Prevalence and predictors of left ventricular dysfunction among patients with chronic kidney disease attending Muhimbili National Hospital in Tanzania – a cross-sectional study

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Purpose: Chronic kidney disease (CKD) is prevalent in sub-Saharan Africa and is a significant cause of mortality, which may result from kidney failure or congestive heart failure – a frequent complication of CKD. There is however scarcity of documented literature on the magnitude and associated factors of echocardiographically determined left ventricular (LV) dysfunction among CKD patients in Tanzania.

Patients and methods: A prospective cross-sectional study was conducted from May 2014 to January 2015 at Muhimbili National Hospital (MNH) in Dar es Salaam, Tanzania. Patients ≥ 18 years with CKD were consecutively enrolled. Clinical characteristics, cardiovascular risk profiles, and laboratory findings including serum creatinine, urea, hemoglobin, and cholesterol levels were collected. Echocardiography was performed to assess LV function using standard criteria.

Results: One hundred and ninety-one CKD patients fulfilled the inclusion criteria. The mean $\pm$ SD age was 48 ± 13 years, and 54.5% were men. A total of 98.4% of the patients were hypertensive, and diabetes was present in 22.8% while 97.9% had end-stage renal disease. The prevalence of LV systolic and diastolic dysfunction was 16.2% and 68.6%, respectively. A clinical finding of heart failure was the only independent predictor of LV systolic dysfunction (odds ratio [OR] = 2.9, $p = 0.012$), while independent predictors of LV diastolic dysfunction were anemia (OR = 4.9, $p = 0.01$), severe hypertension (OR = 9.2, $p = 0.001$), and female gender (OR = 1.7, $p = 0.002$).

Conclusion: LV dysfunction is prevalent among CKD patients seen at MNH and is associated with clinical heart failure, anemia, severe hypertension, and female gender. Echocardiography should be performed in patients with CKD to detect overt or subclinical LV dysfunction.

Keywords: chronic kidney disease, left ventricular dysfunction, sub-Saharan Africa

Introduction

The cardiovascular system is closely related to functions of the kidneys, and it is well known that impairment of kidney function can affect cardiac performance leading to heart failure, which consequently worsens kidney function.^{1,2} The fact that impairment of one component of the cardiorenal system aggravates dysfunction of the other is clinically very important, and there is evidence that treating congestive heart failure can prevent progression to chronic kidney disease (CKD) and vice versa.^{3,4} Cardiac failure and CKD share a number of common risk factors and pathophysiological pathways such as activation of the renin–angiotensin–aldosterone system and

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sympathetic nervous system, inflammation, and increased oxidative stress.¹

Patients with CKD and congestive heart failure have worse survival when compared to patients with CKD but without congestive heart failure, irrespective of other clinical parameters. In a large prospective study by Harnett et al, patients with CKD and congestive heart failure had a mean survival of only 36 months compared with 62-month survival of patients with CKD without heart failure.⁵ Furthermore, the risk of mortality was increased by 50% after a 3-month follow-up of CKD patients who had congestive heart failure when starting dialysis as compared to CKD patients without congestive heart failure.⁶

In sub-Saharan Africa, CKD is very prevalent, and in this part of the world, the disease characteristically affects young adults in their productive years, causing significant morbidity and mortality.^{7,8} When compared to Western cohorts, sub-Saharan patients with CKD have more severe disease and show fast progression to end-stage renal disease (ESRD) and death,^{7,8} which may partly be explained by the high prevalence of heart failure among sub-Saharan CKD patients.^{9,10}

Subclinical systolic and diastolic left ventricular (LV) dysfunction often precedes symptomatic heart failure, and the former can be detected by use of echocardiography. However, only few echocardiographic studies have been conducted among CKD patients in sub-Saharan Africa,^{11,12} and there is scarcity of documented literature on the magnitude and associated factors of echocardiographically determined LV dysfunction. The current study was therefore set out to determine the magnitude of LV dysfunction and associated factors in patients with CKD attending care and treatment at a tertiary hospital in Dar es Salaam, Tanzania.

Patients and methods

Study design

This is a descriptive cross-sectional study.

