**KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH,**

**BELAGAVI, KARNATAKA**

# " IMPACT OF TRANSTHORACIC ECHOCARDIOGRAPHY ON DECISION MAKING IN CHILDREN PRESENTING WITH ACUTE RESPIRATORY DISTRESS AND HEART FAILURE - A ONE YEAR PROSPECTIVE OBSERVATIONAL STUDY."

**REG NO.BM0120010)**

## Dissertation

*Submitted to the*

*KLE Academy of Higher Education and ResearchBelagavi, Karnataka*

*In Partial Fulfillment of the requirements for the degree of*

**M. D. (Doctor of Medicine)**
**IN**
**PEDIATRICS**

**DEPARTMENT OF PEDIATRICS,**

**JAWAHARLAL NEHRU MEDICAL COLLEGE**

**BELAGAVI, KARNATAKA**

**JUNE-JULY –**

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**Dr. TANMAYA METGUD** M.D.
Professor & Head,
Department of Pediatrics,
J. N. Medical College,
Nehru Nagar,
Belagavi-590010

Date: 3/1/2023
Place: Belagavi.

KAHER BELAGAVI J.N. MEDICAL COLLEGE

**Dr. N.S. MAHANTASHETTI** M.D.,
Principal
J.N. Medical College,
Nehru Nagar,
Belagavi-590010.

PRINCIPAL J.N. Medical College, BELAGAVI-590 010

Date: 3/1/2023
Place: Belagavi.

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*Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA*

0831 – 2471350 0831 – 2470759 www.jnmc.edu principal@jnmc.edu


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Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
**Reg. No. BM0120010,**
Postgraduate Student,
2020-21 Batch,
Department of Paediatrics,
J. N. Medical College, Belagavi.

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K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
**JAWAHARLAL NEHRU MEDICAL COLLEGE,**
**NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)**
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

**Ref: MDC/DOME/ \58** **Date: 25/01/2021**

To.
**REG NO. BM0120010**
PG student in Paediatrics,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

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**(Dr. Smita Sonoli)**
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

**(Dr. Harsha Hegde)**
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

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# ABSTRACT

## "IMPACT OF TRANSTHORACIC ECHOCARDIOGRAPHY ON DECISION MAKING IN CHILDREN PRESENTING WITH ACUTE RESPIRATORY DISTRESS AND HEART FAILURE - A ONE YEAR PROSPECTIVE OBSERVATIONAL STUDY.".

**INTRODUCTION:** Respiratory distress is a clinical state characterized by abnormal respiratory rate or effort respiratory distress includes increased work of breathing (e.g., tachypnoea or hyperventilation), inadequate respiratory effort (e.g., hypoventilation or bradypnea), and irregular breathing. Regardless of the cause, if it is not recognized and managed, it can accelerate to respiratory failure and cardiopulmonary arrest. Heart failure (HF) is a term that is simple to implement but challenging to define. Only a few of the many definitions of HF that have been proposed have gained widespread acceptance[1]."HF as a complex clinical syndrome that results from any structural or functional impairment of ventricular filling or ejection of blood" is how the American College of Cardiology (ACC)/American Heart Association (AHA) guidelines define the condition.

**OBJECTIVE**-

To evaluate the value of Echocardiography (ECHO) in diagnosis and decision-making in critically ill children suffering from acute respiratory distress associated with heart failure admitted in PICU.

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**METHODS-**

A one-year prospective observational study, from January 2021 to Dec 2021, at a tertiary care centre. 75 patients between the ages of 1month and 18 years admitted in PICU with signs of respiratory distress with manifestations of heart failure were enrolled in the study. Patients were diagnosed with heart failure according to Modified Ross Heart Failure Classification for Children and Framingham criteria for heart failure. Echocardiographic examination was performed on all cases to confirm diagnosis and the following parameters were also assessed : Ratio of E wave and A wave (E/A), Fractional Shortening (FS) %, Ejection Fraction (EF) %, pulmonary artery pressure, and inferior vena cava (IVC) collapse with respiration

**RESULTS-**

After ECHO examination 41.3% of the cases were diagnosed with (CHD) as compared to only 22.6% diagnosed on admission, similarly 4% of the cases were diagnosed with (RHD) and 18.6 % were diagnosed with myocarditis who were previously missed on clinical examination. ECHO helped in change in the management in 45.9% of patients with CHD, 16.3% of patients with pulmonary HTN and in 37.7% patients with reduced LVF. ECHO improved our decision making by initiation of inotropes in 21.3% of cases, increasing inotropes in 8% of cases, initiation of diuretics in 30.6% of cases, fluid loading in 10.6% of cases, fluid restriction in 6.6% of cases, initiation of digoxin in 37.3% of cases, need of initiation of IVIG therapy was suggested in 15(20%) cases, and surgical intervention was suggested in 34.6% of cases.

## CONCLUSION-

Echocardiography is a rapid, non-invasive safe technique that can provide comprehensive information about cardiac structure and function. It can help in diagnosis and guide with therapy in critically ill infants and children admitted to PICU suffering from acute respiratory distress associated with heart failure. ECHO also has an impact on decision-making by suggesting the initiation of IVIG therapy in a patient diagnosed with myocarditis.

**Keywords:**

Heart Failure, Echocardiography, Pediatric Intensive Care Unit

Assessment, Respiratory distress.

# LIST OF ABBREVIATIONS USED

| | |
|---|---|
| AHA | American Heart Association |
| ACC | The American College of Cardiology |
| HF | Heart Failure |
| PICU | Pediatric Intensive Care Unit |
| TTE | Transthoracic Echocardiography |
| ECHO | Echocardiography |
| ICU | Intensive Care Unit |
| CCU | Critical Care Echocardiography |
| CHF | Congestive Heart Failure |
| CHD | Congenital Heart Disease |
| DCM | Dilated Cardiomyopathy |
| VSD | Ventricular Septal Defect |
| PDA | Patent Ductus Arteriosus |
| ECD | Endocardial Cushion Defect |

| | |
|---|---|
| LV | Left Ventricular |
| NYHA | New York Heart Association |
| POCUS | Point Of Care Ultrasound |
| ESPNIC | The European Society for Pediatric and Neonatal Intensive Care |
| IVC | Inferior Vena Cava |
| ESC | European Society of Cardiology |
| JVP | Juglar Venus Pressure |
| US | United States |
| UK | United Kingdom |
| GBD | Global Burden of Disease |
| RHD | Rheumatic Heart Disease |
| HFrEF | Heart Failure with reduced Ejection Fraction |
| HFpEF | Heart Failure with preserved Ejection Fraction |
| HFmrEF | Heart Failure with Mid-Range Ejection Fraction |
| LVEF | Left Ventricular Ejection Fraction |
| NPE | Neonatologist Perform Echocardiography |

| | |
|---|---|
| SD | Standard Deviation |
| Min | Minimum |
| Max | Maximum |
| DSS | Dengue Shock Syndrome |
| CMP | Cardiomyopathy |
| HFNC | High Flow Nasal Cannula |
| E/A Ratio | Early and Late Atrial Ventricular Filling Velocity |
| PAP | Pulmonary Artery Pressure |
| FS | Fractional Shortening |
| EF | Ejection Fraction |
| IVIg | Intravenous Immunoglobulin |
| HTN | Hypertension |
| LVF | Left Ventricular Function |
| C | Chi-Square Test |
| BNP | Brain Natriuretic Peptide |
| NT-ProBNT | N-Terminal pro b-type natriuretic peptide |

| | |
|---|---|
| ECG | Electrocardiography |
| CO | Cardiac Output |
| ASD | Atrial Septal Defect |
| PFO | Patent Foramen Ovale |

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# INTRODUCTION

A clinical condition known as respiratory distress is defined by an irregular breathing effort or rate. It encompasses symptoms ranging from agonal gasps to tachypnoea with retractions. Regardless of the underlying cause, respiratory distress can progress to respiratory failure and cardiopulmonary arrest in a very short time of span if it is not identified and treated early.

The symptoms of respiratory distress include irregular breathing patterns, insufficient respiratory effort, such as hypoventilation or bradypnea, and increased work of breathing (such as tachypnoea or hyperventilation) and irregular breathing regardless of the underlying reason, if it is not diagnosed and treated early, it can quickly progress to cardiac arrest and respiratory failure.

That is why the management of critically ill patients with respiratory distress requires immediate management and monitoring. Overlapping symptoms and multi-organ involvement bamboozle clinicians many a time. Hemodynamic evaluation of critically ill patients, whether with or without ultrasound, has recently earned top priority in patient care.

Term Heart failure is elementary to implement but difficult to define. Only very few definitions of HF have gained widespread acceptance. It's a pathophysiological situation where the heart either doesn't pump blood with a rate enough to fulfil the needs of the tissues that are metabolizing it or pumps blood only at elevated filling pressures due to an anomaly in cardiac structure or function discussed in a similar study done by Braunwald et al., 1992.[1]

American College of Cardiology (ACC) and American Heart Association (AHA) recommendations, which were also noted by Yancy et al. 2013 in a study, define HF as a multiplex clinical condition that occurs from any structural or functional impairment of

ventricular filling or ejection of blood. In accordance with the ESC's recommendations which Ponikowski et al. 2016, also included in their study, HF is a clinical syndrome with classic manifestations (such as difficulty in breathing, lower limb swelling and fatigue) that may be manifest with few signs (such as elevated pressures in jugular vein (JVP), pulmonary crepitation's, rales, and peripheral edema) caused by any anatomical and/or functional cardiac defects, resulting in decreased cardiac output and/or elevated intracardiac pressures at rest or during stress.[2]

There is very less research on pediatric HF and there is strict need to validate its study findings. That's why critically ill paediatric HF patients should be managed on the basis of proper prospective studies rather than just on the basis of clinical experience.

Admission of children with respiratory distress and cardiac disease to the PICU is increasing. The commonest indication for admission to the PICU is respiratory distress and acute deterioration of cardiac functions. The management of such patients is a perplexing task that needs prioritization and efficient time management. Multiple system involvement with overlapping symptoms frequently makes the clinical picture more challenging. Hemodynamicmonitoring is now given top priority in the care of severely ill patients. Wheeler et al.2009.[3]

Respiratory distress and a sudden decline in cardiac function are the most frequent reasons for admission to the PICU, around 30-48% of all admissions in a

pediatric intensive care unit (PICU) are children and infants with cardiac disease and respiratory distress. The pediatric intensive care unit's clinical service includes echocardiography as a crucial part (PICU).

Echocardiography is recognized as a convenient bedside imaging modality and an accurate diagnostic technique that examines a key body system and provides a clear image highlightingthe child's hemodynamic status. It has also been shown to be a key component of the clinical decision-making process as suggested by Rimington et al., 1996.[4]Echocardiography is the single most useful diagnostic test in the evaluation of patients with HF, according to the American College of Cardiology/American Heart Association's (ACC/AHA) guidelines for the diagnosis and treatment of heart failure (HF), which are also supported by Kirkpatrick et al., 2007[(a)]. In the HF population, echocardiographic imaging can be used to study myocardial structure, function, and valvular disease. It can also be used to determine the causes of hemodynamic instability and quickly direct therapy. As stated by Heinle et al. 1995, it has several advantages, including being a risk-free, noninvasive treatmentthat may be performed serially and in real time.

The use of echocardiography in the ongoing treatment of critically ill patients alters the course of their therapy in a positive aspect. The mounting evidence has supported the use oftransthoracic echocardiography (TTE) in the intensive care unit (ICU) Stanko et al., 2005, Orme et al2009).[5,6]

It helps in giving a thorough assessment of cardiac function. While measuring severely ill patients' hemodynamic variables like cardiac output and stroke volume, thermodilution techniques work well Slama et al.,2006, Goldstein et al.,1988).[7,8]

In the intensive care unit, cardiac conditions that cause shock are common.

These conditions include ventricular systolic dysfunction, acute valvular dysfunction, cardiac tamponade, and pulmonary embolism (Joseph et al.,2004). [9] Additionally, echocardiography contributes in measuring cardiac output, preload, and volume responsiveness; these measurements favor in contrast with traditional methods Mousavi et al.,2010, Charron et al .,2006. Also aid in diagnosing pulmonary embolism and its treatment with the help of echocardiography Mookadam et al., 2010.[10,11]

TTE is non-intrusive and can be performed quickly by qualified staff. However, cardiologists have traditionally been the field to which it belongs, atypical respiratory rate or attempts are

clinical signs of respiratory distress, it includes retractions to agonal gasps as well as signs of tachypnea. It includes breathing that requires more effort (such as tachypnea or hyperventilation), less effort (such as hypoventilation or bradypnea), and irregular breathing.

Regardless of the underlying cause, respiratory distress has the potential to end up into respiratory failure and cardiopulmonary arrest if it is not recognized and treated on time. Therefore, prompt management and monitoring are required for subjects who are gravely ill and experiencing respiratory distress. Many a time, overlapping symptoms and multiorgan involvement confound the clinician.

When seriously ill children or infants are who are getting admitting to the pediatric intensive care unit (PICU) and are experiencing acute respiratory distress linked to heart failure, an echocardiogram, a quick, non-invasive, safe procedure, can help with diagnosis and treatmentplanning.

Critical care echocardiography (CCE), which has gained widespread

acceptance in the last 10 years, has become a crucial area of critical care ultrasonography. Its usefulness, both as a diagnostic method and for hemodynamic assessment, has significantly increased, having an impact on current cardiorespiratory management.

The monitoring of vital information about the structure and ongoing functional changes in ventricular loading situations is enhanced by echocardiography. The functional alterations, current compliance, and overall biventricular performance are controlled by frequent bedside evaluation.

Monitoring changes in cardiac function helps determine how well a treatment is working. Thecause of the underlying disease, management strategies, and length of treatment all have an impact on cardiac output and hemodynamics.

# OBJECTIVES

**PRIMARY OBJECTIVES:**

1. To assess baseline adherence to 6-Mercaptopurine(6MP) during maintenance phase chemotherapy in children undergoing treatment for Acute Lymphoblastic Leukaemia (ALL) and to evaluate the impact of smart pill box and parent education in improving the adherence

**SECONDARY OBJECTIVES:**

1. To identify factors that influence adherence to 6-MP in children with ALL.
2. To compare subjective method using Morisky medication adherence scale (MMAS-8) with objective method using 6-MP metabolite levels in assessing the adherence.
3. To study the pattern of Thiopurine methyl transferase (TPMT) genotype in the given cohort and to correlate it with 6MP metabolite level.

# REVIEW OF LITERATURE

## HISTORY

Heart failure's brief past-

William Harvey explains the circulation for the first time in the sixteenth century. While William Withering wrote a paper on the use of digitalis for medical reasons in the late seventeenth century. The benefits and drawbacks of using digitalis in youngsters were the subjects of a brand-new debate. Up until the early eighteenth century, the diagnosis of heart failure could only be made clinically. René Laennec invented the stethoscope in 1819, which helped distinguish the murmur from normal heart sounds. Wilhelm Röntgen's creation of x-rays in 1895 made further diagnostics considerably simpler.

Digoxin and restriction of fluids alone were not enough for the management of heart failure, the mortality rate was much higher, and hence more medication was under trial for the treatment of cardiac failure. The nineteenth century was the first occasion for the use of diuretics. In 1958 Thiazide diuretics are made available. In 1954 Ultrasound is used by Inge Edler and Hellmuth Hertz to image cardiac structures. The father of echocardiography was Inge Edler and Christiaan Barnard performed the first human heart transplant in 1967. There was ongoing research on diagnostic as well as therapeutic treatment of heart failure, in 1987 CONSENSUS-I study showed unequivocal survival benefits of angiotensin-converting enzyme inhibitors in severe heart failure whereas another study was done in 1995 by the European society of cardiology released recommendations for identifying heart failure.

**DEFINITIONS**

Heart failure (HF) is defined as a defect in the heart's structure or in its functional capacity that is preventing it from pumping blood adequately to meet the body's tissues' oxygen demand while preserving normal filling pressures (or only at the expense of increased filling pressures)[12]

According to the most recent ACC/AHA guidelines for the diagnosis and management ofheart failure, Echocardiography is the single most effective diagnostic and theopoetic technique in the assessment of patients with heart failure (HF) found by Kirkpatrick et al. and others, in a studyin 2007[13]

HF in children has gotten far less attention than it has in adults due to a number of issues, where it has been the focus of in-depth study and the creation of evidence-based recommendations. In comparison to adult cases of HF, which often involve coronary artery disease and hypertension, childhood cases of HF have quite different origins.

Children with HF are described as having a progressive clinical and pathophysiological illness brought on by circulatory, neurohormonal, and molecular abnormalities that produce edema, respiratory distress, growth failure, and exercise intolerance.

Research on the treatment of HF in adults has been extensively studied, but pediatric HF has received far less attention, and what little research there is usually consists of small, retrospective studies, but now it has substantially evolved throughout time as a result of the limited pediatric literature, clinical skills, extrapolation of adult data, and other causes.

The goal of this review is to give a thorough overview of pediatric HF with a focus on diagnosisand treatment.

**GLOBAL INCIDENCE OF PAEDIATRIC HEART FAILURE:**

Due to the absence of a common definition for HF**,** It is challenging to quantify the actual global incidence and prevalence of HF among children. Each year, 11,000–14,000 children in the US are hospitalized with heart failure [14]. The majority of the children who seems to get HF are born with CHD, and depending upon age, 25-75% of pediatric HF patients also have CHD [15,16]. Recently, a study done by Shaddy et al., 2018 revealed that "the incidence of HF ranged from 0.87/100,000 (UK and Ireland), 7.4/100,000 (Taiwan), and 83.3/100,000 (Spain)" [17]. In children all throughout the world, CHD is probably the most prevalent underlying cause of heart failure. CHD occurs in around 8/1000 live births. HF associated with CHD occurs in approximately 20% of all patients.

It has been estimated that the annual incidence of HF as a result of congenital abnormalities isbetween 1 and 2 per 1000 live births and that many infants with CHD undergo early surgical surgery.[18] Following the development of early surgical procedures, the result of HF associated with CHD has changed significantly. In the "early surgical period," the incidence of symptomatic HF has also decreased. Only 10% of the patients reported by Massin et al. who were treated in a tertiary pediatric cardiology setting experienced asymptomatic heart attacks.[19]

Additionally, a considerable portion of pediatric patients who report with heart failure symptoms has cardiomyopathy. According to Rossano et al. from the United States, around 27% (or about 3000) of the 10,000–14,000 children

hospitalized each year with heart failure have abnormalities of the heart muscle as an underlying cause. [20] Cardiomyopathies affect

between 0.8 and 1.3 cases per 100,000 children in affluent nations between the ages of 0 and 18, although their prevalence in children between the ages of 0 and 1 month is ten times higher. [21,22] 90% of pediatric cardiomyopathies are dilated cardiomyopathies.

The prognosis for children with cardiomyopathy is still poor, in contrast to HF owing to CHD,with a 5-year risk for death or heart transplantation of roughly 50% for those with dilated cardiomyopathy (DCM).

