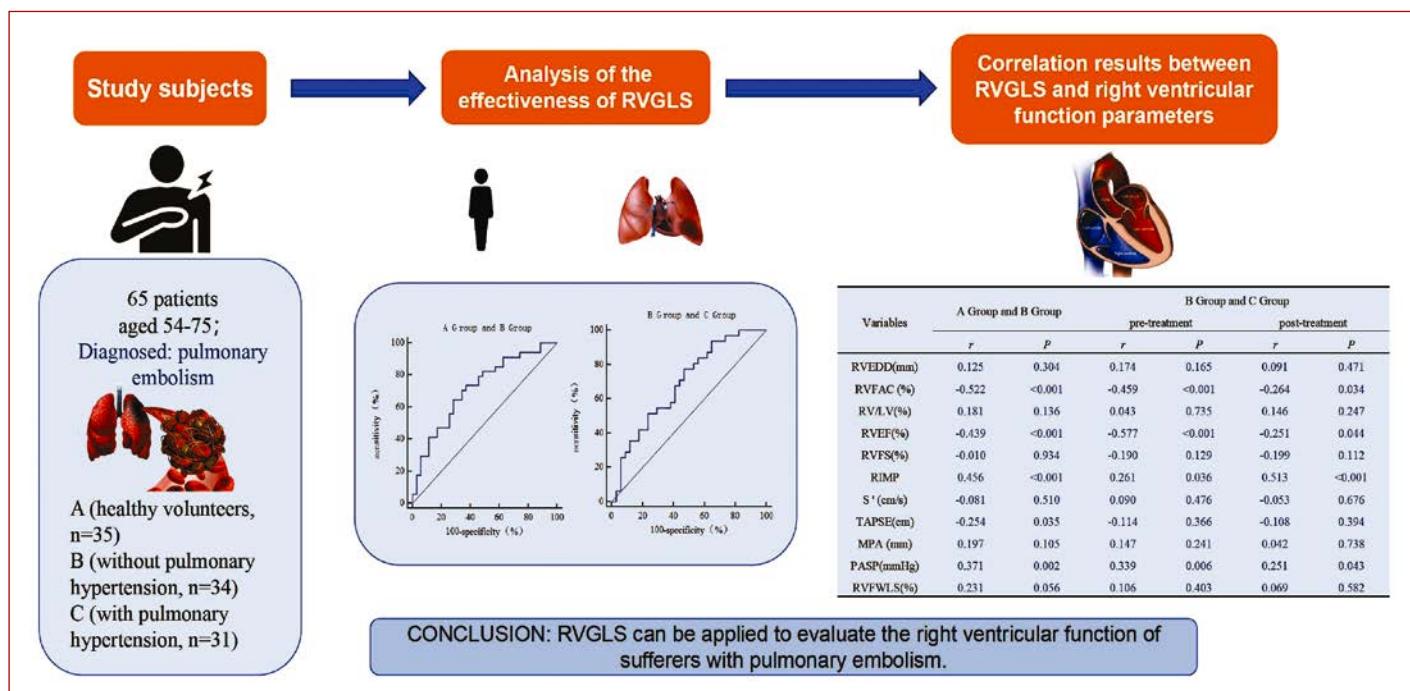


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RIGHT VENTRICULAR MYOCARDIAL GLOBAL LONGITUDINAL STRAIN ASSESSMENT OF RIGHT VENTRICULAR FUNCTION IN PATIENTS WITH PULMONARY EMBOLISM

Objective	To explore the clinical application value of right ventricular (RV) myocardial global longitudinal strain (RVGLS) in assessing changes in RV function in patients with pulmonary embolism.
Material and methods	Patients with pulmonary embolism who were treated successfully in our hospital from January 2022 to December 2023 were enrolled in this study. Included were 34 pulmonary embolism patients without pulmonary hypertension (Group B), 31 with pulmonary hypertension (Group C), and 35 healthy volunteers, matched by gender and age (Group A). Clinical data and RV function-related variables of these groups were compared.
Results	Compared with pre-treatment values of Group A, the following variables of Groups B and C had higher pre-treatment values ($p<0.05$): RV end-diastolic diameter (RVEDD), RV to left ventricular diameter ratio (RV/LV), RV work index (RIMP), main pulmonary artery diameter (MPA), pulmonary artery systolic pressure (PASP), RVGLS, RV free wall longitudinal strain (RVFWLS). The following variables had lower values ($p<0.05$): RV area change fraction (RVFAC), RV ejection fraction (RVEF), RV short-axis shortening rate (RVFS), tricuspid annular peak systolic velocity (S'), tricuspid annular systolic excursion (TAPSE). After therapy, significant differences were observed in the aforementioned indicators between Group C (with pulmonary hypertension) and Group A (healthy controls), with Group C showing persistently elevated RVEDD, RV/LV ratio, RIMP, MPA, PASP, RVGLS, and RVFWLS, alongside reduced RVFAC, RVEF, RVFS, S', and TAPSE compared to Group A (all $p<0.05$). Compared to pre-treatment values in Group B (without pulmonary hypertension), pre-treatment Group C demonstrated significantly higher RVEDD, RV/LV ratio, RIMP, MPA, PASP, RVGLS, and RVFWLS, and significantly lower RVFAC, RVEF, RVFS, S', and TAPSE (all $p<0.05$). Post-treatment comparisons between Groups B and C revealed that these differences remained significant (all $p<0.05$). ROC curve analysis revealed that $RVGLS > 20.59\%$ is the best cutoff value for predicting the occurrence of pulmonary embolism, and $RVGLS > -17.42\%$ is the best cutoff value for predicting the occurrence of pulmonary hypertension in patients with pulmonary embolism. The results of multivariable logistic regression model analysis showed that $RVGLS > -20.59\%$ is independently related to the occurrence of pulmonary embolism, and $RVGLS > -17.42\%$ is independently related to pulmonary embolism complicated by pulmonary hypertension ($p<0.05$). In Groups A and B, RVGLS was negatively correlated with RVFAC, RVEF, and TAPSE ($p<0.05$ for all) and positively correlated with RIMP and PASP ($p<0.05$ for all). In Groups B and C, RVGLS was negatively correlated with RVFAC and RVEF in patients with pulmonary embolism before and after treatment (for all <0.05) and positively correlated with RIMP and PASP ($p<0.05$ for all).
Conclusion	RVGLS can be applied to evaluate the RV function of patients with pulmonary embolism. $RVGLS > -20.59\%$ is independently related to pulmonary embolism, and there is a significant correlation between RVGLS and RVFAC, RVEF, RIMP, and PASP in patients with pulmonary embolism before and after treatment.
Keywords	Right ventricular longitudinal strain; pulmonary embolism; right ventricular function; pulmonary hypertension
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Central illustration. Right Ventricular Myocardial Global Longitudinal Strain Assessment of Right Ventricular Function in Patients with Pulmonary Embolism



