"COMPARISON OF VENOUS EXCESS ULTRASOUND SCORE AND INFERIOR VENA CAVA COLLAPSIBILITY INDEX IN THE ASSSESSMENT OF VENOUS CONGESTION IN CONGENITAL HEART DISEASE PRESENTING WITH HEART FAILURE"

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ABBREVIATIONS

CHD	-	Congenital Heart Disease
HF	-	Heart Failure
RHD	-	Rheumatic Heart Disease
VExUS	-	Venous Excess Ultrasound Score
IVCCI	-	Inferior Vena Cava Collapsibility Index
CVP	-	Central Venous Pressure
POCUS	-	Point-of-Care Ultrasound
HFC	-	Heart Failure Classification
PVPI	-	Portal Vein Pulsatility Index
IVC	-	Inferior Vena Cava
ASD	-	Atrial Septal Defect
VSD	-	Ventricular Septal Defect
ECMO	-	Extracorporeal Membrane Oxygenation
CAD	-	Coronary Artery Disease
TORCH	-	Toxoplasmosis, Other infections, Rubella, Cytomegalovirus, and Herpes simplex
RAAS	-	Renin-Angiotensin-Aldosterone System
GFR	-	Glomerular Filtration Rate
PKU	-	Phenylketonuria
NSAIDs	-	Non-Steroidal Anti-Inflammatory Drugs
HFrEF	-	Heart Failure with Reduced Ejection Fraction
LMICs	-	Low- and Middle-Income Countries
DiGeorge Syndrome	-	Genetic disorder associated with congenital heart defects

PDA	-	Patent Ductus Arteriosus
HCM	-	Hypertrophic Cardiomyopathy
DCM	-	Dilated Cardiomyopathy
VAD	-	Ventricular Assist Device
AKI	-	Acute Kidney Injury
PPCM	-	Peripartum Cardiomyopathy

ABSTRACT

Introduction: Congenital heart disease (CHD) is a leading cause of heart failure (HF) in children, with significant differences in prevalence and management between high-income and low- and middle-income countries. Advances in diagnostic and surgical treatments have improved survival rates, leading to more children living with chronic HF.Accurate assessment of venous congestion, a key factor in HF, is essential for optimal treatment. Tools like the Venous Excess Ultrasound Score (VExUS) and Inferior Vena Cava Collapsibility Index (IVCCI) have emerged for non-invasive monitoring of venous congestion. However, data comparing their use in pediatric CHD patients is limited. This study aims to evaluate the effectiveness of VExUS and IVCCI in assessing venous congestion in children with CHD and HF. The findings could help optimize care, reduce complications, and improve outcomes for pediatric heart failure patients globally, especially in resource-limited settings.

Aims and Objectives: The primary objective of this study is to compare Venous excess ultrasound (VExUS) score with Inferior vena cava collapsibility Index (IVCCI) in assessment of venous congestion in congenital heart failure patients presenting in heart failure.The secondary objective is to assess the correlation between the VExUS score and clinical signs of right heart failure, providing insights into the clinical relevance of this non-invasive tool in managing venous congestion in CHD-related heart failure.

Methodology: This study is a cross-sectional, single-centre study conducted at KLE Dr. Prabhakar Kore Hospital, Belagavi, over a one-year period from August 2023 to September 2024. It aims to compare the Venous Excess Ultrasound (VExUS) score with the Inferior Vena Cava Collapsibility Index (IVCCI) for assessing venous

congestion in children with congenital heart disease (CHD) and heart failure. The sample size is 60, determined using previous study data. Inclusion criteria are children aged 1 month to 17 years admitted with CHD and heart failure. Exclusion criteria include inadequate ultrasound windows, IVC thrombus, cirrhosis with portal hypertension, and renal failure. Data collection includes detailed history, clinical examination, echocardiography, and bedside ultrasound, including IVCCI and VExUS assessments. Statistical analysis will include descriptive statistics, comparisons of quantitative and categorical variables, and testing for normality. Ethical clearance has been granted, and informed consent is obtained from parents or guardians as appropriate. The study will contribute to understanding the utility of these non-invasive tools in monitoring venous congestion in pediatric heart failure patients.

Results & Conclusion: This study analyzed 60 pediatric patients with congenital heart disease (CHD) and heart failure (HF), focusing on diagnostic indices like the Inferior Vena Cava Collapsibility Index (IVCCI), Portal Vein Pulsatility Index (PVPI), and Venous Excess Ultrasound Score (VExUS) to assess venous congestion and its association with clinical outcomes. The cohort had a mean age of 5.42 years, with a higher proportion of female patients. Common clinical features included failure to thrive, respiratory distress, and hepatomegaly. Atrial Septal Defect and Ventricular Septal Defect were the most common diagnoses. VExUS, IVCCI, and PVPI were reliable for detecting venous congestion, with significant associations observed between VExUS scores and the Modified Ross Heart Failure Classification, as well as IVCCI and PVPI. While patients with higher VExUS scores showed trends toward worse outcomes, including higher mortality, the results were not statistically significant. This research suggests that while the IVC Collapsibility Index remains a

useful tool, it is less sensitive than the VExUS Score in detecting venous congestion. The VExUS Score incorporates multiple venous flow parameters thus providing a more comprehensive assessment and hence a superior method in CHD patients presenting with HF

Keywords: VExUS score, Point of care Ultrasound, Venous Congestion, IVC Collapsibility Index, Heart Failure, Congenital Heart Disease

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INTRODUCTION

Congenital heart disease (CHD) encompasses a spectrum of anatomical and functional heart and major vessel abnormalities that develop in the fetus. With progress in diagnostic tools and surgical methods, survival rates for individuals with CHD have risen markedly, leading to an expanding group of both children and adults living with these conditions.

In high-income countries, cardiac failure in children is primarily driven by congenital heart anomalies, which affects nearly 8 to 12 for every 1,000 newborns, with 10-15% of these children developing heart failure (HF), while cardiomyopathies contribute 0.87 to 1.13 cases per 100,000 children annually. A U.S. study estimated that paediatric HF affects about 14,000 to 15,000 children annually.[1,2] In developing nations, paediatric heart failure (HF) poses a greater challenge due to the elevated occurrence of unaddressed congenital cardiac anomalies and acquired conditions such as rheumatic carditis, which plays a significant role in HF cases across regions like Africa and certain areas of Asia, impacting 1-5 out of every 1,000 children. Additionally, illnesses like Chagas disease in Latin America further increase the strain of HF in these populations.[3,4]

However, heart failure remains a common and critical complication[5] Detailed examination for venous overload in these patients is crucial for guiding therapy and improving outcomes. Venous congestion, marked by elevated jugular venous pressure and impaired venous return, has a pivotal impact in pathophysiology of cardiac failure. It contributes to organ dysfunction, oedema, and poor perfusion. Monitoring venous congestion traditionally relies on physical examination and invasive circulatory measurements such as Central Venous Pressure (CVP). However, these

methods have limitations: clinical signs can be subtle and nonspecific, while invasive monitoring carries risks and is resource-intensive.[6] Thus, non-invasive methods for evaluating venous congestion have gained prominence in recent years.

Heart failure in India is driven by a high burden of rheumatic carditis, congenital heart disease (CHD), cardiomyopathies, infections, and malnutrition. Untreated or late-diagnosed CHD and postoperative complications contribute to paediatric heart failure, while Rheumatic Heart Disease (RHD) remains a major cause in young adults due to recurrent streptococcal infections. Cardiomyopathies, including dilated and hypertrophic forms, are linked to genetic factors, infections, and nutritional deficiencies. Viral myocarditis and bacterial endocarditis further exacerbate heart failure progression. Additionally, premature coronary artery disease (CAD), rising childhood obesity, hypertension, severe anaemia, and malnutrition increase the risk of heart failure across all age groups. Congenital heart disease (CHD) arises from genetic, chromosomal, and environmental factors. Genetic mutations (e.g., NKX2-5, GATA4) and chromosomal anomalies like Down syndrome, Turner syndrome, and DiGeorge syndrome contribute significantly. Maternal infections (TORCH), particularly rubella, increase risk, while teratogenic drugs such as isotretinoin, thalidomide, and lithium can cause structural defects. Maternal conditions like diabetes, phenylketonuria, and alcohol use, along with radiation exposure, also elevate risk. CHD often results from multifactorial interactions between genetic predisposition and environmental insults.[7,8]

CHD and heart failure contribute significantly to infant and paediatric mortality in India, with CHD causing 10% of infant deaths and 80,000 annual fatalities due to delayed diagnosis and limited surgical access. Nearly 25% of infants with critical CHD die within the first year without timely intervention.

Postoperative mortality remains high due to inadequate critical care, while chronic consequences include elevated pulmonary vascular resistance and neurodevelopmental delays. Heart failure has a one-year mortality rate of 20% and a 30-day readmission rate of 25%, driven by late diagnosis, poor healthcare access, economic barriers, and infectious complications.[8]

The Venous Excess Ultrasound Score (VExUS) and the Inferior Vena Cava Collapsibility Index (IVCCI) are two ultrasound-based tools increasingly used in the evaluation of venous overload. VExUS is a semi-quantitative scoring system that incorporates Doppler ultrasound evaluation of liver, portal, and kidney veins to yield an integrated assessment of venous congestion. It has been validated in adult populations.[9] In contrast, IVCCI evaluates the compressibility of IVC during breathing, providing an indirect estimate of CVP. IVCCI is implemented in healthcare practice due to its simplicity and ease of measurement, though it is influenced by factors such as mechanical ventilation, intrathoracic pressure, and body habitus.[10]

The application of these modalities in CHD patients presenting with heart failure poses unique challenges and opportunities. The altered hemodynamics, varied anatomical substrates, and increased prevalence of venous congestion in this population underscore the need for robust, reliable, and reproducible methods of assessment. For instance, the presence of shunts, valvular regurgitation, or Fontan circulation may influence venous flow patterns and limit the utility of conventional tools such as IVCCI.[11] VExUS, with its focus on venous Doppler flow patterns, may provide a more nuanced understanding of congestion in these complex patients.

Heart failure in CHD is frequently associated with increased venous pressures due to impaired ventricular compliance, obstructive lesions, or pulmonary hypertension. Elevated venous pressures contribute to the redistribution of blood volume, organ congestion, and worsening cardiac output. As such, early detection and quantification of venous congestion can inform therapeutic interventions, such as diuretics, fluid management, or advanced therapies like extracorporeal membrane oxygenation (ECMO).[12]

Despite their potential utility, comparative studies of VExUS and IVCCI in the CHD population are sparse. Most existing research focuses on adult populations or general heart failure cohorts, limiting the generalizability of findings to pediatric and congenital settings. A systematic evaluation of these tools in CHD patients presenting with heart failure is essential to determine their relative accuracy, feasibility, and prognostic value.[13] Furthermore, integrating these tools into routine clinical practice could optimize patient monitoring and enhance the precision of therapy, specifically in economically disadvantaged regions where invasive monitoring is not feasible.

The choice of VExUS and IVCCI as comparator tools is driven by their complementary strengths. VExUS, with its focus on organ-specific venous flow patterns, provides a comprehensive assessment of systemic congestion, making it particularly relevant in patients with complex anatomy or extracardiac complications such as renal dysfunction. IVCCI, on the other hand, offers a rapid and straightforward estimation of Central Venous Pressure (CVP), making it ideal for bedside assessment and serial monitoring. Understanding the interplay between these modalities could refine the approach to venous congestion assessment, leading to tailored and effective interventions and treatments.[14]

Moreover, this study aligns with the growing emphasis on close surveillance of hemodynamics non-invasively in vulnerable patients. Innovations in ultrasonography, combined with growing accessibility of handheld devices, have made these tools more accessible and user-friendly. As a result, their integration into routine clinical workflows is both feasible and desirable. The findings of this study could inform future guidelines and pave the way for larger, multicenter trials aimed at validating these tools in diverse populations.[15]

Despite the utility of these tools, there is limited comparative data evaluating reliability of VExUS and IVCCI in children having CHD presenting in HF. Integrating these advanced imaging modalities with clinical classification systems like the Modified Ross Heart Failure Classification may improve diagnostic accuracy and therapeutic precision. This study aims to compare VExUS and IVCCI in their ability to assess venous congestion in children with CHD with HF, addressing a significant gap in evidence. The findings could enhance the understanding of venous congestion in paediatric HF, improve the synergy between clinical and imaging-based assessments, and lead to more tailored management strategies for this vulnerable population.[16]

The worldwide burden of congenital heart defects mortality is high, with India accounting for a significant portion of CHD-related deaths. Around 10% infant deaths in India is attributed to CHD, causing approximately 80,000 deaths annually due to untreated or undiagnosed heart defects. Critical CHD, requiring early surgery, leads to a high percentage of neonatal deaths, especially in underserved regions. Delayed diagnosis and limited surgical access significantly impact survival rates, with 25% of infants with critical CHD dying within the first year if untreated. Postoperative mortality remains high due to limited cardiac care facilities. Morbidity includes

complications such as pulmonary hypertension, growth retardation, and neurodevelopmental delays, with frequent hospitalizations for infections and heart failure. Heart failure has substantial influence on both quality of life and survival rates in India, with a 20% mortality rate within a year of diagnosis. The prevalence of hospital stays due to cardiac failure is rising, with a 25% 30-day readmission rate, particularly in children with CHD, cardiomyopathies, and rheumatic heart disease.

The study aims to compare VExUS and IVCCI in assessing venous overload in CHD cases presenting with heart failure. Specifically, it seeks to evaluate their correlation with clinical markers of congestion, their reproducibility across different operators, and their ability to predict adverse outcomes. By addressing these objectives, this research hopes to bridge the gap in evidence and provide clinicians with practical insights into the management of this vulnerable population. By investigating the effectiveness of these two non-invasive tools, this study will provide valuable insight into their relative utility in detecting fluid overload in complex cases. This study is expected to guide clinical practice, leading to improved diagnostic approaches and tailor effective treatment in pediatric congenital heart disease patients. Ultimately, the goal is to optimize patient care and outcomes through better monitoring of venous congestion and fluid status.

NEED FOR STUDY

Venous congestion is a critical determinant of disease progression in cardiac failure, especially in the context of congenital heart disease (CHD). Precise evaluation of venous overload is essential for guiding treatment and monitoring response to therapy. Traditional clinical assessments often fail to quantify venous congestion effectively, particularly in pediatric populations with CHD, where symptoms can be

nonspecific. Point-of-care ultrasound (POCUS) is an effective resource for non-invasive evaluation of venous overload, with the Venous Excess Ultrasound Score (VExUS) and Inferior Vena Cava Collapsibility Index (IVCCI) being prominent parameters. The VExUS score is a comprehensive ultrasonography tool that incorporates multiple venous flow patterns to assess systemic venous congestion. It provides detailed insights into venous pressure and its systemic effects. Meanwhile, the IVCCI, which measures the dynamic changes in IVC diameter during respiration, is a simpler and widely used metric to estimate right atrial pressure and volume status. Although IVCCI is frequently employed in clinical practice, its reliability may depend on age, respiratory patterns, and mechanical ventilation. In contrast, VExUS is potentially more robust in detecting venous congestion across a wider range of clinical scenarios. To date, comparative statistics on the utility of VExUS and IVCCI in pediatric patients with CHD presenting in HF is inadequate. Understanding the relative strengths and limitations of these methods is crucial for optimizing the assessment of venous congestion in this unique population. This study aims to compare VExUS and IVCCI in their ability to assess venous congestion in children with CHD and HF, addressing a significant gap in evidence. The findings could pave the way for improved diagnostic strategies and tailored management approaches, ultimately enhancing clinical outcomes in this vulnerable population.

AIMS AND OBJECTIVES

Primary objective:

- Compare Venous excess ultrasound (VExUS) score with Inferior vena cava collapsibility Index (IVCCI) in the assessment of venous congestion in congenital heart disease presenting in heart failure.

Secondary objective

- Assess the correlation of Venous Excess Ultrasound score with clinical signs of right heart failure.

REVIEW OF LITERATURE

Heart failure (HF) is distinct from its adult counterpart, with diverse aetiologies that vary by age, geography, and underlying health conditions. Unlike adult HF, which is largely due to ischemic heart disease or hypertension, paediatric HF is commonly associated with congenital heart defects (CHDs), cardiomyopathies, and, in certain regions, infectious diseases such as rheumatic fever and myocarditis. The prevalence of HF in the paediatric age group is less frequent compared to adults but remains a critical public health concern due to its substantial burden on healthcare systems, morbidity, and mortality rates.

Global Prevalence

Developed Countries

In high-income countries, HF in children is primarily driven by congenital heart disease (CHD), which affects 8 to 12 for every 1,000 newborns. Studies suggest that between 10-15% of children with CHD develop heart failure. Other notable contributors include cardiomyopathies, accounting for 0.87 to 1.13 cases per 100,000 children annually.[3]

Paediatric HF impacts about 14,000 to 15,000 children every year in USA.[4] Recent breakthroughs in therapeutic strategies led to better patient outcomes for CHD, increasing the number of children living with chronic HF.

Developing Countries

In developing nations, the impact of paediatric HF is higher due to a significant prevalence of untreated CHD and acquired conditions such as rheumatic carditis. RHD is a primary etiological factor of HF in children in Africa and South Asia.[17] Infections like Chagas disease in Latin America also contribute to HF prevalence.

Age-Specific and Aetiology-Based Prevalence

Neonatal and Infant Population

HF in neonates and infants is largely secondary to critical congenital cardiac diseases, including hypoplastic left heart syndrome (HLHS) and ventricular septal defects (VSDs). The incidence of HF in neonates with ductal-dependent CHDs is approximately 30-50% prior to surgical correction .[18]

Cardiomyopathies

Paediatric cardiomyopathies, particularly dilated cardiomyopathy (DCM), account for 1-3 cases per 100,000 children annually. Genetic mutations and metabolic disorders often underlie these conditions.[19]

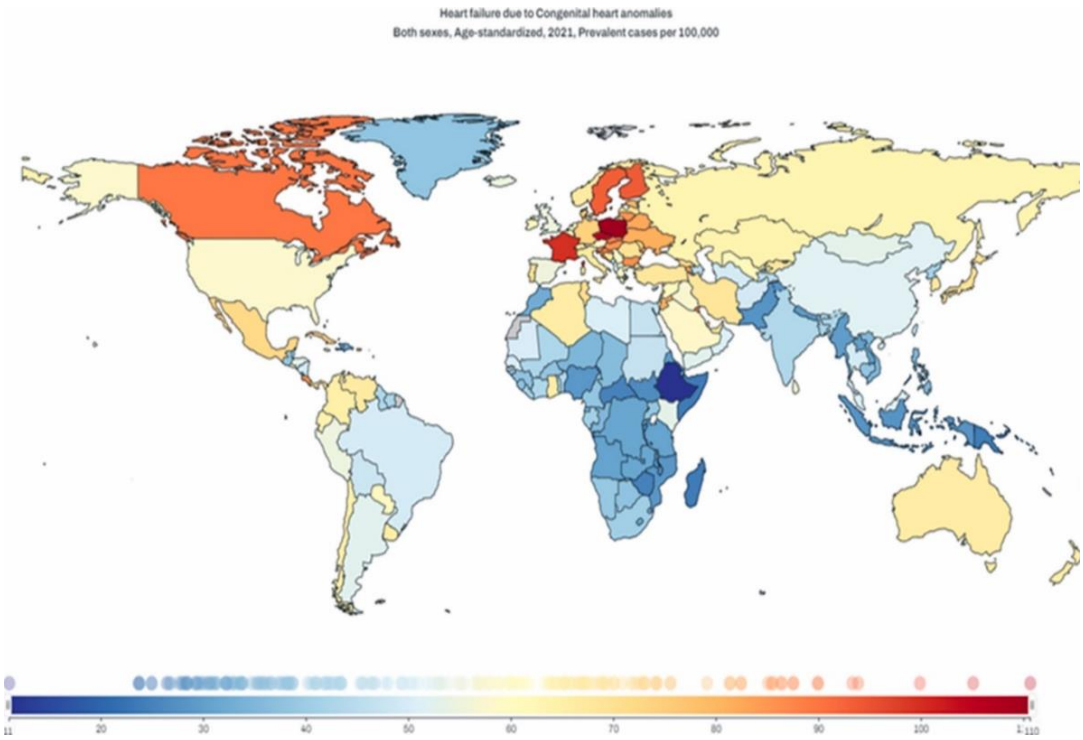


Figure 1: Country specific prevalence for every 100,000 of cardiac failure in 2021.[20]

Acquired Causes

Inflammatory conditions such as myocarditis contribute to 0.5-1.0 cases per 100,000 children annually. In Low and Middle Income Countries (LMICs), infectious aetiologies like HIV-associated cardiomyopathy and malaria-related myocarditis are more prominent. [21]

Rising Non-Congenital Causes: Improved vaccination and healthcare systems in LMICs are reducing infectious causes of HF, but the burden of malnutrition and undiagnosed cardiomyopathies remains significant.

Prevalence of heart failure in India

The prevalence of cardiac failure in pediatric age group with congenital heart disease (CHD) depends on defect, severity, and availability of timely surgical and medical interventions.[21] Among children with CHD, heart failure prevalence ranges from 10–30%, depending on factors such as the specific defect and access to healthcare.

Studies conducted across different regions of India report CHD prevalence rates varying from 0.8 to 26.4 per 1,000 children, with higher rates noted in urban and hospital-based cohorts due to better diagnostic capabilities.[1] The disparity in prevalence data arises due to differences in study design, age group selection, and diagnostic methodologies. Notably, most prevalence studies have been conducted among school-aged children (5–15 years), potentially underestimating the burden among neonates and infants, where HF due to CHD is more common.[2] Furthermore, in low-resource settings, undiagnosed and untreated CHD progresses to heart failure, significantly contributing to paediatric cardiac mortality.

Apart from CHD, rheumatic heart disease (RHD) remains a notable contributor to paediatric heart failure in India, particularly in low-income populations with inadequate access to early antibiotic prophylaxis. Although the prevalence of RHD has declined over the years, it remains a concern in some regions, with studies indicating a prevalence of 0.5–2 per 1,000 school-aged children.[22] Additionally, infectious causes such as viral myocarditis and endocarditis have been reported as contributors to paediatric heart failure, particularly in children presenting with acute-onset cardiac dysfunction.[23]

In resource-rich settings, early detection and timely surgical interventions for CHD have substantially decreased the incidence of heart failure in neonates and infants. However, in developing nations, the prevalence of HF remains much higher due to delayed diagnosis and lack of access to advanced care. Reports suggest that up to 30–50% of children with uncorrected critical CHD in LMICs experience heart failure, compared to 10–15% in high-income countries.[24] Even after successful surgical correction, some children develop postoperative heart failure as a result of residual defects, pulmonary hypertension, or long-term ventricular dysfunction.[19]

Globally, congenital heart disease accounts for approximately 30% of all paediatric heart failure cases. CHD-associated HF has significant implications for long-term outcomes, including a higher risk of arrhythmias, growth retardation, and neurodevelopmental challenges. Survivors of HF in the context of CHD may also face progressive ventricular dysfunction and chronic HF as they age.[3] Addressing these challenges requires increased access to early intervention and comprehensive care, especially in LMICs, where the burden of CHD-related HF is disproportionately high.

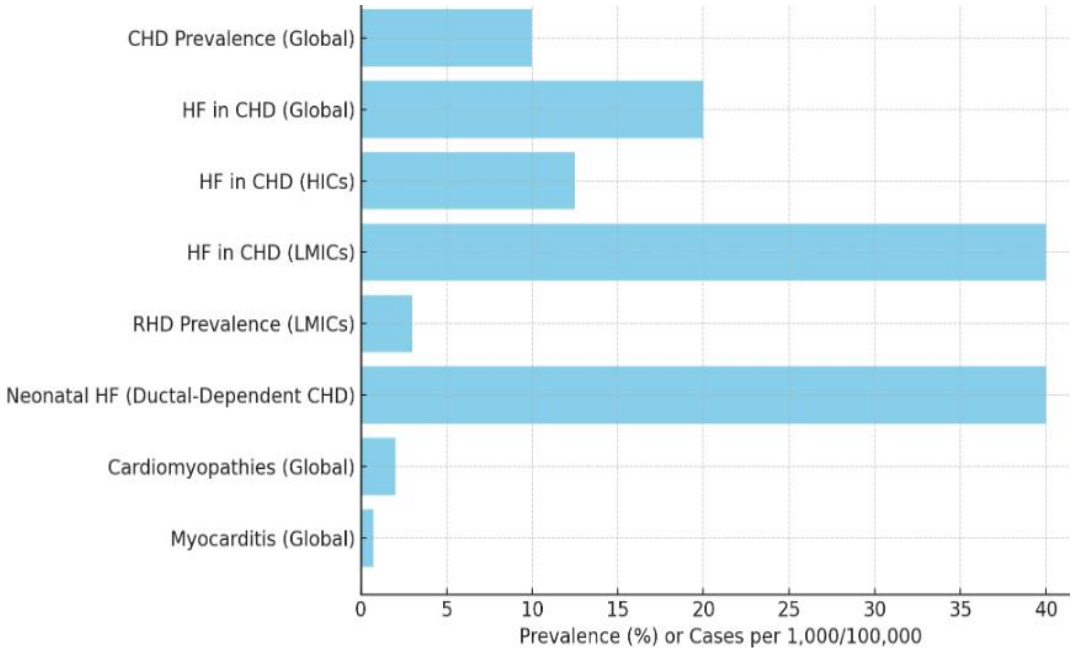


Figure 2: Global prevalence and causes of paediatric heart failure. [20]

Prior meta-analyses[25,26] have revealed a worldwide escalation in the birth prevalence of congenital heart defects (CHD), yet persistently low rates in impoverished nations. These observations are largely due to increased accessibility of diagnostic tools in developing nations, combined with the ongoing underdiagnosis of CHD in childhood in underdeveloped settings. However, 'missed cases' at birth only partially indicate the possible gaps in medical care for children with CHD. In this context, international trends in availability of medical care for infants diagnosed with CHD are scarce. Epidemiological investigations focusing on pediatric populations of school age have revealed heterogeneous estimates of CHD prevalence, spanning a range of 0.5 to 18 per 1,000.[27] Unlike investigations into congenital heart defects among live births, which leverage large-scale birth registries, research on school-aged children is often characterized by relatively small sample sizes, thus limiting the generalizability of findings from any single study. [28,29]

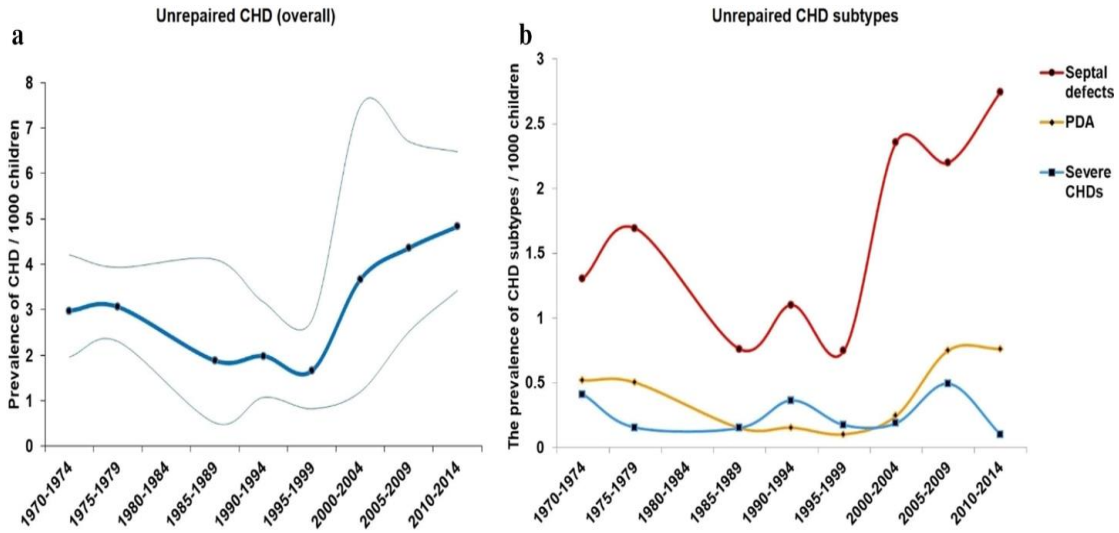


Figure 3: Trends in the worldwide prevalence of unrepaired congenital heart defects and their subtypes among school-aged children between1970 and 2014 [30]

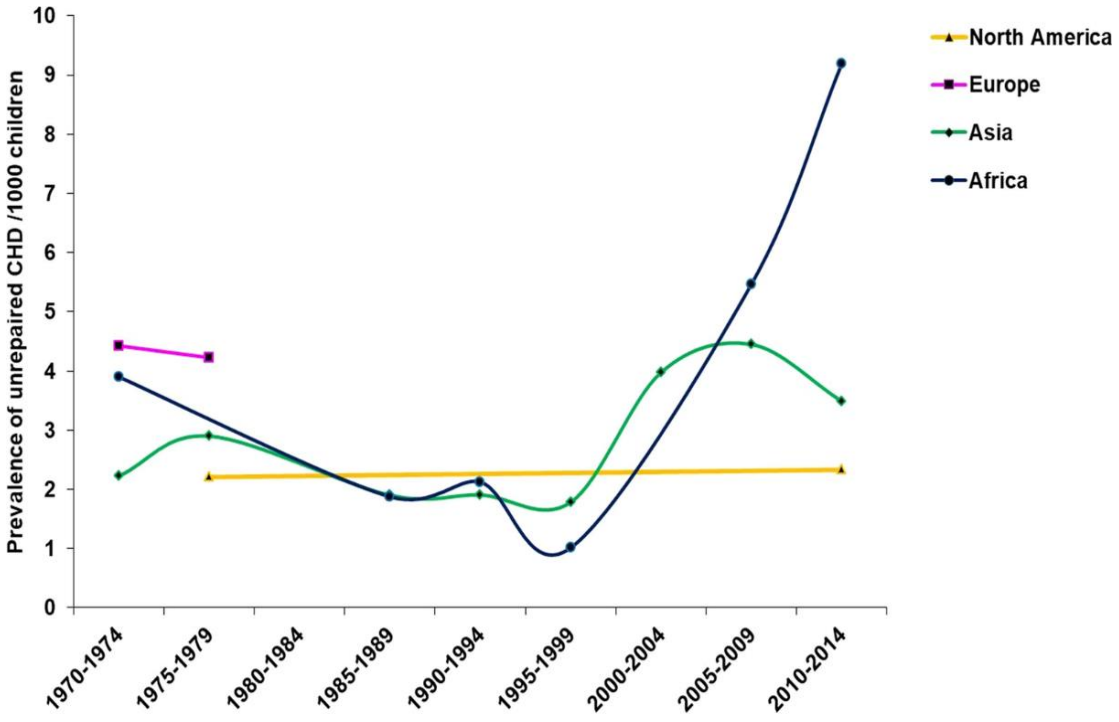


Figure 4: A geographic breakdown of the data showed upward trend in the reported prevalence of unrepaired CHD in Asia (p = 0.034) and Africa (p = 0.029) over the period from 1995 to the present. [30]

AETIOLOGY OF CONGENITAL HEART DISEASE (CHD) IN INDIA

CHD is a major cause of illness and death among children in India, with a substantial prevalence of 8 to12 for every 1,000 newborns. The aetiology of CHD is multifactorial, involving genetic predispositions, maternal health factors, environmental influences, and nutritional deficiencies.

