ORIGINAL ARTICLE

Diastolic Dysfunction as a Determinant of Pulmonary Hypertension in Patients with End Stage Renal Disease and Preserved Left Ventricular Ejection Fraction

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Background: The prevalence of pulmonary hypertension (PH) in patients with end-stage renal disease (ESRD) varies among different studies.

Objective: To determine the prevalence and echocardiographic determinants of PH in patients with ESRD and preserved left ventricular (LV) ejection fraction.

Materials and Methods: Patients with ESRD who underwent comprehensive transthoracic echocardiography were enrolled. PH was defined as mean pulmonary artery pressures of 25 mmHg or greater or pulmonary artery systolic pressure of 50 mmHg or greater. The propensity score matching, and multivariable logistic regression analyses were performed.

Results: Three hundred two patients with a mean age of 49.1±14.6 years were included, of which 47% were female. The prevalence of PH was 42.7%. Diabetes mellitus, right ventricular dimension at basal level, LV ejection fraction, LV dimension, LV mass index, left atrial volume (LAV) index, early (E) and late diastolic velocity of mitral inflow, deceleration time of E and ratio of E, and tissue Doppler early diastolic velocity of mitral annulus (e') were univariately associated with PH. Multivariate factors associated with PH were LAV index (OR 1.09, 95% CI 1.05 to 1.12, p<0.001), and E/e' ratio (OR 1.12, 95% CI 1.05 to 1.20, p<0.001). In the propensity matched analysis, LAV index (OR 1.10, 95% CI 1.05 to 1.14, p<0.001) and E/e' ratio (OR 1.12, 95% CI 1.08 to 1.29, p<0.001) remained as independent determinants of PH.

Conclusion: In patients with ESRD and preserved LV ejection fraction, PH is common and the link between diastolic dysfunction and PH has been demonstrated.

Keywords: Chronic kidney disease; Diastolic dysfunction; End-stage renal disease; Left atrial volume; Pulmonary hypertension

Received 5 September 2022 | Revised 10 November 2022 | Accepted 2 December 2022

J Med Assoc Thai 2023;106(1):41-8

Website: http://www.jmatonline.com

Pulmonary hypertension (PH) is a common and serious cardiac complication, occurring as a consequence of cardiac and non-cardiac conditions. It is a progressive disease of pulmonary vessels that has a poor prognosis. This may be even more significant in patients with end stage renal disease (ESRD)[1-3]. Etiologic mechanisms have been proposed as the cause of PH in patients with ESRD, including co-existing left ventricular (LV) systolic or diastolic dysfunction, endothelial dysfunction associated with other co-morbidities with an increased risk of atherosclerosis, volume overload, an arterio-venous fistula, vascular calcification and stiffness, exposure to dialysis membrane, or severe anemia[4-7]. As previously reported, the prevalence of PH in patients with chronic kidney disease (CKD) or ESRD varies widely among different study populations, but most studies were retrospective studies with a small sample size[4,5,8-12]. However, a recent systematic review and meta-analysis by Tang et al. demonstrated that PH was prevalent in patients with CKD and ESRD, and associated with increased risk of death and cardiovascular events[13]. Early diagnosis of PH in these population is of clinical importance in determining proper intervention. Echocardiographic examination is a non-invasive and reliable tool to evaluate the pulmonary artery pressure (PAP) in clinical practice. The present study aimed to

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How to cite this article:

Ratanasit N, Karaketklang K, Chanwanitkulchai R, Rasmeehirun P. Diastolic Dysfunction as a Determinant of Pulmonary Hypertension in Patients with End Stage Renal Disease and Preserved Left Ventricular Ejection Fraction. J Med Assoc Thai 2023;106:41-8.

DOI: 10.35755/jmedassocthai.2023.01.13738

investigate the prevalence and echocardiographic determinants of PH in patients with ESRD and preserved LV ejection fraction (LVEF).

Materials and Methods

Patient selection

The present study was a single-center cross-sectional study of prospectively enrolled patients with ESRD having clinical signs and symptoms or indications that warranted a comprehensive transthoracic echocardiography. Data on clinical characteristics, physical examination, laboratory tests, and echocardiographic findings were recorded. Patients with ESRD were defined as those with stage 5 of CKD or those with a glomerular filtration rate of less than 15 mL/minute/1.73 m^2[14]. All adult patients with ESRD were considered for inclusion in the present study, regardless of the mode of dialysis or clinical symptoms. Patients with moderate to severe left-sided valvular disease, any prosthetic cardiac valve, LV systolic dysfunction with LVEF of less than 50%, congenital heart disease, permanent pacemaker, history of coronary artery disease or myocardial infarction, prior cardiac surgery or coronary revascularization, chronic pulmonary or liver disease, connective tissue disease, history of drug-induced PH, limited or poor-quality echocardiographic study, or incomplete data were excluded.

