TO STUDY AST/ALT RATIO AS AN INDICATOR OF FUNCTIONAL SEVERITY IN CHRONIC HEART FAILURE WITH REDUCED LEFT VENTRICULAR EJECTION FRACTION AT DR. PRABHAKAR KORE HOSPITAL, MRC, AND BELAGAVI, ONE YEAR PROSPECTIVE CROSS-SECTIONAL STUDY"

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ABSTRACT:

Introduction: Chronic Heart Failure is a progressive condition characterised by gradually failure of heart in pumping sufficient blood. There are various established cardiovascular biomarkers (NT-pro BNP & Troponin) and emerging biomarkers (miRNAs, mimecan, orexin). The AST/ALT ratio can be new feasible cardiovascular biomarker as AST is released from the myocardium and the liver while release of ALT occurs only from the liver so this ratio can be used in myocardial injury.

Materials and methods: A one year prospective cross sectional study of 100 subjects of LVEF of ≤ 40% fulfilling the inclusion criteria conducted in KLE Dr.Prabhakar kore Hospital and MRC, Belagavi. Based on the AST/ALT ratio, the study participants were divided into 2 groups - AST/ALT ratio <1 (group 1) and AST/ALT ratio ≥1 (group 2). LVEF, Clinical findings and laboratory parameters were the outcome variables compared by Chi square test. Applicability of AST/ALT ratio to predict the severity of chronic heart failure with reduced EF is checked by Logistic regression and Receiver Operating Characteristic (ROC) curves.

Results: In present study of 100 subjects, there was a statistically significant difference between two groups with respect to ALT, AST/ALT ratio, and ALP. There was a significant correlation between the APRI index and FIB-4 with AST/ALT ratio. Diagnostic analysis of AST/ALT ratio to predict the severity of chronic heart failure with reduced EF, the area under the curve (AUC) was 0.547 (P-value = 0.5654) with a 95% confidence interval of 0.299 – 0.795 with an optimal cut-off value of 0.6, sensitivity of 96.70%, and specificity of 33.33%.

Conclusion: The AST/ALT ratio is increased in patients with chronic heart failure patients with reduced left ventricular ejection fraction. It is a simple predictor of left ventricular dysfunction in patients with heart failure with reduced ejection fraction.

Keywords: Chronic Heart Failure, Reduced Left Ventricular Ejection Fraction, Aspartate transaminase, Alanine aminotransferase, AST/ALT ratio, Predictive validity, Functional severity

ABBREVIATIONS

Glossary	Abbreviations
ACC	American College of Cardiology
ACM	All-cause mortality
AHA	American heart association
ALT	Alanine aminotransferase
APRI	AST to platelet ratio index
ARIC	Atherosclerosis Risk in Communities
AST	Aspartate aminotransferase
BNP	Brain natriuretic peptide
CAD	Coronary artery disease
CHF	Chronic heart failure
CI	Confidence interval
CVD	Cardiovascular system diseases
EF	Ejection fraction
ELISA	Enzyme-linked immunosorbent assay
ESC	European Society of Cardiology
FIB-4	Fibrosis-4 score
HF	Heart failure
HFimpEF	HF with improved ejection fraction
HFmrEF	HF with mildly reduced ejection fraction
HFrEF	HF with reduced ejection fraction
HFSA	Heart Failure Society of America
HRS	Hazard ratios

JHFS	Japanese heart failure society
LV	Left ventricle
LVEDP	Left ventricle end-diastolic pressure
LVEF	LV ejection fraction
NAFLD	Non-alcoholic fatty liver disease
NT-proBNP	N-terminal pro-brain natriuretic peptide
NYHA	New york heart association
PCI	Percutaneous coronary intervention
PCWP	Pulmonary capillary wedge pressure
RAAS	Renin-angiotensin-aldosterone
RHD	Rheumatic heart disease
SNS	Sympathetic nervous systems
US	United states

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INTRODUCTION

Heart failure occurs when the heart cannot pump enough blood and oxygen for supporting other organs in the body. Heart failure (HF) can be described as a compound clinical syndrome that is characterized by functional impairment or structural impairment of ventricles which in turn leads to symptomatic dysfunction of the left ventricle (LV).[1] It can result from disorders of the pericardium, myocardium, endocardium, heart valves, great vessels, or some metabolic abnormalities.[2]

An estimated 64.3 million people are living with heart failure worldwide.[3] The prevalence of heart failure is around 1% to 2% of the general adult population in developed countries.[4] It has also been estimated that more than half of all heart failure patients have a preserved LVEF.[5,6] It has been estimated that heart failure prevalence in India ranges from 1.3 million to around 4.6 million. There is an increase in the prevalence of Chronic heart failure in spite of the advances made in the management of cardiovascular diseases over the past few decades.

There are several well-known cardiovascular biomarkers like "N-terminal pro-brain natriuretic peptide (NT-proBNP) and troponin" and emerging biomarkers like "miRNAs, mimecan, and orexin". But they are not routinely used, stressing the need for novel biomarkers that are feasible, reproducible, and accurate for screening the functional severity in subjects with HFrEF.

There have been multiple theories regarding the cardiovascular system and liver that have explored the results of abnormal liver function tests in cardiovascular diseases like HF[7], ischemic heart disease[8], and atherosclerosis.[9]

The development of hepatic fibrosis is associated with the dysfunction of multiple systems and is not considered currently an isolated hepatic disease. For monitoring and evaluation of hepatic fibrosis, there are non-invasive have been

developed.[10] like("Aspartate aminotransferase (AST)/Alanine aminotransferase (ALT) ratio[11], AST to platelet ratio index (APRI)[12], Fibrosis-4 score (FIB-4)[13], and non-alcoholic fatty liver disease (NAFLD) fibrosis score").[14] AST/ALT ratio is also known as the De Ritis ratio. It was initially proposed for studying the etiology of hepatitis and has been used for differentiating the etiology of hepatic diseases.[15] AST/ALT ratio has also become an effective biomarker for diseases of other systems like cardiovascular system diseases (CVD)[16], cancers[17], and T2DM.[18]

NEED OF THE STUDY

There are very limited studies that have evaluated the role of liver transaminases, especially AST/ALT ratio in predicting cardiovascular diseases, that is also specifically in HFrEF. The release of AST occurs from the liver and myocardium while the release of ALT occurs only from the liver. There would be an increase in the AST/ALT ratio in case of injury to the myocardium.[19] The probability of occurrence of hepatic fibrosis is also higher with an increased AST/ALT ratio. It is also associated with the pathogenesis of CVD through various mechanisms, such as an increase in the number of inflammatory mediators in the plasma, resistance to insulin, metabolic syndrome, and oxidative stress.[20]

Ewid M et al[21] in their study observed that there is potential for increased AST/ALT ratio for predicting a functional status decline in subjects with HFrEF. But there are no studies to our knowledge in India, which have evaluated the predictive validity of the AST/ALT ratio in HFrEF. Hence the present study was carried out to evaluate the role of the AST/ALT ratio as a predictor of functional severity in subjects with chronic HF with reduced LVEF.

AIMS & OBJECTIVES

AIM:

To study “the role of AST/ALT ratio as an indicator of the functional severity in chronic heart failure with reduced left ventricular ejection fraction”.

REVIEW OF LITERATURE

I. HEART FAILURE:

Heart failure is one of the major causes of morbidity and mortality, globally.[3,22] It is a common and potentially fatal disease, if left untreated. It is the final common stage of many diseases of the heart.

Definition of Heart failure (HF):

Heart failure (HF) can be described as a compound clinical syndrome that is characterised by functional impairment or structural impairment of ventricles which in turn leads to symptomatic dysfunction of the left ventricle (LV).[23,24]

The **2016 ESC criteria[25]** define Heart Failure as "a clinical syndrome characterized by typical symptoms (e.g. breathlessness, ankle swelling and fatigue) that may be accompanied by signs (e.g. elevated jugular venous pressure, pulmonary crackles and peripheral oedema) caused by a structural and/or functional cardiac abnormality, resulting in a reduced cardiac output and/or elevated intracardiac pressures at rest or during stress".[25] It incorporates signs and symptoms with objective measures of cardiac dysfunction like echocardiography, natriuretic peptides or other cardiac imaging. But many patients with proven HFpEF have normal natriuretic peptide levels.[4]

According to ESC guidelines, there are 3 major phenotypes of HF based on LVEF and this is essential because of their differences in response to management:

1. Heart failure with reduced ejection fraction (HFrEF): EF less than or equal to 40%
2. Heart failure with preserved EF (HFpEF): EF is greater than or equal to 50%
3. Heart failure with mid-range EF (HFmrEF) - EF is 41% to 49%

Figure 1: Classification of Heart Failure (2016 ESC)[25]

Type of HF		HFrEF	HFmrEF	HFpEF
CRITERIA	1	Symptoms ± Signs[a]	Symptoms ± Signs[a]	Symptoms ± Signs[a]
	2	LVEF <40%	LVEF 40–49%	LVEF ≥50%
	3	–	1. Elevated levels of natriuretic peptides[b]; 2. At least one additional criterion: a. relevant structural heart disease (LVH and/or LAE), b. diastolic dysfunction (for details see Section 4.3.2).	1. Elevated levels of natriuretic peptides[b]; 2. At least one additional criterion: a. relevant structural heart disease (LVH and/or LAE), b. diastolic dysfunction (for details see Section 4.3.2).

BNP = B-type natriuretic peptide; HF = heart failure; HFmrEF = heart failure with mid-range ejection fraction; HFpEF = heart failure with preserved ejection fraction; HFrEF = heart failure with reduced ejection fraction; LAE = left atrial enlargement; LVEF = left ventricular ejection fraction; LVH = left ventricular hypertrophy; NT-proBNP = N-terminal pro-B type natriuretic peptide.

[a]Signs may not be present in the early stages of HF (especially in HFpEF) and in patients treated with diuretics.

[b]BNP>35 pg/ml and/or NT-proBNP>125 pg/mL.

"In response to the necessity for a consensus definition for HF, in 2020 a writing committee comprised of members of the *Heart Failure Society of America* (HFSA), the *Heart Failure Association of the European Society of Cardiology* (HFA/ESC), and the *Japanese Heart Failure Society* (JHFS) across 14 countries and 6 continents assembled a consensus document resulting in the new Universal Definition and Classification of HF.[26] The proposed universal definition emphasizes HF is a clinical syndrome with symptoms and/or signs caused by a structural and/or functional cardiac abnormality and corroborated by elevated natriuretic peptide levels and/or objective evidence of pulmonary or systemic congestion".

The American College of Cardiology/American Heart Association (ACC/AHA) has characterised HF based on symptoms as fundamental in progression of disease and severity of the disease.[27]

The four stages are defined as below (Table 1)

STAGE	CHARACTERISTICS
Stage A	At risk for HF
Stage B	Pre-HF
Stage C	Symptomatic HF
Stage D	Advanced HF

Then, based on LVEF, a revised classification was proposed:[26]

It classified HF as

"A. HF with reduced ejection fraction (HFrEF) – symptomatic HF with LVEF ≤40%

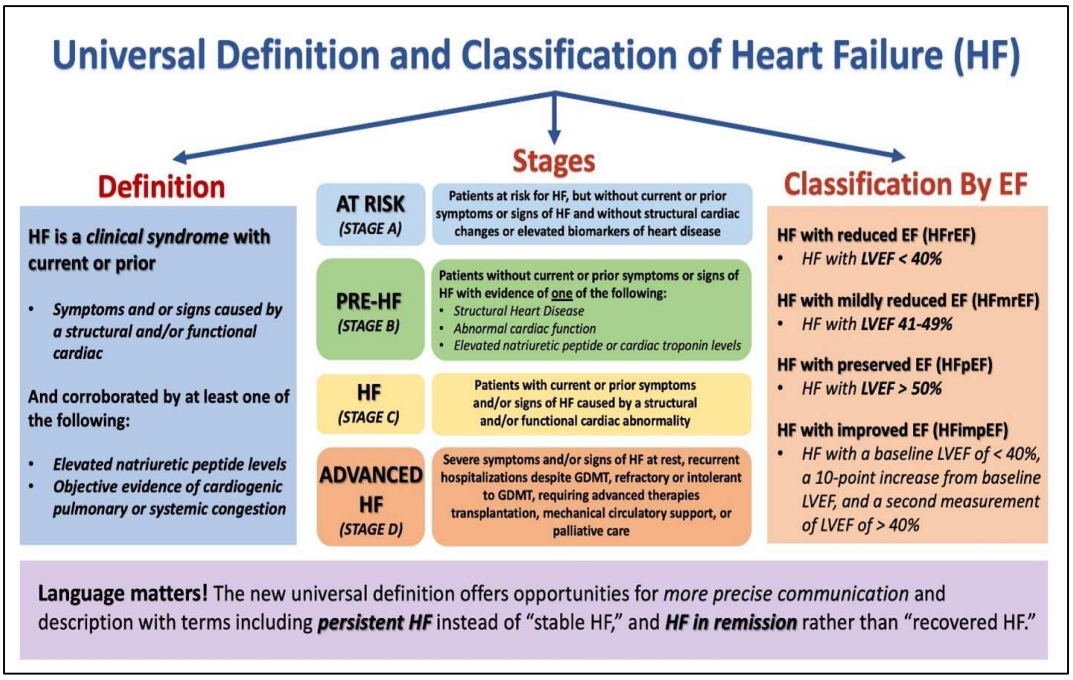
B. HF with mildly reduced ejection fraction (HFmrEF) – symptomatic HF with LVEF 41-49% (previously labeled as HF with mid-range ejection fraction)

C. HF with preserved ejection fraction (HFpEF) – symptomatic HF with LVEF ≥50%

D. HF with improved ejection fraction (HFimpEF) – a new classification which is distinctly defined as symptomatic HF with a baseline LVEF ≤40%, a ≥10-point increase from baseline LVEF, and a second measurement of LVEF >40%".

In subjects with HFrEF, there will be concomitant diastolic dysfunction but on the other hand, diastolic dysfunction can occur without systolic dysfunction.

Figure 2: Universal Definition and classification of Heart Failure:[26]



Courtesy: *Gibson GT, Blumer V, Mentz RJ, Lala A*

https://www.acc.org/latest-in-cardiology/articles/2021/07/12/12/31/universal-definition-and-classification-of-heart-failure

Epidemiology of Heart failure:

An estimated 64.3 million people are living with heart failure worldwide.[3] The prevalence of heart failure is around 1% to 2% of the general adult population in developed countries.[4] It has also been estimated that more than half of all heart failure patients have a preserved LVEF.[5,6] About **6.2 million** adults in the United States have heart failure.[1] In 2018, heart failure was mentioned on 379,800 death certificates (13.4%).[28] HFpEF is more common than HFrEF [median prevalence 4.9% and 3.3% respectively]. The prevalence of diastolic dysfunction is on the rise and currently higher than that of systolic dysfunction, which seems to have decreased in the 21st century".[29] This will amount for increase in prevalence by 46 %.[30] "The EPidemiologia da Insuficiencia Cardiacae Aprendizagem (Epidemiology of Heart Failure and Learning – EPICA) study" in Europe observed that the prevalence of HF increased with age

starting from 1.36 % in the age group of 25 to 49 years to 12.67 % in the age group of 70 to 79 years and 16.14 % in subjects aged more than 80 years.[31] It has been estimated that the prevalence of HF in china is 1.3 % amounting to around 4.2 million.[32] It has been estimated that around 1.3 to 4.6 million in India have HF, amounting to 0.12% to 0.44% of the population.[33] The annual incidence has been estimated to be ranging from 4,91,600 to around 1.8 million.[33] In India, the incidence of HF annually in CHD subjects is estimated to be ranging from 0.4% to 2.3%.[34,35]

Aetiology

There are several conditions that can lead to HF. The aetiology varies between developed and developing countries.[34] In rich countries, the most common causes are COPD and Ischemic heart disease. In poor countries, the common conditions leading to HF are "Hypertensive heart disease, rheumatic heart disease, cardiomyopathy, and myocarditis".[36] It has been reported that around two-thirds of all HF cases occur due to "attributable to ischemic heart disease, COPD, hypertensive heart disease, and rheumatic heart disease".

Figure 3: Aetiology of Heart Failure

Predominant clinical aetiology/situations for systolic and diastolic heart failure

Systolic heart failure	**Diastolic heart failure**
Coronary artery disease	Diabetes mellitus
Arterial hypertension	Arterial hypertension
Valvular heart disease (Volume load)	Valvular heart disease (pressure load)
Arrhythmia	Hypertrophic cardiomyopathy
Inflammatory diseases	Restrictive cardiomyopathy
Idiopathic cardiomyopathy	Constrictive pericarditis
Toxic cardiomyopathy (alcohol)	Amyloidosis (storage disease)

Pathophysiology of heart failure and heart failure with reduced LVEF:

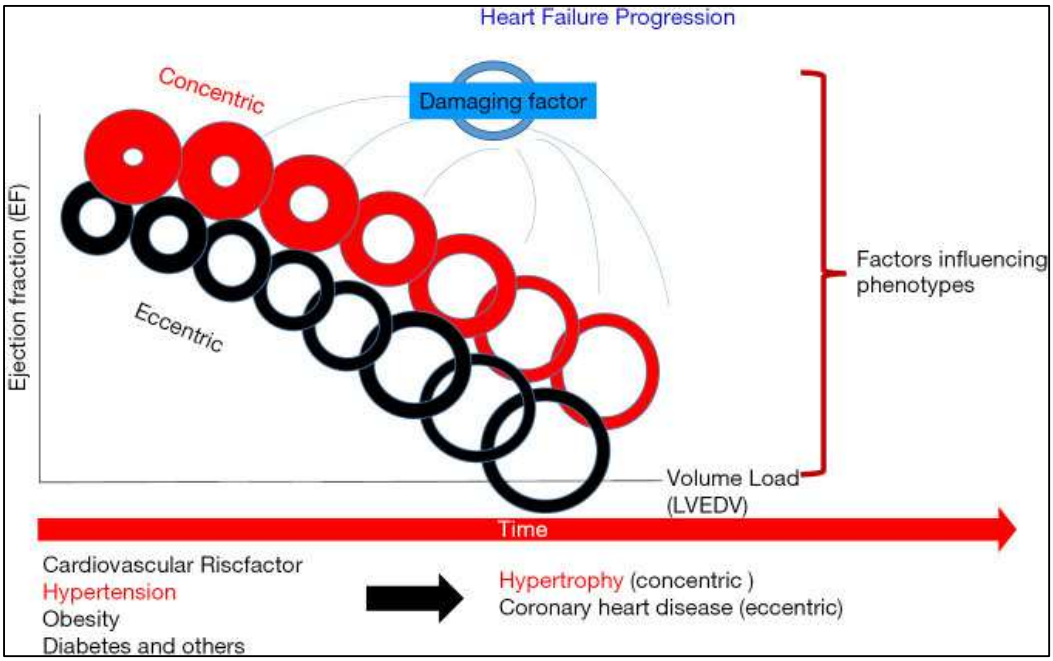
Heart Failure involves a complex pathophysiology. Normal physiological functioning of the heart involves several mechanisms to maintain structural balance, cellular balance and neurohumoral balance.[2] The function of left ventricle and the stroke volume is determined by

1. Preload
2. Myocardial contractility and
3. After load.

The venous return and ventricular end-diastolic volume contribute to pre-load while the wall stress and impedance during ejection from aorta contribute to post-load. "The Frank-Starling curve explains the relationship between stroke volume/cardiac output and left ventricle end-diastolic pressure (LVEDP) or pulmonary capillary wedge pressure (PCWP) in which there is a steep and positive relationship between increased cardiac filling pressures and increased stroke volume/cardiac output". In advanced disease, higher pressure is required to achieve the same output. The increase in LVEDP and venous return will not be able to increase the stroke volume.

In HFrEF, there is eccentric remodelling. It occurs together with chamber dilatation, overload of volume resulting in forward failure and occurs usually due to typically large anterior MI. In HFpEF, there is impaired relaxation of ventricles leading to increase in ventricular stiffness. This ultimately leads to pressure overload, elevated filling pressure resulting in concentric remodelling and/or ventricular hypertrophy and backward failure.

Figure 4: Pathophysiology of Heart failure[37]



The subjects with HFpEF commonly are old, obese belonging to female gender. They have a history of systemic hypertension, AF.[38] There is no significant evidence-based management strategies for them for improving outcomes. In HFrEF subjects, underlying diseases like CHD, valvular heart disease should be effectively treated.[37]

The presentation of HF can be acute due to MI or due to an acute hypertensive emergency. It can also present in patients who are stable, but decompensate suddenly such as due to increased intake of fluids or due to increased heart rate in AF. The most common type of presentation of acute HF clinically is by decompensation of chronic heart failure.

In HFpEF, there is concentric LVH compared to eccentric LVH in HFrEF. The pressure-volume curve is shifted to the left in HFpEF.

In HF, there is activation of neurohumoral systems – "sympathetic nervous systems (SNS), renin-angiotensin-aldosterone system (RAAS), antidiuretic hormone,

and other vasoactive substances (brain natriuretic peptide (BNP), nitric oxide, and endothelin)". The carotid baroreceptor response is decreased in HF. It leads to increased sympathetic activity. This inturn causes increased heart rate, cardiac contractility and increase in the after-load due to vasoconstriction. Because of decrease in the renal perfusion, there is RAAS activation (angiotensin II increases) leading to salt and water retention and hence increased pre-load. This leads to further stress in the ventricular wall and remodelling of ventricles. Again this leads to further HF by worsening of the functioning of ventricles. There is negative remodelling of the heart due to these compensatory mechanisms.[37]

Clinical manifestations:

The symptoms and presentation of HF is mainly determined by the type of HF, characterised by the side of dysfunction (right sided, left sided or biventricular). In left ventricular dysfunction, there is increased pulmonary pressure and backward failure , finally leading to congestion of lungs resulting in increase in the respiratory rate, dyspnoea and lung signs on auscultation.[37] With failure of peripheral circulation, that is in forward failure, cardiac cachexia develops due to malabsorption of nutrients, dysfunction of kidneys and malperfusion in the peripheries. Symptoms of volume overload dominate in a chronic state due to the compensatory mechanism leading to congestion of liver, accumulation of water in third space leading to symptom as and signs of ascites and oedema. Dyspnoea is further worsened by fatigue of muscles of diaphragm and accessory muscles of respiration.[37,39] In spite of the increase in the filling pressure on the left side, pulmonary venous congestion signs such as rales, are not seen in CHF. It is due to chronic lymphatic hypertrophy, resulting in prevention of alveolar oedema.[40]

A typical third or fourth heart sound also known as protodiastolic gallop can be seen in conditions of ventricular volume overload. The symptoms of HF involves all the organ systems in the body and hence can be regarded as a systemic disease. [41]

The clinical criteria established by the **Framingham Heart Study**, requires two major criteria for congestive heart failure, such as elevated jugular venous pressure, pulmonary rales, or a third heart sound, or one major criterion and two minor criteria, including peripheral edema, dyspnea on exertion, or hepatomegaly.[42] The disadvantage is poor sensitivity, especially for early heart failure.[4]

Figure 5: Framingham Heart Failure Criteria

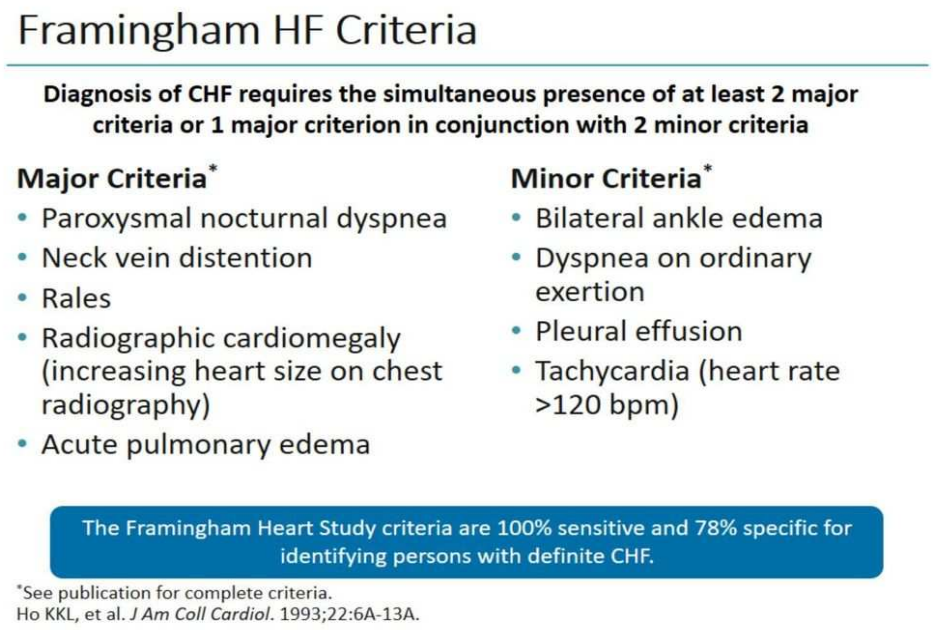


Figure 6: The New York Heart Association (NYHA) functional classification of HF

Class I: HF does not cause limitations to physical activity; ordinary physical activity does not cause symptoms.

Class II: HF causes slight limitations to physical activity; the patients are comfortable at rest, but ordinary physical activity results in HF symptoms.

Class III: HF causes marked limitations of physical activity; the patients are comfortable at rest, but less than ordinary activity causes symptoms of HF.

Class IV: HF patients are unable to carry on any physical activity without HF symptoms or have symptoms when at rest.

I CHRONIC HEART FAILURE WITH REDUCED LVEF

Chronic heart failure (CHF) remains the only cardiovascular disease with an increasing hospitalization burden and an ongoing drain on health care expenditures.[41] The prevalence of CHF increases with advancing life span, with diastolic heart failure predominating in the elderly population.

