ORIGINAL ARTICLE

Higher prevalence of left ventricular hypertrophy and dysfunction in haemodialysis patients compared with peritoneal dialysis at a single centre in South Africa

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ABSTRACT

Background: Abnormalities of cardiac function and structure are highly prevalent among end-stage kidney disease (ESKD) patients and contribute to cardiovascular morbidity and mortality. Left ventricular hypertrophy (LVH) is one of the most common abnormalities and is associated with an increased risk for cardiac events. Despite a high burden of disease, African patients are underrepresented in the literature. Studying LVH prevalence, and risk factors in African HD and PD patients offers novel insights to inform context-specific care.

Methods: A cross-sectional study was conducted at Charlotte Maxeke Johannesburg Academic Hospital, South Africa. Echocardiography was performed on consecutive patients receiving haemodialysis (HD) and those on continuous ambulatory peritoneal dialysis (PD). LVH was diagnosed according to Framingham criteria.

Results: 128 patients were studied; 84 were on HD while 44 received PD. Prevalence of LVH was higher among the HD patients (74%) compared to PD patients (31%); P = 0.011. HD patients displayed a higher frequency of concentric LVH (P = 0.004) whereas left ventricular concentric remodelling was more frequently seen in PD patients (P = 0.023). Diastolic dysfunction was more common in HD patients (64%) compared with PD patients (46%); P = 0.041. Risk factors associated with LVMI among PD patients were anaemia (P = 0.020) and in the HD patients, male gender (P = 0.009) and systolic BP (P < 0.001).

Conclusion: Abnormalities of cardiac structure and function are highly prevalent among patients with end-stage kidney disease receiving dialysis. Routine echocardiographic evaluation should be incorporated into clinical assessment for early identification and timely initiation intervention to reduce cardiovascular morbidity and mortality.

Keywords: Left ventricular hypertrophy, haemodialysis, continuous ambulatory peritoneal dialysis.

INTRODUCTION

Cardiovascular complications are a major clinical problem in patients with end-stage kidney disease (ESKD) on dialysis [1-3]. Abnormalities of cardiac function and structure are highly prevalent among ESKD patients and contribute significantly to the increased morbidity and mortality seen in them [4]. Left ventricular hypertrophy (LVH) is one of the most common abnormalities and is associated with diastolic and systolic dysfunction [4]. It is an independent risk factor for arrhythmias, heart failure, myocardial infarction and sudden death [5]. Hence, identification and early treatment of cardiac abnormalities and LVH are of both diagnostic and prognostic importance among ESKD patients. Several factors are known to contribute to these cardiac abnormalities, some of which are CKD-related such as anaemia, abnormalities of mineral and bone metabolism, and poorly controlled hypertension among others [3]. The type of kidney replacement therapy (KRT) the patient is receiving may also contribute to the cardiovascular risk to the patient, hence studying both haemodialysis (HD) and peritoneal dialysis (PD) becomes imperative.

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Despite the availability of more refined techniques based on nuclear magnetic resonance, echocardiography has remained the most frequently used means of evaluating abnormalities in left ventricular mass and function [6]. It allows detailed examination of the pericardium, valvular apparatus, atria and ventricles, quantification of LVM and left ventricular function in addition to being simple, non-invasive and widely available. Cardiovascular disease is the leading cause of morbidity and mortality among patients receiving kidney replacement therapy, with LVH and left ventricular (LV) systolic and diastolic dysfunction representing the predominant structural and functional cardiac abnormalities in this population, but data representing African patents is lacking in the literature. In this study, we report on the prevalence of LVH, its patterns as well as LV function using echocardiography among a predominantly Black African population with ESKD on dialysis (maintenance HD and CAPD).

METHODS

This cross-sectional study was conducted at Charlotte Maxeke Johannesburg Academic Hospital in South Africa. We examined 128 patients with ESKD who had been on dialysis for three months or more. Patients were categorised into two groups based on the type of KRT they were receiving. Eighty-four patients were on HD whereas 44 received chronic ambulatory peritoneal dialysis. Excluded were patients with congestive heart failure (NYHA class III and IV) and those with obvious infections. All HD patients received a 4-hour HD session three times a week using polysulfone dialysers and standard bicarbonate buffer, whereas PD patients were treated with four 2-litre exchanges of 2.5% glucose solutions per day. Blood pressure was estimated by averaging all pre-dialysis and post-dialysis blood pressure recordings taken during the month of enrolment in the study and for PD patients; blood pressure values were derived from the average of three measurements, two recorded during prior clinic visits and one taken at enrolment.

