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Original Research Article

# Correlate the Echocardiographic Findings with Hypertension and Different Bio-Chemical Parameters in CKD patients

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**Abstract:** Relationship of Echocardiographic findings with hypertension and biochemical parameters in Chronic Kidney disease (CKD) patients has not been clarified. We aim to correlate the echocardiographic findings with different biochemical parameters in chronic renal failure patients. In this study we included 50 cases of CKD and 30 controls. Detailed history, clinical examination and relevant investigations with 2D and Colour Doppler Echocardiography were conducted in all cases and control. We found that CKD patients have highly increased incidence of pericardial effusion, left ventricular hypertrophy (LVH), diastolic dysfunction, valvular regurgitation and left ventricular dilatation. Diastolic dysfunction significantly correlated with blood urea levels, serum creatinine levels and decrease in hemoglobin levels. LV dilatation correlated with decrease in Hemoglobin levels. LVH was related to the presence of hypertension in CKD cases while diastolic dysfunction was not found to correlate with hypertension or LVH, which indicated that many other factors were responsible in causing diastolic dysfunction. Thus, timely identification and treatment of these cardiovascular abnormalities could alter the course of the disease and help in reducing morbidity and mortality of CKD patients.
**Keywords:** Chronic Kidney Disease, Hypertension, Echocardiography.

## INTRODUCTION

Chronic kidney disease (CKD) is a pathophysiologic process with multiple etiologies, resulting in the inexorable attrition of nephron number and function and frequently leading to end-stage renal disease (ESRD) [1] ESRD represents a clinical state or condition in which there has been an irreversible loss of endogenous renal function which results into impairment of excretory, metabolic and endocrine functions leading to development of the clinical syndrome of uraemia. [2].

CKD is not uncommon but fortunately treatable and it is recognized worldwide as a public health problem [3] Patients with CKD are associated with a variety of metabolic cardiovascular, endocrine and haematological complications. [1]. Diabetic and Hypertensive nephropathy are the leading underlying etiologies of both CKD and ESRD. [1].

Patients with CKD are at significantly increased risk for both morbidity and mortality from cardiovascular disease (CVD). The magnitude of the problem has become more apparent as patients now survive longer on maintenance haemodialysis. [2]. CVD in CKD is treatable and potentially preventable, and CKD appears to be a risk factor for CVD. In 1998, the National Kidney Foundation (NKF) Task Force on CVD in CKD issued a report emphasizing the high risk of CVD in CKD. This report showed that there was a high prevalence of CVD in CKD and that mortality due to CVD was 10 to 30 times higher in dialysis patients than in the general population. CVD is the single most important cause of death among patients receiving long-term dialysis; accounting for 44% of overall mortality [4]. The task force recommended that patients with CKD be considered in the "highest risk group" for subsequent CVD events and that treatment recommendations based on CVD risk stratification should take into account the highest-risk status of patients with CKD [3-4].

2-D Echocardiography and Colour Doppler are two useful noninvasive procedures to assess cardiac changes related to renal failure. This study was conducted to investigate the uremic patients for the

assessment of their cardiovascular status and to correlate the echocardiographic findings with each other and with hypertension and different bio-chemical parameters.

## MATERIAL AND METHODS

The clinically stable patients of chronic renal failure were included in the study, taking care to exclude any patient with previous organic cardiac involvement and acute decompensation of CKD. The study included 50 cases of CKD and 30 controls comprising of 15 hypertensive and 15 non hypertensive, age and sex matched controls. Before conducting a study, ethical approval was taken from institutional ethical committee. Once all the criteria were satisfied, a written informed consent was taken and the patient was included in the study. A detailed history of every patient symptoms such as Breathlessness, Edema, Nausea, Vomiting, Loss of appetite, Weakness, Bone pain, Oliguria, Polyuria, Nocturia, Haematuria and clinical examination of Signs: Pallor, Hypertension, Pericardial rub, Cardiac murmur, Raised JVP, Irregular pulse, Gallop Rhythm, Cardiomegaly) with 2D and Colour Doppler Echocardiography was conducted in all cases and control.