In HFrEF, there is substantial loss of cardiomyocytes. This loss could be acute or chronic leading to systolic dysfunction.[43,44] As a consequence, there could be eccentric remodelling resulting in excessive fibrotic tissue. There is elongated and thinner cardiomyocytes with lower myofibrillar density.[45] HF with mildly reduces EF can develop into either HF with preserved EF or reduced EF, although the phenotype will be dominated by CAD as in HFrEF.[46] There is no significant evidence based

management strategies for them for improving outcomes in HFpEF. But in HFrEF subjects, underlying diseases like CHD, valvular heart disease can be effectively treated to get favourable outcomes.[37] In HFrEF, there is eccentric remodelling. It occurs together with chamber dilatation, overload of volume resulting in forward failure and occurs usually due to typically large anterior MI. "HFrEF patients show up more often with coronary heart disease (myocardial infarction), valve disease (aortic stenosis, mitral regurgitation) or uncontrolled hypertension; these more exactly defined underlying diseases in HFrEF are to be treated more effectively via medication, surgery or intervention".[37]

In CHF, the initial management is constructed on diuretics, ACE inhibitors, beta-blockers and mineralocorticoid receptor antagonists. "The new European Society of Cardiology (ESC) guidelines published in 2016 introduced angiotensin-receptor-neprilysin inhibitors, such as sacubitril/valsartan (LCZ 696) as new therapeutic agents in patients with chronic and progressive heart failure. New subgroup analyses for LCZ 696 have been published showing a beneficial effect in the context of various comorbidities, such as renal insufficiency, diabetes and hypotension. Furthermore, new data are available on intravenous iron substitution in chronic heart failure and on the indications for implantable converter defibrillators, cardiac resynchronization therapy and other cardiac devices. Medicinal therapy of acute heart failure is still limited. For patients who cannot be treated with medicinal therapy, mechanical circulatory support, such as extracorporeal membrane oxygenation (ECMO) should be recommended".[47]

II. PREDICTORS OF CHRONIC HEART FAILURE WITH REDUCED LVEF

"Biomarkers, such as N-terminal prohormone brain natriuretic peptide and troponin, may potentially function in this role; however, the cost-effectiveness and target populations for these strategies remain unsettled".[48,49]

"National Academy of Clinical Biochemistry has set forth comparable goals in a consensus document stating that a biomarker in HF ideally enables clinicians to: (i) identify possible underlying (and potentially reversible) causes of HF; (ii) confirm the presence or absence of the HF syndrome; and (iii) estimate the severity of HF and the risk of disease progression. Multiple biomarkers have been classified depending on their putative functional impact on cardiac myocytes and the resulting pathophysiological changes in patients with HF and include (a) myocyte stretch biomarkers; (b) myocyte necrosis biomarkers; (c) systemic inflammation biomarkers; (d) oxidative stress biomarkers; (e) extracellular matrix turnover biomarkers; (f) neuro-hormone biomarkers; and (g) biomarkers of extra-cardiac processes, such as renal function".[50]

Table: 2 Specific biomarkers in HF and correlation to the underlying pathophysiology Adapted from Ahmad et al., 2012.[51];

Myocardial Stress	Myocardial Injury	Matrix and Cellular Remodeling	Inflammation	Oxidative Stress	Neuro-Hormones	Vascular System	Cardio-Renal Syndrome
Natriuretic	Cardiac troponins	Osteopontin	C-reactive protein	Oxidized LDL	Nor-epinephrine	Homocysteine	Creatinine
peptides	High sensitivity cardiac troponins	Galectin-3	sST2	Myeloperoxidase	Renin	Adhesion molecules	Cystatin C
Mid-regional	Myosin light-chain kinase 1	sST2	Tumor necrosis factor	Urinary biopyrrins	Angiotensin-II	ICAM, P-selectin	NGAL
Pro-adrenomedullin	Heart-type fatty acid binding protein	GDF-15	FAS (APO-1)	Urinary and plasma isoprostanes	Co-peptin	Endothelin	Trace protein
Neuregulin	Pentraxin 3	MMPs	GDF-15	Plasma malondialdehyde	Endothelin	Adiponectin	
sST2		TIMPs	Pentraxin 3			C-type natriuretic peptide	
		Collagen propeptides	Adipokines				
			cytokines				
			Procalcitonin				
			Osteoprotegerin				

"APO, apoptosis antigen; GDF, growth differentiation factor; ICAM, intercellular adhesion molecule; MMPs, matrix metalloproteinases; NGAL, neutrophil gelatinase-associated lipocalin; sST2, soluble ST2; TIMPs, matrix metalloproteinase tissue inhibitors".

Emerging investigation in heart failure

1. NT pro Brain natriuretic peptide (BNP):

Natriuretic peptides form in heart primarily. Then they get released into circulation as a response to increase in the wall tension.[52] But contrary to ANP, BNP is secreted both from the atria and ventricles, specifically in heart failure patients. The

natriuretic peptide family mainly consists of ANP (synthesized and secreted by atrial myocytes), BNP, and C-type natriuretic peptide (CNP).[53]

"ANP, BNP, and their N-terminal pro-hormones (N-terminal pro-atrial natriuretic peptide (NT-proANP) and N-terminal pro-brain natriuretic peptide (NT-proBNP))—are raised in both symptomatic and asymptomatic patients with left ventricular dysfunction.[54,55] Recent smaller studies suggest that BNP and NT-proBNP may be superior to ANP and NT-proANP in the detection of left ventricular dysfunction.[55,56] .Recently a reliable and less time consuming enzyme linked immunosorbent assay (ELISA) method for the analysis of NT-proBNP has been developed and NT-proBNP may therefore be a suitable peptide for a diagnostic assay".[57]

2. mi RNAs:

MicroRNAs are also known as miRNAs. They are small endogenous non-coding RNAs, that are about 22 nucleotides long and regulate post-transcriptional expression of the gene.[58] MicroRNAs can inhibit the translation of mRNA (messenger RNA) or induce specific mRNA degradation. There has been documented association between several miRNA pattern expressions with various HF mechanisms.[59] They play an important role in HF pathogenesis. It was reported from a systematic review that the evidence available currently is insufficient to use miRNAs in clinical circumstances.[60]

3. orexin:

Orexins also known as hypocretins cause centrally mediated effects on CVS. They are seen exist in two forms functionally

1. OR-A or orexin A
2. OR-B or orexin B

A 130 amino acid precursor peptide gives rise to them both. They exert their action via the orexin 1 and orexin 2 receptors.[61] Orexin and Orexin 2 receptor containing nerve fibres are identified in CNS in the paraventricular nucleus, which is a vital area for integrating cardiovascular function with sympathetic outflow.[62,63] .There is a significant negative correlation in HF, between Orexin 2 receptor expression and NYHA clinical symptom based disease severity.[63]

Correlation of thyroid function test in heart failure (Free T3, Free T4, Serum TSH)

With increase in age, there is increase in prevalence of CVD, systemic hypertension and hypothyroidism. In hypothyroidism, the most sensitive test for detection is TSH. In heart failure (HF) the main alteration of thyroid function is referred to as "low-triiodothyronine (T3) syndrome" (LT3S) characterized by decreased total serum T3 and free T3 (fT3) with normal levels of thyroxine (T4) and thyrotropin (TSH).[64] Even if commonly interpreted as an adaptive factor, LT3S may have potential negative effects, contributing to the progressive deterioration of cardiac function and myocardial remodelling in HF and representing a powerful predictor of mortality in HF patients.[64]

Triiodothyronine, also known as T3 is responsible for the significant effects of thyroid hormones on cardiovascular system. T3 leads to increase in force and speed of systolic contraction. It also leads to increase in speed of diastolic relaxation. It lowers vascular resistance.[65] Myocardial hypertrophy can occur both physiologically and pathologically due to the effect of thyroid hormones. Relaxation of vascular smooth muscles occurs due to the direct effect of T3. HF results in down-regulation of the heart's thyroid hormone signalling system.[65,66] .In subjects with HF, thyroid hormone

replacement has proven beneficial effects in terms of improving the cardiac contractility.[67,68] .An increase in the action of thyroid hormones either by increased serum T3 levels or levels of T3 receptors can lead to enhanced functioning of the heart without any harmful effects. HF can lead to a hypothyroid cardiac state because of lower TR levels.[65-67]

In hypothyroidism, there is lowering of compliance of the vascular system and vasorelaxation mediated by the endothelium. This leads to increase in the diastolic BP.[69] In hyperthyroidism, there is increased lowered vascular resistance in the periphery, which leads to increase in the blood volume and increase in venous return known as "high output failure" or "a congestive state". "Clinically relevant heart failure implies that despite adequate venous return the heart cannot pump all the blood that returns to it. However, this is not the case in uncomplicated hyperthyroidism where there is a high output state not unlike that which may occur with a peripheral arteriovenous fistula, severe anaemia, pregnancy, or severe liver disease". Besides the overt dysfunction of thyroid, HF is becoming associated with subclinical states of hyperthyroidism and hypothyroidism. There has also been documented association between high normal thyroid function and mild increase in risk of development of AF.[70]

III. AST/ALT RATIO AS A PREDICTOR OF CHRONIC HEART FAILURE WITH REDUCED LVEF

The aspartate transaminase (AST)-to-alanine aminotransferase (ALT) ratio, is used to measure liver injury and has been found to be biomarker for diseases of the cardiovascular system[16], various cancers[17], and T2DM.[18] The concept of aspartate transaminase/alanine transaminase (AST/ALT), also termed the De Ritis ratio, was first

proposed for the study of hepatitis etiology_and commonly used to differentiate varying causes of liver disease such as fatty liver".[15] There has also been evidence from retrospective studies that AST/ALT ratio has been associated with prognosis of pancreatic cancer.[71]

Almost before 50 years, De Ritis described the AST/ALT ratio.[15,72,73] They mainly indicate hepatocellular damage or death when they (ALT and AST) are released from liver cells in abnormal quantities to the bloodstream.

The hepatic proportion of AST/ALT is 2.5:1 but as AST is removed from serum by the liver sinusoids quickly ($t_{½}$=18 h) compared to ALT ($t_{½}$=36 h), both of them have a fairly similar upper reference limits at approximately 30 IU/L to 40 IU/L for both AST and ALT.[74]

AST is found in human tissues with the maximum levels in heart, liver, skeletal muscle, kidney and brain. An increased levels of AST may reflect tissue damage(plasma membrane disruption or apoptosis), plasma membrane bleb formation, increased tissue expression and macroenzymes (complexes of AST with plasma proteins). Severe myocardial ischemia or myocardial cell necrosis in acute myocardial infarction is a common cause of increased serum AST activity.[75]

In acute myocardial infarction, AST start raising 6 to 8 hours after the symptom starts, reaches the peak level at 24 to 36 hours and returns to normal in 3 to 7 days. Reperfusion by thrombolysis or balloon angioplasty shortens the time to AST peak value. Previously AST was of diagnostic value for patients with ischemic damage and risk stratification of patients with acute myocardial infarction. Since more sensitive biomarkers of myocardial ischemia/necrosis became available, AST is no longer used for the diagnosis of acute myocardial infarction.[75]

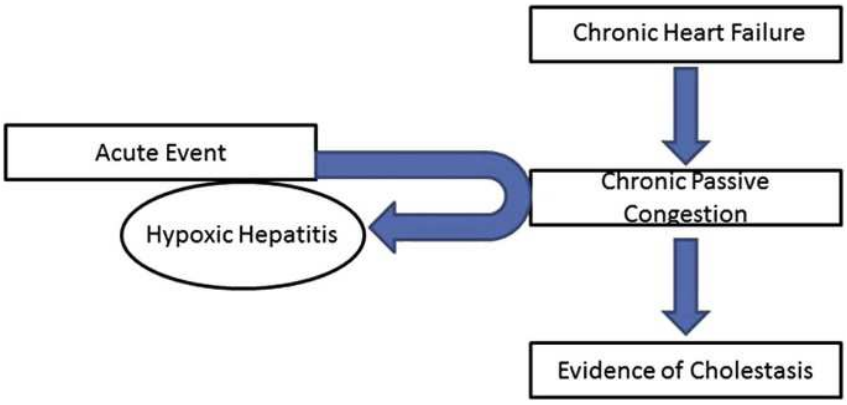


Figure 7: Consequence of Chronic heart failure

Chronic heart failure leads to passive congestion of liver ("nutmeg liver")leads to liver function panel consistent with cholestasis. Patients with chronic heart failure who experienced an acute decompensation showed evidence of hypoxic hepatitis. Necrosis can extend to mid zonal hepatocytes with prolonged ischemia, there is a rapid elevation of aminotransferase and lactate dehydrogenase levels to 10 to 20 times normal within 1 to 3 days of haemodynamic insult.[76]

But release of ALT occurs only from liver. There would an increase in AST/ALT ratio in case of injury to the myocardium.[19] The probability of occurrence of hepatic fibrosis is also higher with increased AST/ALT ratio. It is also associated with pathogenesis of CVD through various mechanisms, such as increase in the amount of inflammatory mediators in the plasma, resistance to insulin, metabolic syndrome and oxidative stress.[20]

There are very limited studies that have evaluated the role of liver transaminases, especially AST/ALT ratio in predicting cardiovascular diseases, especially in HFrEF. Ewid M et al[21] in their study observed that there is potential for increased AST/ALT ratio for predicting functional status decline in subjects with HFrEF. There have been studies reported that have evaluated the role of hepatic transaminases as predictors of CVD. It was observed by Lazo et al[77] in their study

observed that increase in liver transaminases levels correlated with levels of troponin T and NT-pro BNP. But the problem in their study was the failure to enrol HF subjects. Another study done by Yokoyama et al[78], observed that high AST/ALT ratio correlated positively with levels of NT-proBNP. But in their study, the study population was not limited to HFrEF subjects. In a study done by Zoppini et al[79], it was observed that AST/ALT ratio had positive correlation with mortality in CVD, in subjects with T2DM. But their study was also limited by the fact that their study was not limited to subjects with HFrE.

Most causes of liver cell injury are associated with a greater increase in ALT than AST. An AST to ALT ratio of 2:1 or greater is suggestive of alcoholic liver disease, especially with elevated GGT.[80] The ratio can also be increased in non-alcoholic steatohepatitis. It is also frequently elevated in an alcoholic liver disease pattern in hepatitis C with cirrhosis. An AST elevation, but confined to AST/ALT ratio<2 can be seen in wilson's disease or cirrhosis due to viral hepatitis.

When the AST is higher than ALT, a muscle source of these enzymes should be considered like dermatomyositis.

Table 3: Significance of AST/ALT ratio:

	De Ritis Ratio Decision Limit			
Condition	**<1.0**	**1.0 to <1.5**	**1.5 to <2.0**	**≥ 2.0**
Healthy	Women (up to 1.7)		Children	Neonate
	Men (up to 1.3)			
Acute Viral Hepatitis	Resolving		Worsening	Fulminant
Alcoholic Hepatitis	Resolving		Alcohol Abuse	Acute Hepatitis
Chronic Liver Disease	Stable	Fibrosis risk		Other Causes
Muscle Disease	Chronic	Resolving		Acute

Healthy limits are derived from reference[81]

Image source[73]:

Normal serum AST ranges are 0 U/L to 45 U/L and normal ranges for ALT are 0 U/L to 50 U/L. "According to Enzymatic rate method according to the International Federation of Clinical Chemistry recommendation (BS-820 M, Mindray System-Mindray Diagnostics)".[82] AST divided by ALT gives rise to AST/ALT ratio.

In the literature, ratio of AST/ALT ratio more than or equal to one high specificity for cirrhosis of liver in infection with chronic Hepatitis C virus.[11]

IV. MOST RELEVANT STUDIES

Liu X et al[83] (2022) explored "the association between AST/ ALT ratio and all-cause mortality in stable coronary artery disease (CAD) patients treated by percutaneous coronary intervention (PCI). The study is a secondary analysis of a retrospective cohort study involving 203 stable CAD patients. Patients were divided into two groups, based on the optimal AST/ALT ratio threshold calculated by the ROC curve (low group: AST/ALT ratio < 1.40; high group: AST/ALT ratio ≥ 1.40).In the Kaplan-Meier analysis, an elevated AST/ALT ratio was associated with increased ACM in stable ACD patients. An elevated AST/ALT ratio was still found to be an independent prognostic factor for ACM after adjusting for potential confounders". They concluded that increased AST/ALT ratio has the potential to be an independent prognostic factor for ACM in patients with stable ACD.

Liang W et al[84] (2021) in their study evaluated "the prognostic significance of LFTs in well-defined HFpEF patients. They conveyed a *post-hoc* analysis of the Treatment of Preserved Cardiac Function Heart Failure with an Aldosterone Antagonist Trial (TOPCAT). The primary outcome was the composite of cardiovascular mortality, HF hospitalization, and aborted cardiac arrest, and the secondary outcomes were cardiovascular mortality and HF hospitalization. In Cox proportional hazards models, aspartate transaminase (AST) and alanine transaminase (ALT) were not associated with any of the outcomes. On the contrary, increases in total bilirubin (TBIL) and alkaline phosphatase (ALP) were associated with increased risks of the primary outcome .They concluded that elevated TBIL and ALP, had significant association with poorer outcome in subjects with HFpEF and without chronic hepatic diseases, but not with increased ALT and AST.

Ewid M et al[21] (2020) in their study evaluated the role of AST/ALT ratio as a predictor of functional severity in chronic HF with reduced LVEF. "105 patients previously diagnosed with HFrEF from Buraidah-Al Qassim province, Saudi Arabia were included in this retrospective cross-sectional study. Data on study variables, including demographic data, left ventricular ejection fraction, NYHA class, and AST/ALT ratio, were collected from patients' records. The patients were divided into two groups, namely group-1 (AST/ALT ratio<1) and group-2 (AST/ALT ratio≥1), to identify any differences in their cardiac function profiles. NYHA class and NT-proBNP were higher and LVEF was lower in group-2 than in group-1. They found a mild significant correlation between AST/ALT ratio and APRI, FIB-4 score, NYHA-class, and LVEF (r=0.2, 0.25, 0.26, and −0.24, respectively; P<0.05). Multivariate linear regression analysis model and ROC curve showed that AST/ALT ratio could independently predict HFrEF functional severity with a best cut-off value of 0.9, sensitivity of 43.6%, and specificity of 81.4%". They concluded that AST/ALT ratio can independently predict functional severity of HFrEF.

Liu H et al[85] (2021) in their study assessed "the relationship between AST/ALT ratio and all-cause and cardiovascular mortality in patients with hypertension. By March 31, 2020, a cohort of 14,220 Chinese hypertensive patients was followed up. The end point was all-cause and cardiovascular death. They concluded that increase in AST/ALT ratio levels can predict all-cause and cardiovascular mortality in Chinese patients with hypertension.

Sankar K et al[86] (2016) conducted a prospective study in subjects with HF to assess the relationship between severity of HF and impaired function of kidneys and liver in HF subjects. They observed that patients with ejection fraction ≤40-85% had increased

bilirubin, 92.5% had increased serum glutamic oxaloacetic transaminase, 92.5% had increased serum glutamic pyruvic transaminase, and 22.5% had increased alkaline phosphatase. In patients with ejection fraction, ≤40-57.5% had increased urea and 62.5% had increased creatinine". They concluded that degree of HF considerably increased with increased involvement of liver and kidneys.

Yokoyama M et al[78] (2016) did their longitudinal cohort study on 3,494 Japanese subjects, with a follow-up of 10 years. "The AST/ALT ratio increased with increasing BNP levels. And multivariate logistic analysis showed that the AST/ALT ratio was significantly associated with a high BNP (≥100 pg/mL). There were 250 all-cause deaths including 79 cardiovascular deaths. Multivariate Cox proportional hazard regression analysis revealed that a high AST/ALT ratio (>90 percentile) was an independent predictor of all-cause and cardiovascular mortality after adjustment for confounding factors. Kaplan-Meier analysis demonstrated that cardiovascular mortality was higher in subjects with a high AST/ALT ratio than in those without. They concluded that AST/ALT ratio was associated with an increase in BNP and was predictive of cardiovascular mortality in a general population". They concluded that AST/ALT ratio assessed during routine health check-ups can be used as a cost-effective and a simple marker for mortality in cardiovascular diseases.

Zoppini G et al[79] (2016) assessed "the relationship between the AAR and mortality risk in a well-characterized cohort of patients with type 2 diabetes. A cohort of 2529 type 2 diabetic outpatients was followed-up for 6 years to collect cause-specific mortality. Over the 6-year follow-up period, 12.1% of patients died, 47.5% of whom from CV causes. The AAR was independently associated with an increased risk of both all-cause and CV mortality in patients with type 2 diabetes". They concluded that an

increased AAR reflects systemic derangements more than the liver damage and further evidence is needed to substantiate their hypothesis.

Lazo M et al[77] (2015) in their cross-sectional analysis assessed 8668 participants from the Atherosclerosis Risk in Communities (ARIC) Study, without any evidence of cardiovascular disease clinically. They assessed ALT, AST and GGT without history of elevated alcohol consumption as non-invasive surrogates of NAFLD. "They used highly sensitive cardiac troponin T (hs-cTnT) and N-terminal pro-Brain natriuretic peptide (NT-proBNP) as biomarkers of myocardial damage and function. In their population-based study ,higher levels of ALT, AST, and GGT, even within the normal range, were significantly and independently associated with detectable (hs-cTnT >3 ng/L) and elevated (hs-cTnT ⩾14 ng/L) concentrations of hs-cTnT. The adjusted odds ratios (95% confidence interval) for elevated liver enzymes (vs. normal levels) with elevated hs-cTnT were: 1.65 (1.28-2.14) for ALT, 1.90 (1.36-2.68) for AST, and 1.55 (1.13-2.12) for GGT. Furthermore, there was evidence for inverse associations of ALT and AST with NT-proBNP". They concluded that increase in liver enzymes without elevated alcohol consumption has association with subclinical injury of the myocardium.

Samsky MD et al[7] (2013) in their review described the effects of HF on hepatic function. In this study, they highlighted the key differences in clinical presentation, histological findings, and biochemical profiles of patients who present with both acute and chronic liver injury secondary to HF. They discussed the use of liver function tests as prognostic markers in patients with HF, as well as the implications of liver injury on drug metabolism in this patient population.

Lofthus DM et al[8] (2012) evaluated elevation of liver enzymes in subjects with STEMI. They also evaluated correlation of liver enzymes with creatine kinase-MB and associations with outcomes. "The Complement Inhibition in Myocardial Infarction Treated with Angioplasty and Complement Inhibition in Myocardial Infarction Treated with Thrombolytics trials evaluated 1903 patients with STEMI. A core lab analyzed liver enzymes at baseline, days 1, 6, and 14, and CK-MB measured sequentially over 72 h. The GUSTO model for 30-day mortality was used to predict clinical endpoints. A total of 1783 patients were included in the analysis. Aspartate transaminase (AST) was elevated above the upper limit of normal in 85.6% and alanine transaminase (ALT) was elevated in 48.2% of patients at baseline or day 1. CK-MB area under the curve correlated with maximum AST (r=0.727) and maximum ALT (r=0.456). Both AST and ALT elevations were independent predictors of worse outcomes in multivariable adjusted analysis, even after adjustment for CK-MB. Hazard ratios and 95% confidence intervals of AST elevation were 1.12 (1.05-1.19) for all-cause mortality, and 1.08 (1.02-1.13) for the composite endpoint of death, congestive heart failure, shock, or stroke. Hazard ratios and 95% confidence intervals of ALT elevation were 1.15 (1.04-1.27) for mortality and 1.47 (1.10-1.98) for the composite endpoint. They concluded that AST and ALT elevations are common in STEMI". They concluded that both markers correlated with levels of CK-MB and had independent association with poor mortality and outcomes.

Alvarez AM et al[87] (2011) discussed the pathophysiology of congestive hepatomegaly and liver diseases in heart failure and cardiac diseases. They observed that ALT/LDH ratio of < 1.5 can differentiate ischemic injury from other causes of acute hepatitis.

MATERIALS AND METHODS

Source of Data:

The study was conducted with detailed history and examination in tertiary care, KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi in patients of Heart failure, fulfilling the inclusion criteria.

Study design:

A prospective cross-sectional study.

Study period:

January 2021 to December 2021

Sample size:100

The minimum sample size formula based on the prevalence rate is

$$n = \frac{z_a^2 P(1-P)}{d^2}$$

where P is the percentage of prevalence and d is the percentage likely difference in the prevalence.[107]

Z_a is linked with the level of significance. For the 5% level of significance z = 1.96.

With P= 46% and d = 25% of P = 11.50%, the sample size is 72.

To seek confirmative results the sample size will be increased to 100.

Sample method: a prospective cross-sectional study. all consecutive patients fulfilling the inclusion criteria with being included in the study, statistical analysis will be done by chi-square test

INCLUSION CRITERIA:

- The age group of more than 18yrs of age
- Left ventricular ejection fraction <=40%

EXCLUSION CRITERIA:

- Primary liver disease
- Sepsis
- Shock
- Malignancy
- Pregnancy & Lactating women
- Renal failure

ETHICAL CONSIDERATION

The study was approved by the institutional human ethics committee. Informed written consent was obtained from all the study participants and only those participants who signed the informed consent were included in the study.

METHODOLOGY

1. All the patients fulfilling the inclusion criteria and willing to participate were included in the study
2. Informed consent will be obtained.
3. Further, they will be subjected to detailed history and examination.
4. Study groups are divided into
    - AST/ALT ratio <1(group 1)
    - AST/ALT ratio >=1(group 2)

5. Laboratory investigations and calculation of liver fibrosis indices: Complete blood count, liver function tests, Renal function tests, thyroid profile, and were recorded.
6. We calculated the following standard non-invasive hepatic fibrosis indices:
    - AST/ALT ratio.
    - AST to platelet ratio index (APRI) = ([AST/ top normal AST] / platelet count [10^9 / liter]) × 100.
    - Fibrosis-4 (FIB-4) = (Age × AST) / (Platelet count × ALT square root).
7. Transthoracic echocardiography: was performed according to the recommendations of both the American Society of Echocardiography and the European Association of Cardiovascular Imaging. The modified Simpson's rule was used to estimate left ventricular (LV) volumes and measure the ejection fraction.
8. Estimation of systolic pulmonary arterial pressure (SPAP): was estimated using the modified Bernoulli formula (SPAP [mmHg] = 4 × tricuspid regurgitation velocity^2 + right atrial pressure).
9. The variables were compared between two groups and the correlation of AST/ALT ratio with NYHA class (NEW YORK HEART ASSOCIATION), LVEF <=40% APRI, and FIB-4 score, to know if AST/ALT ratio acts as an independent indicator in detecting the severity of heart failure with reduced LVEF<=40% patients and to confirm the previous study done on a similar study.

INVESTIGATION PERFORMED ON PATIENTS ARE

1) Complete blood count

   I Haemoglogin(g/dl)

   II Platelet (X $10^3/\mu l$)

   III WBC(X $10^3/\mu l$)

   IV PT –INR, APTT

2) Liver function test

   a. Total bilirubin(mg/dl)

   b. Direct bilirubin (mg/dl)

   c. Total protein (g/dl)

   d.AST(U/L)

   e.ALT(U/L)

   f.ALP(U/L)

   g.Serum Albumin(gm/dl)

   II AST/ALT RATIO

   III. APRI INDEX

   IV. FIB-4 SCORE

   RBS (mg/dl)

3)Serum Urea (mg/dl)

4)Serum Creatinine (mg/dl)

5)Serum Sodium(meq/l)

6)Serum Potassium (meq/l)

7)USG Abdomen and pelvis

8)Serum TSH (micu/ml) Free T3 (pg/ml), Free T4 (ng/dl)

9)Echocardiogram

10) TRANSTHORACIC ECHOCARDIOGRAPHY

- Ejection Fraction %
- Valves (Mitral, Aortic, Tricuspid, Pulmonary)
- Chambers (Left Ventricle, Right Ventricle, Left Atrium, Right Atrium)
- Septae
- Great Arteries (Aortal, Pulmonary Artery)
- Regional Wall Motion Abnormality
- Pericardial Effusion
- Clots /Vegetation

STATISTICAL ANALYSIS:

Data is analysed using statistical software R version 4.2.1 and Microsoft Excel. Categorical variables given in the form of frequency tables. Continuous variables given in Mean ± SD / Median (Min, Max) form. Chi square test (by monto carlo stimulation test) is used check the association between categorical variables. Normality of variable is checked by Shapiro Wilk test and QQ plot. Two sample t test is used to compare the means of variables over groups. Mann Whitney U test is used to compare the distribution of variables over groups. Spearman's rank correlation test is used to check the correlation between the variables. Applicability of AST/ALT ratio to predict the severity of chronic heart failure with reduced EF is checked by Logistic regression and Receiver Operating Characteristic (ROC) curves. Optimal Cut off value is obtained by using Youden index method. P-value less than or equal to 0.05 indicates statistical significance

RESULTS

In our study 100 subjects are taken whose age ranges between 19 to 89 years with mean age of 59.44 ± 14.68 years. They are divided into two groups based on AST/ALT ratio.

- Group 1: - AST/ALT <1
- Group 2: - AST/ALT ≥ 1

Table 4: Comparison of demographic variables in Group 1 & Group 2.

Variables	Sub Category	Group 1 (n=21)	Group 2 (n=79)	Total (n=100)	p-value
Age group (years)	≤ 40	3 (14.29%)	7 (8.86%)	10 (10%)	0.5027
	41-50	4 (19.05%)	8 (10.13%)	12 (12%)	
	51-60	5 (23.81%)	17 (21.52%)	22 (22%)	
	61-70	4 (19.05%)	30 (37.97%)	34 (34%)	
	>70	5 (23.81%)	17 (21.52%)	22 (22%)	
	Total	21(100%)	79(100%)	100(100%)	
	Mean ± SD	56.71 ± 16.97	60.16 ± 14.04	59.44 ± 14.68	0.2784
Gender	Female	2 (9.52%)	35 (44.3%)	37 (37%)	**0.006***
	Male	19 (90.48%)	44 (55.7%)	63 (63%)	

Abbreviation: * indicates statistical significance.