RVEDD: Right ventricular end-diastolic diameter (mm); RVFAC: Right ventricular fractional area change (%); RV/LV: Right ventricular to left ventricular diameter ratio; RVEF: Right ventricular ejection fraction (%); RVFS: Right ventricular short-axis shortening rate (%); RIMP: Right ventricular index of myocardial performance; S': Tricuspid annular peak systolic velocity (cm/s); TAPSE: Tricuspid annular plane systolic excursion (cm); MPA: Main pulmonary artery diameter (mm); PASP: Pulmonary artery systolic pressure (mmHg); RVFWLS: Right ventricular free wall longitudinal strain (%).

Introduction

Pulmonary embolism is a common disease in which emboli in the veins or right heart system break off and block the pulmonary artery or pulmonary artery branches, causing pulmonary circulation disorders. The main clinical manifestations of pulmonary embolism are hypoxia, tachypnea, and tachycardia [1, 2]. Patients with pulmonary embolism can be divided into acute and chronic groups. The main pathological changes of acute pulmonary embolism are pulmonary obstruction and reduction of the pulmonary vascular bed, as well as increased pulmonary circulatory resistance and right heart load [3].

If the patient has a pulmonary embolism for a long time or the condition recurs repeatedly, it progresses to chronic pulmonary embolism, with specific clinical manifestations such as exertional dyspnea, fatigue, chest pain, syncope, cardiopulmonary insufficiency, and right heart failure, including cardiopulmonary insufficiency and right heart failure. In severe cases, pulmonary embolism can be life-threatening [4, 5]. The incidence and mortality of pulmonary embolism are high [6].

Pulmonary hypertension after pulmonary embolism is life-threatening and is an important cause of early death [7]. The clinical manifestations of pulmonary hypertension are a syndrome of dyspnea, reduced exercise tolerance, and a rapid increase in pulmonary artery systolic pressure. Pulmonary hypertension is the main complication of pulmo-

nary embolism and can easily lead to acute right heart failure or death [8, 9]. Studies have shown that patients with pulmonary embolism and pulmonary hypertension have severe right ventricular (RV) dysfunction and poor prognosis [10]. Therefore, it is particularly important to diagnose patients with pulmonary embolism and access their RV function early and provide targeted treatment.

Due to the special anatomical location and complex morphological structure of the RV, it is difficult to accurately assess its function. Currently, clinical evaluation mainly relies on imaging to evaluate RV function. Echocardiography has become the preferred method to evaluate RV function due to its non-invasive, economical, convenient, good repeatability, and high accuracy [11, 12]. Ultrasound speckle tracking imaging technology is a new quantitative ultrasound evaluation method based on cardiac motion mechanics and myocardial motion deformation, which can effectively evaluate the overall and local function of myocardium [13]. RV longitudinal strain parameters can quantitatively evaluate the overall and local deformation characteristics of the RV myocardium [14].

This investigation used ultrasound speckle tracking imaging technology to detect RV myocardial global longitudinal strain (RVGLS) before and after treatment of patients with pulmonary embolism with or without pulmonary hypertension. The aim was to analyze the impact of RVGLS on RV function in patients with pulmonary embolism.

Material and methods

General information

The study included 65 patients with pulmonary embolism in our hospital from January 2022 to December 2023. There were 36 males and 29 females, aged 54 to 75 yrs, with an average age of 66.2 ± 5.4 yrs.

Mean pulmonary artery pressure (mPAP) was measured via right heart catheterization or estimated non-invasively using echocardiography by applying the simplified Bernoulli equation to the peak tricuspid regurgitant velocity. Patients with mPAP <25 mm Hg at rest were classified as Group B (without pulmonary hypertension), while those with mPAP ≥ 25 mm Hg were classified as Group C (with pulmonary hypertension). Among the 65 patients with pulmonary embolism, 34 were classified as Group B (without pulmonary hypertension) and 31 as Group C (with pulmonary hypertension).

Inclusion criteria:

- 1) Diagnosed with pulmonary embolism;
- 2) Normal left ventricular function;
- 3) Complete clinical data;
- 4) Informed consent from patient and their family.

Exclusion criteria:

- 1) Acute coronary syndrome, obvious valvular disease, or obvious abnormality of the interatrial septum;
- 2) RV outflow tract stenosis or pulmonary artery stenosis;
- 3) Severe liver, lung, or renal insufficiency;
- 4) Malignant tumors;
- 5) Mental disorders, cognitive impairment, and inability to communicate normally;
- 6) A history of cardiac surgery or poor ultrasound image quality;
- 7) Incomplete follow-up.

Another 35 healthy volunteers with matching gender and age were selected as Group A. This group included 18 males and 17 females, aged 54 to 74 yrs, with an average age of (65.3 ± 5.6) years. Cardiopulmonary diseases were excluded by transthoracic echocardiography, electrocardiogram and chest X-ray.

Thrombolytic treatment

All patients received thrombolytic therapy with a total dose of urokinase (20,000 IU/kg) administered intravenously over 2 hours. The activated whole blood coagulation time and activated partial thromboplastin time were measured every 4 hr. When the activated partial thromboplastin time returned to 1.5 to 2.0 times the normal control value, 0.3 ml of low molecular weight heparin sodium is injected subcutaneously once every 12 hr. All patients with pulmonary embolism underwent ultrasound examination and data collection within 24 hr before and 14 days after thrombolytic treatment.

