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Evaluation of Risk Factors for Development of Pulmonary Hypertension in Patients with End Stage Renal Disease Undergoing Hemodialysis

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Abstract

Background: Pulmonary arterial hypertension (PAH) has recently been identified as a common complication in patients with end-stage renal disease (ESRD) who are undergoing hemodialysis (HD) or peritoneal dialysis (PD). The risk factors for the development of PAH in those patients are not well understood.
Objective: To investigate the risk factors for the development of PAH in patients with ESRD on HD.
Patients and Methods: This is a hospital-based cross-sectional study of 50 ESRD patients undergoing HD. The pulmonary artery systolic pressure (PASP) was measured using echocardiography. PASP > 25 mmHg at rest was defined as PAH. As a result, patients were divided into two groups: those who had PAH and those who did not. Each patient's demographic, biochemical, and echographic findings were documented.
Results: Out of 50 patients, 19 (38%) had PAH, while the remaining 31 (62%) had normal PASP. In multivariate analysis, HD duration > 3.4 years (OR= 2.13, 95%CI=1.45-31.38, p= 0.025), hypertension as a cause of ESRD (OR=6.12, 95%CI=1.4-26.77, p=0.031), hemoglobin (Hb) ≤ 10.0 g/dl (OR= 4.35, 95%CI=1.88-9.84, p= 0.018), and left ventricular ejection fraction (LVEF)≤ 55% (OR= 6.75, 95%CI=1.87-23.74, p=0.021) were independent factors associated with PAH. PASP had a significant positive correlation with the rate of fistula flow (r= 0.295, p= 0.038) and E/A ratio (r= 0.368, p= 0.008), but a significant negative correlation with LVEF (r= -0.345, p= 0.014). PASP had a positive significant correlation with each of rate of fistula flow (r= 0.295, p= 0.038) and E/A ratio (r= 0.368, p= 0.008), while it has a negative significant correlation with LVEF (r= -0.345, p= 0.014).
Conclusion: Longer duration of HD, hypertensive nephropathy as a cause of ESRD, Hb≤ 10 g/dl, and LVEF ≤55% are among the demographic, biochemical, and clinical factors associated with the development of PAH in patients with ESRD under HD. The PASP has a positive correlation with fistula flow rate and E/A ratio and a negative correlation with LVEF.
Keywords: Pulmonary arterial hypertension, End stage renal disease, Hemodialysis.

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Introduction

Pulmonary arterial hypertension (PAH) is a common dyspnea-fatigue illness caused by a progressive increase in vascular resistance of the lung with an ultimate right ventricular failure. According to the European Society of Cardiology (ESC), PAH is defined as an increase in the mean pulmonary arterial pressure (mPAP) more than 25 mm Hg at rest [1]. In contrast, the Sixth World Symposium on Pulmonary Hypertension defined PAH as mPAP > 20 mm Hg at rest [2].

Echocardiographic measures contribute in thorough risk assessment in patients with PAH. Several other parameters including right atrial area and the tricuspid annular plane systolic excursion (TAPSE) to pulmonary arterial systolic pressure (PASP) ratio are other key prognostic indices for PAH [3].

Patients with ESRD, defined by a glomerular filtration rate of less than 15 mL/min [4,5], are generally managed by dialysis or a conservative, non-dialysis pathway [6]. PAH is exceedingly common in both pre-dialysis and dialysis populations, yet it is commonly neglected and under-diagnosed [7]. It has a median prevalence of 38% in all dialysis patients, 40% on HD and 19% on peritoneal dialysis (PD) [8].

Predisposing factors for PAH in HD patients are uncertain and have received little attention. Previous studies have shown that patient's age, type of atrioventricular fistula (AVF), abnormalities in bone minerals, duration of HD, and dysfunction in systolic and diastolic heart beats may all have some role [9-12].

The gold standard for HD access is an arteriovenous fistula. It causes increased venous return with a corresponding increase in cardiac output, as well as decreased systemic vascular resistance [13]. Increased cardiac output caused by AVF, endothelial dysfunction in the pulmonary vasculature resulting in plexiform lesions, disparities in vascular tone caused by nonstandard vasoactive mediator production, limited and general inflammation, vasoconstriction, and vascular sclerosis generated by dialysis circuit microbubbles has all been linked to PAH pathogenesis [14,15].

