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Research Article

STUDY ON CARDIAC MANIFESTATIONS IN CKD PATIENTS IN A TEACHING HOSPITAL

VINAYAK INKALAGI[1]*, PRIYANKA KATTI B[2]

[1]Department of General Medicine, Al Ameen Medical College, Bijapur, Karnataka, India. [2]Department of Obstetrics and Gynaecology, Belgaum Institute of Medical Sciences, Belagavi, Karnataka, India.
***Corresponding author: Dr. Vinayak Inkalagi; Email: drvinayakinkalagi22@gmail.com**

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ABSTRACT

Objective: The aim of the study was to study cardiac manifestations in CKD.

Methods: The study involved 140 instances of CKD that were admitted to the general medicine department of Al Ameen Medical College in Bijapur, Karnataka. Patients who were known CKD patients, who experienced symptoms for at least 3 months with serum creatinine more than 3 mg% and creatinine clearance <30 mL/min and individuals with Type 2 or type 3 parenchymal alterations and bilaterally constricted kidneys on abdominal ultrasonography with inadequate corticomedullary differentiation. Although they did not develop kidney damage as a result of the underlying condition, patients with autosomal dominant polycystic kidney disease (ADPKD) and obstructive neuropathy were also included in study and cardiovascular assessment is done in these patients. Parameter noted were chambers size, elative wall thickness, systolic function, diastolic function, left ventricular wall motion abnormalities , pericardial effusion and valvular abnormalities

Results: According to our study, there were 31.4% (44/140) instances of left ventricular hypertrophy (LVH), 13.5% (19/140) of LAD, 18.1% (20/140) of conduction abnormalities, 15.7% (22/140) of ischemia, 2.1% (3/140) of arrhythmias, 2.8% (4/140) of paroxysmal defects, and 21.4% (30/140) of normal cases. In 2D echo results showed LVH in 55.7% (78/140) of patients, and EF<35% in 78.9% (110/140) and >35% in 21.4% (30/140) of cases. 7.1% (10/140) had pericardial effusion; 40% (56/140) had diastolic dysfunction :18.5%(26/140) had systolic dysfuntion, and 14.2% (20/140) had dialted left atrium.

Conclusion: Echocardiography is a non-invasive, secure, user-friendly, and precise method for evaluating heart function in patients with chronic renal disease. When it comes to identifying LVH, echocardiography is more accurate. When it comes to CKD patients, cardiovascular problems are the main cause of morbidity and death.

Keywords: Chronic kidney disease, Echocardiography, Left ventricular hypertrophy.

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INTRODUCTION

Chronic kidney disease (CKD) is a worldwide issue that affects both industrialized and developing nations' populations equally. With about 40% of hospital admissions and nearly 50% of deaths among CKD patients, cardiovascular disease (CVD) is by far the most common cause of morbidity and mortality in these individuals [1,2].

It has been discovered that well-known, "traditional" atherosclerotic risk factors — such as diabetes, hypertension (HTN), dyslipidemia, and advanced age — are independent indicators of CVD in people with CKD. Furthermore, some characteristics of renal insufficiency, such as hemodynamic and metabolic variables including volume overload, anemia, calcium and phosphorus imbalance, chronic infection, and a hypercoagulable environment, may raise the risk and cause CVD [3].

About 10% of people are thought to be afflicted with CKD, a systemic illness; yet, studies revealed that CKD prevalence varies between nations [4]. Due to the aging of the global population and the rise in the incidence of diabetes mellitus (DM), which is now the main cause of CKD, the prevalence of CKD has significantly grown during the past several decades. These days, CKD is regarded as a public health issue because of its link to CVDs, which raises mortality rates in the community [5]. As a result, CKD is a substantial risk factor for CVDs and is common in people with CVDs. In addition, those with CKD have a higher chance of cardiovascular events or mortality compared to the advancement of end-stage renal disease [6].

The idea that individuals with renal illness experience accelerated aging is supported by several researches [7]. This accelerates the emergence of diseases, such as CVDs, which are often linked to advanced age.

