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Original Research Article

Study of complications of congenital heart diseases with special reference to pulse oximetry and anthropometry

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ABSTRACT

Background: This study was done to determine prevalence of congenital heart disease (CHD), its complications especially effect on growth parameter and role of pulse oximeter as a diagnostic modality.
Methods: This study was a hospital based prospective observational study conducted on hospitalized children in paediatric wards and PICU of Civil hospital, B. J. medical college Ahmedabad, for one year which is a tertiary care teaching hospital.
Results: Prevalence of CHD in our institute was 1.2% during the period of one year. Signs of failure (tachycardia, tachypnoea, pallor and hepatomegaly) were seen in more than 50% of patients. Nearly 29.5% patients had hypoxia on arrival on pulse oximeter reading while 74% patients had normal saturation. Complications commonly found in CHD are congestive heart failure and growth failure.
Conclusions: CHD is a chronic disease and has all its consequences; complications are commonly found in congenital heart diseases are congestive heart failure and growth failure. Pulse oximeter can be used as screening test but not as the reliable diagnostic method.

Keywords: Congenital heart disease, Prevalence, Pulse oximetry, Complications, Growth failure

INTRODUCTION

CHD are a group of gross structural abnormality of heart that is present at birth.[1] CHD occurs in approximately 0.8% of live births.[2] With various diagnostic modalities and screening techniques CHDs are now diagnosed early. One of them is pulse oximeter; it is readily available can easily detect certain cyanotic CHD in which early detection and intervention is must. CHD is a chronic systemic disease with associated co-morbidities and complications among them. Many studies have been conducted on CHDs but most of them have studied epidemiology and clinical profile; there are very few data available on complications of CHD. Children with CHD are at an increased risk for many complications directly related to the malformation or indirectly in form of growth failure. Malnutrition among these children contributes to their inherent risk of infections (as in lesions with left to right shunt) and the risk of death. In CHD, infections and malnutrition form a vicious cycle which is difficult to break. So, early diagnosis and prevention of complications is very important in CHDs as it affect prognosis and overall outcome.

METHODS

A hospital based prospective observational study carried out in our paediatric wards and PICU at Civil hospital, B. J. medical college, Ahmedabad for one year during period of June 2018 to June 2019.

All patients of CHD of age group one month to 12 years who were admitted were included in study.

Patients who already had gone through surgical intervention, who had major congenital malformation other than CHD, patients with endocrinal cause of growth retardation were excluded.

A prior institutional ethical committee approval was obtained.

A detailed history and clinical examination were carried out through a preformed performa in patients in whom CHD was suspected and evaluated according to Nada's criteria. Patients with a clinical diagnosis of heart disease were further evaluated with pulse oximetry, chest radiography, twelve-lead electrocardiograms. The confirmation of the diagnosis was done using 2D echocardiography by a trained cardiologist at U. N. Mehta institute of cardiology (our allied institute for cardiology).

Nutritional status was assessed by detailed dietary history (recall method) and various age specific criteria were applied to categorise their status of malnutrition if any.

The criteria applied for age group <5 years were: IAP classification of malnutrition in patients with age group <5 years;[3] WHO classification of malnutrition;[4] UNICEF criteria for detecting severe acute malnutrition (SAM) in age group of 6-60 months.[5]

The criteria applied for age group >5 years to 12 years completed was that of BMI (body mass index). A child's BMI percentile is calculated by comparing that child's BMI to growth charts for children who were the same age and sex as that child.

IAP adaptation of WHO growth charts were plotted in each patient to study the growth faltering.[6]

Information regarding demographic data and detailed history was collected using a structured questionnaire. Necessary investigations were ordered as per individual clinical need for each enrolled patient. Complete blood count, renal function tests, serum electrolytes were sent in all patients. Liver function test, blood gas analysis and other specific investigations were ordered as and when required. Data was recorded in excel sheet and summarized in the form of frequency tables and conclusions were drawn accordingly. Appropriate statistical analysis was done using software operated by social science statistics.

RESULTS

Total admissions in our institute during study period were 8297, among which total no of patients diagnosed with CHDs were 105. Thus, prevalence of 1.2% was found during this study period.

Among which male children were 56 (53.3%) and female children were 49 (46.6%).

The present study shows male to female ratio of 1.1:1 with slightly higher male preponderance.

In present study highest frequency of CHD distribution was found in the age group of infancy which indicated most of the CHDs present early; however nearly 5% patients were also diagnosed late in early adolescent who were relatively asymptomatic during their infancy and probably due to less severity of CHD (Figure 1).

