"TO STUDY CARDIAC STATUS BY ECHOCARDIOGRAPHY IN CIRRHOSIS OF LIVER PATIENTS AND ITS CORRELATION WITH CHILD PUGH SCORE"

By

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DOCTOR OF MEDICINE
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I hereby declare that this dissertation entitled, "**TO STUDY CARDIAC STATUS BY ECHOCARDIOGRAPHY IN CIRRHOSIS OF LIVER PATIENTS AND ITS CORRELATION WITH CHILD PUGH SCORE"** is a bonafide and genuine research work carried out by me under the guidance of

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LIST OF ABBREVIATIONS USED

2D-STE	:	Two-Dimensional Speckle-Tracking Echocardiography
AD	:	Autonomic Dysfunction
ANP	:	Atrial Natriuretic Peptide
BNP	:	Brain Natriuretic Peptide
CCM	:	Cirrhotic Cardiomyopathy
CI	:	Chronotropic Incompetence
CLD	:	Chronic Liver Disease
CMO	:	Carbon Monoxide
CMR	:	Cardiac Magnetic Resonance Imaging
CO	:	Cardiac Output
CPT	:	Child-Pugh-Turcotte
CT	:	Computerized Tomography
DNA	:	Deoxyribonucleic Acid
DT	:	Deceleration Time
EF	:	Ejection Fraction
HCC	:	Hepatocellular Carcinoma
HCV	:	Hepatitis C Virus
HR	:	Heart Rate
HSC	:	Hepatic Stellate Cells
IVRT	:	Isovolumic Relaxation Time
LV	:	Left Ventricular
LVDD	:	Left Ventricular Diastolic Dysfunction
LVSF	:	Left Ventricular Systolic Function

LVSW	:	Left Ventricular Stroke Work
MChR	:	Muscarinic Acetylcholine Receptors
MELD	:	Model For End-Stage Liver Disease
MRI	:	Magnetic Resonance Imaging
NASH	:	Nonalcoholic Steatohepatitis
NO	:	Nitric Oxide
PKA	:	Protein Kinase A
PRA	:	Plasma Renin Activity
RAAS	:	Renin Angiotensin Aldosterone System
SNS	:	Sympathetic Nervous System
SV	:	Stroke Volume
TDI	:	Tissue Doppler Imaging
TIPS	:	Transjugular Intrahepatic Portosystemic Shunt
βAR	:	Badrenergic Receptors

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ABSTRACT

Background: Cirrhosis of liver is the end result of all chronic liver disease. The severity of liver cirrhosis can be assessed by child-Pugh score. Cardiac abnormalities in cirrhosis are usually attributed to the toxic effect of alcohol on the heart. However, it is increasingly recognised that cirrhosis per se can cause cardiac dysfunction. Diastolic dysfunction is present in the vast majority of patients with cirrhotic cardiomyopathy, and simple echocardiographic indices such as the E/A (early/late diastolic volume) ratio may detect diastolic dysfunction even at rest.

Objective of the study: To study cardiac status by echocardiography in cirrhosis of liver patients and its correlation with child pugh score

Methods: This was a prospective cross sectional study was conducted on 60 Patients admitted with diagnosis of Cirrhosis of liver to Raichur Institute of Medical Sciences, Raichur during Period of August 2022 to February 2024. Adult patients with diagnosis of Cirrhosis of liver of any etiology were included in the study. Detailed history and clinical examination of the patient was collected in a pre designed proforma and all basic investigations were done. Data collected was entered into Microsoft excel sheet after appropriate data filtration. Date was analysed using SPSS version 26 software. Chi-square test ‘p’ value of less than 0.05 was considered significant.

Results: The majority of cases fall within the 30-50 years age group (55%). The highest percentage of Child pugh Class C patients are found in the 50-70 years age

group. 98% of the cases being male and only 2% female. The study showed 75% of patients with cirrhosis of liver had left ventricular diastolic dysfunction(LVDD). Grade 1 is the most common with 58%, followed by Grade 2 (15%), Grade 3 (2%), and Nil (25%). Left ventricular diastolic dysfunction is more prevalent in higher Child PUGH classes with significant association. Other echo findings such as Tricuspid regurgitation, Pulmonary artery hypertension, RV dysfunction, Pericardial effusion, Reduced Left ventricular ejection fraction, Mitral regurgitation were also seen. Other parameters, such as PT, APTT, RBS, Urea, Sr. Creatinine, Sodium, and Potassium, show varying levels of significance, indicating the clinical relevance of these measures. High positive correlations was seen with PT, APTT, Urea, Sr. Cr, Sr. Bil, SGOT, SGPT, and INR indicate that these markers increase with disease severity. Negative correlation was present in Sodium and Sr. Albumin suggests that these markers decrease as the PUGH score increases, indicating worsening liver function.

Conclusion : Cardiac dysfunction is a common but often overlooked complication in patients with liver cirrhosis. In our study majority of the patients were in middle age group (30-50 years)predominantly males. Among all the 2D Echo findings, LVDD was predominantly seen and noticed early. Grade 1 LVDD was more common. Our Study demonstrate that as the Child Pugh Score increases, the severity of LVDD also increases. Therefore, it is essential for all patients with cirrhosis to undergo echocardiography to evaluate the extent of left ventricular diastolic dysfunction, which serves as a predictor of poor prognosis in these patients.

Keywords: LVDD; Cardiac dysfunction; echocardiography; cirrhosis of liver ; child pugh score;

INTRODUCTION

Cirrhosis of liver refers to a progressive condition that disrupts the normal architecture of the liver. Chronic liver disease(CLD) is a pathological entity which is associated with a spectrum of clinical manifestations. Cirrhosis is the end result of all chronic liver disease. Interactions between the functions of the heart and the liver have been described, with liver diseases affecting the heart, heart diseases affecting the liver, and conditions that simultaneously affect both. Results of experimental and clinical studies have shown impaired myocardial contractility as well as electrophysiological abnormalities in patients with cirrhosis.[1]

Alcohol being one of the most common causes of liver cirrhosis can itself cause cardiomyopathy, which is termed as “Alcoholic cardiomyopathy”. These abnormalities were initially thought to be a manifestation of alcoholic cardiomyopathy. But in the mid 1980’s, studies in nonalcoholic patients and in experimental animal models showed a similar pattern of blunted cardiac contractile responsiveness.[2-4] Thus these cardiovascular changes are grouped as a separate entity and now termed Cirrhotic cardiomyopathy.[5-7]

The severity of liver cirrhosis can be assessed by child-Pugh score and model for end-stage liver disease (MELD) score. Child-Pugh score has higher sensitivity than MELD score. Child-Pugh score depends on serum bilirubin, ascites, encephalopathy, serum albumin levels and prothrombin time.[8]

Cardiac abnormalities in cirrhosis are usually attributed to the toxic effect of alcohol on the heart. However, it is increasingly recognised that cirrhosis per se can cause cardiac dysfunction.

Diastolic dysfunction is present in the vast majority of patients with cirrhotic cardiomyopathy, and simple echocardiographic indices such as the E/A (early/late diastolic volume) ratio may detect diastolic dysfunction even at rest. This may therefore represent the best available screening tool to diagnose cardiac dysfunction.[9]

Hence, this study was undertaken with the objective to assess the cardiac status by echocardiographically in patients with cirrhosis of liver and its correlation with child pugh score[10].

OBJECTIVES OF THE STUDY

- To study cardiac status by echocardiography in cirrhosis of liver patients and its correlation with child pugh score.

REVIEW OF LITERATURE

HISTORICAL ASPECTS:

- The word "cirrhosis" was derived from the Greek term kirrhos, meaning "tawny" (to describe the orange-yellow or tan color of the diseased liver).
- John Browne (1642–1700) was an English surgeon who first described cirrhosis, in 1685, as the “liver appearing glandoulous”. He also provided the first description of necrotising pancreatitis, in 1684**.**
- In 1761, the transformation of the liver in cirrhotic patients was identified by the first anatomic pathologist, Gianbattista Morgagni after conducting 500 autopsies.
- In 1819, René Laennec coined the term "cirrhosis" in a brief footnote to his 1819 treatise about auscultation.
- The term cirrhosis was overlooked until William Osler mentioned it as so-called the cirrhosis of Laennec in Principles and Practice of Medicine,[11]
- In 1851, primary biliary cirrhosis was described by Addison and Gull at Guy's Hospital Report, the title of the study being, "On a certain affectation of skin-vitiligoiedea-alpha plana-beta tuberosa".
- In 1892, Hanot reported a similar study on primary biliary cirrhosis which was titled "La cirrhosis hypertrophique avec ictere et chroniqe".
- In 1930, the first theory explaining the pathogenesis of this disorder was presented by Roessle and terms such as parenchymal degeneration, regeneration and scarring were mentioned.
- In 1950, Ahrens et al. coined the term primary biliary cirrhosis.[12]

DEFINITION:

Cirrhosis is an irreversible liver disease with definable pathological changes such as chronic damage of the hepatocytes which is not reversible, fibrosis of the liver distorting the architecture and reactive nodular regeneration. Cirrhosis is defined anatomically as a diffuse process with fibrosis and nodule formation.

Cirrhosis of Liver is defined as the histological development of regenerative nodules surrounded by fibrous bands in response to chronic liver injury, that leads to portal hypertension and end stage liver disease.

At present, liver transplantation remains the only curative option for a selected group of patients, but pharmacological therapies that can halt progression to decompensated cirrhosis or even reverse cirrhosis are currently being developed[13].

Pathogenesis and pathophysiology of cirrhosis:

Fibrosis describes encapsulation or replacement of injured tissue by a collagenous scar. Liver fibrosis results from the perpetuation of the normal wound healing response resulting in an abnormal continuation of fibrogenesis (connective tissue production and deposition). Fibrosis progresses at variable rates depending on the cause of liver disease, environmental and host factors. Cirrhosis is an advanced stage of liver fibrosis that is accompanied by distortion of the hepatic vasculature. It leads to shunting of the portal and arterial blood supply directly into the hepatic outflow (central veins), compromising exchange between hepatic sinusoids and the adjacent liver parenchyma, i.e., hepatocytes. The hepatic sinusoids are lined by fenestrated endothelia which rest on a sheet of permeable connective tissue (the space of Disse) which contains hepatic stellate cells (HSC) and some mononuclear cells. The other side of the space of Disse is lined by hepatocytes which execute most of the known

liver functions. In cirrhosis the space of Disse is filled with scar tissue and endothelial fenestrations are lost, a process termed sinusoidal capillarization. Histologically, cirrhosis is characterized by vascularized fibrotic septa that link portal tracts with each other and with central veins, leading to hepatocyte islands that are surrounded by fibrotic septa and which are devoid of a central vein (Figure 1). The major clinical consequences of cirrhosis are impaired hepatocyte (liver) function, an increased intrahepatic resistance (portal hypertension) and the development of hepatocellular carcinoma (HCC). The general circulatory abnormalities in cirrhosis (splanchnic vasodilation, vasoconstriction and hypoperfusion of kidneys, water and salt retention, increased cardiac output) are intimately linked to the hepatic vascular alterations and the resulting portal hypertension. Cirrhosis and its associated vascular distortion are traditionally considered to be irreversible but recent data suggest that cirrhosis regression or even reversal is possible.[14,15]

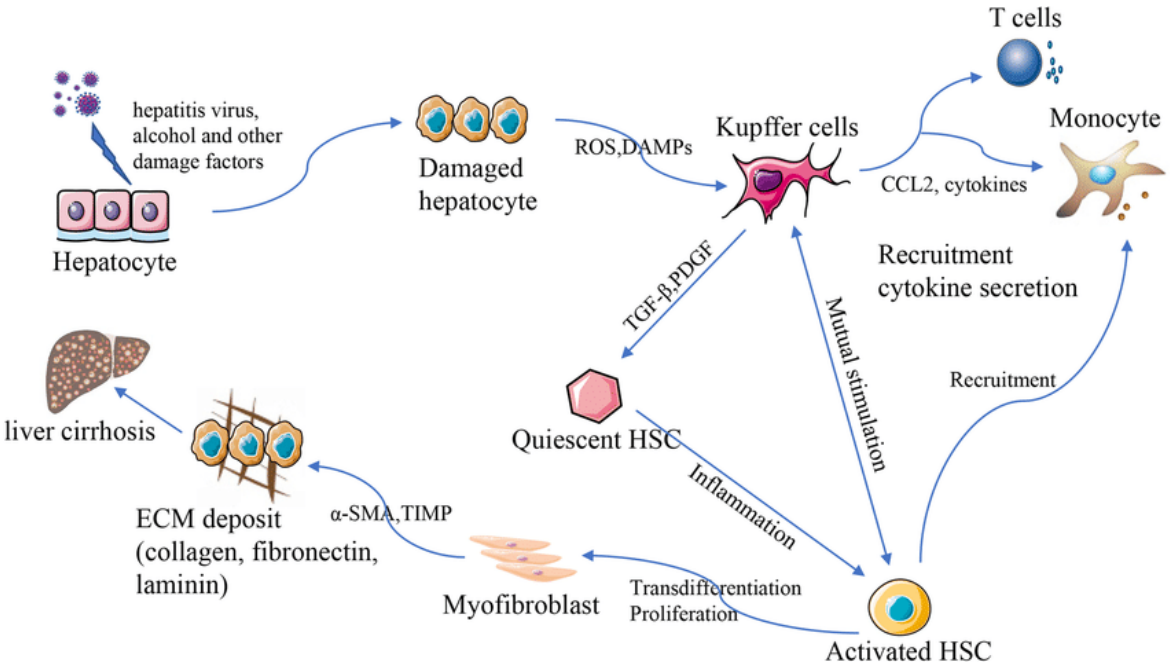

Figure 1: Pathogenesis of liver cirrhosis(From Harrison 21st edition textbook)

EPIDEMIOLOGY:

Chronic liver disease is a major cause of mortality and morbidity worldwide, accounting for approximately 2 million deaths per year. Moreover, there has been a 46% increase in cirrhosis mortality in the world from 1980 to 2023. The major causes of cirrhosis of liver are alcohol and viral hepatitis.[16] Other causes of liver cirrhosis include autoimmune diseases, fatty liver disease, and several inherited metabolic disorders.

In India too, cirrhosis of liver is a major health problem. According to the latest WHO data published in 2023, liver disease deaths in India reached 259,749 or 2.95% of total deaths, accounting for one-fifth (18.3%) of all cirrhosis deaths globally.[17] With the rapidly growing economy and changes in lifestyle and nutrition, it is presumed that the etiological factors of liver cirrhosis in India might have changed over the past few years. It has been reported that in India, alcohol consumption increased by 55% from 1992 to 2022 with doubling of per capita consumption between 2005 and 2022.

Since compensated cirrhosis often goes undetected for prolonged periods of time, a reasonable estimate is that up to 1% of populations may have histological cirrhosis[18].

ETIOLOGY OF CIRRHOSIS:

The etiology of cirrhosis are typically discerned through a combination of patient history, serological tests, and histological assessments. In the Western world, alcoholic liver disease and hepatitis C infection are predominant, whereas hepatitis B is more prevalent in Asia and sub-Saharan Africa. Since the discovery of the hepatitis C virus in 1989 and the recognition of nonalcoholic steatohepatitis (NASH) in obese

and diabetic individuals, cases of cryptogenic cirrhosis—where the cause is unclear—have become rare. Understanding the etiology of cirrhosis is crucial as it can predict complications and guide treatment decisions. Additionally, it facilitates discussions about preventive measures, such as counseling family members of patients with alcoholic cirrhosis or chronic viral hepatitis, and considering genetic testing and preventive advice for relatives of individuals with genetic disorders like hemochromatosis or Wilson's disease.

Epidemiological studies often reveal that multiple factors contribute to cirrhosis development. For instance, chronic hepatitis C is associated with regular (moderate) alcohol consumption, age over 50 years, and male gender. Meanwhile, NASH is linked to older age, obesity, insulin resistance/type 2 diabetes, hypertension, and hyperlipidemia - collectively known as features of the metabolic syndrome.[19,20]

CLINICAL PRESENTATION:

Cirrhosis often progresses silently without symptoms and may go undetected until complications of liver disease manifest. A significant number of patients remain asymptomatic and undiagnosed, with cirrhosis frequently discovered only during autopsy. The diagnosis of asymptomatic cirrhosis typically occurs when incidental screening tests, such as abnormal liver enzyme levels or radiological findings suggestive of liver disease, prompt further evaluation and liver biopsy. The awareness that approximately 20% of patients with hepatitis C virus (HCV) and possibly up to 10% of those with nonalcoholic steatohepatitis (NASH) may advance to cirrhosis has led to increased use of biopsies in these high-risk groups before clinical symptoms of cirrhosis appear. However, many patients still present clinically when their cirrhosis decompensates, often marked by severe and life-threatening complications such as

variceal hemorrhage, ascites, spontaneous bacterial peritonitis, or hepatic encephalopathy[21].

Table 1: Clinical Features of Cirrhosis(From Harrison 21st edition textbook)

GENERAL FINDINGS	**DESCRIPTION**	**ETIOLOGY**
Jaundice	Yellow discoloration of skin, cornea and mucous membranes	Compromised hepatocyte excretory function, occurs when serum bilirubin >2mg/dl
Spider angiomata	Central arteriole with tiny radiating vessels, mainly on trunk and face	Elevated estradiol, decreased estradiol degradation in liver
Nodular liver	Irregular, hard surface on palpation	Fibrosis, irregular regeneration
Splenomegaly	Enlarged on palpation or in ultrasound	Portal hypertension, splenic congestion
Ascites	Proteinaceous fluid in abdominal cavity, clinically detected when ≥1.5 L	Portal hypertension
Caput medusa	Prominent veins radiating from umbilicus	Portal hypertension, reopening of the umbilical vein that shunts blood from the portal vein
Cruveilhier Baumgarten syndrome	Epigastric vascular murmur	Shunts from portal vein to umbilical vein branches, can be present without Caput medusa
Palmar erythema	Erythema sparing the central portion of the palm	Elevated estradiol, decreased estradiol degradation in liver
White nails	Horizontal white bands	Hypoalbuminemia

GENERAL FINDINGS	DESCRIPTION	ETIOLOGY
	and/or proximal white nail plate	
Hypertrophic osteoarthropathy/Finger clubbing	Painful proliferative osteoarthropathy of long bones	Hypoxemia due to right-to-left shunting, porto-pulmonary hypertension
Dupuytren's contracture	Fibrosis and contraction of the palmar fascia	Enhanced oxidative stress, elevated hypoxanthine (alcohol exposure or diabetes)
Gynecomastia, loss of male hair pattern	Benign proliferation of glandular male breast tissue	Enhanced conversion of androstenedione to estrone and estradiol, decreased estradiol degradation in liver
Hypogonadism	Mainly in alcoholic cirrhosis and hemochromatosis	Direct toxic effect of alcohol or iron
Flapping tremor (asterixis)	Asynchronous flapping motions of dorsiflexed hands	Hepatic encephalopathy, disinhibition of motor neurons
Foetor hepaticus	Sweet, pungent smell	Volatile dimethylsulfide, especially in portosystemic shunting and liver failure
Anorexia, fatigue, weight loss, muscle wasting	Occurs in >50% of cirrhotics	Catabolic metabolism by diseased liver, secondary to anorexia
Type 2 diabetes	Occurs in 15-30% of cirrhotics	Disturbed glucose utilization and/or decreased insulin removal by the liver

Table 2: Laboratory Findings in Cirrhosis(From Harrison 21st edition textbook)

LABORATORY TEST	DESCRIPTION	ETIOLOGY
AST and ALT	Often normal or moderately elevated	Leakage from damaged hepatocytes; AST to ALT ratio often above 1, especially in alcoholic cirrhosis (relative vitamin B6 deficiency)
ALP	Elevated <3-fold, except for PBC and PSC	Cholestasis
GGT	More specific for liver than ALP, high in active alcoholics	Cholestasis
Bilirubin	Elevated later than GGT and ALP, important predictor of mortality	Cholestasis, decreased hepatocyte and renal excretory function (exacerbated by systemic inflammation)
Albumin	Decreased in advanced cirrhosis	Decreased hepatic production, sequestration into ascites and interstitium (exacerbated in systemic inflammation), DD: malnutrition, protein losing enteropathy
Prothrombin time	Decreased in advanced cirrhosis	Decreased hepatic production of factor V/VII (While thrombin production is maintained), DD: vitamin K deficiency (e.g., due to mechanical biliary obstruction)
Immune globulins	Increased (mainly IgG)	Shunting of portal venous blood carrying (intestinal) antigens to lymph tissues with resultant stimulation of plasma cells
Sodium imbalance	Hyponatremia	Unability to excrete free water via the kidneys due to increased activity of antidiuretic hormone (vasopressin 2

LABORATORY TEST	DESCRIPTION	ETIOLOGY
		receptor effect)
Anemia	Macro-, normo- or microcytic anemia	Folate deficiency, hypersplenism, direct toxicity (alcohol), gastrointestinal blood loss (e.g., via esophageal varices)
Thrombocytes and leukocytes	Thrombocytopenia (Leukopenia)	Hypersplenism, dysfibronogenemia, reduced hepatic thrombopoietin production

Imaging of cirrhosis:

Ultrasonography, computerized tomography (CT) and magnetic resonance imaging (MRI) are not sensitive to detect cirrhosis, and final diagnosis still relies on histology. However, their specificity is high when an obvious cause is present and imaging reveals an inhomogeous hepatic texture or surface, rarefied hepatic central vein, an enlarged caudate lobe, splenomegaly or collateral veins.[22] However, other etiologies such as portal vein thrombosis, parasitic diseases or hematological malignancies need to be excluded, and normal radiographic findings do not exclude compensated cirrhosis. The major role of radiography is for the detection and quantitation of complications of cirrhosis, i.e., ascites, HCC, and hepatic or portal vein thrombosis.

Ultrasonography provides important information on hepatic architecture, is cheap and widely available. Nodularity and increased echogenicity of the liver are often found in cirrhosis but are also present in steatosis.

Conventional CT and MRI are not useful to define the severity of cirrhosis, while helical CT and MRI with contrast are the modalities of choice when HCC or vascular lesions are suspected. MRI has also been shown to be effective in determining hepatic iron and fat content in hemochromatosis and liver steatosis, respectively.

Elasticity measurement (Fibroscan) is a promising technique based on the velocity of an elastic wave via an intercostally placed transmitter. Shear wave velocity is determined by pulse ultrasound and correlates with liver stiffness, i.e., fibrosis. The examination is limited by morbid obesity, ascites and small intercostal spaces. Elasticity scans have the ability to sample 1/500 of the liver and represent a useful, non-invasive test for diagnosing or excluding cirrhosis[23].

Liver biopsy:

Biopsy is considered the gold standard for diagnosis of cirrhosis, and sequential histological grading of inflammation and staging of fibrosis can assess risk of progression. However, biopsy is prone to considerable sampling variability in all liver diseases.[24] Thus when staging fibrosis in hepatitis C patients using the METAVIR system which is simple and uses only 4 stages (stage 4 being cirrhosis), one third of scores differed by at least one stage when a biopsy from the left liver lobe was compared to that from the right lobe, and with similar results for grading of inflammation. In hepatitis C, correct staging was only achieved for 65% and 75% of cases when biopsies were 15 mm and 25 mm in length, respectively, while in clinical practice only 16% of biopsies reach 25mm in length. Despite these shortcomings, biopsy is still required to confirm cirrhosis in patients with compensated liver function and to suggest its cause. Biopsy confirmation of cirrhosis is not necessary when clear signs of cirrhosis, such as ascites, coagulopathy, and a shrunken nodular appearing liver are present[25].

CLASSIFICATION SYSTEM:

Numerous studies have endeavored to devise a classification system capable of both characterizing the extent of liver damage and predicting outcomes for patients with cirrhosis based on clinical and laboratory parameters.

The Child-Pugh-Turcotte (CPT) classification is widely adopted due to its simplicity and reasonably effective predictive value.[26] For patients categorized as CPT A, B, and C, the one-year survival rates are 100%, 80%, and 45% respectively. CPT classification also forecasts the likelihood of complications such as variceal hemorrhage and the response to surgical interventions.

In recent years, the Model for End-Stage Liver Disease (MELD) has gained prominence, especially in the context of limited organ donors for transplantation. MELD more accurately assesses short-term mortality risk, predicting three-month survival across cirrhosis etiologies. It incorporates creatinine, bilirubin, and INR levels, prioritizing patients at highest risk of death without a transplant, such as those with hepatorenal syndrome.

In the United States, replacing the previous wait-time-based allocation system with MELD has reduced waiting list mortality without impacting post-transplant outcomes. Ongoing refinements consider additional factors like hepatocellular carcinoma (HCC) and hyponatremia (<130 mEq/L). Both CPT and MELD scores can fluctuate significantly with medical interventions such as albumin substitution, ascites drainage, or diuretic therapy. An increasing MELD score over time better reflects the severity and progression of cirrhosis.

Table 3: Child Pugh Turcotte (CPT) classification(From Harrison 21st edition textbook)

POINTS	**1**	**2**	**3**
Encephalopathy	absent	medically controlled	poorly controlled
Ascites	absent	controlled medically	poorly controlled
Bilirubin (mg/dL)	< 2	2-3	> 3
Albumin (g/dL)	< 3.5	2.8-3.5	< 2.8
INR	< 1.7	1.7-2.2	> 2.2

	CPT A: 5-6 POINTS	**CPT B: 7-9 POINTS**	**CPT C: 10-15 POINTS**
Life expectancy (years)	15-20	4-14	1-3
Perioperative mortality (abdominal surgery) (%)	10	30	80

INR, international normalized ratio.

Child-pugh scoring is a scoring system developed for staging cirrhotic liver disease. It is used for assessing prognosis and operative mortality of cirrhosis patients. This scoring system is based on both clinical and laboratory parameters namely the levels of bilirubin, albumin, prothrombin time prolongation, the presence of ascites and encephalopathy. Patients are classified as Child class A, B, or C according to the total score of 5–6, 7–9, and 10–15 respectively. The prognosis worsens with increasing score[27].

CIRRHOSIS AND ITS COMPLICATIONS:

Cirrhosis is a condition that is defined histopathologically and has a variety of clinical manifestations and complications, some of which can be life-threatening. In the past, it has been thought that cirrhosis was never reversible; however, it has become

apparent that when the underlying insult that has caused the cirrhosis has been removed, there can be reversal of fibrosis. This is most apparent with the successful treatment of chronic hepatitis C; however, reversal of fibrosis is also seen in patients with hemochromatosis who have been successfully treated and in patients with alcohol associate liver disease who have discontinued alcohol use.

Regardless of the cause of cirrhosis, the pathologic features consist of the development of fibrosis to the point that there is architectural distortion with the formation of regenerative nodules. This results in a decrease in hepatocellular mass, and thus function, and an alteration of blood flow. The induction of fibrosis occurs with activation of hepatic stellate cells, resulting in the formation of increased amounts of collagen and other components of the extracellular matrix.

Clinical features of cirrhosis are the result of pathologic changes and mirror the severity of the liver disease. Most hepatic pathologists provide an assessment of grading and staging when evaluating liver biopsy samples. These grading and staging schemes vary between disease states and have been developed for most conditions, including chronic viral hepatitis, nonalcoholic fatty liver disease, and primary biliary cholangitis. Advanced fibrosis usually includes bridging fibrosis with nodularity designated as stage 3 and cirrhosis designated as stage 4.

Patients who have cirrhosis have varying degrees of liver function, and clinicians need to differentiate between those who have stable, compensated cirrhosis and those who have decompensated cirrhosis. Patients who have developed ascites, hepatic encephalopathy, or variceal bleeding are classified as decompensated. They should be considered for liver transplantation, particularly if the decompensations are poorly controlled. Many of the complications of cirrhosis will require specific therapy. Portal hypertension is a significant complicating feature of decompensated

cirrhosis and is responsible for the development of ascites and bleeding from esophagogastric varices, two complications that signify decompensated cirrhosis. Loss of hepatocellular function results in jaundice, coagulation disorders, and hypoalbuminemia and contributes to the causes of portosystemic encephalopathy. The complications of cirrhosis are basically the same regardless of the etiology. Nonetheless, it is useful to classify patients by the cause of their liver disease , patients can be divided into broad groups, including those with alcohol-associated cirrhosis, cirrhosis due to chronic viral hepatitis, biliary cirrhosis, nonalcoholic fatty liver disease, and other, less common causes, such as cardiac cirrhosis, cryptogenic cirrhosis, and other miscellaneous causes[28].

Table 4: Causes of Cirrhosis(From Harrison 21st edition textbook)

Alcohol **Chronic viral hepatitis** • Hepatitis B • Hepatitis C **Autoimmune hepatitis** **Nonalcoholic steatohepatitis** **Biliary cirrhosis** • Primary biliary cholangitis • Primary sclerosing cholangitis • Autoimmune cholangiopathy	**Cardiac cirrhosis** **Inherited metabolic liver disease** • Hemochromatosis • Wilson's disease • α1 Antitrypsin deficiency • Cystic fibrosis **Cryptogenic cirrhosis**

CIRRHOSIS AND THE CARDIOVASCULAR SYSTEM:

The cardiovascular effects of cirrhosis are wide-ranging and encompass not only effects on the systemic and splanchnic circulation but also on the heart itself.

Cirrhosis involves the cardiovascular system in the following ways.

1) Cirrhotic cardiomyopathy
2) Autonomic dysfunction
3) Hyperdynamic circulation
4) QT prolongation

Cirrhosis is associated with an increased cardiac output and heart rate as well as decreased systemic peripheral vascular resistance and blood pressure. Splanchnic arterial vasodilatation and impaired autonomic activity play a role.[29]

CIRRHOTIC CARDIOMYOPATHY:

Cirrhotic cardiomyopathy is recognized with abnormal cardiac contractility particularly with pharmacological and physiological stress.A reduction in myocardial β adrenergic receptor signal transduction plays a role, perhaps due to changes in the lipid content of the cardiac plasma membrane or an inhibitory effect of jaundice on adenyl cyclase. Experimental studies have shown decreased β adrenergic receptor density and receptor desensitisation in cardiocytes of cirrhotic rats. In addition, leucocytes of cirrhotic patients also present decreased abundance of β adrenoreceptor.[30]

Heart receptor and post receptor defects are supported by the demonstration of reduced function and expression of cardiac G proteins in cirrhotic animals and impaired cardiac excitation-contraction coupling in portal hypertensive rats. Plasma membrane fluidity and ion channel function are impaired in cirrhosis.

Left ventricular(LV) wall thickness may be increased. The cause of increased cardiac wall thickness is not fully understood. But the role of renin angiotensin aldosterone system (RAAS) and adrenergic hyperactivity has been considered. While systolic function is well preserved at rest there is chronotropic and inotropic incompetence on exercise. Left ventricular ejection fraction is decreased on exercise. The failure of the ejection fraction to increase despite an increase in venous return is suggestive of diastolic dysfunction. Cardiac dysfunction may be subclinical, presenting only after liver transplantation. This may presumably be due to the reduction in after-load that accompanies the generalized vasodilatation of advanced liver disease.

Studies using brain natriuretic peptide (BNP) as a marker of high ventricular pressure reflecting Cardiac dysfunction have shown that the levels of BNP correlated closely with the severity of Cirrhosis as measured by Child- Pugh class and also the degree of Portal hypertension as measured by hepatic venous pressure gradient.Changes were seen equally in patients with alcoholic and nonalcoholic liver diseases[31].

AUTONOMIC DYSFUNCTION (AD)

Autonomic Dysfunction can contribute to the hyper dynamic circulatory syndrome in several ways. A sympatho-vagal imbalance with a prevalent parasympathetic dysfunction would lead to a defective inhibitory vagal tone on the cardiac pacemaker. An acceleration of heart rate would ensue and contribute to increased cardiac output along with an enhanced cardiac preload. Moreover, in the subset of patients with defective sympathetic function, this may contribute to the blunted

cardiovascular response to adrenergic vasoconstrictors and maneuvers enhancing the sympathoadrenergic drive, which have been described in cirrhosis.[32]

AD is commonly detected in patients with cirrhosis, and its prevalence increases in parallel with the severity of liver disease, as does the hyper dynamic circulatory syndrome. Moreover, the presence of both abnormalities heralds an adverse prognosis.

Desperate events may contribute to the development of AD, including factors affecting nerve integrity, such as alterations in lipid metabolism, vitamin E deficiency, alcohol intake, immunologic mechanisms, and retention of toxic metabolites. Moreover, vagal function may be inhibited by elevated angiotensin II production.

This abnormality of the nervous system appears to be unrelated to the toxic effects of chronic alcohol use, because cross-sectional studies have shown an equal prevalence of AN in alcohol and non–alcohol related liver disease. The autonomic neuropathy is probably related to the changes resulting from liver disease, although the precise mechanism remains unclear.

Cold pressor tests suggest that cirrhotic patients have relative hyporesponsiveness to ά-adrenoreceptor stimulation. The failure of these compensatory mechanisms in the presence of overwhelming stress may explain the apparent increase in mortality in patients with chronic liver disease and autonomic neuropathy[33].

HYPERDYNAMIC CIRCULATION:

Hyperdynamic circulation is a common and long-recognized feature of patients with advanced cirrhosis, consisting of elevated cardiac rate and output and reduced peripheral vascular resistance, so that arterial pressure is tendentially or frankly

reduced. Owing to the importance of autonomic function in cardiovascular homeostasis, it may be involved in the pathogenesis of the hyperdynamic circulation. The setting of the hyperdynamic circulatory syndrome is the pathogenetic background of complications such as renal sodium and water retention and hepatorenal syndrome.[34]

QTc PROLONGATION:

AD is associated with an impairment of free water generation and hyponatremia and is likely involved in the pathogenesis of prolonged electrocardiographic Q-T interval, a common finding in advanced cirrhosis with an adverse prognostic significance.

The QT interval is a measurement that represents the total time from ventricular depolarization to complete repolarization. The QT interval on ECG is frequently prolonged. Correction of the prolonged QT interval has been documented in post transplant patients.

Recently, Ward and colleagues described a decrease in K+ current in ventricular myocytes of cirrhotic rats, which would result in a tendency to prolong QT intervals.[35] This is in agreement with the results of Bernardi and colleagues showing a prolonged QT interval and other electrophysiological abnormalities in cardiac excitation and repolarisation in cirrhotic patients. It has been suggested that androgen deficiency may also cause Q-Tc prolongation in male patients with cirrhosis[36].

DEFINITION OF CIRRHOTIC CARDIOMYOPATHY:

According to the 2022 World Congress of Gastro- enterology, cirrhotic cardiomyopathy**(**CCM) is a chronic cardiac dysfunction characterized by impaired contractile responsive ness to stress stimuli, and/or impaired diastolic relaxation, and electrophysiological abnormalities with prolonged QT interval, in the absence of other known cardiac disease.[37,38] On the other hand, patients with cirrhosis display primarily left ventricular diastolic dysfunction (LVDD) with normal systolic function at rest.

Patients with cirrhosis develop a progressive impairment in their circulatory and cardiac function during the course of their illness. In addition to systemic circulatory dysfunction, the clinical course of patients with liver disease is complicated by a progressive impairment in heart function. Cardiac function abnormalities in cirrhosis are clinically not apparent, probably because of the low SVR presented by these patients, which reduces the cardiac afterload. Initially the impaired left ventricular (LV) performance in cirrhotic patients was thought to be due to the direct toxic effect of alcohol.[39]

FACTORS RELATED TO THE INDUCTION OF CIRRHOTIC CARDIOMYOPATHY:

Heart wall thickness changes are common in patients with cirrhosis and portal hypertension. These abnormalities may be an adaptive response to the hyperdynamic circulation and the trophic effects of several neurohumoral systems. In addition, the clinical evidence indicates a link between the degree of liver insufficiency and the severity of CCM. A recent study in advanced cirrhosis documented an association between the extent of CCM and the Model for End- Stage Liver Disease (MELD)

score. Changes in diastolic function appear most prominent in patients with severe decompensation. Patients with ascites have worse LVDD compared to those without ascites. Further, the authors showed lack of response of the LV systolic and chronotropic function to peripheral arterial vasodilation and activation of the sympathetic nervous system (SNS).[40] Therefore, hepatocellular failure and portal hypertension have been considered as possible factors for cardiac changes in patients with cirrhosis.