The classification and diagnosis of CKD have changed over time. However, according to current international recommendations, CKD is defined as reduced kidney function, as demonstrated by a glomerular filtration rate of <60 mL/min per 1.73 m^2 or by kidney damage indicators, or both, for at least three months, independent of the underlying cause. When circulatory abnormalities in CKD are identified early, morbidity and mortality can be decreased, and quality of life can be raised [8]. Our aim is to study cardiac manifestations in CKD.

METHODS

The study was conducted in patients with CKD admitted in the Department of General Medicine, Al Ameen Medical College, Bijapur, Karnataka.

Inclusion criteria

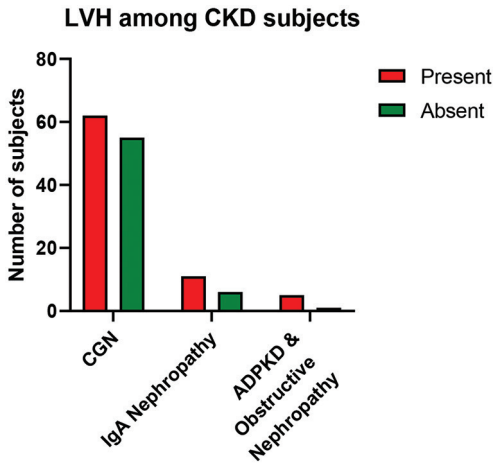
The following criteria were included in the study:

1. Patients who were known CKD patients
2. Those who experienced symptoms for at least 3 months
3. Patients with serum creatinine more than 3 mg% and creatinine clearance <30 mL/min
4. Individuals with Type 2 or type 3 parenchymal alterations and bilaterally constricted kidneys on abdominal ultrasonography with inadequate corticomedullary differentiation. Although they did not develop kidney damage as a result of the underlying condition, patients with autosomal dominant polycystic kidney disease

Table 1: Correlation of CKD diagnosis with LVH findings

CKD diagnosis	LVH		p-value
	No. of cases		
	Present	Absent	
CGN	62	55	0.4402 ns
IGA nephropathy	11	6	
Obstructive nephropathy	4	0	
Autosomal dominant polycystic kidney disease	1	1	
Total	78	62	

CKD: Chronic kidney disease, LVH: left ventricular hypertrophy, Fischer's exact test,*, P<0.05=significant, ns=not significant



(ADPKD) and obstructive nephropathy were also included in the research.

Exclusion criteria

The following criteria were excluded from the study:

1. Individuals with valvular heart disease, coronary heart disease, or DM
2. Individuals with a history of HTN years before the development of chronic renal disease
3. Patients who underwent dialysis after admission
4. Patients who are older than fifty
5. Patients who were alcoholics.

A thorough medical history was obtained for each patient, paying particular attention to how long the symptoms had persisted. Cardiovascular symptoms were seen, including pallor, pedal edema, dyspnea, and chest discomfort. Three blood pressure readings were obtained, and the average was calculated. A cardiovascular assessment was conducted. Serum lipid profile, serum creatinine, serum electrolytes, serum phosphate, calcium, and uric acid, and blood urea were all measured. In addition, chest X-rays and abdominal ultrasonography were performed on the patients. The CockCroft-Gault equation had been used to determine the creatinine clearance in each patient:

$$\text{Estimated Creatinine} = \frac{(140 - \text{age}) \times \text{body weight (kg)}\ \text{clearance(mL/min)}}{72 \times \text{serum creatinine (mg/dL)}}$$

This is a male-only equation. For women, it is doubled by 0.85. In the posteroanterior view of the chest X-ray, the following conditions were examined for: Cardiomegaly, pulmonary interstitial edema, and pleural effusion. The electrocardiogram (ECG) was examined for signs of ischemia, low voltage complexes, and left ventricular hypertrophy (LVH).

Finally, echocardiography was done. The following parameters were looked for:

Chamber size

Measurements of the interventricular septum, left ventricular internal diameter, and left ventricular posterior wall thickness were taken in both systole and diastole using 2D and M-mode echocardiography. Concentric LVH is indicated in patients with interventricular septal thickness and left ventricular posterior wall thickness in diastole more than 1.1 cm. Distinguishing between pathologic and physiological hypertrophy is challenging.