Genetic Factors

Genetic abnormalities play a crucial role in CHD development. Chromosomal disorders such as Down syndrome (Trisomy 21), Turner syndrome, and DiGeorge syndrome are associated with specific cardiac malformations.[31] Mutations in genes regulating cardiac morphogenesis, such as NKX2-5, GATA4, and TBX5, have also been implicated in CHD pathogenesis.[32]

Familial inheritance patterns suggest a strong genetic component in CHD, with an increased risk among siblings and first-degree relatives. Studies indicate that having a first-degree relative with CHD has increased likelihood of contracting the condition, highlighting the involvement of inherited genetic variants and shared environmental influences.

Chromosomal Anomalies

Several genetic syndromes are strongly associated with congenital heart defects (CHDs), each presenting with characteristic cardiac anomalies. Down Syndrome (Trisomy 21) is commonly linked to atrioventricular (AV) septal defects and ventricular septal defects (VSD) due to abnormal endocardial cushion development. Turner Syndrome (45,X) frequently presents with coarctation of the aorta and aortic valve malformation, leading to increased cardiovascular

complications. DiGeorge Syndrome (22q11.2 deletion) is linked to congenital heart defects involving the outflow tracts, including Tetralogy of Fallot and truncus arteriosus, resulting from abnormal neural crest cell migration. Trisomy 18 (Edwards Syndrome) commonly features VSD, patent ductus arteriosus (PDA), and polyvalvular dysplasia, contributing to significant cardiac dysfunction. Trisomy 13 (Patau Syndrome) often involves VSD, atrial septal defect (ASD), and PDA, with complex structural abnormalities contributing to high neonatal mortality. Understanding these syndromic associations aids in early diagnosis, genetic counselling, and management of congenital heart defects.

Infections (TORCH Group)

Infections during pregnancy can lead to congenital heart defects in the fetus. Toxoplasmosis is one such infection that can cause a range of birth defects, though its cardiac effects are less defined. Other infections, including syphilis, varicella-zoster, and parvovirus B19, can also contribute to congenital anomalies, though they typically cause a broader spectrum of systemic effects. Rubella is strongly associated with patent ductus arteriosus (PDA), pulmonary stenosis, and septal defects, particularly when the mother contracts the infection during the first trimester. Cytomegalovirus (CMV) infection can lead to cardiomyopathy and septal defects in affected infants, causing significant cardiac and systemic complications. Although herpes simplex virus is rarely associated with congenital heart defects, it has been documented to cause myocardial damage in some cases.[35]

Teratogenic Drugs

Certain medications during pregnancy can lead to congenital heart defects and other malformations. Isotretinoin (retinoic acid), commonly used for acne treatment,

has been linked to conotruncal defects. Thalidomide, once used as a sedative, is associated with severe limb and heart defects, including abnormalities in the great vessels and other structural heart malformations. Lithium, used for bipolar disorder, is known to cause Ebstein's anomaly. Anticonvulsants like phenytoin and valproate have been linked to septal defects and conotruncal defects, particularly when used in the first trimester. Lastly, the use of NSAIDs in late pregnancy can result in the abnormal early closing of ductus arteriosus, and thus possible elevated pulmonary pressures and other serious cardiovascular complications in the new-born.

Maternal and Environmental Factors

Maternal conditions significantly influence CHD risk. Poorly controlled maternal diabetes increases the likelihood of congenital heart defects, including transposition of the great arteries and ventricular septal defects (VSD).[32] Additionally, maternal infections, particularly rubella, have been linked to congenital rubella syndrome.

Environmental factors, including exposure to teratogenic substances such as alcohol, tobacco, and certain medications (e.g., isotretinoin and thalidomide), contribute to CHD incidence. Air pollution and heavy metal exposure are associated with an increased likelihood of adverse outcomes, particularly in highly industrialized regions. Phenylketonuria (PKU), a metabolic disorder resulting from insufficient phenylalanine hydroxylase, can lead to obstructive defects like coarctation of the aorta and hypoplastic left heart syndrome if not well-controlled during pregnancy[36]

Nutritional Deficiencies

Prenatal deficiencies in folic acid, iodine, and vitamin D are linked to higher likelihood of CHD. Folic acid supplementation has been shown to reduce neural tube defects and certain congenital cardiac anomalies (Singh et al., 2020). Iodine deficiency disrupts fetal thyroid function, affecting overall fetal development, including cardiac structure.[33]

AETIOLOGY OF HEART FAILURE IN INDIA

Heart failure in India has distinct epidemiological patterns compared to Western populations, largely due to the high burden of rheumatic heart disease, congenital defects, and infectious causes.[33]

Congenital Heart Defects and Post-Surgical cardiac Failure

Untreated or late-diagnosed CHD have increased susceptibility for developing cardiac failure due to chronic volume or pressure overload. Postoperative heart failure is a concern in children undergoing CHD corrective surgery, particularly in cases of residual shunts or pulmonary hypertension.[7]

Rheumatic Heart Disease (RHD)

Rheumatic carditis remains a leading cause of heart failure in India, primarily affecting young adults. Recurrent streptococcal infections and inadequate access to prophylactic antibiotics contribute to the high prevalence of valvular heart disease leading to heart failure.

Cardiomyopathies

Dilated cardiomyopathy (DCM) and hypertrophic cardiomyopathy (HCM) are prevalent in both paediatric and adult populations in India. DCM is often idiopathic but can be secondary to infections, nutritional deficiencies, or genetic predisposition. HCM, commonly linked to sarcomeric gene mutations, accounts for a leading proportion of sudden cardiac fatalities in young sport participants.

Myocarditis and Infectious Causes

Viral myocarditis, particularly due to Coxsackie virus and enteroviruses, is an emerging cause of heart failure in Indian children. Bacterial endocarditis, often secondary to congenital or rheumatic heart disease, exacerbates heart failure progression.[8]

Peripartum Cardiomyopathy (PPCM)

Peripartum cardiomyopathy is a significant contributor to heart failure in Indian women, particularly in rural areas with high rates of malnutrition and anaemia. PPCM is often underdiagnosed and associated with poor maternal outcomes.

Coronary Artery Disease (CAD) and Hypertension

Childhood obesity and hypertension are emerging contributors to paediatric heart failure.[32]

Anaemia and Malnutrition

Severe anaemia, common in Indian children and pregnant women, increases cardiac workload, leading to high-output heart failure. Protein-energy malnutrition affects myocardial function, exacerbating cardiac failure in children with underlying cardiac anomalies.[34]

MORTALITY AND MORBIDITY OF CONGENITAL HEART DISEASE (CHD) AND HEART FAILURE

Mortality and Morbidity Due to CHD

The global burden of CHD mortality is high, and India accounts for a substantial proportion of CHD-related deaths. Approximately 10% of infant mortality in India is attributable to CHD, with an estimated 80,000 infant deaths annually due to undiagnosed or untreated heart defects. Critical CHD, requiring early surgical intervention, accounts for a large percentage of neonatal deaths, especially in rural and underserved regions.[37]

Delayed diagnosis and lack of access to surgical correction significantly impact survival rates.[38] Postoperative mortality in CHD patients remains high due to limited cardiac critical care facilities, particularly in government hospitals.

Morbidity associated with CHD includes long-term complications such as pulmonary hypertension, growth retardation, and neurodevelopmental delays. Children with CHD often experience recurrent hospitalizations due to infections and heart failure exacerbations.[39]

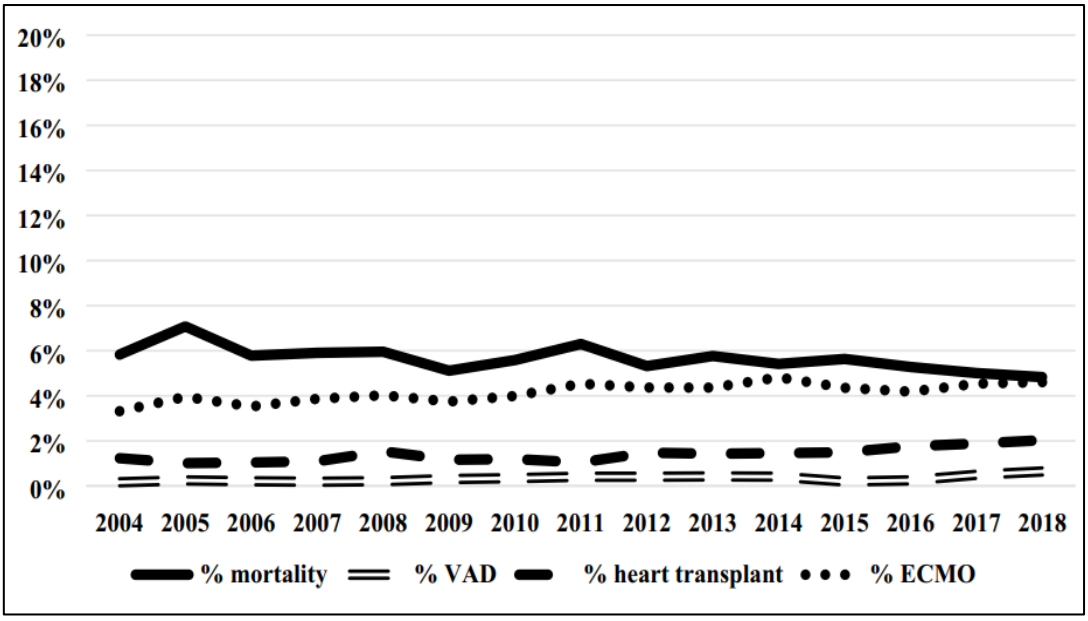


Figure 5: Pediatric HF with CHD trend in USA[40]

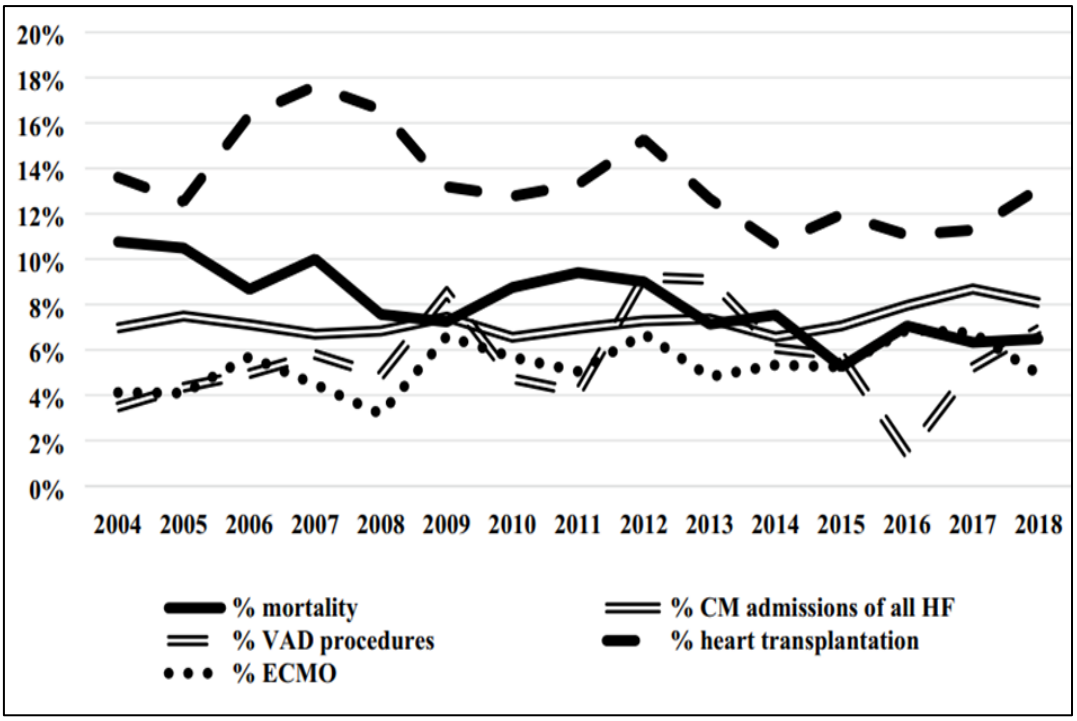


Figure 6: Cardiomyopathy-related heart failure trends in US pediatric population [40]

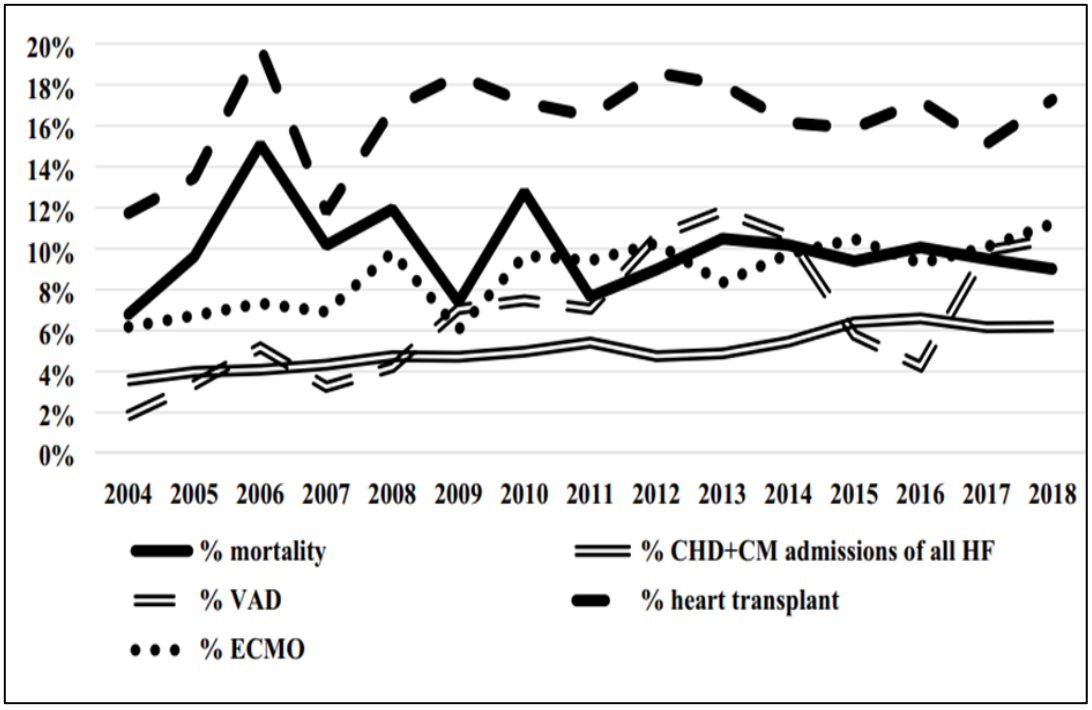


Figure 7: Trends in pediatric heart failure attributed to cardiomyopathy and congenital heart anomalies in the United States[40]

Mortality and Morbidity Due to Heart Failure in India

Heart failure in India is marked by high rates of complications and fatalities, largely due to late-stage presentation and inadequate healthcare infrastructure. The estimated mortality rate for heart failure in India is approximately 20% within one year of diagnosis, significantly higher than in developed countries. The prevalence of admissions due to cardiac failure has been rising, with a reported 30-day readmission rate of nearly 25%.[41]

Factors Contributing to High Mortality and Morbidity in India

Several factors lead to high mortality and morbidity of CHD and heart failure in India*:* Delayed Diagnosis: Many cases of CHD go undiagnosed until late infancy or childhood due to inadequate neonatal screening programs. Limited Access to Pediatric Cardiac Surgery: There is a shortage of specialized pediatric cardiac centers, particularly in rural areas. Economic Barriers: The high cost of cardiac surgery and long-term heart failure management is prohibitive for many families, leading to poor outcomes. Infectious Complications: Bacterial endocarditis and viral myocarditis exacerbate heart failure progression in CHD patients. High Burden of Rheumatic Heart Disease: RHD remains a significant contributor to heart failure mortality in young adults.[8]

VENOUS CONGESTION IN HEART FAILURE

Venous congestion is a critical pathophysiological component of heart failure due to a decline in the heart's contractile function, and thus impaired blood flow, leading to a backlog of blood in the venous system. This impaired venous blood flow leads to elevated venous pressures throughout the body. In heart failure, particularly

congestive heart failure (CHF), this venous congestion can have systemic effects, leading to complications such as pulmonary edema, peripheral edema, hepatomegaly, ascites, and renal dysfunction.[42] These symptoms are a consequence of the increased venous pressure that interferes with normal organ function. Venous congestion in CHF is most often due to decreased cardiac output and altered circulatory dynamics, which leads to impaired contractile function. However, the pathophysiology of venous congestion is not as well understood in patients with heart defects since birth, especially when they present with heart failure, making diagnosis and treatment more complicated.

In patients with CHF, elevated central venous pressure (CVP) leads to congestion of the systemic venous system..[43] For example, when the right atrium and ventricle fail to empty properly, blood backs up into the vena cavas, and from there into the hepatic veins, renal veins, and eventually the portal vein. As venous pressures rise, fluid begins to leak from the vascular system into surrounding tissues, resulting in edema in the lower limbs, ascites in the abdomen, and hepatomegaly due to liver congestion. Additionally, the kidneys are affected by the elevated venous pressure[44,45]

Although these clinical features of venous congestion are established in adult patients with CHF, their presence and significance in patients with congenital heart defects are less understood. Children with CHD, particularly those who have undergone surgeries or interventions to correct heart defects, often present with complex circulatory patterns, making the diagnosis of fluid overload more challenging. These children may have mixed circulatory states due to residual shunts, ventricular dysfunction, or abnormal pulmonary pressures, which complicate the interpretation of traditional clinical signs of venous congestion .[46] The symptoms of venous congestion in CHD patients may not always manifest in the same way as in

adults and can be subtle, especially in early stages. Additionally, CHD patients are more likely to have abnormal hemodynamics, which can lead to inconsistent or atypical manifestations of congestion .[47]

The presence of venous congestion in CHD patients can significantly affect their prognosis and outcomes. Without accurate and timely monitoring, the undiagnosed or inadequately managed venous congestion can worsen heart failure symptoms, leading to frequent hospitalizations, extended hospital stays, and increased adverse outcomes. In some cases, uncontrolled venous congestion can cause irreversible damage to organs such as the liver and kidneys, contributing to organ failure and ultimately decreasing survival rates .[48,49] The development of more precise diagnostic tools to assess venous congestion in these patients is essential to optimize treatment strategies and improve patient outcomes.

Traditional methods for monitoring fluid overload and venous congestion, such as physical examination and invasive jugular venous pressure measurements, have limitations. Physical signs such as peripheral oedema or ascites can be difficult to interpret in paediatric patients, and these signs may not appear until congestion is advanced. Furthermore, CVP measurements, while helpful in assessing venous pressure, are invasive and carry risks, including infection, thrombosis, and arrhythmias .[50] Non-invasive and accurate diagnostic tools are crucial in detecting and managing venous congestion, especially in paediatric CHD patients who may not exhibit typical symptoms.

In the current era, Point-of-care ultrasound (POCUS) has gained prominence as a real-time imaging tool for venous congestion and fluid overload evaluation. Ultrasound techniques such as measuring the variations in the diameter of the inferior

vena cava (IVC) during respiration have been shown to provide valuable insights into the venous overload. The Inferior Vena Cava Collapsibility Index (IVC-CI) is one such technique that estimates right atrial pressure and fluid status by assessing the extent of collapse of IVC on respiration [51]. This method is easy to perform and has been validated as an effective tool in adult and pediatric heart failure management. However, while IVC-CI provides a reliable measure of venous pressure, it may not fully capture the extent of venous congestion, especially in patients with complex circulatory disorders such as CHD.

PATHOPHYSIOLOGY OF VENOUS CONGESTION IN CONGENITAL HEART DISEASE

Venous congestion has significant impact in the pathological mechanisms underlying cardiac failure, and its complexity is pronounced in patients with congenital cardiac anomalies. CHD comprises a broad range of anatomical anomalies that impair the heart's capacity for efficient blood circulation. These abnormalities result in altered hemodynamics that contribute to venous congestion, which is defined by the impaired return of blood from the peripheral circulation to the heart, resulting elevated venous pressures in various parts of the circulatory system. Venous congestion can occur both systemically (involving organs such as the liver and kidneys) and pulmonary (involving the lungs), leading to various complications, including organ dysfunction, fluid overload, and worsening heart failure symptoms.

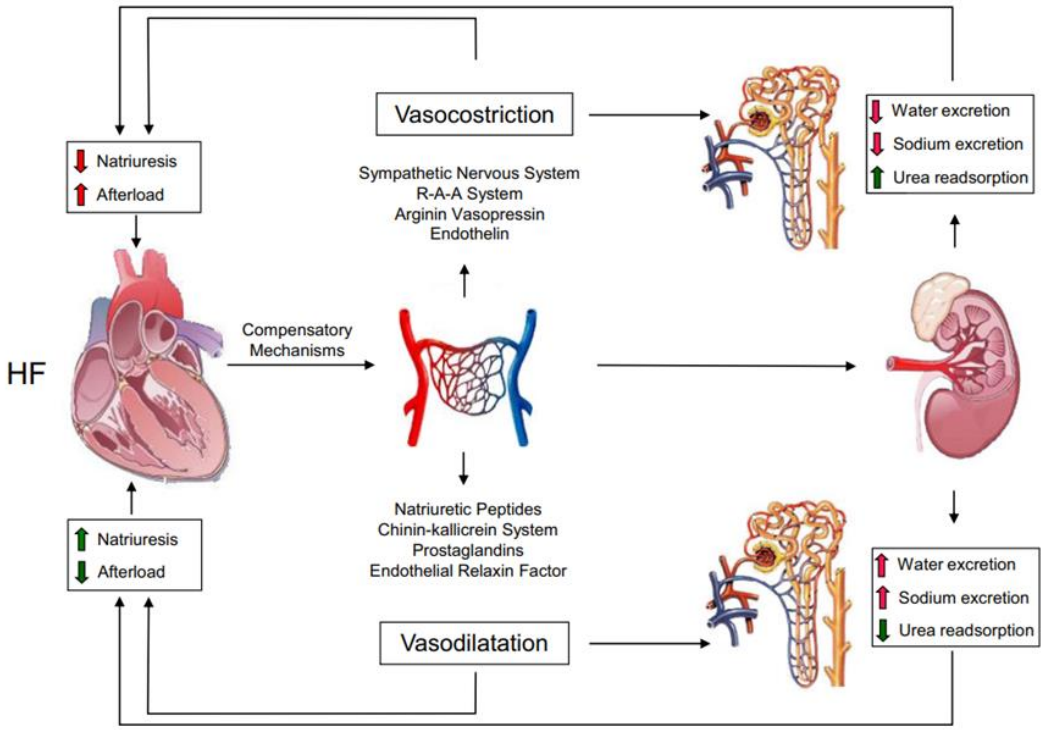


Figure 8: Pathophysiology of venous congestion [52]

Hemodynamic Abnormalities in Congenital Heart Disease

Congenital heart disease comprises structural defects, like atrial septal defect, ventricular septal defect, obstructive defects (e.g., coarctation of the aorta), and complex anomalies like single ventricle physiology [53] These conditions lead to altered circulatory dynamics, where the normal pathways of blood flow are disrupted, and the distribution of blood volume is not optimal. In particular, in CHD patients with right ventricular dysfunction or increased pulmonary venous pressures, the impaired right heart causes elevated right atrial and ventricular pressures, which have direct consequences on venous circulation.

For example, in conditions like single ventricle physiology, the heart's ability to effectively separate the systemic and pulmonary circulations is impaired, leading to more venous blood flow to the right heart causing right heart failure, and backup of

blood in the systemic venous system, contributing to increased jugular venous pressures. As CVP increases, blood accumulates in the inferior vena cava (IVC), hepatic veins, and renal veins, resulting in venous congestion .[54]

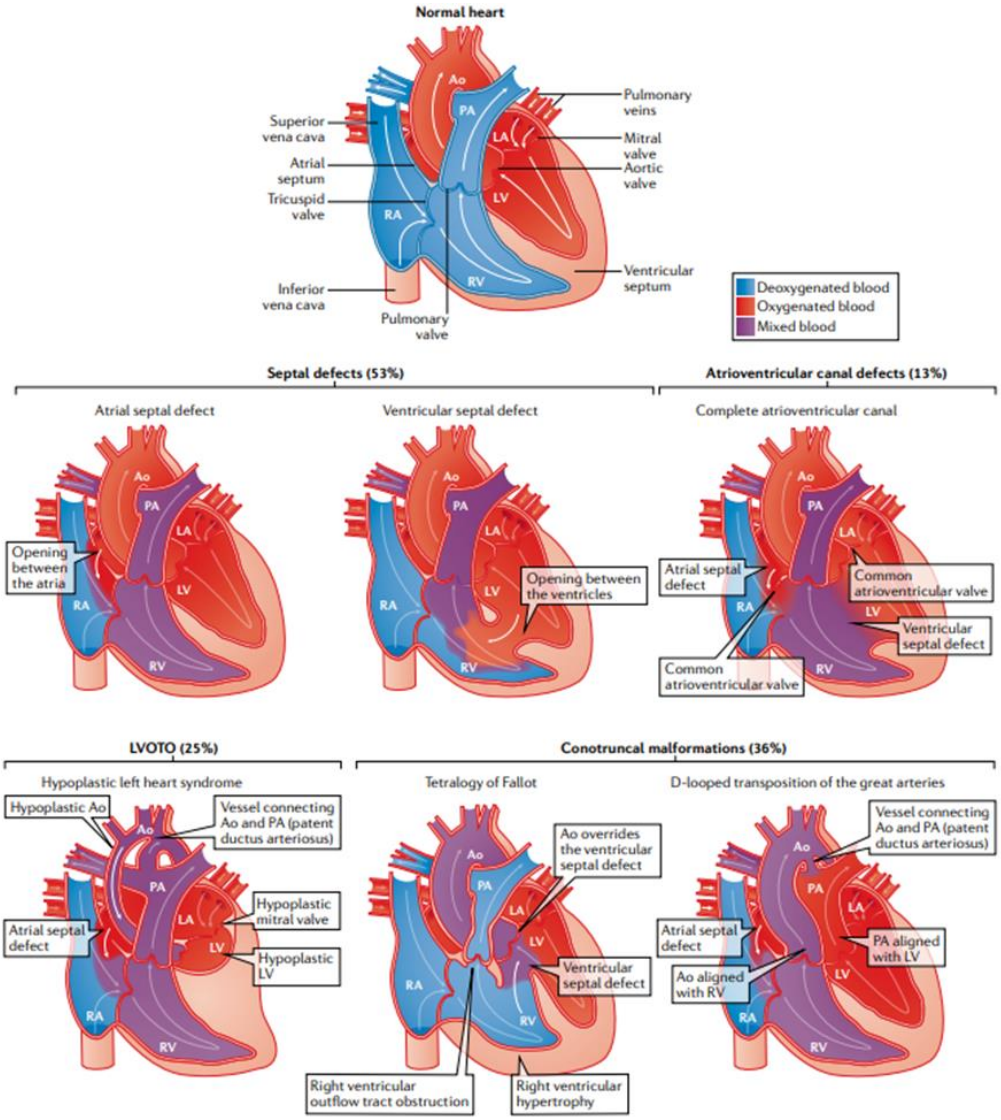


Figure 9: Common types of congenital heart disease[55]

Impaired Right-Sided Cardiac Function

Right ventricular failure leads to an elevation in CVP, which is reflected in the increased pressure in the IVC. The increased pressure in the IVC is propagated backward into the hepatic veins and kidneys. This increased pressure in the hepatic

veins causes hepatic congestion, resulting in hepatomegaly (enlargement of the liver), which is often observed in heart failure patients. Similarly, renal venous congestion impairs renal function, leading to fluid retention, decreased glomerular filtration rate (GFR), and worsening fluid overload [56,57] Venous congestion exacerbates organ dysfunction, further compromising the heart's ability to manage circulation effectively.

