TAJ December 2008; Volume 21 Number 2

ISSN 1019-8555
The Journal of
Teachers Association
RMC, Rajshahi

Original Article

Pattern and Clinical Profile of Congenital Heart Disease in A Teaching Hospital

L Shamima Sharmin[1], M Azizul Haque[2], M Iqbal Bari [3], M Ayub Ali[4]

Abstract

Objectives: To see the (a) type of congenital heart disease, (b) Clinical presentation of the cases, (c) association with extra-cardiac anomalies and disease, (d) complications of different CHD, (e) outcome of patients during hospital stay. Methodology: it was a prospective study conducted in the department of pediatrics of Rajshahi Medical College & Hospital over a period of one year.115 children from birth to 12 years of age who had congenital heart disease confirmed by echocardiography were included. All patients were treated conservatively and observed for immediate out come during the hospital stay. Result: major types of CHD were VSD (42.6%), TOF (18.3%), ASD (14.8%), PDA (7.8%). Male outnumbers female child. Common symptoms were breathlessness (60%), fatigue (54.8%), cough (43.5%), poor weight gain (41.7%), recurrent chest infection (34.8%), fever (28.7%), feeding problems (26.1%), palpitation (21.7%) and bluish discoloration of lips and fingertips (20%). Murmur with or without thrill and cardiomegaly was the most important cardiac finding. Frequently observed complications were heart failure, pulmonary hypertension and growth failure

TAJ 2008; 21(2): 58-62

Introduction

Congenital heart disease (CHD) is the commonest of all congenital lesions and is the most common type of heart disease among children.[1] Congenital heart disease, in a definition proposed by Mitchell et al is "a gross structural abnormality of the heart or intrathoracic great vessels that is actually or potentially of functional significance." [2]

The incidence of congenital heart disease is approximately 8 per 1000 live birth, with a higher rate in stillbirth, spontaneous abortion and prematurity.[3,4] It is believed that this incidence has remained constant worldwide.[5] From population survey in Bangladesh, the prevalence of congenital heart disease was found 0.18%.[6] World Health Organization (WHO) reports, among all cardiovascular disease, the incidence of Congenital heart disease in Bangladesh is 6%, 15% in India, 6% in Burma, 10% in Srilanka.[7] The relative frequency of the most common lesions varies with different reports but nine common lesions form 80% of congenital heart disease (Jackson et al).[8]

These are Ventricular Septal Defect (36%), Atrial Septal Defect (5%), Patent Arterial Duct (9%), Atrioventricular Septal Defect (4%), Pulmonary Stenosis (9%), Aortic Stenosis (5%), Coarctation of Aorta (5%), Transposition of Great Arteries

[1] Student, FCPS part-2, Bangabandhu Sheikh Mujib Medical University, Dhaka.
[2] Resident Physician, Department of Medicine, Rajshahi Medical College Hospital, Rajshahi.
[3] Professor, Department of Paediatrics, Rajshahi Medical College.
[4] Assistant Professor, Department of Cardiology, Rajshahi Medical College.

(4%), Tetralogy of Fallot (4%). The other 20% of congenital heart disease consists of many rare or complex lesions.[8] Congenital heart disease as a whole occurs with equal frequency in male and females but some lesions such as aortic stenosis, coarctation of aorta, transposition of great vessels and tetralogy of Fallot are more common in males whereas atrial septal defects are more common in females. [9] The cause of most Congenital heart defects is unknown. Most cases of congenital heart disease are thought to be multifactorial and result from a combination of genetic predisposition and environmental stimulus.[9]

The clinical presentation of congenital heart disease varies according to the type and severity of the defect.[10] In neonatal period the presenting feature 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).[10] In infancy and childhood the usual presenting features are cyanosis, digital clubbing, murmur, syncope, squatting, heart failure, arrhythmia, failure to thrive.[11] The adolescent and adults present with heart failure, murmur, arrhythmia, cyanosis, hypertension, late consequences of previous cardiac surgery (e.g., arrhythmia, heart failure).[11]

As a common congenital anomaly, CHD not only contribute to a significant morbidity and mortality but also causes a tremendous psychological stress and economical burden to the whole family. However, if the problems are recognized at earlier age, the chance of long term complications are less and the outcome is better. As a result of improved medical and surgical management, more children with CHD are surviving into adolescence and adulthood.[12] Thus there is a need for an increased awareness amongst general physicians and cardiologists of the problem posed by these individuals. Except a few scattered observations, the incidence and detail clinical profile of CHD in Bangladeshi children are not well documented. This study was undertaken to find out the pattern and clinical profile 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.