Cardiac dysfunction in patients with cirrhosis occurs in the setting of a circulatory dysfunction characterized by a marked splanchnic arterial vasodilation. At the initial stages of cirrhosis, the circulatory dysfunction is compensated by the development of a hyperdynamic circulation. Later, during the course of the disease, the progression of liver disease and portal hypertension results in progressive vasodilatation, leading to reduction in the effective arterial blood volume which, in turn, activates the renin-angiotensin-aldosterone system (RAAS) and the SNS. These circulatory changes can lead to the cardiac dilatation of the left chambers[41] and the development of functional changes in the heart. High norepinephrine levels are known to cause impairment of β-adrenergic receptor function.

Factors other than the SNS activity and aldosterone have been implicated in the pathogenesis of cardiac dysfunction in cirrhosis, including nitric oxide (NO), carbon monoxide (CMO) and endogenous cannabinoids. Accumulation of these substances through porto-systemic shunts could act as negative inotropic agents and also participate in the pathogenesis of LVDD in CCM.[42]

Inflammation may play an important role in the pathogenesis of cardiac dysfunction specially in decompensated cirrhosis. It has been postulated that intestinal bacterial overgrowth, altered gut permeability and bacterial translocation (i.e.,

lipopolysaccharide, bacterial DNA) from the intestinal lumen to the circulation may exert continuous pressure on the immune system. Specialized receptors of monocytes and lymphocytes recognise those factors and release inflammatory mediators such as cytokines, reactive oxygen and nitrogen species. These humoral factors may exert inhibitory effects on LV function. Cytokines can affect myocardial function via the effects on both the myocyte contractility and the extracellular matrix. Lipid metabolic abnormalities in patients with cirrhosis facilitate the incorporation of cholesterol into cell plasma membranes. The major factors which lead to the elevated membrane cholesterol content in cirrhosis are probably an increase in plasma cholesterol levels and a decrease in blood lecithin cholesterol acyltransferase activity.[43,44]

PATHOGENIC MECHANISMS:

Cardiovascular autonomic dysfunction

Cardiac contractility is regulated primarily by the SNS through β-adrenergic receptors (β AR). Cardiovascular autonomic dysfunction is frequent in advanced cirrhosis. The incidence of autonomic neuropathy varies from 35% to 80% and is related to the severity of hepatic dysfunction. Autonomic and cardiac dysfunction includes impaired baroreflex sensitivity and heart rate variability. Impaired cardiac response to standing is the most frequently abnormal test and is probably due to blunted baroreflex function in the setting of increased activity of the SNS.[45] The major triggers of the SNS overactivity appear to be baroreceptor-mediated stimulation owing to reduced central and arterial blood volumes. Enhanced sympathetic tone with increased cellular exposure to noradrenalin for longer periods may cause myocardial injury, receptor internalization, sequestration, and down regulation which results in a decrease of β-adrenergic receptor density on the plasma membrane.[46]

β -adrenergic receptor function

The βAR system is critical in modulating the contractility of cardiac muscle cells. Activation of βAR by epinephrine and norepinephrine couples with Gs protein and leads to the stimulation a membrane- bound adenylate cyclase and the subsequent release of cAMP. The second messenger, cAMP, activates a cAMP- dependent protein kinase A (PKA). PKA phosphorylates several intracellular proteins such as L-type calcium channels, phospholamban, troponin Ⅰ, ryanodine receptors thus leading to Ca2+ entering the cell. The cytosolic-free Ca2+ binds to the protein troponin C and interacts with tropomyosin between the actin and myosin filaments with allows the contraction of the myofibrils (systole)[47].

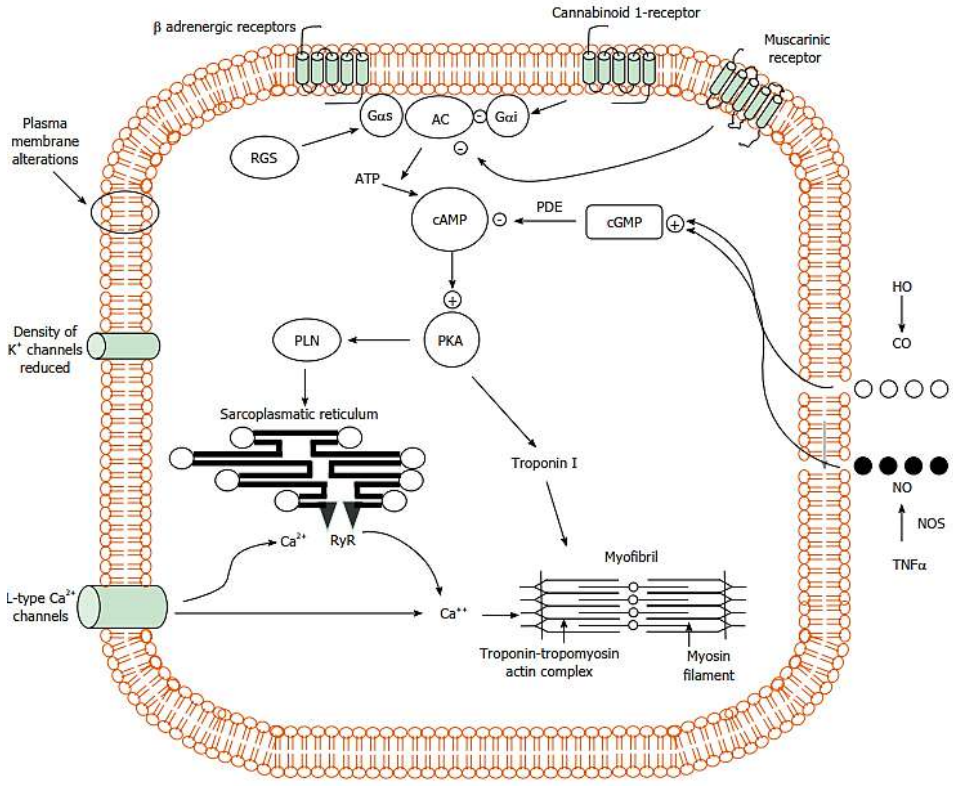

Figure 2: Pathogenic mechanisms of cardiomyocyte contraction in cirrhosis.

(From Harrison 21st edition textbook)

Membrane alteration:

Changes in membrane fluidity have also been observed in cirrhotic patients as well as in experimental cirrhosis. It has been demonstrated that the fluidity of plasma membranes from the erythrocytes in cirrhotic patients becomes more rigid and less permeable with increases in membrane cholesterol content. These metabolic abnormalities in the plasma membrane of cardiac myocyte interfere with the activation of βAR and calcium channels embedded in the membrane. Moreau et al have shown altered control of vascular tone by Ca2+ and K+ channels.[48] Such alterations in membrane properties are likely to play an important role in inducing ECG abnormalities in cirrhosis. The altered membrane fluidity may also impair stimulation of cardiac muscarinic acetylcholine receptors (M-ChR) that modulate pacemaker activity via If and IK. ACh, atrioventricular conduction, and directly or indirectly force of contraction. Alterations of cardiac M2-ChR responsiveness and defective signal transduction to cAMP has been reported in experimental cirrhosis[49].

SYSTOLIC DYSFUNCTION:

Systolic dysfunction is mostly latent in patients with cirrhosis. Although left ventricular systolic function (LVSF) at rest, assessed by invasively and non invasively methods, are normal in cirrhotic patients subtle alterations could be detected under conditions of stress or by using new echocardiographic techniques at rest. Patients with cirrhosis have documented blunted responsiveness to volume and postural challenge, exercise or pharmacological infusion. Contractile dysfunctions are common in pre-ascitic cirrhotic patients; likewise, these patients show increasing end-systolic volumes as a result of sodium loads. This involvement is more important in patients with ascites despite a decrease in both pre-load and afterload. The altered

response to active tilt in cirrhotic patients also suggests an impaired myocardial contractility. During 5 min of standing, cirrhotic patients experienced a decrease in the LV end-systolic volume, SVR and cardiac indexes despite marked increments in HR and in the activity of neurohumoral systems. On the other hand, in patients with cirrhosis there is an abnormal LV response during exercise manifested by an increase in CO and ejection fraction (EF) less than expected in relation to normal subjects.

Gould et al, have reported increasing ventricular filling pressures and an unaffected cardiac stroke index in patients undergoing exercise. Kelbaek et al also observed LV contractile function and ventricular wall compliance was reduced in cirrhotic patients. Another noninvasive tool to evaluate ventricular contractile performance is the measurement of systolic time intervals. The preejection period/left ventricular ejection time ratio has been seen to increase from baseline after exercise in cirrhotic patients[50].

DIASTOLIC DYSFUNCTION:

Abnormalities of diastolic function are an early marker of CCM. Patients with CCM display frequently LVDD. The mechanisms underlying the development of diastolic dysfunction remain unclear. Defects in the passive tension of myofilament proteins as well as impaired myocardial relaxation, possibly related to abnormalities in calcium exchange through the sarcoplasmic reticulum, may play a role in the pathogenesis of LVDD. The sarcomere is made up of filaments of various sizes and contains titin. Titin is responsible for the elasticity of the relaxed striated muscle and thus an important determinant of diastolic stiffness in cardiomyocytes. Additionally, diseases that alter diastolic function also alter the myocardial extracellular matrix. In BDL

animals, has been observed a decrease in PKA which can reduce phosphorylation of titin and increase passive tension. In addition, the levels of collagen-I mRNA in the BDL group were significantly higher than in the sham animals contributing to the pathogenesis of diastolic function.

There has been some data indicating that salt retention may play a part in the development of LVDD. LVDD in the CCM results are most likely a result of LV hypertrophy. Liver cirrhosis can lead to heart wall thickness changes. Study observed that 75% patients with LVDD and cirrhosis had cardiac hypertrophy.

Traditionally, LVDD is divided into three different filling patterns: normal, pseudonormal, and restrictive. However, conventional Doppler echocardiographic indices (E/A ratio) have clear limitations (age and load conditions) and rarely allow for the accurate differentiation between normal and pseudonormal LV diastolic pattern. TDI can overcome some of these factors. The tissue velocity recorded at the basal and septal corner of the annulus mitral (e') is a more sensitive parameter for abnormal myocardial relaxation than mitral variables. TDI velocities have demonstrated a significant correlation with invasive indices of LV relaxation and minimal effect of preload in the setting of impaired relaxation.[51]

The American Society of Echocardiography has suggested that LVDD is characterized by the presence of septal e′ < 8 cm/s, lateral e′ < 10 cm/s, mitral inflow patterns and LA volume index (LAVI) ≥ 34 mL/m2. The degree of severity can be graded according to average E/e′ ratio. The prevalence of LVDD is relatively high in patients with cirrhosis (43%-70%) despite a normal EF and is not related to the etiology of liver disease.[52]

However, a variety of comorbid conditions have been associated with development of LVDD.Therefore, these patients should be excluded in the assessment

of the prevalence of this condition in cirrhosis. Patients with tense ascites show an improvement in LVDD after paracentesis although LVDD in these patients is still abnormal as compared with healthy controls.

LVDD seems to be independently associated with the presence of bacterial endotoxin. Cirrhotic patients have elevated levels of endotoxemia due to bacterial translocation. A recent study by Karagiannakis et al showed that the severity of LVDD determined by the E/e′ ratio correlated with the serum levels of lipopolysaccharide-binding protein, a marker of exposure to bacterial endotoxin.[53]

DIAGNOSIS

The diagnosis of cirrhotic cardiomyopathy remains challenging due to the absence of specific diagnostic tools. Diagnosis can be done by various blood parameters, electrophysiological parameters, and ECHO. But in our study we are mainly focusing on ECHO.

2D ECHO

A 2D ECHO test, also known as a two-dimensional echocardiogram, is a medical imaging procedure used to evaluate the structure and function of the heart. It is a non-invasive test that utilizes ultrasound waves to create detailed images of the heart in real-time.

During a 2D echo test, a technician or a cardiologist places a small device called a transducer on the patient's chest. T he transducer emits high-frequency sound waves that bounce off the structures of the heart and create echoes. The echoes generated from the ultrasound waves are transformed into visual images, which are subsequently displayed on a monitor for examination and analysis.

The images provides valuable information about the heart's chambers, valves, and surrounding structures. It allows to assess the size, shape, and movement of the heart, as well as the functioning of the valves and the pumping of blood. It can also provide information about the presence of any abnormalities or diseases, such as heart valve disorders, congenital heart defects, heart muscle abnormalities, and fluid accumulation around the heart.

The 2D echo test is a common diagnostic tool used in cardiology to help diagnose and monitor various heart conditions. It is typically painless, non-invasive, and carries no significant risks or side effects for the patient.

An echocardiography examination begins with transthoracic 2D scanning from four standard transducer positions: the parasternal, apical, subcostal, and suprasternal windows[54].

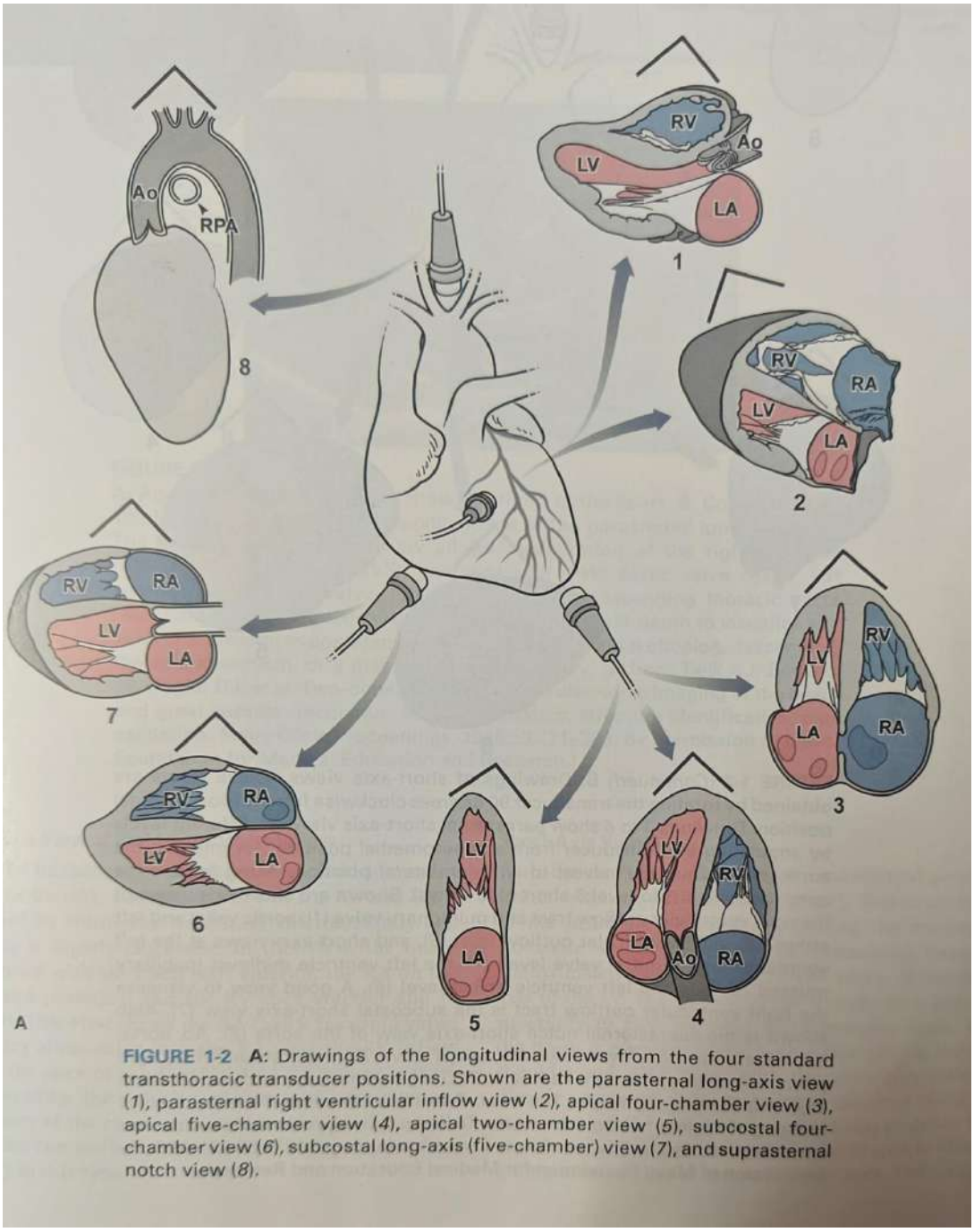

Figure 3: Transducer positions and cardiac view(Figure from south asian 2decho manual)

TABLE 5: Transducer positions and cardiac views(Table from south asian 2decho manual)

Transducer positions and cardiac views
Parasternal position Long axis view RV inflow LV outflow Short-axis view LV apex Papillary muscles (mid level) Mitral valve (basal level) Aortic valve-RV outflow Pulmonary trunk bifurcation
Apical position Four-chamber view Five-chamber (or long-axis) view Two-chamber view
Subcostal position Inferior vena cava and hepatic vein RV and LV inflow LV-aorta RV outflow
Suprasternal notch position Long-axis aorta-short-axis pulmonary artery Short-axis aorta-long-axis pulmonary artery Long-axis aorta and superior vena cava

DOPPLER ECHOCARDIOGRAPHY

Doppler echocardiography measures velocities of blood flow and myocardial tissue according to the Doppler effect .The Doppler effect describes the phenomenon that sound frequency increases as a sound source moves toward the observer (or the transducer) and decreases as the source moves away from the observer.

In the heart, the moving target can be the red blood cell or myocardial tissue such as mitral annulus. When an ultrasound beam with known frequency (fo) is reflected by the red blood cells (RBCS) or myocardial tissue, the frequency of the reflected ultrasound waves (fr) increases when the red blood cells or the myocardium is moving toward the source of ultrasound.

Doppler echocardiography is performed either by the pulsed wave or by the continuous wave .In the pulsed wave mode, a single ultrasound crystal sends and receives sound beams from a single location by placing the "sample volume." The crystal emits a short burst of ultra sound at a certain frequency (pulse repetition frequency).

The ultrasound is reflected from moving red blood cells and is received by the same crystal. Therefore, the maximal frequency shift that can be determined to one direction by pulsed wave Doppler is one-half the pulse repetition frequency; this is called the Nyquist frequency.

If the frequency shift is higher than the Nyquist frequency, aliasing occurs; that is, the Doppler spectrum or recording is cut off at the Nyquist frequency, and the remaining frequency shift (translated into velocity) is recorded on the top or bottom of the opposite side. In other words, when the highest velocity a pulsed mode can measure is 130 cm/s in one direction, any velocity higher than the aliasing velocity is recorded from the top (when the actual flow moves away from the transducer) or from the bottom (when the actual flow moves toward the transducer) of the recording screen or paper.[55]

The pulsed repetition varies frequency inversely with the depth of the sample volume: the shallower the location of the sample volume, the higher the pulsed repetition frequency and the Nyquist frequency. In the continuous wave mode, the transducer has two crystals: one to send and the other to receive the reflected waves continuously. Therefore, the maximal frequency shift that can be recorded is not limited by the pulsed repetition frequency or the Nyquist phenomenon.

Unlike pulsed wave Doppler, continuous wave Doppler measures all the frequency shifts (i.e., velocities) present along its beam path; hence, it is used to detect and record the highest flow velocity accessible. Occasionally, recording of a high-velocity flow is the first clue to an unsuspected lesion within the path of a continuous wave Doppler beam. Continuous wave Doppler is usually performed with either an image guided or a non imaging transducer[56].

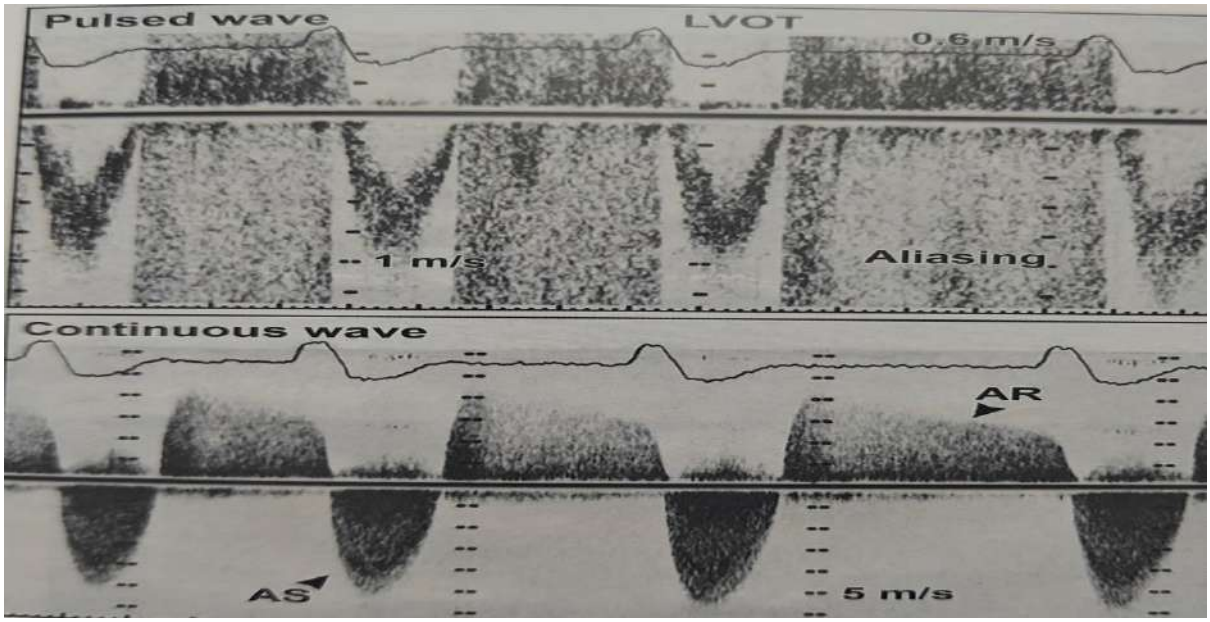

Figure 4: Pulsed wave and continuous wave doppler(Figure from south asian 2decho manual)

TISSUE DOPPLER IMAGING

Tissue Doppler imaging (TDI) records the velocity of myocardial tissue by a pulsed-wave Doppler echocardiography modified from the traditional pulsed-wave Doppler, which records and displays the velocities of blood flow with the erythrocytes as the moving targets. Their normal velocity ranges from 10 cm/s in the venous circulation to 150 cm/s in the arterial circulation. However, the velocities of myocardial tissue are much lower (1-20 cm/s), but their amplitudes are greater than those produced by blood.

Therefore, Doppler ultrasound instruments have been modified to record the low velocities of myocardial tissue and to reject the high velocities generated by blood flow. TDI requires a high frame rate. A special function key needs to be selected to activate TDI.

The subsequent operation is identical to that of regular pulsed-wave Doppler echocardiography, except that the TDI gain needs to be lowered from the regular gain setting used for blood flow Doppler recordings and the velocity scale needs to be adjusted to a lower aliasing velocity (about 20-30 cm/s or even lower) to optimize TDI signals.

Some ultrasound units adjust these variables automaticall y when the TDI function is selected. Also, TDI can be displayed in the color mode, just as in color imaging of blood flow. Tissue velocities are color coded by auto correlation: red for tissue moving toward the transducer and blue for tissue moving away from the transducer.

The ultrasound beam needs to be in parallel as much as possible with the direction of the myocardial movement, especially for the lateral annulus of the mitral and tricuspid valve. Movement and velocities of cardiac structures are regulated by the underlying systolic function and diastolic function of the heart. They are promised to be a more sensitive tool for evaluation of systolic function than ejection fraction and also are an integral part of diastolic function assessment.

Myocardial relaxation, which is the basic dia-stolic property, can be reliably assessed by tissue Doppler, and certain types of myopathies demonstrate characteristic strain imaging pattern, which is helpful in identifying myocardial pathologies. Diastolic strain abnormality persists several hours after termination of myocardial ischemia, which can be helpful during stress echocardiography or evaluation of chest pain syndrome.

Early diastolic velocity (e') of the mitral annulus measured with TDI is a reliable indicator of LV myocardial relaxation, being the cornerstone of diastolic evaluation in the new guidelines. Longitudinal motion of the mitral annulus can be appreciated visually from the parasternal long-axis and apical four-chamber views, but TDI records the velocity of the longitudinal motion in numerical value.

In the normal heart with normal myocardial relaxation, e' increases with an increasing transmitral gradient, increasing preload, exercise, and dobutamine infusion. However, when myocardial relaxation is impaired because of aging or a disease process, e' is affected less or even unchanged by preload or transmitral gradient.

Velocities of longitudinal mitral annulus motion are best obtained from apical views. Although various locations of the mitral annulus can be interrogated with TDI, the two most frequently used locations are the septal (or medial) and lateral mitra l annulus. Usually, e' from the lateral annulus is higher (normally >15 cm/s in young healthy subjects) than that from the medial annulus (normally >10 cm/s). The most r ecent 2016 guidelines recommends the use of averaged e' velocity from both annuli, but the use of e' from one location is acceptable in most clinical situations.

Late diastolic velocity (a') of the mitral annulus at the time of atrial contraction increases during early diastolic dysfunction, as is the case for the mitral inflow A wave, but decreases as atrial function deteriorates. a' has been correlated with left atrial (LA) function[57].

DIASTOLIC DYSFUNCTION

Diastolic function assessment is now an essential part of cardiac function evaluation and can be assessed reliably in most patients by echocardiography. Although there are many variables and parameters representing diastolic function, we can evaluate the presence of diastolic dysfunction and estimate diastolic filling pressures reliably using several simple measures by echocardiography.

Normal diastolic function allows adequate filling of the ventricles during rest and exercise without an abnormal increase in diastolic pressures. Adequate diastolic filling ensures normal stroke volume, according to the Frank Starling mechanism. LV filling consists of a series of hemodynamic events that are affected by numerous intrinsic and extrinsic factors.

Table 6: Factors that Influence Distensibility of the Left Ventricular (LV) Chamber During Diastole(Table from south asian 2decho manual)

Factors that Influence Distensibility of the Left Ventricular (LV) Chamber During Diastole
Factors extrinsic to LV chamber Pericardial restraint Right ventricular loading Coronary vascular turgor (erectile effect) Extrinsic compression by tumor, pleural pressure, and others
Factors intrinsic to LV chamber Passive elasticity of LV wall (stiffness or compliance when myocytes are completely relaxed) Thickness of LV wall Composition of LV wall (muscle, fibrosis, amyloid, hemosiderin), including both endocardium and myocardium Temperature, osmolality Active elasticity of LV vwall due to residual cross-bridge activation (cycling and/or latch state) through part or all of diastole Slow relaxation affecting early diastole only Incomplete relaxation affecting early, mid, and end-diastolic distensibility Diastolic tone, contracture, or rigor Elastic recoil (diastolic suction) Viscoelasticity (stress relaxation, creep)

The initial diastolic event is myocardial relaxation, an active energy-dependent process that causes pressure to decrease rapidly in the LV after the end of contraction and during early diastole, ready for sucking necessary blood volume into the LV.

The normal cycle of cardiac contraction and relaxation requires a precise, transient increase and decrease in the intracellular concentration of calcium ions. The sarcoplasmic reticulum helps orchestrate the movement of calcium during each contraction and each relaxation.

The contraction of cardiac muscle is initiated by the cellular action potential that causes the opening of L-type sarcolemma calcium channels through which calcium ions enter the cytosol. These calcium ions bind to troponin C, which ultimately disinhibits the interaction of actin and myosin and results in the formation of cross-bridges .

Myocardial relaxation is removal of accomplished primarily by the calcium ions from troponin C by an enzyme in the sarcoplasmic reticulum, called sarcoplasmic reticulum calcium adenosine triphosphatase (SERCA,), and the sarcolemmal sodium-calcium exchanger. In humans, approximately 75% of calcium ions are removed by SERCA, and 25% by the sodium-calcium exchanger[58].

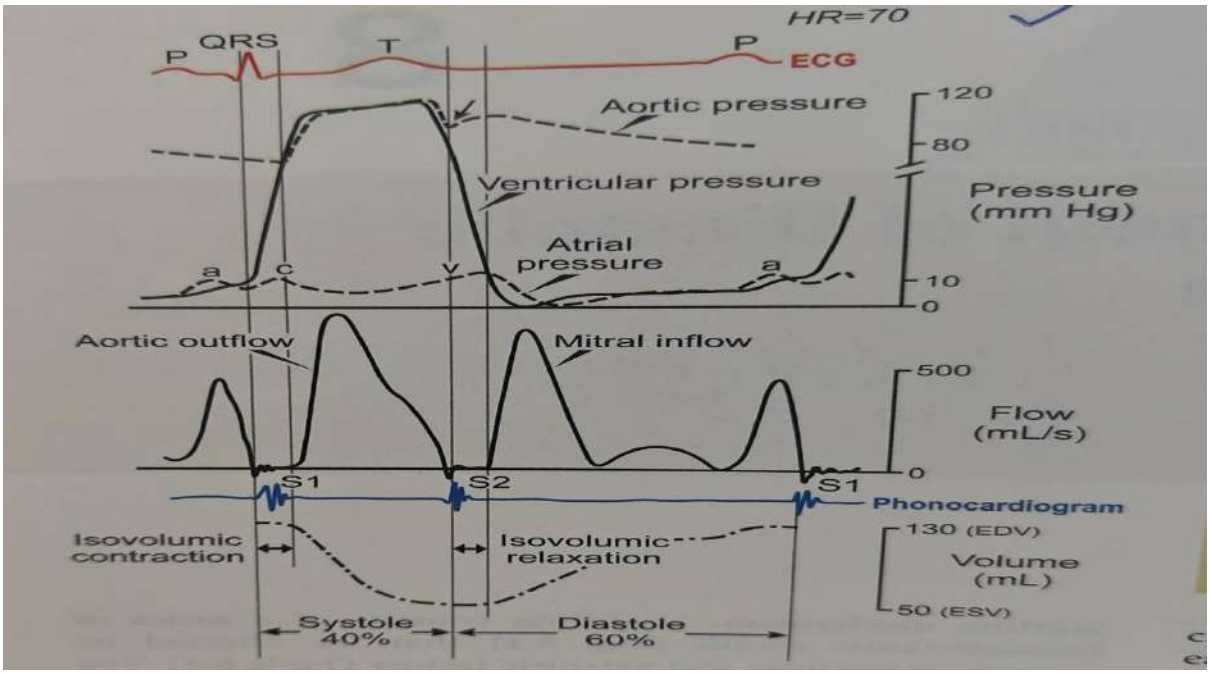

Figure 5: Diagram of intracardiac pressures(top), aortic outflow and mitral inflow(middle), and volumetric changes in left ventricle(bottom) (Figure from south asian 2decho manual).

DOPPLER FLOW VELOCITIES

Mitral Flow Velocities

Diastolic filling usually is classified initially on the basis of the peak mitral flow velocity of the early rapid filling wave (E),peak velocity of the late filling wave due to atrial contraction(A), and the E/A ratio .To measure E And A velocities reliably, the Doppler velocity recording should be satisfactory.

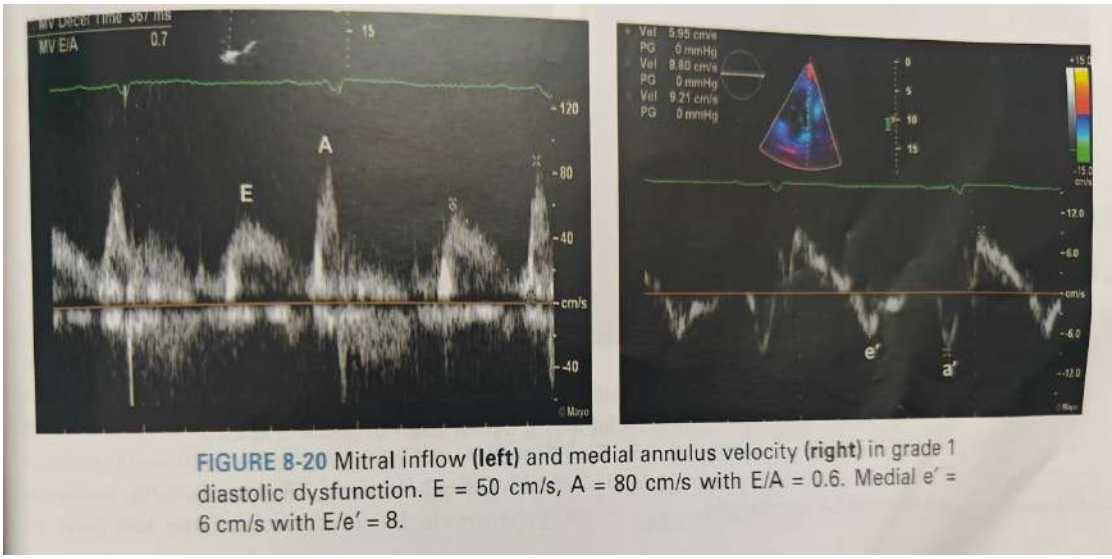

FIGURE 8-20 Mitral inflow **(left)** and medial annulus velocity **(right)** in grade 1 diastolic dysfunction. E = 50 cm/s, A = 80 cm/s with E/A = 0.6. Medial e′ = 6 cm/s with E/e′ = 8.

Figure 6: DOPPLER FLOW VELOCITIES(Figure from south asian 2decho manual)

In patients with a relaxation abnormality as the predominant diastolic dysfunction, DT is prolonged because, with a slower and continued decrease in LV pressure until mid to late diastole, it takes longer for LA and LV pressures to equilibrate. DT is shortened if there is rapid filling due to vigorous LV relaxation and elastic recoil, as in normal young subjects, or, conversely, if there is a decrease in LV compliance or marked increase in LA pressure[59].

Mitral Annulus Velocities

Mitral Annulus velocities are recorded from the apical four- chamber view by placing a 2- to 5-mm sample volume over lateral or medial portion of the mitral annulus using tissue Doppler imaging. Normally, three distinct velocities are recognized: systolic (s'), early diastolic (e), and late diastolic (a') velocities. Additional isovolumic or mid-diastolic velocities may be recorded. E' velocity has been shown to correlate well with invasive measure of myocardial relaxation,

In normal subjects, e' increases as the transmitral gradient increases with exertional or increased preload; however, in patients with impaired myocardial relaxation, e is reduced at baseline and does not increase as much as in normal subjects with increased preload .Therefore, a decrease in e' is one of the earliest markers for diastolic dysfunction, and this decrease is present in all stages of diastolic dysfunction.

At Mayo Clinic, the annulus velocities are obtained from mitral lateral, tricuspid lateral, and medial annulus, but the medial annulus e' velocity is mainly used for diastolic function assessment and estimation of filling pressure.

To measure IVRT (i.e., the interval from aortic valve closure to mitral valve opening), a 3- to 4-mm sample volume is placed in the area of the mitral leaflet tips. Next, the transducer beam is angulated toward the LV outflow tract until aortic valve closure appears above and below the baseline. An alternative technique is to use continuous wave Doppler echocardiography to record aortic and mitral flow simultaneously[60].

GRADING OF DIASTOLIC DYSFUNCTION

The Mayo Clinic Echocardiography Laboratory proposed a grading system (grading 1 to 4) similar to that of the New York Heart Association Functional Classification.

Grade l = impaired relaxation pattern.

Grade 2 = pseudonormalized pattern with moderately increased filling pressure.

Grade 3 = reversible restrictive pattern with severely increased filling pressure.

Grade 4 = irreversible restrictive pattern with severely increased filling pressure.

Normal Diastolic Filling Pattern

In normal young subjects, LV elastic recoil is vigorous and myocardial relaxation is swift; therefore, most filling is completed during early diastole, with only a small contribution at atrial contraction .Therefore, E/A is usually >1.5, septal e' > 10 cm/s.