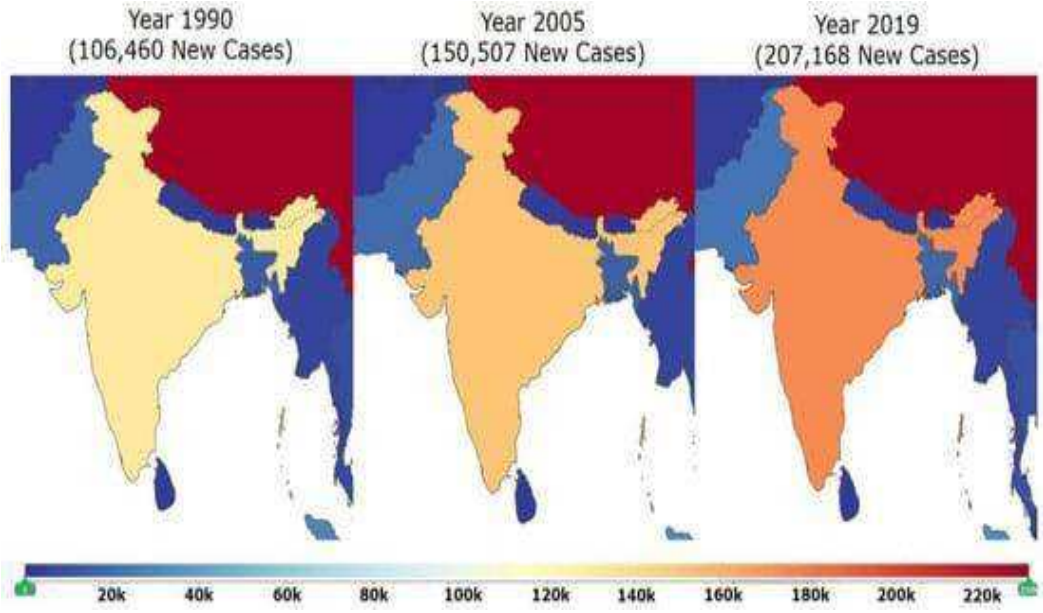


Figure 1 - This map of India (derived from the Global Burden of Disease; GBD) displays the risein DCM incidence there from 1990 to 2019.

**CAUSES OF HEART FAILURE -**

Numerous factors can contribute to heart failure syndrome. The most common causes of CHF are congenital or acquired heart disease, myocardial abnormalities causing volume or pressureoverload, or both. Heart failure can occur at any age as a result of tachyarrhythmias and heart block. Congenital heart defects account for the vast majority of causes of CHF in infants (CHDs). Myocardial dysfunctions with different aetiologies are significant contributors to CHF after childhood. Anemia, pulmonary illnesses, collagen vascular diseases, systemic hypertension or pulmonary hypertension, neuromuscular problems, and medications like anthracyclines are a few of the uncommon causes of CHF.

**CONGENITAL HEART DISEASE-**

The most frequent causes of CHF in the first six months of infancy are volume overload lesionslike ventricular septal defect (VSD), patent ductus arteriosus (PDA), and endocardial cushion defect (ECD). The timing of CHF onset in infancy varies predictably depending on the type ofdefect.

**ACQUIRED HEART DISEASE-**

CHF can result from a variety of acquired cardiac conditions. The age at which CHF manifestswith acquired heart disease is less predictable than with CHD.

1. Following infancy, dilated cardiomyopathy is likely the most prevalent cause of CHF.At any age during childhood and adolescence, it may result in CHF. The majority of

   cases of dilated cardiomyopathy are idiopathic, however, it can also be caused byautoimmune diseases; viral, endocrine, or metabolic disorders;

2. In older children and adolescents, CHF may be brought on by cardiomyopathies associated with Friedreich's ataxia and muscular dystrophy.
3. Children aged 1 to 4 years commonly get myocarditis in conjunction with Kawasakidisease.
4. Children under 1 appear to be more susceptible to viral myocarditis than older children.

It seldom affects newborns and has a fulminating clinical course with a bad prognosis.

5. CHF can occasionally be caused by acute rheumatic carditis, which is most common inschool-age children.

**MISCELLANEOUS CAUSES CHF-**

The following are additional causes of CHF:

1. CHF in infants can result from metabolic problems, including severe hypoxia, acidosis,hypoglycemia, and hypocalcemia
2. Endocrine disorders like hyperthyroidism.
3. Supraventricular tachycardia (SVT) can be a cause of CHF in early infancy.
4. Severe anemia could be the cause of CHF at any age.
5. Hydrops fetalis may contribute to CHF in newborns, whereas severe sickle cell diseasecan cause it later in life.

**PRESENTATION OF HEART FAILURE IN CHILDREN**

Children typically exhibit the following symptoms: Fever, nasal flaring (in infants), breathing problems, colds or coughs, tachypnoea, crepitations or decreased air entry, chest retractions, feeding difficulties, diaphoresis, and inadequate weight gain[23].

1. As a result of decreased heart function, the following are observed:

- Usually they present with tachycardia, gallop rhythm, and thin, feeble pulses.
- There is virtually always cardiomegaly. Medical examinations are less reliable thanchest radiographs for evaluating cardiomegaly.
- Increased sympathetic discharges are noted (e.g., growth failure; perspiration, cold andwet skin).

**2.** The following symptoms are caused by pulmonary venous congestion (i.e. left-sidedfailure) **:**

- Tachypnea is a typical and early sign of CHF in infants.
- Children frequently experience dyspnea on exertion, which is comparable to poorfeeding in small infants.
- Older children may exhibit orthopnoea.
- Wheezing and pulmonary crackles.

3. As a result of systemic venous congestion (i.e. Right-sided failure) resulting in thefollowing consequences:

- Hepatomegaly (enlarged liver span) is common, but not always associated with CHF. It can be present ininfiltrative liver disease, diseases including asthma, bronchiolitis, etc, that result in hyperinflated lungs and can also

cause the enlarged liver to be palpable. On the otherhand, the absence of hepatomegaly does not rule out CHF.

- Swollen eyelids are common in infants.
- Infants do not exhibit adult-like features, such as ankle edema or dilated neck veins.

According to research by Owayed AF et al., most patients with recurrent pneumonia are known to have an underlying illness and the most common underlying cause found was congenital heart abnormalities (9%) in the majority of individuals with repeated infections. Due to structural heart abnormalities that might exacerbate an already compromised respiratory condition, Children with CHD are at risk for increased morbidity from lower respiratory tract infections[14]**.**

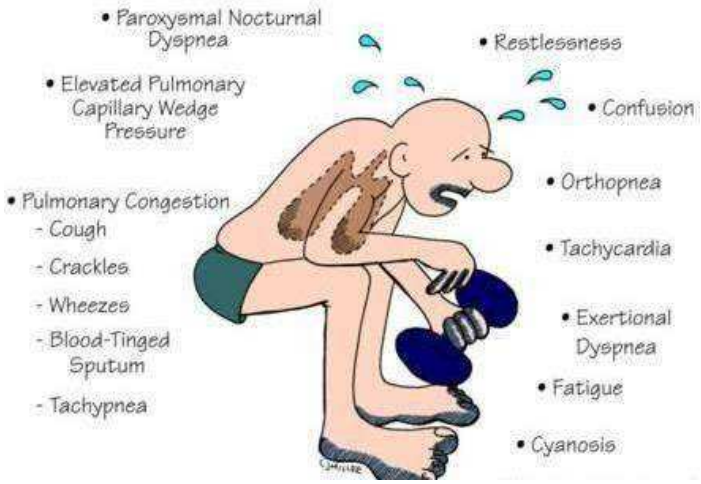


Figure 2- Left-sided heart failure


**Figure 3 -Right-sided heart failure'**

**RESPIRATORY DISTRESS**

- **Pale or bluish skin color** - Check around the lips, eyes, hands, and especially the nail beds.

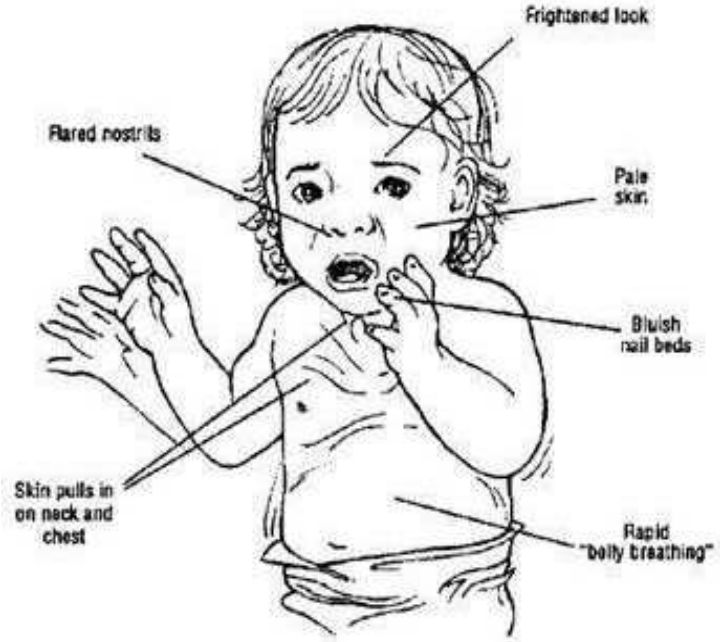


**Figure 4- Signs of respiratory distress in a child**

- **Increased breathing rate** – Monitor the number of breaths for a complete 1minute. Is the child breathing faster than usual?
- **Retractions** – closely observe the area around the collarbone and the ribs to seeif the chest contracts with each breath.
- **Nasal flaring** - Check to see if nostrils widen when breathing in.
- **Noisy breathing** - Breath sounds like grunting, wheezing
- **Clammy skin** – Feel the child's skin to see if it is cool but also sweaty.
- **Mood change** – If the child is drowsy, difficult to wake, fussier than usual, or"just not acting like himself."
- **Change in body position** – Like leaning forward or tilting his head up orbackward to try to breathe easier.

The current criteria for HF only apply to manifest stages with overt clinical symptoms and signs. However, the majority of patients have asymptomatic structural or functional heart conditions including left ventricular (LV) hypertrophy, LV systolic or diastolic dysfunction, and valve disorders, all of which are well-known risk factors for heart failure (HF).

It is crucial to recognize and treat developed HF due to its dismal prognosis. Therefore, by regulating these HF precursor factors, overt HF could be prevented or delayed.

Controlling these HF precursor conditions could therefore stop or delay the development of overt HF. Four stages of HF are recognized by the ACC/AHA, Yancy

et al., 2013, to reflect the importance of the predisposing variables in the condition's cause (Table 1).

Stages A and B are pre-HF forms,

Stages C and D are clinically discernible forms.

Further classifications of the underlying etiology, ejection fraction, temporal course, and severity of HF are possible.

Table 1 ACC/AHA Stages of HF.

| Stages | Definitions | Examples |
|---|---|---|
| Stage A | At high risk for HF but w/o structural heart disease or symptoms of HF | hypertension |
| Stage B | Structural heart disease but w/o symptoms or signs of HF | LV hypertrophy and dysfunction |
| Stage C | Structural heart disease with previous or present symptoms of HF | Examples of Chronic HF |
| Stage D | Refractory HF, in need of specialized interventions | End-stage heart failure |

**Types of HF:**

Finding the HF's underlying etiology is an essential step in the diagnostic process. Systolic and/or diastolic dysfunction of the LV is the most typical cause of HF worldwide. RHD is still one of the most persistent causes of HF in India", *Ramakrishnan et al 2009.* In addition, structural heart defects, metabolic disorders, endocardial, pericardial, and heart rhythm, and conduction abnormalities, as well as

systemic and metabolic conditions, can all contribute to HF. In some cases, more than one etiology may play a role in the onset and progression of HF. Reduced, mid-range, and preserved

**STAGES OF HF**

ejection fraction in heart failure Table2)

**Table 2- Reduced, mid-range, and preserved ejection fraction in heart failure.** *Ponikowski et al., 2016*

| | |
|---|---|
| HFrEF (HF with reduced EF) | LVEF ≤ 40% |
| HFpEF (HF with preserved EF) | LVEF ≥ 50% |
| HFmrEF (HF with mid-range EF) | LVEF 41–49% |

Classification of HF in Children:

The Ross Heart Failure Classification was created to give an overall evaluation of the severity of heart failure in infants, but it has since been altered to cover all pediatric age groups.

The modified Ross Classification assigns a numerical score that is comparable to the NYHA (New York Heart Association) classification for adults and takes into account issues with feeding, growth, and exercise intolerance. Children's NYHA (>6 years of age) and Modified Ross Heart Failure Classification (<6 years of age.) (Table 3)

**Table 3: NYHA and Modified Ross Heart Failure Classification**

**Source (Reference no 7)**

| | NYHA | MODIFIED ROSS CLASSIFICATION |
|---|---|---|
| **Class I:** | no limitation of physical activity | Asymptomatic |
| **Class II:** | May experience fatigue, palpitations, dyspnea, or angina during moderate exercise but not during rest | In Infants- Mild tachypnea or diaphoresis with feeding<br>Older Children - Dyspnea on exertion |
| **Class III:** | Symptoms with minimal exertion that interfere with normal daily activity | In Infants- Marked tachypnea or diaphoresis with feeding and prolonged feeding times with growth failure. Older Children- marked dyspnea on exertion in older children |
| **Class-IV:** | Unable to carry out any physical activity because they typically have symptoms of HF at rest that worsen with any exertion | Tachypnea, retractions, grunting or diaphoresis at rest. |

## HEART FAILURE'S RELATIONSHIP TO RESPIRATORY DISTRESS

An important and frequent non-pulmonary cause of respiratory distress is cardiac disease. Respiratory distress and increased work of breathing are most frequently seen in heart disorders brought on by significant left-to-right shunts, abnormalities of the systemic ventricle, and vascular lesions that obstruct the airway.

Respiratory distress is frequently a symptom of cardiac failure, regardless of the reason. Infants with CHD result in significant left-to-right shunt-producing pulmonary vascular engorgement, edema formation, and impaired lung compliance, exhibiting tachypnoea, difficulty in breathing, and as wellas grunting. compression of intrathoracic airways by vascular engorgement and interstitial edema may cause wheezing or cardiac asthma.

Tachypnea, dyspnea, grunting, and diaphoresis are symptoms of acute myocarditis, which is usually of a viral etiology. The physical examination reveals tachycardia and diminished heart sounds, and a chest x-ray reveals a significantly enlarged heart. Cardiomyopathy can be inherited, congenital, toxic, or metabolic in origin, familial, oridiopathic. It is important to look for additional reasons for heart failure, such as severe hypertension, renal failure, and severe anemia. Increased pulmonary vascular engorgement and edema accompany systemic ventricular failure brought on by obstructive lesions, such as aortic stenosis, coarctation of the aorta, or mitral stenosis, and produce the same symptoms as a significant left-to-right shunt. Systemic blood flow may be reduced depending on the degree of the left ventricular outflow obstruction, which could lead to poor perfusion and metabolic acidosis.Some pediatricians have had to deal with the challenge of treating a child who has both CHD and respiratory problems. It has even been postulated that congenital cardiovascular anomalies are significantly associated with congenital and acquired respiratory disorders.

Even the hypothesis has been made that congenital and acquired respiratory illnesses have a strong correlation with congenital cardiovascular defects. Although it is unknown whether asthma and/or airway hyper-responsiveness are more common in

children with CHD than in the general population, some writers have made the claim that this is the case. (24,25)

In two Nigerian children, Bode Thomas et al. discovered a coexisting ventricular septal defect and hyperactive airway; they treated the children with bronchodilators and steroids, and the children did well [26].

The attending physician may observe that the coexistence of these two disease entities in developing nations like ours may be primarily due to the possibility that both could present with comparable symptomatology. This might delay diagnosis, particularly if the level of suspicion is low [27,28,29,30].

**ROLE OF ECHOCARDIOGRAPHY IN CHILDREN PRESENTING WITH ACUTE RESPIRATORY DISTRESS AND HEART FAILURE –**

Some pediatricians have been faced with the dilemma of encountering a child where both CHD and respiratory disease coexist. It has even been postulated that congenital cardiovascular anomalies are significantly associated with congenital and acquired respiratory disorders. Although the prevalence of asthma and/ or airway hyperresponsiveness in children with CHD is not known, some authors have suggested that hyperactive airway disease is more common in children with CHD than in the general population. [2,3] For instance, Bode-Thomas et al. [4] noted a coexistence of ventricular septal defect and hyperactive airway in two Nigerian children, they managed with bronchodilators and steroids, and the children responded well.

In developing countries like ours, the attending physician may note that the coexistence of these two disease entities may lie mainly in the fact that both could

present with similar symptomatology. This could lead to a delayed diagnosis, especially when there is a low indexof suspicion. [5-8]

Echocardiography has emerged as the imaging technique of choice for the diagnosis and assessment of congenital and acquired cardiac illness in infants, children, and teenagers.

The preferred tool for cardiac evaluation is transthoracic echocardiography (TTE), which is non-invasive, portable, and efficient in providing precise anatomical, hemodynamic, and physiologic parameters about the child's heart.[31]

**IMPACT OF ECHOCARDIOGRAPHY ON DECISION-MAKING IN PICU**

Prioritization and careful time management are necessary for the treatment and care of criticallyill pediatric patients who are experiencing acute respiratory distress. Multisystem involvement and symptom overlap can sometimes make it more challenging to form clinical impressions.

The importance of hemodynamic assessment in the treatment of seriously ill patients has significantly increased in recent years, according to a 2015 study by Ahmed et al. [32],With respiratory distress and cardiac problems, children and infants are admitted more frequently topediatric intensive care unit (PICU) (almost 30–50% of all cases).

The most frequent causes of PICU admission, according to a 2008 study by Pasquali et al., were respiratory distress and an abrupt loss in cardiac function, including hyper cyanosis episodes, acute heart failure, heart failure linked to a chest infection, arrhythmias, and impending respiratory failure.[33]

Comorbid heart failure and respiratory distress can be brought on by a number of different conditions, such as pathology in one or more cardiovascular

system component (the myocardium, pericardium, heart valves, or endocardium), or they can develop as an unintended side effect of a chronic systemic or metabolic condition. Since heart failure does not always involve congestion, the term heart failure (HF) is preferable to congestive heart failure (CHF), Suggested by Rugolotto and others in their study held in 2001.[34]

Echocardiography is a cornerstone of clinical management in the PICU. It is considered as a convenient, realistic, reliable, bedside diagnostic tool that facilitates the study of a vital bodily system and provides a detailed and clear image of the hemodynamic state of the critically ill patient. It is also crucial to be able to promptly identify the source of hemodynamic instability and administer treatment. Benefits of the technique include being non-invasive, risk-free, and ableto be performed serially in real-time, as demonstrated by Heinle et al., 1995.[35]

Many studies, such as those conducted by Manasia et al. in 2005 and Croft et al. in 2006, haveshown the value of echocardiography in the treatment of seriously ill patients, changing their treatment plan in almost 30% to 60% of cases after tests were completed [37]. Acute respiratory distress needs to be treated very away, and Nohria et al. (2005) found that early subject evaluation is crucial for appropriate therapy selection and management. After thediagnosis was suspected, to confirm the diagnosis of abrupt cardiac failure, echocardiographyis necessary.[36] Evaluation of the left ventricular function is the most common reason for echocardiograms, according to the majority of adult pediatrictric research Vignon et al., Stanko et al., 2005; 1994; Orme et al., 2009. [5,6,][38]. The most common reason to perform echocardiography has been found by virtue of many studies conducted in different parts of the world is to assess leftventricular function.

It aids in clinical resolution, which usually take place when the treating physician is unsure ofwhether to support cardiac function or providing fluid support with a focus on sepsis should bethe top priority as supported by Arntfield et al., 2012. [(22)], the use of Echocardiography may bea very valuable indication in such a critical situation.

Tam et al. (1999) found that people who received unexpected ECHO results were more likely(58%) than those who received predicted results (32%) to change their management strategy. According to Tam et al. (1999),[40] the study's description of a modification in treatment methods includes both medication adjustment and the introduction or discontinuation of new

invasive or non-invasive investigative techniques. Similarly, use of POCUS in critically ill infants and children are now available. Bedside goal-directed echocardiography has been the first POCUS application in pediatric practice with guidelines for implementation.