Study site

The study was conducted at the nephrology clinic and nephrology wards of Muhimbili National Hospital (MNH) in Dar es Salaam, Tanzania. The hospital is the topmost referral health facility in the country and serves as the teaching hospital for Muhimbili University of Health and Allied Sciences. It receives patients from lower-level hospitals all over the country.

Study population

The study included patients aged ≥ 18 years with a confirmed diagnosis of CKD as per Kidney Disease Outcomes Quality Initiative criteria,¹³ regardless of their primary causes. Patients known to have primary cardiac diseases, such as rheumatic heart disease, were excluded. Patients were consecutively enrolled in the study as they attended the nephrology outpatients clinic as well as when admitted at the nephrology wards. The study was conducted from May 2014 to January 2015.

Data collection procedure

Clinical data

A structured questionnaire was used to collect information on demographic parameters, clinical presentation, history of previous heart failure, and other cardiovascular risk factors including smoking, history of diabetes, and family history of cardiovascular diseases.

Height was measured using a stadiometer (CEO123; Seca, Hamburg, Germany), with subjects wearing no shoes and averaged to the nearest centimeter. Weight was measured by a weighing scale (Momert, Dunaujvaros, Hungary) and recorded in kilogram. Body mass index (BMI) was calculated as weight in kilogram/height in meter squared, and obesity was defined as BMI ≥ 30 kg/m².

For all participants, a thorough history and physical examination was done. Anemia, uremic signs, and signs of heart failure were examined and recorded. Patients were categorized as having heart failure clinically when they had a combination of shortness of breath, orthopnea, cough, or paroxysmal nocturnal dyspnea with an S₃ gallop on auscultation. Current medications used by the patients were recorded.

Blood pressure was taken using a mercury sphygmomanometer. This was done when the patient has had a 5-minute rest and seated comfortably in a chair with the back and left arm supported, legs uncrossed, and the upper arm at the level of the right atrium. The first and fifth Korotkoff sounds were taken as systolic and diastolic blood pressures, respectively. Three measurements were taken, and the average of the last two was recorded as the patient's blood pressure. Hypertension was defined as systolic blood pressure of ≥ 140 mmHg and/or diastolic blood pressure of ≥ 90 mmHg or use of antihypertensive medications, and was categorized as grade 1 (140–159/90–99 mmHg), grade 2 (160–179/100–109 mmHg), and grade 3 ($\geq 180/\geq 110$ mmHg) according to the European Society of Cardiology guidelines.¹⁴

In this study, inclusion criteria included creatinine level that was used to classify patients as having CKD; for patients who were not on dialysis, the latest creatinine levels were taken, and for patients on dialysis, pre-dialysis creatinine levels were taken. A fasting blood sample was taken and analyzed for cholesterol panel, blood glucose, and full blood count. Urine dipstick (Medi Test Combi 11) test was done to determine overt albuminuria. Serum creatinine levels were used to calculate estimated glomerular filtration rate (eGFR), using the Modification of Diet in the Renal Disease (MDRD) equation.¹⁵ The validated MDRD formula is expressed as

$$\text{eGFR (mL/min/1.73 m}^2\text{)} = 175 \times (\text{Scr}/88.4)^{-1.154} \times (\text{age})^{-0.203} \times 0.742 \text{ (if female)} \times 1.212$$

Only patients with an eGFR of <60 mL/min/1.73 m² (i.e., CKD stage ≥3) were included in this study.

Echocardiogram

The echocardiographic examinations were performed at MNH echocardiography laboratory. General Electric VIVID S5 machine was used for examination. Images from two-dimensional, motion-mode, and Doppler (color and tissue) recordings were taken. All measurements were taken during the echocardiographic examination, and data were retrieved from computer-generated reports inbuilt in the echocardiogram machine. The obtained data were then transferred to precoded recording papers for each patient. Images were also

stored on the echocardiogram machine hard disk as well as external hard disk for later rereading, when this was needed.

