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Peripheral venous pressure measurements to evaluate congestion in pulmonary arterial hypertension

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Abstract

Background: Right atrial pressure (RAP) is an important prognostic criterion in pulmonary hypertension. Elevated mean RAP measured by cardiac catheterization is an independent risk factor for mortality. Accurate bedside assessment of congestion in the management of patients with PH remains challenging. As a continuous conduit of circulating fluid, systemic congestion represented by high RAP may be reflected by peripheral venous pressure (PVP). We evaluated the reliability of PVP measurements for assessing congestion beyond conventional clinical assessments.

Methods: We performed conventional congestion assessments and PVP measurements in 138 patients undergoing right heart catheterization. PVP was measured via the 16-gauge peripheral venous access placed in the upper extremity.

Results: The mean RAP and PVP were 8.7 ± 4.2 mm Hg and 10.7 ± 4.3 mm Hg, respectively. PVP exhibited a strong linear correlation with RAP (Pearson $R = 0.839$; $P < .001$). PVP demonstrated significant discriminatory power for both RAP < 8 mm Hg (area under the curve: 0.91 [95% confidence interval: 0.86 to 0.96]; sensitivity: 72%; specificity: 94% cutoff: 8.5 mm Hg) and RAP ≥ 12 mm Hg (area under the curve: 0.92 [0.87 to 0.97]; sensitivity: 82%; specificity: 89% cutoff: 12.5 mm Hg).

Conclusion: PVP measured via peripheral venous access strongly correlates with invasively obtained RAP. PVP measurements may improve current bedside assessments of congestion.

Abbreviations: AUC = area under the curve, CVP = central venous pressure, IV = intravenous, PAH = pulmonary arterial hypertension, PH = pulmonary hypertension, PVP = peripheral venous pressure, RAP = right atrial pressure, RHC = right-sided heart catheterization, WHO = World Health Organization.

Keywords: cardiac catheterization, peripheral venous pressure, right atrial pressure, systemic congestion

1. Introduction

Pulmonary arterial hypertension (PAH) is a rare, progressive disease which is associated with persistent, abnormal increase in pulmonary artery pressure.[1] PAH progressively causes decreased pulmonary vascular compliance, increased pulmonary vascular resistance, leading to right ventricular heart failure, and ultimately death.[1] The diagnosis of PAH is a multistep process and often requires the skillful use of several tests. The diagnosis of PAH relies on systematic hemodynamic measurements of the right heart and pulmonary circulation which are obtained through right-sided heart catheterization (RHC). RHC is limited in the longitudinal evaluation of patients with PAH as it is an invasive method and may pose risk for potential complications.[1]

Right atrial pressure (RAP) is a valuable parameter in the hemodynamic evaluation of patients which provides noninvasive estimation of pulmonary artery pressures.[2] RAP enables us to estimate intravascular volume, which is critical for optimal management and patient care.[2] Elevated mean RAP measured by RHC has been revealed as a risk factor for poor survival in the Registry to Evaluate Early and Long-term PAH Disease Management and other previous studies.[3–6] Although invasive monitoring is the gold standard method for RAP assessment, various techniques can also be used to evaluate RAP noninvasively.[2] Several echocardiographic methods including indices

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The datasets generated during and/or analyzed during the current study are not publicly available, but are available from the corresponding author on reasonable request.

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obtained from the inferior vena cava, systemic and hepatic veins, tissue Doppler parameters, and right atrial dimensions may be used for evaluation of RAP.[2] As noninvasive assessment of RAP involves indirect measurements, multiple factors must be taken into consideration to provide the most accurate estimate of RAP.[2]

Measurement of RAP or its surrogate central venous pressure (CVP) via a catheter inserted into the central vein (such as the jugular vein, subclavian, or femoral vein) may provide complementary data to clinical examination in selected cases. However, inserting a central line requires a certain level of expertise, which may be associated with essential complications depending on both the catheter insertion procedure (such as pneumothorax, hemothorax) and the presence of a venous catheter as a vascular access (such as catheter and/or air embolization, venous thrombosis, cardiac arrhythmias and sepsis).[7]

The measurement of pressures through peripherally inserted venous cannula such as peripheral venous pressure (PVP) might be an alternative method to central line insertion.

