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Clinical Presentation and Complications of Different Congenital Heart Disease in Children

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Abstract: The clinical presentation of congenital heart disease varies according to the type and severity of the defect. *Aim of* the study: The aim of this study is to observe the clinical presentation and complications of different congenital heart disease. Material & Methods: It was a hospital based Cross-sectional study carried out prospectively in the department of pediatrics of Rajshahi medical college & hospital in from July 2011 to December 2011. The study population was all the children up to 12 years of age admitted in three pediatric units of this hospital during the study period. History included the presenting complaints of the patient's e.g. breathlessness, cough, palpitation, undue fatigability, bluish coloration or lips, tongue and extremities, history suggestive of cyanotic spell, feeding problem, poor weight gain, recurrent chest infection etc. Onset of symptoms and duration were noted. Chest X-ray reports were done by radiologists, ECG reports and echocardiography were done by cardiologists of RMCH. Among the 147 clinically suspected cases congenital heart disease was confirmed in 110 cases by echocardiography. Results: Total 110 patients with different types of congenital heart disease were included in this study. Male: female ratio were 2:1. the commonest lesion was VSD present in 39.1 (%) patients and single ventricle with single A-V canal defect in 0.9 (%). Rest patients had multiple lesions. The commonest presentation was recurrent chest infection which present in 69.1 (%) of patients. The commonest physical finding was anemia, present in 72.7 (%) of patients. Among 110 patients, 101 presented with different complications. Heart failure was present in total 15 cases, among them 40 (%) of VSD, 33.3 (%) of PDA and 26.7 (%) of multiple lesions patients presented with heart failure. Growth failure was observed in total 23 cases, among them 13 (%) of VSD, 21.7 (%) of ASD, 43.5 (%) of TOF, 8.7 (%) of PDA and 13.1 (%) of other patients presented with growth failure. Pneumonia was noted in 54.3 (%) of VSD, 22.9 (%) of ASD, 17.1 (%) of PDA and 5.7 (%) of other patients. Recurrent chest infection was noted in 70 (%) of VSD and 30 (%) of PDA patients. Pneumonia with heart failure was noted in 54.3 (%) of VSD, 22.9 (%) of ASD, 17.1 (%) of PDA and 5.7 (%) of other patients. Cerebral abscess occurred only in TOF cases. Conclusion: Fast breathing, chest indrawing, cough, poor weight gain, feeding problems, anemia, cyanosis, clubbing, easy fatigability, recurrent chest infection and murmur detected in routine cardiac examination

Keywords: Congenital Heart Disease, Clinical Presentation, Complications, Atrial Septal Defect, VSD, PDA, Pulmonary Stenosis

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1. Introduction

Congenital heart disease as a whole occurs with equal frequency in male and females but some lesions such as transposition of great vessels and left sided obstructive lesions are slightly more common in boys whereas atrial septal defect, VSD, PDA and pulmonary stenosis are more common in girls. [1] The clinical presentation of congenital heart disease varies according to the type and severity of the defect.² In neonatal period the presenting features of congenital heart disease are cyanosis (with or without respiratory distress), heart failure (with or without cyanosis), collapse & an abnormal clinical sign detected on routine examination (e.g. absent femoral pulse or a heart murmur). [2, 3] In infancy and childhood the usual presenting features are cyanosis, digital clubbing, murmur, syncope, frequent respiratory tract infection, squatting, heart failure, arrhythmia and failure to thrive.³ The adolescent present with heart failure, murmur, arrhythmia, cyanosis, hypertension & late consequences of previous cardiac surgery (e.g., arrhythmia, heart failure). [3] In over 90% of instances the cause of CHD is unknown. However, multifactorial inheritance with both genetic and environmental inputs is suspected. [1] Here, there is combined action of environmental influence and two or more mutant genes having additive effect. The genetic component exerts a dosage effect- the greater the number of inherited deleterious genes, the more severe the expression of the disease. Because environmental factors significantly influence the expression of these genetic disorders, the term polygenic inheritance is misleading. [4] There is two to tenfold increase in the incidence of congenital heart disease in sibling of an affected patient or children of an affected parent, pointing to genetic influences. On the other hand, monozygotic twin has only a 10% concordance for ventricular septa) defects. [5] There is well-defined sex preponderance for certain specific defects. Overall, males are more often involved. Thus, there are many kinds of genetic influence, but the twin studies make clear that environmental influences although rarely identifiable, must also contribute. Clinical presentation of congenital heart disease differs conferring to the category and severity of the defect. [6] At present the offering feature of congenital heart disease are cyanosis, heart failure with or without cyanosis, collapse, an abnormal clinical sign detected on routine examination. [6] In early stages and childhood the typical symptoms are cyanosis, digital clubbing, murmur, syncope, squatting, heart failure, arrhythmia, failure to thrive. [7] The teenage and grownups faces heart failure, murmur, arrhythmia, cyanosis, hypertension, late consequences of previous cardiac surgery. [7] By way of a mutual congenital anomaly, CHD not only subsidize to a noteworthy morbidity and mortality but also causes a great psychological stress and economic load to the whole family. Yet, if the complications are familiar at earlier age, the coincidental of long-term difficulties are less and the outcome is healthier. In place of a result of enhanced medical and surgical management, more children with CHD are enduring into adolescence and adulthood. [8] Therefore,