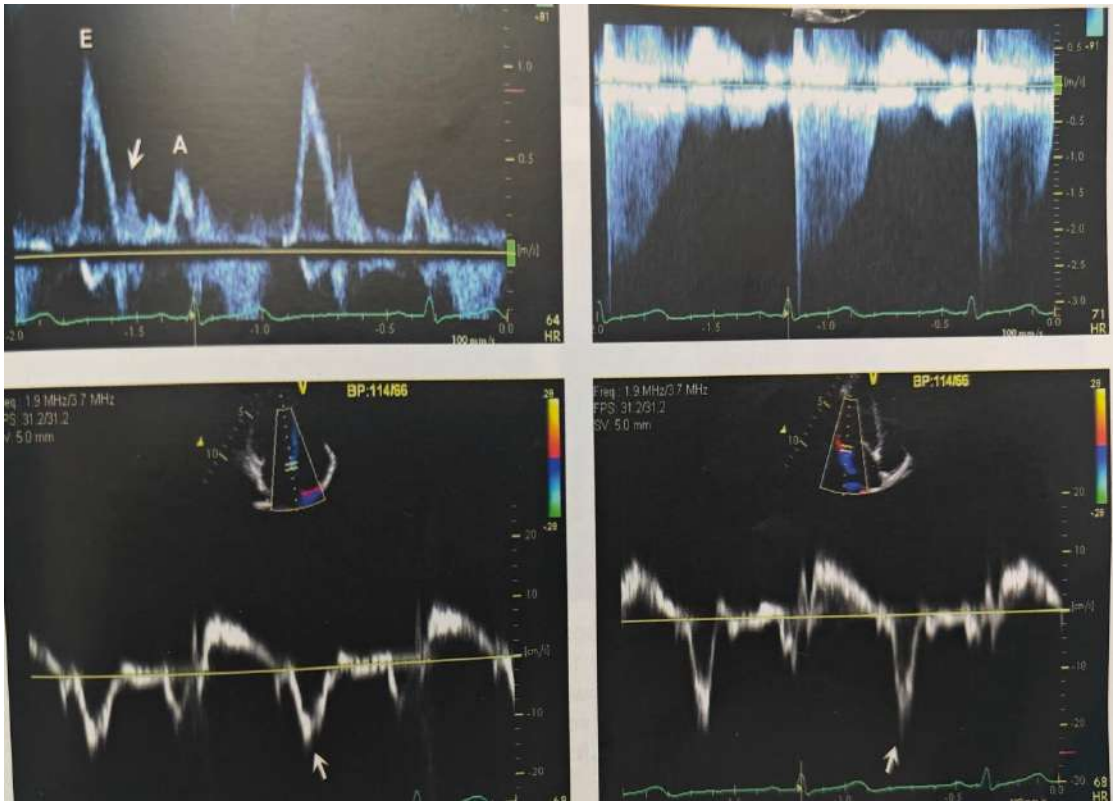

Figure 7: Normal Diastolic Filling Pattern(Figure from south asian 2decho manual)

Grade 1 diastolic Dysfunction

With delayed or impaired myocardial relaxation, e' is reduced; septal e<7 cm/s and lateral e' < 10 cm/s. Mitral E velocity is decreased and A velocity is increased, producing an E/A Ratio < 0.8, with prolonged DT.

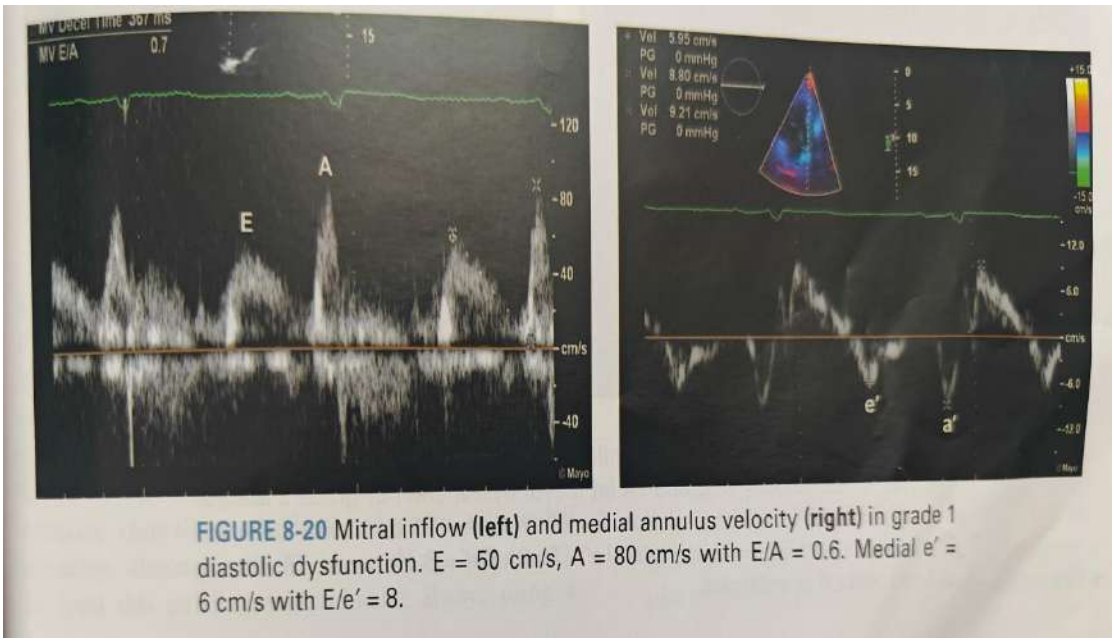

FIGURE 8-20 Mitral inflow **(left)** and medial annulus velocity **(right)** in grade 1 diastolic dysfunction. E = 50 cm/s, A = 80 cm/s with E/A = 0.6. Medial e′ = 6 cm/s with E/e′ = 8.

Figure 8: Grade 1 Diastolic Dysfunction(Figure from south asian 2decho manual)

Grade 2 Diastolic Dysfunction

As diastolic function deteriorates, the mitral inflow pattern goes through a phase that resembles a normal diastolic filling pattern, that is, the E/A 0.8 to 2.0, and DT is normal at 160 to 240 milliseconds. This is the result of a mild to moderately increased LA pressure superimposed on a relaxation abnormality. This is referred to as the "pseudonormalized" mitral flow filling pattern.

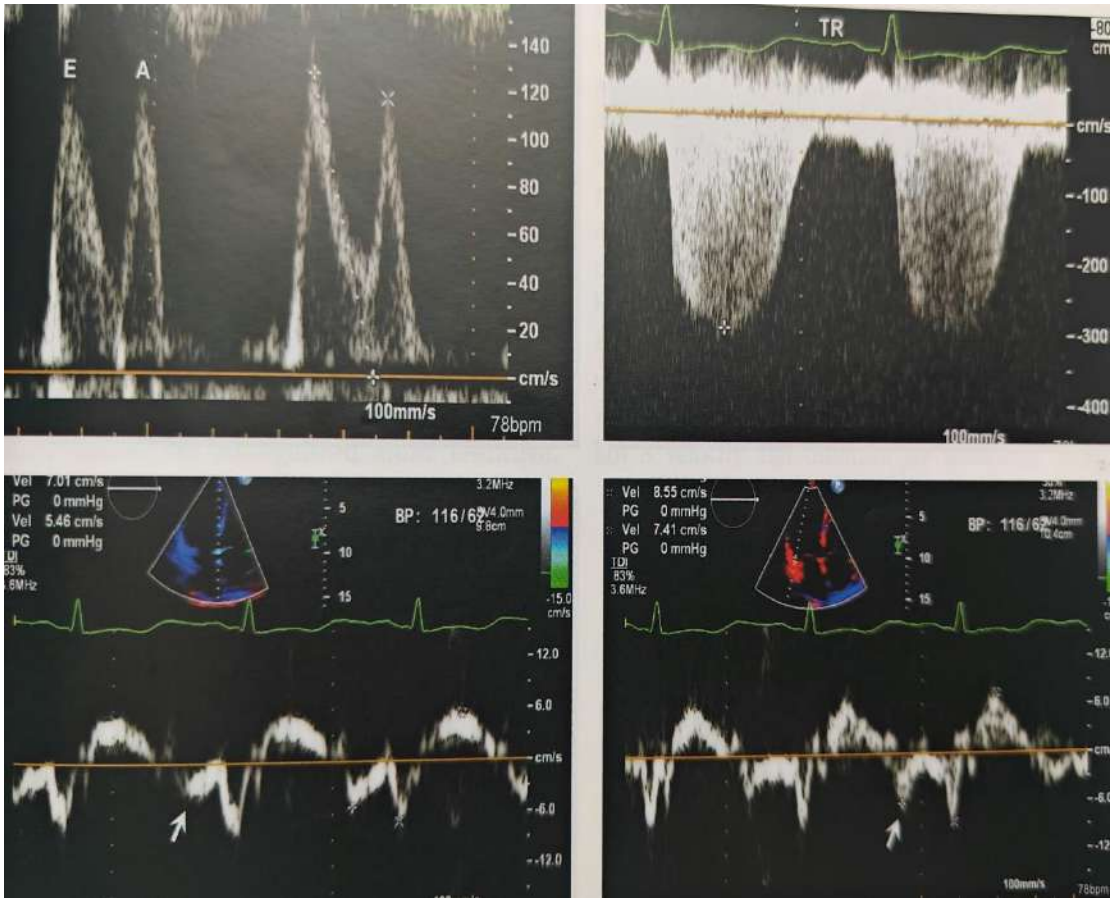

Figure 9: Grade 2 Diastolic Dysfunction (Figure from south asian 2decho manual)

Grade 3 diastolic dysfunction

The term restrictive diastolic filling or restrictive physiology, should be distinguished from restrictive cardiomyopathy. Restrictive physiology can be present in any cardiac abnormality or in a combination of abnormalities that produce decreased LV compliance and markedly increased LA pressure.

Examples include decompensated congestive systolic heart failure, advanced restrictive cardiomyopathy, Severe coronary artery disease, acute severe aortic regurgitation, and constrictive pericarditis.

Increased LA pressure shortens IVRT, and high E velocity. Early diastolic filling in a noncompliant LV causes a rapid increase in early LV diastolic pressure, with rapid equalization of LV and LA pressures producing a shortened DT. Atrial contraction increases LA pressure, but A velocity and duration are shortened because LV pressure increases even more rapidly[61].

When LV diastolic pressure is markedly increased, there may be diastolic mitral regurgitation during mid-diastole or with atria relaxation. Therefore, restrictive restrictive physiology or Grade 3 diastolic dysfunction is characterized by mitral flow velocities that show increased E velocity, decreased A velocity (E/A > 2.0), and shortened DT (<I60 milliseconds) and IVRT (<70 milliseconds). It should be emphasized however, that myocardial relaxation continues to be impaired in patients with restrictive filling and mitral annulus e' velocity is expected be very low (medial e < 7 cm/s, and usually <5 cm/s) with E/e > 15.

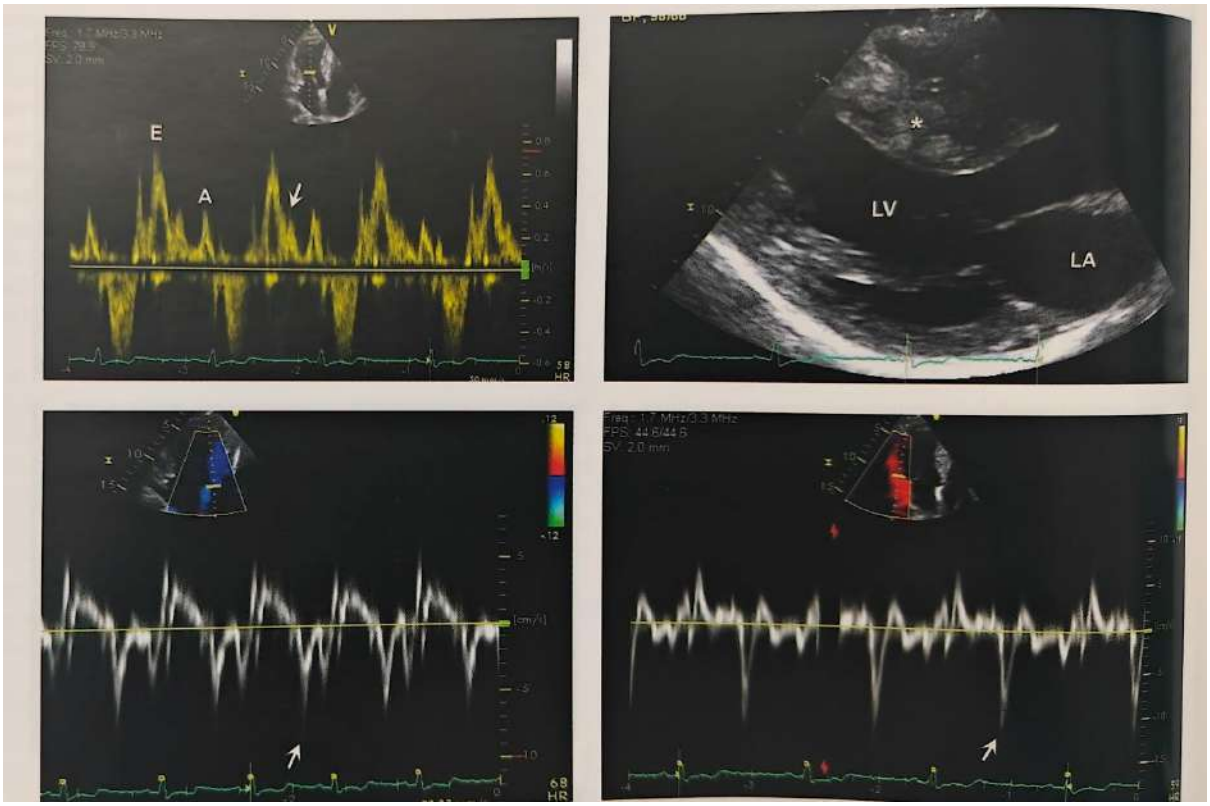

Figure 10: Grade 3 diastolic dysfunction(Figure from south asian 2decho manual)

Grade 4 diastolic dysfunction

The Valsalva maneuver is rarely necessary in classifying grade 3 diastolic dysfunction except that it may reverse a restrictive filling pattern to a grade 1 or 2 pattern, indicating the reversibility of high filling pressure.

However, even if the restrictive filling pattern does not change with the Valsalva maneuver, reversibility cannot be excluded because the Valsalva maneuver may not be adequate or filling pressure may be too high to be altered by the maneuver. Therefore, the grade 4 dysfunction indicating "irreversible restrictive" filling is not included in the ASE/EACV recommendation. So even in our study only, three grades of diastolic dysfunction are considered[62].

Table 7: Grades of Diastolic Dysfunction(Table from Feigenbaum textbook)

Grades of Diastolic Dysfunction
Normal E/A 0.8 to 1.5 E^1 > 10 E/e^1 < 8 IVRT <70 ms DT 140-240 ms
Grade 1 E/A < 0.8 E^1 < 7 E/e^1 < 8 IVRT > 90 ms DT > 240 ms
Grade 2 E/A 0.8 to 1.5 E^1< 7 E/e^1 > 14 IVRT 70 -90 ms DT 140-240 ms
Grade 3 E/A > 1.5 E^1 < 5 E/e^1 > 14 IVRT < 70 ms DT < 140 ms

LV SYSTOLIC FUNCTION

There are numerous variables that echocardiography can measure as an expression of Systolic function of the heart. These include ejection fraction, fractional shortening , stroke volume and cardiac index, systolic tissue velocity of the mitral annulus and myocardium, tissue tracking, and regional wall motion analysis.

The most popular expression of global LV function is the LVEF. LVEF is a simple measure of how much of the end diastolic volume is ejected out of the LV with each contraction[63].

LVEF can be calculated from M Mode, 2D and 3D echocardiography. In our study LVEF was calculated from M Mode.

Grades of ejection fraction(EF)

Normal : > 55%.

Mildly reduced: 40 – 55%.

Moderately reduced: 30-40%.

Severely reduced: < 30%.

PULMONARY ARTERY HYPERTENSION

Mean PA Pressure

While RV systolic pressure is the most commonly measured and reported PA hemodynamic by echocardiography, the presence of PH is defined based on the mean PA pressure (mPAP), at a value greater than 25 mm Hg. The mean PA pressure can be estimated in a variety of ways by echocardiography.

mPAP by PASP

There is a relatively fixed relationship between the PA systolic (PASP) and PA mean pressure across the spectrum of clinical pressures with the mPAP = (0.67 x PASP) + 0.5. If can be estimates PASP by echo, then the PA mean pressure estimated in turn by the mean PASP 2/3rd equating to the PA systolic pressure.

mPAP by mean TR gradient

Mean PA pressure can also be estimated from the mean pressure gradient between the RA and the RV. This gradient can be measured by tracing the systolic Doppler profile of the tricuspid regurgitant jet. This is likely the most accurate mPAP pressure estimate but is dependent on a clear and complete TR systolic envelope[64].

Grades of PAH(based on mPAP)

Mild : 25-40 mm Hg.

Moderate : 41- 55 mm Hg.

Severe : > 55 mm Hg.

PERICARDIAL EFFUSSION

Accumulation of fluid or blood in the potential pericardial space result in a pericardial effusion, detected as an echo free space. An effusion is deemed small if the pericardial space is less than 1 cm wide in diastole, moderate if 1 to 2 cm, large if greater than 2 cm and very large if greater than 2.5 cm.

Among all the echo findings , diastolic dysfunction was predominatly involved in various studies. So in our study, we primarly aim at correlating diastolic dysfunction with child pugh score[65].

MITRAL REGURGITATION

Color doppler imaging is the primary echocardiographic tool for initial detection and quantitation of mitral regurgitation. There are several potential sources of color Doppler flow signal in the left atrium, not all color Doppler signals appearing within the left atrium represent mitral regurgitation.

The characteristics of a true mitral regurgitation jet are: Evidence of proximal flow acceleration , flow conforms to the appearance of a the true jet, downstream (left atrial) appearance is consistent with a volume of blood being ejected through a relatively constraining orifice ,the flow signal is appropriately confined to systole.

Grading of mitral regurgitation(based on MR jet LA%)

MR jet LA% is percentage of left atrial area encompassed by the mitral regurgitation jet with color flow Doppler imaging[66].

Mild: < 15.

Moderate: 15-50.

Severe : > 50.

TRICUSPID REGURGITATION

Tricuspid regurgitation is diagnosed by noting reversed color Doppler flow signals moving from the right ventricle into the right atrium during systole. Similar to mitral regurgitation, its severity can be estimated by noting the size of the regurgitant jet in relation to the size of the right atrium taken in the same frame, which shows the largest area of regurgitation[67].

Grading of tricuspid regurgitation(based on jet area- central jets cm^2)[68]

Mild:<5.

Moderate: 5-10.

Severe: > 10.

RV DYSFUNCTION

Normal RV systolic function is a complex process with a number of contributing Factors, which includes inherent myocardial contractility, systemic venous return determining preload, pulmonary vascular status determining RV afterload, interventricular septal contraction and pericardial compliance.

RV systolic function is assessed in echo cardiography by TAPSE – Tricuspid annular plane systolic excursion. TAPSE less then 17 mm indicates RV systolic dysfunction[69].

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LITERATURE RELATED TO THE STUDY:

Chandey M et al. (2020) conducted a cross-sectional study on 90 patients with liver cirrhosis to assess cardiovascular dysfunction using electrocardiography and echocardiography, based on the Child-Pugh score. In Child-Pugh Class A, 2 (33%) patients had grade 1 diastolic dysfunction. In Class B, 23 (59%) patients had grade 1 diastolic dysfunction. In Class C, 3 (7%) patients had grade 1 diastolic dysfunction, 33 (73%) patients had grade 2 diastolic dysfunction, and 1 (2%) patient had grade 3 diastolic dysfunction, with a p-value of 0.04, indicating significance. Systolic function was found to be normal in all patients. The study concluded that diastolic dysfunction and QTc interval prolongation are both related to the severity of liver cirrhosis and are major criteria for cirrhotic cardiomyopathy.[70]

Uthaya SM et al. (2020) conducted a cross-sectional study involving 93 patients to assess the prevalence of cirrhotic cardiomyopathy and its correlation with the Child-Pugh score. The study revealed a significant increase in the presence of cirrhotic cardiomyopathy in patients with end-stage liver disease compared to previous studies. Systolic dysfunction was found to be statistically significant. However, when correlating with the Child-Pugh score, only diastolic dysfunction showed significance.[71]

Mishra A et al. (2020) conducted a cross-sectional study on 60 patients with liver cirrhosis to examine the correlation between the severity of liver dysfunction, as assessed by the Child-Pugh score, and cardiac function on echocardiography. The study found no statistically significant correlation between the Child-Pugh score and systolic function in cirrhotic patients. However, it showed that both diastolic dysfunction and relevant echocardiographic changes were associated with liver

cirrhosis. The study concluded that liver cirrhosis has a significant correlation with diastolic dysfunction.[72]

Karki N et al. (2019) conducted a cross-sectional study on patients admitted with cirrhosis to examine cardiac dysfunction using the Child-Pugh score. Diastolic dysfunction was observed in 61.9% of patients and was more common in the alcoholic group (63.2% vs. 58.6%). Systolic dysfunction was seen in 6.6% of alcoholic patients. Cirrhotic cardiomyopathy was present in 51.4% of patients. Prolonged QTc (>0.44 seconds) was noted in 79% of patients, mainly in Child-Pugh Class C, with a model for end-stage liver disease (MELD) score >10. The study concluded that cardiac dysfunction, particularly diastolic dysfunction, is prevalent in a significant number of cirrhotic patients, regardless of etiology. QTc prolongation may be an early indicator of cardiac dysfunction and is directly correlated with Child-Pugh and MELD scores.[73]

Jahangiri S et al. (2023) conducted a comprehensive study involving 425 patients with cirrhosis to explore potential associations between electrocardiogram (ECG) changes and both the etiology of cirrhosis and the Child-Pugh score, a clinical tool used to assess the severity of liver disease. The study identified that prolonged QT intervals and the presence of an early transitional zone were the most frequent ECG alterations, observed in 24.7% and 19.8% of the patients, respectively. These ECG changes were significantly correlated with the underlying cause of cirrhosis and the Child-Pugh classification. The researchers concluded that a prolonged QT interval and the presence of an early transitional zone in cirrhotic patients may be indicative of underlying cardiac dysfunction. Therefore, these findings suggest that further cardiac evaluation is warranted in this patient population to better understand and manage potential cardiac complications associated with cirrhosis.[74]

Solanki R et al. (2023) conducted a study on 203 cirrhosis patients to assess the association of left ventricular diastolic dysfunction (LVDD) with factors affecting the severity, complications, and survival of cirrhosis patients. The study concluded that a higher Child-Turcotte-Pugh (CTP) score, prolonged QTc interval, higher ascitic fluid protein levels, and poor survival are significantly associated with LVDD. Additionally, ascitic fluid protein levels >1 g/dL could serve as an indicator for evaluating LVDD.[75]

Al Atroush HH et al. (2022) conducted a study to evaluate cardiac dysfunction in patients with end-stage liver disease, examining its correlation with the Child-Pugh classification and its impact on the clinical outcomes of patients categorized as Child B and Child C. The findings revealed that the majority of these patients exhibited cardiac dysfunction, with diastolic dysfunction being particularly prevalent, affecting 87.5% of the cohort. The study highlighted that the prevalence of diastolic dysfunction in end-stage liver disease is best assessed using the E/É ratio obtained through Tissue Doppler Imaging (TDI), which proved to be more accurate than the traditional E/A ratio. The study concluded that diastolic dysfunction is a critical and sensitive marker for diagnosing cirrhotic cardiomyopathy and is often the earliest affected parameter.[76]

Nirmal A et al. (2021) conducted a cross-sectional study involving 150 individuals with liver cirrhosis to evaluate cardiac dysfunction and its prevalence among these patients. The study found that left ventricular (LV) diastolic dysfunction was statistically significant and correlated well with the severity of liver cirrhosis. The study concluded that cardiac dysfunction was directly correlated with the severity of liver cirrhosis according to the Child-Pugh scoring criteria, suggesting that the observed cardiac changes were due to cirrhosis and not alcohol consumption.[77]

Lanzieri PG et al. (2017) conducted a cross-sectional study on 67 patients with cirrhosis to analyze QT intervals according to cirrhosis severity, as measured by the Child-Pugh classification. The study found a positive correlation between the QT interval and the Child-Pugh score in individuals with Child-Pugh scores ≥ 7 ($r = 0.50$, $p < 0.05$) and QT intervals ≥ 440 ms. The results showed longer QT intervals in patients with Child-Pugh C cirrhosis, reinforcing the relationship between cirrhosis severity and electrocardiographic findings of cirrhotic cardiomyopathy.[78]

Balde J et al. (2016) conducted a cross-sectional study on 42 patients with cirrhosis to assess the association between echocardiography and cirrhosis severity using the Child-Pugh score. Diastolic dysfunction was found in 22 patients (52.3%), with 19 patients in CPS Grade B and three in Grade C. The study found no significant association between echocardiographic changes and the Child-Pugh score in patients with liver cirrhosis.[80]

MATERIALS AND METHODS

Source of data:

Patients admitted to Raichur Institute of Medical Sciences, Raichur during Period of August 2022 to February 2024

Study design:

Prospective cross sectional study

Study period:

August 2022 to February 2024

Place of study:

Raichur Institute of Medical Sciences, Raichur

Sample size:

60 Patients admitted with Cirrhosis of liver

Inclusion Criteria:

1) Adult patients with diagnosis of Cirrhosis of liver of any etiology.

Exclusion Criteria:

1) Patients with less then 18 years.
2) Patients with established hypertension.
3) Patients with diabetes mellitus.

4) Patients with known case of coronary heart disease.
5) Previous with history of cardiac surgery.
6) Known case of congenital heart disease.
7) Chronic kidney disease patients.

Methodology:

- After getting institutional ethics committee clearance and written informed consent 60 patients admitted in Intensive Care Unit/ward in RIMS Teaching hospital during period of August 2022 to February 2024 were considered.
- Patients admitted with diagnosis of cirrhosis of liver during the period of study were included in this study.
- Detailed history and clinical examination of the patient was collected in a pre designed proforma and all basic investigations were done.

Cirrhosis was labeled on the basis of:

- Clinical (reduced liver span <8 cm on clinical exam with ascites/or splenomegaly).
- Biochemical (prolonged prothrombin time>12 seconds and reduced level of serum albumin <3.5g/dl).
- Radiological (increased liver echo pattern, shrunken liver <8cm in mid-clavicle line, portal vein diameter >1.3 cm and spleen size >13 cm longitudinally).

The severity of liver cirrhosis was assessed and according to the child Pugh score, patients were grouped into:

- Class A: patients of Liver cirrhosis with child Pugh Score 5-6.
- Class B: patients of Liver cirrhosis with child Pugh Score 7-9.
- Class C: patients of Liver cirrhosis with childPugh score 10-15.

ECG was done in all the patients. ECG abnormalities were noted.

2D Echocardiography:

2D M mode Colour Doppler Echocardiography was done by commercially available Echocardiography machine, and used to assess cardiac status with special reference to left atrial diameters, left ventricle end diastolic volume, I.V. septal thickness, left ventricular posterior wall thickness and to assess E/A ratio where E(meter/sec)stands for early maximum left ventricular filling velocity, and A(meter/sec) for late diastolic left ventricle filling velocity. E^1 was measured in meter/second.

Left ventricular systolic function was assessed by ejection fraction. Left ventricular diastolic function was assessed by E/A ratio. Grades of diastolic dysfunction:

- Grade 1: impaired relaxation pattern with normal filling pressures.
- Grade 2: pseudo-normalised pattern.
- Grade 3: reversible restrictive pattern.
- Grade 4: irreversible restrictive pattern.

Primary outcome:

1) Diastolic function to be affected more compared to systolic function.
2) Decrease in cardiac function will be correlating to child pugh score.

Investigations:

Hematological investigations:

- Hb
- TLC
- DLC

- Platelet count
- PCV
- MCH
- MCV

Biochemical investigations:

- RBS
- Blood urea
- Serum creatinine
- Sodium
- Potassium
- Serum bilirubin
- Serum albumin
- SGOT
- SGPT
- ALP

USG Abdomen

2D ECHO

Data analysis:

Data collected was entered into Microsoft excel sheet after appropriate data filtration. Date was analysed using SPSS version 26 software. Standard deviation, mean, ratio and proportion was calculated for descriptive data and data were represented in suitable graphs and tabular forms. Appropriate inferential statistics like chi-square test were applied wherever necessary. Chi-square test ‘p’ value of less than 0.05 was considered significant.

SAMPLE SIZE ESTIMATION

Suitable statistical method will be used at time of data analysis after consulting biostatistician

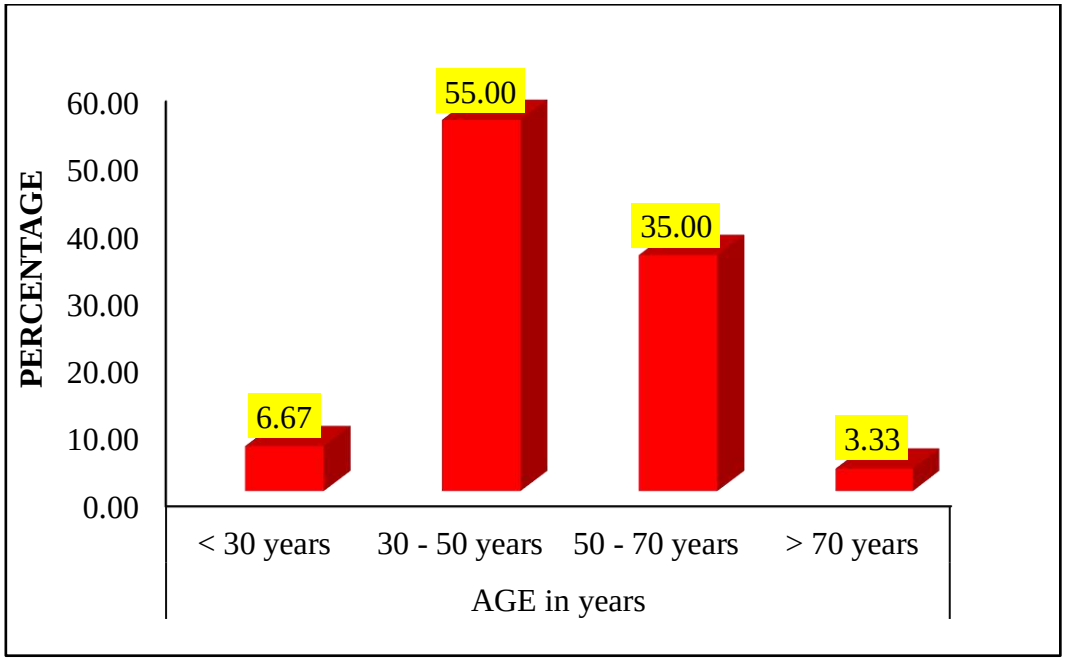
n=4PQ/e2

e is 20%, P is 62, sample size is 60

RESULTS

Table 8: Age Distribution

		No. of Case	Percentage
AGE in years	< 30 years	4	6.67
	30 - 50 years	33	55.00
	50 - 70 years	21	35.00
	> 70 years	2	3.33
TOTAL		60	100

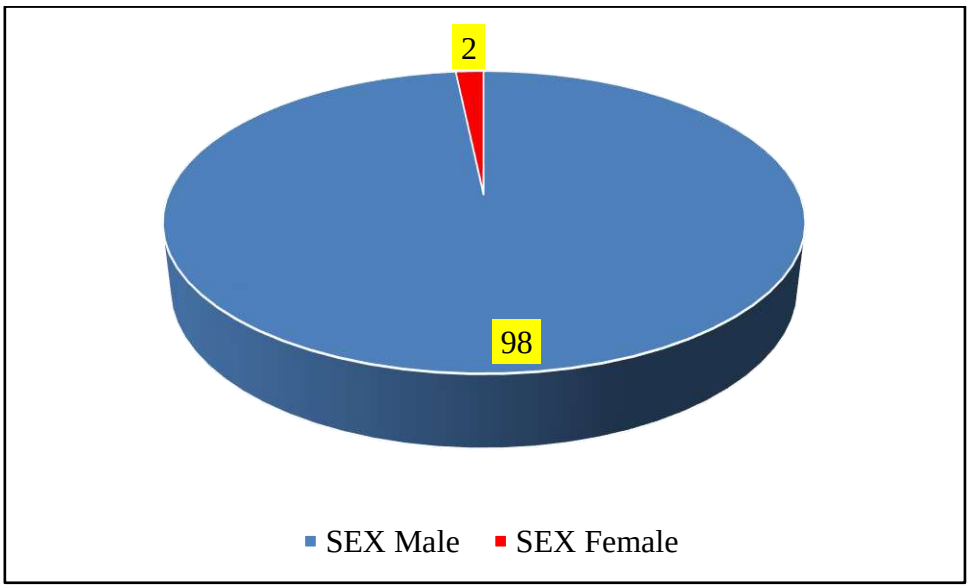


Graph 1: Age Distribution

This table categorizes the sample population based on age groups. The majority of cases fall within the 30-50 years age group (55%), followed by the 50-70 years group (35%). Very few cases are under 30 years (6.67%) or over 70 years (3.33%).

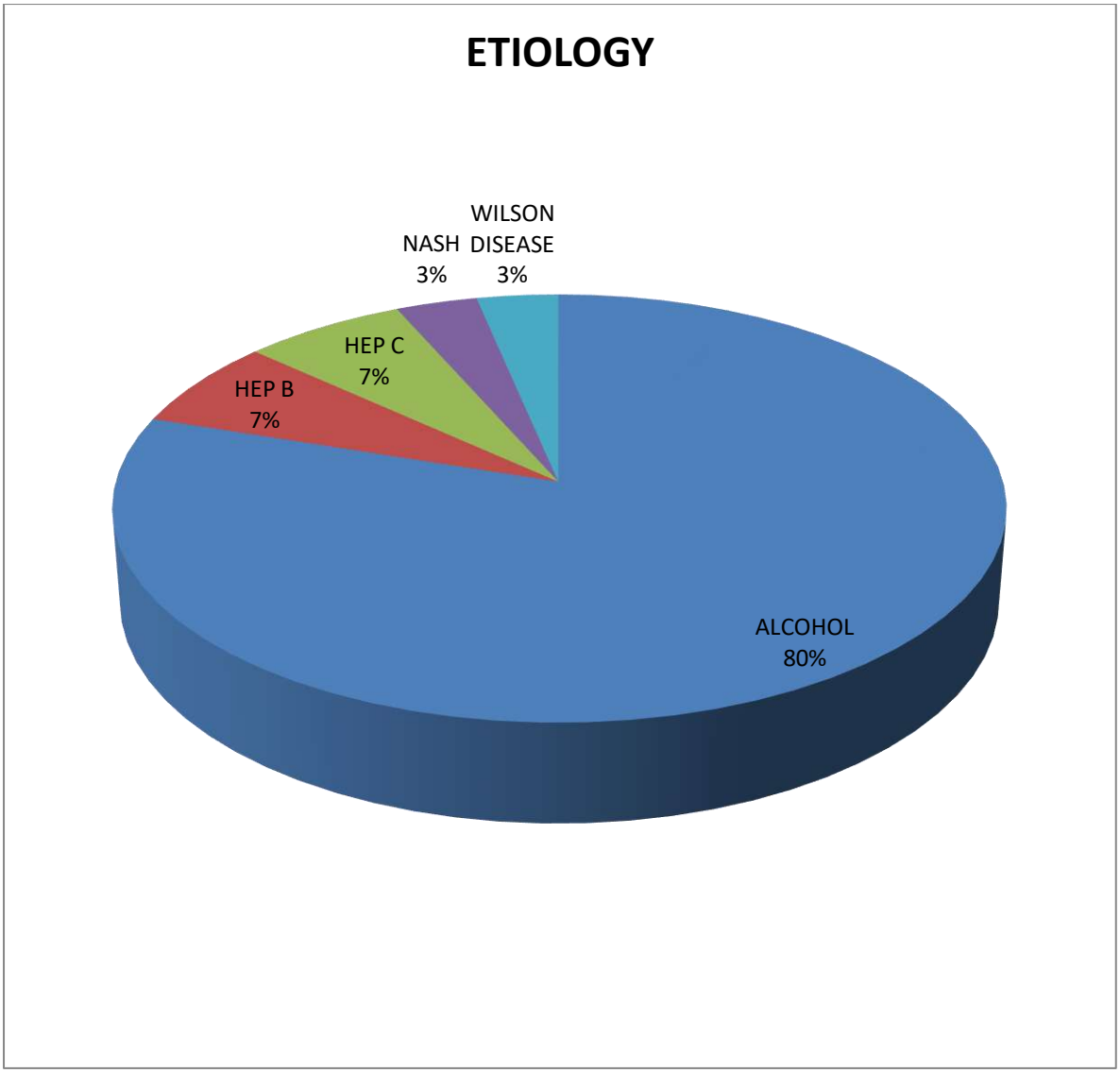
Table 9: Sex Distribution

		No. of Case	Percentage
SEX	Male	59	98
	Female	1	2
TOTAL		60	100



Graph 2: Sex Distribution

This table categorizes the sample population based on sex. The table shows an overwhelming majority of male cases (98%), with only 2% female cases. The male predominance is significant. The high male percentage suggests a possible gender-related factor influencing the prevalence or diagnosis rate.