All echocardiographic examinations were performed by one experienced cardiologist (PC). LV ejection fraction was determined using the Teicholz method from motion-mode guided parasternal long-axis images of the left ventricle (Figure 1) and was taken as a measure of LV systolic function. Ejection fraction of <50% was considered as systolic dysfunction.¹⁶

LV filling was recorded at the level of the mitral leaflets tips. The leading edge of the mitral flow pattern was traced to derive peak early (E) and atrial (A) velocities, E/A ratio, and E-deceleration time (Figure 2). Isovolumic relaxation time was measured from the leading edge of the aortic valve closure spike to the leading edge of the mitral valve opening spike. The medial early diastolic mitral annular velocity (E') was measured by spectral tissue Doppler imaging in apical four-chamber views (Figure 3). The ratio of E to E' velocity (E/E' ratio) was taken as an estimation of LV filling pressure and was considered increased when it was ≥15.¹⁷ LV diastolic dysfunction was defined as mild (impaired relaxation), moderate (pseudonormal pattern), and severe (restrictive pattern) based on transmitral inflow in combination with the diastolic mitral annular velocities.^{18,19}

LV mass was calculated using the anatomically validated formula by Devereux et al²⁰ and indexed to body surface area. LV hypertrophy was defined as LV mass index >95 kg/m² in women and >115 kg/m² in men.¹⁶

Figure 1 Motion-mode guided parasternal long-axis image of the left ventricle.

Figure 2 Left ventricular filling recorded at the level of the mitral leaflets tips.

Figure 3 Medial early diastolic mitral annular velocity (E') measured by spectral tissue Doppler imaging in apical four-chamber view.

Data handling and analysis

Data entry and analysis were done using SPSS version 20. Data is presented as mean $\pm$ SD for continuous variables and as percentages for categorical variables. Groups of patients were compared using χ^2 test, unpaired Student's

t-test, or one-way analysis of variance (ANOVA) as appropriate. Uni- and multivariate logistic regression analyses were performed to identify predictors of LV systolic and diastolic dysfunction. The risk of having LV systolic or diastolic dysfunction is expressed as odds ratio (OR) with

95% confidence interval. A p -value of <0.05 was considered statistically significant.

Ethical consideration

The study was conducted in accordance with the Declaration of Helsinki of research on human subjects. Ethical clearance was obtained from the directorate of research and publication at Muhimbili University of Health and Allied Sciences. All patients signed an informed consent form before any data were collected.

Results

Demographic and clinical characteristics of the study population

During the 9-month study duration, a total of 191 patients fulfilled the inclusion criteria and were enrolled in the study. There were 104 (54.5%) men and 87 (45.5%) women. The mean $\pm$ SD age of the total study population was 48 ± 13 years (range 18–85 years). History of hypertension was self-reported by 98.4% of the total population, and the mean duration of hypertension was 4.7 ± 4.6 years. Sixty-two patients were on dialysis therapy (61 on hemodialysis and one on peritoneal dialysis). Patients on dialysis did not differ from those not on dialysis by age, gender, or other sociodemographic factors, all $p > 0.05$. Family history of cardiovascular disease was present in 46 (24.1%) of the total population studied. The mean systolic blood pressure was found to be significantly higher among men (157 mmHg versus 149 mmHg, $p = 0.02$), but there was no significant

difference in the mean diastolic blood pressures between men and women (93 mmHg versus 90 mmHg, $p = 0.137$). Table 1 summarizes the demographic and clinical characteristics of the study population.

Palpitation was the most common cardiovascular symptom among patients with CKD attending MNH nephrology units, being present in 87.8% of the total population, followed by lower limbs edema (78.7%) and shortness of breath (64.5%). The most common uremic symptoms reported were vomiting (66%), hiccups (42.1%), and body itching (18.3%) (Figure 4). Fifty-three (27.7%) patients were found to have heart failure clinically, that is, following their symptoms and physical findings.

Majority of the patients were using a calcium channel blocker (85.3%) for treatment of hypertension. Over two-thirds were on a diuretic, and 46.7% were on a beta

Table 1 Demographic and clinical characteristics of patients with CKD attending MNH

Characteristics	N = 191
Age (years)	48 ± 13
Body mass index (kg/m ²)	23 ± 3
Proportion with obesity, n (%)	7 (3.7)
Systolic blood pressure (mmHg)	154 ± 24
Diastolic blood pressure (mmHg)	92 ± 14
Proportion with hypertension, n (%)	188 (98.4)
Proportion with diabetes, n (%)	45 (22.8)
History of smoking, n (%)	21 (10.7)

Note: Results are mean $\pm$ SD, unless stated otherwise.