Material and Methods

This prospective study was carried out over a period of one year among the admitted children in the department of paediatrics of Rajshahi medical college & hospital, age ranging from newborn to 12 years. The cases were included in the study when the diagnosis of CHD was established by echocardiography. After enrolment, detailed history of the studied patients was taken to know their clinical presentation. Moreover, thorough clinical examination was done to evaluate specific heart lesion. Apart from echocardiography other investigations like chest X-ray, electrocardiography and other relevant investigations were also done. All the data related to history, clinical examination, investigation and treatment were noted in a preformed datasheet with structured questionnaire. After checking all the data analysis was done using SPSS version 12.

Results

In this study the total number of patients suffering from CHD was 115. Commonest lesion was VSD (42.6%) followed by TOF (18.3%), ASD(14.8%), PDA(7.8%) and others (15.5%) [Table-1]. Male and female ratio was 1.3:1. Males were predominant in VSD, TOF, A-V canal defect and single ventricle with single A-V canal defect. Females were predominant in ASD, PDA, COA, TGA and multiple lesion but equal distribution in PS [Fig-1]. Major clinical presentations were breathlessness (60%), fatigue (54.8%), cough (43.5%), poor weight gain (41.7%), recurrent chest infection (34.8%), feeding problem (26.1%), cyanosis (20%), clubbing (17.4%), oedema (10.4%), anaemia (18.3%), polycythemia (19.1%), tachycardia (37.4%) and fast breathing (43.5%) [Table-2 & 3]. Murmur with or without thrill and cardiomegaly were the most important cardiac finding [Table-4]. Frequently observed complications were heart failure (18.3%), pulmonary hypertension (13%) and growth failure (41.7%) [Table-5].

Table – 1: Types of congenital heart disease in all patients from birth to 12 years of age (N-115)

Type of lesion	No. of patient	Percentage
VSD	49	42.6%
TOF	21	18.3%
ASD	17	14.8%
PDA	9	7.8%
A-V canal defect	4	3.5%
PS	2	1.7%
COA	2	1.7%
TGA	1	0.9%
Single ventricle with single AV canal defect	1	0.9%
Multiple lesions	9	7.8%
Total	115	100%

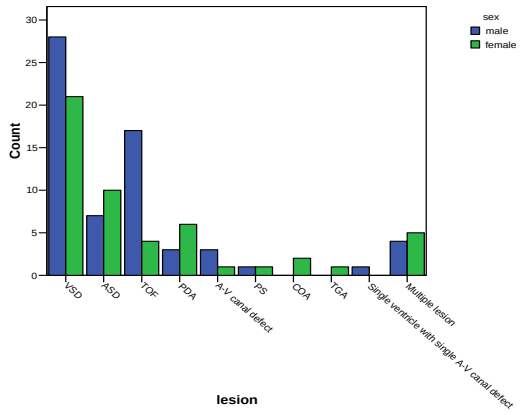

Fig: Sex distribution of different congenital heart disease (N-115)

Table-2: Symptomatology of congenital heart disease (N=115)

Symptoms	No. of cases	Percentage
Breathlessness	69	60%
Fatigue	63	54.8%
Cough	50	43.5%
Poor weight gain	48	41.7%
Recurrent chest infection	40	34.8%
Fever	33	28.7%
Feeding problem	30	26.1%
Palpitation	25	21.7%
Cyanotic spell	15	13%
Convulsion	2	1.7%

Table-3: Important physical findings in CHD (N=115)

Physical findings	No. of cases	Percentage
Dyspnea	53	46.1%
Fast breathing	50	43.5%
Tachycardia	43	37.4%
Chest indrawing	38	33%
Crepitation	29	25.2%
Cyanosis	23	20%
Polycythemia	22	19.1%
Anemia	21	18.3%
Enlarged tender liver	21	18.3%
Clubbing	20	17.4%
Rhonchi	15	13%
Edema	12	10.4%
Engorged neck vein	8	7%
Hypertension	2	1.7%
Radio-femoral delay	2	1.7%

Table-4: Important cardiac findings in different CHD (N=115)