Venous Congestion in Complex CHD

In patients with complex conditions like univentricular physiology or transposition of the great arteries, the pathophysiology of venous congestion is even more intricate. For instance, in univentricular heart disease, the single ventricle must supply both systemic and pulmonary circulations. This leads to an increased volume to the right heart, causing it to be more prone to failure and venous congestion. The right heart, when unable to pump blood efficiently, causes systemic venous pressure to increase, leading to backflow into the hepatic and renal veins .[58]

Furthermore, shunts—either residual or at birth—create abnormal flow pathways between the systemic and pulmonary vascular systems. These shunts lead to more venous blood flowing to heart, exacerbating the pressure on the right atrium and ventricle, thus increasing venous congestion. The increased venous return further contributes to elevated CVP and systemic venous pressure, leading to the manifestation of organ congestion .[59] Shunt-related hemodynamic changes in patients with complex CHD lead to an increased workload on the heart, resulting in a cycle of exacerbated venous congestion and worsening heart failure symptoms.

Fluid Retention and Organ Dysfunction

Fluid overload is a hallmark of heart failure and is crucial in venous congestion. The kidneys, when exposed to increased venous pressures and decreased renal perfusion, initiate compensatory mechanisms aimed at preserving circulatory volume. This process involves activation of the renin-angiotensin-aldosterone system (RAAS), which causes the body to retain sodium and water. This compensatory fluid retention exacerbates venous congestion and causes further fluid buildup in organs such as the lungs (leading to pulmonary oedema), liver (leading to hepatomegaly), and peripheral tissues (leading to peripheral oedema and ascites).[60]

In CHD patients, the interaction between fluid overload and venous congestion is particularly pronounced. As fluid retention worsens, it leads to increased systemic vascular resistance and worsens the elevated venous pressures, creating a vicious cycle of fluid accumulation and worsening heart failure. Fluid overload contributes to both the pathophysiology and the clinical manifestations of venous congestion in CHD patients, such as peripheral oedema, ascites, and difficulty in respiratory function.[61]

The Role of Pulmonary Venous Congestion

While systemic venous congestion is often the most noticeable consequence of right heart dysfunction, pulmonary venous congestion is also a significant feature in some CHD patients. In cases where there is left heart dysfunction or elevated pulmonary vascular resistance, blood can back up into the pulmonary veins, causing pulmonary oedema and respiratory distress. In children with CHD who have elevated pulmonary pressures or residual left-to-right shunts, pulmonary venous congestion can significantly impair oxygenation and increase the risk of respiratory failure.[62]

Pulmonary venous congestion can further worsen the overall circulatory system's ability to manage venous return, contributing to systemic congestion.

The Impact of Shunts and Surgical Repair on Venous Congestion

Shunts, either inherent in the disease process (e.g., ASD, VSD) or created surgically (e.g., Fontan procedure), significantly influence the development of venous congestion. In conditions such as Fontan circulation, where a surgical procedure directs venous blood flow into the pulmonary arteries without a functional right ventricle, venous congestion can occur if the pulmonary vascular resistance increases or if there is inadequate venous drainage. The Fontan circulation is associated with increased systemic venous pressures, which can lead to elevated pressures in the IVC, hepatic veins, and other venous structures, contributing to long-term venous congestion.[63]

Venous Congestion in Paediatric Heart Failure

Paediatric heart failure, particularly in those with CHD, presents a unique challenge in the treatment of venous congestion. In infants and young children, physical signs of venous congestion, such as edema or hepatomegaly, may not be as pronounced as in adults, and the clinical presentation can be subtle or masked by other comorbidities. Furthermore, diagnostic tools such as central venous pressure monitoring can be invasive and difficult to implement in pediatric patients. Non-invasive methods, such as echocardiography, point-of-care ultrasound, and the use of the Inferior Vena Cava Collapsibility Index (IVC-CI) or VExUS (Venous Excess Ultrasound Score), have proven valuable in assessing fluid status and venous congestion in these patients .[64,65]

Traditional Methods for Assessing Venous Congestion

Several methods have been traditionally used to assess venous congestion in heart failure patients, including physical examination, central venous pressure (CVP) measurements, and laboratory markers. Physical examination remains the cornerstone of clinical evaluation, with signs including peripheral edema, jugular venous distention, ascites, and increased liver span serving as indicators of systemic venous congestion. However, these clinical signs can be absent in the initial stages of congestion and can be subjective in their assessment. Furthermore, they may not always correlate with the degree of venous overload without overt symptoms.

Central venous pressure (CVP) measurement is another common method used to estimate the level of right atrial pressure and venous congestion. While CVP provides useful information, its accuracy can be influenced by numerous factors, including positioning of the catheter, patient body position, and the presence of arrhythmias. Additionally, invasive CVP monitoring carries significant risks, such as infection, thrombosis, and the potential for increased mortality in critically ill cardiac patients. As a result, increased interest in less invasive alternatives that can provide real-time insights into venous congestion and fluid overload.

Fluid Overload in Heart Failure

Fluid overload is a critical complication in heart failure patients, contributing significantly to morbidity and mortality. Fluid retention, particularly in the venous circulation, is associated with elevated venous pressure, which leads to systemic congestion and impaired organ function. In paediatric patients with congenital heart defects, fluid overload is common due to the structural and functional abnormalities of the heart, which alter normal circulatory dynamics. Fluid overload can result from

volume overload, altered hemodynamic, or impaired myocardial function, and it exacerbates heart failure symptoms, including oedema, ascites, hepatomegaly, and renal dysfunction.[66,67]

In paediatric heart failure, especially in those with congenital heart anomalies, the mechanisms of venous congestion differ from those seen in adults. The unique hemodynamic abnormalities in CHD patients often include residual shunts, abnormal ventricular function, and right heart failure, which can complicate the clinical management of fluid overload. Therefore, the early detection of fluid overload and venous congestion becomes paramount to optimizing treatment strategies and minimizing adverse clinical outcomes.[68,69]

Pathophysiology of Fluid Overload in Congenital Heart Disease

Congenital heart disease comprises anatomical anomalies present from birth, many of which result in abnormal circulatory patterns. These can lead to volume overload or elevated pulmonary or systemic venous pressures. As venous congestion worsens, the elevated pressure leads to systemic fluid retention, particularly in the hepatic, renal, and portal veins, contributing to abdominal distention, impaired renal function, and other manifestations of fluid overload.[70] In response to increased venous pressure, the kidneys retain more sodium and water, leading to worsening congestion and further volume overload.[71] This vicious cycle can result in heart failure decompensation, organ failure, and poor clinical outcomes in paediatric CHD patients.

In spite of significance of fluid overload in the pathophysiology of heart failure, assessing the exact volume status and venous congestion in CHD patients presents a significant challenge. Traditional methods such as physical examination

and invasive measurements of central venous pressure (CVP) may be insufficient for detecting subtle or early-stage congestion[72,73] Therefore, accurate and non-invasive assessment tools are urgently needed to better manage fluid overload in this population.

Traditional Methods for Assessing Fluid Overload

The traditional assessment of fluid overload and venous congestion typically involves physical examination, CVP measurements, and laboratory markers of renal and hepatic dysfunction. Clinical signs of congestion, such as edema, ascites, and jugular venous distention, are useful but often lack sensitivity in detecting early fluid overload, particularly in pediatric patients.[74,75] These signs may also be subjective, depending on the clinician's experience, and may not be reliably present until congestion is severe.

Central venous pressure (CVP) has historically been a key measure for assessing venous congestion and fluid status. CVP is thought to reflect the pressure within the right atrium and, by extension, the systemic venous pressure. However, invasive CVP measurement carries several risks, including infection, thrombosis, and mechanical complications, and its reliability has been questioned in certain patient populations.[76] Moreover, CVP may not always correlate with clinical signs of fluid overload, especially in patients with complicated hemodynamics such as those with congenital heart disease, who may have abnormal venous pressures due to congenital defects, residual shunts, or other structural abnormalities.[77]

Thus, non-invasive techniques have gained prominence in the management of cardiac failure and fluid overload, particularly for pediatric patients. Among these, point-of-care ultrasound (POCUS) is an essential aid for assessing venous congestion,

offering real-time, bedside evaluation of fluid volume and venous pressures without the risks associated with invasive methods.

Point-of-Care Ultrasound in Fluid Overload Assessment

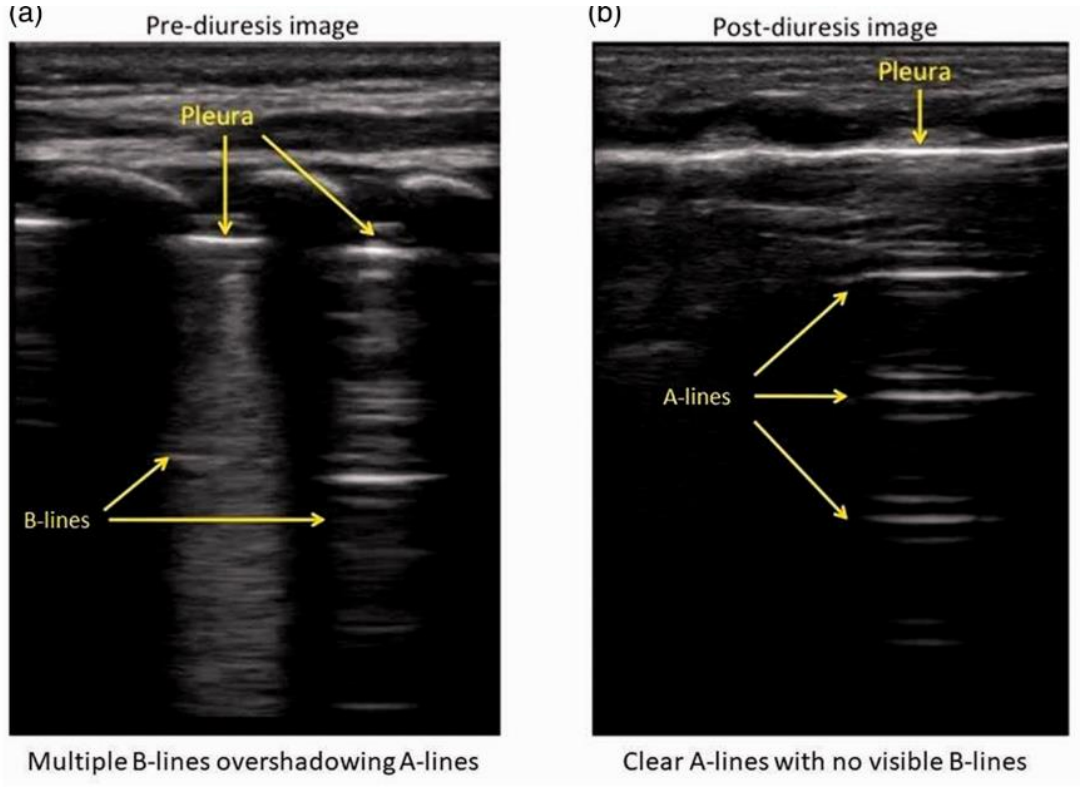


Figure 10:. Conventional bedside lung ultrasound for assessment of pulmonary edema in a 9-week-old baby.[78]

Point-of-care ultrasound (POCUS) has revolutionized the way healthcare providers evaluate fluid overload in real-time. POCUS is widely used in critical care settings to monitor fluid status and guide clinical judgement in cardiac failure. One of the primary ultrasound measurements for assessing venous congestion is the Inferior Vena Cava Collapsibility Index (IVC-CI), which relies on changes in the diameter with breathing cycle. [78]

Inferior Vena Cava Collapsibility Index (IVC-CI)

The IVC-CI is an accessible tool for evaluating fluid status in patients with cardiac failure. The principle behind the IVC-CI is that the diameter of the IVC changes with breathing cycle: during inspiration, a decrease in intrathoracic pressure causes the IVC to collapse, and the extent of this collapse shows a reciprocal relationship to right atrial pressure and jugular venous pressure. Studies have shown that a larger collapse (greater variation) indicates a lower central venous pressure, while a smaller collapse suggests increased venous pressure and fluid overload.[79,80]

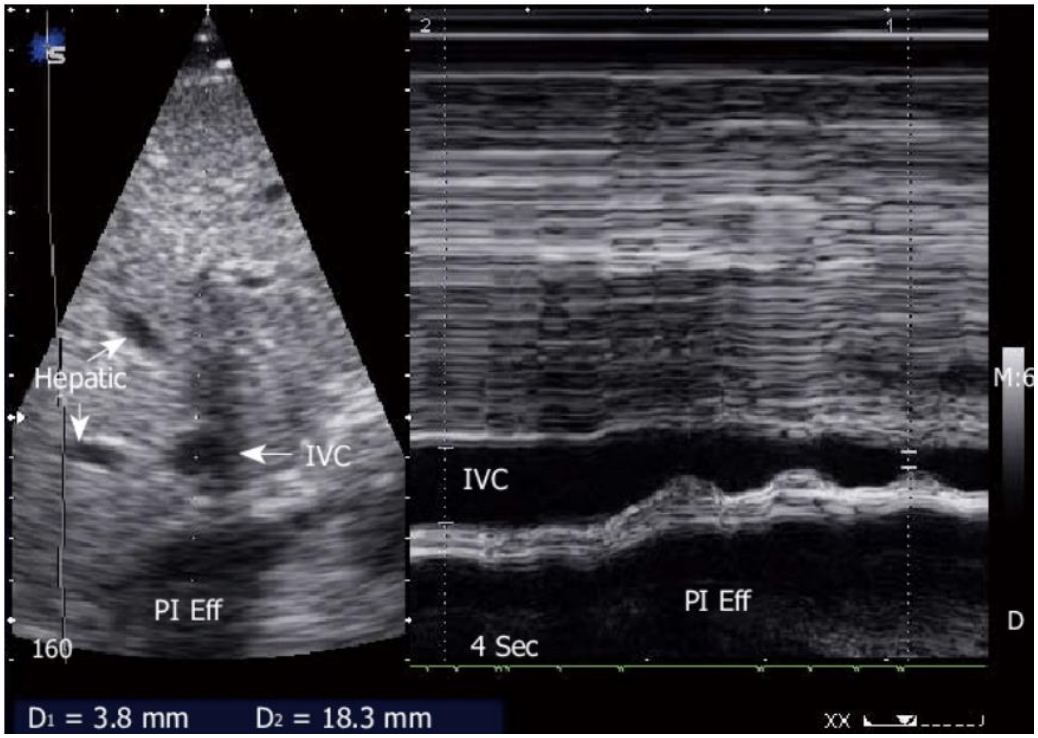


Figure 11: . IVC-CI[80]

The ultrasound image includes two panels[81,82]: the left (B-mode) shows the inferior vena cava (IVC) and hepatic veins, with a pleural effusion (PI Eff) and IVC diameters of 3.8 mm and 18.3 mm, likely for assessing collapsibility and volume status; the right (M-mode) illustrates IVC diameter changes during respiration, with

significant collapsibility (>50%) indicating hypovolemia and a non-collapsing IVC suggesting fluid overload or high right atrial pressure. IVC-CI > 40% suggests hypovolemia and the need for fluid resuscitation, while IVC-CI < 15% indicates volume overload or high right atrial pressure. In paediatric congenital heart disease, IVC-CI is a reliable marker of fluid status but may be inconsistent in certain cases. The IVC-CI is calculated by subtracting the end-expiratory from the end-inspiratory IVC diameter, dividing by the expiratory diameter, and multiplying by 100%. In this case, IVCCI = 79.2%, indicating hypovolemia.[83,84,85]

Venous Excess Ultrasound Score (VExUS)

The Venous Excess Ultrasound Score (VExUS) is a newer, more comprehensive ultrasound technique that evaluates not only the IVC but also Doppler flow patterns in the hepatic, renal, and portal veins to look for venous overload. The VExUS score quantifies changes in these veins that occur due to elevated central venous pressure, such as abnormal flow patterns, decreased flow velocities, and increased pulsatility.[86] As venous congestion worsens, these changes become more pronounced, allowing for a graded assessment of congestion severity.

The VExUS score has been shown to be highly sensitive in detecting venous congestion and has been correlated with clinical outcomes, including the severity of heart failure and fluid overload. Additionally, the VExUS score provides a more detailed picture of venous congestion than the IVC-CI alone, as it evaluates multiple venous structures and allows for the identification of early changes in venous flow.[87] This makes it a potentially valuable tool in paediatric patients with congenital heart disease, where abnormal venous dynamics are common.

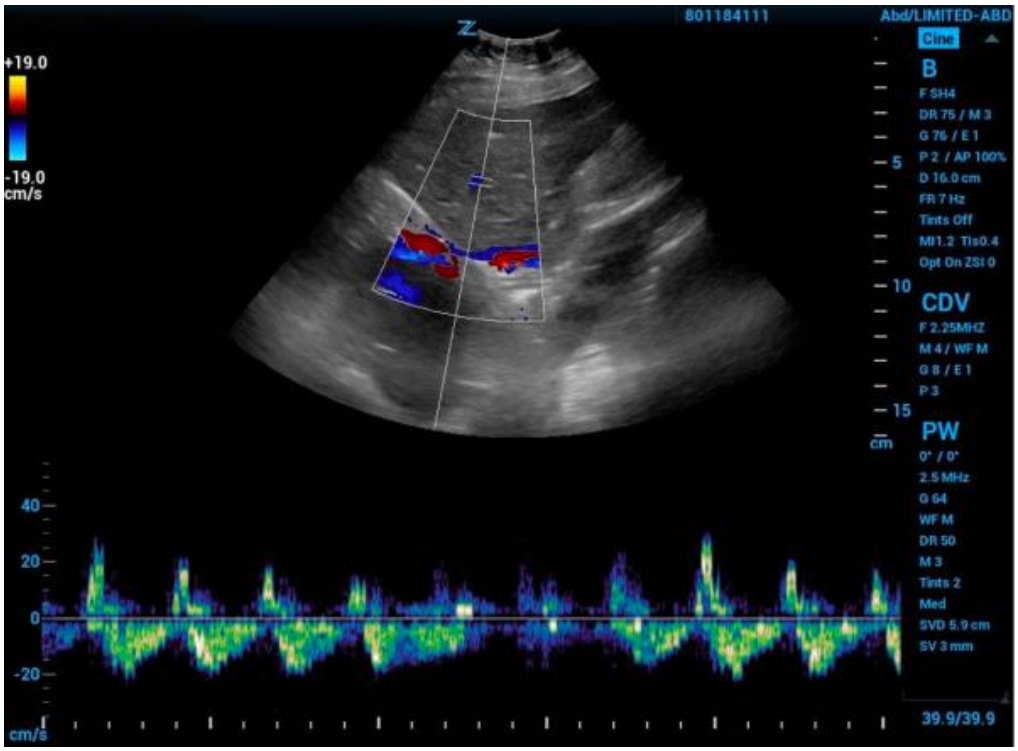


Figure 12: Hepatic vein Doppler with severe abnormality grade tracing[88]

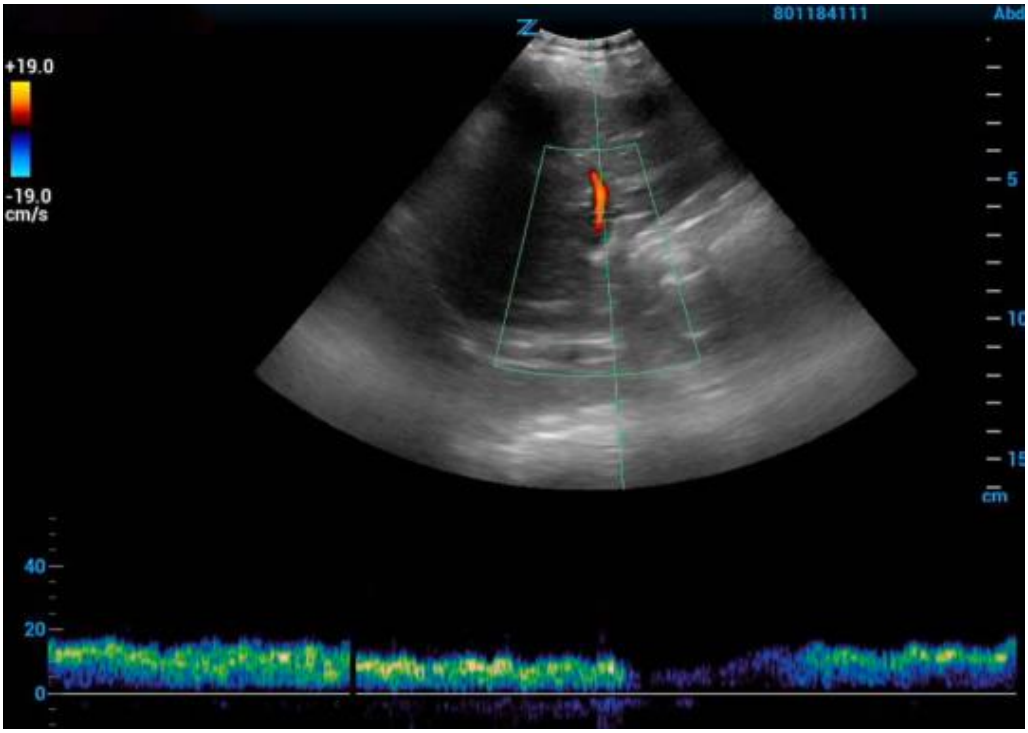


Figure 13: Portal vein doppler with normal grade tracing[88]

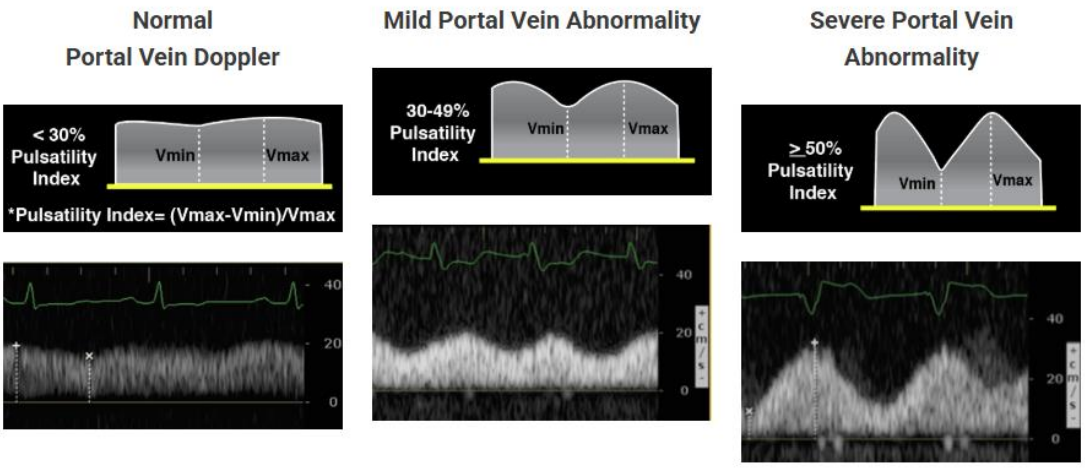


Figure 14: ***Portal Vein Ultrasound Doppler findings***[89]

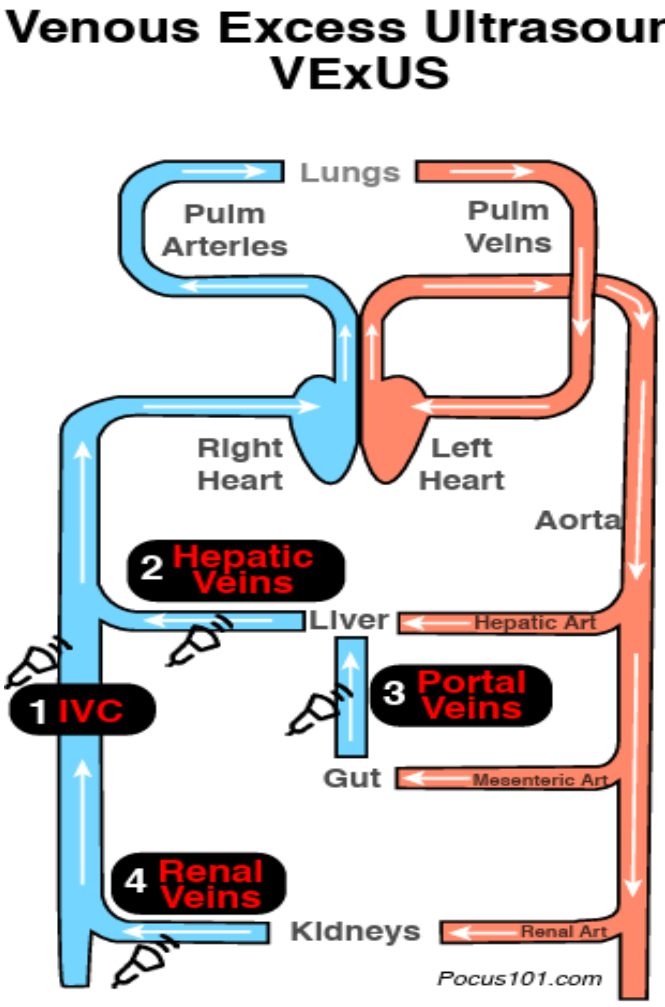


Figure 15: VExUS Score[89]

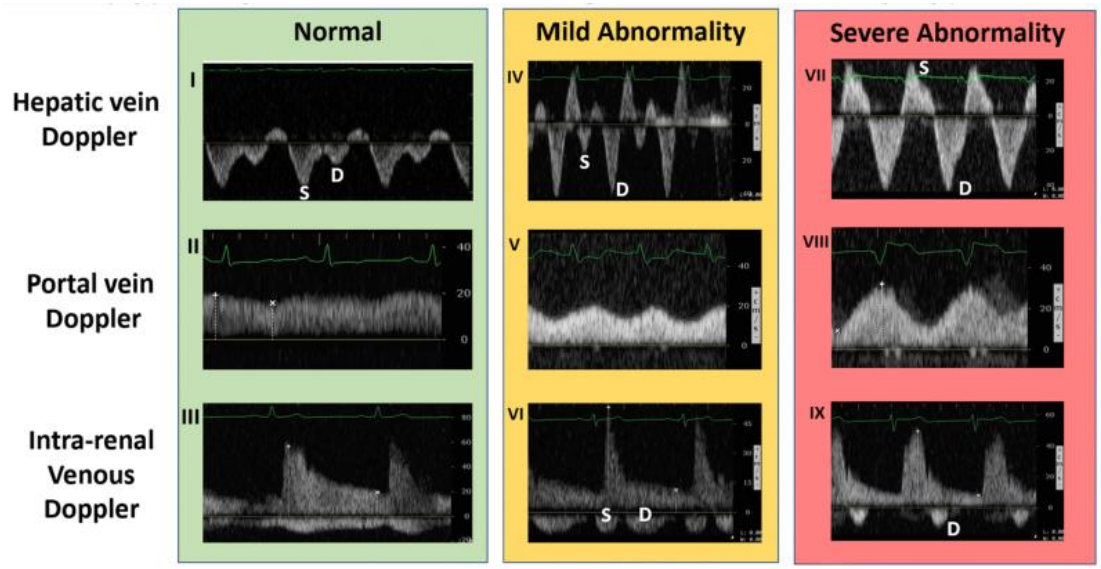


Figure 16: VExUS scoring system and interpretation[88]

The Modified Ross Heart Failure Classification[16]

The Modified Ross Heart Failure Classification is widely accepted for assessing the degree of heart failure (HF) in children, addressing the unique clinical manifestations such as refusal to feed, growth failure, and respiratory distress, which differ from adult presentations. This four-tier system categorizes HF from Class I (asymptomatic) to Class IV (severe symptoms even at rest), guiding therapy, monitoring progress, and standardizing research outcomes. Its age-appropriate design and simplicity make it a valuable resource in paediatric cardiology; however, it has limitations, including subjectivity and the potential for symptom overlap with other conditions. Clinically, it helps streamline treatment decisions, predict prognosis, and improve outcomes, as higher Ross classes correlate with poorer prognoses. Studies like those by Ross et al.[90] and subsequent guidelines, such as the Canadian Cardiovascular Society's recommendations, underscore its role in improving diagnostic precision and therapy. Despite its utility, further refinements and validation are necessary to optimize its application in diverse clinical settings.