To avoid this, Relative wall thickness was calculated in all patients using the following equation:

Relative wall thickness =IVS(D) × LVPW(D) LVID(D)

Relative wall thickness >0.45 cannot occur in physiologic hypertrophy and it signifies pathologic hypertrophy.

A dilated left ventricle is indicated in the parasternal long-axis view by a left ventricular internal diameter in diastole more than 5.6 cm.

The left atrial anteroposterior diameter more than 3.8 cm represents dilated left atrium.

Systolic function

M-mode measurements are the primary method used to evaluate the systolic function. The two parameters that are employed are fractional shortening and ejection fraction (EF). The EF is defined as the ratio of stroke volume to end-diastolic volume.

EF =Enddiastolic volume−Endsystolic volume×100 End diastolic volume Normal values of EF are 55–75 45. Grading of systolic dysfunction:

i. Mild 45–55%.
ii. Moderate 35–45%.
iii. Severe <35%. Fractional shortening is calculated by the following equation: Fractional shortening=LVID (D) – LVID (S) × 100 LVID (D)

Diastolic function

Using the E/A data, pulsed wave Doppler evaluates the diastolic function. Mitral flow, which results in ventricular filling after the mitral valve opens, is indicated by the letter E (m/s). Ventricular filling brought on by atrial systole is shown by a (m/s). Typically, E/A is more than 1.

Less than 1 indicates diastolic dysfunction. Diastolic dysfunction can be graded as follows:

Grade 1=impaired relaxation

Grade 2=pseudo normalized pattern

Grade 3=reversible restrictive pattern

Grade 4=irreversible restrictive pattern

Left ventricular wall motion abnormalities

There are several methods for evaluating left ventricular function. The left ventricular wall is segmented into many parts. Compiling the wall motion score requires figuring out how each part moves.

Pericardial effusion

The amount of echo-free space around the heart is used to measure pericardial effusion.

It is possible to rate the pericardial effusion as minimal: Echo-free space is visible in the posterior atrioventricular groove alone during the systolic phase. It is an example of typical pericardial fluid.

- Mild pericardial effusion: Echo free space <1 cm
- Moderate pericardial effusion: Echo free space 1–2 cm
- Large Pericardial effusion: Echo free space >2 cm.

Valvular abnormalities

The valves were looked for stenotic lesions, regurgitant lesions, calcifications, or vegetations.

Statistical analysis

Frequency tables, bar graphs, and pie charts were created from the data after it was examined using tests such as Chi-square and Analysis of variance.

RESULTS

The current study's age distribution ranges from 20 to over 80 years old, with the bulk of participants being between 41 and 60 years old and 61 and 80 years old. The average age is 59.83±11.19 years.

In our research, the proportion of males (71.4%) was higher than that of girls (28.5%). Ratio of male to female: 2.5:1.

(Table 1) The study found that in 68.5% of patients (96/140) with systolic blood pressure (SBP) 140–159 mmHg, and in 31.4% of cases (44/140) with SBP>160 mmHg. Diastolic blood pressure (DBP) 80–89 mmHg was recorded in 50.7% (71/140) of the patients in our research, 90–99 mmHg in 42.1% (59/140) of the cases, and >100 mmHg in 7.1% (10/140) of the cases.

Only 14.2% (20/100) of the cases in the present study had a length of 6 months, compared to 21.4% (30/140) who had more than 5 years and 1–5 years identified among 64.2% (90/100) cases.

There was easy fatiguability in 130 out of 140 instances or 92.8%. The other prevalent symptoms were dyspnea and pedal edema, which made up 37.15 (52/140) and 27.1 (38/140), respectively. Chest discomfort was reported by 7.1% (10/140) of the cases, palpitations by 14.2% (20/140) of the cases, and vomiting by 14.2% of the cases.

