Right ventricular dysfunction in patients with end-stage renal disease on regular hemodialysis

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Background
Although there are considerable data on the changes in left ventricular function in hemodialysis (HD) patients, only a few studies on right ventricular (RV) function can be found in the literature. We investigated the changes in RV function in HD patients.

Methods
We examined 74 individuals grouped as follows: healthy controls ($n=24$) and HD patients ($n=50$). Echocardiography including tissue Doppler imaging (TDI) of the RV was performed in all patients.

Results
HD patients had significantly lower RV systolic indices than control participants in right ventricle fractional area change (normal 35–63%) (37.54 ± 9.86 vs. 43.5 ± 4.8%, $P<0.001$), tricuspid plane systolic excursion (normal 1.6–3 cm) (2.09 ± 0.49 vs. 2.61 ± 0.36 cm, $P<0.001$), STDIS′ wave (7.99 ± 1.37 vs. 9.66 ± 1.86 cm/s, $P<0.001$), and LTDIS′ wave (peak systolic velocity at lateral tricuspid annulus; normal: 10–19 cm/s) (11.86 ± 2.86 vs.16.04 ± 3.60, $P<0.001$). HD patients had statistically significantly higher systolic pulmonary pressure (normal <35 mmHg at rest) compared with those in the control group (32.75 ± 10.11 vs. 25.23 ± 3.99, $P<0.001$). There were no statistically significant correlations between systolic pulmonary pressure and RV dimensions or RV function indices.

Conclusion
Subclinical RV dysfunction – as estimated by RV function indices; tricuspid plane systolic excursion, right ventricle fractional area change, and LTDIS′ – is increased among HD patients. A high prevalence of pulmonary hypertension was found among HD patients and this was not associated significantly with RV or left ventricular dysfunction in these patients.

Keywords:
hemodialysis, pulmonary hypertension, right ventricular dysfunction

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Introduction

Chronic renal failure (CRF) is associated with significantly increased morbidity and mortality. Chronic renal failure affects almost every system of the body and results in various functional and structural abnormalities. Cardiovascular complications are the main cause of death in patients with chronic kidney disease (CKD) undergoing hemodialysis (HD) therapy [1,2], accounting for 40% of deaths in international registries [3].

The traditional risk factors for cardiovascular disease do not completely explain this high risk, which seems to be influenced by the so-called nontraditional risk factors associated with CKD [4]. This set of factors accelerates the course of coronary artery disease [5] and is associated with a higher prevalence of ventricular hypertrophy, myocardial fibrosis, valvulopathies, arrhythmias, and sudden death [6].

The prevalence of clinical manifestations of cardiac disease at the start of end-stage renal disease (ESRD) therapy is high and these manifestations predict death independently [7,8]. More than 50% of the individuals starting a dialysis program present some type of pre-existent cardiovascular disease [9]. Clinical manifestations of cardiovascular disease were highly prevalent at the start of ESRD therapy: 14% had coronary artery disease, 19% had angina pectoris, 31% had cardiac failure, 7% had dysrhythmia, and 8% had peripheral vascular disease [10].

There is increasing evidence of the pivotal role of echocardiography in the improvement of quality of global clinical evaluation of advanced CKD patients. The current literature and clinical practice have emphasized the usefulness of the method for the diagnosis of clinical and subclinical cardiac dysfunction, the prediction of cardiovascular risk, and in the orientation and follow-up of treatment strategies. Guidelines recommend the echocardiogram for all HD patients 1–3 months after the start of renal replacement therapy and in intervals of 3 years subsequently, irrespective of the symptoms [11].

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ESRD is associated with a variety of cardiac alterations including left ventricular hypertrophy (LVH), left ventricular (LV) dilation, and reduction in systolic and diastolic function, with only 16% of new dialysis patients presenting with normal cardiac morphology and function [12]

On echocardiography, 15% had systolic dysfunction, 32% had LV dilatation, and 74% had LVH [13]. Patients undergoing chronic HD show an increased prevalence of pulmonary hypertension during treatment, 15–20% [1,2].