Table 1 summarised the presenting complaints of patients among which most of the patient of CHD presented with cough (60.9%) and difficulty in feeding (52.5%) as presenting symptom which was comparable to Kumar et al study accounting 51.7% and 50% respectively; although Karthiga et al study showed more number of patients with fever as presenting complaint (87.6%).

On clinical examination majority of the patients had murmur as prominent sign comparable to Karthiga et al study and Nithya et al study.[9] Among those patients with murmur, 20% patients had palpable thrills indicating severity of CHD.

Among 105 patients of CHDs acyanotic CHD accounted nearly 77% (n=81) of total CHDs and cyanotic CHDs accounted 23% (n=24) of total CHD.

Present study showed that out of 105 patients 78 patients had normal saturation between 90-99%, while 22 patients had hypoxia on presentation (Figure 2).

In present study out of 81 patients of acyanotic CHD 57 patients discharged (70.3%); highest number of discharges were done within 8-14 days of treatment course whereas 22.2% mortality in acyanotic heart disease among which nearly 55% patient died within 4-7 days of stay at hospital.

Meanwhile out of 24 patients of cyanotic CHD 15 patients were discharged (60%); highest no of discharges were done within 4-7 days of treatment course, with 16.6% mortality in cyanotic heart disease among which nearly 50% patient died within 1-3 days of stay at hospital.

Overall mortality of CHD was found 20.9% in present study. Mortality in acyanotic CHD was more compared to cyanotic CHD; although, with p=0.2507 it is not statistically significant.

In present study out of 18 patients expired of acyanotic CHD; 12 patient were of VSD among which 8 patients (72.7%) expired due to congestive heart failure indicating severity of disease.

Table 1: Comparison study of presenting complaint.

Symptoms	No of patients (n=105)	Kumar study et al[7] (n=105)	Karthiga study et al[8] (n =66)
	N (%)	N (%)	N (%)
Cough	64 (60.9)	58 (51.7)	-
Difficulty in feeding	58 (55.2)	56 (50)	12 (18.1)
Fever	52 (49.5)	38 (33.9)	52 (78.7)
Difficulty in breathing	43 (40.9)	-	48 (72.7)
Poor weight gain	22 (20.9)	-	27 (40.9)
Decreased urine output	9 (8.5)	-	-
Seizures	1 (0.9)	2 (1.8)	-
Chest pain	1 (0.9)		-

Table 2: Low SpO_2 associatio n with acyanotic CHD.

Acyanotic CHD	No. of patients (n=81)	LRTI	SpO_2 <90% on admission	SpO_2 >90 with oxygen
Ventricular septal defect	05	Yes	Yes	Yes
Atrial septal defect	01	Yes	Yes	Yes
Patent ductus artriosus	01	Yes	Yes	Yes

Table 3: Low pulse oxymetry finding associated with cyanotic CHD.

Cyanotic CHD	No. of patients (n=24)	LRTI found in no. of patients	SpO_2 <90% on admission	SpO_2 <90% after oxygen
Tetralogy of fallots	21	8	Yes	Yes
Double outlet right ventricle	2	-	Yes	Yes
Transposition of great vessels	1	-	Yes	Yes
Total anomalous pulmonary venous circulation	-	-	-	-

Table 4: Pulse oximetry and ABGA findings in acyanotic CHDs with LRTI.

Acyanotic CHD	SpO_2	PaO_2
VSD		
1	80±3	92
2	85±3	94
3	87±3	122
4	86±3	88
5	80±3	95
ASD	86±3	112
PDA	84±3	100

Table 5: Pulse oximetry and ABGA findings in cyanotic CHDs with LRTI.

Cyanotic CHD	SpO_2	PaO_2
TOF patients		
1	70±3	65
2	77±3	72
3	75±3	60
4	72±3	58
5	65±3	78
6	80±3	80
7	70±3	73
8	74±3	55

Table 6: Assessment of wasting.

Weight for age	PEM grade (acc. to IAP classification)	No. of pateints (n=100)	Acyanotic (n=78)	Cyanotic (n=22)	P value
		N (%)	N (%)	N (%)	
>80	Normal	38 (38)	28 (35.8)	10 (45.4)	
71 -80	Grade 1	20 (20)	18 (23.0)	2 (9.09)	
61-70	Grade 2	10 (10)	6 (7.6)	4 (18.1)	0.263
51-60	Grade 3	15 (15)	11 (14.1)	4 (18.1)	
<50	Grade 4	17 (17)	15 (19.2)	2 (9.0)	

Table 7: Assessment of stunting.

Height for age	Total no. of pateints (n=100)	Acyanotic (n=78)	Cyanotic (n=22)	P value
	N (%)	N (%)	N (%)	
Normal	63 (63)	57 (73.0)	6 (27.2)	
BW 2SD-3SD	23 (23)	14 (17.9)	9 (40.9)	0.0076
<3SD	14 (14)	7 (8.9)	7 (31.8)	

Table 8: Comparison of complications between acyanotic and cyanotic CHDs.