Out of 100 participants, Group 1 had 21 participants and Group 2 had 79 participants among which most of them where in a age group of 51-70 years of age with a mean age of 56.71 ± 16.97 years in group 1 and mean age of 60.16 ± 14.04 years in group 2 . The difference in proportion of group 1 and group 2 across the age group was statistically not significant with p value 0.5027.

In gender analysis it is observed that number of male patients are higher than female that is 63 male and 37 female. The difference in proportion of group 1 and group 2 between gender was statistically significant with a p value of 0.006.

Figure 8: Distribution of age in Group 1and Group 2

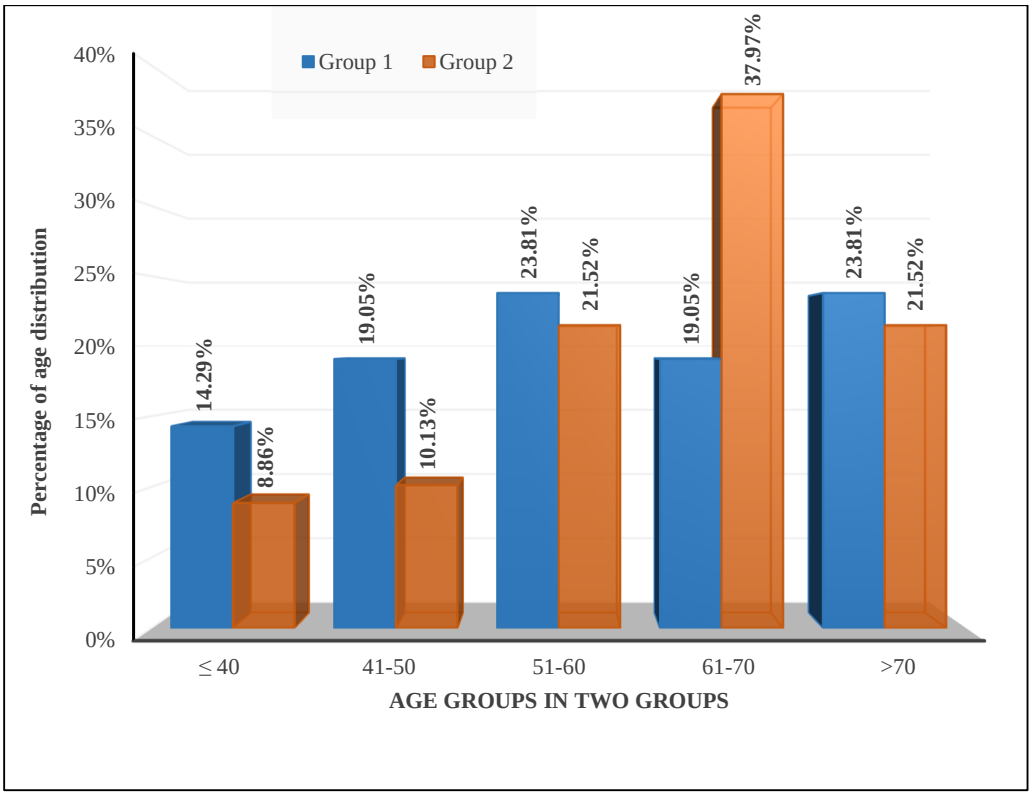


Table 5: Comparison of symptoms and signs of heart failure in Group 1 & Group 2

Symptoms & Signs heart failure	Group 1 (n=21)	Group 2 (n=79)	Total (n=100)	p-value
Cough	2 (9.52%)	6 (7.59%)	8 (8%)	1
Dyspnea	21 (100%)	79(100%)	100 (100%)	
Duration of Dyspnea (years) Mean ± SD	3.52 ± 2.87	2.9 ± 1.92	3.03 ± 2.15	0.193
Paroxysmal Nocturnal Dyspnea	5 (23.81%)	21 (26.58%)	26 (26%)	0.7968
Orthopnea	12 (57.14%)	18 (22.78%)	30 (30%)	**0.0055***
Pedal Oedema	13 (61.9%)	35 (44.3%)	48 (48%)	0.2299
Abdominal Distension	3 (14.29%)	10 (12.66%)	13 (13%)	1
JVP (cm of H2O) Mean ± SD	10.52 ± 0.87	10.61 ± 1	10.6 ± 0.97	0.4405
Hepatomegaly	2(9.52%)	8(10.13%)	10(10%)	1
Pulmonary rales	11(52.38%)	39(49.37%)	50(50%)	0.5572

Abbreviation: * indicates statistical significance.

From Chi square test, it is observed that, dyspnea was the common symptom in both groups with a mean duration of (3.52 ± 2.87) years in group 1 vs (2.9 ± 1.92) in group 2 which was not statistically significant and PND in group 1 were 23.81% vs group 2 were 26.58%,pedal oedema were 61.9% in group 1 vs 44.3% in group 2,abdominal distension were 14.29% in group 1 vs 12.66% in group 2,JVP found was a mean of 10.52 ± 0.87 in group 1 vs 10.61 ±1 in group 2 , Hepatomegaly were found 9.52% in group 1 vs 10.13% in group 2 , Pulmonary rales were found 52.38% in group 1 vs 49.37% in group 2 suggesting is no significant difference in the distribution of above mentioned symptoms and signs between Group 1 &Group 2. However there is significant difference in the distribution of Orthopnea between group 1(57.14%) and

group 2(22.78%) with a p value 0.0055. There is no significant difference in the distribution of other symptoms and signs between Group 1 and Group 2.

Table 6: Comparison of vitals in Group 1 & Group 2.

Vitals	**Group 1 (n=21)** Mean ± SD	**Group 2 (n-79)** Mean ± SD	**Total (n=100)** Mean ± SD	**p-value**
Pulse (bpm)	85.95 ± 12.53	84.94 ± 12.66	85.15 ± 12.58	0.6344
SBP (mm Hg)	110.1 ± 13.05	111.29 ± 9.39	111.04 ± 10.2	0.4953
DBP (mm Hg)	70 ± 10.49	71.39 ± 6.74	71.1 ± 7.64	0.249

Abbreviation: SBP – Systolic blood pressure, DBP – Diastolic blood pressure

From the test, it is observed that, there is no significant difference in the distribution of pulse, SBP and DBP, between group 1 and group 2.

Table 7: Comparison of laboratory investigation in Group 1 & Group 2.(part 1)

Investigation	**Group 1 (n=21)** Mean ± SD	**Group 2 (n=79)** Mean ± SD	**Total (n=100)** Mean ± SD	**p-value**
Hemoglobin(g/dl)	12.44 ± 2.38	12.2 ± 2.07	12.25 ± 2.13	0.2693
Platelets (X 10^3/µl)	271.52 ± 79.14	247.27 ± 90.69	252.36 ± 88.56	0.1478
WBC (X 10^3/µl)	9.61 ± 3.3	10.3 ± 3.44	10.15 ± 3.41	0.3764
PT-INR	1.31 ± 0.32	1.3 ± 0.48	1.31 ± 0.45	0.6114
APTT	1.03 ± 0.22	1.12 ± 0.37	1.1 ± 0.34	0.1867

Abbreviation: WBC – White blood cells, PT-INR - Prothrombin time, APTT – Activated Partial Thromboplastin clotting time.

From the test, it is observed that, there is no significant mean difference in the distribution of hemoglobin, platelets, WBC, PT-INR and APTT between Group 1 and Group 2 a p value >0.05 suggesting no statistical significance.

Table 8: : Comparison of laboratory investigation in Group 1 & Group 2.(part 2)

Investigation	Group 1 (n=21) Mean ± SD	Group 2 (n=79) Mean ± SD	Total (n=100) Mean ± SD	p-value
RBS(mg/dl)	165.52 ± 91.35	180.23 ± 108.37	177.14 ± 104.76	0.4068
Serum Urea (mg/dl)	40.59 ± 19.65	43.39 ± 27.72	42.81 ± 26.17	0.9595
Serum Creatinine (mg/dl)	1.12 ± 0.33	1.13 ± 0.38	1.13 ± 0.37	0.9797
Serum Sodium (mEq/L)	135.81 ± 3.87	136.19 ± 4.45	136.11 ± 4.32	0.7216
Serum Potassium (mEq/L)	4.34 ± 0.52	4.37 ± 0.82	4.37 ± 0.76	0.8717
Serum TSH (mIU/L)	2.23 ± 2.23	4.79 ± 13.31	4.26 ± 11.91	0.3856
FreeT3 (ng/dL)	1.68 ± 0.88	1.67 ± 0.85	1.67 ± 0.85	0.9529
Free T4 (ng/dL)	1.19 ± 0.44	1.21 ± 0.54	1.21 ± 0.52	0.8305

Abbreviation * indicates statistical significance.

RBS – Random blood sugar, TSH – Thyroid stimulating harmone,

From the test, it is observed that, there is no significant mean difference in the distribution in laboratory investigation between Group 1 and Group 2 with a p value >0.05 suggesting no statistical significance.

Table 9: Comparison of Liver function test in Group 1 & Group 2

Liver function test	Group 1 (n=21) Mean ± SD	Group 2 (n=79) Mean ± SD	Total (n=100) Mean ± SD	p-value
Total bilirubin(mg/dl)	0.71 ± 0.37	1.27 ± 1.58	1.16 ± 1.43	0.3995
Direct bilirubin(mg/dl)	0.3 ± 0.21	0.45 ± 0.5	0.42 ± 0.46	0.1859
Total protein(g/dl)	6.23 ± 0.83	6.54 ± 0.9	6.47 ± 0.89	0.1905
AST(U/L)	43.71 ± 46.42	74.04 ± 102.44	67.67 ± 94.12	0.0911
ALT(U/L)	87.05 ± 138.99	35.63 ± 46.26	46.43 ± 77.66	**0.0013***
ALP(U/L)	119.67 ± 58.01	96.19 ± 36.02	101.12 ± 42.36	0.1244
Serum Albumin (gm/dl)	3.37 ± 0.43	3.4 ± 0.64	3.4 ± 0.6	0.6559
AST/ALT Ratio	0.68 ± 0.21	2.08 ± 1.31	1.79 ± 1.3	< **0.001***
APRI Index	0.49 ± 0.67	0.85 ± 1.18	0.78 ± 1.1	**0.0389***
FIB-4	5.36 ± 19.65	3.11 ± 3.01	3.58 ± 9.27	< **0.001***

(Abbreviation: * indicates statistical significance. AST – Aspartate Aminotransferase, ALT – Alanine Transaminase, ALP – Alkaline phosphatase, ALB – Albumin Blood Test, APRI – Aspartate aminotransferase to platelet ratio index, FIB-4 – Fibrosis 4 index.)

In Liver function test, the mean difference of Total bilirubin(mg/dl), Direct bilirubin(mg/dl), Total protein(g/dl), ALP(U/L), Serum albumin(gm/dl) between Group 1 and Group 2 is no statistically significant

The mean difference of AST(U/L) between Group 1 and Group 2 is significant. The mean difference of ALT(U/L), AST/ALT ratio, APRI Index, FIB-4 between Group 1 and Group 2 is very significant (p value <0.05).

Table 10: Comparison of Risk factors of Group 1 & Group 2

Risk factors	Group 1 (n=21)	Group 2 (n=79)	Total (n=100)	p-value
T2DM	11 (52.38%)	32 (40.51%)	43 (43%)	0.3286
HTN	5 (23.81%)	16 (20.25%)	21 (21%)	0.7866
AF	(0%)	1 (1.27%)	1 (1%)	1
Hypothyroidism	0 (0%)	3 (3.8%)	3 (3%)	0.5987

Abbreviation: T2DM – Type 2 Diabetes Mellitus, HTN - Hypertension, AF – Atrial fibrillation.

From Chi square test, it is observed that, there is no significant difference in the distribution of risk factors between Group 1 and Group 2 with no statistical significance.

Table 11: Etiological Diagnosis among Group 1 & Group 2

Diagnosis	Group 1 (n=21)	Group 2 (n=79)	Total (n=100)	p-value
IHD	15 (71.43%)	58 (73.42%)	73 (73%)	0.8552
DCM	5 (23.81%)	21 (26.58%)	26 (26%)	0.7968
RHD	1 (4.76%)	(0%)	1 (1%)	0.2214

Abbreviation: IHD – Ischemic Heart disease, DCM – Dilated cardiomyopathy, RHD – Rheumatic heart disease.

From Chi square test, it is observed that, there is no significant difference in the distribution of diagnosis among Group 1 and Group 2 with no statistical significance.

Table 12: Comparison of Chronic heart failure according to EF (%) in Group 1 & Group 2.

Chronic heart failure (EF%)	Group 1 (n=21)	Group 2 (n=79)	Total (n=100)	p-value
(EF (31-40%)	18 (85.71%)	73 (92.41%)	91 (91%)	0.4073
(EF<30%)	3 (14.29%)	6 (7.59%)	9 (9%)	
Mean ± SD	33.1 ± 6.02	34.57 ± 4.82	34.26 ± 5.09	0.3166

Abbreviation: EF =EJECTION FRACTION

From the test, it is observed that in EF (31-40%) group 1 were 85.7% vs group 2 were 92.41% whereas in (EF<30%) group 1 were 14.29% and group 2 were 7.59% and with a mean standard deviation of 33.1 ± 6.02 in group 1 and and with a mean standard deviation 34.57 ± 4.82 in group 2 suggesting there is no significant difference in the distribution of chronic heart failure (EF%) between Group 1 and Group 2.

Figure 9: Distribution of chronic heart failure according to EF in Group 1 & Group 2.

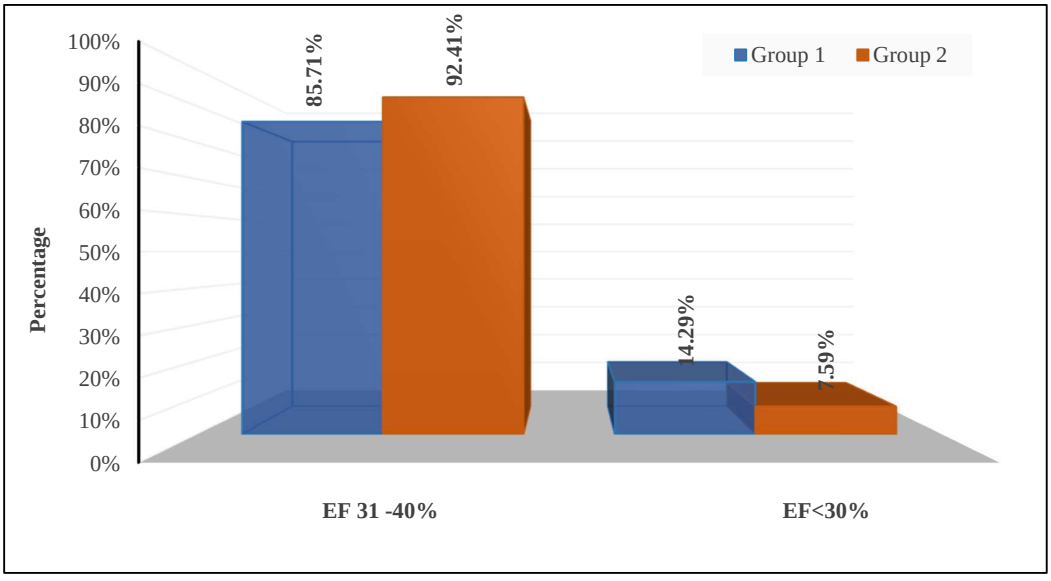


Table 13: Correlation of AST/ALT Ratio with NYHA, APRI index, FIB-4 and EF%.

Variables	Correlation coefficient	p-value
NYHA class	0.0297	0.7707
APRI index	0.3632	**0.0002***
FIB-4	0.5457	**< 0.001***
EF%	0.064	0.5271

Abbreviation: * indicates statistical significance.

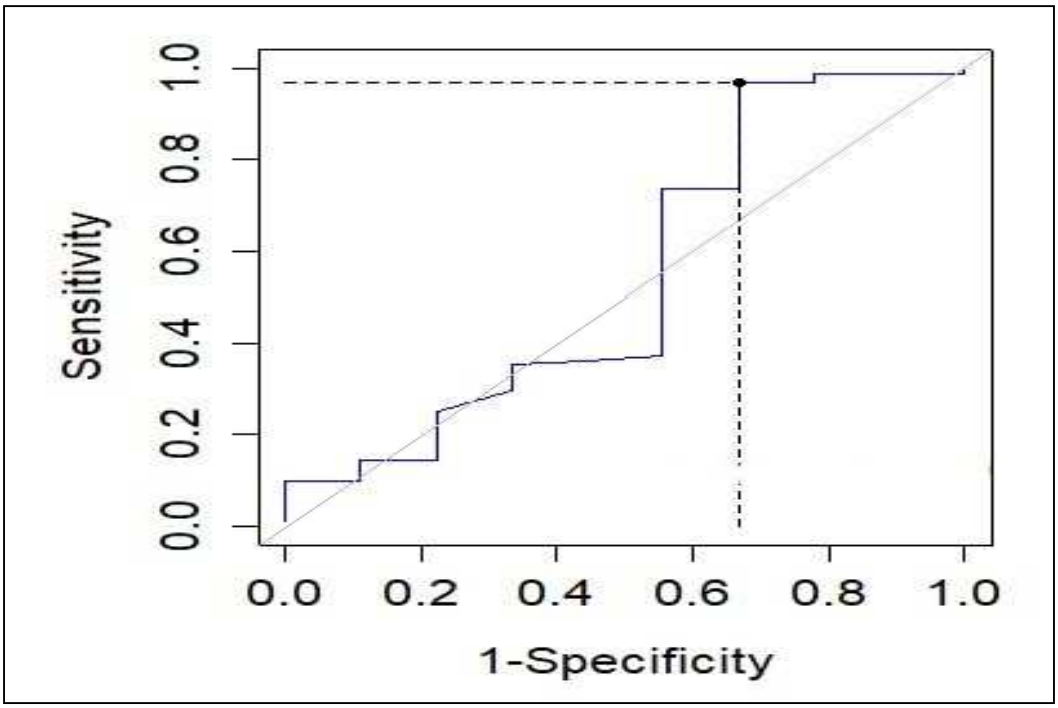
From correlation test, it is observed that, there is significant correlation of APRI index with AST/ALT ratio. There is significant correlation of FIB-4 over AST/ALT ratio. There is no significant correlation of EF% with AST/ALT ratio. There is no significant correlation of NYHA class with AST/ALT ratio.

Table 14: Diagnostic analysis of AST/ALT ratio to predict the severity of chronic heart failure with reduced EF.

	AST/ALT ratio
Cut off	(<) 0.6
Sensitivity (95% CI)	96.70% (90.67% - 99.31%)
Specificity (95% CI)	33.33% (7.49% - 70.07%)
PPV (95% CI)	93.62% (70.36% - 98.64%)
NPV (95% CI)	50% (24.88% - 82.40%)
AU-ROC (95% CI)	0.547 (0.299 - 0.795)
p-value	0.5654

The area under the curve (AUC) was 0.547 (P-value = 0.5654) with a 95% confidence interval of 0.299 – 0.795 with optimal cut-off value of 0.6, sensitivity of 96.70% and specificity of 33.33%.

Figure 10: ROC curve of AST/ALT ratio to predict the reduced EF.



Note: The American Society of Echocardiography cut-off value (EF < 30%) is used for diagnosing patients with severely impaired EF. All subjects had EF≤ 40%. Hence, cut off 40% is not used for analysis.

Note: Multiple linear regression could not be done as there is no significant correlation between EF % and AST/ALT ratio.

DISCUSSION

In our study 100 subjects are taken whose age ranges between 19 to 89 years with a mean age of 59.44 ± 14.68 years. They are divided into two groups based on AST/ALT ratio. Majority (34%) of the subjects were aged between 61 to 70 years in the present study. 63% of the study subjects were males and 37% were female in the present study. **Ewid M et al[21] (2020)** in their study included 105 patients and observed the mean age was 50 ± 14.71 years and the range was between 16 to 88 years. 65% of the subjects were males in their study compared to 63% in the present study. The baseline study population was comparable between their study and the present study. There was no statistically significant difference between group 1 and group 2 with respect to age groups and mean age in the present study, similar to that observed by **Ewid M et al[21] (2020)** with regards to the mean age of study participants. There was a statistically significant difference in gender distribution across the groups in the present study. The prevalence of HF has been reported to be higher in men compared to women in the literature.[89] This could be due to the relatively easy presentation and access of hospitals to males compared to females.

In the present study, 43% of the subjects had a past history of Type 2 DM while 21% had a history of systemic hypertension, and 1% had a history of Atrial fibrillation. It is observed that there is no significant difference in the distribution of risk factors between group 1 and group 2. **Ewid M et al[21] (2020)** in their study observed that more than 50% of their study participants had hypertension and Type 2 DM respectively.

In our study it is observed that dyspnea was the common symptom in group 1 and group 2 with a mean duration of dyspnea (3.52 ± 2.87) years in group 1 vs (2.9 ± 1.92) in group 2 which was not statistically significant. **Ewid M et al[21] (2020)** in their

study observed that duration of heart failure was significantly higher in group 1 compared to group 2 with statistical significance. The symptoms of HF observed in the present study (shown in Table 5) are similar regardless of the EF. There was no statistically significant difference between Group 1 and Group 2.However higher proportion of patients in Group 1 had orthopnoea (57.14%) compared to Group 2 (22.78%) and this difference was statistically significant. In our study there is no significant correlation with AST/ALT ratio and NYHA-class. **Ewid M et al[21] (2020)** in their study found a mild significant correlation between AST/ALT ratio and NYHA-class.

There was no statistically significant difference between group 1 and group 2 with respect to vital parameters in the present study which was found similar to **Ewid M et al[21] (2020).**

There was no statistical significance in signs of heart failure among group 1 and group 2 in the present study(shown in Table 5)

The **Laboratory parameters** performed here (Table 7 & 8) showed there is no significant mean difference in the distribution in laboratory investigation between Group 1 and Group 2. **Ewid M et al[21] (2020)** in their study also observed similarly that the groups were comparable with respect to these parameters with no statistical significance. **Sankar K et al[86] (2016)** in their study observed that in patients with ejection fraction ≤40%, 57.5% had increased urea and 62.5% had increased creatinine. They concluded that the incidence of renal involvement increased considerably with the degree of heart failure. The liver function test in our study showed there was no statistically significant difference between group 1 and group 2 with respect to total bilirubin, direct bilirubin, total protein, Albumin, APRI index, and FIB-4. There was a statistically significant difference between groups with

respect to ALT, AST/ALT ratio, and ALP. But AST was comparable between the groups (43.71 ± 46.42 in Group 1 vs 74.04 ± 102.44 in Group 2).

ALT was higher in Group 1 at 87.05 ± 138.99 compared to 35.63 ± 46.26 in Group 2. ALP was higher in Group 1 at 119.67 ± 58.01 compared to 96.19 ± 36.02 in Group 2. **Liang W et al[84] (2021)** in their study observed that elevated Total bilirubin and ALP were significantly associated with a poor outcome in HFpEF patients without chronic hepatic diseases, while elevated ALT and AST were not. **Sankar K et al.[86] (2016)** in their study observed that in patients with ejection fraction ≤40%, 85% had increased bilirubin, 92.5% had increased serum glutamic oxaloacetic transaminase, 92.5% had increased serum glutamic pyruvic transaminase, and 22.5% had increased alkaline phosphatase. They concluded that the incidence of liver involvement increased considerably with the degree of heart failure.

There was no statistically significant difference in mean LVEF between group 1 and group 2 in the present study. The mean LVEF in Group 1 was 33.1± 6.02% compared to 34.57± 4.82% in Group 2. But **Ewid M et al[21] (2020)** in their study observed LVEF was higher in Group 1 compared to Group 2 (28.93% ± 10.26 Vs 23.83% ± 7.04) and this difference was statistically significant. They observed that in their multivariate model that included age, body mass index (BMI), diabetes mellitus, hypertension, and NT-proBNP, the AST/ALT ratio was a significant independent predictor of LVEF%.

The aspartate transaminase (AST)-to-alanine aminotransferase (ALT) ratio, which is used to measure liver injury, has been found to be associated with some chronic diseases and mortality. AST is released from many tissues, including the myocardium and the liver, while ALT is only released from the liver. Therefore, more severe myocardial pathology would lead to an anticipated increase in the AST/ALT

ratio.[19] AST/ALT ratio was higher in Group 2 (2.08 ± 1.31) compared to Group 1 (0.68 ± 0.21) and this difference was statistically very significant (p=0.001) in the present study. **Yokoyama et al.[78]** recently concluded that an increased AST/ALT ratio positively correlates with NT-proBNP levels. Zoppini et al.[79] studied patients with type 2 diabetes mellitus and found that the AST/ALT ratio positively correlated with CVD mortality; however, as in the above studies, they did not include HFrEF patients. **Ewid M et al[21]** in their study observed that an increased AST/ALT ratio could predict a functional status decline in HFrEF patients. But there are no studies to our knowledge in India, evaluating the predictive validity of AST/ALT ratio in HFrEF.

In the present study from the correlation test, it is observed that there is a significant correlation between the APRI index and FIB-4 with AST/ALT ratio. But there is no significant correlation between EF% with the AST/ALT ratio**.** On diagnostic analysis of the AST/ALT ratio to predict the severity of chronic heart failure with reduced EF, the area under the curve (AUC) was 0.547 (P-value = 0.5654) with a 95% confidence interval of 0.299-0.795 with an optimal cut-off value of 0.6 sensitivity of 96.70% and specificity of 33.33%. In this study, multiple linear regression could not be done as there is no significant correlation between EF% and NYHA class with AST/ALT ratio. **Ewid M et al[21] (2020)** in their study found a mild significant correlation between AST/ALT ratio and APRI, FIB-4 score, NYHA-class, and LVEF (r=0.2, 0.25, 0.26, and −0.24, respectively; P<0.05). The multivariate linear regression analysis model and ROC curve showed that AST/ALT ratio could independently predict HFrEF functional severity with the best cut-off value of 0.9, sensitivity of 43.6%, and specificity of 81.4% in their study. **Liu X et al.[83] (2022)** in their study observed that an elevated AST/ALT ratio is an independent prognostic

factor for All cause mortality in stable coronary artery disease patients. **Liu H et al.[85] (2021)** in their study observed that increased AST/ALT ratio levels were predictive of all-cause and cardiovascular mortality among Chinese hypertensive patients. **Yokoyama M et al.[78] (2016)** in their longitudinal cohort study observed that AST/ALT ratio was associated with an increase in BNP and found cardiovascular mortality was higher in subjects with a high AST/ALT ratio than in those without.

STRENGTH AND LIMITATION OF STUDY

Strength of the study:

AST/ALT ratio is a simple, easy tool to detect the functional severity in chronic heart failure with reduced left ventricular ejection fraction as it is routinely performed, repeatable and feasible investigation. To our knowledge, the study is one of the first of its kind in India in evaluating the role of AST/ALT ratio in HFrEF

Limitations of the study:

The present study is a hospital-based single-center study. The design is also cross-sectional and the sampling methods are not random. In this study, AST/ALT ratio could not be used as a prognostic marker of mortality and morbidity in heart failure and could not correlate with BNP levels.