Abbreviations: CKD, chronic kidney disease; MNH, Muhimbili National Hospital.

Figure 4 Symptoms among chronic kidney disease patients attending Muhimbili National Hospital

Abbreviations: SOB, shortness of breath; PND, paroxysmal nocturnal dyspnea.

blocker. Notably, angiotensin-converting enzyme inhibitor and angiotensin receptor blockers were infrequently used, being prescribed in 3.6% and 16.8%, respectively (Figure 5).

Laboratory findings of the study patients

Patients in this study population had markedly raised mean serum creatinine levels ($1173 \pm 688 \mu\text{mol/L}$, range 318–5802 $\mu\text{mol/L}$, median 968 $\mu\text{mol/L}$, $Q_1 = 776 \mu\text{mol/L}$, and $Q_3 = 1310 \mu\text{mol/L}$). Likewise, the mean serum urea was high ($28 \pm 12 \mu\text{mol/L}$) (Table 2). Both mean serum creatinine ($1305 \mu\text{mol/L}$ versus $1009 \mu\text{mol/L}$) and urea ($31 \mu\text{mol/L}$ versus $26 \mu\text{mol/L}$) levels were significantly higher in men than women, $p < 0.05$ for both. The proportions of patients with CKD of stages 3, 4, and 5 were 0%, 2.1%, and 97.9%, respectively, in the total population.

The mean hemoglobin level of the total population was $9.1 \pm 2.4 \text{ g/dL}$, and anemia, defined as hemoglobin $<13 \text{ g/dL}$ in men and $<12 \text{ g/dL}$ in women, was present in 91.1% of the total population studied (Table 2). There was no statistically significant difference in mean serum glucose, cholesterol, high-density lipoprotein, low-density lipoprotein, and triglycerides between men and women.

Echocardiographic findings of the study patients

The mean LV interventricular septum and posterior wall thickness were 1.53 cm and 1.45 cm, respectively, in the total population and were significantly higher in men than women, $p < 0.01$ for both (Table 3). LV hypertrophy was present in

Table 2 Laboratory findings of patients with CKD attending MNH

Characteristics	N = 191
Blood glucose (mmol/L)	5.4 ± 2.7
Total cholesterol (mmol/L)	4.7 ± 1.5
HDL-cholesterol (mmol/L)	1.08 ± 0.5
LDL-cholesterol (mmol/L)	3.03 ± 1.3
Serum triglycerides (mmol/L)	1.39 ± 0.95
Hypercholesterolemia, n (%)	53 (27.7)
Hemoglobin (g/dL)	9.1 ± 2.4
Proportion with anemia, n (%)	174 (91.1)
Serum urea ($\mu\text{mol/L}$)	28 ± 12
Serum creatinine ($\mu\text{mol/L}$)	1173 ± 688
Estimated GFR (mL/min/1.73 m^2)	6.93 ± 3.4

Note: Results are mean $\pm$ SD unless stated otherwise.

Abbreviations: CKD, chronic kidney disease; MNH, Muhimbili National Hospital; HDL, high-density lipoprotein; LDL, low-density lipoprotein; GFR, glomerular filtration rate.

all but two patients in the total population. Patients without LV hypertrophy were both females.

In the total population, the mean $\pm$ SD fractional shortening, ejection fraction, and stroke volume were $35 \pm 7\%$, $63 \pm 10\%$, and $67 \pm 21 \text{ mL}$, respectively (Table 3). Thirty-one (16.2%) patients were found to have LV systolic dysfunction.

The E/A ratio was significantly higher in men (1.32 ± 0.7) than women (1.09 ± 0.5), $p < 0.01$ (Table 4). Both the E-deceleration time and the isovolumic relaxation time did not differ between men and women (Table 4). The mean E/E' ratio was 14.0 ± 5.4 in the total population, and it did not differ between men and women. Seventy-two patients

Figure 5 Cardiac medications used by patients with chronic kidney disease attending Muhimbili National Hospital.

Abbreviations: CCB, calcium channel blocker; ARB, angiotensin receptor blocker; ACE-I, angiotensin-converting enzyme inhibitor.