Type of Lesion	Cardio-megaly (%)	Thrill (%)	Palpable P_2 (%)	Paraster-nal heave (%)	Fixed splitting S_2 (%)	Single S_2 (%)
VSD	39(79.6)	45(91.8)	7(14.3)	8(16.3)	0	0
ASD	1(5.9)	0	3(17.6)	3(17.6)	17(100)	0
TOF	3(14.3)	7(33.3)	0	0	0	14(66.7)
PDA	4(44.4)	9(100)	2(22.2)	2(22.2)	0	0
PS	1(50)	2(100)	1(50)	1(50)	0	2(100)
COA	2(100)	0	0	0	0	0
AV canal defect	2(50)	0	0	0	0	0
TGA	1(100)	0	0	0	0	0
Single ventricle	0	0	0	0	0	0
Multiple lesions	7(77.8)	6(66.7)	1(11.1)	1(11.1)	1(11.1)	0
Total	60(52.2)	69(60)	14(12.2)	15(13)	18(15.6)	15(13)

Table-5: Complications of different CHD (N=115)

Complications	VSD (N=49)	ASD (N=17)	TOF (N=21)	PDA (N=9)	Others (N=19)
Heart failure	15(30.6)	0	1(4.8)	1(11.1)	4(21.1)
PH	7(14.2)	3(17.6)	0	4(44.4)	1(5.2)
Growth failure	21(42.8)	5(29.4)	17(80.9)	3(33.3)	2(10.5)
Rec. chest infection	22(44.9)	7(41.1)	1(4.8)	5(55.5)	2(10.5)
Cerebral abscess	0	0	1(4.8)	0	0

Discussion

In this study the commonest type of Congenital heart disease was ventricular septal defect. This correlates with many studies [2,3,13,14]. But this differs from Rahman et al, Siddique et al and Fatema et al. [15,16,17] 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.[15,16] 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.[17] However all these studies found TOF as the commonest acyanotic congenital heart disease.[2,3,13,14,15,16,17] This finding is quite similar to the current study.

In this study male and female ratio was 1.3:1, of which males were found more frequent in VSD, TOF, A-V canal defect and single ventricle whereas females were more frequently noted in ASD, PDA, COA, and TGA but equal distribution in PS. This gender distribution correlates partially with the observation of Mollah et al and Rao & Reddy.[14,18]

Of the different clinical features, breathlessness, cough, fatigue, poor weight gain, feeding problems, palpitation, cyanosis, clubbing oedema, - were the major ones and this observation were correlated well with other studies in Bangladesh [13,1416] , India [19,20] and western countries [21,22,23]. 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.[11]

Cardiac findings revealed murmur with or without thrill and cardiomegaly were the most frequently observed feature. Cardiomegaly was found mostly in VSD cases. This observation is similar to that of Keith.[24] Pansystolic murmur was found in all (100%) cases of VSD, similar with Keith.[24] Ejection systolic murmur was present in all (100%) cases of TOF; consistent with the findings of Naik et al [25] and also 88.2% cases of ASD; consistent with Siddique et al [16]. Continues machinery murmur was found in 100% cases of PDA; similar to Siddique et al.[16]

Conclusion

In the light of analysis and interpretation of the present study findings following recommendations can be made:

1) All newborn babies should be examined thoroughly for any evidence of CHD and a follow up examination should be advised in late infancy.
2) Children with undue fatigability, recurrent chest infection, failure to thrive should give due attention to exclude CHD.
3) Heart failure in infancy and childhood should be evaluated cautiously for presence of CHD.
4) Local pediatricians should be trained about specialized cardiac care and specialized cardiac centers should be established locally so that patients can be managed effectively without delay.