The effect of AVF as a risk factor for PAH in HD patients depends mainly on the type and duration of AVF and flow rate by which the blood crosses the AVF [16,17].

This study aimed to look at the determining the incidence of PAH among ESRD patients and assess the risk factors associated with this disease.

Patients and Methods

This is a hospital-based cross-sectional study that was conducted on 50 ESRD patients undergoing HD at the dialysis unit in Al-Imamain Al-Kadhumain Medical City from 1st April 2020 to 1st April 2021. The Iraqi Board for Medical Specialization approved the study. Each participant signed a written consent form.

Adult patients over the age of 18 who have been receiving maintenance HD via AVF for at least 6 months due to ESRD are eligible for the study. Patients with valvular heart disorders, pulmonary obstructive illnesses, connective tissue pathologies, pulmonary thromboembolism, chronic liver disease, and

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thyroid dysfunction were excluded from the study.

All patients had their demographics, biochemical characteristics, and echocardiographic data were collected. The patients were categorized into those with and without PAH. The following data were collected from each patients.

Clinical characteristics such as age, gender, BMI, hypertensive nephropathy, causes of ESRD, diabetic nephropathy, obstructive uropathy, and neurogenic bladder. Dialysis-related factors such as HD duration, AVF duration, and flow using Doppler ultrasonography (GE healthcare, Voluson730 pro V, Austria). Laboratory parameters like hemoglobin, serum albumin, and blood urea. One hour following HD, echocardiography (Vivid E9, GE Healthcare, Milwaukee, WI, USA) was utilized to examine structural and functional heart alterations. The parameters assessed include left ventricular ejection fraction (LVEF), TAPSE, and the diastolic transmitral inflow velocity to early diastolic mitral annulus velocity ratio (E/A).

Bernoulli equation was utilized calculate the pulmonary artery systolic pressure (PASP) from tricuspid regurgitant velocity (TRV) and right atrial (RA) pressure (PASP = 4(TRV)2+RA pressure) to be greater than 25 mmHg, [7].

Statistical Analysis

The t-test and analysis of variance were employed to compare continuous variables. The Chi-square test was accomplished to match categorical data. The odds ratio (OR) and its 95% confidence interval (CI) were assessed using multivariate logistic regression analysis to evaluate the risk factors for PAH. To study the probable association between PASP and other factors, Pearson's correlation was used. Statistical significance was defined as P-values less than 0.05.

Results

Demographic patient characteristics

The patients' average age was 49.88±12.34 years (range 25-69 years). Males made up slightly more than half of the patients (56%). The average BMI was 26.25±7.77 kg/m2. The most common underlying cause of CKD was hypertension (42%), followed by diabetes (40%). Both dialysis and the fistula lasted 3.39±2.3 and 3.04±1.75 years, respectively. The fistula flow rate ranged from 520 to 1400 ml/min with a mean of 763.76±189.71 ml/min as indicated in Table (1).

Table (1): Patients' characteristics and demographic data (n=50)

Variables	Values
Age, years	49.88± 12.34
Gender	
Male	28(56%)
Female	22(44%)
Body mass index, kg/m2	26.25± 7.77
Duration of dialysis, years	3.39±2.3 (1-10)
Duration of fistula, years	3.04±1.75 (0.25-7.0)
Fistula flow, ml/min	763.76±189.71 (520-1400)
Causes of CKD	
Hypertensive nephropathy	21(42%)
Diabetic nephropathy	20(40%)
Others*	9(18%)

*including obstructive uropathy and neurogenic bladder. The data are presented as mean±SD and range

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Biochemical and echocardiographic parameters

With a mean Hb level of 10.0± 0.89 g/dl, the vast majority of patients were anemic. Serum creatinine and blood urea levels increased by a mean of 10.1±2.71 mg/dl and 79.68±21.03 mg/dl, respectively. The average LVEF was 54.24± 9.57%. TAPSE measurements ranged from 11 to 27 mm with a mean of 17.5±3.34mm. The E/A ratio ranged between 0.6 and 2.1 with a mean of 1.14±0.4. Finally, the mean PASP was 26.8±8.97 mmHg as shown in Table (2).

