Bulletin of Environment, Pharmacology and Life Sciences
Bull. Env. Pharmacol. Life Sci., Vol 11 [6] May2022 : 160-171

Online ISSN 2277-1808
Journal's URL:http://www.bepls.com
CODEN: BEPLAD

ORIGINAL ARTICLE **OPEN ACCESS**

Clinical Study of Cardiac Changes in End Stage Renal Disease with Reference to Electrocardiography, Chest X-Ray and 2D Echocardiography

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ABSTRACT
Chronic renal failure is a condition in which the kidney is no longer able to maintain biochemical equilibrium due to the progressive and permanent death of nephrons, regardless of the origin. Patients with chronic kidney disease have a high prevalence of cardiovascular disease (CVD). Dysrhythmias, cardiac arrest, and sudden cardiac death (SCD) are more common in patients on maintenance dialysis.This study was conducted to identify the prevalence of cardiac abnormalities in end stage renal disease (ESRD). The present Prospective cross sectional hospital based observational study was conducted 100 patients with ESRD. A detailed clinical history was taken from all the patients regarding symptoms of ESRD. The data was collected and entered into a spread sheet application (Microsoft9 Excel 2010). Mean age of the study population was 51.88 years. Hypertension (37%) was found to be the major etiology in the study population followed by diabetes mellitus (21%). Major chest x-ray finding observed was Pleural Effusion (22%) followed by Cardiomegaly (11%) and Cardiomegaly along with Pleural Effusion (4%). Major ECG changes notices were Left ventricular hypertrophy (24%) followed by Ischemic changes (22%) and Prolonged QT interval (4%). Regional wall motion abnormality was observed in 9%, pericardial effusion found in 13%. Patients with End Stage Renal Disease are at significantly increased risk for cardiovascular disease. Left ventricular dysfunction is commonest cardiovascular abnormality detected. Echocardiography is a more sensitive diagnostic procedure to detect left ventricular dysfunction.
Keywords:*Cardiac disease, end-stage renal disease, hypertension, haemodialysis, diabetes mellitus*

Received 11.04.2022 Revised 16.05.2022 Accepted 30.05.2022

INTRODUCTION

The Kidney Disease Outcomes Quality Initiative (KDOQI) Clinical Practice Guidelines on Chronic Kidney Disease: Evaluation, Classification, and Stratification of Risk published by the National Kidney Foundation in 2002 were the first to provide a definition of CKD that was independent of cause, as well as a severity classification based on GFR level[1].

Criteria for defining chronic kidney disease (CKD) include:

1.Kidney damage defined as structural or functional abnormalities of the kidney, with or without decreased GFR, manifested by either pathological abnormalities or markers of kidney damage, such as abnormalities in the composition of the blood or urine, or abnormalities in imaging tests, lasting for more than 3 months.

2. With or without renal impairment, GFR <60ml/min/l.73 m^2 for more than 3 months.

Regardless of diagnosis, the existence of kidney damage and degree of kidney function (glomerular filtration rate) should be used to determine the presence of chronic kidney disease[2].

Patients with CKD should be classified into stages based on their renal function, regardless of their diagnosis according to the KDOQI-CKD classification.

The best indicator of overall kidney function is the glomerular filtration rate. The typical GFR varies depending on age, gender, and body size. GFR in young adults ranges from 120 to 130 ml/min per 1.73 m2, and it decreases with age. A GFR of less than 60 ml/min per 1.73 m2 indicates that half or more of an adult's normal renal function has been lost. Below this level, the prevalence of complications of chronic kidney disease increases.

Kidney failure is defined as either

1. A level of GFR to <15 ml/min/1.73m2 which is accompanied in most cases by signs and symptoms of uremia, (OR)
2. A need for initiation of renal replacement therapy (dialysis or transplantation).

