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Study of clinical, Electrocardiographic and 2D-Echocardiography findings in Chronic kidney disease patients

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Abstract
Background: *Cardiovascular abnormalities commonly encountered in patients with chronic kidney disease (CKD) include left ventricular hypertrophy (LVH), left ventricular dilatation, and left ventricular systolic and diastolic dysfunction. Echocardiography should be performed early in the course of CKD and may be valuable in the monitoring of therapy of the patients.*
Aims and objectives: *The present study was conducted to study cardiac disorders using clinical, Electrocardiographic and 2D-Echocardiography findings in Chronic kidney disease patients.*
Material and methods: *The study was performed on 70 patients at Baba Saheb Ambedkar Medical College, Delhi. Electrocardiography was done to look for various changes like p wave morphology, PR interval, QRS complex, T wave morphology, ST-segment, QT interval etc. and to interpret the positive findings for the study. 2D Echocardiography was done for the structural and functional changes in the heart in the study group along with history clinical examination and laboratory investigations.*
1. ***Results****: In the 2D echo, 48.5% of patients had LVDD and 15.7% of patients had LVSD and mitral regurgitation was found 15.7%. Highest LVH was recorded 48.5% in 2D echo. LVH was found in 23 (32.8%) patients which was highest than other finding of ECG. Ischemia was 17.2%, LAD was found 11.4% while LBBB, RAS and RBBB was found 7.14%, 5.7% and 4.3% respectively in ECG, Among the patients with LVH(Left ventricular hypertrophy) in ECG, Diabetes(DM) and Hypertension (HTN) both co morbidities were present in 63.64% and only hypertension was found 36.36% patients.*
2. Among patients with LVH(left ventricular hypertrophy) in Echocardiography, diabetes and Hypertension both co morbidities were found in 56.5% and only diabetes and only hypertension was found 21.73% separately.
3. Shortness of breath was found 32.9%, body swelling was found 32.9% and generalized weakness was found 28.6% as the major symptoms at presentation.
4. Pedal edema was present in 90% at presentation, raised blood pressure was found 55.7%, pallor and cardiomegaly(on chest x ray) was Present in 54.3% patients.

Conclusion: *Most common ECG abnormality was Left ventricular hypertrophy(LVH). Left ventricular hypertrophy was also common in echocardiography along with left ventricular diastolic dysfunction , echocardiography is a more sensitive diagnostic procedure to detect left ventricular hypertrophy. Conduction abnormality is common in CKD patients. Pericardial effusion was less frequent in the study population , availability of hemodialysis can be an important factor for this. Hypertension and diabetes were common etiologic causes in the study population. Anaemia and electrolyte abnormalities were frequently seen in these patients. Most of the patients presented with body swelling/edema and shortness of breath. Cardiovascular abnormalities were clearly more than normal population.*

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I. Introduction

In healthy individual, the volume and composition of body fluids vary within normal limits and the kidneys are largely responsible for maintaining this state. The kidneys also subserve a host of metabolic and endocrine functions. Chronic kidney disease is a pathophysiological process with multiple etiologies, resulting in inexorable attrition of nephron number and function leading to end stage renal disease. End stage renal disease is a clinical condition in which there has been an irreversible loss of endogenous renal function, of a degree sufficient to render the patient permanently dependent upon renal replacement therapy.

Chronic kidney disease (CKD) is defined according to KDIGO (Kidney Disease Improving Global Outcomes) 2012 guidelines as a functional or structural kidney abnormity, lasting for at least 3 months. CKD could be classified according to etiology, glomerular filtration rate and/or albuminuria.[1]

According to epidemiological data, CKD G3-G5 affects approximately 10% of adult population. Therefore, it is a state with serious medical, social and economic consequences. The most frequent CKD causes include especially type 2 diabetes mellitus, vascular disease (hypertension and renal artery disease), primary and secondary glomerulopathy or polycystic kidney disease in the Western countries.[2] The age adjusted cardiovascular complications and mortality is about 30 times higher in end stage renal disease than in general population.[3] Besides the traditional risk factors like age, gender, etc there are many risk factors specific to chronic kidney disease like anemia, hyperparathyroidism, hyperhomocysteinemia, proteinuria, hypoalbuminemia, activated renin angiotensin system which contribute to cardiovascular disease.

Cardiovascular abnormalities commonly encountered in patients with CKD or ESRD include left ventricular hypertrophy (LVH), left ventricular dilatation, and left ventricular systolic and diastolic dysfunction. Uremic Cardiomyopathy is thought to be the pathological cardiac hypertrophy, indicating the influence of impaired renal function on the myocardium. It is the result of pressure overload, volume overload, and the uremic state itself. LV pressure overload occurs frequently from hypertension and arteriosclerosis, and occasionally from aortic stenosis; LV volume overload occurs as a result of the presence of an arteriovenous fistula, anemia, and hypervolemia.[4]

There are several possible explanations for poor prognosis of CKD patients, including traditional cardiovascular risk factors (i.e., hypertension, diabetes mellitus, and dyslipidemia), nontraditional factors (e.g., malnutrition, inflammation, and oxidative stress), and CKD-related risk factors (e.g., atherosclerosis ,anemia, altered calcium phosphate metabolism).8e15 These factors may contribute to the development and deterioration of the coronary artery disease (CAD), microvasculopathy, valvulopathy, cardiomyopathy, and arrhythmias.[5]

Manjunath (2003)[6] et al. reported that patients with GFR ≤59 mL/min/1.73 m2 have a 38% higher risk of cardiovascular disease (CVD) development as compared to those having GFR ≥90 mL/min/1.73 m2. CVD is associated with >50% of the deaths in CKD patients. Patients of CKD having CVD had three to thirty times higher risk of mortality as compared to the general population.[7] In addition, mortality among cardiovascular patients has been found to be twofold higher in CKD stage 2 patients and threefold higher in patients with stage 3 CKD, when collated to patients with normal renal function.[8]

CKD increases cardiovascular morbidity and mortality even in the milder stages. In end-stage renal disease (ESRD) patients, the mortality is approximately 10 times higher than in age-matched controls. The understanding of the cardiovascular disease etiopathogenesis is a mandatory step in the attempt to lower ESRD mortality. Echocardiography has a pivotal role similarly to other cardiac diseases.[9]

Cardiac abnormalities, especially abnormal left ventricular (LV) geometry and functions, are frequently detected in CKD patients and have been proven to be correlated with high cardiovascular mortality/morbidity and all-cause mortality.[10,11] This increased risk of CVD may begin during early stage of CKD much before the onset of kidney failure. This high burden of CVD mortality is well illustrated by comparing CVD mortality in dialysis population to general population. The mortality due to CVD is 10-30 times higher in dialysis patients.[12] Early identification of such high-risk patients should thus allow physicians to optimize the therapeutic interventions, which may lower morbidity and mortality.[13] Echocardiography should be performed early in the course of CKD and may be valuable in the monitoring of therapy of these patients.[14]

These phenomena increase vulnerability to increase electrical excitability, leading to sudden cardiac death among these patients.[15] Echocardiography is a gold standard diagnostic modality for the determination of cardiac structural and functional abnormalities. Therefore, the evaluation of echocardiographic parameters in patients of CKD can help to determine the risk and prognosis of CVD in patients of CKD.[16] In the present study, we evaluated the echocardiographic findings in patients of CKD.

II. Aims And Objectives

AIM-

To study cardiac dysfunction in chronic kidney disease patients.

Primary objective-

To find out electrocardiographic and echocardiographic changes in chronic kidney disease patients.

Secondary objective-

To find out the changes which are consistently more common to most chronic kidney disease patients.

III. Review Of Literature

CHRONIC KIDNEY DISEASE:

The kidney has such considerable functional reserves that in healthy men reduction of renal mass by half cause no disturbances of clinical importance. However when renal mass is reduced further, excretion of waste products of metabolism cannot be achieved without their levels rising in serum and when sufficient nephron loss occurs, control of many physiological processes become inadequate and symptoms and abnormal physical signs results.

Chronic kidney disease is a pathophysiological process with multiple etiologies, resulting in inexorable attrition of nephron number and function leading to end stage renal disease. End stage renal disease is a clinical condition in which there has been an irreversible loss of endogenous renal function, of a degree sufficient to render the patient permanently dependent upon renal replacement therapy (dialysis or transplantation) in order to avoid life threatening uremia. Uremia is the clinical and laboratory syndrome, reflecting dysfunction of all organ systems as a result of untreated or undertreated chronic renal failure.

STAGES OF CHRONIC KIDNEY DISEASE[17]

A widely accepted international classification divides chronic kidney disease into a number of stages defined by clinical estimation of glomerular filtration rate.

Stages	Description	GFR
	At increased risk	90
1	Kidney damage with normal or increased GFR	90
2	Kidney damage with mildly decreased GFR	60-89
3	Moderately decreased GFR	30-59
4	Severely decreased GFR	15-29
5	Renal failure	<15

PREVELANCE:

The Third National Health and Nutrition Examination Survey (NHANES III) revealed following finding related to CKD.[18]

- 6.2 million individuals had a serum creatinine equal to or greater than 1.5 mg per dL, which is a 30-fold higher prevalence of reduced kidney function compared with the prevalence of treated ESRD during the same time interval.
- 2.5 million individuals had a serum creatinine equal to or greater than 1.7 mg per dL.
- 800,000 individuals had a serum creatinine equal to or greater than 2.0 mg per dL.
- Of individuals with elevated serum creatinine, 70% have hypertension.
- Only 75% of patients with hypertension and elevated serum creatinine received treatment, with only 27% having a blood pressure (BP) reading lower than 140/90 mm Hg and 11% having their BP reduced to lower than 130/85 mm Hg.[19]

PATHOPHYSIOLOGY OF CARDIOVASCULAR DISEASES IN CHRONIC KIDNEY DISEASE:

In addition to traditional risk factors which are frequently present in patients with chronic kidney disease, there are few more risk factors specific to chronic kidney disease which contributes to the increased burden of cardiovascular diseases.

In chronic kidney disease, compensatory and adaptive mechanisms maintain acceptable health until the GFR is about 10-15ml/min and life sustaining the renal excretory and homeostatic function continue until the glomerular filtration rate is less than 5ml/hr.[20]

The metabolic product of protein and amino acids depends primarily on the kidneys for excretion unlike fats and carbohydrates, which are eventually metabolised to Co2 and water substance, which are readily excreted even in uremic subjects via lungs and skin.

The pathophysiology of the uremic syndrome is divided into those sets of abnormalities consequent to the accumulation of end products of protein metabolism and the abnormalities consequent to the loss of fluid and electrolyte homeostasis and synthesis of certain hormones{e.g.:- erythropoietin EPO, 1.25-dihydroxycholecalciferol}.

The kidney normally catabolizes a number of circulating plasma protein and polypeptide which is reduced in renal failure. Furthermore, plasma levels of parathyroid hormone {PTH}, Insulin, Glucagon, Luteinizing hormone and prolactin hormone rise with renal failure-not only because of renal catabolism but also

because of enhanced glandular secretion. PTH is an important toxin because of its adverse effect of elevating cellular cytosolic Ca2+ levels in several tissue and organ.[21]

CARDIOVASCULAR COMPLICATION OF CHRONIC KIDNEY DISEASE:

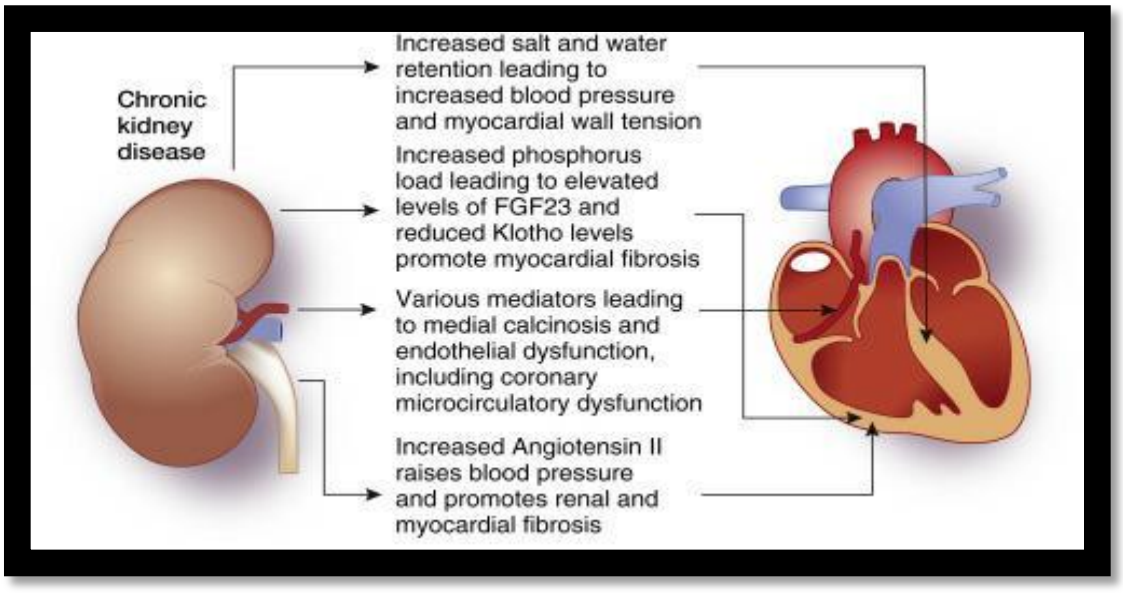
Cardiovascular diseases are a leading cause of death in end stage renal disease largely as a result of the progressively increasing age of ESRD patient and the broad constellation of uraemia associated factors that can adversely affect cardiac function. This increased risk of cardiovascular disease may begin during the earlier stages of CKD before the onset of kidney failure. Notably, patients with CKD have a very high prevalence of cardiovascular disease risk factors such as diabetes and hypertension, but they are also exposed to other non-traditional, uraemia-related cardiovascular disease risk factors.[22]

Cardiovascular disease (CVD) is the leading cause of mortality, accounting for nearly 45% of deaths; approximately 20% of cardiac deaths are attributed directly to acute myocardial infarction. At all ages in both men and women, mortality due to CVD is 10 to 30 times higher in dialysis patients comparing to general population.[23]

The anatomic and hemodynamic alteration of cardiovascular system in CKD is

- Increased total body and vascular volume.
- Increased blood pressure
- Left ventricular hypertrophy.
- Increased pulmonary capillary permeability.
- Increased cardiac index.
- Increased left ventricular chamber size.
- Increased serosal membrane permeability.
- Increased total peripheral resistance.
- Impaired left ventricular contractile function {decreased ejection fraction}.

Image showing pathogenesis of cardiovascular changes in CKD



TRADITIONAL RISK FACTORS FOR CARDIOVASCULAR DISEASES

- Hypertension
- Older age
- Diabetes mellitus
- High LDL Physical inactivity
- Low HDL
- Smoking
- Left ventricular hypertrophy

CHRONIC KIDNEY DISEASE RELATED RISK FACTORS[24]
SPECIFIC TO CKD

- Blood pressure
- Anemia
- Calcium phosphate
- Sodium retention
- Hypervolemia
- Hypoalbuminemia
- Angiotensin II
- Aldosterone
- Depression and sleep disorders

EMERGING RISK FACTORS

- ↑ Homocysteine
- ↑ Nitric oxide synthesis
- ↑ Lipoprotein (a)
- ↑ Insulin resistance

For every increase of systolic blood pressure by 5 mm of Hg and every 10 year rise in age, there is an increase in the risk of left ventricular hypertrophy by 11 and 25 percent respectively. The male gender has an increased risk by 40 percent.

