EFFECT OF PULMONARY HYPERTENSION ON INTRADIALYTIC HYPOTENSION AMONG PATIENTS WITH END STAGE RENAL DISEASE

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ABSTRACT

Background: Intradialytic hypotension (IDH) is an important problem in end stage renal disease (ESRD). Therefore, this study aimed to assess the effect of pulmonary hypertension (PHT) on IDH among patients with ESRD using transthoracic echocardiography.

Methods: In this prospective etiognostic study, transthoracic echocardiography was performed among patients with ESRD in Burapha University Hospital, Thailand. The hemodialytic flow chart data of patients in the hemodialysis unit was collected to ascertain whether these patients presented IDH. The baseline clinical hemodialysis profiles and echocardiographic findings were analyzed using univariate predictors of IDH. Multivariate risk regression was used to identify independent predictors of IDH.

Results: A total of 35 patients with ESRD were enrolled between June 2020 and March 2021. Of these, 16 had PHT (45.7%). The incidence of IDH was 48.5%. All patients exhibited a normal left ventricular ejection fraction. No significant difference was observed of RVSP between frequent-IDH group and occasional-IDH group (45.33 ± 11.62 mmHg and 41.06 ± 13.78 mmHg, respectively, p=0.401). Using univariate analysis, being female, left ventricular mass index, left ventricular ejection fraction and PHT were significantly associated with IDH. No factors were indicated related to IDH occurrence using multivariate analysis. Nevertheless, female patients with ESRD presenting PHT illustrated a tendency to have IDH. This was evidenced by the risk ratio of being female and patients with PHT being 3.13 (95% CI: 0.74-13.30) and 2.18 (95% CI: 0.34-7.06), respectively.

Conclusion: Patients with ESRD presenting PHT showed a higher tendency of developing IDH during hemodialysis than patients with ESRD without PHT. The difference however was statistically insignificant.

Keywords: End stage renal disease, Intradialytic hypotension, Pulmonary hypertension

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Introduction

Intradialytic hypotension (IDH) is one of the complications associated with dialysis procedures, occurring at about 20 to 48%[1-3] of patients with end stage renal disease (ESRD). The condition is important in clinical settings because it could lead to even more serious complications such as an increase of mortality rate[4, 5] and myocardial ischemia from decreasing coronary blood flow.[6]

IDH condition has been hypothesized to cause a lower cardiac output. In fact, both left-sided and right-sided cardiac outputs are closely related. When the right-sided cardiac output is lower, whatever the cause, low blood pressure will occur. Therefore, hemodialytic sessions involving a removal of body fluid from blood vessels, resulting in a decreasing preload in the right ventricle, should be particularly cautioned because it may reduce the right-sided cardiac output. Special attention should be paid to the occurrence of associated conditions such as pulmonary hypertension (PHT).

Approximately 26 to 66% of patients with ESRD(7-11) already being treated by hemodialysis present PHT and are correlated with cardiovascular deaths.[9] Also, PHT is a strong independent predictor of mortality among patients undergoing hemodialysis.[12]

No related studies have significantly documented the relationship between PHT and IDH. Therefore, this study aimed to assess the effect of PHT on IDH among patients with ESRD using transthoracic echocardiography.

Methods

Study design and population

This constituted a prospective etiognostic study that performed echocardiography among patients with ESRD in Burapha University Hospital, Thailand between June 2020 and December 2020. After that, the hemodialysis flow chart data of patients in the hemodialysis unit was collected to ascertain whether these patients presented IDH. The protocol was approved for ethics considerations by the BUU Ethics Institutional Review Board (HS021/2563). All patients provided written informed consent.

The sample size was calculated by comparing the mean of two independent groups. A related study[13] indicated that on average, patients with ESRD receiving hemodialysis exhibited pulmonary artery systolic pressure about 33.9±10.6 mmHg. Moreover, experts suggested that patients with ESRD and IDH should have higher pulmonary artery systolic pressure than patients with ESRD without IDH. The difference was expected to be about 10 mmHg, which is a value predicting the emergence of poor long term survival.[13] Thus, patients with ESRD and IDH are expected to have pulmonary artery systolic pressure equal to 43.9 mmHg. The sample size was calculated to include 48 patients with ESRD in the study (24 patients in each group).