Chronic renal failure is a condition in which the kidney is no longer able to maintain biochemical equilibrium due to the progressive and permanent death of nephrons, regardless of the origin. Biochemical alterations and clinical symptoms are variable and usually non-specific in this illness. Any damaging and progressive illness affecting both kidneys might lead to chronic renal failure. It indicates that both the glomerular and tubular functions have failed. By convention, acquired tubular anomalies or isolated congenital defects aren't taken into account when determining the presence of chronic renal failure.The kidney has a high reserve capacity. Before renal failure develops, about 80% of renal function will be lost. Before the serum creatinine concentration rises above the upper limit of normal, GFR must decline to about half of its normal level. The organization Kidney Disease Improving Global Outcomes(KDIGO) recently recommended retaining the KDOQI definition of CKD, integrating clinical diagnosis and albuminuria stages to the classification, as well as GFR stages, and using these stages to establish risk categories.

Although the kidneys account for only around 1% of total body weight, they account for roughly 20% of resting cardiac output, which is essential for ultrafiltration. The volume of urine produced is the result of two massive, directionally opposing processes: ultrafiltration of more than 180L/day of plasma water and reabsorption of more than 99 percent of this filtrate in the renal tubules through transport[3].

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
2. A set of progressive mechanisms, involving hyper filtration and hypertrophy of the remaining viable nephrons, that are a common consequence following long term reduction of renal mass, irrespective of underlying etiology[4].

Vasoactive hormones, cytokines, and growth factors are involved in the responses to a decrease in nephron number. The increased pressure and flow predispose to sclerosis and dropout of the remaining nephrons, so these short-term adaptations of hypertrophy and hyperfiltration eventually become maladaptive. Increased renin-angiotensin axis intrarenal activity appears to contribute to both early adaptive hyperfiltration and subsequent maladaptive hypertrophy and sclerosis, the latter due in part to transforming growth factor stimulation (TGF). This process explains why a drop in renal mass caused by a single insult can result in a long-term decline in renal function[5].Among patients with end-stage renal disease (ESRD), cardiovascular disease has long been recognized as the leading cause of death. In a study conducted by Sarnak and Levey, when dialysis patients and the general population's cardiovascular mortality rates were compared, the death rates ranged from about a 120-fold difference between patients 25 to 34 years old, to a 15-fold difference between patients 55 to 64 years old, and as much as a 3-fold difference in patients over 85 years old.CVD mortality rates in the general population (2 million deaths) were compared to CVD mortality rates in dialysis patients as part of the National Kidney Foundation Task Force study on CVD (50,000 deaths). These findings revealed that annual CVD mortality rates are much higher in dialysis patients, regardless of gender, age group or race. When compared to the general population, younger dialysis patients had a 500-fold increase in CVD mortality rate, and rates remain approximately five times higher even among the oldest patients. Dialysis patients had a higher prevalence of clinical ischemic heart disease and congestive heart failure than the general population, according to several studies.

Furthermore, among dialysis patients, the percentage of individuals having left ventricular hypertrophy (LVH) is as high as 75%. Cardiovascular illness is a complication of chronic renal disease. It demands special attention because

1) In people with chronic renal disease, cardiovascular events are more common than renal failure.
2) Chronic renal illness appears to be a CVD risk factor and
3) CVD is treatable and may be avoided in patients with chronic renal disease.

Patients with chronic kidney disease should be considered to be at "highest risk" for subsequent CVD events, according to the NKF Task Force on Cardiovascular Disease in Chronic Renal Disease's 1998 report, and the majority of interventions that are effective in the general population should also be applied to patients with chronic kidney disease[6].

Patients with chronic kidney disease have a high prevalence of cardiovascular disease. According to the NEORICA (New Opportunities for Early Renal Interventions through Computerized Assessment) study, patients with CKD stages 3-4 who have impaired renal function but are not yet in ESRD have a 25% chance of developing ischemic heart disease, more than double the risk for patients without CKD. Left

ventricular hypertrophy (LVH) occurs in 27% of patients with chronic kidney disease (CKD) who have a creatinine clearance of greater than 50 ml/min, 31% of patients with a creatinine clearance of 25-50 ml/min, and 45% of patients with a creatinine clearance of less than 25 ml/min. The prevalence of severe systolic dysfunction (left ventricular ejection fraction of 25%), ventricular dilatation, and LVH is 15, 32, and 74 percent, respectively, in patients requiring chronic dialysis.