According to our study, there were 31.4% (44/140) instances of LVH, 13.5% (19/140) of LAD, 18.1% (20/140) of conduction abnormalities, 15.7% (22/140) of ischemia, 2.1% (3/140) of arrhythmias, 2.8% (4/140) of paroxysmal defects, and 21.4% (30/140) of normal cases. In 2D echo results showed LVH in 55.7% (78/140) of patients, and EF<35% in 78.9% (110/140) and >35% in 21.4% (30/140) of cases. 7.1% (10/140) had pericardial effusion; 40% (56/140) had diastolic dysfunction; 18.5% (26/140) had systolic dysfunction; and 14.2% (20/140) had dilated left atrium (Tables 2 and 7).

In the present study, 11.2% of patients had Stage II CKD, 49.2% had Stage III CKD, 21.4% had Stage IV CKD, and 14.2% had Stage V CKD.

About 83.5% (117/140) of the study's cases of CKD were attributed to chronic glomerulonephritis. In our investigation, chronic glomerulonephritis accounted for 83.5% (117/140) of the cases with CKD in which Obstructive nephropathy (2.8%; 4/140), IGA nephropathy (17/140%), and ADPKD 1.4% (2/140).

DISCUSSION

The present study's age distribution ranges from 20 to over 80 years old, with the bulk of participants being between 41 and 60 years old and 61 and 80 years old. It is 59.83±11.19 years, mean±standard deviation.

In Saha *et al*. [9] study, 22.7% of respondents were under 30, 19.3% were between 30 and 40, 17% were between 40 and 50, 19.3% were between 50 and 60, and 21.7% were 60 years of age or older. The patients' average age was 43.5±15.3 years, with 14 and 80 years old as the lowest and highest ages, respectively.

In Girish *et al*. [10] study, the age distribution is 18–70 years old. The age group of 41–50 years accounted for 50% of the cases (50/100),

Table 2: Correlation of CKD diagnosis with pericardial effusion findings

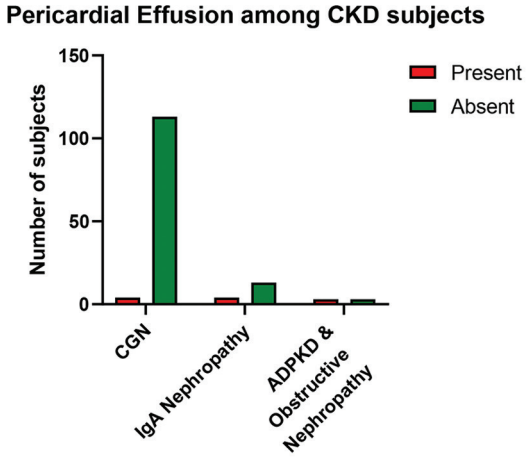
CKD diagnosis	Pericardial effusion		p-value
	No. of cases		
	Present	Absent	
CGN	4	113	0.0093**
IGA nephropathy	4	13	
Obstructive nephropathy	2	2	
Autosomal dominant polycystic kidney disease	1	1	
Total	11	129	

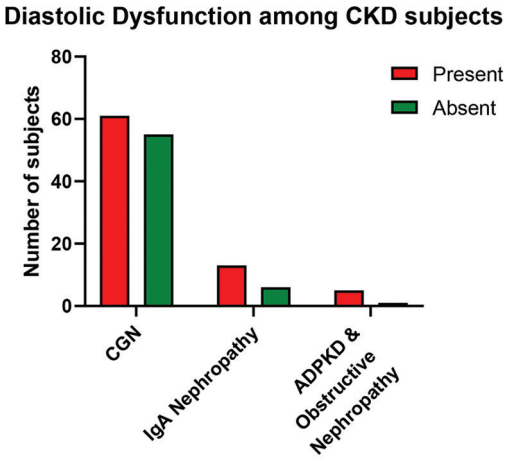
CKD: Chronic kidney disease, Fischer's exact test, *, P<0.05=Significant, ns=Not significant

Table 3: Correlation of CKD diagnosis with pericardial effusion findings

CKD diagnosis	Diastolic dysfunction		p-value
	No. of cases		
	Present	Absent	
CGN	61	56	0.2236ns
IGA nephropathy	13	4	
Obstructive nephropathy	4	0	
Autosomal dominant polycystic kidney disease	1	1	
Total	79	61	

CKD: Chronic kidney disease, Fischer's exact test, *, P<0.05=Significant, ns=Not significant





followed by 51–60 years (25/100), 61–70 years (18/100), and 18–30 years (2%, or 02/100) for the fewest instances at this moment.