Table (2): Biochemical and echocardiographic parameters

Variables	Values
Hemoglobin, g/dl	10.0± 0.89
Creatinine, mg/dl	10.1±2.71
Blood urea, mg/dl	79.68±21.03
LVEF, %	54.24± 9.57
TAPSE, mm	17.5±3.34
E/A ratio	1.14±0.4
PASP, mmHg	26.8±8.97

*SD: standard deviation, LVEF: left ventricle ejection fraction, TAPSE: Tricuspid annular plane systolic excursion, PASP = pulmonary artery systolic pressure

According to the European Society of Cardiology (ESC)/European Respiratory Society guidelines, 19 (38%) patients had PAH, while the remaining 31 (62%) patients had normal PASP.

Association of demographic characteristics of the patients with PAH

Three demographic characteristics were found to be significantly related to PAH. There was a significant difference in the prevalence of hypertension as a cause of CKD among those with PAH versus those without PAH (68.42% versus 25.81%). Similarly, those with PAH had a significantly longer mean duration of HD (4.37±2.91 years) than those without PAH (2.8±1.61 years) with a significant difference. Furthermore, the rate of fistular flow was significantly higher in those with PAH than in those without PAH (835.79±237.94 ml/min versus 719.61±139.7 ml/min, respectively) as demonstrated in Table (3).

Table (3): Association of demographic characteristics of the patients with PAH

Variables	Without PAH (n=31)	With PAH(n=19)	p-value
Age, years	48.79± 14.35	50.55±11.14	0.630
Gender Male Female	 17(%54.84) 14(45.16%)	 11(57.89%) 8(42.11%)	0.833
Body mass index, kg/m2	26.6± 5.38	25.66±3.6	0.504
Duration of dialysis, years	2.8±1.61	4.37±2.91	**0.018**
Duration of fistula, years	2.73±1.81	3.54±1.58	0.113
Fistula flow, ml/min	719.61±139.7	835.79±237.94	**0.034**
Causes of CKD Hypertensive nephropathy Diabetic nephropathy Others	 8(25.81%) 15(48.9%) 8(25.81%)	 13(68.42%) 5(26.32%) 1(5.26%)	**0.010**

*CKD: chronic kidney disease

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Association of biochemical and echocardiographic parameters with PAH

Hemoglobin was the only biochemical parameter that was found to be significantly related to PAH. In those with PAH, the mean Hb concentration was 9.61±0.8 g/dl compared to 10.23± 0.87 in those without PAH. Three echocardiographic parameters were also found to be significantly related to PAH. Patients with PAH had significantly lower mean LVEF and TAPSE (48.84±7.45% and 15.98±2.95 mm, respectively) than those without PAH (57.55±9.31% and 18.44±3.27 mm, respectively).

Patients with PAH, on the other hand, had a significantly higher E/A ratio than patients without PAH (1.33±0.43 versus 1.02±0.33, respectively). The mean PASP in patients with PAH was significantly higher than that in patients without PAH (35.37±9.32 mmHg versus 21.55±2.09 mmHg, respectively), as shown in Table (4).

Table (4): Association of biochemical and echocardiographic parameters with PAH

Variables	Without PAH (n=31)	With PAH (n=19)	p-value
Hemoglobin, g/dl	10.23± 0.87	9.61±0.8	**0.016**
Creatinine, mg/dl	9.93±2.4	10.36±3.19	0.592
Blood urea, mg/dl	78.8±18.59	81.11±25.0	0.711
LVEF, %	57.55±9.31	48.84±7.45	**0.001**
TAPSE, mm	18.44±3.27	15.98±2.95	**0.010**
E/A ratio	1.02±0.33	1.33±0.43	**0.006**
PASP, mmHg	21.55±2.09	35.37±9.32	**<0.001**

* LVEF: left ventricular ejection fraction, TAPSE: Tricuspid annular plane systolic excursion, E/A: E wave/ A wave, PASP: pulmonary artery systolic pressure

Multivariate Analysis

Each variable with a p-value of 0.15 in the primary analysis was entered into a multivariate logistic regression model to find independent factors associated with PAH. Each quantitative variable was divided into two categories for this analysis by using an appropriate cut-off value. The findings revealed that HD duration > 3.4 years (OR= 2.13, 95%CI=1.45-31.38, p= 0.025), hypertension as a cause of CKD (OR=6.12, 95%CI=1.4-26.77, p=0.031), Hb≤ 10.0 g/dl (OR= 4.35, 95%CI=1.88-9.84, p= 0.018), and LVEF≤ 55% (OR= 6.75, 95%CI=1.87-23.74, p=0.021) were independent factors associated with PAH as indicated Table (5).

