

Original Research Article

Cardiac function's in patients with chronic kidney disease

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ABSTRACT

Background: To assess the cardiac functions in patients with chronic kidney disease (CKD).

Methods: 150 patients with CKD were randomly selected. 12 lead ECG were performed to detect CVD. All Patients were diagnosed with CKD. The left ventricular ejection fraction (LVEF) and fractional shortening (FS) were taken as measures of LV systolic function. Diastolic function was determined by measuring early to late peak velocities (E/A) ratio by spectral Doppler LV inflow velocity.

Results: Male: female 95 and 55, hypertension 67% was leading cause of CKD. Diastolic dysfunction as denoted by E/A ratio of less than 0.75 or more than 1.8 was present in 64% of patients. Regional wall motion abnormality (RWMA) was present in 14%. LVH was present in 74%. Systolic dysfunction as measured by reduced fractional shortening (<25%) and decreased LVEF (<52%) was present in 8% and 12% respectively. PE was noted in 15% of patients. Valvular calcification in 8% of CKD patients. Mean LV internal diameter in diastole was 41±6 mm. Mean Interventricular septum diameters in systole was 11.9±1.21 mm. Mean LA diameter was 29±4 mm. Statistically significant difference was noted in LVH and E/A ratio in hypertensive group as compared to normotensive group.

Conclusions: LV diastolic dysfunction also happens in patients who having the early stage of CKD. Hypertensive patients along with CKD had found higher widespread presence of diastolic and systolic dysfunction as compared to normotensive.

Keywords: CKD, Dysfunction, Echocardiography, Hypertension

INTRODUCTION

Cardiovascular disease (CVD) is the supreme cause of morbidity and mortality amid patients with CKD. In spite of alteration for known CAD risk factors, including hypertension and diabetes and mortality risk dynamically intensifying with worsening condition of CKD.¹ Chronic kidney disease is a non-communicable disease typically caused by diabetes and hypertension.² The extremity of CKD can be proficient by a reasonable serum creatinine-based estimated glomerular filtration rate (eGFR), which

also indicates excretory kidney function, and elevated urinary albumin measured by the urinary albumin-to-creatinine ratio (ACR), which is a best predictor of kidney damage.³ In 2017, the worldwide prevalence of CKD was 9.1% (95% uncertainty interval [UI] 8.5 to 9.8), which is roughly estimated 700 million cases. Since 1990, the prevalence of CKD has intensified by 29.3% (26.4 to 32.6), but age-standardized prevalence has remained untouched during this period (1.2%, -1.1 to 3.5). A worthwhile rise was noted in age-standardized incidence of end-stage kidney disease (ESKD) which is treated by

renal replacement therapy, with dialysis and kidney transplantation. The global strengthen in mortality from CKD since 1990 was 41.5% (95% UI 35.2 to 46.5), such that mortality from CKD, and cardiovascular disease deaths in debt to impede kidney function caused 4-6% (4.3 to 5) of worldwide deaths in 2017, which manifested that CKD is the 12th leading cause of death globally in 2017, as an increase from 17th in 1990.⁴