PATHOPHYSIOLOGY OF CARDIOVASCULAR ABNORMALITY

Left ventricular hypertrophy

LVH is highly prevalent in both stages 3 and 4 CKD and dialysis patients and represents a physiologic adaptation to a long term increase in myocardial work requirements.[25]

Pathogenesis

LVH may be thought of as resulting from either pressure or volume overload. Pressure overload results from increased cardiac after load, often due to hypertension, aortic stenosis, and reduced arterial compliance from arteriosclerosis. Volume overload may be related to anaemia, developing when the heart attempts to compensate for decreased peripheral oxygen delivery.[26]

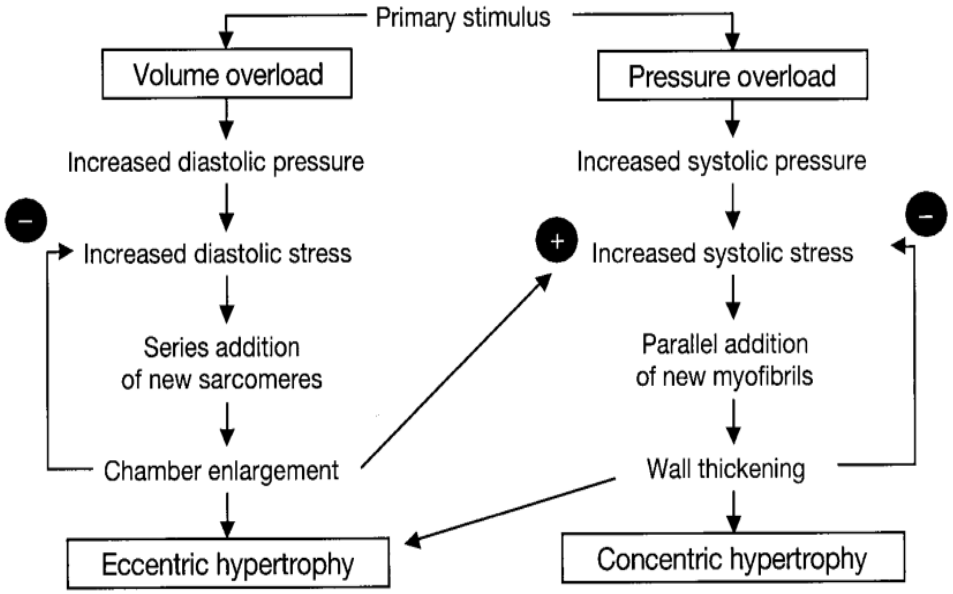


FIGURE : PATHOGENESIS OF LVH

An Indian study in the year 2003 done by Dhangri P and his colleague in 60 patients divided into 2 groups of 30 each into mild to moderate chronic kidney disease (S. Creatinine 1.5-6.0 mg/dl) and advanced chronic kidney disease(S. Creatinine >6.0mg/dl) found in mild to moderate group 40% patient had LVH, and in advanced group 97% of patients had LVH.

Another study done in India by Kale SA (2001)[27] and his friends in a prospective study including 161 patients from 1997-1999 with mean age group of patient being 40.57 found left ventricular disease was found in 105(65.2%) patients. Systolic dysfunction was noticed in 42(37.8%) patients. Left ventricular hypertrophy was seen in 88 (54.7%) patients.

Robert in Canada in the year 2000 on 227 patients had found cardiac failure at inception of dialysis, while 14 patients developed cardiac failure in the first year of dialysis therapy. 36 of remaining 137 patients developed new onset cardiac failure after 1 year. In this group, LV mass index had increased on average by 17g/m2, compared with mean change of 0g/m2 in those remaining free of cardiac failure after 1 year.[28]

Cardiomyopathy (Systolic or Diastolic Dysfunction)

The prevalence of systolic or diastolic dysfunction, or overt LVH, is estimated to be at least 75% at dialysis initiation. De novo and recurrent heart failure occurs in a substantial proportion of patients on dialysis, hence increase in its impact both on morbidity and mortality, as well as the ability to deliver adequate dialysis.[29]

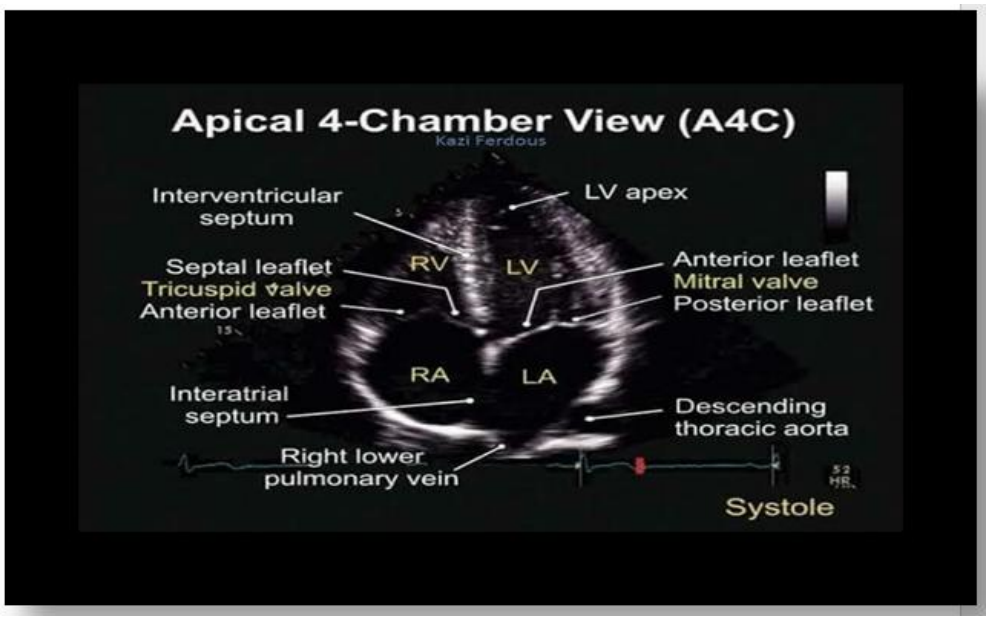
Cardiomyopathy results from both LV pressure overload and LV volume overload. Hypertension causes LV pressure overload and induce concentric LV hypertrophy, with increased myocyte thickness. Salt and water overload anaemia and arterio-venous fistula cause eccentric LV hypertrophy with increase in myocyte length and increase in LV volume. This arteriosclerosis induces further LV hypertrophy by diminished arterial distensibility, enhanced arterial reflections and consequent increased pulsatile work by the heart.

A study from United Kingdom done by Graham and his colleague in the year 2004 on 296 patients found progressive increase in LVH with loss of renal function, so that 80% of patients on renal replacement therapy have LVH, the dominant pattern being concentric LVH . The prevalence of diastolic dysfunction increased in parallel with changes in left ventricular mass but systolic dysfunction and ventricular mass did not.[30]

ECHOCARDIOGRAPHY

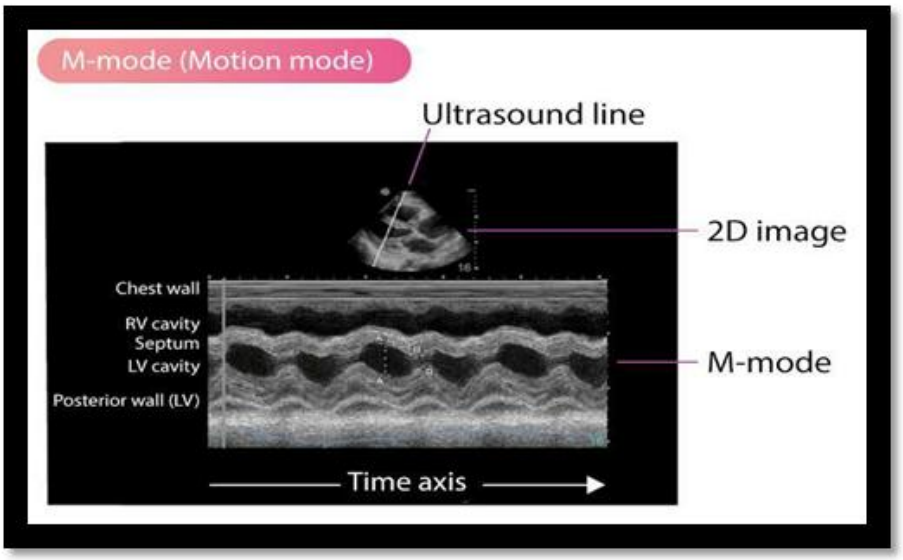
Marchand and others demonstrated hearts electrical activity during last half of 19th century, closely followed by direct recording of cardiac potentials by Walles in 1887. Invention of string Galvanometer by William Einthoven in 1901 provided a reliable and direct method for registering electrical activity of the heart. By 1910 string alvanometer become very popular in clinical practice. Subsequent ECG became the first and most common bioelectric signal to be computer processed and most commonly used cardiac diagnostic test.[31]

- Roman architect 'vitruvius' first coined the word "ECHO".
- Abbe Lazzaro Spallanzani is refered as father of ultrasound.
- Karl Dussik was first to apply ultrasound for medical diagnosis in 1941.
- Edler and Hertz of Sweden were the first to record the movements of cardiac structures by the ultrasound.[32]



- The present day standard instruments are developed by Thurston and Von Ramm.[33]

Echocardiography is one of the frequently used techniques for diagnosing cardiovascular diseases. It is so versatile, with clinical application in the entire spectrum of cardiovascular diseases. Echocardiography uses high frequency ultrasound to evaluate the structural, functional and hemodynamic states of cardiovascular diseases.
An echocardiographic examination begins with trans thoracic two dimensional (2D) scanning from four standard transducer positions: the parasternal, apical, subxiphoid and suprasternal windows. Quantitative measurements of cardiac dimensions, area and volume are derived from 2D images or 2D derived M-mode. In addition, 2D Echocardiography provides the framework for Doppler and color-flow imaging.



ELECTROCARDIOGRAPHIC CHANGES

Patients on maintenance dialysis therapy are at increased risk for dysrhythmias, cardiac arrest, and SCD. Dialysis patients with underlying structural or functional CVD are at much higher risk for these dysrhythmias and cardiac arrest because of increased dysrhythmogenicity due to dynamic changes in electrolytes, volume status, blood pressure and the use of multiple medications. Even nondiabetic dialysis patients have a markedly increased cardiac event rate and decreased event-free survival as compared to the general population.[34]

Ischemic heart disease is present in many patients even at the time of initiation of dialysis. CKD Stage 5 patients with either symptomatic or asymptomatic coronary artery disease are at increased risk for dysrhythmias and SCD. This risk is potentiated with concomitant presence of anaemia and left ventricular hypertrophy or increased left ventricular mass index, often present in CKD patients at the initiation of dialysis therapy.

The prevalence of baseline ECG abnormalities and the development of new dysrhythmias and silent myocardial ischemia is related to the concomitant presence of CAD, and is also directly proportional to the duration of dialysis. Potentially lifethreatening ventricular dysrhythmias and silent myocardial ischemia has been noted.[35]

Risk factors for increased arrhythmogenicity include compromised myocardium (due to either underlying CAD, decreased coronary reserve blood flow, or the consequences of uraemia on myocardial function and structure), increased QTc interval or dispersion, electrolyte abnormalities, intradialytic hypotension, concomitant presence of LVH (present in most of the patient on dialysis), and autonomic dysfunction (with or without diabetes).

Dialysis patients have frequent electrolyte abnormalities such as fluctuating levels of potassium, ionized calcium, magnesium, and other divalent ions. Due to the intermittent nature of the dialysis procedure, patients on HD have wide fluctuations in volume status, and potassium and bicarbonate levels, in between dialysis treatments.

These fluctuations are partly driven by the level of potassium and calcium in the dialysate fluid used during the prior session of treatment, and wide variability in eating habits due to varying adherence to dietary modifications necessary to control the calcium-phosphate product. All these factors culminate in and dysrhythmogenic diathesis.[36]

Soman and his colleague divide 9,554 patients into 5 groups based on creatinine clearance, where mean age group of patients being 63.4±13.8 years and they observed atrial fibrillation in 4.1 to 9.4%, complete heart block in 0.7-3.5%, RBBB in 3.5-7.1%, LBBB in 3.6-8.3% and LVH in 7.1-18.5%. Unstable angina was documented 33.2%- 39.9% in different groups of chronic kidney disease.[37]

The chronic renal insufficiency cohort study done by Soliman and his friend in on 3,267 patients with mean estimated glomerular filtration rate of 43.6±13.0 found atrial fibrillation as the most common arrhythmia in CKD patients, nearly one in every five participants had evidence of atrial fibrillation.[38]

The Electrocardiographic manifestation of the left ventricular hypertrophy due to systolic overload[39]

1. Abnormalities of the QRS complex
- Increased magnitude of QRS deflection.
- Attenuation of the small q wave in left oriented leads.
- An increased time in left ventricular activation time.
- A small equiphasic complex in lead AVF.
- Counter clockwise electric rotation.

2. Abnormalities of ST segments and t waves

T wave may be inverted in lead V5 , V6 lead I and AVL, upright in right oriented leads. The associated ST segment in the left oriented leads is usually minimally depressed. This is an indication of hypertrophied left ventricular strain probably the expression of relative ischemia.

3. Inversion of u wave in the left precordial leads.
4. Left atrial enlargement.
5. Abnormality of the QRS and T wave.

Early and uncomplicated left ventricular hypertrophy, causes no change in the directional of the mean plane QRS vector. With relatively long standing left ventricular hypertrophy, the QRS axis begin to deviate to the left side.

The mean frontal T wave vector is directed to the right reflecting very compromised left ventricle.

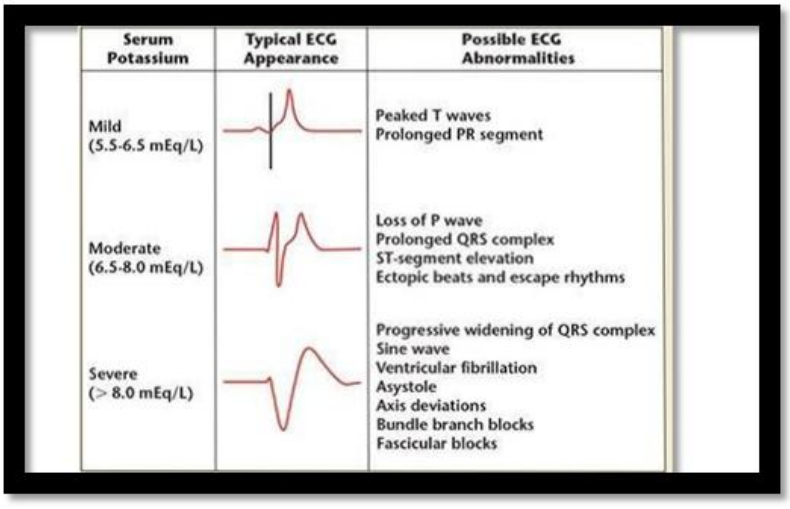
The Electrocardiographic manifestation of diastolic overload.

1. Tall R waves in the left oriented leads.
2. Deep narrow Q waves in the left oriented leads.
3. Relatively tall symmetrical T waves in the left precordial leads.
4. Minimally elevated S-T segment in the left precordial leads.
5. Inverted U waves in the left precordial leads.

The Electrocardiographic manifestation of the cardiomyopathy

The ECG of the dilated cardiomyopathy often shows sinus tachycardia, or atrial fibrillation, ventricular arrhythmias, left atrial abnormalities, diffused non-specific ST-T wave abnormalities and sometimes intraventricular conduction defect and low voltage.