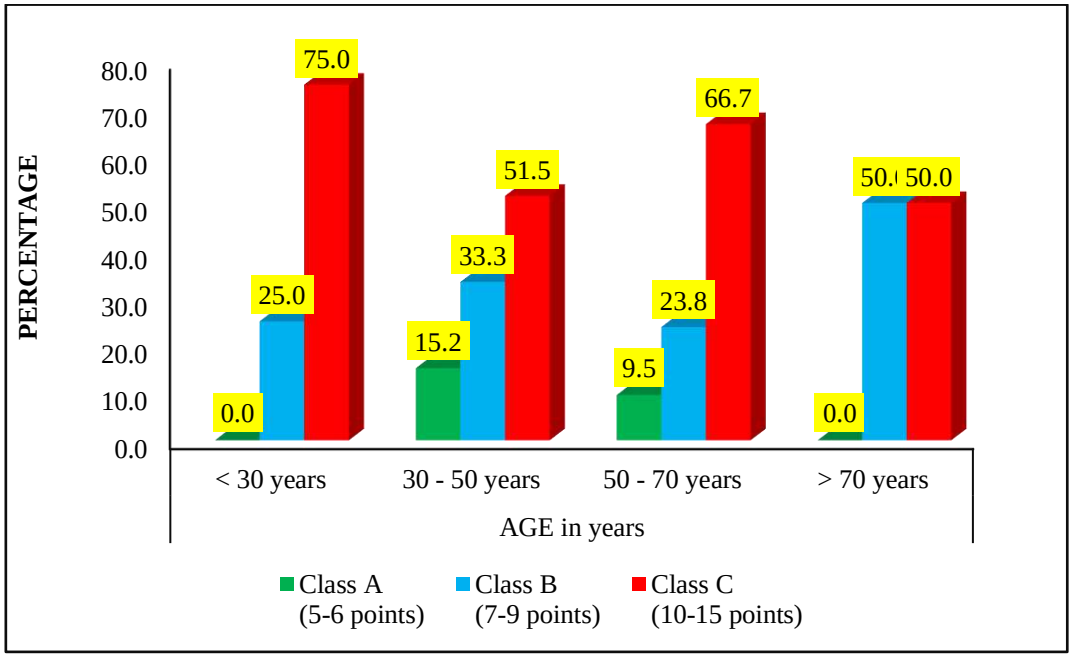


Graph 3: Etiology Distribution

This graph categorizes the etiology. Alcohol being the most common etiology, with 80% followed by hep B(7%) , hep C (7%), NASH(3%), wilson disease(3%).

Table 10: Child PUGH Score and Age

		Child PUGH score						Total		Chi-square	P Value
		Class A (5-6 points)		Class B (7-9 points)		Class C (10-15 points)					
		No.	%	No.	%	No.	%	No.	%		
AGE in years	< 30 years	0	0.0	1	25.0	3	75.0	4	100	2.544	0.864
	30 - 50 years	5	15.2	11	33.3	17	51.5	33	100		
	50 - 70 years	2	9.5	5	23.8	14	66.7	21	100		
	> 70 years	0	0.0	1	50.0	1	50.0	2	100		
TOTAL		7	11.7	18	30.0	35	58.3	60	100		

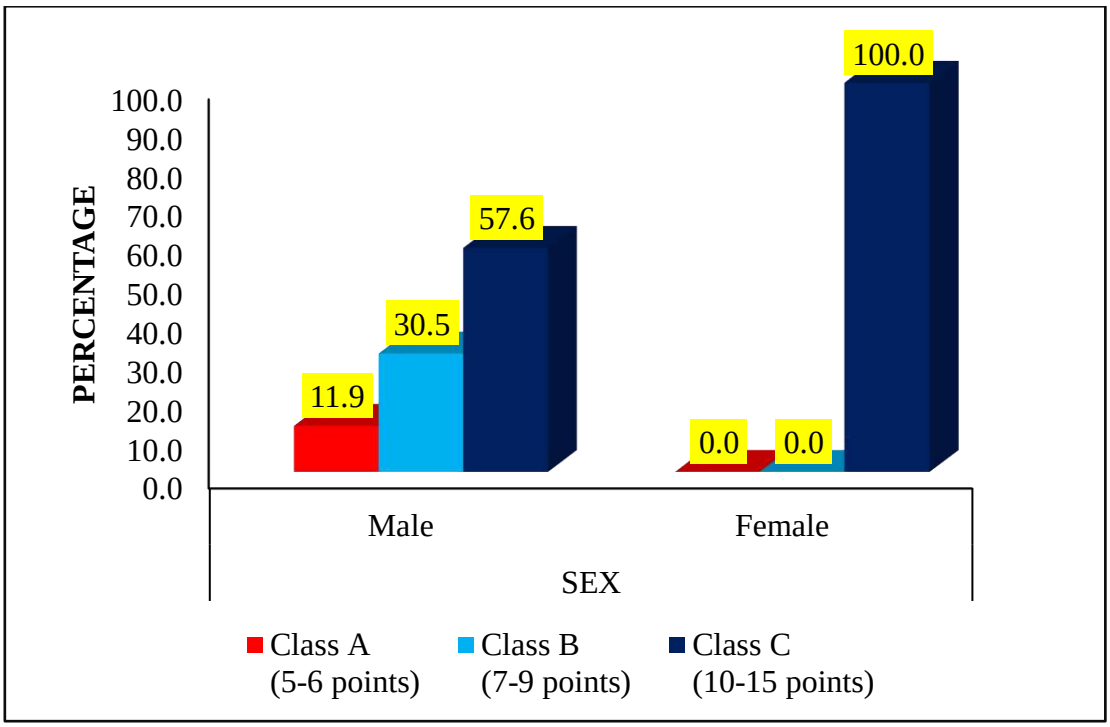


Graph 4: Child PUGH Score and Age

This table categorizes patients based on their Child PUGH score. Most patients fall within the 30-50 and 50-70 age groups. Severity increases with age; the highest percentage of Class C patients are found in the 50-70 years age group. The difference showed no significant association between age and the Child PUGH score(p = 0.864).

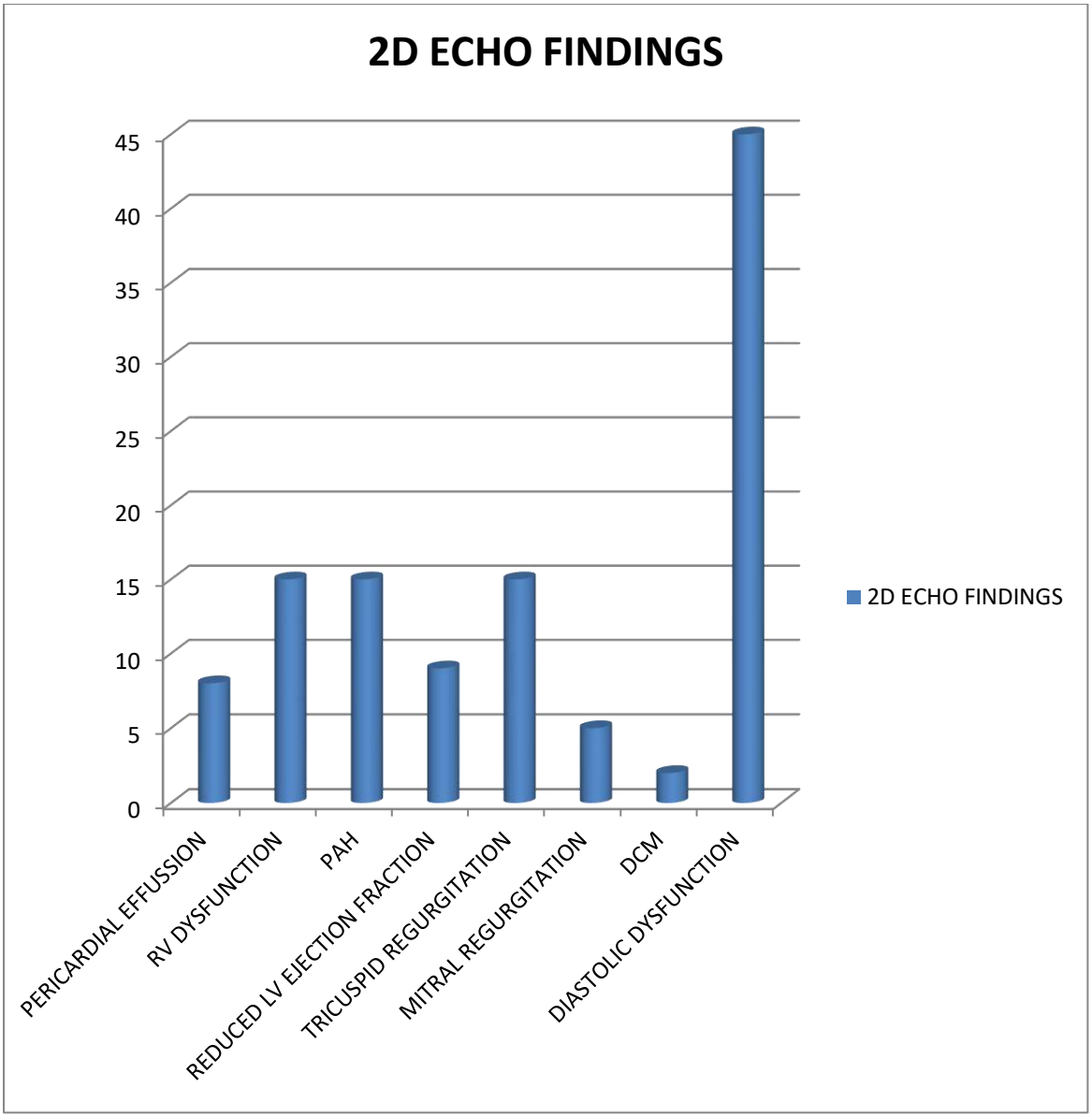
Table 11: Child PUGH Score and Sex

		Child PUGH score						Total		Chi-square	P Value
		Class A (5-6 points)		Class B (7-9 points)		Class C (10-15 points)					
		No.	%	No.	%	No.	%	No.	%		
SEX	Male	7	11.9	18	30.5	34	57.6	59	100	.726[a]	0.695
	Female	0	0.0	0	0.0	1	100.0	1	100		



Graph 5: Child PUGH Score and Sex

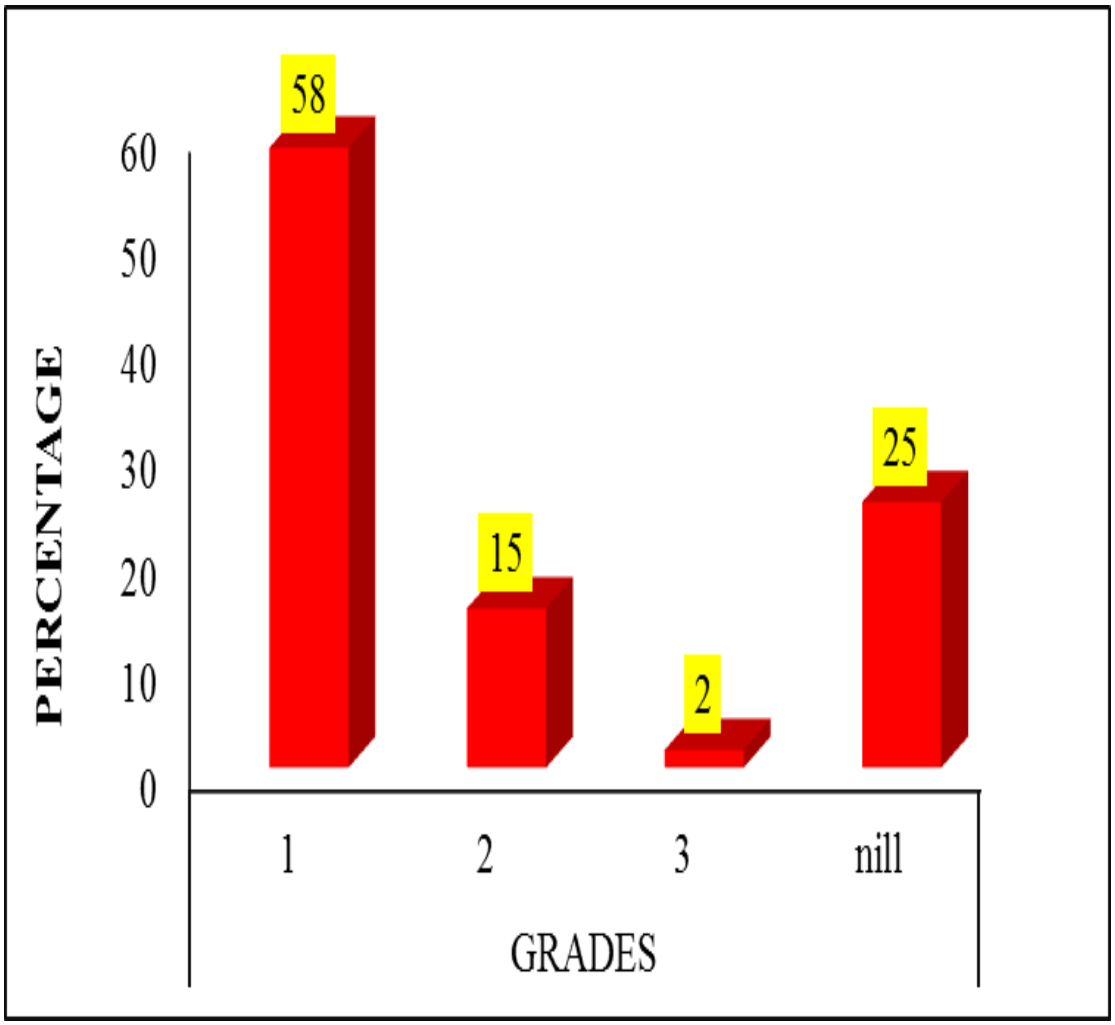
This table shows the distribution of Child PUGH scores across sexes. Males predominantly represent the patient sample. All females fall within Class C. The difference showed no significant difference between sex and Child PUGH score (p= 0.695).



Graph 6: 2D ECHO findings

Among all the 2d echo findings Diastolic dysfunction was predominantly noticed in 45(75%) of patients, which was followed by Pulmonary artery hypertension 15(25%), Tricuspid regurgitation 15(25%), RV dysfunction 15(25%) Reduced LV ejection fraction 9(16%), Pericardial effusion 8(13%), Mitral regurgitation 5(8%), DCM 2(3%).

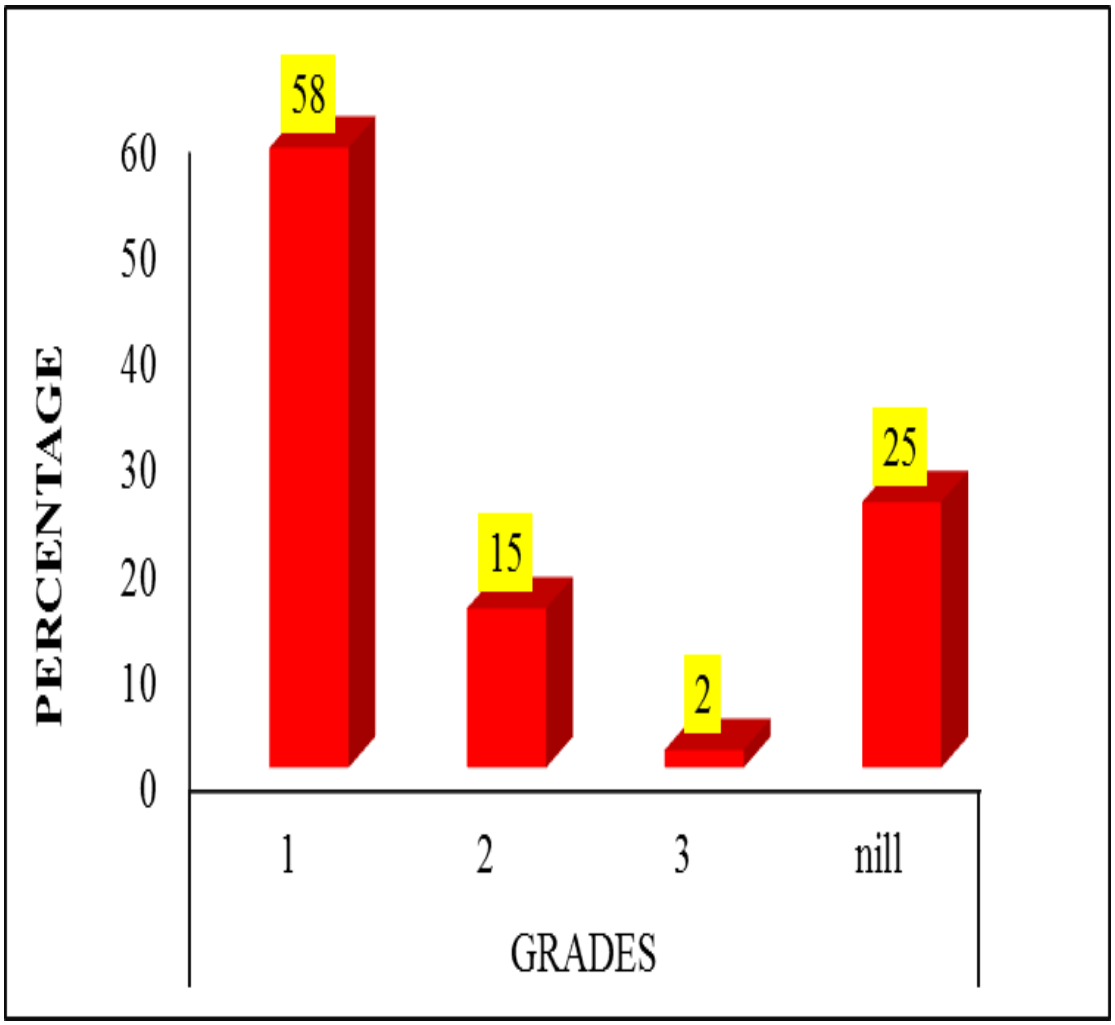
E/A



Graph 7: Distribution of Grades of LV diastolic dysfunction using E/A

Taking E/A into consideration, Grade 1 diastolic dysfunction is the most common with 35(58%), followed by Grade 2 diastolic dysfunction 9 (15%), Grade 3 diastolic dysfunction 1(2%), and Nil 15(25%).

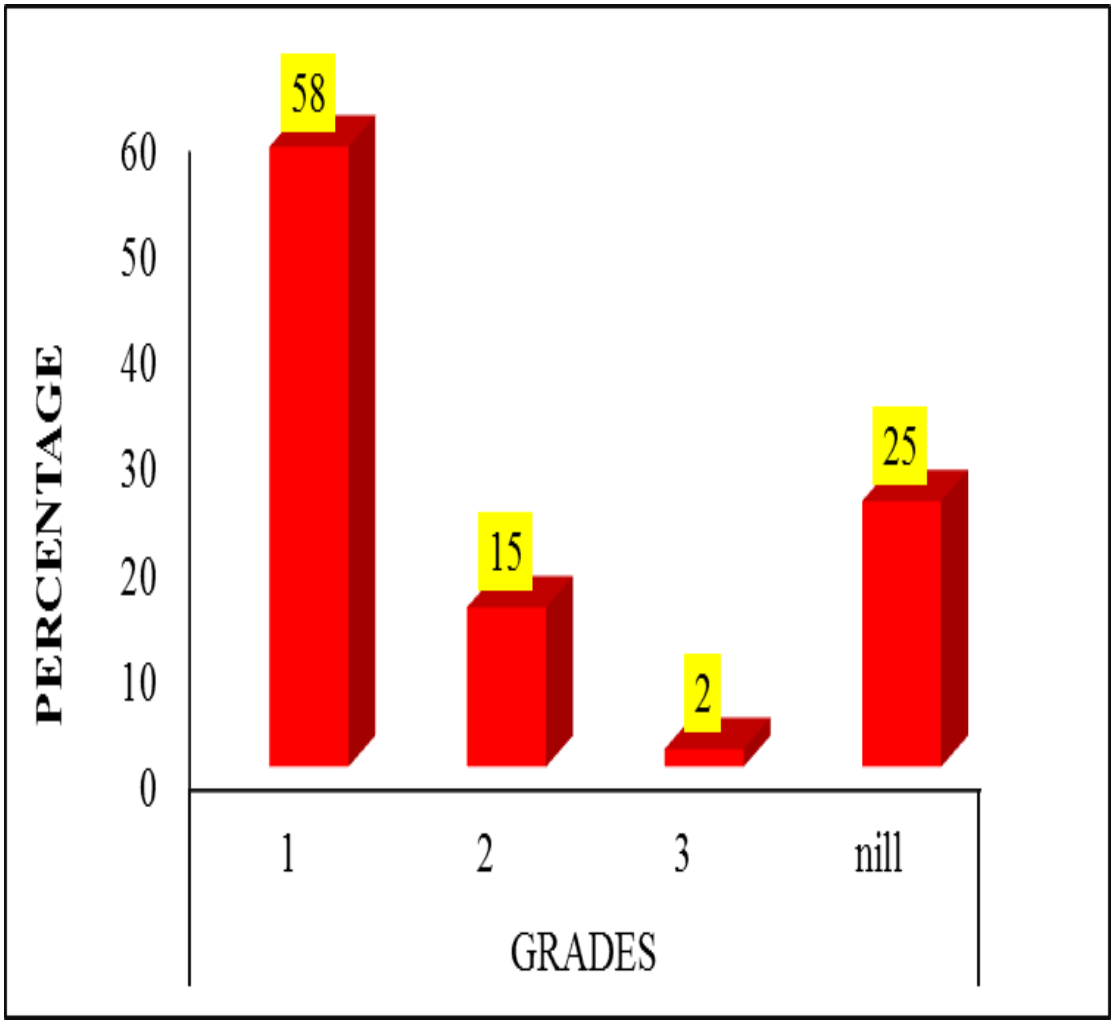
E^1



Graph 8 : Distribution of Grades of LV diastolic dysfunction using E^1

Taking E^1 into consideration, Grade 1 diastolic dysfunction is the most common with 35(58%), followed by Grade 2 diastolic dysfunction 9 (15%), Grade 3 diastolic dysfunction 1(2%), and Nil 15(25%).

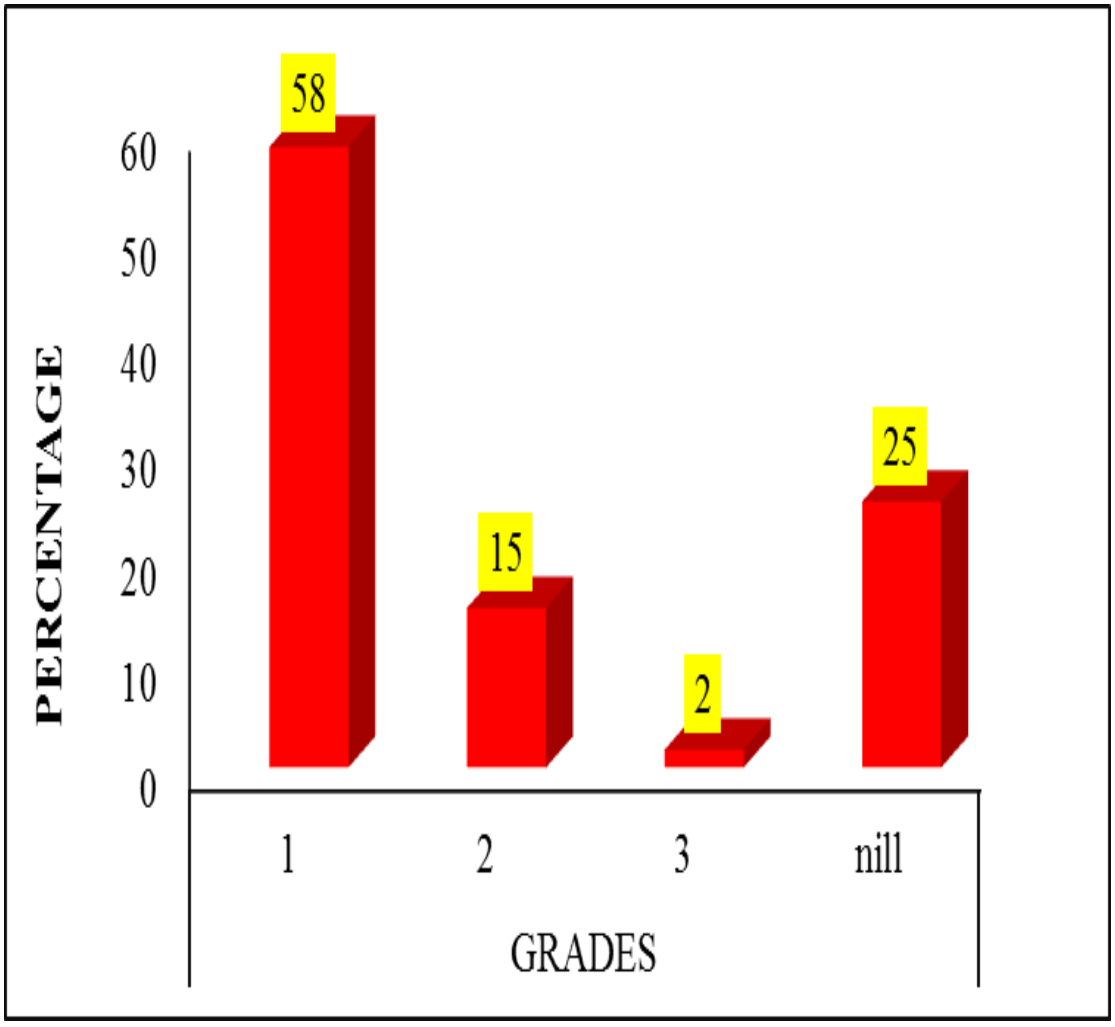
E/E^1



Graph 9 : Distribution of Grades of LV diastolic dysfunction using E/E1

Taking E/E^1 into consideration, Grade 1 diastolic dysfunction is the most common with 35(58%), followed by Grade 2 diastolic dysfunction 9 (15%), Grade 3 diastolic dysfunction 1(2%), and Nil 15(25%).

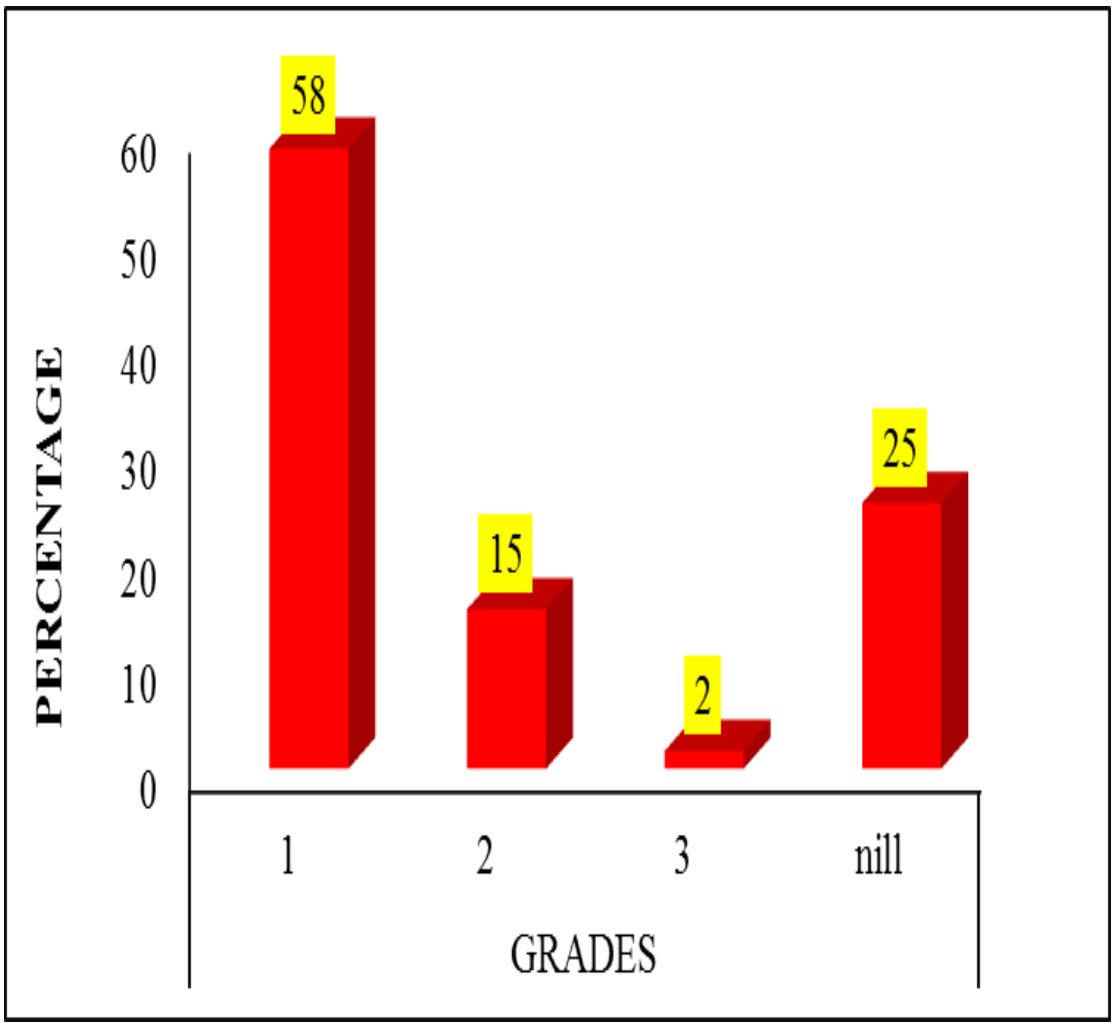
IVRT (ISOVOLUMETRIC RELAXATION TIME)



Graph 10: Distribution of Grades of LV diastolic dysfunction using IVRT

Taking IVRT into consideration, Grade 1 diastolic dysfunction is the most common with 35(58%), followed by Grade 2 diastolic dysfunction 9 (15%), Grade 3 diastolic dysfunction 1(2%), and Nil 15(25%).

DT (DECELERATION TIME)

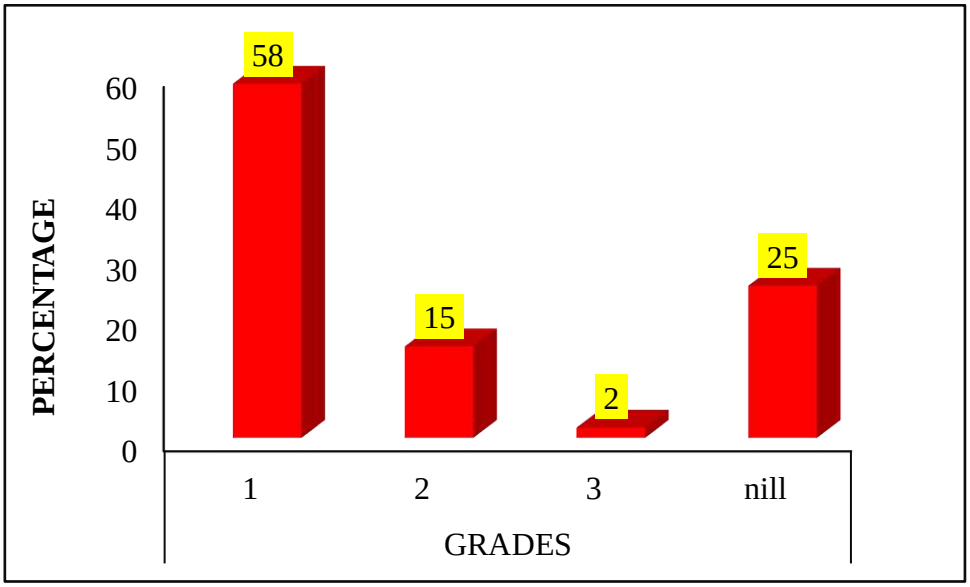


Graph 11: Distribution of Grades of LV diastolic dysfunction using DT

Taking DT into consideration, Grade 1 diastolic dysfunction is the most common with 35(58%), followed by Grade 2 diastolic dysfunction 9 (15%), Grade 3 diastolic dysfunction 1(2%), and Nil 15(25%).

Table 12: Diastolic Dysfunction Grades Distribution

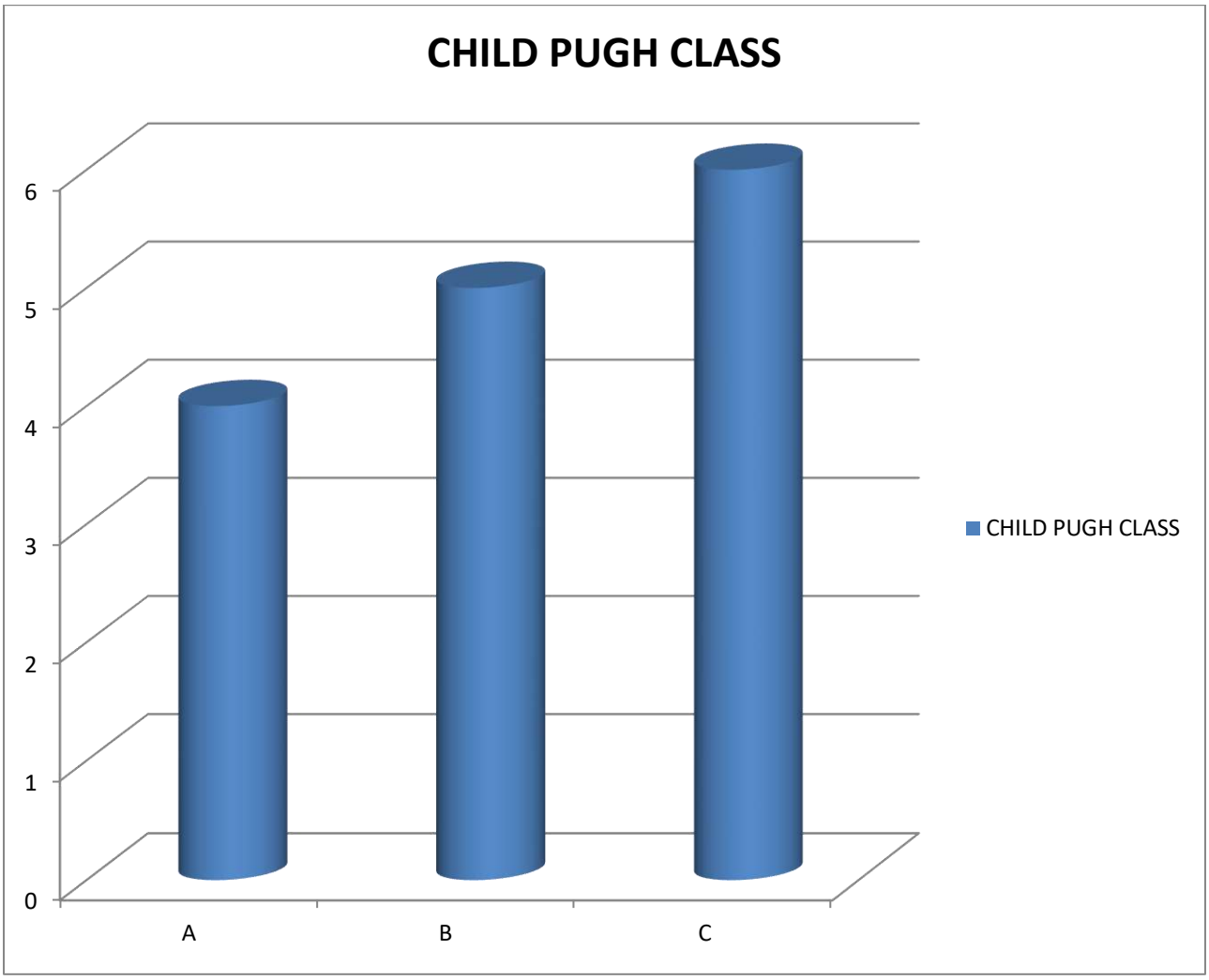
		No. of Case	Percentage
GRADES	1	35	58
	2	9	15
	3	1	2
	nil	15	25
TOTAL		60	100



Graph 12: Diastolic Dysfunction Grades Distribution

This table categorizes grades of diastolic dysfunction. Grade 1 is the most common with 58%, followed by Grade 2 (15%), Grade 3 (2%), and Nil (25%)

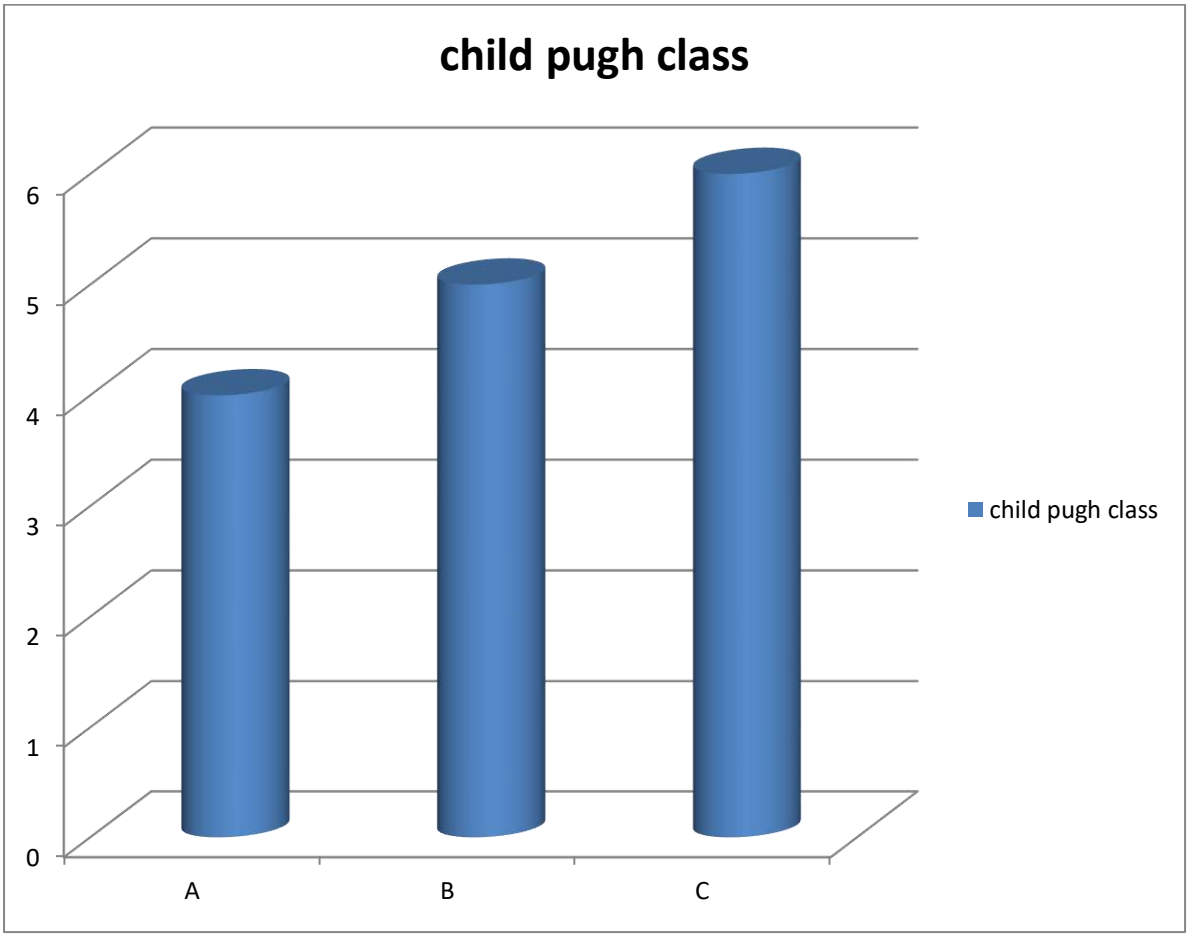
Tricuspid regurgitation



Graph 13: Tricuspid regurgitation Distribution

Among 15 cases of Tricuspid regurgitation , 4(27%) were in child pugh class A, 5(33%) were seen in child pugh class B , 6(40%) were seen in child pugh class C. All were Mild TR.