The Institutional Review Board of Siriraj Hospital, Mahidol University (Bangkok, Thailand) approved the present study protocol (certificate of approval no. 235/2556). Potential participants were informed about the study, gave written informed consents, and enrolled in the study.

Echocardiography

Each participant underwent a comprehensive transthoracic echocardiographic examination, which consisted of two-dimensional, M-mode, Doppler, and tissue Doppler imaging (TDI). All echocardiographic measurements were performed according to the standard guidelines with the average of three to five consecutive cardiac cycles for the analyses[15]. The assessment of PAP, including pulmonary artery systolic pressure (PASP), pulmonary artery end-diastolic pressure, mean PAP, and pulmonary vascular resistance were obtained using Doppler echocardiography[16-19]. PH was defined as a mean PAP of 25 mmHg or higher or PASP of 50 mmHg or higher[20-23]. The severity of pulmonary and tricuspid regurgitation was determined using multiparametric approach, such as color-flow imaging and Doppler echocardiography[24]. Significant pulmonary or tricuspid regurgitation was defined as a moderate or greater degree of regurgitation. Right ventricular (RV) wall thickness, dimensions, systolic function, and index of myocardial performance by the pulsed Doppler method and TDI were assessed according to the standard recommendation by the American Society of Echocardiography[15]. Impaired RV systolic function was defined as a tricuspid annular plane systolic excursion (TAPSE) of less than 17 mm or peak systolic myocardial velocity of the lateral tricuspid annulus (S'TV) of less than 9.5 cm/second[15]. Doppler echocardiography of transmitral inflow and TDI of medial and lateral mitral annulus were used to assess the LV diastolic function[25]. Peak early (E) and late (A) diastolic velocities of mitral inflow and deceleration time of E were measured using pulsed-wave Doppler study with the sample volume at the tip of mitral valve. Longitudinal early (e') and late diastolic myocardial velocities were measured using TDI in apical 4-chamber view with the sample volume at the medial and lateral aspects of mitral annulus. The average of medial and lateral e' was used for the E/e' ratio. The cutoff value for high E/e' ratio was greater than 14, indicating the elevation of left atrial (LA) pressure[25]. LA volume (LAV) and LV mass were measured using the recommendations by the American Society of Echocardiography[15] and indexed with body surface area. LA enlargement was defined as LAV index of greater than 34 mL/m^2[25]. LV hypertrophy was defined as LV mass index greater than 115 g/m^2 in male and greater than 95 g/m^2 in female[15]. LV diastolic dysfunction was diagnosed if three or more of the following parameters met the cutoff values, septal e' of less than 7 cm/second or lateral e' of less than 10 cm/second, average E/e' ratio greater than 14, LAV index greater than 34 mL/m^2, and peak tricuspid regurgitation velocity greater than 2.8 m/second[25].

Statistical analysis

Categorical variables were summarized as number and percentage (%) of patients, and continuous variables as mean ± standard deviation for normally distributed variables and median (percentile 25th and 75th) for non-normally distributed variables. Normality of distribution of variables was examined by Kolmogorov-Smirnov test. Comparisons of categorical variables between PH and non-PH groups were performed using chi-square test or Fisher's exact test. Continuous variables were compared using

Table 1. Baseline characteristics and laboratory data in all patients and the comparisons between patients with and without pulmonary hypertension

