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Role of the coronary sinus in risk stratification and prognostic evaluation of idiopathic pulmonary arterial hypertension

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Abstract

Objective: The coronary sinus (CS) can reflect the pressure and volume load of the right heart chambers. Idiopathic pulmonary arterial hypertension (IPAH) is a progressive, life-threatening disease in which risk assessment plays a critical role. We aimed to evaluate (a) the correlation between CS diameter and risk assessment parameters in IPAH patients and (b) the utility of CS diameter in predicting mortality and hospitalization.

Methods: This retrospective study included 25 IPAH patients. All patients underwent echocardiographic and laboratory examinations, 6-minute walk test, and cardiopulmonary exercise test on the same day. The follow-up was 16.8 ± 10.1 months. The primary endpoint was mortality or hospitalization. The relationship between CS diameter, established risk parameters, and prognosis was analyzed.

Results: Six patients (24%) were hospitalized, and three patients (12%) died during the follow-up . The mean CS diameter was 9.9 ± 3.7 mm and showed a moderate positive correlation with age ($r = 0.560$, $p = 0.004$) and NT-proBNP levels ($r = 0.625$, $p = 0.001$); a weak positive correlation with functional class ($r = 0.483$, $p = 0.017$); and moderate negative correlations with 6-min walking distance ($r = –0.553$, $p = 0.005$) and peak oxygen uptake ($r = –0.506$, $p = 0.038$). Greater CS diameter was associated with older age, higher NT-proBNP levels, and worse functional class, while reduced exercise capacity and peak VO_2 were accompanied by increased CS diameter. A CS diameter > 9 mm predicted mortality and hospitalization with a sensitivity of 77.8% and specificity of 75.0% (area under the curve [AUC]: 0.788; 95% CI: 0.580–0.996; $p = 0.019$). The Kaplan–Meier curve showed that as CS diameter increased, mortality and hospitalization rates increased significantly.

Conclusion: The CS diameter is a simple, readily available, noninvasive echocardiographic parameter that may be a valuable adjunct to current risk assessment models in IPAH.

Keywords
Coronary sinus · Idiopathic pulmonary arterial hypertension · Risk parameters · Mortality · Hospitalization

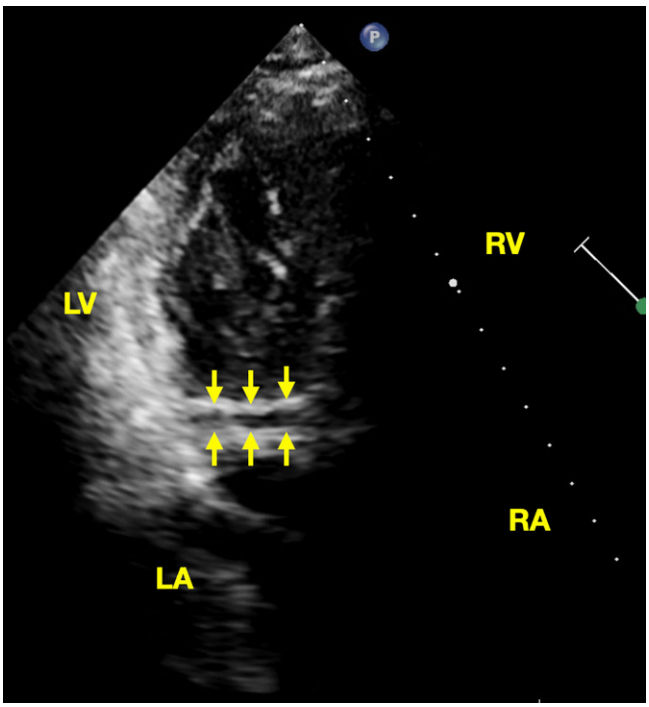


Fig. 1 ◀ Transthoracic echocardiography, modified apical four-chamber view. Arrows indicate the coronary sinus draining into the right atrium (RA). *LA* left atrium, *LV* left ventricle, *RV* right ventricle

Introduction

The coronary sinus (CS) is a tubular venous structure located in the posterior atrioventricular groove. It starts as a continuation of the great cardiac vein and drains into the right atrium (RA). The CS may dilate due to increased pressure on the right side of the heart [1]. A limited number of studies have investigated the diameter of the CS in patients with pulmonary hypertension (PH), but none of them focused on idiopathic pulmonary arterial hypertension (IPAH) and examined the relationship between CS diameter and clinical outcomes.

Idiopathic pulmonary arterial hypertension is a severe and life-threatening disease characterized by pathological changes in the pulmonary vasculature leading an elevation in pulmonary arterial pressure and pulmonary vascular resistance, right ventricular dysfunction, and sudden death [2]. It is a progressive disease in which risk stratification is the cornerstone of follow-up. Several well-defined clinical, noninvasive and invasive parameters that predict the prognosis of the disease have been described [3, 4]. Identifying potential risk stratification factors for IPAH is essential in clinical practice. Hence, in the present study, we aimed to investigate the correlation of CS diameter with the well-known risk assessment parameters in IPAH patients and to evaluate the utility of CS diameter in the prediction of mortality and hospitalization.