Evaluation indicators

Basic patient information was collected from hospital files. This information included age, gender, body mass index (BMI), blood pressure, medical history. Echocardiographic examinations were performed with subjects in the left lateral decubitus position using a Philips iE33 color Doppler ultrasound system equipped with an S5-1 transducer (frequency range 1–5 MHz). The following right ventricular (RV) functional parameters were measured:

- RV end-diastolic diameter (RVEDD),
- RV to left ventricular diameter ratio (RV/LV),
- RV work index (RIMP) = (isovolumic relaxation time + isovolumic contraction time)/ejection time,
- Main pulmonary artery diameter (MPA),
- RV area change fraction (RVFAC) = [(RV end-diastolic area – RV end-systolic area)/RV end-diastolic area] \times 100%,
- RV ejection fraction (RVEF),
- RV short-axis shortening rate (RVFS),
- Tricuspid annular peak systolic velocity (S'),
- Tricuspid annular plane systolic excursion (TAPSE).

Pulmonary artery systolic pressure (PASP) was calculated as the peak tricuspid regurgitation pressure gradient plus estimated right atrial pressure. Apical four-chamber views centered on the RV were acquired at a frame rate of 70–80 frames/second. Speckle-tracking analysis was performed offline using EchoPAC software (GE Healthcare). The end-systolic RV endocardial border was manually traced to generate a region of interest, and the software automatically derived RV global longitudinal strain (RVGLS) and RV free wall longitudinal strain (RVFWLS).

Statistical methods

Data analysis was performed by a SPSS 21.0 software package. Numerical data are presented as number (percentage). These data were analyzed with a chi squared test. Data with normal distribution are presented as mean \pm standard deviation (SD). Continuous variables with normal distribution were compared using Student's t-test (for two groups) or one-way ANOVA (for three groups). Non-normally distributed data were analyzed using the Mann-Whitney U test (for two groups) or Kruskal-Wallis test (for three groups). Intergroup comparisons were performed using one-way analysis of variance (ANOVA) followed by post-hoc Tukey's test for multiple comparisons. Medcalc software was used to generate receiver operating characteristic (ROC) curves. The optimal cutoff value for each parameter was determined by maximizing the Youden index (calculated as sensitivity + specificity – 1). The area under the curve (AUC) was calculated to evaluate diagnostic accuracy. Multifactor logistic regression was used to analyze the relationship between RVGLS $> -20.59\%$ and the occurrence of pulmonary em-

bolism. Pearson correlation analysis was used to evaluate the correlation between RVGLS and RV function parameters under different conditions. $p<0.05$ was considered statistically significant.

Results

Comparison of baseline data of the three groups of patients

No statistically significant differences were found for gender, age, BMI, systolic blood pressure, diastolic blood pressure, heart rate, left ventricular ejection fraction, hypertension, diabetes, dyslipidemia, obesity, smoking history, and drinking history among the the groups ($p>0.05$ for all, Table 1).

Comparison of RV echocardiographic characteristics of the three groups

Compared to Group A, the before treatment values of Groups B and C for RVEDD, RV/LV, RIMP, MPAPAS, P, RVGLS, and RVFWLS were significantly higher ($p<0.05$ for all), and RVFAC, RVEF, RVFS, S', and TAPSE were significantly lower ($p<0.05$ for all). After treatment, significant differences were observed in all the above variables between Groups C and A ($p<0.05$). Compared with Group B values before treatment, before treatment Group C, RVEDD, RV/LV, RIMP, MPA, PASP, RVGLS, and RVFWLS were significantly higher, and RVFAC, RVEF, RVFS, S', and TAPSE were significantly lower ($p<0.05$ for all). Significant differences were found in the above indicators between Group B and Group C when comparing post-treatment values with pre-treatment values ($p<0.05$).

Analysis of the effectiveness of RVGLS in reflecting the condition of patients with pulmonary embolism

ROC curve analysis showed that $RVGLS > -20.59\%$ is the best cutoff value for predicting the occurrence of pulmonary embolism, and $RVGLS > -17.42\%$ is the best cutoff value for predicting the occurrence of pulmonary hypertension in patients with pulmonary embolism. See Table 3 and Figure 1.

Multivariable logistic regression model analysis

In accordance with the outcomes of multivariable logistic regression model analysis, $RVGLS > -20.59\%$ is independently related to the occurrence of pulmonary embolism, and $RVGLS > -17.42\%$ is independently related to pulmonary embolism complicated by pulmonary hypertension. See Tables 4 and 5.

Correlation between RVGLS and RVfunction

In Groups A and B: RVGLS showed significant negative correlations with RVFAC ($r = -0.522$, $p < 0.001$), RVEF ($r = -0.439$, $p < 0.001$), and TAPSE ($r = -0.254$, $p = 0.035$).

RVGLS was positively correlated with RIMP ($r = 0.456$, $p < 0.001$) and PASP ($r = 0.371$, $p = 0.002$).

In Groups B and C (pre-treatment): RVGLS negatively correlated with RVFAC ($r = -0.459$, $p < 0.001$) and RVEF ($r = -0.577$, $p < 0.001$). RVGLS positively correlated with RIMP ($r = 0.261$, $p = 0.036$) and PASP ($r = 0.339$, $p = 0.006$).

In Groups B and C (post-treatment): RVGLS remained negatively correlated with RVFAC ($r = -0.264$, $p = 0.034$) and RVEF ($r = -0.251$, $p = 0.044$). RVGLS was positively correlated with RIMP ($r = 0.513$, $p < 0.001$) and PASP ($r = 0.251$, $p = 0.043$) (Table 6).