Effect of electrolyte disturbance on ECG



Uraemia presents with manifestation of hypocalcaemia and associated with hyperkalaemia and or acidosis. The hypocalcaemia causes a prolong ST segment and there by the QT interval. The hyperkalaemia causes tall T waves.The prolongation of QT interval is inversely proportional to the level of serum calcium. QT interval in hyperkalaemia is either normal or actually decreased. If the hyperkalaemia is associated with hypocalcaemia the QT interval may be prolonged. The QT interval is the interval beginning from the Q wave to the end of T wave. The QT interval shortens with tachycardia and prolong with bradycardia. It is thus evident that QT interval cannot be viewed in absolute term but must be corrected to the effect of associated he heart rate.
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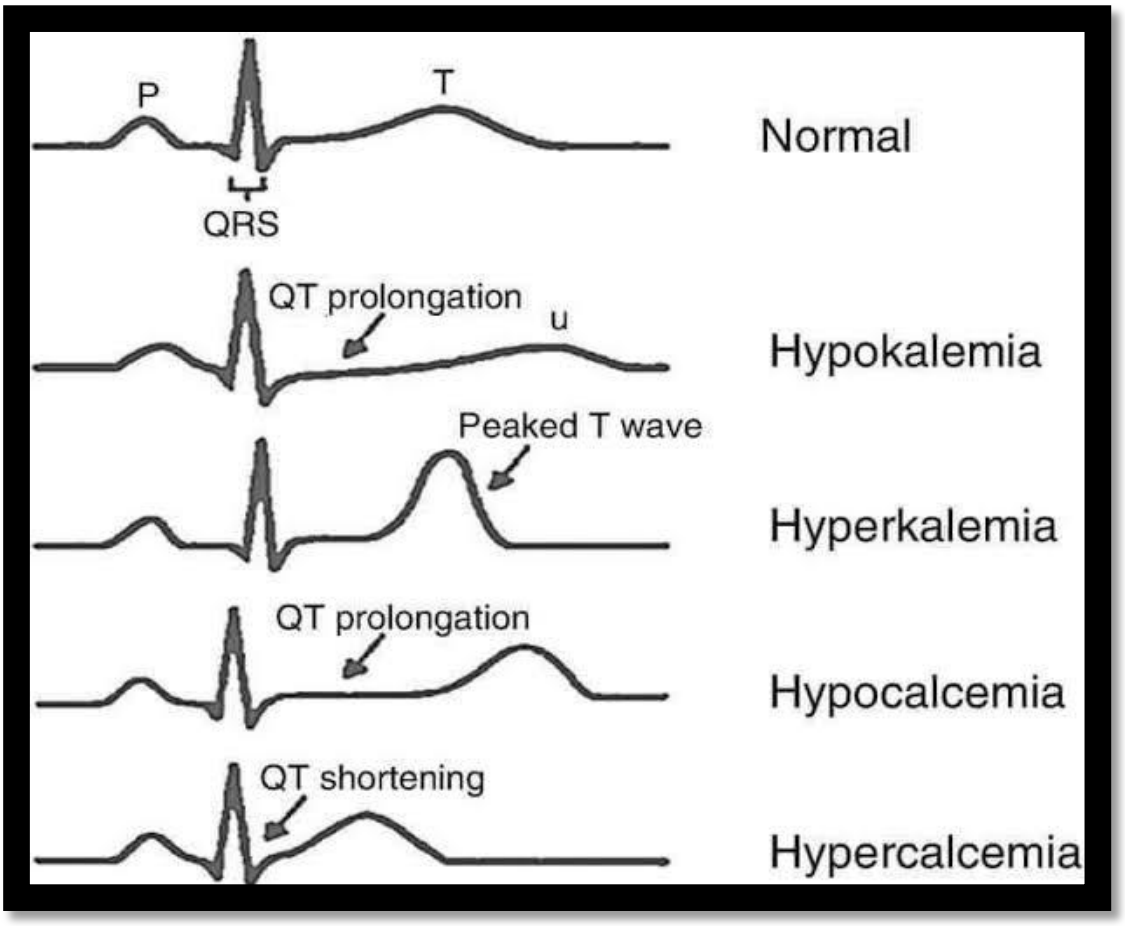


Drechsler C (2009)[40] et al in his study found atrial fibrillation in 8 to 12% of patients, signs of MI in 13 to 17%, LVH in 12 to 14%, ventricular conduction defect in 7 to 8% of patients in CKD patients with different ranges of HBA1c.

Graham (2005)[41] with his colleague observed increased QT interval and QT dispersal were associated with poor renal function. Arrhythmia were more common with poor renal function and in presence of uremic cardiomyopathy proven by echocardiogram.

IV. Literature Review

Barde R (2015)[42] et al assessed and analysed the echocardiographic changes in chronic kidney disease patients on maintenance hemodialysis. They have performed prospective study of echocardiographic changes in chronic kidney disease (CKD) patients undergoing on maintenance hemodialysis at their institute. They performed M-mode echocardiography in 100 CKD patients without obvious clinical evidence of coronary artery disease, Valvular heart disease, congenital heart disease.100 Patients Undergoing Hemodialysis were included. Of them Echocardiography finding showed LV dilation and diastolic dysfunction in 49(49%), left ventricular hypertrophy (LVH) in 57 (57%), systolic dysfunction and pericardial effusion in 28(28%) and 13(13%) patients respectively. RWMA was present in 10% and Valvular calcification was seen in 5 patient. In sub-group of patients with Hb<10 gm%, LVH was present in 77.41% (48) vs 23.68% (9) in patient group with Hb ≥ 10 gm% ($p < 0.01$). Other Sub Group of Patients with BP > 140/90mmhg, LVH Was Present in 64.47 %(49**)** vs 33.33 %(8) in patients group with BP< 140/90 mm hg ($p=0.02$). In both sub group p value for systolic dysfunction, RWMA & pericardial effusion was statistically not significant. They have shown that LV diastolic dysfunction

and hypertrophy were most common echocardiographic findings. There was statistically significant correlation between anemia and presence of LVH and positive correlation between presence of hypertension and LVH.

Krishnasamy R (2015)[43] et al investigated the prognostic value of GLS over EF in patients with advanced Chronic Kidney Disease (CKD). The study included 183 patients (57% male, 63% on dialysis) with CKD stage 4, 5 and 5Dialysis (D). 112 (61%) of patients died in a follow up of 7.8 ± 4.4 years and 41% of deaths were due to cardiovascular (CV) disease. GLS was calculated using 2-dimensional speckle tracking and EF was measured using Simpson's biplane method. Cox proportional hazard models were used to assess the association of measures of LV function and all- cause and CV mortality. The mean GLS at baseline was -13.6 ± 4.3% and EF was 45 ± 11%. GLS was a significant predictor of all-cause [Hazard Ratio (HR) 1.09 95%; Confidence Interval (CI) 1.02–1.16; p =0.01] and CV mortality (HR 1.16 95%; CI 1.04–1.30; p = 0.008) following adjustment for relevant clinical variables including LV mass index (LVMI) and EF. GLS also had greater predictive power for both all- cause and CV mortality compared to EF. Impaired GLS (>-16%) was associated with a 5.6-fold increased unadjusted risk of CV mortality in patients with preserved EF. It was concluded that this cohort of patients with advanced CKD, GLS was a more sensitive predictor of overall and CV mortality compared to EF.

Goornavar SM (2015)[44] et al studied to identify the Echocardiographic changes in patients with CKD and to know the prevalence of each Echocardiographic change in CKD. A total of 50 CKD patients admitted to S. Nijalingappa Medical College Hospital from January 2013 to December 2013, were included in the study. The patients were evaluated as per the history, general physical examination, systemic examination, Blood Urea, Serum Creatinine, Urine Routine, Electrocardiograph (ECG) and Echocardiography. In the present study echocardiographically determined cardiovascular abnormalities were observed in 86% of patients. Left Ventcular Hypertrophy (LVH) in 36% patients. Ischemic heart disease (IHD)16%, LVH and Ischemic Heart Disease in 22%, dilated cardiomyopathay in 4.0%, Pericardial effusion in 6.0%, Septal hypertrophy in 2.0% is observed. It was concluded that LVH was the commonest morphological abnormality observed. They could screen CKD patients before undergoing renal transplant to detect and correct Coronary Artery Disease (CAD) and echo was a tool to detect moderate and massive Pericardial Effusion and to advice pericardiocentesis and adequate dialysis.

Dhamija JP (2016)[45] et al conducted a study to evaluate and analyze the echocardiographic changes in the end stage renal disease patients on maintenance hemodialysis. End stage renal disease (ESRD) patients who were on maintenance Haemodialysis for at least 3 months, in MG hospital were included in the study. They performed 2D echocardiography in 35 ESRD patients during inter-dialytic period. Patients with clinical evidence of coronary artery disease, valvular heart disease, congenital heart disease and pericardial effusion were excluded from the study. Out of 35 ESRD patients, echocardiography revealed LV dilatation and diastolic dysfunction in 18 patients (51.2%), LV hypertrophy in 17 patients (48%), systolic dysfunction and pericardial effusion in 10 patients (28.57%) and 6 patients (17.14%) respectively. RWMA was present in 3 patients (8.5%) and no valvular calcification was seen in any patient. In a sub group of 21 patients with Hb <10g%, LVH was present in 15 patients (71.42%) vs 2 out of 14 patients (14.28%) in patients group with Hb >10 g%. Hypertensive patients were 27 of 35 ESRD patients, 13 out of 27 had higher prevalence of LVH (51.85%). Systolic dysfunction and RWMA was absent in normotensive group. They reported that LV diastolic dysfunction and hypertrophy were most common echocardiographic findings. There was statistically significant correlation between anemia and presence of LVH and positive correlation between presence of hypertension and LVH.

Ahmed HA (2016)[46] et al tried to identify the major echocardiographic abnormalities in end-stage renal disease (ESRD) patients on maintenance hemodialysis. A case–control study was conducted that included 40 patients with ESRD on maintenance hemodialysis and 10 apparently healthy volunteers as controls. All participants were thoroughly interrogated, examined clinically, and subjected to complete blood count, kidney function tests, evaluation of serum electrolytes, serum calcium, PO4 level, lipid profile, fasting blood sugar (FBS), post prandial blood sugar (PPBS), HbA1c, and serum parathyroid hormone, and to transthoracic echocardiography. Patients were classified into two groups according to the presence or absence of echocardiographic changes: group 1 (G1), with echocardiographic changes, and group 2 (G2) without echocardiographic changes. Echocardiographic changes were seen in 75% correct of the studied dialysis patients (30/40). The major echocardiographic changes were: concentric left ventricular hypertrophy in 80% of G1 patients, diastolic dysfunction in 53.3% of G1 patients, valvular calcifications in 40% of G1 patients, systolic dysfunction in 36.3% of G1 patients, and regional wall motion abnormalities in 33.3% of G1 patients. Left atrium was dilated in 26.6% of G1 patients, whereas pericardial effusion was seen in 16.7% of G1 patients and pulmonary hypertension in 16% of G1 patients. Our study supported the high prevalence of echocardiographic changes in hemodialysis patients (75%) with predominance of left ventricular hypertrophy (80%) and diastolic dysfunction (53.3%).

A hospital based study conducted by Shrama M (2017)[47] et all from March 2014 to March 2015 in Guwahati Medical College where CKD patients were evaluated for presence of any cardiovascular morbidity. Cardiomegaly on chest x-ray was present in 64% of the patients. Electrocardiography and 2D echocardiography of patients revealed LVH in76% and 84% of patients. Left ventricular systolic dysfunction (LVSD) was found in 52 % of patient of which 34 % had mild dysfunction (LVEF= 45% -54%) and 18 % had moderate dysfunction (LVEF= 35% -44%). Diastolic dysfunction was found in 54 % of patient. It was concluded that cardiovascular complications were common in patients with chronic kidney disease, which was an important cause of morbidity and mortality in these patients and the most common morbidity found in this study was left ventricular hypertrophy.

Sachdeva A (2017)[48] et al did documentation of various cardiovascular abnormalities in sixty patients with Chronic Kidney Disease at Government Medical College Patiala, using Electrocardiography and Echocardiography as investigation procedures. ECG was normal in 15 out of 60 cases of CKD(25%), LVH present in 20 out of 60(33.33%), Left axis deviation in 9 out of 60(15%), Conduction disturbances in 10 out of 60(16.67%), Ischemia in 12 out of 60(20%), Arrhythmias in 2 out of 60(3.33%) and P-mitrale was found in 4 out of 60 cases(6.67%). The most common ECG change associated with cases with CKD was LVH (33.33%).The most common abnormality found on echo in CKD cases under study was LVH(56.67%) followed by Diastolic Dysfunction(38.33%). They were concluded that left ventricular hypertrophy was the commonest abnormality observed in CKD both on ECG and Echocardiography. Echocardiography was a more sensitive diagnostic procedure to detect left ventricular hypertrophy.

Behera B and Sanjay M (2017)[49] et al studied the prevalence of left ventricular hypertrophy (LVH) by echocardiography in patients with chronic kidney disease (CKD) and to find out correlation of left ventricular hypertrophy with severity of chronic kidney disease. From November 2012 to September 2014, 100 chronic kidney disease patients who were admitted in hospital or attended on OPD basis for dialysis were taken for study. Detailed history, clinical evaluation, laboratory investigations and echocardiography was carried out. The diagnosis of CKD was made on basis of serum creatinine more than 1.5 mg/dl which remained constantly for more than 3 months. Patients with mild, moderate and severe CKD were having serum creatinine level 1.5-3mg/dl, 3-6mg/dl and > 6mg/dl respectively. Glomerular filtration rate (GFR) was calculated by modification of diet in renal disease (MDRD) equation. Cut-off for CKD was taken to be <60ml/min / 1.73m2 as per existing guidelines. Out of 100 patients studied, 67 were males and 33 were females. All patients were selected randomly. Majority of the patients were in the age group of 61 -70 years (41%). In the present study, it was found that left ventricular mass index (LVMI) which reflects LVH showed a progressive rise in severity of renal failure with 17 % of mild category of CKD having LVH as compared to 26% of moderate category and 57% of severe category of CKD. The study revealed that patients with CKD have LVH, which was more marked in patients with severe CKD. So, these patients should have a thorough cardiovascular evaluation even if there were no symptoms, and efforts should be made to prevent LVH, during the early course of renal insufficiency, such as strict control of hypertension, anaemia.

Ramegowda RB (2018)[50] et al studied to identify echocardiographic changes in patients with chronic kidney disease. A total of 50 chronic kidney disease patients admitted to Adichunchanagiri medical college & hospital, were included in this study. The patients were evaluated as per the history, general physical examination, systemic examination, blood urea, serum creatinine, urine routine and echocardiography. In the present study echocardiographic abnormalities were observed in 35 patients (70%). Left ventricular hypertrophy was seen in 24 patients (48%). Regional wall motion abnormalities were seen in 7 patients (14%). LA+LV dilatation was seen in 2 patients (4%). Pericardial effusion was seen in 2 patients (4%). The study showed that left ventricular hypertrophy is the commonest morphological abnormality observed. Left ventricular dysfunction was commonest cardiovascular abnormality detected.