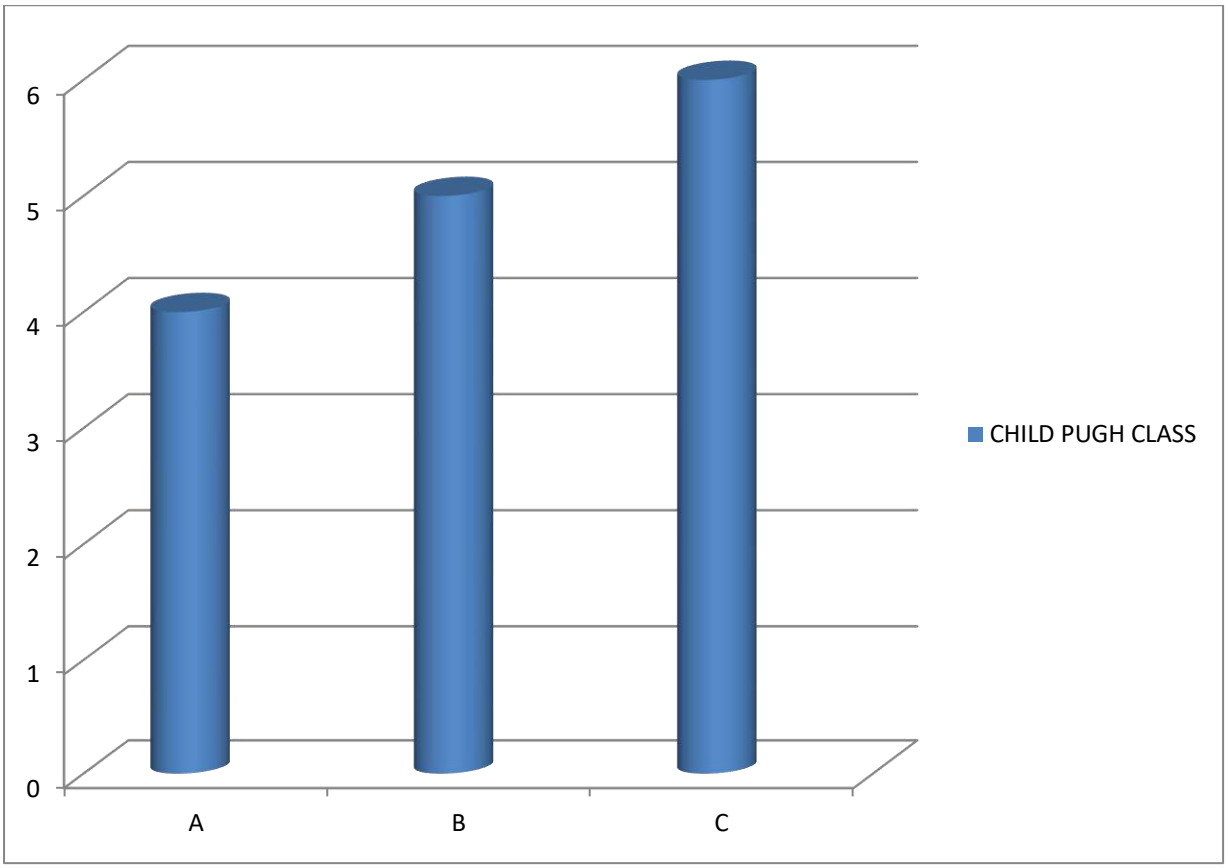
PAH(Pulmonary Artery Hypertension)



Graph 14: PAH Distribution

Among 15 cases of PAH, 4(27%) were in child pugh class A, 5(33%) were seen in child pugh class B , 6(40%) were seen in child pugh class C. All were mild PAH.

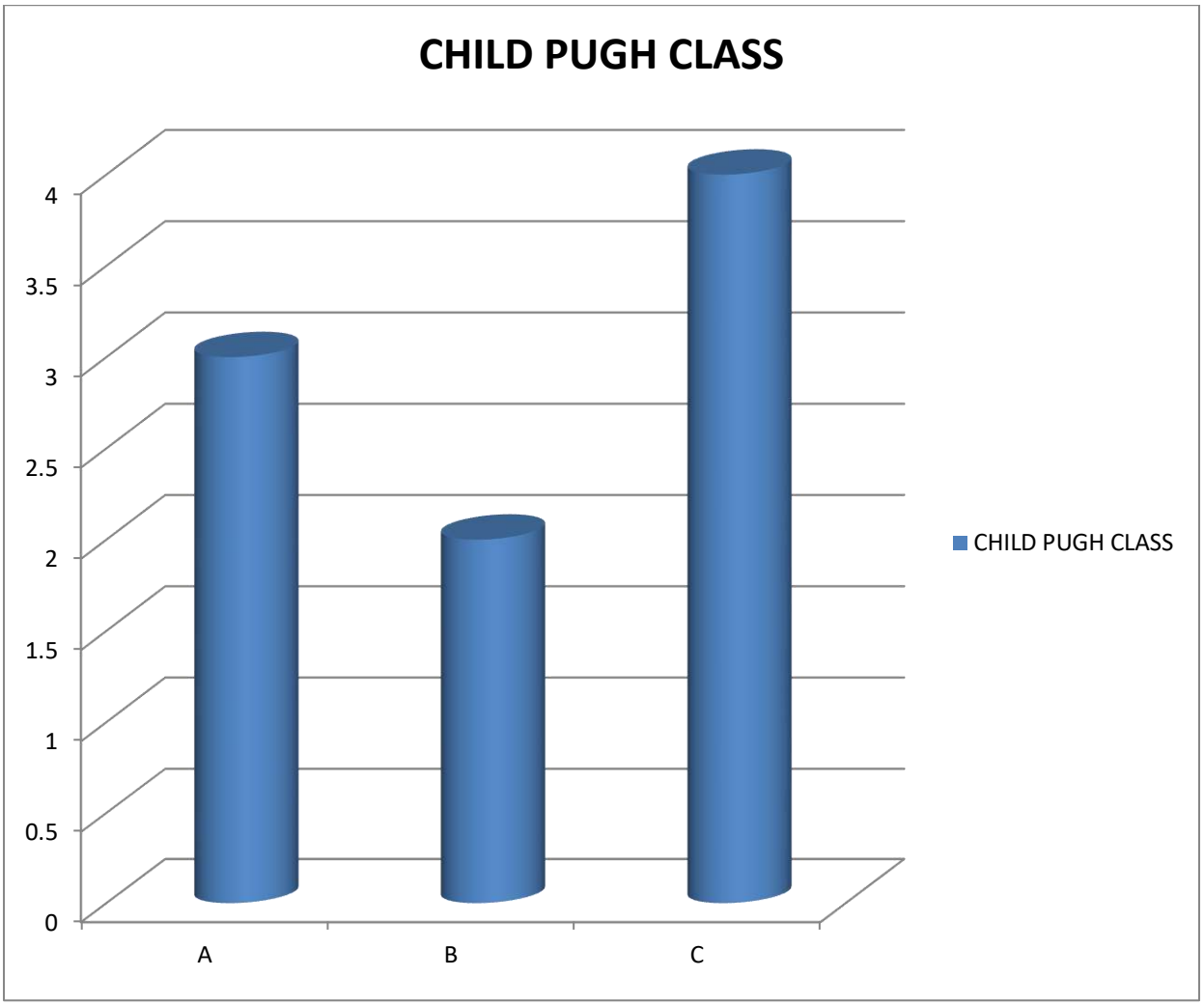
RV dysfunction



Graph 15: RV Dysfunction Distribution

Among 15 cases RV dysfunction , 4(27%) were in child pugh class A, 5(33%) were seen in child pugh class B , 6(40%) were seen in child pugh class C.

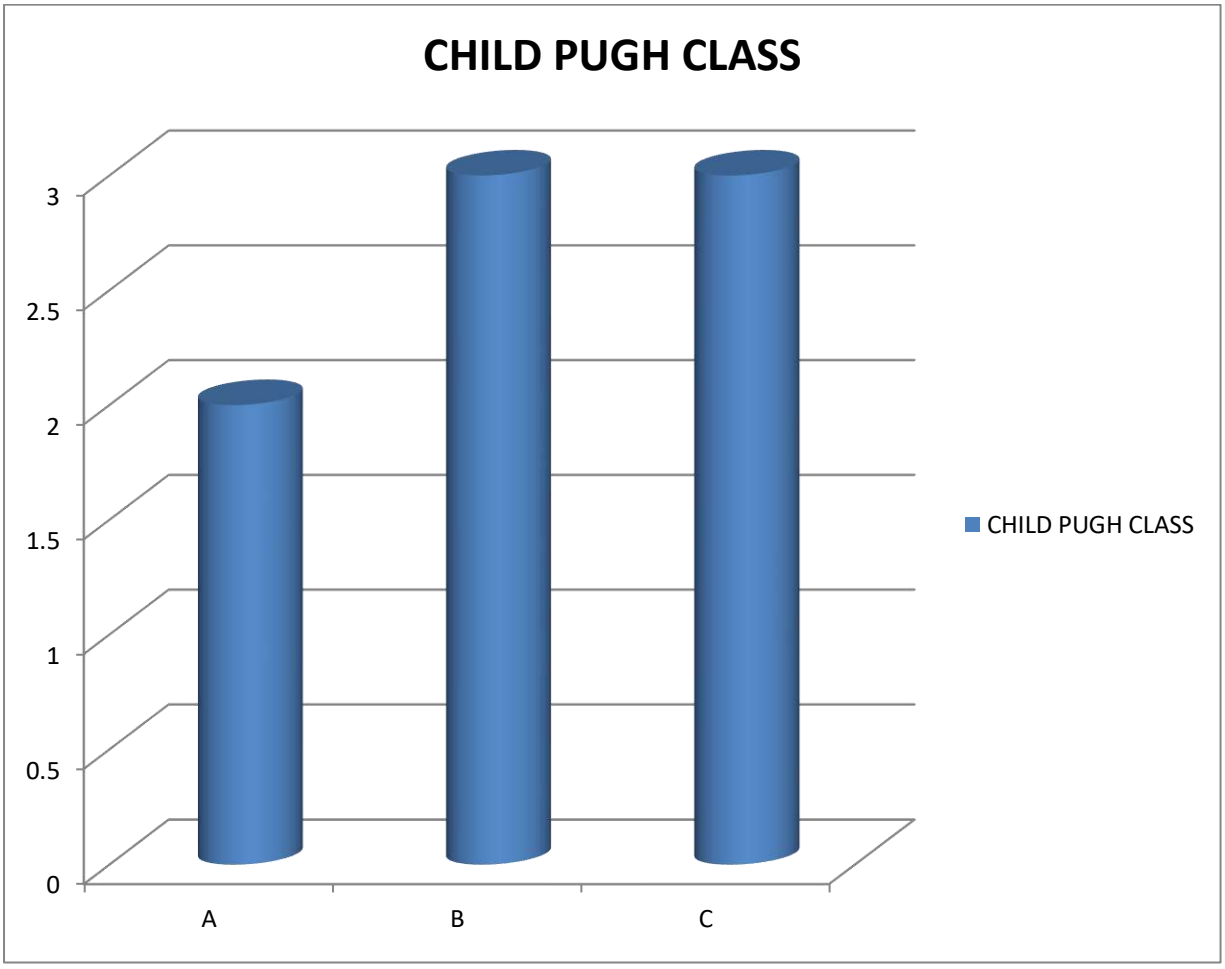
Reduced LV Ejection fraction



Graph 16: Reduced LV Ejection fraction Distribution

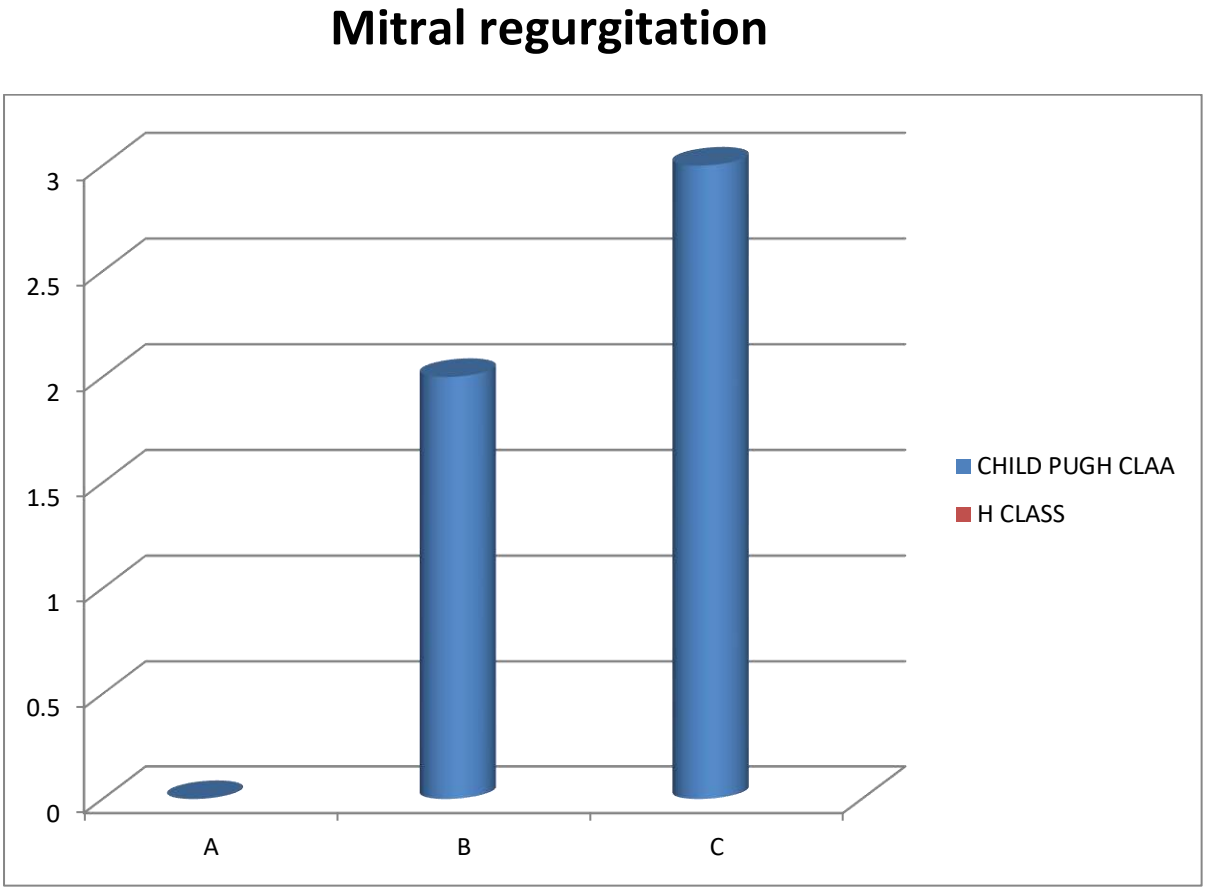
Among 9 cases of Reduced LV Ejection Fraction, 3(33%) were in child pugh class A, 2(23%) were seen in child pugh class B , 4(44%) were seen in child pugh class C.

Pericardial effusion



Graph 17: Pericardial Effusion Distribution

Among 8 cases of Pericardial effusion , 2(26%) were in child pugh class A, 3(37%) were seen in child pugh class B ,3(37%) were seen in child pugh class C.

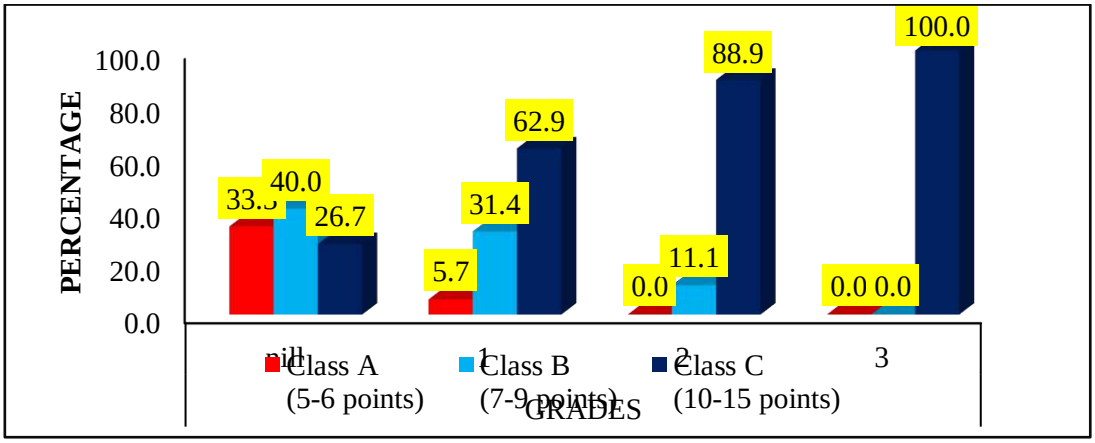


Graph 18: Mitral Regurgitation Distribution

Among 5 cases of Mitral regurgitation , 0(0%) were in child pugh class A, 2(40%) were seen in child pugh class B , 3(60%) were seen in child pugh class C. All were mild MR.

Table 13: Child PUGH Score and Left ventricular Diastolic Dysfunction Grades Distribution

		Child PUGH score						Total		Chi-square	P Value
		Class A (5-6 points)		Class B (7-9 points)		Class C (10-15 points)					
		No.	%	No.	%	No.	%	No.	%		
GRADES	N	5	33.3	6	40.0	4	26.7	15	100	14.599[a]	**0.024**
	1	2	5.7	11	31.4	22	62.9	35	100		
	2	0	0.0	1	11.1	8	88.9	9	100		
	3	0	0.0	0	0.0	1	100.0	1	100		



Graph 19: Child PUGH Score and Left ventricular Diastolic Dysfunction Grades Distribution

Among 35 patients with Grade 1 diastolic dysfunction, 2(5.7%) patients were in Child pugh Class A, 11(31.4%) patients were in Child pugh Class B, and 22(62.9%) patients were in Child pugh Class C.

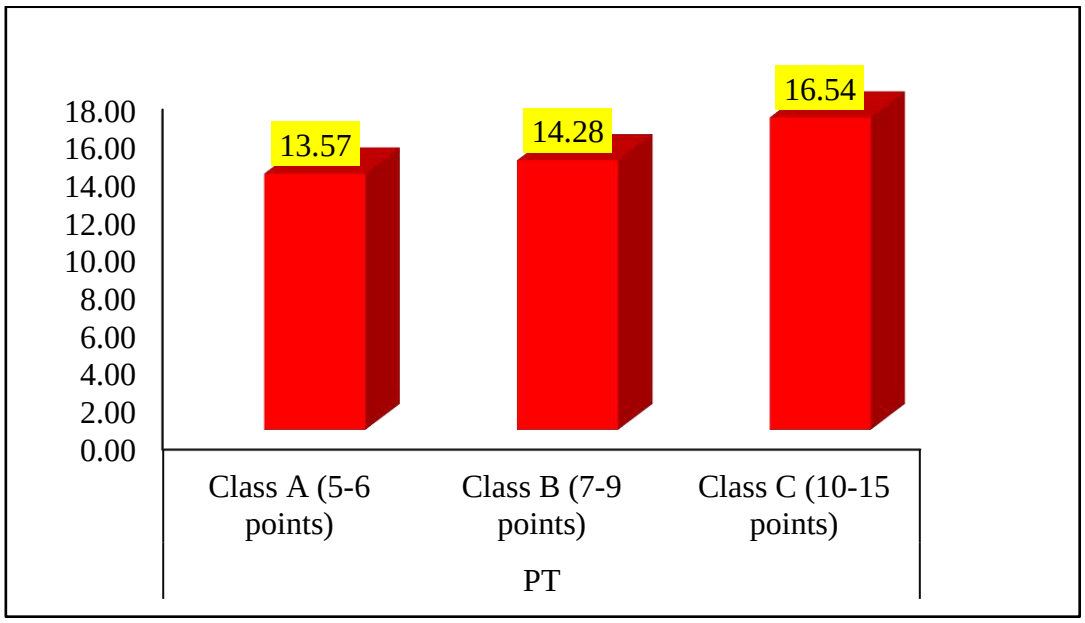
Among 9 patients with Grade 2 diastolic dysfunction , 1(11.1%) patients were in Child pugh Class B, and 8(88.9%) patients were in Child pugh Class C.

Only one patient was in Grade 3 diastolic dysfunction , 100% of the patients were in Child pugh Class C.

The difference indicate that the distribution of grades across the Child PUGH score classes is statistically significant(p=0.024).

Table 14: Descriptive Statistics for Prothrombin Time (PT)

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		F Value	P Value
						Lower Bound	Upper Bound		
PT	Class A (5-6 points)	7	13.57	0.535	0.202	13.077	14.066	3.029	0.056
	Class B (7-9 points)	18	14.28	2.081	0.490	13.243	15.313		
	Class C (10-15 points)	35	16.54	4.792	0.810	14.897	18.189		



Graph 20: Descriptive Statistics for Prothrombin Time (PT)

This table provides descriptive statistics for PT across different Child PUGH score classes. Mean PT increases progressively from Class A (13.57) to Class C (16.54), with a higher standard deviation in Class C indicating greater variability. The mean PT values increase from Class A to Class C, reflecting the worsening liver function.

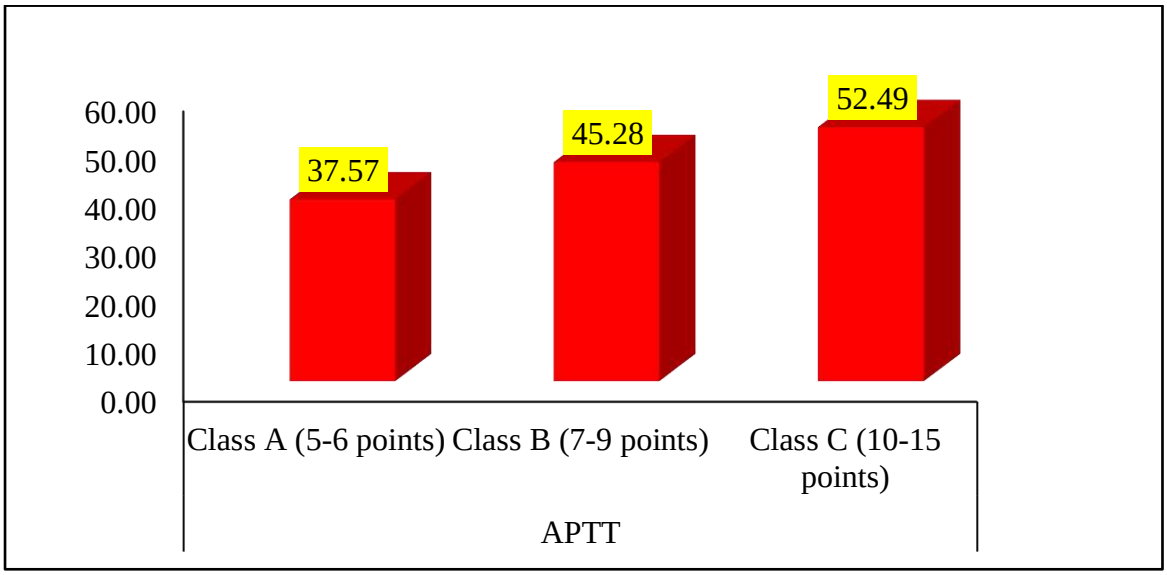
Table 15: Multiple Comparisons for Prothrombin Time (PT) using Bonferroni

Multiple Comparisons							
Bonferroni							
Dependent Variable			Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
PT	Class A	Class B	-0.70635	1.72618	1.000	-4.9643	3.5516
		Class C	-2.97143	1.60452	0.208	-6.9293	0.9864
	Class B	Class A	0.70635	1.72618	1.000	-3.5516	4.9643
		Class C	-2.26508	1.12401	0.146	-5.0377	0.5075
	Class C	Class A	2.97143	1.60452	0.208	-0.9864	6.9293
		Class B	2.26508	1.12401	0.146	-0.5075	5.0377
*. The mean difference is significant at the 0.05 level.							

This table presents multiple comparisons for PT between different Child PUGH score classes using the Bonferroni correction. No significant differences in PT between the classes using the Bonferroni correction.

Table 16: Descriptive Statistics for APTT (Activated Partial Thromboplastin Time) by Class

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		F Value	P Value
						Lower Bound	Upper Bound		
APTT	Class A (5-6 points)	7	37.57	2.992	1.131	34.804	40.339	5.864	**0.005**
	Class B (7-9 points)	18	45.28	8.837	2.083	40.883	49.672		
	Class C (10-15 points)	35	52.49	13.602	2.299	47.813	57.158		



Graph 21: Descriptive Statistics for APTT (Activated Partial Thromboplastin Time) by Class

This table presents the APTT values across three classes defined by their points. The mean APTT value in Class A was 37.57±2.992, in Class B 45.28±8.837 and in Class C was 52.49±13.602. Significant differences were found between Class A and Class C(p=005). APTT increases with higher class points.

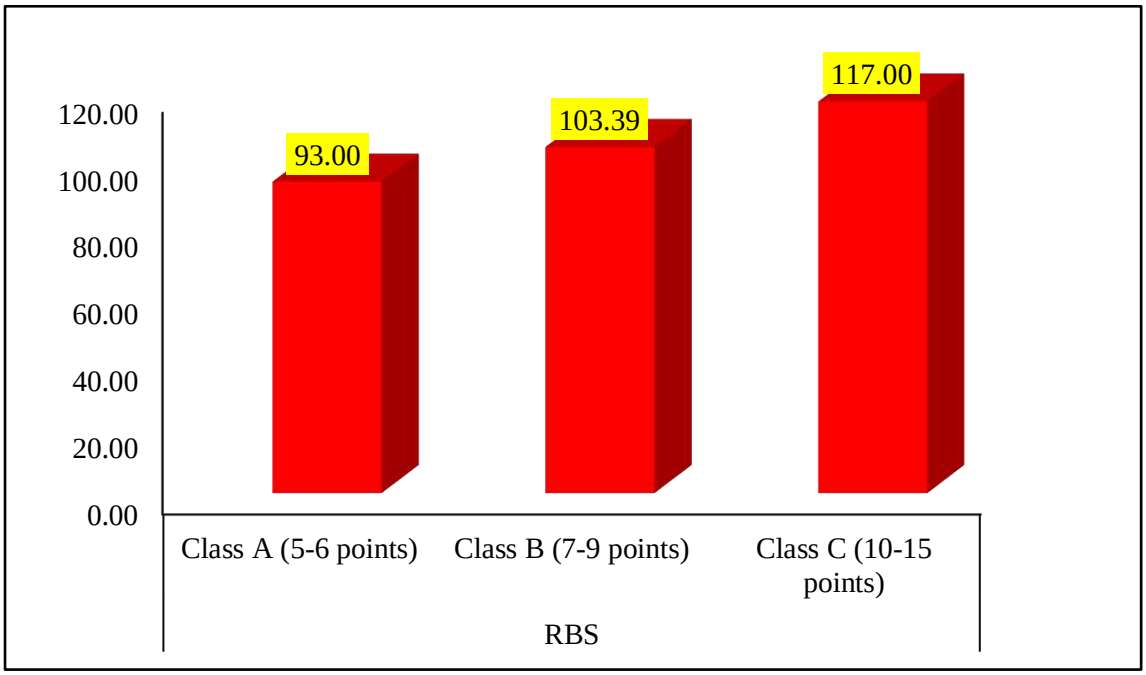
Table 17: Multiple Comparisons for APTT using Bonferroni

Multiple Comparisons							
Bonferroni							
Dependent Variable			Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
APTT	Class A	Class B	-7.70635	5.16777	0.424	-20.4536	5.0409
		Class C	-14.91429*	4.80353	0.009	-26.7631	-3.0655
	Class B	Class A	7.70635	5.16777	0.424	-5.0409	20.4536
		Class C	-7.20794	3.36501	0.109	-15.5084	1.0925
	Class C	Class A	14.91429*	4.80353	0.009	3.0655	26.7631
		Class B	7.20794	3.36501	0.109	-1.0925	15.5084
*. The mean difference is significant at the 0.05 level.							

This table presents multiple comparisons for APTT by Class using the Bonferroni correction. No significant differences in APTT between the classes using the Bonferroni correction.

Table 18: Descriptive Statistics for RBS by Class

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		F Value	P Value
						Lower Bound	Upper Bound		
RBS	Class A (5-6 points)	7	93.00	18.956	7.165	75.469	110.531	2.658	0.079
	Class B (7-9 points)	18	103.39	23.786	5.606	91.560	115.217		
	Class C (10-15 points)	35	117.00	32.867	5.556	105.710	128.290		



Graph 22: Descriptive Statistics for RBS by Class

This table shows the RBS values across three classes defined by their points. RBS shows an increasing trend with higher class points. The mean RBS in Class A was 93.00±18.956, in Class B 103.39±23.786 and in Class C 117.00±32.867. The differences between the classes are not statistically significant(p=0.079).

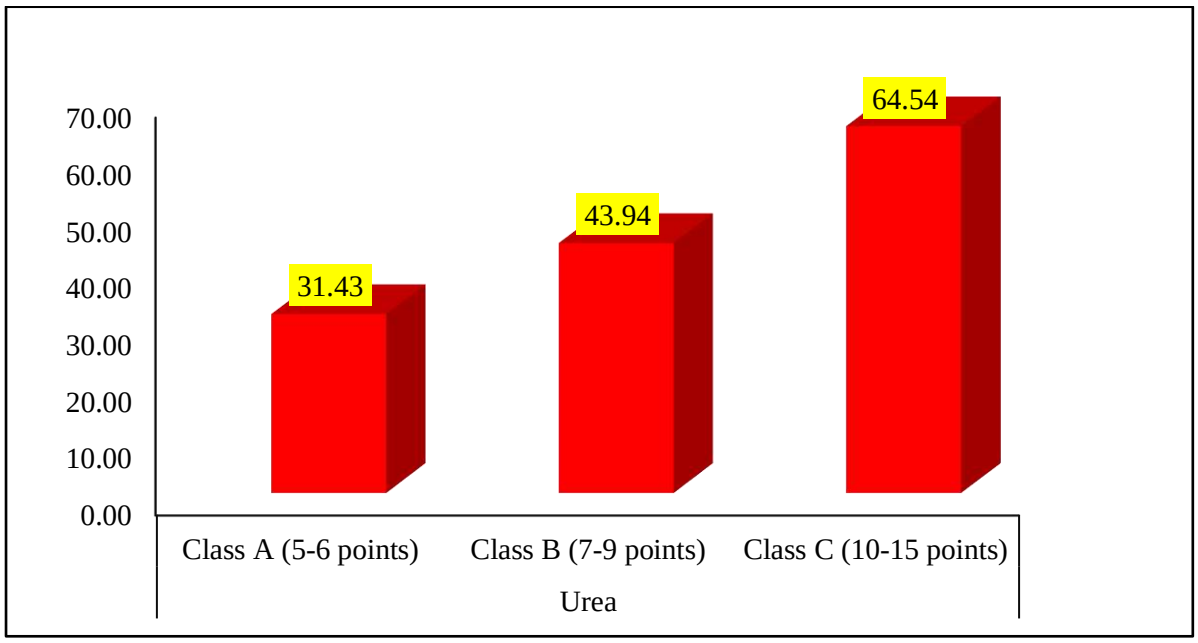
Table 19: Multiple Comparisons for RBS using Bonferroni

Multiple Comparisons							
Bonferroni							
Dependent Variable			Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
RBS	Class A	Class B	-10.38889	12.99357	1.000	-42.4400	21.6622
		Class C	-24.00000	12.07773	0.155	-53.7920	5.7920
	Class B	Class A	10.38889	12.99357	1.000	-21.6622	42.4400
		Class C	-13.61111	8.46080	0.340	-34.4813	7.2590
	Class C	Class A	24.00000	12.07773	0.155	-5.7920	53.7920
		Class B	13.61111	8.46080	0.340	-7.2590	34.4813
*. The mean difference is significant at the 0.05 level.							

This table presents multiple comparisons for RBS by Class using the Bonferroni correction. No significant differences in RBS between the classes using the Bonferroni correction.

Table 20: Descriptive Statistics for Urea by Class

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		F Value	P Value
						Lower Bound	Upper Bound		
Urea	Class A (5-6 points)	7	31.43	3.457	1.307	28.231	34.626	9.289	**0.000**
	Class B (7-9 points)	18	43.94	13.353	3.147	37.304	50.585		
	Class C (10-15 points)	35	64.54	27.285	4.612	55.170	73.916		



Graph 23: Descriptive Statistics for Urea by Class

This table shows the mean Urea values across three classes defined by their points. The mean urea in Class A was 31.43 mg/dL, in Class B 43.94 mg/dL and in Class C mean Urea was 64.54 mg/dL. And the difference was statistically significant increase in Urea levels with severity ($P < 0.05$).