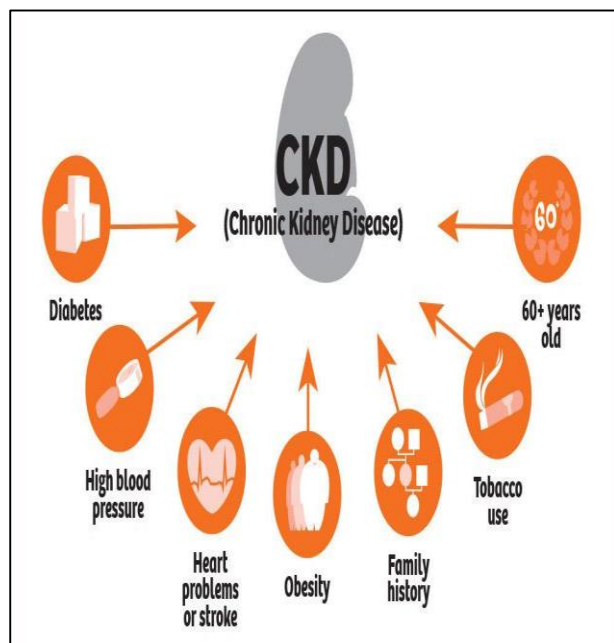


Figure 1: Risk factor of kidney disease.⁵

Hypertension and CKD are intimately interlinked pathophysiologic phenomenon, such that associated with hypertension can lead to worsening kidney function and progressive lowering in kidney function and can contrariwise lead to worsening blood pressure (BP) control.⁶ As per WHO high blood pressure as systolic blood pressure of ≥ 140 mmHg or diastolic blood pressure of ≥ 90 mmHg or receiving any medication for high blood pressure.⁷ High blood pressure (BP) is the most considerable risk factor for the development and progression of CKD as well as more deaths and disease worldwide than any other single health risk factor.⁸⁻¹⁰ Early detection of high BP and its appropriate management is required as possible as to makes a difference in the prevention of CKD progression and control of the CKD health burden.¹¹ Furthermore inexpensive interventions for diabetes, hypertension, and CKD can have a meaningful effect on clinical and societal outcomes.¹² If left uncontrolled, hypertension can cause stroke, myocardial infarction, cardiac failure, dementia, renal failure and blindness. An estimated worldwide 22% of adults aged 18 years and over around the world had raised blood pressure (defined as a systolic and/or diastolic blood pressure $\geq 140/90$ mmHg) in 2014.¹³

Coronary artery disease which includes myocardial infarction, congestive heart failure (CHF) and pericardial

disease are the frequent disclosures of vital cardiovascular abnormalities in the ESRD. Approximately 30% of patients reaching ESRD already have subclinical and clinical evidence of ischemic heart disease or CHF. Furthermore, patients with alleviate glomerular filtration rate (GFR) are presumed to die of CVD even before they are to develop ESRD. Heart failure description for about 15%, myocardial infarction for about 10% and pericarditis for about 3% of dialysis accompanying mortality.¹⁴ Coronary calcification is widespread among patients with CKD although the prognostic value is probably similar to that in the local population, the progression of coronary calcification is expeditious with intensifying CKD.¹⁵

CKD is a state of promoted atherosclerosis, the most common demonstration in ESRD are heart failure and sudden cardiac death rather than myocardial infarction; this reflects the impact of abnormalities like high prevalence of left ventricular dysfunction and secondary to the LVH in dialysis rather than atheromatous coronary heart disease.¹⁶⁻¹⁷ ESRD is the irreversible decline of renal function which results into impairment of excretory, metabolic and endocrine functions leading to evolution of the clinical syndrome of uremia.¹⁸ Thereafter, CV risk intensifying gradually and is maximal in patients with ESRD requiring dialysis and prompted effective treatment with age adjusted CV risk at least 20 times that of the common population.¹⁹⁻²⁰ Whereas standard clinical guidelines perceived CKD as a “modifying factor” to be scrutinized using the standard risk equations, they do not formally encompass kidney-specific variables, even though eGFR is effortlessly available.²¹ We aimed to assess the cardiac functions in CKD patients. After all laboratory test and Echocardiographic findings of hypertensive and normotensive patients, data were calculated and summarize. Comorbid patients were also included in the study and statistically significant difference was found in LVH and E/A ratio in hypertensive group as compared to normotensive group.

METHODS

An observational study was carried out in 150 patients from department of medicine and cardiology from Parul Sevashram hospital, Vadodara, Gujarat India between Sep 2021 to 15 March 2022 after obtaining an approval from institutional ethics committee. Data were collected in the predesigned Patient Profile Form along with complete laboratory reports and all relevant history. All the patients were gone through two-dimensional and M mode echocardiography for determination of their cardiac functions. the analysis made from the data was reported in predesigned forms which includes information such as patient demographic details (BP, all vitals, weight, medical & medication history) and required laboratory information (Serum creatinine, GFR).

All patients were evaluated physically, clinically, biochemically and radiological test were done as per

discretion of physician. Additionally, all required examinations were performed as and when required.

Echocardiography was executed using a cardiac ultrasound unit with a 2-3.5 MHz transducer. TDI was performed in all patients with images taken. Left ventricular end-diastolic, systolic dimensions, end-diastolic, and systolic wall thickness of the inter-ventricular septum and left ventricular wall were determined using standard echocardiography 2-D and M-mode measurements. M mode recording perpendicular to the long axis of and through the center of the left ventricle at the papillary muscle level were taken as standard measurements of the systolic and diastolic wall thickness and chamber dimensions. The LVEF and fractional shortening (FS) were taken as measure of left ventricular systolic function. Diastolic function was determined by measuring E/A ratio by special Doppler inflow velocity (E is peak early diastole velocity and A is peak atrial filling velocity of left ventricle across mitral valve). E/A ratio less than 0.75 and more than 1.8 was considered as diastolic dysfunction. LVH was diagnosed when interventricular septum thickness or left ventricular posterior wall thickness was ≥ 12 mm.