Laboratory measurements

Biochemical parameters such as haemoglobin, serum albumin, calcium, phosphorus, parathyroid hormone, urea and creatinine were analysed by standard procedures at the National Health Laboratory Service. The adequacy of dialysis was assessed by urea kinetic modelling (Kt/V).

Echocardiograms were recorded in the Cardiology Unit using the Acusson Sequoia C256 machine (Acusson Corporation, Mountain View, CA, USA) equipped with 2–4-MHz probes allowing M-mode, 2-D, and pulsed Doppler measurements. All echocardiographic measurements were performed according to the recommendations of the American Society of Echocardiography [7] and during the interdialytic days in HD patients and with an empty abdomen in PD patients. LV internal dimension measurements in end-diastole (LVEDD), and end-systole (LVESD), were taken at the level of the mitral valve leaflet tips in the parasternal long-axis view. The interventricular wall thickness during diastole (IVSTd) and posterior wall thickness during diastole (PWTd) were measured by M-mode. Left ventricular mass was calculated according to the formula described by Devereux and Reichek [8]. LVM was divided by body surface area (BSA) to calculate the LVM index (LVMI). The cut-off points for LV hypertrophy (LVH), using the American Society of Echocardiography (ASE)/European Association of Echocardiography (EAE) guidelines, are >115 g/m^2 for males and >95 g/m^2 for females [9].

Systolic function was assessed by measuring the LV ejection fraction and fractional shortening. Patients with ejection fraction <50% were considered to have systolic dysfunction. Diastolic function was assessed by the E/A ratio, which is derived by measuring flow velocities across the mitral valve by pulsed Doppler. An E/A ratio less than 1 suggests impaired relaxation of the left ventricle, indicating diastolic dysfunction.

Relative wall thickness (RWT) was used to characterise LVH in terms of eccentric and concentric LVH. RWT > 0.45 in the presence of LVH is indicative of concentric hypertrophy and eccentric hypertrophy if <0.45 in the presence of LVH [9]. An RWT of ≥0.45 with normal LVMI is considered LV remodelling.

Ethical clearance to conduct the study was obtained from the Human Ethics Committee of the University of the Witwatersrand (ethics clearance certificate numbers M080452 and M030802).

Statistics

Data were reported as mean ± standard deviation. Comparison between groups was made by Student's t-test for continuous variables and the chi-squared (χ^2) test or Fisher's exact test for categorical variables, as appropriate. The relationship between LVMI and other variables was tested using multiple linear regression analysis with age, gender, haemoglobin (Hb), smoking, systolic blood pressure (SBP), diastolic blood pressure (DBP), serum albumin, calcium, phosphate, parathyroid hormone (PTH), duration of dialysis, and treatment modality included as independent variables. All analysis was conducted using Stata version 8.0 statistical software (Stata Corporation, College Station, TX, USA).

RESULTS

Table 1 shows the baseline clinical and laboratory characteristics of the study population. Males constituted 53% and 58% of the HD and PD groups, respectively. The HD patients were older than those on PD; the respective mean ages were 40.1 ± 10.4 years and 38.1 ± 12.4 years, although the difference was not significant (P = 0.336). The HD patients had longer dialysis vintage (P = 0.0003), lower DBP (P < 0.001), lower Hb (P < 0.0001) but higher serum albumin (P = 0.015) and PTH (P < 0.001) compared with PD patients. SBP and BMI were similar in both groups. Eighteen patients (21%) reported as current smokers whereas 25 (30%) were former smokers. Fifty-seven HD patients (80%) and 27 PD patients (61%) were receiving various combinations of anti-hypertensive medications. The mean Kt/V was 1.31 ± 0.37 among patients receiving haemodialysis, compared with 1.72 ± 0.32 among those on peritoneal dialysis.

Hypertension was the most common cause of ESKD in both groups, followed by glomerulonephritis in the HD group and unknown among PD patients (Table 2).

The LVPWD, IVSD, and LVMI were significantly higher in HD patients than in PD patients (Table 3).