An expert statement was published in 2011, followed by the United Kingdom Expert Consensus Statement on Neonatologists Performed Echocardiography (NPE) and recommendations for NPE in Europe. Non-cardiac POCUS may carry more opportunities for use as well as a numberof benefits for both patients and providers. However, the lack of guidelines is a barrier to widespread adoption.

Therefore, the European Society for Paediatric and Neonatal Intensive Care (ESPNIC) assembled a group of international pediatric POCUS key opinion leaders to create evidence-based guidelines for the use of current and emerging POCUS applications in the neonatal (NICU) and paediatric intensive care units (PICU) by any clinician working in these units.

# MATERIAL AND METHODS

## STUDY SOURCE

This study was conducted in the department of Pediatrics, in tertiary care hospital, Belagavi asa part of the MD academic curriculum.

## STUDY DURATION

The study was conducted between 1st January 2021 to 31st December 2021

## ETHICAL CLEARANCE

Clearance was taken from the Ethical Committee of the Institution

## STUDY-DESIGN

Hospital-based cross-sectional study.

## INCLUSION CRITERIA

All children from age 2 months to 18 years having signs of respiratory distress with the manifestation of heart failure in the form of (tachypnoea, tachycardia, chest indrawing, intercostal retraction, enlarged tender liver, cyanosis, altered level of consciousness) will be included. Patients diagnosed as having heart failure according to Modified ROSS Criteria for Heart Failure will be enrolled in the study. [4]

Class I- No limitations or symptoms

Class II- Infants: Mild tachypnoea or diaphoresis with feeding Older children: Mild to moderate dyspnea on exertion

Class III - Infants: Growth failure and marked tachypnoea diaphoresis with feeding.

Older children: Marked dyspnoea on exertion

Class IV - Symptoms at rest such as tachypnoea, retractions, grunting,or diaphoresis.

**EXCLUSION CRITERIA**

- Cases with chronic illnesses: renal disease,
- Chronic liver diseases
- Cases of poisoning
- Cases of head injury
- Surgical cases like pneumothorax, chest trauma,
- Abdominal trauma
- Hematology and oncology cases present with
- Breathing difficulties
- Guillain barre syndrome

**INFORMED CONSENT** Written informed consent will be taken from the parent or guardian of all children who will be recruited prior to the study.

**STATISTICAL ANALYSIS:**

Continuous variables are expressed as mean ± SD. Total counts and percentages are reported for categorical variables. Four outcomes of interest were identified:

1) Unexpected new ECHO findings. ECHO findings resulting in management.
2) Change in terms of ionotropic and decongestive therapy.
3) ECHO findings resulting in surgical intervention.
4) ECHO results that confirm expected findings.

**SAMPLE SIZE**

A minimum sample size that can be taken is =75

The formula used for sample size calculation is $n = p\ (100 - p)\ Z^{2}$

$E2$

n is the sample size required.

p is the percentage occurrence of a state or condition (proportion or prevalence).E is the percentage maximum error required.

Z is the value corresponding to the level of confidence required.

a. ECHO changes our diagnosis of study cases in CHD from 13 cases (21.7%) to 16 cases (26.7%) after ECHO examination [1], with a percentage of maximum error of 10% ata 95% confidence level.

The sample size is given by-

$$n = \frac{26.7 \times (100 - 26.7) \times (1.96)^2}{10^2}$$

$$n = 75.18 \approx \mathbf{7}5$$

Particularly important views for pediatric examination include the right parasternal,suprasternal notch, and subxiphoid (or subcostal). The acquisition of images by the pediatric TTE and their proper display are crucial components.


**Figure 5- 2-D ECHO machine**

# RESULTS

## METHODS:

Excel and SPSS software version 21 are used for data analysis. Frequency tables are used to present categorical variables. The format for continuous variables is Mean SD/ Median (Min, Max). To examine the relationship between categorical variables, apply the chi-square test. A P-value of 0.05 or less suggests statistical significance.75 participants were observed, ranging in age from 1 month to 18 years, with mean age 60.57 ± 60.87 months. The distribution of participants among various factors is shown in the following table.

**Table 4: Distribution of subjects based on demographic variables.**

| Variables | Sub Category | Number of Subjects (%) |
|---|---|---|
| Age | 1 month – 1 year | 26 (34.66%) |
| | 1 year – 2 years | 5 (6.66%) |
| | 2 years – 5 years | 18 (24%) |
| | 5 years – 12 years | 17 (22.66%) |
| | 12 years -18 years | 9 (12%) |
| | Mean ± SD* | 60.57 ± 60.87 |
| | Median (Min, Max) | 42 (1, 192) |
| Gender | Male | 41 (54.7%) |
| | Female | 34 (45.3%) |

*Age-mean and SD are calculated in months.

The age ranged from 1 to 2 year 5 patients (6.66%), while 18 patients (24%) were among 2 to 5 years of age, 17 patients (22.66%) among 5 to 12 years of age, and 9 patients (12%) were above 12 years till 18 years of age. Gender of subjects are 41 (54.7%) of male and 34 (45.3%)of population are female.

The below graph depicts the same-

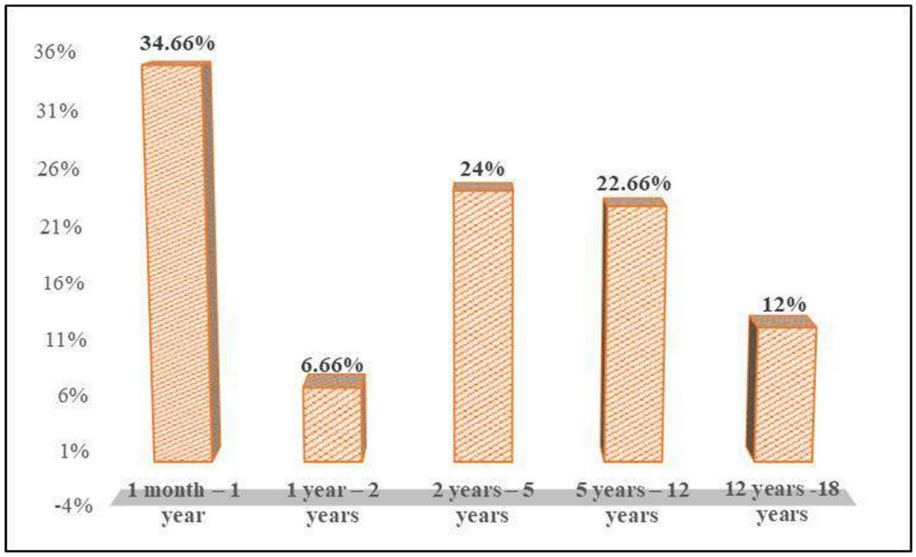


**Figure 6: Distribution of subjects based on Age.**

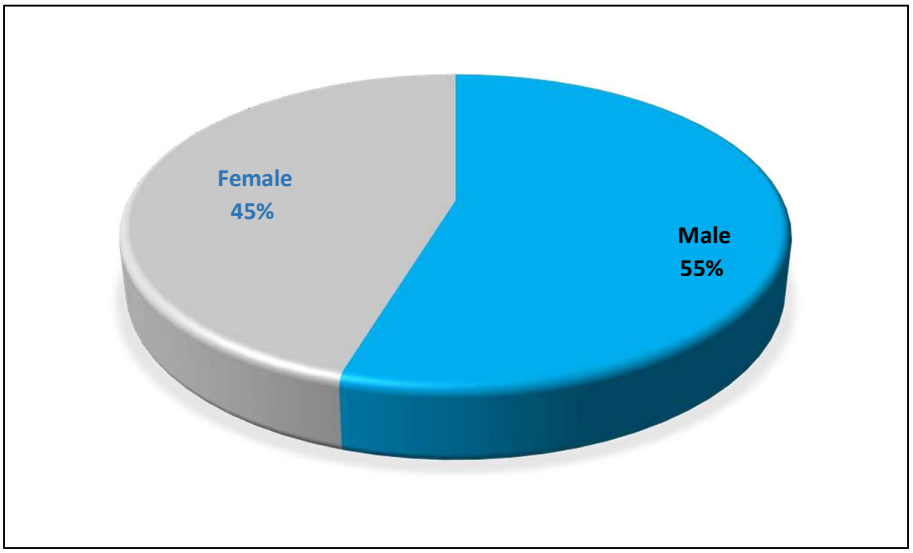


**Figure 7: Distribution of subjects based on Gender.**

**Table 5: Distribution of subjects based on the diagnosis.**

| | | |
|---|---|---|
| Diagnosis -Before ECHO | Chest Infection (Bronchopneumonia/ LRTI) | 33 (44%) |
| | Acute Bronchiolitis | 4 (5.33%) |
| | DSS | 17 (22.66%) |
| | Cardiomyopathy | 0 (0%) |
| | CHD | 21 (28%) |
| | RHD | 0 (0%) |
| Diagnosis -After ECHO | Chest Infection (Bronchopneumonia and Lobar pneumonia) | 14 (18.6%) |
| | Cardiomyopathy | 2 (2.6%) |
| | Acute Bronchiolitis | 2 (2.6%) |
| | Myocarditis | 17 (22.6%) |
| | CHD | 31 (41.3%) |
| | RHD | 3 (4%) |
| | DSS | 6 (8%) |

It can be observed that 33(44%) of subjects were diagnosed with chest infection, 21(28%) withCHD, 17(22.66%) with DSS, and 4(5.33%) with Acute Bronchiolitis before ECHO.

After ECHO, it was observed that 31(41.3%) were diagnosed with CHD, 17(22.6%) withMyocarditis followed by 14(18.6%) with chest infection.

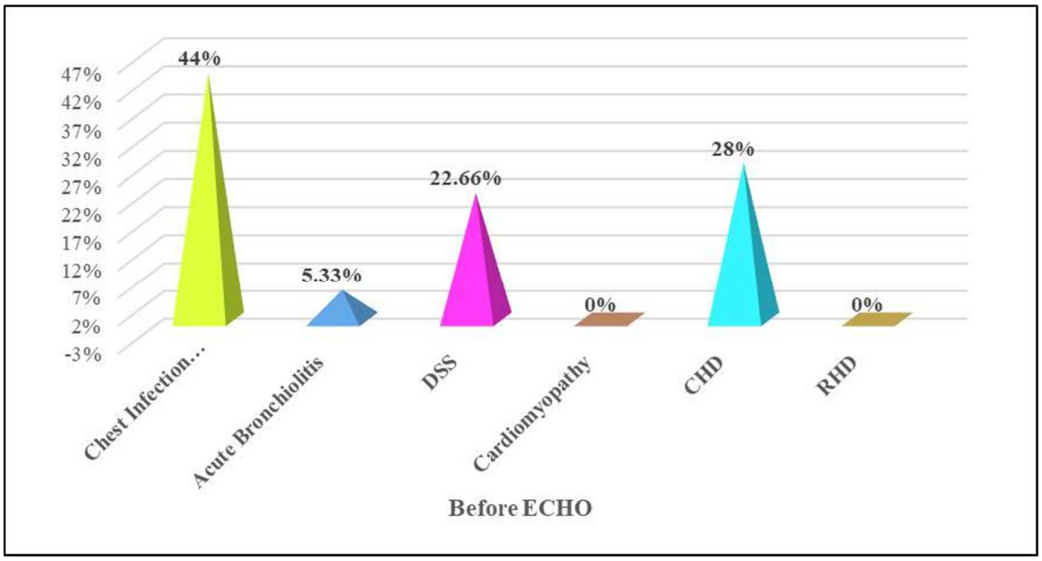


**Figure 8: Distribution of subjects based on Diagnosis-Before ECHO.**

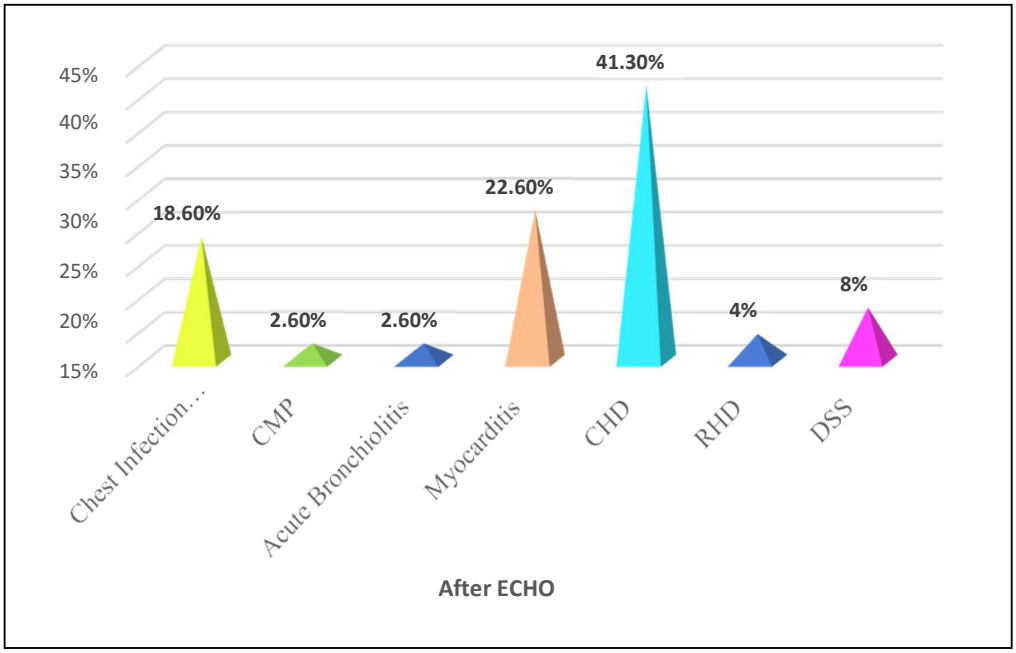


**Figure 9: Distribution of subjects based on Diagnosis-After ECHO.**

**Table 6: Distribution of subjects on Respiratory Support.**

| | **Oxygen by Mask** | **HFNC** | **Ventilator** |
|---|---|---|---|
| **Respiratory Support** | 34 (45.3%) | 36 (48%) | 1 (1.3%) |

**Table 7: Distribution of subjects based on Echocardiographic findings.**

| **ECHO Findings** | **Range** | **Mean ± SD** |
|---|---|---|
| E/A Ratio | 0.03 - 2 | 1.26 ± 0.26 |
| PAP (mmHg) | 25 - 72 | 35.93 ± 13.78 |
| FS% | 10 - 30 | 24.41 ± 5.45 |
| EF% | 17 - 65 | 51.74 ± 12.33 |

It can be observed from ECHO findings that the mean E/A ratio is 1.26 with a 0.03-2 range.PAP with a mean of 35.93 and a range of 25-72. FS% with a mean of 24.41 and a range of 10-

30. EF% has a mean of 51.74 and ranges from 17-65. The Majority of IVC collapse onrespiration is no with 64(85.3%).

**Table 8: Echocardiography guiding fluid status of patients**

| | Sub Category | Number (%) |
|---|---|---|
| IVC Collapse on Respiration | Dilated | 3 (4%) |
| | No | 64 (85.3%) |
| | Yes | 8 (10.7%) |

It can be observed that the majority of the subjects did not have IVC collapse 64 (85.3%), followed by 8 (10.7%) had IVC collapse, which required fluid boluses.

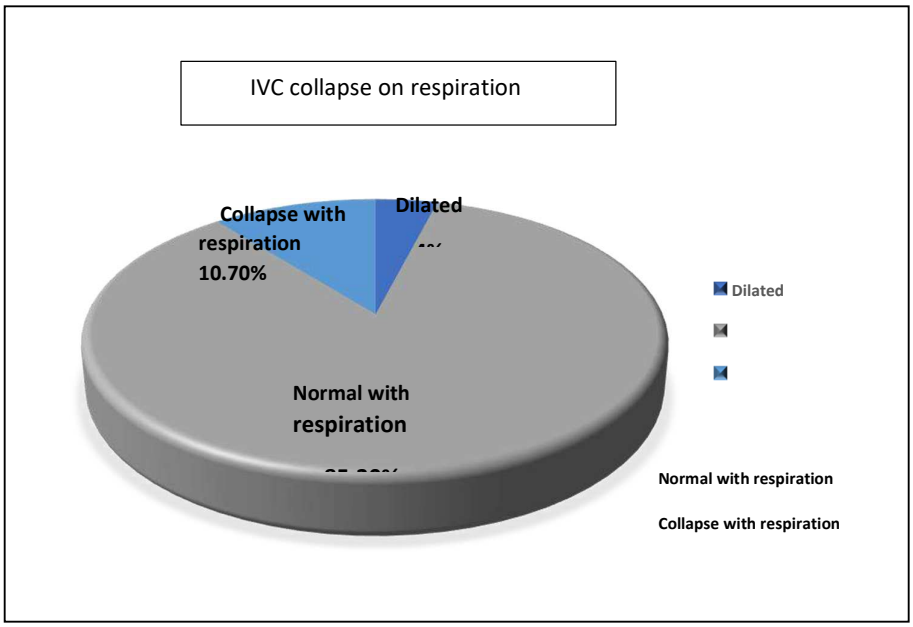


**Figure 10- pi-chart showing IVC relation with respiration**

It can be observed that 8 (10.7%) of subjects underwent Echocardiographic guiding fluid therapy

**Table 9- The following table shows the Distribution of subjects based on the impact of ECHO in guiding the change in treatment.**

| Intervention | No (%) |
|---|---|
| Administering Fluid bolus | 8 (10.6%) |
| Fluid Restriction | 5 (6.6%) |
| Initiation of Inotropes | 16 (21.3%) |
| Increase of Inotropes | 6 (8%) |
| Initiation of diuretics | 23 (30.6%) |
| Initiation of digoxin | 28 (37.3%) |
| Surgical Intervention | 26 (34.6.) |
| IVIg initiation | 15(20%) |
| No Intervention | 12 (16.0%) |

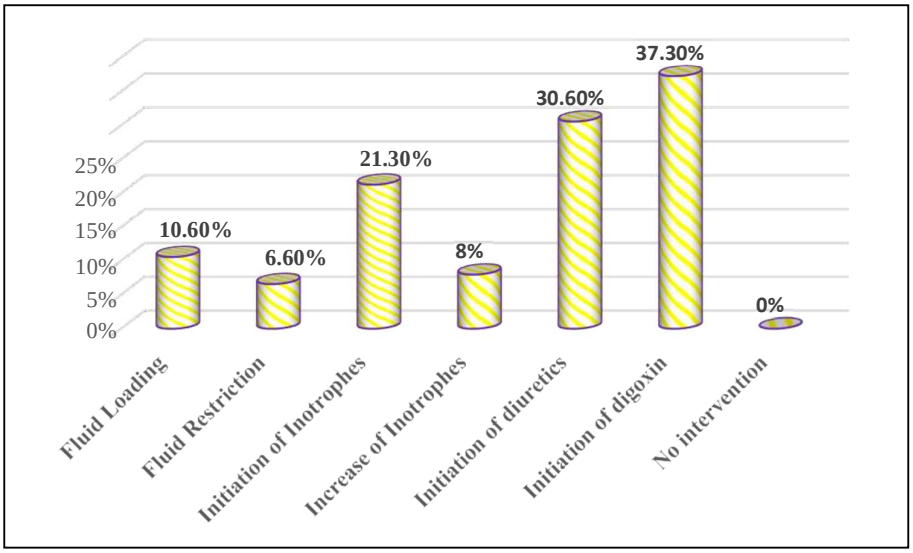


**Figure 11: Distribution of subjects based on intervention on the impact of ECHO on decision making.**

ECHO has changed our management in patients with CHD in 28(45.9%). In patients with pulmonary HTN and patients with reduced LVF, it is noteworthy that the p-value of all resultswas <0.005.