Structure of the Classification

1. **Class I**:
    - Asymptomatic
    - Normal activity and feeding without any signs of fatigue, dyspnoea, or diaphoresis.
2. **Class II**:
    - Mild symptoms with exertion or feeding.
    - May exhibit mild tachypnea, sweating, or fatigue during physical activity or feeding.
3. **Class III**:
    - Marked limitation of activity.
    - Infants may exhibit difficulty feeding with poor weight gain.
    - Older children might experience significant fatigue, tachypnoea, and dyspnoea with mild exertion.
4. **Class IV**:
    - Severe symptoms at rest.
    - Severe feeding intolerance, recurrent hospitalizations, or profound fatigue with minimal activity.

The Modified Ross Classification is particularly helpful in paediatric cardiology for the following reasons:

- Guiding Therapy: It helps clinicians determine the urgency and type of treatment, ranging from diuretics and inotropes to advanced interventions like ventricular assist devices or transplantation.

- Monitoring Progress: Regular application allows for tracking disease progression or improvement following therapy.
- Standardization in Research: By providing a uniform framework, the classification facilitates multicenter studies and comparison of outcomes in pediatric HF research.

REVIEWED STUDIES

Denault AY, et al.[91] introduced the VExUS grading system in their study, "The Venous Excess Ultrasound (VExUS) Grading System: A Step-by-Step Tutorial," which aimed to provide clinicians with a systematic tool to identify venous congestion. The study outlined the technical aspects of measuring hepatic, portal, and renal vein Doppler flow patterns, grading venous congestion into clinically actionable categories. It emphasized the applicability of VExUS in diverse settings, including heart failure and critical care.

Beaubien-Souligny W et al.[92] validated the VExUS system in a prospective study of critically ill patients. Their findings demonstrated that the VExUS score, which incorporates Doppler ultrasound findings, was more predictive of renal dysfunction and systemic congestion than traditional markers like central venous pressure or physical examination findings. The study reinforced the role of VExUS in optimizing volume management and guiding clinical interventions.

Via G, Tavazzi G, and Price S examined the IVC collapsibility index for non-invasive monitoring of venous congestion in cardiac patients. They highlighted that while IVC collapsibility was a convenient bedside tool, its accuracy diminished in patients with intra-abdominal hypertension or altered venous anatomy. The authors

suggested combining the IVC collapsibility index with other advanced ultrasound techniques for better diagnostic precision.[93]

Simonovic D, Ristic AD, and Milic N compared the VExUS score and IVC collapsibility index in congenital heart disease patients presenting with heart failure. Their study concluded that the VExUS score, which integrates multiple Doppler flow patterns, was more effective in detecting systemic venous congestion in patients with complex hemodynamics, such as those with single-ventricle physiology or abnormal venous anatomy.[94]

Duchesne J, de la Hoz A, and Engelhardt T focused on systemic venous congestion using VExUS in patients with decompensated heart failure. They found that higher VExUS scores correlated strongly with renal dysfunction, elevated central venous pressure, and worse clinical outcomes. The study emphasized the importance of VExUS in stratifying risk and tailoring diuretic therapy in heart failure patients.[95]

Mackenzie DC, Noble VE, and Liteplo AS evaluated the role of IVC ultrasound in managing heart failure and guiding fluid therapy. The study showed that while IVC measurements provided a quick estimate of volume status, their diagnostic accuracy was influenced by factors such as patient positioning, respiratory mechanics, and operator expertise, necessitating supplemental tools like VExUS for improved reliability.[96]

Kalra PR, Guha K, and Haldar S conducted a comprehensive review of ultrasound modalities for assessing venous congestion in advanced heart failure. Their analysis highlighted the limitations of the IVC collapsibility index in detecting systemic congestion and suggested that Doppler-based tools like VExUS offered better diagnostic accuracy, particularly in patients with refractory symptoms.[97]

Beaubien-Souligny W, Rola P, and Haycock K explored the predictive utility of VExUS in detecting acute kidney injury (AKI) following cardiac surgery. The study demonstrated that abnormal Doppler flow patterns in the portal and renal veins were strongly associated with post-operative AKI, underscoring the role of VExUS in perioperative risk assessment.[98]

Rola P, Beaubien-Souligny W, and Haycock K conducted a real-world study on the use of VExUS in critically ill patients. They demonstrated that VExUS provided superior prognostic insights compared to conventional methods, aiding clinicians in early detection of venous congestion and improving individualized treatment strategies.[99]

Nagueh SF, Smiseth OA, and Appleton CP investigated the correlation between IVC collapsibility and right atrial pressure in heart failure patients. While their findings confirmed that IVC collapsibility was a reliable non-invasive marker under controlled conditions, they also highlighted its reduced accuracy in mechanically ventilated patients and those with increased intra-abdominal pressures, suggesting the need for complementary methods like VExUS.[100]

Cohen S, Adler L, and Davidson K studied the role of ultrasound in assessing venous congestion in pediatric congenital heart disease patients. They reported that the IVC collapsibility index often failed in children with complex venous anatomy, whereas VExUS offered more comprehensive and reliable assessments of systemic venous congestion.[101]

Shah S, Agarwal R, and Sharma V investigated the use of VExUS and IVC collapsibility in patients with HFpEF. Their study revealed that VExUS was more effective in identifying subclinical venous congestion and predicting hospitalizations,

particularly in patients with preserved ejection fraction, where traditional tools often underperformed.[102]

Ibrahim K, Sabri A, and Patel V studied the impact of venous congestion on outcomes in patients with Fontan physiology, demonstrating that VExUS outperformed IVC collapsibility in detecting systemic venous congestion. Their findings underscored the importance of incorporating VExUS into routine follow-up of Fontan patients to prevent long-term complications like hepatic dysfunction.[103]

Chen J, Rao R, and Matta A compared ultrasound-based measures of venous congestion in pediatric congenital heart disease patients. The study highlighted the limitations of IVC collapsibility in this population and emphasized the superior diagnostic accuracy of VExUS in detecting elevated central venous pressure and guiding clinical decisions.[104]

Barjaktarevic I, Franke R, and Agarwal S evaluated the utility of IVC ultrasound in assessing hemodynamic congestion in critically ill patients. They found that while IVC collapsibility could reliably indicate fluid responsiveness, it was insufficient for detecting systemic venous congestion, particularly in mechanically ventilated patients.[105]

Nadim MK, Forni LG, and Bihorac A explored the relationship between venous congestion and renal outcomes in patients with HFrEF. Their findings demonstrated that higher VExUS scores were strongly predictive of worsening renal function, highlighting its role in optimizing fluid management strategies.[106]

Gammelgaard SK, Tirosh Y, and Guo L used a multimodal ultrasound approach to assess venous congestion in heart failure patients. By combining VExUS, IVC collapsibility, and echocardiographic parameters, they showed that the integration of these modalities improved diagnostic accuracy, with VExUS emerging as the most sensitive tool for detecting systemic congestion.[107]

Ahmed M, Siddiqui S, and Patel K evaluated the impact of VExUS-guided fluid management on clinical outcomes in heart failure patients. Their study demonstrated that using VExUS to guide diuretic therapy significantly reduced complications related to fluid overload compared to traditional methods relying on IVC measurements alone.[108]

Kulkarni AV, Sharma V, and Iyer P assessed venous congestion in complex congenital heart disease patients using advanced ultrasound techniques. They highlighted that VExUS was superior to IVC collapsibility in detecting venous congestion in patients with unique hemodynamic challenges, such as those with single-ventricle physiology or Fontan circulation.[109]

Goldberg R, Cohen J, and Mahajan P explored the prognostic value of venous ultrasound in patients with right heart failure. They found that VExUS was more strongly associated with adverse outcomes than IVC collapsibility, emphasizing its role in guiding treatment decisions and improving patient prognosis.[110]

MATERIALS AND METHODS

Source of Data: Children with Congenital Heart Disease from 1 months to 17 years of age admitted to Paediatric ICU and wards in KLE Dr. Prabhakar Kore hospital, Belagavi.

Study Design: A cross-sectional, single-centre study.

Study period: One-year study period from August 2023 to September 2024

Sample Size:

$n = Z^2_{\alpha/2}\, S_E(100\text{-}S_E)/ d^2$

where S_E-sensitivity, d-error

Considering $\alpha = 0.05$, $Z\alpha/2 = 1.96$, sensitivity=96%, d = 5% from previous study.

Substituting the values in the above equation:

$n = (1.96)^2 \times 96(100\text{-}96)/5^2$

$n \sim 60$

Hence, sample size (n) is **60 nos**.

Sampling technique: Consecutive sampling

Inclusion Criteria:

All children from 1 months to 17 years of age admitted to Paediatric ICU and wards in KLE Dr. Prabhakar Kore hospital, Belagavi, with congenital heart disease presenting with heart failure.

Exclusion Criteria:

1. Inadequate window
2. IVC Thrombus
3. Known case of cirrhosis with portal hypertension
4. Known case of acute or chronic renal failure

Study Procedures: Children aged 1 months to 17 years with congenital heart disease and heart failure admitted to the PICU or wards at KLE Dr. Prabhakar Kore Hospital, Belagavi, were enrolled. The child if able to understand or the parent or guardian were briefed about the study. After their approval written informed consent was taken from parents of children less than 7 years, verbal consent from children between 7-12 years with written informed consent from their parents. Written informed consent was taken from parents and their children above 12 years. Detailed history and duration of symptoms were noted. Blood investigations, Chest Xray and Echocardiography were done. Echocardiography was done to confirm the diagnosis of congenital heart disease presenting in heart failure. Administration of intravenous fluids and diuretics were documented. Patient characteristics and clinical diagnosis were entered in a structured proforma. Patients were classified clinically according to modified Ross classification for assessing the severity of heart failure.

Modified Ross heart failure classification for children is classified as:[90]

Class I: Asymptomatic

Class II: Mild tachypnea or diaphoresis with feeding in infants; Dyspnea on exertion in older children

Class III: Marked tachypnea or diaphoresis with feeding in infants and prolonged feeding times with growth failure; marked dyspnea on exertion in older children

Class IV: Tachypnea, retractions, grunting or diaphoresis at rest.

Bedside ultrasonography was done using Mindray machine using curvilinear abdominal probe of 8-3 MHz by a single radiologist trained in POCUS who was blinded regarding the diagnosis of the patient. Patient is positioned in supine with head end elevated between 0-30 degrees. IVC is visualized about 2 cm caudal to the entry of hepatic veins. Respiratory variations of IVC were noted. IVCCI is calculated as: IVCCI=(IVC expiratory diameter-IVC inspiratory diameter)/IVC expiratory diameter x 100. IVCCI was categorized into 3 based on literature: IVCCI<=15% systemic venous congestion, 16-40% normal, >40%- intravascular depletion.[111] If the IVC is dilated, more than age specific references[112,113], the rest of the protocol was followed. Any one hepatic vein can be viewed in the anterior right hypochondrium. Physiologic waveform has two waves- Systolic (S) >diastolic(D) wave, mildly abnormal- S<D, severely abnormal-reversal of flow of S wave. Portal vein is next visualized in the right mid -posterior axillary line over liver. Portal vein pulsatility index (PI) calculated as: PI=(Vmax-Vmin)/Vmax. Physiologic tracing has minimal variability with portal vein pulsatility index<30%, mild abnormality is pulsatility 30-49%, and severe is >50%. In this study renal vein doppler is not done which is originally defined in VExUS score. With the above components, VExUS score is interpreted as: grade 0- IVC diameter normal, no hepatic/portal vein abnormality; grade1- dilated IVC with normal or mild abnormal doppler patterns; grade2- Dilated IVC with severe flow abnormalities in at least one doppler pattern; grade3- Dilated IVC with severe flow abnormalities in multiple doppler patterns.[114]

Statistical Methods:

A descriptive analysis was performed using mean and standard deviation for quantitative variables, frequency, and proportion for categorical variables. Non-normally distributed quantitative variables were recapitulated by median and

interquartile range (IQR). Data visualization done through graphical representations like bar diagram, pie diagram and box plots. All Quantitative variables underwent normal distribution within each category of explanatory variable by using visual examination of histograms and normality Q-Q plots. Shapiro- wilk test was employed to assess normal distribution. Shapiro wilk test p value of >0.05 was indicative of normal distribution. Categorical outcomes were compared across study groups using Chi square test or Fisher's Exact test (Fisher's exact test was employed when the total sample size was < 20 or when the expected number in any cell is < 5). For normally distributed Quantitative parameters the mean values were compared between groups using statistical tests, with p-value < 0.05 considered statistically significant. Statistical analysis was performed using IBM SPSS version 22.

Ethical clearance: The study was granted ethical clearance by institutional ethics committee.

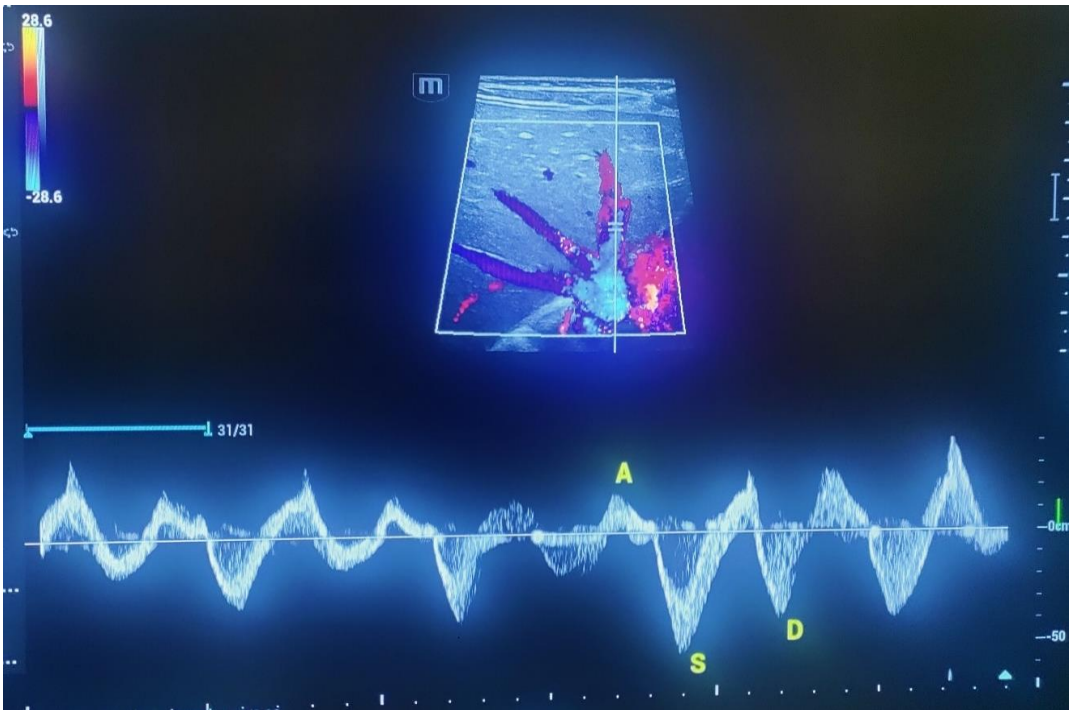


Figure 17: Normal hepatic flow doppler image

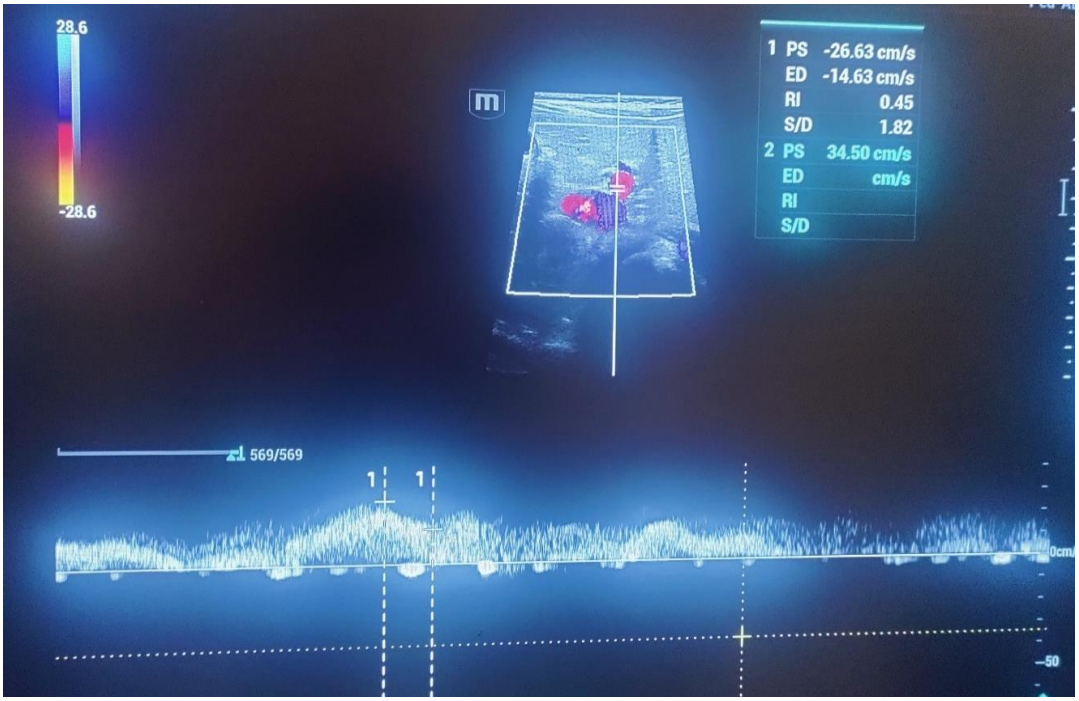


Figure 18: Measurement of portal vein pulsatility index. Mild abnormality seen in portal venous Doppler

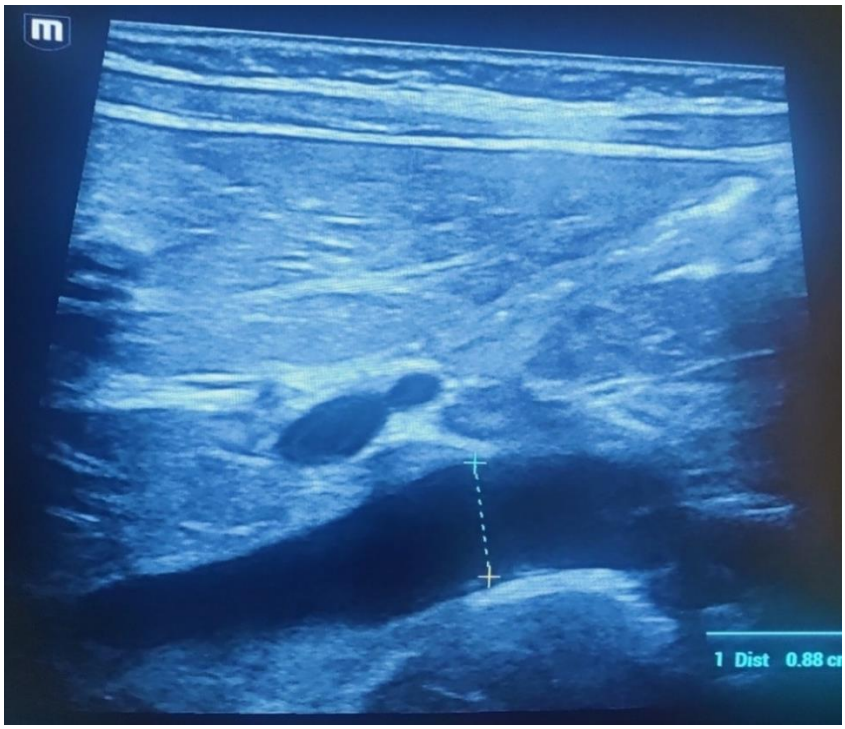


Figure 19: Measurement of IVC diameter

Figure 20: Measurement of IVC diameter using bedside ultrasound machine curvilinear probe

Figure 21: Bedside Mindray machine used for pocus assessment

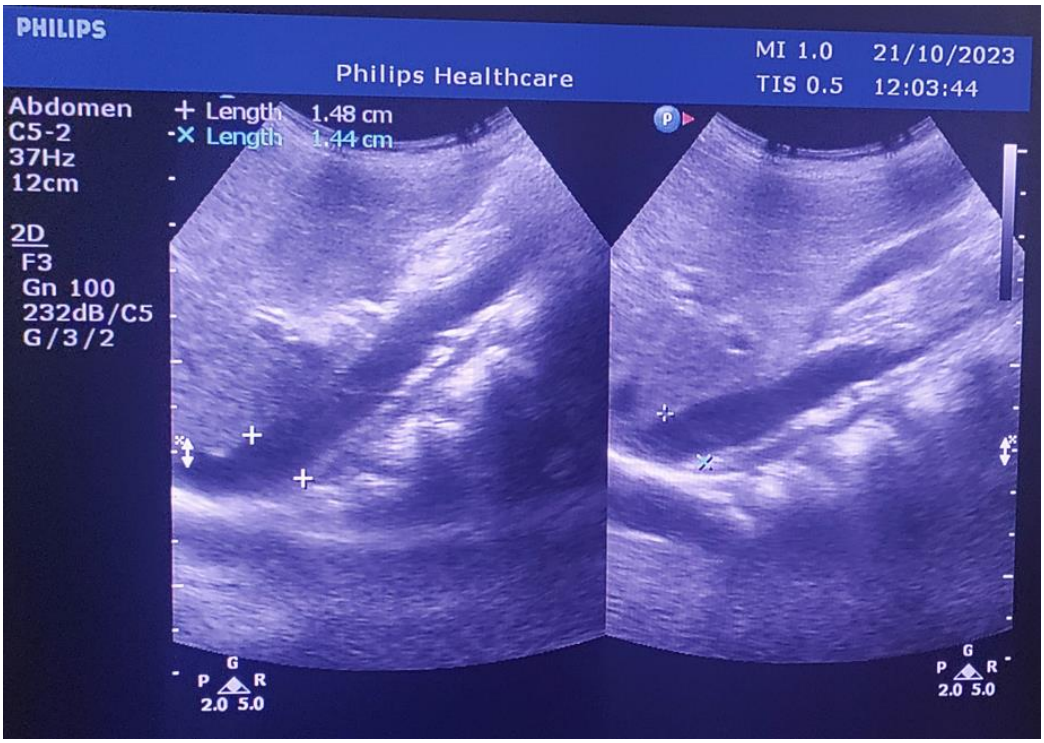


Figure 22: Measurement of ivc expiratory and inspiratory diameter using bedside ultrasound machine

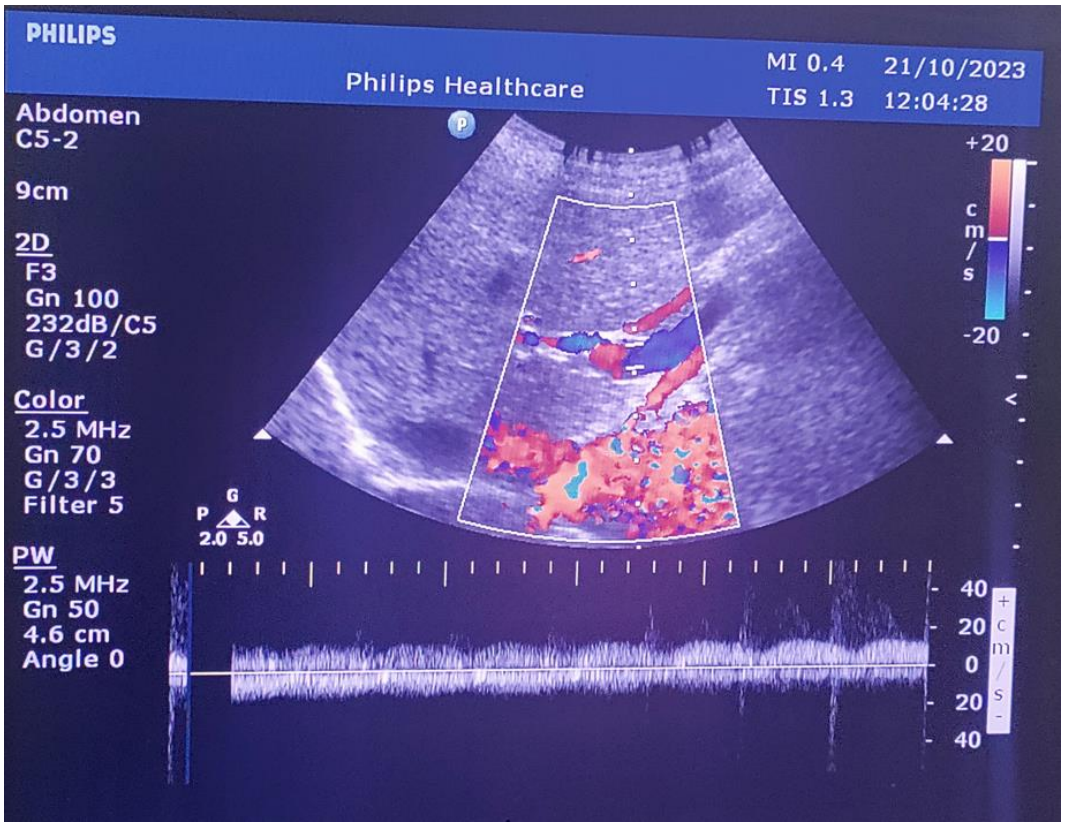


Figure 23: Normal portal vein Pulsatility index

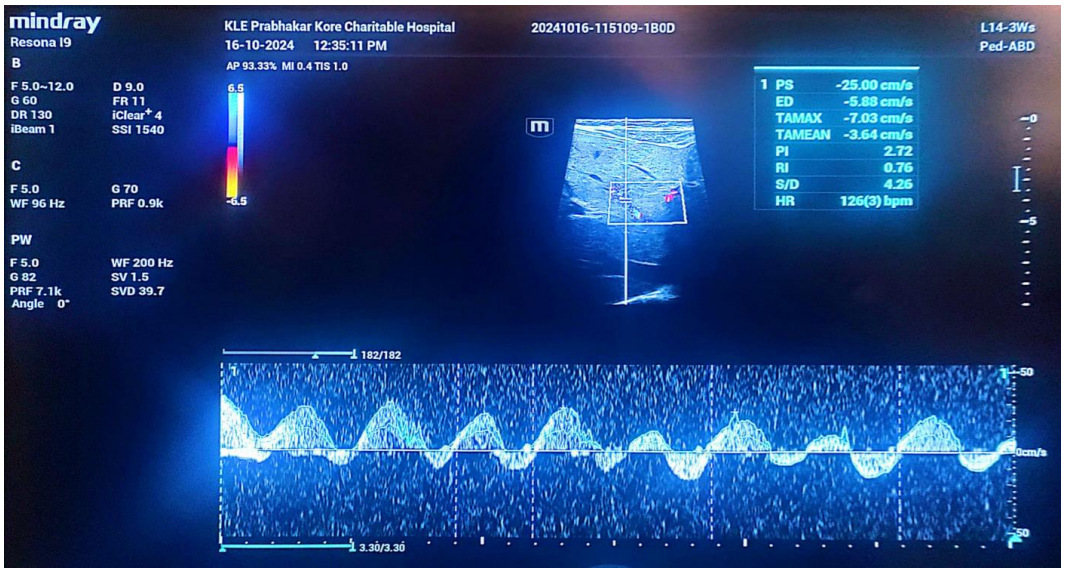


Figure 24: Abnormal hepatic venous flow

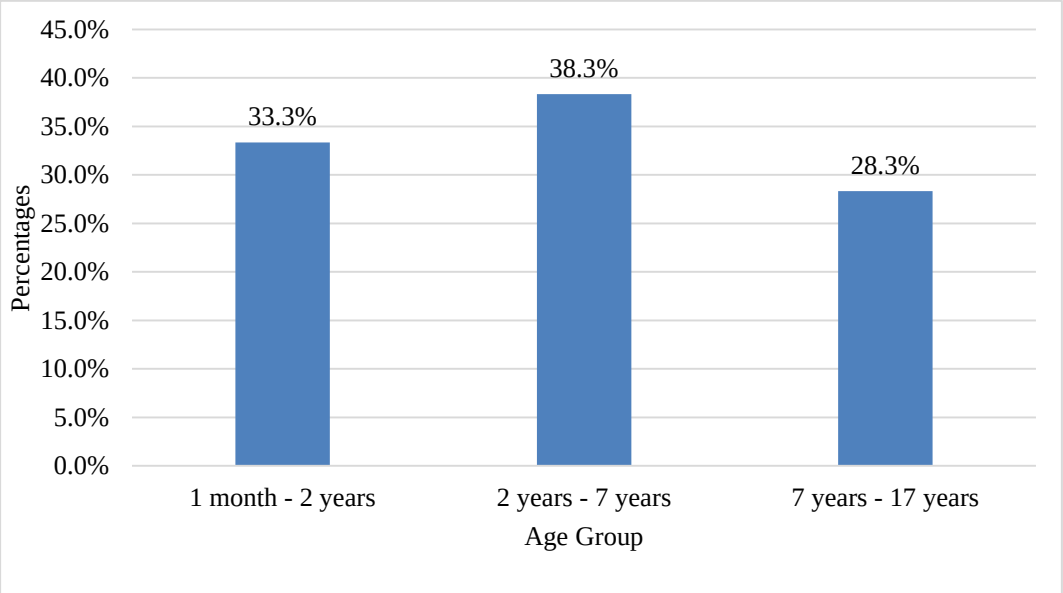
RESULTS

Descriptive analysis of age group in the study population

Table 1: Descriptive analysis of age group in the study population (N=60)

Age Group	Frequency	Percentages
1 month - 2 years	20	33.3%
2 years - 7 years	23	38.3%
7 years - 17 years	17	28.3%

***Figure 25*: Bar chart of age group in the study population (N=60)**



The table presents the distribution of the study population (N=60) based on age group and gender. The age groups are categorized as follows: 1 month - 2 years with 20 patients (33.3%), 2 years - 7 years with 23 patients (38.3%), and 7 years to 17 years with 17 patients (28.3%). These age groups provide insight into the demographic spread of children with congenital heart disease presenting with heart failure.