there is a necessity for an improved responsiveness amongst general physicians and cardiologists of the problem modeled by these individuals. Excluding a few scattered annotations, the incidence and detail clinical profile of CHD in Bangladeshi children are not well recognized. This study was commenced to find out the pattern and clinical presentation and complications of congenital heart disease among the admitted children in Rajshahi Medical College Hospital. It may help to detect and treat congenital heart disease at an earlier age and thus give the affected children and their parents hope of a better life.

2. Methodology and Materials

It was a hospital based Cross-sectional study carried out prospectively in the department of pediatrics of Rajshahi medical college & hospital in from July 2011 to December 2011. The study population was all the children up to 12 years of age admitted in three pediatric units of this hospital during the study period. In all cases detail history was obtained from the parents. History included the presenting complaints of the patient's e.g. breathlessness, cough, palpitation, undue fatigability, bluish coloration or lips, tongue and extremities, history suggestive of cyanotic spell, feeding problem, poor weight gain, recurrent chest infection etc. Onset of symptoms and duration were noted. Chest Xray reports were done by radiologists, ECG reports and echocardiography were done by cardiologists of RMCH. Among the 147 clinically suspected cases congenital heart disease was confirmed in 110 cases by echocardiography. After confirming the diagnosis data was noted in a preformed data sheet.

- 1) Inclusion Criteria
 - a) All cases of clinically diagnosed congenital heart disease admitted into all pediatric units of Rajshahi Medical College Hospital, Rajshahi.
- 2) Exclusion Criteria
 - b) The patients who are critically ill and age more than 12 years will be excluded from the study.

3. Results

Total 110 patients with different types of congenital heart disease were included in this study. Among them male patient is 67% and female is 33%. So male: female ratio is 2:1. It is evident from Table 1 that the commonest lesion was VSD present in 39.1 (%) patients followed by PDA in 17.3 (%), TOF in 15.5 (%), ASD in 11.8 (%), A-V canal defect in 0.9 (%), PS in 1.8 (%), COA in 1.8 (%), TGA in 1.8 (%) and single ventricle with single A-V canal defect in 0.9 (%). Rest patients had multiple lesions. Table 2 shows detailed clinical presentation of CHD cases. In the present series, among 110 patients of CHD, the commonest presentation was recurrent chest infection which present in 69.1 (%) of patients. Then in order of frequency other presentations were poor weight gain in 51.8 (%), respiratory distress in 46.4 (%), cough in 44.5 (%), fever in 44.5 (%), feeding problem in 36.4 (%), dyspnea