SUMMARY

1. A total of 100 subjects aged more than 18 years with LVEF of ≤ 40% were included in this hospital-based prospective cross-sectional study to investigate the AST/ALT ratio as an indicator of the functional severity in chronic heart failure with reduced LVEF.
2. The 100 subjects whose age ranges between 19 to 89 years with a mean age of 59.44 ± 14.68 years.
3. Based on the AST/ALT ratio, the study participants were divided into 2 groups - AST/ALT ratio <1 (Group 1) and AST/ALT ratio ≥1 (Group 2).
4. Out of 100 participants, Group 1 had 21 participants and Group 2 had 79 participants. Majority (34%) of the subjects were aged between 61 to 70 years in the present study. There was no statistically significant difference between Group 1 and Group 2 with respect to age groups and mean age.
5. In gender analysis, it is observed that the number of male patients is higher than a female that is 63 males and 37 females. There was a statistically significant difference between Group 1 and Group 2 with respect to gender distribution between the groups.
6. 43% of the subjects had a past history of Type 2 DM while 21% had a history of systemic hypertension.
7. There was no statistically significant difference between Group 1 and Group 2 with respect to the mean duration of dyspnoea.
8. A higher proportion of subjects in Group 1 had orthopnoea (57.14%) compared to Group 2 (22.78%) and this difference was statistically significant. There was no statistically significant difference in other signs and symptoms of HF between Group 1 and Group 2

9. There was no statistically significant difference between groups with respect to vital parameters.
10. There was no statistically significant difference between groups with respect to hemoglobin, WBC count, platelet count, PT-INR, APTT, serum urea, creatinine, sodium, potassium and random blood sugar, and thyroid function test.
11. There was no statistically significant difference in mean LVEF between the groups. The mean LVEF in Group 1 was 33.10± 6.02% compared to 34.57± 4.82% in Group 2.
12. There was no statistically significant difference between groups with respect to total bilirubin, direct bilirubin, or total protein.
13. There was a statistically significant difference between groups with respect to ALT, and AST/ALT ratio. But AST was comparable between the groups (43.71 ± 46.42 in Group 1 vs 74.04 ± 102.44 in Group 2).
14. ALT was higher in Group 1 at 87.05 ± 138.99 compared to 35.63 ± 46.26 in Group 2
15. ALP was higher in Group 1 at 119.67 ± 58.01 compared to 96.19 ± 36.02 in Group 2.
16. AST/ALT ratio was higher in Group 2 (2.08 ± 1.31) compared to Group 1 (0.68 ± 0.21) and this difference was statistically very significant (p=0.001).
17. From the correlation test, it is observed that there is a significant correlation between the APRI index and with AST/ALT ratio. There is a significant correlation between FIB-4 over AST/ALT ratio. There is no significant correlation between EF% with AST/ALT ratio. There is no significant correlation between NYHA class and with AST/ALT ratio.

18. Diagnostic analysis of AST/ALT ratio to predict the severity of chronic heart failure with reduced EF. The area under the curve (AUC) was 0.547 (P-value = 0.5654) with a 95% confidence interval of 0.299 – 0.795 with an optimal cut-off value of 0.6, sensitivity of 96.70%, and specificity of 33.33%.

CONCLUSION

The AST/ALT ratio is increased in patients with chronic heart failure patients with reduced left ventricular ejection fraction. It is a simple predictor of left ventricular dysfunction in patients with heart failure with reduced ejection fraction.

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ANNEXURES 2- CONSENT FORM

ENGLISH/KANNADA/MARATHI/HINDI

INFORMED CONSENT

Dear Mr./Mrs./Dr. _____, you are kindly requested to enroll yourself in a research study tilted, "To study AST/ALT ratio as an indicator of functional severity in chronic heart failure with reduced left ventricular ejection fraction at Dr Prabhakar Kore Hospital and MRC, Belagavi, one year observational cross sectional study **– A one year PROSPECTIVE CROSS SECTIONAL STUDY in KLE's Dr.Prabhakar Kore Hospital and Medical Research Centre, Belagavi"** being conducted by REGISTRATION NO: BG0120017, a post graduate student in M.D. General Medicine and the study will be carried out under the direct supervision and guidance of Dr. _____, Professor, Department of General Medicine, Jawaharlal Nehru Medical College, Belgaum.

You have been requested to participate in this as you fit into the laid-out criteria for a study 'subject'/ participant.

Your participation in study is voluntary. During the study you will be asked some questions and you are supposed to answer to the best of your knowledge. Your decision whether or not to participate in the study will not affect your treatment in any form. If you decide to participate you are free to withdraw at any time.

TITLE OF THE STUDY:

"To study AST/ALT ratio as an indicator of functional severity in chronic heart failure with reduced left ventricular ejection fraction at Dr Prabhakar Kore Hospital and MRC,

Belagavi, one year observational cross sectional study **– A one year PROSPECTIVE CROSS SECTIONAL STUDY in KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi".**

PURPOSE OF THE STUDY:

"To study AST/ALT ratio as an indicator of functional severity in chronic heart failure with reduced left ventricular ejection fraction at Dr Prabhakar Kore Hospital and MRC, Belagavi, one year prospective cross sectional study – A one year Prospective Cross Sectional Study in KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi".

PROCEDURES INVOLVED:

If you agree to enroll yourself in my study, you will be interviewed regarding your present, past and family history then you will be clinically examined in detail and investigated accordingly.

Then you will be subjected to a few investigations namely

Complete blood count

I Haemoglogin(g/dl)

II Platelet (X $10^3/\mu l$)

III WBC (X $10^3/\mu l$)

IV PT –INR, APTT

Liver function test

a. Total bilirubin(mg/dl)

b. Direct bilirubin (mg/dl)

c.Total protein (g/dl)

d.AST(U/L)

e.ALT(U/L)

f.ALP(U/L)

g.Serum Albumin(gm/dl)

II AST/ALT RATIO

III. APRI INDEX

IV. FIB-4 SCORE

RBS (mg/dl)

3)Serum Urea (mg/dl)

4)Serum Creatinine (mg/dl)

5)Serum Sodium(meq/l)

6)Serum Potassium (meq/l)

7)USG Abdomen and pelvis

8)Serum TSH (micu/ml) Free T3 (pg/ml), Free T4 (ng/dl)

9)Echocardiogram

10) Transthoracic Echocardiography

Ejection Fraction %

Valves (Mitral, Aortic, Tricuspid, Pulmonary)

Chambers (Left Ventricle, Right Ventricle, Left Atrium, Right Atrium)

Septae

Great Arteries (Aortal, Pulmonary Artery)

Regional Wall Motion Abnormality

Pericardial Effusion

Clots /Vegetation

CONCLUSION

RISKS AND BENEFITS:

There are no potential risks involved in this study.

Benefits of taking part in this research:

By taking part in this study, AST/ALT ratio will be an independent indicator of functional severity in chronic heart failure with reduced left ventricular ejection fraction of <=40%

VOLUNTARY PARTICIPATION / WITHDRAWAL FROM THE STUDY:

Taking part in the study is voluntary. You may choose not to enroll yourself in this study and may choose to leave the study anytime in between.

ALTERNATIVES:

Your decision regarding participation in study will not change present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. You would simply be excluded from the study if you wish to, and all your details shall be kept confidential and you will get the routine line of management.

PRIVACY AND CONFIDENTIALITY:

All data collected or disclosed by you during the course of participation of study, will be kept fully confidential. If however during the course it becomes necessary for the progress of the course to disclose the identity, it would be done so only after your informed & written consent.

The only people to know that you are a research subject are members of the research team. No information about you will be disclosed to other without your written permission except:

In emergency to protect your rights AND welfare.

If required by law.

AUTHORIZATION TO PUBLISH RESULT:

The results of the study may be used to publish an article. When the results of research published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information obtained in connection with this study and that can be identified with you will remain confidential.

FINANCIAL INCENTIVES FOR PARTICIPATION:

No additional costs shall be incurred upon you for the purpose of this study. It is purely being done with the idea of research and all the cost of study will be borne by the investigator.

COMPENSATION:

In the event that you become injured as a result of taking part in this study, treatment will be offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum, or you will be given information about where to receive medical care. However, no reimbursement, compensation or free medical care will be given.

QUESTIONS/CONTACT DETAILS:

You shall be free to contact the below mentioned name & addresses anytime during the study period for any clarification or help as you may desire for.

In case of the queries during study or in future you may contact following persons,

1. Dr. HARSHA HEDGE,
Head of Ethical Committee for
Human Research
9448113403

2. Dr. _____
MD (GENERAL MEDICINE),
DNB, MNAMS, FGSI
PROFESSOR AND UNIT CHIEF,
Dept of General Medicine,
JNMC, Belagavi.

3. REG.NO: BG0120017
Investigator,
PG in General Medicine,
JNMC, Belagavi.

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has Been read to me, this consent form and have had all the questions answered

Signature / Left Thumb print of the Participant or legally authorized representative

Participant's name :……...

Signature / Left thumb impression :……...

of the participant

Name of the legally authorized :……...

representative / guardian

Signature / Left thumb impression :……...

Witness' name :……...

Signature / Left thumb impression :……...

Investigator's name and signature :……...

Date:

Place:

माहितीपूर्ण संमती

प्रिय श्रीमती / श्रीमती / डॉ. _____, आपणास विनम्र विनंती आहे की आपणास झुकलेल्या एका संशोधन □ भ्यासामध्ये नाव नोंदवावे "डॉ.प्रभाकर कोरे हॉस्पिटल आणि एमआरसी, बेलागावी येथे डाव्या वेंट्रिक्युलर इजेक्शन कमी झाल्यास तीव्र हृदय □ पयशाच्या क्रियात्मक तीव्रतेचे सूचक म्हणून एएसटी / एएलटी गुणोत्तर □ भ्यासण्यासाठी, एक वर्ष वेधशाळा क्रॉस विभागीय □ भ्यास - केएलई मध्ये एक वर्षाचा भावी क्रॉस विभागीय □ भ्यास. " डॉ.प्रभाकर कोरे हॉस्पिटल □ँड मेडिकल रिसर्च सेंटर, बेलागावी". REGISTRATION NO: BG0120017, एम.डी. जनरल मेडिसीन मधील पदव्युत्तर विद्यार्थी घेत आहेत आणि हा □ भ्यास, जवाहरलाल नेहरू मेडिकल कॉलेज, बेळगाव येथील डॉ. _____, सामान्य चिकित्सा विभाग, जवाहरलाल नेहरू मेडिकल कॉलेज, बेळगाव यांच्या थेट देखरेखीखाली आणि मार्गदर्शनानुसार केला जाईल.

आपण □ भ्यासाच्या 'विषय' / सहभागीच्या निकषांनुसार बसत □ सल्यामुळे यामध्ये सहभागी होण्याची विनंती केली गेली आहे.

□ भ्यासात आपला सहभाग ऐच्छिक आहे. □ भ्यासादरम्यान आपल्याला काही प्रश्न विचारले जातील आणि आपल्या सर्वोत्तम उत्तरासाठी आपल्याला उत्तर द्यावे लागेल. □ भ्यासामध्ये भाग घ्यायचा की नाही या निर्णयाचा तुमच्या उपचारांवर कोणत्याही प्रकारचा परिणाम होणार नाही. आपण सहभागी होण्याचे ठरविल्यास आपण कधीही माघार घेण्यास मोकळे आहात.

बेळगावी.

□ भ्यासाचे शीर्षकः

"डॉ.प्रभाकर कोरे हॉस्पिटल आणि एमआरसी, बेलागावी येथे डाव्या वेंट्रिक्युलर इजेक्शन कमी झाल्यास तीव्र हृदय □ पयशाच्या क्रियात्मक तीव्रतेचे सूचक म्हणून एएसटी / एएलटी गुणोत्तर □ भ्यासण्यासाठी, एक वर्ष वेधशाळा क्रॉस विभागीय □ भ्यास - केएलई मध्ये एक वर्षाचा भावी क्रॉस विभागीय □ भ्यास. " डॉ.प्रभाकर कोरे हॉस्पिटल □ँड मेडिकल रिसर्च सेंटर, बेलागावी".

□ भ्यासाचा हेतू:

“डॉ.प्रभाकर कोरे हॉस्पिटल आणि एमआरसी, बेलागावी येथे डाव्या वेंट्रिक्युलर इजेक्शन कमी झाल्यास तीव्र हृदय

□ पयशाच्या क्रियात्मक तीव्रतेचे सूचक म्हणून एएसटी / एएलटी गुणोत्तर □ भ्यासण्यासाठी, एक वर्ष वेधशाळा क्रॉस

विभागीय □ भ्यास - केएलई मध्ये एक वर्षाचा भावी क्रॉस विभागीय □ भ्यास. " डॉ.प्रभाकर कोरे हॉस्पिटल □ँड मेडिकल

रिसर्च सेंटर, बेलागावी".

प्रक्रिया समाविष्ट:

माझ्या □ भ्यासामध्ये आपण स्वतःस नावनोंदणी घेण्यास सहमत □ सल्यास, आपली वैद्यकीयदृष्ट्या सविस्तर तपासणी

केली जाईल आणि त्यानुसार खाली दिलेल्या तपासणीसाठी चौकशी केली जाईल.

मग आपल्यावर काही तपासणी केल्या जातील बहुदा

1. संपूर्ण रक्त गणना

2. यकृत कार्य चाचणी

3. सीरम युरिया

4. सीरम क्रिएटिनिन

5. सीरम सोडियम

6. सीरम पोटॅशियम

7. ट्रान्सस्टोरॅसिक इकोकार्डियोग्राफी

8. इलेक्ट्रोकार्डियोग्राफी

9. थायरॉईड प्रोफाइल (टीएसएच, टी 3, टी 4)

जोखीम आणि फायदे:या □ भ्यासामध्ये कोणतेही संभाव्य धोके गुंतलेले नाहीत.

या संशोधनात भाग घेण्याचे फायदे:

या □ भ्यासामध्ये भाग घेतल्यास, एएसटी / एएलटी गुणोत्तर <= 40% च्या कमी डाव्या वेंट्रिक्युलर इजेक्शन

□ंशानुसार

तीव्र हृदय □ पयशाच्या कार्यशील तीव्रतेचे स्वतंत्र सूचक □ सेल.

ऐच्छिक सहभाग / □ भ्यासामधून पैसे काढणे:

□ भ्यासामध्ये भाग घेणे ऐच्छिक आहे. आपण या □ भ्यासामध्ये स्वत: ची नावनोंदणी न करणे निवडू शकता आणि

दरम्यान □ भ्यास कधीही सोडणे निवडू शकता.

विकल्पः

□ भ्यासात सहभागासंदर्भातील तुमचा निर्णय केएलईएस डॉ. प्रभाकर कोरे हॉस्पिटल आणि वैद्यकीय संशोधन केंद्र,

बेळगाव येथे तुम्हाला देऊ केलेल्या सध्याच्या किंवा भविष्यातील आरोग्य सेवा बदलणार नाही. आपली इच्छा □ सेल तर

आपल्याला □ भ्यासापासून वगळले जाईल आणि आपले सर्व तपशील गोपनीय ठेवले जातील आणि आपल्याला व्यवस्थापनाची नियमित रूंदी मिळेल.

गोपनीयता आणि गोपनीयता:

□ भ्यासाच्या सहभागादरम्यान आपण गोळा केलेला किंवा जाहीर केलेला सर्व डेटा पूर्णपणे गोपनीय ठेवला जाईल. □ र्थात

कोर्सच्या दरम्यान ओळख जाहीर करणे आवश्यक झाले तर ते तुमच्या माहिती व लेखी संमतीनंतरच केले जाईल.

आपण संशोधन विषय आहात हे फक्त लोकांनाच माहित आहे की ते संशोधन पथकाचे सदस्य आहेत. आपल्या लेखी

परवानगीशिवाय इतर आपल्याबद्दल कोणतीही माहिती उघड केली जाणार नाही:

- आपत्कालीन परिस्थितीत आपले हक्क आणि कल्याण यांचे संरक्षण करण्यासाठी.

- कायद्याने आवश्यक □ सल्यास.

निकाल प्रकाशित करण्यासाठी □ धिकृतताः

□ भ्यासाचा निकाल लेख प्रकाशित करण्यासाठी वापरला जाऊ शकतो. जेव्हा एखाद्या संशोधनाचे निकाल

कॉन्फरन्समध्ये प्रकाशित केले जातात किंवा त्यावर चर्चा केली जाते तेव्हा आपली ओळख उघडकीस आणणारी

कोणतीही

माहिती दर्शविली जाणार नाही. या □ भ्यासाच्या संदर्भात प्राप्त केलेली कोणतीही माहिती आणि ती आपल्याशी

ओळखली

जाऊ शकते ती गोपनीय राहील.

सहभागासाठी आर्थिक प्रोत्साहन:

या □ भ्यासाच्या हेतूने आपल्यावर कोणत्याही प्रकारची □ तिरिक्त किंमत आकारली जाणार नाही.

हे निव्वळ संशोधनाच्या कल्पनेने केले जात आहे आणि □ भ्यासाचा सर्व खर्च तपासनीस करेल.

भरपाई:

या □ भ्यासामध्ये भाग घेतल्यामुळे आपण जखमी झाल्यास, केएलईएस डॉ. प्रभाकर कोरे हॉस्पिटल आणि

मेडिकल

रिसर्च सेंटर, बेळगाव येथे तुम्हाला उपचार देण्यात येतील किंवा तुम्हाला वैद्यकीय सेवा कोठून घ्यावी याविषयी

माहिती

दिली जाईल. तथापि, कोणतेही प्रतिपूर्ती, भरपाई किंवा विनामूल्य वैद्यकीय सेवा दिली जाणार नाही.

प्रश्न / संपर्क तपशील:

□ भ्यासाच्या कालावधीत कोणत्याही स्पष्टीकरणासाठी किंवा तुम्हाला पाहिजे □ सलेल्या मदतीसाठी तुम्ही

खाली नमूद

केलेल्या नावाने व पत्त्यांशी कधीही संपर्क साधू शकता.

आपल्याला संशोधन सहभागी म्हणून हक्कांबद्दल काही प्रश्न □ सल्यास आपण संपर्क साधू शकता

डॉ. हर्षा हेगडे

चे□ रपर्सन, मानवी विषयांसाठी संशोधन संस्थाची नीतिशास्त्र समिती

काहेर, जे.एन. मेडिकल कॉलेज, बेलागावी - 590010

संपर्क क्रमांक : 9480422500

डॉ. _____ (सामान्य चिकित्सा), डीएनबी, एमएनएएमएस, एफजीएसआय

प्राध्यापक आणि युनिट चीफ,

सामान्य औषध विभाग,

जे.एन.एम.सी, बेळगावी.

मोबाइल - 9448231298

REGISTRATION NO: BG0120017

□ न्वेषक, पदव्युत्तर विद्यार्थी

सामान्य औषध विभाग,

जे.एन.एम.सी, बेळगावी.

मोबाइल - 7019980017S

संमती फॉर्म

मी खाली स्वाक्षरी करून या □ भ्यासात भाग घेण्यास स्वेच्छेने सहमत आहे. मी कधीही माघार घेऊ शकतो. या फॉर्मवर

सही करून मी माझा कोणताही कायदेशीर हक्क सोडत नाही. खाली माझी स्वाक्षरी सूचित करते की मी हा संमती फॉर्म

वाचला आहे किंवा हा संमती फॉर्म मला वाचला आहे आणि मला सर्व प्रश्नांची उत्तरे दिली आहेत

सहभागी किंवा कायदेशीररित्या □ धिकृत प्रतिनिधीची सही / डावा □ंगठा प्रिंट

सहभागीचे नाव: ....

स्वाक्षरी / डावा □ंगठा ठसा: ....

सहभागीचा

कायदेशीररीत्या □ धिकृत नाव: ....

प्रतिनिधी / पालक

स्वाक्षरी / डावा □ंगठा ठसा: ....

साक्षीचे नाव: ....

स्वाक्षरी / डावा □ंगठा ठसा: ....

□ न्वेषकांचे नाव आणि स्वाक्षरीः ....

तारीख:

ठिकाणः

ತಿಳುವಳಿಕೆಯ ಸಮ್ಮತಿ

ಆತ್ಮೀಯ ಶ್ರೀ / ಶ್ರೀ. / ಡಾ. _____, ಓರೆಯಾಗಿರುವ ಸಂಶೋಧನಾ

ಅಧ್ಯಯನಕ್ಕೆ ನಿಮ್ಮನ್ನು ಸೇರಿಸಲು ನಿಮ್ಮನ್ನು ದಯೆಯಿಂದ ವಿನಂತಿಸಲಾಗಿದೆ, "ಎ ಎಸ್ ಟಿ / ಎ ಎಲ್ ಟಿ ಅನುಪಾತವನ್ನು ದೀರ್ಘಕಾಲದ ಹೃದಯ ವೈಫಲ್ಯದ ಕ್ರಿಯಾತ್ಮಕ ತೀವ್ರತೆಯ ಸೂಚಕವಾಗಿ ಡಾ. ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಎಂಆರ್ಸಿ ಬೆಳಗಾವಿಯಲ್ಲಿ ಒಂದು ವರ್ಷದ ವೀಕ್ಷಣಾ ಅಡ್ಡ ವಿಭಾಗದ ಅಧ್ಯಯನ. – ಒಂದು ವರ್ಷದ ಅವಲೋಕನ ಅಡ್ಡ ವಿಭಾಗೀಯ ಅಧ್ಯಯನ - ಕೆಎಲ್ಇಯ ಡಾ. .ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಮತ್ತು ವೈದ್ಯಕೀಯ ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಬೆಳಗಾವಿ " ಎಂ.ಡಿ. ಜನರಲ್ ಮೆಡಿಸಿನ್‌ನಲ್ಲಿ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ REGISTRATION NO: BG0120017, ಅವರು ನಡೆಸಲಿದ್ದಾರೆ ಮತ್ತು ಬೆಳಗಾವಿನ ಜವಾಹರಲಾಲ್ ನೆಹರು ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿನ ಜನರಲ್ ಮೆಡಿಸಿನ್ ವಿಭಾಗದ ಪ್ರೊಫೆಸರ್ ಡಾ. _____ ಅವರ ನೇರ ಮೇಲ್ವಿಚಾರಣೆ ಮತ್ತು ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ಈ ಅಧ್ಯಯನವನ್ನು ನಡೆಸಲಾಗುವುದು.

ಅಧ್ಯಯನದ 'ವಿಷಯ' / ಭಾಗವಹಿಸುವವರಿಗೆ ನೀವು ನಿಗದಿಪಡಿಸಿದ ಮಾನದಂಡಗಳಿಗೆ ಸರಿಹೊಂದುವಂತೆ ಇದರಲ್ಲಿ ಭಾಗವಹಿಸಲು

ನಿಮ್ಮನ್ನು ವಿನಂತಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನಿಮ್ಮನ್ನು ಕೆಲವು ಪ್ರಶ್ನೆಗಳನ್ನು

ಕೇಳಲಾಗುತ್ತದೆ ಮತ್ತು ನಿಮ್ಮ ಉತ್ತಮ ಜ್ಞಾನಕ್ಕೆ ನೀವು ಉತ್ತರಿಸಬೇಕಾಗುತ್ತದೆ. ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಬೇಕೆ ಅಥವಾ ಬೇಡವೇ ಎಂಬ

ನಿಮ್ಮ ನಿರ್ಧಾರವು ನಿಮ್ಮ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಯಾವುದೇ ರೂಪದಲ್ಲಿ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ನಿರ್ಧರಿಸಿದರೆ ನೀವು

ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂತೆಗೆದುಕೊಳ್ಳಬಹುದು.

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ:

"ಎ ಎಸ್ ಟಿ / ಎ ಎಲ್ ಟಿ ಅನುಪಾತವನ್ನು ದೀರ್ಘಕಾಲದ ಹೃದಯ ವೈಫಲ್ಯದ ಕ್ರಿಯಾತ್ಮಕ ತೀವ್ರತೆಯ

ಸೂಚಕವಾಗಿ ಡಾ. ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಎಂಆರ್ಸಿ ಬೆಳಗಾವಿಯಲ್ಲಿ ಒಂದು ವರ್ಷದ ವೀಕ್ಷಣಾ ಅಡ್ಡ

ವಿಭಾಗದ ಅಧ್ಯಯನ. - ಒಂದು ವರ್ಷದ ಅವಲೋಕನ ಅಡ್ಡ ವಿಭಾಗೀಯ ಅಧ್ಯಯನ - ಕೆಎಲ್ಇಯ ಡಾ. .ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಮತ್ತು ವೈದ್ಯಕೀಯ ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಬೆಳಗಾವಿ "

ಅಧ್ಯಯನದ ಉದ್ದೇಶ:

"ಎ ಎಸ್ ಟಿ / ಎ ಎಲ್ ಟಿ ಅನುಪಾತವನ್ನು ದೀರ್ಘಕಾಲದ ಹೃದಯ ವೈಫಲ್ಯದ ಕ್ರಿಯಾತ್ಮಕ ತೀವ್ರತೆಯ

ಸೂಚಕವಾಗಿ ಡಾ. ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಎಂಆರ್ಸಿ ಬೆಳಗಾವಿಯಲ್ಲಿ ಒಂದು ವರ್ಷದ ವೀಕ್ಷಣಾ ಅಡ್ಡ

ವಿಭಾಗದ ಅಧ್ಯಯನ. - ಒಂದು ವರ್ಷದ ಅವಲೋಕನ ಅಡ್ಡ ವಿಭಾಗೀಯ ಅಧ್ಯಯನ - ಕೆಎಲ್ಇಯ ಡಾ. .ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಮತ್ತು ವೈದ್ಯಕೀಯ ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಬೆಳಗಾವಿ "

ಒಳಗೊಂಡಿರುವ ಕಾರ್ಯವಿಧಾನಗಳು:

ನನ್ನ ಅಧ್ಯಯನಕ್ಕೆ ನಿಮ್ಮನ್ನು ಸೇರಿಸಲು ನೀವು ಒಪ್ಪಿದರೆ, ನಿಮ್ಮನ್ನು ಪ್ರಾಯೋಗಿಕವಾಗಿ ವಿವರವಾಗಿ ಪರಿಶೀಲಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಅದರ

ಪ್ರಕಾರ ಈ ಕೆಳಗಿನ ತನಿಖೆಗಳಿಗಾಗಿ ತನಿಖೆ ಮಾಡಲಾಗುತ್ತದೆ.

ನಂತರ ನಿಮ್ಮನ್ನು ಕೆಲವು ತನಿಖೆಗಳಿಗೆ ಒಳಪಡಿಸಲಾಗುತ್ತದೆ

1. ಸಂಪೂರ್ಣ ರಕ್ತದ ಎಣಿಕೆ

2. ಪಿತ್ತಜನಕಾಂಗದ ಕಾರ್ಯ ಪರೀಕ್ಷೆ

3. ಸೀರಮ್ ಯೂರಿಯಾ

4. ಸೀರಮ್ ಕ್ರಿಯೇಟಿನೈನ್

5. ಸೀರಮ್ ಸೋಡಿಯಂ

6. ಸೀರಮ್ ಪೊಟ್ಯಾಸಿಯಮ್

7. ಟ್ರಾನ್ಸ್ಥೊರಾಸಿಕ್ ಎಕೋಕಾರ್ಡಿಯೋಗ್ರಫಿ

8. ಎಲೆಕ್ಟ್ರೋಕಾರ್ಡಿಯೋಗ್ರಫಿ

9. ಥೈರಾಯ್ಡ್ ವಿವರ (ಟಿಎಸ್ಹೆಚ್, ಟಿ 3, ಟಿ 4)

ಅಪಾಯ ಮತ್ತು ಪ್ರಯೋಜನಗಳು:

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಯಾವುದೇ ಸಂಭಾವ್ಯ ಅಪಾಯಗಳಿಲ್ಲ.

ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವ ಪ್ರಯೋಜನಗಳು:

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವ ಮೂಲಕ, ಎಎಸ್ಟಿ / ಎಎಲ್ಟಿ ಅನುಪಾತವು ದೀರ್ಘಕಾಲದ ಹೃದಯ ವೈಫಲ್ಯದಲ್ಲಿ ಕ್ರಿಯಾತ್ಮಕ ತೀವ್ರತೆಯ

ಸ್ವತಂತ್ರ ಸೂಚಕವಾಗಿರುತ್ತದೆ </ 40% ನಷ್ಟು ಕಡಿಮೆ ಎಡ ಕುಹರದ ಎಜೆಕ್ಷನ್ ಭಾಗದೊಂದಿಗೆ

ಸ್ವಯಂಪ್ರೇರಿತ ಭಾಗವಹಿಸುವಿಕೆ / ಅಧ್ಯಯನದಿಂದ ಹಿಂತೆಗೆದುಕೊಳ್ಳುವಿಕೆ:

ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದು ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ಈ ಅಧ್ಯಯನಕ್ಕೆ ನಿಮ್ಮನ್ನು ಸೇರಿಸಿಕೊಳ್ಳದಿರಲು ನೀವು ಆಯ್ಕೆ ಮಾಡಬಹುದು

ಮತ್ತು ಈ ನಡುವೆ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನವನ್ನು ಬಿಡಲು ಆಯ್ಕೆ ಮಾಡಬಹುದು.

ಪರ್ಯಾಯಗಳು:

ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವ ಬಗ್ಗೆ ನಿಮ್ಮ ನಿರ್ಧಾರವು ಕೆಎಲ್ಇಎಸ್ ಡಾ. ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಬೆಳಗಾವಿ ವೈದ್ಯಕೀಯ

ಸಂಶೋಧನಾ ಕೇಂದ್ರದಲ್ಲಿ ನಿಮಗೆ ನೀಡುತ್ತಿರುವ ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಆರೋಗ್ಯ ಸೇವೆಗಳನ್ನು ಬದಲಾಯಿಸುವುದಿಲ್ಲ. ನೀವು

ಬಯಸಿದರೆ ನಿಮ್ಮನ್ನು ಅಧ್ಯಯನದಿಂದ ಹೊರಗಿಡಲಾಗುವುದು, ಮತ್ತು ನಿಮ್ಮ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಮತ್ತು ನೀವು

ವಾಡಿಕೆಯ ನಿರ್ವಹಣೆಯನ್ನು ಪಡೆಯುತ್ತೀರಿ.

ಗೌಪ್ಯತೆ ಮತ್ತು ಗೌಪ್ಯತೆ:

ಅಧ್ಯಯನದ ಭಾಗವಹಿಸುವಿಕೆಯ ಸಮಯದಲ್ಲಿ ನೀವು ಸಂಗ್ರಹಿಸಿದ ಅಥವಾ ಬಹಿರಂಗಪಡಿಸಿದ ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಸಂಪೂರ್ಣವಾಗಿ

ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ. ಕೋರ್ಸ್ ಸಮಯದಲ್ಲಿ ಪ್ರಗತಿಗೆ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸುವುದು ಅಗತ್ಯವಿದ್ದರೆ, ನಿಮ್ಮ ಮಾಹಿತಿ ಮತ್ತು

ಲಿಖಿತ ಒಪ್ಪಿಗೆಯ ನಂತರವೇ ಇದನ್ನು ಮಾಡಲಾಗುತ್ತದೆ.

ನೀವು ಸಂಶೋಧನಾ ವಿಷಯ ಎಂದು ತಿಳಿದುಕೊಳ್ಳುವ ಏಕೈಕ ಜನರು ಸಂಶೋಧನಾ ತಂಡದ ಸದಸ್ಯರು.

ನಿಮ್ಮ ಲಿಖಿತ

ಅನುಮತಿಯಿಲ್ಲದೆ ನಿಮ್ಮ ಬಗ್ಗೆ ಯಾವುದೇ ಮಾಹಿತಿಯನ್ನು ಇತರರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ:

- ನಿಮ್ಮ ಹಕ್ಕುಗಳು ಮತ್ತು ಕಲ್ಯಾಣವನ್ನು ರಕ್ಷಿಸಲು ತುರ್ತು ಪರಿಸ್ಥಿತಿಯಲ್ಲಿ.

- ಕಾನೂನಿನ ಪ್ರಕಾರ ಅಗತ್ಯವಿದ್ದರೆ.

ಫಲಿತಾಂಶಗಳನ್ನು ಪ್ರಕಟಿಸಲು ಅಧಿಕಾರ:

ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳನ್ನು ಲೇಖನವನ್ನು ಪ್ರಕಟಿಸಲು ಬಳಸಬಹುದು. ಸಂಶೋಧನೆಯ ಫಲಿತಾಂಶಗಳು ಪ್ರಕಟವಾದ ಅಥವಾ

ಚರ್ಚಿಸಿದಾಗ, ಸಮ್ಮೇಳನದಲ್ಲಿ, ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸುವ ಯಾವುದೇ ಮಾಹಿತಿಯನ್ನು ಪ್ರದರ್ಶಿಸಲಾಗುವುದಿಲ್ಲ. ಈ

ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ಪಡೆದ ಯಾವುದೇ ಮಾಹಿತಿಯು ಮತ್ತು ಅದನ್ನು ನಿಮ್ಮೊಂದಿಗೆ ಗುರುತಿಸಬಹುದು.

ಭಾಗವಹಿಸುವಿಕೆಗೆ ಆರ್ಥಿಕ ಪ್ರೋತ್ಸಾಹ:

ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ಯಾವುದೇ ಹೆಚ್ಚುವರಿ ವೆಚ್ಚಗಳು ನಿಮ್ಮ ಮೇಲೆ ಆಗುವುದಿಲ್ಲ.

ಇದನ್ನು ಸಂಪೂರ್ಣವಾಗಿ ಸಂಶೋಧನೆಯ ಆಲೋಚನೆಯೊಂದಿಗೆ ಮಾಡಲಾಗುತ್ತಿದೆ ಮತ್ತು ಅಧ್ಯಯನದ ಎಲ್ಲಾ ವೆಚ್ಚವನ್ನು ತನಿಖಾಧಿಕಾರಿ ಭರಿಸುತ್ತಾರೆ.

ಪರಿಹಾರ:

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಂಡ ಪರಿಣಾಮವಾಗಿ ನೀವು ಗಾಯಗೊಂಡರೆ ಬೆಳಗಾವಿನ ಕೆಎಲ್ಇಎಸ್ ಡಾ. ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ
ಮತ್ತು ವೈದ್ಯಕೀಯ ಸಂಶೋಧನಾ ಕೇಂದ್ರದಲ್ಲಿ ನಿಮಗೆ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗುವುದು ಅಥವಾ ವೈದ್ಯಕೀಯ ಆರೈಕೆಯನ್ನು ಎಲ್ಲಿ ಪಡೆಯಬೇಕು
ಎಂಬ ಬಗ್ಗೆ ನಿಮಗೆ ಮಾಹಿತಿ ನೀಡಲಾಗುವುದು. ಆದಾಗ್ಯೂ, ಯಾವುದೇ ಮರುಪಾವತಿ, ಪರಿಹಾರ ಅಥವಾ ಉಚಿತ ವೈದ್ಯಕೀಯ
ಸೌಲಭ್ಯವನ್ನು ನೀಡಲಾಗುವುದಿಲ್ಲ.

ಪ್ರಶ್ನೆಗಳು / ಸಂಪರ್ಕ ವಿವರಗಳು:

ನೀವು ಬಯಸಿದಂತೆ ಯಾವುದೇ ಸ್ಪಷ್ಟೀಕರಣ ಅಥವಾ ಸಹಾಯಕ್ಕಾಗಿ ಅಧ್ಯಯನದ ಅವಧಿಯಲ್ಲಿ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಈ ಕೆಳಗಿನ
ಹೆಸರು ಮತ್ತು ವಿಳಾಸಗಳನ್ನು ಸಂಪರ್ಕಿಸಲು ನೀವು ಮುಕ್ತರಾಗಿರಬೇಕು.

ಸಂಶೋಧನಾ ಪಾಲ್ಗೊಳ್ಳುವವರಾಗಿ ನೀವು ಹಕ್ಕುಗಳ ಬಗ್ಗೆ ಯಾವುದೇ ಪ್ರಶ್ನೆಗಳನ್ನು ಹೊಂದಿದ್ದರೆ ನೀವು ಸಂಪರ್ಕಿಸಬಹುದು

ಡಾ. ಹರ್ಷ ಹೆಗ್ಡೆ, ಅಧ್ಯಕ್ಷರು,
ಮಾನವ ವಿಷಯಗಳ ಸಂಶೋಧನೆಗಾಗಿ ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿ,
ಕಾಹೇರ್, ಜೆ.ಎನ್. ವೈದ್ಯಕೀಯ ಕಾಲೇಜು, ಬೆಳಗಾವಿ - 590010
ದೂರವಾಣಿ ಸಂಖ್ಯೆ: 9480422500

ಡಾ_____ ಎಂಡಿ (ಜೆನೆರಲ್ ಮೆಡಿಸಿನ್), ಡಿಎನ್‌ಬಿ, ಎಂಎನ್ಎಎಂಎಸ್, ಎಫ್‌ಜಿಎಸ್‌ಐ
ಪ್ರೊಫೆಸರ್ ಮತ್ತು ಯುನಿಟ್ ಚೀಫ್,
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜೆಎನ್ಎಂಸಿ, ಬೆಳಗಾವಿ.

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ತನಿಖಾಧಿಕಾರಿ, ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜೆಎನ್ಎಂಸಿ, ಬೆಳಗಾವಿ.

ಒಪ್ಪಿಗೆ ಪತ್ರ

ಕೆಳಗೆ ಸಹಿ ಮಾಡುವ ಮೂಲಕ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪುತ್ತೇನೆ. ನಾನು ಯಾವುದೇ

ಸಮಯದಲ್ಲಿ ಹಿಂತೆಗೆದುಕೊಳ್ಳಬಹುದು. ಈ ಫಾರ್ಮ್ಗೆ ಸಹಿ ಮಾಡುವ ಮೂಲಕ ನಾನು ನನ್ನ ಯಾವುದೇ ಕಾನೂನು ಹಕ್ಕುಗಳನ್ನು

ಬಿಟ್ಟುಕೊಡುತ್ತಿಲ್ಲ. ಕೆಳಗಿನ ನನ್ನ ಸಹಿ ನಾನು ಈ ಒಪ್ಪಿಗೆಯ ಫಾರ್ಮ್ ಅನ್ನು ಓದಿದ್ದೇನೆ ಅಥವಾ ಈ ಸಮ್ಮತಿಯ ಫಾರ್ಮ್ ಅನ್ನು ನನಗೆ

ಓದಿದ್ದೇನೆ ಮತ್ತು ಎಲ್ಲಾ ಪ್ರಶ್ನೆಗಳಿಗೆ ಉತ್ತರಿಸಿದೆ ಎಂದು ಸೂಚಿಸುತ್ತದೆ

ಭಾಗವಹಿಸುವವರ ಅಥವಾ ಕಾನೂನುಬದ್ಧವಾಗಿ ಅಧಿಕೃತ ಪ್ರತಿನಿಧಿಯ ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಮುದ್ರಣ

ಭಾಗವಹಿಸುವವರ ಹೆಸರು:

…… …

ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: …… …

ಭಾಗವಹಿಸುವವರ ಕಾನೂನುಬದ್ಧವಾಗಿ ಅಧಿಕಾರ ಪಡೆದವರ ಹೆಸರು: …… …

ಪ್ರತಿನಿಧಿ / ರಕ್ಷಕ ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: …… …

ಸಾಕ್ಷಿ ಹೆಸರು: …… …

ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: …… …

ತನಿಖಾಧಿಕಾರಿ ಹೆಸರು ಮತ್ತು ಸಹಿ: …… …

ದಿನಾಂಕ:

प्रिय श्री / श्रीमती / डॉ. _____, आपसे विनम्र अनुरोध है कि आप

स्वयं को एक शोध अध्ययन में नामांकित करें, "डॉ. प्रभाकर कोरे अस्पताल और एम आर सी, बेलगावी में कम बाएं वेंट्रिकुलर इजेक्शन अंश के साथ क्रॉनिक हार्ट फेल्योर के एक संकेतक के रूप में एएसटी / एएलटी अनुपात का एक वर्ष का अवलोकन पार अनुभागीय अध्ययन - केएलई के डॉ प्रभाकर कोरे अस्पताल और चिकित्सा अनुसंधान केंद्र, बेलगावी,में एक साल का प्रॉस्पेक्टिव क्रॉस अनुभागीय अध्ययन". एमडी जनरल मेडिसिन में स्नातकोत्तर छात्र डॉ. REGISTRATION NO: BG0120017 द्वारा संचालित किया जा रहा है और यह अध्ययन डॉ. _____, प्रोफेसर, जनरल मेडिसिन विभाग, जवाहरलाल नेहरू मेडिकल कॉलेज, बेलगाम के प्रत्यक्ष पर्यवेक्षण और मार्गदर्शन में किया जाएगा. आपसे यह अनुरोध किया गया है कि आप इसमें एक अध्ययन 'विषय' / प्रतिभागी के निर्धारित मानदंडों में फिट हों। अध्ययन में आपकी भागीदारी स्वैच्छिक है। अध्ययन के दौरान आपसे कुछ प्रश्न पूछे जाएंगे और आप अपने ज्ञान का

सबसे अच्छा जवाब देने वाले हैं। अध्ययन में भाग लेने या न लेने का आपका निर्णय किसी भी रूप में आपके उपचार को प्रभावित नहीं करेगा। यदि आप भाग लेने का निर्णय लेते हैं तो आप किसी भी समय वापस लेने के लिए स्वतंत्र हैं।

अध्ययन का शीर्षक:

"डॉ. प्रभाकर कोरे अस्पताल और एम आर सी, बेलगावी में कम बाएं वेंट्रिकुलर इजेक्शन अंश के साथ
क्रॉनिक हार्ट फेल्योर के एक संकेतक के रूप में एएसटी / एएलटी अनुपात का एक वर्ष का अवलोकन
पार अनुभागीय अध्ययन - केएलई के डॉ प्रभाकर कोरे अस्पताल और चिकित्सा अनुसंधान केंद्र,
बेलगावी,में एक साल का प्रॉस्पेक्टिव क्रॉस अनुभागीय अध्ययन".

अध्ययन का उद्देश्य:

"डॉ. प्रभाकर कोरे अस्पताल और एम आर सी, बेलगावी में कम बाएं वेंट्रिकुलर इजेक्शन अंश के साथ क्रॉनिक हार्ट फेल्योर

के एक संकेतक के रूप में एएसटी / एएलटी अनुपात का एक वर्ष का अवलोकन पार अनुभागीय अध्ययन - केएलई के डॉ

प्रभाकर कोरे अस्पताल और चिकित्सा अनुसंधान केंद्र, बेलगावी,में एक साल का प्रॉस्पेक्टिव क्रॉस अनुभागीय अध्ययन".

शामिल प्रक्रियाएं:

यदि आप मेरे अध्ययन में खुद को नामांकित करने के लिए सहमत हैं, तो आपको अपने वर्तमान, अतीत और परिवार के इतिहास के बारे में साक्षात्कार दिया जाएगा, फिर आपको नैदानिक रूप से जांच की जाएगी और तदनुसार जांच की जाएगी।

फिर आपको कुछ जांचों के अधीन किया जाएगा

1. पूर्ण रक्त गणना

2. लिवर फंक्शन टेस्ट

3. सीरम यूरिया

4. सीरम क्रिएटिनिन

5. सीरम सोडियम

6. सीरम पोटासियम

7. ट्रान्सथोरासिक इकोकार्डियोग्राफी

8. इलेक्ट्रोकार्डियोग्राफी

9. थायरॉयड प्रोफाइल (टीएसएच, टी 3, टी 4)

जोखिम और लाभ:

इस अध्ययन में कोई संभावित जोखिम शामिल नहीं हैं।

इस शोध में भाग लेने के लाभ:

इस अध्ययन में भाग लेने से, एएसटी / एएलटी अनुपात कम बाएं वेंट्रिकुलर इजेक्शन अंश के साथ क्रोनिक हार्ट विफलता में कार्यात्मक गंभीरता का एक स्वतंत्र संकेतक होगा = 40%

अध्ययन से स्वैच्छिक भागीदारी / निकासी:

अध्ययन में भाग लेना स्वैच्छिक है। आप इस अध्ययन में खुद को नामांकित नहीं करना चुन सकते हैं और बीच में कभी भी अध्ययन छोड़ने का विकल्प चुन सकते हैं।

विकल्पः

अध्ययन में भाग लेने के बारे में आपका निर्णय के एल ई एस डॉ। प्रभाकर कोरे अस्पताल और चिकित्सा अनुसंधान केंद्र,

बेलगाम में आपके लिए पेश की गई वर्तमान या भविष्य की स्वास्थ्य देखभाल सेवाओं को नहीं बदलेगा। यदि आप चाहें, तो

आपको अध्ययन से बाहर रखा जाएगा और आपके सभी विवरणों को गोपनीय रखा जाएगा और आपको प्रबंधन की नियमित लाइन मिल जाएगी।

गोपनीयता और गोपनीयता:

अध्ययन की भागीदारी के दौरान आपके द्वारा एकत्र या प्रकट किए गए सभी डेटा को पूरी तरह से गोपनीय रखा जाएगा। हालांकि यदि पाठ्यक्रम के दौरान यह आवश्यक है कि पाठ्यक्रम की प्रगति के लिए पहचान का खुलासा करना आवश्यक

है, तो यह आपकी सूचना और लिखित सहमति के बाद ही किया जाएगा।

केवल यह जानने के लिए कि आप एक शोध विषय हैं, अनुसंधान टीम के सदस्य हैं। आपके लिखित अनुमति के बिना आपके बारे में कोई भी जानकारी का खुलासा नहीं किया जाएगा:

- अपने अधिकारों और कल्याण की रक्षा के लिए आपातकाल में।

- यदि कानून द्वारा आवश्यक हो।

परिणाम प्रकाशित करने के लिए प्राधिकरण:

अध्ययन के परिणामों का उपयोग एक लेख प्रकाशित करने के लिए किया जा सकता है। जब एक सम्मेलन में प्रकाशित या

चर्चा की गई शोध के परिणाम, कोई भी जानकारी प्रदर्शित नहीं की जाएगी जो आपकी पहचान का खुलासा करेगी। इस अध्ययन के संबंध में प्राप्त की गई कोई भी जानकारी और जिसे आप के साथ पहचाना जा सकता है, गोपनीय रहेगी।

भागीदारी के लिए वित्तीय प्रोत्साहन:

इस अध्ययन के उद्देश्य से आपके ऊपर कोई अतिरिक्त लागत नहीं लगेगी।

यह विशुद्ध रूप से अनुसंधान के विचार के साथ किया जा रहा है और अध्ययन का सारा खर्च अन्वेषक

द्वारा वहन किया जाएगा।

भरपाई :

इस अध्ययन में भाग लेने के परिणामस्वरूप आप घायल हो जाते हैं, तो केएलईएस डॉ। प्रभाकर कोरे अस्पताल और चिकित्सा अनुसंधान केंद्र, बेलगाम में उपचार की पेशकश की जाएगी, या आपको चिकित्सा देखभाल कहाँ प्राप्त होगी,

इसके बारे में जानकारी दी जाएगी। हालांकि, कोई प्रतिपूर्ति, मुआवजा या मुफ्त चिकित्सा देखभाल नहीं दी जाएगी।

अध्ययन के दौरान या भविष्य में प्रश्नों के मामले में आप निम्नलिखित व्यक्तियों से संपर्क कर सकते हैं,

प्रश्न / संपर्क विवरण:

आप किसी भी स्पष्टीकरण या मदद के लिए अध्ययन अवधि के दौरान किसी भी समय नीचे दिए गए नाम और पते से संपर्क करने के लिए स्वतंत्र हो सकते हैं।

डॉ. हर्षा हेगड़े,

अध्यक्ष, मानव विषय के अनुसंधान संस्थागत आचार समिति,

फॉरेंसिक मेडिसिन विभाग, काहेर, जे.एन.

मेडिकल कॉलेज, बेलागवी - 590010

संपर्क नंबर: 9480422500

डॉ. _____ (जनरल मीडिया), डीएनबी, एमएनएएमएस, एफजीएसआई

प्रोफेसर और यूनिट मुख्य,

सामान्य चिकित्सा विभाग,

जे.एन.एम.सी, बेळगावी.

मोबाइल :9448231298

REGISTRATION NO: BG0120017

अन्वेषक, स्नातकोत्तर छात्र

सामान्य चिकित्सा विभाग,

जे.एन.एम.सी, बेळगावी.

मोबाइल :9448231298

सहमति पत्र

मैं स्वेच्छा से नीचे हस्ताक्षर करके इस अध्ययन में भाग लेने के लिए सहमत हूं। मैं किसी भी समय वापस ले सकता हूं।

मैं

इस फॉर्म पर हस्ताक्षर करके अपने किसी भी कानूनी अधिकार को नहीं छोड़ रहा हूं। नीचे दिए गए मेरे हस्ताक्षर से

संकेत

मिलता है कि मैंने इस सहमति फॉर्म को पढ़ा है, या यह मेरे लिए पढ़ा गया है, यह सहमति फॉर्म और उत्तर दिए गए प्रश्नों

के

उत्तर हैं

तिभागी या कानूनी रूप से अधिकृत प्रतिनिधि का हस्ताक्षर / बायाँ अंगू□ा प्रिंट

प्रतिभागी का नाम: ....।

हस्ताक्षर / बाएं अंगू□ा का निशान: ....।

प्रतिभागी का

कानूनी रूप से अधिकृत का नाम: ....।

प्रतिनिधि / अभिभावक

हस्ताक्षर / बाएं अंगू□ा का निशान: ....।

साक्षी का नाम: ....।

हस्ताक्षर / बाएं अंगू□ा का निशान: ....।

अन्वेषक का नाम और हस्ताक्षर: ....।

दिनांक:

जगह

To study AST/ALT ratio as an indicator of functional severity in chronic heart failure with EF <= 40%.

PROFORMA

CASE NO:

NAME: AGE/SEX:

IP NO. OCCUPATION

ADDRESS:

PRESENTING COMPLAINT:

☐ COUGH ☐ ORTHOPNEA ☐ PEDAL OEDEMA

☐ DYSPNEA ☐ PAROXYSMAL NOCTURNAL DYSPNEA

☐ ABDOMINAL DISTENSION

Past history:

Family history

Personal history

Treatment history

GENERAL PHYSICAL EXAMINATION:

PALLOR- YES/NO

ICTERUS-YES/NO

LYMPHADENOPATHY-YES/NO

CYANOSIS- YES/NO

CLUBBING-YES/NO

EDEMA-YES/NO

VITALS:

PULSE

RESPIRATORY RATE

BLOOD PRESSURE

TEMPERATURE

JVP (cm of H_2O)

SYSTEMIC EXAMINATION:

C.V.S.: ☐ S3 ☐ S4

R.S:

CREPITATION ☐

C.N.S.:

P.A.:

ASCITES ☐ HEPATOMEGALY ☐

INVESTIGATIONS

1) Complete Blood Count

I Haemoglobin(G/Dl)

II Platelets (X $10^3/\mu l$)

III WBC (X $10^3/\mu l$)

IV. PT –INR, APTT

2) Liver Function Test

a. Total Bilirubin (mg/dl)

b. Direct Bilirubin (mg/dl)

c. Total Protein(g/dl)

d.AST(U/L)

e.ALT(U/L)

f.ALP(U/L)

g. Albumin(gm/dl)

II AST/ALT RATIO

III. APRI INDEX

IV. FIB-4 SCORE

RBS (mg/dl)

3)Serum. Urea (mg/dl)

4)Serum Creatinine (mg/dl)

5)Serum Sodium (meq/l)

6)Serum Potassium (meq/l)

7) USG Abdomen and Pelvis

8)Serum Tsh (micu/ml) FREE T3 (pg/ml), Free T4 (ng/dl)

9) Echocardiography

10) Transthoracic Echocardiography

- Ejection Fraction %
- Valves (Mitral, Aortic, Tricuspid, Pulmonary)
- Chambers (Left Ventricle, Right Ventricle, Left Atrium, Right Atrium)
- Septae
- Great Arteries (Aortal, Pulmonary Artery)
- Regional Wall Motion Abnormality
- Pericardial Effusion
- Clots /Vegetation

CONCLUSION

MASTER CHART

To study AST/ALT ratio as an indicator of functional severity in chronic heart failure with EF <= 40%

				Chief Complaint							Vitals				
SL.NO	IP No.	AGE	Sex	Cough	Orthopnea	Dyspnea	Paroxysmal nocturnal dyspnea	Abdominal distension	Icterus	Edema	Pulse	BP	JVP(cm of H2o)	respiratory sytsem	PA
1	1083723	79	F	-	15 days	3 years		10 days	No	Yes B/L grade III	98	120/80	12	B/L Coarse Crepitation	Uniformally distended, Non tender
2	1138927	54	M	-	-	4 yrs	-	-	No	No	76	110/70	10	B/L Normal vesicular breath sounds	Soft non tender
3	1109441	60	M	-	-	2 yrs	15 Days	-	No	No	83	120/80	10	B/L Normal vericular breath sounds	Soft non tender
4	1092384	57	M	-	-	2 yrs	-	-	No	No	86	120/80	8	B/L Coarse Crepitation	Soft non tender
5	1041447	89	F	-	7 days	4 yrs	-	-	No	yes B/L grade II	62	140/60	10	B/L min Crepitation	Soft non tender
6	1067809	65	M	-	-	2 yrs	-	-	No	yes B/L grade I	79	110/70	10	B/L Coarse Crepitation	Soft non tender
7	1079816	66	M	-	-	4 yrs	-	-	No	yes B/L grade II	66	90/60	9	B/L Normal vericular breath sounds	Soft non tender
8	1127697	23	F	-	-	1yr	-	10 days	No	yes B/L grade II	94	120/70	10	B/L Coarse Crepitation	Soft non tender
9	1077143	53	F	-	-	2 yrs	-	-	No	yes B/L grade II	84	90/60	10	B/L NVBS	Soft non tender
10	993513	73	F	-	-	3 yrs	-	-	No	yes B/L grade I	106	110/70	11	B/L NVBS	Soft non tender
11	6386875	62	M	-	-	2 yrs	-	-	No	No	86	110/70	11	B/L NVBS	Soft non tender
12	4652128	55	M	-	-	2 yrs	-	-	No	yes B/L grade II	80	110/70	10	B/L Coarse Crepitation	Soft non tender
13	1115248	55	M	-	-	4 yrs	-	-	No	Yes	90	130/80	8	B/L Coarse Crepitation	Soft non tender
14	1091606	80	M	-	-	2 yrs	chest pain 1 week	-	No	yes B/L grade I	62	130/80	12	B/L Coarse Crepitation	Soft non tender
15	1101377	66	F	4 days	-	4 yrs	4 days	-	No	No	130	110/70	11	B/L Coarse Crepitation	Soft non tender
16	1043272	42	M	-	-	10 days	-	-	No	yes B/L grade II	80	90/60	11	B/L Coarse Crepitation	Soft non tender
17	1081140	81	M	-	chest pain 1 day	3yrs	3 months	-	No	yes B/L grade II	76	110/70	10	B/L Coarse Crepitation	Soft non tender
18	1080384	63	F	-	-	2 yrs	-	-	No	yes B/L grade I	107	100/60	11	B/L Coarse Crepitation	Soft non tender
19	1115084	61	F	7 days	7 days	4yrs	-	7 days	No	yes B/L grade II	98	120/80	12	B/L Coarse Crepitation	Soft distended
20	1055592	64	M	-	-	1yrs	-	14 days	No	yes B/L grade II	67	90/60	10	B/L Coarse Crepitation	Soft distended
21	1091176	45	M	-	7 days	4 yrs	-	15 days	No	yes B/L grade II	90	110/70	12	B/L Coarse Crepitation	non tender
22	1048821	20	F	-	-	4 yrs	-	-	No	Yes B/L grade 11	76	110/70	11	B/L NVBS	non tender
23	1065766	70	M	-	15 days	2 years	-	-	No	No	87	110/70	9	B/L Coarse Crepitation	Soft non tender
24	1059301	24	M	-	15 days	3 yrs	-	-	No	yes B/L grade II	108	90/60	10	B/L Coarse Crepitation	Soft non tender
25	993361	62	F	-	-	3 yrs	-	-	Yes	yes B/L grade I	111	110/70	11	B/L Coarse Crepitation	Soft non tender
26	1113093	48	F	-	-	4 yrs	-	10 days	No	yes B/L grade II	86	110/70	11	B/L Coarse Crepitation	Soft non tender
27	6522537	71	M	-	-	1 yrs	-	7 days	No	yes B/L grade II	106	120/80	12	B/L Coarse Crepitation	Soft non tender