Descriptive analysis of age distribution in the study population

Table 2: Descriptive analysis of age distribution (N=60)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C. I	
					Lower	Upper
Age	5.42 ± 4.7	4.5	0.1	17.0	4.2	6.6

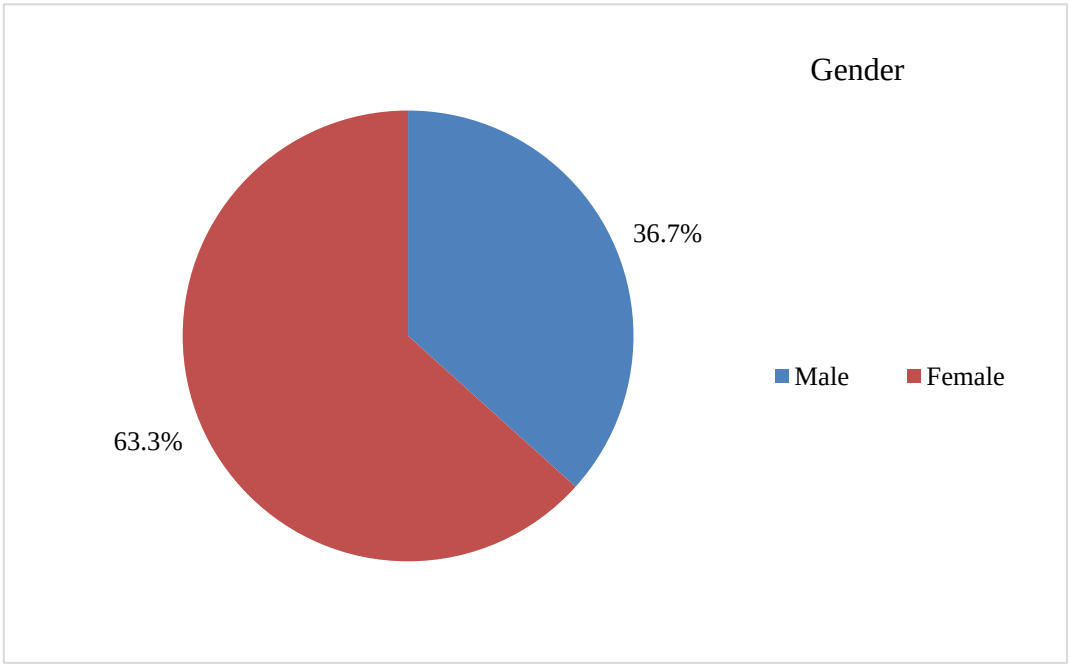
The table provides a detailed breakdown of the age distribution in the study population consisting of 60 paediatric patients with congenital heart disease presenting with heart failure. The table presents the mean age (5.42 ± 4.7 years) and standard deviation (SD), indicating the average age and variability of ages within the cohort. The median age (4.5 years) provides the middle value, where half of the patients are younger and half are older. The minimum age (0.1 years) represents the youngest participant, while the maximum age (17.0 years) indicates the oldest participant in the cohort.

Descriptive analysis of Gender in the study population

***Table 3*: Descriptive analysis of Gender in the study population (N=60)**

Gender		
Male	22	36.7%
Female	38	63.3%

Figure 26: Pie chart of gender in the study population (N=60)



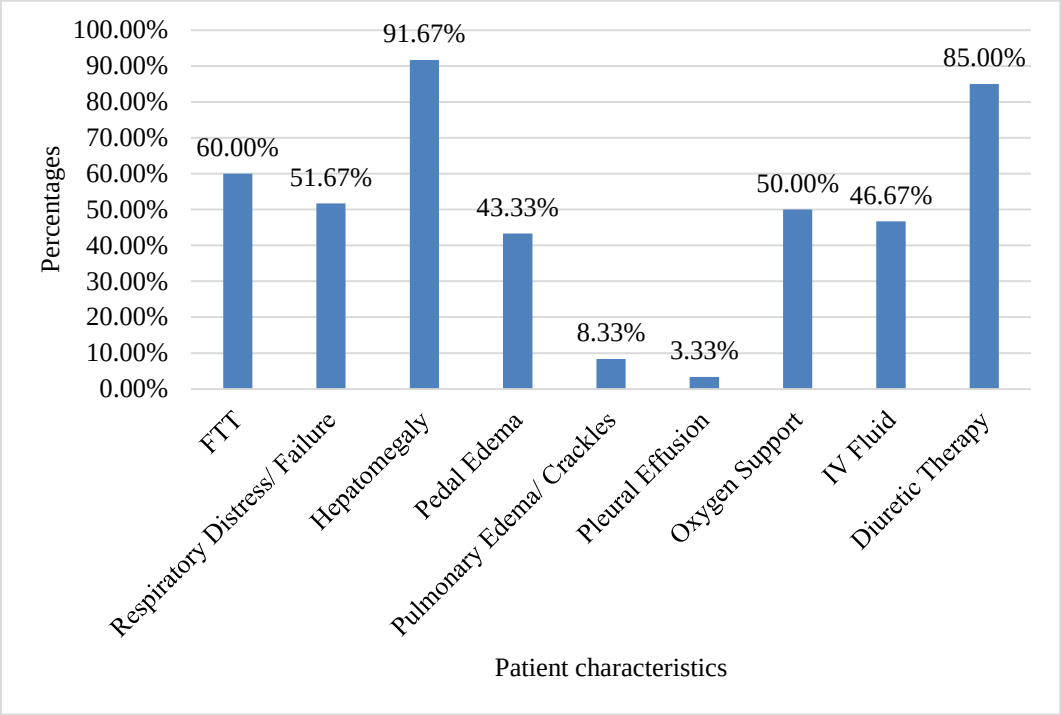
The gender distribution shows that 22 male patients (36.7%) and 38 female patients (63.3%) participated in the study. The pie chart illustrates that the female population is significantly larger than the male population, comprising nearly two-thirds of the total.

Descriptive analysis of patient characteristics in the study population

***Table 4*: Descriptive analysis of patient characteristics in the study population (N=60)**

Parameters	Frequency	Percentages
FTT		
Yes	36	60.00%
No	24	40.00%
Respiratory Distress/ Failure		
Yes	31	51.67%
No	29	48.33%
Hepatomegaly		
Yes	55	91.67%
No	5	8.33%
Pedal Edema		
Yes	26	43.33%
No	34	56.67%
Pulmonary Edema/ Crackles		
Yes	5	8.33%
No	55	91.67%
Pleural Effusion		
Yes	2	3.33%
No	58	96.67%
Oxygen Support		
Yes	30	50.00%
No	30	50.00%
IV Fluid		
Yes	28	46.67%
No	32	53.33%
Diuretic Therapy		
Yes	51	85.00%
No	9	15.00%

Figure 27: Bar chart of patient characteristics in the study population (N=60)



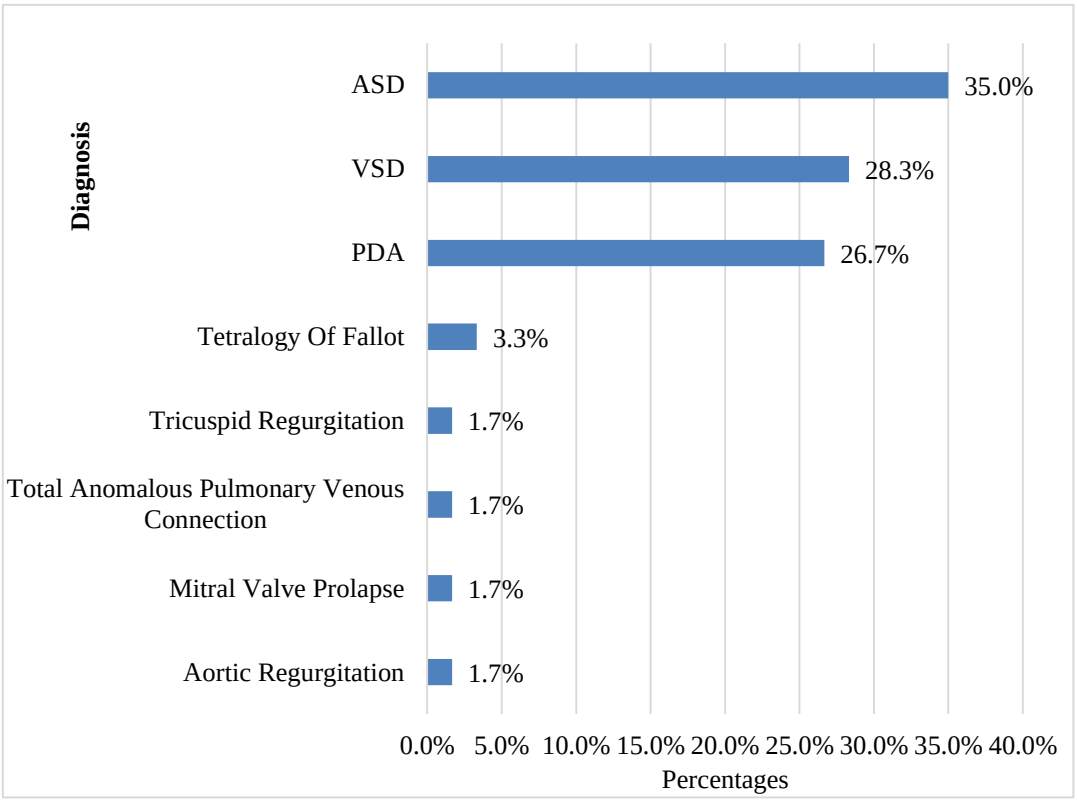
The table outlines various clinical characteristics of the study population, with data for 60 paediatric patients diagnosed with congenital heart disease and heart failure. Failure to Thrive (FTT) is observed in 36 patients (60.00%), indicating a common concern in the cohort. Respiratory Distress/Failure is present in 31 patients (51.67%), highlighting respiratory involvement in more than half of the patients. Hepatomegaly is observed in 55 patients (91.67%), a frequent finding in venous congestion. Pedal Edema is seen in 26 patients (43.33%), while Pulmonary Edema/Crackles is present in only 5 patients (8.33%). Pleural Effusion is noted in 2 patients (3.33%). Regarding treatments, Oxygen Support is used in 30 patients (50.00%), while IV Fluid administration is required in 28 patients (46.67%). The majority of patients, 51 (85.00%), receive Diuretic Therapy, suggesting its importance in managing venous congestion. The distribution suggests that hepatomegaly and diuretic therapy are the most common features, while pleural effusion is the least frequently observed characteristic.

Descriptive analysis of diagnosis in the study population

***Table 5*: Descriptive analysis of diagnosis in the study population (N=60)**

Diagnosis	Frequency	Percentages
ASD	21	35.00%
VSD	17	28.3%
PDA	16	26.7%
Tetralogy of Fallot	2	3.3%
Aortic Regurgitation	1	1.7%
Mitral Valve Prolapse	1	1.7%
Total Anomalous Pulmonary Venous Connection	1	1.7%
Tricuspid Regurgitation	1	1.7%

Figure 28: Bar chart of diagnosis in the study population (N=60)



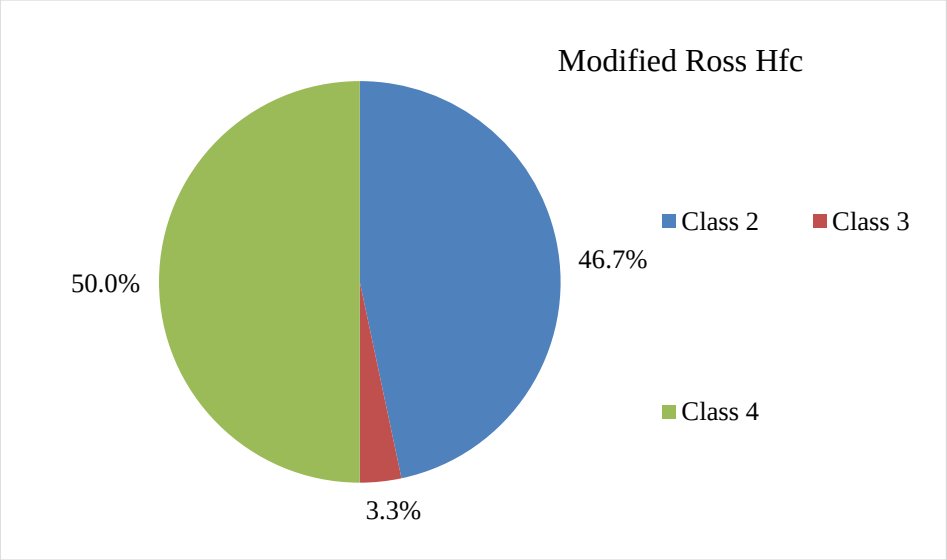
The table and bar chart illustrate the distribution of congenital heart disease diagnoses among a study population of 60 pediatric patients. The most common conditions are Atrial Septal Defect (ASD) in 21 patients (35.0%), Ventricular Septal Defect (VSD) in 17 patients (28.3%), and Patent Ductus Arteriosus (PDA) in 16 patients (26.7%). Less frequent diagnoses include Tetralogy of Fallot in 2 patients (3.3%) and Aortic Regurgitation, Mitral Valve Prolapse, Total Anomalous Pulmonary Venous Connection, and Tricuspid Regurgitation, each affecting 1 patient (1.7%). Both the table and chart emphasize that ASD, VSD, and PDA are the most prevalent congenital heart defects in this cohort.

Descriptive analysis of Modified Ross Heart failure Classification in the study population

Table 6: Descriptive analysis of Modified Ross HFC in the study population

Parameter	Frequency	Percentages
Class 2	28	46.7%
Class 3	2	3.3%
Class 4	30	50.00%

Figure 29: Pie chart of modified ross HFC in the study population (N=60)



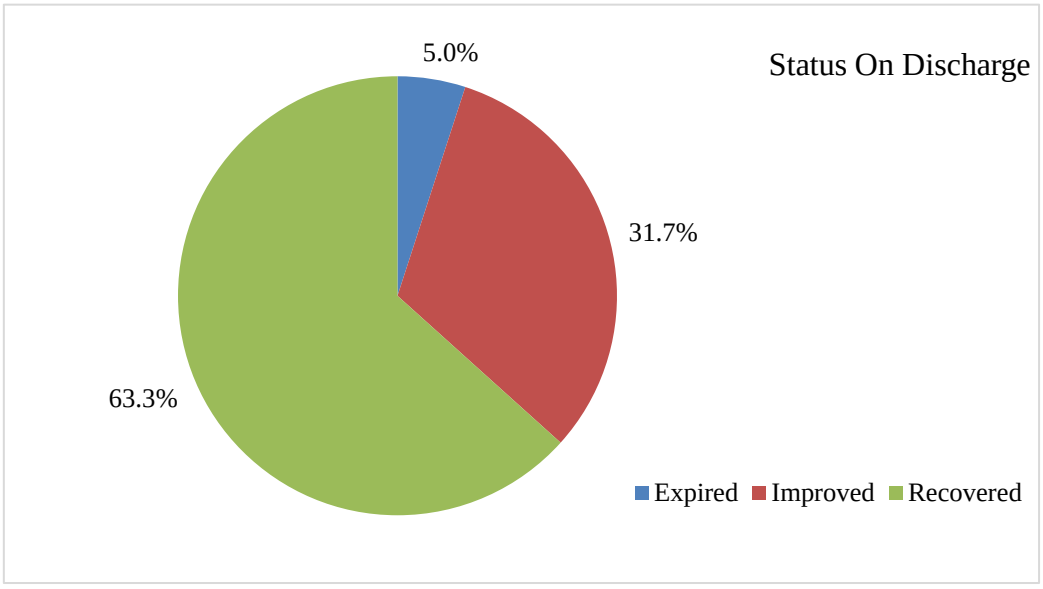
The table and graph present a descriptive analysis of the Modified Ross Heart Failure Classification (HFC) in a study population of 60 pediatric patients, categorizing them based on heart failure severity. The majority of patients, 30 (50.0%), fall into Class 4, indicating severe heart failure with significant clinical compromise. Class 2, representing mild heart failure with symptoms like tachypnea or mild feeding difficulties, includes 28 patients (46.7%). Only 2 patients (3.3%) are classified as Class 3. This distribution highlights that most patients experience severe heart failure (Class 4), with nearly half in a moderate condition (Class 2) and very few in Class 3.

Descriptive analysis of status on discharge in the study population

***Table 7*: Descriptive analysis of status on discharge in the study population (N=60)**

Status on Discharge	Frequency	Percentages
Expired	3	5.00%
Improved	19	31.7%
Recovered	38	63.3%

Figure 30: Pie chart of status on discharge in the study population (N=60)



The table presents the distribution of patient status upon discharge from the study, comprising a total of 60 paediatric patients with congenital heart disease and heart failure. The Status on Discharge is categorized as follows: Expired in 3 patients (5.00%), indicating those who did not survive; Improved in 19 patients (31.7%), reflecting those who showed some clinical improvement but did not fully recover; and Recovered in 38 patients (63.3%), indicating patients who returned stable. The graph majority of patients recovered, while a smaller portion showed improvement, and a minimal percentage did not survive.

Descriptive analysis of IVC diameter in the study population

***Table 8*: Descriptive analysis of IVC diameter in the study population (N=60)**

Parameter	Mean ± SD	Minimum	Maximum	Median	95% C.I	
					Lower	Upper
IVC Exp Diameter (mm)	8.25 ± 2.79	3.7	14.8	8.3	7.5	9.0
IVC Insp Diameter (mm)	7 ± 2.52	2.2	14.4	7.0	6.4	7.7

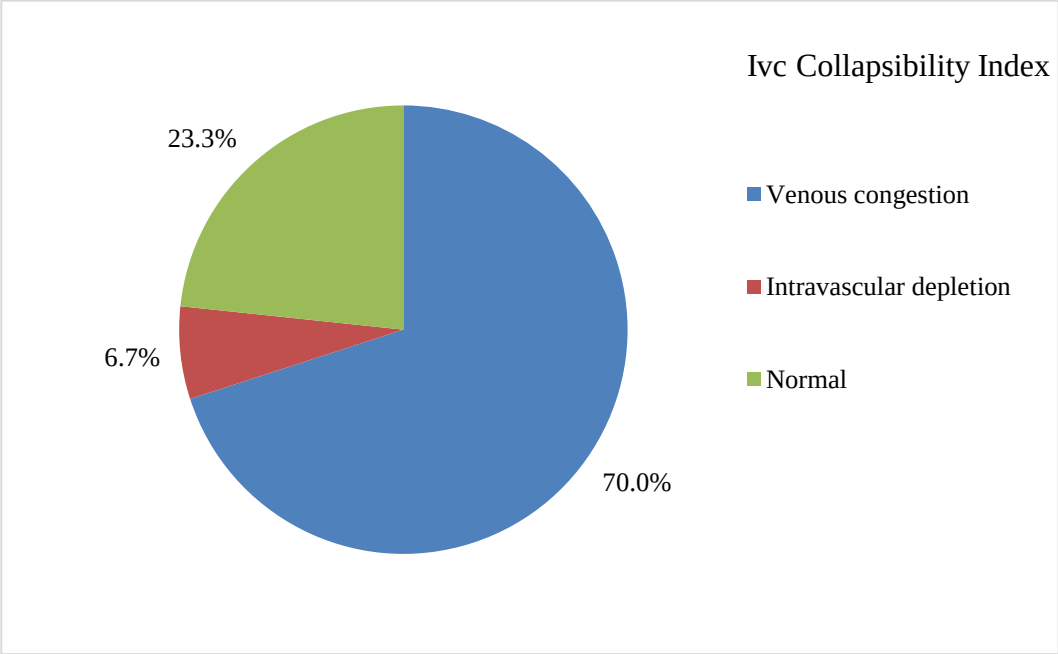
The table provides the descriptive statistics for the expiratory and inspiratory diameters of the inferior vena cava (IVC) in the study population. The IVC expiratory diameter has a mean of 8.25 ± 2.79 mm, with a median of 8.3 mm, indicating the central tendency of the measurements. The range of measurements spans from a minimum of 3.7 mm to a maximum of 14.8 mm, highlighting the variability in IVC diameters among patients. The 95% confidence interval (C.I.) for the expiratory diameter is 7.5 to 9.0 mm, providing an estimate of precision for the mean value. Similarly, for the IVC inspiratory diameter, the mean is 7 ± 2.52 mm, with a median of 7.0 mm, a minimum of 2.2 mm, and a maximum of 14.4 mm.

Descriptive analysis of venous Congestion by IVC Collapsibility Index in the study population

***Table 9*: Descriptive analysis of venous congestion by IVC Collapsibility Index in the study population (N=60)**

Methods	Frequency	Percentages
Ivc Collapsibility Index		
Venous Congestion	42	70.00%
Intravascular depletion	4	6.7%
Normal	14	23.3%

Figure 31: Pie chart of venous congestion by IVCCI index in the study population (N=60)



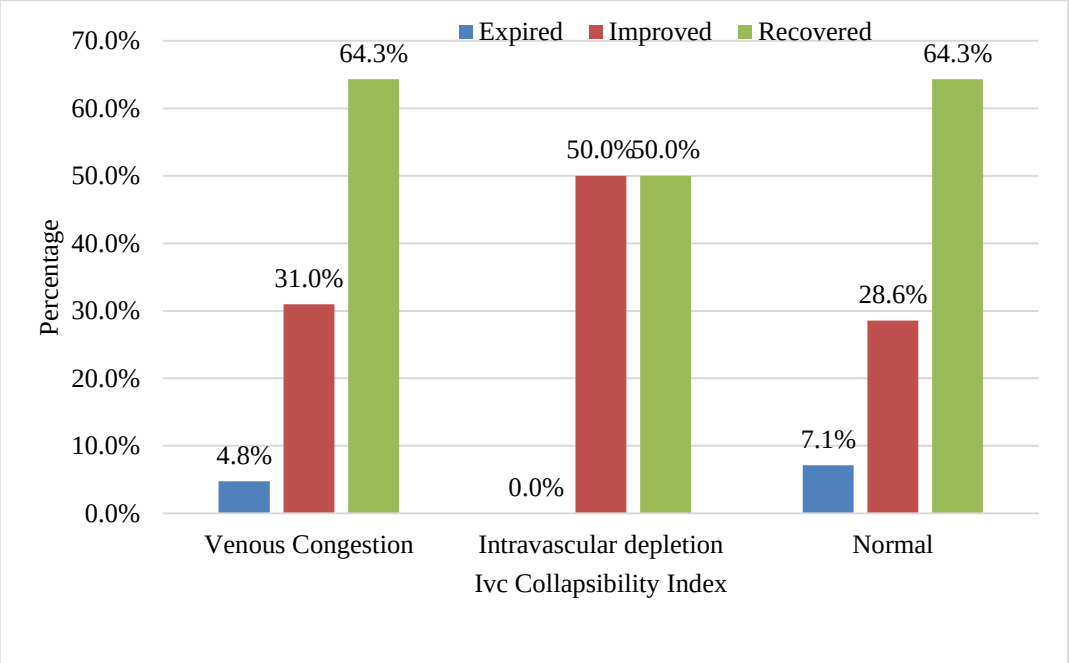
The table and pie chart illustrate the distribution of venous congestion based on the IVC Collapsibility Index in a study population of 60 pediatric patients. The majority, 42 patients (70.0%), exhibit venous congestion, while 14 patients (23.3%) have normal findings, and 4 patients (6.7%) show signs of intravascular depletion. The pie chart visually emphasizes this distribution, with venous congestion being the most prevalent condition, followed by normal findings and a smaller proportion experiencing intravascular depletion. This suggests that venous congestion is a significant concern in this cohort.

Comparison of status on discharge with IVC collapsibility index

Table 10: Comparison of status on discharge with IVC collapsibility index

Status on Discharge	IVC Collapsibility Index			Chi-Square	P-value
	Venous Congestion (N=42)	Intravascular Depletion (N=4)	Normal (N=14)		
Expired	2 (4.8%)	0 (0%)	1 (7.1%)	0.927	0.921
Improved	13 (31%)	2 (50%)	4 (28.6%)		
Recovered	27 (64.3%)	2 (50%)	9 (64.3%)		

Figure 32: Cluster bar chart of comparison of status on discharge across ivc collapsibility index (N=60)



The table and bar graph compare discharge outcomes across different categories of the IVC Collapsibility Index (Venous Congestion, Intravascular Depletion, and Normal) in a study population of 60 paediatric patients. The chi-square

value of 0.927 and P-value of 0.921 indicate no statistically significant association between the IVC Collapsibility Index and discharge status. Among patients with venous congestion (N=42), 64.3% recovered, 31% improved, and 4.8% expired. In the intravascular depletion group (N=4), 50% recovered and 50% improved, with no reported mortality. Similarly, among patients with normal IVC collapsibility (N=14), 64.3% recovered, 28.6% improved, and 7.1% expired. Thus there was no statistically significant association between the IVC Collapsibility Index and discharge status.