on exertion in 29.1 (%), hyper cyanotic spell in 23.6 (%) and convulsion in 4.8 (%) patients. It is evident from Table 3 that some important physical findings in CHD patients. Here the commonest physical finding was anemia, present in 72.7 (%) of patients. Other findings were tachycardia in 47.3 (%), fast breathing in 47.3 (%), cyanosis in 27.3 (%), clubbing in 15.5 (%), edema in 10.9 (%), conjunctival congestion in 15.5 (%), enlarged tender liver in 15.5 (%), hypertension in 27.3 (%), chest indrawing in 31.8 (%), crepitation in 47.3 (%) and rhonchi in 30.9 (%) patients. Table 4 shows the cardiac findings in CHD cases. Thrill was mostly present in 60.5 (%) of VSD cases, in 42.1 (%) of PDA, in 50 (%) of A-V canal defect and 10 (%) of multiple lesions patients. Palpable P2 was present in 27.9 (%) of VSD cases, 15 (%) of TOF, 50 (%) of PS and 30 (%) of multiple lesions patients. Left parasternal heave was found in 25.6 (%) of VSD cases, 15.6 (%) of ASD, 88.2 (%) of TOF, 50 (%) of PS and 40 (%) of multiple lesions patients. Wide and fixed splitting S2 was present in 84.6 (%) cases of ASD and 10 (%) of multiple lesions. Single S2 was found in all (100%) cases of PS, 30 (%) of multiple lesions and 88.2 (%) cases of TOF. Table 5 shows important radiological findings (of X-ray chest) in different CHD patients. Cardiomegaly was present in 5.3% of PDA, 50% of COA, 50% of PS and 20% of multiple lesions cases. Plethoric lung field with cardiomegaly & consolidation was observed in 10% of VSD & 21% of PDA cases. Cardiomegaly with consolidation was observed in 11.6% of VSD & 15.6% of ASD cases. Cardiomegaly with plethoric lung field was observed in 46.2% of VSD, 63.2% of PDA, 76.9% of ASD, 50% of AV canal defect & TGA cases. Boot shape heart with oligamic lung was observed in 88.2% of TOF patients. Oligaemic lung was noted in 11.8% of TOF 20% of multiple lesions cases. Pulmonary opacity/consolidation was found in 4.7% of VSD cases. Table 6 shows detailed ECG findings in CHD patients. RVH with RAD was noted in 4.7% of VSD cases, in 30.8% of ASD cases, in 82.4% of TOF, in 100% of PS and 50% of other CHD patients. LVH with LAD was found in 46.5% of VSD cases, in 38.8% of ASD, in 63.9% of PDA and in 50% of COA patients. RVH+ LVH+ Tall P (P pulmonale) was present in 7% of VSD cases, 7.7% of ASD, 50% of AV canal defect and TGA patients. In this study 37.2% of VSD, 15.4% of ASD, 26.3% of PDA, 50% of PS and 50% of multiple lesion patients had normal ECG tracing. Table 7 shows among 110 patients, left ventricular and atrial dimension was increased in most cases of VSD, PDA and COA. Right atrial and right ventricular dimension was increased in most cases of ASD, PS, single ventricle, TOF and a few cases of VSD & other

CHD. Narrow RVOT and Overriding of aorta was present in all cases of TOF. Table 8 shows among 43 VSD patients 53.5 (%) were peri- membranous, 34.9 (%) were muscular and 11.6 (%) were supracristal variety. Among 43 VSD patients 11.6 (%) were small, 30.2 (%) were medium and 53.5 (%) were large variety. Table 9 shows among 13 ASD patients 7.7 (%) were osteum primum, 92.3 (%) were osteum secondum variety. Table 10 shows complications of different CHD patients. Among 110 patients, 101 presented with different complication. Heart failure was present in total 15 cases, among them, 40 (%) of VSD, 33.3 (%) of PDA and 26.7 (%) of multiple lesions patients presented with heart failure. Growth failure was observed in total 23 cases, among them, 13 (%) of VSD, 21.7 (%) of ASD, 43.5 (%) of TOF, 8.7 (%) of PDA and 13.1 (%) of other patients presented with growth failure. Pneumonia was noted in 54.3 (%) of VSD, 22.9 (%) of ASD, 17.1 (%) of PDA and 5.7 (%) of other patients. Recurrent chest infection was noted in 70 (%) of VSD and 30 (%) of PDA patients. Pneumonia with heart failure was noted in 54.3 (%) of VSD, 22.9 (%) of ASD, 17.1 (%) of PDA and 5.7 (%) of other patients. Cerebral abscess occurred only in TOF cases.