Table 3 Echocardiographic systolic parameters in CKD patients attending MNH

Variables	Total, N = 191	Male, n = 104	Female, n = 87	p-value
Interventricular septum in diastole (cm)	1.53 ± 0.27	1.59 ± 0.25	1.45 ± 0.28	0.001
LV posterior wall in diastole (cm)	1.45 ± 0.4	1.5 ± 0.3	1.4 ± 0.4	0.001
Interventricular septum in systole (cm)	1.9 ± 0.3	2.0 ± 0.4	1.8 ± 0.3	<0.001
LV posterior wall in systole (cm)	1.8 ± 0.4	1.97 ± 0.4	1.7 ± 0.4	<0.001
LV internal diameter in diastole (cm)	4.7 ± 0.8	4.9 ± 0.7	4.5 ± 0.8	<0.001
LV internal diameter in systole (cm)	3.1 ± 0.7	3.25 ± 0.7	3.0 ± 0.8	0.019
LV mass index (g/m ²)	249 ± 79	273 ± 71	221 ± 80	0.438
Proportion with LV hypertrophy, n (%)	189 (99)	104 (100)	85 (97.7)	0.206
Fractional shortening (%)	35 ± 7	34 ± 7	35 ± 8	0.404
Ejection fraction (%)	63 ± 10	63 ± 10	64 ± 10	0.308
Proportion with reduced EF, n (%)	31 (16.2)	19 (18.3)	12 (13.8)	0.403
End diastolic volume (mL)	108 ± 36	118 ± 37	94 ± 30	<0.001
End systolic volume (mL)	41 ± 21	45 ± 23	35 ± 18	0.001
Stroke volume (mL)	67 ± 21	73 ± 21	60 ± 17	<0.001

Note: Results are mean ± SD unless stated otherwise.

Abbreviations: CKD, chronic kidney disease; MNH, Muhimbili National Hospital; LV, left ventricular; EF, ejection fraction.

Table 4 Echocardiographic diastolic parameters in CKD patients attending MNH

Variables	Total, N = 191	Male, n = 104	Female, n = 87	p-value
E (m/s)	0.79 ± 0.2	0.8 ± 0.3	0.77 ± 0.2	0.333
A (m/s)	0.71 ± 0.2	0.66 ± 0.2	0.77 ± 0.2	0.001
E/A ratio	1.21 ± 0.6	1.32 ± 0.7	1.09 ± 0.5	0.006
E-deceleration time (ms)	197 ± 62	193 ± 66	200 ± 56	0.470
Isovolumic relaxation time (ms)	100 ± 52	103 ± 68	95 ± 18	0.276
E' (cm/s)	5.98 ± 1.59	6.12 ± 1.51	5.83 ± 1.69	0.215
E/E' ratio	14.0 ± 5.4	13.8 ± 4.9	14.3 ± 5.9	0.506
Proportion with E/E' ratio ≥15, n (%)	72 (37.7)	41 (39.4)	31 (35.6)	0.590

Abbreviations: CKD, chronic kidney disease; MNH, Muhimbili National Hospital; E, early velocity; A, atrial velocity; E', medial early diastolic mitral annular velocity.

(37.7%) had raised E/E' ratio indicating raised LV filling pressures (Table 4).

Sixty (31.4%) patients were found to have normal LV diastolic function, while 131 (68.6%) had different degrees of LV diastolic dysfunction, of which 34% had impaired relaxation (grade 1 LV dysfunction), 20.4% had pseudonormal LV diastolic pattern (grade 2 LV dysfunction), and 14.1% had restrictive LV diastolic pattern (grade 3 LV dysfunction) (Figure 6).

The proportions of patients with systolic, diastolic, and both dysfunctions in men, women, and in the total population are summarized in Figure 7.

Predictors of LV dysfunction

Predictors of LV systolic dysfunction

Clinical diagnosis of heart failure was independently associated with presence of LV systolic dysfunction (OR = 2.9, $p = 0.012$) in multivariate logistic regression analysis (Table 5). Other variables included in the regression model were gender, age >45 years, diabetes status, smoking, proteinuria, anemia and hypertension severity (Table 5).

■ Normal ■ Impaired relaxation ■ Pseudonormal ■ Restrictive

Figure 6 Left ventricular diastolic patterns in the total population.