Recently published studies have shown that PVP exhibits a strong correlation with RAP, CVP in patients with acute heart failure, heart failure with preserved left ventricular ejection fraction,[8,9] and Fontan circulation.[10]

A simple tool such as PVP measurement, which can indicate the absolute value and changes in RAP elicited by treatment without the need for central line insertion, can provide an estimate of intravascular volume, which is a critical component for optimal patient care and management in patients with PAH.

2. Materials and methods

2.1. Study design

This tertiary single-center prospective observational study was conducted between October 2021 and September 2023 to evaluate the diagnostic accuracy of PVP measurement versus invasively obtained hemodynamic parameters (such as mean RAP) in patients with PAH scheduled for RHC (Table 1). The clinical indication for RHC included confirmation of the diagnosis of newly diagnosed PAH patients or evaluation of treatment effectiveness in patients hospitalized with PAH. Of the 257 patients with a high probability of pulmonary hypertension (PH) detected by echocardiography, 234 patients performed RHC following detailed evaluation for PH (Fig. 1). A total of 192 patients diagnosed with PH by RHC were categorized into 2 groups (precapillary PH or postcapillary PH) according to the World Health Organization (WHO) PH hemodynamic classification (Fig. 1). They were then categorized into 5 groups (WHO group 1–5) according to the WHO clinical classification. Thirty-five of the patients were diagnosed with postcapillary PH (WHO group 2 or group 5) hemodynamically, and the remaining 157 patients were diagnosed with precapillary PH (Fig. 1). One hundred thirty-eight of the patients classified as precapillary PH were evaluated as PAH (WHO group 1 PH). A total of 54 patients with WHO group 2–5 PH were excluded from the study (Fig. 1).

A cohort of 138 consecutive patients who met WHO diagnostic criteria for PAH (group 1 PH) based on clinical evaluation, PVP measurements, and RHC were prospectively assessed (Fig. 1). Comprehensive testing was performed at baseline only. Group 1 patients with PAH included those with idiopathic PAH, PAH associated with collagen vascular disease, and congenital heart disease-related PAH (Eisenmenger's syndrome, PAH associated with systemic-to-pulmonary shunts, PAH after defect correction). Patients who were monitored in the intensive care unit, those under mechanical ventilation, those under continuous positive airway pressure, those requiring oxygen support with nasal cannula or mask on room air, those requiring parenteral pulmonary vasodilators, those requiring inotrope or vasopressor support, those hospitalized due to clinical deterioration, and those requiring parenteral diuretics were excluded from the study without RHC.

