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Prevalence of congenital heart disease in neonatal ICU in moderate high altitude

Bisht Dinesh[1], Negi Prakash Chand[1], Bhardwaj Parveen[2], Sood Mangla[2], Rana Jitender[1]
[1] Department of Cardiology, Indira Gandhi Medical College, Shimla, Himachal Pradesh, India
[2] Department of Pediatric, Indira Gandhi Medical College, Shimla, Himachal Pradesh, India

Abstract
534 neonates with saturation less than 95 percent with Persistent oxygen dependency, murmur, tachypnea more than 55 per minute were taken for Echocardiography in Kamla Nehru Hospital, a branch of IGMC Shimla. The prevalence of CHD in NICU was 15 percent which simulated with prevalence described previously. Clinical tools were at par with echo study.
Among Cyanotic CHD; TOF- 2 case, TGA- 1 case, DORV-1 case, Mitral Atresia -2 cases.
PPHN was seen in 250 cases. Among Acyanotic CHD, ASD-1 case, VSD- small-1 case, VSD Moderate size -1 case, Large VSD -2 cases, PDA- Large -31 cases, AVSD-1 case, Moderate MR -21 cases, Severe biventricular dysfunction-19 cases, Significant Biventricular hypertrophy-21 cases.
SHD was detected 2 -6 day of age with significant p value (0.004) as compared to Non SHD group. Oxygen saturation less than 95 percent was significantly associated with SHD with a significant p value (0.06).
Cyanotic CHD presented earlier than Acyanotic CHD with a significant p value (0.08). Higher maternal age showed significant correlation with Cyanotic CHD (p value - 0.04). Male babies had more cyanotic CHD than female babies (p value-0.07)
Gestational age, birth weight, tachypnea, retraction, tachycardia, paternal age did not show any significance with acyanotic or cyanotic CHD.
On logistic regression analysis, for diagnosis of SHD, HR increase by 10 per minute, 1.78(1.07-2.96) P value=0.02, and detection of murmur 3.0(1.29-7.3),p value=0.01 showed highly significant p values.
PPHN was seen in 50 percent of neonates. Sildenafil was started in neonates with Persistent oxygen dependency. The Hospital stay was reduced from 6 weeks to 3 weeks. The cause of Biventricular dysfunction was Birth asphyxia, HMD, Pneumonia with sepsis. The cause of Significant Mitral Regurgitation was LV dysfunction. The cause of Significant Biventricular hypertrophy was Gestational Diabetes and Inborn errors of metabolism.

Keywords: CHD- congenital heart disease, SHD- structural heart disease, HR-heart rate, RR-respiratory rate. PPHN-Persistent Pulmonary Hypertension

Introduction

High altitude has been linked with high incidence of CHDs like patent ductus arteriosus (PDA) and atrial septal defect (ASD) and their progression. Both PDA and ASD are suspected clinically and later confirmed on investigations, especially Echocardiography.
Failure of lower oxygen tension to constrict the ductus leads to patency of ductus arteriosus while the presence of high pulmonary vascular resistance and right atrial pressure at high altitude inhibits early closure of foremen ovale. With physical development of the child and stretching of fossa ovalis along with incompetence of flap, ASD is established. Analogy can be drawn from high prevalence of ASD in TOF as the right ventricular pressure is high and right ventricular compliance is low from birth.
At high altitude, these two anomalies are due to hypoxemia-induced failure of normal neonatal processes.
The prevalence of CHD in India varies from 2.25 to 5.2/1000 live births.
High altitude gives insight into the pathophysiology of both cyanotic and acyanotic heart disease in an interesting way. The patients with cyanotic CHD have more blunted hypoxic ventilatory response, which develops as early as 7–8 years, while the most blunted ventilatory response is seen in patients with maximum desaturation, which is corrected once the patient is surgically treated and normalized. Another important distinction between native highlander and patients with cyanotic heart disease is the fact that though both have arterial hypoxemia, highlanders have lowered alveolar oxygen tension. For patients with VSD, left to right shunting of blood decreases at high altitude. Patients with single ventricle physiology and postoperative Glenn and Fontan tolerate high altitude very poorly due to hypoxia and increased pulmonary vascular resistance.
Studies performed in Northern India, especially the hilly areas of Jammu and Kashmir, India, revealed that 88.5% of all CHD were the acyanotics, and 11.5% were cyanotic heart patients. Among the acyanotic heart diseases, VSD was the most frequent lesion seen in 31.2%, followed by PDA in 24.3% children. Among the cyanotic heart diseases, TOF was the most frequent, cyanotic heart disease seen in 48.0% patients. The prevalence of CHD was 1.1/1000 hospital attending patients.
In a Chinese study the incidence and spectrum of CHD in neonatal intensive care unit (NICU) was done in order to depict a truer picture of CHD at high altitude. They found that babies were hospitalized because of pneumonia (62.6%), asphyxia (13.2%), or hyperbilirubinemia (7.3%). The incidence of CHD was 26.0% (1,096; 658 boys). Mild and moderate CHD accounted for 97.6% (1,070), including 580(52.9%) secundum atrial septal defect, 224(20.4%) patent ductus arteriosus, 18(1.6%) ventricular septal defect,