Complications	No. of patients of acyanotic CHD (n=81)	No. of patients of cyanotic CHD (n=24)	P value
	N (%)	N (%)	
Growth failure	44 (54.3)	15 (62.5)	0.4781
Pulmonary hypertension	31 (38.2)	04 (16.6)	0.0486
Congestive heart failure	22 (27.1)	15 (62.5)	0.0014
Recurrent infections	8 (9.8)	10 (41.6)	0.00032
Infective endocarditis	2 (2.4)	-	-
CNS complications	-	1 (4.1)	-

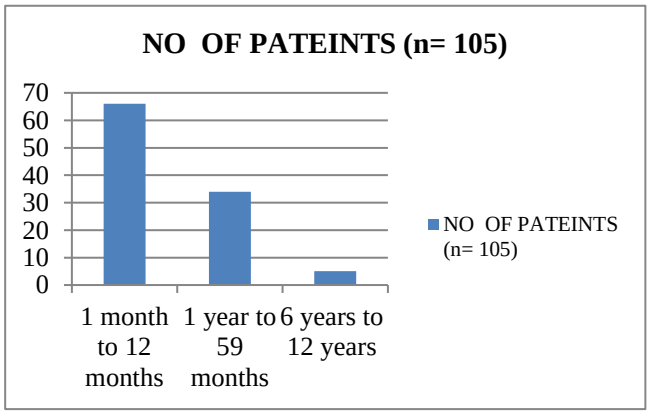

Figure 1: Age presentation.

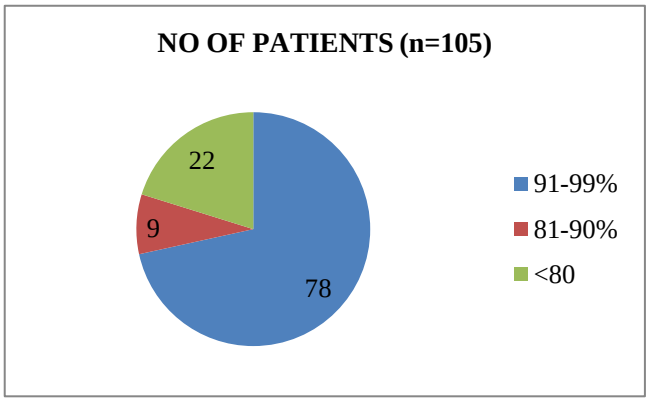

Figure 2: Pulse oximetry findings.

In cyanotic CHD out of 24 patients 4 patients expired of which majority (75%) died due to CCF.

DISCUSSION

In community-based studies from India the prevalence of CHD ranged from 0.8-5.2/1000 patients.[10]

According to a systemic review and meta-analysis, the reported total congenital heart disease birth prevalence increased substantially from 0.6 per 1,000 live births (95% confidence interval CI: 0.4 to 0.8) in 1930 to 1934 to 9.1 per 1,000 live births (95% CI: 9.0 to 9.2) after 1995. The increase over time was S-shaped, with a first steep increase from 1930 to 1960, followed by

stabilization around 5.3 per 1,000 live births from 1961 to 1975, a second steep increase from the late 1970s until 1995, and eventually stabilization around 9.1 per 1,000 live births in the last 15 years.[11]

In the present study as Figure 1 shows that the most of the patients were under infancy period about 62.8% which indicates most of the CHDs present early; however nearly 5% patients were also diagnosed late in early adolescent who were relatively asymptomatic during their infancy and probably due to less severity of CHD.

Acyanotic CHD accounted nearly 77% of total CHDs in present study. Majority patients of acyanotic CHD found in my study were of ASD. Only 12 patients in our study had PDA. As per literature, patent ductus arteriosus occurs in 5% to 10% of all CHDs, excluding premature infants.[12] Thus very few patients of PDA were seen after infancy, the similar observation was supported by our study as well as both reference studies.

In present study cyanotic CHD accounted for nearly 23%, among which highest frequency was of TOF.

Acynotic CHDs usually maintain saturation above 90% which can fall below 90%, due to lower respiratory tract infections like pneumonia or bronchitis it can get improved with oxygen and antimicrobial therapy (Table 2). However, 1 patient of VSD associated with pneumonia did not improve in our study and cyanosis remained the same which was due to eisenmengerization and saturation of that patient was found to be 75-78%.

All patients with cyanotic CHDs had low oxygen saturation which did not improve on oxygenation (Table 3) which was probably due to pathophysiology of cyanotic CHD itself.