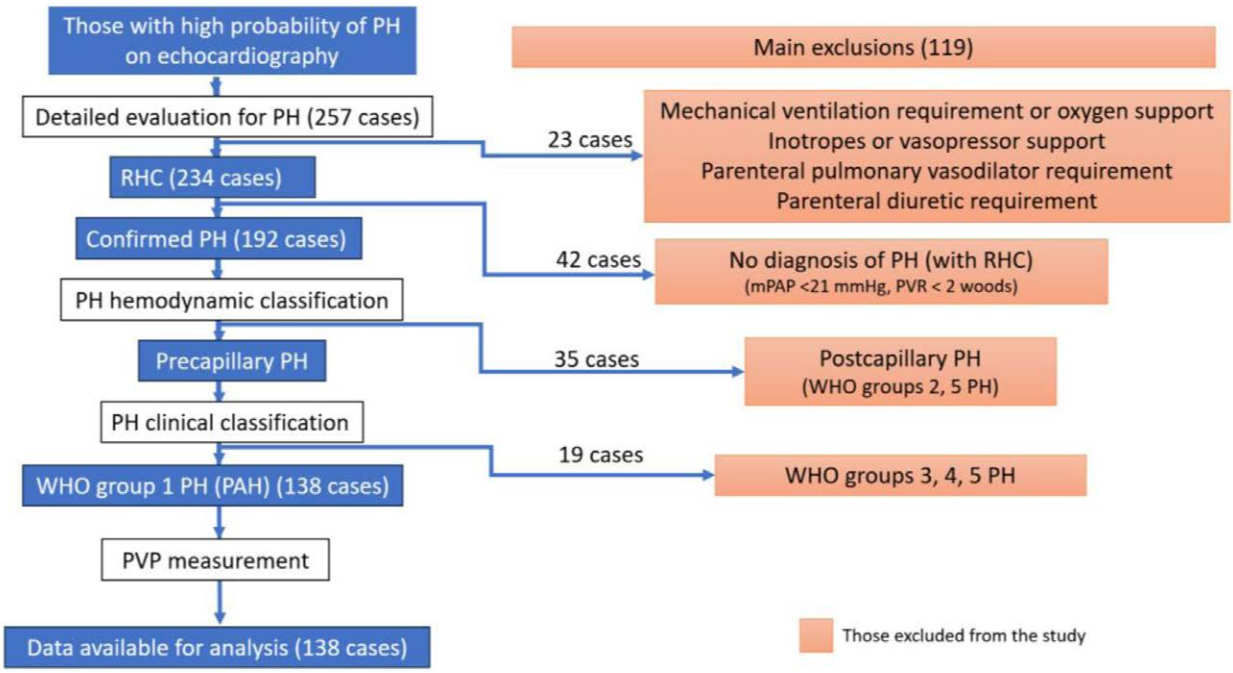

Figure 1. Flow chart of patient selection. Notes: Postcapillary PH was defined as mPAP > 20 mm Hg and PAWP > 15 mm Hg. Precapillary PH was defined as mPAP > 20 mm Hg, PAWP ≤ 15 mm Hg and PVR ≥ 2 woods. mPAP = mean pulmonary artery pressure, PAH = pulmonary arterial hypertension, PAWP = pulmonary arterial wedge pressure, PH = pulmonary hypertension, PVP = peripheral venous pressure, PVR = pulmonary vascular resistance, RHC = right-sided heart catheterization, WHO = World Health Organization.

The diagnosis of PAH was was made in a group of PH patients characterized hemodynamically by the presence of precapillary PH, which is defined by a mean pulmonary artery pressure > 20 mm Hg, a pulmonary artery wedge pressure ≤ 15 mm Hg and a pulmonary vascular resistance ≥ 2 Wood units in the absence of other causes of precapillary PH.

Prior to enrollment of the study patients, peripheral venous access was obtained using a 16-gauge intravenous (IV) in the upper extremity. Blood collection for routine laboratory tests, including brain-type natriuretic peptide, was obtained via this route. PVP was immediately measured by connecting the pressure line of the transducer (TruWave; Edwards Lifesciences, Irvine) directly to the peripheral IV while the pressure transducer remained zeroed at the phlebostatic axis. The arm of the patient is located in a parallel manner to maintain the peripheral IV at mid-chest level. To confirm the continuity between peripheral IV line and central venous system, an increase in the venous pressure waveform was expected after manually occluding the extremity proximal to the catheter. If the pressure waveform failed to increase appropriately, data were not obtained, and the patient was recorded as a technique failure. PVP measurements were performed by 2 experienced investigators who were blinded to the clinical data. Furthermore, the physicians who attended to the study were also unaware of the PVP results. Those who underwent mastectomy due to breast cancer, those with upper extremity lymphedema, those with upper extremity amputation, venous system vasculitis, those with venous thoracic outlet syndrome, those with a history of radiotherapy to the thoracic region, those at risk of superior vena cava syndrome, those with a history of hemodialysis were excluded from the study because they could affect the PVP measurement.

After right femoral central venous access was obtained, a 6-French pulmonary artery catheter was advanced into the pulmonary artery under fluoroscopic guidance. Before obtaining invasive measurements, baseline clinical and demographic data were recorded. Subsequently, right atrium, right ventricle, pulmonary artery, pulmonary artery wedge pressure were measured at end-expiration using standard pressure transducers after being zeroed at the phlebostatic axis. Fick method was used to measure cardiac output and index. RHC measurements were performed by 2 experienced investigators who were blinded to the clinical data. Furthermore, the physicians who attended to the study were also unaware of the RHC results.