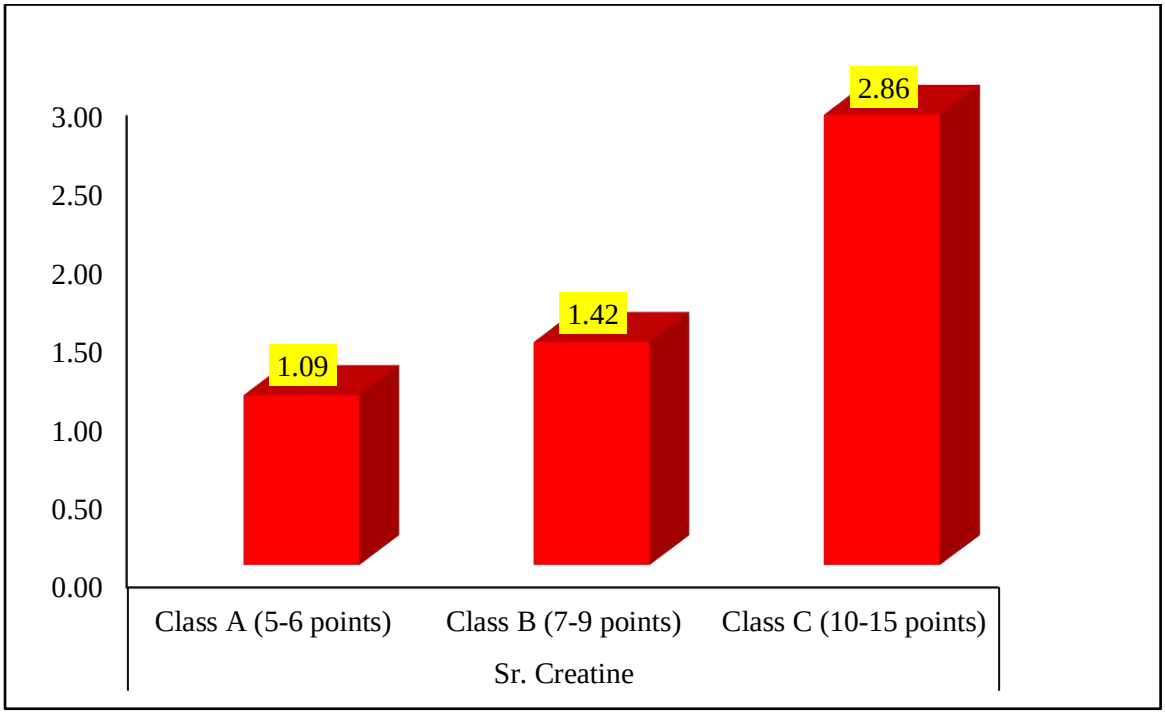
Table 21: Multiple Comparisons for Urea using Bonferroni

Multiple Comparisons							
Bonferroni							
Dependent Variable			Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
Urea	Class A	Class B	-12.51587	9.94543	0.640	-37.0481	12.0164
		Class C	-33.11429*	9.24444	0.002	-55.9174	-10.3111
	Class B	Class A	12.51587	9.94543	0.640	-12.0164	37.0481
		Class C	-20.59841*	6.47600	0.007	-36.5727	-4.6242
	Class C	Class A	33.11429*	9.24444	0.002	10.3111	55.9174
		Class B	20.59841*	6.47600	0.007	4.6242	36.5727
*. The mean difference is significant at the 0.05 level.							

This table presents multiple comparisons for Urea by Class using the Bonferroni correction. No significant differences in Urea between the classes using the Bonferroni correction.

Table 22: Descriptive Statistics for Sr. Creatinine by Class

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		F Value	P Value
						Lower Bound	Upper Bound		
Sr. Creatine	Class A (5-6 points)	7	1.09	0.204	0.077	0.897	1.274	4.950	**0.010**
	Class B (7-9 points)	18	1.42	0.447	0.105	1.200	1.644		
	Class C (10-15 points)	35	2.86	2.410	0.407	2.035	3.691		



Graph 24: Descriptive Statistics for Sr. Creatinine by Class

This table presents the Sr. Creatinine levels across three classes defined by their points. Sr. Creatinine levels increase with higher class points. The mean Sr. Creatinine in Class A was 1.09 mg/dL, in Class B 1.42 mg/dL and in Class C was 2.86 mg/dL. Significant differences are found between Class A and Class C(p=0.010).

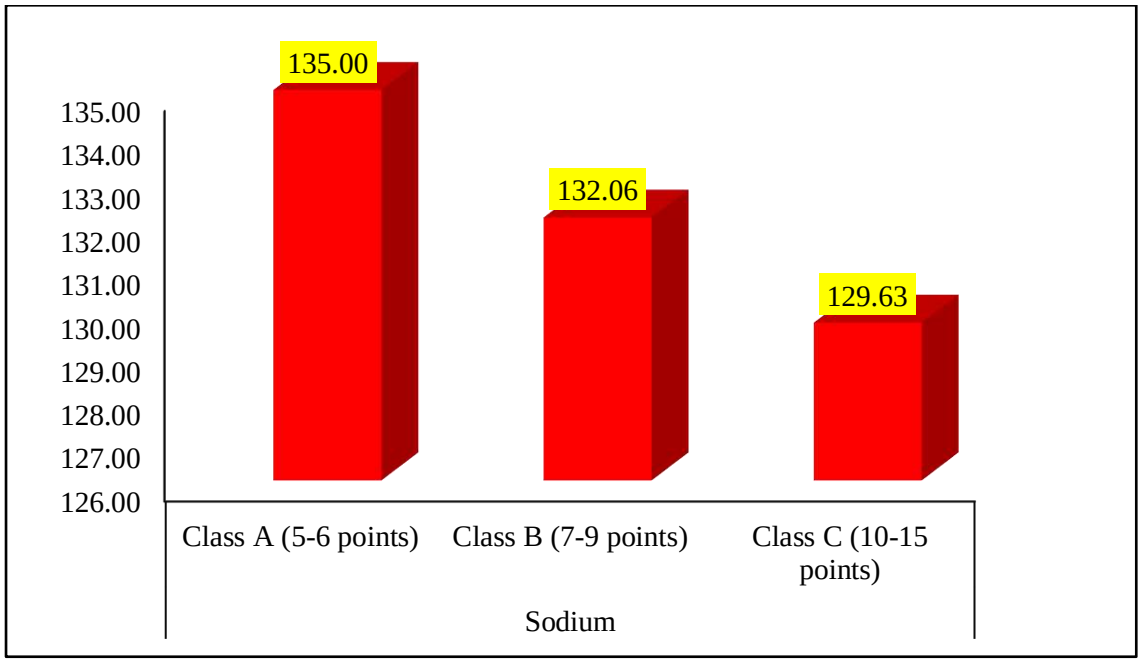
Table 23: Multiple Comparisons for Sr. Creatinine using Bonferroni

Multiple Comparisons							
Bonferroni							
Dependent Variable			Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
Sr. Creatine	Class A	Class B	-0.33651	0.83667	1.000	-2.4003	1.7273
		Class C	-1.77714	0.77770	0.078	-3.6955	0.1412
	Class B	Class A	0.33651	0.83667	1.000	-1.7273	2.4003
		Class C	-1.44063*	0.54480	0.032	-2.7845	-0.0968
	Class C	Class A	1.77714	0.77770	0.078	-0.1412	3.6955
		Class B	1.44063*	0.54480	0.032	0.0968	2.7845
*. The mean difference is significant at the 0.05 level.							

This table presents multiple comparisons for Sr. Creatinine by Class using the Bonferroni correction. No significant differences in Sr. Creatinine between the classes using the Bonferroni correction.

Table 24: Descriptive Statistics for Sodium by Class

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		F Value	P Value
						Lower Bound	Upper Bound		
Sodium	Class A (5-6 points)	7	135.00	4.509	1.704	130.830	139.170	2.503	0.091
	Class B (7-9 points)	18	132.06	5.150	1.214	129.494	134.617		
	Class C (10-15 points)	35	129.63	7.030	1.188	127.214	132.043		



Graph 25: Descriptive Statistics for Sodium by Class

This table shows the Sodium levels across three classes defined by their points. The Mean Sodium in Class A was 135.00 mEq/L, in Class B 132.06 mEq/L and in Class C was 129.63 mEq/L. The result showed a decreasing trend in Sodium levels with severity, though not statistically significant ($P > 0.05$).

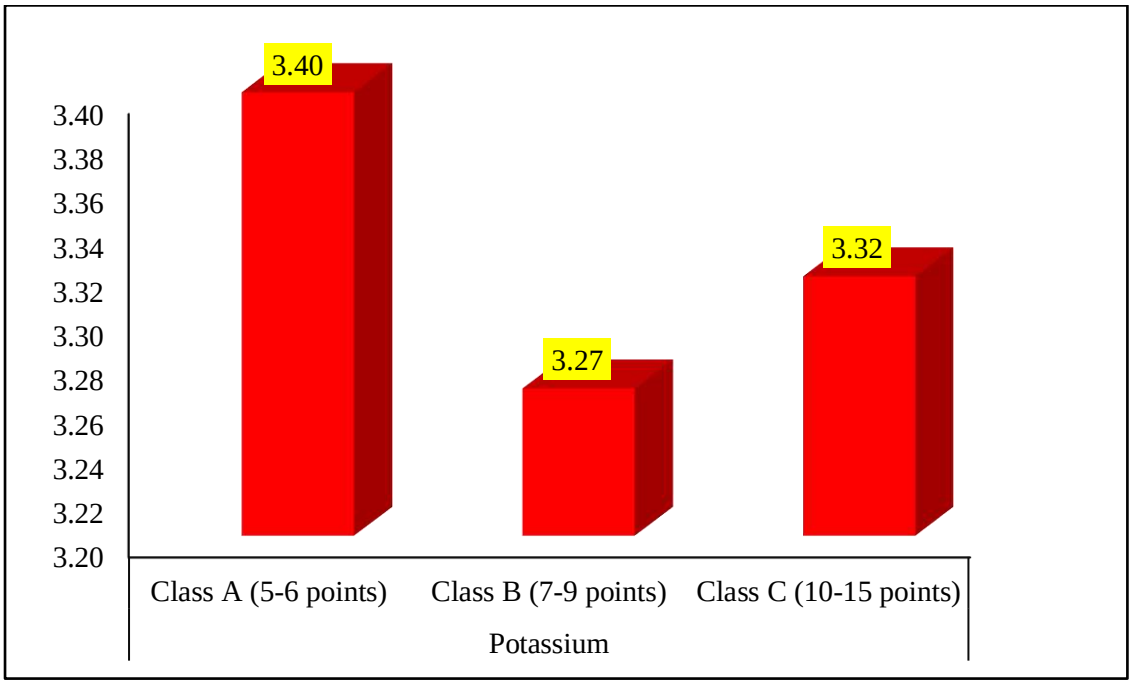
Table 25: Multiple Comparisons for Sodium using Bonferroni

Multiple Comparisons							
Bonferroni							
Dependent Variable			Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
Sodium	Class A	Class B	2.94444	2.80052	0.893	-3.9636	9.8525
		Class C	5.37143	2.60313	0.131	-1.0497	11.7925
	Class B	Class A	-2.94444	2.80052	0.893	-9.8525	3.9636
		Class C	2.42698	1.82357	0.566	-2.0712	6.9252
	Class C	Class A	-5.37143	2.60313	0.131	-11.7925	1.0497
		Class B	-2.42698	1.82357	0.566	-6.9252	2.0712
*. The mean difference is significant at the 0.05 level.							

This table presents multiple comparisons for Sodium by Class using the Bonferroni correction. No significant differences in Sodium between the classes using the Bonferroni correction.

Table 26: Descriptive Statistics for Potassium by Class

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		F Value	P Value
						Lower Bound	Upper Bound		
Potassium	Class A (5-6 points)	7	3.40	0.645	0.244	2.803	3.997	0.063	0.939
	Class B (7-9 points)	18	3.27	0.700	0.165	2.918	3.615		
	Class C (10-15 points)	35	3.32	0.957	0.162	2.988	3.646		



Graph 26: Descriptive Statistics for Potassium by Class

This table shows the Potassium levels across three classes defined by their points. The Mean Potassium in Class A was 3.40 mEq/L, in Class B 3.27 mEq/L and in Class C was 3.32 mEq/L. There was no significant difference in Potassium levels with severity ($P > 0.05$).

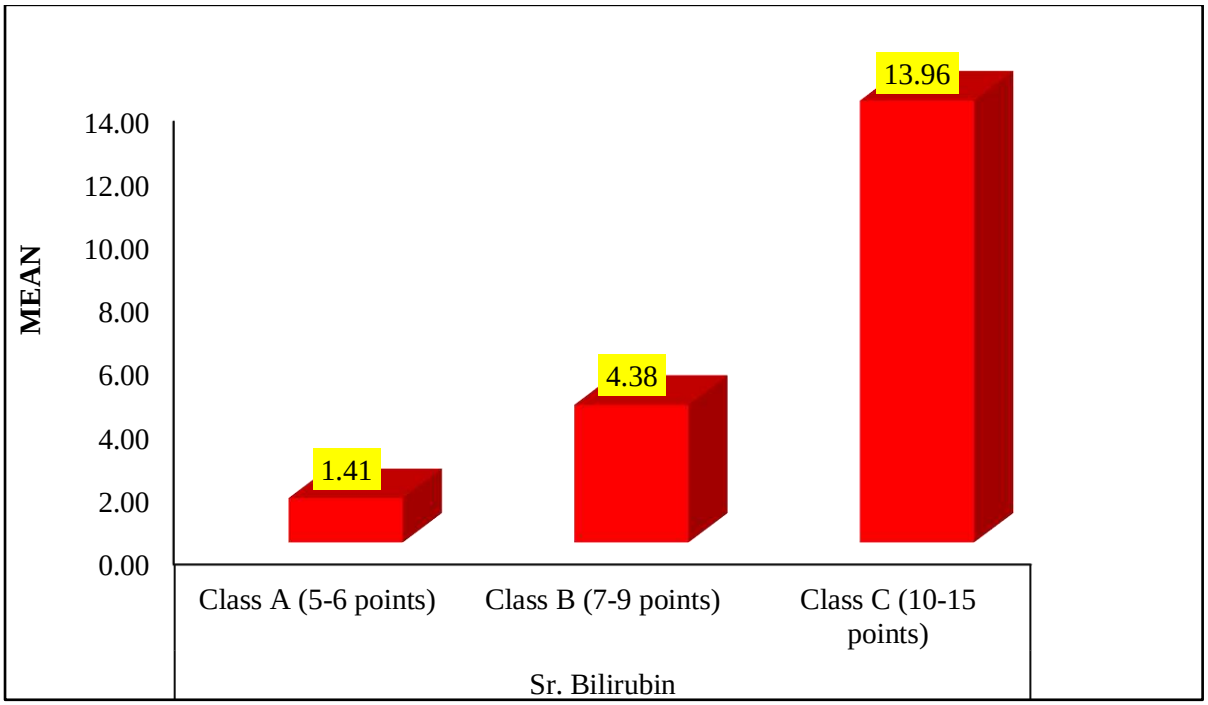
Table 27: Multiple Comparisons for Potassium using Bonferroni

Multiple Comparisons							
Bonferroni							
Dependent Variable			Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
Potassium	Class A	Class B	0.13333	0.38231	1.000	-0.8097	1.0764
		Class C	0.08286	0.35537	1.000	-0.7937	0.9594
	Class B	Class A	-0.13333	0.38231	1.000	-1.0764	0.8097
		Class C	-0.05048	0.24894	1.000	-0.6645	0.5636
	Class C	Class A	-0.08286	0.35537	1.000	-0.9594	0.7937
		Class B	0.05048	0.24894	1.000	-0.5636	0.6645
*. The mean difference is significant at the 0.05 level.							

This table presents multiple comparisons for Potassium by Class using the Bonferroni correction. No significant differences in Potassium between the classes using the Bonferroni correction.

Table 28: Descriptive Statistics for Sr. Bilirubin by Class

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		F Value	P Value
						Lower Bound	Upper Bound		
Sr. Bilirubin	Class A (5-6 points)	7	1.41	0.631	0.238	0.831	1.998	4.514	**0.015**
	Class B (7-9 points)	18	4.38	3.221	0.759	2.776	5.980		
	Class C (10-15 points)	35	13.96	17.216	2.910	8.043	19.871		



Graph 27: Descriptive Statistics for Sr. Bilirubin by Class

This table shows the Sr. Bilirubin levels across three classes defined by their points. The Mean Sr. Bilirubin in Class A was 1.41 mEq/L, in Class B 4.38 mEq/L and in Class C was 13.96 mEq/L. The result showed a statistically significant increase in Sr. Bilirubin levels with severity ($P < 0.05$).

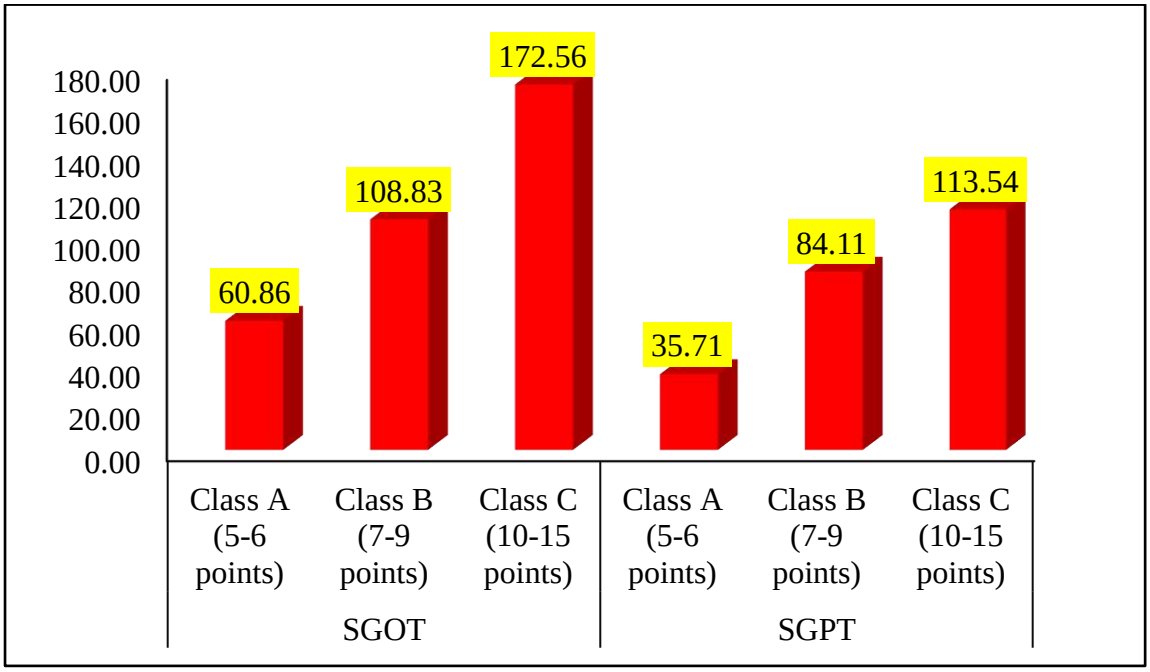
Table 29: Multiple Comparisons for Sr. Bilirubin using Bonferroni

Multiple Comparisons							
Bonferroni							
Dependent Variable			Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
Sr. Bilirubin	Class A	Class B	-2.96349	5.97507	1.000	-17.7021	11.7751
		Class C	-12.54286	5.55393	0.083	-26.2427	1.1569
	Class B	Class A	2.96349	5.97507	1.000	-11.7751	17.7021
		Class C	-9.57937	3.89069	0.051	-19.1765	0.0177
	Class C	Class A	12.54286	5.55393	0.083	-1.1569	26.2427
		Class B	9.57937	3.89069	0.051	-0.0177	19.1765
*. The mean difference is significant at the 0.05 level.							

This table presents multiple comparisons for Sr. Bilirubin by Class using the Bonferroni correction. No significant differences in Sr. Bilirubin between the classes using the Bonferroni correction.

Table 30: Descriptive Statistics for SGOT and SGPT by Class

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		F Value	P Value
						Lower Bound	Upper Bound		
SGOT	Class A (5-6 points)	7	60.86	43.291	16.363	20.819	100.895	5.666	**0.006**
	Class B (7-9 points)	18	108.83	46.201	10.890	85.858	131.808		
	Class C (10-15 points)	35	172.56	114.725	19.392	133.148	211.966		
SGPT	Class A (5-6 points)	7	35.71	26.756	10.113	10.969	60.460	3.548	**0.035**
	Class B (7-9 points)	18	84.11	46.354	10.926	61.060	107.162		
	Class C (10-15 points)	35	113.54	89.186	15.075	82.906	144.179		



Graph 28: Descriptive Statistics for SGOT and SGPT by Class

This table shows the SGOT & SGPT across three classes defined by their points. The Mean SGOT in Class A was 60.86 U/L, in Class B 108.83 U/L and in Class C was 172.56 U/L. The result showed a statistically significant increase in SGOT levels with severity ($P < 0.006$).

This table shows the SGPT levels across three classes defined by their points. The Mean SGPT in Class A was 35.71 U/L, in Class B 84.11 U/L and in Class C was 113.54 U/L. The result showed a statistically significant increase in SGPT levels with severity ($P < 0.035$).

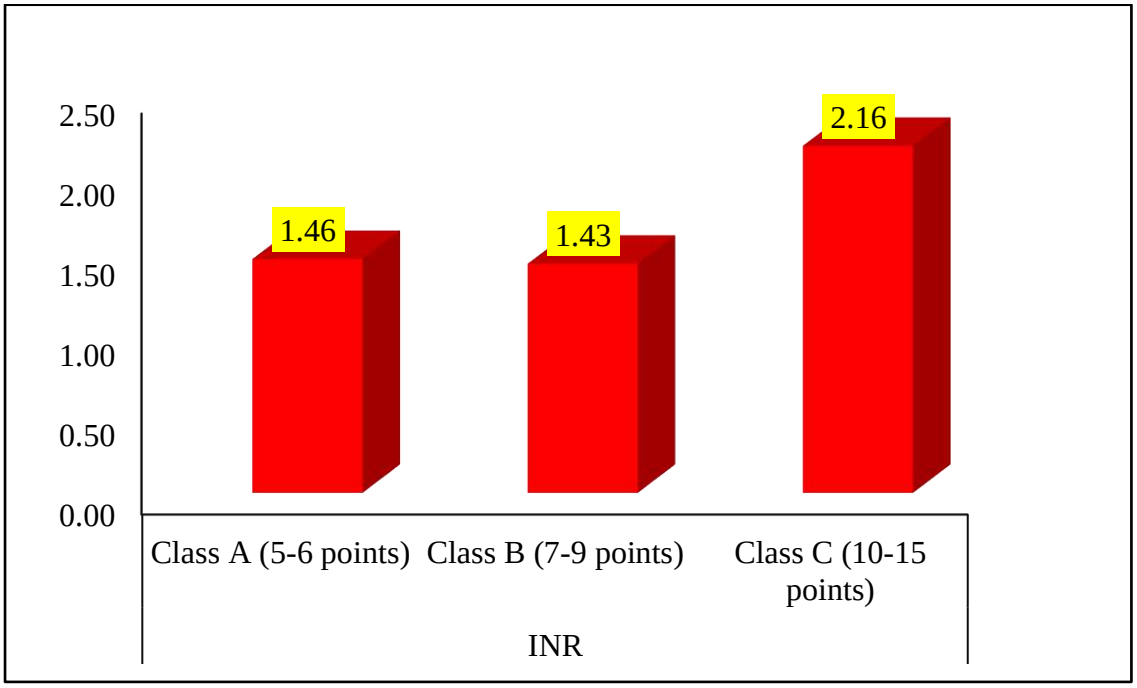
Table 31: Multiple Comparisons for SGOT and SGPT using Bonferroni

Multiple Comparisons							
Bonferroni							
Dependent Variable			Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
SGOT	Class A	Class B	-47.97619	41.51098	0.758	-150.3708	54.4184
		Class C	-111.70000*	38.58512	0.016	-206.8774	-16.5226
	Class B	Class A	47.97619	41.51098	0.758	-54.4184	150.3708
		Class C	-63.72381	27.02999	0.066	-130.3983	2.9507
	Class C	Class A	111.70000*	38.58512	0.016	16.5226	206.8774
		Class B	63.72381	27.02999	0.066	-2.9507	130.3983
SGPT	Class A	Class B	-48.39683	32.91635	0.441	-129.5912	32.7975
		Class C	-77.82857*	30.59627	0.041	-153.3000	-2.3571
	Class B	Class A	48.39683	32.91635	0.441	-32.7975	129.5912
		Class C	-29.43175	21.43357	0.525	-82.3017	23.4382
	Class C	Class A	77.82857*	30.59627	0.041	2.3571	153.3000
		Class B	29.43175	21.43357	0.525	-23.4382	82.3017
*. The mean difference is significant at the 0.05 level.							

This table presents multiple comparisons for SGOT & SGPT by Class using the Bonferroni correction. No significant differences in SGOT & SGPT between the classes using the Bonferroni correction.

Table 32: Descriptive Statistics for INR by Class

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		F Value	P Value
						Lower Bound	Upper Bound		
INR	Class A (5-6 points)	7	1.46	0.310	0.117	1.170	1.744	5.721	**0.005**
	Class B (7-9 points)	18	1.43	0.208	0.049	1.324	1.531		
	Class C (10-15 points)	35	2.16	1.046	0.177	1.805	2.523		



Graph 29: Descriptive Statistics for INR by Class

This table shows the INR levels across three classes defined by their points. The Mean INR in Class A was 1.46, in Class B 1.43 and in Class C was 2.16. Higher mean values in Class C. The result showed a statistically significant increase in INR levels with severity ($P < 0.005$).

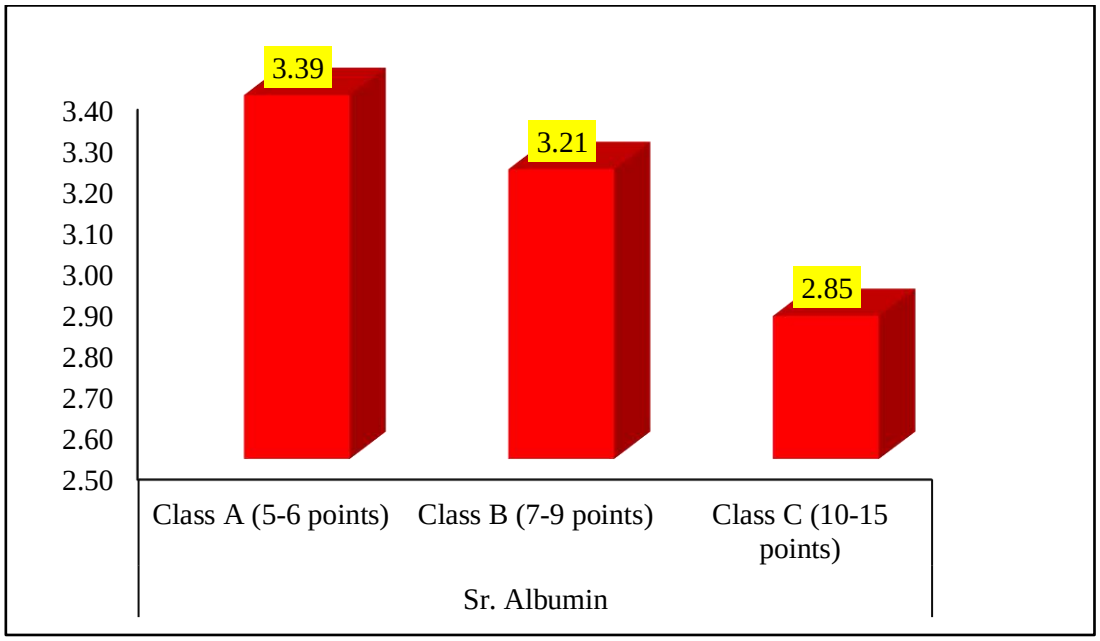
Table 33: Multiple Comparisons for INR using Bonferroni

Multiple Comparisons							
Bonferroni							
Dependent Variable			Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
INR	Class A	Class B	0.02937	0.36620	1.000	-0.8739	0.9327
		Class C	-0.70686	0.34039	0.127	-1.5465	0.1328
	Class B	Class A	-0.02937	0.36620	1.000	-0.9327	0.8739
		Class C	-.73622*	0.23845	0.009	-1.3244	-0.1480
	Class C	Class A	0.70686	0.34039	0.127	-0.1328	1.5465
		Class B	.73622*	0.23845	0.009	0.1480	1.3244
*. The mean difference is significant at the 0.05 level.							

This table presents multiple comparisons for INR by Class using the Bonferroni correction. No significant differences in INR between the classes using the Bonferroni correction.

Table 34: Descriptive Statistics for Sr. Albumin by Class

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		F Value	P Value
						Lower Bound	Upper Bound		
Sr. Albumin	Class A (5-6 points)	7	3.39	0.279	0.106	3.127	3.644	6.985	**0.002**
	Class B (7-9 points)	18	3.21	0.409	0.096	3.002	3.409		
	Class C (10-15 points)	35	2.85	0.461	0.078	2.690	3.007		



Graph 30: Descriptive Statistics for Sr. Albumin by Class

This table shows the Sr. Albumin levels across three classes defined by their points. The Mean Sr. Albumin in Class A was 3.39 g/dL, in Class B 3.21 g/dL and in Class C was 2.85 g/dL. Higher mean values in Class A. The result showed a statistically significant decrease in Sr. Albumin levels with severity ($P < 0.002$).

Table 35: Multiple Comparisons for Sr. Albumin using Bonferroni

Multiple Comparisons							
Bonferroni							
Dependent Variable			Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
Sr. Albumin	Class A	Class B	0.18016	0.19159	1.000	-0.2924	0.6528
		Class C	.53714*	0.17809	0.011	0.0979	0.9764
	Class B	Class A	-0.18016	0.19159	1.000	-0.6528	0.2924
		Class C	.35698*	0.12476	0.018	0.0492	0.6647
	Class C	Class A	-.53714*	0.17809	0.011	-0.9764	-0.0979
		Class B	-.35698*	0.12476	0.018	-0.6647	-0.0492
*. The mean difference is significant at the 0.05 level.							

This table presents multiple comparisons for Sr. Albumin by Class using the Bonferroni correction. No significant differences in Sr. Albumin between the classes using the Bonferroni correction.

Table 36 : Multiple correlation Analysis

Correlations		
		PUGH SCORE
PT	Pearson Correlation	.482**
	Sig. (2-tailed)	**0.000**
	N	60
APTT	Pearson Correlation	.540**
	Sig. (2-tailed)	**0.000**
	N	60
RBS	Pearson Correlation	0.179
	Sig. (2-tailed)	0.172
	N	60
Urea	Pearson Correlation	.583**
	Sig. (2-tailed)	**0.000**
	N	60
Sr. Cr	Pearson Correlation	.541**
	Sig. (2-tailed)	**0.000**
	N	60
Sodium	Pearson Correlation	-.381**
	Sig. (2-tailed)	**0.003**
	N	60
Potassium	Pearson Correlation	-0.120
	Sig. (2-tailed)	0.361
	N	60
Sr. Bil	Pearson Correlation	.472**
	Sig. (2-tailed)	**0.000**
	N	60
SGOT	Pearson Correlation	.519**
	Sig. (2-tailed)	**0.000**
	N	60
SGPT	Pearson Correlation	.448**
	Sig. (2-tailed)	**0.000**

	N	60
INR	Pearson Correlation	.650**
	Sig. (2-tailed)	**0.000**
	N	60
Sr. Alb	Pearson Correlation	-.610**
	Sig. (2-tailed)	**0.000**
	N	60
**. Correlation is significant at the 0.01 level (2-tailed).		
*. Correlation is significant at the 0.05 level (2-tailed).		

In the present study:

Positive Correlations: Significant positive correlations between the PUGH score and PT (0.482), APTT (0.540), Urea (0.583), Sr. Cr (0.541), Sr. Bil (0.472), SGOT (0.519), SGPT (0.448), and INR (0.650) was seen

Negative Correlations: Significant negative correlations between the PUGH score and Sodium (-0.381) and Sr. Albumin (-0.610) was seen

DISCUSSION

Cirrhosis of liver is associated with a wide range of cardiovascular abnormalities, like Left ventricular diastolic dysfunction, Systolic dysfunction, Pulmonary artery hypertension, Pericardial effussion ,RV dysfunction, Tricuspid regurgitation and changes in various vascular territories such as the renal and cerebral vasculature. These conditions highlight the extensive impact of cirrhosis beyond the liver, affecting multiple organ systems and leading to significant clinical complications.

In cirrhosis, scar tissue replaces normal, healthy liver tissue blocking the flow of blood through the organ. On ultrasonography, liver of long-standing cirrhosis appears shrunken. The normal echo pattern is lost, and it appears as coarse echo pattern often associated with splenomegaly, ascites and portal hypertension.

Cardiac dysfunction in cirrhosis often remains ignored. In 2023, the World Congress of Gastroenterology proposed diagnostic criteria for cirrhotic cardiomyopathy. These criteria include assessments of systolic and diastolic dysfunction using echocardiography. Additionally, the criteria encompass other supportive measures such as electrophysiological changes, serum biomarkers, and alterations in cardiac geometry. The comprehensive nature of these criteria underscores the multifaceted nature of cirrhotic cardiomyopathy and the importance of a thorough diagnostic approach to identify and manage this condition effectively.[101]

A total of 60 patients of cirrhosis of liver were selected for cross-sectional study by simple random sampling method. All patients were evaluated for cardiac changes in echo specially diastolic dysfunction.

The prevalence of cirrhotic cardiomyopathy among cirrhotic patients in the literature has been estimated between 50% and 70% in several studies.[102] In our study, the prevalence of cirrhotic cardiomyopathy reached 75% when assessing diastolic dysfunction. The higher prevalence of LVDD reported in our study may be due to the fact that our study population included more patients with decompensated cirrhosis.

The prevalence of LVDD (68.4%) in a study by Solanki R et al[92] similar to that in a recent study from India by Behera et al[103], reported a prevalence of 66.3%. In another study by Uthaya SM et al[96], 77 (82.8%) patients had one or more features of cirrhotic cardiomyopathy.

In the present study, overwhelming majority of male cases (98%), with only 2% female cases was seen. The male predominance is significant. The high male percentage suggests a possible gender-related factor influencing the prevalence or diagnosis rate. The overwhelming majority of male cases (98%) suggest potential gender-related susceptibility or bias in the sample population.

In a study, by Chandey M et al[95], found that cirrhosis was significantly more common in males (91.1%) compared to females (8.89%). Similarly, Solanki et al[92] reported that 85% of their patients were male. In a study by Uthaya SM et al[96], 83% of patients with cirrhotic cardiomyopathy were male. Nirmal A et al[94], also observed a high male predominance with a male-to-female ratio of 8:1.

In the present study, 30-50 years group has the highest number of cases. Only 3.33% of cases are above 70 years. There is a notable concentration of cases in the middle age groups (30-70 years), suggesting a higher prevalence or detection rate in these age ranges. There might be a correlation between age and the prevalence of diastolic dysfunction, particularly in the 30-50 and 50-70 age groups.

In the study conducted by Balde J et al[100], the majority of patients were males over 50 years old. This finding aligns with a Danish study by Dam Fialla A et al[104], which reported a higher incidence of cirrhosis among men. Similarly, Chandey M et al[95] and Lanzieri et al[99] also observed a significant number of male patients in the age group of 41-60 years in their respective studies on cirrhosis, with Lanzieri et al. reporting 50% male among 38 cirrhotic patients. Furthermore, Karki N et al[98], found that 70.5% of their cirrhotic patients were male and 29.5% were female and 54.3% of patients fell within the 40-60 years age group.

Among all the 2d echo findings Diastolic dysfunction was predominantly noticed in 45(75%) of patients, which was followed Tricuspid regurgitation 15(25%), Pulmonary artery hypertension 15(25%), RV dysfunction 15(25%) , Reduced LV ejection fraction 9(16%), Pericardial effusion 8(13%), Mitral regurgitation 5(8%), DCM 2(3%).

Among 15 cases of Tricuspid regurgitation , 4(27%) were in child pugh class A, 5(33%) were seen in child pugh class B , 6(40%) were seen in child pugh class C. In 15 cases of PAH, 4(27%) were in child pugh class A, 5(33%) were seen in child pugh class B , 6(40%) were seen in child pugh class C. In 15 cases of RV dysfunction , 4(27%) were in child pugh class A, 5(33%) were seen in child pugh class B , 6(40%) were seen in child pugh class C. In 9 cases of Reduced LVejection

Fraction, 3(33%) were in child pugh class A, 2(23%) were seen in child pugh class B , 4 (44%) were seen in child pugh class C. In 8 cases of Pericardial effusion, 2 (26%) were in child pugh class A, 3(37%) were seen in child pugh class B , 3(37%) were seen in child pugh class C.