248(22.6%) multiple defects with left to right shunt, 1(0.1%) bicuspid aortic valve, 7(0.6%) pulmonary stenosis, 2(0.2%) aortic stenosis, 6(0.5%) atrioventricular septal defect. Critical CHD accounted for 2.4% (26), including 5(0.5%) complete transposition of the great arteries (TGA), 6(0.5%) hypoplastic right heart, 3(0.3%) hypoplastic left heart, 3(0.3%) double outlet right ventricle, 3(0.3%) tetralogy of Fallot, 2(0.2%) truncus arteriosus, 2(0.2%) total anomalous pulmonary venous connection, 2(0.2%) severe aortic stenosis, 2(0.2%) severe pulmonic stenosis. Among those with CHD, pulmonary arterial hypertension occurred in 2.8% (54/1,943) (systolic pressure 69±24 mmHg). By 2-12 months follow-up in 26 patients with critical CHD, 17 died before cardiac surgery, 1 of the 4 survivors had corrective operation of TGA, and 5 lost track. Thus they concluded that the incidence of CHD in NICU at high altitude is about 20 folds higher than that at low altitude, with substantially less incidence of critical CHD but with high mortality. Routine echocardiography and follow-up should be implemented in all NICU patients to provide early intervention.

Persistent pulmonary hypertension in the newborn can occur due to several reasons. Lung parenchymal conditions such as meconium aspiration syndrome (MAS), pneumonia, respiratory distress syndrome (RDS), and sepsis contribute to persistent pulmonary hypertension in the newborn] Other etiological factors include oligohydramnios, pulmonary hypoplasia, infants of diabetic mothers, in utero closure of ductus arteriosus, small and large for gestational age status. Maternal risk factors such as obesity, diabetes, pre-eclampsia, chorioamnionitis, smoking, selective serotonin reuptake inhibitors (SSRI), and NSAID use during pregnancy can also contribute to persistent pulmonary hypertension in the newborn.Congenital anomalies such as transposition of great arteries (TGA) and congenital diaphragmatic hernia (CDH) are also associated with persistent fetal circulation in the immediate neonatal period.Go to:EpidemiologyThe overall incidence of persistent pulmonary hypertension in newborns is 1.8 per 1000 live births.However, contrary to popular belief, the incidence of persistent pulmonary hypertension in newborns is higher in late preterm infants at 5.4 per 1000 live births. In term infants, the incidence is 1.6 per 1000 live births. Mortality ranges from 7.6 to 10.7%, depending on the severity of the condition.