Cerebrovascular disease, peripheral vascular disease, congestive heart failure, and coronary vascular disease all occur with a prevalence of 13, 22, 36, and 39% in patients with ESRD. As previously noted, the incidence of CVD in ESRD patients is also very high. Patients starting dialysis who are 25 years or younger have a death rate (typically owing to CVD) that is comparable to that of 75-80-year-old individuals in the general population. The strong link between CKD and CVD makes determining the role of CKD as a CVD risk factor difficult. The hazard ratios for cardiovascular events was 1.4 in patients with eGFR > 60 ml/min, 2.0 in those with eGFR 45-59 ml/min, 2.8 in those with eGFR 0 - 44 ml/min, and 3,4 in those with eGFR 15-29 ml/min, respectively. Additionally, the prevalence of recurrent cardiovascular events was 20.4 percent (patients in stage 2 CKD) and 28.4 percent (patients in stage 3 CKD) among patients with established CVD and decreased renal function but not in ESRD (patients in stage 3 - 4 CKD).

A number of factors may alter cardiovascular dynamics in renal failure, including anaemia, hypertension,volume overload, and electrolyte imbalance, edema, and arteriovenous fistulas. In chronic uremia, cardiomyopathy manifests as systolic function, concentric LVH,or LV dilatation[7].

Dysrhythmias, cardiac arrest, and SCD are more common in patients on maintenance dialysis. Due to heightened dysrhythmogenicity caused by dynamic changes in electrolytes, volume status, blood pressure, and medication use, dialysis patients with underlying structural or functional CVD are substantially more prone to these dysrhythmias and cardiac arrest. Even patients not on dialysis have a much greater rate of cardiac events and a worse event-free survival when compared to the general population[8].

Ischemic heart disease is present in many patients even when dialysis is started. Dysrhythmias and SCD are more common in CKD Stage 5 patients who have either symptomatic or asymptomatic coronary artery disease. This risk is heightened by the presence of anaemia and left ventricular hypertrophy, or an increased left ventricular mass index, both of which are common in CKD patients at the start of dialysis therapy.

The prevalence of CAD is associated with the development of dysrhythmias and silent myocardial ischemia, and the length of dialysis is directly proportional to the prevalence of baseline ECG abnormalities, new dysrhythmias, and silent myocardial ischemia. There have been reports of potentially life-threatening ventricular dysrhythmias and asymptomatic myocardial ischemia.

Increased arrhythmogenicity is caused by myocardial compromise (due to underlying coronary artery disease, decreased coronary reserve blood flow, or the effects of uremia on myocardial function and structure), prolonged QTc interval or dispersion, electrolyte abnormalities, intradialytic hypotension, concomitant presence of LVH (present in the majority of dialysis patients), and autonomic dysfunction (with or without diabetes)[3].

Electrolyte abnormalities, such as fluctuating potassium, ionised calcium, magnesium, and other divalent ions, are common in dialysis patients. Patients on HD exhibit substantial changes in volume status, potassium, and bicarbonate levels in between dialysis treatments due to the intermittent nature of the dialysis method.

The level of potassium and calcium in the dialysate fluid used during the previous treatment session, as well as considerable variation in eating habits due to variable adherence to dietary adjustments necessary to manage the calcium-phosphate product, contribute to these fluctuations. All of these elements come together to create a dysrhythmogenic diathesis.

The use of echocardiography to assess left and right ventricular function is well-established. In ESRD patients, LV diastolic dysfunction (LVDD) is a major cause of cardiac morbidity. Diastolic dysfunction appears to be the first symptom of LV dysfunction, and it may even occur before LVH.