Fractional shortening (s) was calculated as:

$FS (\%) = (LVDd - LVDs) \times 100$ normal range being 25% to 45% (LVDd)

LVDd: Left ventricle internal diameter in diastole and LVDs: Left ventricle internal diameter in systole

Ejection fraction was calculated as:

$LV\ EF (\%) = (LVVd - LVVs) \times 100$ normal = $59.2 \pm 6\%$ (LVVd)

LVVd: Left ventricle volume in diastole and LVVs: Left ventricle volume in systole

Study criteria

Inclusion criteria

Patients with age about > 18 years, GFR should be < 90 ml/min/ $1.73\ m^2$, serum creatinine > 3 mg/dl and subjects having confirm diagnosis of CKD > 6 month were included in the study.

Exclusion criteria

Pregnant, lactating women, mentally ill or other psychological subjects, subject who are on antineoplastic medication, post traumatic patient, patient who had severe course of COVID-19 and other comorbid disease or condition which can interfere with study as per investigators discretion were excluded from the study.

Biochemical estimations

Physical examination, all vitals, GFR, serum creatinine, CBC, cardiac biomarker, kidney function test and

echocardiography were performed. Additional tests were performed based on investigator discretion as applicable.

Statistical analysis

The data was represented graphically in MS-excel with median values.

RESULT

This study included 150 patients with 95 (63.33 %) male and 55 (36.66 %) female who were diagnosed with chronic kidney disease (stage 1 to 5) or End stage renal disease based on laboratory interpretation of GFR (< 90 ml/min/ $1.73\ m^2$) and serum creatinine (> 3 mg/dl). Among that 75 (50 %) patients were hypertensive (BP $> 140/90$ mmhg) and 75 (50%) were normotensive.

Table 1: Subject's demography including clinical characteristic, (n=150).

Variables		Total, n (%)
Age (Years)		18- 75 (Mean 55 ± 10)
Gender	Male	95 (63.33)
	Female	55 (36.66)
BMI (mean) (kg/m ²)		25.5
Clinical characteristics	Hypertensive	101 (67)
	Normotensive	50 (33)
	Haemodialysis	60 (40)
	End-stage renal disease (Stage 4 and 5)	66 (44)
	Diabetes	56 (37)
	Reduced urine output	108 (71.3)
	Nocturia	42 (27.9)
	Haematuria	18 (11.8)
	Pruritus	19 (12.9)
	Pallor	96 (63.2)
	Pedal oedema	93 (61.7)
	Proteinuria	72 (47.7)
Educational status	Primary	78 (52)
	Secondary	28 (18.66)
	Tertiary	18 (12)
	None	26 (17.33)
Occupation	Employed	60 (40)
	Unemployed	57 (38)
	Others	33 (22)
Marital status	Married	88 (58.66)
	Single	21 (14)
	Others	41 (27.33)

The subjects were classified in to 5 groups based on GFR. Among that 60 patients were on hemodialysis. We also found that E/A increased in parallel with the severity of kidney dysfunction, apart from patients with very advanced CKD (group 5).

Table 2: Analyzed patients based on CKD stages, (n=75).

Stage of CKD	Hypertensive	Normotensive
Stage 1 (GFR 90 or higher)	6	9
Stage 2 (GFR 89 to 60)	9	9
Stage 3a (GFR 59 to 45)	9	12
Stage 3b (GFR 44 to 30)	12	18
Stage 4 (GFR 29 to 15)	24	15
Stage 5 (GFR less than 15)	15	12

Echocardiography showed that major contributing factors for left ventricular hypertrophy and diastolic dysfunction were hypertension. Major contributing factor for systolic dysfunction was RWMA due to ischemic heart disease. Echocardiography showed that left ventricular hypertrophy (LVH) was present in 111 (74%). Systolic dysfunction as measured by reduced fractional shortening (<25%) and decreased LVEF (<52%) was present in (13) 8% and 18 (12%) respectively.

Diastolic dysfunction as denoted by E/A ratio of less than 0.75 or more than 1.8 was present in 96 (64%) of patients. RWMA was present in 21 (14%). Pericardial effusion was noted in 23 (15%) of patients. Valvular calcification was noted in 12 (8%) of CKD patients. Echocardiography was more sensitive for detecting LVH and minimal pericardial effusion prior to clinical detection. Statistically significant difference was noted in LVH and E/A ratio in hypertensive group as compared to normotensive group.