High PVR relative to systemic vascular resistance is essential to normal intrauterine fetal circulation. Fluid-filled alveoli and hypoxia-induced pulmonary vasoconstriction in the presence of circulating vasoconstrictors such as endothelin-1 and thromboxane maintain high PVR in the fetal phase Lak. Conversely, circulating levels of vasodilators such as nitric oxide and prostaglandins are low Pulmonary reactivity to vasodilators increases with increasing gestational age. During normal transition after birth, several events occur simultaneously, which results in a smooth transition to extrauterine life. A drastic fall in pulmonary arterial pressure following the first breath accompanies increased pulmonary blood flow when the umbilical cord is clamped. The increased partial pressure of oxygen and the initiation of ventilation also stimulate the production of vasodilators such as nitric oxide and prostacyclins; cyclic guanosine monophosphate (cGMP) and cyclic adenosine monophosphate (cAMP) mediate pulmonary vasodilation through endothelial nitric oxide. Cyclooxygenase enzyme mediates the conversion of arachidonic acid to prostacyclin and is a rate-limiting enzyme. COX-1 is found in the lung and is upregulated when the fetus reaches term gestation. Prostacyclins increase cAMP levels which in turn cause vasorelaxation by decreasing intracellular calcium concentration. Nitric oxide - cyclic guanosine monophosphate (NO- cGMP) and nitric oxide- cyclic adenosine monophosphate (NO-cAMP) pathways are extensively studied in the pathophysiology of persistent fetal circulation. Subsequent clinical studies supported the widely accepted inhaled nitric oxide therapy in persistent pulmonary hypertension in newborns.

Persistent pulmonary hypertension in the newborn is categorized into three types:Maladaptation: abnormal pulmonary vascular response in lung parenchymal disorders such as meconium aspiration syndromeUnderdeveloped vasculature: decreased pulmonary vasculature as seen in small for gestational age or oligohydramniosIdiopathic persistent pulmonary hypertension in the newborn, likely due to excessive pulmonary vascular smooth muscle thicknessThese categories can overlap in any given condition.High PVR decreases pulmonary blood flow. This causes ventilation-perfusion mismatch and right to left shunting of blood across the foramen ovale and ductus arteriosus resulting in refractory hypoxemia. Extra-cardiac shunting across patent ductus arteriosus (PDA) results in more than a 10% differential between pre and post-ductal saturations. Infants with persistent pulmonary hypertension are vulnerable to wide swings in oxygen saturation.

Material and Methods

534 neonates with saturation less than 95, Persistent oxygen requirement, murmur, tachypnea more than 55 were taken for Echocardiography in Kamla Nehru Hospital, a branch of IGMC Shimla.

Ethical approval was taken from the IGMC Shimla ethical committee.

Mindray portable echo machine with 12 S Neonatal probe was used and bedside Echocardiography was done.

The data was recorded on the Proforma sheet.

Google forms were filled for each neonate separately.

Google responses were noted for 534 responses.

Excel sheet was derived from the Google responses.

Frequency and Prevalence for each Congenital heart disease was calculated.

P value was determined in two groups with or without structural heart diseases.

P value were also determined between two groups of cyanotic and acynotic heart disease.

Results.

Table 1: demographic and clinical features of newborn babies with and without structural heart disease

Characteristics	Overall study population	Group with structural heart disease	Group without structural heart disease	P value
Age (days) (median IQ)	5(3.0-12.0)	4.0(2.0-6.0)	6.0(3.0-12.0)	0.004
Gender (male)	341(63.8)	44(63.7)	297(63.8)	0.48
Gestation age	32.7± 4.4	32.7± 3.9	32.7± 4.5	0.87

Birth-weight	1.9± (0.7)	1.9± (0.6)	1.9± (0.7)	0.50
Maternal age	26.4± (3.0)	26.4± (2.7)	26.4± (3.0)	0.47
Paternal age	29.8±3.4	29.9±3.0	29.7±3.4	0.45
Maternal H/O Diabetes	3(0.56)	0	3(0.65)	0.32
Maternal history of Hypertension	4(0.75)	0	4(0.79)	0.39
H/O drug intake during first trimester	7(1.31)	1(1.45)	6(1.29)	0.42
Tachypnoea (RR≥55/minute)	428(80.1)	59(85.5)	369(79.3)	0.11
Tachycardia (HR≥200	3(0.56)	0	3(0.65)	0.32
Inter-costal retraction (yes)	408(76.4)	56(81.1)	352(75.7)	0.16
Oxygen saturation ≤95%	429(80.3)	60(86.9)	369(79.3)	0.06
Failure to maintain oxygen saturation ≥90%	422(79.0)	58(84.0)	364(78.2)	0.13

