Original Article

Evaluation of CHA_2DS_2-VASc Score and R_2CHADS_2 Score in Patients with Acute Pulmonary Thromboembolism and Right Ventricular Dysfunction

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Abstract

Background and Aim: Acute pulmonary embolism (APE) is one of the fatal emergencies. Imaging methods are not available to all physicians as an emergency and depend on the individual. For this reason, besides imaging methods, more easily applicable and individual-independent parameters are investigated to help the diagnosis. Some scoring systems are also used according to the patient's current disease status. R_2CHADS_2 score is used more in patients with chronic renal failure (CRF). The aim of the study was to compare CHA_2DS_2-VASc and R_2CHADS_2 scores in patients with APE and right ventricular dysfunction (RVD). **Methods:** The study was conducted retrospectively. The patient group of the study consisted of 392 patients diagnosed with APE. Patients with nonmassive pulmonary embolism and submassive pulmonary embolism (SPE) without RVD were defined as Group 1 ($n = 213$) and massive pulmonary embolism and SPE with RVD were defined as Group 2 ($n = 179$). CHA_2DS_2-VASc and R_2CHADS_2 scores were compared as a scoring system in patients. **Results:** CHA_2DS_2-VASc scores and R_2CHADS_2 scores were evaluated in the two groups. The R_2CHADS_2 score was statistically significant in Group 2 ($P < 0.001$). There was a statistically significant difference between the groups in terms of CRF and estimated glomerular filtration rate (15 [7.2%] vs. 29 [16.4%], $P < 0.001$, and 57.6 vs. 46.4 mL/min/1.73 m^2, $P < 0.001$). **Conclusions:** Our findings show that the CHA_2DS_2-VASc score and R_2CHADS_2 are independent predictors of RVD in patients with APE. However, the R_2CHADS_2 scoring system is observed better than the CHA_2DS_2-VASc scoring system in patients with APE and RVD.

Keywords: Acute pulmonary embolism, CHA_2DS_2-VASc, chronic kidney disease, echocardiography, R_2CHADS_2

Introduction

Acute pulmonary embolism (APE) is an emerging form of pulmonary thromboembolism (PTE), a medical emergency that can result in death.[1] Inhospital mortality rate varies according to the clinical course. Clinical course of APE can be examined in three groups as massive, submassive, and nonmassive. This classification of cases with APE is very important as it is decisive for the management of treatment to be applied to patients. APE – as a result of pulmonary arterial occlusion – can lead to life-threatening acute but potentially reversible right ventricular dysfunction (RVD). Inhospital mortality of patients with massive pulmonary embolism (MPE) varies from 25% to 50%.[2] RVD is the most important predictor of mortality in submassive APE and its detection is vital,[3] and these patients should be given thrombolytic treatments. The fastest and simplest method to detect RVD is

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echocardiography (ECHO) in APE, however, it is cardiologist dependent, and also in some patients, it is not possible to evaluate right ventricular function due to imaging difficulties. Therefore, in cases where there is no cardiologist or the right ventricle is not evaluated clearly due to imaging difficulties, a different marker predicting RVD is required. We thought that CHA_2DS_2-VASc and R_2CHADS_2 scores, which are especially used in the risk stratification of atrial fibrillation (AF), may help the risk stratification in AF patients with APE.

The CHA_2DS_2-VASc (C: congestive heart failure [HF] or left ventricular systolic dysfunction, H: hypertension, A: age of ≥75 years, D: diabetes mellitus, S: previous stroke, V: vascular disease, A: age between 65 and 74 years, and Sc: female gender) score is a scoring system that is used to determine the risk of thromboembolism in nonvalvular AF and also enables the management of anticoagulant therapy in diagnosed patients.[4] Moreover, it has also been shown in the literature that it can be used in thrombotic cases that develop after percutaneous coronary interventions.[5,6]

R_2CHADS_2 score was described as $CHADS_2$ score plus one point for renal dysfunction (estimated glomerular filtration rate [eGFR] <60 ml/min/1.73 m^2). Impaired renal function was shown to be a predictor of stroke and systemic embolism in AF patients without valvular disease.[7] In Rivaroxaban Once-Daily, Oral, Direct Factor Xa Inhibition Compared With Vitamin K Antagonism for Prevention of Stroke and Embolism Trial in AF, the study demonstrated that R_2CHADS_2 score was superior to predict stroke risk than $CHADS_2$ score.[7] All components of R_2CHADS_2 had predictive value for both prognosis and complications of ischemic heart disease and AF.

