



ORIGINAL ARTICLE

Relationship between severity of pulmonary hypertension and coronary sinus diameter



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KEYWORDS

Pulmonary hypertension;
Coronary sinus;
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Abstract

Introduction and Objective: We investigated the relationship between coronary sinus (CS) diameter and pulmonary artery systolic pressure (PASP) in patients with pulmonary hypertension (PH) and normal left ventricular systolic function.

Methods: A total of 155 participants referred for transthoracic echocardiography were included in the study. The study population consisted of 100 patients with chronic PH and 55 control subjects. Patients with PH were divided into two groups according to PASP: those with PASP 36–45 mmHg, the mild PH group (n=53); and those with PASP >45 mmHg, the moderate to severe PH group (n=47). CS diameter was measured from the posterior atrioventricular groove in apical 4-chamber view during ventricular systole according to the formula: mean CS=(proximal CS+mid CS+distal CS)/3.

Results: Mean CS diameter was significantly higher in the moderate to severe PH group than in the controls and in the mild PH group (1.12±0.2 cm vs. 0.82±0.1 cm and 0.87±0.1 cm, respectively; p<0.001). It was significantly correlated with right atrial (RA) area (r=0.674, p<0.001), RA pressure (r=0.458, p<0.001), PASP (r=0.562, p<0.001), inferior vena cava diameter (r=0.416, p<0.001), right ventricular E/A ratio (r=-0.290, p<0.001), and E/Em ratio (r=0.235, p=0.004). RA area (β=0.475, p<0.001) and PASP (β=0.360, p=0.002) were found to be independent predictors of CS diameter.

Conclusions: A dilated CS was associated with moderate to severe pulmonary hypertension, and RA area and PASP were independent predictors of CS diameter.

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PALAVRAS-CHAVE

Hipertensão pulmonar;
Seio coronário;
Área auricular direita;
Doppler tecidual onda de pulso

Relação entre a gravidade da hipertensão pulmonar e o diâmetro do seio coronário**Resumo**

Introdução e objetivos: Os autores analisaram a relação entre o diâmetro do seio coronário (SC) e a pressão sistólica arterial pulmonar (PSAP) em doentes com hipertensão pulmonar (HP) que apresentaram funções sistólicas ventriculares esquerdas normais.

Métodos: Um total de 155 participantes referidos para ecocardiografia transtorácica foram incluídos no estudo. A população do estudo é composta por 100 doentes com HP crónica e 55 indivíduos no grupo controlo. Os doentes com HP foram divididos em dois grupos de acordo com a PSAP. Os doentes que tiveram PSAP de 36 a 45 mmHg foram definidos como o grupo de HP ligeiro (n = 53) e aqueles com PSAP > 45 mmHg foram selecionados como o grupo de HP moderada-grave (n = 47). O diâmetro do SC foi medido desde o sulco auriculoventricular posterior na incidência apical quarto câmaras durante a sístole ventricular de acordo com a fórmula: media do SC = (SC proximal + SC médio + SC distal)/3.

Resultados: O diâmetro médio do SC foi significativamente superior no grupo da HP moderada-grave do que no grupo controlo e no grupo da HP suave (1,12±0,2 cm versus 0,82±0,1 cm e 0,87±0,1 cm; p < 0,001, respetivamente). Verificou-se uma correlação, estatisticamente significativa, com a área da aurícula direita (AD) (r = 0,674, p < 0,001), pressão da AD (r = 0,458, p < 0,001), PSAP (R = 0,562, p < 0,001), veia cava inferior (r = 0,416, p < 0,001), rácio ventricular direito E/A (r = -0,290, p < 0,001) e rácio E/Em (r = 0,235, p = 0,004). Área da AD (β = 0,475, p < 0,001) e PSAP (β = 0,360, p = 0,002) foram encontradas como fatores preditores independentes do diâmetro do SC.

Conclusões: O SC dilatado foi associado à hipertensão pulmonar moderada a grave e a área da AD e da PSAP foram fatores preditores independentes do diâmetro do SC.

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Introduction

Pulmonary hypertension (PH) is routinely diagnosed in cardiology and pulmonary clinics. Chronic PH causes several adaptive changes in the right heart chambers due to pressure overload. Myocardial hypertrophy is the initial adaptive response, followed by progressive contractile dysfunction.¹ Chamber dilatation ensues to allow compensatory preload and maintain stroke volume despite reduced fractional shortening. As contractile weakening progresses, clinical evidence of right ventricular (RV) failure occurs, characterized by rising filling pressures, diastolic dysfunction, and diminishing cardiac output.² As a part of these processes, the inferior vena cava (IVC), which opens into the right atrium (RA), adapts to these changes, which results in reduced collapse with inspiration and dilatation due to pressure overload.

The coronary sinus (CS) is a venous structure that drains myocardial blood primarily from the left ventricle (LV) into the RA, which also receives blood from the IVC. CS diameter has been correlated significantly with pulmonary artery systolic pressure (PASP) in patients with PH resulting from chronic lung disease and LV dysfunction.³ In patients with chronic heart failure (CHF), a dilated CS is possibly a part of the process of cardiac remodeling and may provide useful information to predict the severity of CHF and poor functional class.⁴ Thus, it is unclear whether the underlying cause of CS dilatation is secondary to PH or a part of cardiac remodeling in patients with CHF.

In our echocardiographic study, we aimed to investigate the relationship between CS diameter and PASP, and to identify predictors of dilated CS in patients with PH.

Methods**Study patients**

This was a cross-sectional clinical study that included 155 participants in stable sinus rhythm prospectively referred for transthoracic echocardiography (TTE). The study population consisted of 100 patients with chronic PH and 55 control subjects. The study was conducted according to the guidelines of the Declaration of Helsinki and was approved by the local Ethics Committee, and informed consent was obtained from all patients.

Patients with a possible diagnosis of PH resulting from thrombotic and/or embolic disease, left ventricular (LV) systolic dysfunction, LV wall motion abnormalities and/or dilatation, more than mild LV diastolic dysfunction,⁵ more than mild aortic and mitral regurgitation, or any grade of valvular stenosis, severe tricuspid regurgitation, renal dysfunction (serum creatinine >1.5 mg/dl), cirrhotic liver disease, or inadequate imaging of the coronary sinus (CS) and/or IVC were excluded from the study. The subjects were divided into three groups according to PASP: patients with PASP ≤35 mmHg (control group, n=55), those with PASP 36–45 mmHg (mild PH group, n=53) and those with PASP

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