Material and methods

Study population

In total, 28 patients with IPAH who were admitted to our pulmonary hypertension outpatient clinic between October 2018 and November 2020 were included in this retrospective study. The inclusion criteria were as follows: age ≥ 18 years; diagnosis of IPAH confirmed by right heart catheterization (RHC); stable clinical status under medical treatment; availability of all medical records. Exclusion criteria were patients with PH other than IPAH diagnosis; patients with unstable clinical status or hospitalized patients; patients with persistent left superior vena cava (PLSVC); cases with inadequate image quality and missing data. After applying the exclusion criteria, a total of 25 patients with IPAH were included in the study. All patients underwent detailed echocardiographic and laboratory examinations, the 6-minute walking test (6 MWT), and cardiopulmonary exercise testing (CPET) on the same day. The study was conducted in accordance with the principles of the Helsinki Declaration and approved by the local ethics committee. Written informed consent was obtained from each patient.

Echocardiographic examination

All transthoracic echocardiographic (TTE) examinations were performed by two experienced cardiologists who were blinded to patient information and in accordance with the American Society of Echocardiography (ASE)/European Society of Cardiology (ESC) guidelines [5]. A Philips Epiq 7C (Philips Healthcare, Andover, MA, USA) device with an X5–1 transducer was used. To measure the CS, the apical window was chosen, as this was considered the best view for visualizing the CS [6]. From the apical four-chamber (A4C) view, when the transducer is slightly tilted posteriorly to the level of the dorsal wall of the heart, in the posterior aspect of the left atrioventricular groove, the CS and its entry into the RA can be seen (◘ **Fig. 1**). Maximum CS diameter was measured during ventricular systole. To exclude PLSVC, agitated saline contrast was injected into a peripheral vein of the left arm.

Systolic pulmonary artery pressure (sPAP) was calculated from the peak tricuspid regurgitation (TR) jet velocity, using the simplified Bernoulli equation and adding the estimated RA pressure (RAP). The RAP was estimated from the inferior vena cava (IVC) diameter and from respiratory changes. A normal IVC diameter (≤ 2.1 cm) and inspiratory collapse of > 50% suggests an estimated RAP of 3 mm Hg (range, 0–5 mm Hg); a normal IVC diameter without inspiratory collapse of > 50% and dilatation (> 2.1 cm) with inspiratory collapse (> 50%) suggests an estimated RAP of 8 mm Hg (range, 5–10 mm Hg); and dilatation (> 2.1 cm) without inspiratory collapse of > 50% suggests an estimated RAP of 15 mm Hg [7].

The RA area was measured in A4C at end-systole, on the frame just prior to tricuspid valve (TV) opening, by tracing the RA blood–tissue interface, excluding the area under the TV annulus [5]. The right ventricular (RV) area was manually traced along the RV endocardial border from the lateral TV annulus along the free wall to

the apex and back to the medial TV annulus, along the interventricular septum at end-diastole. Trabeculations, papillary muscles, and the moderator band were included in the cavity area [5]. The left ventricular (LV) area was calculated in a similar manner. Right ventricular function was evaluated by measuring tricuspid annular plane systolic excursion (TAPSE); TAPSE was acquired by placing an M-mode cursor through the TV annulus and measuring the amount of longitudinal motion of the annulus at peak systole [7]. Left ventricular ejection fraction (LVEF) was measured from the A4C view using the modified Simpson's biplane method [5]. The acquired images were reanalyzed independently by two cardiologists to obtain the inter- and intraobserver variabilities.

Laboratory examination

Venous blood samples were drawn from IPAH patients prior to the 6 MWT and CPET from an antecubital vein into EDTA Vacutainer tubes (Mediost BV, Doesburg, The Netherlands). To determine the level of N-terminal prohormone of brain natriuretic peptide (NT-proBNP), an electrochemiluminescence immunoassay (ProBNPElecsys, Roche Diagnostics GmbH, Mannheim, Germany) was used.

Six-minute walking test

Patients rested for at least 10 min before the 6 MWT, and the dose and timing of any medications the patient was receiving were recorded. Heart rate, blood pressure, and oxygen saturation were measured. The levels of tiredness and dyspnea were determined according to the Borg scale both before and after the test. The resting period, if any, and the reason for stopping the test and other symptoms were recorded. Subsequently, the 6MW distance (6MWD) was calculated [8].