The prevalence of LVH was significantly higher among the HD patients (73: 87%) than in the PD patients (30: 68%); χ^2 = 6.44, P = 0.011. HD patients also had a significantly higher prevalence of concentric LVH (P = 0.004), whereas left ventricular concentric remodelling was observed in four PD patients and in none of the HD group (P = 0.023). Although eccentric LVH was observed among patients undergoing CAPD, this difference did not reach statistical significance (χ^2 = 2.25, P = 0.134).

The proportion of patients with normal echocardiographic findings was comparable between the two groups. As expected, diastolic dysfunction was more common among HD patients (P = 0.041). Systolic dysfunction was observed in 17 HD patients (20%) and 3 PD patients (7%); the difference was not statistically significant (P = 0.084); Table 4.

The presence of valvular abnormalities was similar in both groups; 33% in HD subjects and 29% among CAPD patients. In the patients on PD, 13 (29%) demonstrated various forms of valvular abnormalities, including mitral regurgitation, aortic regurgitation and various other combinations of mitral and tricuspid regurgitation. Nine patients on CAPD had pericardial effusions of variable sizes (between 0.46 cm and 1.46 cm) posteriorly. None of the patients demonstrated wall motion abnormalities. Among HD patients, the valvular abnormalities seen included: 7 (9%) with mitral valve regurgitation, 11 (15%) with tricuspid regurgitation, and aortic regurgitation (1), whereas 9 (11%) had various other combinations of mitral, tricuspid and aortic regurgitation. No HD subjects had pericardial effusion nor wall motion abnormalities, while 7 (8%) patients had calcification of their heart valves.

Table 1. Comparison of clinical and laboratory characteristics of HD and PD patients.

Parameter	Group 1 HD patients (n = 84)	Group 2 PD patients (n = 44)	P value
Age (years)	40.1 ± 10.4	38.1± 12.4	0.336
Male	44 (52%)	26 (59%)	0.469*
Duration of dialysis (months)	45.9 ± 45.1	19.5 ± 20.7	< 0.001
Body mass index (kg/m^2)	24.45 ± 5.03	24.76 ± 3.58	0.717
Systolic BP (mmHg)	147.1 ± 18.1	145 ± 28.1	0.609
Diastolic BP (mmHg)	83.94 ± 13.26	92 ± 17	< 0.001
BP >140/90 mmHg	47 (56%)	25 (57%)	0.925*
Haemoglobin (g/dL)	9.48 ± 1.49	10.99 ± 2.14	< 0.001
Serum albumin (g/L)	39.74 ± 3.96	37.5 ± 6.26	0.015
Calcium (mmol/L)	2.31 ± 0.26	2.26 ± 0.33	0.349
Phosphate (mmol/L)	1.55 ± 0.50	1.68 ± 0.62	0.201
Parathyroid hormone (ng/L)	556.13 ± 466.92	83.23 ± 58	< 0.001
Total Kt/V	1.31 ± 0.37	1.72 ± 0.32	

Abbreviation: BP, blood pressure.
* Chi-squared test.

Table 2. Aetiology of end-stage kidney disease among study subjects.

Variable	Group 1 HD patients (n = 84) (%)	Group 2 PD patients (n = 44) (%)
Hypertension	43 (51)	20 (46)
Chronic glomerulonephritis	17 (20)	3 (7)
Chronic pyelonephritis/reflux	7 (8)	0 (0)
Congenital abnormalities	4 (5)	6 (14)
Lupus nephritis	4 (5)	0 (0)
Polycystic kidney disease	2(2)	0 (0)
Diabetes	0 (0)	3 (7)
Others*	2 (2)	0 (0)
Unknown	5 (6)	12 (27)

* Others include: obstructive uropathy =1; trauma =1.

Factors associated with higher LVMI among HD subjects were systolic blood pressure and male gender (Table 5), whereas among PD patients only haemoglobin was associated with LVMI (ß −1.80, 95% CI: −3.22 to −0.38, P = 0.020).

DISCUSSION

In this study, a high prevalence of LVH was observed among Black African patients with ESKD who were receiving HD (87%) and PD (68%). This is in keeping with reports from other studies, which also noted a high prevalence of LVH among ESKD patients [2,10-15]. In a retrospective study involving 202 patients undergoing chronic dialysis (comprising 111 on HD and 91 on PD), of whom 132 had echocardiograms, Luyckx et al. documented a high prevalence of LVH at 68% [15].

Table 3. Echocardiographic measurements in the study population.