However, although most available studies focused on LV function in dialysis patients, the impact of dialysis treatments on the development of right ventricular dysfunction (RVD) has not been fully investigated. However, recently, a retrospective study in which Paneni *et al.* [14] investigated the impact of different dialysis treatments on right ventricular (RV) function showed that compared with peritoneal dialysis, HD increases the risk of RVD, particularly in the presence of brachial ateriovenous fistula (AVF). A limitation of this study is its retrospective design.

Patients and methods

This is a prospective study that included 50 patients with end-stage renal failure on regular HD (group I). They were recruited from the nephrology and dialysis department, Cairo University (Kasr El-Ainy) hospital, from May 2011 to December 2011. We also included 24 age-matched and sex-matched healthy volunteers (without any cardiac or renal diseases) who served as the control group (group II).

Selection of patients

Patients on HD had been on regular 4 h HD sessions three times per week for at least 3 months using bicarbonate-buffered dialysate. All patients underwent full clinical evaluation and they also underwent chest radiography, standard 12-lead ECG, and arterial blood gas analysis. The exclusion criteria were defined by clinical or echocardiographic evidence of ischemic heart disease, LV dysfunction, valvulopathy, or previous renal transplantation. We also excluded patients with clinical conditions that might predispose to pulmonary hypertension (chronic obstructive pulmonary disease, interstitial lung diseases, connective tissue disorders, chronic thromboembolic disease, congenital left-to-right shunts, and primary pulmonary hypertension). Every participant provided informed consent and all the diagnostic procedures were approved by our institute's ethics committee.

Echocardiography

The entire study population underwent transthoracic echocardiography, including both conventional and tissue Doppler imaging (TDI) of the LV and the RV. Echocardiography was performed within 1 h after the completion of HD while the patients were at optimal dry weight to avoid any overestimation of pulmonary pressure because of volume overload. Images were obtained using a Philips iE33 (Philips Healthcare, Massachusetts, USA) with 2 and 2.5 MHz sector transducer equipped with the TDI mode. The study was carried out according to the criteria of the American Society of Echocardiography [15]. LV volumes were estimated using the z-derived method. Ejection fraction (EF) of the LV was calculated using the Teicholz formula [16] and further confirmed using Simpson's technique in the four-chamber view. The maximal tricuspid regurgitation velocity was measured by continuous wave Doppler echocardiography from the apical four-chamber view. The highest peak velocity was recorded and the average peak velocities from three beats were calculated. RV diameters were measured in the long axis view. EF of the RV was calculated using Simpson's formula from the apical four-chamber view.

Data management and statistical analysis

The data were coded and entered on an IBM compatible computer using the statistical package SPSS version 16.0 (IBM Corporation, Armonk, New York, USA). All data sets were assessed for normality and distribution and appropriate tests were used accordingly. Descriptive statistics using the suitable measures of central tendency and dispersion were calculated to summarize the given data sets. The mean and the SD were mostly used for quantitative data. For qualitative data sets, percentages were used. Inferential statistics were used to assess differences between the studied groups and their comparison. Student's *t*-test was used to assess differences between two unrelated groups in normally distributed quantitative variables. Qualitative variables were assessed using the χ^2-test. The association between two quantitative variables was tested using the univariate analysis approach for quantitative variables, namely, simple linear correlation – regression. The Pearson correlation coefficient (r) was used to test the strength of the relationship, which varies from −1 to 1, where 0 indicates no relationship and 1 indicates a perfect linear relationship. The sign identifies the direction of the relationship, whether direct in case of a positive sign or inverse in case of a negative sign. All the above-mentioned statistical levels rejected the null hypothesis at a P value less than 0.05.

Results

No statistically significant differences were found among the groups in their age, sex, BMI and smoking, and systolic blood pressure; however, there was a significant increase in diastolic blood pressure and heart rate (Table 1).

Laboratory investigations showed a significant increase in blood urea, serum creatinine, potassium, and phosphors in the HD group compared with the control group and a statistically significant decrease in blood hemoglobin and calcium (Table 2).