Despite the fact that out of 21 patients of TOF 8 patients had LRTI, oxygen therapy had no influence on pulse oximetry finding. Hence, pulse oximetry was not a reliable method to find out hypoxia due to other causes (e.g. LRTI) in patients with cyanotic CHD. And hence, other methods like ABGA can be considered for assessment and monitoring of such patients in ICU settings.

Table 4 shows that the most of the patients with acyanotic CHDs who had shown hypoxemia on pulse oximeter were not found to be hypoxemic on blood gas analysis. It showed weakly statistically significant correlation (correlation coefficient r value 0.3486).

Pulse oximetry was found to be insufficiently sensitive to rule out the presence of pneumonia in children 2 years of age who presented with respiratory complaints to an emergency room.[13]

In all patients of TOF almost similar findings were seen in readings of pulse oximeter and ABGA (Table 5). There was moderate positive correlation between pulse oximeter and ABGA findings (correlation coefficient r value 0.5889).

In terms of processes, pulse oximetry had been shown to reduce the number of arterial blood gas samples taken in various populations. However, sometimes this reduction had been as modest as 10% and pulse oximetry may not always reduce arterial blood gas sampling and analysis. Using standardized protocols for reducing blood gas sampling and analysis along with pulse oximetry will help to ensure the most efficient use of blood gas analyses.[13]

Table 6 shows that out of 100 patients who were below 5 years of age about 38% patients had normal nutrition; while 62% patients some or other form of malnutrition. Out of these patients with malnutrition 17% patients had severe wasting with their weight for age falling into range of <50.

Though the frequency of distribution of wasting was more common in acyanotic CHD compare to cyanotic CHD, the observation was not statistically significant (p value 0.263).

Previous data from developing country showed prevalence of pre-operative undernutrition in children with CHD was up to 45%.[15,16]

Severe wasting (grade 4) was seen in 17% of patients of CHD.

Table 7 shows that 63% of patients have normal height for age; however, 37% patients had some form of stunting. When stunting was compared between cyanotic and acyanotic it was found to be higher in cyanotic CHD which was statistically signigicant (p=0.0076).

As per literature, cyanotic patients were affected in growth, depending on the severity of tissue hypoxemia and on the degree of physiological adaptation. Weight and height were affected equally in cyanotic patients. Acyanotic lesions, especially in combination with septal defect, left to right shunt, will affect weight more than height. Acyanotic lesions were related to acute malnutrition (affecting weight predominently), whereas cyanotic lesions were related to chronic malnutrition (affecting weight and height both).[17]

Study showed majority of patients of acyanotic CHDs had normal height for age.

Table 7 shows that majority of patients (40.9%) of cyanotic CHD had stunting with height for age between 2SD-3SD.

Out of 100 patients below 5 years of age 21 patients had SAM. Out of 21% of SAM patients 90.4% patients were of acyanotic CHD, which was statistically not significant

(p=0.2436). 33.33% had weight for height Z score <3 SD, 23.8% had MAC <11.5 cm while b/l pedal odema was found in 42.85%. However, here we cannot attribute this odema due to SAM only as CCF also can present with b/l pedal odema as presenting complaint.

A high prevalence percentage of low WFH was reported most commonly in patients with chronic congestive failure, chronic shunt hypoxemia and nosocomial postoperative acute and chronic states. Various studies among children with various cardiac diseases (e.g. CHD, idiopathic dilated cardiomyopathy) showed prevalence rates between 18 and 64% on admission. The highest rates were found in cardiac surgical patients and in children with CHDs and left-to-right shunt.[17]

In the present study, only 5 patients were above 5 years of age with 90% of patients were undernourished.

We can see from Table 8 that growth failure was seen in more than half of the patients; pulmonary hypertension seen in more than 1/3rd of the patients; while only 2 patients had infective endocarditis of which both were belonging to the age group of 6-12 years.

The highest frequency of complication observed was congestive heart failure (62.5%) closely followed by growth failure (54.1%). Only one patient of cyanotic CHD (TOF) had CNS complication in form of brain abscess.

Growth failure was seen in higher no in case of acyanotic CHD compared to cyanotic CHD but it was not statistically significant.

However, pulmonary hypertension was common in acyanotic CHD while recurrent infections were more common in cyanotic CHD which was statistically more significant (p<0.005).

The limitations of present study included single center-based enrolment and no priority sample size calculation.

CONCLUSION

Pulse oximeter is an important tool for detecting hypoxia and monitoring of blood oxygen level in cases of CHD as non-invasive method. However, in resource constraint settings, it can be used as a safe non-invasive modality for early detection of hypoxia. Nevertheless, it has caveats with it as in cases of shock and hypoperfusion one cannot rely only on that.

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