1. Age- SHD was detected 2 -6 day of age with significant p value as compared to Non SHD group. (0.004)
2. Gender did not have any significant relation with SHD.
3. Gestational age was also not significantly related to SHD.
4. Birth weight also did not predispose to SHD.
5. Maternal age did not contribute to SHD.
6. Paternal age also did not contribute to SHD as a significant risk factor.
7. Maternal diabetes did not contribute to occurrence of SHD.
8. Gestational Hypertension was not a significant contributor to SHD.
9. History of any drug intake was not found to have any significant P value in contributing to SHD.
10. Tachypnea was not significantly related to SHD, the reason being it was primarily due to lung causes.
11. Tachycardia also did not significantly contribute to SHD as it was due to other reasons of tachycardia.
12. Intercostals retractions had no significance to SHD as it was due to lung causes.
13. Oxygen saturation less than 95 percent was significantly associated with SHD with a significant p value. (0.06)

Table 2: Demographic and clinical features of new born babies with cyanotic and Acyanotic CHD

Characteristics	Overall group with structural heart disease	Group with cyanotic CHD	Group with A cyanotic CHD	P value
Age Day(median IQ)	4(2.0-6.0)	1.0(1.0-2.0)	5(2.0-6.5)	0.08
Gender (male)	44(63.7)	5(100)	39(60.9)	0.04
Birth weight	1.9±0.6	2.3±1.1	1.8±0.6	0.5
Gestational age	32.7±3.9	34.4±3.9	32.6±3.9	0.2
Maternal age	26.4±2.7	28.6±3.4	26.2±2.7	0.07
Paternal age	29.9±3.0	31.4±4.1	29.8±2.9	0.43
H/O maternal Diabetes	0	0	0	
History of Maternal HT	0	0	0	
Tachypnoea (RR≥55 per minute)	59(85.5)	5(100)	54(84.3)	0.22
Tachycardia (HR≥200 BPM)	0	0	0	0
Oxygen saturation (Oxygen saturation ≤95%)	60(86.9)	5(100)	55(85.9)	0.24
Intercostals recession (yes)	56(81.1)	4(100)	52(81.2)	0.44

Interpretation

1. Cyanotic CHD presented earlier than Acyanotic CHD with a significant p value. (0.08)
2. Male gender also showed significant correlation with Cyanotic CHD. (0.04)
3. Maternal age also showed association with cyanotic CHD with a significant p value. (0.07)
4. Gestational age, birth weight, tachypnea, retraction, tachycardia, desaturation and paternal age did not show any significance with cyanotic CHD.

Table 3: Predictors of structural heart disease in new born babies requiring ICU care.

Characteristics	Odds ratio (95% C.I.)	Two sided p value
Gestational age	1.0(.94-1.0)	0.92
Gender (male)	.99(.58-1.68)	0.98
Birth-weight	.88(0.62-1.26)	0.5
	1.73(.83-3.61)	1.47
Respiratory rate increase by 10 per minute	1.17(0.73-1.87)	0.49
Heart rate increase by 10 beats per minute	1.78(1.07-2.96)	0.02
Murmurs	3.0(1.29-7.3)	0.01
Not maintaining arterial oxygen saturation	1.46(.74-2.89)	0.27