Discussion

Pulmonary embolism is the third leading cause of cardiovascular death worldwide after stroke and heart attack. Although common, pulmonary embolism is often elusive as a diagnosis and requires a high index of suspicion in patients with clinical cardiopulmonary symptoms because the consequences of missed or delayed diagnosis of pulmonary embolism can be serious [15, 16]. In recent years, with the emergence of local thrombolysis, the treatment of pulmonary embolism has made progress. Also, other treatment methods, such as mechanical extraction devices, hemodynamic support devices, and surgical embolectomy have gradually been improved [17]. Multidisciplinary pulmonary embolism response teams have been developed nationwide to optimize the care of patients with venous thromboembolism. Despite multidisciplinary advancements, challenges persist in managing pulmonary embolism, including delayed diagnosis, suboptimal response to anticoagulation or thrombolysis, high bleeding risk in vulnerable populations, and limited access to advanced therapies (e.g., catheter-directed interventions) [18]. Therefore, early assessment and prediction of the progression of pulmonary embolism are of great significance.

Studies have shown that persistent RV dysfunction and pulmonary hypertension are important outcomes following pulmonary embolism. Increased pulmonary artery pressure caused by pulmonary embolism increases right heart load secondary to increased RV afterload, increased afterload and myocardial ischemia. The result may be acute RV dysfunction. Persistent RV pressure overload secondary to pulmonary embolism leads to RV dilation and dysfunction. This can impair LV filling via ventricular interdependence, resulting in reduced cardiac output, myocardial ischemia, and interventricular dysynchrony due to altered septal motion [19, 20].

RVGLS based on ultrasound speckle tracking imaging technology is a prognostic, reliable, and accurate tool for evaluating RV systolic function in cardiovascular diseases [21]. Strain parameter analysis can more sensitively and accurately detect RV dysfunction in patients with pulmonary embolism than conventional echocardiographic RV function parameters [22]. In addition, RVGLS has also been

Table 1. Comparison of general information of three groups

Variable	Group A (n= 35)	Group B (n= 34)	Group C (n= 31)	χ^2 / F	p
Gender(male/female)	18/17	19/15	17/14	0.150	0.928
Age (yrs)	66.78±7.59	67.54±7.16	66.94±7.08	0.355	0.702
BMI (kg/m ²)	25.36±4.30	26.02±4.13	25.14±4.05	0.026	0.975
Systolic blood pressure (mmHg)	131.5±12.6	132.8±11.5	130.7±13.4	1.145	0.322
Diastolic blood pressure (mmHg)	78.56±10.91	77.23±11.58	79.57±11.29	0.289	0.750
Heart rate (min ⁻¹)	77.69±6.29	78.14±6.05	77.23±6.84	0.118	0.889
LVEF (%)	61.17±4.30	61.54±4.17	60.36±4.28	0.644	0.527
Hypertension	15/20	16/18	14/17	0.123	0.940
Diabetes mellitus	8/27	5/29	6/25	0.748	0.688
Dyslipidemia	11/24	10/24	8/23	0.257	0.880
Obesity	19/16	17/17	19/12	0.846	0.655
Smoking history	8/27	10/24	9/22	0.470	0.791
Drinking history	10/25	12/22	11/20	0.478	0.787

Data are mean±SD or ratios. Statistical tests: χ^2 for categorical variables; F-value for continuous variables with normal distribution.

P-values derived from one-way ANOVA (for continuous variables) or χ^2 test (for categorical variables).

LVEF: Left ventricular ejection fraction (%); BMI: Body mass index (kg/m²).

Table 2. RV echocardiographic characteristics

Variable	Group A (n= 35)	Group B (n=34)		Group C (n=31)	
		pre-treatment	post-treatment	pre-treatment	post-treatment
RVEDD (mm)	30±7	37±7*	31±6#	41±8*	35±7**
RVFAC (%)	19±7	34±7*	39±6#	30±7*	35±6**
RV/LV (%)	0.7±0.3	1.0±0.3*	0.8±0.3*	1.2±0.4*	0.9±0.3**
RVEF (%)	68±4	52±6*	65±4**	46±6*	60±6**
RVFS (%)	35±6	30±6*	33±6#	23±6*	29±6**
RIMP	0.4±0.1	0.5±0.1*	0.4±0.1*	0.6±0.1*	0.5±0.1**
S' (cm/s)	14±1	10±2*	13±2#	8±2*	12±2**
TAPSE (cm)	2.6±0.5	1.8±0.5*	2.5±0.7#	1.4±0.5*	2.1±0.7**
MPA (mm)	21±2	27±3*	23±2**	30±3*	25±3**
PASP (mmHg)	17±1	25±2*	17±1#	60±2*	47±1**
RVGLS (%)	-22±5	-17±6*	-21±6#	-13±5*	-18±5**
RVFWLS (%)	-29±6	-26±6*	-28±8#	-15±7*	-22±8**

Data are mean±SD. *Compared with Group A, p<0.05; #Compared with the same group before therapy, p<0.05.

RVEDD: Right ventricular end-diastolic diameter (mm); RVFAC: Right ventricular fractional area change (%); RV/LV: Right ventricular to left ventricular diameter ratio; RVEF: Right ventricular ejection fraction (%); RVFS: Right ventricular short-axis shortening rate (%);

RIMP: Right ventricular index of myocardial performance; S': Tricuspid annular peak systolic velocity (cm/s);

TAPSE: Tricuspid annular plane systolic excursion (cm); MPA: Main pulmonary artery diameter (mm); PASP: Pulmonary artery systolic pressure (mmHg); RVGLS: Right ventricular global longitudinal strain (%); RVFWLS: Right ventricular free wall longitudinal strain (%).