Variables	All patients (n=302)	PH (n=129)	No PH (n=173)	p-value
Age (years); mean±SD	49.1±14.6	49.3±16.2	48.9±13.3	0.856
Female; n (%)	142 (47.0)	65 (50.4)	77 (44.5)	0.311
BSA (m^2); mean±SD	1.6±0.20	1.59±0.20	1.64±0.19	0.035
BMI (kg/ m^2); mean±SD	22.8±4.4	22.5±5.0	23.0±3.9	0.285
Systolic BP (mmHg); mean±SD	134.9±20.9	139.7±20.9	131.3±20.2	0.001
Diastolic BP (mmHg); mean±SD	74.5±14.3	74.4±14.2	74.5±14.5	0.967
Dyspnea; n (%)	122 (40.4)	57 (44.2)	65 (37.6)	0.247
Edema; n (%)	66 (21.9)	38 (29.5)	28 (16.2)	0.006
Mode of dialysis; n (%)				0.457
No dialysis	14 (4.6)	8 (6.2)	6 (3.5)	
Peritoneal dialysis	39 (12.9)	18 (14.0)	21 (12.1)	
Hemodialysis	249 (82.5)	103 (79.8)	146 (84.4)	
Comorbidities; n (%)				
Hypertension	271 (89.7)	120 (93.0)	151 (87.3)	0.104
Dyslipidemia	112 (37.1)	54 (41.9)	58 (33.5)	0.138
Diabetes mellitus	74 (24.5)	40 (31.0)	34 (19.7)	0.023
Smoking	73 (24.2)	26 (20.2)	47 (27.2)	0.159
Family history of premature CAD	32 (10.6)	15 (11.6)	17 (9.8)	0.615
History of stroke	23 (7.6)	14 (10.9)	9 (5.2)	0.067
History of heart failure	89 (29.5)	44 (34.1)	45 (26.0)	0.127
Atrial fibrillation	17 (5.8)	11(8.9)	6(3.6)	0.056
Laboratory data				
Hemoglobin (g/dL); mean±SD	10.4±1.8	10.1±1.9	10.6±1.8	0.110
Hematocrit (%); mean±SD	32.7±5.5	31.9±5.2	33.2±5.6	0.109
Albumin (g/dL); mean±SD	4.2±0.8	4.0±0.7	4.3±0.9	0.004
Calcium (mg/dL); mean±SD	9.3±1.4	9.3±1.8	9.3±1.0	0.855
Phosphorus (mg/dL); median (P_{25}-P_{75})	4.7 (3.7 to 5.8)	4.5 (3.8 to 5.6)	4.7 (3.7 to 5.9)	0.404
Cholesterol (mg/dL); mean±SD	182.3±52.2	181.7±55.3	182.9±49.9	0.906
Triglyceride (mg/dL); median (P_{25}-P_{75})	120.0 (83.0 to 162.0)	98.0 (61.0 to 138.0)	131.5 (93.5 to 177.5)	0.002

BMI=body mass index; BP=blood pressure; BSA=body surface area; CAD=coronary artery disease; PH=pulmonary hypertension; SD=standard deviation

Student's t-test or Mann-Whiney U test. Univariate and multivariate predictors of PH were evaluated using logistic regression analysis (backward stepwise method) and presented as odds ratio (95% confidence interval). The estimation of the propensity score matching was assessed using logistic regression for balancing the distribution of patient characteristics as age, gender, mode of dialysis, hypertension, diabetes mellitus, and dyslipidemia, between PH and non-PH groups. After propensity score matching, the calculation for the appropriate predictors of PH in the multivariable logistic regression was performed. For all tests performed, a two-tailed p-value less than 0.05 was considered to be statistically significant. PASW Statistics, version 18.0 (SPSS Inc., Chicago, IL, USA) was used to perform all statistical analyses.

Results

Patient characteristics

There were 302 patients with ESRD enrolled and included for analyses. Table 1 shows baseline characteristics and laboratory data in all patients as well as the comparisons between patients with and without PH.

Echocardiographic data

PH was present in 129 or 42.7% of the patients. There were 166 or 55.0% and 125 or 41.4% patients with PASP of 35 mmHg or greater and mean PAP of 25 mmHg or greater, respectively. The mean PAP and PASP in patients with hemodialysis, peritoneal dialysis and no dialysis were 25.6±8.5, 26.0±8.6, and 24.9±9.7 mmHg, and 42.3±15.9, 42.9±13.8 and 37.8±15.6 mmHg, respectively. Table 2 shows

Table 2. Echocardiographic data in all patients and the comparisons between patients with and without pulmonary hypertension