The following table shows the association between changing interventions after Echo and CHD, Pulmonary HTN, and Reduced LV function LVH dilatation.

**Table 10: Changing interventions after Echo**

| | **Altered drug Therapy** | **No difference** | **$X^2$** | **p-value** |
|---|---|---|---|---|
| | **N= 64** | **N=11** | | |
| CHD | 28(45.9%) | 0(0%) | 10.255 | 0.0014[C◦] |
| Pulmonary HTN | 10(16.3%) | 8(57.1%) | 10.366 | 0.0055 [MC ◦] |
| Reduced LV function LVH dilation | 23(37.7%) | 0(0%) | 7.6135 | 0.012 [MC ◦] |

*Abbreviation: C – Chi square test, MC – Chi square test with Monte Carlo simulation, * indicates statistical significance.*

From Chi square test, it is observed that, there is significant association of CHD, pulmonary HTN and Reduced LV function LVH dilation with changing intervention. The odds of CHD are 0.0405 (95% CI: 0.0023 - 0.7099) time less among those whose interventions are not changed compared to those whose interventions are changed. The odds of pulmonary HTN is 6.8 (95% CI: 1.9348 - 23.8996) time more among those whose interventions are not changed compared to those whose interventions are changed. The odds of Reduced LV function LVH dilation is 0.056 (95% CI: 0.0032 - 0.9919) time less among those whose interventions are not changed compared to those whose interventions are changed.

**Table 11: Distribution of subjects based on outcome of the study after ECHO.**

| Primary outcome | No. of subjects | Survival | Death |
|---|---|---|---|
| Pneumonia | 14 | 14 | 0 |
| CHD | 31 | 28 | 3 |
| DSS | 6 | 6 | 0 |
| Cardiomyopathy | 2 | 2 | 0 |
| RHD | 3 | 3 | 0 |
| Myocarditis | 17 | 15 | 2 |

It can be observed that among the outcomes, death was observed in 2 subjects with Myocarditis, 3 with CHD.

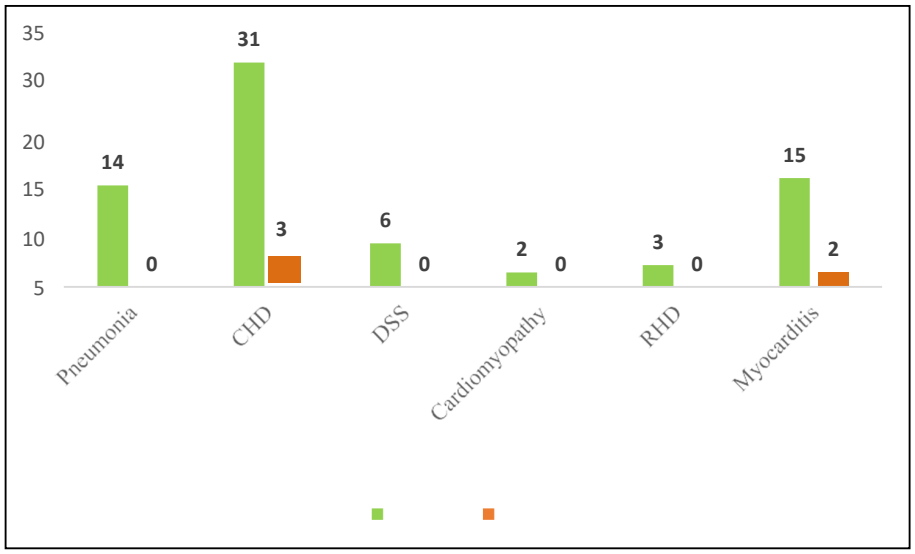


**Figure 12- Graph showing outcome of the study .**

# DISCUSSION

Echocardiography is based on ultrasound technology to image the heart and associated vascularstructures. From the reflected energy or so-called ultrasound echoes, a cardiac image is created.Standard views used for pediatric examinations are-

- Parasternal window (obtained from the high left chest just lateral to the sternum,)
- Apical window is (the left lateral chest just inferior and lateral to the nipple)
- subcostal window (sub-xiphoid area)
- suprasternal window (the suprasternal notch)

Acute respiratory distress needs to be treated immediately, and the key to providing effective management is early patient evaluation.[32] Diagnosis of Heart Failure is not always straightforward. Its diagnosis relies on several sources of clinical findings, including history, physical examination, chest radiographs, and echocardiographic studies. There is no single particular test that can diagnose CHF.

Cardiomegaly on a chest film (or echocardiographic scan), in addition to physical signs, is practically a requisite sign of CHF. According to Nir et al. (2005), most children with pressure overload or volume overload cardiac lesions had higher levels of BNP and the N terminal segment of its prohormone (NT-ProBNT) than normal children. Because an appropriate reference range hasn't been established, the utility of the amounts of these peptides, however, seems to be constrained. Depending on the commercial testing kits utilized, the values of these peptides vary.[41] An electrocardiogram (ECG) is possibly the least important test for the diagnosis of CHF, but it helps in identifying the cause of heart failure .Respiratory distress and cardiac failure were factors in 30 to 50 percent of admissions to the pediatric intensive care

unit, according to a 2008 research by Pasquali and his colleagues. Acute heart failure, cyanotic episodes, arrhythmias, heart failure associated with a lower respiratory tract infection, and imminent respiratory failure are the most frequent causes. However, multisystemic involvement might occasionally make the clinical picture more difficult, which raises the morbidity rate in these children. Therefore, a thorough examination of the children'shemodynamics is required.[33]

According to the American College of Critical Care Medicine's 2017 guidelines, a detailed hemodynamic assessment includes the evaluation of parameters such as temperature, sensorium, capillary refill, heart rate, rhythm by electrocardiography, oxygen saturation monitoring, blood pressure, and superior vena cava saturation monitoring. However, diagnosis of heart failure appeared to be always difficult due to the presence of several markers influencing cardiac function. When performed by expert hands, echocardiography looks to be a safe, non-invasive, and quick method of obtaining extensive information on ventricular function. As a result, echocardiography confirmation of acute heart failure preserves the golden hour, allowing for prompt therapy and improved results.

Echocardiography is the most beneficial non-invasive investigation that determines the severity of heart failure and validates the diagnosis of heart failure is echocardiographic. It could also aid in determining the etiology of heart failure. It also helps in the evaluation and monitoring of patients with acute HF because it can come up with both diagnostic and prognostic information [42]. Additional hemodynamic data may also be collected, including cardiac output (CO), end-diastolic volume, both global and local ventricular function, and valvular anomalies.By using Doppler techniques, echocardiographic examinations can demonstrate enlargement of the ventricular chambers, impaired LV systolic performance (reduced

fractional shortening or ejection fraction), as well as impaired diastolic function. The severity of valve regurgitation ,the degree of left and right ventricular function, and a non-invasive assessment of filling pressures can all be obtained via echocardiography. Heart failure (HF) is a composite clinical syndrome resulting from varied primary and secondary causes and shared pathways of disease progression, correlating with significant mortality, morbidity, and cost". The global burden of HF is not known, as many children do not have access to medical services and die of HF each year [43]. Confirmation of the diagnosis of acute heart failure by Echocardiography is essential and should be performed in the next to no time following suspicion of the diagnosis; Echocardiography is quick, non-invasive, and safe and provides substantial particulars on heartfailur.[51]

The present observational study was conducted in the department of Paediatrics at JNMC Medical College, Belgaum from Jan 2021 to Dec 2021. A total of 75 study participants were enrolled. In this study, we observed that the majority 44(%) of subjects are of age within 5 years, and 17 patients (22.66%) among 5-12 years of age.

Our study reveals that males 41 (54.7%) had a greater risk of developing heart failure than females 34 (45. 3%). In contrast, research conducted by Grayburn et al. (2005) found that girlswere at greater risk than boys, and this can be explained by the fact that in our culture there isa preferential referral to boys over girls in some families. Our findings are consistent with thoseof Kutty et al. 2014, who found that boys (58%) more often than girls (42%) need ECHO in pediatric critical care. [32] our study is also supported by the findings of the 2016 study by Rabah et al. There were 57 (56.4%) male patients and the majority (n = 41, 40.6%) were infantsunder 1-year-old.

In our study, chest infection (pneumonia & bronchiolitis),44% was the most frequent cause of HF with respiratory distress, and congenital heart disease (CHD)22.6% was the 2nd most prevalent cause, supported by the study done by Ahmed et al. (2015), These results were supported by another study done by Ayat et al. 2019 where congenital heart disease was the commonest indication for ECHO in 47 patients (27.65%) followed by respiratory distress in 24 patients 14.12%. These results were in contrast to another study conducted by Rabah et al, 2016 observed normal echocardiography was the most common finding (31.7%) followed by congenital heart disease (24.7%).[26][49,50]

Our study found that cardiomyopathy is still an important cause of heart failure, which is in line with research by Ahmed et al. (2015), Grayburn et al. (2005), and Nishimura and Tajik (2009).[32][44,45]

The presence of a heart murmur, a positive history of prior attacks, and a throat infection all point to a cardiac etiology for rheumatic fever. But few studies revealed that auscultation had low sensitivity and specificity in detecting abnormal murmurs. Subclinical RHD was introduced with the use of ECHO to acknowledge the possibility of RHD being silent. A studyperformed by Marijon et al in Cambodia and Mozambique, compared auscultatory to echocardiographic screening in over 5000 children. Ten times more RHD was detected through an echocardiograph. Additional studies persistently emphasized the superiority of echocardiography in detecting latent RHD [46,47].

Cases with myocarditis lack the clue from the history and examination and were detected whiledoing echocardiography for unexplained respiratory distress. This highlights the value of an echocardiographic assessment in children who have unexplained respiratory distress, especially if there is tachycardia. In our study,

18.6% of cases were diagnosed after ECHO which remains undiagnosed at the time of admission, supported by Nagueh et al.,2007[45]. In myocarditis, Electrocardiographic changes are considered to be present commonly, particularly ST-T anomalies, but they are also nonspecific and can imitate coronary heart disease.

All cases underwent echocardiography, and of the total echocardiography parameters, five were chosen for analysis:

- The E/A Ratio.
- The Pulmonary Artery Pressure
- The Fractional Shortening, and
- The Ejection Fraction.

These parameters were used for diagnosis, and to evaluate our management, Inferior vena cava (IVC) collapse during respiration was used.[32]

Accurate assessment of the diastolic function of the heart by echocardiography is an evolving field that has made notable progress in the recent past. Diastolic heart failure and its effects onpostoperative care also need to be taken into account. It's important to consider diastolic heart failure and how it affects postoperative care.

Diastolic dysfunction is indicated by a prominent pulmonary vein atrial reversal wave (a wave). This observation shows pronounced flow reversal into the pulmonary veins during atrial systole as a result of a non-compliant ventricular chamber. Additionally, the mitral inflow Doppler pattern can be a helpful index of diastolic dysfunction. it consists of two waves, An‘A’ wave reflects active filling because of atrial systole, and an ‘E wave, signifies early passive ventricular filling (preload dependent).

In patients with diastolic dysfunction, the E: A ratio can be altered i.e rate of E wave deceleration, and the duration of the A wave. According to our study, critically unwell infantsand children had a notable reduction in E-wave and E/A ratio, which suggests LV diastolic dysfunction.

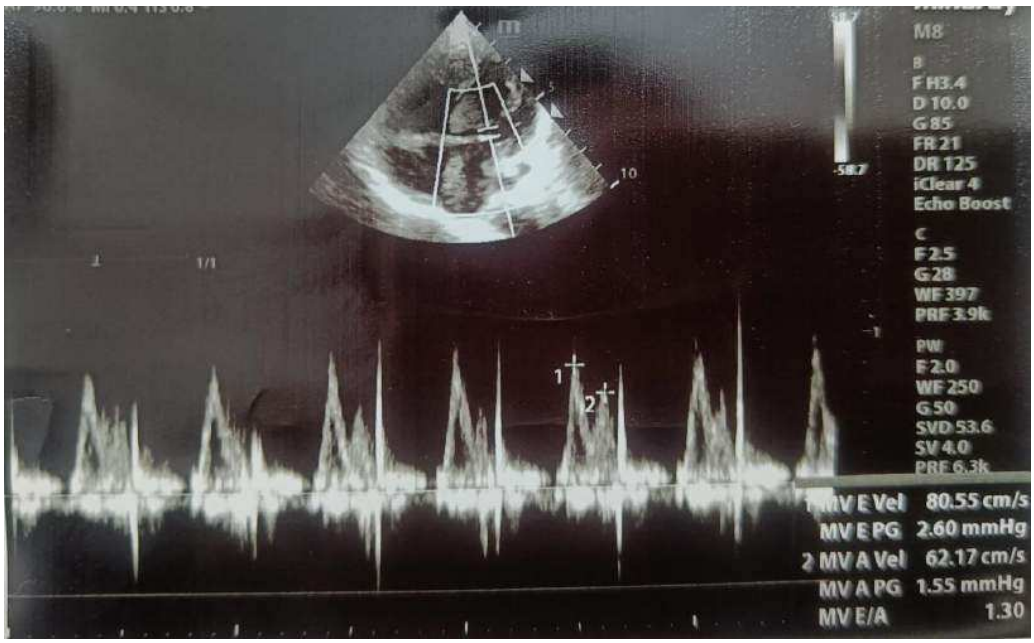


**Figure-13. Echocardiography showing E/A ratio of 1.3 suggestive of normal left ventricle diastolicfunction.**

According to Our study, there was a significant decrease in E-wave and E/A ratio in gravely illinfants and children, suggesting LV diastolic dysfunction. This is fairly consistent with El- Khuffash and McNamara et al. (2011), who suggest that E/A ratios less than 1 signify diastolic dysfunction and poor myocardial relaxation.[8]

This might be explained by the fact that the isovolumic relaxation time increases as left ventricular relaxation deteriorates. E-wave is attenuated, and the E-wave deceleration time is extended, as this condition involves a compensatory rise of the A-wave, the E/A ratio becomesless than 1.[48]

As an extension to a physical examination, echocardiographic evaluation of left ventricular (LV) function has been shown to improve both the timing and efficacy of therapy. By using echocardiography, the left ventricular systolic function can be evaluated both qualitatively and quantitatively

- Qualitative Analysis of the LV Systolic Function-

It involves the examiner's visual interpretation in connection to the myocardial contractile function preferred in PICU settings and also by Non-echocardiographers toassess LV function using the qualitative examination of the LV systolic function.

The parasternal (long and short views), the apical and the subcostal views, and numerous echocardiography views are used to visually evaluate the left ventricular ejection fraction (EF). This evaluation is done by looking at the myocardial thickness during systole and the decrease in the diameter of the ventricular chamber during systole compared to diastole, which is provided by the motion of the ventricular wall during systole. Subjectively stated, LV function is divided into four categories: normal (EF 55%)

Slightly reduced (EF 41%-55%)

Considerably reduced (EF 31%-40%), andsignificantly impaired (EF 30%).

- Quantitative Analysis of the LV Systolic Function-

LV ejection fraction and cardiac output/index measures are used in the quantitative investigation of the LV systolic function. This information enables the physician to make decisions about therapeutic choices like fluids and/or inotropic drugs.

The M mode or two-dimensional mode can be used to measure the ejection fraction. Its calculation in the M mode is the most widely used in clinical practice,

especially in pediatric patients, and is obtained from the fractional shortening (FS) measurement. In all cases of

cardiomyopathy and viral myocarditis, it was noticed that ejection fraction and fractional shortening were substantially affected or lowered, with ejection fraction occasionally falling below 15%. It was noted that ejection fraction and fractional shortening severely affected or decreased in all cases with cardiomyopathy and viral myocarditis, as ejection fraction may reach less than 15, This is in strong concordance with Nosir et al 2009, whereas the ejection fraction might range from 30% to 54% in other forms of heart failure. Therefore, the ejection fraction and fractional shortening may be considered reliable indications of dilated cardiomyopathy and myocarditis together with increased cardiac dimension, however using these two characteristics alone, we cannot distinguish between the two cases. Therefore, the need for initiation of IVIG therapy was required in 15(20%) cases diagnosed with myocarditis after ECHO.

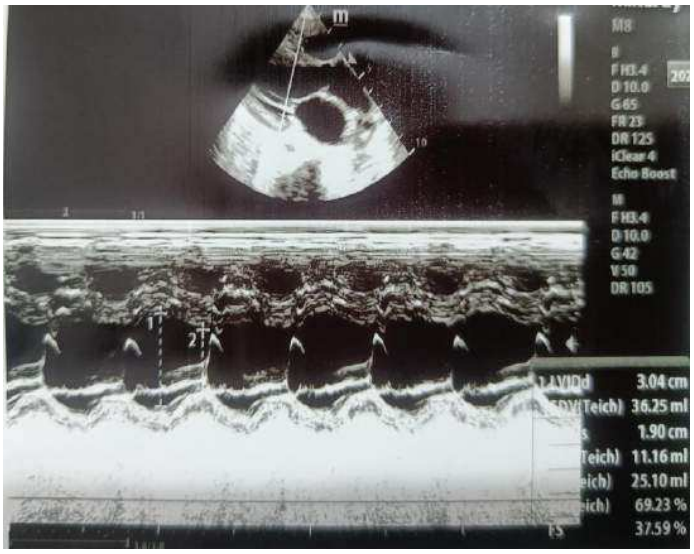


**Figure-14. Echocardiography showing fractional shortening of 37.59% and ejection fraction of69.23% with good biventricular function in a patient of the ventricular septal defect.**

Regarding changes in pulmonary artery pressure, the mean PAP was 375.65, which is in the upper normal range; in 19/75 cases, there was mild to moderate pulmonary hypertension, and

in 4/75 cases, there was severe pulmonary hypertension (CHD), which could be attributed to the underlying disease's characteristics. According to several studies Manasia et al., 2005; Croft et al., 2006), the use of echocardiography in the care of critically ill patients has a positiveeffect that changes the course of treatment in 30% to 60% of instances.[37,]

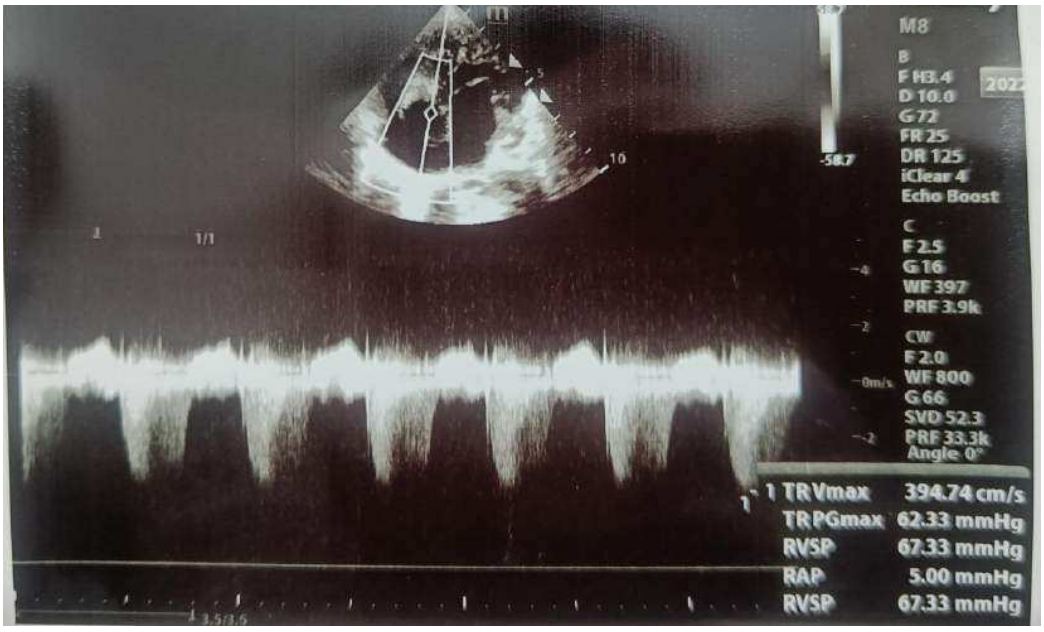


**Figure- 15. Echocardiography showing Pulmonary artery pressure is 67.33 mm Hg, signifying severe pulmonary artery hypertension.**

The inferior vena cava (IVC) diameter is the first method used in echocardiography to evaluate preload and fluid responsiveness. The most common echocardiographic technique for evaluating fluid responsiveness is respiratory changes in IVC diameter, which involves analyzing the IVC diameter change with respiration while receiving positive pressure ventilation (inspiration and expiration)

Our study showed that Echocardiography also aids in decision-making in the studied cases byassessing inferior vena cava collapse with respiration, our study showed that: some cases need fluid loading (10.6%), and others needed fluid restriction (6.6%). Our findings are supported by another study done by Heloisa et.al.,2015 proved a strong correlation between respiratory change in IVC and the patient's fluid responsiveness, The authors demonstrated a linear relationship between respiratory changes in the IVC and elevated Cardiac output.