The echocardiogram allows for the assessment of ventricular mass and volume, as well as the discovery of hypertrophy, the determination of its geometric pattern (concentric or eccentric), and the quantification of systolic function with high precision. Furthermore, Doppler-derived approaches can provide information on ventricular relaxation and filling dynamics, as well as the existence of anomalies in the heart valves and pericardium[9].

In patients on HD, left ventricular systolic dysfunction (LVSD) is a powerful indication of a poor prognosis. Diastolic dysfunction is defined by changes in ventricular relaxation and compliance, which are often followed by a compensatory rise in filling pressure in later stages. Regardless of the origin, the latter occurrence is generally to blame for the symptoms of heart failure. A prevalence of LVDD in CKD patients ranging from 50 to 60% has been found in a few studies[10].

The ECG and different kinds of echocardiography (two-dimensional, stress echocardiography) findings should provide the most important details among the diagnostic procedures. Determination of cardiac disease in patients with ESRD can provide guidelines for treatment. Researchers from all around the world have worked hard to identify the underlying pathology and assist persistently morbid patients with renal failure. Understanding the mechanism of cardiovascular illnesses in chronic renal disease allows for early detection, prevention, and intervention to manage complications. Hence, the current study was carried out with the aim of determining the prevalence of cardiac abnormalities in end-stage renal disease with reference to electrocardiography, chest x-ray, and 2D echocardiography.

MATERIAL AND METHODS

SourceofData:The present Prospective crosssectional hospital based observational study was conducted 100 patients with ESRD from August 2019 to September 2021.

The study was conducted after taking permission from institutional ethics committee. Patients with a diagnosis of ESRD were taken up for the study. A detailed clinical history was taken from all the patients regarding symptoms of ESRD. Patients were examined for signs and symptoms of ESRD.

Investigations:

The patients were subjected for detailed investigations; Complete blood count, Renal function test/Serum electrolytes, Liver function test, Urine R/M, Electrocardiography, Chest X-Ray PA view, USG Abdomen, 2D echocardiography, Fasting Lipid Profile, Glycosylated haemoglobin (HbA1c).

Study Type

- Descriptive and cross-sectional study.

InclusionCriteria

- All patients previously diagnosed as CKD
- Patients with ESRD stage 5(GFR less than 15ml/min)
- Patients on hemodialysis

Exclusion Criteria

- Documented ischemic heart disease
- Congenital heart disease
- Valvular heart disease
- Primary cardiomyopathies
- Age less than 18 years.

Data Analysis

The data was collected and entered into a spread sheet application (Microsoft Excel 2010) prior to being exported to the SPSS version 20 data editor page (SPSS Inc., Chicago, Illinois, USA).

Percentages, means, and standard deviations were computed as part of descriptive statistics. Independent sample t-test and chi-square test were used as statistical tests in this study. The confidence interval and p-value were set at 95% and 5%, respectively.

RESULTS

This study includes 100 subjects with CKD from the Department of General Medicine and Department of Nephrology at our institute from August 2019 to September 2021.

Table 1: Comparison of age and gender distribution of the study population

R4	Sex		Total
	Female	Male	
<307	2	6	8
	6.7%	8.6%	8.0%
31-40	3	4	7
	10.0%	5.7%	7.0%
41-50	5	14	19
	16.7%	20.0%	19.0%
51-60	13	25	38
	43.3%	35.7%	38.0%
61-70	7	19	26
	23.3%	27.1%	26.0%
71-80	0	2	2
	.0%	2.9%	2.0%
Total	30	70	100
	100.0%	100.0%	100.0%
Mean±SD	51.90±11.0	51.87±12.7	0.992 (NS)**
p-value	0.840 (NS)*		

Test applied: Chi-square test*, Independent sample t-test**

Mean age of the study population was 51.88 years. Majority (38%) of the patients belongs to 51-60 years. Mean age of the female patients was 51.90 years and male 51.87 years (p=0.992). Majority of the patients were male (70%) and female (30%).

