"A STUDY OF THE CLINICAL PROFILE, LEFT VENTRICULAR MASS INDEX AND DIASTOLIC DYSFUNCTION IN PATIENTS WITH DIABETIC NEPHROPATHY ON HAEMODIALYSIS"

By

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Under the guidance of

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I hereby declare that this dissertation titled **"A STUDY OF THE CLINICAL PROFILE, LEFT VENTRICULAR MASS INDEX AND DIASTOLIC DYSFUNCTION IN PATIENTS WITH DIABETIC NEPHROPATHY ON HAEMODIALYSIS"** is a bonafide and genuine research work carried out by me under the guidance and supervision of **Dr. VIDYA SAGAR.C.R.** MD, Associate Professor, Department of General Medicine, Sri. Devaraj Urs Medical College, Tamaka, Kolar.

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LIST OF ABBREVIATIONS

%	-	Percentage
APKD	-	Adult Polycystic Kidney Disease
AER	-	Albumin Excretion Rate
AGE	-	Advanced Glycation End Products
BSA	-	Body Surface Area
BV	-	Blood volume
CGN	-	Chronic Glomerulonephritis
CHF	-	Congestive Heart Failure
CKD	-	Chronic Kidney Disease
CRF	-	Chronic Renal Failure
CVD	-	Cardiovascular Disease
CVS	-	Cardiovascular System
Ca2+	-	Calcium
Chol	-	Cholesterol
Cms	-	Centimeters
Cr	-	Creatinine
CrCl	-	Creatinine Clearance
DM	-	Diabetes Mellitus
DN	-	Diabetic Nephropathy
ECM	-	Extra cellular Matrix
ESRD	-	End Stage Renal Disease
GFR	-	Glomerular Filtration Rat**e**
HD	-	Haemodialysis

Hb	-	Hemoglobin
HDL	-	High Density Lipoproteins
HR	-	Heart Rate
HTN	-	Hypertension
IVSDd	-	Thickness of the Interventricular Septum
K+	-	Potassium
Kg	-	Kilogram
E	-	Mitral Inflow peak early Diastolic Velocity
A	-	Mitral Inflow peak late Diastolic Velocity
E/A	-	Early to late peak Mitral Inflow Velocity
LDL	-	Low Density Lipoproteins
LVIDd	-	Internal Diameter of the Left Ventricle at End Diastole
LV	-	Left Ventricle
LVH	-	Left Ventricular Hypertrophy
LVM	-	Left Ventricular Mass
LVMI	-	Left Ventricular Mass Index
M	-	Meters
P	-	Phosphorus
PWd	-	Thickness of the Posterior Wall in the End Diastole
Sr	-	Serum
Tg	-	Triglycerides

ABSTRACT

BACKGROUND AND OBJECTIVES :

In recent years, prevalence of Chronic Renal failure has increased in the community, resulting in increasing number of admission and thus decreasing the life expectancy.

Diabetes is fast gaining the status of a potential epidemic in India with more than 62 million diabetic individuals currently diagnosed with the disease. In 2000, India topped the world with 31.7 million, the highest number of people with diabetes mellitus and the prevalence of diabetes is predicted to double globally from 171 million in 2000 to 366 million in 2030 with a maximum increase in India.

Diabetes has many long-term complications, despite intensive treatment, causing serious handicaps at relatively younger age. Diabetic nephropathy is one of the major complications that develops in up to 30% of patients and is the leading cause of end-stage renal disease (ESRD). Besides the eventual loss of kidney function, the need for dialysis treatment and transplantation, this complication also increases the risk of early death from cardiovascular disease.

Chronic Renal Failure affects almost all systems of body and results in various abnormalities. Of various causes, infection and cardiovascular events contribute towards large proportion of increased morbidity and mortality. In patients with chronic kidney disease (*CKD)*, uremic toxins and left ventricular (*LV*) dyssynchrony are the factors that may lead to LV dysfunction and conduction abnormalities and thus contribute to high cardiac mortality.

The increase in extra-cellular fluid volume (ECV) is predominantly seen in haemodialysis (HD) patients and correlates positively with increments in blood pressure (BP) and cardiovascular (CV) mortality. It predisposes to the left ventricular hypertrophy (LVH) and contributes to the diastolic heart failure and myocardial ischemia. It is thus not surprising that CV mortality among dialysis patients is much higher than in the general population.

Left Ventricular Hypertrophy (LVH) and diastolic dysfunction are the major Echocardiographic findings in patients with Chronic Renal Failure (CRF) and there is scanty information on the prevalence of Left Ventricular Hypertrophy, nature of LVH, and diastolic dysfunction in patients with CRF.

Aim of the present study is to estimate the prevalence of Left Ventricular Hypertrophy by Echocardiography in patients with Diabetic Nephropathy and to find out the Correlation of Left Ventricular Hypertrophy and Diastolic Dysfunction with severity of Chronic Renal Failure.

METHODOLOGY:

A total of 50 diabetic nephropathy patients of age group 40 to 60 years undergoing haemodialysis admitted or who visited on out patient basis to R. L. Jalappa Hospital attached to SDUMC, Tamaka, Kolar between February 2013 and February 2014 formed the study population.

Detailed history, Clinical evaluation, Laboratory investigations, Echocardiography was carried out

RESULTS:

The Prevalence of Left Ventricular Hypertrophy in Chronic Renal failure was 72%, consisting of 70% males and 30% females, and the prevalence of Diastolic dysfunction was 40% consisting of 75% males and 25% females.

In the present study, we found that Left Ventricular Mass Index (LVMI) which reflects Left Ventricular Hypertrophy (LVH) showed a progressive rise with increase in severity of renal failure with 17% of Mild CRF category having LVH as compared to 30% of Moderate CRF category and 53% of patients of Severe CRF category having LVH.

In the present study, we also found that Diastolic Dysfunction also showed a progressive rise with increase in severity of renal failure with 100% of patients of Severe CRF category having Diastolic Dysfunction.

CONCLUSIONS:

Prevalence of Left Ventricular Hypertrophy in Chronic Renal failure was 72% and that of Diastolic Dysfunction was 40 %. The present study shows that patients with chronic renal failure have higher left ventricular mass index, higher prevalence of left ventricular hypertrophy (LVH) and diastolic dysfunction which is more marked in patients with severe chronic renal failure.

The high prevalence of Left ventricular hypertrophy and diastolic dysfunction in these population on echocardiography implies that these patients require detailed cardiovascular evaluation despite absence of symptoms, and also that various efforts aimed at prevention and control should be started early during the course of renal insufficiency, such as effective control of anemia, diabetes

KEYWORDS: Left Ventricular Hypertrophy, Left Ventricular Mass Index, Diastolic Dysfunction, Chronic Renal Failure, Diabetes.

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INTRODUCTION

Chronic Kidney Disease (CKD) is one of the major public health problems. Incidence is increasing over years and is often under diagnosed and under-treated, the reason being treatment cost is high and outcome is poor. CKD is becoming increasingly common due to rising incidence of diabetes, hypertension, obesity and ageing population in India. Once the patients develop CKD, progress to End Stage Renal Disease (ESRD) and require renal replacement therapy, but many of them die of non-renal causes particularly premature cardiovascular events.

Chronic Renal Failure (CRF) is the common condition which a physician comes across in day to day practice. Chronic Renal Failure affects every aspect of the lives of the patients who suffer it and involves all systems of body and results in various abnormalities.[1, 2, 3]

Chronic renal failure is defined as irreversible, substantial and usually long standing loss of renal function causing ill health.[1]

The exceedingly high cardiovascular (CV) mortality rate among patients with ESRD is a challenge for kidney disease research. In the 45-54 year age group, the CV mortality rate among dialysis patients is about 65 times higher than in the general population.[4] It is estimated that cardiovascular diseases (CVD) account for 40 to 50 % of all ESRD mortality.[2,3,5,6,7,8,9,10] Of the various causes, infection and cardiovascular events contributes towards large proportion of increased morbidity and mortality.[3,10]

Fluid retention inevitably leads to dilatation of the heart compartments and a direct relationship is found between Blood Volume (BV) and left ventricular (LV) diameter in Haemodialysis (HD) patients.[12] LVH is an independent predictor of survival, present in approximately 70 % of ESRD patients, and as many as 50 % of HD patients may have diastolic dysfunction of the LV.[11,13,14] Prevalence of LVH increases with decline of renal

function. Data available suggests that age, sex, hypertension, anemia are significantly associated with LVH and they are independent risk factors.

Left Ventricular Hypertrophy (LVH) is a major Echocardiographic finding in Chronic Renal Failure (CRF).[1,3,15,16] Doppler-derived indices are used for the estimation of LV diastolic function. There is scanty information on the prevalence of Left Ventricular Hypertrophy and nature of LVH in patients with CRF.[2]

It is recommended that all ESRD patients be evaluated for LV systolic function and valvular disease after HD initiation.[17] The most cost-effective test is echocardiography and should be performed early in the course of CRF as may be valuable in the monitoring of therapy of these patients.[3]

AIMS AND OBJECTIVES

1. To Study the Prevalence of Left Ventricular Hypertrophy by Echocardiography in Patients with Diabetic Nephropathy on Haemodialysis.

2. To Find out Correlation of Left Ventricular Hypertrophy and Diastolic Dysfunction with Severity of Chronic Renal Failure**.**

REVIEW OF LITERATURE

End Stage Renal Disease (ESRD)

The term End Stage Renal Disease (ESRD) was developed only after renal replacement therapy became available with haemodialysis, peritoneal dialysis and renal transplantation during early1960s.

Chronic kidney disease (CKD)

Chronic kidney disease encompasses a spectrum of different pathophysiologic processes associated with abnormal kidney function, and a progressive decline in glomerular filtration rate (GFR).[18]

Chronic Renal Failure

The term *chronic renal failure* applies to the process of continuing significant irreversible reduction in nephron number, and typically corresponds to Chronic kidney disease **(**CKD) stages [3,11,18,19]

Diabetic nephropathy

Diabetic nephropathy is defined as a multifaceted pathologic entity which spans the continuum from early renal hypertrophic changes to late stages of advanced structural distortion of glomeruli, renal vasculature, interstitium, and tubules.

End Stage Renal Disease

The term *end-stage renal disease* represents a stage of CKD where the accumulation of toxins, fluid, and electrolytes normally excreted by the kidneys results in the *uremic syndrome*. This syndrome leads to death unless the toxins are removed by renal replacement therapy, using dialysis or kidney transplantation.[18]

Severity of Chronic Renal Failure:

Classification of Chronic Kidney Disease (CKD) Based on GFR[12]	
Stage	**GFR, mL/min per 1.73 m^2**
0	>90[a]
1	>90[b]
2	60–89
3	30–59
4	15–29
5	<15

[a]With risk factors for CKD.

[b]With demonstrated kidney damage (e.g., persistent proteinuria, abnormal urine sediment, abnormal blood and urine chemistry, abnormal imaging studies

Based on Serum Creatinine [2] :

Mild CRF - Sr. Creatinine - 1.5 to 3 mg/dl

Moderate CRF - Sr. Creatinine - 3 to 6 mg/dl

Severe/Advanced CRF - Sr. Creatinine - >6 mg/dl

Glomerular Filtration Rate (GFR)[20]

Normal Glomerular Filtration Rate (GFR) :

Human kidneys contain approximately 1 million glomeruli. Each glomerulus is approximately 150 to 200 micrometer in diameter. The total surface area provided for filtration is approximately 1 meter square. Normally 125 ml/min GFR produces approximately 180 lts/day of tubular fluid from the rich renal plasma flow by the process of ultra filtration.

The kidney has many functions including: glomerular filtration, reabsorption and secretion in the tubules, concentration and dilution of urine, acidification of urine and production and metabolism of hormones.

The most important parameter in assessing kidney function and the progression of renal disease is glomerular filtration rate (GFR), the renal excretory capacity.

Glomerular filtration rate and the concept of renal clearance

GFR is the measurement of how much filtrate is made by the glomeruli. It is an excellent measure of the excretory function of the kidney. For each individual nephron the filtration is determined by the plasma flow, the net pressure gradient, the capillary surface area and the capillary permeability.

The GFR of the whole organism corresponds to the sum of GFR for all nephrons (approximately 1 million per kidney). GFR is described in terms of renal clearance, a concept originally developed by Homer Smith.

The clearance of a substance is the volume of plasma from which all the substance is removed and excreted into the urine per unit time, and is a 'virtual' volume. In clinical practice, endogenous creatinine is usually the substance chosen for clearance measurements.

More accurate estimation of GFR requires the administration of an exogenous substance by continuous infusion or single injection, for example inulin, iohexol, or Cr-EDTA.

The renal clearance of a filtration marker, C, is defined as:

$$C = \frac{UV}{P}$$

Where, P is the plasma concentration of the marker,

U is the urine concentration, and

V is the urine flow rate.

In actual clearance studies, the time over which urine is collected is required. Therefore this formula is written as

$$C = \frac{Uex}{AUC}$$

Where, Uex is the amount of urine excretion of the marker and

AUC is the area under plasma concentration curve during collection of urine.

This formula can be rewritten as:

$$C = \frac{Uex}{(P't)}$$

Where P' is the mean plasma concentration of the marker during the urine collection

t is the period of urine collection.

The clearance of any chosen marker substance only equals GFR if a number of criteria for the measured substance are met. The substance must be freely filterable at the glomerulus, and neither secreted nor reabsorbed by the tubules. It must be in steady state concentrations in the blood, with no extra renal route of excretion. It must also be easily and accurately measured.

Measurement of GFR in clinical practice

Creatinine clearance is the usual method in clinical practice for estimation of GFR. Even simpler are the measurement of plasma urea and creatinine; these however have significant limitations.

Plasma urea:

An elevated plasma urea suggests impaired renal function. However, urea is the main end product of protein catabolism, and individuals with a high protein intake may have elevated plasma urea without any renal dysfunction.

Plasma urea also rises disproportionately to creatinine in the context of extra-cellular volume depletion, because tubular reabsorption of urea is increased.

Plasma creatinine:

Creatinine is an endogenous substance mainly produced in muscle cells from creatine and phosphocreatine. The production rate is almost constant. Hence, the steady state concentration of plasma creatinine depends on its excretion, which mainly reflects GFR. However, large changes in GFR correspond to only small changes in plasma creatinine when patients have near normal renal function, making plasma creatinine a less sensitive marker of GFR in the early stages of renal disease. Moreover, Creatinine is not an ideal GFR marker since tubular secretion of creatinine is enhanced when renal function is reduced.

Most important is that production of creatinine depends on muscle mass, so plasma creatinine under estimates renal impairment in those with reduced muscle mass, including women, children, the elderly and those with malnutrition.

Creatinine clearance:

The Creatinine Clearance (Ccr) is defined as:

$$Ccr = Ucr\ V / Pcr$$

Where, Pcr is the plasma concentration of creatinine,

Ucr is the urine concentration of creatinine, and

V is the urine flow rate.

Ccr is steady state estimation and therefore cannot be interpreted when renal function is rapidly changing, for example during the course of acute renal failure. Although superior to plasma creatinine as a measurement of GFR, Ccr has other significant limitations.

Timed urine collection

Measurement of Ccr requires a timed collection of urine with a single blood sample, ideally drawn during the collection. Incomplete urine collection is a major source of inaccuracy in Ccr measurement in clinical practice.

The patient should be instructed to pass urine into the toilet on rising, and then collect all urine subsequently passed over the next 24 h including the urine passed on rising the following day.

Major inaccuracies are suggested by a low urine volume, but completeness of the urine collection is better assessed by comparing urinary creatinine with normal daily creatinine production: men 20-25 mg/kg/day (0.18-0.22 mmol/kg/day); women 15-20 mg/kg/day (0.13-0.18 mmollkg/day).

Tubular secretion:

Creatinine is secreted by the tubules as well as filtered by the glomerulus; therefore, Ccr consistently exceeds true GFR.

Cimetidine, trimethoprim, and probenecid inhibit proximal secretion of creatinine, elevate plasma creatinine and diminish creatinine clearance. Oral administration of cimetidine 600 mg before Ccr measurement has been recommended to inhibit creatinine secretion, thus producing values of Ccr closer to those of GFR, although this is not widely used in clinical practice.

Creatinine Generation:

Calculation of Ccr depends on the assumption that the concentration of plasma creatinine is constant. However, plasma creatinine level and urinary excretion of creatinine are both higher during the day than at night, mainly due to the absorption of exogenous creatinine contained in the diet. A transient increase in plasma creatinine and urinary creatinine excretion are observed after eating cooked meat, which contains a considerable amount of creatinine. For this reason, Ccr calculated from short-term daytime urine collections is about 20% higher than that calculated from a 24-h urine collection.

Assay methods for Creatinine:

There are two routine methods for assaying creatinine: a method using the Jaffe reaction and an enzymatic method. The Jaffe reaction in which creatinine reacts with an alkaline solution of picrate is still widely used but is not specific for creatinine. Non creatinine chromogens such as glucose, acetoacetate, ascorbic acid, and some cephalosporins, particularly cefoxitin, react positively and cause the Jaffe reaction to overestimate plasma creatinine.

The Jaffe reaction over-estimates plasma creatinine by about 0.2 mg/dl for normal plasma creatinine values and by up to 0.4 mg/dl when plasma creatinine is 10 mg/dl. Over-estimation of plasma creatinine is offset by tubular secretion of creatinine, with the net effect that Ccr by the Jaffe assay is close to GFR in normal individuals.

The effect of the positive bias by the Jaffe assay becomes negligible as plasma creatinine increases, but creatinine secretion is greater with reduced GFR; hence the difference between Ccr, and GFR expands in patients at lower GFR.

The enzymatic method, now widely available in clinical use, is much more accurate and is recommended for Creatinine measurement.

The dissociation between Ccr, by the enzymatic method and GFR is seen regardless of renal function. Enzymatic assays are also prone to interference by drugs.

***Predicted creatinine clearance* - the Cockcroft-Gault formula**

Rapid estimation of creatinine clearance from plasma creatinine values, without recourse to urine measurement, is clinically useful. Creatinine generation decreases linearly with advancing age, as muscle mass falls, and a number of formu- lae for the estimation of creatinine excretion have been derived to account for this. The most widely used formula is that of Cockcroft and Gault. The formula over-estimates the true Ccr values in obese patients, and also in those on a low protein diet. Equations that takes body fat into account predict Ccr, better than the Cockcroft-Gault formula, but are more complicated and not widely used.

$$\text{CrCl} = \frac{\{140 - \text{Age(yrs)}\} \times \text{Weights (Kgs)}}{\text{Plasma Creatinine} \times 72} \quad \text{For Males}$$

$$\text{CrCl} = \frac{\{140 - \text{Age(yrs)}\} \times \text{Weights (Kgs)}}{\text{Plasma Creatinine} \times 72} \times 0.85 \quad \text{For Females}$$

***Predicted GFR* - a new equation from the MDRD study:**

An equation that predicts GFR from plasma creatinine concentration was developed from data obtained in the Modification of Diet in Renal Disease (MDRD) Study. This equation proved more accurate than measured Ccr, in both the MDRD study, and the African-American Study of Hypertension and Kidney Disease.

The equation has not yet been validated in children, the elderly or pregnant women, nor where there are extreme values for serum albumin concentration. This equation is less quid than the Cockcroft-Gault formula to use in everyday clinical practice and a nomogram to speed the estimation.

Other measurements of GFR:

Methods of measuring renal clearance more accurate than Ccr and its calculated estimates are required in some clinical settings and are also a valuable research tool. To avoid the in-accuracies of urine collection, the total plasma clearance is measured of an exogenous filtration marker which has little or no extra renal excretion.

Plasma clearance with continuous injection - inulin clearance

Plasma clearance of a filtration marker, C, is defined as:

C = *Tex / AUC*

Where, Tex is the total amount excreted from plasma and

AUC is the area under the plasma concentration curve.

When a filtration marker is infused intravenously or subcutaneously at a constant rate, the plasma concentration reaches a steady state. At this point, the urinary excretion of the marker will be identical to the rate of infusion. Therefore, GFR can be calculated from plasma concentration and infusion rate of the marker.

When the plasma concentration does not reach a steady state, GFR will be overestimated; therefore this method requires a relatively long study period (3-24 h). Although cumbersome, this is the most accurate method and inulin clearance with continuous infusion is the 'gold standard' for measurement of GFR.

Inulin, a 5200-Da uncharged polymer of fructose, satisfies the criteria for an ideal clearance substance since it is freely filtered at the glomerulus and is not reabsorbed, secreted, synthesized or metabolized by the tubules.

But, as well as the practical disadvantages of a continuous infusion technique, the Anthrone method, often used to assay inulin, is complicated and high glucose concentrations can give false-positive reactions. The alternative enzymatic assay of inulin is not widely available.

Plasma clearance with single injection

After a bolus intravenous injection, the disappearance curve of the plasma concentration represents two components: a rapid phase reflecting the distribution of the marker from intravascular space to extravascular space and a slow phase reflecting the renal excretion of the marker.

A two compartment model is required to describe the entire plasma disappearance curve, but requires frequent blood sampling, which is impractical. Therefore, a one-compartment model is widely used which matches only the slow phase of the plasma disappearance-curve.

This model is expressed as :

$C(t) = Ae^{-BT}$

$\ln C(t) = -Bt + A$

Where, $C(t)$ is the plasma concentration of the marker at a given time (t),

A is the zero- time intercept, and

B is the rate constant, the falling slope of the marker in semilogarithmic plot.

The line can usually be determined by two blood samples during the slow phase (e.g., at 90 and 120 min after injection), although a relatively long time (3-24 h) may be required to obtain the accurate falling slope of the marker in patients with impaired renal function.

Plasma clearance is slightly overestimated in this simple one-compartment model, since it does not account for that part of the *AVC* resulting from the rapid phase. Brochener-Mortensen introduced the following equation to correct the clearance values:

Corrected plasma clearance = 0.990778C - 0.001218C

Where, C is the original clearance from the one-compartment model.

To enhance the convenience of these techniques, a single sample method has been developed, using a sample taken 3-5 h after the bolus injection. Zero time intercept is calculated by dividing the injected dose by a distribution space of the marker estimated from body weight.

However, the estimation of the distribution space is difficult in patients with severe edema or ascites.

Epidemiology of ESRD[21,22,23,24]

Chronic Renal Failure is a world wide health problem. According to World Health Organization (WHO) Global Burden of Disease Project diseases of kidney and urinary tract contribute to Global burden with approximately 8,50,000 deaths every year and 115,010,107 disability adjusted life year.

Chronic Renal failure is the 12th leading cause of Death and 17th cause of Disability. This Global problem may be grossly under-estimated for a number of reasons.

Patients with CRF are at high risk for cardiovascular (CVD) and Cerebrovascular (CNS) diseases and they are more likely to die of CVD than to develop End Stage Renal failure.

The population of the India exceeds one billion and is projected to become the major reservoir of chronic diseases like diabetes and hypertension. Since 25-40% of these may develop chronic renal failure, the End stage renal disease (ESRD) burden will rise and health care system would need to take care of them.[21]

With the transition of demographic profile from 'younger' to 'older' populations, there is a gradual, concurrent epidemiological transition taking place worldwide.[22] Old age together with unhealthy diets, tobacco usage, obesity and sedentary life styles is contributing to the increase in chronic diseases like cancers, cardiovascular diseases, diabetes, hypertension, neurological and psychiatric diseases.

It is evident that all the countries in the world, irrespective of their economic development are facing an increasing trend in non-communicable diseases (NCDs), which are expected to account for 73 per cent of deaths and 60 per cent of disease burden by the year 2020.[22]

The prevalence of End stage renal disease varies from country to country, and depends on the incidence of the particular diseases.[23]

There is an epidemiological transition taking place in India, with the decline in communicable diseases and a growing burden of chronic disease.[23]

In a recent review, Reddy and others noted that 53 percent of deaths in India were due to chronic disease. The principal named categories of chronic disease in their report were cardiovascular disease, cancer, chronic respiratory disease and diabetes.[23]

Notably, chronic kidney disease (CKD) was not a category on its own merits but most likely included under the 'other' category.

The World Health Organization laid down certain criteria for a major non communicable disease (NCD), namely, [25,26]

(i) being a major cause of morbidity and mortality,

(ii) being axenable to prevention by community based strategies, and

(iii) sharing common risk factors with other NCDs.

Though CKD meets these criteria, it does not find a place in this category.

There is no reason to suspect that the global epidemic of CKD does not have its counterpart in India and epidemiologic indicators suggest that it is likely to be sizeable.[23]

Type 2 diabetes, the most common type in all populations and the number of patients has rapidly increased in the past few decades and is still rising further. In 2002, the number of diabetes patients worldwide was estimated at 173 million and has been predicted to increase to 350 million in 2030.

India has been described as the Diabetes capital of the world, every fifth diabetic in the world being Indian.[21,23]

The increasing prevalence of diabetes, hypertension and associated risk factors such as obesity, hypercholesterolemia and the metabolic syndrome underscores the potential for sustained and explosive growth of this epidemic.

The epidemiology of CKD in India is very different from the West. Patients are roughly two decades younger, and a substantial proportion present with small kidneys, so the etiology of CKD is unclear.[21]

Chronic kidney disease (CKD) is a global threat to health in general and for developing countries in particular, because therapy is expensive and life-long.[24]

In India >90% patients cannot afford the cost. Over 1 million people worldwide are alive on dialysis or with a functioning graft. Incidence of CKD has doubled in the last 15 years. Risk factors for developing CKD differ between races and countries.

It would be interesting to know the incidence of CKD and its causes in India, The currently reported incidence of CRF in India is based on extrapolated data from the US. As yet, no large-scale population studies are available.

According to two studies conducted.[21,23,24]

(i) a population screening in New Delhi and

(ii) a second prospective study that involved 48 hospitals.

Thirty-seven were found to have chronic renal failure (prevalence rate of 0.78%). If these data are applied to India's 1 billion population there are >7.85million CRF patients in India.

Etiologically:

1. Diabetes (41%),
2. Hypertension (22%),
3. Chronic glomerular nephritis (16%),
4. Chronic interstitial disease (5.4%),
5. Ischaemic nephropathy (5.4%),
6. Obstructive uropathy (2.7%),
7. Miscellaneous (2.7%) and
8. Unknown cause (5.4%)

All these constituted the spectrum.

The second study was more representative, as 48 centers were distributed all over India. Data were based on prospective investigations conducted over a period of 1 (33 hospitals) to 3 months (15 hospitals) comprising 4145 CKD patients.

It showed the following etiological pattern:[21,24]

1. Diabetes (29.7%),
2. Chronic glomerulonephritis (19.3%),
3. Hypertension (14%),
4. Chronic interstitial disease and vesico-ureteral reflux (12.6%),
5. Obstruction and calculus (9.3%),
6. ADPKD and Alport Syndrome (8.4%),
7. Undiagnosed (6.2%).

This study shows that the prevalence of CRF in India is >0.8%. If the two studies are combined [21,23,24]

- Diabetes has emerged as the most frequent cause (30–40%) followed by
- Hypertension (14–22%),
- CGN (16–20%),
- CIN (5.4–12.7%),
- Heredofamilial disease (8.4%),
- Obstruction including calculus (2.9%).

