

The Echocardiographic and Radiographic Characteristics of Children with Clinically Confirmed Congenital Heart Disease: An Observational Study

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Abstract

Aim: The aim of this study evaluate the echocardiographic and radiological profile of clinically diagnosed congenital heart disease in children

Methods: The Prospective, Observational study was conducted in the Department of Pediatrics, Vardhman institute of medical sciences, Pawapuri, Nalanda, Bihar, India, for 1 year. 100 Children from 1 month to 18 years of age with clinically suspected cases of CHD were included in this study. All children in the age group of 1 month to 18years of age with complaints of recurrent chest infection, respiratory distress, cyanotic spells, squatting episodes, poor feeding, failure to thrive and other relevant history and examination findings suggestive of CHD were included in this study. Informed consent was taken from parents or caregivers.

Results: Out of 4588 patients selected for study 120 patients were found to have CHDs and the hospital incidence of CHD is 2.62%. In our study in clinical features, breathlessness was the commonest and it was in 65% followed by cough in 80% and fever 42 % of cases. In our study the distribution of CHD cases by clinical diagnosis was 70% for ACHD and 30 % for CCHD cases. In our study as per the chest x-ray findings following were there cardiomegaly, Increased pulmonary vascular markings (Pulmonary plethora), Decreased Pulmonary vascular markings (Pulmonary oligemia), RVH/LVH, Pneumonia in 73 %, 41 %, 33 % 51 % and 15 % respectively. In our study the correlation of clinical profile with chest x-ray was 81 %. The clinical diagnosis correlating with echo was 81%.

Conclusion: The most common presenting symptoms in CHD were cough and

breathlessness. Chest X-ray is important corroborative evidence in the diagnosis of CHD and was abnormal in 81% of cases with the presence of cardiomegaly in 73 % of all cases.

Keywords: CHD, cardiomegaly, echocardiography

Introduction

In the under-5 age group, bronchopneumonia is one of the leading causes of morbidity and mortality. It accounts for almost fifteen percent of the deaths below 5 years of age. It has been accounted for 808,694 deaths in 2017 alone. It can be caused by bacteria, viruses and fungi.

On the other hand, CHD are the commonest congenital defects globally accounting for almost 6 - 8 per 1000 live births.¹ Studies have shown that there is increased risk of mortality in children with CHD presenting as bronchopneumonia. Various factors have contributed for the increasing incidence of bronchopneumonia in children with CHD including increased vasculature, alterations in the immune system reflected with change in lymphocyte population and difference in serum zinc levels. Several predisposing factors have been reported for recurrent respiratory tract infections, including malnutrition, congenital or acquired immune deficiency conditions, some children with CHD may have respiratory problems triggered by enhanced pulmonary blood flow causing pulmonary oedema, or airway obstruction due to enlarged or dilated vascular and heart structures. Patients with CHD have a high prevalence of abnormal ciliary motion, which is associated with an increased risk of respiratory symptoms.²

A predisposing factor for recurrent respiratory tract infections is congenital heart disease which causes increased pulmonary blood flow. Ventricular Septal Defect (VSD), Atrial Septal Defect (ASD), Patent Ductus Arteriosus (PDA) are typical acyanotic congenital heart disease predisposed to recurrent respiratory tract infections in childhood. Shunting from left to right induces increased flow of pulmonary blood and pulmonary oedema. Pulmonary oedema progresses to congestive heart failure and becomes a nidus for lower respiratory tract infection. Pneumonia and congestive cardiac failure may be the early symptoms of congenital heart disease underlying it. The age at which symptoms begin in children with congenital heart disease depends on the severity of the defects. Large-scale ventricular septal defects and patent ductus arteriosus arise early and have more serious diseases resulting in CCF.³ It has been documented that the mortality rate caused by uncontrolled heart failure in children with CHD and pneumonia receiving conservative medical care is 11.9 % to 56 %. Controversial care for children with left to right shunt CHD concomitant with chronic pneumonia is still present. Increased pulmonary blood flow, pulmonary arterial pressure, right ventricle afterload, and cardiac dilatation increase the probability of pulmonary congestion, and with imperfect development of immune system function, the infants are more susceptible to pneumonia.

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These infants generally manifest with recurrent pneumonia, will be less responsive to medical therapy alone, and have recurrent pulmonary infections that will be difficult to eradicate. Pneumonia is difficult to treat, or may progress into serious pneumonia, and then heart and respiratory failure will occur. Assessment of cardiac function in children with CCF is based on electrocardiogram, ultrasonic cardiogram, and clinical data that

is not precise. CCF's clinical characteristics include weight gain or oedema, dysphoria, cyanosis, poor reaction, light skin and milk intake reduction. All those features can be used to diagnose CCF early. These symptoms can also be seen in children with CHD, however. Thus, these clinical results were not unique to CCF and inadequate for correct diagnosis. Increased heart rate, pneumonia, or infection of the respiratory tract which often accompanies CHD can induce CCF. Although many studies have shown that there is increased risk of bronchopneumonia in children suffering from CHD, studies looking at the pattern of CHDs presenting as bronchopneumonia have not been completely available. Through this prospective cohort study we would like to look into the pattern of CHDs presenting as bronchopneumonia. We will also get additional information about the subpopulation of CHDs landing in congestive cardiac failure or those associated with high mortality or morbidity.⁴⁻⁶