This study aimed to evaluate the comparison of CHA_2DS_2-VASc score and R_2CHADS_2 score to predict RVD in patients with APE, and we aimed to assist clinicians in the decision of APE risk classification in cases where ECHO cannot be performed.

Methods

Study population and design

We retrospectively collected the data of patients with acute pulmonary embolism who applied to the emergency department between January 2016 and June 2019, we retrospectively attended the study. A total of 392 patients were included in the study. The patients over 18 years of age, who presented to the emergency department with symptoms of shortness of breath, hemoptysis, and chest pain and who were diagnosed with APE after undergoing pulmonary computed tomography angiography with a prediagnosis of APE, were included in the study.

All patients had transthoracic ECHO within 48 h of hospitalization that it is routinely (using the General Electric Vivid 3 ultrasound system, Chicago, IL, USA). A review of patient files revealed that ECHO images had been obtained from all cases in semi-supine position, apical four chambers, parasternal short-long axis, and subcostal positions. ECHO was carried out by using a 3.4 MHz transducer probe with two dimensional, classical, and tissue Doppler. Systolic pulmonary artery pressure (sPAP) was calculated by using Bernoulli equation devaluating tricuspid regurgitation pressure gradient (TIPG = 4 × tricuspid regurgitation velocity^2) in addition to right atrial pressure. Pulmonary hypertension was defined as sPAP >36 mmHg.[8] The tricuspid annular plane systolic excursion (TAPSE) method was used to evaluate the RVD. A value of TAPSE ≤15 was accepted to confirm the presence of the RVD. Laboratory parameters were recruited from our hospital electronic database. The blood parameters of the patients consisted of the values when they first applied with APE. The demographic characteristics of the patients, comorbidities, patient histories, vital signs, treatment regimens at the hospital, monitoring findings daily, and the duration of hospital stays were obtained from the medical files of the patients.

Patients with chronic obstructive pulmonary disease, sleep breathing disorders, intracardiac lesions, sepsis or septic shock, serious pericardial and pleural effusions, acute renal failure, malignancies, collagen tissue disease, congenital heart disease, and valvular heart disease were excluded from the study.

The APE cases were assigned to three subgroups, according to their hemodynamic and radiological characteristics. The patients developing hypotension were assigned to the massive PTE subgroup (MPE), the patients with stable hemodynamics but with RVD detected by ECHO were assigned to the submassive PTE group (SPE), and the patients with stable hemodynamics with no RVD confirmed by ECHO were assigned to the nonmassive PTE group (nonmassive pulmonary embolism [NPE]).[9] We organized these three subgroups into two groups according to RVD in accordance with the purpose of our study. Patients with NPE and SPE without RVD were defined as Group 1 and MPE and SPE with RVD were defined as Group 2.

The CHA_2DS_2-VASc score was calculated by summing the points assigned to each of the risk factors, which include congestive HF (1 point), hypertension (1 point), age ≥75 (2 points), diabetes mellitus (1 point), previous stroke, transient ischemic attack or thromboembolism (2 points), vascular disease (history of myocardial infarction, peripheral arterial disease, or complex aortic plaques) (1 point), age between 65 and 74 years (1 point), and female gender (1 point).

The R_2CHADS_2 score was calculated by summing the points assigned to each of the risk factors, which include renal dysfunction (2 points), congestive HF (1 point), hypertension (1 point), age ≥75 (2 points), diabetes mellitus (1 point), previous stroke, transient ischemic attack, or thromboembolism (2 points).

The simplified Pulmonary Embolism Severity Index (sPESI) scores of the patients were calculated using data obtained from FONET software program (version 3.1.1.6 b5) FONET, Ankara, Turkey. The sPESI score was calculated

by summing the points assigned to each risk factor, which include history of cancer (1 point), age >80 (1 point), history of HF or chronic lung disease (1 point), pulse rate >110 beats/min (1 point), systolic blood pressure <100 mmHg (1 point), and SaO_2 <90% (1 point). The patients with sPESI risk scores of 0 were accepted to have low sPESI scores. The patients with sPESI scores of 1 were accepted to have high sPESI scores.

Definition of congestive HF was based on a previous diagnosis of HF. HF was defined according to the criteria recommended by the working group on HF of the European Society of Cardiology.[10] Hypertension was defined as systolic blood pressure >140 mmHg and/or diastolic blood pressure >90 mmHg in at least two separate measurements or if a patient was already receiving antihypertensive medications. Diabetes mellitus was defined as a patient already receiving antidiabetic drugs and/or insulin or if fasting blood glucose level was ≥126 mg/dL. Stroke and transient ischemic attack were assessed with patient history, and only the events owing to thromboembolism were included as a component of CHA_2DS_2-VASc score. Stenosis of ≥50% in noncoronary arteries was defined as peripheral arterial disease.