Pathophysiology of chronic kidney disease and ESRD [27]:

The Pathophysiology of CKD involves two broad sets of mechanisms of damage:

1. Initiating mechanisms specific to the underlying etiology (e.g., immune complexes and mediators of inflammation in certain types of glomerulonephritis, or toxin exposure in certain diseases of the renal tubules and interstitium); and a set of progressive mechanisms, involving hyperfiltration and hypertrophy of the remaining viable nephrons, that are a common consequence following long-term reduction of renal mass, irrespective of underlying etiology.

   The responses to reduction in nephron number are mediated by vasoactive hormones, cytokines, and growth factors. Eventually, these short-term adaptations of hypertrophy and hyper filtration become maladaptive as the increased pressure and flow predisposes to sclerosis and dropout of the remaining nephrons.

   Increased intra-renal activity of the renin-angiotensin axis appears to contribute both to the initial adaptive hyper filtration and to the subsequent maladaptive hypertrophy and sclerosis, the latter, in part, owing to the stimulation of transforming growth factor (TGF-beta). This process explains why a reduction in renal mass from an isolated insult may lead to a progressive decline in renal function over many years.

Pathophysiology and Biochemistry of Uremia [27]

Although serum urea and creatinine concentrations are used to measure the excretory capacity of the kidneys, accumulation of these two molecules themselves do not account for many symptoms and signs that characterize the uremic syndrome in advanced renal failure. The uremic syndrome and the disease state associated with advanced renal impairment involve more than renal excretory failure.

A host of metabolic and endocrine functions normally undertaken by the kidneys are also impaired, and this results in anemia, malnutrition, and abnormal metabolism of carbohydrates, fats, and proteins.

In summary, the pathophysiology of the uremic syndrome can be divided into manifestations in three spheres of dysfunction:

1. Those consequent to the accumulation of toxins normally undergoing renal excretion, including products of protein metabolism;
2. Those consequent to the loss of other renal functions, such as fluid and electrolyte homeostasis and hormone regulation; and
3. Progressive systemic inflammation and its vascular and nutritional consequences

   Hundreds of toxins that accumulate in renal failure have been implicated in the uremic syndrome.

Uremic toxins identified:

Include byproducts of protein and amino acid metabolism. Urea - 80% of total (excreted nitrogen).

Guanidino compounds: Guanidine, Methyl guanidine, Creatinine, Guanidino succinic acid

Urates and hippurates

- End products of nucleic acid metabolism
- End products of aliphatic amino acid metabolism
- End products of aromatic amino acid metabolism

Other nitrogenous substances

- Polyamines
- Myoinositol
- Phenols
- Benzoates
- Indolin

- Advanced glycation end products
- Inhibitors of ligand-protein binding
- Glucuronoconjugates and aglycones
- Inhibitors of somatomedin and insulin action

Compounds with a molecular mass between 500 and 1500 Da, the so-called middle molecules, are also retained and contribute to morbidity and mortality.

Cardiovascular Complications in End Stage Renal Disease

Chronic renal failure (CRF) affects almost all systems of the body.[2, 3]End stage renal disease and cardiac disease seem to be inextricably linked. Of various causes, infection and cardiovascular events contribute towards large proportion of increased morbidity and mortality.[2]

As early as 1827 Richard Bright drew attention to the common presence of left ventricular hypertrophy and thickening of the aortic wall in patients with end stage renal disease.[25,28]

Today, cardiovascular complications are a major clinical problem in ureamic patients accounting for 44% of all deaths in this population.[25,29,30,31,32,33,34]

Death from cardiac causes is 10- 20 times more common in patients with renal failure than in matched segments of the general population.

Several structural and non-structural alterations of the heart and the vasculature are present in the uremic patients, and they presumably contribute to the increased cardiovascular risk in renal failure.

Recent clinical and experimental studies clearly document that the pathogenesis of cardiovascular abnormalities in renal failure is much more complex than initially thought. Apart from elevated BP, hypervolemia and anemia, activation of local systems such as the rennin-angiotensin system (RAS) and endothelin (ET) system plays an important role.[18]

Structural and Functional changes of the heart in renal failure [18,35]

Structural:

1. Left ventricular hypertrophy
2. Hypertrophy of Cardiomyocytes, alterations in myocytes number
3. Intermyocytic fibrosis
4. Coronary heart disease
5. Micro vascular disease - Arteries, capillaries

Functional:

1. Reduction of insulin mediated glucose uptake
2. Reduction in the activity of the insulin dependent glucose transporter
3. Reduced stability of the energy rich nucleotides
4. Abnormal control of intracellular calcium in cardiomyocytes
5. Reduction of the inotropic and chronotropic response to alpha adrenergic stimulation

The risk of cardiovascular disease (CVD) in patients with Chronic renal failure appears to be far greater than in the general population.[26]

Patients with CRF should be considered in the highest risk group for subsequent cardiovascular disease CVD events. Treatment recommendations based on CVD risk stratification should take into account, this "highest risk" status of the patient with CRF.[26]

Among patients treated by haemodialysis or peritoneal dialysis, the prevalence of Coronary Artery Disease is approximately 40% and the prevalence of Left Ventricular Hypertrophy is approximately 75%.[19,26.29,30,36,37]

The burden of Cardiovascular Disease varies inversely with the level of renal function. An inverse relationship between the level of renal function and the prevalence of Left ventricular Hypertrophy was reported by Levin et al.[26,30,38]

Patients with end-stage renal disease (ESRD) are at a much higher risk of Cardiovascular disease than the general population.[39] Evaluating the Cardiovascular organs of potential recipients for the purpose of preventing or, at least, delaying the development of cardiac abnormalities, understanding the determinants of Cardiovascular disease, and careful preparation of interventions aimed at correcting them is very important in the management of ESRD patients.[39]

There is growing evidence suggesting that prevalence of Cardio Vascular (CV) disease among ESRD patients is already high by the time renal replacement treatment is initiated The results of a number of studies suggest that factors leading to the development of CV abnormalities begin to operate very early in the progression of chronic kidney disease, well before patients reach ESRD.[39]

The annual mortality from cardiovascular disease in CRF patients is substantially higher in the general population.[40]

Cardiac risk factors in Chronic Uremia [34, 40]

Traditional Cardiac risk factors

1. Diabetes Mellitus
2. Hyperlipidemia
3. Hypertension
4. Physical inactivity
5. Tobacco use

Risk factors Altered by Uremia

1. Dyslipidemia
2. High lipoprotein (a)
3. Prothrombotic factors
4. Hyperhomocysteinemia

Uremia Related risk factors

1. Haemodynamic overload
2. Anemia
3. Increased oxidant stress
4. Hypoalbuminemia
5. Inadequate dialysis
6. Divalent ion abnormalities
7. Metabolic acidosis
8. Hypo/hyperkalemia.

Pathophysiology of cardiac hypertrophy [41]

Physiologically, left ventricular hypertrophy is primarily an adaptive remodeling process, compensating for an increase in workload placed on the heart with the aim of minimizing ventricular wall stress. Two contrasting models of adaptation may develop depending on the patterns of stress imposed.[41]

Pressure overload, caused, for example, by hypertension or aortic stenosis, requires the generation of greater intracavitary pressure during ventricular contraction. This is achieved by arraying contractile protein units in parallel. Relatively, an increase in wall thickness and a fall in cavity volume take place.

Concentric hypertrophy, as this process is known, leads to decreased diastolic compliance and may place the myocardium at risk of ischemia, even without coronary artery disease.

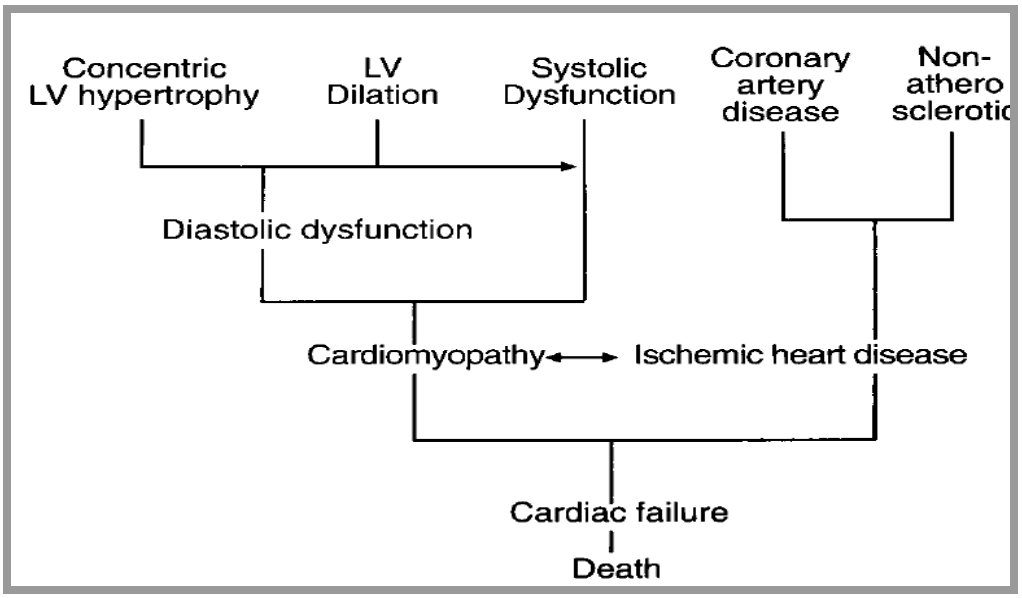
In conditions of volume overload, such as anemia or aortic incompetence, lengthening of contractile units leads to a physiologically useful increase in systolic stroke volume, according to Starling's Law. Unopposed, this process of left ventricular dilation leads to increased wall tension, a state known to increase oxygen requirements and myocytes burnout.

According to the Law of Laplace, the wall tension of a hollow spherical body is directly proportional to radius and pressure and inversely proportional to wall thickness.

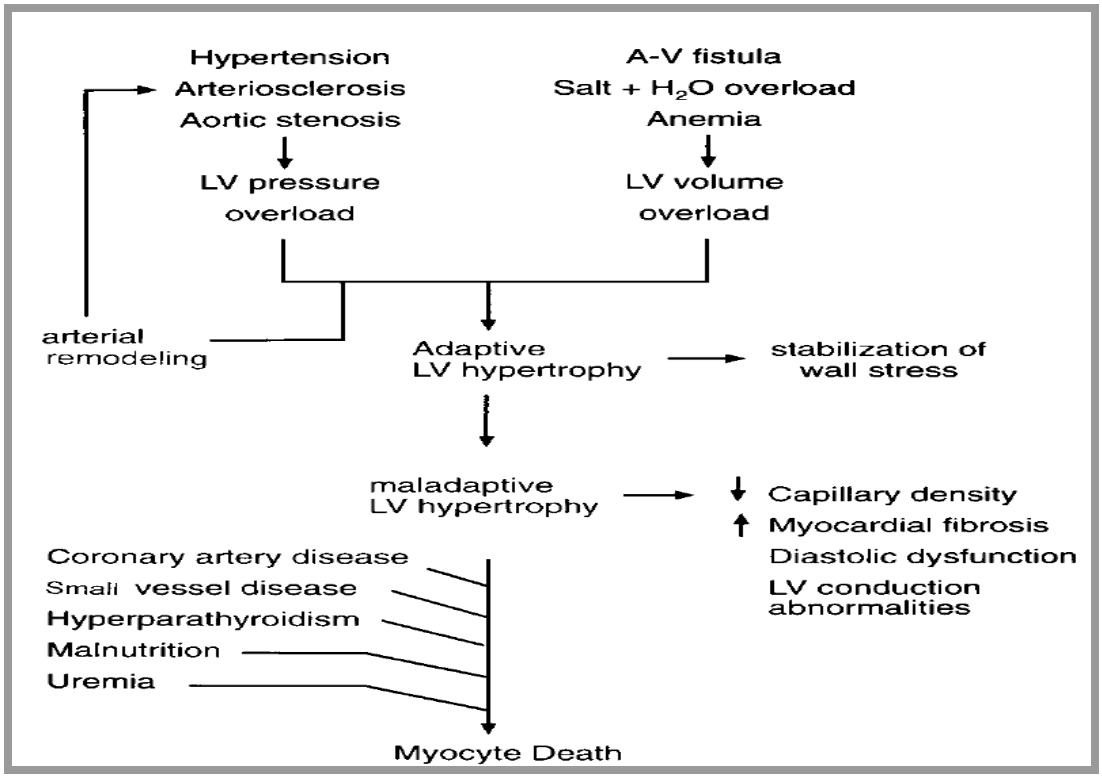
Thus, in states of left ventricular dilation, wall thickening and left ventricular hypertrophy are useful secondary order adaptations that tend to decrease wall tension.

The molecular mechanisms that underlie these processes are slowly being unraveled. Subtle signaling changes can lead from physiologic adaptation to pathologic maladaptation. With continuing pressure and volume overload, cardiac myocyte apoptosis accelerates. In addition, fibrosis accelerates.

Hypertrophy, apoptosis, and fibrosis are influenced by constitutional and genetic factors, hormones, growth factors, and cytokines such as endothelin 1, angiotensin II, insulin-like growth factor, and tumor necrosis factor a. The balance of these factors and downstream intracellular signals can alter the balance among hypertrophy, apoptosis, and fibrosis.



Cardiomyopathy and Ischemic Heart Disease in Chronic Uremia [40]



Left Ventricular pressure overload, Left Ventricular volume overload and Myocyte death in Chronic Uremia [40]

Diagnosis and Classification[41]

Left ventricular hypertrophy is a histologic entity. Myocardial biopsy is rarely performed, so it is rarely possible in practice to prove that maladaptive pathologic features, especially fibrosis, are present. Instead, have to rely on measures of left ventricular size, geometry, and function.

Echocardiography is non-invasive and provides an accurate assessment of each of these parameters. For each parameter, superior techniques exist but are not routinely used because of expense, unavailability, or invasiveness.

Thus, magnetic resonance imaging seems to be a superior technique to assess left ventricular mass and cavity volume in patients with end-stage renal disease (ESRD). Similarly, cardiac function is measured better with invasive techniques.[2, 19,41]

In practice, echocardiography is a reasonable overall tool and is highly suited to longitudinal research studies. [2,3,19,31,41,42] Left ventricular mass is calculated by assuming that the ventricle is a hollow spheroid.

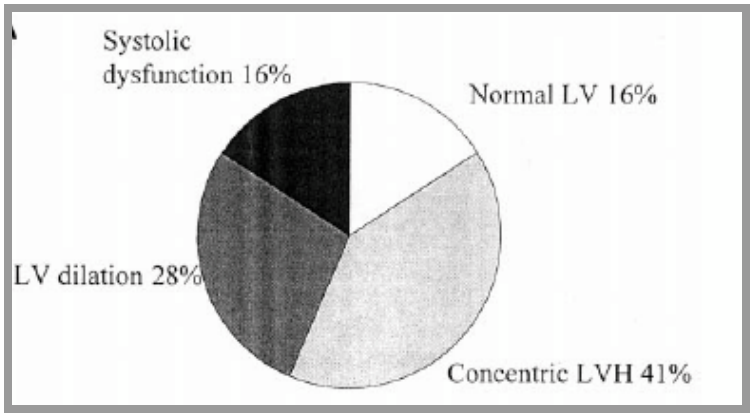
In the general population, left ventricular mass increases with age, male gender, and body size. For comparative purposes, left ventricular mass usually is normalized to some index of body size.

The ideal method remains a matter of debate. However, normalization to body surface area is the most commonly used method.

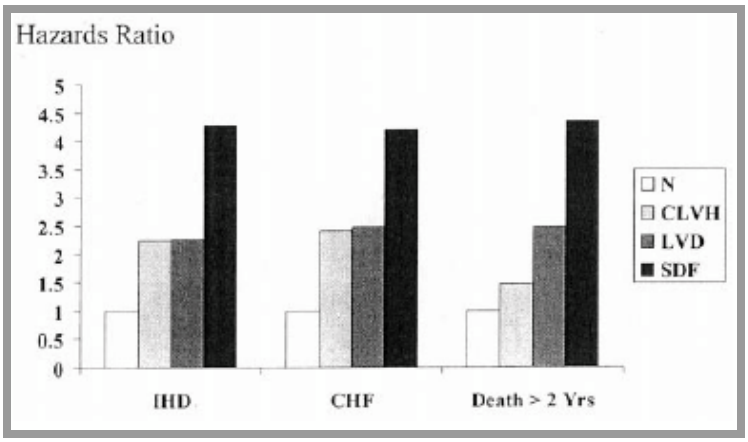
In the healthy adult, Framingham population, the upper limits of normal are:

131 g/m2 for males and

100 g/m2 for females



Prevalence of Left Ventricular Disorders, identified by Echocardiography [19,29,41]



Outcome, adjusted for age, gender, and diabetes mellitus, according to type of left ventricular abnormalities at inception of dialysis therapy. [41]

Where in , N-Normal; CLVH-Concentric left ventricular hypertrophy;

LVD-Left ventricular dilatation; SDF-Systolic dysfunction;

IHD-Ischemic heart disease; CHDF- Congestive heart failure.

Risk factors for Left Ventricular disorders [25,29,30,43,44,45]

Many risk factors for left ventricular hypertrophy have been suggested in chronic renal failure patients [7,27,41]

Not Easily Reversible:

1. Older age
2. Diabetes mellitus
3. Abnormally stiff large arteries

Easily Reversible:

1. Arteriovenous connections
2. Anemia
3. Hypertension
4. Extracellular fluid volume expansion
5. Uremic internal milieu
6. Abnormalities of calcium phosphate homeostasis

Anemia

The association of anemia with Chronic Renal Failure has been recognized since the early 19th century[25] Moreover, various studies done over the years have shown not only a higher incidence of anemia, but also a significantly higher incidence of cardiac complications, particularly Left ventricular hypertrophy. In fact, anemia has been cited as an independent risk factor for the development of LVH and morbidity and mortality in CRF patients.

The importance of anemia in CRF dialysis patients was shown by the observation that decrease in hemoglobin level of 1 g/dl incrementally increased mortality by 18-25% and LVH by 50%.[7,25] Enlargement of the ventricle was related to the degree of anemia and the hemodynamic effect of the arteriovenous fistula.[7,13,27,46,47,48]

The study of Levin *et al.* showed a robust association between modest declines in hemoglobin levels, from a baseline level of 12.8 g/dl, and progressive left ventricular growth in patients with early renal insufficiency.

Anemia in the long term was associated with progressive LV dilation, new- onset cardiac failure, and death in the CRF patients.[7,8,27] These findings are consistent with several observational studies that have suggested a dose-response association between the severity of anemia, mortality, and hospitalization in haemodialysis patients

Several treatment studies demonstrated that partial correction of anemia leads to a decrease in left ventricular dimensions, without leading to full correction of left ventricular hypertrophy and dilation.[8,25,49] In addition, partial correction of anemia has led consistently to improved quality of life, exercise capacity, and cognitive function.

The impact of complete correction of renal anemia is a reasonable question in light of these findings. Several studies have examined this issue, and more are in progress.

The United States Normalization of Hematocrit Trial studied 1233 haemodialysis patients with symptomatic ischemic heart disease or cardiac failure. The primary outcome was either myocardial infarction or death. The Canadian Normalization of Hemoglobin study also compared normalization of hemoglobin to partial correction of anemia in haemodialysis patients.

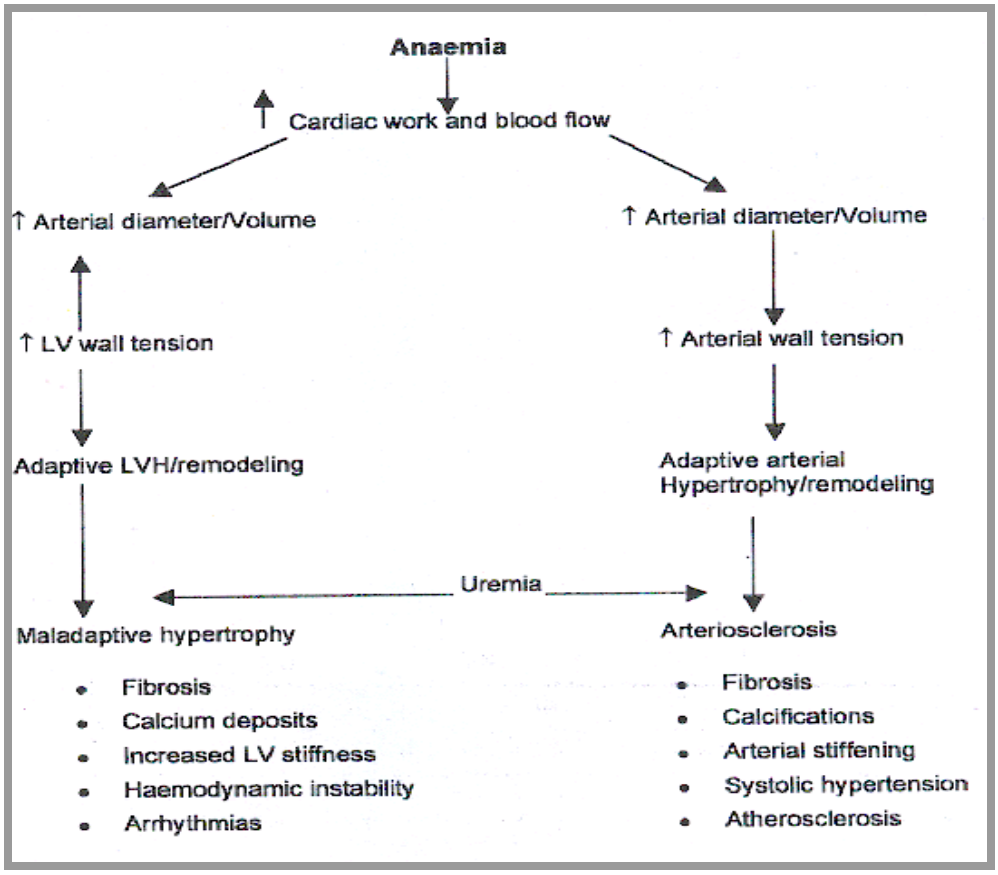
A total of 146 haemodialysis patients with either asymptomatic concentric left ventricular hypertrophy or left ventricular dilation were randomly assigned to maintain hemoglobin levels of 10 g/dl or to be ramped upward, to 13.5 g/dl.

In the left ventricular dilation group, the changes in cavity volume were equivalent in both hemoglobin groups. In the concentric LV hypertrophy group, the changes in left ventricular mass index were similar; those assigned to higher targets, however, were less likely to have developed left ventricular dilation. Patients in the higher hemoglobin arm had less depression and fatigue and improved relationships. There was no increase in the rate of dialysis access loss.

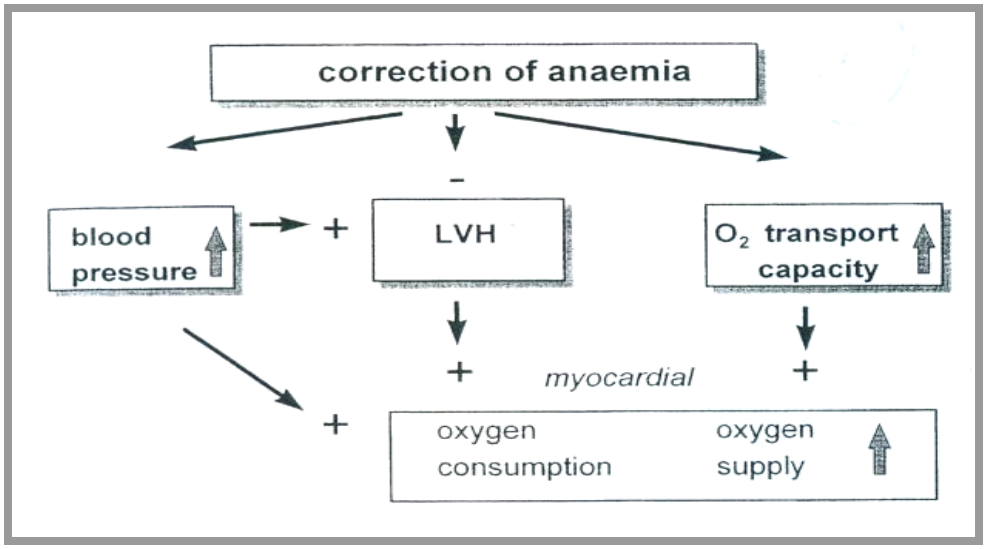
It is likely that the left ventricular changes observed in uremic patients may be more inherently susceptible to prevention than to treatment. The evidence against target hemoglobin levels below 11 to 12 g/dl is persuasive.

Normalization of hemoglobin is very likely to be associated with enhanced quality of life and physical performance, but the safety and cost of this approach and its impact on cardiovascular outcomes remain open questions, as do the relative effects of early and late intervention.[27]

The etiology of anemia in CRF is multifactorial and varies between patients, but the primary underlying defect is erythropoietin deficiency.[7,8,25]



Schematic representation of adaptive and maladaptive remodeling of the cardiovascular system



Potential compromise of the beneficial effects of anemia correction on LVH and myocardial oxygen supply by an uncontrolled increase in blood pressure[49]

Hypertension

The systolic blood pressure is an important factor influencing the occurrence of left ventricular hypertrophy in CRF patients and also affects the left ventricle geometry in this population.[7,8,29,30,39]

The prevalence of hypertension in CRF is approximately 60% to 100%, depending on the target population, cause of renal disease, and level of renal function.[26,30]

Hypertension is associated with cardiovascular disease outcomes in all CRF target populations. Hypertension can be either a cause or a consequence of CRF.

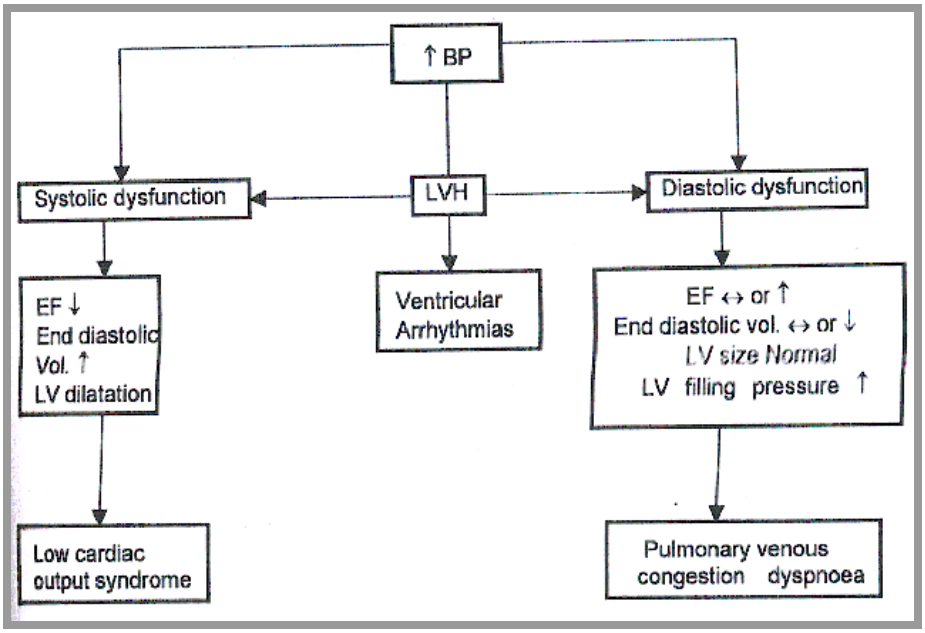
Based on epidemiological data from the National High Blood Pressure Education Program and National Health and Nutrition (NHANES), stroke, myocardial infarction and CHF have decreased by approximately 15% to 40%, whereas hypertensive nephropathy as a cause of ESRD in increasing at annualized rate of 10% for the last several years.[26]

Studies have shown that an increase in systolic blood pressure of 5mmhg is associated with an increase of 3% risk of LVH.