All patients underwent 2D directed M mode echocardiography performed on Hewlett Packard SIM 7000, in left lateral decubitus position using 3.5 MHz transducer by a consultant physician experienced in echocardiography. The left ventricular ejection fraction (EF) and fractional shortening (FS) were taken as measures of LV systolic function. EF was determined by measuring left ventricular volumes in apical 2-chamber view. Left ventricular volumes were measured by Area length method, [5] both in end diastole (LVVd) and in end systole (LVVs):

$$EF = LVVd - LVVs/ LVVd$$

The mean EF in normal population is taken as 59.2 ± 6%. [6, 7] EF was considered decreased if it was <50%. Fractional shortening (FS) was determined by measuring left ventricular internal diameter in diastole (LVIDd) and left ventricular internal diameter in systole (LVIDs) by 2D directed M mode echo at the level of papillary muscle:

$$FS = (LVIDd-LVIDs)/ LVIDd \times 100$$

Normal reference value in adults for FS is 35±8%. [8] FS of ≤25% was taken as index of systolic dysfunction. [9] Diastolic function was determined by ratio of peak early diastole velocity (E)/peak atrial filling velocity (A) of LV, i.e., (E/A), measured by spectral Doppler LV inflow velocity with sample volume at the level of mitral valve. Normal value of Doppler LV diastolic function index was taken as: Peak velocity E (m/sec): 0.61m/sec±0.14, peak velocity A (m/sec): 0.48m/sec±0.14 with a normal E/A ratio: l.40 ±0.549. LV diastolic dysfunction was considered if E/A velocity were found to be ≤0.8 [10].

The results were statistically analysed between groups and within group's comparison of the groups, the p value was kept significant at 0.05 levels and compared with previous studies and conclusions derived appropriately.

## RESULTS

**Table 1: Incidence of Various Echocardiographic Abnormalities In CKD Cases And Controls**

| Type of abnormality | CKD cases | | Controls | | 'Z' value | 'P' value |
|---|---|---|---|---|---|---|
| | Patients | % | Patients | % | | |
| Pericardial effusion | 15 | 30 | 0 | 0 | 4.63 | P<0.0001 |
| LVH | 30 | 60 | 5 | 16.7 | 4.46 | P<0.0001 |
| Systolic dysfunction | 7 | 14 | 0 | 0 | 3.16 | P<0.005 |
| Diastolic dysfunction | 23 | 46 | 2 | 6.6 | 4.69 | P<0.0001 |
| Systolic+diastolic dysfunction | 2 | 4 | 2 | 6.6 | 0.50 | P>0.05 |
| LV dilatation | 10 | 20 | 1 | 3.3 | 2.55 | P<0.05 |
| Cardiac Valvular Calcification | 1 | 2 | 2 | 6.6 | 0.94 | P>0.05 |
| Valvular regurgitation | 14 | 28 | 1 | 3.3 | 3.45 | P<0.0001 |

**Table 2: Incidence of Systemic Hypertension and in CKD patients with Diastolic Dysfunction and LVH**

| | Hypertensive | | Non Hypertensive | | Total | P value |
|---|---|---|---|---|---|---|
| | No. | % | No. | % | | |
| **Diastolic dysfunction** | 15 | 53.5 | 8 | 36.3 | 23 | p>0.05 |
| **LVH** | 20 | 74 | 10 | 43.4 | 30 | p<0.05 |

**Table 3: Relation of LVH And Diastolic Dysfunction In CKD patient**

| LVH | DIASTOLIC DYSFUNCTION | | | | Total | P value |
|---|---|---|---|---|---|---|
| | Present | | Absent | | | |
| | No. | % | No. | % | | |
| Present | 17 | 74 | 13 | 48 | 30 | >0.05 |
| Absent | 6 | 26 | 14 | 52 | 20 | |
| **Total** | **23** | **100** | **27** | **100** | **50** | |