Definition of chronic renal failure (CRF) was based on a creatinine clearance of <60 mL/minute, which was calculated by the Modification of Diet in Renal Disease formula and utilizing the baseline creatinine level.[11]

The study was conducted in accordance with the principles of the Declaration of Helsinki.

Statistical analysis

All analyses were performed using SPSS for Windows version 18.0 (SPSS, Chicago, Illinois, USA). Quantitative data are presented as means ± standard deviations (SD) for parametric variables or medians with interquartile ranges (lower and upper quartiles) for nonparametric variables. Kolmogorov–Smirnov test was used to assess the compatibility of our data with normal distribution. Comparisons of parametric values among groups were performed by one-way analysis of variance. Comparisons of nonparametric values among groups were performed by the Kruskal–Wallis test. Tukey's HSD (for parametric variables) and Bonferroni adjustment Mann–Whitney *U*-test (for nonparametric variables) were used as *post hoc* test for multiple comparisons among the groups. Multiple linear regression analysis was performed for parameters affecting the presence of RVD. Normally distributed data are expressed as mean ± SDs and nonnormally distributed data are expressed as percentage. $P < 0.05$ was considered statistically significant. Receiver-operating characteristic (ROC) curves were estimated for CHA_2DS_2-VASc and R_2CHADS_2. ROC analysis was used to determine the cutoff values of CHA_2DS_2-VASc and R_2CHADS_2 in predicting RVD in patients with APE.

Ethical statement

Approval was obtained from the Ethics Committee of the Tokat Gaziosmanpasa University – date: April 30, 2020, number: 20-KAEK-081 – where our institution was conducted.

Results

The study population consisted of 392 patients (mean age, 60.2 ± 10.4 years and 55.3% of females). In this study, according to RVD, we divided the patients into two groups: the NPE and SPE without RVD group (n = 120, 56.33%) determined as Group 1 (n = 213) and the MPE and SPE with RVD (n = 3, 1.67%) determined as Group 2 (n = 179). The main characteristics of PTE subgroups are shown in Table 1. Systolic PAB was 42.23 ± 19.24 mmHg in Group 2 and 35.62 ± 18.21 mmHg in Group 1 ($P < 0.001$). Group 2 was older than those in Group 1 (68.2 ± 9.4 vs. 52.2 ± 11.4, $P < 0.001$). There were a total of 217 female patients in the study, and there was no significant difference between the groups in terms of gender ($P = 0.134$). Cardiac diseases were detected at a higher rate in Group 2, and this finding was statistically significant (63 [29.6] vs. 91 [50.7%], $P = 001$).

Newly detected deep vein thrombosis (DVT) was statistically significantly higher in Group 2 (87 [40.7%] vs. 107 [60%], $P = 0.001$). Again, compared to other groups, inhospital mortality rate was detected to be higher in Group 2 (2 [1.1%] vs. 16 [8.9%], $P < 0.001$).

The CHA_2DS_2-VASc scores and R_2CHADS_2 scores were classified into two groups as follows: the scores between 0 and 1 were classified as the low-risk group and the scores of >1 were classified as the high-risk group [Table 1]. When CHA_2DS_2-VASc score was compared in high-risk groups, it was found 108 in Group 1 and 123 in Group 2 ($P < 0.001$). When the R_2CHADS_2 score was compared in high-risk groups, it was found 119 in Group 1 and 141 in Group 2 ($P < 0.001$). The associations of the variables with these groups are summarized in Table 2. The R_2CHADS_2 score was statistically significant in Group 2 ($P < 0.001$). There was a statistically significant difference between the groups in terms of CRF and eGFR (15 [7.2%] vs. 29 [16.4%], $P < 0.001$, and 57.6 versus 46.4 mL/min/1.73 m^2, $P < 0.001$). In addition, the sPESI scores are used to estimate the early mortality rate. sPESI score of all patients included in the study has been calculated. while it was 1.2 ± 0.61 in Group 1, it was found 3.8 ± 0.8 in Group 2 ($P < 0.001$). Our results indicated that a CHA_2DS_2-VASc score of ≥2 could be used as a predictor of RVD in patients presenting with APE with a sensitivity of 76% and a specificity of 68%, an area under the curve of 0.808, and a 95% confidence interval (CI) of 0.756–0.859. $RCHA_2DS_2$ score of ≥2 could be used as a predictor of RVD in patients presenting with APE a sensitivity of 82% and a specificity of 64%, an area under the curve of 0.882, and a 95% CI of 0.844–0.921 [Figure 1].