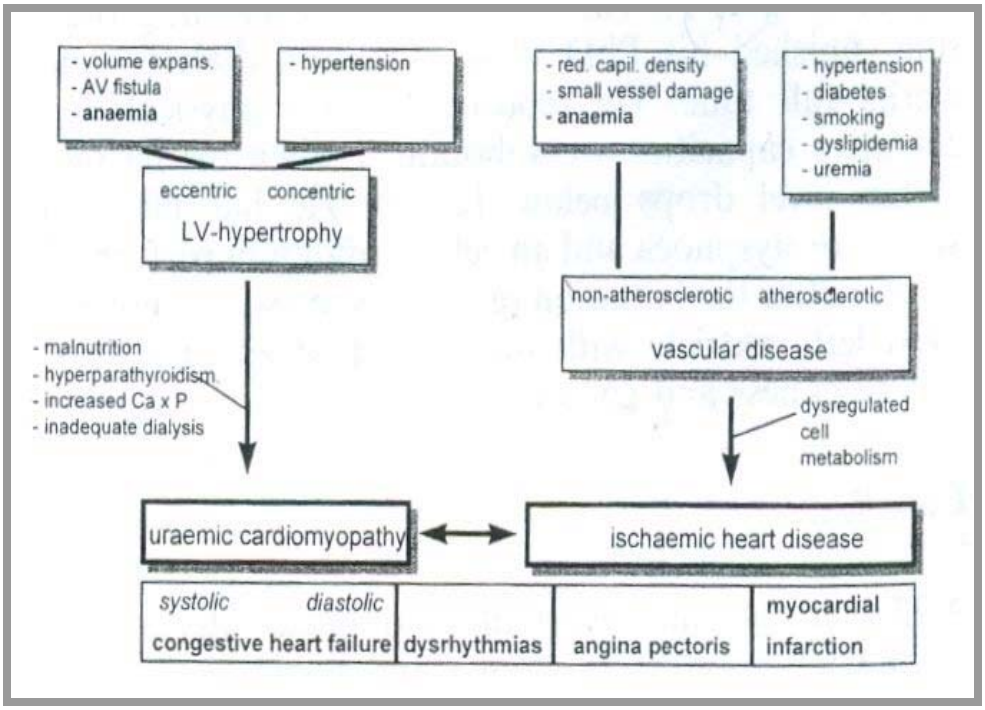
The clinically more important pathogenetic mechanisms of hypertension in CRF are[26]

1. Extracellular fluid volume expansion
2. Renin-angiotensin aldosteron system stimulation
3. Increased sympathetic activity
4. Endogenous digitalis like factors
5. Prostaglandins/bradykinins
6. Alterations in endothelium derived factors (endothelin/nitric oxide)
7. Increased body weight
8. Erythropoietin administration

9. Parathyroid hormone secretion/ increased intracellular calcium /hypercalcaemia
10. Calcified arterial tree
11. Pre-existing essential hypertension
12. Renal vascular disease and renal artery stenosis
13. Chronic allograft dysfunction
14. Cadaver allografts, especially from a donor with a family history of hypertension
15. Cyclosporine, other immune suppressive and corticosteroids therapy



Consequence of systolic and diastolic dysfunction related to hypertension [40]



Pathogenesis and the main contributory of cardiac morbidity and complication in the patients with renal failure.[49]

Diabetes

Renal disease is a common and often severe complication of diabetes. There is a independent correlation between diabetes and LVH .[9,30,50]

Diabetic nephropathy is the cause of ESRD in 15-30% of patients with approximately equal proportions of patients having type I and type II diabetes.[51] Extra-renal complications of diabetes include macrovascular disease and microvascular disease. The overall risk of death from myocardial infarction in a patient with diabetes is approximately three times that of the general population, with younger patients at higher risk and CVD is the leading cause of death [24,52]

Better glycemic control was shown to reduce risk of microvascular complications and better blood pressure control has been demonstrated to reduce risk of cardiovascular events and progression of diabetic nephropathy.[53] Once established on dialysis therapy, the survival for diabetic patients is considerably worse than for patients with other causes of ESRD with a median survival on dialysis survival of 2.5 years.[54] This is mainly attributable to the excess risk of vascular disease.

However the development of nephropathy, heralded by the presence of albuminuria, enhances the risks of further macrovascular complications as well as the risk of progressing to ESRD.[55]

Diabetes is associated with increasing LV mass index in the subset with concentric Left Ventricular Hypertrophy when compared to those with normal echocardiogram. Diabetes has been identified as a predictor in CRF. [29]

The UK prospective diabetes study (UKPDS) and diabetes control and complications trial (DCCT) have established an initiating role for hyperglycemia in developing microvascular complications such as diabetic nephropathy.[56,57]

There are four main hypotheses about how hyperglycemia causes diabetic nephropathy:

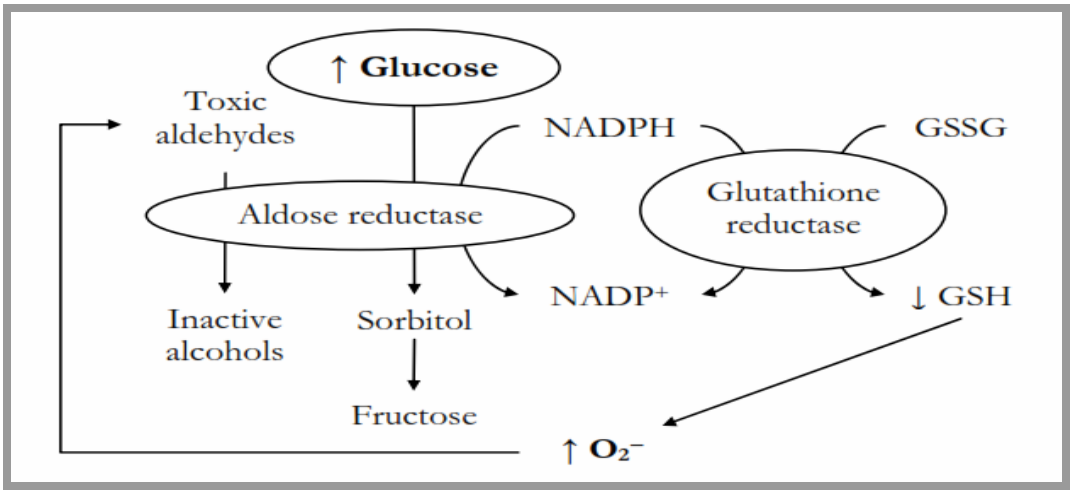
1) increased polyol pathway flux,
2) increased advanced glycation end product formation,
3) activation of the protein kinase C isoforms,
4) increased hexosamine pathway flux.

Polyol pathway flux :

Aldose reductase is the first enzyme in the polyol pathway and an oxidoreductase that catalyses the NADPH-dependent reduction of a wide variety of carbonyl compounds,such as glucose. Aldose reductase has a low affinity for glucose and in the normal situation the metabolism of glucose by this pathway is minimal.

In a hyperglycemic environment, increased intracellular glucose results in its increased enzymatic conversion to sorbitol, with concomitant decrease in NADPH. In the polyol pathway, sorbitol isoxidized to fructose by the enzyme sorbitol dehydrogenase with NAD being reduced to NADH. The most accepted theory how the polyol pathway is involved in causing diabetic nephropathy is as follows. Reduction of glucose to sorbitol consumes NADPH.

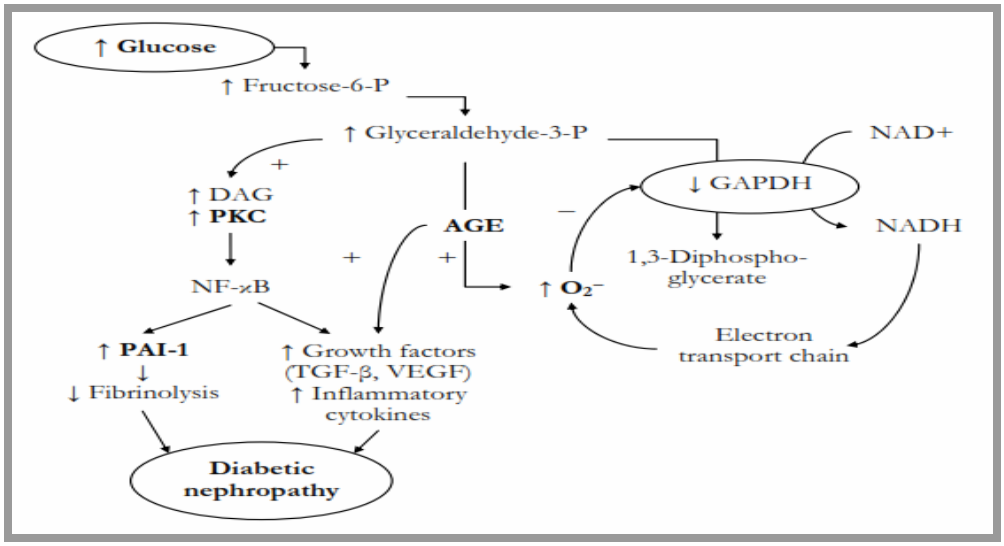
As NADPH is required for generating glutathione, and glutathione protects cells from oxidative stress, this could induce intracellular oxidative stress.[58,59]



Advanced glycation end product formation

Advanced glycation end products (AGE) are irreversibly damaged proteins or lipids resulting from a chain of chemical reactions after an initial glycation reaction.[60] AGE formation is increased in diabetes due to increased intracellular glucose. Intracellular and extracellular AGEs and its precursors damage cells by three mechanisms.

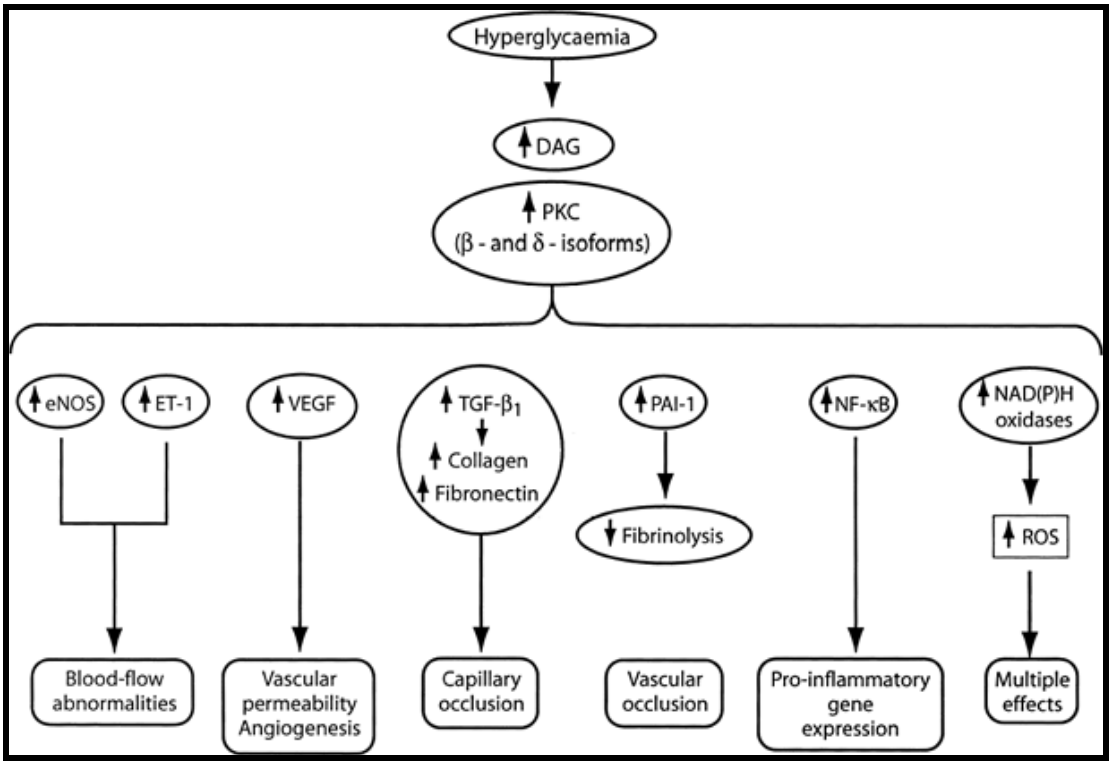
1. Intracellular proteins modified by AGEs have an altered function.
2. Extracellular matrix components modified by AGE precursors interact abnormally with other matrix components and with the receptors for matrix proteins, such as integrins on cells.
3. Plasma proteins modified by AGE precursors bind to AGE receptors on endothelial cells, mesangial cells and macrophages, inducing receptor-mediated production of reactive oxygen species.



Protein kinase C isoforms

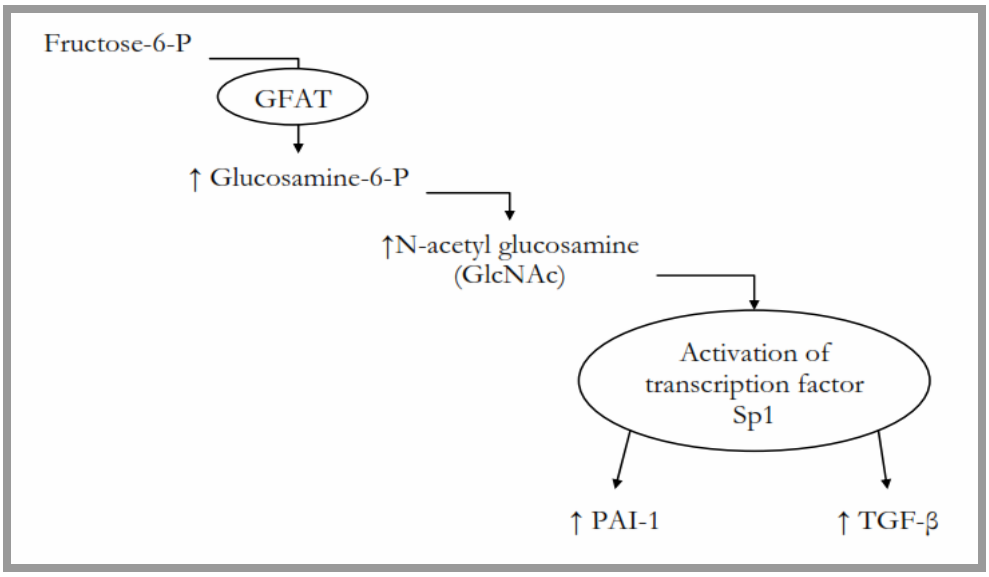
Intracellular increase of glucose augments the synthesis of a molecule called diacylglycerol (DAG), which is a critical activating cofactor for the classic iso forms of protein kinase C (PKC), ß, d and a. PKC has an effect on expression of a variety of genes.

Its over activation leads to blood flow abnormalities, vascular permeability, capillary and vascular occlusion, pro-inflammatory gene expression and oxidative stress.[61,62,63]



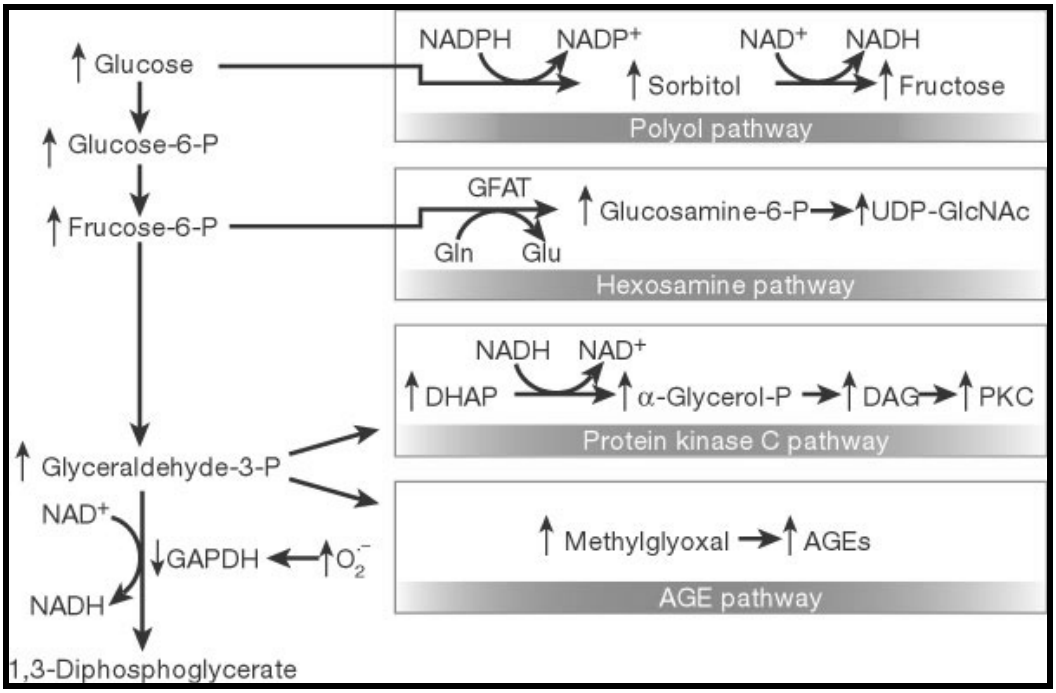
Hexosamine pathway flux

In the hexosamine pathway, fructose-6-phosphate is diverted from glycolysis leading to an increase in uridine diphosphate-*N*-acetylglucosamine. Overmodification by this glucosamine of serine and threonine residues leads to pathological changes in gene expression and protein function. Although a role for this relatively newly identified pathway in diabetic nephropathy is evident the exact pathogenic mechanisms still need to be established.[64,65]



Brownlee[66] hypothesized that these four pathways can be linked together by a common initiating factor: superoxide formation by the mitochondria. Increased hyperglycemia-derived electron donors from the tricarboxylic acid cycle (also known as the citric acid cycle), NADH and $FADH_2$, generate a high mitochondrial membrane potential, by pumping protons across the mitochondrial inner membrane. This inhibits electron transport, increasing the half-life of free radical intermediates of co-enzyme O, which reduce O_2 to superoxide. Hyperglycemic induced superoxide formation by

the mitochondria decreases GAPDH which converts glyceraldehyde-3-P into 1,3-diphosphoglycerate in the tricarboxylic acid cycle. As a result glyceraldehyde-3-P,which is a precursor for AGE and PKC, increases. This initiates AGE formation and PKC activation. Due to the reduced conversion by GAPDH, the molecules upstream the tricarboxylic acid cycle will also increase. These are fructose-6-P, activating the hexosamine pathway, and glucose itself, increasing the polyol pathway activity.



Early in the course of diabetes, abnormalities in blood flow and increased vascular permeability occur. In this stage, there is decreased activity of vasodilators, such as nitric oxide and increased activity of vasoconstrictors, such as angiotensin II and endothelin-1 and elaboration of permeability factors such as vascular endothelial growth factor (VEGF). Later, abnormalities in the extracellular matrix contribute to an irreversible increase in vascular permeability. With time, microvascular cell loss occurs, in part as result of apoptosis, and there is progressive capillary occlusion due

to both extracellular matrix overproduction induced by growth factors such as transforming growth factor ß (TGF-ß) and deposition of hyaline material. Together, these changes lead to oedema, high blood pressure in the glomerulus and ischemia, finally leading to glomerulosclerosis.

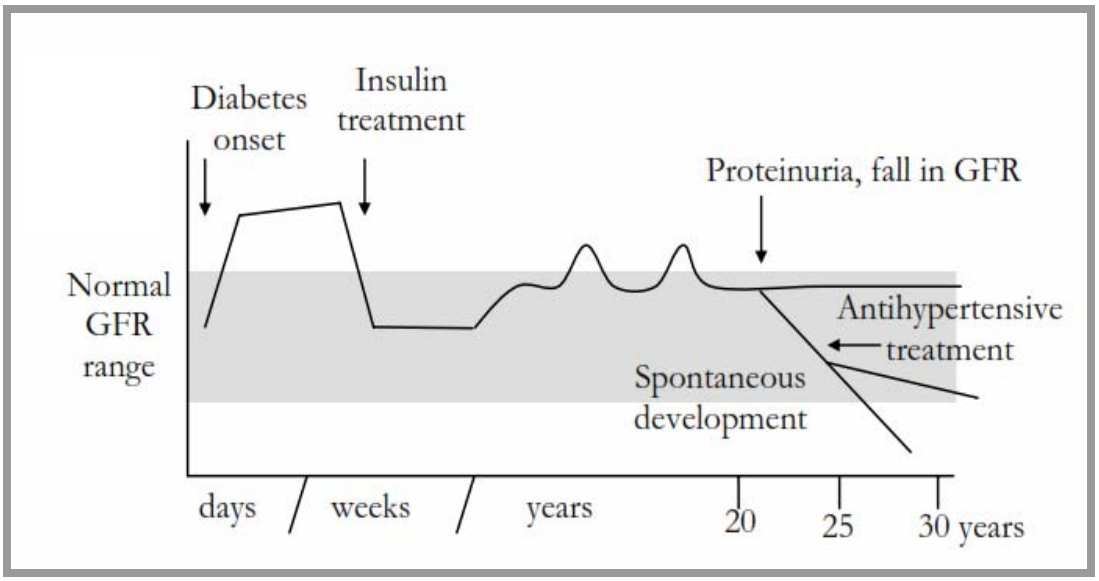
Haemodynamics of the kidneys during diabetes

In a hyperglycaemic environment the kidneys are affected in several ways.Reabsorption of glucose from the urine is normally very effective, but during hyperglycaemia all the glucose can not be absorbed and is instead secreted. Water is also eliminated due to osmotic effects, resulting in large urine volumes and dehydration.

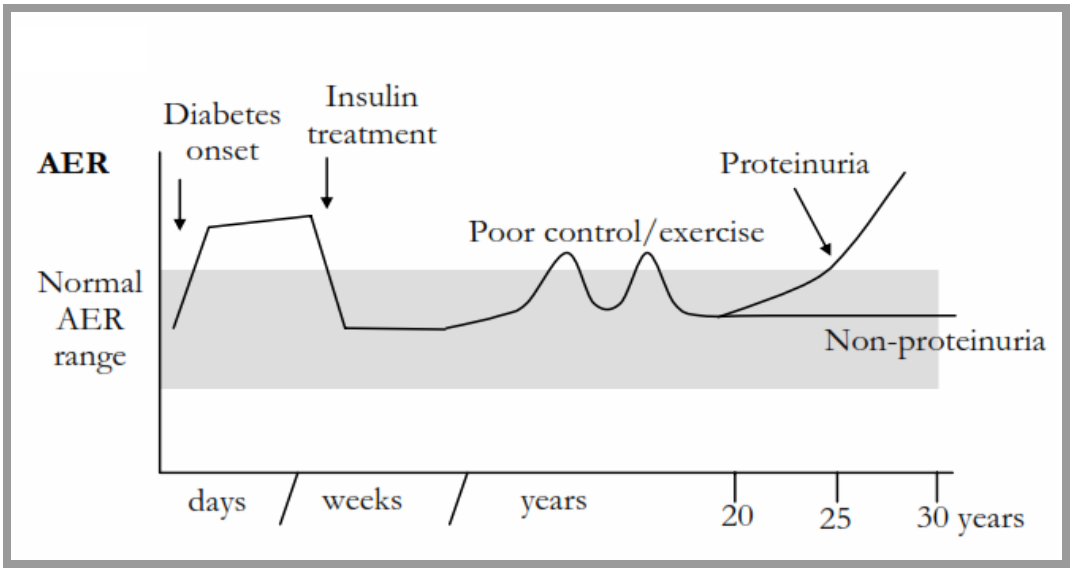
It has been shown that patients with elevated GFR are at increased risk of developing DN.[67,68] During the years with diabetes, the haemodynamics of the kidneys and the GFR mostly depends on metabolic control and blood pressure. Before insulin treatment is started, the GFR is increased, but it is normalized with the initiation of insulin treatment.[69] However, the GFR remains slightly elevated in at least 25% of the patients, even if it is within normal range, it is likely that these patients have a higher risk of developing DN later on.[70,71] The GFR can be temporarily increased during periods of poor metabolic control but this can be reversed by effective insulin treatment.[72,73] Increased blood pressure also increases the GFR and if antihypertensive medication is used, the rate of decline in kidney function can be markedly reduced.[74,75,76,77] The albumin excretion rate (AER) is at first affected the same way as the GFR. The AER is increased at the onset of diabetes, but when insulin treatment normalizes the GFR, also the AER is normalized (fig. 1b).

Albumin leakage into the urine is a well-known early marker for the development of DN and also the first clinical sign of kidney damage.[67,78]

If DN progresses, GFR slowly decreases due to the reduced filtration surface while albumin leakage increases. At first there will be relatively small amounts of urinary albumin, microalbuminuria (also called incipient nephropathy). As the kidney damage progresses the amount of albumin increases and the patient develops macroalbuminuria (overt nephropathy). Albuminuria is also a marker for other complications such as proliferative retinopathy and cardiovascular complications.[79] With decreased GFR, hypertension will develop, which in turn accelerates the process.[80] During the development of DN there are morphological changes in the glomeruli, thickening of the glomerular basement membrane, expansion of the mesangium and accumulation of extracellular matrix (ECM). The leakage of albumin per se accelerates the progression of kidney disease, probably because albumin accumulates in the mesangium and the extracellular structures in the glomeruli, which may stimulate cell proliferation and accumulation of ECM.[81] There is also loss of podocytes due either to apoptosis or cell detachment, or both which leads to increased permeability for albumin molecules.[82] Later on there is sclerosis of the glomeruli which reduces the filtration surface and the reduced capability of some glomeruli to filtrate the blood increases the workload for the others, damaging also the remaining glomeruli. [83,84]



GFR changes with duration of diabetes.



AER changes with duration of diabetes[85]

Dyslipidemia [40]

The prevalence of hyperlipidemia in chronic renal disease is higher than in the general population, but varies depending on the type of lipid, target population, cause of renal disease and level of renal function.[9,40,86]

In the general population, hyper-cholesterolaemia and dyslipidaemia are inter-changeable in terms of prevalence and risk implication. In ESRD, however, this is not the case. ESRD patients typically have either normal or slightly increased low-density lipoprotein (LDL), increased very low-density lipoprotein (VLDL) and intermediate-density lipoprotein (IDL) leading to elevated triglyceride levels, and decreased levels of high-density lipoprotein (HDL). There are also qualitative changes in dyslipidaemia with a shift to a more atherogenic LDL particle size toward a small, dense apo-B-rich LDL Predominance.[87] The prevalence of dyslipidaemia in chronic kidney disease patients is very high. In the general population, the relationship between hyperlipidaemia (dyslipidaemia) and cardiovascular disease (predominantly coronary artery disease) is well established and there are proven benefits of lipid-lowering with statins.[88,89,90,91,92,93] Only limited epidemiological and even more limited interventional, data exist on the relationship between dyslipidaemia and cardiovascular disease in ESRD. In patients receiving maintenance haemodialysis, reports suggest either no relationship or paradoxical correlations, the so called "reverse epidemiology", where a lower total cholesterol level has been associated with a higher risk of death or conversely, a higher serum cholesterol level has been found in long-term dialysis survivors.[94,95,96,97]

Hyper-cholesteremia in chronic uremia is confounded by the impact of malnutrition, which lowers serum cholesterol. Chronic uremia is associated with higher lipoprotein (a) level, chylomicron remnants, and altered lipoprotein particles, which are potentially atherogenic.