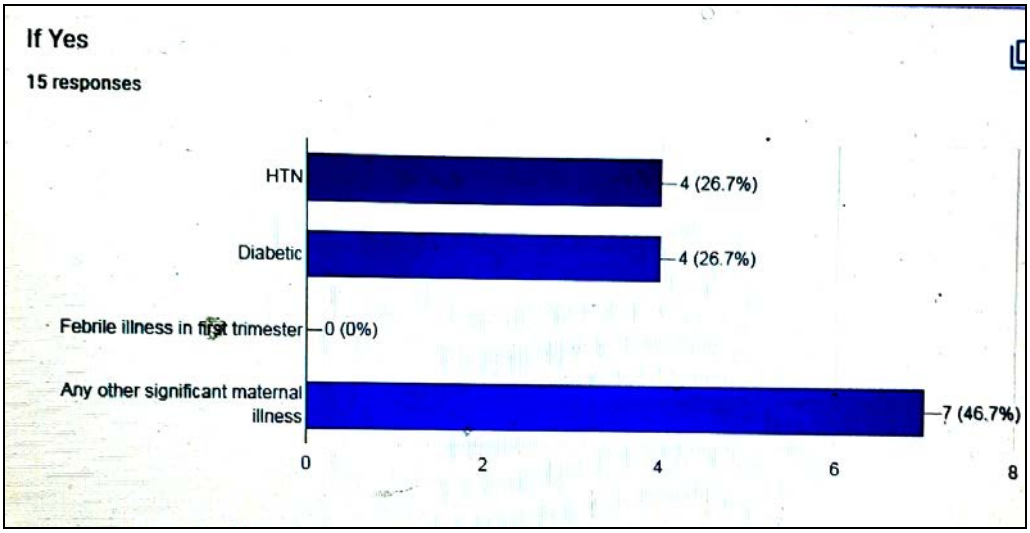
Interpretation

On logistic regression analysis, diagnosis of SHD, HR increase 10 per minute, 1.78(1.07-2.96) P value=0.02, and detection of murmur 3.0(1.29-7.3), p value=0.01 showed highly significant p value.

Age of neonate was 1 day to 42 days

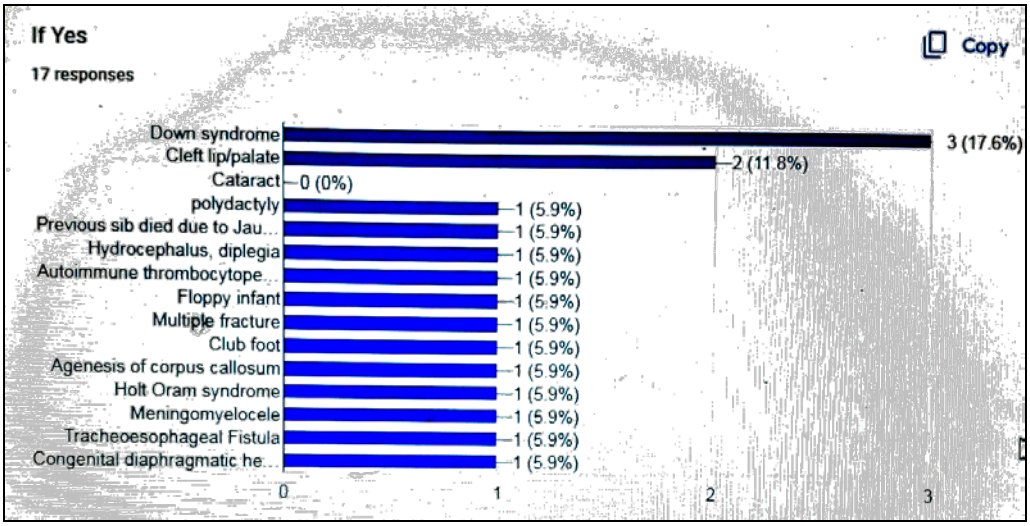
Gestational age was 26 weeks to 42 weeks
Male: Female was 63.7: 36.3
Baby weight was 700 grams to 3.6 kg
Maternal Age was 20 years to 39 years
Maternal diseases were present in 3 percent