Mean left ventricular internal diameter in diastole was 41 ± 6 mm. Mean Interventricular septum diameters in systole was 11.9 ± 1.21 mm. Mean left atrium diameter was 29 ± 4 mm. E/A was elevated in group 5 as measure up with group 4, even though the difference was not significant, and E was significant lower in groups 1, 2, 3, and 4 compared with group 5. These results suggest that the left ventricle filling pressure may be higher in group 5 than groups 1, 2, or 3. However, there is flexibility that it is affected by intrinsic volume overload in hemodialysis patients. Even a moderate decline in kidney function is associated with a significant worse prognosis in patients with under-lying CHF. E/A ratio were lower in CKD subjects, these results show that left ventricular diastolic dysfunction is present in all patients with CKD, including those with an early stage of the disease due to hypertension.

Echocardiography was executed using a cardiac ultrasound unit with a 2-3.5 MHz transducer. TDI was performed in all patients with images taken. Left ventricular end-diastolic, systolic dimensions, end-diastolic, and systolic wall thickness of the inter-ventricular septum and left ventricular wall were determined using standard

echocardiography 2-D and M-mode measurements. The ratio of early to late peak velocities (E/A), and deceleration time of E velocity the echocardiography's of the patient on dialysis were performed before dialysis.

Table 3: Echocardiographic findings in ESRD study cases.

Echocardiographic finding in cases of ESRD	No. of cases	Percentage (%)
Left ventricular hypertrophy	112	74
Fractional shortening (<25%)	12	8
Ejection fraction (<50%)	18	12
E/A ratio (<0.75 or >1.8)	90	60
RWMA	18	12
Pericardial effusion (<10 mm)	21	14
Valvular calcification	12	8

Echocardiography show that left ventricular hypertrophy was present in 74%. Dysfunction as measured by reduced fractional shortening (<25%) and decreased LVEF (<52%) was present in 8 and 12% respectively. Diastolic dysfunction is an abnormality of relaxation, filling, or dispensability of the left ventricle that is associated with augmented cardiovascular mortality. Trans-mitral pulsed Doppler is a non-invasive method of evaluation of diastolic dysfunction, but is influenced by factors such as the loading condition of the left atria and heart rate. In contrast, TDI can be used to measure mechanical wall function directly by calculate in the velocity of myocardial longitudinal movement and to monitor diastolic function of the myocardium more effectively. In the present study diastolic dysfunction as denoted by E/A ratio of less than 0.75 or more than 1.8 was present in 64% of patients. E/A ratio were lower in CKD subjects, these results show that left ventricular diastolic dysfunction is present in all patients with CKD, including those with an early stage of the disease. These results indicate that Doppler indices combined with conventional and TDI can detect subtle changes of diastolic function caused by kidney dysfunction. RWMA was present in 14%. Pericardial effusion was noted in 15 % of patients. Valvular calcification was noted in 8 % of CKD patients.

We also found that E/A increased in parallel with the severity of kidney dysfunction, apart from patients with very advanced CKD (group 5). E/A was elevated in group 5 as measure up with group 4, even though the difference was not significant, and E was significant lower in groups 1, 2, 3, and 4-compared with group 5. These results suggest that the left ventricle filling pressure may be higher in group 5 than groups 1, 2, or 3. However, there is flexibility that it is affected by intrinsic volume overload in hemodialysis patients. Even a moderate decline in kidney function is associated with significant worse prognosis in patients with underlying CHF.

Table 4: Correlation analyses according to echocardiography finding in hypertensive and normotensive ESRD study, (n=75).