A longitudinal study has demonstrated that a high lipoprotein (a) level is a risk factor for further cardiac events.[40]

Echocardiography

Introduction[98]

The term echocardiogram refers to the evaluation of cardiac structure and function with images and recordings produced by ultrasound. In the past three decades it has rapidly become a fundamental component of the cardiac evaluation.

Currently, echocardiography ("echo") provides essential (and sometimes unexpected) clinical information and has become the second most frequently performed diagnostic procedure.

What began as a one-dimensional (ID) method performed from the precordial area to assess cardiac anatomy has evolved into a two-dimensional (2D) modality performed from either the thorax or from within the esophagus capable of also delineating flow and deriving hemodynamic data.

Newly evolving technical developments likely will extend the capacity of ultrasound to routine 3D visualization as well as to the assessment, in conjunction with contrast agent, of myocardial perfusion.

History[98]

The development of echocardiography is usually credited to Elder and Hertz in 1954.Primitive cross-sectional images of the excised human heart were produced in 1957; however, for nearly two additional decades, clinical echocardiography consisted primarily of 10 time-motion (M-mode) recordings, as popularized by Feigenbaum.

In the mid-1970s, Bom and associates developed a multi element linear- array scanner that could produce spatially correct images of the beating heart.

2D images of superior quality were soon achieved by mechanical sector scanners and ultimately by phased-array instruments as developed by Thurston and Von Ramm, which are the present-day standard.[99]

In the past several years, 3D instruments capable of real-time volumetric imaging have been developed. Miniaturization of ultrasound transducers has also led to their incorporation into gastro scopes and cardiac catheters to achieve trans-esophageal and intravascular images.

Although efforts to use the Doppler principle to measure flow velocity by ultrasound were begun in the early 1970s by Baker et al, clinical application of this technique did not thrive until the work of Hatle in the early 1980s.

Pulsed and continuous-wave Doppler recordings soon were expanded to full 2D color-flow imaging.

Most recently, Doppler velocity recordings have been obtained from myocardium itself enabling measurement of tissue velocities and regional strain.

Left Ventricular Hypertrophy

Left Ventricular Hypertrophy (LVH) plays a central role in chronic adaptation to pressure or volume overload of the systemic circulation.[100,101] The degree of hypertrophy parallels the severity of overload and detection of extreme hypertrophy may indicate a poor prognosis. Thus, logically, serial determination of left ventricular muscle mass (LVM) should be an essential element in the study of such disorders.

However, assessment of LVH in man has been limited by the lack of an accurate, well-validated, widely applicable and readily repeatable method for quantitating LVM.

The biplane angiographic method of Rackley et al. is accurate by comparison with autopsy LVM, but has seen limited use because of its technical complexity and invasive methodology. The non-invasive basis and wide applicability of echocardiography make it an appealing method for the systematic serial evaluation of LVH.

Several studies have indicated a close statistical relationship between echocardiographic and angiographic estimates of LVM. However, the reliability of three-dimensional data derivations from M-mode echocardiography has recently been regarded with considerable skepticism.

Moreover, the critical comparison between echocardiographic LVM and true anatomic LVM has not yet been made. Finally, existing Echocardiographic studies have each evaluated a single method for LVM.

None has systematically assessed the individual Echocardiographic variables which determine the accuracy of such an estimate.[100]

Left Ventricular Mass (LVM) is determined by the equation developed by Devereux et al [2, 3, 19]

LVM = 0.8 {1.04 [(LVIDd + IVSd + PWd) 3 - (LVIDd)3]} + 0.6 g

Where in: LVIDd – internal diameter of the left ventricle at end diastole

IVSDd – thickness of the interventricular septum

PWd - thickness of the posterior wall in the end diastole

Left Ventricular Mass Index (LVMI) calculated by:

$$\textbf{LVMI} = \frac{\textbf{Left Ventricular Mass}}{\textbf{Body surface area}}$$

Where in:

Body surface area calculated by using Dubois

$$BSA = 0.007184 \times W^{0.425} \times H^{0.725}$$

Where in: W – Weight in Kilograms and

H – Height in Centimeters

Diastolic dysfunction

Diastolic dysfunction refers to abnormal mechanical properties of the Left Ventricle (LV) and is defined as an abnormality of distensibility, filling or relaxation of the LV during diastole.[102] Due to impaired capacity of the LV to accept blood, left atrial pressure increases. Abnormalities in diastolic function can occur in the presence or absence of a clinical syndrome of heart failure and with normal or abnormal systolic function.[103] Diastolic heart failure is defined as a clinical syndrome characterized by the symptoms and signs of heart failure, a preserved ejection fraction and abnormal diastolic function.[103]

The prevalence of diastolic dysfunction increases with age, with an approximate incidence of 15 % to 25 % in patients <60 years of age, 35 % to 40 % in those between 60 and 70 years of age, and 50 % in patients >70 years of age.[104,105,106]

Disturbed LV diastolic function has been reported as many as in 70 - 80 % of patients on Haemodialysis (HD) included in cross-sectional studies.[107,108,109,110] It is prevalent in adults and children with mildly to moderately impaired renal function.[111,112,113,114,115] Further, the prevalence of diastolic dysfunction increases with deteriorating renal function.[116,117]

About 80 % of dialysis patients have LVH and a strong correlation between LVH and diastolic dysfunction has been observed in these patients.[118,119]

Diabetes, independently of LVH, is also related to diastolic dysfunction in patients with CRF.[120]

Doppler tissue imaging measures the velocity of the mitral annulus motion during the diastole. Myocardial fibers have a common insertion on the fibrous mitral annulus and the mitral annular motion in the longitudinal axis is determined by the sum of longitudinally oriented fibers.[121]

Thus, the mitral annulus velocity represents the velocity of changes in the LV long axis dimensions. In healthy persons, the mitral annular motion during the diastole is almost a mirror image of the transmitral flow pattern and consists of two negative velocities E` (mitral annular early diastolic velocity) and atrial A (mitral annular late diastolic velocity).[122]

Normal ageing causes a decrease in E` and a substantial increase in A` myocardial velocities.[123,124] An increase in Heart Rate (HR) reduces E`, but increases A`.[125] The main limitation of the method is that the E` is a regional index and errors can occur when results are extra polated to the entire ventricle.

METHODOLOGY

1. Source of Data

Diabetic nephropathy patients of age group 40 to 60 years undergoing hemodialysis admitted or who visited on out patient basis during the study period from December 2012 to September 2014.

2. Method of Collection of Data

The data for this study was collected from fifty patients fulfilling the inclusion /exclusion criteria admitted in R.L.Jalappa Hospital and patients on Dialysis on OPD Basis during the study period from December 2012 To September 2014, using a proforma specially designed for the study.

Study Design Descriptive Study

Sample Size 50 cases

Study Duration December 2012 to September 2014.

Inclusion Criteria:

The study population consists of 40 to 60 years age group of diabetic nephropathy patients with mild, moderate and severe Chronic Renal Failure attending the hospital and patients on dialysis. Where in [1,2]

(i) Mild Chronic Renal Failure - includes patients with Serum Creatinine 1.5-3 mg/dl

(ii) Moderate Chronic Renal Failure –includes patients with Serum Creatinine values 3.0-6.0 mg/dl and

(iii) Severe Chronic Renal Failure - includes patients with Serum Creatinine value > 6 mg/dl.

Exclusion Criteria:

1. Patients with polycystic kidney disease.
2. Patients with chronic kidney disease of other/undetermined etiology.
3. Patients with chronic heart diseases like cardiomyopathy, ischaemic heart diseases, congenital heart diseases, rheumatic heart diseases.
4. Patients with poor Echo window

Methods of Sample Collection

The following set of investigations was asked for in the patients included in the study.

1. Complete Haemogram
2. Renal function tests
3. Random Blood Sugar, Glycated Haemoglobin
4. Liver function test
5. Urine analysis & culture
6. Renal ultrasound
7. Lipid profile
8. Serum electrolytes, Serum Calcium, Serum Phosphorous
9. Chest skiagram
10. Electrocardiography-12 lead
11. Fundus Examination
12. 2D Echocardiography

All patients under went 2 dimensional directed M- Mode Echocardiography performed in left lateral position.

The following measurements were taken in to account by using the Penn convention methods[1,2,3]

- Thickness of Interventricular septum (IVSd)
- Thickness of Posterior wall in end diastole (PWd)
- Internal diameter of Left ventricle at end diastole (LVIDd)
- Mitral Inflow Peak Early Diastolic Velocity (E)
- Mitral Inflow Late Diastolic Velocity (A)
- Early to Late Peak Mitral Inflow Velocity $\frac{E}{A}$

Left ventricular mass (LVM) and Left ventricular mass index (LVMI) - were calculated by using ECHO CUBE Formula recommended by American society of Echocardiography[1,2,3]

Left Ventricular Mass (LVM) = 0.8 {[1.04x (LVIDd+IVSd+PWd)3 -LVIDd3]}+0.6g

Left Ventricular Mass Index (LVMI) = $\frac{\textbf{LVM}}{\textbf{Body surface area}}$

Body surface area calculated by Dubois formula

BSA = 0.007184 x $W^{0.425}$ x $H^{0.725}$

W– Weight in kilograms (Kgs)

H – Height in Centimeters (Cms)

Left Ventricular Hypertrophy is defined in absolute terms as: [1,2,3]

- LVMI – More than 131 g/m2 in men
- LVMI – More than 100 g/m2 in women

Creatinine Clearance **(CrCl)** is calculated according to the formula derived from Cockcroft-Gault

Cockcroft-Gault equation:

CrCl = $\frac{\{140 - \text{Age(yrs)}\} \times \text{Weights (Kgs)}}{\text{Plasma Creatinine} \times 72}$ For Males

CrCl = $\frac{\{140 - \text{Age(yrs)}\} \times \text{Weights (Kgs)}}{\text{Plasma Creatinine} \times 72 \times 0.85}$ For Females

Normal values of Creatinine :

In Men - 90- 139ml/min

In women - 80-135ml/min

Diastolic Dysfunction : $\frac{\mathbf{E}}{\mathbf{A}} > \mathbf{1}$

Where in : E - Mitral Inflow Peak Early Diastolic Velocity.

A - Mitral Inflow Late Diastolic Velocity.

$\frac{E}{A}$ - Early to Late Peak Mitral Inflow Velocity.

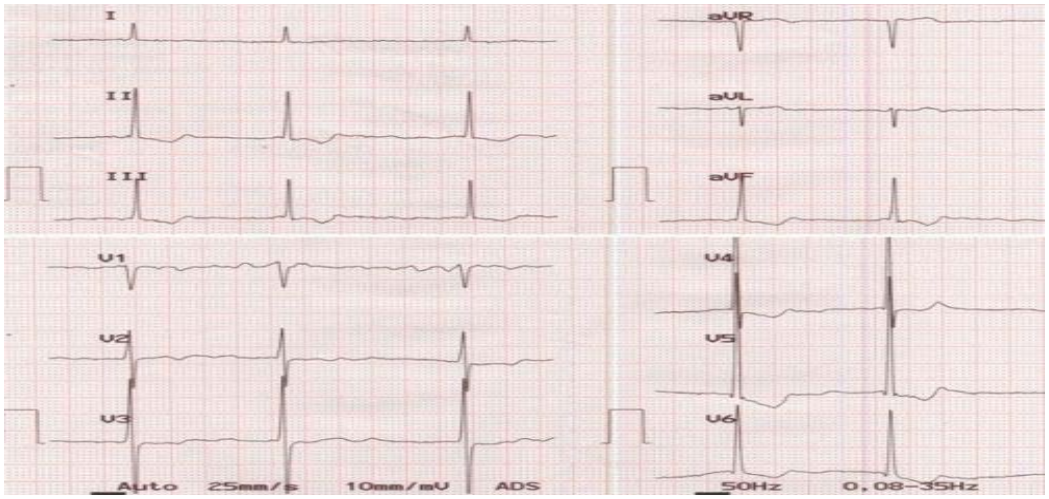


Fig 1 : 12 LEAD ELECTROCARDIOGRAM SHOWING LEFT VENTRICULAR HYPERTROPHY WITH STRAIN

(Name of the Patient : Shankarappa)

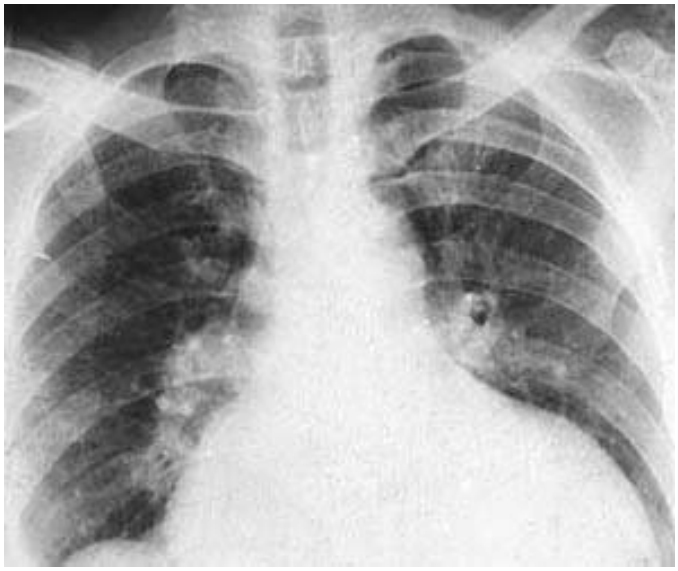


Fig 2. CHEST X RAY SHOWING CARDIOMEGALY (LV APEX)

(Name of the Patient : SaiKrishnana)

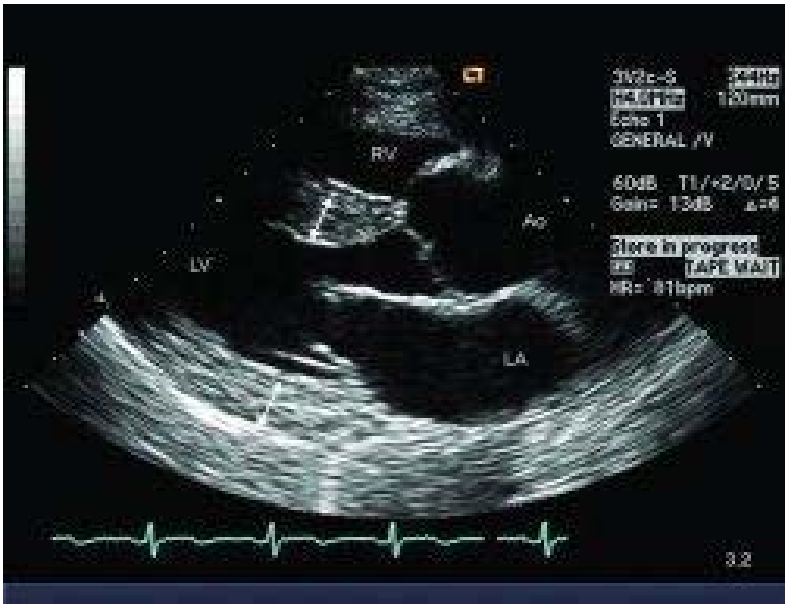


Fig 3a. Parasternal long axis view on transthoracic echocardiogram shows concentric left ventricular hypertrophy

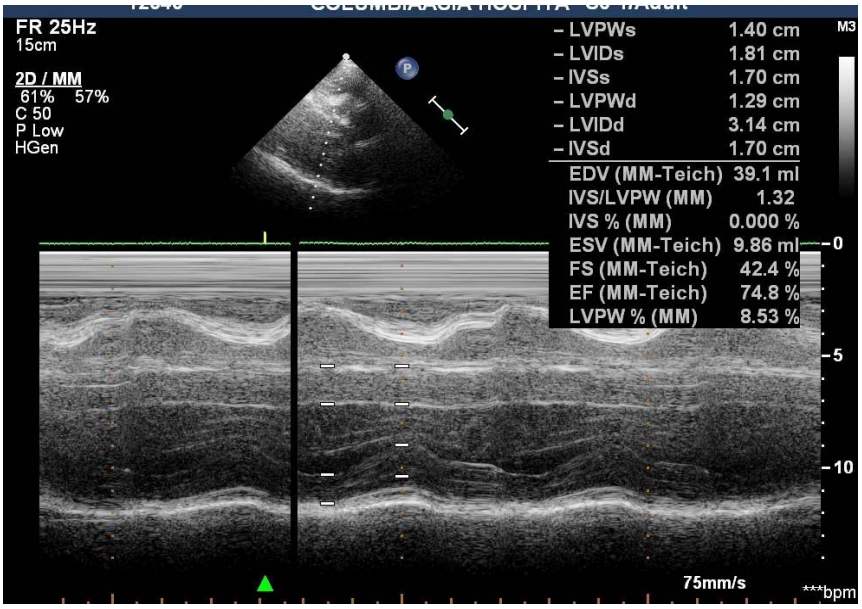


Fig 3b. Echocardiography showing the measurement of Gradients in Left Ventricular Hypertrophy

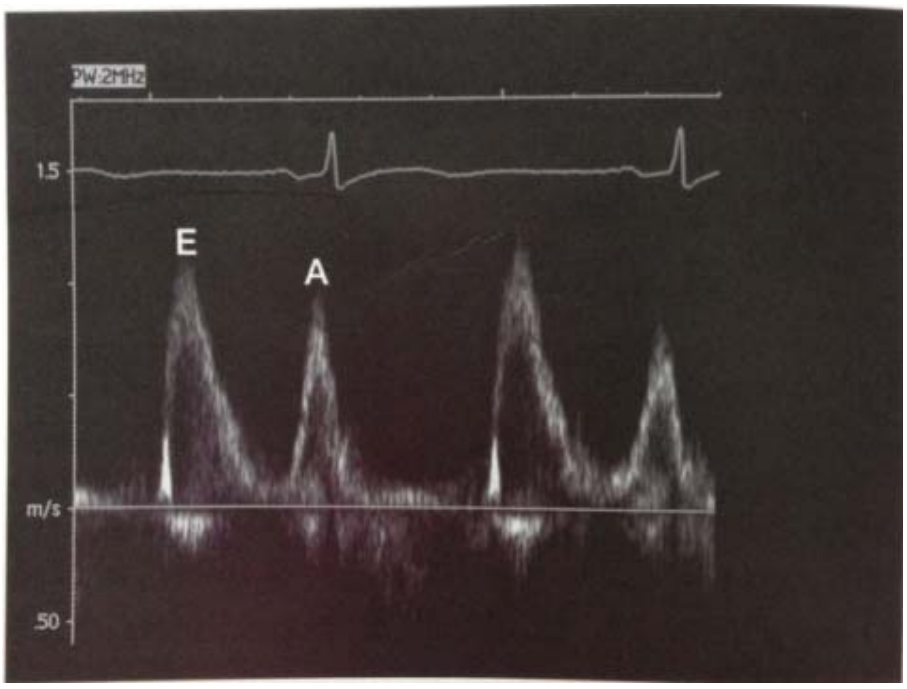


Fig 4. Echocardiography showing Diastolic Dysfunction

RESULTS

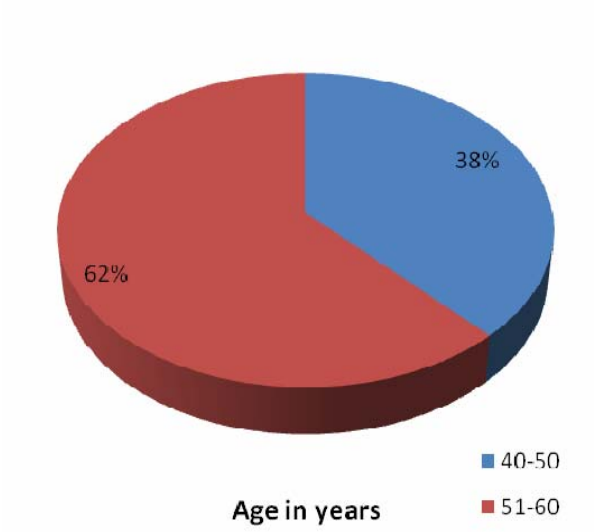
The present study comprised 50 cases of diabetic nephropathy undergoing hemodialysis admitted to R.L.Jalappa Hospital, Kolar, during the study period from December 2012 To September 2014.

1. Age Distribution:

Table – 1: Showing age distribution of 50 cases of chronic renal failure

Age in years	No. of patients	%
40-50	19	**38.0**
51-60	31	**62.0**
Total	**50**	**100.0**

Graph – 1: Age distribution



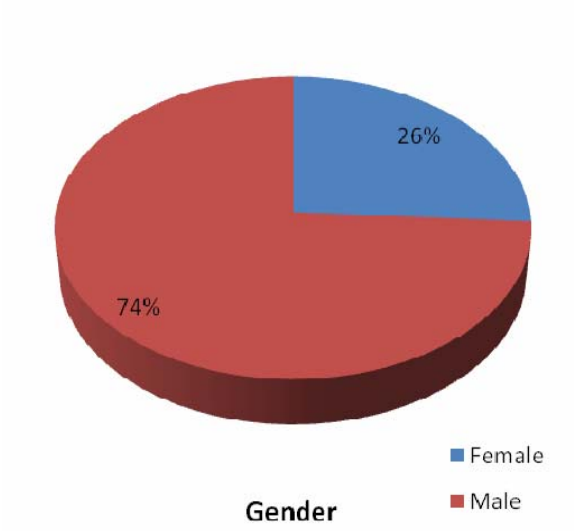
As shown in the Table 1 and Graph 1, in the present study the Age variation was from 41 to 60 years. Majority of patients were in the age group of 51-60 years that included 31 patients (62 %)

2. Gender Distribution :

Table 2: Showing the Gender Distribution

Gender	No. of patients	%
Female	13	**26.0**
Male	37	**74.0**
Total	**50**	**100.0**

Graph 2: Gender distribution



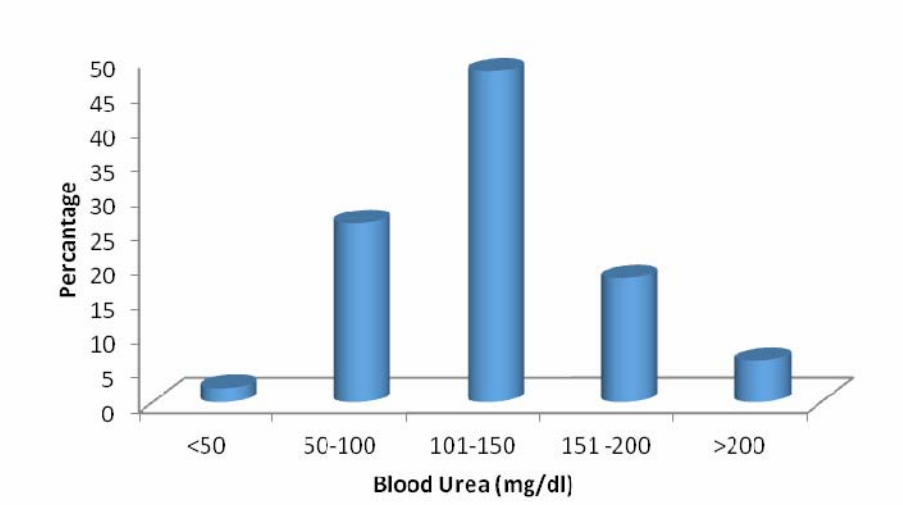
As shown in the table 2 and graph 2, the present study group consisted of 74% males and 26% females.

3. Blood Urea Levels

Table – 3: Showing the distribution of Blood Urea levels

Blood Urea (mg/dl)	No. of patients	%
<50	1	**2.0**
50-100	13	**26.0**
101-150	24	**48.0**
151 -200	9	**18.0**
>200	3	**6.0**
Total	50	**100.0**

Graph 3: Blood urea (mg/dl)



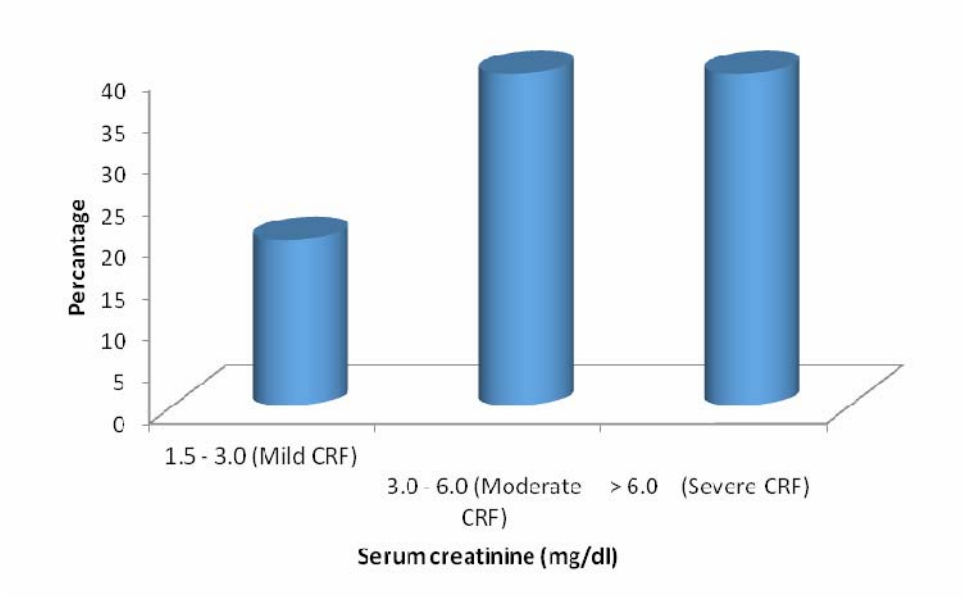
The range of blood urea level in the present study was between 50-222 mg/dl. Maximum number of patients i.e., 24 patients (48%) had blood urea in the range of 101-150 mg/dl, followed by in 13 patients (26%) had blood urea in the range of 50- 100 mg/dl

4. Serum Creatinine levels:

Table – 4: Showing the Distribution of Serum Creatinine

Serum creatinine (mg/dl)	No. of patients	%
1.5 - 3.0 (Mild CRF)	10	**20. 0**
3.0 - 6.0 (Moderate CRF)	20	**40.0**
> 6.0 (Severe CRF)	20	**40.0**
Total	**50**	**100.0**

Graph 4: Serum creatinine (mg/dl)



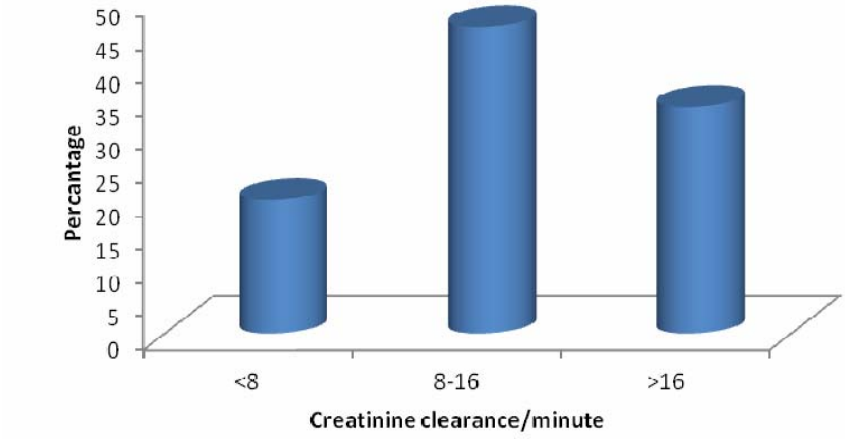
The range of serum Creatinine level in the present study was between 1.5 - 20.8 mg/dl. However, 20 patients (40%) were equally distributed in moderate and severe CRF group (i.e., 40% in each group) and remaining 20% were in the Mild CRF group.