Saxena NK, Mehra D (2018)[51] aimed a study to understanding Dilated cardiomyopathy (DCM) in correlation with electrocardiographic and echocardiographic less than 40 year patients. A total of 60 patients (30 males and 30 females) of dilated cardiomyopathy were studied. ECG and 2D Echocardiography was done among all these patients using standard techniques. Diagnosis of dilated cardiomyopathy done by echocardiography. Both males and females were affected but and middle aged male population were found to be predominantly affected. Ventricular ectopics, Sinus tachycardia, Left and right bundle branch block and ST-T changes were common ECG abnormalities. They were concluded that the ECHO findings in patients revealed a dilated LV cavity with low ejection fraction. Mitral regurgitation were seen in 73.3% of patients.

Chen S (2018)[52] et al conducted a review to survey the clinical outcomes of CKD using cardiac and vascular markers including echocardiographic parameters, systolic time intervals, electrocardiography, heart rate variability, ankle-brachial index, pulse wave velocity, differences between interarm and interankle blood pressure, and vascular calcification. They concluded that CV diseases were common in patients with CKD, and CKD was a major risk factor for CV diseases. Many traditional and nontraditional risk factors also contribute to CV diseases. Functional and structural cardiac abnormalities, high P wave dispersion on 12 lead ECG, low

HRV, ΔHRV, ABI < 0.9 or > 1.3, high PWV, four limb BP difference and vascular calcification may play important roles in CV and renal prognosis in patients with CKD. Therefore, it was important for physicians to recognize the patients at risk and implement early prevention and treatment strategies.

Veeramani Kartheek AS, Srinivas Reddy VC (2019)[53] assessed the prevalence of cardiac dysfunction in patients with chronic kidney disease with end stage renal disease, at the time of initiation of haemodialysis. Patients with chronic kidney disease with End Stage Renal Disease admitted in King George Hospital, Visakhapatnam during the period from November 2018 to March 2019 were included in the study. A total of fifty CKD patients with ESRD were studied to determine the range of abnormalities of cardiac function. The predominant gender in the study group was male, constituting 68%. Hypertension was the most common aetiology of CKD with 21 (42%) patients, followed by hypertension & diabetes together (22%) and diabetes mellitus alone (20%). Other causes were NSAID abuse (6%), IgA Nephropathy (2%), Polycystic Kidney Disease (2%) & unknown aetiology (6%). Cardiovascular abnormalities were observed in large number of patients with ESRD (76%). LVH was the most common echocardiographic abnormality in CKD cases. Diastolic function was deranged more when compared to systolic function in patients with CKD. They have concluded that high prevalence of left ventricular hypertrophy, diastolic dysfunction on echocardiography implieed that these patients require detailed cardiovascular evaluation despite the absence of symptoms.

Kashioulis P (2019)[54] et al conducted a study to identify sub-clinical cardiac abnormalities by echocardiography in patients with chronic kidney disease (CKD) stages 3 and 4 and to investigate underlying mechanisms. Ninety-one patients with CKD stages 3 and 4, without a diagnosis of heart disease, and 41 healthy matched controls were included in this cross sectional study. Cardiac morphology and function were analysed with Doppler echocardiography and coronary flow velocity reserve (CFVR) in response to adenosine was measured in the left anterior descendent artery to detect coronary microvascular dysfunction (CMD). All study subjects had a left ventricular (LV) ejection fraction > 50%. Patients with CKD showed statistically significant increases in left atrial volume index and transmitral and pulmonary vein flow velocities during atrial contraction and prolonged LV isovolumetric relaxation time. Patients with CKD had significantly reduced CFVR vs. controls (2.74 ± 0.86 vs. 3.40 ± 0.89, $P < 0.001$), and 43% of patients were classified as having CMD compared with 9% of controls ($P = 0.001$). They revealed that patients with CKD stages 3 and 4, without a diagnosis of heart disease, showed early abnormalities in LV diastolic function that did not fulfil the criteria for LV dysfunction according to current guidelines. A large proportion of CKD patients had CMD, suggesting that microvascular abnormalities may have a pathogenic role in the development of heart failure in this patient group.

Jameel F (2020)[55] et al evaluated the echocardiographic findings in patients of CKD on maintenance hemodialysis. This cross-sectional study was conducted in the nephrology unit of Jinnah Postgraduate Medical Center between March 2019 to October 2019. A total of 100 patients who were on maintenance for more than one year were included in the analysis. Two-dimensional transthoracic echocardiography was done in each patient for the determination of cardiac structural and functional parameters such as LV hypertrophy, LV systolic dysfunction, and LV diastolic dysfunction. The mean age of the patients was 46.9±12.8 years. There was male dominance with male/female ratio 63/37. There were 39% hypertensive and 62% anemic patients. LV dysfunction was diagnosed in 31% of patients, LV diastolic dysfunction in 47% patients, and left ventricular hypertrophy (LVH) in 55% of patients. LVH was found in 74.3% hypertensive patients versus only 42.6% non-hypertensive patients (p-value 0.001). LV systolic dysfunction was also high in hypertensive patients, 46.1% versus 21.3% patients in non-hypertensive patients (p-value 0.008). The data showed that there was a high frequency of cardiac functional and structural abnormalities in CKD patients on HD especially in patients having concomitant hypertension. LVH was the most common structural defect and LV diastolic dysfunction was the most common functional cardiac defect in CKD patients on hemodialysis.

Nakazone M (2020)[56] et al evaluated the risk prediction performance of the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation and anemia in CKD patients. From 2000 to 2010, a total of 232 patients were studied in a single center retrospective study. CKD was defined as creatinine clearance <60 mL/min/1.73m2 according to CKD-EPI equation. Anemia was defined as hemoglobin <12 g/dL (women) and <13 g/dL (men). Cox proportional hazards models were used to establish predictors for death. At baseline, 98 individuals (42.2%) had criteria for CKD and 41 (17.7%) for anemia. During follow-up, 136 patients (58.6%) died. Independently, CKD and anemia were not associated with all-cause mortality. However, when they coexisted, an additional risk was attributed for these patients. Cox proportional hazard models analysis identified systolic blood pressure, implantable cardioverter defibrillator, left anterior fascicular block, left ventricular end-diastolic diameter, and serum sodium as independent predictors for death. They concluded that

CKD and anemia were not independent predictors for long-term mortality in CKD patients. However, the prognosis is poorer in individuals with both comorbidities.

V. Material And Methods

Source of data -Patient admitted to or in OPD follow up from Dr. BSA Medical College and Hospital with features suggestive of chronic kidney disease.

Method of collection of data - 70 patients with features suggestive of chronic kidney disease were taken.

Calculation of sample size- using **Cochran formula** for a undefined population

$N= z^2*p(1-p)\e^2$

z- z-score, e-margin of error, p-population proportion

z =1.94 (for 90% confidence interval)

p=50%, e= 10%

N=1.64 *1.64 *0.5(1-0.5)\0.1*0.1

N=67.24 ~ 67

This means 67 or more measurements are needed to have a confidence level of 90% with real value with in +\- 10% of the measured.

Sample technique: simple randomisation technique

Study design-cross-sectional study

Study population: A randomly selected patients who admitted to or in OPD follow up from Dr. BSA Medical College and Hospital with features suggestive of chronic kidney disease

Selection criteria-

1. All the proven cases of CKD admitted or in follow up from the Dr.Baba Saheb Ambedkar Medical College and Hospital.
2. Azotemia atleast 3months.
3. Ultrasound showing bilateral shrunken kidneys
4. Broad casts in the urine.

Exclusion criteria-

1. Congenital heart disease

Informed Consent- written informed consent was taken from all the patients participating in the study.

Following investigations were carried along with detailed History taking and clinical examination involving all the organ systems

Urine examination-To look for urine protein blood cells or casts etc.

Blood tests- haemoglobin, blood sugar, blood urea, serum creatinine, serum electrolyte.

X-ray –x-ray chest to look for significant cardiac finding.

Electrocardiography-To look for various changes, p wave morphology, PR interval, QRS complex, T wave morphology, ST-segment, QT interval etc. and to interpret the positive findings for the study

Ultrasound whole abdomen-(For diagnostic purpose only, not included in result) look for the kidney size and features of chronic kidney disease.

2D-Echocardiography –To look for the structural and functional changes in the heart in the study group. E.g. chamber sizes, ejection fraction, flow across valves using doppler, presence or absence of pericardial effusion

Methodology

Dilated cardiomyopathy (DCM) is defined as left ventricular chamber dilation with decreased systolic function (FVEF <40%) in the absence of coronary artery disease or conditions that impose a chronic pressure overload.

It was measured by echocardiography

The degree of left ventricular dilatation is highly variable and depends on the stage of disease and severity of left ventricular dysfunction.

A comprehensive echocardiographic study following standardized protocols, was performed for all subjects and all recorded studies were revised by an echocardiographer accredited by the European Association of Cardiovascular Imaging.

From the parasternal window, parasternal short axis views were obtained by placing the transducer in the left third or fourth intercostal space adjacent to the sternum with the knob pointing toward the right shoulder. The transducer was then angulated superiorly and inferiorly to obtain the papillary muscle level. From the papillary muscle level after confirming a true short axis view that was perpendicular to the center of the true long axis of the left ventricle (LV), measurements for the LV posterior wall thickness at end diastole (PWd), interventricular septum at end diastole (IVSd), LV internal dimensions at end diastole (LVEDD) and end systole (LVESD) were obtained. Measurements were made from the leading edge of the septal endocardium to the leading edge of posterior wall endocardium. Left ventricular hypertrophy (LVH) was diagnosed using American society of

echocardiography criteria, left ventricular mass/ body surface area >95g/m2 in females and more than 115g/m2 in males.

The left ventricular internal dimensions at end diastole (LVDd) and end systole (LVDs) were measured in the standard parasternal long-axis view at the level of the mitral valve leaflet tips obtained from two-dimensional echocardiographic images. End diastole was defined as the frame in the cardiac cycle in which the respective LV dimension is the Simpson's technique was used to determine ejection fraction (EF), EF<50 was taken as abnormal or left ventricular systolic dysfunction (LVSD) .The peak velocity of mitral regurgitation(MR) was measured by using continuous-wave Doppler echocardiography.

Diastolic dysfunction (LVDD) occurs when the left ventricular myocardium is non-compliant and not able to accept blood return in a normal fashion from the left atrium. This can be a normal physiologic change with aging of the heart or result in elevated left atrial pressures leading to the clinical manifestations of diastolic congestive heart failure. There are four grades of diastolic dysfunction as described below. Echocardiography is the gold standard to diagnose diastolic dysfunction.

Grade I (impaired relaxation): This is a normal finding and occurs in nearly 100% of individuals by the age of 60. The E wave velocity is reduced resulting in E/A reversal (ratio < 1.0). The left atrial pressures are normal. The deceleration time of the E wave is prolonged measuring > 200 ms. The e/e' ratio measured by tissue Doppler is normal.

Grade II (pseudonormal): This is pathological and results in elevated left atrial pressures. The E/A ratio is normal (0.8 +- 1.5), the deceleration time is normal (160-200 ms), however the e/e' ratio is elevated. The E/A ratio will be < 1 with Valsalva. A major clue to the presence of grade II diastolic dysfunction as compared to normal diastolic function is the presence of structural heart disease such as left atrial enlargement, left ventricular hypertrophy or systolic dysfunction. If significant structural heart disease is present and the E/A ratio as well as the deceleration time appear normal, suspect a pseudonormal pattern. Valsalva distinguishes pseudonormal from normal as well as the e/e' ratio. Diuresis can frequently reduce the left atrial pressure relieving symptoms of heart failure and returning the hemodynamics to those of grade I diastolic dysfunction.

Grade III (reversible restrictive): This results in significantly elevated left atrial pressures. Also known as a "restrictive filling pattern", the E/A ratio is > 2.0, the deceleration time is < 160 ms, and the e/e' ratio is elevated. The E/A ratio changes to < 1.0 with Valsalva. Diuresis can frequently reduce the left atrial pressure relieving symptoms of heart failure and returning the hemodynamics to those of grade I diastolic dysfunction.

Grade IV (fixed restrictive): This indicates a poor prognosis and very elevated left atrial pressures. The E/A ratio is > 2.0, the deceleration time is low and the e/e' ratio is elevated. The major difference distinguishing grade III from grade IV diastolic dysfunction is the lack of E/A reversal with the Valsalva maneuver (no effect will be seen with Valsalva). Diuresis will not have a major effect on the left atrial pressures and clinic heart failure is likely permanent. Grade IV diastolic dysfunction is present only in very advanced heart failure and frequently seen in end-stage restrictive cardiomyopathies such as amyloid cardiomyopathy.

Statistical analysis

The data was coded and entered into Microsoft Excel spreadsheet. Analysis was done using SPSS version 20 (IBM SPSS Statistics Inc., Chicago, Illinois, USA) Windows software program. Descriptive statistics included computation of percentages, means and standard deviations.

VI. Observation And Results

OBSERVATION AND RESULTS

Table 1: descriptive data of the study

	Minimum	Maximum	Mean	Std. Deviation
Age	25	95	56.43	15.32
Weight	45.00	120.00	59.45	12.35

Mean age was 56.43 years, with range 25-95 years and mean weight was 59.45 Kg with range of 45-120 kg.

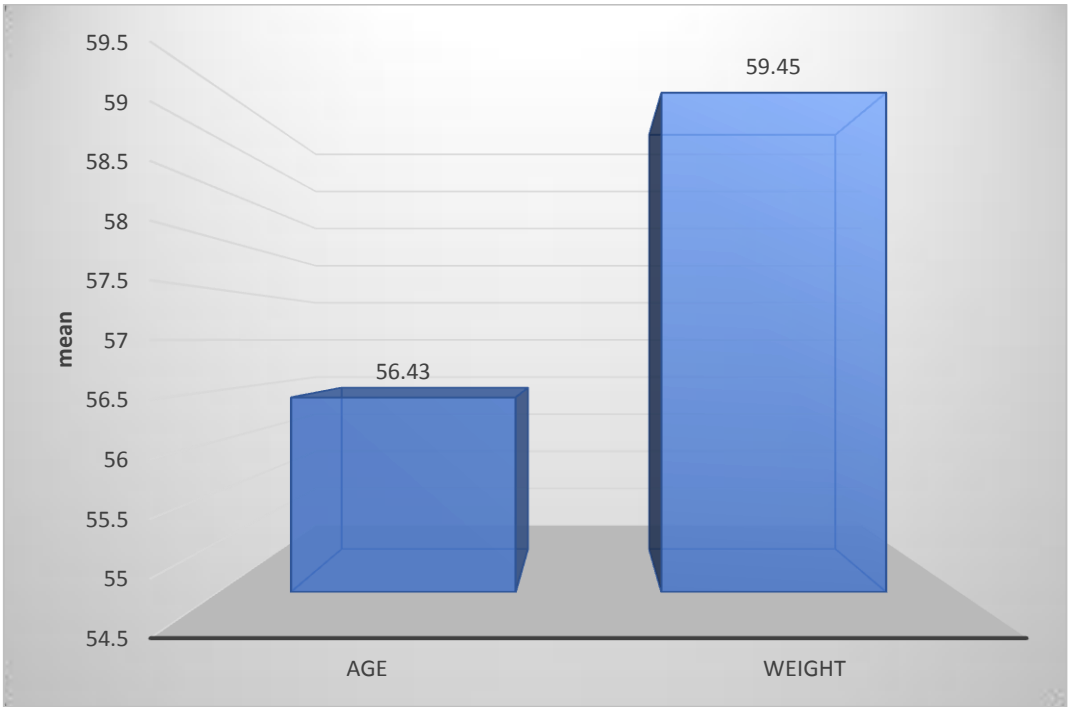


Table 2: blood reports wise distribution of the study

	Minimum	Maximum	Mean	Std. Deviation
Blood urea	46	392	125.23	55.31
Serum creatinine	2.20	18.50	7.0746	3.47
hb%(haemoglobin)	3.00	11.00	8.4029	1.73
NA+(sodium)	109.00	148.00	132.92	7.27
K+(potassium)	2.80	8.00	4.72	1.04
ca+(calcium)	5.40	10.00	7.94	1.07
P(phosphorus)	2.00	12.00	4.88	1.92
Rate	60	120	85.65	16.07