Variables	All patients (n=302)	PH (n=129)	No PH (n=173)	p-value
Pulmonary artery hemodynamic data				
Peak TR velocity (m/s); median (P_{25}-P_{75})	2.8 (2.5 to 3.2)	3.2 (3.0 to 3.6)	2.6 (2.4 to 2.8)	0.011
RAP (mmHg); median (P_{25}-P_{75})	5.0 (5.0 to 10.0)	10.0 (5.0 to 10.0)	5.0 (5.0 to 10.0)	<0.001
PASP (mmHg); mean±SD	42.1±15.6	53.7±15.4	33.1±8.0	<0.001
PAEDP (mmHg); mean±SD	13.2±4.8	16.6±4.3	10.0±2.3	<0.001
Mean PAP (mmHg); mean±SD	25.6±8.5	32.4±7.0	19.2±3.0	<0.001
PVR (Wood unit); median (P_{25}-P_{75})	1.7 (1.5 to 2.1)	2.0 (1.6 to 2.5)	1.6 (1.4 to 1.9)	<0.001
Left ventricle				
LVEF (%); mean±SD	67.1±10.4	64.8±12.8	68.9±7.8	0.002
LV diastolic dimension (mm); mean±SD	48.8±7.6	49.9±7.9	48.0±7.2	0.027
LV systolic dimension (mm); mean±SD	30.5±7.6	32.4±9.2	29.1±5.7	<0.001
LV mass index (g/m^2); mean±SD	161.1±53.2	184.9±54.1	143.3±45.0	<0.001
LV hypertrophy; n (%)	267 (88.4)	128 (99.2)	139 (80.8)	<0.001
LV diastolic function				
E (cm/s); mean±SD	90.7±32.5	109.9±33.6	76.4±22.8	<0.001
A (cm/s); mean±SD	88.5±29.9	95.2±33.1	83.8±26.5	0.002
E/A ratio; mean±SD	1.06±0.48	1.21±0.52	0.96±0.42	<0.001
Deceleration time of E (ms); mean±SD	214.0±51.0	199.6±53.9	224.8±46.0	<0.001
e' (cm/s); mean±SD	7.3±4.2	6.9±3.2	7.5±4.8	0.212
E/e' ratio; mean±SD	14.0±7.1	17.8±8.4	11.4±4.3	<0.001
High E/e' ratio; n (%)	111 (36.8)	75 (58.1)	36 (20.8)	<0.001
LA diameter (mm); mean±SD	44.6±7.8	47.7±7.6	42.3±7.1	<0.001
LAV index (mL/m^2); mean±SD	43.7±14.9	52.6±15.5	36.9±10.3	<0.001
LA enlargement; n (%)	221 (73.2)	122 (94.6)	99 (57.2)	<0.001
Diastolic dysfunction; n (%)	134 (44.4)	98 (76.0)	36 (20.8)	<0.001
Right ventricle				
RV wall thickness (mm); median (P_{25}-P_{75})	5.3 (4.4 to 6.4)	5.5 (4.4 to 6.8)	5.2 (4.4 to 6.0)	0.264
Basal RV dimension (mm); mean±SD	30.1±13.5	32.5±14.1	28.2±12.9	0.008
Mid-cavity RV dimension (mm); mean±SD	24.7±12.2	25.7±12.2	23.9±12.1	0.220
Longitudinal RV dimension (mm); mean±SD	57.5±25.8	59.0±27.2	56.4±24.7	0.406
RV end diastolic area (cm^2); mean±SD	19.4±7.7	20.9±8.6	18.2±6.7	0.003
RV end systolic area (cm^2); mean±SD	9.9±4.2	11.0±5.4	9.2±2.9	0.001
RVFAC (%); mean±SD	48.0±8.4	47.0±9.8	48.7±7.2	0.098
TAPSE (mm); mean±SD	23.9±5.9	24.6±6.7	23.4±5.1	0.072
S'TV (cm/s); median (P_{25}-P_{75})	13.2 (11.3 to 15.2)	13.6 (11.8 to 15.4)	12.8 (11.1 to 15.0)	0.048
RIMPDopp; median (P_{25}-P_{75})	0.32 (0.24 to 0.37)	0.33 (0.25 to 0.39)	0.32 (0.24 to 0.36)	0.319
RIMPTDI; median (P_{25}-P_{75})	0.37 (0.31 to 0.43)	0.38 (0.29 to 0.44)	0.37 (0.31 to 0.43)	0.913
Impaired RV systolic function; n (%)	34 (11.3)	16 (12.4)	18 (10.4)	0.612

A=late diastolic velocities of mitral inflow; E=peak early diastolic velocities of mitral inflow; e'=longitudinal early diastolic myocardial velocity; LA=left atrium; LAV=left atrial volume; LV=left ventricular; LVEF=left ventricular ejection fraction; PAP=pulmonary artery pressure; PAEDP=pulmonary artery end-diastolic pressure; PASP=pulmonary artery systolic pressure; PH=pulmonary hypertension; PVR=pulmonary vascular resistance; RA=right atrium; RIMPDopp=right ventricular index of myocardial performance by Doppler method; RIMPTDI=right ventricular index of myocardial performance by tissue Doppler method, RV right ventricular; RVFAC=right ventricular fractional area change; S'TV=peak systolic myocardial velocity of lateral tricuspid annulus; SD=standard deviation; TAPSE=tricuspid annular plane systolic excursion; TR=tricuspid regurgitation

echocardiographic data in all patients as well as the comparisons between patients with and without PH. LV hypertrophy, LA enlargement, and diastolic dysfunction were significantly more common in patients with PH than in those without (p<0.001). Significant tricuspid and pulmonary regurgitation were present in 6.3% and 12.6% of patients, respectively, and both were significantly more common in patients with PH at 15.0% versus 0%, and 24.2% versus 4.0%, respectively (p<0.001).