By echocardiography, compared with the control group, the HD group showed a statistically significant increase in

interventricular septal thickness (IVST; 0.99 ± 0.21 vs. 0.83 ± 0.13 cm, $P<0.001$), posterior ventricular wall thickness (PWT; 1.01 ± 0.17 vs. 0.83 ± 0.13 cm, $P<0.001$), and LV mass index (117.43 ± 46.72 vs. 80.13 ± 13.40 g/m^2, $P<0.001$) (Table 3). The prevalence of LVH by ECG was 28% (14 patients) versus 52% (26 patients) by echocardiography. The sensitivity and specificity was 50 and 95.83%, respectively, and the positive predictive value and negative predictive value were 92.85 and 63.88%, respectively (Table 4).

However, patients on HD showed a nonsignificant increase in RV dimensions compared with the control group, except for the long dimension of RV, which was higher in the HD group (7.23 ± 0.87 vs. 6.36 ± 0.79 cm, $P<0.001$) (Table 5). The prevalence of an increase in RV diameters according to the mean lower limits of RV dimensions is shown in (Table 6). Compared with the control group, HD patients had significantly lower RV systolic indices and there was a statistically significant decrease in right ventricular fractional area change (RVFAC; 37.54 ± 9.86 vs. 43.5 ± 4.8%, $P<0.001$), tricuspid plane systolic excursion (TAPSE; 2.09 ± 0.49 vs. 2.61 ± 0.36 cm, $P<0.001$), STDIS′ wave (7.99 ± 1.37 vs. 9.66 ± 1.86 cm/s, $P<0.001$), and LTDIS′ wave (11.86 ± 2.86 vs. 16.04 ± 3.60, $P<0.001$) (Table 7).

Table 1 Characteristics of the study population

Variables	Mean ± SD		
	HD ($n=50$)	Control ($n=24$)	*P* value
Age (years)	37.3 ± 12.92	39.6 ± 12.65	0.80
Sex [*n* (%)]			
Male	31 (62)	15 (62.2)	0.96
Female	19 (38)	9 (37.8)	
BMI (kg/m^2)	23.37 ± 3.71	24.71 ± 2.49	0.116
Smoking [*n* (%)]			
Smoker	15 (30)	5 (21)	0.27
Nonsmoker	35 (70)	19 (79)	
Duration on dialysis (months)	37 ± 18	–	–
Hypertension (%)	78	–	–
Systolic BP (mmHg)	140 ± 20.07	119.7 ± 8.90	0.10
Diastolic BP (mmHg)	86.04 ± 10.78	76.45 ± 4.77	**<0.001**
Pulse rate (beats/min)	78.56 ± 8.76	75.29 ± 6.84	**<0.001**

Bold values indicates a significant increase in diastolic blood pressure and heart rate in hemodialysis patients.
BP, blood pressure; HD, hemodialysis.

Table 2 Laboratory data of the study population

Variables	Mean ± SD		
	HD ($n=50$)	Control ($n=24$)	*P* value
Hemoglobin (g/dl)	9.56 ± 2.02	14.52 ± 0.57	**<0.001**
Creatinine (mg/dl)	10.09 ± 2.5	0.64 ± 0.08	**<0.001**
Urea (mg/dl)	146.12 ± 50.97	34.75 ± 6.32	**<0.001**
Na (mEq/l)	143.68 ± 5.65	140.40 ± 1.35	0.314
K (mEq/l)	5.44 ± 0.85	3.93 ± 0.28	**<0.001**
Calcium (mg/dl)	8.42 ± 1.31	9.31 ± 0.35	**<0.001**
Phosphorus (mg/dl)	5.42 ± 1.72	3.31 ± 0.40	**<0.001**
Cholesterol (mg/dl)	164.32 ± 56.50	151.70 ± 13.31	0.380
Triglycerides (mg/dl)	149.20 ± 82.85	129.90 ± 15.87	0.503
FBG (mg/dl)	101.82 ± 21.51	91.45 ± 13.24	0.637

Bold values indicate a significant increase in blood urea, serum creatinine, potassium, and phosphors in the HD group compared with the control group and a statistically significant decrease in blood hemoglobin and calcium.
FBG, fasting blood glucose; HD, hemodialysis.

