

# Prevalence and factors associated with Pulmonary Arterial Hypertension on maintenance Hemodialysis Patients in Kinshasa, the Democratic Republic of the Congo

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## Research article

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# Abstract

**Background:** Cardiovascular diseases in particular Pulmonary arterial hypertension (PAH) is associated a high morbi-mortality in chronic hemodialysis, but it's magnitude remains paradoxically unknown in sub-Saharan Africa. The aim of this study was to evaluate the prevalence of PAH and associated factors in chronic hemodialysis patients.

**Patients and method:** In a cross-sectional study, patients treated with HD for at least 6 months in 4 hemodialysis centers were examined. PAH was defined as estimated systolic pulmonary arterial pressure (sPAP)  $\geq 35$  mmHg using transthoracic Doppler echocardiography performed 24 hours after the session.

**Results:** A total of 85 HD patients were included. Their average age was  $54.6 \pm 14.3$  years. 57 patients (67.1%) were male. Mean duration of HD was  $13.3 \pm 11$  months. With reference to vascular access, 12 (14.1%), 29 (34.1%) and 44 (51.8%) patients had AVF, tunneled cuff and temporary catheter, respectively. The underlying cause of ESRD was diabetes in 30 patients (35.3%). The prevalence of PAH was 29.4%. In multivariate analysis, no secure healthcare funding (adjusted OR 5, 95% CI [1.24-8.27]), hyponatremia (adjusted OR 2, 95% CI [1.61-10.01]), arrhythmia (aOR 3, 95% CI [1.06 -5.85]), vascular access change (aOR 4, 95% CI [1.12-6.23]) and diastolic dysfunction (aOR 5, 95% CI [1.35-9.57]) were independently associated with PAH.

**Conclusion:** One third of hemodialysis patients exhibit PAH, which is associated with diastolic dysfunction and arrhythmia. Therefore, early detection and control of PAH and associated factors may help to tackle PAH associated morbidity and mortality in maintenance hemodialysis patients.

**Keywords:** Pulmonary hypertension, Hemodialysis, Systolic pulmonary arterial pressure, Cardiovascular disease.

## Introduction

Excessive cardiovascular mortality in patients with end-stage renal disease (ESRD) has been described in epidemiological and clinical studies. It accounts for about 50% of deaths in dialysis [1] of which about 29–30 % due to pulmonary hypertension [2]. Pulmonary arterial hypertension (PAH), defined as a rise in pulmonary arterial pressure (PAP) resulting from heart, lung or systemic disorders, is a common finding in patients on maintenance hemodialysis [3, 4] and an independent predictor of all-cause and cardiovascular mortality in maintenance hemodialysis patients [5 - 8]. The prevalence of PHT varies from 30 to 40%, as detected by Doppler echocardiography in patients on chronic hemodialysis (HD). Several studies have reported PAH-related mortality: "The Jackson Heart Study" on 27% of patients with PAH hospitalized for heart failure for an average of 6 years, had a death rate of 44% [9]. Once PAH was established, it is associated with high morbidity and mortality [10], in a large prospective study in patients with chronic renal failure who were not dependent on dialysis, PAH was associated with a risk of 15% death and 28% cardiovascular events while hemodialysis is recognized as a factor that promotes the occurrence of PAH [11].

The possible causes of PAH in haemodialysis patients are classified into three categories: 1) an increase in cardiac output caused by arteriovenous fistula, anemia or hypervolemia [12, 13]; 2) increased pulmonary vascular resistance caused by uremia-induced endothelial dysfunction, pulmonary embolism, calcification of the pulmonary artery or other comorbid diseases, including chronic obstructive pulmonary disease or connective tissue disease, and 3) elevation of pulmonary capillary pressure caused by systolic and diastolic heart failure [14]. Barak and Katz's hypothesized that microbubbles, which originate from the dialysis tubes or filter, may be trapped in the pulmonary circulation [15]. Early intervention to reduce pressure in the pulmonary artery can prevent the worsening of heart failure and death [16–19]. The purpose of this study was to evaluate the prevalence, associated characteristics and determinants of PAH in chronic hemodialysis patients.