Table 3. Baseline characteristics after the propensity score matching in all patients and the comparisons between patients with and without pulmonary hypertension

Variables	All patients (n=178)	PH (n=85)	No PH (n=93)	p-value
Age (years); mean±SD	46.1±13.6	45.4±15.3	46.7±12.0	0.524
Female; n (%)	83 (46.6)	45 (52.9)	38 (40.9)	0.107
Mode of dialysis; n (%)				
No dialysis	12 (6.7)	5 (5.9)	7 (7.5)	0.678
Peritoneal dialysis	27 (15.2)	15 (17.6)	12 (12.9)	
Hemodialysis	139 (78.1)	65 (76.5)	74 (79.6)	
Comorbidities; n (%)				
Hypertension	175 (98.3)	83 (97.6)	92 (98.9)	0.607
Dyslipidemia	72 (40.4)	35 (41.2)	37 (39.8)	0.850
Diabetes Mellitus	72 (40.4)	39 (45.9)	33 (35.5)	0.158

PH=pulmonary hypertension; SD=standard deviation

Table 4. Univariate and multivariate analyses of factors associated with pulmonary hypertension in all patients and propensity matched population

Factors	All patients				Propensity matched population			
	Univariate		Multivariate		Univariate		Multivariate	
	Odd ratio (95% CI)	p-value	Odd ratio (95% CI)	p-value	Odd ratio (95% CI)	p-value	Odd ratio (95% CI)	p-value
Edema	2.16 (1.24 to 3.76)	0.006	-	-	-	-	-	-
Diabetes mellitus	1.84 (1.08 to 3.12)	0.024	-	-	-	-	-	-
Basal RV dimension	1.03 (1.01 to 1.05)	0.010	-	-	-	-	-	-
LV ejection fraction	-	-	-	-	0.96 (0.92 to 0.99)	0.009	-	-
LV diastolic dimension	1.04 (1.01 to 1.07)	0.030	-	-	1.04 (1.001 to 1.09)	0.047	-	-
LV mass index	1.02 (1.01 to 1.02)	<0.001	-	-	1.02 (1.01 to 1.03)	<0.001	-	-
LAV index	1.11 (1.08 to 1.14)	<0.001	1.09 (1.05 to 1.12)	<0.001	1.12 (1.08 to 1.16)	<0.001	1.10 (1.05 to 1.14)	<0.001
E /e' ratio	1.22 (1.15 to 1.30)	<0.001	1.12 (1.05 to 1.20)	<0.001	1.27 (1.17 to 1.38)	<0.001	1.12 (1.08 to 1.29)	<0.001

CI=confidence interval; E=peak early diastolic velocities of mitral inflow; e'=longitudinal early diastolic myocardial velocity; LAV=left atrial volume; LV=left ventricular; RV=right ventricular

Propensity matched population

There were 178 patients after the propensity score matching. No significant differences were reported regarding age, gender, mode of dialysis and comorbidities such as hypertension, dyslipidemia, or diabetes mellitus, between patients with and without PH (Table 3).

Determinants of PH

Table 4 shows the univariate and multivariate parameters associated with PH in patients with ESRD before and after the propensity matched analyses. The LAV index and E/e' ratio, representing diastolic dysfunction, were independent echocardiographic determinants of PH.

Discussion

The present study demonstrated that PH was common in patients with ESRD. The LAV index and E/e' ratio were independently associated with PH. LV hypertrophy, LA enlargement, high E/e' ratio and diastolic dysfunction were more common in patients with PH than in those without. The present study demonstrated a link between diastolic dysfunction and PH in patients with ESRD and preserved LVEF.