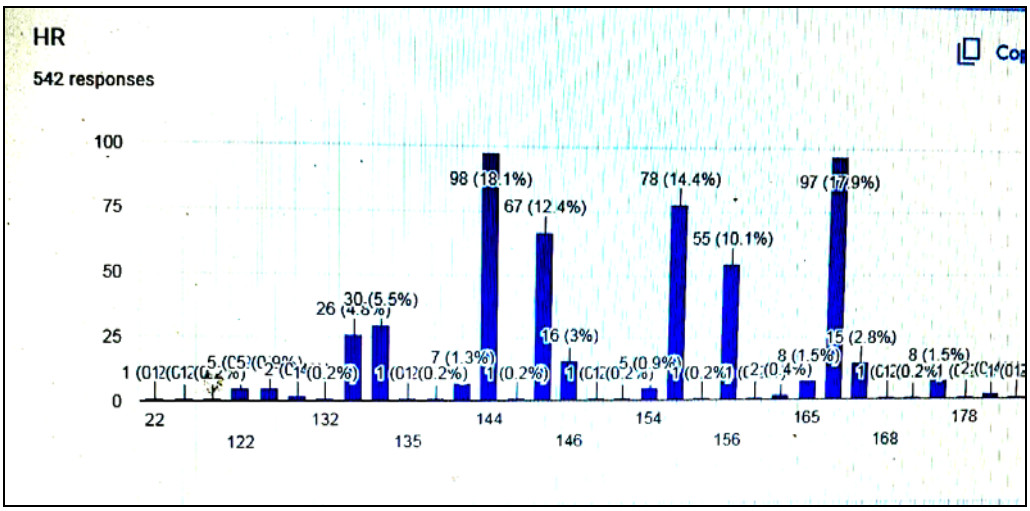
Out of 3 percent mothers 26 percent had Gestational Hypertension, 26 percent had Gestational Diabetes and 46 percent had hypothyroidism and SLE.
Maternal history of Drug intake
1.3 percent mothers were taking drugs



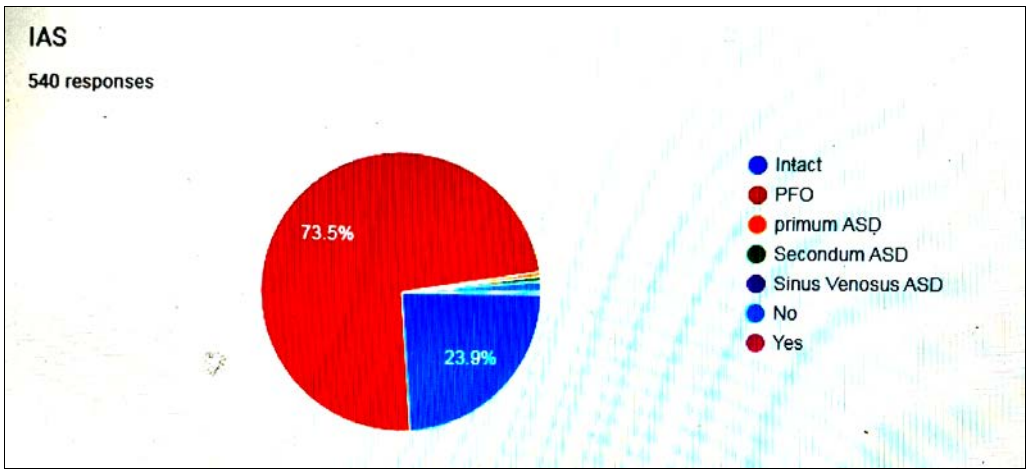
Paternal Age was between 20 yrs to 40 yrs
Extracardiac anomalies were found in 3 percent, of which Down syndrome in 17 percent, cleft palate in 11 percent, polydactyly in 5 percent, Hydrocephalus in 6 percent and other anomalies.

Intercostal retraction were found in 76 percent
Respiratory rate more than 44 per minute was seen in 84 percent.
Saturation right upper limb off oxygen less than 95 was seen in 85 percent