Pretibial edema is graded according to the depth of the indentation and the time it takes for the skin to recover after pressure is applied. Grade 1 (+1) indicates mild pitting with a slight indentation that quickly disappears at a depth of approximately 2 mm. Grade 2 (+2) indicates moderate pitting with a slight indentation that resolves within a few seconds at a depth of approximately 4 mm. Grade 3 (+3) is characterized by deep pitting that persists for 10 to 20 seconds, approximately 6 mm deep.

The Institutional Review Board of our Hospital approved this study (study approval number: 2558). Written informed consent to participate in the study was obtained from all participants. The principles of the study are in accordance with the Declaration of Helsinki.

Table 1

Hemodynamic parameters of all patients in this study.

Pulmonary arterial wedge pressure, mm Hg	11.8 ± 4.1
Systolic pulmonary artery pressure, mm Hg	85.7 ± 31.0
Diastolic pulmonary artery pressure, mm Hg	33.7 ± 17.1
Mean pulmonary artery pressure, mm Hg	53.5 ± 21.6
Pulmonary vascular resistance, woods	8.97 ± 6.54
Systolic systemic blood pressure, mm Hg	130.5 ± 25.9
Diastolic systemic blood pressure, mm Hg	71.5 ± 15.8
Mix venous oxygen saturation, %	83.7 ± 10.1

2.2. Statistical analysis

All statistical tests were 2-sided, and P values < .05 were considered significant. Statistical analysis was performed using Stata (version 13, StataCorp LP, College Station).

Continuous variables are expressed as median (interquartile range) and categorical variables as n (%). The primary outcome of the study included the degree of correlation between PVP and CVP measurements. Pearson's correlation coefficients were calculated to assess this correlation. Any relationship between peripheral edema and mean PVP and RAP levels was also investigated.

3. Results

One hundred thirty-eight WHO group 1 PAH patients (42 [30.4%] men and 96 [69.6%] women), who were aged between 18 and 80 (mean ± SD, 43.0 ± 17.8) were included in this study. Of these, 44 (31.9%) had idiopathic PAH, 16 (11.6%) had connective tissue-related PAH, and 75 (54.3%) had congenital heart disease-related PAH (Table 2). Of the congenital heart disease-related PAH patients, 42 (56%) patients had Eisenmenger's syndrome, 10 (13.3%) patients had PAH associated with systemic-to-pulmonary shunts, and 23 (30.7%) patients had PAH after defect correction (Table 2).

A significant correlation was detected between peripheral edema and mean RAP (R = 522; P < .001). A significant correlation was also detected between peripheral edema and mean PVP (R = 58; P < .001; Fig. 2).

Receiver operating characteristic (ROC) curve analysis revealed that a mean PVP lower than 6.5 mm Hg was associated with peripheral edema <1+ in patients with PAH, with a

Table 2

Demographic, clinical, and laboratory data of all patients in this study.

Age, yr		43.0 ± 17.8
Sex, female, n (%)		96 (69.6%)
Heart rate, bpm		87.0 ± 15.4
Six-min walk distance, m		280.0 (IQR 40–600)
NYHA class	I, n (%)	6 (4.3)
	II, n (%)	16 (11.6)
	III, n (%)	80 (58)
	IV, n (%)	36 (26.1)
Peripheral edema	0, n (%)	30 (21.7)
	1+, n (%)	46 (33.3)
	2+, n (%)	39 (28.3)
	3+, n (%)	21 (15.2)
	4+, n (%)	2 (1.4)
PAH clinical classification	Idiopathic PAH	44 (31.9%)
	Connective tissue-related PAH	16 (11.6%)
	Congenital heart disease-related PAH	75 (54.3%)
	Eisenmenger's syndrome	42 (56%)
	PAH associated with systemic-to-pulmonary shunts	10 (13.3)
	PAH after defect correction	23 (30.7)
Medications	Endothelin receptor antagonists, n (%)	70 (51)
	Phosphodiesterase 5 inhibitors, n (%)	68 (49)
	Prostacyclin receptor agonist, n (%)	8 (6)
	Soluble guanylate cyclase stimulator, n (%)	12 (9)
	Prostacyclin analogs, n (%)	9 (7)
	Diuretics, n (%)	69 (50)
	Treatment-naive, n (%)	58 (42)
Laboratory data	Pro-brain natriuretic peptide, pg/L	399.8 (IQR 10–6287)
	High-sensitive troponin, pg/L	100 (IQR 1–949)