Comparison of IVC inspiratory and expiratory diameter between age groups

***Table 11*: Comparison of IVC inspiratory and expiratory diameter between age groups (N=60)**

Parameter	Age Group (Years)			P Value
	1 Month - 2 Years (N=20)	2 Years - 7 Years (N=23)	7 Years - 17 Years (N=17)	
IVC Exp Diameter (mm)	6.26 ± 1.62	7.99 ± 2.34	10.95 ± 2.3	<0.001
IVC Insp Diameter (mm)	5.48 ± 1.56	6.72 ± 1.79	9.17 ± 2.87	<0.001

The table compares the IVC Expiratory Diameter and IVC Inspiratory Diameter across three different age groups (1 Month - 2 Years, 2 Years - 7 Years, and 7 Years - 17 Years) in the study population (N=60). The mean values for IVC Expiratory Diameter are 6.26 ± 1.62 mm in the 1 Month - 2 Years group, 7.99 ± 2.34 mm in the 2 Years - 7 Years group, and 10.95 ± 2.3 mm in the 7 Years - 17 Years group. The mean values for IVC Inspiratory Diameter are 5.48 ± 1.56 mm in the 1 Month - 2 Years group, 6.72 ± 1.79 mm in the 2 Years - 7 Years group, and 9.17 ± 2.87 mm in the 7 Years - 17 Years group. Both IVC Expiratory Diameter and IVC Inspiratory Diameter show statistically significant differences across age groups, with p-values of <0.001 for both parameters. This indicates that as age increases, the IVC Expiratory and Inspiratory Diameters also tend to increase, suggesting age-related physiological changes in the size of the inferior vena cava.

Comparison of mean of IVC expiratory and inspiratory diameter (mm) with gender

***Table 12*: Comparison of mean of IVC expiratory and inspiratory diameter (mm) with gender**

Parameter	Gender (Mean± SD)		P value
	Male (N=22)	Female (N=38)	
IVC Exp Diameter (mm)	7.6 ± 2.74	8.63 ± 2.78	0.172
IVC Insp Diameter (mm)	6.45 ± 2.47	7.32 ± 2.53	0.200

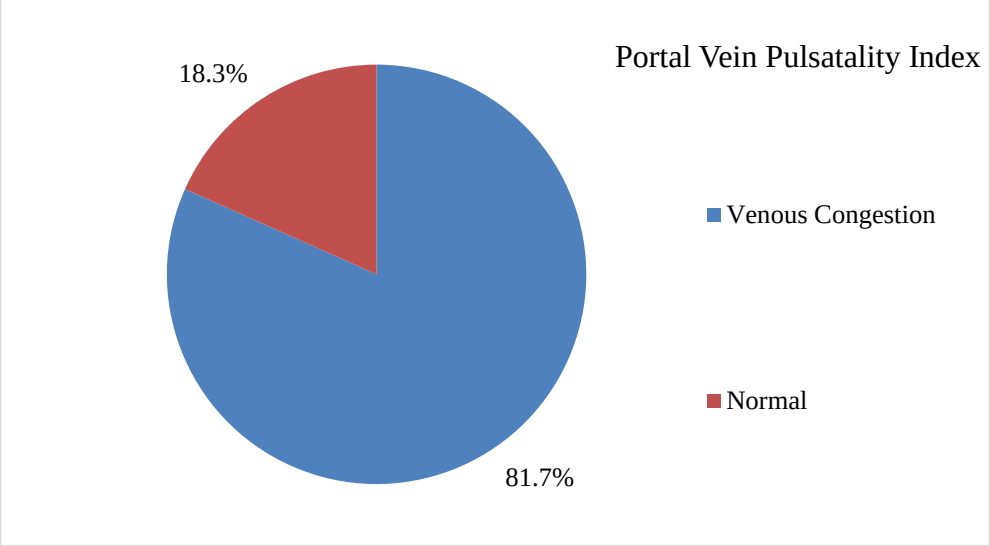
The table compares the mean IVC Expiratory Diameter and IVC Inspiratory Diameter between male and female patients in the study population (N=60). For IVC Expiratory Diameter, the mean is 7.6 ± 2.74 mm in males (N=22) and 8.63 ± 2.78 mm in females (N=38). Similarly, for IVC Inspiratory Diameter, the mean is 6.45 ± 2.47 mm in males and 7.32 ± 2.53 mm in females. The p-values for both comparisons are 0.172 for IVC Expiratory Diameter and 0.200 for IVC Inspiratory Diameter, indicating no statistically significant difference between genders for either parameter. These results suggest that gender does not play a major role in the variation of IVC diameters in the studied cohort, indicating that other factors may influence the measurements.

Descriptive analysis of venous congestion by Portal Vein Pulsatality Index in the study population

***Table 13*: Descriptive analysis of venous congestion by Portal Vein Pulsatality Index in the study population (N=60)**

Methods	Frequency	Percentages
Portal Vein Pulsatality Index		
Venous Congestion	49	81.7%
Normal	11	18.3%

Figure 33: Descriptive analysis of venous congestion by Portal Vein Pulsatality Index in the study population (N=60)



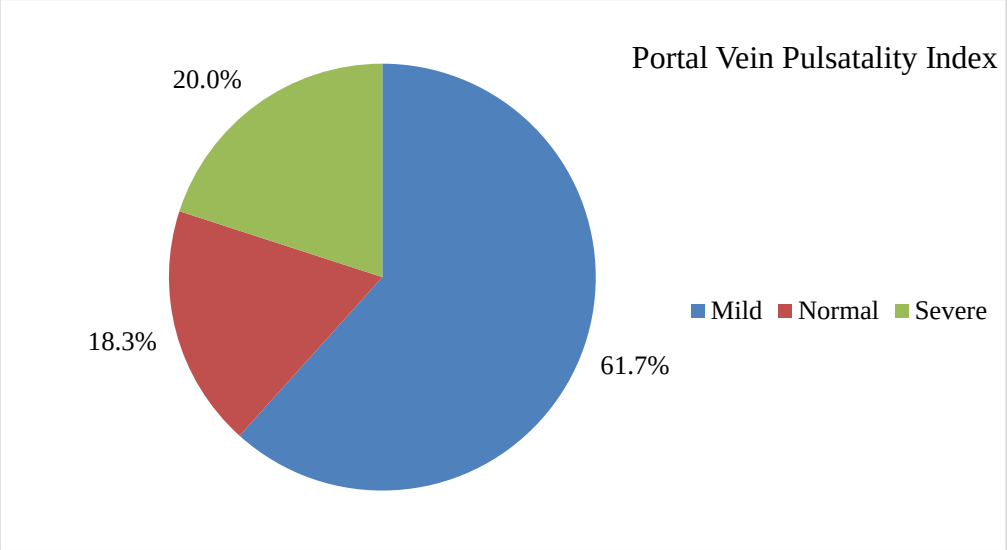
The table and graph illustrate the distribution of venous congestion based on the Portal Vein Pulsatility Index in a study population of 60 pediatric patients. The majority, 49 patients (81.7%), exhibit venous congestion, while 11 patients (18.3%) have normal findings. The graph visually reinforces this distribution, with venous congestion (81.7%) being the predominant condition, compared to the smaller proportion of normal cases (18.3%). These findings highlight a high prevalence of congestion in the portal venous system among the study population.

Descriptive analysis of Portal Vein Pulsatality Index in the study population

***Table 14*: Descriptive analysis of Portal Vein Pulsatality Index in the study population (N=60)**

Portal Vein Pulsatality Index	Frequency	Percentages
Mild	37	61.7%
Normal	11	18.3%
Severe	12	20.0%

Figure 34: Pie chart of portal vein pulsatality index in the study population (N=60)



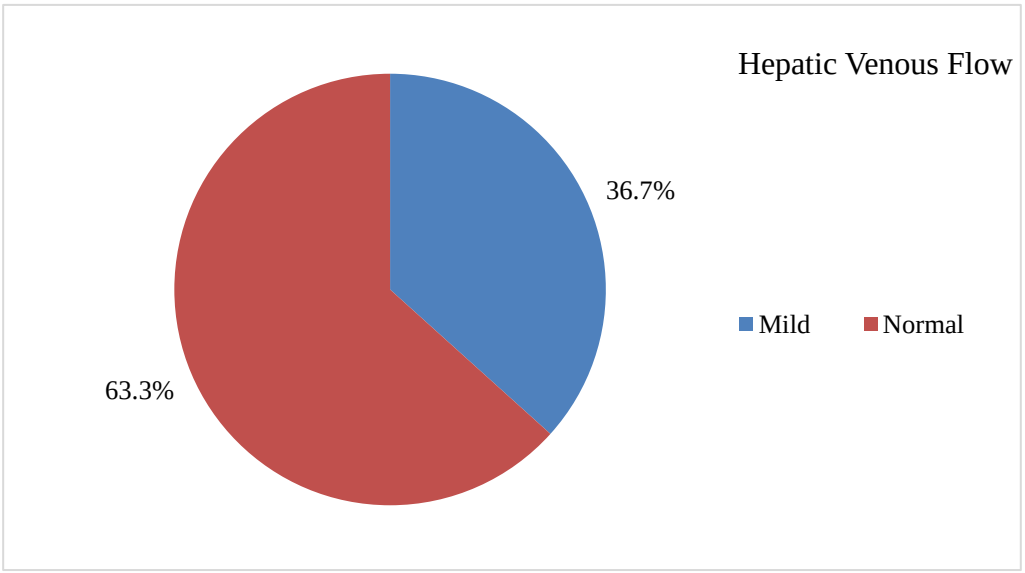
The table and graph illustrate the classification of the Portal Vein Pulsatility Index in a study population of 60 pediatric patients. The majority, 37 patients (61.7%), fall into the Mild category, while 12 patients (20.00%) exhibit Severe pulsatility, and 11 patients (18.3%) have Normal findings. The distribution suggests that while mild portal vein pulsatility is the most prevalent, a notable proportion of patients also experience severe pulsatility, indicating varying degrees of portal venous congestion within the cohort.

Descriptive analysis of Hepatic Venous Flow in the study population

***Table 15*: Descriptive analysis of Hepatic Venous Flow in the study population (N=60)**

Hepatic Venous Flow	Frequency	Percentages
Mild	22	36.7%
Normal	38	63.3%

Figure 35: Pie chart of Hepatic Venous Flow index in the study population (N=60)



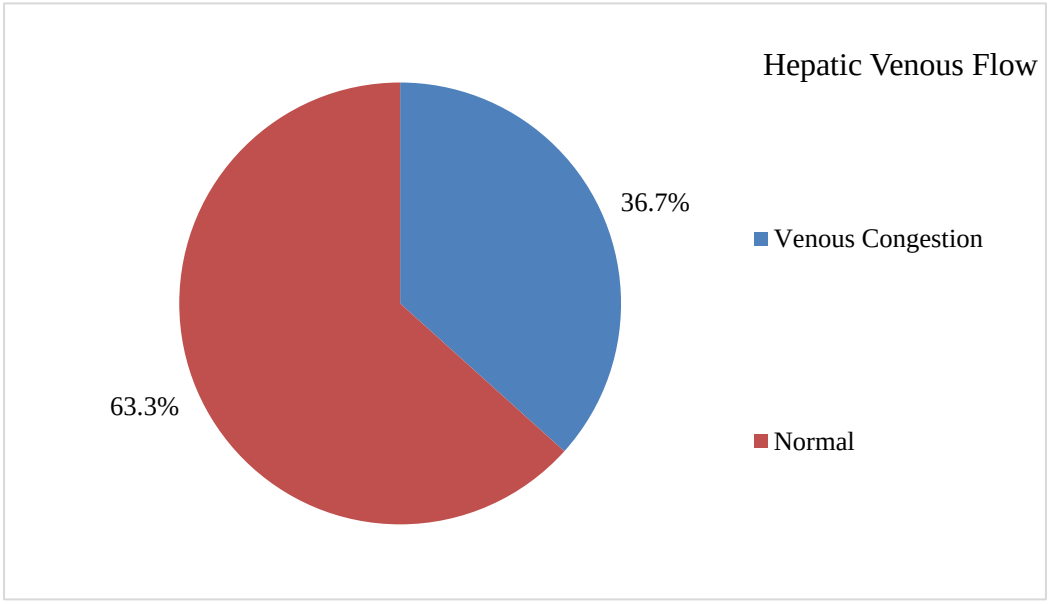
The table and pie chart present the distribution of Hepatic Venous Flow in a study population of 60 paediatric patients. The majority, 38 patients (63.3%), exhibit Normal hepatic venous flow, while 22 patients (36.7%) show Mild changes. The pie chart visually emphasizes this distribution, with Normal flow (63.3%) being the predominant category (red), while Mild alterations (36.7%) represent a smaller proportion (blue). These findings suggest that most patients do not have significant hepatic venous flow abnormalities, though a notable percentage exhibit mild changes.

Descriptive analysis of venous congestion by Hepatic Venous Flow in the study population

***Table 16*: Descriptive analysis of venous congestion by Hepatic Venous Flow in the study population (N=60)**

Methods	Frequency	Percentages
Hepatic Venous Flow		**Percentages**
Venous Congestion	22	36.7%
Normal	38	63.3%

Figure 36: Pie chart of venous congestion by Hepatic Venous Flow in the study population (N=60)



For Hepatic Venous Flow, Venous Congestion is noted in 22 patients (36.7%), while Normal hepatic venous flow is seen in 38 patients (63.3%). The majority, 63.3%, falls under the Normal category (red), while 36.7% corresponds to Venous Congestion (blue). This indicates that a significant proportion of cases exhibit normal hepatic venous flow, with a smaller but notable percentage experiencing congestion.

Predictive Validity of portal vein pulsatality and Hepatic Venous Flow index in predicting Venous Congestion

***Table 17*: Predictive Validity of portal vein pulsatality and Hepatic Venous Flow index in predicting Venous Congestion (n=60)**

Parameter	Value	95% CI	
		Lower	Upper
Portal Vein Pulsatality Index			
Sensitivity	97.92%	88.93%	99.95%
Specificity	83.33%	51.59%	97.91%
False positive rate	16.67%	2.09%	48.41%
False negative rate	2.08%	0.05%	11.07%
Positive predictive value	95.92%	86.02%	99.50%
Negative predictive value	90.91%	58.72%	99.77%
Diagnostic accuracy	95.00%	86.08%	98.96%
Hepatic Venous Flow			
Sensitivity	45.83%	31.37%	60.83%
Specificity	100.00%	73.54%	100.00%
False positive rate	0.00%	-	26.46%
False negative rate	54.17%	39.17%	68.63%
Positive predictive value	100.00%	84.56%	100.00%
Negative predictive value	31.58%	17.50%	48.65%
Diagnostic accuracy	56.67%	43.24%	69.41%

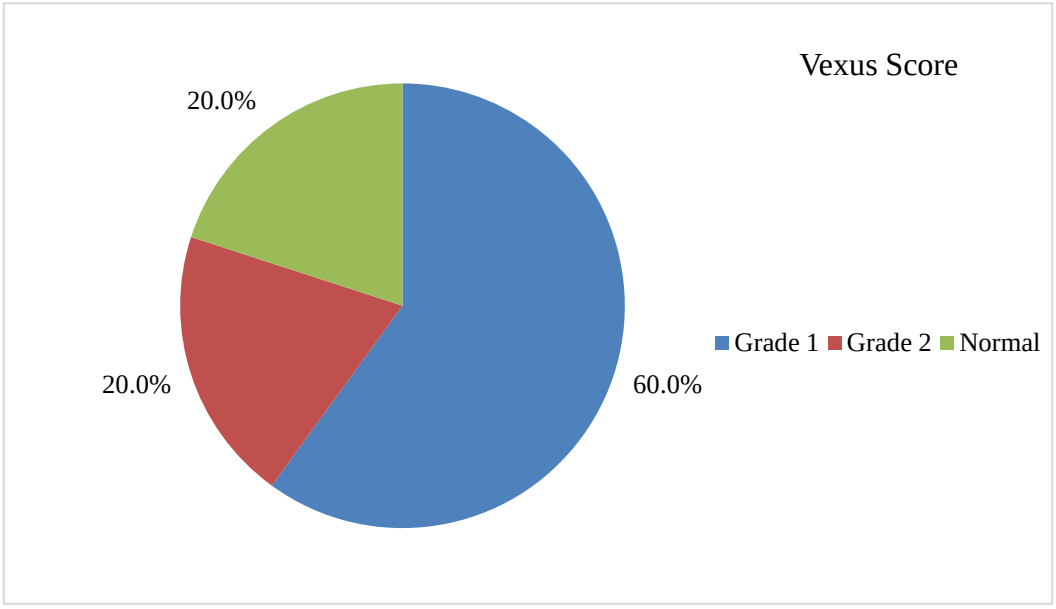
The table presents the predictive validity of the Portal Vein Pulsatility Index and Hepatic Venous Flow index in predicting venous congestion among pediatric patients with congenital heart disease (N=60). The Portal Vein Pulsatility Index demonstrates high sensitivity (97.92%, 95% CI: 88.93% - 99.95%) and specificity (83.33%, 95% CI: 51.59% - 97.91%), indicating its excellent performance in detecting venous congestion and accurately identifying patients without it. The false positive rate is 16.67% (95% CI: 2.09% - 48.41%), and the false negative rate is 2.08% (95% CI: 0.05% - 11.07%). The positive predictive value is 95.92% (95% CI: 86.02% - 99.50%), and the negative predictive value is 90.91% (95% CI: 58.72% - 99.77%), highlighting the test's reliability in confirming or excluding venous congestion. The Hepatic Venous Flow index shows lower sensitivity (45.83%, 95% CI: 31.37% - 60.83%) but perfect specificity (100%, 95% CI: 73.54% - 100%), meaning it is highly accurate in identifying patients without venous congestion but has a relatively high false negative rate (54.17%, 95% CI: 39.17% - 68.63%). The positive predictive value is 100% (95% CI: 84.56% - 100%) but the negative predictive value is 31.58% (95% CI: 17.50% - 48.65%). The diagnostic accuracy of the Portal Vein Pulsatility Index is 95.00% (95% CI: 86.08% - 98.96%), while the Hepatic Venous Flow index has a lower diagnostic accuracy of 56.67% (95% CI: 43.24% - 69.41%). These results indicate that the Portal Vein Pulsatility Index is a highly reliable diagnostic tool for detecting venous congestion compared to the Hepatic Venous Flow index.

Descriptive analysis of VExUS Score in the study population

***Table 18*: Descriptive analysis of VExUS Score in the study population (N=60)**

VExUS Score	Frequency	Percentages
Grade 1	36	60.00%
Grade 2	12	20.00%
Normal	12	20.00%

Figure 37: Pie chart of VExUS Score index in the study population (N=60)



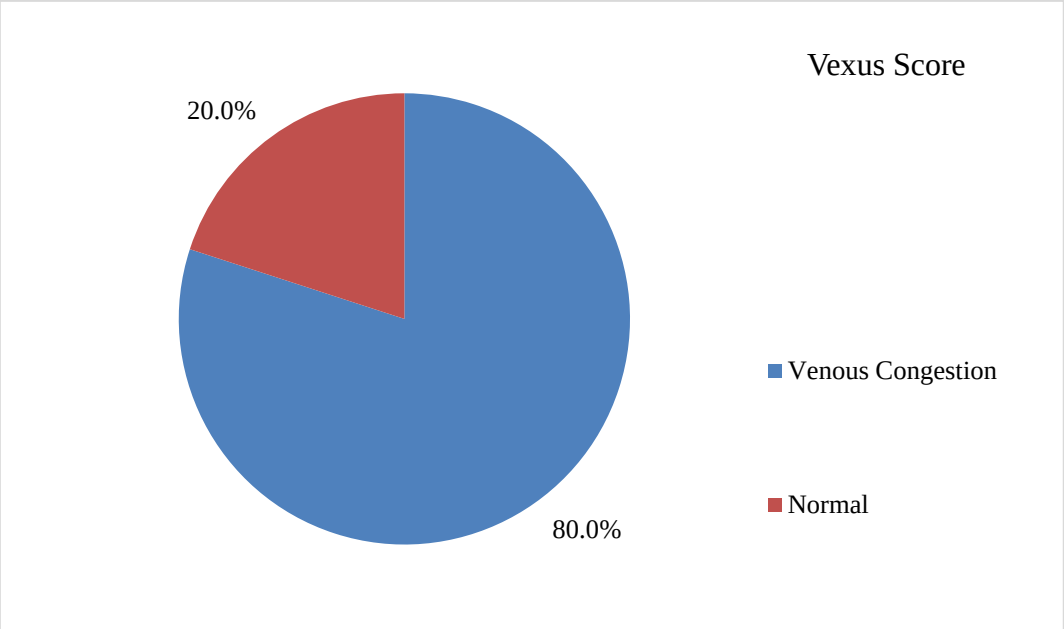
The VExUS Score is divided into Grade 1 in 36 patients (60.00%), Grade 2 in 12 patients (20.00%), and Normal in 12 patients (20.00%). The pie chart represents the distribution of VExUS Score across three categories: Grade 1, Grade 2, and Normal. The largest portion, Grade 1, is depicted in blue and accounts for 60.0%, indicating that the majority fall under this category. The Grade 2 category, shown in red, makes up 20.0%, while the Normal category, represented in green, also constitutes 20.0%.

Descriptive analysis of VExUS Score in the study population

***Table 19*: Descriptive analysis of VExUS Score in the study population (N=60)**

Methods	Frequency	Percentages
VExUS Score		
Venous Congestion	48	80.00%
Normal	12	20.00%

Figure 38: Pie chart of VExUS Score in the study population (N=60)



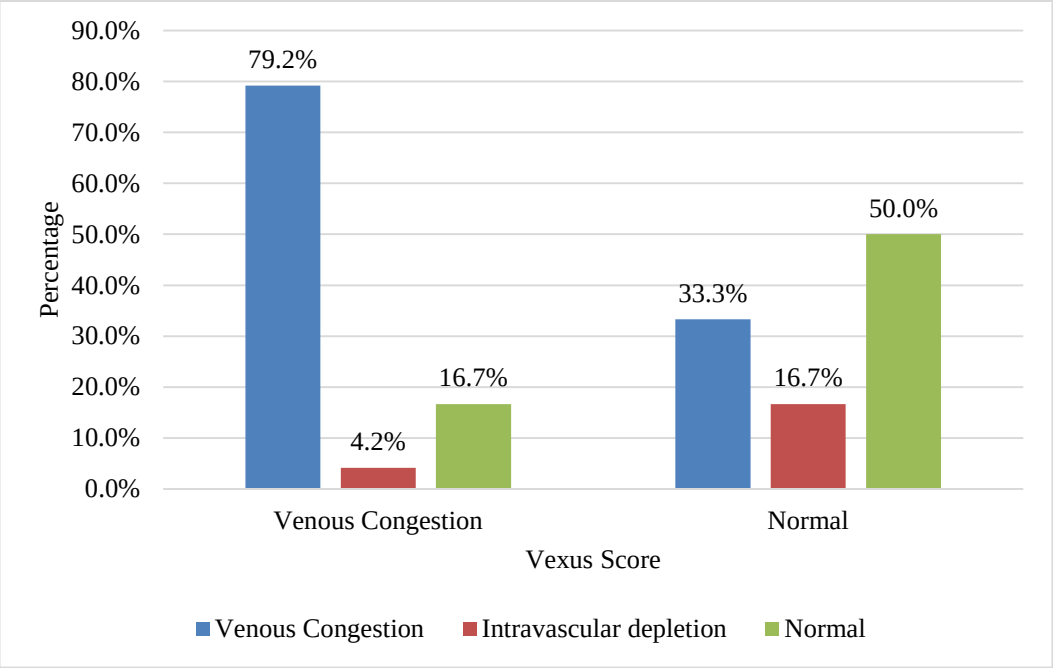
The VExUS Score shows Venous Congestion in 48 patients (80.00%), with 12 patients (20.00%) classified as Normal. The majority, 80.0%, falls under Venous Congestion (blue), while 20.0% represents the Normal condition (red). This indicates that a significant proportion of cases experience venous congestion based on the VExUS Score.

Comparison of IVC collapsibility index with VEXUS score

***Table 20*: Comparison of IVC collapsibility index with VEXUS score**

IVC Collapsibility Index	VEXUS Score		Chi square	P value
	Venous Congestion (N=48)	Normal (N=12)		
Venous Congestion	38 (79.2%)	4 (33.3%)	9.702	0.008
Intravascular Depletion	2 (4.2%)	2 (16.7%)		
Normal	8 (16.7%)	6 (50%)		

Figure 39: Cluster bar chart of comparison of IVC collapsibility index with VEXUS score (N=60)



The table and graph compare the IVC Collapsibility Index across VEXUS score categories in the study population. A chi-square value of 9.702 and a P-value of 0.008 indicate a statistically significant association between the VEXUS score and venous congestion. Among patients in the venous congestion by VEXUS group

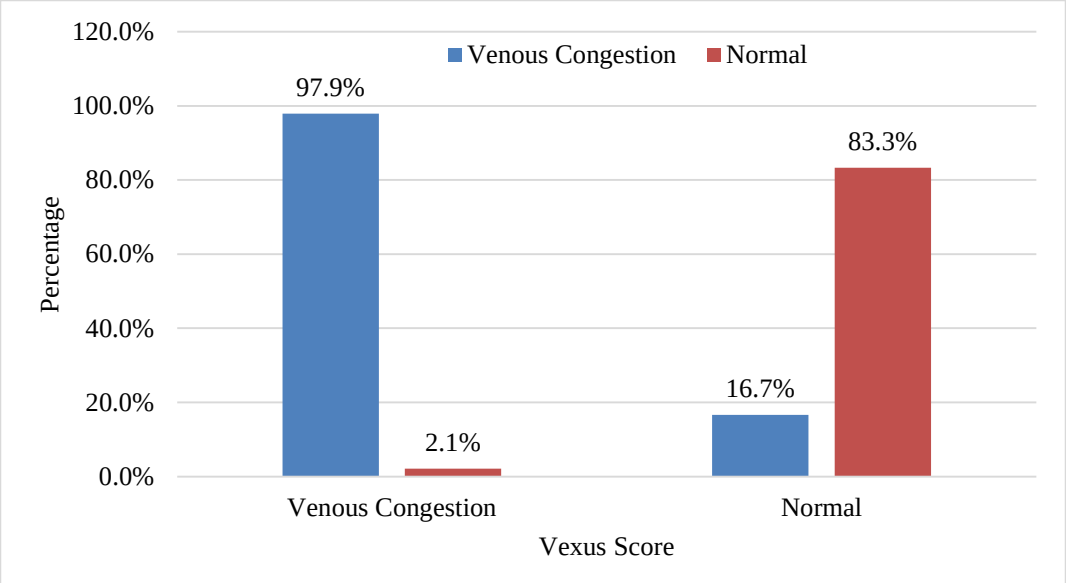
(N=48), 79.2% exhibited venous congestion, 4.2% had intravascular depletion, and 16.7% had normal IVC collapsibility. In contrast, within the normal VEXUS group (N=12), 33.3% showed venous congestion, 16.7% had intravascular depletion, and 50% had normal IVC collapsibility. Thus, VEXUS score demonstrates statistically significant superiority over IVCCI in assessing venous congestion as indicated by chi-square value=9.702 and p-value of 0.008.