Parameter	Group 1 HD patients	Group 2 PD patients	P value
IVSd (cm)	1.41 ± 0.33	1.08 ± 0.34	< 0.001
IVSs (cm)	1.63 ± 0.45	1.21± 0.33	< 0.001
LVEDD (cm)	4.90 ± 0.79	5.60 ± 0.71	0.563
LVESD (cm)	3.25 ± 0.78	4.50 ± 0.98	0.335
LVPWd (cm)	1.28 ± 0.26	1.12 ± 0.17	< 0.001
LVPDWs (cm)	1.57 ± 0.38	1.44 ± 0.30	0.036
LVMI (g/m^2)	162.1 ± 56.2	164 ± 78	0.874
RWT	0.54 ± 0.13	0.38 ± 0.07	< 0.001
E/A ratio	1.30 ± 0.42	1.65 ± 1.17	0.015
Ejection fraction (%)	60.87 ± 11.75	58.13 ± 8.10	0.169
Shortening fraction (%)	33.09 ± 7.98	31.07 ± 5.63	0.137

Abbreviations: LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; IVSd, interventricular septal thickness in diastole; IVSs, interventricular septal thickness in systole; LVPWd, left ventricular posterior wall end-diastole; LVPWs, left ventricular posterior wall end-systole; LVMI, left ventricular mass index; RWT, relative wall thickness; E wave: peak early diastolic filling velocity; A wave: late diastolic filling velocity.

Table 4. Types of left ventricular hypertrophy and its geometric models among study subjects.

Parameter	Group 1 HD patients (n = 84) (%)	Group 2 PD patients (n = 44) (%)	χ^2	P value
Left ventricular hypertrophy	73 (87)	30 (68)	6.44	0.011
Normal	11 (13)	10 (23)	1.95	0.162
Concentric LVH	61 (73)	19 (43)	10.68	0.001
Eccentric LVH	12 (14)	11 (25)	2.25	0.134
LV concentric remodelling	0 (0)	4 (9.1)	5.17	0.023
LV diastolic dysfunction	54 (64)	20 (46)	4.20	0.041
LV systolic dysfunction	16 (19)	3 (7)	2.52	0.113

Abbreviations: LVH, left ventricular hypertrophy; LV, left ventricle.

Table 5. Risk factors associated with LVMI in haemodialysis patients.

LVMI	Coefficient ß	SE ß	P value	95% confidence interval
Smoker/Non-smoker	−14.13	9.41	0.137	–32.85–4.60
Male/female	−43.57	16.21	0.009	–75.83– –11.32
Systolic BP (mmHg)	1.27	0.59	0.036	0.09–2.45
Diastolic BP (mmHg)	0.75	0.81	0.355	–0.86–2.36

Abbreviations: SE ß, standard error beta; BP, blood pressure.

LVH is the most common abnormality in patients with ESKD on HD, with a prevalence ranging from 49% to 79% [10]. A Canadian prospective cohort study of 432 patients who underwent echocardiography within one year of starting dialysis revealed that only 16% had normal cardiac function and dimensions, 42% had concentric LVH, and 23% had eccentric LVH [11]. Zoccali et al. [12] reported that LVH was present in 77% of ESKD patients on HD, Huting et al. [13] found that LVH was present in 84% of the CAPD patients they studied, whereas Mehmet et al. [14] also reported a high prevalence of LVH of up to 70% among 74 CAPD patients in Turkey. Several factors are known to contribute to these cardiac abnormalities, some of which are CKD-related such as anaemia, abnormalities of mineral and bone metabolism, extracellular fluid overload and poorly controlled hypertension, among others [3]. More than half of our study subjects had suboptimal blood pressure control. Anaemia was also prevalent among our patients as indicated by the mean low HB. Similarly, abnormalities in mineral bone metabolism were observed particularly among the HD subjects; all of these factors may have contributed to the high prevalence of LVH in our patients.