Echocardiographic finding in cases of ESRD	Hypertensive, n (%)		Normotensive, n (%)	
	Present	Absent	Present	Absent
Left ventricular hypertrophy	72 (96)	03 (04)	39 (52)	36 (48)
Fractional shortening (<25%)	9 (12)	66 (88)	03 (04)	72 (96)
Ejection fraction (<50%)	12 (16)	63 (84)	06 (08)	69 (92)
E/A ratio (<0.75 or >1.8)	66 (88)	09 (12)	24 (32)	51 (68)
RWMA	15 (20)	60 (80)	03 (04)	72 (96)
Pericardial effusion (<10 mm)	15 (20)	60 (80)	06 (08)	69 (92)
Valvular calcification	09 (12)	66 (88)	03 (04)	72 (96)

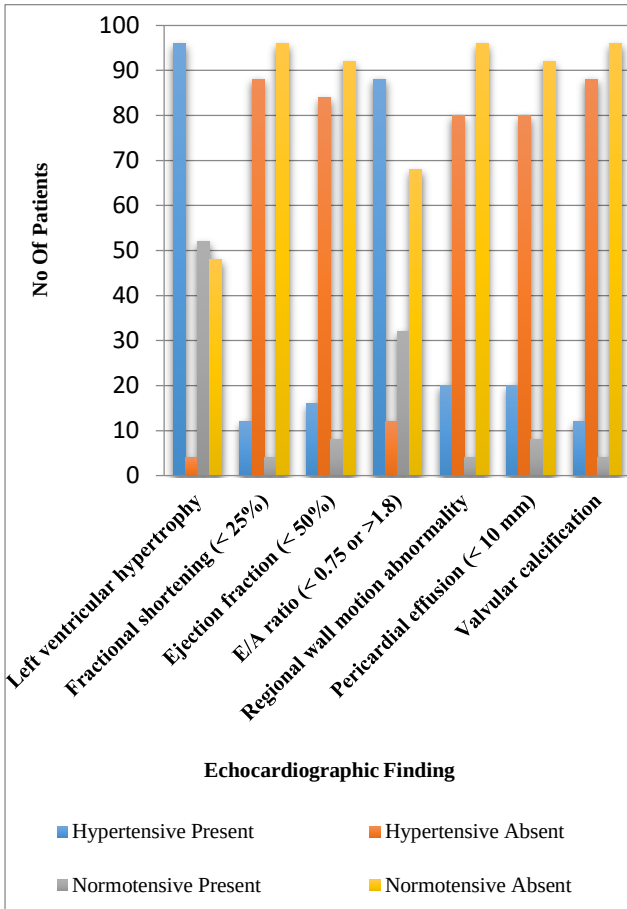


Figure 2: Correlation analyses according to echocardiography finding in hypertensive and normotensive ESRD study.

In hypertensive patients with CKD, LVH was present in 96%, diastolic dysfunction was present in 88% as deliberate by abnormal E/A ratio, systolic dysfunction as considered by reduced LVEF was present in 16% and pericardial effusion observed in 20%. In normotensive patient with CKD, LVH was present in 52%, diastolic dysfunction was present in 32%, and systolic dysfunction was present in 8% and pericardial effusion observed in 8% patients. Mean left ventricular internal diameter in diastole was 41±6 mm. Mean Interventricular septum diameters in systole was 11.9±1.21 mm. Mean left atrium diameter was 29±4 mm.

Normotensive group was measured along with hypertensive group. Statistically significant difference was noted in LVH and E/A ratio in hypertensive group as compared to normotensive group.

Our result has limited the evidence base for optimal decision making and single center study, Patients with CKD exhibit an elevated cardiovascular risk manifesting as coronary artery disease, heart failure, arrhythmias, and sudden cardiac death.

DISCUSSION

Our current study shows a correlation between CKD, hypertension and systolic and diastolic dysfunction of heart. In CKD, LVDD occurs frequently [97 (64%)] thus related with heart failure. Similarly, Sachdeva et al concluded that based on echocardiographic changes, LVDD was present in 38.33% patients, Krishna et al reported LVDD in 38%, Ladha et al reported LVDD in 61.4% and Singal et al reported in 50%.²²⁻²⁵ Rao et al found that 67.2% of subjects had diastolic dysfunction.²⁶ Losi et al in a cross-sectional study declare that nearly 40% of the patients had diastolic dysfunction.²⁷ Agrawal et al had distinguish a prevalence of diastolic dysfunction of 30% in early stages of CKD and 53.2% in later stages of CKD.²⁸ There was an inclination of increasing prevalence of diastolic dysfunction with collapse renal function (34.8% in CKD stage 1 and 77.8% in CKD stage 5). A similar study conducted by Nitin et al had found that 51.85% of patients with mild/moderate CKD had diastolic dysfunction, whereas 82.6% of patients with severe CKD had diastolic dysfunction.²⁹ Moreover, a study by Singh et al the LVDD was found 43.65%.³⁰