	Investigation					Liver Function Test																		
SL.NO	Hemoglobin(g/dl)	Platelets	WBC	PT-INR	APTT	Total bilirubin(mg/dl)	Direct bilirubin(mg/dl)	Total protein(g/dl)	AST	ALT	ALP	ALB	AST/ALT Ratio	APRI Index	FIB-4	RBS	S.Urea	S.Creatinine	S.sodium	S.Potassium	USG Abdomen and Pelvis	TSH	Free T3	Free T4
1	12.5	198	8100	1.4	0.89	0.3	0.14	5.521	21	11	70	2.5	1.91	0.97	2.53	232	33	0.92	137	3.28	Normal	3.94	1.53	1.12
2	13.5	181	10.8	1.1	0.9	0.74	0.36	8.5	86	47	102	3.5	1.83	1.19	3.72	78	38	1.26	134	4.01	Normal	3.2	1.52	1.14
3	12	353	7.9	1.01	0.91	0.95	0.3	7.2	22	10	135	4.1	2.2	0.16	1.18	83	50	1.69	141	4.55	Normal	0.49	3.75	1.7
4	13.9	265	9.1	0.97	0.95	0.8	0.27	6.5	99	50	115	3.9	1.98	0.93	3.01	362	37	0.97	138	4.86	Normal	0.58	2.05	1.42
5	10.5	173	7.6	1.19	1	0.71	0.23	5.2	13	22	55	3.1	0.6	0.19	1.4	83	20	0.87	135	4.75	B/L mild plueral effersion	0.42	2.88	1.31
6	12.5	298	12.1	1.04	1.22	0.26	0.11	5.1	42	10	56	3.1	4.2	0.35	2.86	268	34	1.49	139	5.05	Enlarged prostate	1.33	1.69	1.15
7	6.9	221	7	1.39	0.78	0.41	0.23	5.3	16	15	125	3.3	1.07	0.18	1.24	96	46	1.36	143	5.9	Enlarged prostate	1.74	2.54	1.16
8	9.7	293	10.47	0.93	1.11	0.3	0.1	6.8	20	14	126	2.7	1.43	0.17	0.42	88	63	0.68	137	5.41	Mild to moderate ascites	95	1.26	0.45
9	11.1	249	7.5	1.24	1.29	0.23	0.12	6.5	16	10	149	2.1	1.6	0.16	1.08	52	50	1.05	127	3.97	B/L mild pleural effusion	5.92	2.54	1
10	9	240	5.7	1.05	1.12	0.75	0.41	6.8	27	17	80	3.6	1.6	0.28	1.99	105	28	1.59	129	4.27	Right mild Hydronepheoeis	0.43	2.93	1.42
11	13.8	207	10.5	1.28	1.02	2.82	0.9	6.8	215	106	81	4.1	2.03	2.6	6.25	164	26	0.81	138	4.16	Enlarged prostate	1.67	2.97	1.74
12	16.7	210	6.6	1.17	1.31	0.54	0.18	6.5	18	15	94	4	1.2	0.21	1.22	83	20	1.05	140	4.9	Normal	0.42	2.18	1.33
13	9.6	179	7.1	1.02	1	0.58	0.13	6.4	51	32	161	3.5	1.6	0.71	2.77	132	27	0.62	133	3.97	B/L few small renal cyst,B/L mild pleural effusion	2.12	2.08	2.08
14	10.6	219	9.2	0.99	0.9	0.4	0.13	5.7	17	12	120	3.1	1.42	0.19	1.8	143	33	1.2	142	2.74	B/L mildB/L mild pleural effersion plueral effersion	2.12	2.78	2.04
15	10.2	127	11.3	1.27	1.01	1.57	0.73	7.3	71	25	66	3.9	2.84	1.4	7.38	130	64	1.64	133	6.22	B/L mild plueral effB/L mild pleural effersionersion	1.28	1.05	3.59
16	16.3	210	7.8	0.93	1.13	0.55	0.24	8.3	41	26	75	5.6	1.58	0.49	1.61	118	33	0.99	139	3.82	B/L mild pleural effersion	1.9	0.9	0.8
17	14.1	257	17.9	1.35	0.82	1.39	0.51	6.3	215	64	89	3.8	3.36	2.09	8.47	326	58	1.43	132	4.16	Normal	5.37	2.66	1.16
18	12.1	196	9.6	1.2	0.96	0.21	0.13	6.9	16	14	85	3.6	1.14	0.2	1.38	206	79	1.04	138	4.15	Left Renal calculi	0.48	2.88	1.31
19	12.1	285	21.1	1.53	1.07	0.62	0.23	6.4	361	109	83	3.2	3.31	3.17	7.4	281	85	1.9	135	4.17	Normal	0.42	1.4	1.2
20	12	85	7.5	1.26	1.01	0.68	0.42	6.1	11	10	67	2.7	1.1	0.32	2.74	114	40	1.55	135	5.44	mild ascitis,B/L mild pleural effersion	1.19	1.08	1.18
21	15.2	333	11	1.08	1.03	0.6	0.26	7.4	43	48	96	3.9	0.9	0.32	0.84	261	22.42	0.95	134	3.89	Right Renal calculi	1.81	1.96	1.57
22	11.1	233	11.1	0.92	1.1	0.36	0.15	4.7	20	12	102	2.5	1.7	0.22	0.49	50	30	0.64	133	4.66	Normal scan	2.21	1.8	1.56
23	9.2	238	15	2.59	1.7	0.85	0.46	6.1	228	47	116	2.7	4.85	2.4	9.7	211	89	1.4	148	3.85	B/L mild pleural effusion	0.73	2.88	1.31
24	14.5	316	11.2	1.62	1.26	4	1	5.8	35	18	52	3.4	1.9	0.28	0.58	93	28	1	135	4.1	Normal scan	3.78	3.05	1.47
25	9.9	275	15.2	4.38	1.02	0.1	0.1	6.4	48	14	159	3.6	3.43	0.44	2.89	218	141	1.62	136	3.95	Normal scan	0.22	1.3	2.01
26	8.6	212	7.7	1.28	0.82	0.26	0.09	5.5	37	20	103	2.4	1.85	0.44	1.87	66	187	1.72	144	3.13	Left renal calculi	4.09	1.37	0.66
27	9	205	12.7	0.96	0.96	0.55	0.28	4.9	41	10	90	2	4.1	0.5	4.5	248	27	1.38	132	4.54	Normal scan	0.11	1.51	1.72

SL.NO	2D Echo	DIAGNOSIS		EF %	NYHA Class	ECG
1	IHD, HYPOKINESIA OF INFERIOR WALL AND INFERO LATERAL WALL, MODERATE DYFUNCTION WITH EF - 40%, CONCENTRIC LVH, TRIVAL MR, SCLEROTIC AORTIC VALVE WITH GRADE I AR, GRADE I TR WITH PPG - 25 MMHG, MILD PAH, NO CLOT	IHD, HTN	AST/ALT ratio= 1.91(GROUP 2)	40	2	SINUS RHYTM,Q WAVES IN LEAD II ,III,AVF
2	DCM, Global hypokinesia of LV, Severe LV dysfunction with LVEF - 30%, RV dysfunction {TAPSE 1.4cm S1 8.6 cm/s}, LV dilated, MAC i.e Grade I MR (2 jets), AV thickened i.e Trivial AR, Grade III TR i.e PPG -36 mmHg, Estimated PA Pressure 36+10=46mmHg, Modrate PAH, no clot, Rim of PE	DCM	AST/ALT = 1.83 (group 2)	30	3	SINUS RHYTHM
3	IHD, Akinesia of inferior wall, inferior septum i.e hypokinesia of inferlateral wall, anterior wall, anteroseptum, apical septum and apex, moderate LV dysfunction i.e LVEF - 35%, RV dysfunction {TAPSE 1.4cm S1 8.2 cm/s}, LV dilated, Grade II MR, AV thickened i.e Trivial AR, Triuial TR i.e PPG - 25 mmHg, Estimated PA Pressure 25+5=30 mmHg, Moderate PAH (ACT - 76ms), No clot/PE	IHD	AST/ALT = 2.2 (group 2)	35	3	SINUS RHYTHM,Twave inversion in lead II,III,AVF;, ST depression in Lead I, avl, V5, V6
4	IHD, Akinesia of apical septum, apex, apicolateral, basal inferio wall and inferolateral wall, hypokinesia of anterior wall, anteroseptum, moderate LV dysfunction i.e EF - 35%, RV dysfunction {TAPSE 1.0cm S1 8.7 cm/s}, Grade II MR, AV thickened i.e Trivial AR, Triuial AR i.e PPG - 25 mmHg, Estimated PA Pressure 41+5=46 mmHg, Moderate PAH (ACT - 70ms), No clot/PE	IHD ,T2DM	AST/ALT = 1.98(group 2)	35	3	SINUS tachycardia, ST depression in Lead I, AVL, VS, V6; ST elevation ,II, III,AVF
5	IHD, Akinesia of anterior wall, anteroseptum, apical septum and apex, apicalateral segment, anterolateral wall i.e hypokinesia of lateral wall, moderate LV dysfunction i.e LVEF - 35%, mitral annulate calcification i.e Grade I MR, AV thickened i.e Trivial AR, Triuial TR i.e PPG - 25 mmHg, Estimated PA Pressure 25+5=30 mmHg, Mild PAH (ACT - 81ms),? Layered LV apical clot, No PE	IHD -T2DM , HTN	AST/ALT = 0.6(group 1)	35	3	SINUS RHYTHM, LAD, T wave inversion V1, V2, V3, V4
6	S/p PTCA, Akinesia of antenor wall, anteroseptum, i.e hypokinesia of apical septum and apex, inferoseptum, moderate LV dysfunction i.e LVEF - 35%, mifd RV dysfunction {TAPSE 1.5cm S1 8.6 cm/s}, LV dilated LA midly duilated, Grade I MR, Grade I TR i.e PPG - 38 mmHg, Estimated PA Pressure 38+5= 43 mmHg, Mild PAH, no clot/ PE	IHD	AST/ALT = 4.2(group 2)	35	3	SINUS RHYTHM, Q waves in lead I,AVL,V5,V6;T wave inersion in v3,v4,v5,v6
7	S/p PTCA, Akinesia of inferalat anteralat and lat wall i.e hypokinesia of inferoseptum , inf wall, moderate LV dysfunction i.e LVEF - 35%, RV dysfunction {TAPSE 1.3 cm S1 7.3 cm/s}, RA and RV midly dilated LA dilated, Grade I MR, Grade II TR i.e PPG - 62 mmHg, Estimated PA Pressure 62+10 = 72mmHg, severe PAH, no clot/ PE	IHD,T2DM ,HTN,	AST/ALT = 1.07(group 2)	35	3	SINUS RHYTHM, ST depression I, AVL, V4, V5, V6
8	DCM, Global hypokinesia of LV, Severe LV dysfunction with LVEF - 35%, RV dysfunction {TAPSE 1.4cm S1 8.6 cm/s}, LV dilated, MAC i.e Grade I MR (2 jets), AV thickened i.e Trivial AR, Grade III TR i.e PPG -36 mmHg, Estimated PA Pressure 36+10=46mmHg, Modrate PAH, no clot, Rim of PE	DCM ,HTN,Hypothyr	AST/ALT = 1.43(group 2)	35	3	SINUS RHYTHM, Lead II, III, AVF, T wve inversion at V2, V3, V4, V5
9	IHD, Akinesia of anterior wall, anteroseptum, apical septum and apex, i.e hypokinesia of inferior wall, inferoseptum. moderate LV dysfunction i.e LVEF - 40%, Grade II MR, AV thickened i.e Trivial AR, Grade II TR i.e PPG - 30 mmHg, Estimated PA Pressure 30+5=35 mmHg, Mild PAH, no clot, Right sided pericardial effusion	IHD	AST/ALT = 1.07(group 2)	40	2	SINUS rhythm, ST depression Lead I, AVL
10	Global Hypokinesia of LV, Severe LV dysfunction with LVEF - 30-35%, RV Dysfunction + (TAPSE - 1.3 cm, S1 - 7.5 cm/s), RA,RV,LA dilated, Mitral Annular calcification with severe MR (VC 0.6cm), Aortic valve thickened with Grade -I AR, Grade- III TR with PPG-40mmHg, Estimated PA pressure - 40+10 = 50mmHg, Servere PAH (Act - 59ms), No clot/pericardial effusion, Note. AF noted during Echo study.	IHD-T2DM ,HTN,	AST/ALT = 1.6(group 2)	30	3	Atrial fibrillation, fast ventilator rate left axis deviation, T wave inversion V5, V6, I, AVL
11	IHD, Akinesia of anterior wall, anteroseptum, apical septum and apex, moderate LV dysfunction i.e LVEF - 40%, Tririal MR, AV thickened i.e Trivial AR, Grade I TR i.e PPG - 35 mmHg, Estimated PA Pressure 35+5=40 mmHg, LV apical clot measuring 1.2x0.9cm, No PE	IHD	AST/ALT = 2.03 (group 2)	40	2	SINUS RHYTHM, ST elevation Lead I, AVL, V2, V3,V4, V5, V6
12	Post PTCA, Dyskinesia of apex with Akinesia of anterior wall, anterostum, apical aeptum and apicolateral segment, Apical septum and apex thinned out, moderate LV dysfunction with EF-40%, Trivial MR, Normal PA pressure, NO Clot/Pericardial effusion	IHD	AST/ALT = 1.2(group 2)	40	2	SINUS RHYTHM, Twave inversion in Lead I, AVL, V5, V6
13	All dilated chambers, global hypokinesia of LV and Akinetic basal mid inferior wall and inferoposteror wall of LV segments, Moderate LV systolic dysfunction (Ef-35%), Mild MR, Sclerotic AOV, Trivial AR/ No AS, Mild RV/Moderate PAH (PG - 48mmHg), No clot/PE, AF during study	DCM	AST/ALT = 1.6 (group 2)	35	3	SINUS RHYTHM, poor R wave progression in V1, V2
14	Post CABG, Dyskinesia of anterior wall, anteror septum, moderate LV dysfunction with EF-40%, Grade II MR, Sclerotuic AV i.e PPG - 26mmHg i.e Grade I AR, Grade I TR i.e PPG - 32 mmHg, Mild PAH, NO Clot/Pericardial effusion	IHD,HTN	AST/ALT=1.42 (group 2)	40	2	SINUS RHYTHM, Lead I, avl, V1, V2, V3, V4, V5, V6 IHD - AWMI wellin T wave
15	DCM,Tachycardic noted on echo (HR-130 bpm), Severe calcific AS i.e PPG/MPG 72/40 mmHg i.e severe AR 215ms, Vmax 4.1ls, Aortic annulus 1.5cm, Mild Ms i.e MVO of 1.9 - 2.0 cm i.e PPG/MG 9/5mmHg i.e Severe MR (VC = 0.9cm), LA, LV dilated, Global hypokinesia of LV, Severe LV dysfunction i.e LVEF-30%, Grade I + TR i.e PPG 35mmHg, Estimated PAP 35+5=40mmHg, Mild PAH, No lA/LA appendage clot, note. AF noted during echo study	DCM,AF	AST/ALT = 2.84(group 2)-	30	3	Afrial fIbrillation with fast ventricular rate with left bundle branch block, T wave, inversion in V5, V6
16	DCM, Global hypokinesia of LV, moderate LV dysfunction i.e LVEF - 35%, RV dysfunction {TAPSE 1.2cm S1 7.9 cm/s}, LV dilated,Trivial MR, TR i.e PPG - 20 mmHg, Mild PAH (Act -95ms), no clot/ PE	DCM	AST/ALT = 1.58 (group 2)	35	3	SINUS RHYTHM, low voltage ORS
17	IHD, Akinesia of anterior wall, anteroseptum, apical septum and apex, lateral segment i.e hypokinesia of inferoseptum. moderate LV dysfunction i.e LVEF - 35%, LV dilated, Grade I MR, AV thickened i.e Trivial AR, Grade I TR i.e PPG - 25 mmHg, Estimated PA Pressure 28+5=33 mmHg, Moderate PAH, no clot/pericardial effusion	IHD,T2DM. AST/AL	AST/ALT =3.36(group 2)	35	3	SINUS RHYTHM,Q waves Lead I, AVL, ST elevation V2, V3, V4, V5, V6.
18	ICM, Akinesia of anterior wall, anteroseptum, apical septum and apex, inferior wall, inferoseptum. Apical septum and apex thinned out, severe LV dysfunction i.e LVEF - 30%, LV dilated , Grade II MR, AV thickened i.e Trivial AR, Grade II MR Mild PAH (ACT - 81 ms), no clot/ pericardial effusion	IHD,T2DM	AST/ALT =1.14(group 2)	30		SINUS RHYTHM ,LAD, BROAD QRS Broad QRS, LBBB
19	IHD, Akinesia of anterior wall, anteroseptum, apical septum and apex, antolateral wall and inferolateral wall, servere LV dysfunction i.e LVEF - 30%, RV dysfunction {TAPSE 1.2 cm S1 8.4 cm/s}, LV dilated, severe MR, AV thickened i.e Trivial AR, Grade III TR i.e PPG - 35 mmHg, Estimated PA Pressure 35+15=50mmHg, Moderate PAH (ACT - 63 mmHg) no clot/ PE	IHD,T2DM	AST/ALT = 3.31 (group 2)	30	3	SINUS RHYTHM, T wave inversion V1,V2, V3, St depresion V3, V4, V5, V6
20	IHD Akinesia of anterior wall, anteroseptum, apical septum and apex, antolateral segment and inferolateral segment, severe LV dysfunction i.e LVEF - 30%, RV dysfunction {TAPSE 0.9 cm S1 6.5 cm/s}, Grade I TR i.e PPG - 31 mmHg, Estimated PA Pressure 31+15=46mmHg, Moderate PAH no clot/ PE, LV apical clot measuring 1.4 x 1.1 cm, Bilateral PE	IHD	AST/ALT = 1.1(group 2)	30	3	A SINUS RHYTHM, ventricular, premature complexes, T wave inversion in V2, V3, V4, V5, V6
21	IHD Akinesia of anterior wall, anteroseptum, apical septum and apex, antolateral segment and inferolateral segment, servere LV dysfunction i.e LVEF - 30%, RV dysfunction {TAPSE 0.9 cm S1 6.5 cm/s}, Grade I TR i.e PPG - 31 mmHg, Estimated PA Pressure 31+15=46mmHg, Moderate PAH no clot/ PE, LV apical clot measuring 1.4 x 1.1 cm, Bilateral PE	IHD,T2DM	AST/ALT = 0.90(GROUP 1)	30	3	SINUS RHYTHM, ST elevation in lead I, AVL, V, V2, V3, V4, V5, V6
22	DCM, Global Hypokinesia of LV, Severe LV dysfunction with LVEF - 27%,LV dilated, Normal PA pressurem, No clot/pericardial effusion	DCM	AST/ALT = 1.7 (group 2)	27	4	SINUS RHYTHM, Low QRS complex
23	S/p PTCA, Akinesia of inferior wall, inferior septum, inferolateral wall i.e hypokinesia ofanterior wall and anterior septum, moderate LV dysfunction i.e LVEF - 35%, RV dysfunction {TAPSE 1.5 cm S1 7.5 cm/s}, Grade I MR, AV thickened i.e Trivial AR, Grade I TR i.e PPG -30 mmHg, Estimated PA Pressure 30+5=45mmHg, Mild PAH, no clot/ PE	IHD,HTN	AST/ALT = 4.85(group 2)	35	3	SINUS RHYTHM, Q waves II III AVF
24	S/P MVR, Gradient across mitralatre i.e PPg /MPG 15/6 mmHg i.e Grade II MR, All dilated chambers, global hypokinesia of LV, severe LV dysfunction i.e LVEF - 20%, RV dysfunction {TAPSE 1.4 cm S1 7.7 cm/s}, severe TR i.e PPG 41 mmHg, Moderate AR PHE (379ms), servere PAH (ACT 49 ms), Estimated PA Pressure 41+15=56mmHg, , No clot/PE,	DCM	AST/ALT = 1.9 (group 2)	20	4	atrial fibrillation with fast ventricular rate
25	DCM, Global hypokinesia of LV, Severe LV dysfunction with LVEF - 25%, RV dysfunction {TAPSE 1.4cm S1 8.6 cm/s}, LV dilated, MAC i.e Grade I MR (2 jets), AV thickened i.e Trivial AR, Grade III TR i.e PPG -36 mmHg, Estimated PA Pressure 36+10=46mmHg, Moderate PAH, no clot, Rim of PE	IHD,T2DM	AST/ALT = 3.43(group 2)	25	4	Monomorphic VT
26	Dilated Cardiomyopathy, Global hypokinesia of LV, LA LV Dilated, Severe LV systolic dysfunction with LVEF - 35%, Grade II MR, Grade I TR, Moderate PAH	DCM, HTN	AST/ALT = 1.85 (group 2)	35	3	SINUS RHYTHM,
27	S/p PTCA, Akinesia of anterior wall, anterolateral, apical septum and apex, apicolateral segment with hypokinesia of interior wall and inferolateral wall, moderate LV dysfunction i.e LVEF - 39.4%, RV dysfunction {TAPSE 1.3 cm, Grade II MR, Grade I TR i.e PPG - 31mmHg, Estimated PA Pressure 31+5=36mmHg, mild PAH, no clot/ PE	IHD,T2DM	AST/ALT = 4.1 (group 2)	39.4	2	SINUS RHYTHM, Poor R wave progression limb leads T wave inversion in V3, V4, V5