## Patients And Methods

- Study design and participants

This present study was a cross-sectional one conducted from Mars 2016 to June 2019 and included ESRD patients  $\geq 18$  years old on maintenance HD for more than 6 months in 4 hemodialysis centers (University Hospital of Kinshasa, Ngaliema Medical center, Afia Medical Care, Medical Center of Kinshasa). ESRD was defined as irreversible and advanced loss of kidney function due to any etiology requiring long term RRT with HD. Patients on chronic hemodialysis with the following characteristics: chronic pulmonary diseases such as chronic obstructive pulmonary disease, pulmonary fibrosis, pregnant patients, pregnant women, chest wall, previous pulmonary embolism, collagen vascular disease, moderate or severe mitral or aortic valve disease and having obstructive sleep apnea syndrome were excluded from the study.

- Hemodialysis method

Patients had two or three sessions per week for 24 hours with anticoagulant (low molecular weight heparin or unfractionated heparin) at each session. Most patients were treated with synthetic dialyser membranes (high flux) and bicarbonate-based dialysis solution at a bicarbonate concentration of 32 mEq / L. Blood flow and dialysate flow rate were 200, respectively at 400 mL / min and 500–800 mL / min, with the generators 4008 and 5008s of Fresenius.

- Echocardiographic measurements

Transthoracic Doppler echocardiograms were performed by a single cardiologist using an ultrasound system with a 3.5 Hz cardiac lead (Vivid 7, GE, Massachusetts, USA) as a non-invasive method, in post dialysis, 24 hours after when patients were at optimal dry weight. PHT was defined as Systolic PAP equal to or greater than 35 mm Hg. Systolic right ventricular (or pulmonary artery) pressure was calculated using the modified Bernoulli equation: PAP = tricuspid systolic jet (TR) + 10–15 mm Hg (estimated right atrial pressure: 15 mm Hg in dilated right atrium and 10 mmHg in normal or slightly enlarged right atrium) [20]. The parameters of interest included the following: age, sex, comorbidities, medications,

etiology of kidney disease, age at time of ESRD, duration of hemodialysis therapy, and blood access location. Laboratory investigations encompassed: blood urea nitrogen (BUN), serum creatinine, natremia, kaliemia, uric acid, hemoglobin, hematocrit, iron, ferritin, ProBNP, Troponin, calcium, phosphorus, and parathyroid hormone level. The results of the predialysis blood samples at the time of echocardiographic study and the mean of the preceding three months values were evaluated.

## Statistical Analysis

Data was collected on an ad hoc file and analyzed using SPSS version 21. Normally distributed continuous variables are expressed mean  $\pm$  standard deviation; categorical variables as absolute (n) and relative (in percent) frequencies. Student t test was used to compare of two groups; Chi square test was used to compare frequencies of categorical variables. Logistic Regression analysis was used to assess the determinants of PAH. P value $<$ 0.05 was considered significant.

## Ethical issues

The research followed the tenets of the Declaration of Helsinki and informed consent was obtained. The study protocol was approved by the Clinical Research Ethics Committee of Kinshasa School of Public Health (number ESP/CE/013/2016).

## Results

A total of 85 patients were enrolled in the study; 57 patients (67.1%) were male. Mean duration of HD was  $13.3 \pm 11$  months. With reference to vascular access, 12 (14.1%), 29 (34.1%) and 44 (51.8%) patients had AVF, tunneled cuff and temporary catheter, respectively. The underlying cause of ESRD was diabetes in 30 patients (35.3%). Twenty five (29.4%) patients (mean age  $54.6 \pm 14.3$  years) had PAH with a mean value for SPAP of  $28.0 \pm 10$  mmHg.