Cardiopulmonary exercise testing

Patients underwent a treadmill exercise ergospirometry test based on a Bruce protocol (Ergometrics 900, Ergoline, Bitz, Germany). Breath-to-breath measurements of oxygen uptake (VO_2), carbon dioxide production (VCO_2), and ventilation (Ve) values were analyzed. Heart rate, blood pressure, and ventilation were recorded continuously during ergospirometry. Ventilatory flow, inspiratory and expiratory oxygen concentration differences, and expiratory carbon dioxide concentration were measured. From these variables, oxygen uptake, peak oxygen uptake, carbon dioxide delivery, respiratory exchange ratio (RER), and ventilatory equivalents for oxygen and carbon dioxide (Ve/VO_2, Ve/VCO_2) were calculated by the machine. Patients were encouraged to exercise to exhaustion or to a RER ≥ 1.0. VO_2 max was defined as the amount of oxygen consumed by the body at the peak of tolerable exercise. The anaerobic threshold (AT) was determined from the plot of carbon dioxide output (VCO_2) against oxygen uptake (VO_2), where the slope of this linear relation increased owing to a rise in VCO_2 (V-slope method). If no agreement was obtained, the AT was considered not identified. Ve/VO_2 and Ve/VCO_2 were defined as ventilator efficiency [9]. The Ve/VCO_2 slope was obtained by linear regression analysis of the relationship between Ve and VCO_2 during exercise. The CPET was safe for all patients without any adverse events during the test.

Study endpoint

The primary endpoint of the study was mortality or hospitalization due to hemodynamic instability. Hemodynamic instability was characterized by a marked increase in right ventricular filling pressures accompanied by a reduction in cardiac output and a consequent decrease in systemic arterial pressure and clinical signs of hypoperfusion. The patients were divided into two groups according to the presence of the endpoint. The two groups were compared in terms of demographics as well as clinical, echocardiographic, and laboratory features.

Statistical analysis

Data were analyzed using the Statistical Package for the Social Sciences, version 24.0 (SPSS Inc., Chicago, IL, USA). To determine whether the variables show a normal distribution, visual (histograms, probability curves) and analytical methods (Kolmogorov–Smirnov or Shapiro–Wilk) were applied. Numerical variables showing a normal distribution are expressed as mean ± standard deviation (SD); numerical variables not showing normal distribution are expressed as median (interquartile range) and categorical variables as percentage (%). Numerical variables were evaluated using Student *t* tests and the Mann–Whitney *U* test between the two groups. The chi-square or Fisher exact test were used to compare categorical variables. A receiver operating characteristic (ROC) curve was generated to investigate the prognostic accuracy of CS diameter for mortality and hospitalization. Event-free survival curves were constructed using the Kaplan–Meier method and compared using the log-rank test. The correlation between CS diameter among the other numerical variables was evaluated by Pearson or Spearman analysis. A value of $p < 0.05$ was considered to be statistically significant.

Results

A total of 25 IPAH patients (mean age: 47.5 ± 18.5 years; male: 7) were enrolled in this study. The mean duration between the first diagnosis and inclusion in the study was 33.2 ± 11.4 months. During a mean follow-up of 16.8 ± 10.1 months, six (24%) patients were hospitalized due to hemodynamic instability, and three patients (12%) died. The mean CS diameter of the study population was 9.9 ± 3.7 mm, median World Health Organization functional class (WHO FC) was 1.5 (1–2), median NT-proBNP was 822 pg/mL (181–1494), mean 6MWD was 389 ± 142 m, and mean peak oxygen uptake (VO_2), was 12.6 ± 4.8 ml/min/kg (**Table 1**).

Four of our patients had positive results on the vasoreactivity test and were therefore under calcium channel blocker therapy. Ten patients were under PAH-specific monotherapy, nine of whom were using endothelin receptor antagonists (ERA) and one was under phosphodiesterase 5 (PDE5) inhibitor treatment. Overall, 11 patients were under PAH-specific combination therapy, ten of whom were receiving ERA + PDE5 inhibitor and one was under ERA + PDE5 inhibitor + prostacyclin analogue treatment.

Table 1 Basal demographic, clinical, echocardiographic, and laboratory features of the study population.