In the present study, the severity of the diastolic dysfunction was studied. Grade 1diastolic dysfunction is the most common with 58%, followed by Grade 2 (15%), Grade 3 (2%), and Nil (25%). Grade 1 was the most common, with 58% of cases. Significant differences are observed in across different Child PUGH score classes, with p-values indicating statistical significance (p=0.024). A significant portion (25%) falls under 'nil,' indicating no grading applicable. Grade 2 and 3 are much less common. The distribution shows a trend towards less severe cases being more common. The grading shows most cases are in the lower severity grades (1 and 2), which might indicate early-stage intervention or a particular patient profile being studied.

In a systemic review done by Stundiene et al[105], it was found that 51.2% of cirrhotic patients had Lv diastolic dysfunction diagnosed and Grade I was almost prevalent (59.2%, P<0.001) among them, the Grade 3 was rarely diagnosed only 5.1%.

In the present study, patients were categorizes based on their Child PUGH score (Class A, B, or C).

Child PUGH score: A scoring system used to assess the prognosis of chronic liver disease, mainly cirrhosis.

- Class A: 5-6 points (least severe)

- Class B: 7-9 points (moderately severe)
- Class C: 10-15 points (most severe)

In the present study, most patients fall within the 30-50 and 50-70 age groups. Severity increases with age; the highest percentage of Class C patients are found in the 50-70 years age group. The Chi-square value is 2.544 with a p-value of 0.864, indicating no significant association between age and the Child PUGH score in this sample. There is a noticeable trend of increasing severity with advancing age. The lack of significant p-value suggests age is not a strong predictor of Child PUGH class in this dataset. Age alone may not be a reliable indicator of liver disease severity as per Child PUGH classification in this population.

In the current study, in distribution of Child PUGH scores across sexes, males predominantly represent the patient sample. All females fall within Class C. And the difference was not significant (p=0.695), indicating no significant difference between sex and Child PUGH score. The male predominance in the sample might skew results. The insignificant p-value suggests sex is not correlated with Child PUGH score. The sample size for females is too small to draw reliable conclusions.

In the present study, the relationship between diastolic function (E, A, E/A, DT, IVRT, LVSD, LVDD) and Child PUGH score was assessed. Diastolic function parameters are compared across Child PUGH Classes. Abnormal diastolic functions (especially LVDD) are more prevalent in higher Child PUGH classes. Only LVDD showed a significant p-value (0.002). LVDD has a significant correlation with higher

Child PUGH scores, indicating a potential link between liver disease severity and diastolic dysfunction. Diastolic function, particularly LVDD, is a crucial factor in liver disease severity.

In studies conducted by Karagiannakis DS , et al[106] and Lee SK et al[107] found that development of LVDD in cirrhotic patients is predictor of worse progonosis. Therefore it is more important to monitor closely cirrhotic patients who developed LVDD. Similar to other studies our studies showed that diastolic dysfunction was found in almost every patient of cirrhotic cardiomyopathy

In a study by Solankia R et al[92], reported that among patients with left ventricular diastolic dysfunction (LVDD), 56% had grade 1 and 44% had grade 2 LVDD. The incidence of LVDD was significantly higher in patients with Child-Turcotte-Pugh (CTP) class C ($P<0.001$). A study by Mishra A et al[97], observed a significant increase in the number of patients with diastolic dysfunction as the Child-Pugh score increased. Most patients in groups B and C exhibited a diastolic dysfunction grade 1, while a smaller proportion of patients in these groups had a grade 3 diastolic dysfunction. In a study by Solankia R et al[92], 68.4% of patients had left ventricular diastolic dysfunction, among these, 56% (n=78) had Grade 1 LVDD, 44% (n=61) had Grade 2 LVDD, and none had Grade 3 LVDD.

In the study, the distribution of patients across different grades and Child PUGH score classes (A, B, and C) were studied. There is a clear trend of increasing severity

(higher grades) with higher Child PUGH score classes, especially noticeable in Grade 1 and Grade 2 distributions. The chi-square test shows significant differences in grade distribution across Child PUGH score classes, emphasizing the clinical relevance of the Child PUGH classification in predicting patient severity. The difference indicate that the distribution of grades across the Child PUGH score classes was statistically significant(p=0.024).

In the present study, mean PT increases progressively from Class A (13.57) to Class C (16.54), with a higher standard deviation in Class C indicating greater variability. The mean PT values increase from Class A to Class C, reflecting the worsening liver function. The standard deviation increases significantly in Class C, suggesting greater variability in PT among patients with more severe liver disease. Increasing mean PT values with higher Child PUGH classes underscore the progressive nature of liver dysfunction. The mean differences between classes are not statistically significant after Bonferroni correction, indicating that while there are observed differences in PT, they are not significant when adjusted for multiple comparisons. The higher standard deviation in Class C for PT highlights the increased heterogeneity in liver function among the most severe cases.

In a study conducted by Siddiqui SA et al, in 171 patients of chronic liver disease, prothrombin time was prolonged in 150(88%) patients.[108]

In the present study, mean APTT value in Class A was 37.57±2.992, in Class B 45.28±8.837 and in Class C was 52.49±13.602. Significant differences were found

between Class A and Class C(p=005). APTT increases with higher class points and the difference was statistically significant(P= 0.005). Higher mean APTT in higher class points. Significant difference between classes A and C was noticed. The analysis showed that as the class points increase, the mean APTT also increases, indicating a possible correlation between the severity (or class points) and APTT values.

In the present study, RBS showed an increasing trend with higher class points. The mean RBS in Class A was 93.00±18.956, in Class B 103.39±23.786 and in Class C 117.00±32.867. The differences between the classes are not statistically significant(p=0.079). Mean RBS increases with higher class points. The mean RBS values show an increasing trend with higher class points, although the differences are not statistically significant. While there is an upward trend in RBS values with higher class points, the lack of statistical significance suggests that other factors might influence RBS levels, and the correlation is not strong.

In the present study, mean urea in Class A was 31.43 mg/dL, in Class B 43.94 mg/dL and in Class C mean Urea was 64.54 mg/dL. Higher urea indicates impaired kidney function. Significant increase in mean Urea from Class A to C. And the difference was statistically significant increase in Urea levels with severity ($P < 0.05$). Mean Urea increases significantly with severity class. Strong positive correlation between Urea levels and severity class, indicating worsening condition with higher Urea. No significant differences in Urea between the classes using the Bonferroni correction was noticed.

In the present study, mean Sr. Creatinine levels in Class A was 1.09 mg/dL, in Class B 1.42 mg/dL and in Class C was 2.86 mg/dL. Significant differences are found between Class A and Class C(p=0.010). There was a statistically significant increase in Sr. Creatinine levels with severity ($P < 0.05$). Higher Sr. Creatinine indicates impaired kidney function. Significant increase in mean Sr. Creatinine from Class A to C. The significant increase in Sr. Creatinine levels with higher class points suggests a correlation between severity and kidney function, as indicated by creatinine levels. Higher class points correlate with increased creatinine, indicating potential kidney impairment or increased severity of the condition.

In the present study, mean sodium in Class A was 135.00 mEq/L, in Class B 132.06 mEq/L and in Class C was 129.63 mEq/L. The result showed a decreasing trend in Sodium levels with severity, though not statistically significant ($P > 0.05$). Sodium levels show a decreasing trend with higher class points. No significant differences are found between the classes. While there is a downward trend in Sodium levels with higher class points, the lack of statistical significance suggests that the correlation is weak. Other factors might influence Sodium levels, and further investigation is needed to determine the exact relationship. Sodium levels can indicate fluid balance and kidney function.

In the study conducted by Al Atroush et al[93], sodium levels did not show clinical significance, with a p-value of 0.35, indicating no meaningful difference between patients and controls.

In the present study, mean potassium in Class A was 3.40 mEq/L, in Class B 3.27 mEq/L and in Class C was 3.32 mEq/L. There was no significant difference in

Potassium levels with severity (P > 0.05). Mean Potassium remains relatively constant across severity classes. Potassium levels can indicate kidney function and electrolyte balance. Little variation in mean Potassium across classes. Potassium levels do not show a strong correlation with severity class.

In the study conducted by Al Atroush et al[93], potassium levels were not clinically significant, with a p-value of 0.89, further suggesting no substantial difference between the two groups in this parameter.

In the present study, mean Sr. Bilirubin in Class A was 1.41 mEq/L, in Class B 4.38 mEq/L and in Class C was 13.96 mEq/L. The result showed a statistically significant increase in Sr. Bilirubin levels with severity (P < 0.05). Higher Sr. Bilirubin indicates liver dysfunction. Mean Sr. Bilirubin increases significantly with severity class. Strong positive correlation between Sr. Bilirubin levels and severity class, indicating worsening condition with higher Sr. Bilirubin.

In the study conducted by Al Atroush et al[93], bilirubin levels were significantly higher in patients compared to controls, with a p-value of less than 0.001, indicating a strong statistical association.

Study showed progressive increase in SGOT mean values from Class A (60.86) to Class C (172.56), with significant differences (p = 0.006). In SGPT also an increasing trend from Class A (35.71) to Class C (113.54), with significant differences (p = 0.035) was seen. The significant increase in SGOT levels with higher class points suggests a correlation between severity and liver function. Significant differences in mean values were found between Class C and Class A, indicating higher liver enzyme levels in more severe classes and indicating potential liver damage or increased

severity of the condition. SGPT levels increase with higher class points(p=0.035). The significant increase in SGPT levels with higher class points suggests a correlation between severity and liver function. Higher class points correlate with increased SGPT levels, indicating potential liver damage or increased severity of the condition.

In the present study, higher mean INR values in Class C (2.16) compared to Class A (1.46) and Class B (1.43) was observed. Higher mean values were in Class C. The result showed a statistically significant increase in INR levels with severity ($P < 0.005$). Significant differences were found between Class C and Class B, with Class C having higher INR values. INR increases with higher class points. The significant increase in INR with higher class points suggests a correlation between severity and blood coagulation status. Higher class points correlate with increased INR, indicating potential coagulation abnormalities or increased severity of the condition. The data indicates a strong positive correlation between class points and INR values, reflecting the impact of disease severity on blood coagulation.

In the study by Uthaya SM et al[96], International Normalized Ratio (INR) values were found to have clinical significance, with a p-value of 0.043. This statistically significant result suggests that INR values are an important factor in the clinical assessment of patients. The p-value indicates a reliable association, highlighting the relevance of INR in the context of the study and its potential implications for patient management and treatment strategies. In the study conducted by Al Atroush et al[93], INR was also significantly elevated in patients compared to controls, with a p-value of less than 0.001, emphasizing its clinical importance.

In the present study, Sr. Albumin levels decrease with higher class points. The Mean Sr. Albumin in Class A was 3.39 g/dL, in Class B 3.21 g/dL and in Class C was 2.85 g/dL. Higher mean values in Class A. The result showed a statistically significant decrease in Sr. Albumin levels with severity ($P < 0.002$). The significant decrease in Sr. Albumin levels with higher class points suggests a correlation between severity and liver function. Higher class points correlate with decreased Sr. Albumin levels, indicating potential liver dysfunction or increased severity of the condition. The data indicates a strong negative correlation between class points and Sr. Albumin levels, highlighting the impact of disease severity on liver function.

In the study conducted by Uthaya SM et al[92], serum albumin levels were found to have clinical significance, with a p-value of 0.038. This indicates a statistically significant association, suggesting that serum albumin levels may play an important role in the clinical evaluation and management of the patients studied. The lower p-value underscores the reliability of this finding in the context of their research.

CONCLUSION

- Cardiac dysfunction is a common but often over looked complication in patients with liver cirrhosis.
- In our study majority of the patients were in middle age group (30-50 years) predominantly males.
- 2D Echo findings seen in cirrhosis of liver patients are Left ventricular diastolic dysfunction, Tricuspid regurgitation, Pulmonary artery hypertension, RV dysfunction, Pericardial effusion, Reduced LV ejection fraction, Mitral Regurgitation.
- Among all the 2D Echo findings, Left ventricular diastolic dysfunction was predominantly seen and noticed early.
- Our Study demonstrate a significant association between liver cirrhosis and Left ventricular diastolic dysfunction. Grade 1 Left ventricular diastolic dysfunction was more common.
- Our Study demonstrate that as the Child Pugh Score increases, the severity of left ventricular diastolic dysfunction also increases. LVDD is a predictor of poor prognosis in cirrhosis of liver patients.
- In our Study we have noticed that as the severity of liver cirrhosis increases, blood markers such has PT,APTT, Urea, SGOT,SGPT,INR also increases.
- Medical management of CCM mainly includes beta blockers like metaprolol, loop diuretics like furosemide , potassium sparing diuretics like spironolactone, ACE inhibitor like ramipril. Newer drugs like Sacubitril-Valsartan(Angiotensin receptor- neprilysin inhibitor), Dapagliflozin(SGLT 2 inhibitor) can also be used in the management of CCM.

- Liver transplantation remains a potential intervention that can lead to the normalization of cardiac function in CCM patients, often resulting in significant improvements post transplantation.

LIMITATIONS OF THE STUDY

- The sample size was small (60).

- This was a single centre study, hence before generalization the study needs to be done in other populations.

SUMMARY

This cross-sectional study was conducted on a total of 60 patients of cirrhosis of liver selected by simple random sampling method in patients admitted to Raichur Institute of Medical Sciences, Raichur during Period of August 2022 to February 2024. The study was aimed to assess cardiac status by echocardiography in cirrhosis of liver patients and its correlation with child pugh score. All patients were evaluated for diastolic dysfunction. All adult patients with diagnosis of Cirrhosis of liver of any etiology were included in the study.

The results of our study are summarized as follows:

- The majority of cases fall within the 30-50 years age group (55%), followed by the 50-70 years group (35%)(p-value= 0.864).
- Severity of cirrhosis of liver increases with age; the highest percentage of Class C patients are found in the 50-70 years age group.
- There is a significant gender disparity, with 98% of the cases being male and only 2% female(p-value= 0.695).
- Among all the 2d echo findings , Left ventricular diastolic dysfunction was predominantly noticed in 45(75%) of patients .
- Grade 1 left ventricular diastolic dysfunction is the most common with 58%, followed by Grade 2 (15%), Grade 3 (2%), and Nil (25%).
- In 15 cases of Tricuspid regurgitation , 4(27%) were in child pugh class A, 5(33%)were seen in child pugh class B , 6(40%) were seen in child pugh class C.

- Among 15 cases of PAH, 4(27%) were in child pugh class A, 5(33%) were seen in child pugh class B , 6(40%) were seen in child pugh class C.

- Among 15 cases of Rv dysfunction, 4(27%) were in child pugh class A, 5(33%) were seen in child pugh class B , 6(40%) were seen in child pugh class C.

- In 9 cases of Reduced LV ejection Fraction, 3(33%)were in child pugh class A, 2(23%) were seen in child pugh class B , 4(44%)were seen in child pugh class C.
- In 8 cases of Pericardial effusion , 2(26%) were in child pugh class A, 3(37%) were seen in child pugh class B , 3(37%) were seen in child pugh class C.
- Abnormal LVDD are more prevalent in higher Child PUGH classes(p-value= 0.002). Significant association between LVDD and Child PUGH score was observed in our study.
- Other parameters, such as PT, APTT, RBS, Urea, Sr. Creatinine, Sodium, and Potassium, show varying levels of significance,as described below indicating the clinical relevance of these measures.
- The mean PT values increase from Class A to Class C, reflecting the worsening liver function(p-value= 0.056).
- Mean PT increases with severity of class.The differences are not statistically significant ($P > 0.05$).
- Higher APTT indicates a longer time for blood clotting. Mean APTT increases significantly with severity of class. Significant increase in mean APTT from Class A to C($P > 0.005$).

- Mean RBS increases with higher class points. No significant difference found between classes(p-value = 0.079).
- Higher mean Urea in higher class points. Significant differences between multiple classes(p-value = 0.000).
- Progressive increase in mean values from Class A (60.86) to Class C (172.56), with significant differences (p-value = 0.006).
- Progressive increasing trend from Class A (35.71) to Class C (113.54). SGPT levels increase with higher class points. Significant differences are found between the classes(p-value = 0.035).
- Mean INR increases with higher class points, higher mean values in Class C (2.16) compared to Class A (1.46). Significant differences are found between the classes(p-value = 0.005).
- Mean Sr. Creatinine increases significantly with severity class. Significant increase in mean Sr. Creatinine from Class A to C(p-value = 0.010).
- Mean Sodium decreases with severity class. Differences are not statistically significant ($P > 0.05$).
- Mean Potassium remains relatively constant across severity classes. Differences are not statistically significant ($P > 0.05$).
- Mean Sr. Bilirubin increases significantly with severity of class. Differences are statistically significant ($P < 0.05$).
- Positive Correlations: High positive correlations with PT, APTT, Urea, Sr. Cr, Sr. Bil, SGOT, SGPT, and INR indicate that these markers increase with disease severity.

- Negative Correlations: Negative correlation with Sodium and Sr. Albumin suggests that these markers decrease as the PUGH score increases, indicating worsening liver function.

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INFORMED CONSENT

I Mr / Mrs _____Son / Daughter/Wife of Mr/ Mrs _____ aged about _____ years, have been explained in a language best understood by me about the "CARDIAC STATUS BY ECHOCARDIOGRAPHY IN CIRRHOSIS OF LIVER PATIENTS AND ITS CORRELATION WITH CHILD PUGH SCORE" at the department of General medicine, RIMS,Raichur conducted by Dr Banda Naveen under the guidance of Dr Hari Prasad S, Associate Professor ,Department of General Medicine, RIMS,Raichur.

I have been explained about the procedures and investigations that will be done during this study. I have no objection in sharing my medical information and details in case records with the investigators of this study. I have been informed that I will not be sharing any incentives. Personal identity will not reveal and data may be used for publication/ dissertation purpose.

I understand my participation in this study is entirely voluntary and I willfully give consent regarding participation in this study for the specified duration. I may withdraw at any time of the study and I will not hold responsibility against any doctor, staff or hospital management if any untoward complications occur.

Date:

Place:

ಒಪ್ಪಿಗೆ ಪತ್ರ

ದಿನಾಂಕ:_____

ಶ್ರೀ/ಶ್ರೀಮತಿ/ಕುಮಾರ/ಕುಮಾರಿ. _____ ಆದ ನಾನು, ಈ ಕೆಳಕಂಡ ವೈದ್ಯಕೀಯ ಸಂಶೋಧನೆ "CARDIAC STATUS BY ECHOCARDIOGRAPHY IN CIRRHOSIS OF LIVER PATIENTS AND ITS CORRELATION WITH CHILD PUGH SCORE "

ಈ ಮೇಲ್ಕಂಡ ಸಂಶೋಧನೆಗೆ ಸಂಬಂಧಿಸಿದಂತೆ, **RIMS , RAICHUR GENERAL MEDICINE** ವಿಭಾಗದ **Dr Banda Naveen** ರವರು **Dr. Hari Prasad S** ಇವರ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ನಡೆಸುತ್ತಿರುವ ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಭಾಗವಹಿಸಲು ನನ್ನ ಸಂಪೂರ್ಣ ಒಪ್ಪಿಗೆ ಇದ್ದು, ಇದರ ಧ್ಯೇಯೋದ್ದೇಶಗಳು, ಉಪಯೋಗಗಳು, ಸಂಭವಿಸಬಹುದಾದ ಅಡ್ಡಪರಿಣಾಮಗಳು ಹಾಗು ತೀವ್ರತೆಯ ಬಗ್ಗೆ ನನಗೆ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಿರುತ್ತಾರೆ.

ಇದಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನನ್ನ ಆರೋಗ್ಯಕ್ಕೆ ಯಾವುದೇ ತೊಂದರೆಯಾದಲ್ಲಿ ಮೇಲ್ಕಂಡ ವೈದ್ಯರು/ಸಂಸ್ಥೆಯನ್ನು ದೂಷಿಸುವುದಿಲ್ಲವೆಂದು ತಿಳಿಯಪಡಿಸುತ್ತೇನೆ.

ಈ ಸಂಶೋಧನೆಯ ಯಾವುದೇ ಹಂತದಲ್ಲಿ ನಾನು ಹೊರನಡೆಯಬಹುದಾಗಿದೆ ಎಂದು ನನಗೆ ತಿಳಿಸಲಾಗಿದೆ.

ರೋಗಿಯ ಸಹಿ

ಎಡ ಹೆಬ್ಬೆಟ್ಟಿಗುರುತು

सहमतिपत्र

अध्ययन शीर्षक CARDIAC STATUS BY ECHOCARDIOGRAPHY IN CIRRHOSIS OF LIVER PATIENTS AND ITS CORRELATION WITH CHILD PUGH SCORE"

अन्वेषक: **Dr Banda Naveen** ,अध्य|पक: **Dr Hari Prasad S** , Assistant PROFESSOR OF MEDICINE, RIMS, RAICHUR .

प्रतिभागीकेपूर्णनाम _____

1. मेरी पुष्टि है की मैंने अध्ययन हेतु सूचना पत्रको पढ़ व समझ लिया तथा मुझे प्रश्न पूछने या मुझे अध्ययन अन्वेशक ने सभी तत्थो को समझा दिया है तथा मुझे प्रशन पूछने के समान अवसर प्रदान किये गए |

2. मैं यहाँ समझा लिया की अध्ययन मे मेरी भागीदारी पूर्णतः स्वैच्छिकहै औरमैंकिसी भी समयकिसी भी कारणके बिना, मेरे इलाज याकानूनी अधिकारोंकोप्रभावितकिये बिना, अध्ययन में भाग न लेनेकेलिएस्वतंत्रहूँ |

3. मैं यह समझ लिया है कि अध्ययन के प्रायोजक, प्रायोजक की तरफ से काम करने वाले लोग, आचार समितिऔरनियामक अधिकारियों को मेरे स्वास्थ्यरिकॉर्ड को वर्तमान अध्ययन या आगे के अध्ययन के सन्दर्भ देखने के लिए मेरी अनुमतिकी जरूरत नहीं है, चाहे मैंने इस अध्ययन से अपना नाम वापस ले लिया हो | हालांकि,मैं यह समझता हूँकिमेरीपहचानको किसी भीतीसरे पक्ष या प्रकाशित माध्यम में नहीं दी जायेगी |

4. मैं इससे सहमत हूँ कि कोई भी डेटा यापरिणाम जो इस अध्ययन से प्राप्त होता है उसका वैज्ञानिकउद्देश्य (ओं) केउपयोग के लिए मेरी तरफ से कोई प्रतिबन्ध नहीं है |

5. मैंभविष्य के अनुसंधानके लिए भंडारितनमूना(ऊतक /रक्त) परअध्ययन के लिएअपनीसहमति देता हूँ | मैंउपरोक्तअध्ययन मेंभागलेने के लिए सहमत हूँ |

अन्वेषककेहस्ताक्षर- प्रतिभागीकाहस्ताक्षर

ETHICAL COMMITTEE CLEARANCE CERTIFICATE

ಕರ್ನಾಟಕ ಸರ್ಕಾರ

Government of Karnataka

ರಾಯಚೂರು ವೈದ್ಯಕೀಯ ವಿಜ್ಞಾನಗಳ ಸಂಸ್ಥೆ, ರಾಯಚೂರು

Raichur Institute of Medical Sciences,Raichur.

(Autonomous Institution) Phone / Fax-08532 238488/89)

Institutional Ethics Committee Approval

Date:22-07-2022

To

Dr. Banda Naveen
Post Graduate,
Dept of General Medicine
RIMS, Raichur

Ref.: Protocol titled " TO STUDY CARDIAC STATUS BY ECHOCARDIOGRAPHY IN CIRPHOSIS OF OF LIVER PATIENTS AND ITS CORRELATION WITH CHILD PUGH SCORE."

with Ref No : RIMS/IEC/Teach. Staff/2022-23/31, Dated: 30-05-2022
Sub : Institutional Ethics Committee approval for the study.
Dear Dr.Banda Naveen

We have received from you, following study related documents vide letter dated 30-05-2022 At the Institutional Ethics Committee meeting held on 08-07-2022 and 16-07-2022, Rectification document submitted date on 16-07-2022, and the above mentioned documents were examined and discussed date: 16-07-2022, After consideration, the committee has decided to approve the above mentioned study proposal,

The following members attended the meeting at which your study proposal was discussed are:

1. Dr.Basawaraj Patil	Member (Session Chairman)
2. Dr. Basavaraj. M.Patil	Principal
3. Dr. Naveen N.S	Member
4. Dr.S.D.Mandolkar	Member
5. Dr.Venkatesh Naik R	Member
6. Dr. Manjunath H	Member
7. Dr. Arvind B. Sangavi	Member
8. Dr.Mohd Arshad	Member
9. Dr.Chandrashekhar R	Member
10. Dr.Harish Murthy	Member
11. Dr.Vijay Kumar Sukhani	Member
12. Dr.Jagadish Gupta	Member, Lions Club
13. Dr. Rahul C Kirte	Member Secretary

All members voted for the proposed study and no members voted against the proposed study. It is to be noted that neither you nor any of your proposed study team members were present during the decision making procedures of the Institutional Ethics Committee. You are required to submit the progress report to the IEC every six months and at the completion of the project.

Yours sincerely,

Chairman
Institutional Ethics Committee.
Raichur Institution of Medical Sciences, Raichur.

Member Secretary,
Institutional Ethics Committee
Raichur Institution of Medical Sciences, Raichur

PROFORMA

Patient Information:

Serial number	
Name	
ID number of the patient	
Age	
Sex	
IP No.	
Address and phone number	
Occupation	
Date of admission	
Date of follow up	

I. History of Present illness:

II. Past History

Personal history

Diet	
Appetite	
Sleep	
Bowel and Bladder habits	
Alcohol consumption	

COMORBIDITIES

Hypertension	
Coronary artery disease	
Hypothyroidism	
COPD	
Others	

General Physical Examination

General Physical Examination	
PALLOR	
ICTERUS	
CYANOSIS	
CLUBBING	
LYMPHADENOPATHY	
OEDEMA	
JVP	

Vital Signs	
Pulse rate(beats per minute)	
Blood pressure(millimeter of mercury)	
Temperature (deg. Fahrenheit)	
Respiratory rate (cycles per min)	

SYSTEMIC EXAMINATION

1.Cardiovascular system	
2. Respiratory System	

3.Abdomen examination	
4.CNS examination	

Diagnosis:

Biochemical investigations:

PT		
APTT		
Random blood sugar(mg/dl)		
Blood urea(mg/dl)		
Serum creatinine(mg/dl)		
Sodium		
Potassium		
Serum Bilirubin(mg/dl)		
SGOT		
SGPT		
INR		
Serum Albumin(g/dl)		

USG ABDOMEN

Liver

2D ECHO

Patient Name: Age: Sex:

UHID: IP: Date:

IVS(d)		IVS(s)		LVID(d)		LVID(s)	
LVpwd		LVpws		Aor		LA	

Diastolic function

E :

A :

E/A :

DT :

IVRT:

E^1 :

E/E^1 :

Vp (colour m mode):

LV Relaxation:

LV Compliance:

LA Pressure:

LV Blood filling:

LV Volume Index:

Pulmonary Venous flow(pw Doppler):

OTHER PARAMETERS

IAS:	IVS:
Tricuspid Valve:	Right Atrium:
Pulmonary Value:	Right Ventricle:
Mitral Value:	Left Atrium:
Aortic Value:	Left Ventricle:
Pulmonary Artery:	Aorta:
Pericardium:	Aortic Arch:
IVC:	RWMA:

IMPRESSION:

DR BASAVARAJ M,

MBBS,MD,DM(CARDIOLOGY).

	Score
Serum albumin	
Serum bilirubin	
Ascites	
Hepatic Encephalopathy	
INR	

CHILD PUGH SCORE:

MASTER CHART

KEYS TO MASTER CHART

E : Early diastolic filling velocity measured in meters/second.

A : Late diastolic filling velocity measured in meters/second.

E^1 : Early diastolic mitral annular velocity measured in meters/second.

DT: Deceleration Time in milli seconds.

IVRT: Isovolumetric Relaxation Time in milli seconds.

LVDD: Left Ventricular Diastolic Dysfunction.

TR : Tricuspid Regurgitation.

PAH: Pulmonary Arterial Hypertension.

RVD: Right Ventricular Dysfunction.

PE : Pericardial Effusion.

RLVEF: Reduced Left Ventricular Ejection Fraction.

MR : Mitral Regurgitation.

DCM: Dilated Cardiomyopathy.

MASTER CHART

					Diastolic function									Bio Chemical investigations												Score					Child PUGH score								
Sl.No	Name	IP/OP	Sex	Age	E	A	E/A	E^1	E/E^1	DT	IVRT	LVDD	Grade	PT	APTT	RBS	Urea	Sr. Cr	Sodium	Potassium	Sr. Bil	SGOT	SGPT	INR	Sr. Alb	Sr Alb	Sr. Bil	Ascites	Heptic Enc	INR		TR	PAH	RVD	PE	RLVEF	MR	DCM	ETIOLOGY
1	Narsappa	20220112461	male	42	0.5	1	0.5	0.068	7.3	245	105	yes	1	19	65	123	78	2.5	125	3.5	10.5	354	213	2	3	2	3	3	2	2	12	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
2	Hanumantha	20220199628	male	48	0.5	1.2	0.4	0.067	7.4	250	98	yes	1	20	67	135	88	3.5	129	3.5	9.8	215	105	1.9	3.2	2	3	2	2	2	11	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
3	Shivappa	20220198231	male	54	1.2	0.9	1.3	0.05	24	180	75	yes	2	21	65	98	98	4.5	126	3.3	15.6	334	214	3.2	2.5	3	3	3	3	3	15	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
4	Narasimhalu	20220197428	male	58	0.5	1	0.5	0.068	7.3	245	100	yes	1	16	60	119	59	1.9	135	3.4	9.8	114	85	2	2.9	2	3	3	2	2	12	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
5	Adeppa	20220199216	male	55	1.2	0.9	1.3	0.19	6.3	180	60	no	n	14	39	143	59	1.9	125	3.5	6.8	115	75	1.5	3.6	2	3	2	2	1	10	NO	NO	NO	NO	NO	NO	NO	HEP B
6	Shamshuddin	2023066267	male	63	1.3	0.9	1.4	0.06	21.6	180	75	yes	2	25	70	198	98	6.7	120	3.5	15.5	335	224	3.5	2	3	3	3	3	3	15	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
7	Bherappa	2023066215	male	55	0.5	1.2	0.4	0.067	7.4	245	100	yes	1	13	59	114	45	1.9	129	3.5	9.5	225	118	1.7	3	2	3	2	2	2	11	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
8	Shankarappa	2023162215	male	45	1.1	0.9	1.2	0.2	5.5	170	65	no	n	14	39	98	68	2.5	130	3.5	4.5	115	94	1.4	3.4	2	3	2	1	1	9	NO	NO	NO	YES	NO	NO	NO	WILSON DISEASE
9	Veeresh	2023161215	male	50	0.5	1.3	0.4	0.065	7.6	250	100	yes	1	19	69	111	67	2.5	131	2.9	10.9	125	75	2.1	3.1	2	3	2	3	2	12	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
10	Gundappa	202361154	male	35	1.3	0.9	1.4	0.2	6.5	180	60	no	n	14	38	112	72	1.8	119	2.5	5.6	114	74	1.6	3.1	2	3	2	2	1	10	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
11	Virupaksha	202361155	male	40	0.5	1.4	0.4	0.067	7.5	245	115	yes	1	14	59	121	46	1.9	129	2.4	3.5	104	94	1.4	3.6	1	3	2	1	1	8	NO	NO	NO	NO	NO	YES	NO	ALCOHOL
12	Devendrappa	202362255	male	45	1.3	0.9	1.4	0.2	6.5	185	62	no	n	13	39	122	34	1.3	129	3.5	2.5	145	94	1.2	3.6	1	2	1	1	1	6	YES	YES	YES	YES	NO	NO	NO	ALCOHOL
13	Gokulappa	202363315	male	40	0.8	0.6	1.3	0.04	20	185	75	yes	2	23	72	98	96	4.5	129	2.5	15.6	412	335	5.6	2.1	3	3	3	3	3	15	NO	NO	NO	NO	NO	NO	YES	ALCOHOL
14	Shamshuddin	202367552	male	65	0.8	0.6	1.3	0.2	4	175	65	no	n	14	59	104	75	1.8	118	2.5	10.6	225	119	1.9	3.1	2	3	3	2	2	12	NO	NO	NO	NO	NO	NO	NO	NASH
15	Mohinuddin	202368552	male	75	0.5	1.4	0.4	0.068	7.4	245	100	yes	1	13	38	139	34	1.4	129	3.5	3.5	75	24	1.4	3.6	1	3	2	1	1	8	YES	YES	YES	NO	YES	NO	NO	ALCOHOL
16	Shareefsab	202315057	male	33	0.5	1.3	0.4	0.065	7.7	245	110	yes	1	14	35	119	59	2.5	135	4.5	10.6	335	215	1.4	2.1	3	3	3	2	1	12	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
17	Ningappa	202378832	male	40	0.5	1	0.5	0.068	7.3	255	95	yes	1	12	59	104	58	1.9	120	2.5	2.9	115	75	1.4	3.1	2	2	2	1	1	8	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
18	Nasir	202381440	male	45	0.9	0.6	1.5	0.2	4.5	180	60	no	n	14	39	106	25	1.1	135	4.5	1.8	75	28	1.4	3.6	1	1	2	1	1	6	YES	YES	YES	NO	YES	NO	NO	ALCOHOL
19	Pratap	202378870	male	38	0.5	1.2	0.4	0.067	7.4	255	100	yes	1	14	39	111	58	1.9	135	2.8	9.8	119	58	1.4	3.1	2	3	2	1	1	9	NO	NO	NO	YES	NO	NO	NO	ALCOHOL
20	Kiran kumar	202383275	male	40	0.4	1.2	0.3	0.06	6.7	260	100	yes	1	13	53	111	45	1.4	129	3.5	15.5	78	30	2.3	3.1	2	3	3	2	3	13	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
21	Ramavva	202315934	female	60	0.4	1.2	0.3	0.065	6.2	242	110	yes	1	15	59	123	58	2.1	118	2.5	9.8	57	38	2	3.5	2	3	3	2	2	12	YES	YES	YES	NO	NO	NO	NO	NASH
22	Hanumantha	202319257	male	30	0.9	0.7	1.3	0.18	5	180	65	no	n	14	39	135	39	1.1	128	3.5	5.9	53	25	1.4	3.1	2	3	2	1	1	9	NO	NO	NO	NO	NO	YES	NO	ALCOHOL
23	Tipanna	202319431	male	45	1.4	1	1.4	0.05	28	185	80	yes	2	25	45	108	89	4.5	137	2.4	25.7	202	99	2.05	2.9	3	3	3	3	2	14	NO	NO	NO	NO	NO	NO	YES	ALCOHOL
24	Jagadish	202319246	male	53	0.9	0.7	1.3	0.17	5.2	185	62	no	n	14	59	104	98	5.2	129	2.5	15.5	152	72	1.6	3.1	2	3	3	2	1	11	NO	NO	NO	NO	NO	YES	NO	ALCOHOL
25	Laxmappa	202319268	male	60	0.4	1.1	0.4	0.06	6.6	255	100	yes	1	14	59	138	88	3.5	125	3.5	9.8	115	25	2.1	3.1	2	3	3	2	2	12	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
26	Yallapa	202318389	male	50	0.5	1.2	0.4	0.068	7.4	250	90	yes	1	16	58	125	86	4.5	129	2.4	16.5	115	85	1.9	3.1	2	3	3	2	3	13	YES	YES	YES	NO	NO	NO	NO	ALCOHOL
27	Abdul sab	202318757	male	55	0.8	0.6	1.4	0.18	4.4	180	65	no	n	14	52	104	39	1.1	132	3.5	4.1	78	52	1.4	2.9	2	3	2	1	1	9	YES	YES	YES	NO	NO	NO	NO	HEP C
28	Durgappa	202319265	male	47	1.8	0.8	2.25	0.04	45	120	50	yes	3	35	85	106	120	8.8	120	2.5	29.9	399	355	5.9	2	3	3	3	3	3	15	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
29	Veeresh	202319093	male	38	0.5	1.1	0.4	0.065	7.7	245	110	yes	1	15	59	118	98	3.5	126	3.5	10.5	112	98	1.8	3.1	2	3	3	3	2	13	NO	NO	NO	YES	NO	NO	NO	ALCOHOL
30	Pratap	202315124	male	38	0.5	1.2	0.4	0.067	7.5	252	100	yes	1	15	35	124	98	3.5	125	2.5	105	75	28	1.9	2.9	3	3	3	2	2	13	YES	YES	YES	NO	NO	NO	NO	ALCOHOL
31	Shantappa	202334473	male	55	1.2	0.9	1.33	0.21	5.7	170	62	no	n	14	36	94	35	1.2	142	3.5	0.8	19	25	1.2	3.2	1	2	1	1	1	6	YES	YES	YES	NO	NO	NO	NO	HEP C
32	Saleem	202335557	male	35	1.1	0.8	1.37	0.19	5.7	180	68	no	n	14	34	63	33	1.2	139	3.2	1.5	45	25	1.9	3.1	2	1	1	1	1	6	NO	NO	NO	YES	YES	NO	NO	ALCOHOL
33	Hampayya	202336103	male	55	0.5	1.1	0.4	0.065	7.7	255	100	yes	1	14	37	98	43	1.5	129	3	2.8	110	78	2	3.1	2	2	2	1	2	9	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
34	Urukundappa	202336158	male	40	1	0.7	1.42	0.16	6.25	170	65	no	n	13	42	97	33	1.2	133	3.3	0.9	22	15	1.3	3.7	1	1	1	1	1	5	NO	NO	NO	NO	YES	NO	NO	ALCOHOL
35	Srinivas	202336058	male	48	0.4	1.1	0.36	0.06	6.6	250	100	yes	1	14	39	92	39	1.5	139	2.4	4.3	113	114	1.3	4.2	3	1	2	1	1	8	YES	YES	YES	NO	NO	NO	NO	ALCOHOL
36	Basavaraj	202336162	male	35	0.5	1.2	0.4	0.065	7.7	250	110	yes	1	15	44	92	42	1.5	129	3.3	3.9	115	104	1.3	3.1	2	3	2	1	1	9	NO	NO	NO	NO	NO	NO	NO	WILSON DISEASE
37	Bajarappa	202335760	male	64	1.2	1	1.2	0.06	20	180	80	yes	2	20	45	63	43	1.2	129	2.9	14.7	237	211	1.3	3.5	1	3	2	2	1	9	YES	YES	YES	NO	NO	NO	NO	ALCOHOL
38	Mehaboob	202336270	male	50	0.5	1.3	0.4	0.067	7.5	242	110	yes	1	15	43	63	39	12	129	3.2	4	113	101	2.06	2.5	3	3	3	2	2	13	YES	YES	YES	YES	YES	NO	NO	ALCOHOL
39	Anjappa	202334933	male	37	1	0.9	1.1	0.06	16	170	80	yes	2	20	55	73	78	1.7	120	2.5	21.8	215	115	2.1	2.5	3	3	2	3	2	13	NO	NO	NO	NO	NO	NO	NO	HEP B
40	Gopal	202332118	male	38	0.5	1.2	0.4	0.065	7.7	245	110	yes	1	13	39	78	29	0.8	136	2.3	1.6	75	23	1.9	3.5	1	1	2	1	1	6	YES	YES	YES	NO	NO	NO	NO	ALCOHOL
41	Gynappa	202334489	male	59	0.5	1.1	0.4	0.065	7.7	240	110	yes	1	14	34	91	31	0.8	131	3.5	0.8	45	40	1.3	3	2	1	1	1	1	6	NO	NO	NO	NO	NO	NO	NO	HEP C
42	Narsappa	202335261	male	25	1.2	1	1.2	0.18	6.6	170	65	no	n	15	34	85	29	0.7	131	3.5	0.8	110	75	1.9	3.1	2	1	2	1	2	8	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
43	Revayya	202332091	male	47	0.4	1.1	0.4	0.06	6.6	246	120	yes	1	14	55	72	32	1.1	132	3.6	2.9	143	101	1.4	3.1	2	2	2	2	1	9	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
44	Shivaraj	202334940	male	36	1	0.8	1.25	0.14	7.14	180	60	no	n	14	55	78	39	1.1	134	3.6	2.9	134	105	1.4	3.1	2	2	2	1	1	8	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
45	Thimappa	202335639	male	31	0.5	1.3	0.4	0.067	7.5	240	100	yes	1	18	53	98	78	1.9	139	2.5	2.3	137	98	1.1	3.1	2	2	2	1	1	8	YES	YES	YES	NO	NO	NO	NO	ALCOHOL
46	B Ramesh	202336720	male	56	0.4	1.2	0.3	0.065	6.2	245	110	yes	1	15	43	93	34	1.1	143	3.1	2.2	353	152	0.8	3.3	2	2	3	2	1	10	NO	NO	NO	YES	YES	NO	NO	ALCOHOL
47	Syed sabjali	202336964	male	58	0.5	1.3	0.4	0.068	7.4	250	130	yes	1	14	53	92	32	0.9	139	2.9	3.6	335	321	1.18	3.1	2	3	3	2	1	11	NO	NO	NO	NO	NO	YES	NO	ALCOHOL
48	Ramesh	202338614	male	55	1.2	1.1	1.09	0.18	6.6	180	68	no	n	14	45	93	28	1.1	139	2.9	1.2	133	143	1.4	2.1	3	1	2	1	1	8	NO	NO	NO	NO	YES	NO	NO	ALCOHOL
49	Basavaraj	202338694	male	32	0.4	1.3	0.3	0.06	6.6	245	110	yes	1	20	54	175	95	1.7	139	7.2	7.9	113	125	1.3	3.1	2	3	2	2	1	10	NO	NO	NO	NO	YES	NO	NO	ALCOHOL
50	Srinivas	2023385585	male	48	0.5	1.2	0.4	0.068	7.4	250	120	yes	1	15	35	185	39	1.1	142	3.9	5.5	2.5	85	1.4	3.2	2	3	2	2	1	10	NO	NO	NO	NO	NO	NO	NO	HEP B