PAH was present in 25 (29.4%) patients (Figure 1). Patients with PAH had hyponatremia ( $p = 0.010$ ), arrhythmia ( $p = 0.025$ ) and received less  $\beta$  blockers and antiplatelet drug ( $p = 0.038$ ) and Aspirin junior ( $p = 0.041$ ). They also did not have a secure funding ( $p = 0.017$ ) and experimented a vascular access change ( $p = 0.038$ ) (Table 1). Compared with patients without PAH, those with PAH had, in average, a significantly higher inter-dialytic weight gain ( $p = 0.014$ ), pulse pressure ( $p = 0.001$ ) (Table 2). These findings were similar for inferior vena cava diameter ( $p = 0.004$ ), filling pressures E / E' ( $p = 0.001$ ) and proportion of diastolic dysfunction ( $p = 0.007$ ) (Table 3).

In logistic multivariate analysis (Table 4), no secure healthcare funding (Adjusted OR 5, 95% CI [1.24–8.27]), hyponatremia (aOR 2, 95% CI [1.61–10.01]), arrhythmia (aOR 3, 95% CI [1.06–5.85]), vascular access change (aOR 4, 95% CI [1.12–6.23]) and diastolic dysfunction (aOR 5, 95% CI [1.35–9.57]) were independently associated with PAH.

## Discussion

The main findings of the present study are as follows. First, roughly 3 out of 10 patients on maintenance hemodialysis had PAH. Second, hyponatremia, arrhythmia, vascular access change, diastolic dysfunction and no secure healthcare funding emerged as the main factors associated with PAH.

One third of patients on maintenance hemodialysis exhibited PAH. This frequency is within the range of 27–57% reported by most studies [21, 22] and similar to that reported by Amin *et al.* in Egypt although echocardiography was performed within 4h after the end of dialysis [16]. However, it is lower than that of 58.6% and 60% found by Fabian in Italy [23] and Hayati *et al.* in Iran, respectively; the latter survey used an average PAP of 25 mmHg to define PAH [24]. Between studies disparities in PAH frequency could be explained, among others, by differences in PAH definition criteria and time of echocardiography perform relative to hemodialysis session. In this regard, the high PAH frequency reported by Hayati *et al.* could be explained by the use of an average PAP of 25 mmHg to define PAH [24]. Although we used the same PAH definition as Ramasubbu *et al.* in the US, systolic arterial pulmonary pressure greater than 35 mmHg, our prevalence was low compared to 47% because, may be, of differences in population studied and methodology applied [25]. The time of echocardiography realization and the importance of interdialytic weight gain (IWG) could also influence the prevalence of PAH. Similar to our study, echocardiography was performed within 24 h after the end of dialysis [18]. When pulmonary hypertension was defined as systolic pulmonary arterial pressure (sPAP) greater than 45 mmHg, the frequency of PAH was only 20% [25]. This may justify our high prevalence compared to 16% of Agarwal [6].

The independent determinants of pulmonary arterial hypertension in our study were: unsecured funding for dialysis, hyponatremia, arrhythmia, vascular access first change, and diastolic dysfunction. The positive association of unsecured funding with PAH is a reflection of irregular dialysis sessions with subsequent significant interdialytic weight gain through renal sodium and water retention [23]. Sodium and water retention and subsequent hemodilution could also explain hyponatremia and its association with PAH. The vascular access change, most often from catheter to more efficient vascular access such as arteriovenous fistula aggravates the pulmonary congestion through increased circulating blood volume. Indeed, PAP may also be increased by high cardiac output resulting from the arteriovenous access and/or concomitant renal anemia, as well as from fluid overload [26, 27]. Diastolic dysfunction in dialysis patients can be due to multiple factors. Left ventricle hypertrophy, mainly as a result of chronic hypertension, is common and strongly associated with impaired left ventricular diastolic function in patients on dialysis. Furthermore, a large number of dialysis patients have diabetes which, by itself, is associated with increased myocardial stiffness and diastolic dysfunction [28]. Diastolic dysfunction, with a rise in filling pressures resulting in post-capillary PAH, could also explain the observed association with PAH. Our finding of an association between diastolic dysfunction and PAH agrees with that reported by Abdelwhab [28, 29] and Agarawal [6]; the latter found an increased left atrium diameter. The finding of our study underscore the role of volumetric overload in the pathogenesis of PAH among maintenance hemodialysis patients. Other studies identified increased age [25], female [16], lower body mass index

[25], high cardiac output [13], lower hemoglobin [12], lower metabolites of nitric oxide [13], upper dialysis cycle [23], lower diastolic blood pressure [23] as additional factors associated with PAH.