Table 3 Left-side echocardiographic parameters in the study population

Variables	Mean ± SD		
	HD ($n=50$)	Control ($n=24$)	*P* value
EF (%)	62.50 ± 11.6	64.54 ± 15.20	0.525
IVST (cm)	0.99 ± 0.21	0.83 ± 0.13	**<0.001**
PWT (cm)	1.01 ± 0.17	0.83 ± 0.13	**<0.001**
LV mass (g)	198.07 ± 73.33	144.91 ± 21.68	**<0.001**
LV mass index (g/m^2)	117.43 ± 46.72	80.13 ± 13.40	**<0.001**
left Atrium (cm)	3.72 ± 0.67	3.78 ± 0.50	0.661
Aorta (cm)	2.88 ± 0.41	2.76 ± 0.34	0.262
Mitral E wave (cm/s)	84.34 ± 25.40	80.40 ± 19.23	0.504
Mitral A wave (cm/s)	75.18 ± 22.25	61.05 ± 12.19	**0.005**
Mitral E/A	1.17 ± 0.67	1.28 ± 0.33	0.279

Bold values indicate a statistically significant increase in left ventricle dimensions and thickness in hemodialysis patients.
EF, ejection fraction; HD, hemodialysis; IVST, interventricular septal thickness; LV, left ventricular; mitral A wave, atrial filling wave velocity; mitral E wave, early diastolic wave velocity; PWT, posterior ventricular wall thickness.

Table 4 Sensitivity and specificity of ECG for the determination LVH

	LVH by echocardiography	No LVH by echocardiography	Total ($n=50$) [*n* (%)]
Positive LVH by ECG	13	1	14 (28)
Negative LVH by ECG	13	23	36 (72)
Total ($n=50$)	26 (52)	24 (48)	50 (100)

Sensitivity = 0.5, specificity = 0.95, positive predictive value = 0.92, negative predictive value = 0.63.
LVH, left ventricular hypertrophy.

Table 5 Right ventricular dimensions in the study population

Variables	Mean ± SD		
	HD ($n=50$)	Control ($n=24$)	*P* value
RV mid (cm)	3.07 ± 0.73	2.95 ± 0.38	0.294
RV basal (cm)	3.39 ± 0.56	3.19 ± 0.37	0.119
RV long (cm)	7.23 ± 0.87	6.36 ± 0.79	**<0.001**
RVOT proximal (cm)	3.28 ± 0.53	3.07 ± 0.46	0.109
RVOT distal (cm)	2.66 ± 0.45	2.49 ± 0.36	0.107
RA major (cm)	4.49 ± 0.56	4.23 ± 0.53	0.61
RA minor (cm)	3.06 ± 0.60	3.66 ± 0.46	0.64

Bold values indicates patients on HD who showed a nonsignificant increase in RV dimensions compared with the control group, except for the long dimension of RV, which was higher in the HD group.
HD, hemodialysis; RA, right atrium; RV, right ventricular; RVOT, right ventricle outflow tract.

Table 6 Prevalence of increased RV dimensions in the HD group

RV parameters	Abnormal (*n*)	Percentage of total ($n=50$)
RV mid (>3.5 cm)	6	12
RV basal (>4.2 cm)	4	8
RV long (>8.6 cm)	2	4
RVOT proximal (>3.3 cm)	21	42
RVOT distal (>2.7 cm)	18	36

HD, hemodialysis; RV, right ventricular; RVOT, right ventricle outflow tract.

From Table 8, it is clear that there were positive correlations between RV long, right atrium (RA) major, RA minor diameters and E and E/A. Also, there were positive correlations between RV mid cavity, proximal right ventricle outflow tract, RA minor diameters and left atrium diameter.