Table 5: Creatinine clearance/minute

Distribution of Creatinine clearance (ml) / minute

Creatinine clearance/minute (ml/min)	No. of patients	%
<8	10	**20.0**
8-16	23	**46.0**
>16	17	**34.0**
Total	**50**	**100.0**

Graph 5: Creatinine clearance/minute



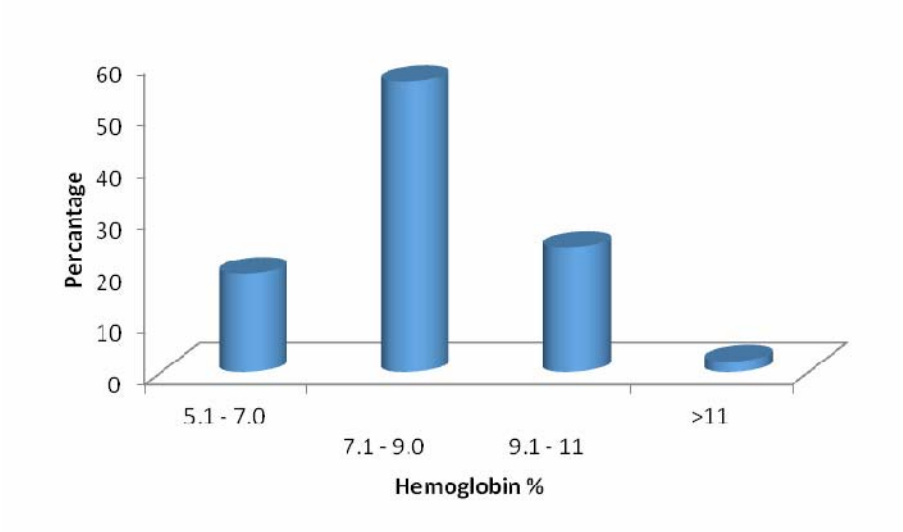
In the present study of 50 patients, 23 patients (46%) had creatinine clearance of 8 – 16 ml/min followed by 17 patients (34%) having creatinine clearance of >16 ml/min.

Table 6: Hemoglobin %

Distribution of levels of hemoglobin

Hemoglobin %	No. of patients	%
5.1 - 7.0	9	**18.0**
7.1 - 9.0	28	**56.0**
9.1 - 11	12	**24.0**
>11	**1**	**2.0**
Total	**50**	**100.0**

Graph 6: Hemoglobin %



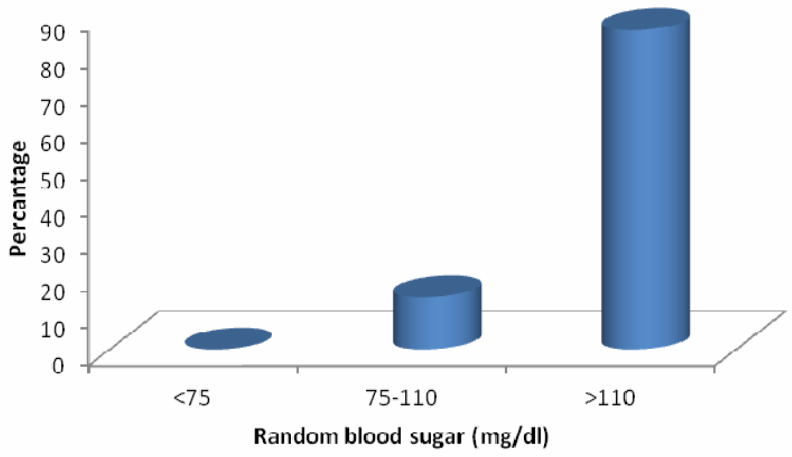
In the present study majority of the patients 28 patients (56%) had hemoglobin levels between 7.1-9 gm%, followed by 12 patients (24%) in between 9.1-11 gm%.

Table 7: Random blood sugar (mg/dl)

Distribution of levels of Random Blood Sugar

Random blood sugar (mg/dl)	No. of patients	%
<75	0	**0.0**
75-110	7	**14.0**
>110	43	**86.0**
Total	**50**	**100.0**

Graph 7: Random blood sugar (mg/dl)



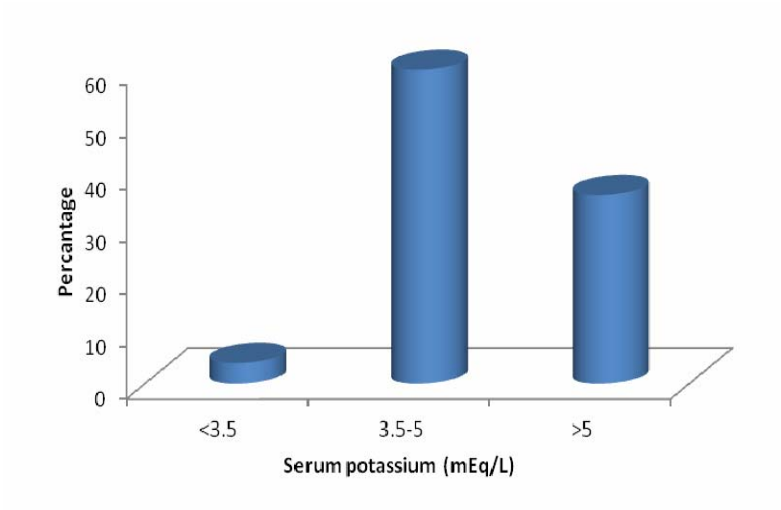
In the present study of 50 patients, 43 (86%) patients had RBS levels of > 110 mg/dl indicating the role of poorly controlled diabetes in the disease process of CRF.

Table 8: Serum electrolytes

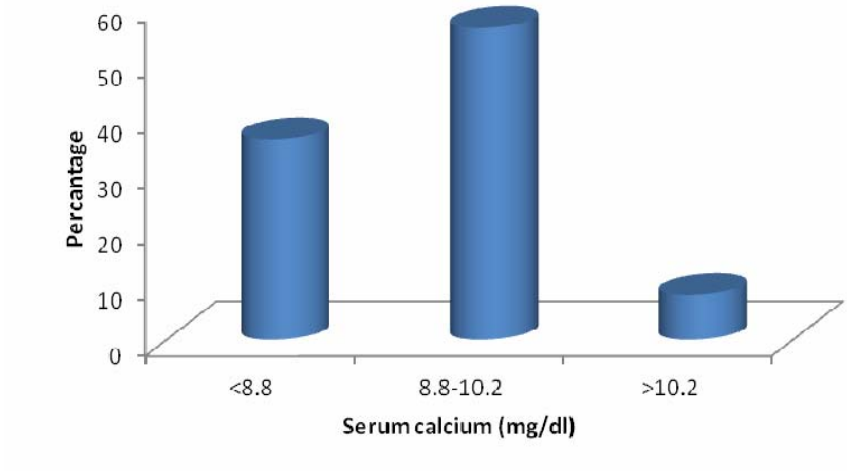
Distribution of Serum Electrolytes

	No. of patients (n=50)	**%**
Serum potassium (mEq/L)		
• **<3.5**	2	4.0
• **3.5-5**	30	60.0
• **>5**	18	36.0
Serum calcium (mg/dl)		
• **<8.8**	18	36.0
• **8.8-10.2**	28	56.0
• **>10.2**	4	8.0
Serum phosphorous (mEq/L)		
• **<2.5**	0	0.0
• **2.5-4.8**	27	54.0
• **>4.8**	23	46.0

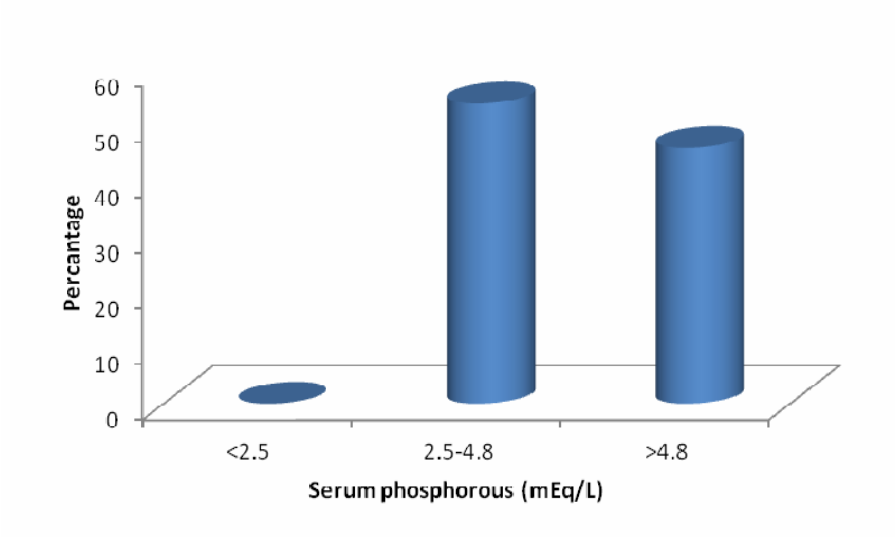
Graph 8: Serum electrolytes



In the present study, the range of Serum Potassium was 3.1 - 6.4 mEq/L and in this, 30 patients (60 %) had Serum Potassium level between 3.5 - 5 mEq/L.



In the present study, the range of serum calcium levels was from 6.9 - 11.2 mg/dl. In 28 patients (56%) serum calcium levels were between 8.8 - 10.2 mg/dl



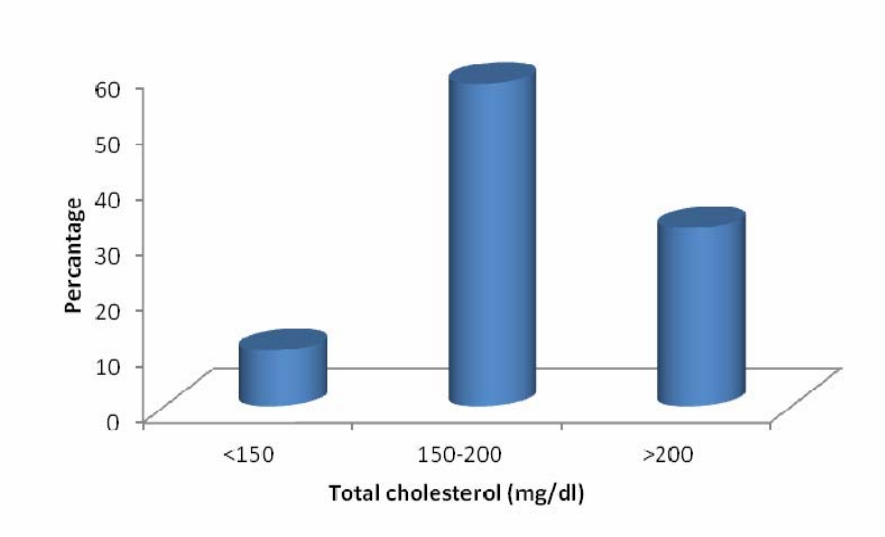
In the present study, the range of serum phosphorus was between 3.1 - 6.4 mg/dl. In 27 patients (54%) the serum phosphorus levels was between 2.5 – 4.8 mg/dl.

Table 9: Lipid parameters of patients studied

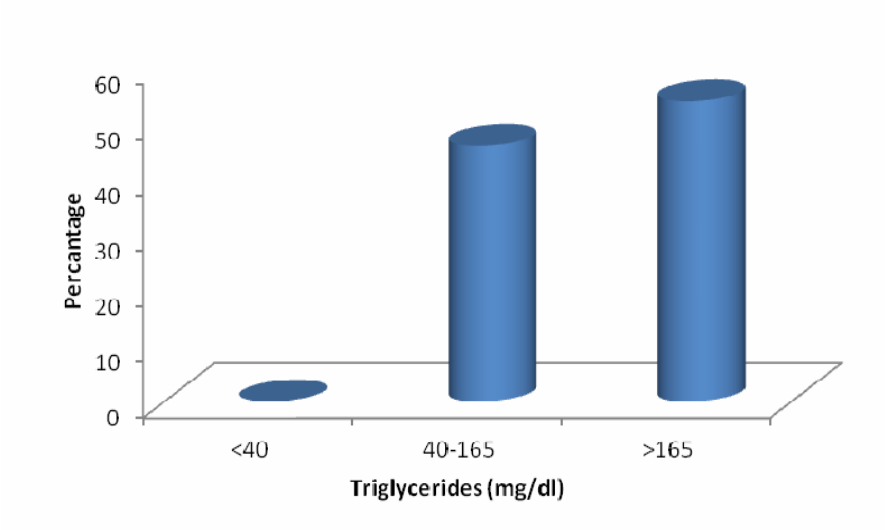
Distribution of Lipids

	No. of patients (n=50)	**%**
Total cholesterol (mg/dl)		
• **<150**	5	10.0
• **150-200**	29	58.0
• **>200**	16	32.0
Triglycerides (mg/dl)		
• **<40**	0	0.0
• **40-165**	23	46.0
• **>165**	27	54.0
Low density cholesterol (mg/dl)		
• **<60**	0	0.0
• **60-150**	50	100.0
• **>150**	0	0.0
High density cholesterol (mg/dl)		
• **<30**	10	20.0
• **30-60**	40	80.0
• **>60**	0	0.0

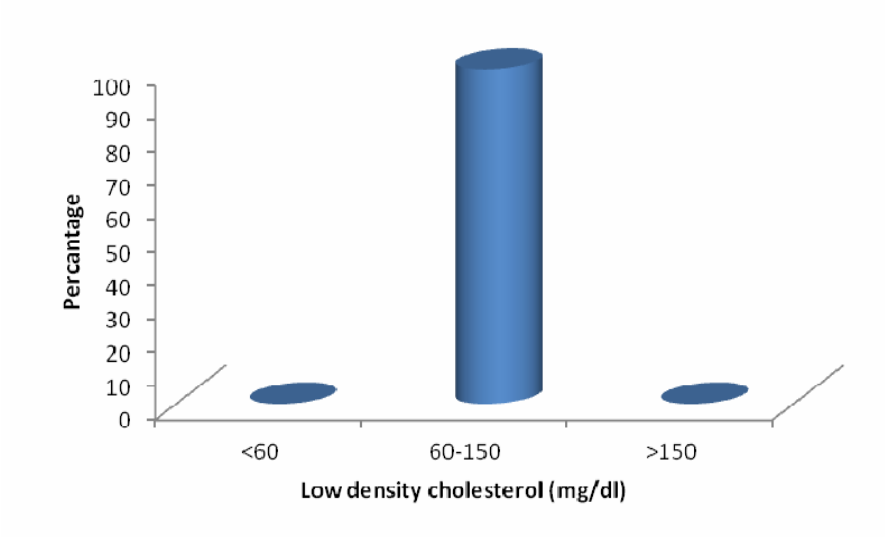
Graph 9: Lipid parameters of patients studied



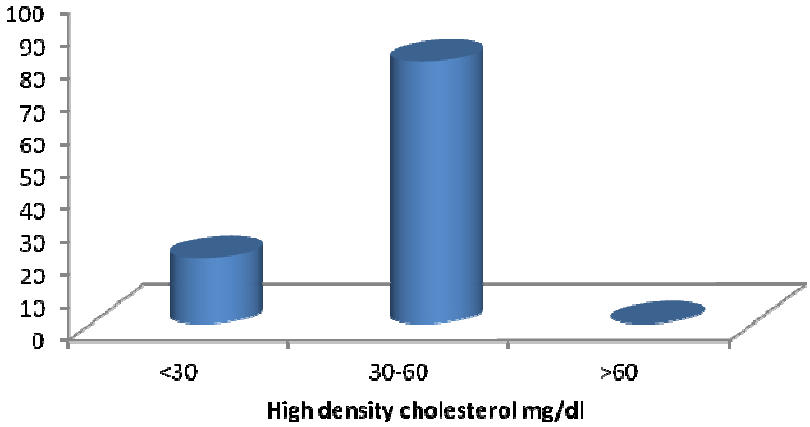
In the present study, the range of total cholesterol was between 146 - 313 m/dl. Total cholesterol between 151-200 mg/dl was observed in 29 patients (58%) and >200 m/dl was observed in 16 patients (32%).



The range of triglycerides levels in the present study was between 142 - 210mg/dl, of which 27 patients (54%) had triglycerides >165 mg/dl.



In the present study, the range of LDL cholesterol levels was between 98-144 mg/dl and all the 50 patients (100%) had LDL cholesterol levels between 60-150 mg/dl.



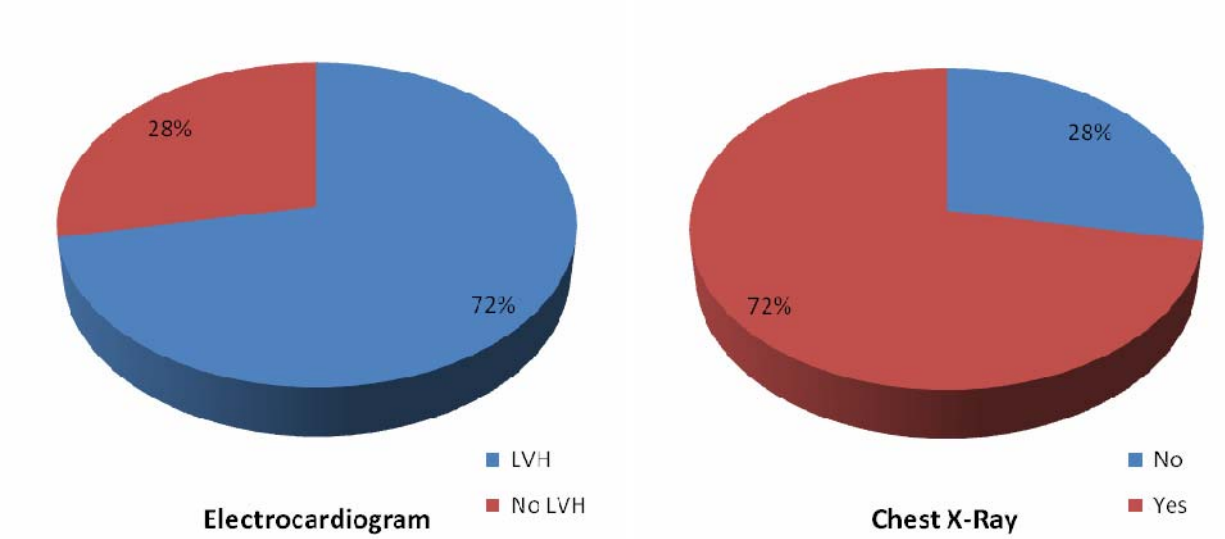
The range of HDL cholesterol in the Present study was 24-48 mg/dl. The HDL cholesterol level of 30 - 60 mg/dl was observed in 40 patients (80%)

Table 10: Radiological assessment

Electrocardiographic and Chest X-Ray Changes

	No. of patients (n=50)	%
Electrocardiogram		
• **LVH**	36	72.0
• **No LVH**	14	28.0
Chest X-Ray		
• **No**	14	28.0
• **Yes**	36	72.0

Graph 10: Radiological assessment



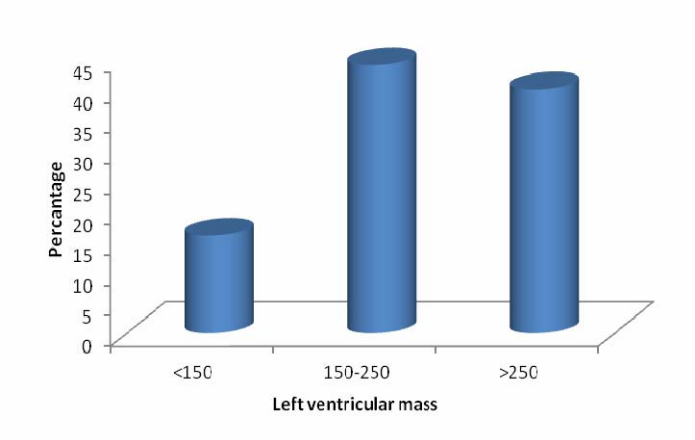
In the present study, Left ventricular hypertrophic changes were equally seen in 36 patients (72%) in Chest X-Ray and Electrocardiogram.

Table 11: Left ventricular mass/ Left ventricular mass index

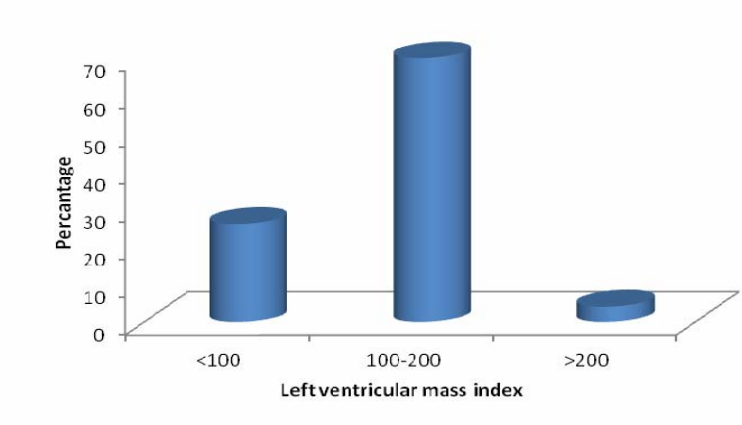
Distribution of Left Ventricular Mass and Left Ventricular Mass Index

	No. of patients (n=50)	**%**
Left ventricular mass		
• **<150**	8	16.0
• **150-250**	22	44.0
• **>250**	20	40.0
Left ventricular mass index (g/m^2)		
• **<100**	13	26.0
• **100-200**	35	70.0
• **>200**	2	4.0

Graph 11: Left ventricular mass/ Left ventricular mass index



In the present study of 50 patients, 22 patients (44%) had Left Ventricular Mass of 150 - 250 folllowed by 20 patients (40%) had >250.



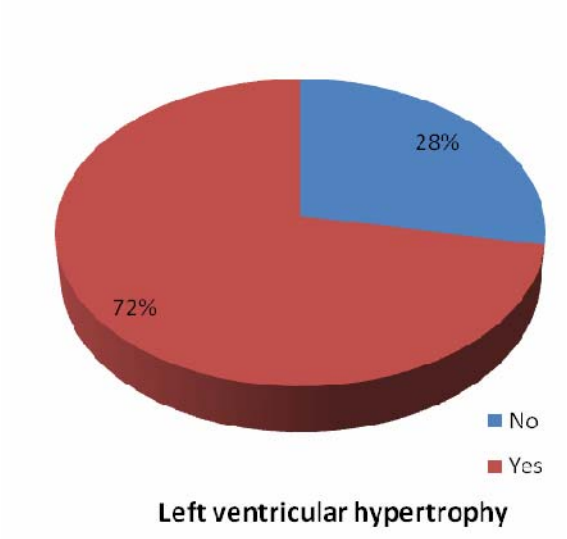
In the present study, majority of the patients- 70% (35 patients) had Left Ventricular Mass Index of 100 – 200 g/m^2

Table 12: Left ventricular hypertrophy on Echocardiogram

Echocardiogram Changes

Left ventricular hypertrophy on Echo	No. of patients	%
No	14	**28.0**
Yes	36	**72.0**
Total	**50**	**100.0**

Graph 12: Left ventricular hypertrophy



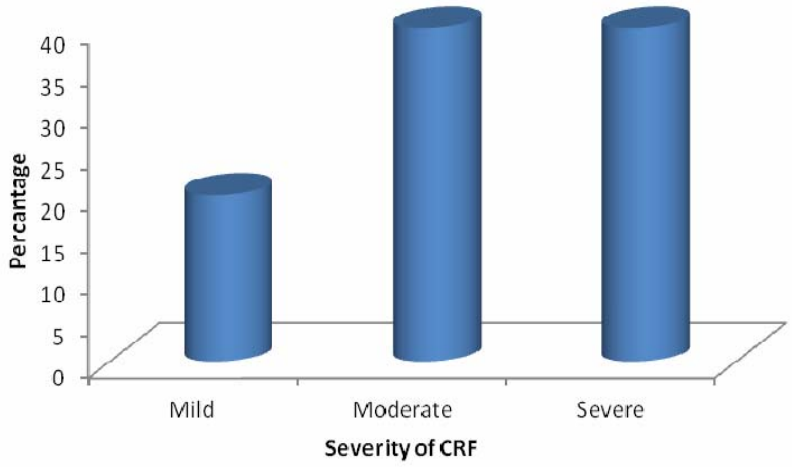
In the present study, Echo changes of Left Ventricular Hypertrophy were seen in 36 patients (72%).

Table 13: Severity of CRF

Distribution of Severity of CRF

Severity of CRF	No. of patients	%
Mild	10	**20.0**
Moderate	20	**40.0**
Severe	20	**40.0**
Total	**50**	**100.0**

Graph 13: Severity of CRF



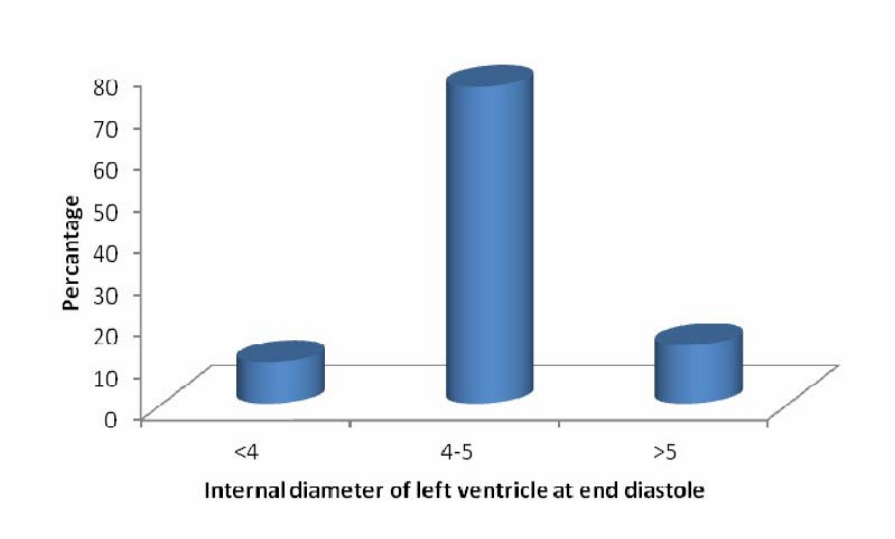
In the present study of 50 patients, equal number of patients are seen in Moderate and Severe categories of CRF – 20 patients (40%) followed by mild CRF- 10 patients (20%).