The mean urea value was 125.23 mg/dl and patients had range of urea between 46 to 392 mg/dl. The mean creatinine value was 7.07 mg/dl and patients had range of creatinine between 2.02 to 18.5 g/dl. The mean value was 8.4 g/dl and patients had range of Hemoglobin between 3 to 11 g/dl. The mean potassium value was 4.88 mEg/dl and patients had range of potassium between 2 to 12 mEg/dl. The mean K+ value was 4.72 mEq/dl and range between 2.8 to 8 mEg/dl. The mean Ca+ value was 7.94 mEg/dl and patients had range of Ca+ between 5.4 to 10 mEg/dl. The mean rate was 85.65 mEg/dl and patients had range between 60 to 120 mEg/dl

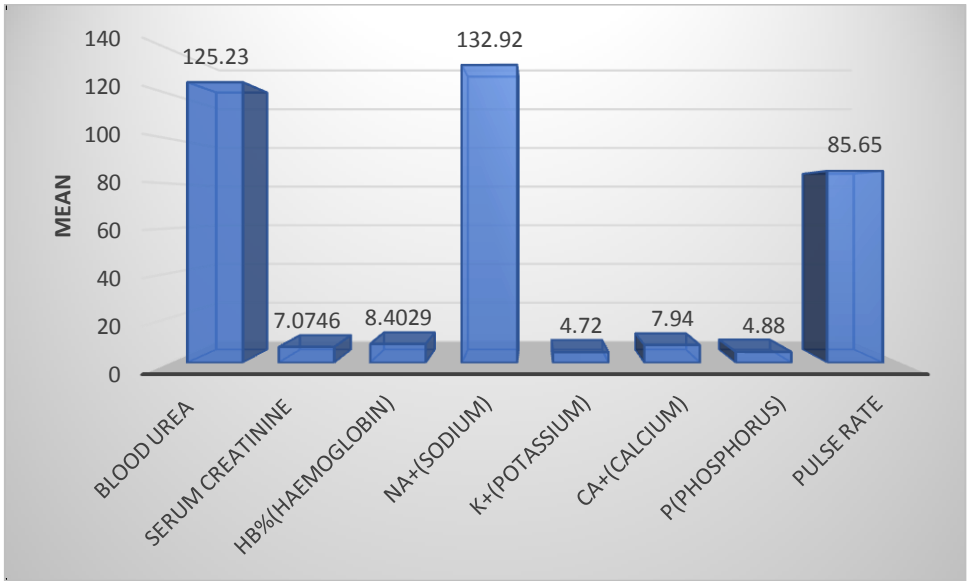


Table 3: gender wise distribution of the study

	Frequency	Percent
F	18	25.7
M	52	74.3
Total	70	100.0

Out Of total 70 patients, 52 (74.3) were males and 18 (25.7%) were females.

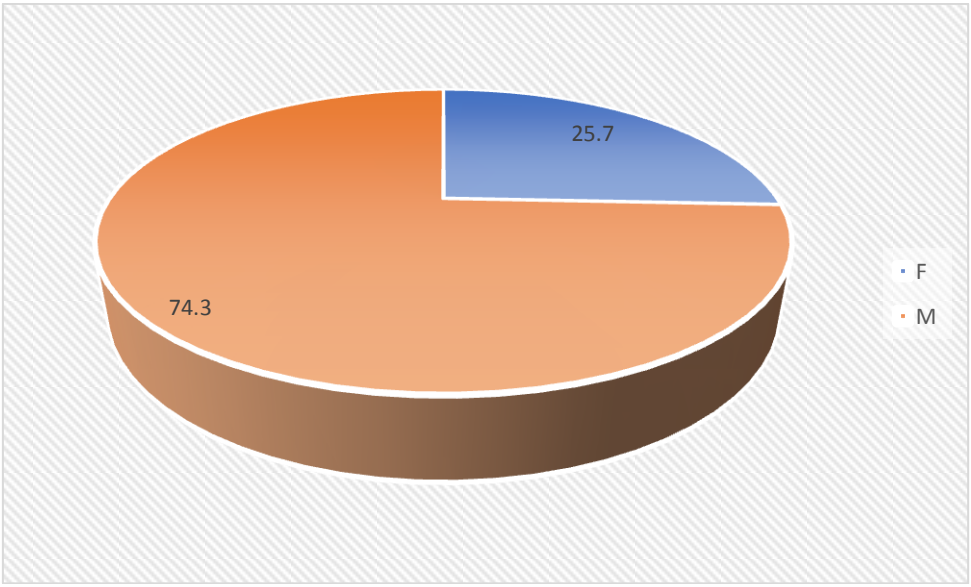


Table 4: Etiology wise distribution of the study

	Frequency	Percent
A-chronic(acute kidney injury progressing to chronic)	5	7.1
AN(Analgesic Nephropathy	5	7.1
CGN(Chronic glomerulonephritis)	10	14.3
DM(Diabetes mellitus)	11	15.7
DM(Diabetes mellitus), HTN(Hypertension)	23	32.9
HTN(Hypertension)	15	21.4
Obstructive Uropathy	1	1.4
Total	70	100.0

Hypertension was responsible for 54.3% cases , diabetes mellitus was responsible for 48.6% cases, only hypertension was responsible in 21.4% cases, only diabetes was responsible in 15.7% and Hypertension and diabetes mellitus were both responsible in 32.9% of patients. CGN was found in 14.3%.

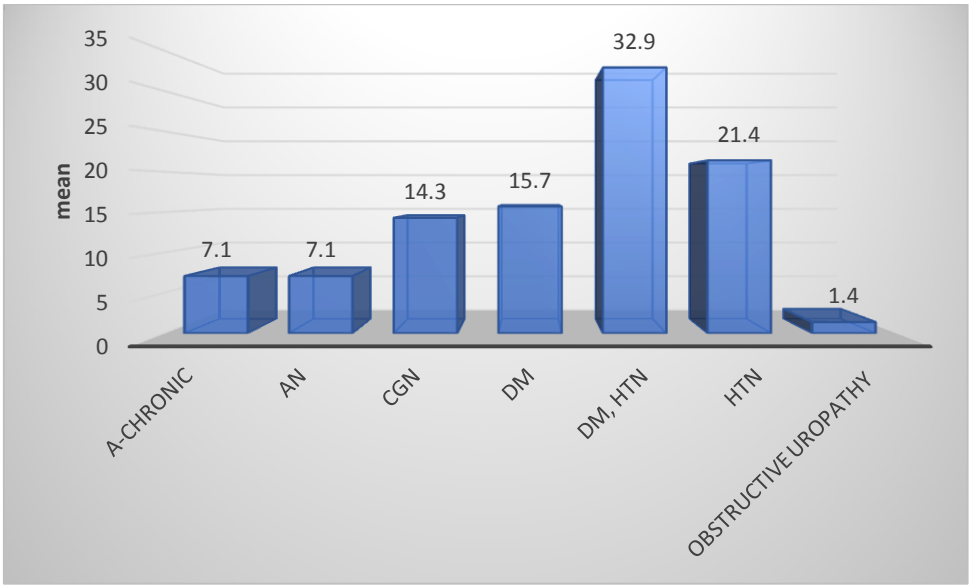


Table 5: Rhythm wise distribution of the study

	Frequency	Percent
IR(irregular)	1	1.4
R(regular)	69	98.6
Total	70	100.0

Regular rhythm was found 98.6% and irregular rhythm was found in 1.4%.

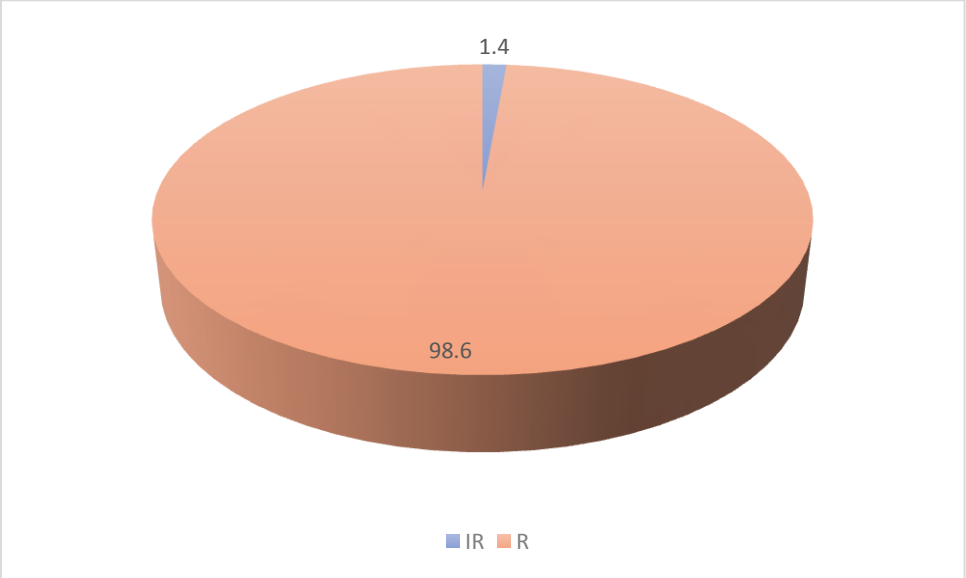


Table 6: ST/T wise distribution of the study

	Frequency	Percent
Normal	54	77.1
ST-T changes / ischemia	12	17.2
Tall T	4	5.7
Total	70	100.0

ST-T changes / ischemia was found in 17.2% and tall T was found in 5.7%

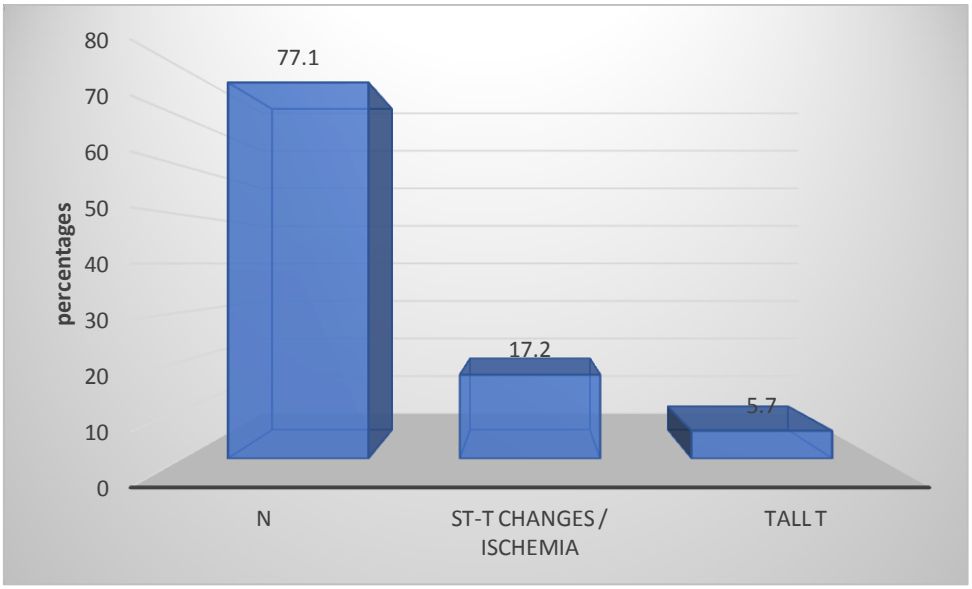


Table 7: Pericardial effusion wise distribution of the study

	Frequency	Percent
Normal	65	92.9
Present	5	7.1
Total	70	100.0

Pericardial effusion was found only in 5 (7.1%) patients

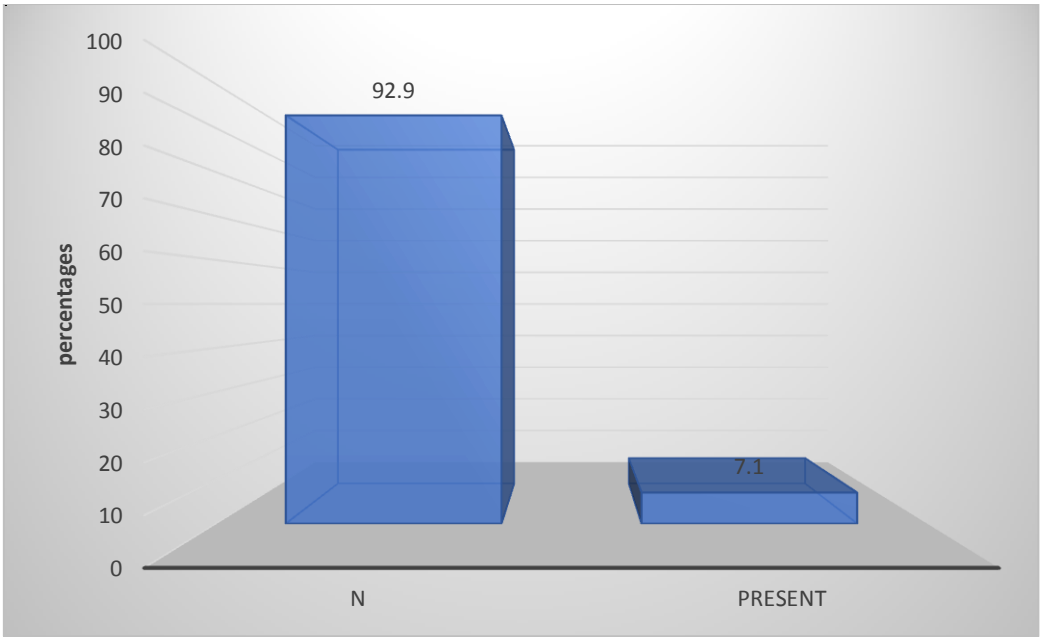


Table 8: ECHO wise distribution of the study

	Frequency	Percent
N	15	21.4
LVDD(Left ventricular Diastolic Dysfunction)	34	48.5
LVSD(Left ventricular systolic Dysfunction)	11	15.7
LVH(Left ventricular hypertrophy)	34	48.5
MR(Mitral regurgitation)	11	15.7
AR(Aortic regurgitation)	2	2.9

TR(Tricuspid Regurgitation)	4	5.7
Dilated cardiomyopathy	2	2.9

Out of 70 patients, 48.5% of patients had LVDD and 15.7% of patients had LVSD and mitral regurgitation was found in 15.7%.
LVH was recorded in 48.5% in the study.