Table 3. Analysis of the diagnostic performance of RVGLS using a cutoff value of $\leq -20.59\%$ in patients with pulmonary embolism

Groups	AUC	Cutoff value	Youden index	Sensitivity (%)	Specificity (%)	95% C	p
Groups A and B	0.718	>-20.59	0.364	73.53	62.86	0.597–0.820	<0.001
Groups B and C	0.689	>-17.42	0.304	77.42	52.94	0.562–0.798	0.004

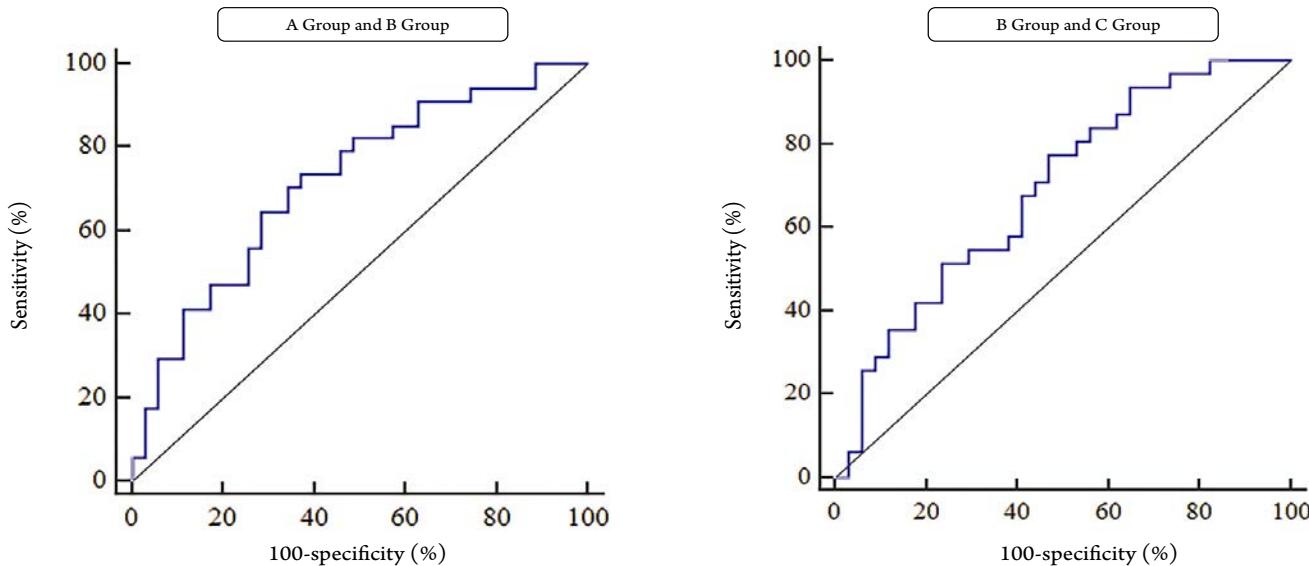
AUC: Area under the ROC curve; Youden index: Sensitivity + Specificity – 1.

Table 4. Single and multi-factor analysis of RVGLS $> -20.59\%$

Variable	Univariate analysis			Multivariate analysis		
	OR	95% CI	p	OR	95% CI	p
Pulmonary embolism	3.855	1.687~14.014	0.015	6.365	1.878~21.575	0.003
PASP	0.105	0.016~0.692	0.019	–	–	–

OR, odds ratio; CI, confidence interval. PASP, pulmonary artery systolic pressure.

Figure 1. ROC curve of RVGLS reflecting the condition of patients with pulmonary embolism



proven to be a factor reflecting RV systolic dysfunction in patients with pulmonary embolism [23]. The findings of this investigation show that the RVGLS of Groups B and C before treatment were higher than that of Group A. The RVGLS of Group B and Group C after treatment was significantly reduced compared with that before treatment. The RVGLS of

Group B before treatment was the same as that of Group C before treatment. This shows that RVGLS in patients with pulmonary embolism increases as the condition worsens, and will be improved accordingly after thrombolytic treatment. Thus, RVGLS has value in predicting the condition of patients with pulmonary embolism.

Table 5. Single and multi-factor analysis of RVGLS > -17.42%

Variable	Univariate analysis			Multivariate analysis		
	OR	95% CI	p	OR	95% CI	p
Pulmonary hypertension	1.511	1.005-2.273	0.047	7.398	1.871-29.252	0.004
Diastolic blood pressure	0.894	0.804-0.959	0.040	-	-	-
RVFS	0.105	0.016-0.692	0.019	-	-	-

OR, odds ratio; CI, confidence interval; RVFS, right ventricular short-axis shortening rate.

Table 6. Correlation results between RVGLS and RV function parameters

Variable	A Group and B Group		B Group and C Group			
	r	p	pre-treatment	post-treatment	r	p
RVEDD (mm)	0.125	0.304	0.174	0.165	0.091	0.471
RVFAC (%)	-0.522	<0.001	-0.459	<0.001	-0.264	0.034
RV/LV (%)	0.181	0.136	0.043	0.735	0.146	0.247
RVEF (%)	-0.439	<0.001	-0.577	<0.001	-0.251	0.044
RVFS (%)	-0.010	0.934	-0.190	0.129	-0.199	0.112
RIMP	0.456	<0.001	0.261	0.036	0.513	<0.001
S' (cm/s)	-0.081	0.510	0.090	0.476	-0.053	0.676
TAPSE (cm)	-0.254	0.035	-0.114	0.366	-0.108	0.394
MPA (mm)	0.197	0.105	0.147	0.241	0.042	0.738
PASP (mmHg)	0.371	0.002	0.339	0.006	0.251	0.043
RVFWLS (%)	0.231	0.056	0.106	0.403	0.069	0.582

RVEDD: Right ventricular end-diastolic diameter (mm); RVFAC: Right ventricular fractional area change (%); RV/LV: Right ventricular to left ventricular diameter ratio; RVEF: Right ventricular ejection fraction (%); RVFS: Right ventricular short-axis shortening rate (%); RIMP: Right ventricular index of myocardial performance; S': Tricuspid annular peak systolic velocity (cm/s); TAPSE: Tricuspid annular plane systolic excursion (cm); MPA: Main pulmonary artery diameter (mm); PASP: Pulmonary artery systolic pressure (mmHg); RVFWLS: Right ventricular free wall longitudinal strain (%).