Moreover, systolic dysfunction in 12 (8%) basically related with coronary artery disease hence major determinant of prognosis. It is present in greater proportion in later stages of CKD. Similarly, a study by Kulkarni et al and Foley et al concluded that systolic dysfunction was present in 29 (41.4%) and 14.8 % patients.^{31,32} A study by Singh et al.³⁰ LV systolic dysfunction was 15.6%. Furthermore, systolic dysfunction was reported 24.6% with CKD in a European multicenter study reported by Chinali et al but higher than the 8.3% reported by Adiele et al.^{33,34} These findings suggest that there is a significant burden of LV systolic and diastolic dysfunction in CKD

patients. We found LVEF was present in 18 (12%). Similar result was also stated by Shin et al that LVEF was present in 57.2% of hemodialysis patients.³⁵ Hensen et al LVEF was observed in 32% of patients although same was higher as compared to our study.³⁶

In our study echocardiography showed that left ventricular hypertrophy (LVH) was present in 111 (74%). Similar result were also produced by Hayashi et al and Foley et al that LV hypertrophy was observed in 63% and 73.4% patient by Ramegowda et al left ventricular hypertrophy was seen in 24 patients (48%).^{32,38,39} In CKD patients, left ventricular hypertrophy (LVH) is a common finding and it is associated with an increased CVD-related mortality.^{32,37} Studies indicates that the pervasiveness of LVH inflation to 60-75% in patients beginning renal replacement therapy, and reaches 70-90% in patients on regular dialysis.³⁹ According to Levin et al the prevalence and severity of LVH enlarged along with the progression of chronic kidney disease.⁴⁰

Valvular calcification was found in 8% cases. Similarly, Rong et al concluded that 22.9% had valvular calcification in patient with CKD.⁴¹ A Chinese study indicated that the relative risk of heart VC increased by 2.22 to 2.66-fold with each decade of life, while the rate of VC remains higher in patients with CKD.⁴² Eventually, vascular calcification strongly contributes to the exceedingly extreme cardiovascular disease mortality in the population.⁴³ Pericardial effusion (PE) (<10 mm) was found in 14% patients. Comparably, in a study by Ravi et al resulted that effusion was moderate to large in 46% of cases. Additionally, in a study by Qian et al pericardial effusion was present in 35% patients.^{44,45} Furthermore, by Ramegowda et al pericardial effusion was seen in 2 patients (4%).³⁸

Ejection fraction (<50%) was detected in 12% patients similar result were also concluded by Mavranakas et al.⁴⁶ That EF was confirmed in 12.7% patients. Another study by Hensen et al have similar result that LVEF was observed in 32% of patients.³⁶ RWMA was present in 12% cases. Assa et al also concluded that 16% patients developed regional left ventricular systolic dysfunction.⁴⁷ Additionally, Regional wall motion abnormalities were seen in 7 patients (14%) by Ramegowda et al.³⁸

Most common Echocardiographic abnormality was Left ventricular hypertrophy followed by conduction abnormalities, E/A ratio, pericardial effusion and RWMA. Left ventricular hypertrophy is the commonest morphological abnormality observed in our study, followed by RWMA. Left ventricular dysfunction is commonest cardiovascular abnormality detected. Echocardiography is a more sensitive diagnostic procedure to detect left ventricular dysfunction in patients with CKD. As the stages of CKD progresses spike ECHO abnormalities were observed like left ventricular hypertrophy, systolic dysfunction, LVDD, regional wall motion abnormalities and pericardial effusion.

Limitation

Our study used only two parameters i.e., GFR and Serum Creatinine level that was probably not enough reveals long standing inflammatory status, co morbid disease and other medical conditions. However, the study also has several limitations. We used conventional and TDI methods to assess the left ventricular diastolic function in CKD patients, less sample size and the duration of renal dysfunction, hypertension, and diabetes was unknown in each group. We also did not examine pulmonary vein flow velocity. Instead of these we analyzed the parameters of left ventricular diastolic function by routine echocardiographic examination. These factors may have affected the statistical analysis.

CONCLUSION

We concluded that left ventricular diastolic dysfunction also occurs in patients having early stage of CKD but patients with hypertensive CKD had higher prevalence of diastolic and systolic dysfunction as compared to normotensive counterparts and that Doppler indices combined with conventional and TDI can be used to detect subtle changes of diastolic function due to kidney dysfunction. Also, CKD causes a systemic, chronic pro-inflammatory state contributing to vascular and myocardial remodeling. In this respect to our study, CKD mimics a hastened aging of the cardiovascular system.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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