig 7)

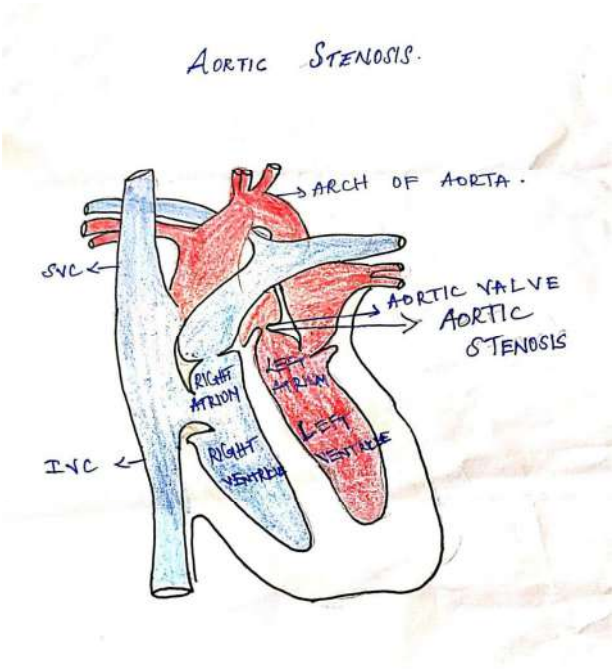

Fig.7. Aortic Stenosis Is Also A Cause Of Acyanotic Heart Disease

6) PULMONARY STENOSIS

It is most common at valve level. Isolated PS constitutes 8-10% of all congenital heart disease. Critical valvular pulmonary stenosis presents as ductus dependent circulation in the neonate or with heart failure. Valvular pulmonary stenosis can occur as in Noonan Syndrome For subvalvular or supravalvular pulmonary stenosis, surgery is the preferred mode of intervention. Peripheral pulmonary artery stenosis needs intervention.(as shown in fig 8)

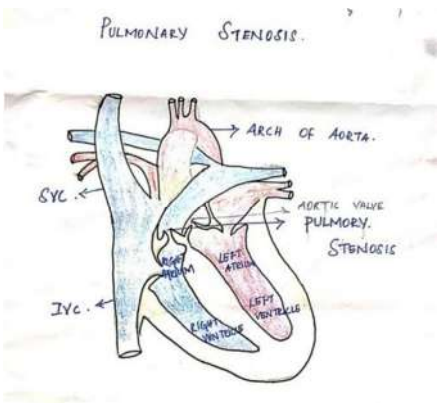

Fig. 8. Pulmonary Stenosis Is Also A Cause Of Acyanotic Heart Disease

7) COARCTATION OF AORTA

6-8% of all congenital heart disease is more in males. So in patients with bicuspid aortic valve, Ventricular septal defect, Patent ductusarteriosus and transposition of great arteries, look for Coarctation of aorta. Neonates with Coarctation of aorta may present with Patent ductusarteriosus shock once closes. Intervened around 1-2 years of age.

1.15 ARRHYTHMIA

Incidence of arrhythmia in children varies in literature with around 55.1 per 1 lakh pediatric emergency[12]. Most of the children with arrhythmias present with non specific complaints like feeding difficulties, fussiness, palpitation, chest heaviness, presyncope[14]. Infants will present with tachypnea, poor feeding and emesis. Prolonged duration of arrhythmia leads to more sicker infants presenting with shock, pallor, peripheral cyanosis and Congestive heart failure. Arrhythmias are classified into tachyarrhythmia, bradyarrhythmia, atrial flutter, atrial fibrillation.[14]Supraventricular tachycardia: It is the term used to refer any arrhythmia originating above the ventricular tissue. Incidence of Supraventricular tachycardia is 1 in 250 [15]to 1 in 1000 children. Half the children with Supraventricular tachycardia present with their first episode in infancy[16-17]. Spontaneous resolution may occur in up to 90 % by the end of infancy. However the second peak occurs in approximately one third of children at mean age of 8 years[17]. Children with a diagnosis made after the first year of life only up to 15 % may have spontaneous resolution[16]. Majority of children with Supraventricular tachycardia have normal heart[18] However the association of underlying congenital heart disease ranges upto 9 % to 32 % , most common being the Ebstein's anomaly and corrected transposition of great arteries[22]. Most regular Supraventricular tachycardia is narrow complex tachycardias other than Supraventricular tachycardia with aberrant conduction.