Patients with IDH under the condition that systolic blood pressure decreased by 20 mmHg or more, the mean arterial pressure decreased by more than 10 mmHg or blood pressure was lower than 100/60 with associated symptoms of hypotension including yawning, cramps, nausea, vomiting, tweaking and dizziness.[14] Patients in the frequent-IDH group needed to be particularly treated during the hemodialysis sessions. Examples of treatments included temporarily stopping an ultrafiltration (UF) operation, providing inotropic agents or saline.[15] Patients with frequent-IDH in this study exhibited the aforementioned condition more than three times during a three-month study, whereas patients with occasional-IDH showed the condition less than three times or none during the same timeframe. The hemodialysis chart of patients was reviewed to collect data about 1) general information including age, sex, body mass index (BMI), underlying diseases, causes of chronic renal failure, medicines used, and laboratory results and 2) cardiovascular disease-related information including previous HF hospitalization, coronary artery disease, history of pulmonary hypertension and history of syncope and 3) kidney dialysis related information including weekly frequency of kidney dialysis, length of time of each kidney dialysis, mean net UF weekly, position of catheter, pre/post HD weight, and pre/post blood pressure and heart rate. The inclusion criteria included patients (1) aged more than 18 years (2) having

treatment of hemodialysis for six months or more, (3) receiving hemodialysis at least twice weekly and at least four hours each session, (4) without history of catheter related infection and (5) without history of abnormal bleeding in the last month. The exclusion criteria included (1) BMI of 40 kg/m2 or more, (2) catheter-related infection in the last month, (3) bleeding disorder in the last month, (4) inadequate hemodialysis defined by losing HD twice in the last month, (5) significant left sided valvular heart disease (any aortic/ mitral stenosis), (6) HIV positive status and (7) pregnancy.

Hemodialysis

All patients with ESRD received dialysis two to three times weekly for four hours each session.

Transthoracic echocardiography

Echocardiography was performed before hemodialysis to collect important parameters including left ventricular ejection fraction (LVEF), left ventricular mass index (LVMi), pattern of left ventricular geometry, LA volume index, diastolic function, E/e', right atrial pressure, tricuspid regurgitation velocity, right ventricular systolic pressure (RVSP), mean pulmonary arterial pressure (mean PAP) using Abbas's formula and right ventricular systolic function (tricuspid annular plane systolic excursion and peak velocity of tricuspid annulus). Echocardiogram was performed by a cardiologist in Burapha Hospital. We used the American Society of Echocardiography guidelines and recommendations to assess and measure these parameters. The cardiologist performed all examinations using a diagnostic ultrasound system (Philips EPIQ CVx with a Philips X5-1 MHz Phased Array Probe).

Statistical Analysis

Subject characteristics were described using descriptive statistics, including frequency and percentage for categorical variables. Continuous variables were reported as means, standard deviation of normally distributed variables and median, minimum and maximum of normally distributed variables. Factors to predict IDH used the independent t-test for continuous variables and categorical variables used the Fisher exact test. Variables found to be significant in the univariate analysis, were entered in a multivariate risk regression analysis (backward elimination). For all tests performed, a two-tailed $p < 0.05$ was considered statistically significant.

Results

The data of 40 patients with ESRD were collected. Three patients were excluded from the study because parameter values using an echo-cardiogram were unable to be collected. Two patients with severe valvular heart disease were excluded. Finally, a total of 35 patients with ESRD were analyzed. Of these, 16 had PHT (45.7%), and incidence of IDH was 48.6%. General information of patients in both groups are shown in **Table 1** and hemodialysis profiles are shown in **Table 2.**

All patients exhibited normal LVEF and right ventricular systolic function (tricuspid annular plane systolic excursion and peak velocity of tricuspid annulus). Most patients presented Grade I diastolic function and concentric hypertrophy of the left ventricular geometric pattern. The echocardiographic data are shown in **Table 3**.

The primary research outcome was that a relationship between PHT and IDH was insignificant. When the dataset was subjected to univariate analysis, variables with a significant correlation with IDH were being female, LVMi, LVEF and PHT. The RVSP of the frequent-IDH group and the occasional-IDH group appeared to be 45.33 ± 11.6224.86 ± 8.16 mmHg, respectively (p=0.234).