RV systolic function parameters detected by conventional ultrasound can be used as auxiliary diagnostic indicators for pulmonary embolism and are used to risk stratify the severity of pulmonary embolism patients [24]. They are also widely used in the diagnosis of RV systolic dysfunction in various cardiovascular diseases [25, 26]. Conventional echocardiographic parameters have inherent limitations in assessing RV function, including angle dependency (e.g., tissue Doppler-based RIMP), load sensitivity (e.g., RVEF influenced by preload and afterload), and geometric assumptions that may not fully capture the RV's complex morphology [27]. RV afterload is the main determinant of normal RV function, and changes in RV afterload can significantly affect RV systolic function [28]. Due to the complex geometric shape of the RV, it is difficult to evaluate RVEF with geometric models using two-dimensional echocardiography [29, 30].

The evaluation indicators of RV systolic function recommended by the latest guidelines include parameters such as RVFAC and RIMP [31, 32]. RVFAC is a two-dimensional parameter that can evaluate the overall function of the RV. RIMP measured by tissue Doppler is angle-dependent, so it can be measured by two-dimensional ultrasound. There are limitations in detecting RV systolic function by cardiology [33], so there is greater dependence on imaging technology. Ultrasound speckle tracking imaging technology is a new technology based on two-dimensional images. It can track the movement of the entire RV and the free wall of the RV in each radial and circumferential direction frame by frame. Ultrasound speckle tracking imaging technology can quantitatively analyze the ultrasonic speckle echo of myocardial tissue, and, thus, accurately evaluate the systolic function of the RV [34]. In addition, ultrasonic speckle tracking imaging technology is not affected by respiratory movement, angle and motion artifacts. It can quantitatively analyze relevant index data of myocardial function offline with high sensitivity, accuracy, and repeatability [35]. Previous studies have shown that the global and local strain parameters of the RV detected using ultrasound speckle tracking imaging technology can accurately and effectively evaluate RV function [36].

The findings of this research revealed a correlation between RVGLS and RVGFAC, RVEF, RIMP, and PASP in patients with pulmonary embolism before and after treatment. The correlation between RVGLS and conventional parameters (e.g., RVFAC, RVEF) in our study ($r = -0.522$ to -0.439) was weaker than reported by Chen et al. ($r = -0.72$ for RVGLS vs. RVFAC) [37], and Chen et al. ($r = -0.70$ for RVGLS vs. RVEF) [38]. This variability may stem from differences in patient cohorts (e.g., inclusion of PH subgroups) or imaging protocols. A possible reason is that only the changes in RV function parameters of pulmonary embolism patients with pulmonary hypertension were analyzed and evaluated, and the risk stratification of pulmonary embolism patients and the severity

of pulmonary hypertension were not classified. It may be that different patients have different risk stratification and different degrees of pulmonary hypertension.

The effects of pulmonary hypertension on RV systolic function vary depending on the pathological stage and loading conditions. In the compensatory phase, RV adapts to increased afterload through myocardial hypertrophy and enhanced contractility, often maintaining normal RVEF, while RVGLS may show early signs of decline [39]. In the compensated phase, chronic pressure overload leads to RV dilation, depletion of contractile reserve, and significant reductions in RVEF and RVFAC, accompanied by further deterioration in RVGLS [40]. In acute PE complicated by PH, a sudden increase in afterload causes acute RV dilation, elevated RV/LV diameter ratio, and increased RIMP, with the degree of RVGLS impairment directly correlating with thrombus burden and the extent of pulmonary artery obstruction [41]. Research has shown that conventional ultrasound indicators lack sensitivity in identifying pulmonary embolism [42]. RVGLS combined with conventional RV ultrasound parameters can significantly improve the sensitivity and specificity in diagnosing pulmonary embolism [43].

Conclusion

In conclusion, this study highlights the clinical utility of RVGLS as a sensitive and specific marker for assessing right ventricular dysfunction in patients with pulmonary embolism, independent of the presence of pulmonary hypertension. RVGLS demonstrates strong correlations with conventional echocardiographic parameters while overcoming limitations related to load dependency and geometric assumptions. Its ability to detect subclinical RV impairment early and stratify disease severity underscores its potential as a valuable tool in the diagnostic and prognostic evaluation of pulmonary embolism. Future multicenter studies are warranted to validate these findings and explore the role of RVGLS in guiding therapeutic decision-making.

Data Availability

The experimental data used to support the findings of this study are available from the corresponding author upon request.