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Table (5): Multivariate analysis

Variables	Patients		p-value	OR(95%CI)
	Without PAH (n=31)	With PAH (n=19)		
HD Duration, years				
≤3.4	22(70.97%)	8(42.22%)	0.025	1.0
>3.4	9(29.03%)	11(57.89%)		2.13(1.45-31.38)
Fistula duration				
≤3.0	21(67.74%)	10(52.63%)	0.155	1.0
>3	10(32.26%)	9(47.37%)		6.19(0.62-14.3)
Causes of CKD				
Other causes	8(25.81%)	1(5.26)	0.175	1.0
Hypertensive nephropathy	8(25.81%)	13(68.42%)	0.031	6.12(1.4-26.77)
Diabetic nephropathy	15(48.39%)	5(26.32%)	0.085	1.39(0.03-8.32)
Hb, g/dl				
>10.0	19(61.29%)	5(26.32%)	0.018	1.0
≤10.0	12(38.71%)	14(73.68%)		4.35(1.88-9.84)
Fistula flow, ml/min				
≤760	20(64.52%)	9(47.37%)	0.193	1.0
>760	11(35.48%)	10(52.63)		1.77(0.1-30.6)
LVEF, %				
>55	21(67.74%)	4(21.05%)	0.021	1.0
≤ 55	10(32.26%)	15(78.94%)		6.75(1.87-23.74)
TAPSE, mm				
>17	23(74.19%)	8(42.22%)	0.075	1.0
≤17	8(25.81%)	11(57.89%)		5.26(0.82-10.32)
E/A ratio				
>1.0	15(48.39%)	16(84.21%)	0.068	1.0
≤1.0	16(51.61%)	3(15.79%)		2.41(0.67-87.11)

Correlation of PASP with other Variables

Pearson's correlation was used to investigate the relationship between PASP and other quantitative variables. As shown in table 6 and figures 1 through 3, PASP has a positive significant correlation with each of the fistula flow rates (r= 0.295, p= 0.038) and E/A ratio (r= 0.368, p= 0.008), but a negative significant correlation with LVEF (r= -0.345, p= 0.014).

Table (6): Pearson's correlation between PASP and other quantitative variables

Variables	Coefficient	p-value
Age, years	0.010	0.948
Body mass index, kg/m2	-0.005	0.974
Duration of dialysis, years	0.121	0.402
Duration of fistula, years	0.118	0.416
Fistula flow, ml/min	**0.295**	**0.038**
Hemoglobin, g/dl	-0.165	0.252
Creatinine, mg/dl	-0.021	0.886
Blood urea, mg/dl	-0.146	0.180
LVEF, %	**-0.345**	**0.014**
TAPSE, mm	-0.191	0.184
E/A ratio	**0.368**	**0.008**

*LVEF: left ventricular ejection fraction, TAPSE: Tricuspid annular plane systolic excursion, E/A: E wave/ A wave

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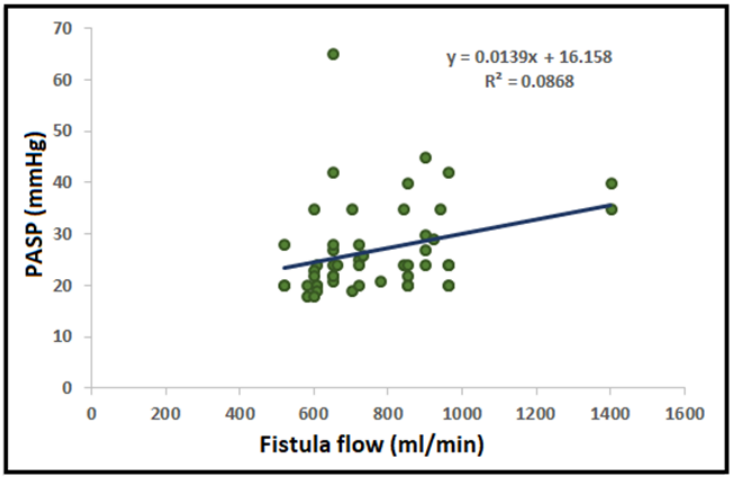