In Adarsh *et al*. [11] study, the mean age of the patients was 59 varied from 47 to 73 years.

Sanath *et al*. [12] noted cardiovascular symptoms are most common in people between the ages of 41 and 50, where they occur 40% of the time (36 instances). These people are followed in order of occurrence by age groups: 51–60, 61–70, 31–40, and 21–30, with incidences of 24.44% (22 cases), 18.88% (17 cases), 13.33% (12 cases), and 3.33% (three cases).

In our study, males (71.4%) were predominant when compared to females (28.5%). Male: Female ratio 2.5:1.

In Saha *et al*. [9] study, there were around 58% men and 42% women, meaning that the male-to-female ratio was roughly 3:2.

In Girish *et al*. [10] study with a male-to-female ratio of 1.6:1, males outnumbered females by 62% (62/100) to 38% (38/100). Male patients made up 64.44% (58 cases) of the Sanath *et al*. research [12], with female patients making up 35.55% (32 cases). There were 168 (67.2%) men and 82 (32.8%) women in the Adarsh *et al*. [11] research (Table 8).

Of the 64.2% of patients in the current investigation, 21.4% had been followed for more than 5 years, and only 14.2% had been followed for one to 5 years (Table 9). About half of the patients in the Girish *et al*. [10] research had symptoms that had lasted more than 5 years, and only 7% of the cases had symptoms that had lasted between one and 5 years.

Easy fatiguability was found in 92.8% of the patients (130/140) in our research. The other prevalent symptoms that were present were dyspnea and pedal edema, which made up 37.15 (52/140) and 27.1 (38/140), respectively. About 7.1% (10/140) of the patients experienced chest discomfort, and 14.2% of the cases had palpitations and vomiting (20/140).

Girish *et al*. [10] noted that the majority of patients (75%) had dyspnea, which was followed by chest discomfort (65%), pedal edema (40%), and palpitations (35%).

In the present study, 20% had DM and 70% had HTN. The low-density lipoprotein (LDL) cholesterol was elevated by 10%.

In Saha *et al*. study [9] among the conventional risk factors, 23% had DM and 83.3% had HTN. About 46.6% of those with dyslipidemia had elevated LDL cholesterol, and 57.3% had elevated triglyceride levels. Of them, 20% suffered from obesity and overweight, and 27% had a smoking habit.

In Girish *et al*. [10] study of 100 patients, 43% (43/100) had a history of DM, 32% (32/100) had a history of HTN, 18% (18/100) had a history of both DM and HTN, and 7% (07/100) had no prior history.

In Sanath *et al*. [12] study 41 instances or 45.55%, the most frequent cause of CKD is DM+HTN, which is followed in 26.66% of cases by DM alone. In 13.33% of cases, HTN was the primary cause, while chronic glomerulonephritis and other conditions were present in only 10% and 4.44% of cases, respectively.

In the present study, 11% of patients had Stage II CKD, 49.2% had Stage III CKD, 21.4% had Stage IV CKD, and 14.2% had Stage V CKD. In the Girish *et al*. [10] research, 6% of patients had Stage II CKD, 54% had Stage III CKD, 30% had Stage IV CKD, and 10% had Stage V CKD.