Patients on HD had statistically significantly higher systolic pulmonary pressure (SPAP) compared with those in the control group (32.75 ± 10.11 vs. 25.23 ± 3.99, $P < 0.001$) (Table 9). There were no correlations between SPAP and EF, IVST, and PWT. There were no statistically significant correlations between SPAP and RV dimensions or RV function indices (Table 10).

Discussion

Cardiovascular disease is the leading cause of mortality in patients undergoing dialysis, accounting for 50% of deaths [17]. In particular, congestive heart failure is the most common finding in these patients and is associated with a poor prognosis [18].

HD, which is usually carried out through a surgically created native AVF, has been associated with an increased risk of pulmonary hypertension. In HD patients, AVF causes a left-to-right shunt, leading to chronic volume overload, independent of the increase in total body water, thus worsening RV overload [2].

Previous studies on the relation between pulmonary hypertension and dialysis have mostly investigated the impact of volume overload on TDI indices of LV function, showing an increased prevalence of diastolic dysfunction in these patients [19]. However, although most available studies focused on LV function in dialysis patients, the impact of dialysis treatments on the development of RVD has not fully been investigated. However, in 2010, a retrospective study in which Paneni *et al.* [14] investigated the impact of different dialysis treatments on RV function showed that HD increases the risk of RVD, particularly in the presence of brachial AVF. One of the important limitations of that study was its retrospective nature.

In the present study, we investigated RV involvement in HD patients, in an attempt to determine the prevalence of RV dysfunction in these patients and find the correlates of RV dysfunction. Our study was prospective and was designed to investigate the impact of chronic dialysis therapy on RV function.

Patients on HD in our study showed a significant increase in LV mass index compared with the controls (117.43 ± 46.72 vs. $80.13 \pm 13.40\ g/m^2$, $P < 0.001$); also, in our study, it was found that HD patients, compared with controls, showed a statistically significant increase in both IVST (0.99 ± 0.21 vs. 0.83 ± 0.13 cm, $P < 0.001$) and PWT

Table 7 Right ventricular function parameters in the study population

	Mean ± SD		
Variables	HD ($n=50$)	Control ($n=24$)	P value
RVFAC (%)	37.54 ± 9.86	43.5 ± 4.8	**<0.001**
TAPSE (cm)	2.09 ± 0.49	2.61 ± 0.36	**<0.001**
LTDIS′ (cm/s)	11.86 ± 2.86	16.04 ± 3.60	**<0.001**
STDIS′(cm/s)	7.99 ± 1.37	9.66 ± 1.86	**<0.001**

Bold values indicates comparison with the control group, HD patients had significantly lower RV systolic indices and there was a statistically significant decrease in right ventricular fractional area change.
HD, hemodialysis; RVFAC, right ventricle fractional area change; TAPSE, tricuspid plane systolic excursion.

Table 9 Comparison of SPAP between the HD group and the control group

Variable	HD ($n=50$)	Control ($n=24$)	P value
SPAP (mean ± SD) (mmHg)	32.75 ± 10.11	25.23 ± 3.99	<0.001

HD, hemodialysis; SPAP, systolic pulmonary pressure.