IQR = interquartile range, NYHA = New York heart association, PAH = pulmonary arterial hypertension.

sensitivity of 60% and a specificity of 89% (AUC = 0.75 [95% CI 0.64–0.87]; $P < .001$; Fig. 3A). In addition, ROC curve analysis showed that a mean RAP lower than 5.5 mm Hg was associated with peripheral edema <1+ in patients with PAH, with a sensitivity of 60% and a specificity of 82% (AUC = 0.78 [95% CI 0.68–0.87]; $P < .001$; Fig. 3B).

Furthermore, ROC curve analysis showed that a mean PVP > 10.5 mm Hg was associated with peripheral edema ≥3+ in patients with PAH, with a sensitivity of 73% and a specificity of 96% (AUC = 0.90 [95% CI 0.84–0.96]; $P < .001$; Fig. 3C). In addition, ROC curve analysis found that a mean RAP >9.5 mm Hg was associated with peripheral edema ≥3+ in patients with PAH, with a sensitivity of 73% and a specificity of 83% (AUC = 0.84 [95% CI 0.76–0.92]; $P < .001$; Fig. 3C).

A significant correlation was found between mean PVP and RAP ($r = 0.839$; $P < .001$; Fig. 4). ROC curve analysis revealed that a PVP lower than 8.5 mm Hg was associated with <8 mm Hg RAP in patients with PAH, with a sensitivity of 72 % and a specificity of 94% (area under the ROC curve = 0.91; 95% CI 0.86–0.96; $P < .001$; Fig. 5). In addition, a PVP higher than 12.5 mm Hg was associated with ≥12 mm Hg RAP in patients with PAH, with a sensitivity of 82% and a specificity of 89% (AUC curve = 0.92; 95% CI 0.87–0.97; $P < .001$; Fig. 5).

4. Discussion

This study aimed to investigate whether the average PVP measured through a catheter placed in the peripheral vein in patients with PAH was related to the average RAP and whether the change in 1 was the cause of the change in the other. This study confirms the strong correlation between PVP and invasively obtained RAP in patients with PAH. In the current study, there was a significant and strong correlation between average PVP and average RAP and the average PVP value was slightly higher than the average RAP value. Additionally, a moderately significant correlation was detected between peripheral edema and mean PVP and mean RAP. Moderate results were obtained in ROC curve analyses.

RHC is a key diagnostic tool for measuring intracardiac pressures in patients with cardiac dysfunction. Nevertheless, in certain circumstances, RHC may not be feasible outside of a catheterization laboratory or an intensive care unit. Using PVP for estimating CVP has been described primarily in the anesthesia[11–13] and critical care studies[14,15]; however, few data are available in patients with cardiovascular disorders.[14,15] We evaluated the ability of PVP to predict RAP in PAH patients, whose definitive diagnosis was made by RHC and who may require serial cardiac catheterization during follow-up.

Previous studies have shown reasonable correlation between CVP and PVP in noncardiac conditions including,