Figure (1): Scatter plot and regression line between Fistula flow and PASP

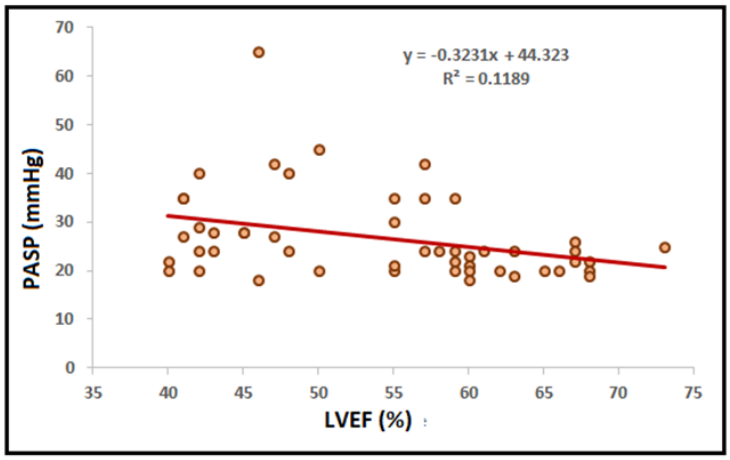

Figure (2): Scatter plot and regression line between LVEF and PASP

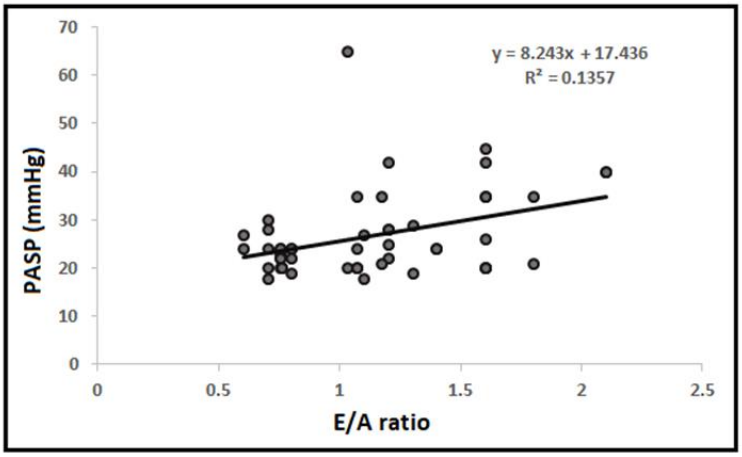

Figure (3): Scatter plot and regression line between E/A ratio and PASP

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Discussion

According to the findings of this study, 19 (38%) ESRD patients had PAH, while 31 (62%) had normal PASP. This rate is in line with international rates. According to a review of 27 studies, the prevalence of PAH ranged from 9-39% in ESRD patients, 18.8-68.8% in HD patients, and 0-42% in PD patients [18]. A very close rate (41%) was recorded on 100 patients in an Indian study [19], who were either on conservative management, HD, or PD. However, higher rates (50% to 77.3%) were reported in many other studies [20-23], and others reported a prevalence rate as low as 26.74% [24].

The variation in the prevalence of PAH among ESRD patients in different studies can be explained by differences in ethnicity as well as demographic characteristics of the study group, such as stage of ESRD, mode of dialysis (HD vs PD), comorbid conditions, and inclusion criteria [25].

Although the exact mechanisms of PAH in ESRD are unknown, several proposed explanations including anemia, fluid overload, an increased cardiac index, left ventricular diastolic dysfunction, and possibly pulmonary artery calcifications are the main culprits [18,26].

Age, gender, and BMI had no significant association with the development of PAH in ESRD patients in this study. Many studies around the world support these findings [10,19,27-29]. All these studies found no significant association between demographic characteristics of the patients and the development of PAH. However, Moniruzzaman et al. (19) discovered a 2:1 male predominance in their study.

The current study found that HD duration > 3.4 years (OR= 2.13, 95%CI=1.45-31.38, p= 0.025) and hypertension as a cause of CKD (OR=6.12, 95%CI=1.4-26.77, p=0.031) were significantly associated with the development of PAH in ESRD patients in multivariate analysis. That means that ESRD patients with more than 3.4 years of HD and those with underlying hypertension are 2.13-fold and 6.12-fold more likely to develop PAH than those with less than 3.4 years of HD and no underlying hypertension.