	Total ($n = 25$)	Patients without adverse event ($n = 16$)	Patients with adverse event ($n = 9$)	p
Age	47.5 ± 18.5	41.4 ± 17.5	58.3 ± 15.7	*0.027*
Gender (male), *n* (%)	7 (28.0%)	5 (31.3%)	2 (22.2%)	1.0
Smoking, *n* (%)	1 (4.0%)	1 (6.3%)	0 (0.0%)	1.0
DM, *n* (%)	2 (8.0%)	0 (0.0%)	2 (22.2%)	0.120
HTN, *n* (%)	11 (44.0%)	4 (25.0%)	7 (77.8%)	*0.017*
AF, *n* (%)	4 (16.0%)	1 (6.3%)	3 (33.3%)	0.116
6MWD (m)	389 ± 142	418 ± 112	330 ± 184	0.209
NT-proBNP (pg/mL)	822 (181–1494)	420 (139–1121)	1494 (887–2464)	*0.012*
Peak VO_2 (mL/min/kg)	12.6 ± 4.8	13.6 ± 4.6	11.3 ± 5.1	0.660
LVEF (%)	58.5 ± 4.9	59.4 ± 5.2	57.1 ± 4.5	0.321
TAPSE (mm)	19.5 ± 5.2	20.5 ± 5.2	17.8 ± 5.0	0.154
RA area (cm^2)	22.5 (18.3–27.8)	20.5 (17.3–24.5)	27.5 (26.3–29.5)	*0.032*
RVED area (cm^2)	26.3 (20.0–28.9)	23.7 (20.2–31.6)	26.8 (17.5–28.9)	0.947
LVED area (cm^2)	22.6 (18.2–28.0)	23.4 (17.0–29.5)	22.0 (18.2–25.5)	0.462
sPAP (mm Hg)	69.0 (53.0–80.0)	69.0 (50.0–80.0)	71.0 (61.3–102.5)	0.401
Coronary sinus (mm)	9.9 ± 3.7	8.5 ± 2.4	12.4 ± 4.2	*0.018*

Data are presented as percentage, mean ± standard deviation, or median (interquartile range)
AF atrial fibrillation, *DM* diabetes mellitus, *HTN* hypertension, *LVED* left ventricular end-diastolic, *LVEF* left ventricular ejection fraction, *NT-proBNP* N-terminal prohormone of brain natriuretic peptide, *RA* right atrium, *RVED* right ventricular end-diastolic, *sPAP* systolic pulmonary arterial pressure, *TAPSE* tricuspid annular plane systolic excursion, *VO_2* oxygen uptake, *6MWD* 6 min walking distance.

Table 2 Correlation of coronary sinus with well-known risk stratification and echocardiographic parameters

	Coronary sinus	
	r	p
WHO FC	0.483	*0.017*
6MWD	–0.553	*0.005*
NT-proBNP	0.625	*0.001*
Peak VO_2	–0.506	*0.038*
RA area	0.224	0.293

WHO FC World Health Organization Functional Class (other abbreviations are as in **Table 1**)

The study population was divided into two groups according to the presence of primary endpoint as patients with adverse events and patients without adverse events. Except for age and hypertension, there was no significant difference in demographics between the groups. Patients in the adverse event group were older (58.3 ± 15.7 vs. 41.4 ± 17.5, $p = 0.027$) and more of them had hypertension (7 vs. 4, $p = 0.017$). NT-proBNP levels (1494 [887–2464] vs. 420 [139–1121], $p = 0.012$), RA area (27.5 [26.3–29.5] vs. 20.5 [17.3–24.5], $p = 0.032$), and CS diameter (12.4 ± 4.2 vs. 8.5 ± 2.4, $p = 0.018$) were significantly higher in patients in the adverse event group. Other clinical, echocardiographic, and exercise parameters including well-known risk stratification parameters such as WHO FC, 6MWD, and peak VO_2 were not significantly different between the two groups (**Table 1**).

The correlation analysis revealed that CS diameter had a moderate positive correlation with age ($r = 0.560$, $p = 0.004$) and NT-proBNP levels ($r = 0.625$, $p = 0.001$) and a weak positive correlation with WHO FC ($r = 0.483$, $p = 0.017$) and moderate negative correlation with 6MWD ($r = –0.553$, $p = 0.005$) and peak VO_2 ($r = –0.506$ $p = 0.038$; **Table 2**, **Figs. 2**, **3** **and** **4**).

We preformed ROC analysis to detect the optimal cut-off value of CS diameter in predicting mortality and hospitalization (**Fig. 5**). A CS diameter of > 9 mm predicted mortality and hospitalization with a sensitivity of 77.8%, specificity of 75.0%, positive predictive value of 63.6%, negative predictive value of 85.7%, and accuracy of 76.0% (area under curve [AUC]: 0.788; 95% CI: 0.580–0.996; $p = 0.019$). A Kaplan–Meier cumulative survival curve was generated for mortality and hospitalization by using this cut-off value (**Fig. 6**). The Kaplan–Meier curve showed that as CS diameter increased, mortality and hospitalization rates increased significantly. Intra-observer and inter-observer differences for echocardiographic data were < 5%.

Discussion

In the present study, we evaluated the correlation between CS diameter and established risk assessment parameters in patients with IPAH, and investigated the prognostic utility of CS diameter in this population. Our findings contribute to the growing body of evidence supporting noninvasive markers that may assist in risk stratification and prognostic evaluation in IPAH.