**Table 4 : Biochemical Parameters in various cardio vascular disorders in CKD patients**

| Diagnosis | | Blood Urea (mg/dL) | Serum Creatinine (mg/dL) | Hb (gm%) | S.Sodium (mEq/Lt) | S. Potassium (mEq/Lt) | Total Cholesterol (mg/dL) | S. Triglycerides (mg/dL) |
|---|---|---|---|---|---|---|---|---|
| LVH | | | | | | | | |
| Present | Mean | 102.73 | 4.64 | 8.750* | 134.50 | 4.827 | 189.03 | 123.63 |
| | Std. Deviation | 31.066 | 2.395 | 1.5469 | 5.224 | .8047 | 88.443 | 42.168 |
| Absent | Mean | 93.10 | 6.18 | 6.780 | 134.70 | 4.705 | 209.45 | 139.55 |
| | Std. Deviation | 41.403 | 4.158 | 1.8452 | 6.658 | .6362 | 100.042 | 39.599 |
| Systolic Dysfunction | | | | | | | | |
| Present | Mean | 81.29 | 4.54 | 6.571* | 136.29 | 4.514 | 267.29* | 148.29* |
| | Std. Deviation | 20.654 | 2.396 | 2.2874 | 6.422 | .7537 | 91.834 | 34.860 |
| Absent | Mean | 101.74 | 5.37 | 8.188 | 134.30 | 4.821 | 185.79 | 127.02 |
| | Std. Deviation | 36.707 | 3.397 | 1.7830 | 5.697 | .7347 | 88.831 | 42.084 |
| Diastolic Dysfunction | | | | | | | | |
| Present | Mean | 98.43 | 4.38 | 7.787 | 134.26 | 4.739 | 190.39 | 134.39 |
| | Std. Deviation | 32.921 | 2.263 | 2.1870 | 6.784 | .8294 | 95.629 | 42.547 |
| Absent | Mean | 99.26 | 5.99 | 8.111 | 134.85 | 4.811 | 203.00 | 126.26 |
| | Std. Deviation | 38.139 | 3.815 | 1.6885 | 4.873 | .6635 | 91.744 | 41.021 |
| Dilated LV | | | | | | | | |
| Present | Mean | 89.90 | 5.34 | 7.480 | 131.30* | 4.860 | 248.60* | 151.10* |
| | Std. Deviation | 31.600 | 2.455 | 1.7542 | 6.395 | .7137 | 103.306 | 38.957 |
| Absent | Mean | 101.13 | 5.23 | 8.083 | 135.40 | 4.758 | 184.35 | 124.73 |
| | Std. Deviation | 36.401 | 3.468 | 1.9615 | 5.387 | .7507 | 86.667 | 40.879 |
| Pericardial effusion | | | | | | | | |
| Present | Mean | 115.20* | 6.35 | 7.033* | 134.87 | 4.807 | 200.07 | 129.87 |
| | Std. Deviation | 42.714 | 4.785 | 2.1976 | 4.926 | .8163 | 96.263 | 41.674 |
| Absent | Mean | 91.89 | 4.78 | 8.360 | 134.46 | 4.766 | 195.97 | 130.06 |
| | Std. Deviation | 29.912 | 2.282 | 1.6677 | 6.166 | .7133 | 92.688 | 42.038 |
| Regurgitation | | | | | | | | |
| Present | Mean | 114.87* | 5.93 | 8.453 | 133.67 | 4.927 | 227.47 | 132.47 |
| | Std. Deviation | 29.969 | 2.700 | 2.0959 | 6.008 | .7353 | 101.064 | 44.231 |
| Absent | Mean | 92.03 | 4.96 | 7.751 | 134.97 | 4.714 | 184.23 | 128.94 |
| | Std. Deviation | 35.819 | 3.479 | 1.8310 | 5.716 | .7397 | 87.344 | 40.901 |
| Valvular Calcification | | | | | | | | |
| Present | Mean | 125.00 | 8.00 | 9.100 | 128.00 | 6.000 | 99.00 | 90.00 |
| | Std. Deviation | | | | | | | |
| Absent | Mean | 98.35 | 5.20 | 7.939 | 134.71 | 4.753 | 199.20 | 130.82 |
| | Std. Deviation | 35.641 | 3.277 | 1.9329 | 5.755 | .7234 | 92.665 | 41.524 |