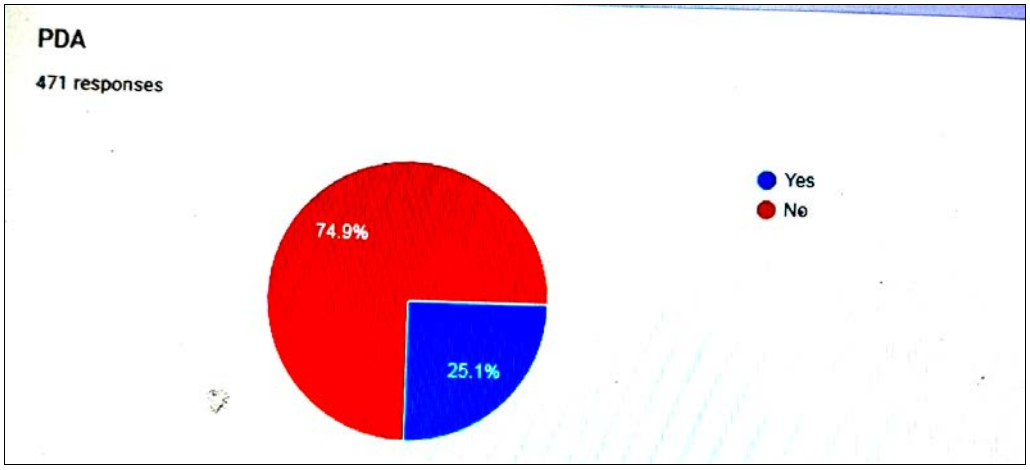




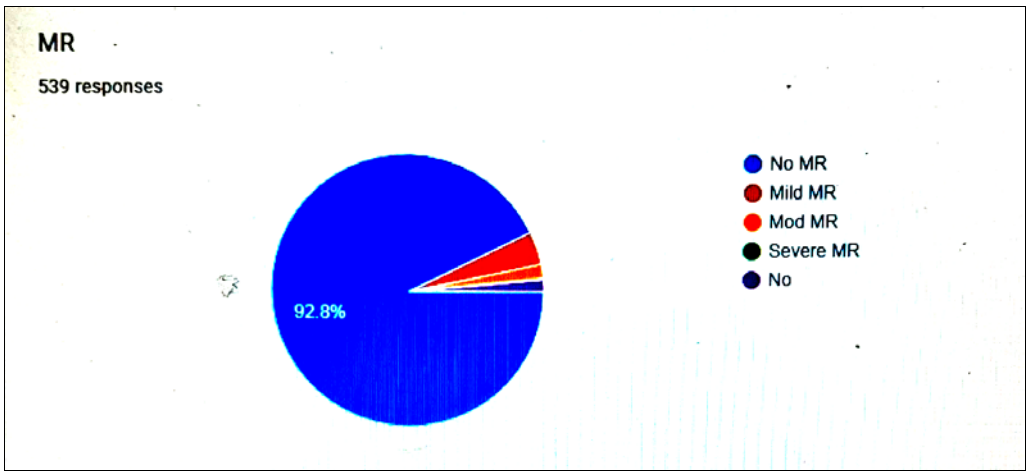
Heart Rate more than 140 was seen in 90 percent
Murmur was present in 5.2 percent
LV dysfunction was present in 5.4 percent
RV dysfunction was present in 5.2 percent



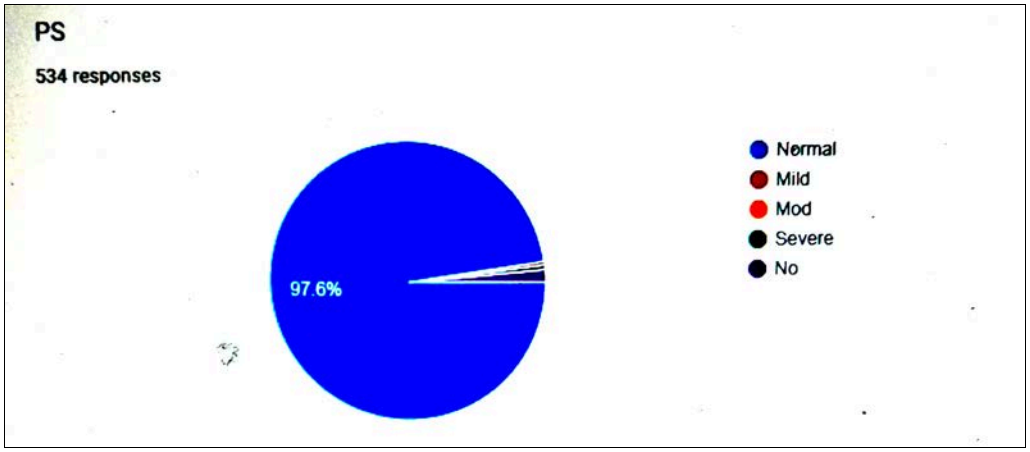
PFO was patent in 73 percent, ASD secondment was seen in 3 babies 0.6 percent
VSD was see in 2.6 percent