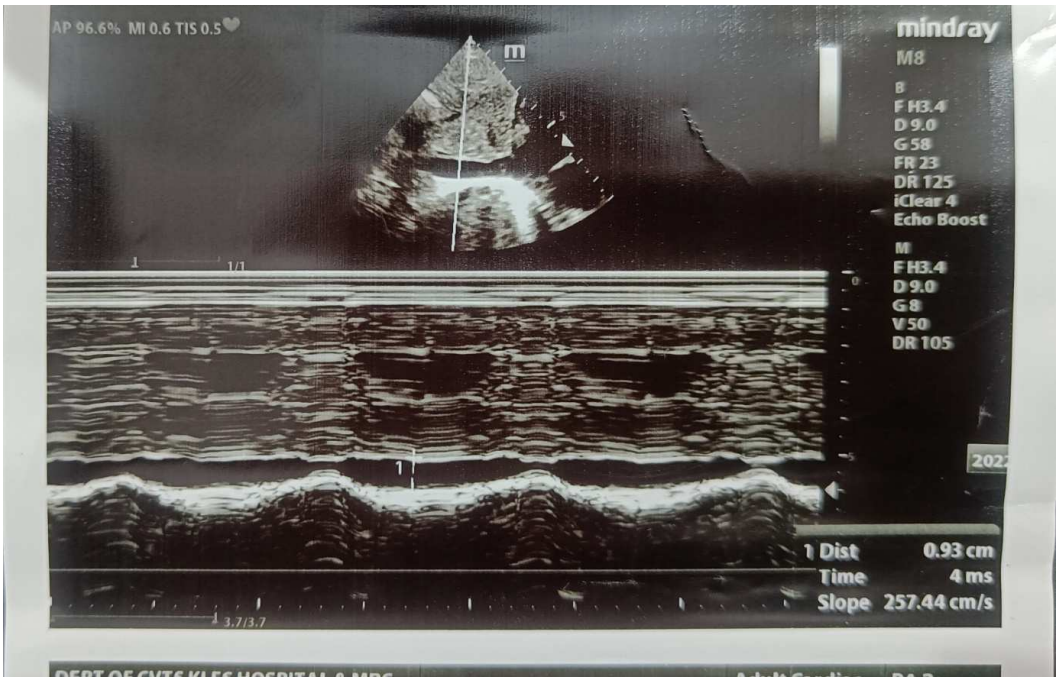


**Figure-16. bedside echocardiography showing change in inferior vena cava diameter with respiration, (0.93 cm) which along with other clinical data showed that fluid resuscitation should be maintained. Neither there is fluid overload nor IVC collapse.**

initial treatment of shock is fluid resuscitation. Aggressive fluid resuscitation, however, could be detrimental to some people in cardiogenic shock. Children's fluid responsiveness cannot be predicted by clinical evaluation or static measurements of filling pressures (central venous pressure and pulmonary wedge pressure), which is

consistent with findings in adults, and examination of pediatric patients is made more difficult by the fact that dynamic indicators like pulse pressure variation and stroke volume variation did not predict fluid responsiveness in children as they did in adults.\

Before, during, and after percutaneous ASD/PFO, PDA device closure, and echocardiography is crucial. In fact, the American Society of Echocardiography recently advocated its use for procedural guiding [28].

Following an echocardiogram, surgical intervention was recommended for 26 (34%) of the patients. A similar study done by Saffa et al, revealed that the P-value of the results for the majority of patients [163 patients (95.88%)] was significant (<0.05%), the echocardiographic results and the therapeutic decision or intervention in this study showed a strong favorable association, the same in Rabah et al, 2016 study a significant P-value of all results was significant.[49]

# CONCLUSION

- Echocardiography is a quick, safe, non-invasive method that is capable of giving detailed information about the structure and function of the heart. It can help with diagnosis and give therapeutic recommendations for seriously ill children who have been admitted to a pediatric intensive care unit (PICU) and are experiencing acute respiratory distress associated with heart failure.
- Echocardiography aids in determining the cause of CHF.
- Serial examination of the effectiveness of therapy using echocardiography is alsoadvantageous.
- ECHO also influences decision-making by recommending the initiation of IVIG therapy for patients diagnosed with myocarditis.
- Patients with congenital heart disease and those exhibiting signs of respiratory distresswere identified to be the most common two reasons for echocardiography in this study.
- Echocardiography helps in prompt referring to a cardiothoracic surgeon if any surgicalintervention is required.

# LIMITATIONS

- The study was conducted in a single location with a limited sample size.
- The lack of blinding and randomization in this trial hindered proper assessment of the impact of echocardiography on the outcome of critically ill children.
- Such restrictions preclude the study's conclusions from being applied generally

# SUMMARY

The study was conducted over a period of 1 year between January 2021 and December 2021 at KLES Dr. Prabhakar Kore Hospital. 75 children from age 2 months to 18 years having signs of respiratory distress with the manifestation of heart failure in the form of (tachypnoea, tachycardia, chest indrawing,intercostal retraction, enlarged tender liver, cyanosis, altered level of consciousness) were enrolled. Patients diagnosed as having heart failure according to Modified ROSS and Framingham Criteria for Heart Failure. All enrolled cases underwent Echocardiographic examination, Standard views used for examinations were-

- Parasternal window (obtained from the high left chest just lateral to the sternum,)
- Apical window is (the left lateral chest just inferior and lateral to the nipple)
- subcostal window (sub-xiphoid area)
- suprasternal window (the suprasternal notch)

Total 5 echocardiographic parameters were chosen for analysis:

- The E/A Ratio.
- The Pulmonary Artery Pressure
- The Fractional Shortening, and
- The Ejection Fraction.

These parameters were used for diagnosis, and to evaluate our management, Inferior vena cava (IVC) collapse during respiration was used.[32]

Excel and SPSS software version 21 was used for data analysis. Frequency tables were used to present categorical variables. The format for continuous variables was Mean SD/ Median (Min, Max).

To examine the relationship between categorical variables, we used the chi-square test. A P-value of 0.05 or less suggests statistical significance.

- In this study, we observed that the majority of 22 (29%) subjects were of age less than 5 years and 17 patients (22.66%) among 5-12 years of age.
- Male: Female ratio was 1.2:1.
- It was observed that before ECHO, 33(44%) subjects were diagnosed with chest infection, 21(28%) with CHD, 17(22.66%) with DSS, and 4(5.33%) with Acute Bronchiolitis.
- After ECHO, it was observed that 31(41.3%) were diagnosed with CHD, 17(22.6%) withMyocarditis followed by 14(18.6%) with chest infection.
- In patients with diastolic dysfunction, the E: A ratio can be altered. According to our study, critically unwell infants and children had a notable reduction in E-wave and E/A ratio, which suggests LV diastolic dysfunction.
- In our study we observed, that the mean E/A ratio is 1.26 with a 0.03-2 range. PAP with a mean of 35.93 and a range of 25-72, similarly FS% with a mean of 24.41 and a range of 10-30, we also observed that EF% has a mean of 51.74 and ranges from 17-65. It was observed that the majority of the subjects did not have IVC collapse 64 (85.3%), followed by 8 (10.7%) had IVC collapse, which required fluid boluses.
- Our study showed there was a significant impact of Echocardiography in guiding the treatment for PICU patients, there was a requirement of initiation of inotropes in 21.3 % of patients after performing ECHO, 8% of patients required an increase in inotropes, we initiated decongestive therapy in 37% of patients, 34.6% patients were referred, for surgical interventions.

- Cases with myocarditis lack the clue from the history and examination and were detected while doing echocardiography for unexplained respiratory distress. This highlights the value of an echocardiographic assessment in children who have unexplained respiratory distress, especially if there is tachycardia.

- In our study, 22.6% of cases were diagnosed with myocarditis after ECHO which remains undiagnosed at the time of admission, out of which 20% of patients required IVIg.

- ECHO has changed our management in patients with CHD in 28(45.9%). In patients with pulmonary HTN and patients with reduced LVF, it is noteworthy that the p-value of all resultswas <0.005.

## BIBLIOGRAPHY


1. Braunwald E. Heart disease: a textbook of cardiovascular medicine Braunwald E, editor; 1992.
2. Yancy CW, Jessup M, Bozkurt B, Butler J, Casey DE Jr, Drazner MH et al. 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2013;62(16):e147-239. doi: 10.1016/j.jacc.2013.05.019, PMID 23747642.Wheeler DS, Wong HR, Shanley TP. Cardiovascular Pediatric Critical Illness and Injury. Springer; 2009 Mar 1.
3. Refer1, Rimington H, Adam G, Chambers JOpen-access echocardiography. Lancet. 1996 Aug 24;348(9026):555-6. doi: 10.1016/S0140-6736(05)64722-8, PMID 8757178.
4. Stanko LK, Jacobsohn E, Tam JW, De Wet CJ, Avidan M. Transthoracic echocardiography: impact on diagnosis and management in tertiary care intensive care units. Anaesth Intensive Care. 2005 Aug;33(4):492-6. doi: 10.1177/0310057X0503300411, PMID 16119491.
5. Orme RM, Oram MP, McKinstry CE. Impact of echocardiography on patient management in the intensive care unit: an audit of district general hospital practice. Br J Anaesth. 2009 Mar 1;102(3):340-4. doi: 10.1093/bja/aen378, PMID 19151420.

6. Slama M, Maizel J. Echocardiographic measurement of ventricular function. Curr Opin Crit Care. 2006 Jun 1;12(3):241-8. doi: 10.1097/01.ccx.0000224869.86205.1a, PMID 16672784.
7. Goldstein M, Vincent JL, Kahn RJ. Evaluation of cardiac function by echo-Doppler studies in critically ill patients. Intensive Care Med. 1988;14(4):406-10. doi: 10.1007/BF00262897, PMID 3403772.
8. Joseph MX, Disney PJS, da Costa R, Hutchison SJ. Transthoracic echocardiography to identify or exclude cardiac cause of shock. Chest. 2004 Nov;126(5):1592-7. doi: 10.1378/chest.126.5.1592, PMID 15539732.
9. Mousavi N, Czarnecki A, Ahmadie R, Tielan Fang KK, Kumar K, Lytwyn M, et al. The utility of tissue doppler imaging for the noninvasive determination of left ventricular filling pressures in patients with septic shock. J Intensive Care Med. 2010;25(3):163-7. doi: 10.1177/0885066609359903, PMID 20444737.
10. Mookadam F, Jiamsripong P, Goel R, Warsame TA, Emani UR, Khandheria BK. Critical appraisal on the utility of echocardiography in the management of acute pulmonary embolism. Cardiol Rev. 2010;18(1):29-37. doi: 10.1097/CRD.0b013e3181c09443, PMID 20010336.
11. Filippatos DK, Poole GM, Van Veldhuisen WP, Keren DA, Ponikowski P, Poole-Wilson PA et al. ESC guidelines for the diagnosis and treatment of acute and chronic heart failure 2008: the Task Force for the diagnosis and treatment of acute and chronic heart failure 2008 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association of the ESC (HFA) and endorsed by the European Society of Intensive Care

Medicine (ESICM). Eur J Heart Fail. 2008;10(10):933-89.ences. doi: 10.1016/j.ejheart.2008.08.005, PMID 18826876.

12. Das BB. Current state of pediatric heart failure. Children (Basel). 2018;5(7):88. doi: 10.3390/children5070088, PMID 29958420.
13. Lipshultz SE, Sleeper LA, Towbin JA, Lowe AM, Orav EJ, Cox GF et al. The incidence of pediatric cardiomyopathy in two regions of the United States. N Engl J Med. 2003 Apr 24;348(17):1647-55. doi: 10.1056/NEJMoa021715, PMID 12711739.
14. Rosenthal D, Chrisant MR, Edens E, Mahony L, Canter C, Colan S et al. International Society for Heart and Lung Transplantation: practice guidelines for management of heart failure in children. J Heart Lung Transplant. 2004 Dec 1;23(12):1313-33. doi: 10.1016/j.healun.2004.03.018, PMID 15607659.
15. Shaddy RE, George AT, Jaecklin T, Lochlainn EN, Thakur L, Agrawal R et al. Systematic literature review on the incidence and prevalence of heart failure in children and adolescents. Pediatr Cardiol. 2018 Mar;39(3):415-36. doi: 10.1007/s00246-017-1787-2, PMID 29260263.
16. Rossano JW, Kim JJ, Decker JA, Price JF, Zafar F, Graves DE et al. Prevalence, morbidity, and mortality of heart failure–related hospitalizations in children in the United States: a population-based study. J Card Fail. 2012 Jun 1;18(6):459-70. doi: 10.1016/j.cardfail.2012.03.001, PMID 22633303.
17. Vaidyanathan B, Sathish G, Mohanan ST, Sundaram KR, Warrier KK, Kumar RK. Clinical screening for congenital heart disease at birth: a

prospective study in a community hospital in Kerala. Indian Pediatr. 2011 Jan;48(1):25-30. doi: 10.1007/s13312-011-0021-1, PMID 20972295.

18. Saxena A, Mehta A, Sharma M, Salhan S, Kalaivani M, Ramakrishnan S et al. Birth prevalence of congenital heart disease: A cross-sectional observational study from North India. Ann Pediatr Cardiol. 2016 Sep;9(3):205-9. doi: 10.4103/0974-2069.189122, PMID 27625516.
19. Saxena A. Congenital heart disease in India: a status report. Indian J Pediatr. 2005 Jul;72(7):595-8. doi: 10.1007/BF02724185, PMID 16077244.
20. Lipshultz SE, Sleeper LA, Towbin JA, Lowe AM, Orav EJ, Cox GF et al. The incidence of pediatric cardiomyopathy in two regions of the United States. N Engl J Med. 2003 Apr 24;348(17):1647-55. doi: 10.1056/NEJMoa021715, PMID 12711739.
21. Packer M. Acute heart failure is an event rather than a disease: Plea for a Radical Change in Thinking and in Therapeutic Drug Development. JACC Heart Fail. 2018;6(1):73-5. doi: 10.1016/j.jchf.2017.05.008, PMID 29284579.
22. Pediatric Bronchoscopy Collaborative Group, Corp-Author. The subspecialty group of respiratory diseases, the society of pediatrics, Chinese medical association. Guide to pediatric bronchoscopy. 2009 ed. Zhonghua ErKeZaZhi. 2009;47:740-4.
23. Qun J, Zhou J, Yu Y. An official Chinese Thoracic Society Clinical Practice guideline: the etiologic assessment of bronchoalveolar lavage in infectious lung disease. Chin J Tuberc Respir Dis. 2017;40:578-83.

24. Bode-Thomas F, Hyacinth I, Yilgwan C. Co-existence of ventricular septal defect and bronchial asthma in two Nigerian children. Clin Med Insights Case Rep. 2010;3:5-8. doi: 10.4137/ccrep.s4584, PMID 20657755.

25. Said SI, Hamidi SA, Gonzalez Bosc L. Asthma and pulmonary arterial hypertension: do they share a key mechanism of pathogenesis? Eur Respir J. 2010;35(4):730-4. doi: 10.1183/09031936.00097109, PMID 20356986.

26. Zhang Y, Chen Y, Chen Z, Zhou Y, Sheng Y, Xu D et al. Effects of bronchoalveolar lavage on refractory Mycoplasma pneumoniae pneumonia. Respir Care. 2014;59(9):1433-9. doi: 10.4187/respcare.03032, PMID 24962224.

27. Lui GK, Saidi A, Bhatt AB, Burchill LJ, Deen JF, Earing MG, et al. Diagnosis and management of noncardiac complications in adults with congenital heart disease: A Scientific Statement from the American Heart Association. Circulation. 2017;136(20):e348-92. doi: 10.1161/CIR.0000000000000535, PMID 28993401.

28. Lee SL, Cheung YF, Leung MP, Ng YK, Tsoi NS. Airway obstruction in children with congenital heart disease: assessment by flexible bronchoscopy. Pediatr Pulmonol. 2002;34(4):304-11. doi: 10.1002/ppul.10164, PMID 12205572.

29. Henry WL, DeMaria A, Gramiak R, King DL, Kisslo JA, Popp RL et al.. Report of the American Society of Echocardiography Committee on Nomenclature and Standards in Two-dimensional Echocardiography. Circulation. 1980 Aug;62(2):212-7. doi: 10.1161/01.cir.62.2.212, PMID 7397962.

30. Mohsen A. Abd el- H, Mohamed I, Abu F. Impact of echocardiography on decision making in PICU. Al-Azhar Assiut Med J;13(1):153–8:2015Jan.

31. Pasquali SK, Hall M, Slonim AD, Jenkins KJ, Marino BS, Cohen MS et al. Off-label use of cardiovascular medications in children hospitalized with congenital and acquired heart disease. Circ Cardiovasc Qual Outcomes. 2008 Nov;1(2):74-83. doi: 10.1161/CIRCOUTCOMES.108.787176, PMID 20031793.

32. Rugolotto M, Hu BS, Liang DH, Schnittger I. Rapid assessment of cardiac anatomy and function with a new hand-carried ultrasound device (OptiGo): a comparison with standard echocardiography. Eur J Echocardiogr. 2001 Dec;2(4):262-9. doi: 10.1053/euje.2001.0121, PMID 11888820.

33. Heinle SK, Tice FD, Kisslo J. Effect of dobutamine stress echocardiography on mitral regurgitation. J Am Coll Cardiol. 1995;25(1):122-7. doi: 10.1016/0735-1097(94)00358-w, PMID 7798488.

34. Nohria A, Mielniczuk LM, Stevenson LW. Evaluation and monitoring of patients with acute heart failure syndromes. Am J Cardiol. 2005;96(6A):32G-40G. doi: 10.1016/j.amjcard.2005.07.019, PMID 16181821.

35. Manasia AR, Nagaraj HM, Kodali RB, Croft LB, Oropello JM, Kohli-Seth R, et al. Feasibility and potential clinical utility of goal-directed transthoracic echocardiography performed by noncardiologist intensivists using a small hand-carried device (SonoHeart) in critically ill patients. J Cardiothorac Vasc Anesth. 2005 Apr;19(2):155-9. doi: 10.1053/j.jvca.2005.01.023, PMID 15868520.

36. Vignon P, Chastagner C, François B, Martaillé JF, Normand S, Bonnivard M, et al. Diagnostic ability of hand-held echocardiography in ventilated critically ill patients. Crit Care. 2003;7(5):R84-91. doi: 10.1186/cc2360, PMID 12974974.