*p<0.05

The age of CKD patients ranged from 16 to 83 year with a mean of 45.57 year. The age distribution in the control group was comparable (18 to 80 year) with a mean of 50.8 year. The CKD study group comprised 60% males and 40% females. The control group comprised 66.6% males and 33.3% females. The duration of symptoms ranged from 3-38 months with a mean duration of 10.6 months. Hypertensive nephropathy was the most common etiology (36%). Other etiologies were chronic glomerulonephritis (32%), obstructive causes (4%), interstitial nephritis (4%) and others or unknown including polycystic kidney disease 24%. Breathlessness on exertion was the most common symptom (76%). Other symptoms were

edema, anorexia, nausea, vomiting, weakness, weight loss, nocturia and oliguria. Pallor was the most common sign (84%), followed by edema (68%), hypertension (54%), murmurs (16%) and pericardial rub (12%). All the patients of CKD in the study group were anemic with Hemoglobin levels ranging from 3.2 to 12.1 gm/dl. The serum creatinine levels in the study group ranged from 2.6 to 10.9 mg/dl with a mean of 4.88 mg/dl.

There was significant high incidence of LVH, Diastolic dysfunction, Systolic Dysfunction, Pericardial effusion and left ventricular dialatation in cases as compared to controls (table 1). The LVH correlated strongly with hypertension in the study group ($p < 0.05$) (Table 2). No significant association was found between LVH and diastolic dysfunction ($p < 0.05$) (Table 3).

The mean ejection fraction in our study group was 60.4% as against 62.66% in controls. There was no significant association of left ventricular ejection fraction with hypertension. An insignificant decrease in EF was found in CKD cases in comparison to controls.

Significant difference in mean Hb was observed in CKD patients with LVH. Patients with Systolic dysfunction had significant lower hemoglobin and higher cholesterol and triglyceride levels as compared to patients without systolic dysfunction. No difference in mean blood urea, serum creatinine, serum sodium, serum potassium, hemoglobin and cholesterol was observed in patients with or without diastolic dysfunction and Valvular Calcification. CKD patients with lower pericardial effusion had lower Hb as compared to CKD patients without pericardial effusion (Table 4).

## DISCUSSION

Changes in cardiac structure and function detected by echocardiography are common in patients with CKD undergoing hemodialysis, and have been recognized as key outcome predictors. Age of the patients of CKD in our study ranged from 16 to 83 years. The majority of the patients belonged to the age group 45 to 54 years (34%). It was comparable to the studies of D'Cruz *et al.;* [11], Greaves *et al.;* [12], and Levin *et al.;* [12] and Paoletti *et al.;* [13]. The male female ratio was 1.5:1. It was comparable to the studies of Foley *et al.;* [14], Owen *et al.;* [15], Caires G *et al.;* [16] and Manes *et al.;* [17].

In our study, the duration of symptoms of CKD ranged from 3 to 38 months with a mean duration of 10.6 months. Maximum patients (62%) had duration of 3 to 10 months. This finding was comparable with the study of Achari *et al.;* [18] and Kleiger *et al.;* [19].

The commonest etiology of CKD in our study was found to be hypertensive nephropathy (36%) followed closely by chronic glomerulonephritis (32%). This is consistent with studies of Andrea Galassi *et al.;* [20] (Hypertensive nephropathy 37.7%), Greaves *et al.;* [9] (CGN in 24%), Levin *et al.;* [13] (CGN in 30%). Interstitial nephritis was found in 4% and obstructive uropathy also in 4%. Other causes included polycystic kidney disease (2%). Cause remained unknown in the remaining cases.

In the present study, dyspnoea on exertion was the most common symptom (76%) with peripheral edema being the next common (68%). Achari *et al.;* [18] studied CKD patients and found edema as the most common symptoms (55%). Satyanarayan *et al.;* [21] studied 50 CKD cases and found breathlessness in 78%. Our results were comparable with all the above studies. The other symptoms in the cases of CKD in the present study included anorexia, nausea, vomiting, weakness, weight loss, oliguria and nocturia.

In the present study, pallor was the most common finding (84%) followed by pedal edema (68%) and hypertension (54%). The cardiac murmurs were found in 28% and pericardial rub in 12%. Our study correlated with these studies. Satyanarayan *et al.;* [21] found hypertension in 40% cases. Paoletti *et al.;* [13] reported hypertension in 66%. Kossowska *et al.;* [22] reported hypertension in 75% cases. Achari *et al.;* [18] reported anemia in the all patients, and hypertension in 90% patients. This very high incidence of hypertension did not correlate with our study. This could be due to differences in the etiological factors in the two studies.