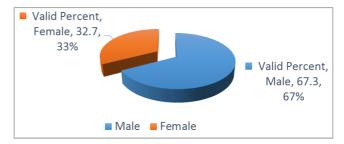


Figure 1. Sex distribution of different congenital heart disease (n-110).

Table 1. Types of congenital heart disease in all patients from birth to 12 years of age (n-110).

Types of Lesion	No. of Patient	Percentage
VSD	43	39.1
ASD	13	11.8
TOF	17	15.5
PDA	19	17.3
A-V canal	1	0.9
PS	2	1.8
TGA	2	1.8
COA	2	1.8
Multiple lesion	10	9.1
Single ventricle with single A-V canal defect	1	0.9
Total	110	100.0

Table 2. Clinical presentation of congenital heart disease (n-110).

Symptoms	No. of cases	Percentage
Respiratory distress	51	46.4%
Fever	49	44.5%
Cough	49	44.5%
Poor weight gain	57	51.8%
Recurrent chest infection	76	69.1%
Feeding problem	40	36.4%
Dyspnea on exertion	32	29.1%

Symptoms	No. of cases	Percentage
Hyper cyanotic spell	26	23.6%
Convulsion	2	4.8%

Table 3. Important physical findings in CHD (n-110).

Physical finding	No. of cases	Percentage
Cyanosis	30	27.3
Clubbing	17	15.5
Edema	12	10.9
Anemia	80	72.7
Hypertension	20	27.3
Conjunctival congestion	17	15.5
Tachycardia	52	47.3
Enlarged tender liver	17	15.5
Fast breathing	50	45.9
Chest indrawing	35	31.8
Crepitation	52	47.3
Rhonchi	34	30.9

Table 4. Important cardiac findings in CHD Patients (n-110).

Types of lesion	Thrill n (%)	Palpable P2 n (%)	Parasternal Heave n (%)	Wide & Fixed splitting S2 n (%)	Single S2 n (%)
VSD	26 (60.5%)	12 (27.9%)	11 (25.6%)	0 (0%)	0 (0%)
ASD	0 (0%)	0 (0%)	2 (15.6%)	11 (84.6%)	0 (0%)
TOF	0 (0%)	15 (88.2%)	15 (88.2%)	0 (0%)	15 (88.2%)
PDA	8 (42.1%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
A-V canal defect	1 (50%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
PS	0 (0%)	1 (50%)	1 (50%)	0 (0%)	2 (100%)
TGA	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
COA	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Multiple lesion	1 (10%)	3 (30%)	4 (40%)	1 (10%)	3 (30%)

Note: Parenthesis indicate percentage in respect of total no, of individual defect.

Table 5. Important radiological findings (of x-ray chest) in CHD patients (n-110).

	X-ray ches	t								
Lesion	Normal	Cardio- megally	Cardiomegally with Plethoric lung	Cardiomegally with consolidation	Plethoric lung with consolidation	Boot shape heart with oligamic lung	Oligamic lung	Cardiomegally with consolidation with plethoric lung	Consolidation	Total
VSD	1 (2.3%)		20 (46.2%)	5 (11.6%)	5 (11.6%)			10 (23%)	2 (4.7%)	43 (100%)
ASD			10 (76.9%)	1 (7.7%)	2 (15.4%)					13 (100%)
TOF						15 (88.2%)	2 (11.8%)			17 (100%)
PDA	2 (10.3%)	1 (5.3%)	12 (63.2%)					4 (21.1%)		19 (100%)
A-V canal	1 (50.0%)		1 (50%)							2 (100%)
PS	1 (50.0%)	1 (50%)								2 (100%)
TGA	1 (50.0%)		1 (50%)							2 (100%)
COA	1 (50.0%)	1 (50%)								2 (100%)
Multiple lesion	5 (50.0%)	2 (20%)	1 (10%)				2 (20%)			10 (100%)
Total	12 (14%)	5 (4.5%)	44 (40%)	6 (5.5%)	7 (6.4%)	15 (13.6%)	4 (5.5%)	14 (12.7%)	2 (1.8%)	110 (100%)

Note: Parenthesis indicate percentage in respect of total no. of individual defect.

 Table 6. ECG findings in CHD patients (n-110).