In our investigation, the following conditions were found in cases: Arrhythmias in 2.1% (3/140), P-mitrale in 2.8% (4/140), LVH in

Table 4: Correlation of CKD diagnosis with systolic dysfunction findings

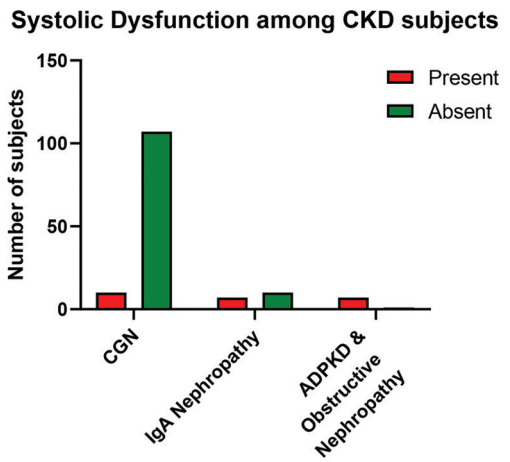
CKD diagnosis	Systolic dysfunction		p-value
	No. of cases		
	Present	Absent	
CGN	10	107	0.2236 ns
IGA nephropathy	7	10	
Obstructive nephropathy	3	1	
Autosomal dominant polycystic kidney disease	1	1	
Total	21	119	

CKD: Chronic kidney disease, Fischer's exact test, *, P<0.05=Significant, ns=Not significant

Table 5: Correlation of CKD diagnosis with dilated LV findings

CKD diagnosis	Dilated LV		p-value
	No. of cases		
	Present	Absent	
CGN	4	113	0.0093**
IGA nephropathy	4	13	
Obstructive nephropathy	2	2	
Autosomal dominant polycystic kidney disease	1	1	
Total	11	129	

CKD: Chronic kidney disease, Fischer's exact test, *, P<0.05=significant, ns=not significant



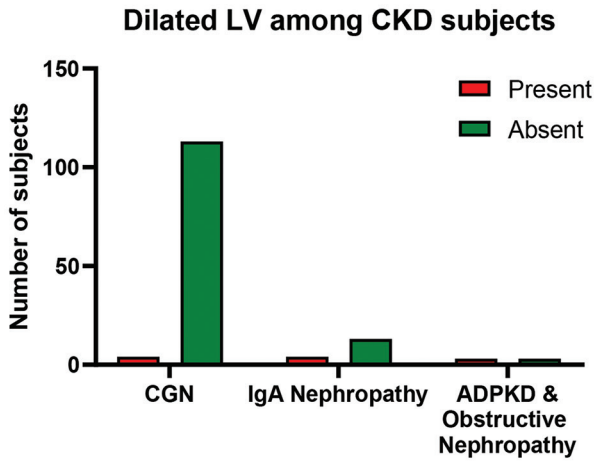


Table 6: Correlation of CKD diagnosis with dilated LA findings

CKD diagnosis	Dilated LA		p-value
	No. of cases		
	Present	Absent	
CGN	4	113	0.0093**
IGA nephropathy	4	13	
Obstructive nephropathy	2	2	
Autosomal dominant polycystic kidney disease	1	1	
Total	11	129	

CKD: Chronic kidney disease, Fischer's exact test, *, P<0.05=significant, ns=not significant

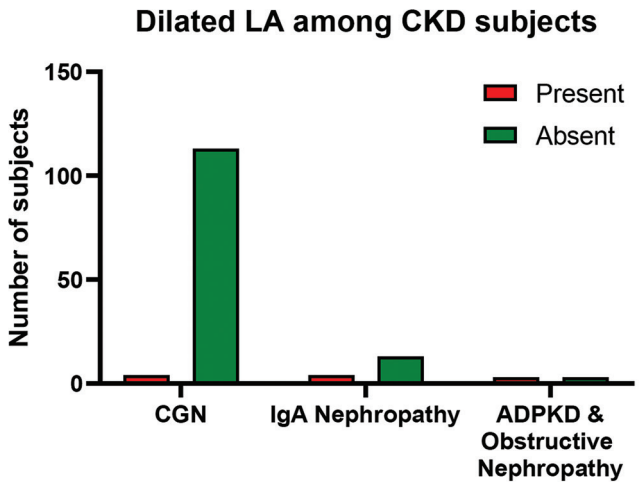


Table 7: Correlation of CKD diagnosis with ejection fraction % findings

CKD diagnosis	One way ANOVA		p-value
	EF %		
	Mean±standard deviation	SEM	
CGN	36.94±7.583	0.7071	0.9698 ns
IGA nephropathy	36.47±8.690	2.108	
Obstructive nephropathy and autosomal dominant polycystic kidney disease	37.17±9.261	3.781	