In the present study patients with PAH had elevated E/E ratio and ProBNP level suggesting a chronic volume overload and subsequent diastolic dysfunction [9]. This chronic volume overload translated in increased inferior vena cava diameter at exhalation that could be the probable cause of an association between an elevated right atrial diameter and PAH. Chronic volume overload was more frequent in patients with suboptimal dialysis and subsequent increased interdialytic weight gain related to irregular dialysis sessions favored by unsecured funding. In fact, in previous studies, a decrease in PAP following short AV fistula compression [12], as well as fluid removal, has been reported [30]. The use of these drugs may therefore be associated with lower pulmonary hypertension (although we have not found a difference in LV mass index between groups). Except from abovementioned hemodynamic factors, biochemical factors could also contribute to PAH in maintenance hemodialysis patients. In this regard, an increase in circulating nitric oxide synthase inhibitors like asymmetric dimethyl arginine (ADMA) accumulate with subsequent poor availability of nitric oxide, oxidative stress, endothelial dysfunction and vasoconstriction that can worsen PAH. [13]. Increased stiffness of the pulmonary capillaries due to hyperparathyroidism and pulmonary vascular calcification is one possible explanation for the PAH [31]. As noted by several other researchers, we have not established an association between pulmonary arterial hypertension, calcium, phosphorus and parathyroid [21, 32].

## Study Limitations

The interpretation of the results of the present study should take into account of some limitations. First, the cross-sectional design of the study precludes the establishment of temporal relationship between the issues of interest. Second, the small sample size did not allow sufficient power to statistical tests to identify potential associations between variables of interest. Third, the unique measurement of parameters of interest could have over- or underestimated their true values. Fourth, computerized tomography was not performed to exclude other potential causes of PAH.

## Conclusion

PAH was a common clinical finding in the present case series of maintenance hemodialysis and associated with volume overload, cardiac dysfunction and arrhythmia, vascular access change towards arteriovenous fistulae, and lack of secured dialysis funding. Therefore, early detection and control of PAH and associated factors could help to reduce PAH associated morbidity and mortality in maintenance hemodialysis patients.

## List Of Abbreviations

CHF: Congestive Heart Failure, DM: Diabetes Mellitus, EF: Ejection Fraction, ESRD: End Stage Renal Disease GFR: Glomerular Filtration Rate, HD: Haemodialysis, HT: hypertension, IVC: Inferior Vena Cava,

IVS: Interventricular Septum, LV: Left Ventricle, LVEDD: Left Ventricle End Diastolic Diameter, LVESD: Left Ventricle End Systolic Diameter, LVH: Left Ventricular Hypertrophy, LVMI: Left Ventricular Mass index, LVPW: Left Ventricular Posterior Wall RUV: Residual urination volume, PAH: Pulmonary Arterial hypertension, sPAP: systolic Pulmonary Arterial Pression

## Declarations

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### *Contribution of the authors*

All authors of the manuscript participated in the conduct and preparation of the study. YE, FL and ES designed the study, acquired, analyzed and interpreted data, drafted and revised the manuscript. AL, AN, YN, J-RM, VM, JB, TM, YL, FK, AN, CI, EK, NN and ES analyzed, interpreted data and revised the manuscript. All authors read and approved the final manuscript

### *Conflicts of interest*

The authors did not declare any conflict of interest.