Our study yielded four key findings:

1. A CS diameter > 9 mm was predictive of mortality and hospitalization, demonstrating a sensitivity of 77.8% and a specificity of 75.0%.
2. CS diameter showed a moderate positive correlation with age and NT-proBNP levels.
3. CS diameter exhibited a moderate negative correlation with 6MWD and peak VO_2, suggesting an association with impaired functional capacity.
4. A weak positive correlation was observed between CS diameter and WHO FC, likely influenced by the subjective nature of WHO FC assessment as reported by patients.

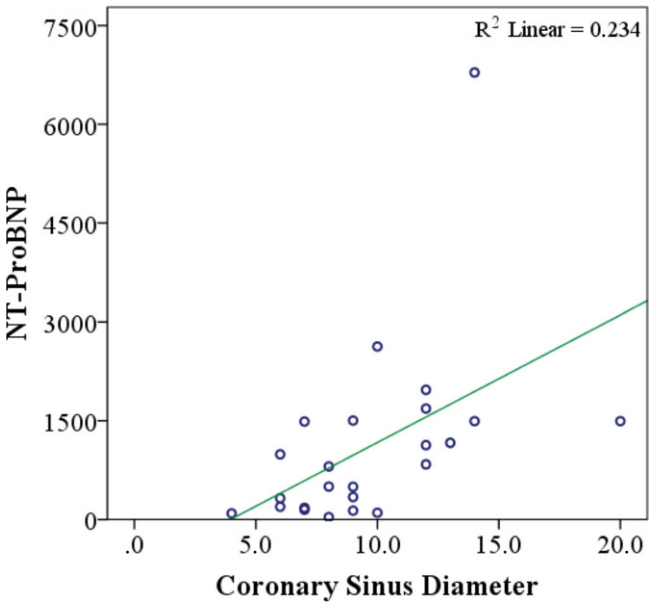


Fig. 2 ▲ Positive correlation between coronary sinus diameter and N-terminal prohormone of brain natriuretic peptide (NT-proBNP)

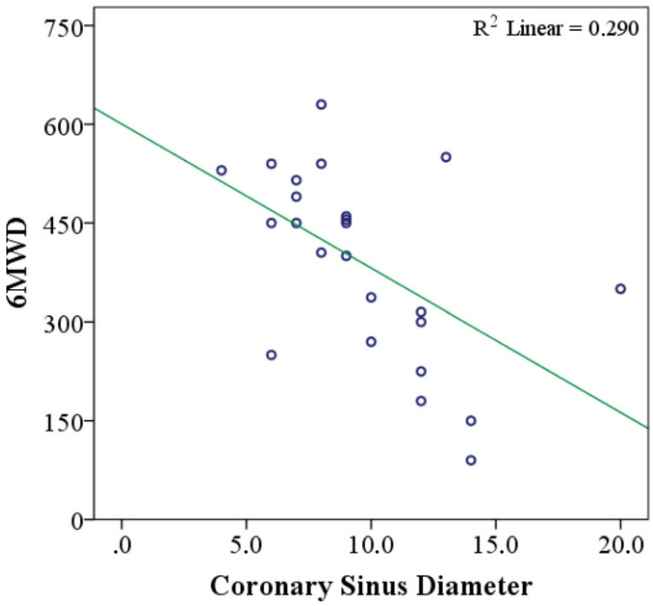


Fig. 3 ▲ Negative correlation between coronary sinus diameter and 6-min walking distance (6MWD)

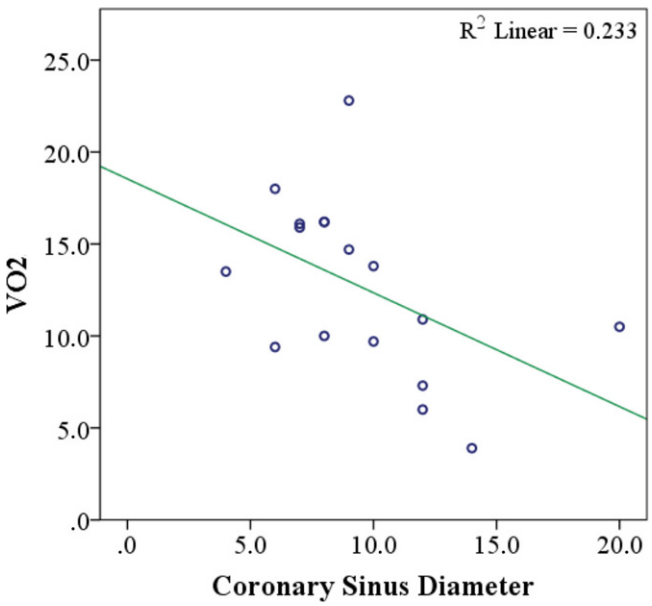


Fig. 4 ▲ Negative correlation between coronary sinus diameter and peak oxygen uptake (VO_2)