In agreement with these findings, Mehta et al. [10] and Fabian et al. [30] found that both diabetes and hypertension had a strong association with PAH. Agarwal et al. [21], on the other hand, demonstrated that DM, but not hypertension, was significantly associated with PAH. Mehta et al. [10] discovered that people with HD for a long time had a higher prevalence of PAH. Emara et al. [31], Patel et al. [19], and Havlucu et al. [32] discovered a similar link. In one study, increasing the duration of dialysis from one to two years increased the prevalence of PAH from 38 to 58% [33].

The most common cause of kidney disease is hypertension, which causes LV diastolic dysfunction and raises pulmonary venous and arterial pressure [34]. The longer a patient has CKD, the longer they are exposed to influence cardiovascular normal physiology, including the synergistic impacts of elevated pulmonary vesicular resistance, elevated cardiac output, and increased PCWP, and thus the probabilities of having more severe PAH [35].

Another intriguing finding in the present study was that Hb levels of <10.0 g/dl (OR= 4.35, 95%CI=1.88-9.84, p= 0.018) were

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significantly related to PAH. There is almost universal agreement among studies on the role of Hb in the development of PAH in ESRD patients. According to Etemadi et al. [36], Hb levels in PAH patients were significantly lower than in normal PAH patients. When patients with and without PAH were compared, the Hb concentration in patients with PAH was found to be significantly lower than in those without PAH [29,37].

It has been demonstrated that severe anemia can have an impact on pulmonary circulation. Low Hb levels may contribute to PAH by exacerbating hypoxia [38]. Chronic hypoxia changes the morphological and biochemical phenotypes of the pulmonary arteries, as well as the biochemical and physiological properties of vascular cells [39]. When alveolar hypoxia continues, there will be pulmonary vasoconstriction, which is compensated by an increase in PAP, which is escort by structural alterations in peripheral pulmonary arteries, involving increased the thickness of artery wall [40].

According to the current study, a higher rate of AVF flow was associated with PAH in univariate analysis but not in multivariate analysis. This could be due to the interdependence of AVF flow and other risk factors like hypertension or Hb level.

In general, previous studies have reported on the effects of AVF on PAH. Mehta et al. [10] discovered that 51 of 57 patients with AVF had PAH, while 21 of 31 patients without AVF had PAH. This demonstrates a strong link between AVF and PAH ($p = 0.002$). Havlucu et al. [32] discovered a similar relationship. In one case report, a brachiocephalic fistula's high output state (3,600 ml/min) contributed to the development of secondary PAH, which was resolved after fistula ligation [41]. This finding, however, was not replicated by Agarwal et al. [18]. This disparity could be explained by differences in AVF duration and population studied.

Only LVEF 55% (OR= 6.75, 95%CI=1.87-23.74, p=0.021) was found to be an independent factor for PAH in the current study. According to these findings, Zhang et al. [29] discovered a significant decrease in LVEF, TAPSE, and mitral annulus velocity (E/E') in PAH patients compared to controls. Another study examining the prevalence and characteristics of PAH in PD patients discovered that PAH patients had lower LVEF than those without PAH [42].

PASP had a positive significant correlation with fistula flow rate and E/A ratio in the current study, but a negative significant correlation with LVEF. In a related Egyptian study, there was a significant positive correlation between PASP and dialysis duration, ferritin level, cardiac output, and AV fistula flow, but a significant negative correlation between PASP and Hb level and serum iron [23].

The present study has two major limitations: it is a single-center study with a relatively small number of patients. Furthermore, no follow-up was performed to evaluate the effect of PAH on morbidity and mortality.

Conclusions

The study concludes that the prevalence of PAH in patients with ESRD under HD is comparable to the global rate. The longer the duration of HD, hypertensive nephropathy as a cause of ESRD, ≤Hb 10 g/dl, and LVEF ≤55% are the demographic, biochemical, and

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clinical factors associated with the development of PAH in patients with ESRD under HD. Finally, the PASP has a positive correlation with fistula flow rate and E/A ratio but a negative correlation with LVEF.