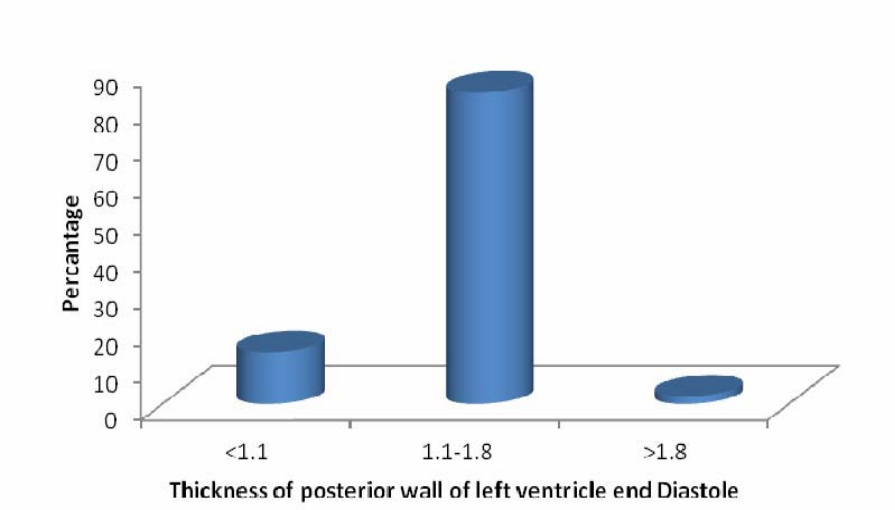
Table 14: Cardiac index parameters

Distribution of Cardiac Index parameters

	No. of patients (n=50)	**%**
Internal diameter of left ventricle at end diastole		
• **<4**	5	10.0
• **4-5**	38	76.0
• **>5**	7	14.0
Thickness of posterior wall of left ventricle end Diastole		
• **<1.1**	7	14.0
• **1.1-1.8**	42	84.0
• **>1.8**	1	2.0
Thickness of interventricular septum		
• **<1.1**	8	16.0
• **1.1-1.8**	41	82.0
• **>1.8**	1	2.0

Graph 14: Cardiac index parameters





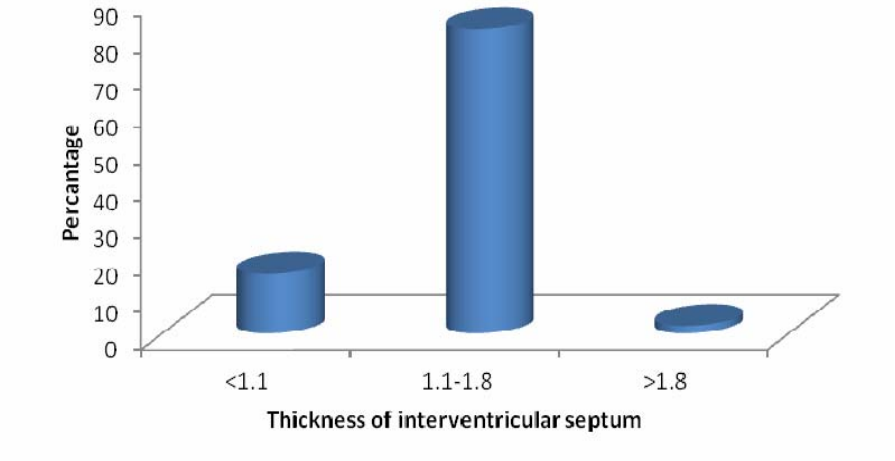
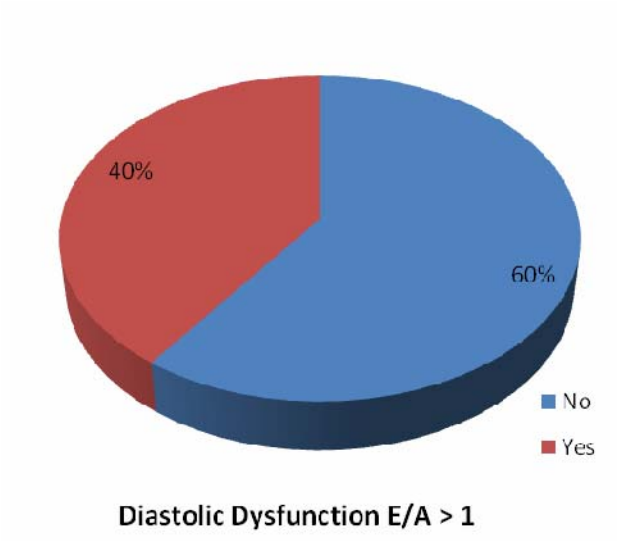


Table 15: Diastolic Dysfunction E/A > 1

Distribution of Diastolic Dysfunction

Diastolic Dysfunction E/A > 1	No. of patients	%
No	30	**60.0**
Yes	20	**40.0**
Total	**50**	**100.0**

Graph 15: Diastolic Dysfunction E/A > 1



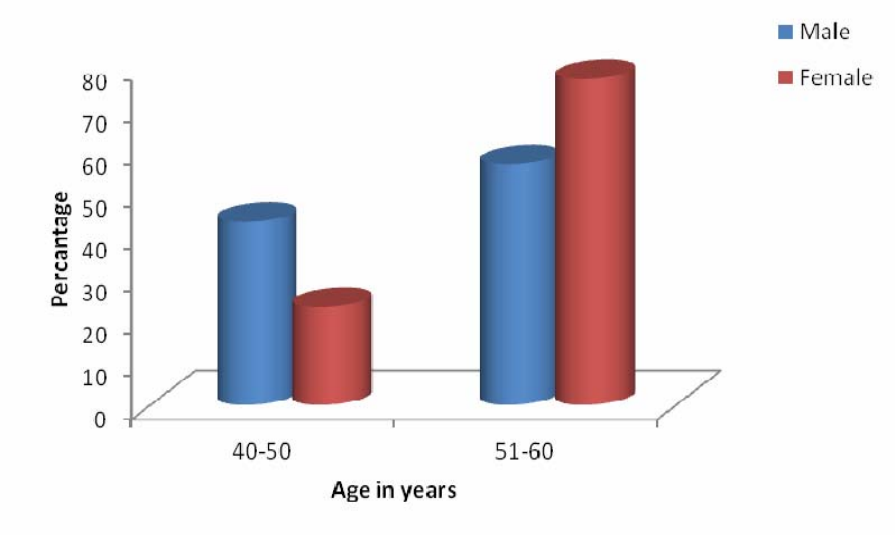
In the present study, Diastolic Dysfunction E/A>1 (Early to Late Peak Mitral Inflow Velocity) was seen in 30 patients (60%).

Table 16: Age distribution of patients studied according to gender

Analysis of patients studied according to gender

Age in years	Gender		Total
	Male	Female	
40-50	16(43.2%)	3(23.1%)	**19(38%)**
51-60	21(56.8%)	10(76.9%)	**31(62%)**
Total	**37(100%)**	**13(100%)**	**50(100%)**

Graph 16: Age distribution of patients studied according to gender



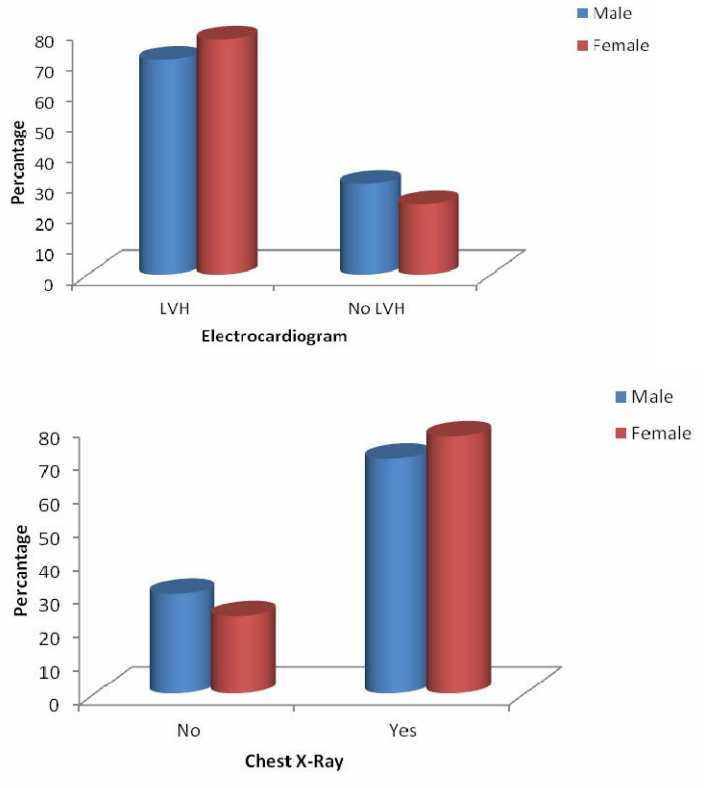
As shown in Table 16 and Graph 16, of the total 50 patients, 19 patients (38%) are of the age group 40 – 50 years with 16 male (43% of total males) and 3 female (23% of total females) and the remaining 31 patients are of 51 – 60 years age group with 21 male (57%) and 10 female (77%).

Table 17: Radiological parameters according to gender

Analysis of Radiological parameters according to gender

	Gender		Total (n=50)
	Male (n=37)	Female (n=13)	
Electrocardiogram			
• **LVH**	26(70.3%)	10(76.9%)	**36(72%)**
• **No LVH**	11(29.7%)	3(23.1%)	**14(28%)**
Chest X-Ray			
• **No LVH**	11(29.7%)	3(23.1%)	**14(28%)**
• **LVH**	26(70.3%)	10(76.9%)	**36(72%)**

Graph 17: Radiological parameters according to gender



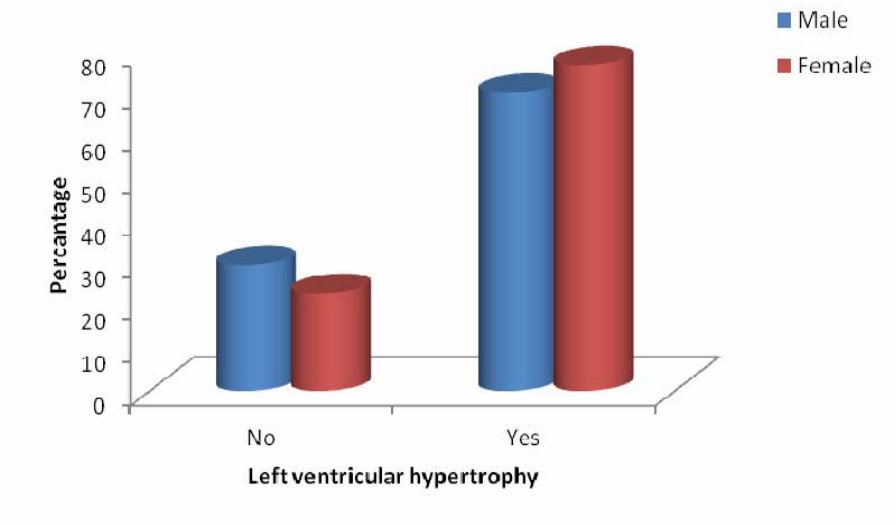
Of the 36 patients (72%), having Left Ventricular Hypertrophy in both Chest X-Ray and Electrocardiogram, 26 patients are male and 10 patients are female.

Table 18: Left ventricular hypertrophy in Echocardiogram according to gender

Analysis of Left ventricular hypertrophy in Echocardiogram according to gender

Left ventricular hypertrophy in Echo	Gender		Total
	Male	Female	
No	11(29.7%)	3(23.1%)	**14(28%)**
Yes	26(70.3%)	10(76.9%)	**36(72%)**
Total	**37(100%)**	**13(100%)**	**50(100%)**

Graph 18: Left ventricular hypertrophy in Echocardiogram according to gender



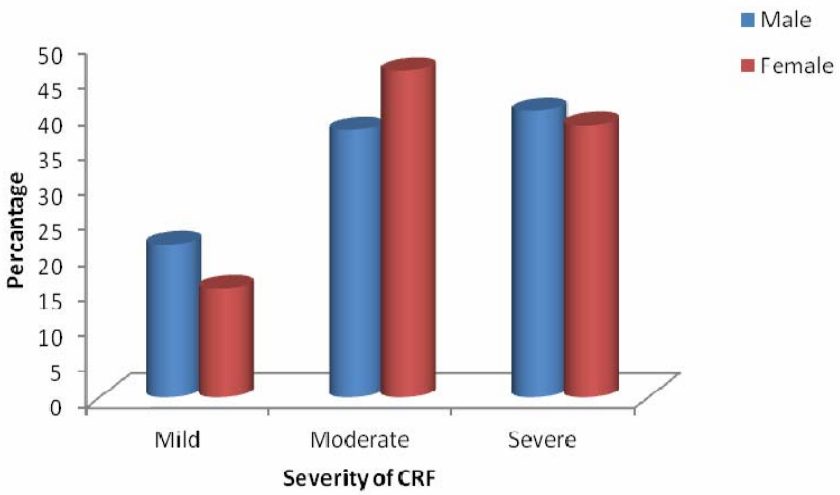
As shown in Table 18 and Graph 18, of the 37 total male patients, 26 (70%) had Left Ventricular Hypertrophic Changes in Echocardiogram as compared to 10 (77%) female patients (total female patients – 13).

Table 19: Severity of CRF according to gender

Analysis of Severity of CRF according to gender

Severity of CRF	Gender		Total
	Male	Female	
Mild	8(21.6%)	2(15.4%)	**10(20%)**
Moderate	14(37.8%)	6(46.2%)	**20(40%)**
Severe	15(40.5%)	5(38.5%)	**20(40%)**
Total	**37(100%)**	**13(100%)**	**50(100%)**

Graph 19: Severity of CRF according to gender



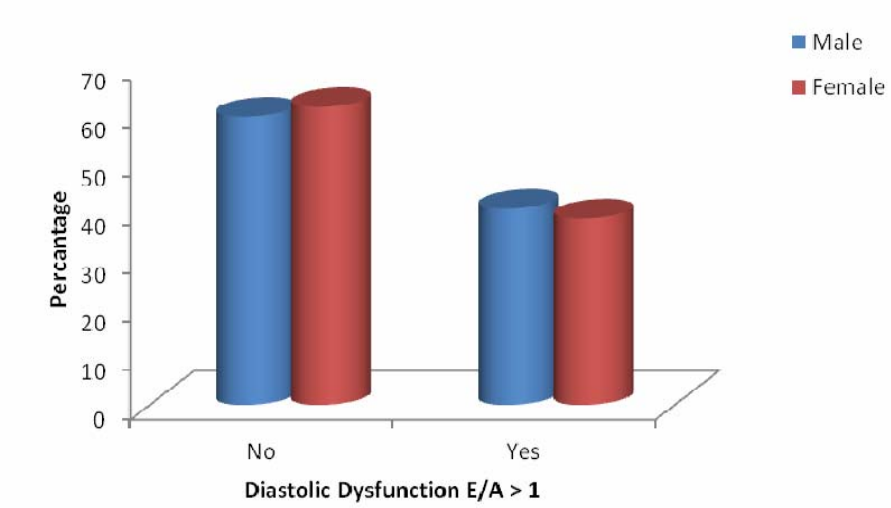
Of the 20 (40%) patients in Moderate CRF category, 14 (37.8%) are male patients and 06 (46%) are female patients as compared to 20 (40%) patients of Severe CRF category having 15 (40.5%) male patients and 05 (38.5%) female patients.

Table 20: Diastolic Dysfunction E/A >1 according to gender

Analysis of Diastolic Dysfunction according to gender

Diastolic Dysfunction E/A > 1	Gender		Total
	Male	Female	
No	22(59.5%)	8(61.5%)	**30(60%)**
Yes	15(40.5%)	5(38.5%)	**20(40%)**
Total	**37(100%)**	**13(100%)**	**50(100%)**

Graph 20: Diastolic Dysfunction E/A >1 according to gender

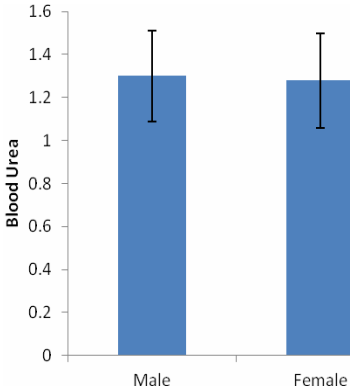


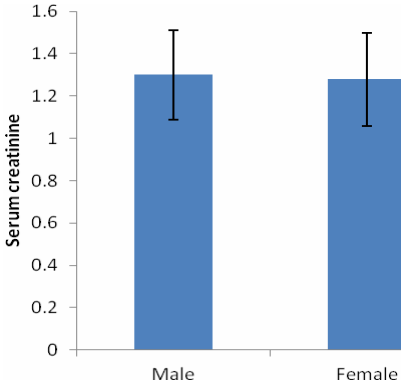
As shown in Table 20 and Graph 20, of the 20 patients (40%) having Diastolic Dysfunction,15 are male patients (40.5% of total male) and 5 are female patients (38.5% of total female).

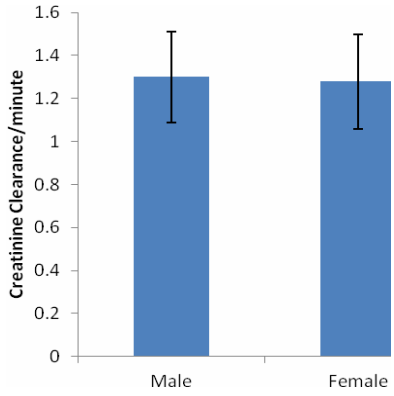
Table 21: Comparison of study variables according to gender

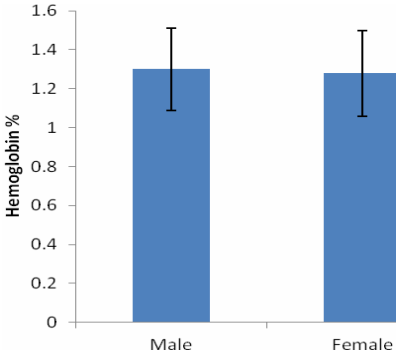
Variables	Gender		Total	P value
	Male	Female		
Blood Urea	115.97±46.39	139.00±47.67	**121.96±47.34**	**0.133**
Serum creatinine	6.19±4.21	6.51±4.40	**6.27±4.21**	**0.816**
Creatinine Clearance/minute	15.06±8.96	13.06±5.61	**14.54±8.21**	**0.457**
Hemoglobin %	8.39±1.27	8.65±1.73	**8.46±1.39**	**0.579**
Random blood sugar	210.95±92.94	179.00±77.39	**202.64±89.51**	**0.273**
Serum potassium	4.68±0.82	4.92±0.87	**4.74±0.83**	**0.394**
Serum calcium	8.87±1.03	9.20±0.85	**8.95±0.99**	**0.302**
Serum phosphorous	4.72±0.92	5.28±1.08	**4.87±0.99**	**0.082+**
Total cholesterol	196.32±37.65	206.69±30.80	**199.02±35.98**	**0.377**
Triglycerides	162.78±15.66	166.62±15.56	**163.78±15.57**	**0.451**
Low density cholesterol	115.81±13.27	120.92±14.91	**117.14±13.75**	**0.253**
High density cholesterol	34.97±6.61	33.31±6.17	**34.54±6.48**	**0.431**
Left ventricular mass	223.00±66.33	217.26±67.14	**221.51±65.90**	**0.790**
Left ventricular mass index	136.53±37.64	133.07±33.36	**135.63±36.28**	**0.771**
Internal diameter of left ventricle at end diastole	4.44±0.45	4.38±0.38	**4.42±0.43**	**0.665**
Thickness of posterior wall of left ventricle end Diastole	1.33±0.25	1.29±0.25	**1.32±0.25**	**0.643**
Thickness of interventricular septum	1.30±0.21	1.28±0.22	**1.29±0.21**	**0.767**

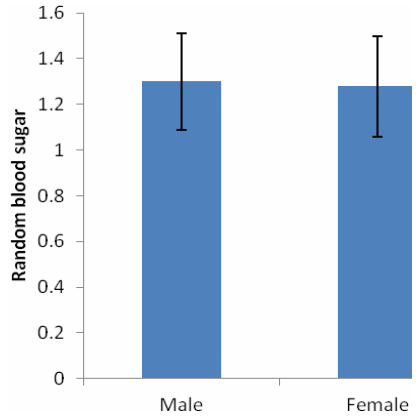
Graph 21: Comparison of study variables according to gender

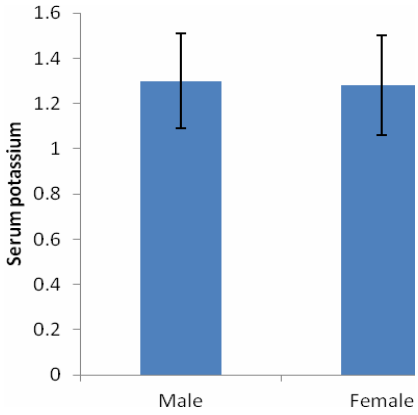

Blood Urea
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female

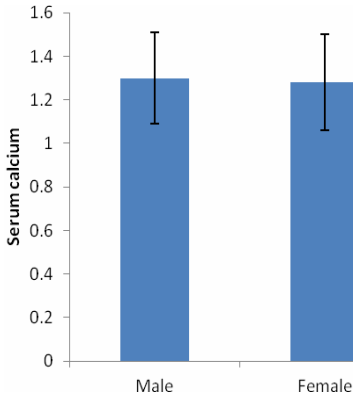

Serum creatinine
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female

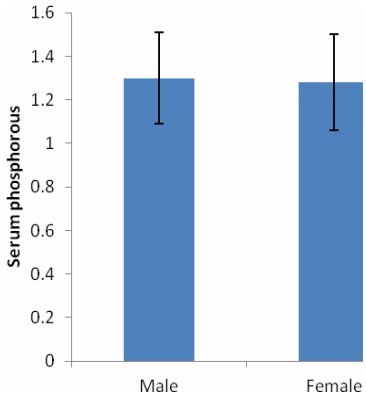

Creatinine Clearance/minute
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female

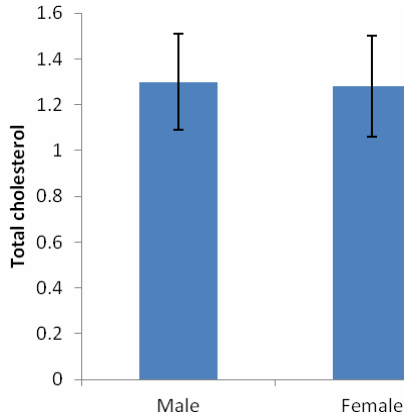

Hemoglobin %
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female

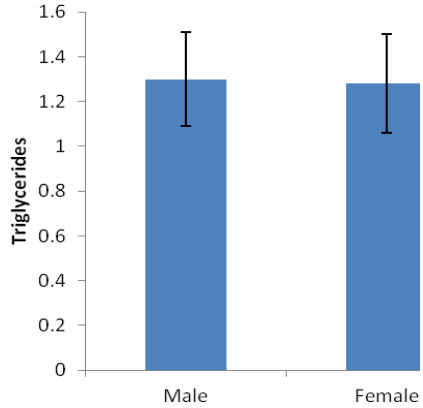

Random blood sugar
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female

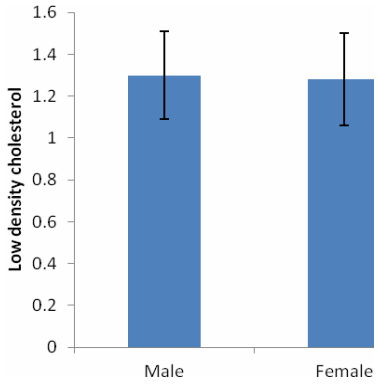

Serum potassium
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female

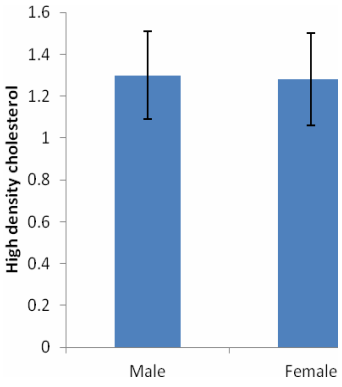

Serum calcium
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female

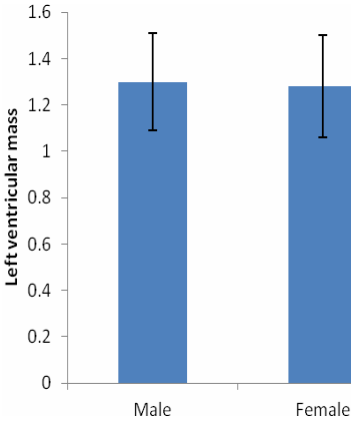

Serum phosphorous
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female

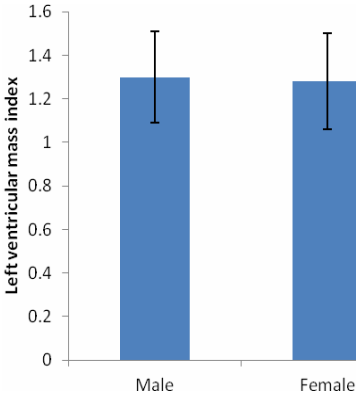

Total cholesterol
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female

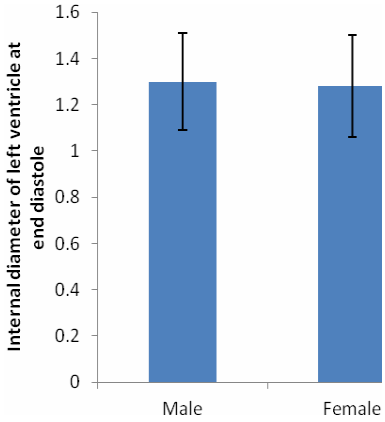

Triglycerides
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female

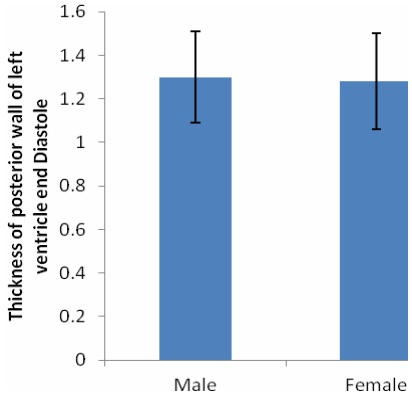

Low density cholesterol
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female

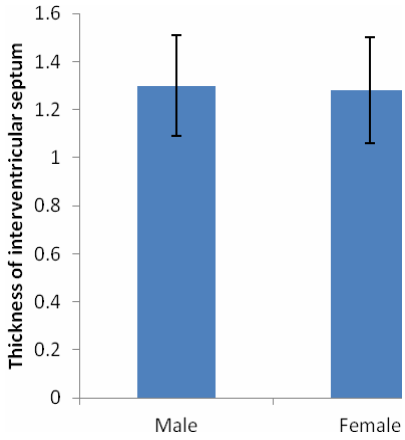

High density cholesterol
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female


Left ventricular mass
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female


Left ventricular mass index
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female


Internal diameter of left ventricle at end diastole
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female


Thickness of posterior wall of left ventricle end Diastole
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female


Thickness of interventricular septum
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Male
Female

Female patients were found to have a higher mean Blood Urea level compared to Male patients. However, the mean difference was not statistically significant.