MASTER CHART

51	Basavaraj	202338759	male	28	1.1	0.9	1.22	0.05	22	155	85	yes	2	13	55	75	28	1.2	138	4.8	23.2	68	58	2.2	2.8	2	3	3	3	2	13	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
52	Lal sab	202338589	male	50	0.5	1.6	0.3	0.065	7.7	242	110	yes	1	13	34	185	38	1.1	127	3.9	3.5	58	52	1.97	3.2	2	3	2	2	2	11	NO	NO	NO	NO	NO	YES	NO	ALCOHOL
53	Praveen	202338981	male	31	1.1	0.9	1.22	0.04	27.5	190	75	yes	2	15	34	75	35	0.9	130	2.4	15.3	77	28	1.8	3.2	2	3	2	2	2	11	NO	NO	NO	NO	YES	NO	NO	HEP C
54	Hanumantha	202339087	male	53	0.5	1.2	0.4	0.065	7.7	245	110	yes	1	14	31	153	38	1.1	141	5.2	4.4	36	32	1.5	3.3	2	2	2	1	1	8	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
55	Urukundappa	202337625	male	29	1.1	0.8	1.3	0.06	18	220	80	yes	2	10	34	138	29	1.1	139	2.9	15.5	135	101	3.2	2.1	3	3	3	2	3	14	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
56	Narasimhalu	202338090	male	35	0.5	1.3	0.4	0.068	7.4	245	110	yes	1	10	52	125	38	1.1	132	4.2	4.4	32	31	1.3	3.2	2	3	2	1	1	9	NO	NO	NO	YES	NO	NO	NO	HEP C
57	Pampanna	202337817	male	25	0.4	1.3	0.3	0.065	6.2	246	110	yes	1	14	54	98	38	1.1	129	3.5	6.5	139	23	2.5	3	2	3	3	2	3	13	YES	YES	YES	NO	NO	NO	NO	ALCOHOL
58	Dhanajay	202339018	male	43	0.5	1.2	0.4	0.065	7.7	242	120	yes	1	14	55	85	29	1.1	129	3.5	6.4	93	74	2.08	2.1	3	3	3	2	3	14	NO	NO	NO	NO	NO	NO	NO	ALCOHOL
59	Ramesh	20241723	male	42	0.5	1.3	0.4	0.067	7.5	245	120	yes	1	14	54	153	37	1.1	135	5	3.1	97	36	1.66	3.1	2	3	2	2	1	10	YES	YES	YES	NO	NO	NO	NO	ALCOHOL
60	Nagaraj	20241777	male	40	0.5	1.2	0.4	0.068	7.4	245	100	yes	1	13	24	74	34	1.1	139	2.9	1	33	21	1.54	2	3	1	3	2	1	10	NO	NO	NO	NO	NO	NO	NO	HEP B

ANNEXURES

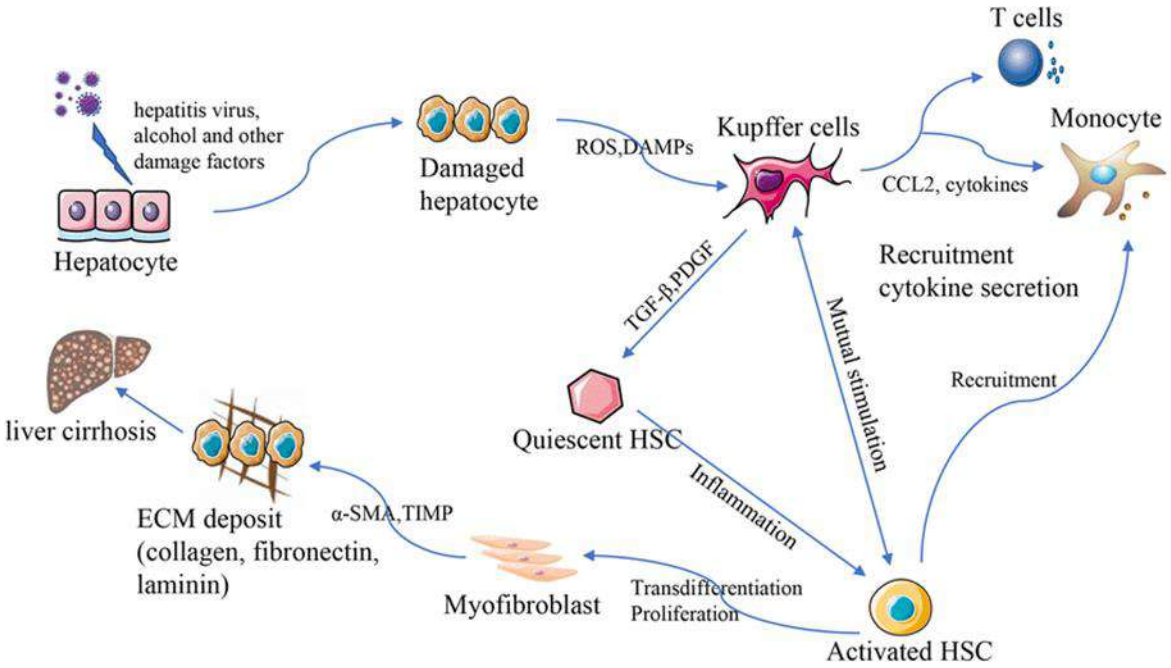

Figure 1: Pathogenesis of liver cirrhosis(From Harrison 21st edition textbook)

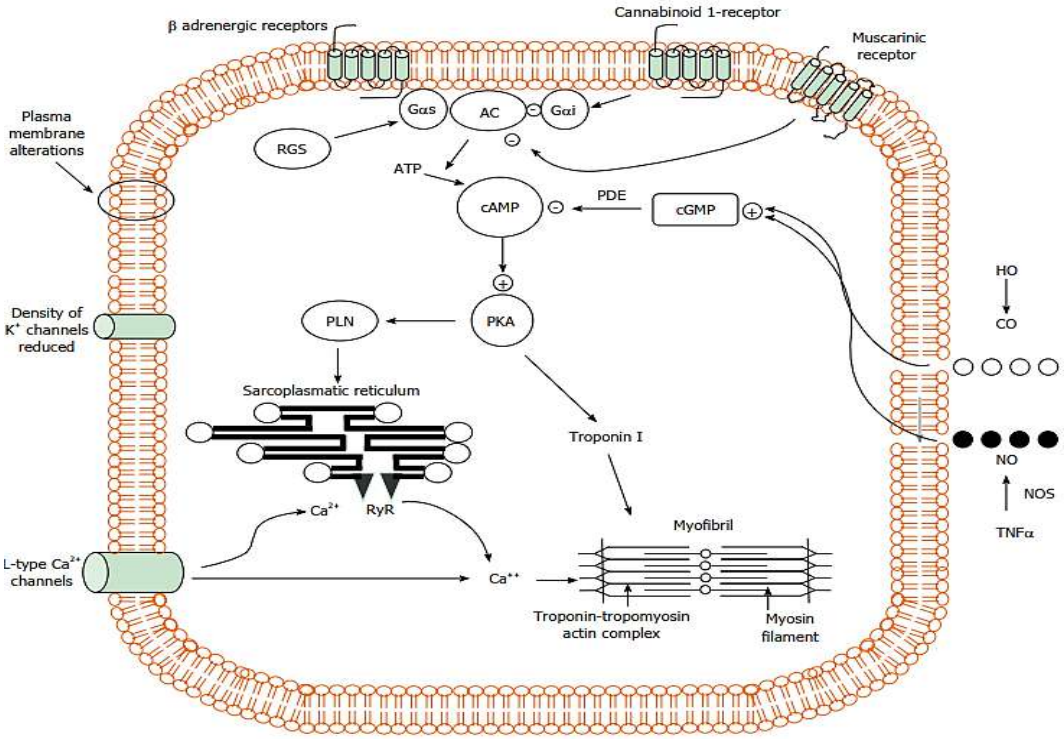

Figure 2: Pathogenic mechanisms of cardiomyocyte contraction in cirrhosis.

(From Harrison 21st edition textbook)

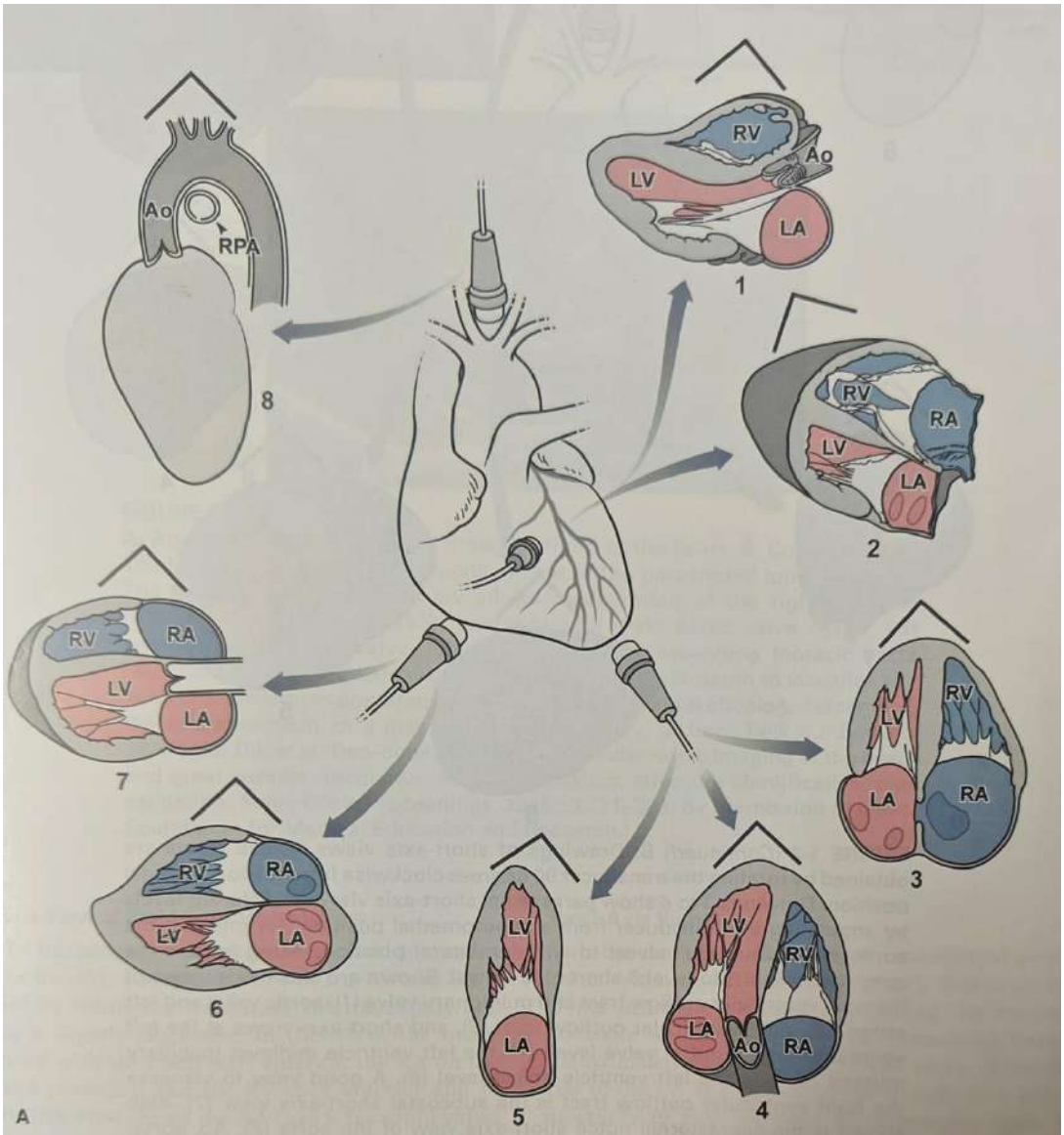

FIGURE 1-2 **A**: Drawings of the longitudinal views from the four standard transthoracic transducer positions. Shown are the parasternal long-axis view (*1*), parasternal right ventricular inflow view (*2*), apical four-chamber view (*3*), apical five-chamber view (*4*), apical two-chamber view (*5*), subcostal four-chamber view (*6*), subcostal long-axis (five-chamber) view (*7*), and suprasternal notch view (*8*).

Figure 3: Transducer positions and cardiac view(Figure from south asian 2decho manual)

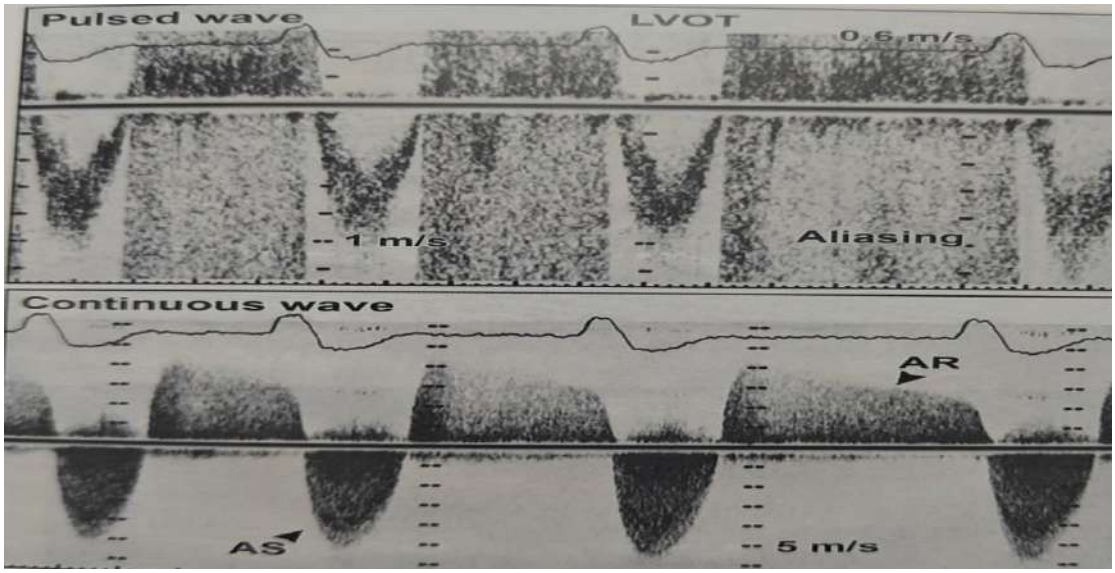

Figure 4: Pulsed wave and continuous wave doppler(Figure from south asian 2decho manual)

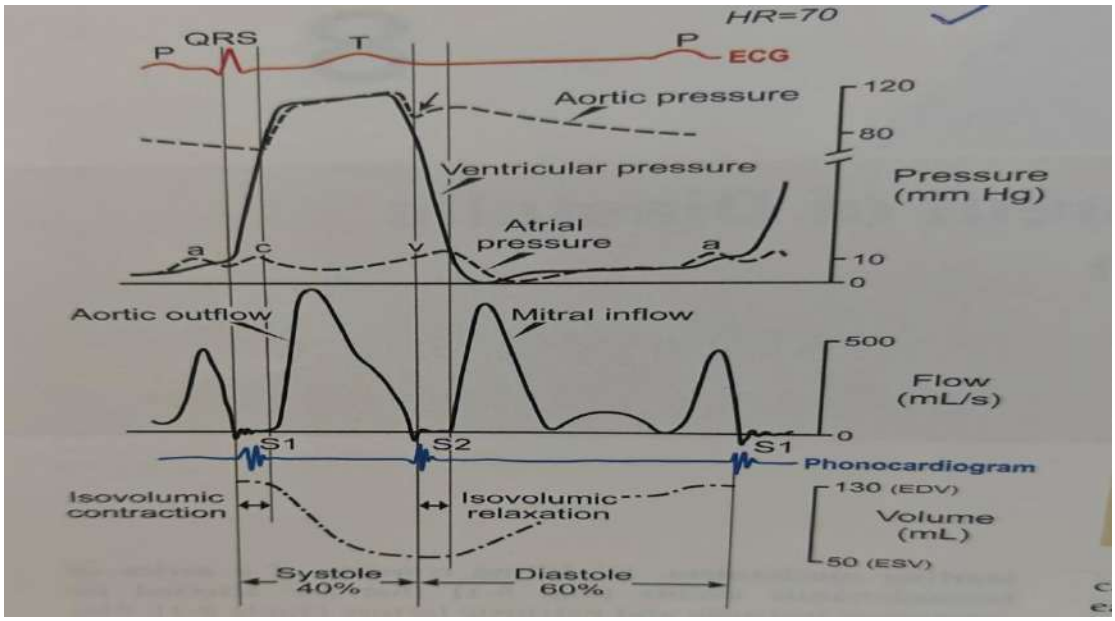

Figure 5: Diagram of intracardiac pressures(top), aortic outflow and mitral inflow(middle), and volumetric changes in left ventricle(bottom) (Figure from south asian 2decho manual).

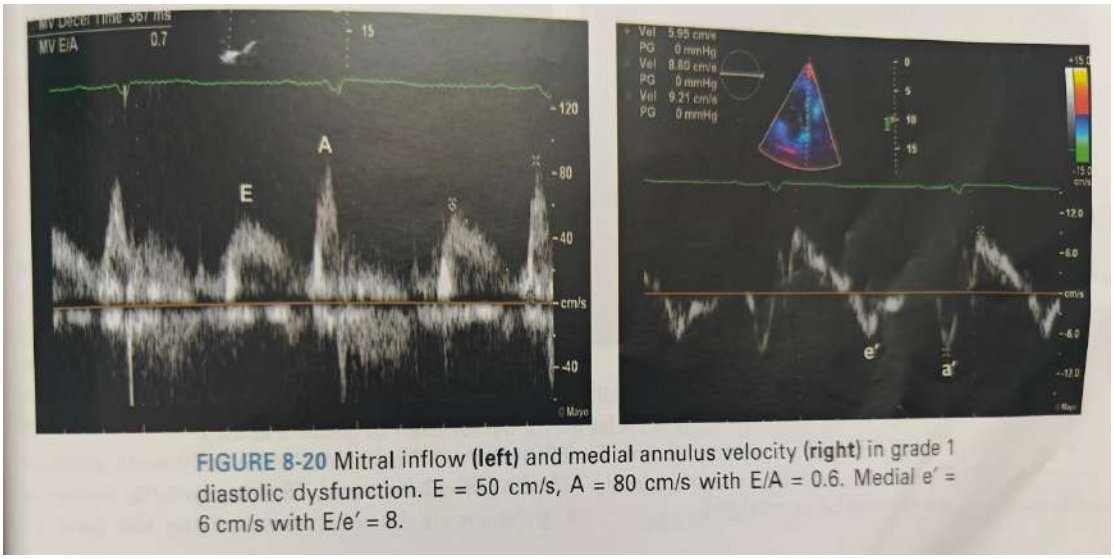

FIGURE 8-20 Mitral inflow **(left)** and medial annulus velocity **(right)** in grade 1 diastolic dysfunction. E = 50 cm/s, A = 80 cm/s with E/A = 0.6. Medial e′ = 6 cm/s with E/e′ = 8.

Figure 6: DOPPLER FLOW VELOCITIES(Figure from south asian 2decho manual)

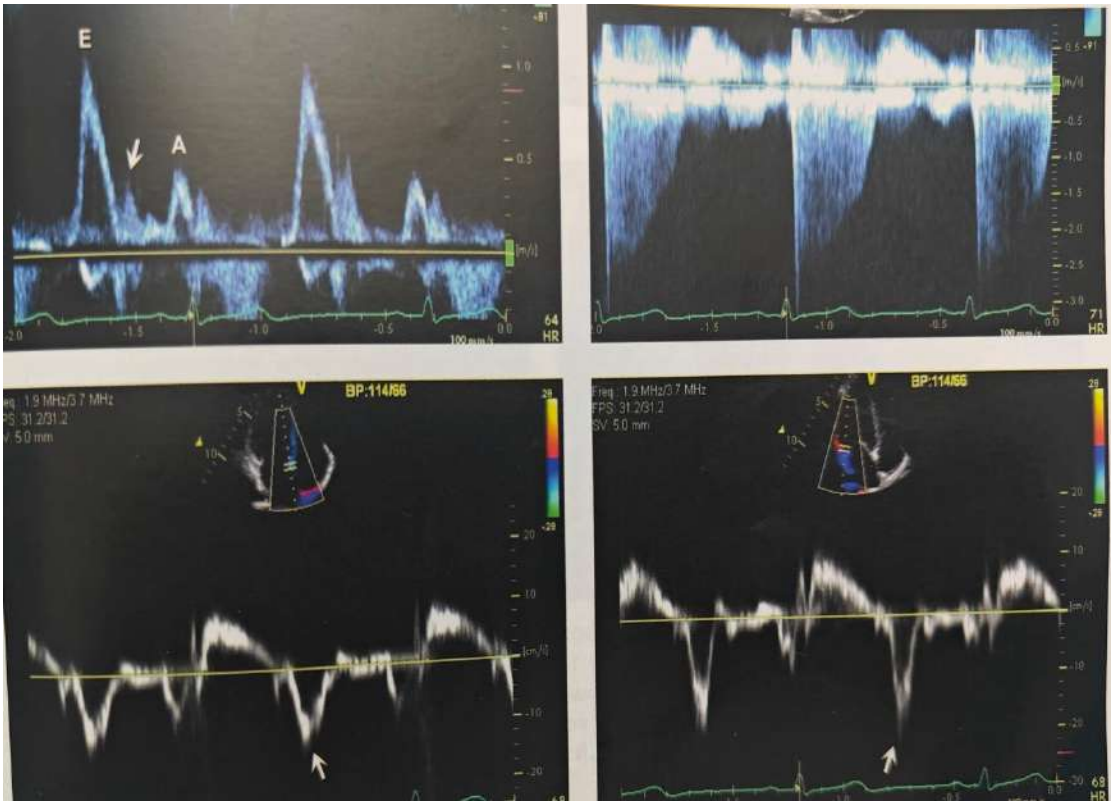

Figure 7: Normal Diastolic Filling Pattern(Figure from south asian 2decho manual)

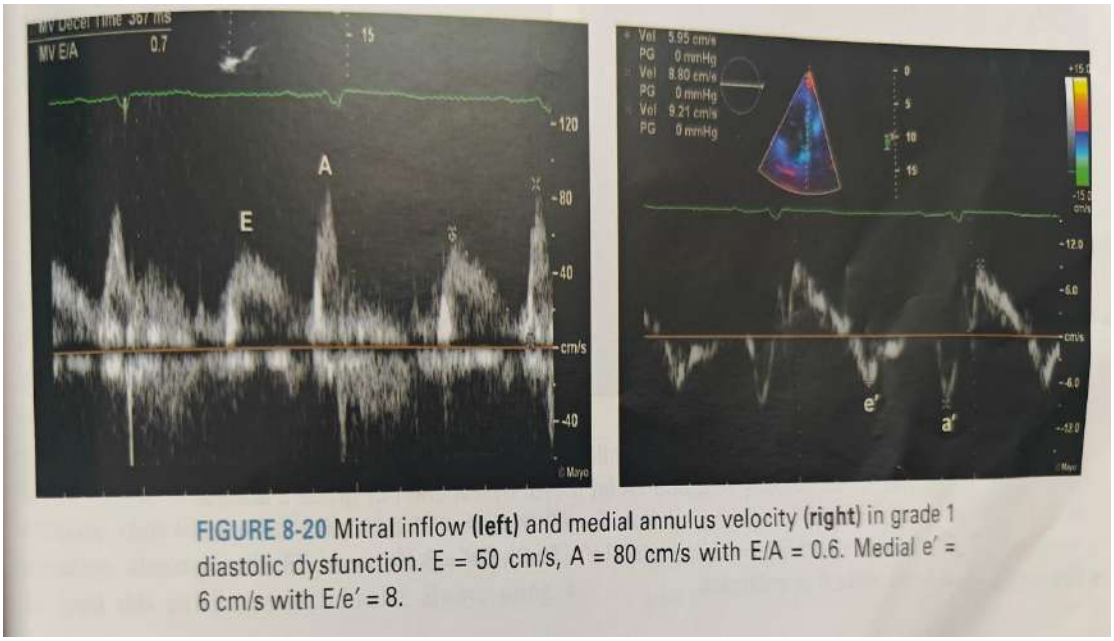

FIGURE 8-20 Mitral inflow **(left)** and medial annulus velocity **(right)** in grade 1 diastolic dysfunction. E = 50 cm/s, A = 80 cm/s with E/A = 0.6. Medial e′ = 6 cm/s with E/e′ = 8.

Figure 8: Grade 1 Diastolic Dysfunction(Figure from south asian 2decho manual)

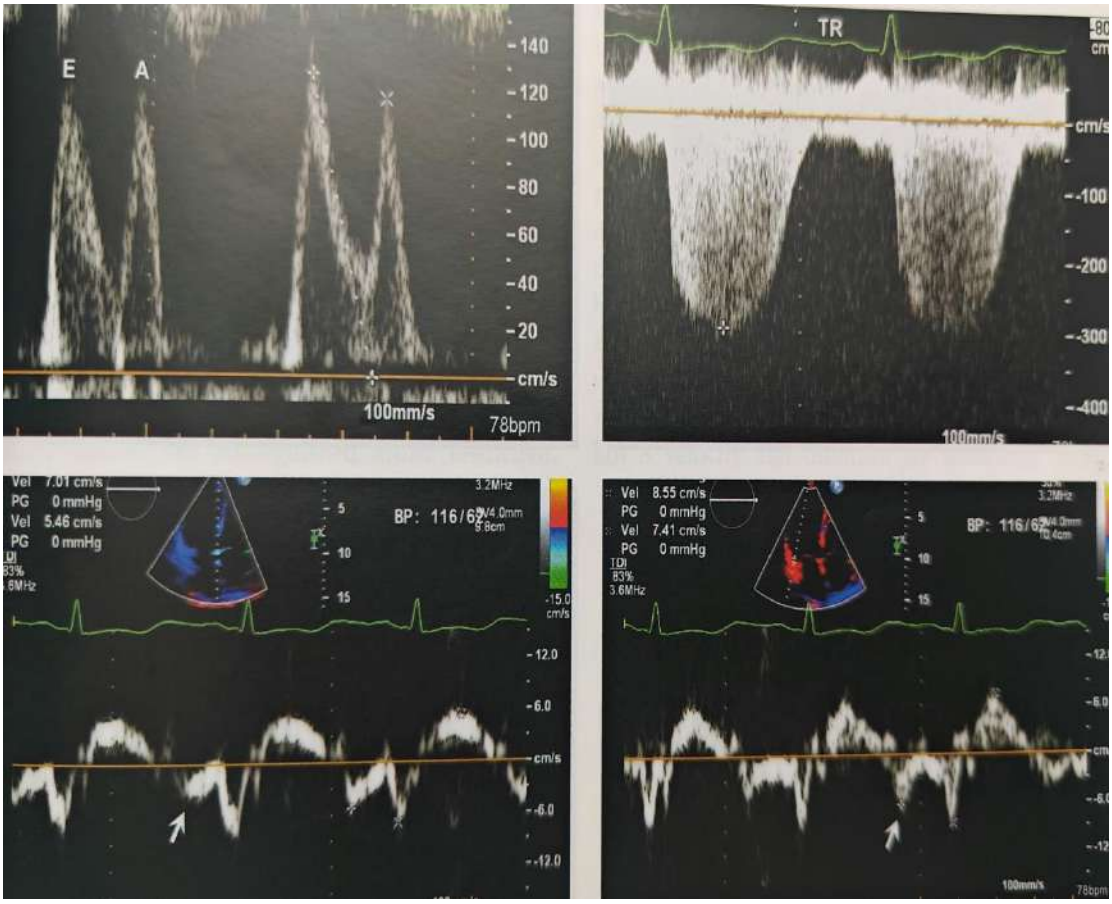

Figure 9: Grade 2 Diastolic Dysfunction (Figure from south asian 2decho manual)

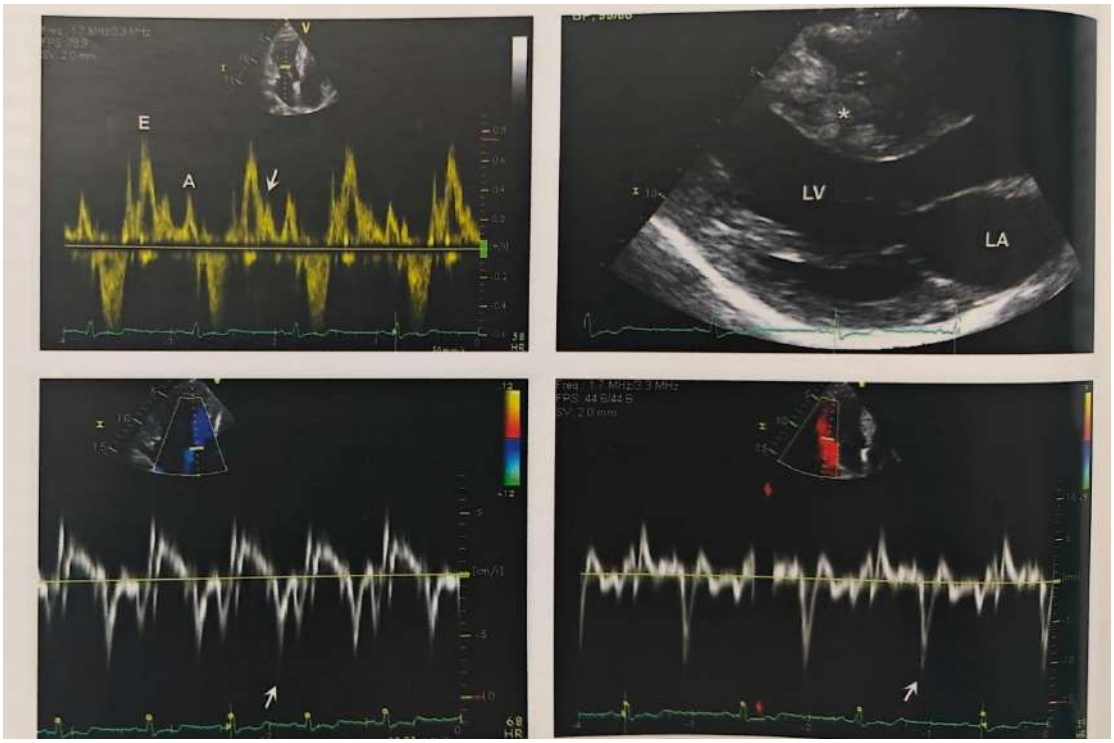

Figure 10: Grade 3 diastolic dysfunction(Figure from south asian 2decho manual)