The HD patients demonstrated a higher frequency of LVH than those on PD. This aligns with the findings of Tian et al. [16] among Chinese subjects with ESKD, among whom they found a significantly higher prevalence of LVH among HD patients (68.8%) compared to those on PD (45.2%) [16]. In another study, by Canziani et al., LVH was present in 93% of HD patients compared with 52% of those on CAPD [17]. This, however, contrasts with the findings of Enia et al., who reported higher LVH among CAPD patients compared to those on HD and attributed this difference to greater volume overload among the PD patients [18]. In contrast, other researchers, such as Gunai et al. [19] and Ellouali et al. [20], found no difference in cardiac structure between the subjects undergoing the two modalities of treatment. It has been proposed that the manner in which each of the KRTs is executed is considered crucial in influencing the onset and advancement of cardiac dysfunction. For instance, cardiovascular risk factors associated with CKD, such as anaemia, mineral metabolism, and inadequately managed hypertension due to fluid overload, may be more significantly affected by the dialysis dose and adequacy than by the specific modality of dialysis employed. The traditional thrice-weekly haemodialysis and, to a certain degree, peritoneal dialysis have not demonstrated efficacy in improving left ventricular hypertrophy. In contrast, more frequent daily or extended dialysis sessions have been linked to improvements in left ventricular hypertrophy [20,21].

Concentric LVH was the predominant type of LVH seen in this study and was significantly more common among HD patients. This may be due to the longer duration of HD; hypertension being more often observed as a cause of CKD among these subjects as well as the more severe mineral bone disorder seen in the HD patients. Furthermore, peritoneal dialysis is a continuous treatment, rendering it more physiological than intermittent haemodialysis.

Concentric LVH is known to be associated with pressure overload and results in increased ventricular stiffness and diastolic dysfunction. Not surprisingly, there was also a predominance of diastolic dysfunction in our study population. Eccentric LVH was observed more frequently in patients undergoing CAPD; however, this finding was not statistically significant in our study. Factors such as chronic volume overload, increased preload, and slower fluid removal all play a role in left ventricular dilatation and the progression of eccentric hypertrophy.

The PD patients had a lower rate of systolic dysfunction than the HD group, though this difference was not significant. The reason for this may be the strict selection criteria for the chronic dialysis programme in South Africa. Another possible explanation may relate to the continuous nature of PD, which is often considered more physiological than intermittent HD. Subjects with severe cardiac disease with an ejection fraction less than 40% were often excluded from the chronic dialysis programme. Systolic dysfunction

was noted in 20% of the HD group, which is similar to the report by Zoccali et al. [10], who found systolic dysfunction in 22% of their patients. Diastolic dysfunction was more prevalent among the HD subjects, most likely due to a higher occurrence of concentric LVH, more severe mineral bone disorder, and cardiac calcification among them.

Among HD subjects, systolic blood pressure and male gender were associated with LVMI. Hypertension is the leading cause of LVH among ESKD patients [23,24]. Several factors contribute to the development of LVH in these patients and include pressure overload from hypertension or decreased vascular compliance from aortic calcification; increased preload from fluid overload, anaemia, and high output arterio-venous fistula has also been implicated in LVH. Other cellular and neurohumoral factors involved are the renin–angiotensin–aldosterone system, vitamin D deficiency, severe secondary hyperparathyroidism and recently fibroblast growth factor-23 (FGF 23) [5,25]. Most of these factors were present in our subjects, especially those on HD. Several studies of HD patients have reported an association between hypertension and LVH [11,12,14,16, 23,24]. Among PD subjects there was no association between LVH and hypertension.

It is noteworthy that pericardial effusion was observed in 20% of patients on CAPD, whereas none of those on HD displayed this finding. This may be attributed to uraemia resulting from inadequate dialysis in those on CAPD, with consequent inflammation manifesting as uraemic pericarditis. Other potential causes include infections such as viral illnesses or tuberculosis, and, more rarely, a direct communication between the peritoneum and the pericardium (peritoneal–pericardial leak).

Our study is limited by its cross-sectional design and the absence of matching between the two groups with respect to disease aetiology, extracellular volume status, and duration of kidney replacement therapy. In addition, data on inter-dialytic weight gain, daily total ultrafiltration, and daily body weight for CAPD patients were not available. These factors could potentially affect the prevalence of the echocardiographic changes observed. However, the study still shows a high prevalence of left ventricular structural and functional abnormalities among ESKD patients in our practice. This calls for the inclusion of assessing cardiac function as part of the routine care of these patients. This is important as early detection of these abnormalities will lead to taking appropriate measures such as improved blood pressure and extracellular volume control, and anaemia management to reduce their effects in causing CVD.

CONCLUSION

This study shows that LVH is highly prevalent among African ESKD patients on kidney replacement therapy and this is notably related to hypertension, male gender and anaemia. Therefore, echocardiography should be part of the routine assessment of patients on kidney replacement therapy.

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Conflict of interest

The authors have no conflicts of interest to declare.

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