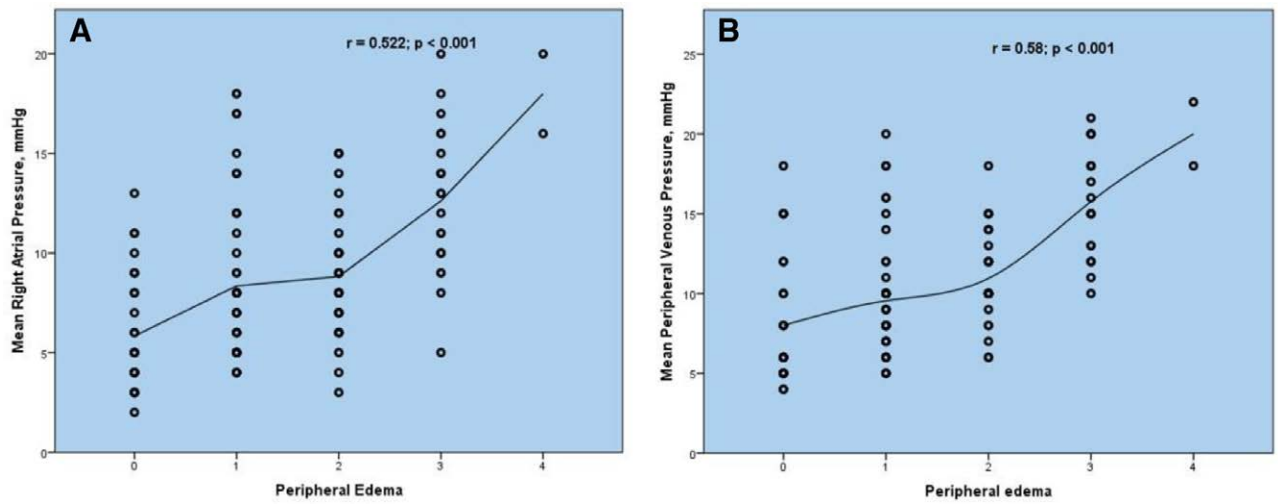

Figure 2. Scatter-dot plot showing the correlation between peripheral edema and mean right atrial pressure (A) and mean peripheral venous pressure (B) separately in patients with PAH. PAH = pulmonary arterial hypertension.

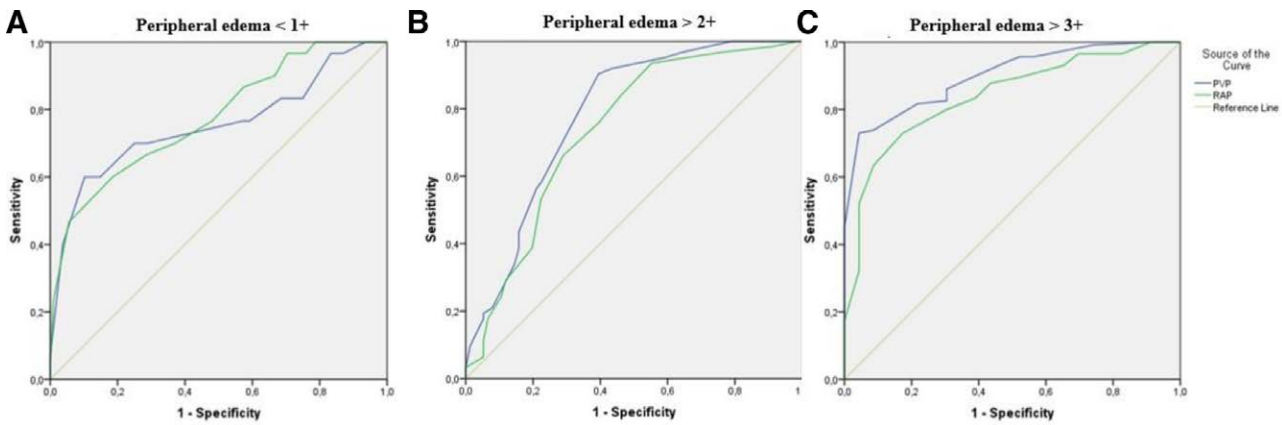

Figure 3. ROC analysis showing the sensitivity and specificity of mean PVP (blue line) and RAP (green line) with peripheral edema status ([A] peripheral edema < 1+, [B] peripheral edema ≥ 2+, and [C] peripheral edema ≥ 3+]) in patients with PAH. PAH = pulmonary arterial hypertension, PVP = peripheral venous pressure, RAP = right atrial pressure, ROC = receiver operating characteristic.

gastrointestinal, surgical, neurosurgical, and pediatric disorders.[16] The findings in children with cardiac disease are conflicting, with poor correlation seen in congenital heart disease but good correlation observed while on cardiopulmonary bypass and in those with Fontan circulation.[16] To our knowledge, the current study is the 1st to validate this relationship in patients with PAH. The results add to the generalizability of PVP measurement among a diverse population.