Table 8 Correlations between RV indices and left ventricle indices

Variables	EF	IVST	PWT	A	E	E/A	Left atrium	Aorta
RV mid *r* (cm)	0.084	−0.010	−0.118	−0.044	0.245	0.230	0.369	−0.031
P	0.564	0.994	0.413	0.762	0.086	0.108	**0.008**	0.830
RV basal *r* (cm)	−0.052	0.029	0.037	−0.125	−0.036	0.092	0.114	−0.024
P	0.718	0.841	0.799	0.387	0.803	0.527	0.429	0.866
RV long *r* (cm)	−0.180	−0.022	0.056	−0.007	0.336	0.387	0.193	0.042
P	0.212	0.879	0.697	0.963	**0.017**	**0.005**	0.180	0.772
RV proximal *r* (cm)	−0.132	0.098	0.043	−0.102	0.040	0.202	0.302	0.036
P	0.362	0.496	0.765	0.483	0.783	0.160	**0.033**	0.804
RV distal *r* (cm)	−0.173	−0.063	−0.122	−0.008	−0.112	0.010	−0.172	−0.063
P	0.229	0.664	0.400	0.955	0.437	0.946	0.232	0.665
RA major *r* (cm)	0.139	0.050	0.061	−0.081	0.269	0.285	0.170	0.250
P	0.336	0.730	0.672	0.574	0.059	**0.045**	0.239	0.080
RA minor *r* (cm)	−0.159	0.051	−0.011	−0.128	0.174	0.286	0.307	0.147
P	0.271	0.723	0.937	0.378	0.227	**0.044**	**0.030**	0.309
RVFAC *r* (%)	0.344	0.134	0.170	0.048	0.160	0.008	0.157	0.020
P	0.141	0.352	0.237	0.743	0.268	0.956	0.277	0.891
TAPSE *r* (cm)	0.164	0.100	0.025	0.047	0.100	0.035	0.057	0.227
P	0.255	0.490	0.862	0.745	0.492	0.809	0.685	0.113
STDIS′ *r* (cm/s)	0.419	−0.194	−0.220	−0.194	0.014	0.052	−0.006	0.104
P	**0.002**	0.178	0.125	0.178	0.923	0.721	0.966	0.473
LTDIS′ *r* (cm/s)	0.301	0.150	0.042	0.018	0.112	0.009	0.048	0.148
P	**0.034**	0.300	0.774	0.904	0.437	0.951	0.739	0.305

Bold values indicates there were positive correlations between RV long, RA major, RA minor diameters and E and E/A.
EF, ejection fraction; IVST, interventricular septal thickness; PWT, posterior ventricular wall thickness; RA, right atrium; RV, right ventricular; RVFAC, right ventricle fractional area change; TAPSE, tricuspid plane systolic excursion.

Table 10 Correlations between SPAP and right-side, left-side parameters

Variables	SPAP *P* value
RV mid (cm)	0.156
RV basal (cm)	0.151
RV long.(cm)	0.062
RA major (cm)	0.423
RVOT proximal (cm)	**0.012**
RVOT distal (cm)	0.800
Lateral TDIS (cm/s)	0.355
RVFAC (%)	0.821
TAPSE (cm)	0.223
EF (%)	0.072
IVST (cm)	0.517
PWT (cm)	0.617
E/A	**0.027**
LA (cm)	**0.019**
Aorta (cm)	0.643

Bold values indicates there were no correlations between SPAP and EF, IVST, and PWT. There were no statistically significant correlations between SPAP and RV dimensions or RV function indices.
EF, ejection fraction; IVST, interventricular septal thickness; LA, left atrium; PWT, posterior ventricular wall thickness; RA, right atrium; RV, right ventricular; RVFAC, right ventricle fractional area change; RVOT, right ventricle outflow tract; SPAP, systolic pulmonary pressure; TAPSE, tricuspid plane systolic excursion.

(1.01 ± 0.17 vs. 0.83 ± 0.13 cm, $P < 0.001$). This finding was similar to that of the study of Said *et al.* [20] who found that HD led to an increase in IVST (1.1 vs. 1.0 cm, $P = 0.01$) and PWT (1 vs. 0.95 cm, $P = 0.009$); the LV mass index was 133.7 g/m^2 (95–186.5 g/m^2) (mean, range).

The prevalence of LVH by ECG in our study was 28% (14 patients) versus 52% (26 patients) by echocardiography. The sensitivity and specificity of ECG criteria were 50 and 95.83%, respectively, and the positive predictive and negative predictive values were 92.85 and 63.8%, respectively. In a recent comparable study carried out by Esquitin *et al.* [21] LV was detected better by echocardiography than by any of the ECG criteria (69 and 34%, respectively, of their study population).