Table 1. Clinical characteristics of frequent-IDH and occasional-IDH groups

Characteristic	Frequent-IDH (n=17)	Occasional-IDH (n=18)	*p*-value
Female, n (%)	14 (73.7)	5 (26.3)	0.002
Age (years, mean ± sd)	68.82 ± 12.51	66.11 ± 16.35	0.567
Body weight (kg, mean ± sd)	59.18 ± 16.14	60.78 ± 12.94	0.747
Body mass index (kg/m^2, mean ± sd)	24.32 ± 5.8	23.61 ± 4.74	0.691
Underlying disease, n (%)			
Hypertension	11 (45.8)	13 (54.2)	0.725
Coronary artery disease	2 (28.6)	5 (71.4)	0.402
Stroke	2 (66.7)	1 (33.3)	0.603
Diabetes mellitus	4 (43.8)	9 (56.2)	0.738
Dyslipidemia	15 (60.0)	10 (40.0)	0.06
Previous heart failure	1 (20.0)	4 (80.0)	0.338
Etiology of ESRD, n (%)			0.49
Diabetes mellitus	8 (44.4)	10 (55.6)	
HTN (HT nephrosclerosis)	5 (62.5)	3 (37.5)	
Glomerulonephritis	0	1 (100.0)	
Unknown	4 (50.0)	4 (50.0)	
Number of Anti HTN drugs (mean ± SD)	1.88 ± 1.22	2.23 ± 1.31	0.433
Nitrate, n (%)	0	2 (100.0)	0.486
Beta blocker, n (%)	4 (33.3)	8 (66.7)	0.289
CCB, n (%)	10 (47.6)	11 (52.4)	0.582
ACEI, n (%)	1 (50.0)	1 (50.0)	0.743
ARB, n (%)	5 (50.0)	5 (50.0)	0.604
Diuretic, n (%)	11 (47.8)	12 (52.2)	0.592
Hydralazine, n (%)	1 (25.0)	3 (75.0)	0.603
Laboratory investigation			
Hb (mg/dL, mean ± SD)	9.45 ± 1.54	9.76 ± 1.39	0.545
Albumin (mg/dL, mean ± SD)	3.8 ± 0.36	3.77 ± 0.31	0.77
Total calcium (mg/dL, mean ± SD)	8.85 ± 0.98	8.92 ± 0.8	0.813
Phosphate (mg/dL, mean ± SD)	4.68 ± 1.51	4.32 ± 0.93	0.415
PTH level (pg/mL, mean ± SD)	262.11 ± 242.11	284.73 ± 287.04	0.811

Values presented as mean±SD or n (%), *p*-values corresponded to independent-t test and Fisher's exact test.

ESRD: end stage renal disease, anti HTN drugs: antihypertensive drugs, ACEI: angiotensin-converting enzyme inhibitors, ARB: angiotensin II receptor blockers, CCB: calcium channel blockers, Hb: hemoglobin, PTH: parathyroid hormone

Table 2. Hemodialysis profiles of frequent-IDH and occasional-IDH groups

Hemodialysis profile	Frequent-IDH group (n=17)	Occasional-IDH group (n=18)	*p*-value
IDH symptoms, n (%)			0.198
None	7 (31.8)	15 (68.2)	
Fainting	4(66.6)	2 (34.4)	
Dyspnea	1 (100.0)	0	
chest pain	1 (100.0)	0	
abdominal pain	1 (100.0)	0	
muscle cramping	2 (66.7)	1 (33.3)	
back pain	1 (100.0)	0	
Frequency of dialysis, n (%)			0.315
2 sessions/week	9 (42.9)	12 (57.1)	
3 sessions/week	8 (57.1)	6 (42.9)	
Mean Net UF, (ml, mean ± sd)	2535.29 ± 829.11	2742.22 ± 1283.69	0.577
BFR, (ml/min, mean ± sd)	329.41 ± 25.36	327.78 ± 30.78	0.865
Dialysis membrane			0.591
HDF 80	9 (60.0)	6 (40.0)	
HDF 100	2 (25.0)	6 (75.0)	
Elisio 170	3 (42.9)	4 (57.1)	
Elisio 190	1 (50.0)	1 (50.0)	
Elisio 210	2 (66.7)	1 (33.3)	
PreHD SBP, (mmHg, mean ± SD)	141.06 ± 27.88	131.28 ± 24.48	0.274
PreHD DBP, (mmHg, mean ± SD)	64.24 ± 17.49	61.89 ± 15.13	0.673
PreHD HR, (bpm mean ± SD)	76.76 ± 15.83	78.06 ± 17.13	0.819
Position of catheters, n (%)			0.67
AVF	10 (47.6)	11 (52.4)	
AVG	1 (33.3)	2 (66.7)	
Perm cath	4 (44.4)	5 (55.6)	
DLC	2 (100.0)	0	

Values presented as mean±SD or n (%), *p*-values corresponded to independent-t test and Fisher's exact test.

Mean net UF: mean net ultrafiltration, BFR: blood flow rate, PreHD SBP: prehemodialysis systolic blood pressure, PreHD DBP: prehemodialyisis diastolic blood pressure, PreHD HR: prehemodialysis heart rate, AVF: arteriovenous fistula, AVG: arteriovenous graft, DLC: double lumen catheter.