37. Arntfield RT, Millington SJ. Point of care cardiac ultrasound applications in the emergency department and intensive care unit--a review. Curr Cardiol Rev. 2012 May;8(2):98-108. doi: 10.2174/157340312801784952, PMID 22894759.

38. Tam JW, Nichol J, MacDiarmid AL, Lazarow N, Wolfe K. What is the real clinical utility of echocardiography? A prospective observational study. J Am Soc Echocardiogr. 1999;12(9):689-97. doi: 10.1016/s0894-7317(99)70018-0, PMID 10477412.

39. Nir A, Nasser N. Clinical value of NT-probnp and BNP in pediatric cardiology. J Card Fail. 2005;11(5);Suppl:S76-80. doi: 10.1016/j.cardfail.2005.04.009, PMID 15948106.

40. Nohria A, Mielniczuk LM, Warner Stevenson L. Evaluation and monitoring of patients with acute heart failure syndromes. Am J Cardiol. 2005;96(6):32-40.

41. Hinton RB, Ware SM. Heart failure in pediatric patients with congenital heart disease. Circ Res. 2017;120(6):978-94. doi: 10.1161/CIRCRESAHA.116.308996, PMID 28302743.

42. Nishimura RA, Tajik AJ. Evaluation of diastolic filling of left ventricle in health and disease: Doppler echocardiography is the clinician's Rosetta Stone. J Am Coll Cardiol. 1997;30(1):8-18. doi: 10.1016/s0735-1097(97)00144-7, PMID 9207615.

43. Nagueh SF, Middleton KJ, Kopelen HA, Zoghbi WA, Quiñones MA. Doppler tissue imaging: A noninvasive technique for evaluation of left ventricular relaxation and estimation of filling pressures. J Am Coll Cardiol. 1997;30(6):1527-33. doi: 10.1016/s0735-1097(97)00344-6, PMID 9362412.

44. Roberts KV, Brown AD, Maguire GP, Atkinson DN, Carapetis JR. Utility of auscultatory screening for detecting rheumatic heart disease in high-risk children in Australia's Northern Territory. Med J Aust. 2013;199(3):196-9. doi: 10.5694/mja13.10520, PMID 23909543.

45. Marijon E, Ou P, Celermajer DS, Ferreira B, Mocumbi AO, Jani D, et al. Prevalence of rheumatic heart disease detected by echocardiographic screening. N Engl J Med. 2007;357(5):470-6. doi: 10.1056/NEJMoa065085, PMID 17671255.

46. Terminology and Diagnostic Criteria Committee of The Japan Society of Ultrasonics in Medicine. Standard measurement of cardiac function indexes. J Med Ultrason (2001). 2006;33(2):123-7. doi: 10.1007/s10396-006-0100-4, PMID 27277734.

47. Rabah F, Al-Senaidi K, Beshlawi I, Alnair A, Abdelmogheth AAA. Echocardiography in PICU: when the heart sees what is invisible to the eye. J Pediatr (Rio J). 2016 Jan 1;92(1):96-100. doi: 10.1016/j.jped.2015.04.011, PMID 26569341.

48. PARKMYUNGKSALAMAT M. Park's pediatric cardiology for practitioners, 7E: South Asia Edition. S.l.: ELSEVIER INDIA; 2020.

49. Members C, COMMITTEE MEMBERS Search for more papers by this author, WILLIAMSJr JOHNF, JOHN F. WILLIAMSJr Search for more papers by this author, Bristow MR, MICHAEL R. BRISTOW Search for more

papers by this author, et al. Guidelines for the Evaluation and management of heart failure [Internet]. Circulation. 1995 [cited 2022Dec3]. Available from: https://www.ahajournals.org/doi/10.1161/01.CIR.92.9.2764.

# ANNEXURE I – CONSENT FORM

CONSENT FOR PARTICIPATION IN RESEARCH

IMPACT OF TRANSTHORACIC ECHOCARDIOGRAPHY ON DECISION MAKING IN CHILDREN PRESENTING WITH ACUTE RESPIRATORY DISTRESS AND HEART FAILURE - A ONE YEAR PROSPECTIVE OBSERVATIONAL STUDY AT KLE,S DR PRABHAKAR KORE HOSPITAL BELGAUM

Principal Investigator: **REG NO. BM0120010**

Co – investigator: DR. _____.

You have been asked to involve your child in the above said research to be conducted at PICU of KLE university's JN medical college hospital, Belagavi by Dr. KAJOL YADAV, PG student in the Department of Paediatrics at Jawaharlal Nehru Medical College, Belagavi.

Introduction

PURPOSE OF THE STUDY:

Participation of your child will help us to know the effects of ECHOCARDIOGRAPHY in diagnosis and decision making in PICU. You are free to discontinue the participation in the study at any time for any reasons and you will not be paid any reimbursement for participation in the research. Hence involving your child in the study is your voluntary decision.

Voluntary participation

Your child's participation in this study is your voluntary decision, whether or not to participate will not affect your current or future relationship with KLEs Dr. Prabhakar Kore Hospital & MRC, Belagavi.

Risk and benefits

There are no risks involved. Reduction in morbidity and mortality.

Privacy and Confidentiality

The only people who will know that you are a research participant are member of the research team. No information about you or provided by you, during research will be disclosed to others without your written consent. When the results of the research are published or discussed in the conferences, no information will be disclosed that would reveal your identity. Any information obtained in connections with this study and that can be identified with you remain confidential and will be disclosed only with your permission.

Queries

If you have any queries you may contact

**REG NO. BM0120010**

Post Graduate Student Department of Paediatrics JNMC, Belagavi-590010.

DR._____ ASSOCIATE PROFESSOR DEPARTMENT OF PAEDIATRICS, JNMC, Belagavi-590010.

If you have any questions about your rights or research participation you may contact Chairman ethical committee:

DR. ROOPA. M.BELLAD MD DCH PROFESSOR
DEPARTMENT OF PAEDIATRICS,
JAWAHARLAL NEHRU MEDICAL COLLEGE, BELGAVI-590010

You will be given a copy of this form for your information and to keep for your records.

STATEMENT OF CONSENT

I hereby voluntarily agree for my participation in this study. I understand that I have the liberty to withdraw at any time. My signature below indicates that I have read or have been told in the language I understand, about this entire consent form including the risks and benefits and have had all my questions answered. I will be given a copy of this consent form.

Signature of the authorized representative/ parent:

Date:

Name:

Relation to the Subject:

Signature of the witness:

Date:

Name:

Signature of investigator:

**PROFORMA**

Date:

Name:

ID NUMBER:

NAME:

MOTHER'S NAME:

FATHER'S NAME:

IP NUMBER:

DATE AND OF BIRTH: GENDER:
DATE OF ADMISSION:

CLINICAL EXAMINATION: FEVER
HR

RR
MAJOR CRITERIA:
ACUTE PULMONARY EDEMA CARDIOMEGALY
HEPATO-JUGLAR REFLEX NECK VEIN DISTENTION
PAROXYSMAL NOCTURNAL DYSPNEA ORTHOPNEA
RALES
THIRD HEART SOUND GALLOP
MINOR CRITERIA:
ANKLE EDEMA DYSPNEA ON EXERTION HEPATOMEGALY NOCTURNAL
COUGH PLEURAL EFFUSION TACHYCARDIA

INVESTIGATIONS:

CBC CRP UREA

CREATININE

X-RAY FINDINGS

DIAGNOSIS AT THE TIME OF ADMISSION

DIAGNOSIS AFTER ECHO

DECONGESTIVE THERAPY

DECONGESTIVE THERAPY

PLANNED BEFORE ECHO

PLANNED AFTER ECHO

IONOTROPIC THERAPY

IONOTROPIC THERAPY

PLANNED BEFORE ECHO

PLANNED AFTER ECHO

SURGICAL INDICATION IF ANY AFTER ECHO:

**NYHA and Modified Ross Heart Failure Classification**

| | NYHA | MODIFIED ROSS CLASSIFICATION |
|---|---|---|
| **Class I:** | no limitation of physical activity | Asymptomatic |
| **Class II:** | May experience fatigue, palpitations, dyspnea, or angina during moderate exercise but not during rest | In Infants- Mild tachypnea or diaphoresis with feeding<br>Older Children - Dyspnea on exertion |
| **Class III:** | Symptoms with minimal exertion that interfere with normal daily activity | In Infants- Marked tachypnea or diaphoresis with feeding and prolonged feeding times with growth failure. Older Children- marked dyspnea on exertion in older children |
| **Class-IV:** | Unable to carry out any physical activity because they typically have symptoms of HF at rest that worsen with any exertion | Tachypnea, retractions, grunting or diaphoresis at rest. |