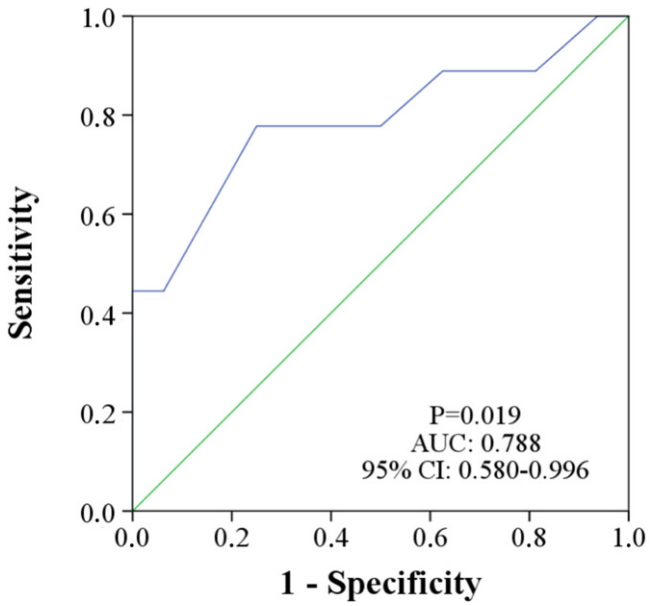


Fig. 5 ▲ Coronary sinus receiver operating characteristics curve for composite outcomes. *AUC* area under curve, *CI* confidence interval

Idiopathic pulmonary arterial hypertension is a life-threatening and progressive disease in which risk assessment plays a pivotal role in guiding management and follow-up strategies. The 2015 ESC and European Respiratory Society (ERS) PH guidelines emphasized the importance of frequent risk assessment [3]. These guidelines proposed a risk assessment framework incorporating clinical (right heart failure symptoms, symptom progression, syncope), functional (WHO FC), exercise capacity (6MWD, CPET), echocardiographic (right atrial area, pericardial effusion), and hemodynamic (RAP, cardiac index [CI], and mixed venous oxygen saturation [SvO_2]) parameters. According to the 2015 ESC/ERS guidelines, a three-strata risk stratification (low/intermediate/high risk) was used in the assessment of patients, both at the time of diagnosis and at each follow-up. The main goal was to achieve a low-risk profile for every patient [3].

Following the 2015 ESC/ERS PH guidelines, three major registries—the Swedish PAH Registry (SPAHR); the Comparative, Prospective Registry of Newly Initiated Therapies for Pulmonary Hypertension (COMPERA); and the French PH Registry—provided further validation of risk assessment models. Kylhammar et al. demonstrated that WHO FC, 6MWD, NT-

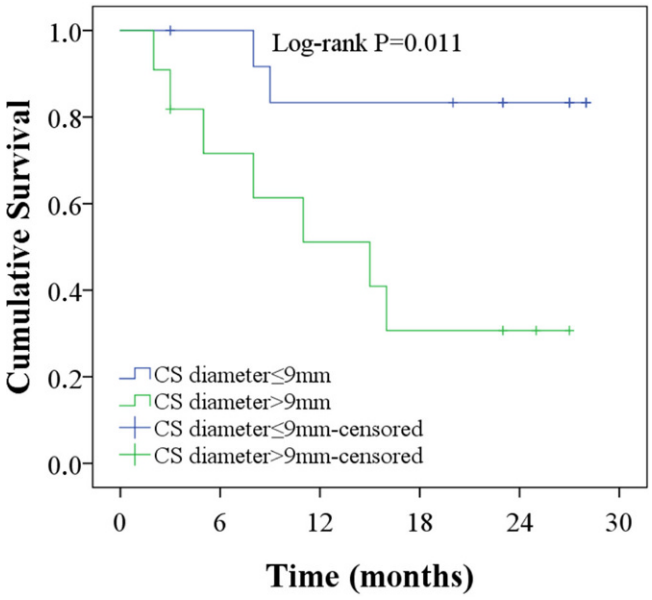


Fig. 6 ◀ Kaplan–Meier cumulative survival curve for composite outcomes

proBNP, right atrial area, mean RAP (mRAP), pericardial effusion, CI, and SvO_2 were significant prognostic indicators in the SPAHR cohort [10]. Similarly, Hoeper et al. assessed newly diagnosed PH patients in the COMPERA registry, identifying WHO FC, 6MWD, BNP or NT-proBNP, RAP, CI, and SvO_2 as essential risk markers [11]. Boucly et al. in the French PH Registry, proposed a four-parameter low-risk criteria model including WHO FC, 6MWD, RAP < 8 mm Hg, and CI ≥ 2.5 L/min/m² [12].