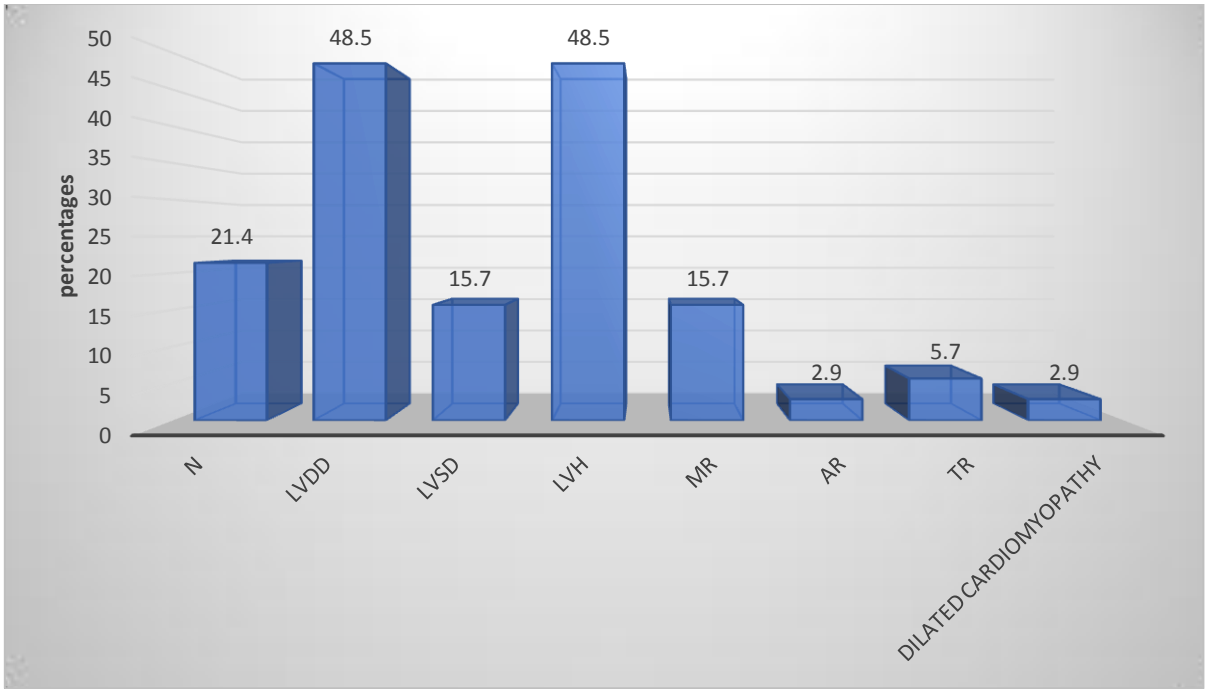


Table 9: ECG wise distribution of the study

	Frequency	Percent
N	16	22.9
AF(Atrial fibrillation)	1	1.4
Hyperkalaemia	3	4.3
Ischemia changes	12	17.2
LAD(Left axis Deviation)	11	15.7
LAE(left atrial enlargement)	2	2.9
LVH(Left ventricular hypertrophy)	23	32.8
LVS(left ventricular strain)	1	1.4
LBBB(Left bundle branch block)	3	4.3
RBBB(Right bundle branch block)	5	7.14
RAS(Right atrial strain)	5	7.1
RAE(Right atrial enlargement)	1	1.4

LVH was found in 23 (32.8%) patients which was highest than other finding of ECG. Ischemic changes were 17.2%, LAD was found in 15.2% while LBBB, RAS and RBBB was found in 4.3%, 7.1% and 7.14% respectively.

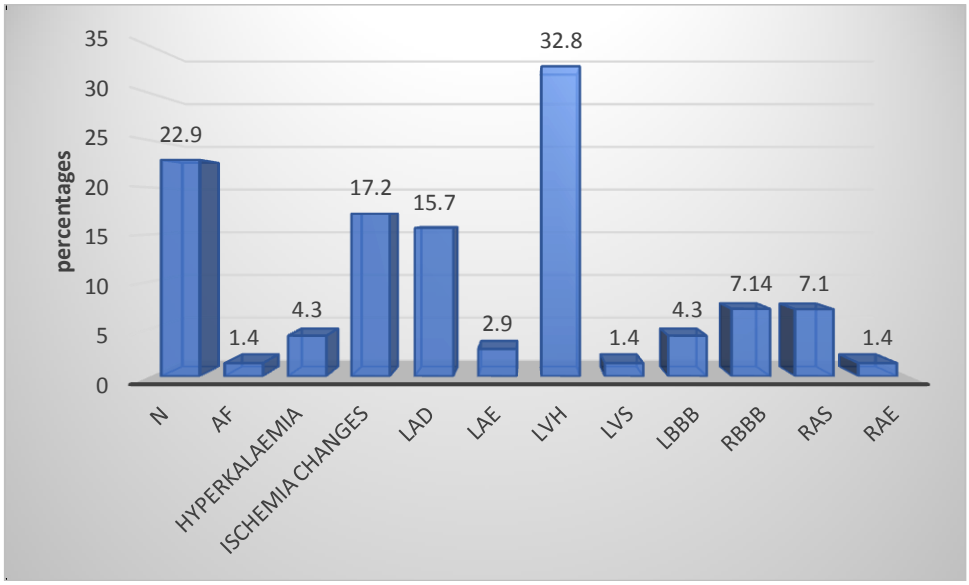


Table 10: VALVES wise distribution of the study

	Frequency	Percent
AR(Aortic regurgitation)	1	1.4
AR(Aortic regurgitation),TR(Tricuspid Regurgitation)	1	1.4
MR(Mitral regurgitation)	10	14.3
MR(Mitral regurgitation),AR(Aortic regurgitation)	1	1.4
MR(Mitral regurgitation),TR(Tricuspid regurgitation)	4	5.7
N	53	75.7
Total	70	100.0

MR was found 14.3% in the study while MR with AR and MR with TR was recorded 1.4% and 5.7% respectively.

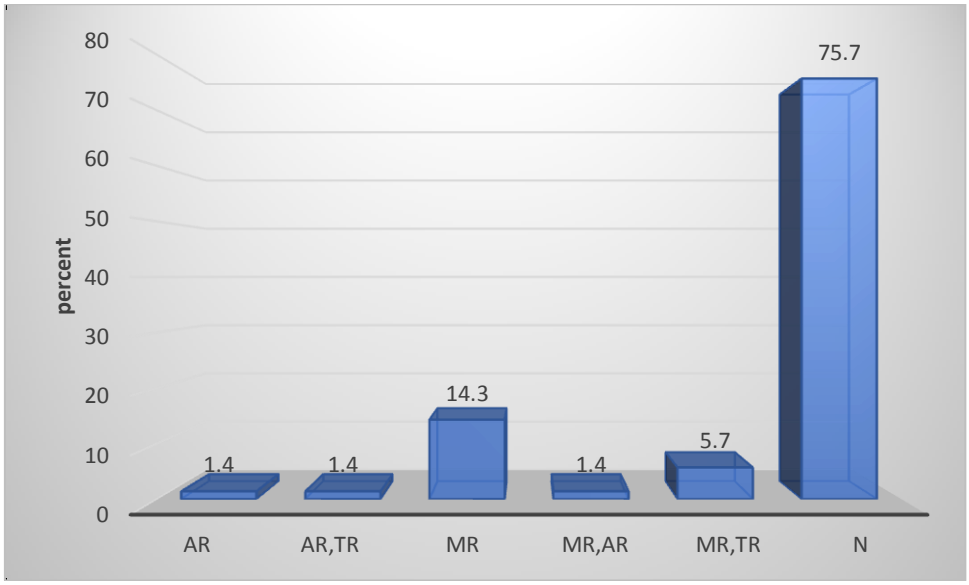


Table 11: Presenting sign wise distribution of the study

	Frequency	Percent
X ray (cardiomegaly)	38	54.3
Pallor	38	54.3
Pedal edema	63	90
Blood pressure(increased)	39	55.7

Pedal edema was present in 90% at presentation, raised blood pressure was found 55.7%, pallor and cardiomegaly(on chest x ray) was Present in 54.3% patients.

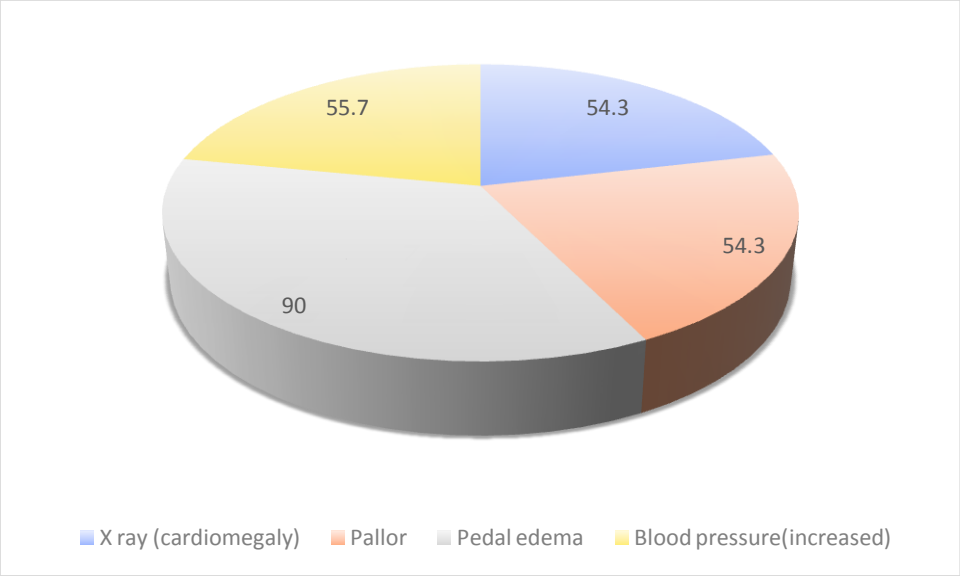


Table 12: Presenting symptom wise distribution of the study

	Frequency	Percent
Chest pain	2	2.9
Decreased urine output	1	1.4
Generalized weakness	20	28.6
Palpitation	1	1.4
Shortness of breath	23	32.9
Body Swelling	23	32.9
Total	70	100.0

Shortness of breath was found 32.9%, body swelling was found 32.9% and generalized weakness was found 28.6% as the major symptoms at presentation.

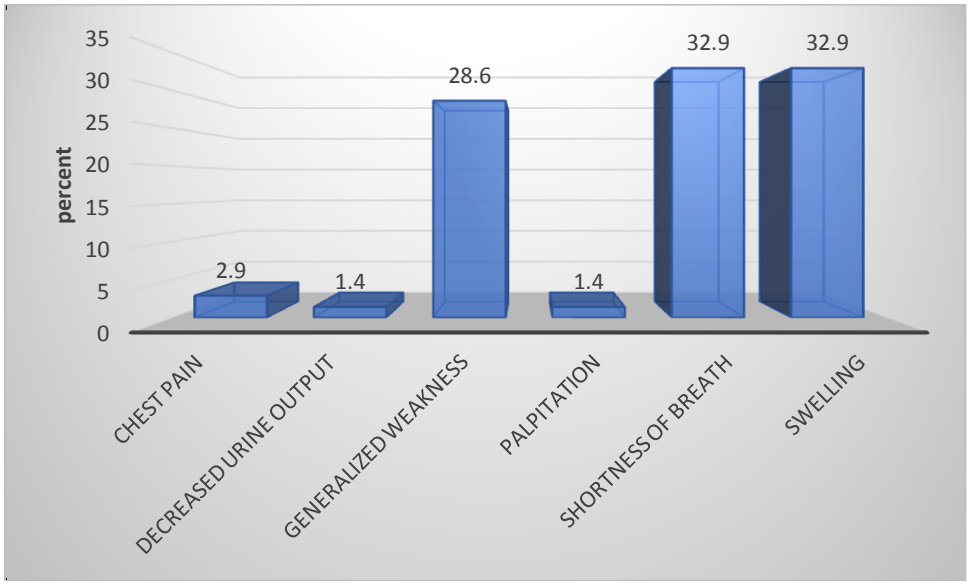


Table 13: Stages wise distribution of the study

eGFR	Frequency	Percent
G4 (Stage 4)	16	22.9
G5 (Stage 5)	54	77.1
Total	70	100.0

77.1% patients were in stage 5 and 22.9% were in stage 4.

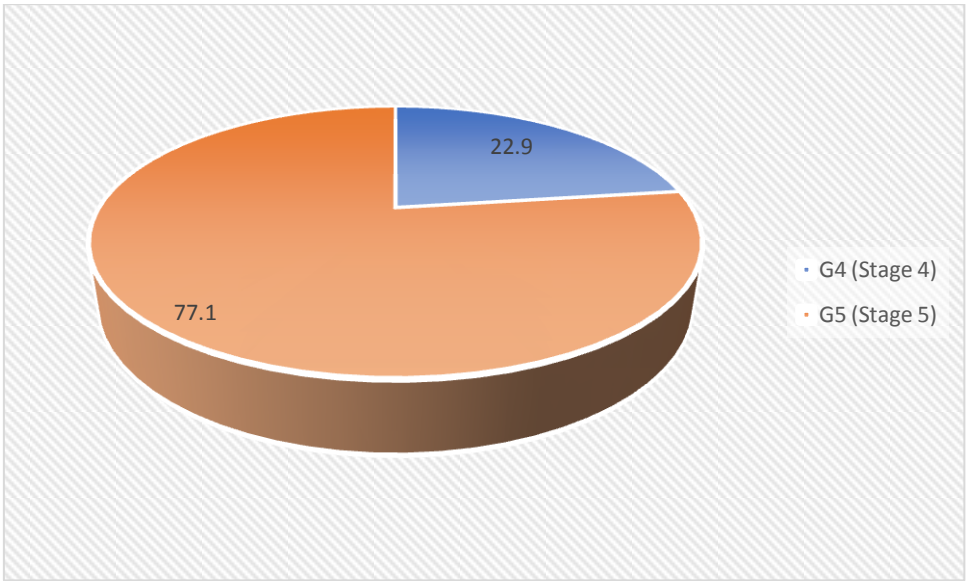


Table 14: Stages and LVH in ECHO wise distribution of the study

eGFR	Frequency	Percent
G4 (Stage 4)	5	14.7
G5 (Stage 5)	29	85.3
Total	34	100

Stage 5 was found 85.3% and stage 4 was found 14.7% among Patients with LVH(Left ventricular hypertrophy).

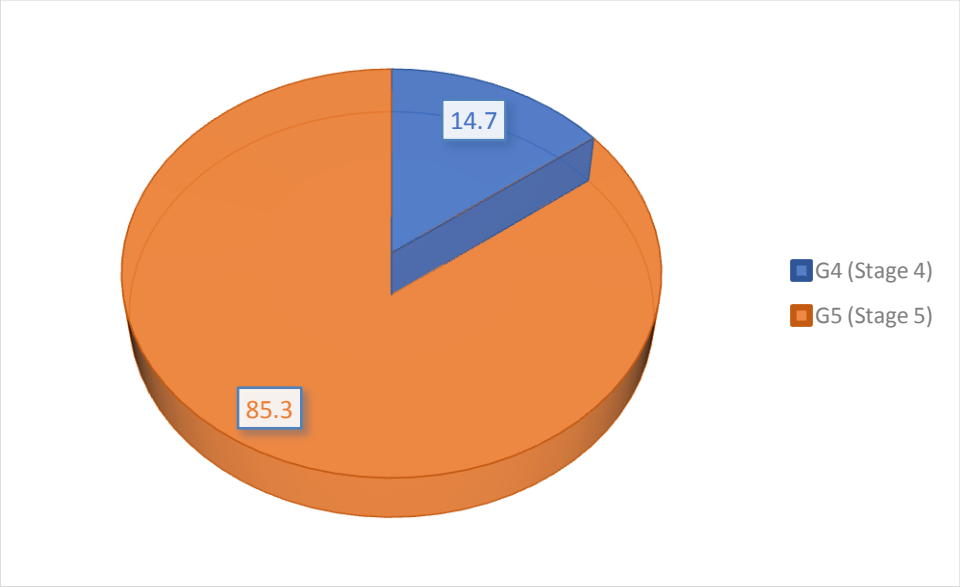


Table 15: Stages and LVH in ECG wise distribution of the study

	Frequency	Percent
G4 (Stage 4)	5	21.7
G5 (Stage 5)	18	78.3
Total	23	100

Among LVH with ECG, Stage 5 was found 78.3% and stage 4 was found 21.7%.