Recommendations

ESRD patients with prolonged HD, those with hypertension, low Hb and low LVEF should undergo regular checking for their PAH because they are under greater risks to develop pulmonary hypertension than their counterpart's patients with no such features.

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Ethical clearance: The study was approved by the Iraqi Council for Medical Specialization.

Conflict of interest: Nil

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تقييم عوامل الخطر لتطور ارتفاع ضغط الدم الرئوي لدى المرضى الذين يعانون من مرض الكلى في نهاية المرحلة والذين يخضعون لغسيل الكلى

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الملخص

خلفية الدراسة: يعد ارتفاع ضغط الدم الشرياني الرئوي من المضاعفات الشائعة في المرضى الذين يعانون من مرض الكلى في مرحلته النهائية والذين يخضعون للغسيل الكلى الدموي أو غسيل الكلى البريتوني. عوامل الخطر المرتبطة بتطور ارتفاع ضغط الدم الشرياني الرئوي في هؤلاء المرضى غير مفهومة جيدًا.

اهداف الدراسة: لتقييم العوامل المرتبطة بتطور ارتفاع ضغط الدم الشرياني الرئوي في مرضى الداء الكلوي بمراحله الأخيرة الذين يتلقون علاجا بالغسيل الكلوي.

المرضى والطرائق: شملت هذه الدراسة المقطعية على 50 مريضًا يعانون من الداء الكلوي بمراحله الأخيرة تحت الغسيل الكلوي الدموي. استخدم تخطيط صدى القلب لقياس الضغط الانقباضي للشريان الرئوي .تم تعريف ارتفاع ضغط الدم الشرياني الرئوي على أنه تخطي الضغط الانقباضي في الشريان الرئوي حاجز 25 مم زئبق عند الراحة. وفقًا لذلك ، تم تصنيف المرضى إلى مجموعتين: مع ارتفاع ضغط الدم الشرياني الرئوي او بدونه. تم تسجيل العوامل الديموغرافية والبيوكيميائية وتخطيط صدى القلب لكل مريض.

النتائج: من بين 50 مريضا أظهر 19 مريضا (38٪) ارتفاعا في ضغط الدم الشرياني الرئوي بينما كان لدى المرضى الآخرين 31 (62٪) ضغطا انقباضيا طبيعيا في الشريان الرئوي. في التحليل متعدد المتغيرات ، كانت مدة الغسيل الكلوي > 3.4 سنوات ((OR= 2.13, 95%CI=1.45-31.38, p= 0.025) ، ارتفاع ضغط الدم كسبب للداء الكلوي بمراحله الأخيرة (OR=6.12, 95%CI=1.4-26.77, p=0.031) وتركيزالهيموكلوبين≥ 10.0 جم / ديسيلتر(OR= 4.35, 95%CI=1.88-9.84, p= 0.018) وقذفة البطين الأيسر≥ 55% (OR= 6.75, 95%CI=1.87-23.74, p=0.021) عوامل مستقلة مرتبطة بارتفاع ضغط الدم الشرياني الرئوي. أظهر الضغط الانقباضي في الشريان الرئوي ارتباطا معنويا موجبا مع كل من معدل تدفق الناسور (r= 0.295, p= 0.038) و نسبة E/A (r= 0.368, p= 0.008)، في حين كان له علاقة معنوية سلبية مع قذفة البطين الايسر (r= -0.345, p= 0.014).

الاستنتاجات: ان كل من طول مدة الغسيل الكلوي ، واعتلال الكلية الناتج عن ارتفاع ضغط الدم كسبب للداء الكلوي بمراحله الأخيرة ونسبة الهيموكلوبين ≥ 10 ملغم/ديسيلتر وقذفة البطين الايسر ≥ 55% هي عوامل مستقلة لتطور ارتفاع ضغط الدم الشرياني الرئوي في مرضى الداء الكلوي بمراحله الأخيرة الخاضعين للغسيل الكلوي. يرتبط الضغط الانقباضي في الشريان الرئوي ارتباطًا إيجابيًا بمعدل تدفق الناسور ونسبة E / A وارتباطا سلبيا مع قذفة البطين الايسر.

الكلمات المفتاحية: ارتفاع ضغط الدم الشرياني الرئوي، الداء الكلوي بمراحله الاخيرة ، الغسيل الكلوي

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