These variables and other variables that were likely to be related to the occurrence of IDH including diabetes, coronary artery disease, albumin level, presystolic blood pressure, prediastolic blood pressure, LVMi and LVEF were then analyzed using multivariable analysis. Results showed that no factors were related to IDH occurrence. Nevertheless, female patients and patients with PHT illustrated a tendency to have IDH. This was evidenced by the risk ratio of being female and patients with PHT at 3.13 (95% CI= 0.74-13.30) and 2.18 (95% CI= 0.34-7.06), respectively. multivariate risk regression analysis to determine factors associated with IDH is shown in **Table 4.**

Table 3. Echocardiographic data between frequent-IDH and occasional-IDH groups

Echocardiographic findings	Frequent-IDH group (n=17) (mean ± SD)	Occasional-IDH group (n=18) (mean ± SD)	*p*-value
LVDd (mm)	44.07 ± 5.8	41.47 ± 5.77	0.193
LV mass index (g/m^2)	128.48 ± 26.52	110.22 ± 21.69	0.032
RWT	0.62 ± 0.14	0.69 ± 0.14	0.173
LV geometry, n (%)			0.125
concentric remodeling	2 (22.2)	7 (77.8)	
concentric hypertrophy	14 (56.0)	11 (44.0)	
LVEF (%)	64.08 ± 11.19	72.87 ± 5.89	0.006
Diastolic function, n (%)			0.429
normal	1 (25.0)	3 (75.0)	
grade I	13 (54.8)	11 (45.8)	
grade II	2 (33.3)	4 (66.7)	
grade III	1 (100.0)	0	
E/e’	17.83 ± 7.01	15.15 ± 6.32	0.243
LA volume index (mL/sqm)	47.38 ± 13.85	50.12 ± 14.92	0.579
TAPSE (mm)	25.42 ± 3.86	25.29 ± 4.78	0.931
Peak velocity of tricuspid annulus (cm/sec)	12.7 ± 2.51	11.99 ±1.62	0.326
mean PAP (mmHg)	28.28 ± 6.94	24.86 ± 8.16	0.234
RVSP (mmHg)	45.33 ± 11.62	41.06 ± 13.78	0.401
PHT, n (%)	11 (68.8)	5 (31.3)	0.044
RAP (mmHg)	8.54 ± 1.82	8.71 ± 2.12	0.788
CO (mL/min)	5.31 ± 2.25	4.32 ± 0.98	0.100

Values presented as mean±SD or n (%), *p*-values corresponded to independent-t test and Fisher’s exact test.

LVDd: left ventricular dimension in diastole, LV mass index: left ventricular mass index, RWT: relative wall thickness, LV geometry: left ventricular geometry, LVEF: left ventricular ejection fraction, LA volume index: left atrial volume index, TAPSE: tricuspid annular plane systolic excursion, Mean PAP: mean pulmonary arterial pressure RVSP: right ventricular systolic pressure, PHT: pulmonary hypertension, RAP: right atrial pressure, CO: cardiac output.

Discussion

IDH is a serious and frequent complication of chronic hemodialysis. IDH is the end result of the interaction between ultrafiltration rates, cardiac output and arteriolar tone.[16] As mentioned previously, we hypothesized that the occurrence of IDH among patients undergoing chronic hemodialysis was related to reduced cardiac output and PHT. When developing IDH, the human body exhibits the following adapted mechanisms.[17] First, interstitial fluid moves to blood vessels to refill the blood volume. Second, increased sympathetic outflow to arteriolar vasoconstriction and increased peripheral vascular resistance, helps to maintain BP. Finally, the heart contracts more, causing the rising heart rate to increase cardiac output and raise BP. When all of these mechanisms fail, IDH eventually strikes. Decreased cardiac output plays a key role in the patho-physiology of IDH. Cardiac output depends on preload, afterload, heart rate and contractility. Changes in preload, determined mainly by

Table 4. Multivariate regression analysis to determine factors associated with intradialytic hypotension