Kleiger *et al.;* [19] reported anemia in virtually each of their 39 patients. Significantly ($p<0.001$) lower level of Hemoglobin is present in study group in comparison to controls. We found significant association between decrease in hemoglobin levels and pericardial effusion as well as with systolic dysfunction. Kleiger *et al.;* [19] reported that a decrease in hemoglobin was independently associated with the presence of LV dilatation.

The blood urea levels in CKD study group ranged from 51 to 198 mg/dL with a mean value of 99 mg/dL. The serum creatinine levels in our CKD study group ranged from 2.6 to 10.9 mg/dL with a mean value of 4.88 mg/dL. Achari *et al.;* [18] showed blood urea levels ranging from 55 to 165 mg/dL and serum creatinine ranging from 2.1 to 10.5 mg/dL. Nand *et al.;* [23] reported serum creatinine value of 6.57 mg/dL. Pehrrson *et al.;* [24] had serum creatinine levels ranging from 4.5 to 17 mg/dL. These results were comparable with the results of our study. Dyslipidemia was present in 42% of CKD cases and 16.6% of controls. There was a significant increase in dyslipidemia in CKD cases as compared to control ($p < 0.01$). It is comparable with

the study of Shoji *et al.;* [25] in 2001 which showed dyslipidemia in >40% cases.

38% patients of CKD showed left ventricular hypertrophy (LVH) on ECG while 16% showed ST-T changes (suggestive of ischaemia in 6% cases and pericarditis in 12% cases) and 10% showed various conduction abnormalities. Of the patients showing left ventricular hypertrophy, majority (13 patients) were hypertensive. Niwa *et al.;* [26] found that LVH was the most frequent (47%) abnormality in the ECG of CKD patients. Satyanarayan *et al.;* [21] found left ventricular hypertrophy in 66% patients and arrhythmias in 16% patients. Kossowska *et al.;* [22] showed ST-T changes in 8% cases. Our study correlated with these studies.

In the present study we found pericardial effusion in 15 patients (30%). Tamponade was not present in any of the patient. One patient showed evidence of constrictive pericarditis. Highly significant increase in pericardial effusion was found in CKD patients in comparison to controls in our study ($p<0.0001$). Ermolenko *et al.;* [27] 1975 reported pericarditis in 27% cases and Chabbra *et al.;* [28] concluded that clinically pericardial effusion was present in 13% cases while on echocardiography, there was effusion in 57% cases. These findings were similar to the present study which indicates that echocardiography is a sensitive means to detect pericardial effusion. Rutsky *et al.;* [29] reported 20% incidence of pericardial effusion. Menon *et al.;* [30] reported 32% incidence in their study. In our study, no statistically significant association could be found between pericardial effusion and blood urea levels ($p>0.05$), serum creatinine levels ($p>0.05$), serum sodium level ($p>0.05$), serum potassium level ($p>0.05$), hemoglobin levels or duration of CKD ($p>0.05$).