Table 2: Distribution of the study population according to etiology

Etiology	Frequency	Percent
Hypertension	37	37.0
Diabetes Mellitus	21	21.0
Unknown	12	12.0
Diabetes Mellitus / Hypertension	11	11.0
Chronic Glomerulonephritis	7	7.0
Polycystic kidney disease	7	7.0
Obstructive uropathy	5	5.0
Total	100	100.0

Hypertension (37%) was found to be the major etiology in the study population followed by diabetes mellitus (21%), unknown cause (12%), Diabetes with hypertension reported in (11%), Chronic Glomerulonephritis (7%), polycystic kidney disease (7%) and Obstructive uropathy (5%).

Table 3: Signs suggesting cardiovascular involvement in the study population

Signs	Frequency	Percent
Pallor	100	100.0
Edema	70	70.0
Raised JVP	28	28.0
Total	100	100.0

All the 100 patients reported pallor as a major sign followed by edema (70%) and Raised JVP (28%).

Table 4: Distribution of Haemoglobin levels in the study population

C	Frequency	Percent
4-6	6	6.0
6-8	24	24.0
8-10	57	57.0
10-12	13	13.0
Total	100	100.0
Mean±SD	8.54±1.5	

Mean Hb levels in the present study was 8.54 gm%. Majority of the study subjects reported Hb levels in the range 8-10 gm% (57%) followed by 6-8 gm% (24%), 10-12 gm% (13%) and 4-6 gm% (6%).

Table 5: Distribution of Urea levels in the study population

Urea (mg/dl)	Frequency	Percent
100-150	48	48.0
150-200	52	52.0
Total	100	100.0
Mean±SD	153.70±21.20	

Overall mean value of urea levels was 153.70 mg/dl. 52% of the patients reported urea levels in the range of 15-200 mg/dl and rest 48% showed urea levels in the range of 100-150 mg/dl.

Table 6: Distribution of Serum Creatinine levels in the study population

Serum Creatinine (mg/dl)	Frequency	Percent
5-10	64	64.0
10-15	30	30.0
15-20	6	6.0
Total	100	100.0
Mean±SD	9.35±2.94	

Overall mean value of serum creatinine levels was 9.35 mg/dl. 64% reported 5-10 mg/dl followed by 10-15 mg/dl (30%) and rest (6%) reported 15-20 mg/dl levels of serum creatinine.

Table 7: Distribution of Random Blood Sugar levels in the study population

Random Blood Sugar (mg/dl)	Frequency	Percent
<100	13	13.0
101-200	72	72.0
201-300	10	10.0
>300	5	5.0
Total	100	100.0
Mean±SD	154.18±59.41	

Mean RBS in the present study was 154.18 mg/dl. Majority (72%) had blood sugar levels 101-200 mg/dl. and 15% had blood sugar levels above 200 mg/dl. 13% reported blood sugar levels below 100%

Table 8: Distribution of Serum Total Cholesterollevels in the study population

Serum Total Cholesterol (mg/dl)	Frequency	Percent
150-200	70	70.0
201-250	28	28.0
>250	2	2.0
Total	100	100.0
Mean±SD	199.67±17.89	

Mean value of serum total cholesterol shown by the study population was 199.67 mg/dl. Majority (70%) showed levels between 150-200 mg/dl followed by 201-250 mg/dl (28%) and 2% reported levels above 250 mg/dl.