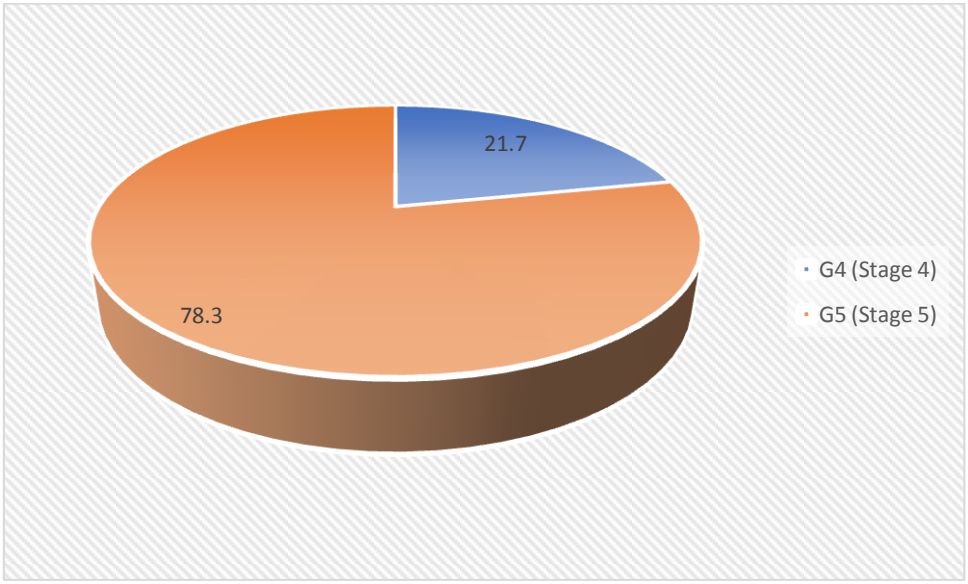


Table 16: Stages and LVDD in ECHO wise distribution of the study

	Frequency	Percent
G4 (Stage 4)	7	20.59
G5 (Stage 5)	27	79.41

Total	34	100

Among patients with Left ventricular diastolic dysfunction(LVDD) in ECHO, S 79.41% were in stage 5 and 20.59% were in stage 4.

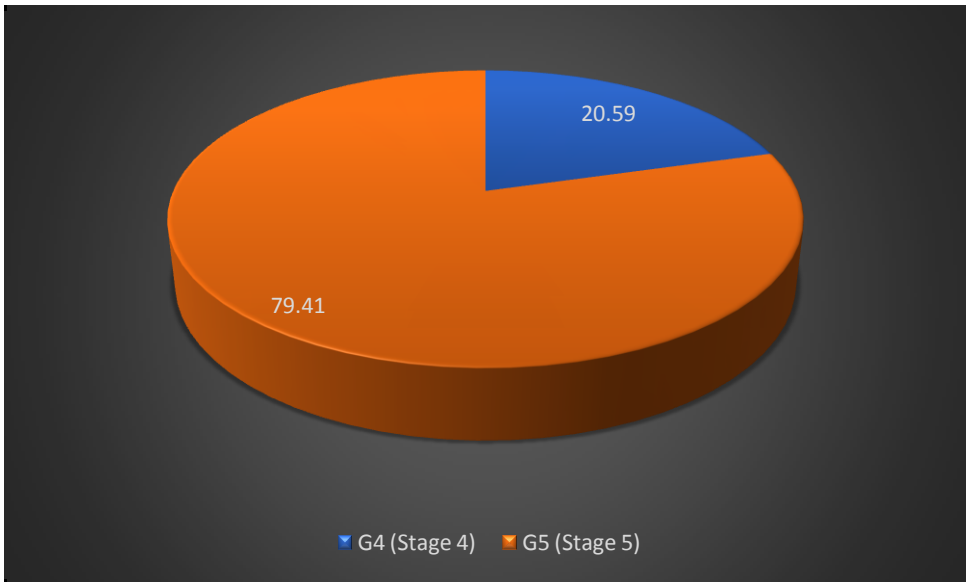


Table 17: LVSD vs stage table wise distribution of the study

			LVSD		Total
Stages	G4		3	27.27%	
	G5		8	72.73	
					11(100 %)

Among patients with left ventricular systolic dysfunction (LVSD) 3(27.27%) and 8(72.73) patients were present in stage 4 and 5 respectively.

Table 18: LVH in ECG wise distribution of the study

	Frequency	Percent
DM	0	0
HTN	4	36.36
DM+HTN	7	63.64
Total	11	100

Among the patients with LVH(Left ventricular hypertrophy) in ECG, Diabetes(DM) and Hypertension (HTN) both co morbidities were present in 63.64% and only hypertension was found 36.36% patients.

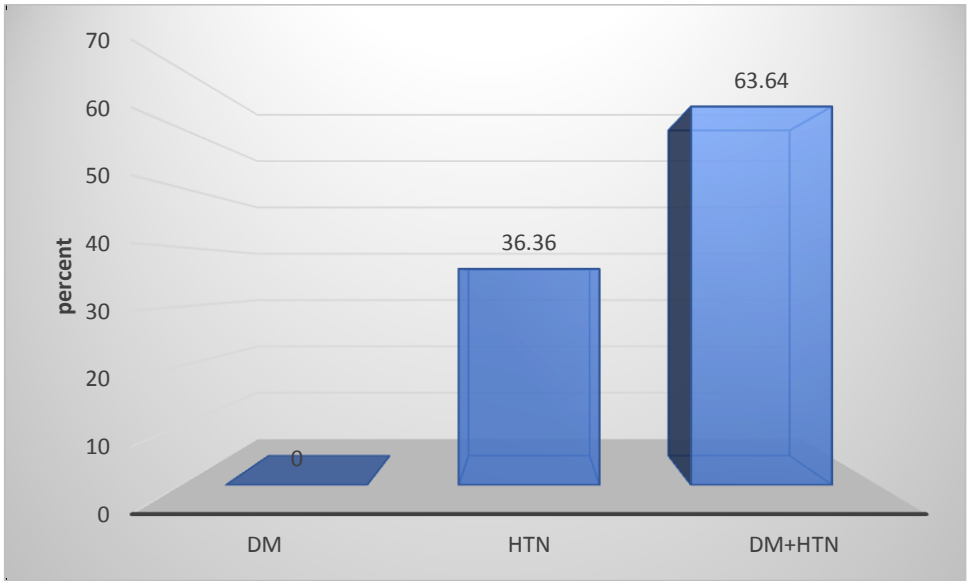


Table 19: LVH in ECHO wise distribution of the study

	Frequency	Percent
DM(Diabetes mellitus)	5	21.73
HTN(Hypertension)	5	21.73
DM+HTN (Diabetes+Hypertension)	13	56.5
Total	23	100

Among patients with LVH(left ventricular hypertrophy) in Echocardiography, diabetes and Hypertension both co morbidities were found in 56.5% and only diabetes and only hypertension was found 21.73% separately.

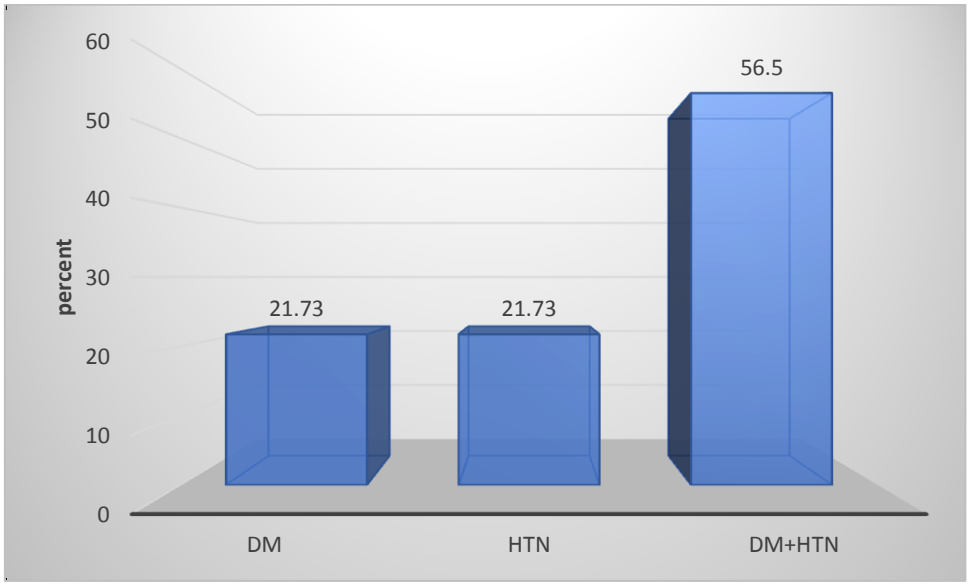


Table 20: LVDD in ECHO wise distribution of the study

	Frequency	Percent
DM(Diabetes mellitus)	3	11.6
HTN(Hypertension)	9	34.6

DM+HTN(Diabetes+Hypertension)	14	53.8
Total	26	100

Among patients with Left ventricular diastolic dysfunction (LVDD) in Echocardiography Diabetes and Hypertension both co morbidities were found in 53.8% and only diabetes and hypertension were found 11.6 and 34.6% respectively.

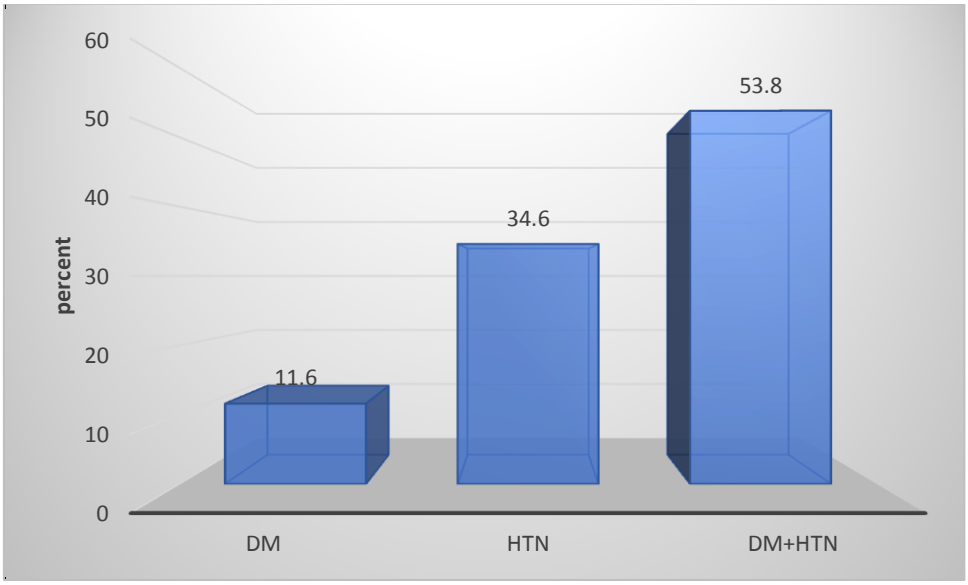
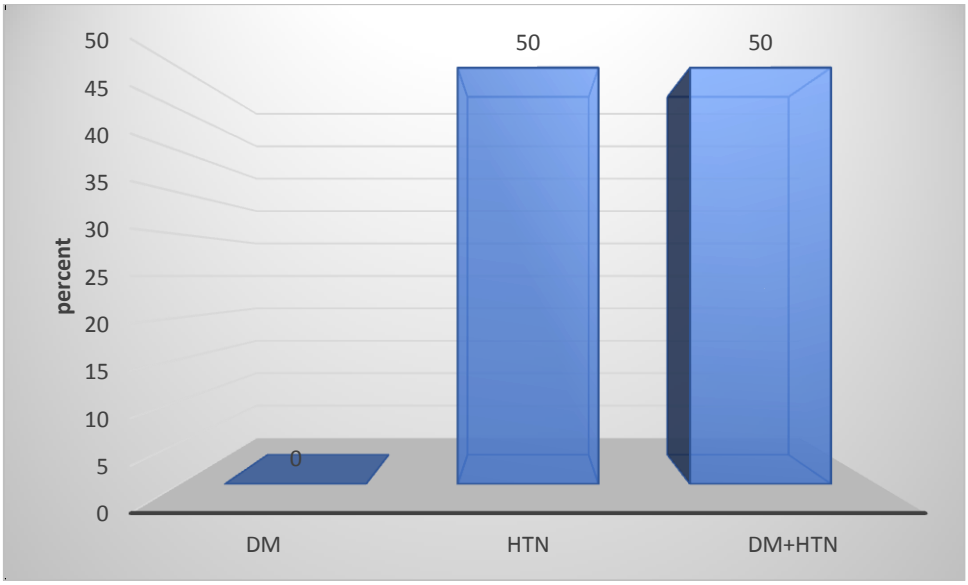


Table 21: LVSD in ECHO wise distribution of the study

	Frequency	Percent
DM(Diabetes mellitus)	0	0
HTN(Hypertension)	3	50
DM+HTN(diabetes + Hypertension)	3	50
Total	6	100

Among patients with Left ventricular systolic dysfunction (LVSD) in echocardiography, diabetes and Hypertension both co morbidities were found 50% and only hypertension was found 50% separately.



VII. Discussion

CKD patients have higher proportions of congestive heart failure that is associated with a higher mortality rate in these patients.[57] Echocardiography is a valuable tool to assess the assess changes in function and structure of the heart that result from CKD. Abnormal LV geometry, reduction in interventricular septum strength, and changes in LV mass index are important parameters that are affected by CKD in patients with preserved EF.[58]

Various diagnostic modalities both invasive and non-invasive such as electrocardiography, echocardiography, magnetic resonance imaging and radionuclide scans are utilised for the diagnosis of cardiovascular dysfunction. Electrocardiography (ECG) and different types of Echocardiographic findings provide the most important details regarding cardiovascular morbidities.

The study included a total of 70 patients. Of these 70 patients, 52 (74.3) were males and 18 (25.7%) were females. The age of the patients varied from 25 to 95 years with the mean age 56.43 years. The mean age of the present study was compared with other studies, the mean age in the others studies are 51 years in Foley (1995)[59] et al, 47.5 years in Ramanan (2005)[60] et al, 47.58 years in Goornavar (2015)[44] et al and 46.9 years in Jameel F (2020)[55] et al.

Hypertension was responsible in 54.3% patients, diabetes mellitus was responsible in 48.6% patients. Only hypertension was responsible 21.4% patients, only diabetes mellitus was responsible in 15.7% while both diabetes and hypertension was found in 32.9% of patients. Sharma M (2017)[47] et al found hypertension in 80%,Ulasi LL (2006)[61] et al their cross sectional study (involving CKD patients) found hypertension in 85.3% of the patients. Levin A(2003)[62]in their study also found prevalence of hypertension and LVH in 87-90% of the patients. A study by Tsilonis K (2016)[16] et al reported diabetes mellitus in 24% of patients and hypertension in 22% of patients of CKD in their study group.

The most common considered mechanisms for hypertension in CKD patients are sodium retention and activation of the renin-angiotensin system. Sympathetic nervous system activation also plays a role. Elevation of plasma catecholamine concentrations, and increased sympathetic nerve traffic has been demonstrated in renal failure.[63,64]

Kale SA (2001)[27] et al identified hypertension as an important risk factor for cardiac dysfunction. There being an independent and significant relationship between systolic, diastolic and mean blood pressure with left ventricular disease. This mandates an aggressive control of hypertension in CKD patients. Reduction of left ventricular hypertrophy with control of hypertension is also being reported.