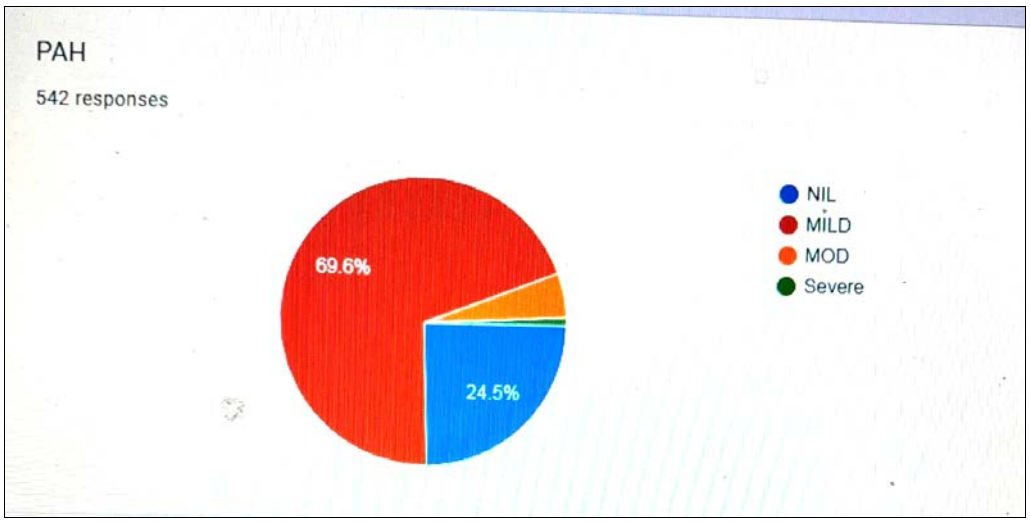
PDA was seen in 25 percent of which Large PDA was seen in 16 percent



MR was seen in 7.2 percent of which Moderate MR was 3.9 percent.
TR was seen in 66 percent of which Severe TR was 11 percent.



PS was seen in 2.4 percent.



PAH was seen in 75 percent.

Final Result
Cyanotic
1.TOF- 2 case
2.TGA- 1
3.Admixture lesions
a. TAPVC -0
b. DORV-1
c. Truncus-0

4. Single ventricle-
a. Mitral Atresia -2
b. Tricuspid Atresia -0

6. PPHN- 250
Acyanotic
A. Shunt lesions
1.ASD-1
2.VSD- small-1
Moderate -1
Large -2

3.PDA-
Large -31
4.APW-0
5.AVSD-1
B. Obstructive lesions
1.Aortic stenosis -0
2.Pulmonary stenosis -0
3.Coarctation-0
C. Regurgitant lesions
1.MR-
Moderate -21

2.AR-0
D. Myocardial diseases
1. Severe biventricular dysfunction-19
2. Significant Biventricular hypertrophy-21

Primary objectives

1. To determine prevalence of congenital heart diseases in NICU in moderate high altitude.

Secondary objectives

1. To determine clinical tools for determination of CHD.
2. To determine risk factors leading to SHD.
3. To determine prevalence of Persistent Pulmonary Hypertension in Moderate high altitude.

Conclusion

The prevalence of CHD in NICU was 15 percent which simulated with prevalence described previously which correlates with the previous study.

Clinical tools were at par with echo study. HR, Desaturation, are established clinical tools for CHD

detection. Maternal age is an important risk factor for CHD occurrence.

PPHN was seen in 50 percent of neonates. Sildenafil was started in neonates with Persistent oxygen dependency. The Hospital stay was reduced from 6 weeks to 3 weeks.

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