CKD: Chronic kidney disease, ANOVA: Analysis of variance, One-way ANOVA*, P<0.05=significant, ns=not significant

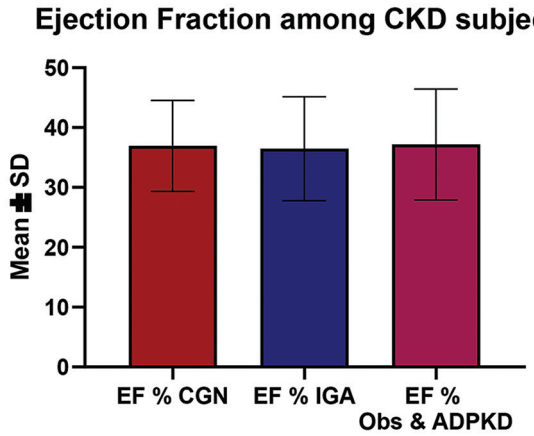


Table 8: Correlation of CKD diagnosis with SBP findings

CKD diagnosis	One way ANOVA		p-value
	SBP		
	Mean±standard deviation	SEM	
CGN	150.4±27.12	2.507	0.3681 ns
IGA nephropathy	141.0±19.17	4.649	
Obstructive nephropathy and autosomal dominant polycystic kidney disease	152.5±22.22	9.073	

SBP: Systolic blood pressure, CKD: Chronic kidney disease, ANOVA: Analysis of variance, One-way ANOVA*, P<0.05=significant, ns=not significant

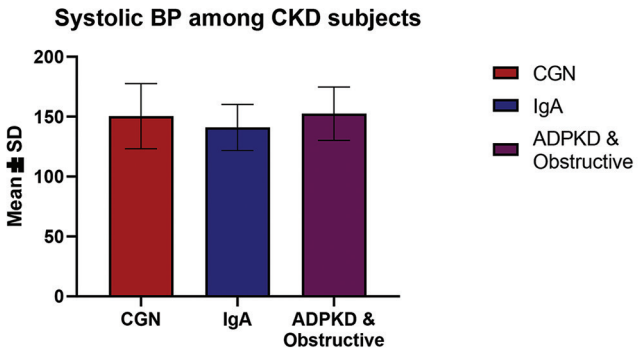
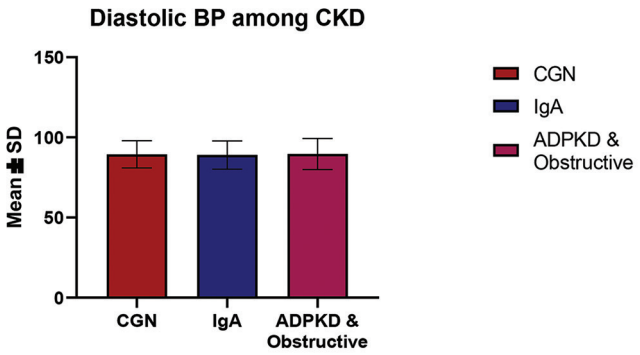


Table 9: Correlation of CKD diagnosis with DBP findings

CKD diagnosis	One way ANOVA		p-value
	DBP		
	Mean±standard deviation	SEM	
CGN	89.45±8.470	0.7830	0.9766 ns
IGA nephropathy	89.00±8.846	2.145	
Obstructive nephropathy and autosomal dominant polycystic kidney disease	89.67±9.688	3.955	

DBP: Diastolic blood pressure, CKD: Chronic kidney disease, ANOVA: Analysis of variance, One-way ANOVA*, P<0.05=Significant, ns=Not significant



31.4%(44/140) cases, LAD in 13.5% (19/140) cases, conduction abnormalities in 18.1%(20/140) cases, ischemia in 15.7% (22/140) cases, and normal in 21.4% (30/140) instances. 2D echo results showed LVH in 55.7% (78/140) of cases, EF <35% in 78.9% (110/140) of cases, and >35% in 21.4% (30/140) of cases. 7.1% (10/140) had pericardial effusion; 40% (56/140) had diastolic dysfunction; 18.5% (26/140) had systolic dysfunction; and 14.2% (20/140) had dilated left atrium (Tables 3-6).