28	1080825	65	M	-	-	2 yrs	-	15 days	No	yes B/L grade II	86	100/60	10	B/L Coarse Crepitation	Soft distended	11.8	253	14.4	1.65	1.08	6.01	3.35	7.5	52	41	156	3.8	1.27	0.51	2.09	114	69	1.08	130	4.68	mild ascites right mild pleural effusion	21	1.5	1.6	IHD, Akinesia of inferior wall, inferior septum and inferolateral segment, moderate LV dysfunction i.e LVEF - 40%, RV dysfunction {TAPSE 1.0 cm S1 8.7 cm/s},RA, RV, PA and IVC dilated, MAC i.e Sev. Eccentric MR, AV thickened i.e Grade I AR, Sev. eccentric TR i.e PPG - 81 mmHg, Estimated PA Pressure 81+15=96mmHg, Sev PAH, No clot/PE	IHD	AST/ALT = 1.27 (group 2)	40	2	SINUS RHYTHM, T wave inversion V2, V3, V4, V5, V6
29	1091560	71	F	-	-	2 yrs	-	-	No	yes B/L grade II	88	110/70	12	B/L Coarse Crepitation	Soft non tender	11.7	447	13.5	1.3	2.9	0.47	0.19	5.9	344	85	71	3	4.05	1.92	5.93	154	38	1.12	135	4.13	Left mild and right mini pleural effusion	0.37	2.86	1.54	IHD Akinesia of anterior wall, anteroseptum, apical septum and apex, antolateral segment and antrolateral segment with hypokinesia of interior wall and inferior septum, , Moderate LV dysfunction i.e LVEF - 35%, Grade I MR , AV thickened i.e Trivial AR, Grade I TR with PPG - 25 mmHg, Estimated PA Pressure 25+15=40mmHg, mild PAH (ACt 89ms), no clot/ PE	IHD, HTN	AST/ALT = 4.05(group 2)	35	3	SINUS Tachycardia, T wave inversion V1, V2, V3
30	1091374	58	F	-	-	2 yrs	-	-	No	No	89	110/70	11	B/L Coarse Crepitation	Soft non tender	13.6	174	12.2	1.24	180	0.49	0.1	6.3	54	29	82	3.9	1.86	0.78	3.34	84	29	0.9	140	3.96	Right Renal calculi	0.68	2.29	1.24	Post PTCA, Dyskinesia of apical septum apex, Akinesia of anterior wall, anterostum, apicolateral segment, Apical septum and apex thinned out, moderate LV dysfunction with EF-40%, moderate MR (VC = 0.5cm), AV thickened i.e PPG - 55mmHg, Estimated PA Pressure 55+5=60mmHg, moderate PAH, No PE, LV apical clot.	IHD	AST/ALT = 1.86 group 2)	40	2	SINUS RHYTHM , T wave inversion in V1, v2, V3, V4, V5, V6
31	6287239	59	M	-	-	2 yrs	-	10 days	No	yes B/L grade I	80	110/70	11	B/L Coarse Crepitation	Soft non tender	13.9	238	8.65	1.59	1.09	0.23	0.09	5.8	44	31	86	4	1.42	0.46	1.96	137	28	1.09	136	3.56	Enlarged prostate	1.26	2.27	0.99	IHD, Akinesia of inferior wall, inferior septum and inferolateral wall, Inferior wall, inferoseptum, Inferolateral wall scared, moderate LV dysfunction i.e LVEF - 40%, Trivial MR , AV thickened i.e Trivial AR, mild PAH, No clot/PE	IHD	AST/ALT = 1.42(group 2)	40	2	SINUS RHYTHM, Q waves Lead II, III, AVF, T wave inversion II III AVF
32	1059632	75	F	-	-	2 yrs	-	-	No	No	80	110/70	11	B/L NVBS	Soft non tender	12.4	163	7	1.2	1.33	0.91	0.53	5.9	27	22	70	3.3	1.23	0.41	2.65	86	38	0.71	137	2.94	B/L mild pleural effusion	1.06	1.71	1.81	IHD Akinesia of anterior wall, anteroseptum, apical septum and apex, apicalateral segment, anterolateral wall with hypokinesia of lateral wall , Moderate LV dysfunction i.e LVEF - 35%, MAC i.e Grade I MR , AV thickened i.e Trivial AR, Trivial TR with PPG - 25 mmHg, Estimated PA Pressure 25+5=30mmHg, mild PAH (ACt 81ms),layered LV apical clot, no PE	IHD	AST/ALT = 1.23 group 2)	35	3	SINUS RHYTHM, LAD, T wave inversion V1, V2, V3, V4, V5
33	1083512	37	M	-	-	2 yrs	-	-	No	No	76	120/80	11	B/L Coarse Crepitation	Soft distended	14.4	121	7.7	2.29	1.26	3.54	1.94	6.5	18	12	50	4.2	1.5	0.37	1.59	96	77	1.52	138	3.37	mild pleural effusion	3.97	2.15	1.81	Ischemic heart disease, LV segmental akinesia, Reduced Biventricular function (LV EF-35%), Moderate MR, Moderate TR, Pulmonary Hypertension, Layered clot at LV apex	IHD	AST/ALT ration = 1.5(GROUP 2)	35	3	SINUS RHYTHM, poor R waves in limb leads
34	1029133	71	F	-	-	4 yrs	-	-	No	No	76	130/80	12	B/L Coarse Crepitation	Soft non tender	11.7	187	7.8	1.16	0.88	0.71	0.29	6.5	16	15	61	3.8	1.07	0.21	1.57	128	22	0.64	145	3.4	Right mild and Left mini pleural effusion	1.81	1.64	1.83	DCM, Global hypokinesia of LV, Severe LV dysfunction with LVEF - 30%, RV dysfunction {TAPSE 1.2cm S1 6.5 cm/s}, All chamber dilated, Grade III MR, AV thickened i.e Trivial AR, Severe TR with PPG -49 mmHg, Estimated PA Pressure 49+15=64mmHg, Severe PAH, no clot, Rim of PE	DCM,HTN	AST/ALT = 1.07 group 2	30	3	Normal SINUS RHYTHM
35	6288630	65	M	-	-	2 yrs	-	-	No	yes B/L grade I	76	120/80	11	B/L Coarse Crepitation	Soft non tender	11.1	409	8.6	1.57	1.02	0.59	0.24	5.3	34	35	78	2.8	0.97	0.21	0.91	362	82	1.38	127	4.82	B/L mild pleural effusion	1.65	2.03	1.44	CAD,RWMA,MODERATE ECCENTRIC MITRAL REGURGITATION LVEF 40%	IHD,T2DM	AST/ALT = 0.97 group 1	40	2	SINUS RHYTHM, Q waves in II, III, AVF
36	1067606	19	F	-	7 days	1 yrs	-	-	No	yes B/L grade II	108	90/60	11	B/L Coarse Crepitation	Soft non tender	10.9	239	9	1.47	1.1	0.8	0.55	6.1	47	91	75	3.5	0.52	0.49	0.23	90	52	0.92	137	4.09	Normal	0.18	1.43	1.17	RHD, MVP i.e severe eccentric MR, Global hypokinesia of LV, Severe LV dysfunction with LVEF - 20-25%, RV dysfunction {TAPSE 1.2cm S1 6.1 cm/s}, LA LV dilated, Grade III TR i.e PPG -38 mmHg, Estimated PA Pressure 38+5=43mmHg, moderate PAH (ACt 74ms), no clot/PE	RHD	AST/ALT = 0.52 (group 1)	20	4	SINUS Tachycardio, P.Pulmonale, T wave inversion in V3, V4, V5, V6
37	1052943	49	M	-	15 days	3 yrs	-	-	No	yes B/L grade II	90	100/60	11	B/L Coarse Crepitation	Soft non tender	13.5	288	12.3	1.07	1.01	0.99	0.57	5.3	19	21	63	3.1	0.9	0.17	0.71	320	34	0.96	138	4.01	B/L mild pleural effusion	4.36	1.6	1.15	DCM, Global hypokinesia of LV, Severe LV dysfunction with LVEF - 30%, RV dysfunction {TAPSE 1.2cm S1 6.5 cm/s}, All chamber dilated, Grade III MR, AV thickened i.e Trivial AR, Severe TR with PPG -49 mmHg, Estimated PA Pressure 49+15=64mmHg, Severe PAH, no clot, Rim of PE	DCM,T2DM	AST/ALT = 0.9(group 1)	30	3	SINUS RHYTHM, T wave inversion in V2, V3, V4, V5
38	1036444	72	M	-	10 days	6 yrs	-	-	No	yes B/L grade II	78	110/70	11	B/L Coarse Crepitation	Soft non tender	14.2	331	6.15	0.96	0.96	0.6	0.2	6.9	20	28	283	3.9	0.71	0.15	0.82	106	44	1.6	137	5	NORMAL	3.2	1.2	1.8	Post PTCA, Akinesia of anterior wall, anterostum, apicolateral segment, Apical septum and apex, Muscle mass preserved, moderate LV dysfunction with EF-40%, Trivial MR, AV thickened i.e Trivial AR Normal PA Pressure No clot/ PE,	IHD, HTN	AST/ALT ratio = 0.71 (Group 1)	40	2	SINUS RHYTHM T wave inversion in lead I, AVL, V5, V6
39	1109137	72	F	-	10 days	10 yrs	-	20 days	No	yes B/L grade I	53	130/80	12	B/L Coarse Crepitation	Soft non tender	10.3	366	11.8	1.02	0.78	0.8	0.23	6.2	32	14	125	3.3	2.29	0.22	1.68	326	65	1.22	141	4.86	B/L moderate pleural effusion	2.53	1.6	1.44	Bradycardia noted on echo (HR 53bpm), IHD, Akinesia of anterior wall, anteroseptum, apical septum and apex, Moderate LV dysfunction with EF - 40%, Grade I MR , AV thickened with PPG -13mmHg with Trivial AR, Grade I TR with PPG - 36 mmHg, Estimated PA Pressure 36+5=41mmHg, mild PAH No clot, no PE	IHD , T2DM	AST/ALT = 2.29 (Group 2)	40	2	SINUS RHYTHM, T wave inversion in Lead I, AVL, V5, V6
40	1066869	55	M	-	-	2 yrs	-	-	No	yes B/L grade II	80	110/60	10	B/L Coarse Crepitation	Soft non tender	14.8	279	7.6	1.16	0.94	1.4	0.5	6.5	153	571	163	2.9	0.27	1.37	1.26	106	80	1.1	135	3.5	Left mild to moderate pleural effusion	2.09	3.81	1.99	PPT, Global hypokinesia of LV, Severe LV dysfunction with LVEF - 30%, RV dysfunction {TAPSE 1.3cm S1 8.7 cm/s} Trivial MR, AV thickened i.e Trivial AR, Mild PAH (ACT 81ms) No clot/ PE,	DCM	AST/ALT = 0.27(group 1)	30	3	SINUS RHYTHM, T wave inversion in V2, V3
41	1091566	67	M	-	-	3 yrs	-	-	No	No	77	110/70	11	B/L NVBS	Soft non tender	11.5	208	5.7	1.1	1.2	0.58	0.28	5.6	80	56	88	3.2	1.43	0.96	3.44	384	48	1.47	139	4.43	Normal	58.21	2.32	0.88	Tachycardic noted on echo (HR-112 bpm), IHD Akinesia of anterior wall, anteroseptum, apical septum and apex, apicalateral segment, Moderate LV dysfunction i.e LVEF - 40%, Grade II MR, AV thickened i.e Trivial AR, Trivial TR i.e PPG - 25mmHg, Estimated PA Pressure 25+10=35mmHg, mild PAH No clot, no PE.	IHD ,T2DM,HTN ,H	AST/ALT = 1.43 (Group 2)	40	2	SINUS RHYTHM, ST depresion Lead I, AVL, V4, V5, V6
42	6294529	64	M	-	20 days	3 yrs	-	-	No	yes B/L grade II	86	90/60	9	B/L Coarse Crepitation	Soft non tender	11.9	266	10.3	1.46	1.05	0.55	0.1	7	26	72	72	3.8	0.36	0.24	0.74	171	34	1.67	128	5.14	Normal	1.48	0.88	0.8	Global hypokinesia of LV, Severe LV dysfunction with LVEF - 25%, RV dysfunction {TAPSE 1.3 cm S1 8.9 cm/s}, LV dilated, Grade III MR, AV thickened i.e Trivial AR, Grade II TR with PPG 44mmHg, Estimated PA Pressure44+10=54mmHg, moderate PAH (ACt 76ms), no clot/PE	IHD, T2DM	AST/ALT = 0.36(Group 1)	25	4	SINUS RHYTHM, Q waves in Lead II, III, AVF, T wave inversion V1, V2, V3
43	1092938	50	M	-		3 yrs	-	-	Yes	No	87	110/70	10	B/L NVBS	Soft non tender	15.5	334	8.2	1.35	1.12	2.48	0.64	7.4	123	41	95	4.5	3	0.92	2.88	130	20	1.21	140	3.34	Normal	1.27	2.53	1.37	IHD Akinesia of anterior wall, anteroseptum, apical septum and apex, Mild LV dysfunction i.e LVEF - 40%, Moderate MR, Normal PA Pressure,Grade I DDF, No clot/no PE	IHD	AST/ALT =3 Group 2	40	2	SINUS RHYTHM, T wave inversion, V2, V3, V4, V5
44	1068365	60	F	-		3 yrs	-	-	No	yes B/L grade I	98	100/60	11	B/L NVBS	Soft non tender	10.1	538	10.81	1.29	2.57	0.4	0.2	7.8	65	8	117	2.8	8.125	0.3	2.56	432	38	0.9	142	5.32	cystitis	1.3	0.53		Tachycardic noted on echo (HR-135 bpm), Global hypokinesia of LV, Moderate LV dysfunction i.e LVEF - 40%, Grade I MR, AV thickened i.e Trivial AR, mild PAH (ACT 85 ms) No clot, no PE.	IHD ,HTN,T2DM	AST/ALT = 8.125 Group 2	40	2	SINUS RHYTHM, ST depression I, II, III, AVF, V4, V5, V6
45	1067265	53	F	-		4 yrs	-	10 days	No	yes B/L grade I	105	120/80	10.5	B/L NVBS	t distended non ten	14.1	310	8.25	1.37	1.21	1.8	0.7	8.1	406	259	129	3.8	1.6	3.27	4.3	107	64	0.8	132	3.1	BL mild PLEURAL EFFUSION	3.71	1.94	1.72	Tachycardic noted on echo (HR-110 bpm), Global hypokinesia of LV, Severe LV dysfunction with LVEF - 30%, RV dysfunction {TAPSE 1.3 cm S1 8.4 cm/s} Severe MR, All Chambers dilated, Severe TR with PPG 60mmHg, Estimated PA Pressure 60+15=75mmHg, Severe PAH (ACt 56ms), no clot/PE	DCM, HTN	AST/ALT = 1.6 Group 2	30	3	SINUS tachycardia
46	1091343	70	F	-		3 yrs	-	-	No	No	78	120/80	11	B/L NVBS	Soft non tender	9.9	184	8.6	1.59	0.97	0.34	0.13	7	203	37	119	4.1	5.49	2.76	12.7	485	73	1.6	131	6.3	cystitis	2.68	1.95	1.77	IHD Akinesia of anterior wall, anterolateral and lateral wall with hypokinesia of anteroseptum and inferolateral wall , Moderate LV dysfunction i.e LVEF - 35%, RV dysfunction {TAPSE 1.0 cm S1 7.2 cm/s} LA Dilated, Severe MR (VC 0.8cm) , AV thickened i.e Trivial AR, Grade ITR with PPG - 31 mmHg, Estimated PA Pressure 31+15=46mmHg, moderate PAH, No clot, no PE	IHD , T2DM	AST/ALT = 5.49 Group 2	35	3	SINUS RHYTHM, T wave inversion in V1, V2, V3, V4, V5, V6
47	1140610	69	M	-	-	4 yrs	-	-	No	No	62	110/70	10	B/L NVBS	Soft non tender	11.7	184	10.5	1.59	0.97	0.34	0.13	7	203	37	119	4.1	5.5	2.76	12.7	485	34	1.04	134	4.22	Enlarged Prostate	2.36	1.78	1.67	IHD Akinesia of anterior wall, anterolateral and lateral wall with hypokinesia of anteroseptum and inferolateral wall , Moderate LV dysfunction i.e LVEF - 35%, RV dysfunction {TAPSE 1.0 cm S1 7.2 cm/s} LA Dilated, Severe MR (VC 0.8cm) , AV thickened i.e Trivial AR, Grade ITR with PPG - 31 mmHg, Estimated PA Pressure 31+15=46mmHg, moderate PAH, No clot, no PE	IHD	AST/ALT = 5.5 Group 2	35	3	SINUS RHYTHM, Biphasic T wave inversion V2, V3, V4
48	1092452	63	M	-	7 days	4 yrs	-	-	No	yes B/L grade III	100	100/60	13	B/L Coarse Crepitation	Soft non tender	13.8	438	9.4	1.39	0.97	1.23	1.03	5.2	87	67	236	2.5	1.298	0.2963	1.55	232	42	0.6	122	5.82	Right mild hydronephrosis	1.87	1.49	1.04	S/p PTCA, Akinesia of anterior wall, anterolateral, apical septum and apex, with hypokinesia of lateral wall and inferor wall, Apical septum apex thined out, moderate LV dysfunction i.e EF - 35%, LA, LV dilated, Grade I MR, Grade I TR with PPG - 25mmHg, Estimated PA Pressure 25+5=30mmHg, mild PAH, Layered LV apical clot, no PE	IHD,T2DM	AST/ALT=1.29(GROUP 2	35	3	SINUS RHYTHM, Q wave Leads I, AVL, T wave inversion V2, V4
49	1140248	68	M	-	-	2 yrs	-	-	No	No	76	120/80	10	B/L NVBS	Soft non tender	12.8	184	9.4	1.41	0.68	0.34	0.13	7	203	37	119	4.1	5.5	2.76	12.7	125	23	0.57	138	3.63	Normal	0.8	0.45	0.8	Post PTCA, Akinesia of anterior wall, anteroseptum, apicolateral segment, Apical septum and apex, lateral wall with hypokinesia of lateral wall and inferor wall, Apical septum apex thined out, moderate LV dysfunction i.e EF - 35%, LA, LV dilated, Grade I MR, Grade I TR, mild PAH (ACT - 85ms), No clot/ PE,	IHD	AST/ALT =5.5(GROUP 2)	35	3	SINUS RHYTHM, Biphasic T wave inversion V1, V2, V3, V4, V5
50		35	M	-	-	2YS	-	-	no	no	87	110/70	11	b/l nvbs	soft non tender	12.7	307	8.3	0.91	1.22	2.3	0.8	6	67	54	74	3.3	1.24	0.55	1.78	112	21	0.67	132	4	Normal	2.83	3.53	1.22	Follow up ear of perscardits, IHD Akinesia of anterior wall, anteroseptum, Muscle mass preserved Moderate LV dysfunction i.e LVEF - 40%, Moderate MR (VC 0.4cm) , Normal PA Pressure, No clot, no PE	IHD	AST/ALT=1.24(GROUP 2)	40	2	SINUS RHYTHM, ST elevation lead I, AVL, V5, V6, T wave inversion in II, III, AVF
51	1092391	75	M	-	-	15 days	-	15 days	No	yes B/L grade III	100	100/60	12	B/L Coarse Crepitation	Uniformly distended	5.3	304	4.2	1.33	1	1.4	0.56	6.3	33	54	86	3.3	0.61	0.2	1.11	288	67	1.49	134	4.93	Mild cystitis	1.39	1.39	1.13	IHD Akinesia of anterior wall, anterosep, apical sep and apex, Moderate LV systolic dysfunction i.e EF - 40%, Grade II MR, Sclerotic AVD i.e PPG - 14 mmHg, Grade I AR, Grade II TR i.e PPG of 51 mmHg, moderate PAH, Type I DDF	IHD,T2DM,HTN	AST/ALT=0.81(GROUP 1)	40	2	SINUS RHYTHM, Left axis deviation, ST depression lead I, V4, V5, V6
52	1140318	60	M	-	-	2 yrs	-	-	No	No	108	110/70	11	B/L NVBS	Soft non tender	12.7	307	8.3	0.91	1.22	2.3	0.8	6.4	67	54	74	3.3	1.24	0.55	1.78	477	29	1.15	131	5.1	Enlarged Prostate	4.41	0.24	0.21	IHD, Akinesia of inferior wall, inferior septum inferolateral wall and lateral wall, moderate LV dysfunction i.e EF - 40%, Grade II MR, AV thickened i.e Trivial AR, Trivial TR I.e PPG - 23mmHg mild PAH, No clot/PE	IHD, T2DM	AST/ALT = 1.24 Group 2	40	2	SINUS RHYTHM
53	1140264	62	M	-	15 days	2 yrs	Chest pain 5days	-	No	yes B/L grade II	80	110/80	8	B/L baral Crepitation	Soft non tender	17.5	202	8.9	1.2	0.81	1	0.1	6.1	53	20	65	3.2	2.65	0.66	3.64	150	35	1.07	133	4.91	B/L mild pleural effusion	2.12	3.25	1.35	Global hypokinesia of LV, Severe LV dysfunction with EF - 23.3%, RV dysfunction {TAPSE 1.4 cm S1 8.1 cm/s}, LV dilated, Trivial MR, AV thickened i.e Trivial AR, Large LV apical clot measuring 2.7 cm x 2.3cm moderate PAH (ACt 85ms), no clot/PE	DCM,T2DM	AST/ALT ration = 2.65 Group 2	23.3	4	SINUS RHYTHM, Q waves in II, III, AVF
54	1039690	48	F	-	-	2 yrs	-	-	No	No	68	110/70	10	B/L NVBS	Soft non tender	14	28	11.1	1.02	0.86	2.3	0.8	6.4	67	54	74	3.3	1.24	5.98	15.63	227	34	0.87	139	3.66	Normal	3.2	1.2	1.6	IHD, Akinesia of inferior wall, inferilateral wall anterolateral wall and anteror wall, ans lateral wall, moderate LV systolic dysfunction i.e LVEF - 35.40%, Muscle mass preserved, MAC i.e severe MR (vc . 0.8 cm) AV thickened i.e Trivial AR, Grade I eccentric TR i.e PPG - 42mmHg, Estimated PA Pressure 42+10=52mmHg, moderate PAH, No clot/PE	IHD	AST/ALT ration = 1.24(Group 2)	35	3	SINUS RHYTHM, T wave inversion in II, III, AVF
55	1049333	63	F	-	-	2 yrs	-	-	No	No	86	110/70	10	B/L Min crepls	Soft non tender	11.1	167	8.9	1.26	0.74	0.85	0.2	6.9	17	9	89	3.8	1.9	0.25	2.14	114	17	0.82	139	3.83	Right Renal calculi	2.12	1.52	1	DCM, Global hypokinesia of LV, Severe LV dysfunction with LVEF - 30%, LV dilated, Grade I MR, AV thickened with PPG -10mmHg with Trivial AR, Mild PAH, no clot, Rim of PE	DCM	AST/ALT = 1.9 Group 2	30	3	SINUS RHYTHM, i.e low QRS complex
56	1067134	47	F	-	2 months	2 yrs	2 months	-	No	yes B/L grade II	88	120/80	10	B/L NVBS	Soft non tender	11.8	218	9.9	2.04	0.98	2.67	0.76	6.9	22	15	84	3.9	1.5	0.25	1.23	110	88	1.59	142	3.73	Left renal calculi	0.76	1.45	1.09	Global hypokinesia of LV, Moderate LV dysfunction with EF - 40%, Grade I MR, AV thickened i.e Trivial AR, Grade I TR with PPG 45mmHg, Estimated PA Pressure 45+5=50mmHg, moderate PAH, no clot, Rim of PE	DCM,	AST/ALT = 1.5 Group 2	40	2	SINUS RHYTHM
57	1140803	29	M	-	10 days	2 yrs	-	-	No	No	91	110/70	10	B/L NVBS	Soft non tender	14.2	261	13.2	1.15	0.85	0.71	0.24	6.3	99	162	119	3.7	0.61	0.94	0.87	184	30	1.27	139	3.97	Normal	0.94	3.18	1.65	IHD Akinesia of anterior wall, anterosep, apical sep and apex and apicolateral segment, Moderate LV dysfunction i.e LVEF - 40%, Trivial MR, Normal PAP, No PE, Lv apical clot measuring 0.9x 0.7cm, 0.7 x 0.5 cm	IHD, T2DM	AST/ALT = 0.61 Group 1	40	2	SINUS RHYTHM, T wave inversion Lead I, AVL, V5, V6
58	1140983	65	F	-	-	2 yrs	2 months	-	No	No	84	110/70	10	B/L NVBS	Soft non tender	10.5	186	6	0.99	0.82	0.35	0.17	7.2	24	12	101	4.5	2	0.32	2.42	137	48	1.52	137	3.58	Normal	48.4	2.28	0.67	K/C/O DCM, Global hypokinesia of LV, Severe LV dysfunction with LVEF - 30%, LV, IVE dilated, Grade II MR, AV thickened with Trivial AR, Grade II TR i.e PPG - 51mmHg, Estimated PA Pressure 51+15=66mmHg, Severe PAH, no clot/ PE	DCM, T2DM, HTN,H	AST/ALT = 2 (Group 2)	30	3	SINUS RHYTHM, low viltage QRS
59	6145432	75	F	-	15	3 yrs	-	-	No	yes B/L grade III	69	100/60	10	B/L basal Crepitation	Soft non tender	10.4	126	8600	1.2	1.3	0.4	0.3	6	36	18	26	2.1	2	0.71	5.05	109	20	1.1	134	3.4	B/L mild pleural effusion	1.1	0.1	0.4	IHD Akinesia of anterior wall, anteroseptum, apical septum and apex, apicalateral segment, anterolateral wall with hypokinesia of lateral wall , Moderate LV dysfunction i.e LVEF - 35%, MAC i.e Grade I MR , AV thickened i.e Trivial AR, Trivial TR with PPG - 25 mmHg, Estimated PA Pressure 25+5=30mmHg, mild PAH (ACt 81ms),layered LV apical clot, no PE	IHD ,T2DM	AST/ALT = 2 (Group 2	35	3	SINUS RHYTHM