In our CKD study group, LVH was detected in 30 patients (60%) of who 20 was hypertensive and 10 was no hypertensive. The mean left ventricular posterior wall thickness in hypertensive cases was 13.38 mm as compared to 11.93 mm in no hypertensive cases. LVH was detected in 5 (16.6%) of control group, of whom 4 were hypertensive. Parfrey *et al.;* [31] noticed LVH in 42% of CKD cases while Kossowska [23] in found LVH in 78% patients. In our study LVH was found to have a statistically significant association with hypertension ($p<0.05$) in CKD patients. Ikaheimo *et al.;* [32] found left ventricular posterior wall thickness significantly more in hypertensive uremic patients as compared to normotensive uremic patients. Facchin *et al.;* [33] found significant correlation between Hypertension and left ventricular posterior wall and interventricular septal wall thickness and left ventricular mass in uremic patients. Our findings match with the above studies. No correlation of LVH could be found with blood urea levels ($p>0.05$), serum creatinine levels ($p>0.05$), serum sodium levels ($p>0.05$), serum potassium level ($p>0.05$). Similar to our study Facchin *et al.;* [33] did not find a correlation of LVH with serum creatinine, blood urea levels and with diastolic dysfunction. In our study 14% of uremic patients had systolic dysfunction. This finding correlated with the study of Foley [14] in (Systolic dysfunction in 15%), Parfrey [31] (16%) and Shutov [34] (22.9%) has no correlation of systolic dysfunction as found in our study with blood urea ($p>0.05$), serum creatinine levels ($p>0.05$), serum sodium ($p>0.05$), sodium potassium ($p>0.05$). In the present study, diastolic dysfunction was found in 46% of CKD patients as against 6.6% of the controls. Manes *et al.;* [17] showed 87% diastolic dysfunction. Our study correlated with these studies. No significant association was found between hypertension and diastolic function or between left ventricular hypertrophy and diastolic dysfunction in uremic cases as well as in controls. Huting *et al.;* [35] found a progressive deterioration of left ventricular diastolic function in CKD patients on hemodialysis. Gupta *et al.;* [36] showed that left ventricular diastolic parameters are significantly altered in End Stage Renal Disease patients and they improved after hemodialysis. Facchin *et al.;* [33] found no significant relationship between left ventricular mass and diastolic dysfunction. There was also no significant difference in diastolic function in CKD normotensive as against CKD Hypertensive patients. Schroeder *et al.;* [37] found that left ventricular diastolic parameters were impaired in cases of CKD due to chronic glomerulonephritis and there was no significant correlation between hypertension and left ventricular diastolic function. Our study correlated well with the above studies. This indicated that there were factors other than left ventricular hypertrophy and hypertension responsible for causing diastolic dysfunction in CKD patients. Gupta *et al.;* [36] in 1993 studied LV diastolic dysfunction in 21 patients with end stage renal disease by Doppler and demonstrated that patients with ESRD have impaired diastolic function that improves with hemodialysis. Our study correlated with the above studies.

Dilated left ventricle was present in 20% of CKD patients in our study as against only 3.3% in controls. Parfrey *et al.;* [31] reported 9% incidence of left ventricular dilatation in CRF patients while Achari *et al.;* [18] reported 4% incidence. No correlation could be found between left ventricular dilatation and various biochemical parameters. In the present study, cardiac calcification was found in 1 patient (2%). This was comparable with Achari *et al.;* [18] who reported a 2% incidence of cardiac valvular calcification in CKD patients.

D'Cruz *et al.;* [11] reported calcification in 30% cases. No statistically significant association could be found between cardiac valvular calcification and the

various biochemical markers (including blood urea, serum creatinine, serum sodium, serum potassium, and hemoglobin. Valvular regurgitation was reported in 14 patients (28%) in our study by echocardiography. Clinically 8 patients (16%) had evidence of systolic murmurs suggestive of regurgitant lesions. Thus echocardiography was a more sensitive technique for determining valvular regurgitation in CKD patients. No statistically significant association could be found between valvular regurgitation and the various biochemical markers (including blood urea, serum creatinine, serum sodium, serum potassium, and hemoglobin) or duration of disease ($p>0.05$).

**CONCLUSION:**

There is a highly increased risk of development of cardiovascular abnormalities during the course of CKD that contributed significantly to the morbidity and mortality of these patients. Echocardiography is a safe and sensitive noninvasive technique to assess the various cardiovascular abnormalities, compared to clinical or electrocardiographic examination that has a low sensitivity. In our study, CKD patients were found to have highly increased incidence of pericardial effusion, LVH, diastolic dysfunction, valvular regurgitation and left ventricular dilatation. Diastolic dysfunction significantly correlated with blood urea levels, serum creatinine levels and decrease in hemoglobin levels. LV dilatation correlated with decrease in Hemoglobin levels.LVH was related to the presence of hypertension in CKD cases while diastolic dysfunction was not found to correlate with hypertension or LVH, which indicated that many other factors were responsible in causing diastolic dysfunction. Thus, timely identification and treatment of these cardiovascular abnormalities could alter the course of the disease and help in reducing morbidity and mortality of CKD patients.

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