A recently published new study showed that PVP was superior to traditional congestion parameters in predicting RAP increases in patients undergoing RHC.[17] Furthermore, the independent prognostic value of PVP measured at discharge was demonstrated in patients hospitalized with heart failure.[18]

CVP is one of the clinical parameters used to guide fluid or diuretic administration in PAH patients. However, central line insertion can be associated with significant complications. Some

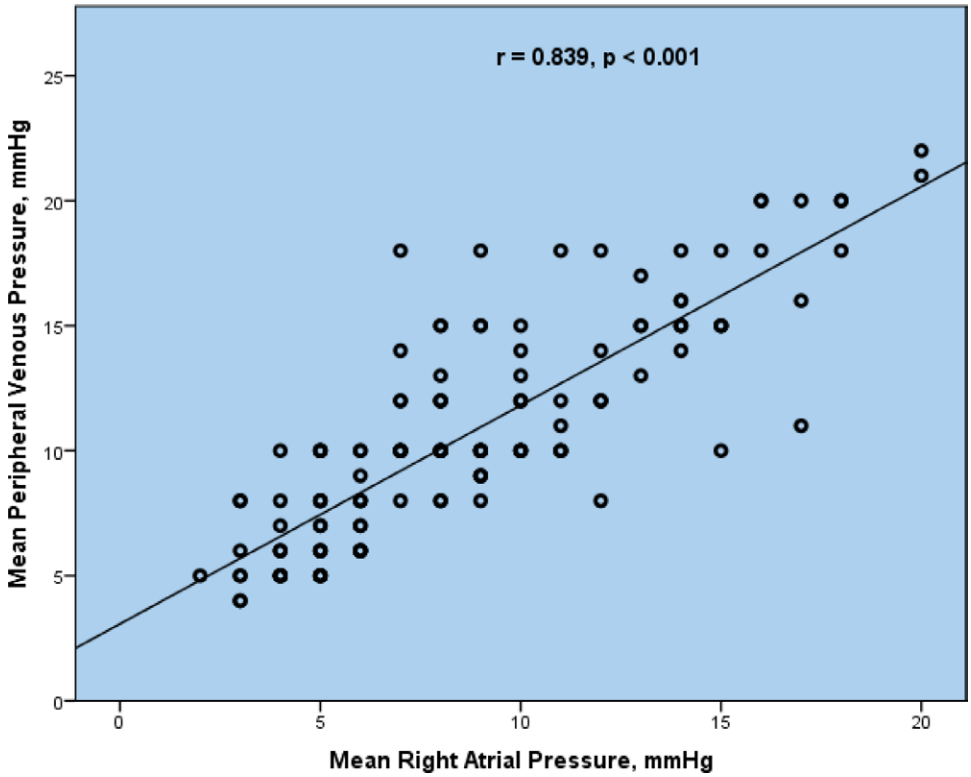

Figure 4. Scatter-dot plot showing the correlation between mean right atrial pressure and mean peripheral venous pressure.