In Saha *et al*. study [9], 9% had LVH, 38% heart failure, 4.7% arrhythmia, and 18.3% ischemic heart disease. Cardiovascular events occurred in around one-third (32.3%) of the patients, 15% in two events, 2.7% in three events, and half the patients had none at all.

Girish *et al*. [10] study, 59% of patients had an ECG with LVH. About16% of patients had conduction abnormalities, while 24% of cases had ischemia. About 10% of patients had arrhythmias, 5% had P-mitrale, and 6% had normal studies. In 59% of patients, 2D ECHO results revealed LVH as the most frequent abnormality. In 36% of the instances, systolic dysfunction was the next commonly observed anomaly. In 20% of instances, valve calcifications were seen. Dilated LV and diastolic dysfunction in 9%, RWMA and pericardial effusion in 6% of patients and 3% of patients had dilated LA. About 3% of patients had dilated LA. Systolic dysfunction was observed in 36 instances in the current investigation. Twelve percent of the patients had severe systolic dysfunction, 18% had moderate systolic dysfunction, and 6% had mild systolic dysfunction.

Nine instances with diastolic dysfunction and five cases with Grade 1 and four cases with Grade II diastolic dysfunction were included in the current investigation.

Adarsh *et al*. study [11] 90 (36%) occurrences of LVH with a pressure overload pattern. Low voltage QRS complexes: 43 instances (17.2%) and 135 cases (54%) with sinus tachycardia. LVH was found to have a mean EF of 55.21 with a standard deviation of 6.13. One hundred and six individuals, or 42.4%, exhibited LVH on both the ECG and echocardiography. Table 4 shows the CKD phases of LVH. Deficit of left ventricle diastolic function 31 (52%) of the 130 patients with left ventricular diastolic dysfunction had Grade 1, 40 (16%) had Grade 2, and 10 (4%) had Grade 3.

In Sanath *et al*. [12] study of the 90 participants, the majority (32.22%) had ischemic alterations, followed by 22.22% with ST-T abnormalities. About 10%, 8.88%, and 6.66% of patients had ECG alterations for LVC, LBBB, and VPC, respectively, while 20% had LVH changes. 28.88% of the 90 patients had LVH ECHO alterations, which were followed by cases with calcified valves, diastolic dysfunction, regurgitation, and pericardial effusion types of ECO changes (22.22%, 20%, 15.55%, and 13.33%, respectively). Among the participants, the majority of cases (43.33%) had mild systolic dysfunction (EF% of 45–55), followed by instances with moderate systolic dysfunction (23.33%) and cases with severe systolic dysfunction (13.33%) with EF% of 35–45%.

In our investigation, chronic glomerulonephritis accounted for 83.5% (117/140) of the cases with CKD in which Obstructive nephropathy.

In Girish *et al*. study [10], diabetic nephropathy, or 45% of cases with CKD, was the most prevalent cause. Chronic glomerulonephritis made up 15% of the next most prevalent cause, which was HTN-associated CKD, or 31%. IgA nephropathy and adult polycystic kidney disease accounted for around 2% (02/100) of cases, with adult polycystic kidney disease accounting for 5% (05/100).

CONCLUSION

In this study, we found that 31.4% of the patients had LVH, 13.5% had LAD, 18.1% had conduction abnormalities, 15.7% had ischemia, 2.1% had arrhythmias, 2.8% had P-mitrale, and 21.4% had normal. EF <35% was observed in 78.9% of patients, >35% in 21.4%, and LVH in 55.7% of cases according to 2D echo results. About 7.1% of patients have pericardial effusion; 40% have diastolic dysfunction; 18.5% have systolic dysfunction; and 14.2% have dilated left ventricle. In chronic renal disease, echocardiography is a non-invasive, safe, simple, and reliable method of assessing heart function. When it comes to identifying LVH, echocardiography is more accurate. For individuals with CKD, cardiovascular illnesses are the primary cause of morbidity and death.

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