Predictors of LV diastolic dysfunction

In multivariate logistic regression analysis that included gender, obesity, diabetes status, smoking, clinical diagnosis of heart failure, presence of proteinuria, presence of anemia, and hypertension severity, female gender (OR = 1.71, $p = 0.002$), presence of anemia (OR = 4.9, $p = 0.01$), and having severe hypertension (OR = 9.18, $p = 0.001$) were

Figure 7 Prevalence of left ventricular systolic, diastolic, and both dysfunctions among patients with chronic kidney patients attending Muhimbili National Hospital.

Table 5 Independent predictors of LV systolic dysfunction obtained by multivariate logistic regression analysis in the total proportion

Variables	Odds ratio	95% confidence interval	p-value
Male gender	1.065	0.453–2.643	0.888
Age >45 years	0.933	0.393–2.324	0.877
Diabetes mellitus	1.449	0.516–3.735	0.454
Smoking	1.569	0.435–5.325	0.478
Clinical heart failure	2.90	1.262–6.578	0.012
Anemia	1.406	0.275–7.206	0.682
Proteinuria	1.059	0.453–2.475	0.895
Hypertension severity			
Controlled/normal (constant)	–	–	–
Mild hypertension	0.983	0.314–3.081	0.976
Moderate hypertension	1.494	0.452–4.941	0.510
Severe hypertension	1.902	0.556–6.501	0.682

Abbreviation: LV, left ventricular.

Table 6 Independent predictors of LV diastolic dysfunction obtained by multivariate logistic regression analysis in the total population

Variables	Odds ratio	95% confidence interval	p-value
Women	1.710	1.532–6.782	0.002
Obesity	4.508	0.299–68.065	0.277
Diabetes mellitus	0.736	0.325–1.667	0.463
Smoking	1.419	0.452–4.448	0.549
Clinical heart failure	1.389	0.624–3.091	0.421
Anemia	4.935	1.476–16.505	0.010
Proteinuria	1.974	0.945–4.121	0.070
Hypertension severity			
Controlled/normal (constant)	–	–	–
Mild hypertension	1.747	0.747–4.088	0.198
Moderate hypertension	1.444	0.561–3.712	0.442
Severe hypertension	9.188	2.390–35.319	0.001

Abbreviation: LV, left ventricular.

significantly independently associated with a finding of LV diastolic dysfunction (Table 6).

Discussion

The present study was done to document the magnitude and covariates of LV dysfunction in patients with CKD attending MNH. The prevalence of LV systolic dysfunction was found to be 16.2% and that of LV diastolic dysfunction to be 68.6% in this population of otherwise advanced CKD patients, most of whom (97.9%) were having ESRD. The study is among the few to report on LV function among CKD patients in the sub-Saharan African region and adds to the existing knowledge on cardiovascular disease in patients with CKD.

The finding that LV systolic dysfunction was present in 16.2% of this CKD population is similar to that obtained by Arodiwe et al in patients with CKD attending care and treatment at Enugu teaching hospital in Nigeria.¹² Of note, the prevalence of LV systolic dysfunction in that study was 15.1%. The similarities are explained by the similar study populations between the current study and the study by Arodiwe et al in terms of the CKD severity and age and the fact that both studies were done at a university referral hospital, most likely receiving CKD patients with ESRD. Although the study by Foley et al involved a different ethnic population, mainly Caucasians in Canada, the prevalence of echocardiographic LV systolic dysfunction was 15%, also similar to the present study.²¹

Other studies found LV systolic dysfunction to be independently associated with mean blood pressure and stage of CKD.^{12,21} In the present study, blood pressure severity, duration of hypertension, or CKD stage was not found to be independently associated with LV systolic dysfunction. The reason for this is most likely due to the fact that the current study population was very homogenous (97.9% had ESRD and 98.4% had hypertension), therefore lacking variability in these parameters. However, our finding that the presence of clinical heart failure predicted LV systolic dysfunction is interesting suggesting that by carefully listening and examining patients, one can predict their LV systolic function, underscoring the importance of proper history taking and physical examination to determine the presence of heart failure in patients with CKD.