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## Tables

**Table 1: General characteristics of the population studied**

<b>Variables</b>	<b>PAH-</b> n=60	<b>PAH+</b> n=25	<b>P</b>
Male, n (%)	39(65.0)	18(72.0)	0.359
Age ≥60 years, n(%)	22(36.7)	13(52.0)	0.143
Unemployed, n(%)	10(16.7)	2(8.0)	0.600
Secure finding	36(60.0)	8(32.0)	<b>0.017</b>
HT, n(%)	54(90.0)	24(96.0)	0.332
DM, n(%)	24(40.0)	8(32.0)	0.330
Tabacco, n(%)	6(10.0)	4(16.0)	0.329
Alcohol, n(%)	14(23.3)	9(36.0)	0.176
Hypervolemia, n(%)	31(51.7)	11(44.0)	0.343
Hyponatremia	10(16.7)	11(44.0)	<b>0.010</b>
Hypocalcemia	40(66.7)	19(76.0)	0.280
Vascular access			0.248
Temporary catheter	34(56.7)	10(40.0)	
Permanent catheter	20(33.3)	9(36.0)	
Fistula	6(10.0)	6(24.0)	
Arrythmia	1(1.7)	4(16.0)	<b>0.025</b>
Vascular access change	29(48.3)	18(72.0)	<b>0.038</b>
Supplement Ca/VitD	31(51.7)	14(56.0)	0.451
Phosphorus chelator	6(10.0)	5(20.0)	0.183
Diuretic	30(50.0)	16(64.0)	0.173
EPO	43(71.7)	19(76.0)	0.451
ACEI	22(36.7)	14(56.0)	0.081
ARAI	12(20.0)	4(16.0)	0.461
β-blockers	9(15.0)	8(32.0)	<b>0.038</b>
Iron supplement	45(75.0)	22(88.0)	0.147
AAS junior	22(36.7)	15(60.0)	<b>0.041</b>
RUV			0.343

<500 ml	29(48.3)	14(56.0)
≥500 ml	31(51.7)	11(44.0)

**Table 2: Clinical, biological parameters**

<b>Variables</b>	<b>PAH-</b>	<b>PAH+</b>	<b>p</b>
Age (years)	52.0±16.5	54.6±14.3	0.482
KT/V hebdo	1.1±0.2	1.1±0.1	0.960
Dry weight, Kg	70.5±16.3	66.9±14.9	0.354
IGW max kg	2.8±1.2	3.4±1.5	<b>0.014</b>
SBP mmHg	153.0±18.1	164.0±18.2	<b>0.012</b>
DBP mmHg	85.6±15.5	81.9±14.8	0.311
PP mmHg	67.3±16.7	82.2±16.8	<b>0.000</b>
Duration in HD, month	14.4±12.2	18.5±10.2	0.264
BMI, kg/m <sup>2</sup>	25.5±5.4	24.6±4.6	0.503
Residual diuresis, mL	625.0 (241.0-1010.0)	774.0(370.0-1680.0)	0.711
Creatinine mg/dL	9.7±3.2	9.9±4.4	0.804
BUN mg/dL	139.2±51.9	147.3±56.6	0.525
Uric Acid, mg/dL	7.3±1.9	7.6±2.0	0.560
K <sup>+</sup> , méq/L	4.6±0.6	4.6±0.6	0.949
PxCa <sup>2</sup> mg <sup>2</sup> /dL <sup>2</sup>	42.8±12.9	44.5±16.0	0.607
Albumin g/L	36.3±5.9	36.1±5.4	0.876
Hb g/dL	9.0±1.6	8.8±1.0	0.509
Hct, %	27.2±5.3	26.7±3.6	0.672
Total Cholestérol	172.2±38.7	183.7±29.7	0.188
HDL	45.5±17.1	50.1±21.5	0.298
LDL	103.4±33.3	118.4±32.3	0.060
Triglycerida	114.2±40.6	118.6±37.2	0.642
Vit D	27,3(0,0-35,0)	26.9 (10.4-38.5)	0.132
PTH	379,3(197,6-509,0)	282.9 (16.1-1308.6)	0.886
Ferritina	339.5 (45.5-20000)	458.4 (212.4-2000.0)	0.817
S. Iron	12.8 (10.2-14.5)	13.1 (9.5-24.9)	0.705
Troponin	15.4 (1.5-48.0)	16.4 (7.6-34.0)	0.670
ProBNP	5907.0 (1509-22222)	12846.0 (4548-25000)	0.425