Table 9: X-ray chest findings in the study population

X-ray chest findings	Frequency	Percent
NIL	63	63.0
Pleural Effusion	22	22.0
Cardiomegaly	11	11.0
Cardiomegaly, Pleural Effusion	4	4.0
Total	100	100.0

Major chest x-ray finding observed was Pleural Effusion (22%) followed by Cardiomegaly (11%) and Cardiomegaly along with Pleural Effusion (4%)

Table 10: Electrocardiographic (ECG) findings in the study population

Electrocardiographic findings	Frequency	Percent
Left ventricular hypertrophy	24	24.0
Ischemic changes	22	22.0
Prolonged QT interval	4	4.0

Major ECG changes notices were Left ventricular hypertrophy (24%) followed by Ischemic changes (22%) and Prolonged QT interval (4%)

Table 11: Echocardiographic parameters and findings in the study population

Echocardiographic parameters	N	Mean	Std. Deviation
Left ventricular internal diameter in diastole (LVIDd) (mm)	100	45.62	6.228
Left ventricular internal diameter in systole (LVIDd) (mm)	100	29.89	6.246
Interventricular septum diameter in systole (IVS) (mm)	100	12.21	1.754
Left ventricular posterior wall diameter (LVPWD) (mm)	100	12.12	1.754
Left atrium diameter (mm)	100	33.07	4.281
Ejection fraction (EF) (%)	100	54.79	9.979
Echocardiographic findings	Frequency		Percent
Regional wall motion abnormality	9		9.0
Pericardial effusion	13		13.0
Mild MR	7		7.0
Sclerotic AV	7		7.0
Total	100		100.0

The mean value of the left ventricular internal diameter in diastole (LVIDd) (mm) is 45.62 mm; the left ventricular internal diameter in systole (LVIDs) (mm) is 29.89 mm, the interventricular septum diameter in systole (IVS) (mm) is 12.21 mm, the left ventricular posterior wall diameter (LVPWD) (mm) is 12.12 mm and the left atrial diameter (mm) is 33.07 mm.
Regional wall motion abnormality observed in 9%, pericardial effusion was found in 13%, Mild MR and Sclerotic AV observed in 7% each.

Table 12: Correlation of lipid profile with echocardiography in the study population

Echocardiographic finding	Lipid Profile		Total	Chi-square value	p-value
	TC<200 (N=70)	TC>200 (N=30)			
Left ventricular hypertrophy	8	16	24	20.217	0.001* (Sig.)
	11.4%	53.3%	24.0%		
Ischemic changes	5	17	22	30.01	0.001* (Sig.)
	7.1%	56.7%	22.0%		
Prolonged QT interval	1	3	4	4.08	0.045* (Sig.)
	1.4%	10.0%	4.0%		

Test applied: Chi-square test
Out of total 24 patients with LVH 8 patients showed lipid profile (TC<200) and rest 16 patients showed lipid profile (TC>200). On chi-square analysis statistically significant correlation exist between LVD and Lipid profile (p=0.001). Similarly, out of 22 patients with Ischemic changes 17 showed lipid profile (TC>200). On chi-square analysis statistically significant correlation exist between Ischemic changes and deranged lipid profile (p=0.001). Prolonged QT interval also showed statistically significant correlation with deranged lipid profile (p=0.045).