Lesion	Electrocardi	Electrocardiogram									
Lesion	Normal	RVH	RVH+RAD	LVH	LVH+LAD	RVH+LVH+P-Pulmonale	Total				
VSD	16 (37.2%)	0 (0%)	2 (4.7%)	2 (4.7%)	20 (46.5%)	3 (7%)	43 (100%)				
ASD	2 (15.4%)	0 (0%)	4 (30.8%)	1 (7.7%)	5 (38.8%)	1 (7.7%)	13 (100%)				
TOF	0 (0%)	2 (11.8%)	14 (82.4%)	0 (0%)	1 (5.9%)	0 (0%)	17 (100%)				
PDA	5 (26.3%)	0 (0%)	1 (5.3%)	1 (5.3%)	12 (63.9%)	0 (0%)	19 (100%)				
A-V canal	1 (50%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (50%)	2 (100%)				
PS	0 (0%)	0 (0%)	2 (100%)	0 (0%)	0 (0%)	0 (0%)	2 (100%)				
TGA	1 (50%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (50%)	2 (100%)				

I:	Electrocardi	Electrocardiogram											
Lesion	Normal	RVH	RVH+RAD	LVH	LVH+LAD	RVH+LVH+P-Pulmonale	Total						
COA	1 (50%)	0 (0%)	0 (0%)	0 (0%)	1 (50%)	0 (0%)	2 (100%)						
Multiple lesion	5 (50%)	0 (0%)	5 (50%)	0 (0%)	0 (0%)	0 (0%)	10 (100%)						
Total	31 (28.2%)	2 (1.8%)	28 (25.5%)	4 (3.6%)	37 (35.6%)	6 (5.5%)	110 (100%						

Note: Parenthesis indicate percentage in respect of total no. of individual defect.

Table 7. Echocardiographic findings of patients under study (n-110).

E: 1:	Ecocardi	Ecocardiography									
Finding	VSD	ASD	TOF	PDA	A-V canal	PS	TGA	COA	Multiple lesion		
Di la	0	0	2	0	0	0	0	0	0		
Right venous hypertrophy	.0%	.0%	100.0%	.0%	.0%	.0%	.0%	.0%	.0%		
D A D + DV/H	2	4	14	1	0	2	0	0	5		
RAD+RVH	7.1%	14.3%	50.0%	3.6%	.0%	7.1%	.0%	.0%	17.9%		
	2	1	0	1	0	0	0	0	0		
Left venus hypertrophy	50.0%	25.0%	.0%	25.0%	.0%	.0%	.0%	.0%	.0%		
LADIMI	20	4	1	11	0	0	0	1	0		
LAD+LVH	54.1%	10.8%	2.7%	29.7%	.0%	.0%	.0%	2.7%	.0%		
N DVOT	0	0	17	0	0	0	0	0	0		
Narrow RVOT	.0%	.0%	100.0%	.0%	.0%	.0%	.0%	.0%	.0%		
Oi di	0	0	17	0	0	0	0	0	0		
Overriding of aorta	.0%	.0%	100.0%	.0%	.0%	.0%	.0%	.0%	.0%		

Note: Parenthesis indicate percentage in respect of total no. of individual defect.

Table 8. Echocardiographic findings of VSD patients under study (n-43).

Types of VSD	Frequency	Percentage	
Peri membranous	23	53.5	
Muscular	15	34.9	
Supracrystal	5	11.6	
Small	7	16.3	
Medium	13	30.2	
Large	23	53.5	

Table 9. Echocardiographic findings of ASD patients under study (n-13).

Types of ASD	Frequency	Percentage
Osteum primum	1	7.7%
Osteum secundum	12	92.3%
Total	13	100.0%

Table 10. Complications of different CHD (n-110).