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REFERENCES

- Hirmerová J, Bílková S, Woznica V. Isolated pulmonary embolism - a specific clinical entity? *Vnitřní lékařství*. 2023;69(1):8–13. DOI: 10.36290/vnl.2023.001
- Demelo-Rodriguez P, Galeano-Valle F, Salzano A, Biskup E, Vriz O, Cittadini A et al. Pulmonary Embolism: A Practical Guide for the Busy Clinician. *Heart Failure Clinics*. 2020;16(3):317–30. DOI: 10.1016/j.hfc.2020.03.004
- Khandait H, Harkut P, Khandait V, Bang V. Acute pulmonary embolism: Diagnosis and management. *Indian Heart Journal*. 2023;75(5):335–42. DOI: 10.1016/j.ihj.2023.05.007
- An J, Nam Y, Cho H, Chang J, Kim D-K, Lee KS. Acute Pulmonary Embolism and Chronic Thromboembolic Pulmonary Hypertension: Clinical and Serial CT Pulmonary Angiographic Features. *Journal of Korean Medical Science*. 2022;37(10):e76. DOI: 10.3346/jkms.2022.37.e76
- Surov A, Akritidou M, Bach AG, Bailis N, Lerche M, Meyer HJ et al. A New Index for the Prediction of 30-Day Mortality in Patients With Pulmonary Embolism: The Pulmonary Embolism Mortality Score (PEMS). *Angiology*. 2021;72(8):787–93. DOI: 10.1177/0003319721993346
- Pulmonary embolism. *Nature Reviews Disease Primers*. 2018;4(1):18031. DOI: 10.1038/nrdp.2018.31
- Jiménez D, Lobo JL, Barrios D, Prandoni P, Yusen RD. Risk stratification of patients with acute symptomatic pulmonary embolism. *Internal and Emergency Medicine*. 2016;11(1):11–8. DOI: 10.1007/s11739-015-1388-0
- Yuriditsky E, Horowitz JM, Lau JF. Chronic thromboembolic pulmonary hypertension and the post-pulmonary embolism (PE) syndrome. *Vascular Medicine*. 2023;28(4):348–60. DOI: 10.1177/1358863X231165105
- Barco S, Mavromanoliki AC, Kreitner K-F, Bunck AC, Gertz RJ, Ley S et al. Preexisting Chronic Thromboembolic Pulmonary Hypertension in Acute Pulmonary Embolism. *Chest*. 2023;163(4):923–32. DOI: 10.1016/j.chest.2022.11.045
- Benza RL, Langleben D, Hemnes AR, Vonk Noordegraaf A, Rosenkranz S, Thenappan T et al. Riociguat and the right ventricle in pulmonary arterial hypertension and chronic thromboembolic pulmonary hypertension. *European Respiratory Review*. 2022;31(166):220061. DOI: 10.1183/16000617.0061-2022
- Kohls N, Konstantinides SV, Lang IM, Funk GC, Huber K. Risk stratification and risk-adapted management of acute pulmonary embolism. *Wiener klinische Wochenschrift*. 2023;135(1–2):22–7. DOI: 10.1007/s00508-022-02104-0
- Konstantinides SV, Vicaut E, Danays T, Becattini C, Bertolotti L, Beyers-Westendorf J et al. Impact of Thrombolytic Therapy on the Long-Term Outcome of Intermediate-Risk Pulmonary Embolism. *Journal of the American College of Cardiology*. 2017;69(12):1536–44. DOI: 10.1016/j.jacc.2016.12.039
- Li Y, Wang Y, Ye X, Kong L, Zhu W, Lu X. Clinical study of right ventricular longitudinal strain for assessing right ventricular dysfunction and hemodynamics in pulmonary hypertension. *Medicine*. 2016;95(50):e5668. DOI: 10.1097/MD.0000000000005668
- Li S-Y, Zhang Y, Shen T-T, Lu T-T, Li X. Measuring of strain parameters reflects changes of right ventricular function before and after thrombolytic therapy in patients with acute pulmonary embolism. *The International Journal of Cardiovascular Imaging*. 2022;38(10):2199–208. DOI: 10.1007/s10554-022-02626-8
- Mabrouk B, Anis C, Hassen D, Leila A, Daoud S, Hichem K et al. Pulmonary thromboembolism: incidence, physiopathology, diagnosis and treatment. *La Tunisie Médicale*. 2014;92(7):435–47. PMID: 25775281
- Mascarello MG, Vannoni G, Indavere A, Waistein KM, Estrella ML, Rodríguez SG et al. Pulmonary embolism. Clinical suspicion and anatomopathological correlation. *Medicina*. 2020;80(2):97–102. PMID: 32282313
- Osho AA, Duzdzinski DM. Interventional Therapies for Acute Pulmonary Embolism. *Surgical Clinics of North America*. 2022;102(3):429–47. DOI: 10.1016/j.suc.2022.02.004
- Lacey MJ, Hammad TA, Parikh M, Tefera L, Sharma P, Kahl R et al. Prospective Experience of Pulmonary Embolism Management and Outcomes. *The Journal of Invasive Cardiology*. 2021;33(3):E173–80. PMID: 33570502
- Samaranayake CB, Royle G, Jackson S, Yap E. Right ventricular dysfunction and pulmonary hypertension following sub-massive pulmonary embolism. *The Clinical Respiratory Journal*. 2017;11(6):867–74. DOI: 10.1111/crj.12429
- Lang IM, Campean IA, Sadushi-Kolici R, Badr-Eslam R, Gerges C, Skoro-Sajer N. Chronic Thromboembolic Disease and Chronic Thromboembolic Pulmonary Hypertension. *Clinics in Chest Medicine*. 2021;42(1):81–90. DOI: 10.1016/j.ccm.2020.11.014
- Edemskiy A.G., Ivanov S.N., Kliver E.N., Novikova N.V., Galstyan M.G., Sevastyanov A.V. et al. Patient's way from acute pulmonary embolism to chronic thromboembolic pulmonary hypertension: diagnostic and treatment options. *Therapeutic Archive*. 2019;91(8):108–14. [Russian: Едемский А.Г., Иванов С.Н., Кливер Е.Н., Новикова Н.В., Галстян М.Г., Севастянов А.В. и др. Путь пациента от острой тромбоэмболии легочной артерии до хронической тромбоэмбологической легочной гипертензии: вопросы диагностики и лечения. Терапевтический архив. 2019;91(8):108–14]. DOI: 10.26442/00403660.2019.08.000316
- Trivedi SJ, Terluk AD, Krishnadas L, Chow V, Chia E-M, Byth K et al. Right ventricular speckle tracking strain echocardiography in patients with acute pulmonary embolism. *The International Journal of Cardiovascular Imaging*. 2020;36(5):865–72. DOI: 10.1007/s10554-020-01779-8
- Mahfoudhi H, Chenik S, Haggui A, Dahmani R, Mastouri M, Lahidheb D et al. Fonction ventriculaire droite après un premier épisode d'embolie pulmonaire : apport du strain 2D. *Annales de Cardiologie et d'Angéiologie*. 2020;69(3):115–9. DOI: 10.1016/j.an-card.2020.03.010
- Dutta T, Frishman WH, Aronow WS. Echocardiography in the Evaluation of Pulmonary Embolism. *Cardiology in Review*. 2017;25(6):309–14. DOI: 10.1097/CRD.0000000000000158
- Guliani S, Das Gupta J, Osofsky R, Marek J, Rana MA, Marinaro J. Protocolized use of catheter-directed thrombolysis and echocardiography is highly effective in reversing acute right heart dysfunction in severe submassive pulmonary embolism patients. *Perfusion*. 2020;35(7):641–8. DOI: 10.1177/0267659119896891
- Chornenki NLJ, Poorzargar K, Shanjer M, Mbuagbaw L, Delluc A, Crowther M et al. Detection of right ventricular dysfunction in acute pulmonary embolism by computed tomography or echocardiography: A systematic review and meta-analysis. *Journal of Thrombosis and Haemostasis*. 2021;19(10):2504–13. DOI: 10.1111/jth.15453
- Agarwal G, Kharge J, Raghu TR, MohanRao PS, Manjunath CN. Incidence and predictors of chronic thromboembolic pulmonary hypertension following acute pulmonary embolism: An echocardiography guided approach. *Indian Heart Journal*. 2021;73(6):746–50. DOI: 10.1016/j.ihj.2021.10.003
- Sanz J, Sánchez-Quintana D, Bossone E, Bogaard HJ, Naeije R. Anatomy, Function, and Dysfunction of the Right Ventricle: JACC State-of-the-Art Review. *Journal of the American College of Cardiology*. 2019;73(12):1463–82. DOI: 10.1016/j.jacc.2018.12.076
- Pargana J, Calé R, Martinho M, Santos J, Lourenço C, Castro Pereira JA et al. Prevalence and predictors of chronic thromboembolic pulmonary hypertension following severe forms of acute pulmonary embolism. *Revista Portuguesa de Cardiologia*. 2023;42(12):947–58. DOI: 10.1016/j.repc.2023.06.007
- Wong DJ, Sampat U, Gibson MA, Auger WR, Madani MM, Daniels LB et al. Tricuspid annular plane systolic excursion in chronic thromboembolic pulmonary hypertension before and after pulmonary thromboendarterectomy. *Echocardiography*. 2016;33(12):1805–9. DOI: 10.1111/echo.13364
- Augustine DX, Coates-Bradshaw LD, Willis J, Harkness A, Ring L, Grapsa J et al. Echocardiographic assessment of pulmonary hypertension: a guideline protocol from the British Society of Echocardiogra-