DISCUSSION

In the present study population, the mean age was 51.88 years. Majority (38%) of the patients belongs to 51-60 years of followed by 61-70 years (26%), 41-50 years (19%). Least number (2%) of patients was reported in the 71-80 years age group. Mean age of the female patients was 51.90 years and male were 51.87 years (p=0.992). Majority of the patients were male (70%) and rest were female (30%). Majority of them were reported in the age group of 51-60 years.
ESRD was observed in a notable majority in males and a dramatic increase in prevalence with age in both genders, similar to the findings of a study undertaken by P. Jungers *et al* (1996) to evaluate the age- and gender-related incidence of chronic renal failure in a French urban region [11].
In another study conducted by Hida M et al. (1985) where they determined the age and sex distribution in chronic renal failure patients at dialysis, they found 50-59 year-old age group had the most cases and most were male [12].
Hypertension (37%) was found to be the major etiology in the study population followed by diabetes mellitus (21%), unknown cause (12%), Diabetes with hypertension reported in (11%), Chronic Glomerulonephritis (7%), polycystic kidney disease (7%) and Obstructive uropathy (5%).
Agarwal SK (2000) observed that chronic glomerulonephritis was observed in 49.4% cases of CKD followed by diabetic nephropathy in 28.4% cases[13]. Coresh J *et al* (2007) in cross-sectional analysis of the most recent National Health and Nutrition Examination Surveys in United States had found that the most common cause of chronic kidney disease was diabetes mellitus followed by hypertension[14].
In our study leading cause of ESRD was hypertension; this may be because of clustering of patients and small sample size. Unknown cause of ESRD may be due to recurrent UTI or anatomical disturbances since childhood like vesicoureteric reflux in female patients.
In the present investigation all the 100 patients reported pallor as a major sign of cardiovascular involvement followed by edema (70%) and raised JVP (28%). Foley RN *et al* (1995) studied clinical manifestations of cardiovascular disease in ESRD. They found 19% angina pectoris, 31% cardiac failure and 7% dysrhythmia. NP Singh *et al* (1999) noticed clinical manifestation in CRF patients: pallor (100%), edema (80%), HTN (82%), pericarditis (15%), and pleural effusion (7%)[15].
In our study 100% of patients had decreased haemoglobin level. Mean Hb level was 8.54 gm%. Majority of the study patients reported Hb levels in the range 8-10 gm% (57%) followed by 6-8 gm% (24%), 10-12 gm% (13%) and 4-6 gm% (6%).
Anemia is a risk factor for cardiac problems on its own. The risk of heart failure rises by 25%, the risk of echocardiographically demonstrable left ventricular hypertrophy rises by 42%, and the risk of death rises by 14% for every 1 g/dl decline in mean haemoglobin[16].

Our results found in agreement with the finding of the studies conducted by DS Chafekar *et al* (1994) who found mean haemoglobin percentage was 7.84 ± 0.98 in ESRD patients. Foley RN *et al* (1995) had found mean haemoglobin percentage 8.4 ±1.7 in ESRD patients. Similar results were observed in a study by Singh NP et at (2000).

Overall mean value of urea levels in the present study was 152.70 mg/dl which is comparable with the study by Foley RN et al. (1995) who observed a mean urea of 117±15.3 and Singh NP et al. (2000) who observed a mean urea of 121.2±30.6. Contrary to the findings observed by Chafekar DS et al. where the mean blood urea level was 77.07±25.39mg/dl[17]. Discrepancy in serum blood urea between their study and our study may be due to small sample size or an exogenous source of urea.

In the present study mean value of serum creatinine levels was 9.35 mg/dl found in agreement with the study conducted by Kale SA *et al* (2001) who observed a mean serum creatinine level of 9.59mg/dl in ESRD patients[18]. Other studies reported mean value less than the present study this might be because of the difference in the sample size and geographic location.

Mean value of serum total cholesterol shown by the study population was 199.67 mg/dl. Serum total triglyceride shown by the study population was 185.9 mg/dl. Serum HDL shown by the study population was 42.42 mg/dl. Mean value of serum LDL shown by the study population was 171.07 mg/dl.

Beman-ali *et al* (2000) observed mean cholesterol levels of 204±41mg/dl; mean triglyceride levels of 196±125mg/dl; mean HDL levels of 30±7mg/dl; mean LDL levels of 117+36mg/dl in ESRD patients[19]. Habib AN *et al* (2006) observed mean cholesterol levels of 208±59mg/dl and mean triglyceride levels of 212±163mg/dl [20]. Hsu CY *et al.* (2009) observed mean cholesterol levels of 226.5±45.8mg/dl [21].Brotman DJ *et al* [22] in a study observed a mean cholesterol level of 218±45mg/dl; mean triglyceride levels of 188±59mg/dl; mean HDL levels of 47±17mg/dl; mean LDL levels of 141±53mg/dl.