Material and Methods

The Prospective, Observational study was conducted in the Department of pediatrics, Vardhman institute of medical sciences, Pawapuri, Nalanda, Bihar, India, for 1 year, after taking the approval of the protocol review committee and institutional ethics committee. 100 Children from 1month to 18 years of age with clinically suspected cases of CHD were included in this study. Patients with acquired heart disease and established cases of Congenital heart disease were excluded from this study.

Data Collection

All children in the age group of 1 month to 18 years of age with complaints of recurrent chest infection, respiratory distress, cyanotic spells, squatting episodes, poor feeding, failure to thrive and other relevant history and examination findings suggestive of CHD were included in this study. Informed consent was taken from parents or caregivers.

The study group was first assessed clinically according to a performed proforma after getting the written consent. They underwent Chest X-ray and Electrocardiogram. The final diagnosis was confirmed by Echocardiography. Clinical diagnosis of the suspected CHD was then correlated with X-ray findings and Echocardiography.

Statistical analysis

Sample of 100 cases attending the hospital during the study period was selected by Simple Random Technique. Data were entered into a Microsoft Excel datasheet and was analyzed using SPSS 21.0 version software. Categorical data was represented in the form of Frequencies and proportions. The Chi-square test was used as a test of significance for qualitative data. Continuous data were represented as mean and SD. P-value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

Table 1: Hospital incidence of CHD during the study period

Total selected cases for study in OPD and IP during the study period	4588
Total CHDS during the study period	120
CHDs was taken into the study during the study period	100
Percentage of CHD in the study	3.7

Table 1: out of 4588 patients admitted 120 patients were found to have CHDs and the hospital incidence of CHD is 3.7%.

Table 2: Distribution of CHD by mode of presentation clinically

Mode of presentation	No cases of CHD	Percentage
Breathlessness	65	65
Chest pain	13	13
Cough	80	80
Cyanosis	28	28
Fever	42	42
Chest pain	37	37
Feeding difficulties	42	42
FTT PEM Malnutrition	54	54
Palpitation	9	9
Fatigue	7	7
Suck rest suck cycle	7	7
Forehead sweating	27	27

Table 2: depicts that cough(80%) and breathlessness (65%) were major symptoms.

Table 3: Distribution of CHD cases by Clinical Diagnosis

Type Of CHD	No of cases	Percentage
CCHD	30	30
AACHD	70	70
Total	100	100

Acyanotic CHD comprises 70% of total cases.

Table 4: Distribution of CHD cases by Chest Xray findings

Chest x-ray findings	No of cases	Percent
Cardiomegaly	73	73
Increased pulmonary vascular marking	41	41
Decreased pulmonary vascular markings	33	33
RVH/LVH	51	51
Pneumonia	15	15

As per the table 4: Cardiomegaly 73%and hypertrophy 51% were major findings.

Table 5: Correlation of Clinical features with Chest X-ray

Correlation present or absent	No of CHD cases	Percentage
Correlating	81	81
Not Correlating	19	19
Total	100	100

Table 6: Correlation of Clinical diagnosis of total CHD with ECHO findings

Correlation with ECHO	No of CHD cases	Percentage
Not Correlating	19	19
Correlating	81	81
Total	100	100

Discussion

The term congenital is derived from the Latin word ('con' means together and 'genitus' meaning born) referring to 'present at birth'. Congenital heart disease (CHD) is defined as an abnormality in 'cardiocirculatory' structure or function that is present since birth, even though it may be discovered later.⁷ Thomas Beville Peacock was a physician who practised in the 1800s at St Thomas 'Hospital in London. He was the first to classify CHD into 4 categories: misplacements of the heart, pericardial abnormalities, cardiac malformations, and irregularities of the primary vessels. Peacock wrote about the causes of CHD, attributing most forms to abnormalities in embryonic development, particularly growth arrest.⁸

The estimated birth prevalence of CHD is 8/1000 live births with a significant geographical difference. A recent systemic review reported the highest prevalence (9.3/1000 live births) in

Asia due to the high birth rate and consanguineous marriages and the lowest prevalence in Africa (8.2/1000 live births). In India, over 180,000 children are born with CHD every year with state-wise variation and contribute to 10% of the present infant mortality. Nearly one-third of the CHD is critical, requiring intervention in the 1st year of life. Most of the CHD is thought to be multifactorial and result from a combination of genetic and environmental insult.⁹ Fetal echocardiography can make a more accurate diagnosis of CHD even before birth. With currently available treatment modalities over 75% of infants born with critical heart disease can survive beyond the first year of life and many can lead a normal life thereafter.¹⁰ Cardiogenesis begins on the 18th day of life with the formation of the carcinogenic crescent of the precardiac mesoderm and normally is completed by the 45th day of life with the formation of the membranous part of the ventricular septum. Cardiovascular maturation continues well after birth.¹¹