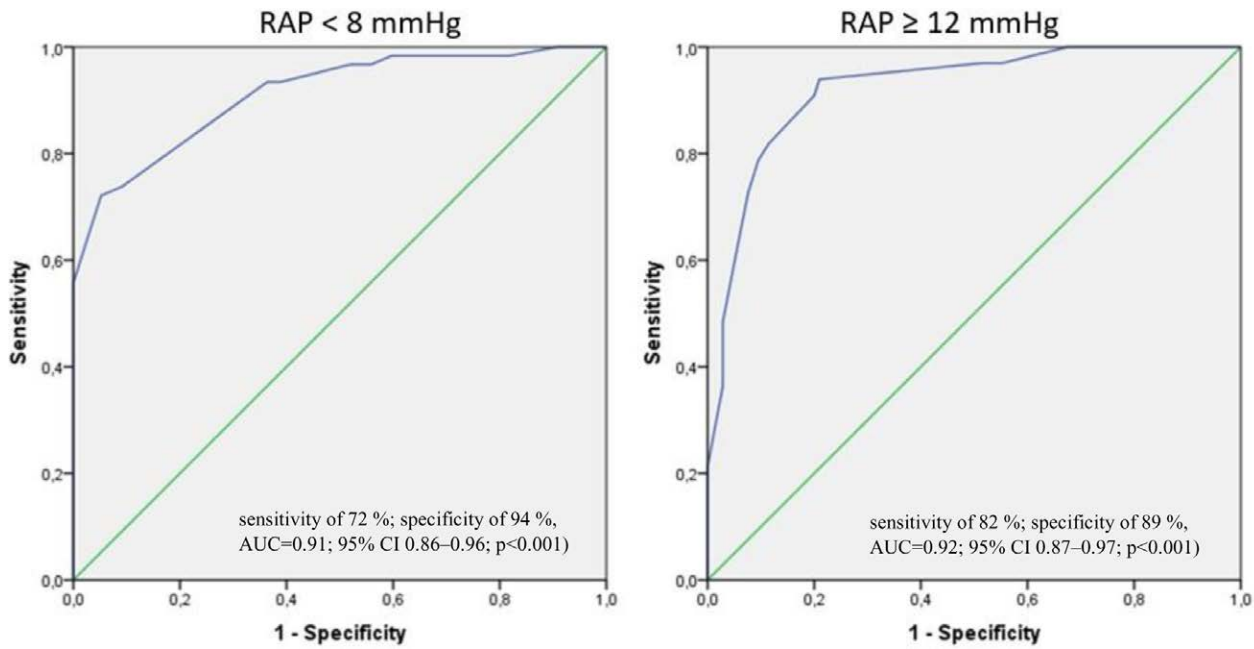

Figure 5. ROC analysis showing the sensitivity and specificity of mean RAP and mean PVP in patients with PAH. PAH = pulmonary arterial hypertension, PVP = peripheral venous pressure, RAP = right atrial pressure, ROC = receiver operating characteristic.

of these complications can be serious. Additionally, central line placement requires expertise and experienced staff. Therefore, estimation of CVP without using a central venous catheter would be valuable in patients with PAH and in conditions where expert assistance for central line insertion is not always immediately available.

This study has significant clinical implications. PVP measurement can also assist practitioners who are less confident at assessing intracardiac pressures during physical examination. PVP may be particularly useful when jugular venous examination is limited due to body habitus or patient positioning. PVP can be used to evaluate clinical deterioration and volume status in PAH patients. Mean RAP, which has been shown to have prognostic value and can be used in follow-up, can also be used in situ without the need for RHC. For this, it needs to be supported by more comprehensive studies.

Restoration of clinical euvolemia is an essential aspect of managing patients with PAH, both to recover functional status and to improve short- and long-term outcomes. However, due to subclinical hemodynamic congestion as well as the potential difficulty in accurately assessing bedside volume status, many patients with PAH are discharged with elevated RAP. More accurate assessment of RAP in the setting of clinical worsening of PAH and at discharge may reduce readmission rates in the 1st few months after hospitalization. Although RHC is indicated for invasive hemodynamic assessment to guide management when there is uncertainty regarding volume status, this approach is not feasible in all patients due to the risks involved. For this reason, mean PVP may serve as a valuable clinical surrogate of mean RAP in patients with PAH and the event of clinical deterioration. Thus, it may eliminate the need for RHC for invasive hemodynamic evaluation in some patients.

5. Conclusion

The simple measurement of PVP provided accurate information on hemodynamic congestion in the management of patients with PAH. The use of this measurement to clinical practice may improve current evaluation of congestion in the management of patients with PAH.

Author contributions

Conceptualization: Mükremin Coşkun, Abdullah Yıldırım, Abdullah Eren Çetin, Emre Sezici, İbrahim Halil Kurt, Mert Evlice.
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