Similar to Said *et al.* [20], we found no difference in the left ventricle ejection fraction (LVEF) between the HD group and the control group (62.50 ± 11.6, 64.54 ± 15.20%, $P = 0.525$, respectively). However, Paneni *et al.* [14] found that the EF was significantly lower in HD patients than in peritoneal dialysis patients and controls (56.1 ± 8.6, 62.4 ± 9.8, 68.3 ± 5.7%, $P = 0.001$, respectively). The difference may be attributed to a relatively shorter duration of dialysis in our study patients than that in the study of Paneni and colleagues (37 ± 18, 44 ± 29 months, respectively).

No difference in RV dimensions was found between the two groups of our study, except for the long dimension of RV, which was higher in the HD group compared with the controls (7.23 ± 0.87 vs. 6.36 ± 0.79 cm, $P < 0.001$). More importantly, a positive correlation was found between E/A and RV long diameters ($P = 0.005$). This means that diastolic dysfunction of LV had an impact on RV dimensions.

The TAPSE is a good parameter to evaluate the RV function and it correlates closely with RVEF and RVFAC in a variety of patient populations [22]. In our study, TAPSE was reduced (<1.6 cm) in 28% of the patients in the HD group. This may not only have been because of the effects of uremia on RV function but also the effect of hyperdynamic circulation because of AVF [23].

RVFAC is another measure of RV systolic function that has been shown to correlate well with RV EF by MRI [24]. RVFAC was reduced in 20 patients (40%) of the HD group. A statistically significant decrease in RVFAC in the HD group than the control group was observed (37.54 ± 9.86 vs. 43.5 ± 4.8%, respectively, $P < 0.001$). However, no correlation was detected between RVFAC and duration of HD, SPAP, LVEF, LV mass index, or E/A. Thus, the reduction in RVFAC was independent of LVH, diastolic dysfunction of the LV, and pulmonary hypertension. Further studies are needed to explore the cause for reduction in RVFAC in HD patients.

Twelve HD patients (24%) showed an abnormal decrease in lateral TDIS′. Patients with HD had statistically significant lower lateral TDIS′ compared with the controls (11.86 ± 2.86, 16.04 ± 3.60 cm/s, $P < 0.001$). Similar results were obtained by Paneni *et al.* [14] and Said *et al.* [20]. Similar to the reduction in RVFAC, the decrease in lateral TDIS′ in the HD group was also independent of LVH, LV diastolic dysfunction, and pulmonary hypertension.

SPAP of 35 mmHg represents the cutoff value for pulmonary hypertension assessed by Doppler echocardiography [25]. The long-term HD patients have a high prevalence of pulmonary hypertension [26]. In our study, SPAP was increased in 17 HD patients (34%). The mean SPAP was 32.75 ± 10.11 mmHg in HD patients and 25.23 ± 3.99 in the control group ($P < 0.001$). Comparable prevalence of pulmonary hypertension in HD patients was found by previous two studies in Egypt. Abdelwhab and Elshinnawy [27], Amin *et al.* [28]; both detected increased SPAP among their HD populations (44.4 and 29%, respectively).

In the current study, on the basis of the Pearson linear correlation coefficient, there was a positive correlation between SPAP and left atrium ($P = 0.019$), E wave ($P = 0.001$), E/A ratio ($P = 0.027$), right ventricle outflow tract proximal diameter ($P = 0.012$), and septal TDIS′ ($P = 0.009$). However, no significant correlation was found between SPAP and systolic right indices, RVFAC ($P = 0.223$), LTDIS′ ($P = 0.335$), TAPSE ($P = 0.223$), or LVEF% ($P = 0.072$). This means that the increased SPAP in the HD population could be attributed to left atrium and LV diastolic dysfunction, and may also be because of AVF, which causes a left-to-right shunt, leading to chronic volume overload.

Conclusion

This study confirmed a high prevalence of pulmonary hypertension among HD patients and that is not significantly associated with RV dysfunction in these patients. Subclinical RV dysfunction, as estimated by RV

function indices, TAPSE, RVFAC, and LTDIS′, is increased among HD patients. The reduction in RV systolic indices was independent of LVH, diastolic dysfunction of the LV, or pulmonary hypertension, whereas diastolic dysfunction of LV had an impact on RV dimensions.

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Conflicts of interest

There are no conflicts of interest.

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