References

1. Schoen FJ. The Heart. In: Cortan RS, Kumar V, Robins SL, 6th ed. Robins Pathologic Basis of Disease. Philadelphia: W.B. Saunders Company, 1999; 543-600
2. Mitchell S.C., Korones S.B. and Berendes H.W., Congenital heart disease in 56,109 births. Incidence and natural history. Circulation 43 (1971), pp. 323–332
3. Fyler DC, Buckley LP, Hellenbrand WE, Cohn HE. Report of the New England Regional Infant Cardiac Program. Pediatrics, 1980; 65(2) Suppl : 375-461.
4. Jordan S. C., Scoll O., 3rd ed. Heart disease in Paediatrics. London: Butterwoths, 1989:38
5. Abdulla R. What is the prevalence of congenital heart disease? Ped Cardiol 1997; 18: 268
6. Malik A: Congenital and Acquired heart disease. A survey 7,062 Persons, Bangladesh Medical Research council Bulletin.2: 115, 1976.
7. Malik A : Problems of Cardiovascular disease in Bangladesh & other developing country. Proceeding of the Bangladesh-Japan joint conference on CVD, Dhaka, Bangladesh, 1984
8. Jackson M, Walsh KP, Peart I, Arnold R. Epidemiology of congenital heart disease in Merseyside – 1979 to 1988. Cardiol Young 1996: 6:272-280.
9. Bernstein D. Congenital heart disease. In Behrman RE, Kligman RM, Jenson HB, 17th ed. Nelson textbook of Pediatrics. Philadelphia: Saunders, 2004; 1499-1553.
10. Kitchiner D J. Cardiovascular disease. In: Mclntosh N, Helms PJ, Smyth RL, 6th ed. Forfer & Arneil's Textbook of Pediatrics. Edinburgh: Churchill Livingstone, 2003; 815-888
11. Bloomfield P, Bradbury A, Grubb NR, Newby DE. Cardiovascular Disease. In: Boon NA, Colledge NR, Walker BR, 20th ed. Davidson's Principle and Practice of Medicine. Edinburgh: Churchill Livingstone, 2006; 519-646
12. Camm AJ, Bunce NH. Cardiovascular Disease. In: Kumar P, Clark M, 6th ed. Kumar & Clark Clinical Medicine. Edinburgh: ElsevierSaunders, 2005; 725-872
13. Hussain M, Hossain M, Amin SK, Molla MR. Pattern of Congenital Heart Disease in Dhaka Shishu Hospital. D S (Child) H J 1992; 8: 35-46
14. Mollah MAH, Begum NA, Islam MN, Mahmud RS, Haq MA, Nahar N, Rashid MA.Clinical Profile of Congenital Heart Diseases (CHD): An Analysis of 218 Cases. Bangladesh heart J 2002; 17: 62-67.
15. Rahman S, Ahmed MN, Rahmatullah KHI, Alam MS. The Incidence of Congenital Heart Diseases Diagnosed by Non-Invasive Technique – Ten Years Study in Bangladesh. D S (Child) H J 1992; 8 : 5-15
16. Siddique FM, Kamal SMM, Huq KMHSS. Clinical Presentation of Congenital Heart Disease in hos[italized patients. Bangladesh Heart Journal 1989; 4: 13-17.
17. Begum NNF, Ahmed QS. Pattern of Heart disease among neonates and their outcome: one year experience in non- invasive cardiac laboratory of Combined Military Hospital, Dhaka. Bangladesh J child health 2001; 25: 48-52.
18. Rao VS, Reddy R. Profile of congenital heart disease in children. Indian J Pediatr 1974; 41: 244-248.
19. Vashestha VM, Kalra A, Kalra K, Jain VK. Prevalence of Congenital Heart Disease in School Children. Indian J Pediatr. 1993;30: 1337-1340
20. Khalil A, Aggarwal R, Thirupuram S, Arora R. Incidence of Congenital Heart Disease among Hospital Live Births in India. Indian J Pediatr. 1994; 31: 519-527.
21. Cameron JW, Rosenthal A, Olsan AD. Malnutrition in hospitalized children with congenital heart disease. Arch Pediatr Adoles Med 1995; 149: 1098-1102.
22. Silove ED. Assessment and management of congenital heart disease in the newborn by district pediatrician. Archives of Diseases in childhood 1994; 71-74
23. Hag AI. Pattern of Congenital Heart Disease in Sudanese Children. East Afr Med J 1994; 71: 580-586
24. Keith JD. Ventricular septal defect. In: Keith JD, Rowe RB, Viad P, 3rd ed. Heart disease in Infancy and childhood. Newyork: Mac Millan publishing Co., 1978; 337-350
25. Naik S, Tandon R, Gopinath N et al. Tetralogy of Fallot : a clinical profile. Indian Heart J 1972; 24: 14-15

All correspondence to:
Laila Shamima Sharmin
Student, FCPS part-2,
Bangabandhu Sheikh Mujib Medical University
Dhaka.