The overall prevalence of PH in patients with ESRD was 42.7%. Previous studies have demonstrated that the prevalence of PH in patients with CKD varies widely, depending on the study design, region, and population, the definition of PH, the stage of CKD, the mode of dialysis, the method, and the technique of PAP estimation[1,2,4,5,8-13]. The prevalence of PH was greater in more advanced stage of CKD and among ERSD, in hemodialysis patients than those on peritoneal dialysis[3,5,8,11,26]. Previous studies have used various definitions of PH based on echocardiography of PASP of 35 mmHg or more in most studies and a range of 25

to 45 mmHg[1,2,4,6,9,12,26,27]. While PAP was measured non-invasively by Doppler echocardiography in most studies, other studies employed right heart catheterization as the diagnostic method and used the mean PAP of 25 mmHg or more as the definition of PH[9,12,20]. The estimation of PAP, including PASP, pulmonary artery end-diastolic pressure, mean PAP, and pulmonary vascular resistance, by Doppler echocardiography has been well-validated and correlated with the values obtained by right heart catheterization[16,17,19]. The cutoff value of PASP obtained by echocardiography to determine the presence of PH in the literatures remains the topic of discussion. The present study defined PH as mean PAP as 25 mmHg or more at rest, according to the standard recommendation[20]. However, in patients in whom peak pulmonary regurgitation velocity could not be obtained to estimate mean PAP, then PASP of 50 mmHg or more was designated to define PH[21,22,28], and to eliminate concerns about the ambiguity imposed by estimated right atrial pressure and the issue of mild PH versus normal variants.

PH is a common and unfavorable consequence in patients with ERSD and is associated with adverse outcomes[1-3]. Multiple possible etiologic factors have been proposed as the pathophysiologic mechanisms of PH in patients with ESRD, such as endothelial dysfunction, vascular calcification, inflammation, hemodialysis arteriovenous fistula, uremia, severe anemia, LV systolic and diastolic dysfunction, LV hypertrophy, and volume overload[4-6,29,30]. Underlying diseases associated with ESRD, such as hypertension, diabetes mellitus, metabolic syndromes, and sleep disorders may also be contributing pathogenic factors[4-7]. LV diastolic dysfunction is also common in patients with CKD and carries a poor prognosis[31,32]. Like the previously published data[32], the present study confirms that ESRD patients with PH had worse diastolic parameters, such as higher E, A, E/A ratio, E/e' ratio, and LAV index, than those without PH. Furthermore, the E/e' ratio and LAV index are independent determinants of PH, representing the link between LV diastolic dysfunction and PH in patients with ESRD. Similarly, the previous study by Reque et al. reported the diastolic dysfunction as a determinant of PH in non-dialysis-dependent CKD patients[3]. LV hypertrophy has been recognized as a physiological consequence of high LV filling pressure and volume overload, leading to LV diastolic dysfunction[33]. The present study found that the E/e' ratio and LAV index were independently associated with PH. The E/e' ratio has been recognized as an important echocardiographic parameter to evaluate LV filling pressure and high E/e' ratio was reported as an indicator of poor prognosis[34,35]. LV diastolic dysfunction may provide the pathological link to PH in patients with CKD through an increase in extracellular water, volume overload, an elevated LA pressure/size and eventually, elevated LV filling pressure[36-38]. The complexity of pathophysiologic relationships between LV diastolic dysfunction and PH requires further studies to reveal the potential mechanisms that may lead to novel approaches to modify the outcomes in patients with ESRD.

Limitation

In the present study, PAP was measured using Doppler echocardiography rather than right heart catheterization. However, the estimation of PAP by Doppler echocardiography has been studied and correlated with the values obtained by right heart catheterization. RV dysfunction was reported in 11.3% of patients in the present study, which might underestimate the Doppler echocardiographic assessment of PASP. The timing for the echocardiographic examination varied relative to the date of dialysis such as pre-dialysis versus immediate post-dialysis, and volume status might affect echocardiographic findings, especially PAP and diastolic parameters. Although all patients had clinically significant ESRD, the duration and etiology of disease were not available in all patients.

Conclusion

PH and associated LV diastolic dysfunction are prevalent in patients with ESRD. The present study has demonstrated and extended knowledge of the link between diastolic dysfunction and PH in patients with ESRD and preserved LVEF.

What is already known on this topic?

PH is prevalent in patients with ESRD and associated with poor outcomes.

What this study adds?

Diastolic dysfunction is common and links to PH in patients with ESRD and preserved LVEF.

Acknowledgement

The present study was supported by Faculty of Medicine Siriraj Hospital, Mahidol University. The authors are indebted to Herbert M Swick, MD for the manuscript preparation and grammatical editing.

Conflicts of interest

All authors have no conflict of interest.

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