The main differences between the fetal and postnatal circulation are: a) Presence of placental circulation, which provides a gas exchange for the fetus. The absence of gas exchange in the collapsed lungs results in very little flow of blood to the lungs and thus little pulmonary venous return to the left atrium

Presence of ductus venosus, joining the portal vein with the inferior vena cava, providing a low, resistance bypass for umbilical venous blood to reach the inferior vena. Widely open foramen ovale to enable oxygenated blood (through umbilical veins) to reach the

left atrium and ventricle for distribution to the coronaries and the brain; and lastly and Wide open ductus arteriosus to allow right ventricular blood to reach the descending aorta, since lungs are non-functioning.¹²

The sudden expansion of lungs with the first few breaths causes a fall in pulmonary vascular resistance and an increased flow into the pulmonary trunk and arteries. The pulmonary artery pressure falls due to the lowering of pulmonary vascular resistance. The pressure relations between the aorta and pulmonary trunk are reversed so that the flow through the ductus is reversed. Instead of blood flowing from the pulmonary artery to the aorta, the direction of flow through the ductus, is from the aorta to the pulmonary trunk.¹³

The relative frequency of the most common lesions varies but nine common lesions form 80 % of CHD. These are VSD (36%) , ASD (5 %), patent arterial duct (9 %), atrioventricular septal defect (AVSD) (4 %), pulmonary stenosis (PS) (9 %), AS (5 %), coarctation of aorta (5 %), transposition of great arteries (4 %), and TOF (4 %).The other 20 % of CHD consists of many rare or complex lesions.¹⁴

Breathlessness, chest retractions, FTT, Feeding difficulty, cyanosis were the common clinical presentations in congenital heart disease. CHD should be suspected in all cases of recurrent chest infections and failure to thrive. A high index of suspicion, a detailed history, physical examination, chest x-ray, electrocardiogram along Echocardiography helps us to diagnose most of the congenital heart disease. With limited resources, clinical acumen forms the backbone for diagnosis for CHD. Early detection and intervention reduce the morbidity and mortality of CHD.¹⁵ In our study out of 4588 patients admitted 120 patients were found to have CHDs and the hospital incidence of CHD is 2.62%.This is comparable with other studies by Rakshit Reddy et al where incidence was found to be 1.85 %.⁷ In a study done by Shymsundar incidence was 2.1 %¹⁶ and 2.5 % in a study done by A.Saxena et al.¹⁷

In our study in clinical features, breathlessness was the commonest and it was in 65% followed by cough in 80% and fever in 42 % of cases whereas in a study done by Karthigaet al the incidence of breathlessness, cough and fever were present in 72%, 70% and 60% repectively.¹⁸ and in a study done by Rakshith et al it was 68 %, 64 % and 58 % respectively.⁷ In our study the distribution of CHD cases by clinical diagnosis was 70% for ACHD and 30 % for CCHD cases. In a study done by Chandra SekharKondapalli it was 85% for ACHD and 15 % for CCHD.¹⁹ In a study done by Rakshit Reddy et al it was 82 % for ACHD and 18 % for CCHD.⁷ In our study as per the chest x-ray findings following were there Cardiomegaly, Increased pulmonary vascular markings (Pulmonary plethora), Decreased Pulmonary vascular markings (Pulmonary oligemia), RVH/LVH, Pneumonia in 73 %, 41 %, 33 % 51% and 15 % respectively. In a study done by Chandra SekharKondapalli incidence was 78 %, 47 %, 20 % and 10 % respectively [3]. whereas in a study done by Laya B et al it was 65 %, 25 %, 10 % and 30 % respectively.²⁰ In our study the correlation of clinical profile with chest x-ray was 81%. In a study done by Chandra Shekar Kondapalli it was 80 %.¹⁹ and in a study done by Akbar et al 3 it was 63%²¹ and in a study done by Laya et al it was 71 %.²⁰ In all these studies clinical diagnosis is strongly correlated and supported by chest x-ray findings. In our study the clinical diagnosis correlating with echo was 81 %. In a study done by Mir D et al the incidence was found to be the same 69% fully correlating and 19% partially

correlating and in a study done by Rakshith Reddy et al full correlation was seen in 73 % and partial correlation in 14 %.⁷ In a study by Swenson et al and Klewer et al the clinic echo correlations were 82 % and 81 % respectively.^{22,23}

Conclusion

The most common presenting symptoms in CHD was cough and breathlessness. Chest X-ray is important corroborative evidence in the diagnosis of CHD and was abnormal in 81 % of cases with the presence of Cardiomegaly in 73 % of all cases. Properly carried out the clinical examination, Chest X-ray and ECHO evaluation are important tools in arriving at a near accurate diagnosis in CHD, however before undertaking surgical intervention echocardiographic confirmation of the diagnosis is required.

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