In this study X-ray findings showed Pleural Effusion (22%) followed by Cardiomegaly (11%) and Cardiomegaly along with Pleural Effusion (4%). Our findings were found in line with studies conducted by Roy *et al* (1994)[23]who observed similar results and Banerjee D *et al*. (2007) who observed fluid overload in 20.2% and pulmonary edema in 21.3%, CHF was in 12.5% of hemodialysis patients[24].

Present study on ECG demonstrated Left ventricular hypertrophy (24%) followed by Ischemic changes (22%) and Prolonged QT interval (4%). Roy *et al* (1994) found LVH in 50% and 26% had ischemic changes[25].Parfrey PS *et al* (1996) observed left ventricular hypertrophy in 39% of patients.Watabe D et al. (2006) found left ventricular hypertrophy in 21% of patients[26]. Variation in ECG findings may be due to small sample size.

Echocardiographic parameters in our study showed mean value of left ventricular internal diameter in diastole (LVIDd) (mm) of 45.62mm, left ventricular internal diameter in systole (LVIDd) (mm) of 29.89 mm, Interventricular septum diameter in systole (IVS) (mm) of 12.21 mm, Left ventricular posterior wall diameter (LVPWD) (mm) of 12.12mm, Left atrium diameter (mm) of 33.07 mm and Ejection fraction (EF) (%) of 54.79%. Regional wall motion abnormality observed in 9%, pericardial effusion found in 13%, Mild MR and Sclerotic AV observed in 7% each.

Foley RN *et al* (1995) discovered that on baseline echocardiography, anomalies of left ventricular structure and function were quite common in ESRD patients: 73.9 percent had hypertrophy in the left ventricle, 35.5 percent had dilatation in the left ventricle, and 14.8 percent had systolic dysfunction.

Menon AS *et al*. (1998) observed diastolic dysfunction in 76%, LVH in 40%, systolic dysfunction 4%, and pericardial effusion in 4% of CRF patients. Singh NP *et al* (2000) observed LVH in 76.92%, diastolic dysfunction in 72% and no systolic dysfunction in CRF patients. Agarwal S *et al* (2003) had observed that 15 percent of patients had systolic dysfunction, whereas 60 percent had diastolic dysfunction.

Zoccali C et al. (2000) discovered that in hemodialysis patients, 77 percent had LVH, 22 percent had systolic dysfunction by LVEF assessment and 48 percent had systolic dysfunction by midwall fractional shortening[27].Mallamaci *et al*. (2001) had found 79% LVH 13% systolic dysfunction[28].Joki N *et al.* (2003) had found 27% had systolic dysfunction that predicted cardiac death in hemodialysis patients[25].

In the present study out of total 24 patients with LVH; 8 patients showed lipid profile (TC<200) and rest 16 patients showed lipid profile (TC>200). On chi-square analysis statistically significant correlation exist between LVD and Lipid profile (p=0.001). Similarly out 22 patients with Ischemic changes 17 showed lipid profile (TC>200). On chi-square analysis statistically significant correlation exist between Ischemic changes and Lipid profile (p=0.001). Prolonged QT interval also showed statistically significant correlation with Lipid profile (p=0.045). Shah BV *et al* had suggested significant hypertriglyceridemia does develop in a majority of CRF patients on conservative line of treatment[29].

CONCLUSION

The current study came to the conclusion that patients with End Stage Renal Disease are at significantly increased risk for cardiovascular disease. Left ventricular dysfunction is commonest cardiovascular abnormality detected. Statistically significant correlation exists between LVD, Ischemic changes, Prolonged QT interval and Lipid profile. Echocardiography is a more sensitive diagnostic procedure to detect left ventricular dysfunction. To decrease morbidity and mortality early attention should be given to all risk factors.

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CITATION OF THIS ARTICLE

P Diggikar, C N Hareshkumar, P Gopal and N. S. S. Saranya. Clinical Study of Cardiac Changes in End Stage Renal Disease with Reference to Electrocardiography, Chest X-Ray and 2D Echocardiography. Bull. Env. Pharmacol. Life Sci., Vol 11[6]May 2022: 161-170