# FRAMINGHAM 'S CRITERIA

**MAJOR CRITERIA:**

ACUTE PULMONARY EDEMA CARDIOMEGALY

HEPATO-JUGLAR REFLEX NECK VEIN DISTENTION

PAROXYSMAL NOCTURNAL DYSPNEA ORTHOPNEA

RALES

THIRD HEART SOUND GALLOP

**MINOR CRITERIA:**

ANKLE EDEMA DYSPNEA ON EXERTION HEPATOMEGALY NOCTURNAL

COUGH PLEURAL EFFUSION TACHYCARDIA

| SNO. | ID NUMBER | NAME | FATHER'S NAME | IP NUMBER | AGE | GENDER | CRITERIA | | INVESTIGATIONS | ECHO FINDINGS | | | | | DIAGNOSIS | | DECONGESTIVE THERAPY | | INOTROPIC THERAPY | | SURGICAL INDICATION AFTER ECHO | RESPIRATORY SUPPORT | | | OUTCOME | | IVIG | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | MAJOR | MINOR | | E/A RATIO | PAP (mmHg) | FS%- | EF% | IVC COLLAPSE ON RESPIRATION | BEFORE ECHO | AFTER ECHO | BEFORE ECHO | AFTER ECHO | BEFORE ECHO | AFTER ECHO | | O2 BY MASK | HFNC | VENTILATOR | DEATH | SURVIVED | GIVEN | NOT GIVEN |
| 1 | 1027971 | Vignesh Goankar | Mahesh Gaonkar | 1027971 | 2 months | M | 1 | 2 | Hb-12.3 TLC-24,700 PLATELETS-5.6, RBC-4.24 ,CRP-4.0, UREA-32, CREAT-0.43, X RAY- | E/A RATIO- 1.3:1 | 60 mm hg | 25% | 55% | NO | CHD | Very large PDA with Dilated LA,LV L to R shunt , Severe hyperkinetic PAH IN CCF | NONE | INJ LASIX, TAB ENVAS | NONE | INJ DOBUTAMINE, INJ MILRINONE, SYRUP DIXIN | PDA DEVISE CLOSURE | | YES | | SURVIVED | | |
| 2 | 1029075 | Vijayshree Hiremath | Veeresh Hiremath | 1029075 | 11 months | F | 3 | 2 | Hb-11.3 TLC-18500 PLATELETS-3.63 RBC-4.11 ,CRP-650, UREA-28, CREAT-0.39 X RAY | E/A RATIO- 2:1 | 40 mmhg | 26%, | 55% | NO | LRTI ?PNEUMONIA | HYPERTROPHIC CARDIOMYOPATHY WITH LRTI | NONE | SYRUP FUROPED,INJ LASIX,TAB ALDECTONE | NONE | NONE | NONE | YES | | | SURVIVED | | |
| 3 | 1030721 | Vedant Sangote | Shrishail Sangote | 1030721 | 2 months | M | 3 | 2 | Hb-12.9 TLC-10,900, PLATELETS-1.23, RBC-4.2, CRP-234.5, UREA-40, CREAT-0.48 X RAY | E/A RATIO- 1.3:1 | 58mm Hg | 29% | 62% | NO | CHD | Acynotic CHD with CCF with Severe PAH with Large PDA | NONE | IV LASIX INFUSION | NONE | INJ DIGOXIN,INJ DOBUTAMINE | ADVISED TO UNDERGO SURGICAL CORRECTION | | YES | | SURVIVED | | |
| 4 | 1030928 | Nishmita Diggavi | Vijyareddy | 1030928 | 1 year 3 months | F | 1 | 2 | Hb-11.0 TLC-20,700 PLATELETS-1.74 RBC-4.35 CRP-86 UREA-12 CREAT X RAY | E/A RATIO- 1.3:1 | 30 mm HG | 29% | 65% | NO | Pneumonia with impending respiratory failure with hypertension with Tachycardia | Pneumonia with impending respiratory failure with hypertension with Tachycardia with mild LV dysfunction with mild LVH | NONE | TAB ENVAS 2.5 MG 1/2 OD , INJ LASIX BD | NONE | NONE | NONE | | YES | | SURVIVED | | |
| 5 | 1035935 | Akshata Talwar | Irrapa | 1035935 | 12yrs | F | 2 | 2 | Hb-12.4 TLC-21.4 PLATELETS-4.19 RBC-4.24 CRP-13.6 UREA-11 CREAT-0.42 X RAY- Rt sided Consolidation | E/A RATIO- 1.3:1 | 30mm Hg | 21% | 44% | NO | Bronchochopneumonia | Covid myocarditis | Inj Lasix | To continue Inj Lasix | Inj Dobutamine | To Hike up Inj Dobutmine | NONE | | | | SURVIVED | | |
| 6 | 1038501 | Kavita Patil | Ramesh | 1038501 | 18 months | F | 1 | 1 | Hb-11.2 TLC-16600 PLATELETS-2.5 lac RBC-3.8 CRP-61.6 UREA-14 CREAT-0.33 X RAY- B/L Diffuse opacities | E/A RATIO- 1.3:1 | 28 mm Hg | 29% | 60% | NO | Viral Pneumonia | Viral Pneumonia | NONE | NONE | NONE | NONE | NONE | YES | | | SURVIVED | | |
| 7 | 1040599 | Mithun | Basawaraj | 1040599 | 4yrs | M | 2 | 2 | Hb-12.3 TLC-18,200 PLATELETS-2.4 LAC RBC-5.25 CRP-68.9 UREA-22 CREAT-0.4 X RAY- PATCH IN LFT LOWER LOBE | E/A RATIO- 1.4:1 | 29mm Hg | 29% | 60% | NO | Bronchopneumonia | Bronchopneumonia | NONE | NONE | NONE | NONE | NONE | YES | | | SURVIVED | | |
| 8 | 1041091 | Rihana | Aftab Mullah | 1041091 | 15 years | F | 1 | 2 | Hb-16.3 TLC-17200 PLATELETS-180000 RBC-5.25 CRP-11.1 UREA-23CREAT-0.6 X RAY- B/; DIFFUSE INFILTRATES | E/A RATIO- 1.4:1 | 27mm HG | 12% | 25% | NO | Dengue with Warning Signs | MISC-myocarditis | NONE | INJ LASIX, TAB ENVAS | NONE | IV DOBUTAMINE | NONE | | YES | | SURVIVED | yes | |
| 9 | 1045371 | Neha | prashant | 1045371 | 5yrs | F | 1 | 2 | Hb-11.4 TLC-17200 PLATELETS-2.8 RBC-5.25 CRP-21.1 UREA-23 CREAT-0.6 X RAY- B/; DIFFUSE INFILTRATES | E/A RATIO- 1.4:1 | 30 mm HG | 25% | 55% | NO | PNEUMONIA | AcyanoticCHD Moderate sized VSD,L->R Shunt, No PAH , In CCF | NONE | INJ LASIX, TAB ENVAS | NONE | NONE | VSD PATCH CLOSURE | YES | | | SURVIVED | | |
| 10 | 1047371 | Mallikarjun | Hanmant | 1047371 | 9 yrs | M | 2 | 1 | Hb-10.5TLC-8,490 PLATELETS-2.0 lac RBC-2.55 CRP-9 UREA-20 CREAT-0.8 X RAY- NORMAL | E/A RATIO- 1.4:1 | 62mmHg | 26% | 58% | NO | ? CHD | Post viral Myocarditis with HOCM in CCF With Severe PAH | Inj Lasix | To continue Inj Lasix , to strt tab Enalapril, tab Lasilactone | Inj Dobutamine | To continue ,Inj Dobutamine | NONE | YES | | | SURVIVED | | |
| 11 | 1048013 | Chandrashekar | Prakash | 1048013 | 5 months | M | 2 | 2 | Hb-12.4TLC-9,700 PLATELETS-4.11lac RBC-5.9 CRP-42 UREA-34 CREAT-0.6X RAY- RIGHT SIDED LOWER LOBE CONSOLIDATION | E/A RATIO- 1.5:1 | 58mm hg | 26% | 56% | NO | Bronchiolitis | Large ASD with L->R shunt with severe PAH | Inj Lasix | To continue Inj Lasix , to strt tab Envas, tab Aldactone | NONE | TO START INJ DOBUTAMINE AND INJ ADRENALINE | SURGICAL REPAIR AFTER STABILISATION(ASD PATCH CLOSURE) | YES | | | SURVIVED | | |
| 12 | 1050189 | Preetam | Umesh | 1050189 | 7 years | M | 1 | 5 | Hb-15.4 TLC-11.8 PLATELETS-8000 RBC-5.46 CRP-6.4 UREA-27 CREAT-0.55 X RAY-B/L PLEURAL EFFUSION ARDS | E/A RATIO- 1.3:1 | 30mm Hg | 30% | 60% | NO | DENGUE SHOCK SYNDROME | DENGUE SHOCK SYNDROME | INJ LASIX | INJ LASIX INFUSION | INJ ADRENALINE,INJ DOBUTAMINE | INJ DOBUTAMINE STOPPED | NONE | YES | | | SURVIVED | | |
| 13 | 1050409 | B/O Alina | Afzal | 1050409 | 3 months | M | 3 | 2 | Hb-9.9 TLC-14,300 PLATELETS-3.06 lac RBC-4.3 CRP-60 UREA-16 CREAT-0.21 X RAY-LEFT SIDED CONSOLIDATION | E/A RATIO- 1.3:1 | 60mm hg | 26% | 55% | NO | CHD with LRTI | Large perimembranous VSD with L->R shunt with S. PAH | INJ LASIX | TO CONTINUE INJ LASIX, DIXIN DROPS, TAB ALDACTONE | NONE | NONE | SURGICAL REPAIR AFTER STABILISATION(VSD PATCH CLOSURE) | | YES | | SURVIVED | | |
| 14 | 1050527 | Farhan | Roshan | 1050527 | 13 years | M | 2 | 4 | Hb-13.2 TLC-3600 PLATELETS-18000 RBC-5.24 CRP-13.6 UREA-14 CREAT-0.44 X RAY- B/L PLEURAL EFFUSION | E/A RATIO- 1.4:1 | 29mm Hg | 12% | 25% | YES | DENGUE with warning signs | Dengue Shock Syndrome with Dengue myoarditis with B/L Pleural effusion | INJ LASIX | INJ LASIX INFUSION ,TAB DIGOXIN , TAB ENVAS 2.5 MG OD | INJ ADRENALINE | INJ DOBUTAMINE, INJ ADRENALINE | NONE | | | YES | DEATH | NO | | NOT GIVEN |
| 15 | 1050973 | Ankita | Ashok | 1050973 | 4 yrs | F | 1 | 3 | Hb-14.8 TLC-5,100 PLATELETS-2.3 lac RBC-5.55 CRP-6.4 UREA-28 CREAT-0.3 X RAY-RT SIDED PLEURAL EFFUSION | E/A RATIO- 1.5:1 | 30mm Hg | 20% | 40% | YES | Dengue fever With Warning signs | Dengue myocarditis with warning signs | NONE | NONE | NONE | IV DOBUTAMINE | NONE | | YES | | SURVIVED | YES | |
| 16 | 1051235 | Prisha | Anand | 1051235 | 2 months | F | 2 | 3 | Hb-9.4 TLC-8400 PLATELETS-3.37 RBC-3.13 CRP-1.2 UREA-39 CREAT-0.45 X RAY- RT Lung consolidation COVID 19 RTPCR- NEGATIVE | E/A RATIO- 1.3:1 | 62mmHg | 25% | 50% | NO | Bronchopneumonia | Large PDA R to L shunt,Severe Suprasystemic PA pressure in CCF | INJ LASIX | INJ LASIX, TAB ENVAS, SYRUP PULMOCEL, TAB BOSENTAN | NONE | NONE | PDA Ligation | | YES | | SURVIVED | | |
| 17 | 1052963 | Maheboob | Hasan | 1052963 | 2.5 months | M | 2 | 3 | Hb-10 TLC-18400 PLATELETS-3.2 RBC-3.13 CRP-20 UREA-29 CREAT-0.44 X RAY- RT Lung consolidation COVID 19 RTPCR- NEGATIVE | E/A RATIO- 1.5:1 | 60mm hg | 24% | 50% | NO | Bronchopneumonia | Large VSD R to L shunt , L to R shunt,Severe Suprasystemic PA pressure in CCF | INJ LASIX | INJ LASIX, TAB ENVAS, TAB BOSENTAN | NONE | INJ DOBUTAMINE | VSD Patch closure | | YES | | SURVIVED | | |
| 18 | 1115507 | Gurubai | Ramchandra | 1115507 | 4 years | F | 3 | 3 | Hb-12.8TLC-16800PLATELETS-3.62 RBC-4.8 CRP-200 UREA-32 CREAT-0.4 X RAY- B/L PLEURAL EFFUSION | E/A RATIO- 1.3:1 | 60mm hg | 25% | 52% | NO | Bronchopneumonia aith CHD | Large VSD L -> R shunt with S. PAH in CCF | INJ LASIX | TO CONTINUE INJ LASIX,TO START TAB DIGOXIN, TAB ENVAS | NONE | INJ DOBUTAMINE | SURGICAL REPAIR AFTER STABILISATION(VSD PATCH CLOSURE) | | YES | | SURVIVED | | |
| 19 | 1055084 | Trishala | Gopinath | 1055084 | 16 years | F | 1 | 3 | Hb-11.0 TLC-4000 PLATELETS-44,000 RBC-4.2 CRP-2 UREA-32 CREAT-0.6 X RAY-RIGHT SIDED PLEURAL EFFUSION | E/A RATIO- 1.3:1 | 28mm hg | 30% | 60% | YES | Dengue Shock Syndrome with LRTI | Dengue Shock Syndrome with LRTI | INJ LASIX | INJ LASIX | INJ DOBUTAMINE, INJ ADRENALINE | INJ DOBUTAMINE, INJ ADRENALINE | NONE | YES | | | SURVIVED | | |
| 20 | 1055109 | Saksham | Shashikant | 1055109 | 2 months 15 days | M | | | Hb-13 TLC-9,400 PLATELETS2.89 LAC RBC- 5.45 CRP-42 UREA-39 CREAT-0.3 X RAY-PATCH IN LEFT LOWER LOBE | E/A RATIO- 1.4:1 | 25mm hg | 29% | 60% | NO | Bronchopneumonia | CHD -LARGE PDA L --> R shunt with no PAH in CCF | NONE | INJ LASIX | NONE | INJ DOBUTAMINE | PDA DEVICE CLOSURE | | YES | | SURVIVED | | |
| 21 | 1055656 | Vihaan | Jagadish | 1055656 | 3 years 6 months | M | 2 | 5 | Hb-7.7 TLC-29400 PLATELETS-6.24 RBC-3.43 CRP-99 UREA-20 CREAT-0.36 X RAY- B/L PLEURAL EFFUSION | E/A RATIO- 1.3:1 | 28mm hg | 13% | 25% | NO | ? Viral Pneumonia with ?CHD IN CCF | ? Viral Myocarditis IN CCF with severe LV systolic dysfnction with moderate MR, TR with Pericardial effusion with B/L pleural effusion | INJ LASIX TID | INJ LASIX INFUSION , TAB ENVAS 2.5 MG OD | INJ DOBUTAMINE | INJ ADRENALINE INFUSION AND INJECTION DOBUTAMINE | NO | | | | SURVIVED | YES | |
| 22 | 1055202 | Adarsh | Shivaji | 1055202 | 6 years | M | 2 | 4 | Hb-13.9 TLC-2.9 PLATELETS-2200 RBC-5.14 CRP-94 UREA-18 CREAT-0.32,COVID IGG IGM-POSITIVE, X RAY- B/L PLEURAL EFFUSION | E/A RATIO- 1.3:1 | 28mm hg | 22% | 45% | YES | Dengue Shock Syndrome in critical phase | Dengue Shock Syndrome with mild LV dysfunction ? MISC Myocarditis in Critical phase | NONE | IV LASIX INFUSION,ORAL DIGOXIN , TAB ENVAS | INJ ADRENALINE | Continue INJ ADRENALINE INFUSION AND INJECTION DOBUTAMINE | NONE | | | | SURVIVED | YES | |
| 23 | 1056803 | Midhat | Mohammad Mustak | 1056803 | 2 months | F | 2 | 1 | Hb-15 TLC-26000 PLATELETS-2.4 RBC-4.5 CRP-0.3 UREA-15 CREAT-0.4 X RAY- Cardiomegaly | E/A RATIO- 1.3:1 | 28mm hg | 29% | 60% | NO | ? CHD WITH Bronchopneumonia | CHD With Moderate sized muscular VSD with Small sized ASD in CCF with Pneumonia | NONE | DIXIN DROPS 0.3 ML OD, FUROPED DROPS 0.3 ML BD | NONE | NONE | NONE | YES | | | SURVIVED | | |
| 24 | 1116959 | Rajeev Kumar | Karan | 1116959 | 11yrs | M | 3 | 3 | Hb-13.0 TLC-3600 PLATELETS-23,000 RBC-5.97 CRP-10.5 UREA-23 CREAT-0.4 ,X RAY-Normal | E/A RATIO- 1.4:1 | 28mm hg | 19% | 40% | NO | Bronchopneumonia | Acyanotic CHD ASD (L-->R) Shunt with np PAH | NONE | SRYP FUROPED | NONE | NONE | NONE | YES | | | SURVIVED | | |
| 25 | 1058529 | Sanchit | Parshram | 1058529 | 7 years | M | 1 | 4 | Hb-11.1 TLC-4200 PLATELETS-1.41 RBC-424 CRP-197.4 UREA-31 CREAT-0.4 COVID IGG IGM POSITIVE ,X RAY-B/L Pleural effusion | E/A RATIO- 0.9:1 | 30mm Hg | 19% | 40% | NO | ? Dengue with warning signs | MIS.C Myocarditis with LV dysfunction with AR | NONE | INJ LASIX | INJ DOBUTAMINE | INJ DOBUTAMINE | NONE | | | | SURVIVED | YES | |
| 26 | 1060088 | Amruta | Mahantesh | 1060088 | 9 years | F | 2 | 3 | Hb-14 TLC-3000, PLATELETS-9000, RBC-4.85 CRP-1 UREA-21 CREAT-0.53 X RAY-R>L Pleural effusion | E/A RATIO- 1.3:1 | 30mm Hg | 28% | 60% | NO | Dengue fever with warning signs with R sided pleural effusion | Dengue fever with Warning Signs with Rt sided Pleural effusion | NONE | INJ LASIX INFUSION | INJ ADRENALINE,INJ DOBUTAMINE | To Continue INJ ADRENALINE,INJ DOBUTAMINE | NONE | | YES | | SURVIVED | | |
| 27 | 1057574 | Rihan | Maula | 1057574 | 16 years | M | 1 | 2 | Hb-16.3 TLC-17200 PLATELETS-23000 RBC-5.93 CRP-11.1 UREA-23 CREAT-0.61 X RAY- NORMAL | E/A RATIO- 1.3:1 | 28mm hg | 12% | 25% | NO | SEVERE DENGUE With Warning Signs | MIS.C Myocarditis | NONE | INJ LASIX , TAB ENVAS | INJ ADRENALINE | INJ ADRENALINE,INJ DOBUTAMINE | NONE | | YES | | SURVIVED | YES | |
| 28 | 1060414 | Sujay | Shrishail | 1060414 | 11 years | M | 2 | 4 | Hb-14.9 TLC-3000 PLATELETS-12,000 RBC-5.49 CRP-10.9 UREA-10 CREAT-0.60 X RAY-R > L Pleural effusion | E/A RATIO- 1.1:1 | 29mm Hg | 29% | 60% | YES | Dengue fever with warning signs with B/L pleural effusion | Dengue Shock Syndrome with B/L Pleural effusion pneumonia | NONE | INJ LASIX | INJ ADRENALINE ,INJ DOBUTAMINE | INJ ADRENALINE ,INJ DOBUTAMINE | NONE | YES | | | SURVIVED | | |
| 29 | 1060569 | Sadanand | Shivraj | 1060569 | 5 years 6months | M | 1 | 4 | Hb-17.6 TLC-8100 PLATELETS-18000 RBC-8.25 CRP-0.9 UREA-17 CREAT-0.2 COVID IGG IGM POSITIVE X RAY- RT Pleural effusion | E/A RATIO- 1.1:1 | 30mm Hg | 18% | 40% | NO | Dengue fever with warning signs with Rt sided Pleural effusion | Dengue Myocarditis with MIS.C with B/L Pleural effusion | NONE | INJ LASIX , TAB ENVAS, TAB ALDACTONE | INJ ADRENALINE | To Continue with INJ ADRENALINE , To Start with INJ DOBUTAMINE | NONE | | | | SURVIVED | YES | |
| 30 | 1059470 | Aadarsh Shinde | Deepak | 1059470 | 1month,15 Days | M | 2 | 2 | Hb-16.1 TLC-9000 PLATELETS-4.03 lac RBC-6.46 CRP-40 UREA-29 CREAT-0.76 X-ray-normal | E/A RATIO- 1.3:1 | 60mm hg | 26% | 55% | NO | CHD With LRTI | CO-Arctation OF Aorta with S.PAH With LRTI | Inj Lasix | To continue Inj Lasix , Syp Dixin , Syp Furoped | NONE | Inj Dobutamine | BALOON DILATATION | | YES | | SURVIVED | | |
| 31 | 1061062 | Shreyas | Arjun | 1061062 | 6 months | M | 1 | 3 | Hb-9.7 TLC-15340 PLATELETS-5.18 lac RBC-3.68 CRP-19 UREA-29 CREAT-0.9 X-ray-diffuse Opacities | E/A RATIO- 1.2:1 | 30mm Hg | 29% | 60% | NO | Bronchiolitis | Bronchiolitis | Inj Lasix | NONE | NONE | NONE | NONE | YES | | | SURVIVED | | |
| 32 | 1061601 | Shankar | Shiddappa | 1061601 | 12yrs | M | 2 | 3 | Hb-16.8 TLC-16.8 PLATELETS-2.32 lac RBC-6.0 CRP-487.5 UREA-75 CREAT-0.8 X-ray-RT LOWERLOBE CONSOLIDATION. | E/A RATIO- 1.3:1 | 25mm hg | 17% | 35% | NO | Bronchopneumonia | RHD with MR in sinus rhythm with Bronchopneumonia | Inj Lasix | To start with Lasix Infusion | Inj Adrenaline | Hike up Inj Adrenaline To start with Inj Dobutamine, Dopamine | NONE | YES | | | SURVIVED | | |
| 33 | 1062761 | B/o Shambal | Vittal | 1062761 | 3 months | M | 2 | 2 | Hb-12.9 TLC-18500 PLATELETS-5.28 RBC-4.7 CRP-90 UREA-9 CREAT-0.23 X RAY-inc Bronchovascular markings | E/A RATIO- 1.3:1 | 25mm hg | 28% | 60% | NO | Bronchiolitis | Acyanotic CHD , Small Fossa Ovalis ASD with Bronchiolitis | NONE | Furoped drops | NONE | NONE | NONE | | YES | | SURVIVED | | |
| 34 | 1066458 | Nidha | Ismail | 1066458 | 11 years | F | 1 | 2 | Hb-10.9 TLC-28600 PLATELETS-734 RBC-3.84 CRP-5.9 UREA-21 CREAT-0.39 COVID IGG-NEGATIVE COVID IGM-POSITIVE X RAY- | E/A RATIO- 1.1:1 | 25mm hg | 28% | 62% | NO | VIRAL PNEUMONIA | VIRAL PNEUMONIA | NONE | INJ LASIX | NONE | NONE | NONE | YES | | | SURVIVED | | |
| 35 | 1066488 | Kansa | Irshad | 1066488 | 4 year | F | 1 | 3 | Hb-11.6 TLC-12.7 PLATELETS-56000 RBC-5.12 CRP-14.6 UREA-52 CREAT-0.47 X RAY- S/C Emphysema B/L Pleural effusion | E/A RATIO- 1.3:1 | 25mm hg | 15% | 28% | YES | Dengue Shock Syndrome with DIC with MIS.C | Dengue Shock Syndrome with DIC with Dengue Myocarditis with MIS.C | INJ LASIX BD | INJ LASIX INFUSION | INJ ADRENALINE ,INJ NORADRENALINE | To continue INJ ADRENALINE ,INJ NORADRENALINE AND START INJ DOBUTAMINE | NONE | | YES | DEATH | | YES | |
| 36 | 1067986 | Harsha | Gangappa | 1067986 | 1.5 year | F | 2 | 4 | Hb-8.6 TLC-15400 PLATELETS-3.85 RBC-3.95 CRP-48.5 UREA-25 CREAT-0.3 X RAY- S/O Bronchopneumonia and L sided Pleural effusion | E/A RATIO- 1.3:1 | 25mm hg | 28% | 62% | NO | Bronchopneumonia with Synpneumonic effusion with Empyema | Bronchopneumonia with Synpneumonic effusion with Empyema | NONE | NONE | NONE | NONE | NONE | | YES | | SURVIVED | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 37 | 1068166 | Shifa Sanadi | Kousar Haiderali | 10868166 | 3.5 years | F | 1 | 2 | Hb-10.1 TLC-10600 PLATELETS-2.81 RBC-4.67 CRP-387 UREA-25 CREAT-0.5 COVID IGM POSITIVE COVID IGG NEGATIVE X RAY-B/L BRONCHOPNEUMONIA | E/A RATIO- 1.3:1 | 26mm hg | 26% | 56% | NO | Dengue Shock Syndrome with Bronchopneumonia with culture proven UTI with Moderate Anemia with Moderate Acute Malnutrition | DSS with Bronchopneumonia with culture proven UTI with Moderate Anemia with Moderate Acute Malnutrition | NONE | NONE | INJ ADRENALINE | INJ ADRENALINE | NONE | | YES | | SURVIVED | | |