Complication	Types of Lo	Types of Lesion							
	VSD	ASD	TOF	PDA	A-V Canal	PS	COA	Multiple Lesion	- Total
Heart failure	6 (40%)	0 (0%)	0 (0%)	5(33.3%)	0 (0%)	0 (0%)	0 (0%)	4(26.7%)	15(100%)
Pneumonia	19(54.3%)	8(22.9%)	0 (0%)	6(17.1%)	0 (0%)	1(2.9%)	0 (0%)	1(2.9%)	35(100%)
Growth failure	3(13%)	5(21.7%)	10(43.5%)	2(8.7%)	1(4.3%)	1(4.3%)	1(4.3%)	0 (0%)	23(100%)
Recurrent respiratory tract infection	8(57.1%)	0 (0%)	0 (0%)	3(21.4%)	1(7.1%)	0 (0%)	1(7.1%)	1(7.1%)	14(100%)
Cerebral abscess	0 (0%)	0 (0%)	4(100%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	4(100%)
Pneumonia with Heart failure	7(70%)	0 (0%)	0 (0%)	3(30%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	10(100%)
Total	43(42.6%)	13(12.9%)	14(13.9%)	19(18.8%)	2(2%)	2(2%)	2(2%)	6(5.9%)	101(100%)

Note: Parentesis indicate percentage in respect of total no. of individual defect.

4. Discussion

This prospective study was conducted in the department of pediatrics of Rajshahi medical college & hospital from July, 2011 to December, 2011. Babies from birth to children up to 12 years of age were included in this study. The aim of this study was to know the clinical pattern of congenital heart disease in this hospital. In this study the commonest type of

Congenital heart disease was ventricular septal defect. This correlates with many studies. [9-13] But this differs from Rahman et al, Siddique et al and Fatema et al [14-18]. They found ASD the commonest lesion. This difference in observation might be due to that Rahman et al and Siddique et al included many adult patients in their study. [16-18] A significant proportion of VSD close spontaneously before adulthood and some untreated patients with large VSD die in childhood from heart failure. On the other hand, ASD

patients may remain asymptomatic in childhood and are diagnosed for the first time when they are adult. The study subject of Fatema et al were all newborn and many small sized VSD and most of the child with TOF may not manifest by that time. [14, 15] However all these studies found TOF as the commonest cyanotic congenital heart disease [10-20]. This finding is quite similar to the current study. In this study male and female ratio was 2:1, of which males are predominant in ASD, TOF, COA, TGA, multiple lesions, PS, A-V canal defect and single ventricle with single AV canal defect whereas females were more frequently noted in VSD and PDA. This gender distribution correlates partially with the observation of Mollah et al, Hussain et al and Rao & Reddy. [21-23] Of the different clinical features, fast breathing, chest indrawing, cough, poor weight gain, feeding problems, anaemia, cyanosis, clubbing - were the major ones and this observation were correlated well with other studies in Bangladesh [21], India [24, 25] and western countries [26, 27]. In this study we found a significant number of ASD cases were asymptomatic and admitted in hospital for some other disease condition. These cases were diagnosed incidentally during routine systemic examination. A small ASD can remain asymptomatic throughout life. [28] Cardiac findings revealed murmur with or without thrill were the most frequently observed feature. Pansystolic murmur was found in all (100%) cases of VSD, similar with Keith [29]. Ejection systolic murmur was present in all (100%) cases of TOF; consistent with the findings of Naik et al [30] and also 76.9% cases of ASD; consistent with Siddique et al [17], Continues machinery murmur was found in 100% cases of PDA; similar to Siddique et al. [17] In this study, most frequent associated disease with CHD was pneumonia present in 35% cases and commonly observed complications were heart failure 15% and growth failure 23%. Fast breathing, chest indrawing, cough, poor weight gain, feeding problems, anemia, cvanosis, clubbing, easy fatigability, recurrent chest infection and murmur detected in routine cardiac examination. Features of heart failure are common mode of presentation of congenital heart disease. Without correction of congenital heart defect outcome of the patients are not satisfactory.

Limitations of the study

This cross-sectional study was conducted in a single community. Limitations of this study were this hospital lacks facilities for curative treatment of different congenital heart disease and this study was done in a selected group of patients. So, an extensive study with large sample size and longer period of time should be conducted to get a real picture of the problem; so that cases can be recognized earlier and prompt action can be undertaken.

5. Conclusion and Recommendations

All newborn babies should be examined thoroughly for any evidence of CHD and a follow up examination should be advised in late infancy. Children with undue fatigability, recurrent chest infection, failure to thrive should give due attention to exclude CHD.

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