Prognostic factor	Frequent-IDH (n=17)	Occasional-IDH (n=18)	Risk Ratio	Adjusted Risk Ratio (95% CI)	*p*-value
PHT, n (%)	11 (68.8)	5 (31.3)	2.18	1.56 (0.34-7.06)	0.565
Sex, n (%)					
male	4 (23.5)	13 (76.5)	1		0.120
female	14 (73.7)	5 (26.3)	3.13	3.14 (0.74-13.30)	
DM, n (%)	8 (47.1)	9 (52.9)	0.89	0.49 (0.13-1.92)	0.308
CAD, n (%)	3 (37.5)	5 (62.5)	0.70	0.81 (0.16-4.18)	0.799
Albumin, (mean±SD)	3.85±0.41	3.77±0.31	1.39	0.53(0.06-4.30)	0.528
PreSBP (mean± SD)	75.0±21.97	114.5±20.63	1.0	0.98 (0.94-1.04)	0.666
PreDBP (mean± SD)	42.17±13.36	59.67±13.91	1.0	0.98 (0.94-1.04)	0.538
LVMi, (mean± SD)	128.48±26.52	110.22±21.69	1.01	1.0 (0.97-1.03)	0.798
LVEF, (mean± SD)	64.08±11.19	72.87±5.89	0.96	0.97 (0.91-1.04)	0.427

PHT: pulmonary hypertension, DM: diabetes mellitus, CAD: coronary artery disease, PreSBP: presystolic blood pressure, PreDBP: prediastolic blood pressure, LVMi: left ventricular mass index, LVEF: left ventricular ejection fraction

intravascular volume, seem to play a major role in the development of IDH.[16] Therefore during our hemodialysis sessions, intravascular fluid has to be removed. Patients presenting pulmonary hypertension may experience induced IDH due to decreasing cardiac output.

multivariate risk regression revealed that such a relationship does not exist. However, the chance that patients with ESRD and PHT condition (indicated by their RVSP being equal or greater than >50 mmHg or their mean PAP by Abbas's formula being equal or greater than >25 mmHg) will develop IDH 2.18 times that of patients without PHT. The pathogenesis of PHT in this population remains poorly understood. Reported associations include arteriovenous fistulae, cardiac dysfunction, fluid overload, bone mineral disorder and non-biocompatible dialysis membranes.[18]

This study also illustrated that female patients had a higher chance of developing IDH than male patients. In particular, our multivariate risk regression analysis indicated that the chance of female patients with IDH was 3.13 times than that of male patients. This finding was consistent with several studies. For example, Andras Tisler et al.[19] found that being female was a statistically significant factor of IDH occurrence. Similarly, Johanna et al.[20] examining the prevalence of IDH among patients with conventional hemodialysis, found that four determining factors of IDH included diabetes, high interdialytic weight gain, being female, and low body weight. Studies of Johanna Kuipers et al.[21] and Orofino L et al.[22] also found similar results, that female patients with low body weight increases the chance of having IDH even further. This could be explained in that females in general have lower body weight and, consequently, have a higher UF rate (mL/h/kg body weight) during hemodialysis for a similar interdialytic weight gain than males.[20]

Patients with frequent IDH exhibit higher LVMi than patients with occasional IDH indicating that these patients have left ventricular hypertrophy (LVH). For patients with chronic renal failure, the prevalence of LVH increases progressively as renal function deteriorates. The development of LVH results from coronary hypoperfusion,

myocardial stunning and renin-angiotensin-aldosterone system dysregulation. Finally, LVH actively contributes to IDH occurrence, through the induction of LV stiffening, myocardial ischemia and arrhythmia.[23]

Barberato et al.[24] observed that patients experiencing ESRD with LVEF less than 50% and LA volume index greater than 35 mL/m2 indicated diastolic dysfunction, which is a factor that can significantly predict the occurrence of IDH. This study found that both groups of patients (frequent-IDH and occasional-IDH) exhibit normal LVEF, i.e., greater than 60%, and normal cardiac output measured by an echocardiogram. Therefore, high LA volume index will not necessarily cause IDH during chronic hemodialysis.

Apart from patients with ESRD, patients with PHT must be treated with noncardiac operations. This patient group represented an important risk factor for increased perioperative morbidity and mortality. In other words, these patients illustrated a significantly increased risk for hemodynamic instability.[25] Thus, physicians have to give special attention to these patients by monitoring closely, optimizing systemic BP, oxygenating and ventilating, avoiding exacerbating factors, and using vasopressors and pulmonary vasodilators whenever necessary as essential elements of management.[26]

One limitation of our study was the small sample size; only 40 patients could be enrolled during the study period. This restriction affected the multivariate risk regression analysis in that its revealed risk of IDH factors was unclear. Although female patients and patients with PHT showed a higher chance of IDH occurrence than their counterparts, the difference was not statistically significant.

Conclusion

Patients with ESRD and PHT measured by RVSP or mean PAP during echocardiography showed a higher tendency of developing IDH during hemodialysis than patients with ESRD without PHT. Therefore, patients with ESRD who were diagnosed having PHT showed clinical significance so that physicians had to closely monitor the possible occurrence of hypotension during hemodialysis.

Conflict of interest

The authors declare they have no conflict of interest.

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