60	1138927	54	M	-	2 months	2 yrs	2 months	-	No	No	86	110/80	11	B/L NVBS	Soft non tender	13.5	181	10.8	1.6	2.11	0.74	0.36	8.5	86	47	102	3.5	1.83	1.19	3.72	266	71	1.39	136	4.4	Normal	1.32	1.74	1.28	IHD, Global hypokinesia of LV, Only basal lateral wall is contaeting, Apical septum and apex thinned out, EF - 24%, EDR - 82ml, ESV - 61ml, LV mildly dilated, Gr II MR, MV Annulus - 3.1 cm, AV - N, Gr I TR i.e PPG - 30 mmHg, Mild RV dysfunction (TAPSE - 1.1 cm), Mild PAH, No clot/PE	IHD,T2DM	AST/ALT = 1.83(Group 2)	24	4	SINUS RHYTHM, low voltage QRS complex
61	1136419	79	M	-	10 days	2 yrs	-	-	No	No	87	120/80	12	B/L NVBS	Soft non tender	11.2	234	12.4	1.77	0.86	0.62	0.32	7.1	53	55	113	4.1	0.96	0.56	0.24	91	29	0.65	141	3.63	Cystitis	10.62	2.04	1.54	Known case of DCM+ POST PTCA, Global hypokinesia of LV, Severe LV dysfunction with LVEF - 30%, LV dilated, Grade I MR, AV thickened with Trivial AR, Grade III TR i.e PPG - 52mmHg, Estimated PA Pressure 52+15=67mmHg, Severe PAH (ACT - 56ms), no clot, Rim of PE	DCM, T2DM	AST/ALT = 0.96(GROUP 1)	30	3	SINUS RHYTHM,
62	1035104	65	F	-	2 months	2 yrs	2 months	-	No	No	80	140/80	11	B/L NVBS	Soft non tender	10.7	358	12.1	1.04	1.06	0.26	0.1	7.9	51	10	154	4.1	5.1	0.36	2.93	245	76	1.93	130	4.98	B/L mild pleural effusion	3.13	0.46	0.6	IHD Akinesia of anterior wall, anteroseptum, apical septum and apex, inferior septum, apicolateral segment, anterolateral segment, Moderate LV dysfunction i.e LVEF - 35%, MAC i.e Grade III MR , AV thickened with Grade l AR, Grade I TR with PPG - 27 mmHg, moderate PAH (ACt 70ms),layered LV apical clot, no PE	IHD, T2DM	AST/ALT = 5.1 Group 2	35	3	SINUS RHYTHM, T wave inversion in I, AVL, V5, V6
63	1139186	50	F	-	15 days	2 yrs	Chest pain 5days	-	No	yes B/L grade II	80	110/80	8	B/L basal Crepitation	Soft non tender	13	242	10.54	0.97	1.06	2.3	0.8	6.4	67	54	74	3.3	1.24	0.69	1.88	129	19	0.55	145	3.96	right renal calculi,B/L mild pleural effersion	1.8	0.4	0.3	S/p PTCA, Akinesia of anterior wall, anteroseptum apical septum and apex,apicolateral segment, moderate LV dysfunction i.e EF -40%, Moderate MR, AV thickened with Trivial AR, Grade II eccentric TR i.e PPG - 48mmHg, Estimated PA Pressure 48+5=53mmHg, Moderate PAH, Layered LV apical clot, no PE	IHD, T2DM	AST/ALT = 1.24 Group 2	40	2	SINUS RHYTHM, T wave inversion in I, AVL, V5, V6
64	6269139	43	M	-	2 months	2 yrs	2 months	-	No	No	76	110/80	11	B/L NVBS	Soft non tender	10.5	217	7	1.1	0.93	0.5	0.1	5.3	26	30	195	3.1	0.625	0.58	1.11	76	19	0.87	135	4.42	Right and mini pleural effersion	4.14	2.51	1.44	IHD, Akinesia of anterior wall, anteroseptum, apical septum and apex, inferoseptum, anterolateral wall i.e hypokinesia of inferior wall zero, lateral and antero lateral segment, ateroseptum, Apical septum, Apex thinned out, Severe LV dysfunction i.e LVEF - 30%, RV dysfunction {TAPSE 1.1 cm S1 6.8 cm/s}, All four chambers dilated, Grade II MR,Grade II TR with PPG - 42 mmHg, Estimated PA Pressure 42+15=57mmHg, Severe PAH (ACT - 49ms), Layered LV apical clot, No PE	IHD, T2DM	AST/ALT = 0.62 (group 1)	30	3	SINUS RHYTHM, T wave inversion V1, V2, V3, V4, V5, V6
65	6138579	80	M	-	2 months	2 yrs	2 months	-	No	yes B/L grade III	106	100/60	10	B/L Coarse Crepitation	Soft non tender	12.2	164	9.97	1.14	1.25	0.6	0.2	4.7	18	15	162	1.9	1.2	0.27	2.27	260	50	1.9	134	6.3	Enlarged prostate	5.22	1.52	1.02	Known case of DCM+ POST PTCA, Global hypokinesia of LV, Severe LV dysfunction with LVEF - 30%, RV dysfunction {TAPSE 1.3 cm S1 7.7 cm/s}, LV dilated, Grade I MR, AV thickened with Trivial AR, Grade III TR i.e PPG - 52mmHg, Estimated PA Pressure 52+15=67mmHg, Severe PAH (ACT - 56ms), no clot, Rim of PE	IHD ,T2DM	AST/ALT = 1.2 Group 2	30	3	SINUS RHYTHM, poor R wave progression in limb leads
66	1056719	82	F	-	15 days	2 yrs	Chest pain 5days	-	No	yes B/L grade II	80	110/80	8	B/L basal Crepitation	Soft non tender	10.2	164	9.97	1.14	1.25	0.6	0.2	4.7	18	15	162	1.9	1.2	0.27	2.27	260	50	1.9	134	6.3	Cysititis	2	1.2	1.1	IHD Akinesia of anterior wall, anteroseptum, apical septum and apex, Apical septum, Apex thinned out, Moderate LV dysfunction i.e LVEF - 40%, MAC i.e Grade II MR , Sclerotic AV i.e PPG 10 mmHg Gr II AR, Grade II Tr i.e PPG - 42 mmHg, Estimated PA Pressure 42+5=47mmHg, moderate PAH, no clot, no PE	IHD, T2DM, HTN	AST/ALT = 1.2 Group 2	40	2	SINUS RHYTHM, st elevation in V2, V3, V4, V5, V6
67	1042490	62	M	-		2 yrs	-	-	No	No	81	130/80	11	B/L NVBS	Soft non tender	10.6	310	12.7	1.03	1.23	0.6	0.3	5.9	14	9	121	3.2	1.56	0.11	0.93	103	33	1.8	137	4.23	Normal	2.15	1.2	1.4	Post PTCA, Akinesia of inferior wall, inferolateral wall and anterolateral wall, moderate LV dysfunction i.e EF - 40%, LV mildly dilated, Grade III MR, AV thickened with Trivial AR, Normal PAP, No clot/ PE,	IHD	AST/ALT = 1.56(group 2)	40	2	SINUS RHYTHM, Q waves in II, III, AVF
68	1104408	67	M	-	15 days	2 yrs	15 Days	-	No	No	86	120/80	11	B/L NVBS	Soft non tender	10.3	263	24.4	1.36	0.91	0.54	0.3	6.2	12	8	78	3.6	1.5	0.11	1.08	220	11.2	0.88	1.35	5.19	Ventral Hernia	1.18	0.42	0.41	IHD Akinesia of anterior wall, anteroseptum, apical septum and apex, Moderate LV dysfunction i.e LVEF - 40%, LV dilated LA mildly dilated, Grade II MR, AV thickened with Trivial AR, Trivial TR i.e PPG - 20mmHg, Normal PAP	IHD , T2DM	AST/ALT = 1.5(GROUP 2)	40	2	SINUS RHYTHM, T wave inversion in lead V2,V3, V4
68	1140759	45	M	-	-	3 yrs	3 days	-	No	yes B/L grade I	88	110/70	11	B/L Coarse Crepitation	Soft non tender	12.6	238	10.8	1.49	1.06	0.54	0.16	6.1	81	39	49	3.3	2.08	0.85	2.45	147	85	1.32	135	4.62	Normal	1.18	2.27	1.24	IHD, Akinesia of inferior wall, inferoseptum, inferilateral anterolateral segment, apical septum and apex, moderate LV dysfunction i.e EF - 32.4%, mild MR (vc . 0.3 cm) AV thickened i.e Trivial AR, Grade III TR i.e PPG - 45mmHg, Estimated PA Pressure 45+5=50mmHg, moderate PAH, No clot/PE	IHD	AST/ALT ratio = 2.08(GROUP 2)	32.4	3	SINUS RHYTHM
70	1141134	51	M	10 days	15 days	2 yrs	-	-	No	No	91	110/70	10	B/L NVBS	Soft non tender	14.4	332	8.3	0.94	0.93	0.39	0.14	7.5	22	27	93	3	0.82	0.16	0.65	95	25	0.8	134	4.62	Normal	1.8	0.43	0.43	IHD, Akinesia of anterior wall, anteroseptum, apical septum and apex, apicolateral segment, Apical septum, Apex, apicolateral apicoanterior segment thinned out, Moderate LV dysfunction i.e LVEF - 35%,LV Dilated , Trivial MR, Mild PAH (ACT - 88ms), No clot, No PE	IHD,HTN	AST/ALT ratio = 0.82(GROUP 1)	35	3	SINUS RHYTHM, Biphasic T wave inversion V2, V3, V4
71	1049804	56	M	-	-	4 yrs	-	-	No	No	76	90/100	11	B/L NVBS	Soft non tender	13.9	113	7	1.07	0.88	0.5	0.2	6.9	19	20	83	3.7	0.95	0.42	2.11	74	10	0.8	133	3.8	Normal	2	1.4	1	Post PTCA, Akinesia of anterior wall, anteroseptum, apical and apex, moderate LV dysfunction i.e EF - 40%, Grade I MR, Mild PAH (ACT -81ms) No clot/ PE,	IHD	AST/ALT = 0.9 5 (Group 1)	40	2	SINUS RHYTHM, ST elevation V2, V3
72	976463	53	M	10 days	-	2 yrs	-	-	No	No	80	110/80	11	B/L NVBS	Soft non tender	13.8	331	11.9	1.25	1.21	0.4	0.31	5.3	17	10	88	3.2	1.7	0.13	0.86	108	10	0.68	133	3.31	Normal	2	1.8	1.6	Global hypokinesia of LV, Moderate LV dysfunction with EF - 35%, RV dysfunction {TAPSE 1.1 cm S1 6.8 cm/s}, LA dilated RA mildly dilated, MAC with Grade I MR, AV thickened i.e Trivial AR, mild PAH, no clot,PE	DCM	AST/ALT = 1.7 Group 2	35	3	SINUS RHYTHM, Low voltage QRS
73	1140715	36	M	-	-	4 yrs	-	-	No	No	80	110/60	9	B/L NVBS	Soft non tender	11.5	217	7	1.8	1.6	0.5	0.1	5.3	26	30	195	3.6	0.87	0.3	1.05	106	53	1.16	141	3.81	B/L mini pleural effersion	0.84	1.52	0.99	IHD, Hypokinesia of inferior wall and inferoseptum, Borderline resting LV systolic function i.e LVEF = 40%, Trivial MR, Normal PAP, No clot/PE	IHD	AST/ALT = 0.87 Group 1	40	2	SINUS RHYTHM, TWAVES IN LEAD II,III AVF
74	1080141	48	M	-	-	4 yrs	-	-	No	yes B/L grade II	95	140/80	10	B/L basal Crepitation	Soft non tender	10.5	217	7	1.8	1.6	0.5	0.1	5.3	26	30	195	2.6	0.87	0.3	1.05	106	53	1.16	142	3.82	Normal	0.84	1.52	0.99	Global hypokinesia of LV, Severe LV dysfunction with EF - 30%, Mild RV dysfunction {TAPSE 1.4 cm S1 9.0 cm/s}, MAC with Grade II MR, AV thickened i.e Trivial AR, Grade I TR i.e PPG - 41mmHg, Estimated PA Pressure 41+10=51mmHg, moderate PAH, no clot, Rim of PE, Pleural effesion, RA collapsing	DCM, T2DM	AST/ALT = 0.87 (Group 1)	30	3	SINUS RHYTHM, low voltage QRS complex
75	1050652	55	M	10 days	-	2 yrs	-	-	No	No	76	110/80	11	B/L basal Crepitation	Soft non tender	18	173	9.18	2.14	1.46	2.3	0.8	6.4	67	54	74	3.3	1.24	0.97	2.9	106	26	0.7	135	4	Cysititis, B/L mini plueral effersion	0.43	1.2	1	Known case of DCM, Global hypokinesia of LV, Severe LV dysfunction with LVEF - 30%, RV dysfunction {TAPSE 1.3 cm S1 7.4 cm/s}, All chamber dilated, Grade I MR, Grade I TR i.e PPG - 27mmHg, Estimated PA Pressure 27+5=32mmHg, Moderate PAH (ACT - 77ms), no clot, Mild pericardial effusion + lateral to Rv-Rim, towards RA 2.3cm, Right sided Pleural effusion	DCM	AST/ALT = 1.24 Group 2	30	3	SINUS RHYTHM
76	1092926	54	M	-	Chest pain 3 day	4 yrs	3 days	2 months	No	Yes	117	110/70	11	B/L Coarse Crepitation	rmly distended non	10.6	287	12.9	1.23	0.83	0.87	0.62	6	28	62	129	3.5	0.45	0.24	0.7	264	37	0.62	135	5.06	Normal	0.42	1	1.2	Known case of DCM, Global hypokinesia of LV, Severe LV dysfunction with LVEF - 25%, LA, LV, IVC dilated, Grade III MR, Grade III TR i.e PPG - 67mmHg, Estimated PA Pressure 67+15+83mmHg, Severe PAH (ACT - 58ms), no clot/PE	DCM	AST/ALT ratio = 0.45 (Group 1	25	4	SINUS Tachycardia, Broad QRS, LBBB
77	716753	65	M	-	15 days	2 yrs	15 Days		no	no	88	112/70	10	bl nvbs	soft nt	13.9	266	8.5	1.2	1.1	0.2	0	7.1	17	24	142	2.7	0.71	0.16	0.85	268	23	0.83	136	4.06	Enlarged prostate	2.3	0.41	0.23	IHD, Hypokinesia of inferior wall and inferoseptum, Borderline resting LV systolic function i.e LVEF = 40%, Trivial MR, Normal PAP, No clot/PE	IHD	AST/ALT = 0.71 (Group 1)	40	2	SINUS RHYTHM, Q waves in Lead II, III, AVF, T wave inversion V1, V2, V3
78	1053142	62	F	-	-	2 yrs	-	-	No	No	82	110/70	10	B/L NVBS	Soft non tender	14.9	252	13.2	1.04	1.08	2.3	0.8	6.4	67	54	74	3.3	1.24	0.67	2.24	132	16	0.74	135	4.54	Normal	3	0.1	0.12	IHD, Akinesia of anterior wall, anteroseptum, apical septum and apex, Mild LV systolic function i.e EF - 40%, Grade I MR, AV thickened i.e Trivial AR, i.e PPG - 10mmHg, Trivial TR with PPG - 25mmHg, Estimated PA Pressure 25+5=30mmHg, Mild PAH, No clot, No PE	IHD	AST/ALT = 1.24 Group 2	40	2	SINUS RHYTHM, T wave inversion in Lead I, AVL, V5, V6
79	1040343	76	F	-	-	2 yrs	-	-	No	No	88	120/80	11	B/L NVBS	Soft non tender	14.2	220	13.9	1.04	0.83	2.3	0.8	6.4	67	54	74	3.3	1.24	0.76	3.15	233	50	0.83	140	4.58	Left renal calculi	3	1	1	IHD, Dyskinesia of apex with Akinesia of anterior wall, apicolateral segment, Apical septum, Apex, thinned out, Moderate LV dysfunction i.e LVEF - 35-40%, LV Dilated , MAC with Grade I MR, AV thickened with Grade I AR i.e PPG - 10mmHg, Grade I TR i.e PPG - 37mmHg, Estimated PA Pressure 47+5=52mmHg, Moderate PAH, No clot, No PE	IHD, T2DM	AST/ALT = 1.24 Group 2	35	3	SINUS RHYTHM, T wave inversion in Lead I, AVL, V5, V6
80	1034673	67	F	-	-	2 yrs	-	-	No	No	80	110/70	11	B/L NVBS	Soft non tender	11.2	314	9.5	1.15	1.11	2.3	0.8	6.4	67	54	74	3.3	1.24	0.53	1.94	208	43	1.33	131	4.61	Normal	2.6	1.2	1.1	IHD, Akinesia of anterior wall, anteroseptum, apical septum and apex and apicolateral segment, Apical septum, Apex, thinned out, and scarred, Moderate LV dyfunction i.e EF - 35%, LA, LV dilated, MAC with Trivial MR, AV thickened with Grade I AR, Grade II eccentric TR with PPG - 25mmHg, Mild PAH, No clot, No PE	IHD , T2DM	AST/ALT = 1.24 Group 2	35	3	SINUS RHYTHM, T wave inversion Lead I, AVL
81	1152972	59	F	-	-	2 yrs	-	-	No	No	81	110/70	11	B/L NVBS	Soft non tender	10.2	323	10.7	1.1	1.2	0.7	0.3	7.7	35	19	83	3.8	1.84	0.27	1.47	430	16	1.6	134	4.2	Normal	1.2	1	1	IHD Akinesia of anterior wall, anteroseptum, apical septum and apex and apricolateral, Moderate LV dysfunction i.e LVEF - 40%, Moderate MR (VC. 0.4 cm), AV thickened i.e Trivial AR, Grade II eccentric TR with PPG 32 mmHg, Estimated PA Pressure 32+5=37mmHg, Mild PAH, no clot, no PE, Grade I IAs aneenysm	IHD, T2DM	AST/ALT=1.84(GROUP 2)	40	2	SINUS RHYTHM T wave inversion in lead I, AVL, V5, V6
82	1152874	62	M	-	-	3 yrs	-	-	Yes	No	81	110/70	11	B/L NVBS	Soft non tender	9.1	193	6.9	1.07	0.51	8.6	0.7	7.1	24	20	101	3.5	1.2	0.31	1.73	104	30	1.18	136	3.85	Normal	1.1	0.1	0.1	Post PTCA, Dyskinesia of apical septum, apex with Akinesia of interior wall, interolateral, anterior lateral, Apical septum and apex thinned out, moderate LV dysfunction with EF-35%, RV dysfunction {TAPSE 1.3 cm S1 7.4 cm/s} RA, RV, LV, IVC dilated, Trivial MR, AV thickened i.e Trivial AR, Grade III TR with PPG 53 mmHg, Estimated PA Pressure 35+15=69mmHg, Severe PAH, NO Clot/Pericardial effusion	IHD	AST/ALT =1.2(group 2)	35	3	SINUS RHYTHM, T wave inversion in Lead II, III, AVF
83	1152947	23	F	-	-	2 yrs	-	-	No	Yes B/L pitting pedal edema	78	110/70	10	B/L Crepitation	Soft non tender	11	233	11	1.1	1.2	3.24	2.05	7.2	642	314	170	2.6	2.04	6.8	2.3	204	30	1.3	137	2.73	Normal	3	1.1	1.2	Global hypokinesia of LV, Severe LV dysfunction with EF - 30%, RV dysfunction {TAPSE 1.4 cm S1 8.7 cm/s}, LV dilated,Moderate MR (VC=0.5 cm), Grade I TR i.e PPG - 35mmHg, Estimated PA Pressure 35+10=45mmHg, mild PAH, no clot/ PE	DCM	AST/ALT =2.04(group 2)	30	3	SINUS RHYTHM,
84	1038701	56	M	-	-	14 yrs	-	-	No	No	78	110/70	11	B/L NVBS	Soft non tender	12.9	348	8.61	1.72	1.44	0.7	0.3	7.7	35	19	83	3.8	1.84	0.25	1.29	103	43	0.7	130	4.9	Right moderate pleural effusion	2.1	1.2	1	IHD, Akinesia of inferior wall, inferoseptum, inferilateral wall, moderate LV dysfunction i.e LVEF - 40%, LV dilated, Severe MR, Grade II TR i.e PPG - 53mmHg, Estimated PA Pressure 43+10=63mmHg, Severe PAH (ACT - 60ms), No clot/PE	IHD	AST/ALT = 1.84(group 2)	40	2	ATRIAL FIBRILLATION
85	1055960	53	M	-	15 days	4 yrs	-	-	No	No	104	110/70	12	B/L basal Crepitation	Soft non tender	12.9	348	8.61	1.72	1.44	0.7	0.3	7.7	35	19	83	3.8	1.84	0.25	1.29	103	43	0.7	130	4.9	minimal ascities,Right mild pleural effusion	2.47	1.31	1.2	IHD, Akinesia of anterior wall, anteroseptum, apical septum and apex and apicolateral segment, moderate LV dysfunction i.e EF - 35%, LV dilated, Grade II MR, AV thickened i.e Trivial AR, Grade I TR i.e PPG - 35mmHg, Estimated PA Pressure 35+10=45mmHg, Moderate PAH Layered LV clot.	IHD	AST/ALT = 1.84(group 2)	35	3	SINUS RHYTHM, LAD,LAFB
86	1041566	70	M	-	-	2 yrs	15 Days	-	No	No	88	110/70	11	B/L NVBS	Soft non tender	14.3	437	18.9	1.2	1.4	0.56	0.3	7.2	35	18	52	3.4	1.9	0.2	1.32	176	42	1.1	138	4.6	normal	1.5	1	1.2	IHD, Akinesia of anterior wall, anteroseptum, apical septum and apex, apicolateral segment, inferior wall, inferoseptum with hypokinesia of Inferolateral and anteroseptum wall. ANteroseptum, apical septum and apex thinned out, Severe LV dysfunction i.e LVEF - 30%, RV dysfunction {TAPSE 0.8 cm S1 7.1 cm/s}, All chamber dilated, Severe MR, Sclerotic AV with PPG - 12 mmHg with Trivial AR, Grade III TR i.e PPG - 55mmHg, Estimated PA Pressure 55+15=70mmHg, Severe PAH, no clot/ pericardial effusion	IHD	AST/ALT = 1.9 (Group 2)	30	3	SINUS RHYTHM, Low voltage QRS
87	1032897	73	M	-	-	3 yrs	2 months	-	No	No	70	110/60	10	B/L NVBS	Soft non tender	15	152	12.7	2.08	0.86	1.63	0.74	5.5	187	399	83	3.4	0.47	3.08	4.5	88	38	1.35	134	4.6	Normal	2.02	0.8	0.6	IHD, Akinesia of anterior wall, anteroseptum, apical septum and apex, apicolateral segment, inferior wall, inferior septum, apical septum and apex thinned out, Severe LV dysfunction i.e LVEF - 30%, RV dysfunction {TAPSE 1.0 cm S1 8.4 cm/s},LA, LV dilated, MAC with Grade - I MR, Grade I TR i.e PPG -25mmHg, Estimated PA Pressure 25+5=30mmHg, Moderate PAH, no clot, Rim of pericardial effusion	IHD	AST/ALT ratio = 0.45 (Group 1)	30	3	SINUS RHYTHM, T wave inversion I, AVL, V5, V6
88	1051140	68	F	-	-	2 yrs	-	-	No	No	76	110/70	12	B/L NVBS	Soft non tender	11.4	323	6.9	1.07	0.51	8.6	0.7	7.1	24	20	101	3.5	1.2	0.19	1.13	450	47	0.73	134	5.37	Normal	3.12	2.1	1.2	IHD, Akinesia of inferior wall, inferolateral segment with Hypokinesia of inferoseptum, moderate LV dysfunction i.e LVEF - 40%, Grade I MR, AV thickened i.e Trivial AR, Normal PA Pressure, No clot/PE	IHD,T2DM	AST/ALT ratio = 1.2 (Group 2)	40	2	SINUS RHYTHM, low voltage QRS complex
89	6029782	45	M	-	-	2 yrs	-	-	No	No	80	110/70	11	B/L basal Crepitation	Soft non tender	12.3	251	10	1.91	0.99	2.21	0.99	5.5	39	35	66	3.4	1.11	0.39	1.18	108	30	1.03	133	4.56	mild ascities,B/L mini pleural effusion	3.6	1.8	1	ICM, Akinesia of anterior wall, anteroseptum, apical septum and apex, apicolateral segment, inferior wall, inferior septum and inferolateral segment, apical septum and apex thinned out, Severe LV dysfunction i.e LVEF - 25%, RV dysfunction {TAPSE 1.0 cm S1 7.4 cm/s}, All chambers dilated, Grade - II MR, AV thickened i.e Trivial AR, Grade II TR i.e PPG -56mmHg, Estimated PA Pressure 56+10=66mmHg, Severe PAH (ACT - 49ms), LV apical clot measuring 1.2x1.3cm pericardial effusion	IHD	AST/ALT = 1.11 (Group 2)	25	4	SINUS RHYTHM
90	4624158	71	M	-	2 months	3 yrs	2 months	-	No	No	80	100/60	11	B/L basal Crepitation	Soft non tender	13.1	205	9.1	1.03	0.84	1.14	0.63	4.9	18	13	99	2.5	1.39	0.22	1.71	123	17	0.96	136	5.14	Enlarged prostate,bl thin rim of pleural effusion	1.58	2.69	1.57	S/p PTCA and PPI, Akinesia of inferior wall, inferolateral wall, anterior septum, apical septum, apex i.e hypokinesia of anterior wall, moderate LV dysfunction i.e EF - 35%, LV dilated, Moderate MR (VC 0.5 cm), Sclerotic AV i.e PPG - 16mmHg i.e Trivial AR, Grade II TR i.e PPG - 50mmHg, Estimated PA Pressure 50+5=55mmHg, Moderate PAH, No clot, no PE	IHD	AST/ALT = 1.39 (Group 2)	35	3	SINUS RHYTHM, T wave inversion in Lead I, AVL, V5, V6

91	1140727	59	M	-	-	2 yrs	2 months	-	No	No	86	110/80	10	B/L NVBS	Soft non tender	13.9	347	10.2	0.99	0.81	0.29	0.13	5.4	11	24	69	3.4	0.46	0.08	0.38	161	44	1.62	141	4.64	Normal	1.15	2.2	1.37	IHD, Akinesia of inferior wall, inferolateral, infro septum, antrolaterial segment, anteri wall i.e hypokinesia of anteroseptum, apical and apex, Severe LV dysfunction i.e EF -30%, RV dysfunction {TAPSE 1.4 cm S1 8.3 cm/s}, LV dilated, Moderate MR (VC 0.5 cm), AV thickened i.e Trivial AR, Grade II TR i.e PPG - 36mmHg, Estimated PA Pressure 36+5=41mmHg, Moderate PAH (ACT 67ms), No clot, no PE	IHD	IHD, AST/ALT = 0.46 (Group 1)	30	3	SINUS RHYTHM, Broad QRS, LAFB
92	1140733	65	M	3 days	-	3 yrs	-	-	No	No	78	110/70	10	B/L NVBS	Soft non tender	14.3	437	18.9	1.1	1.2	0.56	0.3	7.2	16	23	126	3.7	0.7	0.09	91.04	176	56	1.51	136	4.63	B/L mild pleural effusion	3.12	1.12	1.12	S/P CABG, Akinesia of anterio wall anteroseptum, anterolateral, inferolateral segment i.e hypokinesia of apical septum and apex, Moderate LV dysfunction i.e EF - 35%, LV dilated, Grade II MR, AV thickened i.e Trivial AR, mild PAH (ACT 80ms), No clot, no PE	IHD, T2DM, HTN	AST/ALT =0.7 (Group 1)	35	3	SINUS RHYTHM, Q waves in II, III, AVF
93	1041123	71	M	-	-	3 yrs	2 months	-	No	Yes B/L pitting pedal edema	88	110/70	10	B/L Coarse Crepitation	Soft non tender	13	119	5.3	1.1	0.85	0.38	0.1	6.6	49	36	79	3.7	1.36	1.03	4.87	120	26	0.82	140	5.03	Enlarged prostate	3.2	1.2	1.1	Severe Calcific Aortic Stenosis with PPG/MPG 75.40 mmHg with Grade -I AR, V Max 4.3 ms, Aortic annulus 1.5 cm, Global hypokinesia of LV, Severe LV dysfunction with LVEF - 30%, RV dysfunction {TAPSE 1.3 cm S1 6.7 cm/s}, LV, LA dilated, MAC with Grade III eccentric MR, Grade II TR i.e PPG - 57mmHg, Estimated PA Pressure 57+5=63mmHg, Severe PAH, no clot/ PE	DCM	AST/ALT =1.36(Group 2)	30	3	SINUS RHYTHM
94	1030165	66	F	-	-	3 yrs	2 months	-	No	yes B/L grade II	76	110/70	11	B/L NVBS	Soft non tender	10.4	277	10.5	0.99	0.87	0.64	0.15	8	20	19	84	3.7	1.05	0.18	1.08	224	30	1.2	134	4.5	Normal	3	0.1	0.2	IHD, Akinesia of anterior wall, anteroseptum, inferior septum, apical septum and apex apicolateral segment with hypokinesia of inferior wall, mid anteroseptum, apical septum and apex thinned out, Moderate LV dysfunction i.e LVEF - 35%, RV dysfunction {TAPSE 1.1 cm S1 8.4 cm/s}, LV dilated, Grade - II MR, AV thickened i.e Trivial AR, Grade II TR i.e PPG -66mmHg, Estimated PA Pressure 66+10=76mmHg, Severe PAH, no clot, Rim of pericardial effusion	IHD, T2DM	AST/ALT = 1.05(group 2)	35	3	SINUS RHYTHM, T wave inversion in Lead I, AVL, V5, V6
95	1148349	68	M	-	-	3 yrs	2 months	-	No	No	88	110/70	11	B/L NVBS	Soft non tender	13.9	119	6.3	1.1	0.85	0.38	0.1	6.6	49	36	79	3.7	1.36	1.03	4.7	120	26	0.82	140	5.03	Normal	3.49	2.48	1.28	Global hypokinesia of LV, Severe LV dysfunction with EF - 30%, RV dysfunction {TAPSE 1.4 cm S1 8.7 cm/s}, LV dilated,Moderate MR (VC=0.5 cm), Grade I TR i.e PPG - 35mmHg, Estimated PA Pressure 35+10=45mmHg, mild PAH, no clot/ PE	DCM, T2DM.	AST/ALT = 1.36 (Group 2)	30	3	Absent P wave, Irregular R-R interval, Atrial fibrillation
96	1013498	67	F	-	-	3 yrs	2 months	-	No	yes B/L grade II	101	110/70	11	B/L NVBS	Soft non tender	14.7	187	7.8	1.16	0.88	0.71	0.29	6.5	16	15	61	3.8	1.07	0.21	1.48	128	22	0.64	145	3.4	B/L mild pleural effusion	2.34	1.6	1.2	IHD, Akinesia of anterior wall, anterioseptum, apical septum and apex apicolateral mid septum with hypokinesia of infero septum, mid anteroseptum, apical septum and apex thinned out, Moderate LV dysfunction i.e LVEF - 35%, RV dysfunction {TAPSE 7.4 cm S1 8.6 cm/s}, LV, LA dilated, Grade - III MR, AV thickened i.e Trivial AR, Grade III eccentric i.e PPG -53mmHg, Estimated PA Pressure 53+20=73mmHg, Severe PAH (ACT - 49ms), no clot/ pericardial effusion	IHD, T2DM	AST/ALT =1.07 (Group 2)	35	3	SINUS RHYTHM, Left axis deviation LAFB
97	6391471	21	F	-	-	2 yrs	2 months	-	No	yes B/L grade II	88	110/70	10	B/L NVBS	Soft non tender	11.7	188	11.4	1.06	1.17	1.5	0.5	7.1	22	9	173	3.3	2.44	0.29	0.82	76	26	0.7	137	3.9	unbilical hernia	1.17	2.38	1.91	Tachcardic noted on echo (HR -119bpm), Global hypokinesia of LV, Severe LV dysfunction with EF - 30%, RV dysfunction {TAPSE 1.5 cm S1 8.4 cm/s}, Trivial MR, Trivial TR i.e PPG - 23mmHg, Normal PA Pressure, Large LV layered apical clot, No PE	DCM	AST/ALT = 2.44(Group 2)	30	3	Atrial fibrillation
98	1022665	74	M	-	-	3 yrs	-	-	NO	NO	70	112/60	11	BL NVBS	SOFT NT	11.5	437	18.9	1.2	1.4	0.56	0.3	7.2	35	18	52	3.4	1.9	0.2	1.4	176	42	1.1	138	4.6	Normal	3	1	1	Post CABG, + MV repair, Mitral value ring in sltu, MVO of 2.3 - 2.4 cm with PPG/MPG 8/3mmHg with Grade I MR, Akinesia of inferior wall, inferolateral wall i.e hypokinesia of inferior septum, Moderate LV dysfunction i.e EF - 40%, MV mildly dilated, RA, RV and LA dilated, Mild RV dysfunction, Grade - II TR PPG - 53mmHg, Estimated PA Pressure 53+15=68mmHg, Severe PAH, no clot/ pericardial effusion	IHD, T2DM, HTN	AST/ALT =1.9 Group 2	40	2	SINUS RHYTHM, q waves in lead ii, III, AVF
99	1140610	69	M	3 days	-	3 yrs	-	-	No	No	76	120/70	12	bl nvbs	soft nt	11.7	277	10.5	0.99	0.87	0.64	0.15	8	20	19	84	3.7	1.05	0.18	1.14	112	30	1.5	134	4.6	Normal	1.2	0.4	0.2	IHD, Akinesia of anterior wall, anteroseptum, apical septum and apex, apicolateral segment, inferior wall, inferior septum, apical septum and apex thinned out, Severe LV dysfunction i.e LVEF - 30%, RV dysfunction {TAPSE 1.0 cm S1 8.4 cm/s},LA, LV dilated, MAC with Grade - I MR, Grade I TR i.e PPG -25mmHg, Estimated PA Pressure 25+5=30mmHg, Moderate PAH, no clot, Rim of pericardial effusion-	IHD	AST/ALT =1.05 (Group 2)	30	3	Normal SINUS RHYTHM
100	1038787	75	M	5 days	-	5 yrs	-	-	No	No	81	110/70	11	B/L NVBS	Soft non tender	13.4	437	8.9	1.2	1.4	0.56	0.3	7.2	35	18	52	3.4	1.9	0.2	1.4	176	42	1.1	138	4.6	Normal	2.1	1.6	1.4	Tachcardic noted on echo, Global hypokinesia of LV, Moderate LV dysfunction with EF - 35%, RV dysfunction {TAPSE 1.2 cm}, RA,RV, PA dilated and LA mildly dilated, Grade III MR, AV thickened i.e Grade I AR, Grade III TR i.e PPG -53mmHg, Estimated PA Pressure 53+20=73mmHg, Severe PAH, No clot, No PE	DCM,T2DM,HTN A	AST/ALT = 1.9 (Group 2)	35	3	SINUS RHYTHM