1.16 CONGESTIVE HEART FAILURE

Heart failure in the pediatric population differs from that in adults with respect to etiology and prognosis. While ischemia contributes to more than two thirds of heart failure in the adult population, main causes of heart failure in the pediatric age group are congenital heart diseases and cardiomyopathies [19] besides these, there are certain correctable systemic causes of Heart failure. The treatment modalities in the pediatric population are extrapolated from the adult population. The use of novel drugs and mechanical devices are controversial A working definition of HF is a syndrome with both clinical and pathophysiological entities due to cardiac and non cardiac abnormalities presenting with edema, growth failure, respiratory distress and exercise intolerance due to circulatory, neuro-hormonal and molecular derangements. Most of the medical management of heart failure has been extrapolated from adult studies. The use of sympathomimetic amines like dopamine and dobutamine is effective in patients with circulatory failure.[20]In volume overload conditions like Ventricular septal defect, Atrio-ventricular septal defect, children become symptomatic usually after a gap of one to two months when the pulmonary resistance drops. The mainstay of treatment is diuretics but these conditions ultimately require corrective surgery. The first treatment strategy is to identify the underlying cause for heart failure like rhythm abnormality or residual defect and that has to be corrected. The medical treatment for children presenting with heart failure secondary to cardiomyopathies is primarily directed for dilated cardiomyopathy. Pacemaker, in cases of Congenital heart disease with Heart failure is indicated in children with symptomatic bradycardia, loss of atrioventricular synchrony with high grade atrioventricular blocks.[21] The indications for cardiac resynchronization therapy are sparse in this set of patients. The use of circulatory support devices in end stage failure in children is increasing as a bridge to transplantation (BT) indication. Heart transplantation is the treatment of choice in end stage heart failure refractory to all treatments The one year survival rates are more than 80% and the overall survival after two decades is around 40 to 50%.Nutrition plays an important role in heart failure management. A diet plan with high calorie feeds is required to optimize the growth in view of increased metabolic demands in heart failure kids. The future directions in pediatric heart failure should include conducting research with proper randomized trials, directed towards management, which is lacking in this subset of patients. The use of cell based therapy including stem cell treatment for myocardial repair, heart valves, pacemakers and vessels construction, is gaining prominence and can be explored in pediatric patients. [22]

2. CONCLUSION

Congenital heart disease is always a problem in neonates. These cyanotic heart diseases warrant medical intervention and death occurs without proper intervention. Acyanotic heart disease to be intervened when required. This article emphasizes the timing of referral to pediatric cardiologist and pediatric cardiac surgeons which is very essential for pediatricians for adequate counseling of the parents. All pediatricians should know about the natural course of cardiac disease in order to correctly diagnose and refer. Genome sequencing in pediatrics was done for cardiac diseases like cardiomyopathies, laterality defects, and outflow tract obstructions. In Congenital heart disease, Structural variants occur and are being diagnosed by chromosomal microarray but genomic sequence of late is very much diagnostic for pediatric heart diseases. For more information allen HD et al; Heart disease in infants, children and adolescents,7TH Edition, kluwer/ lippincot William and wilkins; Philadalphia; usa; 2008[5] can be referred

3. AUTHOR CONTRIBUTION STATEMENT

Dr. Sathulhak, collected various data needed for the studies, Dr. Paramanantham, reviewed the content and communicated for publication in a journal.

4. CONFLICT OF INTEREST

Conflict of interest declared none.

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