The prevalence of LV diastolic dysfunction of 68.2% found in this study is also very similar to previous studies on prevalence of CKD among Africans,¹¹ as well as in different populations,²² indicating the comparability of the present study with previous reports in literature. For example, in the study by Arodiwe et al that looked at LV diastolic dysfunction in CKD patients in the same hospital in Nigeria, they found the prevalence of LV diastolic dysfunction to be 62.8%,¹¹ while the study by Hayashi et al in Sweden found a prevalence of 65%.²² Many other quoted studies have shown a prevalence of LV diastolic dysfunction of 40–66% regardless of treatment, that is, hemodialysis, peritoneal dialysis, or even after renal transplantation.^{23,24}

In this study, patients with anemia were almost five times more likely to have LV diastolic dysfunction, and the presence of severe hypertension increased the likelihood of having LV diastolic dysfunction ninefold. Moreover, female gender was more likely to have diastolic dysfunction by 70%. Other studies have found LV diastolic dysfunction to be associated with severity of hypertension as well as hypertension duration.¹¹ Gender did not predict LV diastolic dysfunction in the Nigerian study by Arodiwe et al,¹¹ but other studies in hypertensive^{25,26} as well as in general populations²⁷ have found a significant correlation between female gender and presence of LV diastolic dysfunction, similar to the present study. These results may explain the relatively higher incidence in females among patients with diastolic heart failure^{28,29} and higher cardiovascular mortality in female gender.³⁰ The reason for increased LV diastolic dysfunction in females is thought to be that women are more likely to be obese, particularly central obesity which is associated with LV diastolic dysfunction.³¹ Of note, in the current study, the OR for the association between obesity and LV diastolic

dysfunction was 4, although this was not statistically significant, most likely due to the fewer number of patients with obesity in this population.

The finding that anemia was independently associated with LV diastolic dysfunction is similar to the study by Pakfetrat et al, which reported a negative correlation between hemoglobin levels and presence of LV diastolic dysfunction in patients with CKD.³² Anemia is a known associating factor in heart failure, and in a recent study by Makubi et al, anemia was independently associated with heart failure in a cohort of heart failure patients attending care and treatment at MNH, linking the association between anemia and presence of LV dysfunction.³³

The prevalence of clinical heart failure in this study was 27.7%. This is similar to the recorded heart failure prevalence of 30% in a large population of patients with ESRD from Centers for Medicare and Medicaid Services in the US,³⁴ and similar to the 30% prevalence of pulmonary edema among CKD patients attending a tertiary health facility in Ghana.³⁵

We found in this study many demographic similarities with previous studies on CKD patients in the sub-Saharan African region. The mean age of 48 years is similar to previous studies in the region,^{9,11,36} and the present findings add to the general observation that sub-Saharan patients with CKD are younger when compared to those in the developed countries.^{37,38} Moreover, while aiming at collecting patients with CKD of stages 3–5, we found no single patient with stage 3 CKD during our 9-month data collection period; instead, only four patients had stage 4 CKD (2.1%), and the rest of the study population had ESRD (97.9%). This finding is similar to that found in other countries in the region^{9,35} confirming the observation that sub-Saharan patients with CKD present late to the tertiary health facilities where definitive diagnosis and management of CKD are offered.

The high prevalence of hypertension found in this study can be explained by the advanced CKD levels of the study subjects in this population. It is known that hypertension is almost always present with ESRD.³⁹ It cannot however be determined in this cross-sectional study whether hypertension is the cause or the outcome of CKD, although there are reasons to believe that a higher proportion of these patients may have had hypertension as the primary cause of CKD, as hypertension has been reported to be the most common cause of CKD in the sub-Saharan African region.³⁸

Study limitations

Findings from this study cannot be generalized to all health facilities in Tanzania since lower-level health facilities are

likely to receive less severe cases of CKD; however, the study gives a good overview of CKD patients at the country's topmost referral hospital. The cross-sectional nature of the study makes it impossible to confirm a causal relationship between CKD and LV dysfunction as LV dysfunction may have developed before CKD and other risk factors seen in this study population.

Conclusion

LV dysfunction is prevalent among CKD patients attending care and treatment at MNH and is associated with clinical heart failure, anemia, severe hypertension, and female gender. Echocardiography should be performed to detect overt and subclinical LV dysfunction among CKD patients.

Disclosure

The authors report no conflicts of interest in this work.

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