The recently updated 2022 ESC/ERS PH guidelines introduced modifications to the hemodynamic definition of PH and refined risk stratification approaches. The mean pulmonary artery pressure (mPAP) threshold for PH diagnosis was lowered from ≥ 25 to > 20 mm Hg, and for pre-capillary PH pulmonary vascular resistance (PVR) from ≥ 3 to > 2 Wood units, with PCWP ≤ 15 mm Hg introduced as a mandatory criterion. The revised guidelines also reinforced a more dynamic and individualized approach to risk assessment, incorporating multiple parameters at baseline and during follow-up.

In the updated 2022 ESC/ERS Guidelines for Pulmonary Hypertension, at the time of diagnosis, comprehensive risk assessment in pulmonary arterial hypertension with the three-strata model is similar to the 2015 guidelines, categorizing patients into low, intermediate, and high risk. However, a notable difference is the inclusion of cardiac magnetic resonance imaging (CMR) as a distinct component in risk evaluation. Cardiac magnetic resonance imaging is the gold standard imaging modality for evaluation of right ventricular size and function. Additionally, CMR offers the opportunity for tissue characterization, which shows the fibrosis of ventricle. Therefore, CMR is a very important tool for investigating the RV remodeling and reverse remodeling after specific treatments. Echocardiographic parameters have been further detailed, incorporating assessment of the TAPSE/sPAP ratio, which reveals the right ventricle-pulmonary arterial coupling. Moreover, in hemodynamic evaluation, stroke volume index was added as a parameter. However, with the new 2022 guideline, this comprehensive three-strata risk evaluation was recommended for the baseline examination only, not for follow-ups. For the follow-ups, a simplified four-strata risk assessment model (low risk, intermediate–low risk, intermediate–high risk, and high risk) has been introduced, utilizing WHO FC, 6MWD, and BNP or NT-proBNP levels. With this update, the importance of noninvasive monitoring in the clinical follow-up of patients has become increasingly emphasized [4]. Even though the follow-up risk assessment has changed, the main goal is the same: achieving a low-risk profile.

In our study, we included all noninvasive parameters highlighted in the 2015 and 2022 ESC/ERS PH guidelines, as well as those validated in major PH registries. Our results demonstrated a significant correlation between CS diameter and key risk assessment parameters, particularly NT-proBNP, 6MWD, and peak VO_2. The weaker correlation observed with WHO FC may be attributed to its subjective nature, which has been previously noted as a limitation in PH risk stratification. These findings suggest that CS diameter, a readily assessable echocardiographic parameter, may provide additional prognostic value in IPAH.

The relationship between several cardiovascular diseases and CS has been examined in detail, but no study investigated CS in IPAH patients [14–21].Cetin et al. reported that mean CS diameter increased significantly in patients with moderate to severe PH due to pulmonary disease, and that RA area and PASP were independent predictors of CS diameter in patients with PH [14]. However, in our study, we did not find any correlation between CS diameter and RA area. In 2008, Gunes et al. found a correlation between CS diameter and right heart chambers in PH patients regardless of etiology [15]. A study by Mahmoud et al. reported that CS dilation was related to RA size in PH patients [1]. Cetin et al. [14], Gunes et al. [15], and Isaacs et al. included patients with known or suspected PH in their studies [16], while Lee et al. [17] examined PH patients with mitral regurgitation; in all of these studies, the authors reported a correlation between CS diameter and PAP. In our study, by contrast, we did not find any correlation between CS and sPAP. The differences in the results can be explained by the inclusion of different study populations; we included only patients with IPAH. Another potential explanation is the relatively small sample size of our study, which may have limited the statistical power to detect subtle or modest associations, thereby contributing to the absence of a statistically significant correlation in this context. Although these studies evaluated CS in different PH groups, none of them investigated the long-term outcomes. To the best of our knowledge, our study is the first to evaluate CS in the risk assessment and prognosis of IPAH patients.

Interestingly, while parameters such as WHO FC and 6MWD are integral to current risk stratification models, they did

not significantly differ between patients in the adverse event group and those in the no adverse event group in our cohort. This may highlight the limitation of relying solely on functional metrics, which can be influenced by factors such as age, comorbidities, and patient effort. By contrast, CS diameter, as an anatomical marker, may offer a more objective and reproducible measure of chronic right heart burden.

Our study adds to the limited but growing body of literature emphasizing the utility of novel echocardiographic parameters in IPAH. Given the current ESC/ERS guideline emphasis on noninvasive monitoring, incorporating CS diameter into routine echocardiographic evaluation may enhance risk stratification, especially in resource-limited settings where advanced imaging or repeated right heart catheterizations are not feasible.