Higher mean Serum Creatinine was found in Female patients compared to Male patient. But, the mean difference was not statistically significant.

The mean Creatinine Clearance (ml/min) was found to be higher in Male patients as compared to Female patients. However, the difference in mean was not statistically significant.

Hemoglobin (gm%) was found to be lower in Male patients. But the mean difference in Hemoglobin was not statistically significant.

Male patients were found to have a higher mean Random Blood Sugar level compared to Female patients. However, the mean difference was not statistically significant.

Higher mean Serum Potassium (mEq/l) was found in Female patients. But the difference in mean Serum Potassium was not statistically significant.

Lower mean Serum Calcium (mg/dl) was found in Male patients. But the difference in mean Serum Calcium was not statistically significant.

Female patients recorded higher mean Serum Phosphorus (mg/dl). The difference in mean Serum Phosphorus was not statistically significant.

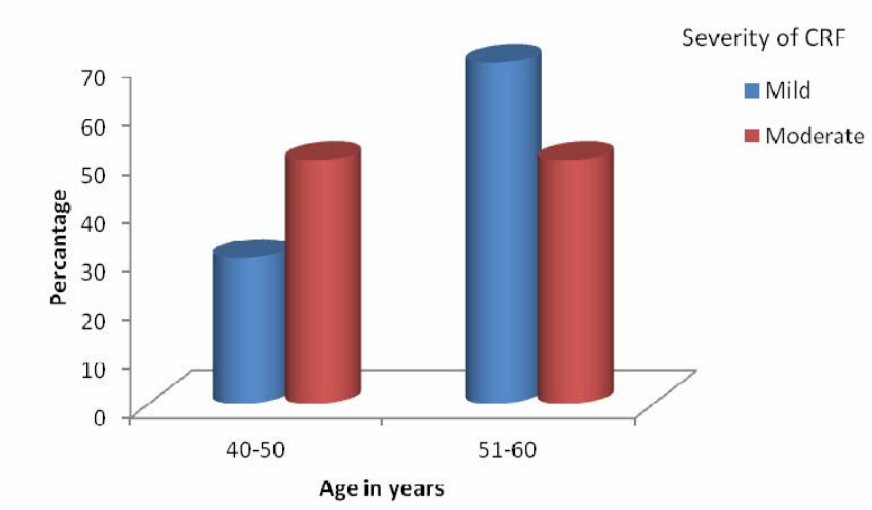
Table 22: Age distribution with Severity of CRF of patients studied

Distribution of Severity of CRF according to the age groups

Age in years	Severity of CRF			Total
	Mild	Moderate	Severe	
40-50	3(30%)	10(50%)	6(30%)	**19(38%)**
51-60	7(70%)	10(50%)	14(70%)	**31(62%)**
Total	**10(100%)**	**20(100%)**	**20(100%)**	**50(100%)**

P=0.361, Not significant, Chi-Square test

Graph 22: Age distribution with Severity of CRF of patients studied



In the present study, 19 patients of the age group 40 – 50 years consisted of 10 patients of Moderate CRF category followed by 06 patients in Severe and 03 patients in Mild CRF category.

31 patients of the age group 51 – 60 years consisted of 14 patients of Severe CRF category followed by 10 patients in Moderate and 07 patients in Mild CRF category.

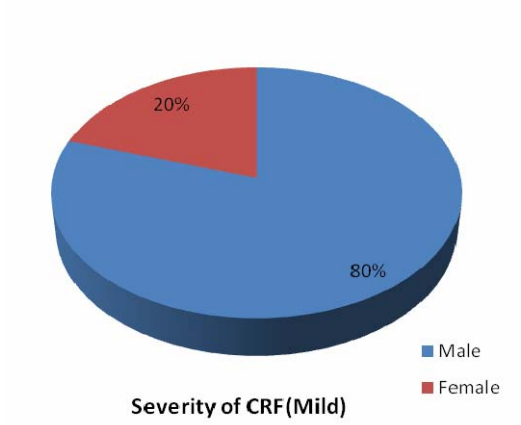
Table 23: Gender distribution with Severity of CRF of patients studied

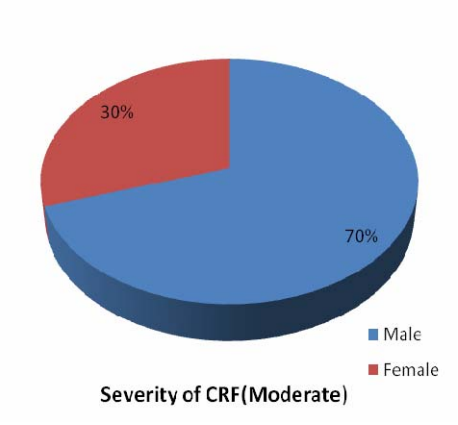
Distribution of Severity of CRF according to gender

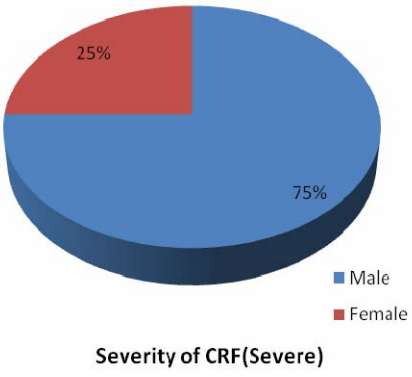
Gender	Severity of CRF			Total
	Mild	Moderate	Severe	
Male	8(80%)	14(70%)	15(75%)	**37(74%)**
Female	2(20%)	6(30%)	5(25%)	**13(26%)**
Total	**10(100%)**	**20(100%)**	**20(100%)**	**50(100%)**

P=0.834, Not significant, Chi-Square test

Graph 23: Gender distribution with Severity of CRF of patients studied







Of the 37 total male patients, 15 are of Severe CRF category followed by 14 patients in Moderate and 08 patients in Mild Categories of CRF

Of the 13 total female patients, 06 are of Moderate CRF category followed by 05 patients in Severe and 02 patients in Mild Categories of CRF.

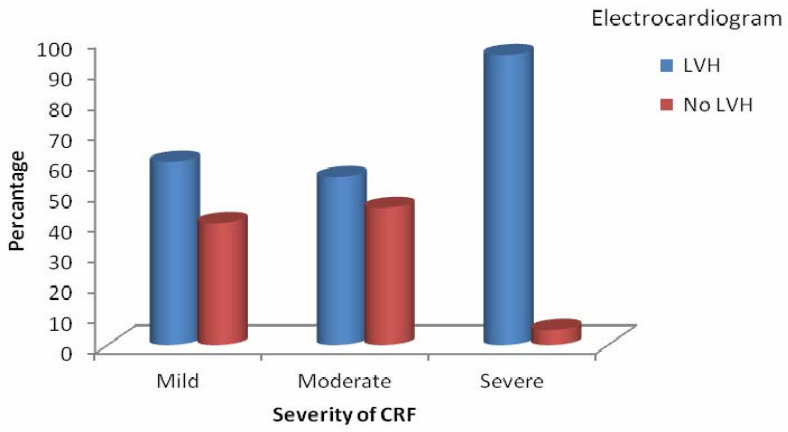
Table 24: Radiographic findings with Severity of CRF of patients studies

Analysis of Radiological parameters according to Severity of CRF

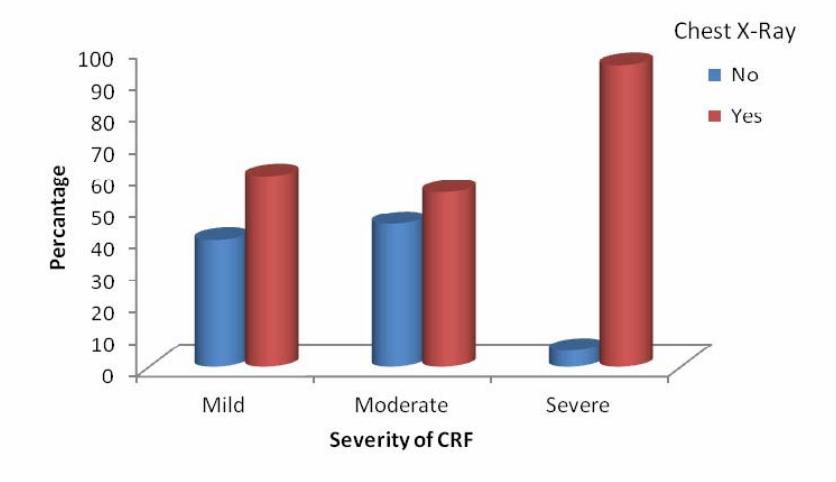
	Severity of CRF			Total (n=50)	P value
	Mild (n=10)	Moderate (n=20)	Severe (n=20)		
Electrocardiogram					
• **LVH**	6(60%)	11(55%)	19(95%)	**36(72%)**	**0.012***
• **No LVH**	4(40%)	9(45%)	1(5%)	**14(28%)**	
Chest X-Ray					
• **No**	4(40%)	9(45%)	1(5%)	**14(28%)**	**0.012***
• **Yes**	6(60%)	11(55%)	19(95%)	**36(72%)**	

P=0.012 Significant, Chi-Square test

Graph 24: radiographic findings with Severity of CRF of patients studied



Of the 36 total patients having Left Ventricular Hypertrophic Changes in Electrocardiogram, 19 patients are Severe CRF category, 11 patients in Moderate and 0 6 patients are Mild Categories of CRF.



Of the 36 total patients having Left Ventricular Hypertrophic Changes in Chest X-Ray, 19 patients are Severe CRF category, 11 patients in Moderate and 06 patients are Mild Categories of CRF.

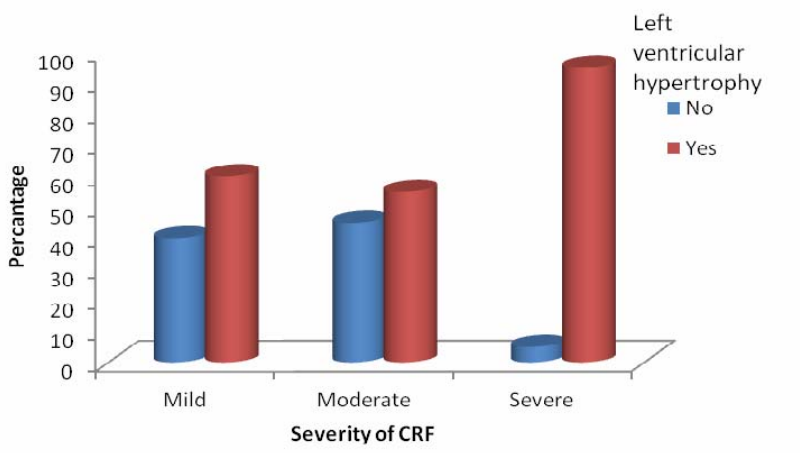
Table 25: Left ventricular hypertrophy with Severity of CRF of patients studied

Analysis of Left ventricular hypertrophy according to Severity of CRF

Left ventricular hypertrophy	Severity of CRF			Total
	Mild	Moderate	Severe	
No	4(40%)	9(45%)	1(5%)	**14(28%)**
Yes	6(60%)	11(55%)	19(95%)	**36(72%)**
Total	**10(100%)**	**20(100%)**	**20(100%)**	**50(100%)**

P=0.012*, Significant, Chi-Square test

Graph 25: Left ventricular hypertrophy with Severity of CRF of patients studied



Of the 36 total patients having Left Ventricular Hypertrophic Changes in Echocardiogram, 19 patients are Severe CRF category, 11 patients in Moderate and 0 6 patients are Mild Categories of CRF.

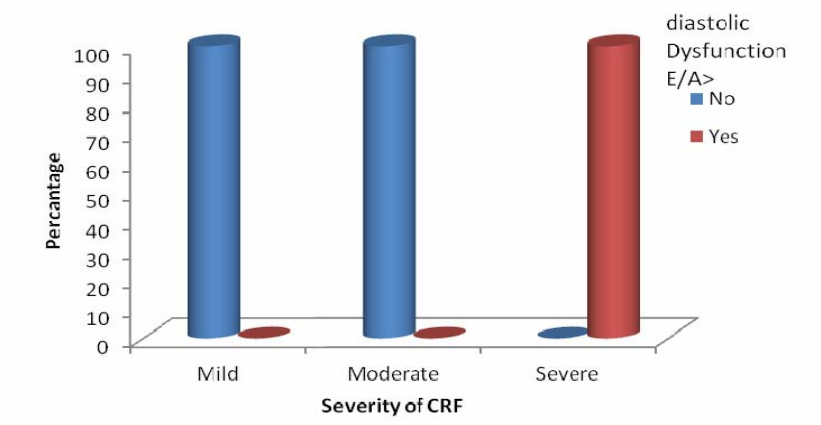
Table 26: Diastolic Dysfunction with Severity of CRF of patients studied

Analysis of Diastolic Dysfunction with Severity of CRF

Diastolic Dysfunction E/A > 1	Severity of CRF			Total
	Mild	Moderate	Severe	
No	10(100%)	20(100%)	0(0%)	**30(60%)**
Yes	0(0%)	0(0%)	20(100%)	**20(40%)**
Total	**10(100%)**	**20(100%)**	**20(100%)**	**50(100%)**

P<0.001**, Significant, Fisher Exact test

Graph 26: Diastolic Dysfunction with Severity of CRF of patients studied

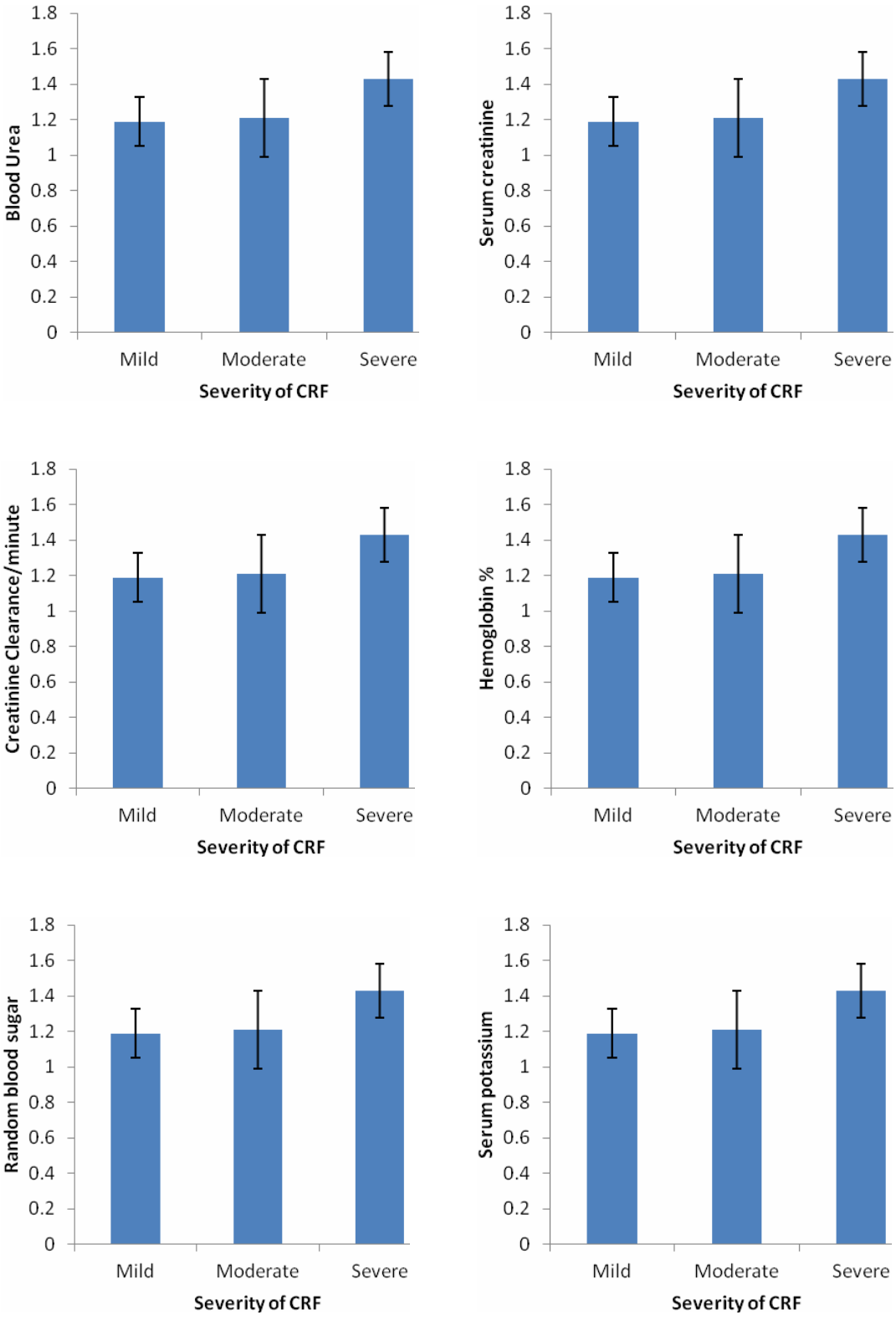


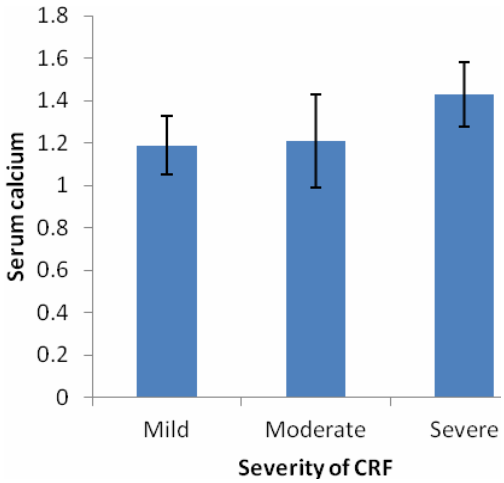
All the 20 patients (40%) having Diastolic Dysfunction are of Severe Category CRF.

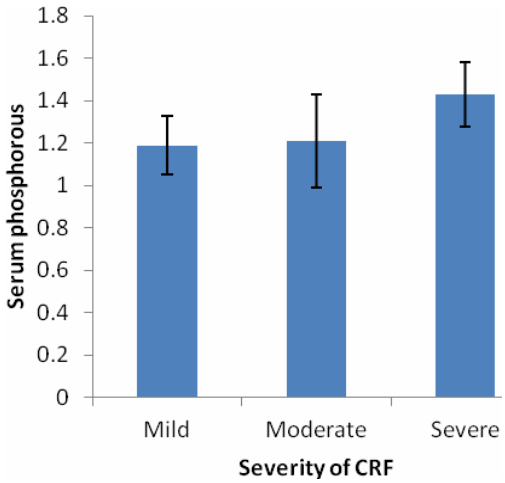
Table 27: Comparison of study variables according to Severity of CRF

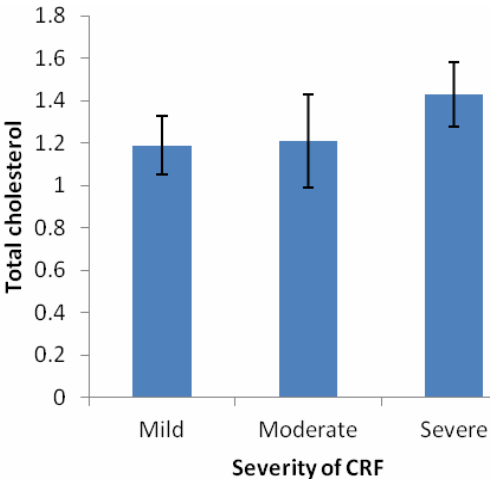
Variables	Severity of CRF			Significance		
	Mild	Moderate	Severe	Mild-Mod	Mild-Severe	Mod-Severe
Blood Urea	97.30±30.25	98.60±34.65	157.65±43.71	**0.996**	**<0.001****	**<0.001****
Serum creatinine	2.40±0.35	4.11±0.71	10.37±3.84	**0.190**	**<0.001****	**<0.001****
Creatinine Clearance/minute	25.84±10.39	15.27±2.22	8.16±2.71	**<0.001****	**<0.001****	**<0.001****
Hemoglobin %	8.93±10	9.05±1.33	7.64±1.24	**0.969**	**0.026***	**0.002****
Random blood sugar	172.50±75.69	243.65±98.94	176.70±72.15	**0.087+**	**0.991**	**0.041***
Serum potassium	4.42±0.79	4.79±1.00	4.87±0.65	**0.500**	**0.359**	**0.950**
Serum calcium	9.40±1.43	8.78±0.86	8.91±0.82	**0.243**	**0.402**	**0.915**
Serum phosphorous	4.97±0.77	5.26±0.85	4.43±1.06	**0.701**	**0.294**	**0.018***
Total cholesterol	191.20±52.35	188.75±26.64	213.20±31.24	**0.982**	**0.241**	**0.077+**
Triglycerides	170.40±14.57	165.85±9.09	158.40±19.59	**0.719**	**0.112**	**0.275**
Low density cholesterol	122.00±13.00	122.25±12.26	109.60±12.54	**0.999**	**0.036***	**0.007****
High density cholesterol	37.50±6.82	34.90±7.31	32.70±4.95	**0.546**	**0.136**	**0.522**
Left ventricular mass	200.17±74.51	188.22±52.24	265.46±49.00	**0.847**	**0.011***	**<0.001****
Left ventricular mass index	125.35±44.73	119.15±30.02	157.25±26.67	**0.873**	**0.036***	**0.001****
Internal diameter of left ventricle at end diastole	4.35±0.53	4.31±0.37	4.57±0.41	**0.967**	**0.375**	**0.135**
Thickness of posterior wall of left ventricle end Diastole	1.26±0.33	1.23±0.22	1.44±0.18	**0.940**	**0.119**	**0.016***
Thickness of interventricular septum	1.19±0.14	1.21±0.22	1.43±0.15	**0.975**	**0.003****	**0.001****

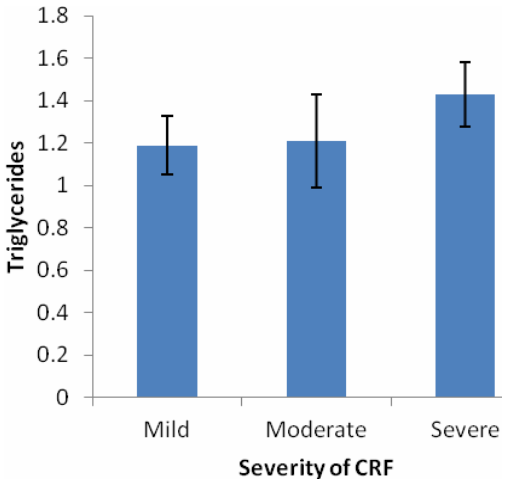
Graph 27: Comparison of study variables according to Severity of CRF

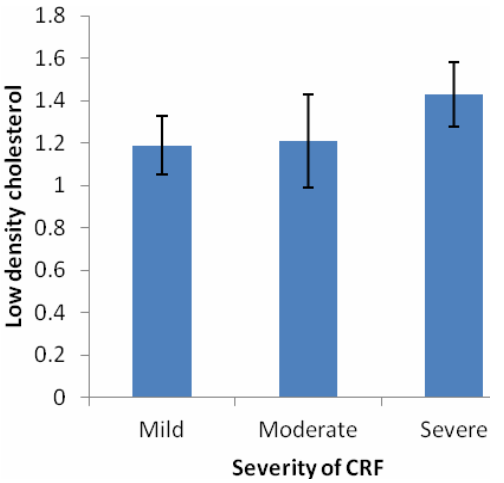


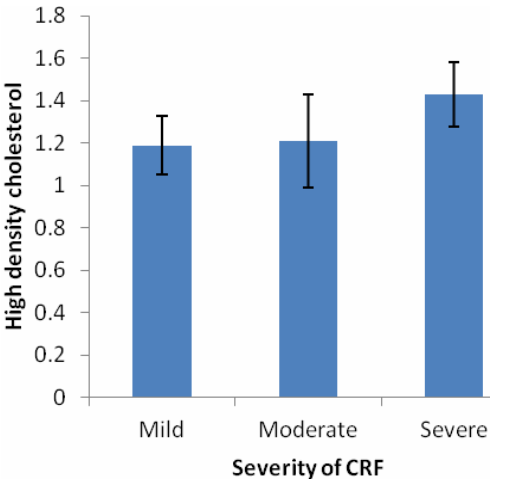

Serum calcium
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Mild
Moderate
Severe
Severity of CRF

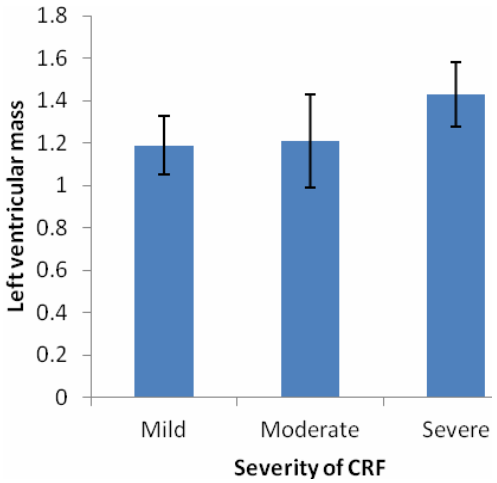

Serum phosphorous
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Mild
Moderate
Severe
Severity of CRF

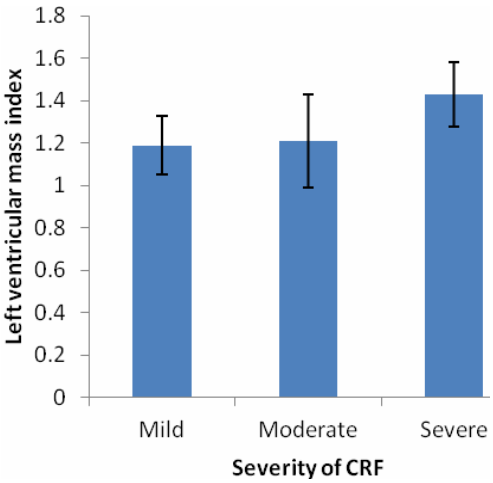

Total cholesterol
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Mild
Moderate
Severe
Severity of CRF

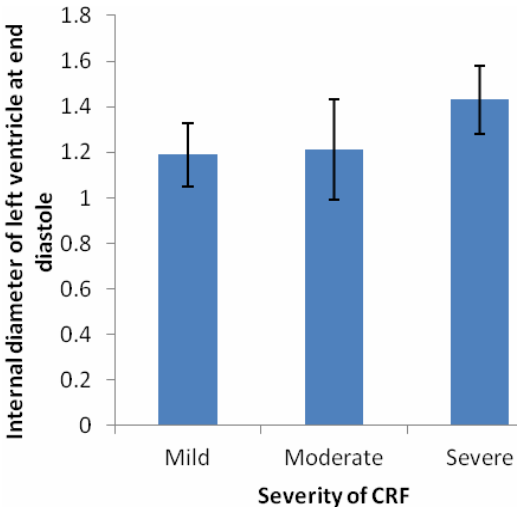

Triglycerides
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Mild
Moderate
Severe
Severity of CRF

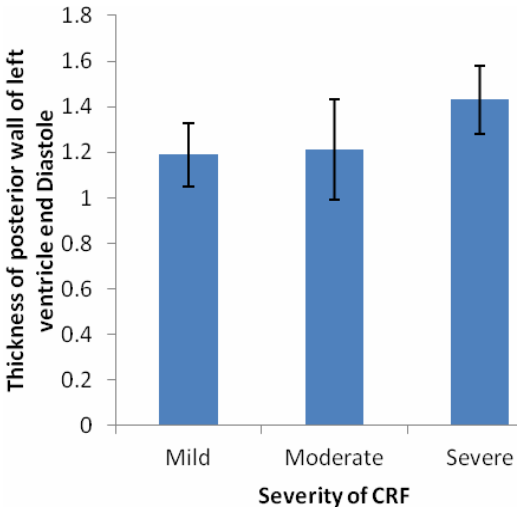

Low density cholesterol
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Mild
Moderate
Severe
Severity of CRF

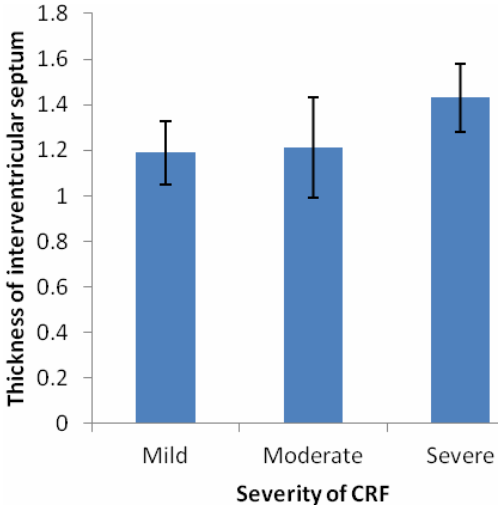

High density cholesterol
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Mild
Moderate
Severe
Severity of CRF


Left ventricular mass
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Mild
Moderate
Severe
Severity of CRF


Left ventricular mass index
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Mild
Moderate
Severe
Severity of CRF


Internal diameter of left ventricle at end diastole
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Mild
Moderate
Severe
Severity of CRF


Thickness of posterior wall of left ventricle end Diastole
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Mild
Moderate
Severe
Severity of CRF


Thickness of interventricular septum
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Mild
Moderate
Severe
Severity of CRF

Patients with severe CRF were found to have a higher mean Blood Urea level compared to patients with moderate and mild CRF and the mean difference between them was found to be statistically significant ($P<0.001$). However, the mean difference between patients with mild and moderate CRF were not statistically significant.