CRP mg/L

33.3 (0.6-76.0)

18.5 (1.9-112.0)

0.186

Data are expressed as mean  $\pm$  standard deviations (ET), absolute frequency (n) and relative frequency (in percent). Abbreviations: SBP, systolic blood pressure DBP, diastolic blood pressure; PP, pulse pressure; BMI, body mass index ; IWG: interdialytic weight gain; eGFR, estimated glomerular filtration rate MDRD, modification of diet in renal disease BUN, blood urea nitrogen PxCa, phosphate calcium product PTH, parathormon HDL-c, high-density lipoprotein-cholesterol LDL-c, low-density lipoprotein-cholesterol ProBNP, Pro brain natriuretic peptid

**Table 3. Echocardiography parameters**

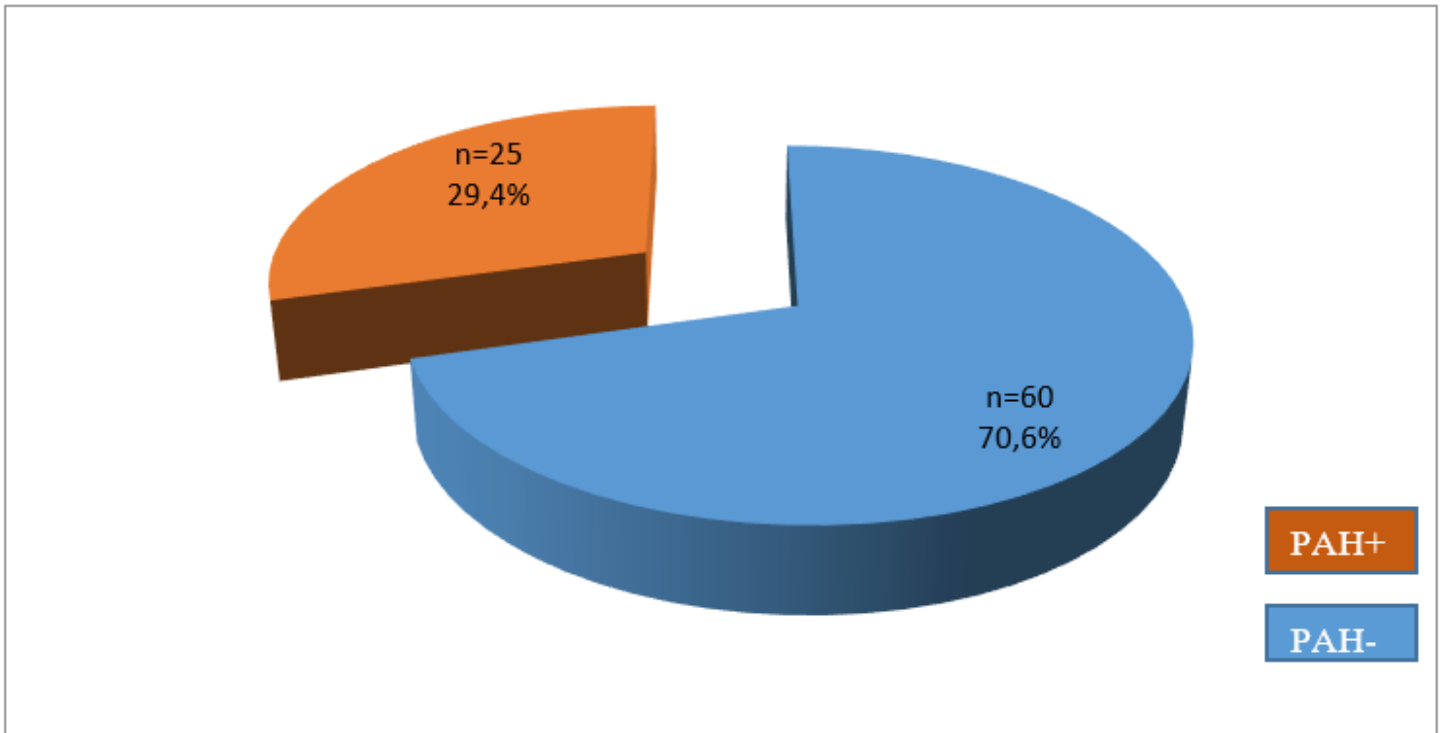
Variables	PAH-	PAH +	P
IVS	13.1 $\pm$ 2.4	13.6 $\pm$ 2.1	0.340
LVPW	11.8 $\pm$ 1.9	12,9 $\pm$ 2.8	<b>0.039</b>
LVEF	64.5 $\pm$ 8.6	64.3 $\pm$ 10.3	0.942
LVEDD	49.9 $\pm$ 6.1	50.4 $\pm$ 5.8	0.737
LVMi	369.4 $\pm$ 139.6	367.0 $\pm$ 170.9	0.948
ICV	13.6 $\pm$ 4.9	17.2 $\pm$ 5.6	<b>0.004</b>
E/A	1.0 $\pm$ 0.6	1.3 $\pm$ 0.9	0.118
E/E'	9.5 $\pm$ 4.1	14.0 $\pm$ 7.2	<b>0.001</b>
TD	217.2 $\pm$ 45.9	243.4 $\pm$ 104.9	0.276
Left Atrium volume	20.6 $\pm$ 5.3	22.9 $\pm$ 6.4	0.080
Right Atrium volume	14.8 $\pm$ 3.0	18.2 $\pm$ 4.5	<b>0.000</b>
RVEDD	20.1 $\pm$ 6.3	21.2 $\pm$ 7.8	0.505
Calcifications	19(31.7)	9(36.0)	0.442
Systolic dysfunction	17(28.3)	10(40.0)	0.211
Diastolic dysfunction	8(14.8)	6(35.3)	<b>0.007</b>
Pericardic effusion	9(15.0)	3(12.0)	0.506