| 38 | 1072258 | Veeraj | Anand | 1072258 | 2 months | M | 1 | 2 | Hb-14 TLC-8600 PLATELETS-4.5 RBC-4.5 CRP-6 UREA-12 CREAT-0.2 X RAY- Bronchoilitis | E/A RATIO- 1.2:1 | 25mm hg | 28% | 60% | NO | Bronchiolitis with AOM | Bronchiolitis with AOM | NONE | NONE | NONE | NONE | NONE | YES | | | SURVIVED | | |
| 39 | 1072254 | Prabha | Chandrakant | 1072254 | 13 years | F | 2 | 5 | Hb-12 TLC-10700 PLATELETS-1.97 RBC-5.97 CRP-3 UREA-50 CREAT-0.67 X RAY-Mild Pleural effusion on R side | E/A RATIO- 2:1 | 30mm Hg | 23% | 50% | NO | CHD | Restrictive Cardiomyopathy | INJ LASIX | IV LASIX INFUSION,ORAL DIGOXIN , TAB ENVAS TAB ALDACTONE | NONE | INJ DOBUTAMNE | NONE | | YES | | SURVIVED | | |
| 40 | 1072444 | Rachita | Ramappa | 1072444 | 6 months | F | 2 | 2 | Hb-9.8 TLC-13400 PLATELETS-2.84 RBC-4.10 CRP-20 UREA-36 CREAT-0.45 X RAY-CARDIOMEGALY, Pulm. Plethora + | E/A RATIO- 1.2:1 | 25mm hg | 27% | 60% | NO | Bronchopneumonia | Bronchopneumonia | NONE | NONE | NONE | NONE | NONE | YES | | | SURVIVED | | |
| 41 | 1072843 | Shivaratna | Sadashiv | 1072843 | 2.5 years | M | 1 | 3 | Hb-8.8 TLC-11800 PLATELETS-250 RBC-5.65 CRP-0.8 UREA-30 CREAT-0.45 X RAY-Bronchopneumonia | E/A RATIO- 1.3:1 | 25mm hg | 14% | 30% | YES | Pneumonia with CCF | Acyanotic CHD (Large Perimembranous VSD L to R) with ASD L to R in CCF with Bronchopneumonia | NONE | DIXIN-P DROPS,TAB ENVAS, TAB DIGOXIN, INJ LASIX | INJ DOBUTAMINE | TO Continue With INJ DOBUTAMINE To start With INJ ADRENALINE | PLANNED VSD PATCH CLOSURE AFTER CORRECTION AND PNEUMONIA | | YES | | SURVIVED | | |
| 42 | 1078595 | Santosh | Basavani | 1078595 | 10 years | M | 3 | 2 | Hb-11.9 TLC-8600 PLATELETS-3.82 RBC-4.43 CRP-2.1 UREA-23 CREAT-0.52 X RAY- NORMAL | E/A RATIO- 1.3:42 | 25mm hg | 28% | 60% | NO | CHD With CCF | RHD with severe AR with Carditis in sinus rhythm with Bronchopneumonia | NONE | TAB ENVAS 2.5 MG 1/2 OD ,TAB DIGOXIN,TAB LACILACTONE | INJ DOBUTAMINE | INJ DOBUTAMINE | NONE | YES | | | SURVIVED | | |
| 43 | 1085012 | Akash | Durgappa | 1085012 | 12 years | M | 2 | 3 | Hb-13.8 TLC-12300 PLATELETS-49000 RBC-4.93 CRP-0.4 UREA-30 CREAT-0.8,COVID IGG IGM - NEGATIVE X RAY- | E/A RATIO- 1.3:1 | 28mm hg | 16% | 35% | DILATED | Severe Dengue with Dengue Shock Syndrome with ?Dengue Hepatitis | Dengue Shock Syndrome with Dengue Myocarditis with Dengue Hepatitis in shock | INJ LASIX INFUSION | INJ LASIX INFUSION | INJ DOBUTAMINE ,INJ ADRENALINE | INJ DOBUTAMINE | NONE | | YES | | SURVIVED | YES | |
| 44 | 1088419 | Dhanush | Sadanand | 1088419 | 5 months | M | 2 | 2 | Hb-13.6 TLC-17,000 PLATELETS-2.6 Lac RBC-5.4 CRP-40 UREA-23 CREAT-0.5 X RAY-b/l Patchy Consolidation | E/A RATIO- 1.3:1 | 58mm hg | 28% | 60% | NO | Viral (bronchopulmonary) Pneumonia | Acyanotic CHD (Large Perimembranous VSD L to R) with moderate apical muscular VSD with S.PAHwith Bronchopulmonary Pneumonia | NONE | INJ LASIX IV BD, SYRUP DIXIN PEDS, TAB ALDACTONE | NONE | NONE | VSD CORRECTION TO BE DONE AFTER CLINICAL STABILISATION | | YES | | SURVIVED | | |
| 45 | 1085852 | Azamat | Rihan | 1085852 | 1 year | M | 3 | 4 | Hb-7.8 TLC-20500 PLATELETS-7.86 RBC-4.41 CRP-269.7 UREA-10 CREAT-0-.22 X RAY-Massive L sided Pleuraleffusion , R sided Pneumonia | E/A RATIO- 1.1:2 | 28mm hg | 28% | 60% | NO | Bacterial Pneumonia with massive L pleural effusion with Respiratory Distress (synpneumonic effusion) | Bacterial Pneumonia with massive L pleural effusion with Respiratory Distress (synpneumonic effusion) | NONE | NONE | NONE | NONE | ICD insertion | | YES | | SURVIVED | | |
| 46 | 1086076 | Manveet | santosh | 1086076 | 10 months | M | 1 | 3 | Hb-8.0 TLC-17,600 PLATELETS-2.4 lac RBC-2.4 CRP-80 UREA-26 CREAT-0.44 X RAY-RT sided Patchy Consolidation of rt lower lobe . | E/A RATIO- 1.1:1 | 26mm hg | 29% | 60% | NO | Rt sided Bronchopneumonia ? Aspiration | Rt sided Bronchopneumonia ? Aspiration | NONE | NONE | NONE | NONE | NONE | | YES | | SURVIVED | | |
| 47 | 1088595 | Khushi | Bhimappa | 1088595 | 2 year 5 months | F | 1 | 3 | Hb-7.4 TLC-12500 PLATELETS-3.77 RBC-5.54 CRP-4.1 UREA-24 CREAT-0.27 X RAY-patchy consolidation in perihilar region | E/A RATIO- 1.3:1 | 26mm hg | 28% | 60% | NO | ? Viral pneumonia WITH bronchpneumonia | ? Viral pneumonia WITH bronchpneumonia | NONE | NONE | NONE | NONE | NONE | YES | | | SURVIVED | | |
| 48 | 1089777 | Mohd Nadaf | Rehman | 1089777 | 3 years | M | 1 | 3 | Hb-9.5 TLC-15820 PLATELETS-7.41 RBC-4.78 CRP-2.1 UREA-42 CREAT-0.26 X RAY- R sided Consolidation | E/A RATIO- 1.1:1 | 48mm hg | 28% | 65% | DILATED | Bronchopneumonia with SAM | Cor Pulmonale (Rt Heart Failure) due to Pulmonary Hypertension secondary to Bronchitic syndrome | NONE | INJ LASIX ,DIXIN PEDS SUSPENSION | NONE | NONE | NONE | | YES | | SURVIVED | | |
| 49 | 1089800 | B/O Kaveri Kalal | Umesh Kalal | 1089800 | 2 months | M | 1 | 2 | Hb-9.5 TLC-18200 PLATELETS-366 RBC-3.19 CRP-positive (42.0) UREA-23 CREAT-0.34 X RAY- patch on right side | E/A RATIO- 1.3:1 | 55mm hg | 26% | 56% | NO | Bronchopneumonia | Acyanotic CHD , co-arctation of aorta with moderate size PDA with ASD | INJ LASIX | INJ LASIX | NONE | INJ DOBUTAMINE | AORTIC BALOON VALVOTOMY | | YES | | SURVIVED | | |
| 50 | 1090414 | Krutika | Siddappa | 1090414 | 15 years | F | 1 | 4 | Hb-11 TLC-15200 PLATELETS-1.94 RBC-5.84 CRP-2.8 UREA-53 CREAT-0.75 X RAY- R sided collapse and consolidation | E/A RATIO- 1.3:1 | 30mm Hg | 29% | 60% | NO | Pneumonia with R sided collpase and consolidation with Respirstory acidosis | Pneumonia with R sided collpase and consolidation with Respirstory acidosis | NONE | NONE | NONE | NONE | NONE | YES | | | SURVIVED | | |
| 51 | 1091031 | Puneet Raj | Annappa | 1091031 | 4 months | M | 1 | 3 | Hb-9.8 TLC-13000 PLATELETS-654 RBC-3.6 CRP-positive (61.0) UREA-22 CREAT-0.23 X RAY- R sided pleural effusion present | E/A RATIO- 1.3:1 | 28mm hg | 28% | 60% | NO | Bronchopneumonia with CHD | Acyanotic CHD , large perimembranous VSD with L -> R shunt with PAH with LRTI ( Bronchopneumonia) | NONE | Inj. Lasix, Tab. Aldactone, Syp. Dixin Peds | NONE | Inj Dobutamine | VSD PATCH CLOSURE | | YES | | SURVIVED | | |
| 52 | 1120246 | Anjna | Siddappa | 1120246 | 2 months | F | 1 | 2 | Hb- 12.0 TLC-11,700 PLATELETS-3.87 LAC RBC-4.45 CRP-40 UREA-18 CREAT-0.7 X RAY-patch in LEFT Upper lobe | E/A RATIO- 1.3:1 | 47mm Hg | 29% | 60% | NO | Bronchopneumonia | Acyanotic CHD (Large ASD) with L-->R With PAH | NONE | Dixin peds drops , Furoped drops | NONE | NONE | NONE | YES | | | SURVIVED | | |
| 53 | 1092130 | Prateek | Arunkumar | 1092130 | 5 years | M | 2 | 2 | Hb- 11.8 TLC-9400 PLATELETS-267 RBC-5.04 CRP-4.6 UREA-52 CREAT-0.48 X RAY-patch in right middle lobe | E/A RATIO- 1.3:1 | 47mm Hg | 25% | 52% | NO | CHD | Acyanotic CHD , Moderate sized perimembranous VSD ( L-> R shunt) with mild LV Dysfunction with LRTI | NONE | Inj. Lasix, Tab. Aldactone, Syp. Dixin Peds | NONE | Inj. Dobutamine | Surgical Patch for VSD Closure | YES | | | SURVIVED | | |
| 54 | 1092512 | Samiksha | Shanmukappa | 1092512 | 2yr,6 months | F | 1 | 4 | Hb- 10.1 TLC- 8,000 PLATELETS-1.12 RBC-3.3 CRP-557UREA-22 CREAT-0.19 X RAY-B/L Pleural Effusion | E/A RATIO- 1.2:1 | 25mm hg | 29% | 62% | NO | Pneumonia with P.effusion | Pneumonia | Inj Lasix | Inj Lasix | NONE | NONE | NONE | YES | | | SURVIVED | | |
| 55 | 1106024 | Preeti Khol | Shahu Khol | 1106024 | 3 Years | F | 1 | 2 | Hb- 11.1 TLC- 13,000 PLATELETS-381 RBC-5.32 CRP-11 UREA-22 CREAT-0.3 X RAY-normal | E/A RATIO- 1.3:1 | 45mm hg | 18% | 40% | NO | Congenital heart disease | Acyanotic CHD ,large PDA,(L->R) with Normal Ventricular Function | Inj. Lasix | TO CONTINUE INJ LASIX ,TO START DIXIN PEDS SUSPENSION | NONE | NONE | PDA Closure | YES | | | DEATH | | |
| 56 | 1106584 | Gudumabi | Maiboob | 1106584 | 9 months | F | 2 | 2 | Hb- 9.5 TLC- 15850 PLATELETS-286 RBC- 3.8 CRP-10 UREA-50 CREAT-0.2 X RAY-cardiomegaly present | E/A RATIO- 1.3:1 | 26mm hg | 10% | 17% | NO | ? Bronchopneumonia ? CHD | Post Viral Myocarditis with severe LV Dysfunction with DCMP in CHF | Inj Lasix | To continue Inj Lasix , TO Start Oral Digoxin , Tab. Aldoctone, Tab Envas | Inj. Dobutamine | To Continue Inj. Dobutamine | None | | YES | | SURVIVED | YES | |
| 57 | 1095625 | B/O Harsha | Nikkhil Pawar | 1095625 | 2 months | M | 2 | 2 | Hb- 12.5 TLC-14700 PLATELETS- 396 RBC-3.63 CRP-18.1 UREA- 23 CREAT- 0.32 X RAY- | E/A RATIO- 1.3:1 | 72mmHg | 30% | 62% | YES | ? CHD | Acyanotic CHD ( large perimembranous VSD),L->R shunt with severe PAH in CCF in Sinus Rhythm | Inj. Lasix | INJ LASIX ,DIXIN PEDS , Tab. Envas | NONE | Inj Dobutamine | VSD Closure | YES | | | SURVIVED | | |
| 58 | 1101094 | Halappa | Vittal | 1101094 | 14yrs | M | 2 | 2 | Hb- 13.2 TLC-4,100 PLATELETS- 5000 RBC-5.09 CRP-4.2 UREA- 21 CREAT- 0.63, covid igM positive X RAY-B/L MILD PLEURAL | E/A RATIO- 1.3:1 | 32mm hg | 14% | 29% | NO | Dengue Shock Syndrome | Dengue Myocarditis With Severe LV Dysfuction | Inj Lasix | Inj Lasix | Inj Adrenaline , Inj Dobutamine | To Continue Inj Adrenaline , Inj Dobutamine | NONE | | YES | | SURVIVED | YES | |
| 59 | 1102830 | Ansh | Krishna | 1102830 | 9 yr | M | 1 | 3 | Hb- 9.6 TLC- 18,000 PLATELETS- 1.93 RBC-3.32 CRP-82 UREA- 14 CREAT- 0.35, X RAY-Patchy Consolidation In LFT Lower Lobe | E/A RATIO- 1.3:1 | 32mm hg | 29% | 65% | NO | Bronchopneumonia With Pleuritis | Bronchopneumonia With Pleuritis | NONE | NONE | NONE | NONE | NONE | YES | | | SURVIVED | | |
| 60 | 1104207 | Saanvi | Nandishwar | 1104207 | 13 yrs | F | 1 | 2 | Hb- 13.5 TLC- 18,200 PLATELETS-268 RBC-4.2 CRP-72 UREA-11 CREAT-0.5 X RAY-B/L Patchy Infiltrates | E/A RATIO- 1.3:1 | 28mm hg | 22% | 45% | NO | Dengue Shock Syndrome? Mis.C | Mis- C myocarditis | Inj Lasix | To continue Inj Lasix ,To Start Oral Digoxin , Tab. Aldoctone, Tab Envas | Inj. Dopamine | To Continue With Inj Dopamine, to startInj. Dobutamine | None | | YES | | SURVIVED | YES | |
| 61 | 1104417 | Suraavi | Suraj | 1104417 | 6 yrs | F | 1 | 2 | Hb-10.6 TLC-7800 PLATELETS-168 RBC-4.13 CRP-340 UREA-39 CREAT-0.87 X RAY- NORMAL | E/A RATIO- 1.2:1 | 28mm hg | 20% | 42% | NO | ? CHD with Cardiogenic Shock | Mis -C myocarditis with cardiogenic shock | Inj. Lasix | Inj Lasix | inj Dobutamine | inj dobutamine | None | | YES | | SURVIVED | YES | |
| 62 | 1104914 | Laxmi | Ganpati | 1104914 | 15 yrs | F | 2 | 2 | Hb-13.1 TLC- 11,600 PLATELETS- 348 RBC-4.9 CRP-negative UREA-22 CREAT-0.46 X RAY-NORMAL | E/A RATIO- 1.2:1 | 25mm hg | FS%- | 60% | NO | CHD | RHD with MR with no signs of CCF without Infective Endocarditis | none | Inj Lasix | none | none | Mitral valve replacement | YES | | | SURVIVED | | |
| 63 | 1105637 | Sinchna | Nagraj | 1105637 | 1.5 months | F | 2 | 2 | Hb- 11.0 TLC-17,900 PLATELETS-1.22lac RBC-3.53 CRP-11.1 UREA-12 CREAT-0.42 X RAY-Normal | E/A RATIO- 1.1:1 | 26mm hg | 12% | 20% | DILATED | ?Dengue Shock Syndrome ?Mis- C | Mis- C myocarditis with DIC with severe LV Dysfunction with mild MR | Inj Lasix | Continue Inj. Lasix , to add Digoxin, Tab Envas | Inj Dobutamine,inj Dopamine | Inj Dobutamine,inj Dopamine,to add IV Calcium Gluconate | none | | YES | | SURVIVED | YES | |
| 64 | 1107107 | Suprita | Sidram | 1107107 | 11 months | F | 2 | 2 | Hb- 10.2 TLC- 18,600 PLATELETS-286 RBC- 3.2 CRP-Positive 90 UREA- 46 CREAT-0.42 X RAY- | E/A RATIO- 1.3:1 | 40mmhg | 22% | 48% | NO | CHD with FTT | Acyanotic CHD with severe Juxta ductal co-actation of aorta and severe valvular AS with mild PAH not in CCF with LRTI with FTT. | none | Inj Lasix | none | none | Balloon dilation of COA and Aortic stenosis after LRTI subsides | | YES | | SURVIVED | | |
| 65 | 1106553 | Sachin Khaddi | Nagappa | 1106553 | 5 yrs | M | 2 | 1 | Hb- 12.0 TLC-16,700 PLATELETS-360 RBC-4.0 CRP-POSITIVE 46 UREA-42 CREAT-0.38 X RAY- | E/A RATIO- 1.2:1 | 25mm hg | 25% | 55% | NO | LRTI (Bronchopneumonia) | Acyanotic CHD with large fossa ovalis without PAH without CCF | none | Syp. Dixin peds, Syp. Furoped, Tab. Aldactone 25 mg 1/2 x OD | NONE | NONE | ASD device closure , device size 22 | YES | | | SURVIVED | | |
| 66 | 1106485 | Rachita | Sunil | 1106485 | 2 Yrs | F | 2 | 1 | Hb-6.6 TLC-12,000 PLATELETS- 2.8LAC RBC-2.0 CRP-10 UREA- 35CREAT-0.4 X RAY- INCREASED BRONCHOVASCULAR MARKINGS | E/A RATIO- 1.3:1 | 60mm hg | 29% | 60% | NO | Bronchopneumonia | Acyanotic CHD Large ASD (L->R) With S. PAH IN CCF | NONE | SYP. Dixin peds, Syp. Furoped, Tab. Aldactone 25 mg 1/2 x OD | NONE | NONE | ASD DEVISE CLOSURE | YES | NO | NO | SURVIVED | | |
| 67 | 1106788 | pratiksha | Mallappa | 1106788 | 5yrs | F | 3 | 2 | Hb- 12.9,TLC- 6000,PLATELETS-6.70 lac RBC-4.67 CRP-58(positive) UREA- 16CREAT- 0.43, X RAY-Increased Bronchovasvular Markings . | E/A RATIO- 1.3:1 | 45mm hg | 26% | 57% | NO | CHD | Acyanotic CHDLarge Sized PDA (L-->R) Shunt | Inj Lasix | to Continue Inj Lasix , To start Oral Digoxin , Tab. Aldoctone, sryp Furoped | NONE | NONE | PDA Ligation | | YES | | SURVIVED | | |
| 68 | 1108389 | Prattik | Prakash | 1108389 | 5 yrs | M | 1 | 2 | Hb- 11.0 TLC-9,700 PLATELETS- 4.93 lac RBC-4.22 CRP-76 UREA-63 CREAT-0.38 X RAY-Normal | E/A RATIO- 1.2:1 | 26mm hg | 22% | 50% | NO | CHD | MIS-C Viral Myocarditis | NONE | NONE | NONE | NONE | NONE | YES | | | SURVIVED | YES | |
| 69 | 1108602 | Sindhu | Ambarish | 1108602 | 2mnths, 15 days | F | 3 | 2 | Hb-12.8 TLC- 18,300 PLATELETS-3.07 LAC RBC-4.91 CRP-74(POSITIVE ) UREA-31 CREAT-0.5 X RAY-Left lower lobe Consolidatin | E/A RATIO- 1.3:1 | 55mm hg | 27% | 56% | NO | CHD | Acyanotic CHD Large VSD (L->R) Shunt with moderate PAH | Inj Lasix | To continue Inj Lasix | NONE | To START INJ DOBUTAMINE . | VSD Ptch Closure | | YES | | DEATH | | |
| 70 | 1108805 | Mallikarjun | Mahadev | 1108805 | 2 months | M | 3 | 2 | Hb-11.6 TLC- 13,000 PLATELETS-5.02 RBC-3.72 CRP-Neg UREA-25 CREAT-0.27 X RAY-RT LOWER LOBE CONSOLIDATION | E/A RATIO- 1.3:10 | 25mm hg | 26% | 57% | NO | CHD | Bronchopneumonia | Inj Lasix | To continue Inj Lasix | inj Dobutamine | inj Dobutamine | NONE | YES | | | SURVIVED | | |
| 71 | 1109905 | Radhabai | Sadeppa | 1109905 | 14yrs | F | 2 | 2 | Hb-13.2 TLC-3,600 PLATELETS-3.2 LAC RBC-5.24 CRP-60 UREA-14 CREAT-0.44 X RAY-PATCH ON LEFT SIDED CONSOLIDATION | E/A RATIO- 1.2:1 | 25mm hg | 29% | 60% | NO | Bronchopneumonia | Large Fossa Ovalis ASD with CHF | NONE | Inj Lasix | NONE | inj Dobutamine | ASD Patch Closure | YES | | | SURVIVED | | |
| 72 | 1117581 | Ronak | Suraj | 1117581 | 8yr | M | 1 | 2 | Hb- 9.7TLC-15,000 PLATELETS-3.2 Lac RBC-3.68 CRP- 62UREA- CREAT-62 X RAY-normal | E/A RATIO- 1.2:1 | 25mm hg | 28% | 62% | NO | Bronchopneumonia | Bronchopneumonia | NONE | NONE | NONE | NONE | NONE | yes | | | SURVIVED | | |
| 73 | 1119581 | Ahamed | Ahamad Rasut | 1119581 | 18 months | M | 1 | 2 | Hb8.9- TLC-9,900 PLATELETS-2.77 LAC RBC-4.64 CRP-254 UREA- 28CREAT-0.25 X RAY-Cardiomegaly | E/A RATIO- 1.3:1 | 53mm hg | 26% | 56% | NO | CHD | Acyanotic CHD Large Perimembranous VSD (L-->R) With Moderate PAH in CCF | Inj Lasix | To continue Inj Lasix To start srp Dixin peds | NONE | NONE | VSD Patch closure | | YES | | SURVIVED | | |
| 74 | 1121818 | | Ramu | 1121818 | 5 yrs | M | 2 | 2 | Hb-12.3 TLC-9,1200 PLATELETS-3.69 lac RBC-4.63 CRP-40 UREA- 14CREAT-0.5 X RAY- normal | E/A RATIO- 1.3:1 | 25mm hg | 25% | 58% | NO | CHD | CHD (Bicuspid Aortic valve) | NONE | NONE | Inj Lasix | NONE | AORTIC BALOON VALVOTOMY | YES | | | SURVIVED | | |
| 75 | 1121018 | Sanvi | Ravi kamble | 1121018 | 6yrs | F | 2 | 3 | Hb- 12.1TLC- 16.6PLATELETS-362k RBC-4.63 CRP negUREA-17 CREAT-0.4 X RAY- NORMAL | E/A RATIO-1.1:1 | 65mm Hg | 26% | 58% | NO | CHD | Acyanotic CHD ,large PDA,(L->R)with severe PAH | inj Lasix | inj Lasix | NONE | NONE | PDA DEVICE CLOSURE | YES | | | DEATH | | |
| 0 | | | | | | | | | | | | | | | | | | | | | | | | | | |
| 0 | | | | | | | | | | | | | | | | | | | | | | | | | | |
| 52 | | | | | | | | | | | | | | | | | | | | | | | | | | |
| 53 | | | | | | | | | | | | | | | | | | | | | | | | | | |