Higher mean Serum Creatinine was found in patients of severe CRF category followed by moderate and mild categories and and the mean difference between them was found to be statistically significant ($P<0.001$). However, the mean difference between patients with mild and moderate CRF were not statistically significant.

The mean Creatinine Clearance (ml/min) was found to be higher in mild category followed by moderate and severe categories. The difference in mean Creatinine Clearance between each group was found to be statistically significant ($P<0.001$).

Hemoglobin (gm%) was found to be lower in patients belonging to severe CRF category as compared to mild and moderate categories respectively. The mean difference in Hemoglobin between mild and severe category as well as between moderate and severe category was found to be statistically significant. But the mean difference in Hemoglobin between mild and moderate categories was not statistically significant.

Patients with Moderate CRF were found to have a higher mean Random Blood Sugar level compared to patients with Severe and Mild CRF and the mean difference between them was found to be statistically significant between Moderate and Severe groups. However, the mean difference between patients with Mild and Moderate CRF and Mild and Severe CRF were not statistically significant.

Higher mean Serum Potassium (mEq/l) was found in patients with severe CRF followed by patients with moderate and mild CRF categories. But the difference in mean Serum Potassium was not statistically significant between any of the categories.

Lower mean Serum Calcium (mg/dl) was found in patients with Moderate CRF followed by patients with Severe and Mild CRF categories. But the difference in mean Serum Calcium was not statistically significant between any of the categories.

Patients in moderate category of CRF recorded higher mean Serum Phosphorus (mg/dl) followed by Mild and Severe categories respectively. The difference in mean Serum Phosphorus was found to be statistically significant between moderate and severe categories. However, statistically significant difference was noticed between mild and severe as well as mild and moderate categories.

Higher mean Total Cholesterol levels were found in Severe CRF category of patients followed by Mild and Moderate categories respectively. But the difference in mean Total Cholesterol levels between the groups was not statistically significant.

Higher mean Triglyceride levels were found in Moderate CRF category of patients followed by Mild and Severe categories respectively. However, difference in mean Triglyceride levels between the groups was not statistically significant.

Patients of Mild and Moderate categories of CRF had higher LDL followed by Severe category CRF. The mean LDL difference between mild and severe category as well as moderate and severe category were found to be statistically significant.

The mean HDL was found to be lower in patients with Severe CRF compared to patients with moderate and mild CRF. However, difference in mean HDL levels between the groups was not statistically significant.

Higher mean Left Ventricular Mass were found in Severe CRF category of patients followed by Mild and Moderate categories respectively. The mean difference between mild and severe category as well as moderate and severe category were found to be statistically significant. But, difference in mean between Mild and Moderate groups was not statistically significant.

The mean Left Ventricular Mass Index was found to be lower in patients with Moderate CRF compared to patients with Mild and Severe CRF. The mean difference between mild and severe category as well as moderate and severe category were found to be statistically significant However, difference in mean between mild and moderate groups was not statistically significant.

DISCUSSION

Premature cardiovascular disease is a significant cause of morbidity and mortality among patients with CRF. Four main structural abnormalities of the heart have been described in patients with CRF: LV hypertrophy, expansion of the nonvascular cardiac interstitium leading to inter-myocardiocytic fibrosis, changes in vascular architecture, and myocardial calcification.

All these abnormalities promote systolic as well as diastolic LV dysfunction which predisposes to symptomatic heart failure, which is a risk factor for premature death.

Various diagnostic modalities, both invasive and noninvasive such as electrocardiography, echocardiography and radionuclide scans are utilized for diagnosing left ventricular hypertrophy and dysfunction.

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.

In the present study, we found that LVMI and diastolic dysfunction showed a progressive rise with increase in severity of renal failure. This is in concordance with the study done by Dangiri P et al[2], Agarwal S et al[3], Adeera Levin et al[11] who also found a similar trend of LVMI in patients of CRF.

In the present study majority of the patients 28 patients (56%) had hemoglobin levels between 7.1-9 gm%. In 60 % of patients Serum Potassium level was between 3.5 - 5 mEq/L. In 28 patients (56%) Serum calcium levels was between 8.8 - 10.2 mg/dl and 27 patients (54%) the serum phosphorus levels were between 2.5 - 4.8 mg/dl, all of which is in concordance with study done by Agarwal S et al 2 and N A Tomilina, et al.[19]

In the present study, HDL cholesterol level <60mg/dl was observed in 100% patients which is similar to study done by Dangri P et al 2. Triglycerides levels >165 mg/dl was observed in 27(54%) of patients and Cholesterol levels >200 m/dl in 16 patients (32 %) the values were much lower as compared to studies by Dangri P et al [2] and Yashpal et al.[28]

In the present study, out of 50 patients 36 (72%) patients had Left Ventricular Hypertrophy on Echocardiography which is comparatively similar to study done by Laddha M et al.[126] in 2014, reported as LVH in 74%, as compared to other studies done by Zoccali C et al [127] (77%), Shivendra et al [128] (48%), Adeera Levin et al 11 (70%), Yashpal et al28 (15.49%), Chafekar D S et al [32] (17.6%), Parfrey P S et al [29] (41%), Rachel J Middleton, et al [129] (41%), Tomilina N A et al (52.6%), Kale S A et al [46] (54.7%), Goran J Paunovic et al [39] (56.9%), Sanchari Datta et al [7] (77%)

Of the 72% of patients with LVH, 06 (17%) patients were from Mild CRF Category, 11(30%) patients were from Moderate CRF category and 19 (53%) patients were from severe Chronic Renal Failure category respectively as compared to 40% in Mild and Moderate and 97% in Severe CRF categories as shown by Dangri P et al[2] and 30% in Mild/Moderate category and 53.2% in Severe Category as shown by Agarwal S et al.[3]

In the present study, out of 50 patients 20 (40%) patients had diastolic dysfunction which is comparatively similar to study done by Laddha M et al[126], reported in 61.4%, as compared to other studies done by Agarwal S. et al[128] (53.2%), McMurray et al.[129] (50%), Kunz K.et al [130] (50 %), Shivendra et al (51%).

CONCLUSION

The present study shows that patients with Chronic Renal Failure have higher Left ventricular mass index (LVMI) and higher prevalence of Left ventricular hypertrophy (LVH) and diastolic dysfunction and also with respect to category of Chronic Renal Failure, the LVH and diastolic dysfunction prevalence progressively increases with increasing severity of Chronic Renal Failure Category.

The high prevalence of Left ventricular hypertrophy and diastolic dysfunction 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.

LVH has got prognostic implications, because this group of ESRD patients will die of diastolic dysfunction or sudden cardiac death.131 Echocardiography is a cost effective noninvasive diagnostic test which can detect early changes in the cardiac parameters. This is important for risk stratification and early preventive measures.

Thus, echocardiographic screening of ESRD patients has both therapeutic and prognostic implications. All asymptomatic ESRD patients should undergo a routine echocardiographic evaluation.

SUMMARY

Randomly selected 50 patients of diabetic nephropathy undergoing hemodialysis of age group 40 to 60 years were studied for the prevalence of Left Ventricular Hypertrophy and Diastolic Dysfunction with the help of Echocardiography.

In the present study, 37 patients were males and 13 patients were females. The maximum number of patients were in the age group of 51-60 years (62%).

In the present study, hemoglobin (gm%) was found to be lower in patients belonging to severe CRF category as compared to mild and moderate categories respectively.

Low mean Serum Calcium (mg/dl) was found in patients with moderate CRF followed by patients with severe and mild categories. Patients in moderate category of CRF recorded higher mean Serum Phosphorus (mg/dl) followed by severe and mild categories respectively. The difference in mean Serum Phosphorus was found to be statistically significant between moderate and severe categories.

Higher mean Total Cholesterol levels were found in severe CRF category of patients followed by mild and moderate categories respectively. Higher mean Triglycerides levels were found in mild CRF category of patients followed by moderate and severe categories respectively. Patients in the moderate category of CRF had higher LDL followed by mild and severe categories and difference in mean LDL was significant between mild and severe and also between moderate and severe categories.

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ANNEXURES

PROFORMA

1. **DEMOGRAPHIC DATA OF THE PATIENT**

Name -	Serial no. case –
Age -	Sex –
Unit -	Date of Admission -
I.P No. / O.P No -	Occupation -
Address -	

2. **History of Presenting Complaints** :

1. Fever	yes / no
2. Cough	yes / no
3. Chest pain	yes / no
4. Breathlessness	yes / no
5. Nausea	yes / no
6. Vomiting	yes / no
7. Hiccups	yes / no
8. Pain abdomen	yes / no
9. Diarrhoea	yes / no
10. Easy fatigability	yes / no
11. Anemia	yes / no
12. Puffiness of face	yes / no
13. Swelling of lower limbs	yes / no
14. Musculoskeletal pain	yes / no

15. Pruritis yes / no

16. Convulsions yes / no

17. Burning micturation yes / no

18. Haematuria yes / no

19. Oliguria yes / no

20. Nocturia yes / no

21. Anuria yes / no

3. Past history :

H/O Similar Complaints in the past yes / no

H/O Renal Disease

H/O Diabetes yes / no Duration –

H/O Hypertension yes / no Duration –

H/O Ischemic heart disease yes / no Duration -

4. **Drug history** :

NSAIDS yes / no Drugs - Duration -

Anti diabetic drugs yes / no Drugs - Duration -

Anti hypertensives yes / no Drugs - Duration -

5. Family history :

H/O Renal diseases yes / no

H/O Cardiac diseases yes / no

H/O Hypertension H/O yes / no

Diabetes yes / no

6. **Personal history** :

Diet	-
Appetite	-
Sleep	-
Bowel and Bladder habits	-
Habits	-

7. **General Physical Examination** :

1. Built - Well / Moderate / Poor
2. Nutrition - Good / Moderate / Poor
3. Uremic odour - Present / Absent
4. Nails - Normal / Pallor / Brittle
5. Pallor - Present / Absent
6. Cyanosis - Present / Absent
7. Oedema - Generalized / Localized / Facial / Abdominal / Pedal
8. Skin - Smooth / Dry / Scaly / Pigmentation
9. Lymphadenopathy

Vital signs :

P.R	-	/bpm	Height	-	mts
B.P	-	mm/hg	Weight	-	kgs
R.R	-	cycles/min	BMI	-	
JVP	-		Body surface area	-	

8. **Systemic examination:**

1. **Cardiovascular system** : Inspection:

Palpation: Percussion: Auscultation:

2. **Respiratory system** : Inspection:

   Palpation : Percussion: Auscultation:

3. **Abdominal examination :**

   Inspection : Palpation : Percussion : Auscultation:

4. **Central nervous system**: Higher Mental Functions: Cranial Nerves:

   Motor system : Sensory system: Cerebellar signs:

   Skull and spine:

Investigations:

1. **Blood routine :**

   Hb - gm%

   TC - /cumm

   DC - P ;L ; M ;E .

   ESR - mm/hr

   RBS - mg/dl HBA1C -

   FBS - mg/dl PPBS - mg/dl

2. **Renal function test**:

   B.Urea - mg/dl

   Sr.Creatinine - mg/dl

   Sr.Calcium - mg/dl

   Sr.Phosphrous - mg/dl

   Sr.Electrolytes - Na+ - meq/lt

   K+ - meq/lt

   Cl- - meq/lt

3**. Liver Function Test**:

T.B D.B SGOT SGPT SAP T.Pr Alb A/G

4. **Urine Analysis** :

Urine routine - Alb - Sugar - Microscopy - cells -

Casts - Urine culture -

5. **ECG -**

6. **Chest X-ray PA view**

7. **Ultrasound Abdomen**

Kidney (size, shape)

8. **Fundus Examination :** (Normal or Diabetic Retinopathy)

9. **2D-Echo** :

LVIDd -

LVPWd-

IVSd -

E -

A -

E/A > 1 yes / no

Left Ventricular Mass :

Left Ventricular Mass Index :

Diastolic Dysfunction :

SUMMARY:

C.R.F : MILD / MODERATE / SEVERE

L.V.H : PRESENT / ABSENT

Diastolic Dysfunction : PRESENT / ABSENT

KEY TO MASTER CHART

%	-	Percentage
Cr.Clear	-	Creatinine Clearance
CXR	-	Chest X ray

Diastolic Dysfunction E/A > 1 - Early to Late Peak Mitral Inflow Velocity.

ECG	-	Electrocardiogram
Echo	-	Echocardiogram
Hb	-	Hemoglobin
HDL	-	High Density Lipoprotein
IVSd	-	Thickness of the interventricular septum in diastole
LDL	-	Low Density Lipoprotein
LVIDd	-	Internal diameter of the left ventricle at end diastole
LVM	-	Left Ventricular Mass
LVMI	-	Left Ventricular Mass Index
RBS	-	Random Blood Sugar
Sr.Ca	-	Serum Calcium
Sr.Phos	-	Serum Phosphorus
Sr.Pot	-	Serum Potassium
T.Chol	-	Total Cholesterol
Tg	-	Triglycerides

MASTER CHART

Sl No	Patient Name	Age	Sex	Blood Urea	S.Cr	Cr.clear/min	Hb %	RBS	Sr. K	Sr. Ca	Sr. Phos	T. Chol	TG	LDL	HDL	ECG	CXR	LVM	LVMI	LVH	Severity of CRF	LVIDd	LVPWd	IVSd	Diastolic Dysfunction E/A > 1
1	Venkatachalam	52 years	Male	128	12.1	8.08	8.8	270	5	9.8	5.9	200	172	110	28	LVH	Y	253.6	130.6	Y	S	4.9	1.2	1.4	Y
2	Muniraju	42 years	Male	218	9.4	8.12	5.7	134	5.2	7.9	4.2	230	160	106	30	LVH	Y	270	165.6	Y	S	5.1	1.3	1.3	Y
3	Vijaya	51 years	Male	210	20.8	3.54	7.4	108	4.8	9.7	3.5	220	176	106	28	LVH	Y	352.8	195.8	Y	S	5.3	1.6	1.4	Y
4	Natarajan	56 years	Male	192	6.1	8.28	7.9	198	4.7	7.6	3.5	180	150	100	36	LVH	Y	192.6	137.5	Y	S	4.1	1.3	1.3	Y
5	Saraswathi	52 years	Female	160	10.6	7.35	8.4	128	5.1	9.8	5	200	150	100	34	LVH	Y	261.8	156.7	Y	S	4.5	1.4	1.5	Y
6	Govinda Reddy	45 years	Male	124	6.2	15.4	9.1	295	4.3	8.6	4.2	156	142	98	42	LVH	Y	355.8	205.6	Y	S	4.4	1.7	1.9	Y
7	Srinivas.B.M.	53 years	Male	122	12.1	8.09	9.2	276	5.2	10.1	7.8	240	156	110	30	LVH	Y	253.6	130.6	Y	S	4.9	1.2	1.4	Y
8	Uma C Rao	56 years	Female	222	13.6	5.03	6	104	4.4	8.6	3.8	258	150	138	28	LVH	Y	245.1	153.1	Y	S	4	1.5	1.6	Y
9	Narayana Reddy	55 years	Male	127	8.5	7	9.5	121	4.4	8.9	3.9	226	166	110	32	LVH	Y	224.4	147.6	Y	S	4.2	1.4	1.4	Y
10	Shankarappa	55 years	Male	198	15.2	7.3	7	183	4.8	7.9	4	220	166	110	30	LVH	Y	284.8	152.2	Y	S	4.6	1.6	1.4	Y
11	Suri Singh	53 years	Male	190	11.3	7.47	7.7	340	5.5	7.7	4.2	200	150	100	30	LVH	Y	284.8	170.5	Y	S	4.6	1.6	1.4	Y
12	Guruppa	48 years	Male	127	10.2	5.43	6	120	5.2	9	3.5	200	130	98	36	LVH	Y	273.8	195.5	Y	S	4.8	1.4	1.4	Y
13	Bibijan	55 years	Female	190	10.4	10.65	5.4	88	5.3	9	5.8	190	168	100	42	LVH	Y	312.84	160.4	Y	S	4.9	1.6	1.4	Y
14	Reddy.A.V.	53 years	Male	197	12.1	6.43	8	113	5.9	8.3	5.1	246	168	110	30	LVH	Y	284.7	172.5	Y	S	4.6	1.6	1.4	Y
15	Muniyamma	53 years	Female	187	15	5.15	7.7	126	4.6	9.2	3.7	230	160	106	28	LVH	Y	294.9	177.6	Y	S	4.4	1.8	1.4	Y
16	Syed Ayub	43 years	Male	113	6.6	11.6	7.6	169	4.2	8.4	3.8	156	126	100	38	LVH	Y	232.7	157	Y	S	4	1.4	1.6	Y
17	Papamma	59 years	Female	108	7	7.42	8.3	178	3.7	8.5	3.9	268	210	136	30	LVH	Y	312.8	167.2	Y	S	4.9	1.4	1.6	Y
18	Rangan.S.	52 years	Male	71	6.9	9.7	6.1	160	4.6	10.3	4	206	126	106	32	LVH	Y	255.4	156.3	Y	S	5.1	1.2	1.3	Y
19	Narayana Shetty	50 years	Male	123	6	10.03	8.3	238	6.4	9	4	250	176	136	28	LVH	Y	192.6	113.2	Y	S	4.1	1.3	1.3	Y
20	Venkatesh	50 years	Male	100	4.2	12.6	10.6	286	4.6	7.8	4.7	239	158	136	30	LVH	Y	190.4	134	Y	Mod	3.9	1.4	1.3	N
21	Haroon Rasheed	40 years	Male	76	3.9	19.4	9.6	186	4.8	9.6	3.8	196	178	132	34	LVH	Y	256.7	156.5	Y	Mod	4.3	1.6	1.6	N
22	Bhavaiah	50 years	Male	56	3.9	17.3	9.6	230	3.8	9	3.8	178	163	128	30	LVH	Y	285.3	163	Y	Mod	4.7	1.2	1.6	N
23	Devamma	59 years	Female	112	3.5	15.9	11	121	5.8	9.6	7	236	170	132	33	LVH	Y	265.1	161.6	Y	Mod	3.9	1.3	1.4	N
24	Krishnamma	52 years	Female	73	4.5	14.6	11	302	5.9	9.7	7	186	166	123	29	LVH	Y	190.4	134	Y	Mod	5	1.1	1.1	N
25	Somaiah	60 years	Male	56	3.1	15.9	9.3	268	3.1	7.8	5.1	246	186	141	26	LVH	Y	207	130.1	Y	Mod	4.2	1.3	1.4	N
26	Shivappa Gowda	54 years	Male	112	3.5	15.9	11	121	5.8	8.8	5.9	190	170	132	33	LVH	Y	265.1	161.6	Y	Mod	4.2	1.4	1.2	N

MASTER CHART

Sl No	Patient Name	Age	Sex	Blood Urea	S.Cr	Cr.clear/min	Hb %	RBS	Sr. K	Sr. Ca	Sr. Phos	T. Chol	TG	LDL	HDL	ECG	CXR	LVM	LVMI	LVH	Severity of CRF	LVIDd	LVPWd	IVSd	Diastolic Dysfunction E/A > 1
27	Achamma	45 years	Female	149	4.2	12.5	8.8	280	5.2	9.2	5.2	196	156	138	30	LVH	Y	168.9	122.3	Y	Mod	4.4	1.1	1.1	N
28	Shivamma	58 years	Female	172	4.5	14.6	11	302	5.9	9.7	5.8	186	166	123	29	LVH	Y	190.4	134	Y	Mod	4.2	1.4	1.3	N
29	Kamala Kannan	45 years	Male	129	5.3	11.93	9.2	138	4.3	8.6	5.4	198	160	110	38	LVH	Y	212.3	132.6	Y	Mod	4.8	1.8	1.6	N
30	Syed Arifunna	60 years	Male	116	4.2	12.6	8.8	286	4.6	8.2	4.7	184	158	128	30	LVH	Y	190.4	134	Y	Mod	4.2	1.4	1.2	N
31	Anjanna Reddy	56 years	Male	82	2.6	22.3	8.9	184	4.6	7	4.6	158	156	110	35	LVH	Y	297.4	185.8	Y	Mild	5	1.4	1.4	N
32	Radha Krishna	60 years	Male	46	1.8	46.6	11.2	84	6	10.8	6	313	154	144	40	LVH	Y	334.5	202.7	Y	Mild	4.8	2.1	1.1	N
33	Noorjan	46 years	Female	155	2.7	18.7	8.9	238	4.6	10.2	4.9	191	174	128	26	LVH	Y	136.1	101.1	Y	Mild	4	1	1.1	N
34	Ajjappa	60 years	Male	58	2.7	20.5	8.8	142	5	11	4.2	240	176	126	30	LVH	Y	235.4	144	Y	Mild	5.1	1.3	1.4	N
35	Nalakappa	52 years	Male	110	2.7	20.5	9	142	5	11.2	4.2	200	176	126	32	LVH	Y	235.4	144	Y	Mild	4.5	1.4	1.3	N
36	Mohammed Ajaz	59 years	Male	103	2.1	21.9	8.9	318	4.2	8.3	4.1	198	148	120	42	LVH	Y	208.9	139.2	Y	Mild	4.7	1.2	1.3	N
37	Babu	44 years	Male	146	7.2	11.14	8.7	185	4	9.8	4.7	188	166	112	42	NO LVH	N	170.2	99.5	N	S	4	1.3	1.2	Y
38	Chikka Venkatarayappa	58 years	Male	111	3.7	17.4	8.1	419	4	9	5.7	180	166	116	28	NO LVH	N	127	78.3	N	Mod	4	1.1	1	N
39	Chowda Reddy	41 years	Male	140	5.3	12.3	8.3	148	5.2	8.9	5.3	180	166	118	42	NO LVH	N	123.2	90.5	N	Mod	4.6	1	1	N
40	Ragavendra Rao	42 years	Male	53	3.7	17.4	8.1	419	6	9	5.7	144	178	98	48	NO LVH	N	127	78.3	N	Mod	4	1	1	N
41	Muniyappa	58 years	Male	102	4.5	14.4	7	243	3.7	8.8	4.8	176	152	110	48	NO LVH	N	151.2	99.4	N	Mod	4	1.1	1	N
42	Deva Reddy	59 years	Male	122	5.6	13.7	7.5	192	3.5	8.9	4.3	164	156	122	30	NO LVH	N	241	122	N	Mod	5.1	1.2	1.2	N
43	Bhavani	47 years	Female	65	3.2	17.9	8.9	160	6	7	4.9	186	160	128	38	NO LVH	N	136.1	75.1	N	Mod	4	1.1	1	N
44	Gowri	59 years	Female	104	3.2	17.2	8	110	3.8	10.1	5.6	190	158	122	44	NO LVH	N	158.7	97.9	N	Mod	4.6	1	1	N
45	Faruq Ulla	42 years	Male	71	4.5	14.4	7	243	3.7	6.9	4.8	176	172	110	30	NO LVH	N	151.2	99.4	N	Mod	4.1	1.1	1.1	N
46	Sureshappa	44 years	Male	53	3.7	17.4	8.1	419	6	9	5.7	144	178	98	48	NO LVH	N	127	78.3	N	Mod	4	1	1	N
47	Anandappa	49 years	Male	99	2	44.2	9	89	4	9.8	6	148	198	130	46	NO LVH	N	150	90	N	Mild	3.8	1	1.1	N
48	Padmamma	51 years	Female	110	2.2	22.8	9	190	3.6	9	6	170	178	98	42	NO LVH	N	151.2	88.9	N	Mild	4.1	1.1	1.1	N
49	Jairam	49 years	Male	102	2.8	19.9	7	102	3.9	8.9	4.9	146	176	110	46	NO LVH	N	125.8	79.6	N	Mild	3.8	1	1.1	N
50	Marigowda	54 years	Male	108	2.4	21	8.6	236	3.3	7.8	4.8	148	168	128	36	NO LVH	N	127	78.2	N	Mild	3.7	1.1	1	N