Almost all patients in the study had Hemoglobin less than 12 g/dl. The mean value was 8.4 g/dl and patients had range of Hemoglobin between 3 to 11 g/dl which was comparable to Foler R (2000)[59] et al (8.4) and Chafekar DS (1994)[65] et al (7.84).

Urea and creatinine were elevated in all patients. The mean urea value was 125.23 mg/dl and patients had range of urea between 46 to 392 mg/dl which was comparable to Singh NP (1999)[66] et al (121.2) and Foley R (2000)[59] et al (117). The mean creatinine value was 7.07 mg/dl and patients had range of creatinine between 2.02 to 18.5 g/dl which was comparable to Singh NP et al (3.5), Chafekar DS (1994)[65] et al (5.75) and Jameel F (2020)[55] et al (5.36). The mean K+ value was 4.72 mEq/dl which was comparable to Singh NP et al (4.73)[66]. The mean potassium value was 4.88 mEg/dl and patients had range of potassium between 2 to 12 mEg/dl while mean Ca+ value was 7.94 mEg/dl and patients had range of Ca+ between 5.4 to 10 mEg/dl which is in concordance with study done by Agarwal S (2003)[14] et al and Tomilina NA (2007)[67] et al.

In the present study **ECG abnormalities** were observed in 54 patients (77.1%). Kartheek ASV and Reddy VC (2019)[53]found 76% of ECG abnormality. Left ventricular hypertrophy was found in 23 patients (32.8%). Other electrocardiographic abnormalities also detected in the study were left axis deviation (11.4%), left atrial enlargement (2.9%), LBBB (4.3%) and RBBB (7.14%).

Signs of ischemia were seen in 12 patients (17.2%). Krivoshiev V (1987)[68] et al and Menon AS (1998)[69] et al found it in 20% and 29.1% respectively. Arrhythmia (Atrial Fibrillation) was seen only in 1 patients (1.4%). Soman SS (2002)[37] et al, Ramanan (2005)[60] et al and Goornavar(2005)[44] et al was reported 5%, 4% and 16% in their study respectively.

Sachdeva S (2017)[70] et al found that Echo was Normal in 14 cases (23.33%), LVH in 34 cases (56.67%), Ischemia in 9 cases(15%), Pericardial Effusion in 5 cases(8.33%).

Ladha A (2014)[71] et al reported that LVH, ischemia, pericardial effusion were 74.3%, 12.9% and 24.3% respectively.

The above observation made in the present study is comparable with studies done by Soman SS (2002) et al and Ramegowda R (2018)[50] et al.

Left ventricular hypertrophy (LVH) is the common abnormality detected. It was found in 23 (32.8%) patients by ECG. In a study done by Parfrey PS (1996)[8] et al in Division of Nephrology, Salvation Army Grace General Hospital, Canada, 41% of patients had concentric left ventricular hypertrophy. Ramegowda R (2018)[50] et al was found 48%. Dai Y (1993)[72] et al has reported an incidence of LVH in 52% of patients. Gruppen MP (2003)[73] et

al has reported LVH in 47% of male patients and 39% of female patients. Stewart GA (2005)[30] et al in their study found LVH in more than 80% of the study group concentric type being dominant. Costa Fde A (2009)[74] et al in their study also found LVH in 83% of CKD patients. The study by GruppenMP (2003)[73] et al was a Dutch cohort study done in young adult patients with end-stage renal disease since childhood. Echocardiographically proved left ventricular hypertrophy is an independent risk factor for cardiovascular morbidity and mortality. Lowering of cardiac size and increase in fractional shortening were both associated with reduced subsequent likelihood of cardiac failure. These associations were independent of baseline age, diabetes mellitus, ischemic heart disease, baseline echocardiographic parameters.[75]

Ischemic-like ST-T changes was found only in 12 (17.2%) patients. Sharma M (2017)[47] et al was found 22% and Shapira OM and Bar-Khayim Y (1992)[76] was found 22-39%. Both studies have higher number of patients as compared to our study.

Echocardiography provides an excellent non-invasive method to delineate details of the anatomy of cardiac cavity, wall dimensions and wall movements. It is now increasingly used in the assessment of cardiac performance and is also invaluable in the demonstration of structural abnormalities such as LVH and pericardial effusion. Left ventricular hypertrophy is the single strongest independent predictor of adverse cardiovascular events. LVH is a major echocardiographic finding in uremic patients.

On **2D echocardiography**, LVH was found in 34 (48.5%) patients, Menon AS (1998) et al found it in 40%, Middleton RJ (2001)[77] et al found it in 41% patients while Shrama M (2017)[47] et al found it in 84% of the patients. Ulasi LL (2006)[63] et al in their cross sectional study (involving CKD patients) found LVH in 95.5% of the cases and 6.7% of controls (95.5% vs. 84%).Levin A(2003)[62]in their study found LVH to be present in 75% of the patients prior to hemodialysis (75% vs. 84%). The variation could be due to difference in the sample size and its composition, selection criteria.

Pericardial effusion was found in 5(7.1%) cases. Gupta A (1986)[78] et al reported an incidence of 8.8% in patients on maintenance hemodialysis. Sharma M (2017)[47] et al was found 20% and IjomaC(2010)[79] et al was found 15.9%.

In our study, 48.5% of patients have LVDD and 15.7% of patients have LVSD. Sharma M et al recorded 52% of LVSD and 54% of LVDD. LV diastolic dysfunction was more common in all stages of CKD.[80]Jameel F (2020)[55] et al found that LV diastolic dysfunction was there in 47% patients and LV systolic dysfunction in 31% of patients.A study conducted by Shivendra S (2014)[81] et al. reported diastolic dysfunction in 51.42% patients, and systolic dysfunction in 28.57% patients of CKD on maintenance HD. Agarwal S (2003)[14] et al. reported LV diastolic dysfunction in 53.2% patients and LV systolic dysfunction in 30% of patients having severe CKD. Another study by Laddha M (2014)[75] et al. reported LV diastolic dysfunction in 61.4% patients, and systolic dysfunction in 24.3% patients. A similar study by Ahmed HA (2016)[46] et al. showed that LV diastolic dysfunction in 53.3% patients, and LV systolic dysfunction in 36.3% patients.In the study conducted on ESRD patients by Parfrey PS et al systolic dysfunction was found in 16% of patients.[8]

Moreover, few studies have explored the relationship between electrocardiographic and echocardiographic LVH. Kim SJ (2012)[82] et al was tried to elucidate the correlation between commonly used criteria of ECG-LVH and echocardiographic LVH, and found that SP and CP criteria correlated more closely with LVMI determined by echocardiography than SV and CV criteria, respectively and that CP criteria provided the highest predictive value for echocardiographic LVH.

High diastolic blood pressure, high pulse pressure and increased BMI as well as male gender were found to be significantly associated with the presence of LVH in the cohort conducted by Amoako YA (2017)[83] et al.

VIII. Conclusion

Most common ECG abnormality was Left ventricular hypertrophy ,in echocardiography left ventricular hypertrophy and left ventricular diastolic dysfunction were common. Echocardiography is a more sensitive diagnostic procedure to detect left ventricular hypertrophy. Conduction abnormality is common in CKD patients. Echocardiography was also helpful to detect left ventricular hypertrophy.

The high prevalence of Left ventricular hypertrophy in these populations on echocardiography implies that these patients require detailed cardiovascular evaluation despite absence of symptoms, and also that various efforts aimed at prevention and control of left ventricular hypertrophy should be started early during the course of renal insufficiency, such as effective control of hypertension, anemia. Pericardial effusion was less frequent in the study population , availability of hemodialysis can be an important factor for this.

Hypertension and diabetes were common etiologic causes in the study population so screening at an early stage can prevent cardiovascular complications and even progression to Chronic kidney disease and also electrocardiographic and echocardiography abnormalities were more commonly associated with those having these comorbidities along with CKD.

Anemia and electrolyte abnormalities were frequently seen in these patients both these abnormalities can lead to cardiac dysfunction ,heart failure is associated with anaemia and various conduction abnormalities are associated with electrolyte abnormalities so frequent monitoring of these parameters is important.

Most of the patients presented with body swelling/edema and shortness of breath which are seen in other conditions also but can give us a valuable clue. Most of the patients in the study group had cardiovascular abnormalities which is clearly more frequent as compared to those with out chronic kidney disease but exact comparison was not done with any control group.

Limitation and recommendation

Participants included in this study randomly were in stage 4 or stage 5 of CKD so a stage wise correlation of findings with the stage could not be made in the study population. Parathyroid levels an important factor in pathogenesis of cardiovascular changes in these patients was not done due to non availability at the place of study.

There are some limitations of echocardiography, including 2DE and 3DE, to evaluate cardiac functions. Good image quality is essential for accurate LV quantification. Technical conditions, such as image quality, echocardiographic resolution, artifacts, may significantly affect the accuracy of cardiac measurement. In addition, although many studies indicate the possible clinical roles of echocardiography in CKD patients, there is no large cohort study to show cost effectiveness of an echocardiographic study and its impact on daily management. Therefore, it is premature to suggest routine echocardiographic study in CKD patients now, especially in those without cardiac symptom/sign. Further large-scale cohort study is necessary to validate the clinical impact and applications of echocardiographic study in CKD patients.

Impact of hyperlipidaemia, secondary hyperparathyroidism, homocysteine levels and markers of inflammation and duration of MHD were not studied in population group.

As a final point, it is worth mentioning the value of cardiac biomarkers, such as troponin T and brain natriuretic peptide, which are useful for diagnostic and prognostic purposes in advanced CKD. Although they do not replace echocardiography, these surrogate markers may progress to play an adjunctive role to echocardiography in assessing cardiovascular risk of CKD subjects.[84]

IX. Summary

This study was performed on 70 patients at Baba Saheb Ambedkar Medical College and hospital, Delhi with findings:

1. Mean age was 56.43 years, with range 25-95 years and mean weight was 59.45 Kg with range of 45-120 kg.
2. The mean urea value was 125.23 mg/dl and patients had range of urea between 46 to 392 mg/dl. The mean creatinine value was 7.07 mg/dl and patients had range of creatinine between 2.02 to 18.5 g/dl. The mean value was 8.4 g/dl and patients had range of Hemoglobin between 3 to 11 g/dl. The mean potassium value was 4.88 mEg/dl and patients had range of potassium between 2 to 12 mEg/dl. The mean K+ value was 4.72 mEq/dl and range between 2.8 to 8 mEg/dl. The mean Ca+ value was 7.94 mEg/dl and patients had range of Ca+ between 5.4 to 10 mEg/dl
3. Out Of total 70 patients, 52 (74.3) were males and 18 (25.7%) were females.
4. Hypertension was responsible for 54.3%, diabetes mellitus was responsible for 48.6% and Hypertension and diabetes mellitus were both responsible in 32.9% of patients. Chronic glomerulonephritis was found 14.3%.
5. Regular rhythm was found 98.6% and irregular rhythm was found 1.4%.
6. ST-T changes / ischemia was found 17.2% and tall T was found 5.7%.
7. LVH was found in 23(32.8%) which was most frequent ECG finding, ischemic changes were found in 17.2%,LAD was found in 11.4% while LBBB,RAS and RBBB was found in 7.14%, 5.7% and 4.3% respectively.
8. Pericardial effusion was found only in 5 (7.1%) patients
9. In the 2D ECHO, 48.5% of patients had LVDD and 15.7% of patients had LVSD and mitral regurgitation was found 15.7%. LVH was recorded in 48.5% in the study.
10. Among the patients with LVH(Left ventricular hypertrophy) in ECG, Diabetes(DM) and Hypertension (HTN) both co morbidities were present in 63.64% and only hypertension was found 36.36% patients.
11. Among patients with LVH(left ventricular hypertrophy) in Echocardiography, diabetes and Hypertension both co morbidities were found in 56.5% and only diabetes and only hypertension was found 21.73% separately.
12. Shortness of breath was found 32.9%, body swelling was found 32.9% and generalized weakness was found 28.6% as the major symptoms at presentation.

13. Pedal edema was present in 90% at presentation, raised blood pressure was found 55.7%, pallor and cardiomegaly(on chest x ray) was Present in 54.3% patients.

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PATIENT PERFORMA

Reg No.: Name:

Age: Gender:

Address: Occupation:

HISTORY OF ANY CHRONIC ILLNESS ASSOCIATED WITH CKD

Chief complaints-

History of present illness-

Past history-

Personal history-

Social history-

CLINICAL EXAMINATION

General Examination

BP- Pulse-

Weight- Height-

Respiratory rate-. JVP-

Pallor- Icterus-

Clubbing- Cyanosis-

Edema- lymphadenopathy-

Any other significant finding-

Systemic Examination

Cardiovascular system-

Respiratory system-

Central nervous system-

Abdominal examination-

BLOOD INVESTIGATION
Hemoglobin-
Blood urea-
Serum creatinine-
Sodium-
Potassium-
Phosphorus-
Calcium-

ECG FINDINGS
Rate-
Rhythm-
Axis-
P-wave-
QRS complex-
ST-T segment
Inference-

X-RAY FINDINGS -

ECHO findings-
RV size-
LVEF-
LVESD-
LVEDD-
LVPW-
Diastolic dysfunction-
Valve-
Pericardial effusion-
Inference-

USG (for diagnostic purpose **)-**

INFORMED CONSCENT FORM

Subject identification number for this trial _____
TITLE OF THE STUDY- To Study of Clinical, Electrocardiographic And 2DEchocardiograpy findings in Chronic kidney disease Patients.
Name of the Principal Investigator: Dr.Pankaj Joshi

I have received the information sheet on the above study and have read and / or
understood the written information. I have been given the chance to discuss the study and ask questions.
I consent to take part in the study and I am aware that my participation is voluntary.
I understand that I may withdraw at any time without this affecting my future care.
I understand that the information collected about me from my participation in this
research and sections of any of my medical notes may be looked
at by responsible persons (ethics committee members / regulatory authorities).
give access to these individuals to have access to my records.
I understand I will receive a copy of the patient information sheet and the informed consent form.

Signature / Thumb Impression of subject Date of signature

सूचित सहमति प्रपत्र

इस परीक्षण के लिएविषय पहचान संख्या_____

परियोजना का शीर्षक: Study of clinical ,Electrocardiographic and 2D-Echocardiography findings in CKD Patients.

जांचकर्ता: डॉ पंकज जोशी

मुझे उपरोक्त अध्ययन पर सूचना पत्र प्राप्त हुआ है और मैंने पढ़ा है और या लिखित सूचना समझा।मुझे अध्ययन पर चर्चा करने और प्रश्न पूछने का मौका दिया गया है।मैं अध्ययन में भाग लेने की सहमति देता हूं और मुझे पता है कि मेरी भागीदारी स्वैच्छिक है।भविष्य की देखभाल को प्रभावित किए बिना किसी भी समय वापस ले सकता हूं।मैं समझताहूं कि इस में मेरी भागीदारी से मेरे बारे में एकत्र की गई जानकारी मेरे किसी भी मेडिकल नोट्स के शोध और अनुभागों को देखा जा सकता है जिम्मेदार व्यक्तियों (नैतिकता समिति केसदस्यों / नियामक प्राधिकरण द्वारा)।मैं मेरे रिकॉर्ड तक पहुंचने के लिए इन व्यक्तियों तक पहुंच प्रदान करें।मैं समझता हूं कि मुझे रोगी सूचना पत्र की एक प्रति प्राप्त होगी और सूचित किया जाएगा

हस्ताक्षर / अंगूठे इंप्रेशन हस्ताक्षर की तिथि