IVS, interventricular septum; PwD, posterior wall in diastolic; LVEF, left ventricular ejection fraction; LVEDds: left ventricular end diastolic diameter; LVMI, left ventricular mass index; ICV, inferior cave veinous; DT, deceleration time; SPAP, systolic pressure arterial pulmonary

**Table 4. Univariate and multivariate factors associated with PAH in logistic regression analysis**

Variables	Univariate analysis		Multivariate analysis	
	P	Unadjusted OR (IC95%)	P	Adjusted OR (IC95%)
Healthcare funding				
Secure		1		1
No secure	<b>0.021</b>	3.19 (1.19-8.55)	<b>0.023</b>	4.76 (1.24-8.27)
Hyponatremia				
No		1		1
Yes	<b>0.010</b>	3.93 (1.39-11.13)	<b>0.020</b>	2.47 (1.61-10.01)
Arrhythmia				
No		1		1
Yes	<b>0.035</b>	4.24 (1.19-6.33)	<b>0.045</b>	2.65 (1.06-5.85)
Vascular access change				
No		1		1
Yes	<b>0.041</b>	2.75 (1.02-7.54)	<b>0.034</b>	4.26 (1.12-6.23)
β-blockers				
No		1		1
Yes	<b>0.018</b>	2.67 (1.89-8.01)	0.865	1.14 (0.25-5.30)
Diastolic dysfunction				
No		1		1
Yes	<b>0.007</b>	3.14 (2.90-10.91)	<b>0.011</b>	4.98 (1.35-9.57)

## Figures



**Figure 1**

Prevalence of PAH