Comparison of portal vein pulsatality index with VEXUS score

***Table 21*: Comparison of portal vein pulsatality index with VEXUS score**

Parameter	VEXUS Score		Chi square	P value
	Venous Congestion (N=48)	Normal (N=12)		
Portal Vein Pulsatality Index				
Venous Congestion	47 (97.9%)	2 (16.7%)	42.328	<0.001
Normal	1 (2.1%)	10 (83.3%)		

Figure 40: Cluster bar chart of comparison of portal vein pulsatality index with VEXUS score (N=60)



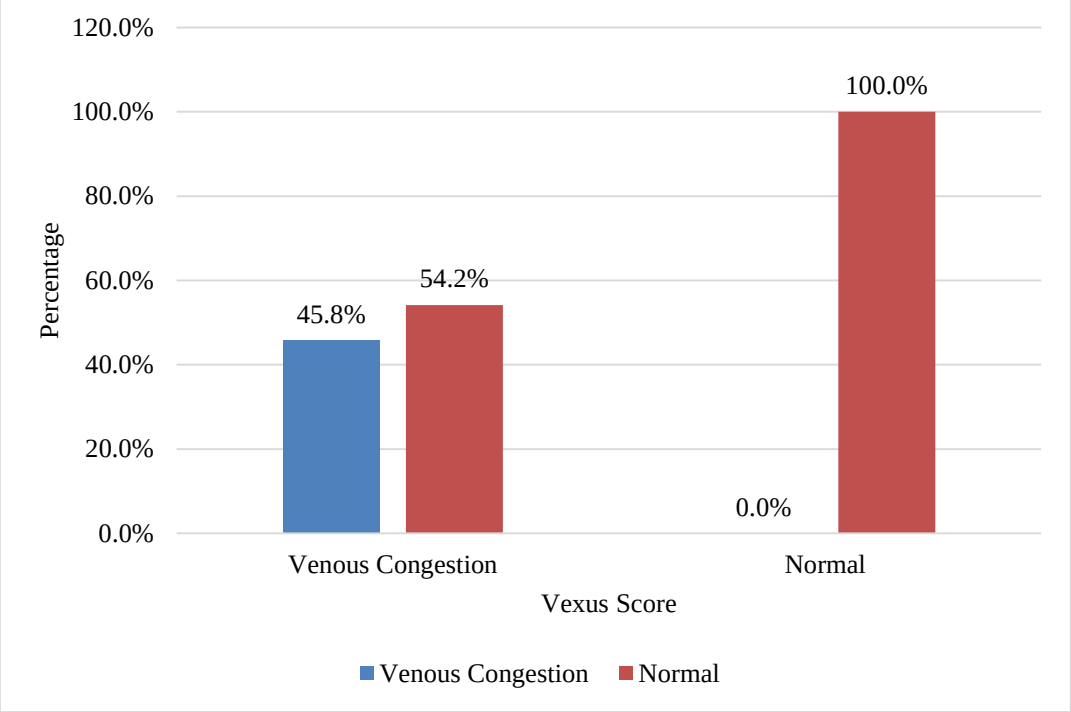
The table and bar graph demonstrate a strong association between the Portal Vein Pulsatility Index and the VEXUS score, as indicated by a highly significant chi-square value of 42.328 ($P < 0.001$). Among patients with venous congestion by VEXUS score (N=48), 97.9% exhibited venous congestion on the Portal Vein Pulsatility Index, while only 2.1% had normal portal vein pulsatility. Conversely, in the normal VEXUS score group (N=12), 16.7% showed venous congestion, whereas 83.3% had normal portal vein pulsatility.

Comparison of Hepatic Venous Flow with VEXUS score

***Table 22*: Comparison of Hepatic Venous Flow with VEXUS score (N=60)**

Parameter	VEXUS Score		Chi square	P value
	Venous Congestion (N=48)	Normal (N=12)		
Hepatic Venous Flow				
Venous Congestion	22 (45.8%)	0 (0%)	8.64	0.003
Normal	26 (54.2%)	12 (100%)		

Figure 41: Cluster bar chart of comparison of Hepatic Venous Flow with VEXUS score (N=60)



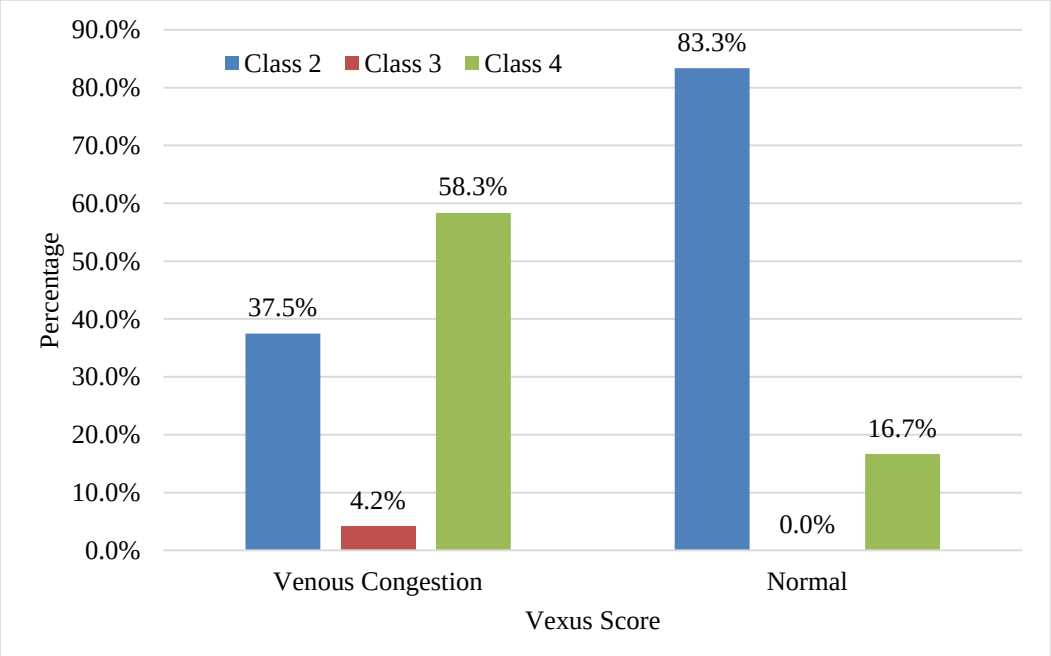
The table and figure present the comparison of Hepatic Venous Flow with the VEXUS score in a sample of 60 participants. Among the 48 individuals with venous congestion by VEXUS score, 22 (45.8%) had abnormal hepatic venous flow. Conversely, normal hepatic venous flow was observed in 26 (54.2%) of those with venous congestion by VEXUS and in all 12 (100%) individuals with a normal VEXUS score. The chi-square test yielded a value of 8.64, with a statistically significant P-value of 0.003, indicating a strong association between hepatic venous flow abnormalities and higher VEXUS scores. A cluster bar chart, visually represents this relationship, demonstrating the higher prevalence of abnormal hepatic venous flow among those with venous congestion, further reinforcing the statistical findings.

Comparison of Modified Ross HFC with VEXUS score according to venous congestion

***Table 23*: Comparison of Modified Ross HFC with VEXUS score according to venous congestion (N=60)**

Parameter	VEXUS Score		Chi square	P value
	Venous Congestion (N=48)	Normal (N=12)		
Modified Ross HFC				
Class 2	18 (37.5%)	10 (83.3%)	8.15	0.017
Class 3	2 (4.2%)	0 (0%)		
Class 4	28 (58.3%)	2 (16.7%)		

Figure 42: Cluster bar chart of comparison of modified Ross HFC with VEXUS score according to venous congestion (N=60)



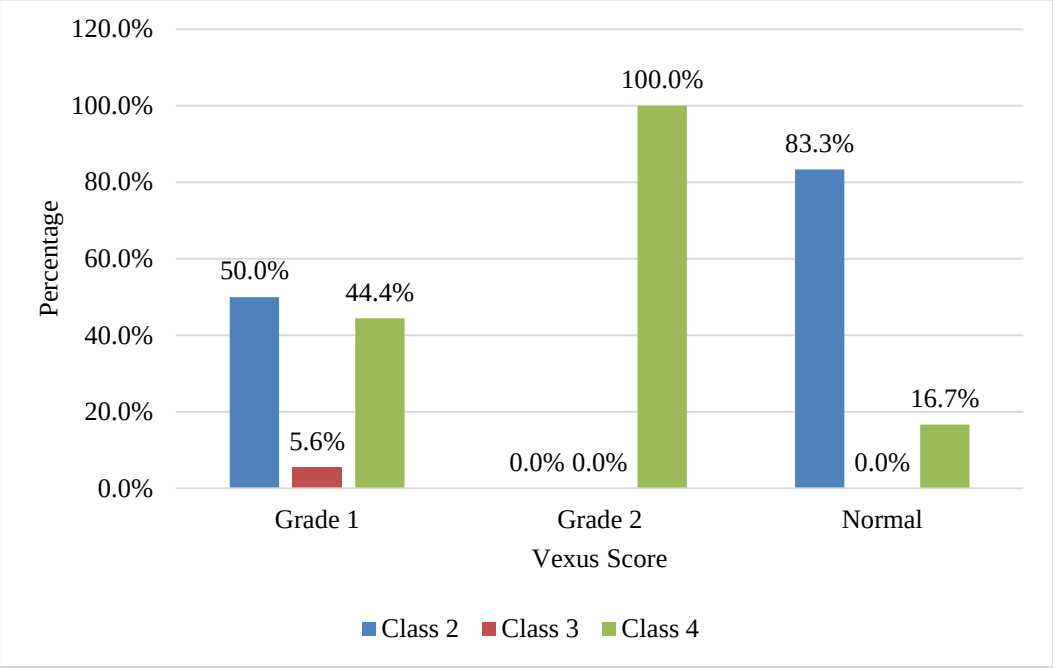
The table and cluster bar chart compare the Modified Ross Heart Failure Classification (HFC) with the VExUS Score to assess venous congestion in paediatric patients with congenital heart disease (N=60). The data show a statistically significant association (Chi-square = 8.15, P = 0.017) between heart failure severity and VEXUS score. In the Venous Congestion group (N=48), 37.5% of patients were classified as Class 2, 4.2% as Class 3, and 58.3% as Class 4 (severe heart failure). In contrast, among patients with a Normal VExUS score (N=12), 83.3% were in Class 2, none in Class 3, and only 16.7% in Class 4. The bar chart highlights that venous congestion is most prevalent in Class 4, with 58.3% of affected patients falling into this category. These findings suggest that worsening heart failure, particularly Class 4, is strongly associated with venous congestion, reinforcing the clinical significance of the VExUS score in assessing disease

Comparison of modified ross HFC with VExUS score according to grading of VExUS

Table 24: Comparison of modified ross HFC with VExUS score according to grading of VExUS (N=60)

Modified Ross HFC	VExUS Score			Chi-Square	P-value
	Grade 1 (N=36)	Grade 2 (N=12)	Normal (N=12)		
Class 2	18 (50%)	0 (0%)	10 (83.3%)	19.36	0.001
Class 3	2 (5.6%)	0 (0%)	0 (0%)		
Class 4	16 (44.4%)	12 (100%)	2 (16.7%)		

Figure 43: Cluster bar chart of comparison of modified ross HFC with VExUS score according to grading of VExUS (N=60)



The table and bar graph compare the Modified Ross Heart Failure Classification (HFC) across different VEXUS score grading (Grade 1, Grade 2, and Normal) in the study population (N=60). A statistically significant association was observed (Chi-square = 19.36, P = 0.001), indicating a strong correlation between the VEXUS score grading and heart failure severity. Among patients with Grade 1 VEXUS (N=36), 50% were classified as Class 2, 5.6% as Class 3, and 44.4% as Class 4. In the Grade 2 VEXUS group (N=12), all patients (100%) were in Class 4, indicating severe heart failure. Conversely, in the Normal VEXUS group (N=12), 83.3% were in Class 2, while 16.7% were in Class 4, with no cases in Class 3.

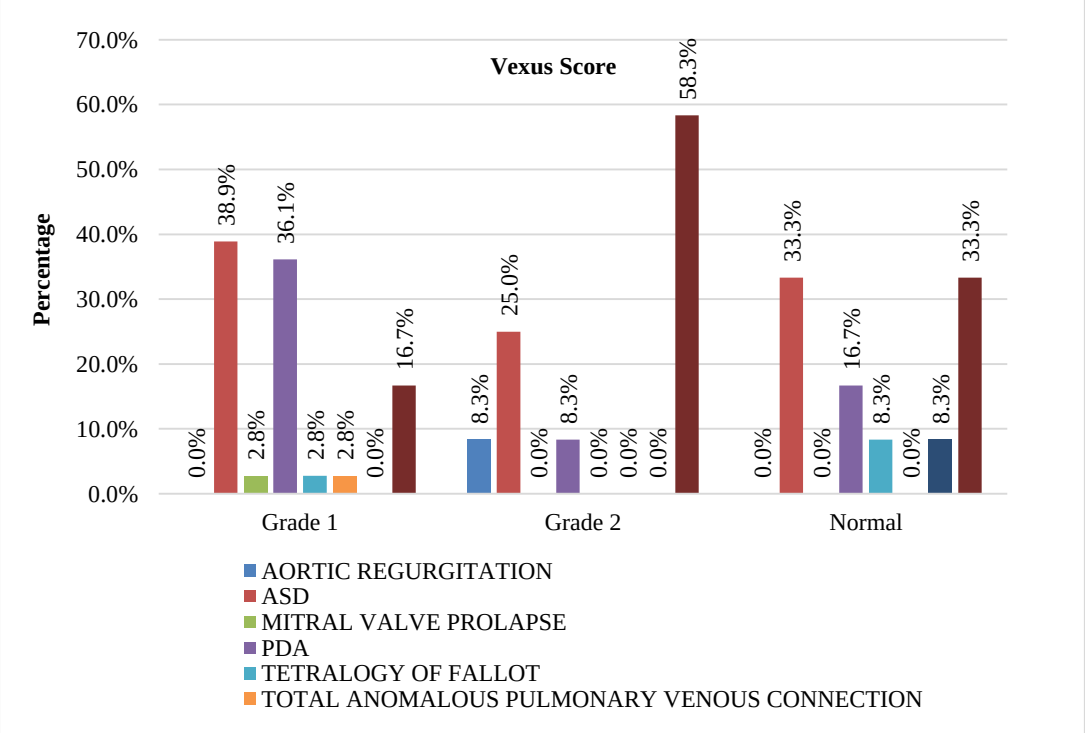
The bar graph further illustrates this distribution, reinforcing that as VEXUS severity increases, the proportion of patients in Class 4 also rises. These findings highlight the strong link between venous congestion severity and worsening heart failure, emphasizing the VEXUS score's clinical significance in assessing disease progression.

Comparison of diagnosis with VEXUS score in the study population

Table 25: Comparison of diagnosis with VEXUS score (N=60)

Diagnosis	VExUS Score			Chi-Square	P-value
	Grade 1 (N=36)	Grade 2 (N=12)	Normal (N=12)		
Aortic Regurgitation	0 (0%)	1 (8.3%)	0 (0%)	19.99	0.131
ASD	14 (38.9%)	3 (25%)	4 (33.3%)		
Mitral Valve Prolapse	1 (2.8%)	0 (0%)	0 (0%)		
PDA	13 (36.1%)	1 (8.3%)	2 (16.7%)		
Tetralogy of Fallot	1 (2.8%)	0 (0%)	1 (8.3%)		
TAPVC	1 (2.8%)	0 (0%)	0 (0%)		
Tricuspid Regurgitation	0 (0%)	0 (0%)	1 (8.3%)		
VSD	6 (16.7%)	7 (58.3%)	4 (33.3%)		

Figure 44: Cluster bar chart of comparison of diagnosis with VExUS score (N=60)



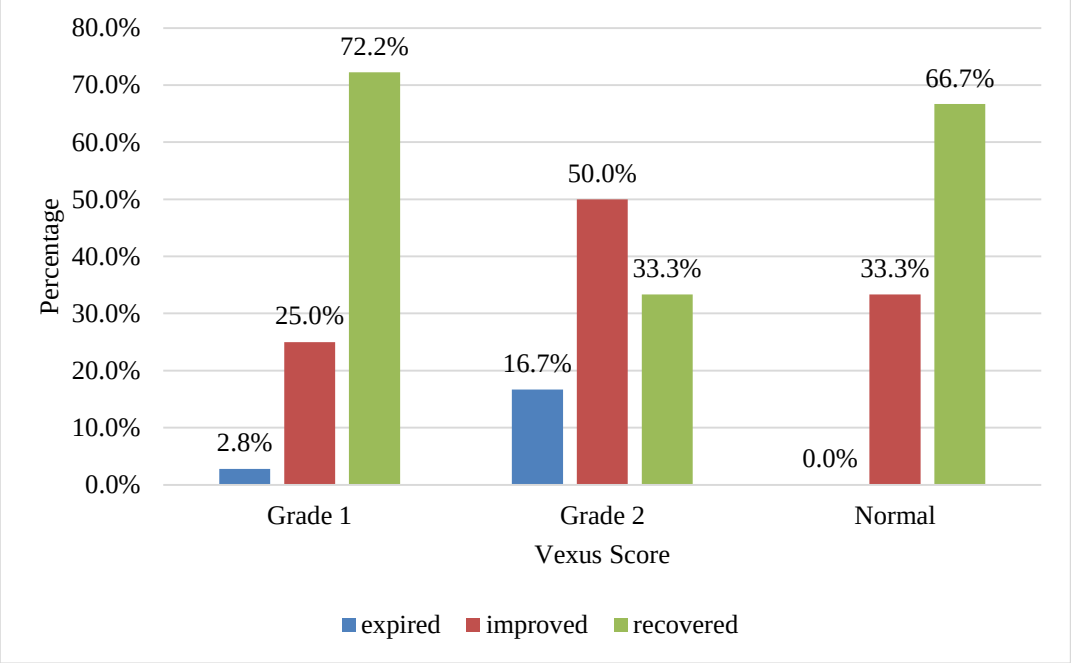
The table and bar graph compare various cardiac diagnoses across different VExUS score categories (Grade 1, Grade 2, and Normal) in the study population (N=60). Although the chi-square value of 19.99 and P-value of 0.131 indicate no statistically significant association between diagnosis and VExUS score, however certain trends emerge in the data. Atrial Septal Defect (ASD) was the most common diagnosis, found in 38.9% of Grade 1, 25% of Grade 2, and 33.3% of the Normal group. Patent Ductus Arteriosus (PDA) was most prevalent in Grade 1 (36.1%), with lower frequencies in Grade 2 (8.3%) and Normal (16.7%). Ventricular Septal Defect (VSD) appeared more frequently in Grade 2 (58.3%) compared to Grade 1 (16.7%) and Normal (33.3%), suggesting a possible link between VSD and severity of VExUS score. Other less common diagnoses included Aortic Regurgitation (8.33% in Grade 2), Tetralogy of Fallot (one case in Grade 1 and Normal groups), Tricuspid Regurgitation (8.33% in the Normal group), and Total Anomalous Pulmonary Venous Connection (2.78% in Grade 1). The bar graph highlights the same distributions, showing ASD as the most prevalent condition across all groups, while VSD appears more frequently in patients with higher venous congestion (Grade 2). Despite these patterns, the lack of statistical significance suggests that a larger sample size may be necessary to establish definitive correlations between specific congenital heart diseases and VExUS score severity.

Comparison of status on discharge with VExUS score in the study population

Table 26: Comparison of status on discharge with VExUS score (N=60)

Status on Discharge	VExUS Score			Chi square	P value
	Grade 1 (N=36)	Grade 2 (N=12)	Normal (N=12)		
Expired	1 (2.8%)	2 (16.7%)	0 (0%)	8.187	0.085
Improved	9 (25%)	6 (50%)	4 (33.3%)		
Recovered	26 (72.2%)	4 (33.3%)	8 (66.7%)		

Figure 45: Cluster bar chart of comparison of status on discharge with VExUS score (N=60)



The table and cluster bar chart compare patient discharge status across different VExUS score categories (Grade 1, Grade 2, and Normal) in the study population (N=60). Although the chi-square value of 8.187 and P-value of 0.085

indicate no statistically significant association between VExUS score and discharge outcomes, the data suggests a trend linking higher venous congestion with poorer prognosis.

Among patients with Grade 1 VExUS scores, 72.2% recovered, 25% improved, and 2.8% expired. In the Grade 2 category, recovery was lower (33.33%), improvement was more common (50%), and mortality was significantly higher (16.7%). In contrast, the Normal VExUS group had a recovery rate of 66.7%, an improvement rate of 33.3%, and no recorded deaths. The cluster bar chart reinforces these findings, showing that recovery rates were highest in the Normal (66.7%) and Grade 1 (72.2%) groups. The absence of mortality in the Normal group suggests that higher VExUS scores correlate with worse outcomes, highlighting the potential role of VExUS score in predicting prognosis. However, the lack of statistical significance (P=0.085) indicates that larger studies are needed to confirm these trends.

DISCUSSION

The evaluation of venous congestion in congenital cardiac anomalies presenting in heart failure is crucial for prognostication and directing effective treatment plan. This research compares Venous Excess Ultrasound Score (VExUS) alongside the Inferior Vena Cava (IVC)collapsibility index as tools for evaluating venous congestion in pediatric CHD cases.

Our findings demonstrates the utility of Venous excess ultrasound and IVC collapsibility index in assessing venous congestion, as evidenced by a study by Longino et al.[115]. The study population consisted of 60 paediatric patients, with a mean age of 5.42 years (SD ± 4.7), and a majority (38.3%) belonging to the 2–7-year age group. The female predominance (63.3%) aligns with Reller et al.[116] indicating a higher prevalence of congenital heart anomalies in females.

Among the clinical characteristics analysed, hepatomegaly (91.7%) and failure to thrive (60%) were the most prevalent findings, underscoring the systemic effects of venous congestion. Additionally, respiratory distress (51.7%) was observed in more than half of the patients, suggesting pulmonary involvement. While pleural effusion was relatively rare (3.3%), pedal oedema (43.3%) and pulmonary crackles (8.3%) were notable, reinforcing the multifactorial presentation of venous congestion in CHD-associated heart failure as evidenced by Gewitz et al.[117]

The IVC diameter analysis revealed a mean expiratory diameter of 8.25 mm and an inspiratory diameter of 7.0 mm, with considerable variability. These findings are consistent with previous studies by Schefold et al.[118] and Orde et al.[119] showing that IVC collapsibility can reflect volume status and venous congestion but may be limited in patients with altered right heart hemodynamics. The Portal Vein Pulsatility

Index, with 61.7% categorized as mild and 20% as severe, further highlights the relevance of portal congestion in evaluating systemic venous pressure. Similarly, hepatic venous flow abnormalities were present in 36.67% of cases, emphasizing the impact of congestive hepatopathy in CHD patients which was similar to the studies done by Beaubien-Souligny et al.[120] and Tamaki et al.[121]

The VExUS score distribution (Grade 1: 60%, Grade 2: 20%, Normal: 20%) indicates that the majority of patients exhibited some degree of venous congestion. Notably, all Grade 2 patients were classified as severe (Class 4) in the Modified Ross Heart Failure Classification, demonstrating the clinical relevance of VExUS in identifying severe heart failure cases. Prior research by Beaubien-Souligny et al (2023).[122] and Denault et al.[123], has supported the use of VExUS in critically ill patients, correlating higher scores with worse outcomes.

This study supports the growing evidence that the VExUS score provides a more comprehensive evaluation of venous congestion compared to IVC collapsibility alone; which has been evidence by Beaubien-Souligny et al.[122] and other study by Balik et al.[124] While IVC diameter remains a useful measure, it may not fully capture systemic venous congestion, particularly in CHD patients with altered right heart physiology. Integrating VExUS assessment alongside traditional markers could enhance the precision of heart failure management in pediatric CHD populations.

The descriptive analysis of venous congestion based on the IVC Collapsibility Index indicated that 70% of patients exhibited venous congestion, whereas 6.7% showed intravascular depletion, and 23.3% had normal findings. Nagueh et al.[125] showed that the IVC Collapsibility Index has been widely used as a surrogate marker for right atrial pressure, which correlates with systemic venous congestion. However,

this method has limitations, particularly in CHD patients where right atrial compliance and ventricular dysfunction can significantly alter venous return dynamics according to Dani et al.[126]

The Portal Vein Pulsatility Index, which demonstrated congestion in 81.7% of patients, is used to evaluate systemic venous congestion. Portal venous flow becomes more pulsatile in response to increased right atrial pressure and systemic congestion according to Denault et al.[123]. Given the higher percentage of congestion detected using this parameter, it suggests a more sensitive detection of systemic venous congestion compared to the IVC Collapsibility Index. Similarly, hepatic venous flow analysis revealed congestion in 36.7% of patients, while 63.3% had normal hepatic venous flow. Beaubien-Souligny et al.[127] showed hepatic venous flow assessment is a crucial part of the VExUS Score and provides insights into systemic venous congestion. The relatively lower proportion of congestion detected compared to the Portal Vein Pulsatility Index suggests that hepatic venous flow assessment alone may not be sufficient to diagnose venous congestion comprehensively in CHD patients. The VExUS Score, which showed venous congestion in 80% of patients, incorporates multiple venous flow parameters, including hepatic, portal, and renal vein waveforms. This scoring system provides a more comprehensive evaluation of venous congestion than isolated assessments such as the IVC Collapsibility Index.[127] The high proportion of congestion detected using VExUS suggests that it is a superior method for identifying systemic congestion in CHD patients with HF.

Furthermore, patient outcomes upon discharge indicate that 63.3% of patients recovered, while 31.7% improved but did not fully recover, and 5% did not survive. This outcome data in accordance with Gould et al.underscores the significance of early and accurate assessment of venous congestion in guiding appropriate

interventions and improving patient prognosis [128] The Modified Ross Heart Failure Classification further stratified patients into different heart failure severity classes, with a majority (50%) classified as Class 4, indicating severe heart failure. This correlates with the high prevalence of venous congestion detected by VExUS and other ultrasound parameters. Lastly, the distribution of congenital heart disease diagnoses within the study population highlights the common underlying conditions contributing to HF in these patients. Atrial Septal Defect (35%), Ventricular Septal Defect (28.3%), and Patent Ductus Arteriosus (26.7%) were the most frequently observed. These structural abnormalities significantly impact pulmonary and systemic circulation, further exacerbating venous congestion and heart failure as per Gewillig & Brown et al.[129]

Overall, the findings suggest that while the IVC Collapsibility Index remains a useful tool, it is less sensitive than the VExUS Score in detecting venous congestion. The VExUS Score's incorporation of multiple venous flow parameters provides a more comprehensive assessment and should be considered a superior method in CHD patients presenting with HF. These results reinforce the need for multimodal ultrasound evaluation.

Venous congestion assessment is particularly crucial in pediatric patients with congenital heart disease (CHD) due to their unique hemodynamic challenges, including volume overload, altered venous compliance, and increased right heart pressures. Early detection and management of congestion can significantly impact outcomes, preventing worsening heart failure and associated complications. The Venous Excess Ultrasound (VExUS) score and the Inferior Vena Cava (IVC) collapsibility index are widely used parameters in evaluating systemic congestion.

The comparison of these indices in the current study highlights their relative strengths and limitations in predicting venous congestion.

In our study the comparison of IVC Collapsibility Index with VExUS Score showed a significant association between the IVC collapsibility index and VExUS score (chi-square = 9.702, P = 0.008) suggests that a higher VExUS score correlates with increased venous congestion, which is evidenced by the study by Beaubien-Souligny et al (2022).[130] However, this correlation may not be universally applicable across all patient subgroups, particularly those with altered venous compliance due to chronic heart failure or post-surgical changes in hemodynamics. Further studies are needed to delineate the specific conditions under which this relationship holds strongest. Denault et al.[117] showed patients with venous congestion exhibited significantly reduced IVC collapsibility, indicating fluid overload and compromised venous return. However, while the IVC collapsibility index is a useful marker, it may not fully capture the nuances of venous congestion, particularly in conditions where venous compliance varies due to chronic heart failure according to Mullens et al.[131]

The portal vein pulsatility index showed a highly significant association with the VExUS score (chi-square = 42.328, P < 0.001), demonstrating its reliability in identifying venous congestion. Nearly 98% of patients with venous congestion exhibited portal vein pulsatility abnormalities, reinforcing its diagnostic value. Similarly, hepatic venous flow abnormalities were significantly associated with the VExUS score (chi-square = 8.64, P = 0.003). The predictive validity of the portal vein pulsatility index was superior, with a sensitivity of 97.92% and specificity of 83.33%, whereas hepatic venous flow had lower sensitivity (45.83%) but perfect specificity (100%). These findings align with previous studies by Beaubien-Souligny et al.[127] emphasizing the role of portal venous pulsatility in evaluating right heart failure.

The association between the Modified Ross Heart Failure Classification (HFC) and VExUS score (chi-square = 8.15, P = 0.017) supports the contribution of VExUS in categorizing the severity of heart failure in CHD patients. A higher proportion of patients in Class 4 heart failure were in the venous congestion group, indicating that systemic venous congestion correlates with worsening heart failure symptoms and correlates with study by Rola et al.[132]

The differences in Inferior Vena Cava (IVC) diameters during expiration and inspiration varied significantly across age groups (P < 0.001), supporting the idea that venous compliance changes with age. In contrast, no notable differences were found between genders, suggesting that gender does not affect IVC diameter in this group of children. These observations align with prior work by Nagueh et al., which points to age-related shifts in IVC characteristics.[133]

Diagnostic Performance of VExUS Score and IVC Collapsibility Index showed **t**he lack of a statistically significant association between cardiac diagnosis and VExUS score (chi-square = 19.99, P = 0.131) suggests that while VExUS is effective in assessing venous congestion, its specificity to particular congenital heart defects remains unclear. Similarly, the lack of a significant association between discharge outcomes and both the VExUS score (P = 0.085) and IVC collapsibility index (P = 0.921) indicates that while these parameters help assess congestion, they may not directly predict clinical outcomes. However, trends observed in the data suggest that patients with higher VExUS scores had worse outcomes, which aligns with existing evidence by Dhayat et al.[134] on systemic venous congestion and prognosis in heart failure. The VExUS score demonstrated strong associations with venous congestion as assessed by multiple ultrasound markers, particularly the portal vein pulsatility index and hepatic venous flow. This highlights its potential role as a key tool in

guiding clinical decision-making for pediatric CHD patients with heart failure, emphasizing the need for its integration into routine congestion assessments. The IVC collapsibility index, while useful, showed limitations in sensitivity and predictive value compared to portal vein parameters. These findings reinforce the clinical utility of the VExUS score as a comprehensive tool for assessing venous congestion in paediatric CHD patients with heart failure.