Limitations of the study

Some limitations of our study should be acknowledged. First, this was a single-center, retrospective study with a relatively small number of patients. The limited number may have reduced the statistical power to detect differences in clinical outcomes, particularly when mortality and hospitalization were analyzed as separate endpoints. Consequently, subtle but potentially meaningful associations between these outcomes and the studied parameters may not have been detected. Second, CS was measured as one dimension with two-dimensional transthoracic echocardiography. Three-dimensional echocardiography may enable the evaluation of CS in two orthogonal directions and in area, thereby increasing the power of the measurements. As an important methodological consideration in echocardiographic studies, inter- and intra-observer variability may influence measurement consistency and potentially affect the reproducibility of the results. Larger, multicenter, prospective studies are warranted to validate these findings and to explore the integration of CS diameter into future risk stratification algorithms.

Conclusion

The diameter of the coronary sinus is a simple, readily available, noninvasive echocardiographic parameter that may serve as a valuable adjunct to current risk assessment models.

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Data Availability. The underlying data are stored in a secure repository and may be made available to qualified researchers upon reasonable request. Such requests should be directed to the corresponding author and must comply with institutional and ethical guidelines.

Declarations

Conflict of interest. B. Uygur, K. Memiç Sancar, Ü. Bulut, S. Kahraman, A. Ateşli Yazıcı, A.R. Demir, H. Zencirkıran Ağuş, G. Coşkun, M. Erturk and M. Yıldız declare that they have no competing interests.

The study was conducted in accordance with the principles of the Helsinki Declaration and approved by the local ethics committee. Written informed consent was obtained from each patient.

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Zusammenfassung

Rolle des Koronarsinus bei der Risikostratifizierung und Prognosebewertung der idiopathischen pulmonalarteriellen Hypertonie

Ziel: Der Koronarsinus (CS) kann die Druck- und Volumenbelastung der rechten Herzkammern widerspiegeln. Die idiopathische pulmonalarterielle Hypertonie (IPAH) ist eine fortschreitende, lebensbedrohliche Erkrankung, bei der die Risikobewertung eine entscheidende Rolle spielt. Ziel unserer Studie war es, den Zusammenhang zwischen dem CS-Durchmesser und Risikobewertungsparametern bei IPAH-Patienten sowie den Nutzen des CS-Durchmessers zur Vorhersage von Mortalität und Krankenhausaufenthalten zu untersuchen.
Methoden: Es wurden 25 IPAH-Patienten in diese retrospektive Studie eingeschlossen. Bei allen Patienten wurde am selben Tag eine Echokardiographie und Laboruntersuchungen, ein 6-Minuten-Gehtest und ein kardiopulmonaler Belastungstest durchgeführt. Die Nachbeobachtungszeit betrug 16,8 ± 10,1 Monate. Der primäre Endpunkt war Mortalität oder Hospitalisierung. Der Zusammenhang zwischen CS-Durchmesser, etablierten Risikoparametern und Prognose wurde analysiert.
Ergebnisse: Sechs Patienten (24 %) wurden hospitalisiert, und drei Patienten (12 %) verstarben während der Nachbeobachtungszeit. Der mittlere CS-Durchmesser betrug 9,9 ± 3,7 mm. Der CS-Durchmesser zeigte eine moderate positive Korrelation mit dem Alter (r = 0,560; $p = 0,004$) und den NT-proBNP-Werten (r = 0,625; $p = 0,001$); eine schwache positive Korrelation mit der Funktionsklasse (r = 0,483; $p = 0,017$); und moderate negative Korrelationen mit der 6-Minuten-Gehstrecke (r = –0,553; $p = 0,005$) und der maximalen Sauerstoffaufnahme (r = –0,506; $p = 0,038$). Ein größerer CS-Durchmesser war mit höherem Alter, höheren NT-proBNP-Werten und einer schlechteren Funktionsklasse verbunden, während eine verringerte körperliche Leistungsfähigkeit und ein höherer VO_2-Spitzenwert tendenziell mit einem größeren CS-Durchmesser einhergingen. Ein CS-Durchmesser > 9 mm sagte Mortalität und Hospitalisierung mit einer Sensitivität von 77,8 % und einer Spezifität von 75,0 % voraus (Fläche unter der Kurve [AUC]: 0,788; 95 %-KI: 0,580–0,996; $p = 0,019$). Die Kaplan-Meier-Kurve zeigte, dass mit zunehmendem CS-Durchmesser die Sterblichkeits- und Hospitalisierungsrate signifikant anstieg.
Schlussfolgerung: Der CS-Durchmesser ist ein einfacher, leicht verfügbarer, nichtinvasiver echokardiographischer Parameter, der eine wertvolle Ergänzung aktueller Risikobewertungsmodelle bei IPAH-Patienten darstellen kann.

Schlüsselwörter
Koronarsinus · Idiopathische pulmonalarterielle Hypertonie · Risikoparameter · Sterblichkeit · Krankenhausaufenthalt