phy. *Echo Research & Practice*. 2018;5(3):G11–24. DOI: 10.1530/ERP-17-0071

32. Slegg OG, Willis JA, Wilkinson F, Sparey J, Wild CB, Rossdale J et al. IMProving PULmonary hypertension Screening by Echocardiography: IMPULSE. *Echo Research & Practice*. 2022;9(1):9. DOI: 10.1186/s44156-022-00010-9

33. Doheny C, Gonzalez L, Duchman SM, Varon J, Bechara CF, Cheung M et al. Echocardiographic assessment with right ventricular function improvement following ultrasound-accelerated catheter-directed thrombolytic therapy in submassive pulmonary embolism. *Vascular*. 2018;26(3):271–7. DOI: 10.1177/1708538117733645

34. Dong J, Jiang X-M, Xie D-J, Luo J, Ran H, Li L et al. Establishment of a canine model of pulmonary arterial hypertension induced by dehydromonocrotaline and ultrasonographic study of right ventricular remodeling. *Clinical and Experimental Hypertension*. 2023;45(1):2190503. DOI: 10.1080/10641963.2023.2190503

35. Zhou C, Lou B, Li H, Wang X, Ao H, Duan F. Incidence, risk factors and prognostic effect of imaging right ventricular involvement in patients with COVID-19: a dose-response analysis protocol for systematic review. *BMJ Open*. 2021;11(5):e049866. DOI: 10.1136/bmjopen-2021-049866

36. Waziri F, Ringgaard S, Mellemkjær S, Bøgh N, Kim WY, Clemmensen TS et al. Long-term changes of right ventricular myocardial deformation and remodeling studied by cardiac magnetic resonance imaging in patients with chronic thromboembolic pulmonary hypertension following pulmonary thromboendarterectomy. *International Journal of Cardiology*. 2020;300:282–8. DOI: 10.1016/j.ijcard.2019.09.038

37. Vitarelli A, Barillà F, Capotosto L, D'Angeli I, Truscelli G, De Maio M et al. Right Ventricular Function in Acute Pulmonary Embolism: A Combined Assessment by Three-Dimensional and Speckle-Tracking Echocardiography. *Journal of the American Society of Echocardiography*. 2014;27(3):329–38. DOI: 10.1016/j.echo.2013.11.013

38. Wang Y, Guo D, Liu M, Zhang X, Hu H, Yang H et al. Assessment of right ventricular remodeling in chronic thromboembolic pulmonary hypertension by 2D-speckle tracking echocardiography: A comparison with cardiac magnetic resonance. *Frontiers in Cardiovascular Medicine*. 2022;9:999389. DOI: 10.3389/fcvm.2022.999389

39. Tello K, Dalmer A, Vanderpool R, Ghofrani HA, Naeije R, Roller F et al. Right ventricular function correlates of right atrial strain in pulmonary hypertension: a combined cardiac magnetic resonance and conductance catheter study. *American Journal of Physiology-Heart and Circulatory Physiology*. 2020;318(1):H156–64. DOI: 10.1152/ajpheart.00485.2019

40. Vonk Noordegraaf A, Westerhof BE, Westerhof N. The Relationship Between the Right Ventricle and its Load in Pulmonary Hypertension. *Journal of the American College of Cardiology*. 2017;69(2):236–43. DOI: 10.1016/j.jacc.2016.10.047

41. Pruszczak P, Goliszek S, Lichodziejewska B, Kostrubiec M, Ciurzyński M, Kurnicka K et al. Prognostic Value of Echocardiography in Normotensive Patients With Acute Pulmonary Embolism. *JACC: Cardiovascular Imaging*. 2014;7(6):553–60. DOI: 10.1016/j.jcmg.2013.11.004

42. Maraziti G, Vinson DR, Becattini C. Echocardiography for risk stratification in patients with pulmonary embolism at low risk of death: a response. *European Heart Journal*. 2021;43(1):86–7. DOI: 10.1093/eurheartj/ehab779

43. Wilson R, Eguchi S, Orihara Y, Pfeiffer M, Peterson B, Ruzieh M et al. Association between right ventricular global longitudinal strain and mortality in intermediate-risk pulmonary embolism. *Echocardiography*. 2024;41(4):e15815. DOI: 10.1111/echo.15815