LIMITATIONS AND SCOPE OF THE STUDY

Limitations of the Study

- The research involved a limited cohort of 60 pediatric patients, which may restrict the ability to apply the findings broadly. A larger group would be necessary to confirm these results.
- Conducted at a single healthcare facility, the study's outcomes might not fully translate to other settings with different patient profiles or care practices.
- The sample may not fully reflect the wider population of pediatric CHD patients, as it could include a higher proportion of severe cases needing complex treatments.
- Additional modalities, such as invasive hemodynamic monitoring, could provide further validation.

Scope of the Study

This research offers important understanding of the clinical features, diagnostic results, and outcomes.It focuses on evaluating venous congestion using key indicators such as IVCCI, PVPI, Hepatic Venous Flow, and the VExUS Score. By analyzing their associations with clinical outcomes, this research highlights the predictive utility of these indices in assessing venous congestion and heart failure severity in pediatric patients.

CONCLUSION

This research sheds light on the clinical traits, diagnostic outcomes, and prognosis of children with congenital heart anomalies and cardiac failure, stressing the need for early identification of venous congestion—a common issue in this group. It assesses various diagnostic tools to gauge the extent of venous congestion and forecast patient outcomes, revealing that many pediatric CHD and HF patients display this condition through tools like IVC Collapsibility Index, PVPI, and VExUS Score. The VExUS Score, integrating multiple clinical and diagnostic factors, showed a robust link to venous congestion and HF severity, positioning it as a promising aid in patient care. The Portal Vein Pulsatility Index stood out for its dependability in spotting venous congestion, boasting a sensitivity of 97.92% and specificity of 83.33%. A notable connection was also found between the Modified Ross Heart Failure Classification and the VExUS Score, suggesting that children with advanced heart failure (Class 4) are more prone to significant venous congestion. This work underscores the VExUS Score's practical value in detecting venous congestion, predicting outcomes, and guiding treatment in young CHD and HF patients. Early recognition and management of venous overload with the VExUS Score can enhance these patients' outlook and lessen the impact of cardiac failure among children with congenital cardiac conditions.

SUMMARY

This study aimed to analyze the clinical characteristics, diagnostic findings, and outcomes of paediatric patients with congenital heart disease (CHD) and heart failure (HF), examining various diagnostic indices and their associations with venous congestion and clinical outcomes. The patients comprised 60 paediatric children, with a mean age of 5.42 years. Gender distribution showed a higher proportion of female patients (63.33%) compared to male patients (36.67%). The study evaluated several clinical, hemodynamic, and diagnostic parameters, including the inferior vena cava (IVC) diameter, portal vein pulsatility index, hepatic venous flow, and VExUS score.

Age and Gender Distribution:

- The age group distribution was as follows: 1 month - 2 years (33.33%), 2 - 7 years (38.33%), and 7 - 17 years (28.33%).
- A higher proportion of female patients (63.33%) compared to male patients (36.67%).

Clinical Features and Treatments:

- Common clinical findings included Failure to Thrive (60%), Respiratory Distress/Failure (51.67%), and Hepatomegaly (91.67%).
- Diuretic therapy was commonly used in 85% of patients, with oxygen support administered in 50%.

Diagnosis of CHD:

- The most common diagnoses were Atrial Septal Defect (ASD, 35%), Ventricular Septal Defect (VSD, 28.33%), and Patent Ductus Arteriosus (PDA, 26.67%).

Heart Failure Classification:

- According to the Modified Ross Heart Failure Classification, the majority of patients (50%) had Class 4 heart failure, indicating severe symptoms. A smaller proportion had Class 2 (46.67%) and Class 3 (3.33%) heart failure.

Status on Discharge:

- On discharge, most patients had improved or recovered, with 63.33% recovering fully, 31.67% improving, and 5% dying.

IVC and Hemodynamic Parameters:

- The IVC expiratory diameter had a mean value of 8.25 mm, and the inspiratory diameter had a mean value of 7 mm. Both IVC diameters exhibited considerable variability across the cohort.

- The Portal Vein Pulsatility Index and Hepatic Venous Flow were also evaluated. The majority of patients showed mild portal vein pulsatility (61.67%) and normal hepatic venous flow (63.33%).

Age-Related Variations in IVC Measurements:

- Statistically significant differences were found in the IVC diameters (both expiratory and inspiratory) across different age groups, with larger diameters seen in older children.

Gender Differences in IVC Measurements:

- No statistically significant differences in IVC diameters between male and female patients were observed.

Predictive Validity of Diagnostic Indices:

- **The Portal Vein Pulsatility Index** showed high sensitivity (97.92%) and specificity (83.33%) in predicting venous congestion, while the **Hepatic**

  Venous Flow index showed high specificity (100%) but lower sensitivity (45.83%).

Venous Congestion Assessment:

- The study assessed venous congestion using multiple methods, including the IVC Collapsibility Index, the Portal Vein Pulsatility Index, and the VExUS Score.

- A large proportion of patients (70%) showed signs of venous congestion based on the IVC Collapsibility Index, while 81.67% had venous congestion on the Portal Vein Pulsatility Index. The VExUS Score identified venous congestion in 80% of patients.

Comparison of Diagnosis and VExUS Score:

- The distribution of diagnoses showed no statistically significant association with the VExUS score, though a trend was noted where patients with Ventricular Septal Defect (VSD) were more likely to have higher venous congestion.

Comparison of Modified Ross HFC and VExUS Score:

- A significant association was found between the Modified Ross Heart Failure Classification and the VExUS score, suggesting that patients with severe heart failure (Class 4) were more likely to have venous congestion ($P = 0.017$).

Comparisons and Associations of:

- **IVC Collapsibility Index and VExUS Score:** A significant association was observed between venous congestion as measured by the IVC Collapsibility Index and the VExUS Score ($P = 0.008$).

- **Portal Vein Pulsatility Index and VExUS Score:** A strong association was also found between the Portal Vein Pulsatility Index and the VExUS Score ($P < 0.001$), indicating that these indices are reliable in identifying venous congestion.

Status on Discharge and VExUS Score:

- A trend toward worse outcomes was observed for patients with higher VExUS scores (Grade 2), with a higher mortality rate (16.67%) and lower recovery rates (33.33%). However, this association was not statistically significant ($P = 0.085$).

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ANNEXURES

ANNEXURE – I - INFORMED CONSENT FORM

INFORMED CONSENT FORM

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH

COMPARISON OF VENOUS EXCESS ULTRASOUND SCORE AND INFERIOR VENA CAVA COLLAPSIBILITY INDEX IN THE ASSESSMENT OF VENOUS CONGESTION IN CONGENITAL HEART DISEASE PRESENTING WITH HEART FAILURE

Principal Investigator: _____

Name of guide: _____

Introduction: You are being invited to participate in this study to find out **COMPARISON OF VENOUS EXCESS ULTRASOUND SCORE AND INFERIOR VENA CAVA COLLAPSIBILITY INDEX IN THE ASSESSMENT OF VENOUS CONGESTION IN CONGENITAL HEART DISEASE PRESENTING WITH HEART FAILURE.** Participation of your child will help us to know the severity of venous congestion using Doppler ultrasound. The above-mentioned study will therefore help us take important clinical decisions regarding treatment of your child. Hence the present study is undertaken. Participation in this study is completely voluntary.

Explanation of procedures: In this study, you will have to answer a few prepared questions regarding your disease symptoms. If you agree to participate, then only questions will be asked to you. At any moment, you can withdraw from the study. Information will be collected using pre-tested-designed questionnaire. The child will be assessed by Doppler ultrasound upon enrollment for study. Child will be examined for clinical signs of heart failure.

Possible Benefits: The study helps in assessing venous congestion in heart failure patients.

Possible Risks: There is no risk involved in this study.

Benefits from the study: The study helps predict the development of venous congestion in pediatric heart failure patients.

Confidentiality: All the data collected will remain confidential and only aggregated data will be published. Your personal identity will not be revealed.

Withdrawal: Your participation in this study is purely voluntary. You may decide to participate or not. Even though you decide not to participate, you will not be deprived of the benefits of this study.

Costs of Participation: The cost of the study will be entirely borne by the researcher. It involves the cost of Doppler ultrasound .There will be no additional cost to you for participating in this study.

Payment of Participation: There will be no incentives to you for participating in this study.

Questions:

If you have any questions about your rights as a study participant, you may contact Dr.Harsha Hedge, Chairperson, Institutional Ethics Committee on Human Subjects' Research, J.N. Medical College, Belagavi -590010, Ph. No 0831-2473777, Extn 4052

Legal Rights: By signing this consent form; you are not waiving any of your Legal rights.

Consent statement:

"I volunteer and consent to participate in the study. I have read (or it has been read to me in the language known to me) the information sheet thoroughly. Full opportunity was given to me to ask questions. I am fully satisfied with the answers to the questions I wanted to ask. I hereby voluntarily agree to participate in this research project".

Name of the Participant	Signature of the participant or Left-HandThumb impression
Name of Investigator	Signature of investigator
Name of Witness	Signature of Witness
Date:	Place:

Assent (<18 years)

I have read the information in this form. After understanding all details about the study, I agree to give assent to be included as a volunteer in the study titled **COMPARISON OF VENOUS EXCESS ULTRASOUND SCORE AND INFERIOR VENA CAVA COLLAPSIBILITY INDEX IN THE ASSESSMENT OF VENOUS CONGESTION IN CONGENITAL HEART DISEASE PRESENTING WITH HEART FAILURE**

Name of the Participant	Signature of the participant Or Left-Hand Thumb impression
Name of the Parent	Signature of the parent
Name of Investigator	Signature of investigator
Name of Witness	Signature of Witness
Date:	Place:

ANNEXURE – II -PROFORMA

Name

IP no

Age

Sex – male/ female

Address

Urban/rural

Socioeconomic status-upper / upper middle/ middle / lower middle/lower class

Parents educational status

Mother-primary school/high school/PUC/degree/University

Father-primary school/high school/PUC /degree/university

Phone no

Details of congenital heart disease history

Age of onset

History of presenting illness-

Cough/fever/suck -rest -suck -cycle / fast breathing/ shortness of breath or

breathlessness/ forehead sweating/ bluish discolouration of oral cavity, tongue/failure

to thrive/swelling of eyelids and feet/decreased urination/ rash/ delayed development milestones/ palpitations

History of recurrent upper or lower respiratory tract infections

History of previous hospital admissions

Treatment history in past

Family history

Consanguinity- yes /no

Pedigree chart

History of cardiac disease in siblings – yes / no

Death of siblings- yes/no

Birth history

Antenatal history

History of acute or chronic infections in various trimesters-fever /rash/maternal rubella/mumps

Other maternal illness- Gestational diabetes mellitus/SLE/gestational HTN / preeclampsia

Maternal intake of drugs-

phenytoin/lithium/valproate/carbamazepine/lithium/hydantoin or alcohol Maternal exposure to radiations/ toxins

Natal history

Birthweight

APGAR score

Postnatal history

History of respiratory distress/feeding difficulty/ infections

History of NICU admission

Immunisation history

VACCINE	PRIMARY	BOOSTER
BCG		
DPT		
OPV		
HEPATITIS B		
MEASLES		
MMR		
TYPHOID		

Development history

Normal/ Developmental delay/ Developmental regression

If development delay present- global/ dissociative

EXAMINATION

General physical examination

Vitals

HR

RR

CFT

Temperature

BP of UL &LL

Anthropometry

	Measured	Expected	Percentile
Weight			
Height			
BMI			

Inference

Pallor – yes/no

Cyanosis -yes/no

Clubbing-yes/no

Oedema-yes/no

Head to toe Examination

Face

Eyes

Ears

Oral cavity

Neck

Chest

Abdomen

Extremities

Congenital markers

Skin

Dysmorphic features- yes/no

Extra cardiac anomalies- yes/no

Systemic examination

Cardiovascular system-

Peripheral

CVS

Pulse- central,

peripheral

rate, rhythm, volume, character ,condition of vessel wall

pulse deficit-yes/no radiofemoral delay- yes/no,

radioradial delay-yes/no

Signs of congestive cardiac failure – JVP-

normal/ elevated

Hepatojugular reflex-yes/no

Ascites -yes/no

Liver span- Peripheral

edema- yes/no

Signs of infective endocarditis- Fever-yes/no

Toxic look- yes/ no

Rash- yes/no

Pallor-yes/no

Clubbing-yes/no

Tachycardia-yes/no

Splinter haemorrhages- yes/no

Oslers nodes-yes/no

Janeway lesion-yes/no

Splenomegaly-yes/no

Palmar erythema-yes/no

Central CVS

Inspection-

Precordial bulge- yes/no

Visible pulsations- yes/no

Apical impulse

Palpation

Apex beat

Precordial pulsations

Heave and thrusts

Palpable p2-yes/no

Percussion

Dullness >2.5cm beyond left sternal border in left second intercostal space yes/no

Auscultation

First heart sound- loud /soft

Splitting of S1- yes/no

Second heart sound- loud /soft

Splitting of S2- yes/no , variable/fixed

Single S2-yes/no

Paradoxical splitting ofS2- yes/no

Third heart sound

Pathological S3- yes/no

Fourth heart sound- Left

atrial S4 gallop- yes/no

Right atrial S4 gallop-yes/no

Clicks/ opening snap- yes/ no

Heart murmur- yes/no

Site of origin, phase, duration, grading , pitch-

Respiratory System

Per abdomen

Hepatomegaly-yes/no

Liver span

Shifting dullness- yes/no

Fluid thrill-yes/no

Central nervous system

INVESTIGATIONS

Blood Investigations	
Hb	
Hematocrit	
Serum Creatinine	

Chest X-ray	
Echocardiography	
Venus Doppler	
IVC diameter	
IVC Collapsibility index	
VExUS	
Hepatic venous flow pattern	
Portal vein pulsatality index	
Intrarenal venous flow	
VexUS grade	

ANNEXURE III – MASTER CHART

NAME	AGE	SEX	IVC EXP DIAMETER	IVC INSP DIAMETER	IVC COLLAPSIBILITY INDEX	PORTAL VEIN PULSATALITY INDEX	HEPATIC VENOUS FLOW	VEXUS SCORE	STATUS ON DISCHARGE	MODIFIED ROSS HFC	FAILURE TO THRIVE	RESPIRATORY DISTRESS/ FAILURE	HEPATOMEGALY	PEDAL EDEMA	PULMONARY EDEMA/ CRACKLES	PLEURAL EFFUSION	OXYGEN SUPPORT	IVFLUID	DIURETIC THERAPY	DIAGNOSIS
SPANDANA DATTATRAY	10M	female	6.1mm	5.4mm	venous congestion	mild	normal	grade1	recovered	Class 3	YES	YES(distress)	YES	NO	NO	NO	NO	NO	YES	VSD
TEJASWINI BABANGOUD	9Y	female	13mm	10.9mm	normal	mild	mild	grade1	improved	Class 3	YES	YES(distress)	YES	YES	NO	NO	NO	NO	YES	ASD
ANUSHREE ASHOK	2M	female	3.7mm	3.09mm	normal	normal	normal	normal	recovered	class2	YES	NO	NO	NO	NO	NO	NO	NO	YES	VSD
LAXMI VEERABHADRA MAHALINGAPUR	10 Y	female	9.94mm	8.9mm	venous congestion	mild	normal	grade1	recovered	class 2	YES	NO	YES	NO	NO	NO	NO	NO	YES	ASD
SHRIRAKSHA RAMAPPA	1Y 10M	female	7.4mm	6.4mm	venous congestion	severe	mild	grade 2	recovered	class 4	YES	YES(distress)	YES	YES	YES	NO	YES (HFNC)	YES	YES	VSD
SOUJANYA NINGAPPA	3 Y	female	13mm	7mm	intravascular depletion	normal	normal	normal	recovered	class 2	NO	NO	YES	NO	NO	NO	NO	NO	YES	VSD
SHRINIVAS TANGADI	4 Y	male	12mm	10mm	normal	normal	normal	normal	recovered	class2	NO	NO	YES	YES	NO	NO	NO	NO	YES	PDA
MUTURAJ AMASIDDA	2M	male	8.4mm	7.6mm	venous congestion	mild	mild	grade1	recovered	class 4	NO	YES(distress)	YES	NO	NO	NO	YES(NP)	YES	YES	TAPVC
BHAIRAVI GANGARAM	8 Y	female	14.8mm	14.4mm	venous congestion	normal	normal	normal	recovered	class2	YES	NO	NO	NO	NO	NO	NO	NO	YES	ASD
SHANKAR VITTAL	7Y	male	6.43mm	5.7mm	venous congestion	mild	mild	grade1	recovered	class2	NO	NO	YES	NO	NO	NO	NO	NO	YES	PDA
AMRUTHA TUKARAM	1 Y	female	9.1mm	8mm	venous congestion	mild	mild	grade1	recovered	class4	NO	YES(distress)	YES	NO	NO	NO	YES(NP)	NO	YES	PDA
KEERTHANA MALLAPPA	6M	female	6.1mm	4mm	normal	mild	normal	grade1	improved	class 4	NO	YES(distress)	YES	NO	NO	NO	YES(NP)	YES	YES	ASD
B/O MAMATA BALACHANDRA HARIJAN	6M	female	5.2mm	4.7mm	venous congestion	mild	mild	grade1	improved	class4	NO	YES(distress)	YES	NO	NO	NO	YES(NP)	YES	YES	PDA
B/O DIVYA SUSHANT PATIL	3M	male	5.4mm	4.6mm	venous congestion	severe	mild	grade2	expired	class 4	YES	YES(failure)	YES	YES	YES	NO	YES (MV)	YES	YES	VSD
SHREYA HANAMANT	12Y	female	9mm	5mm	intravascular depletion	normal	mild	grade1	recovered	class 2	YES	NO	YES	YES	NO	NO	NO	NO	YES	ASD
B/O NANDINI NAYAK DHANASING	2M	male	5.6mm	4.1mm	normal	severe	mild	grade2	expired	class4	YES	YES(failure)	YES	YES	YES	NO	YES(mv)	YES	YES	VSD
LAHARI SIDDAPPA	3Y	female	11.5mm	8.1mm	normal	normal	normal	normal	recovered	class2	NO	NO	YES	YES	NO	NO	NO	NO	YES	ASD
SAMANVI SANTOSH	2M	female	4.2mm	3.8mm	normal	normal	normal	normal	recovered	class2	YES	NO	NO	NO	NO	NO	NO	NO	YES	PDA
AFREEN MAULALI	11Y	female	1.04cm	6.5mm	normal	mild	normal	normal	improved	class2	NO	NO	YES	NO	NO	NO	NO	NO	YES	VSD
KRUTHIKA BASAVARAJ	3Y	female	7.9mm	7.1mm	venous congestion	mild	normal	grade1	recovered	class2	YES	NO	YES	NO	NO	NO	NO	NO	YES	ASD
AAHIL AZIZ KARADI	2Y	male	7.3mm	7mm	venous congestion	mild	normal	grade1	recovered	class2	YES	NO	YES	YES	NO	NO	NO	NO	YES	PDA
RITESH YALLAPPA	9M	male	8.9mm	8.3mm	venous congestion	mild	normal	grade1	improved	class4	YES	YES(distress)	YES	NO	NO	NO	YES(NP)	YES	YES	VSD
RUPALI CHANDRAKANT	17 Y	female	13.5mm	12.6mm	venous congestion	normal	normal	normal	recovered	class2	NO	NO	YES	YES	NO	NO	NO	NO	NO	ASD
CHAITRA KALLAPPA	16 Y	female	13.8mm	11.7mm	venous congestion	mild	mild	grade1	recovered	class2	YES	NO	NO	NO	NO	NO	NO	NO	NO	VSD
RAKSHITA MALLAPPA TIPPANAVAR	10 Y	female	8.3mm	7.9mm	venous congestion	mild	normal	grade1	recovered	class2	YES	NO	YES	YES	NO	NO	NO	NO	NO	PDA
SHARAT PUNDLIK MADAR	1Y	male	6.4mm	5mm	normal	mild	normal	grade1	recovered	class2	YES	NO	YES	NO	NO	NO	NO	NO	YES	ASD
LAVANYA MAHADEV ODEYAR	3Y	female	5.8mm	5.1mm	venous congestion	mild	normal	grade1	recovered	class2	YES	NO	YES	YES	NO	NO	NO	NO	YES	ASD
BIBI SAKINA FAKRUSAB JAMADAR	5 Y	female	7mm	6.7mm	venous congestion	mild	normal	grade1	recovered	class2	NO	NO	YES	NO	NO	NO	NO	NO	YES	ASD

RACHIT ANAND JANAGOUDA	3 Y	male	4.3mm	4mm	venous congestion	mild	normal	grade1	recovered	class2	YES	NO	YES	YES	NO	NO	NO	NO	YES	VSD
FATIMAZAHRE SHARIQUE PARISHWAD	7 y	female	9.7mm	7.9mm	normal	mild	normal	grade1	recovered	class2	NO	NO	YES	NO	NO	NO	NO	NO	NO	PDA
SANDEEP SHRISHAIL VADDAR	6Y	male	8.6mm	7.8mm	venous congestion	mild	normal	grade 1	recovered	class4	YES	YES(distress)	YES	NO	NO	NO	YES(NP)	YES	YES	PDA
ADITYA CHIDANAND YARANAL	3Y	male	4.2mm	2.2mm	intravascular depletion	normal	normal	normal	improved	class4	NO	yes (distress)	YES	NO	NO	NO	YES(NP)	YES	YES	TR
MANJULA VITTAL KAMANE	5Y	female	7.1mm	6.2mm	venous congestion	mild	normal	grade1	recovered	class2	YES	NO	YES	NO	NO	NO	NO	NO	NO	PDA
ZOYA ZAHEER KHATIB	11Y	female	8.9mm	8.1mm	venous congestion	mild	mild	grade1	expired	class4	YES	YES(failure)	yes	YES	NO	YES	YES(MV)	YES	YES	ASD
SHIVPRIYA GANGARAM KAMBALE	4Y	female	5.3mm	5.1mm	venous congestion	mild	normal	grade1	recovered	class2	NO	NO	YES	NO	NO	NO	NO	NO	YES	ASD
HARSHITA YALLAPPA MUNDAGI	2Y	female	6.5mm	6mm	venous congestion	severe	mild	grade2	recovered	class4	YES	YES(distress)	YES	YES	NO	NO	YES(HFNC)	YES	YES	VSD
SANVI SIDARAYI PUJERI	3Y	female	6.8mm	5.5mm	normal	normal	normal	normal	recovered	class2	YES	NO	NO	YES	NO	NO	NO	yes	YES	VSD
VINAYAK RAMESH BHOI	14Y	male	8.2mm	6.9mm	venous congestion	mild	mild	grade1	recovered	class4	YES	YES	YES	YES	NO	NO	YES(NP)	YES	YES	VSD
KUSHAL SHIVANANDA KOLAKAR	6Y	male	9.7mm	8.5mm	venous congestion	mild	normal	grade1	recovered	class4	NO	NO	YES	NO	NO	NO	yes(NP)	YES	YES	ASD
AMEET SANJU SOLLAPURE	6Y	male	5.4mm	4.7mm	venous congestion	mild	mild	grade1	recovered	class4	NO	YES(distress)	YES	NO	NO	NO	yes(NP)	YES	NO	PDA
AZAAN MALIKSAB NADAF	6M	male	4.5mm	4.4mm	venous congestion	severe	mild	grade2	improved	class4	YES	YES(distress)	YES	NO	NO	NO	yes(hfnc)	YES	YES	VSD
GOUTAMI UMESH BIDARI	6Y	female	8.8mm	7.6mm	venous congestion	mild	normal	grade1	recovered	class4	YES	yes(distress)	YES	NO	NO	NO	yes(np)	YES	NO	ASD
ADVITA MAHANTESH SASALATTI	5Y	female	9.6mm	8.9mm	venous congestion	severe	mild	grade2	improved	class4	YES	YES(distress)	YES	NO	NO	NO	yes(mask)	YES	YES	ASD
LAXMI MALAPPA DAWANE	7Y	female	9mm	8.5mm	venous congestion	mild	normal	grade1	improved	class4	YES	yes(distress)	YES	YES	YES	YES	yes(hfnc)	YES	YES	PDA
SANVI JAYAPAL MARABASANNAVAR	8m	female	5mm	4.3mm	venous congestion	mild	normal	grade1	improved	class4	NO	yes(distress)	YES	NO	NO	NO	yes(np)	YES	YES	PDA
SUPRIT KIRAN KAVARI	5Y	male	8.9mm	8mm	venous congestion	severe	mild	grade2	improved	class4	YES	YES(distress)	YES	YES	NO	NO	yes(nrm)	YES	YES	AR
KHUBAIB AHMADALI BARAGIR	1Y	male	3.8mm	3.5mm	venous congestion	normal	normal	normal	improved	class2	YES	NO	YES	NO	NO	NO	NO	NO	YES	ASD
SHANTAVEER VITTHAL DALWAI	6Y	male	8.6mm	7.2mm	normal	mild	normal	grade1	improved	class4	YES	yes(distress)	YES	NO	NO	NO	yes(np)	NO	YES	ASD
HARSH PRAVIN BONGALE	1M	male	6.7mm	6.5mm	venous congestion	mild	normal	normal	improved	class4	NO	YES(distress)	YES	YES	YES	NO	yes(hfnc)	YES	YES	TOF
MOHAMMAD NOORAIN ABBASALI SATTAR	14Y	male	1.43cm	1.34cm	venous congestion	severe	mild	grade2	improved	class4	YES	YES(distress)	YES	NO	NO	NO	yes(nrm)	YES	YES	VSD
MADIHA RAJESAB NADAF	4M	female	8mm	6.7mm	normal	severe	mild	grade2	recovered	class4	NO	YES(distress)	YES	YES	NO	NO	yes(mask)	YES	YES	PDA
JYOTHI LAMANI	12y	female	8.3mm	7.3mm	venous congestion	mild	normal	grade1	improved	class4	YES	YES(distress)	YES	NO	NO	NO	yes(np)	YES	YES	PDA
AISHWARYA ANIL MATHAD	11y	female	8.6mm	7.5mm	venous congestion	severe	mild	grade2	improved	class4	YES	YES(distress)	YES	YES	NO	NO	yes(hfnc)	YES	YES	ASD
ANVISHA SANTHOSH HATTI	2Y	female	6.9mm	6.2mm	venous congestion	severe	mild	grade2	improved	class4	NO	yes(distress)	YES	NO	NO	NO	yes(np)	YES	YES	ASD
AYISHA MAIBUB NAGATHAN	4Y	female	6.4mm	5.7mm	venous congestion	mild	normal	grade1	recovered	class2	NO	NO	YES	YES	NO	NO	NO	NO	YES	ASD
BHARATI SIDDAPPA MULIMANI	8Y	female	9.9mm	7.1mm	normal	severe	mild	grade2	recovered	class4	YES	yes (distress)	YES	YES	NO	NO	yes(hfnc)	YES	YES	VSD
ALFIYA SAIDUSAB GUDDADAMANI	12Y	female	1.18cm	1.12cm	venous congestion	mild	normal	grade1	recovered	class4	NO	yes(distress	YES	NO	NO	NO	yes(np)	NO	NO	VSD
KAVERI CHANDRAPPA KIVUDANNANAVAR	16y	female	1.15cm	1.11cm	venous congestion	mild	normal	grade1	recovered	class2	NO	NO	YES	YES	NO	NO	NO	NO	NO	TOF
BASU SURESH KONNUR	9Y	male	1.19cm	5.4mm	intravascular depletion	mild	normal	grade1	improved	class2	NO	NO	YES	YES	NO	NO	NO	NO	YES	MVP
SUDHARSHAN DHAREPPA MAGADUM	5Y	male	7.7mm	7.1mm	venous congestion	mild	normal	grade1	recovered	class2	YES	NO	YES	YES	NO	NO	NO	NO	YES	PDA