In multivariate logistic regression analysis, CHA_2DS_2-VASc score (odds ratio [OR]: 1.486, 95% CI: 1.024–2.162, $P = 0.030$), R_2CHADS_2 score (OR: 2.225, 95% CI: 1.1892–

Table 1: The association of variables with acute pulmonary embolism subgroups

Variable	Group 1 (*n*=213), *n* (%)	Group 2 (*n*=179), *n* (%)	*P*
Scores			
CHA_2DS_2-VASc score			
0-1	104 (49.1)	55 (30.9)	0.011
≥2	108 (50.8)	123 (69)	0.009
$R_2CHA_2DS_2$-VASc score			
0-1	112 (52.5)	62 (34.7)	0.154
≥2	119 (55.8)	141 (78.8)	<0.001
sPESI	1.2±0.61	3.8±0.8	<0.001
Demographıc data and dıseases			
Age (years)	52.2±11.2	68.2±9.4	<0.001
Female gender	121 (56.9)	96 (53.9)	0.134
Smoking	63 (29.6)	70 (38.9)	0.216
CRF	15 (7.2)	29 (16.4)	<0.001
DVT	87 (40.7)	107 (60)	0.001
Prior pulmonary thromboembolism	12 (5.5)	7 (3.7)	0.564
Cardiac disease	63 (29.6)	91 (50.7)	0.01
Liver disease	6 (2.7)	18 (9.8)	0.028
Neurological disease	27 (12.8)	29 (16.4)	0.525
Prior DVT	12 (5.5)	8 (4.2)	0.534
Diabetes mellitus	31 (14.5)	21 (11.7)	0.155
Symptoms and hemodynamıc status			
AF	42 (19.9)	34 (18.9)	0.536
Systolic blood pressure (mmHg)	126±27	113±32	0.029
Diastolic blood pressure (mmHg)	75±15	73±20	0.405
Dyspnea	167 (78.2)	168 (93.8)	<0.001
Chest pain	98 (45.8)	87 (48.8)	0.133
Hemoptysis	21 (9.8)	19 (10.6)	0.542
O_2 saturation (%)	94±5	92±5	0.089
PaO_2 (mmHg)	76±25	74±24	0.109
Echocardıographıc fındıngs			
LVEF (%)	55±10	50±11	<0.001
TAPSE (mm)	17±4	12±2	<0.001
RV systolic dysfunction	88 (41.3)	108 (60.5)	<0.001
RV dilatation	121 (56.9)	148 (82.6)	<0.001
Laboratory fındıngs			
eGFR (mL/min/1.73 m^2)	57.6±5	46.4±4	<0.001
Creatinine (g/dL)	1.02±0.4	1.32±0.4	<0.001
White blood cell ($\times10^9$/L)	8.68±2.48	8.88±4.49	0.494
Hemoglobin (g/dL)	13.93±16	12.96±1.7	<0.001
Inhospital mortality	2 (1.1)	16 (8.9)	<0.001

Definition: Group 1: Nonmassive PTE and submassive PTE without RVD. Group 2: Massive PTE and submassive PTE with RVD. CHA_2DS_2-VASc=Congestive heart failure, hypertension, age ≥75 years (doubled), diabetes mellitus, prior stroke or transient ischemic attack (doubled), and vascular disease, age 65-74 years, and sex category (female); sPESI=Simplified Pulmonary Embolism Severity Index; CRF=Chronic renal failure; DVT=Deep venous thrombosis; AF=Atrial fibrillation; PaO_2=Partial pressure of oxygen; LVEF=Left ventricular ejection fraction; TAPSE=Tricuspid annular plane systolic excursion; RV=Right ventricle; eGFR=Estimated glomerular filtration rate; PTE=Pulmonary thromboembolism; RVD=Right ventricular dysfunction

3018, $P = 0.02$), sPAP (OR: 1.216, 95% CI: 1.154–1.262, $P < 0.001$), eGFR (OR: 2.281, 95% CI: 2.124–3.221, $P < 0.001$), and DVT (OR: 3.554, 95% CI: 1.249–9.624, $P = 0.006$) were independent predictors of RVD in patients with APE [Table 2]. TAPSE was found to be 17 mm in Group 1 patients, while it was 12 mm in Group 2 patients, and this was statistically significant [Table 1, $P < 0.001$]. This statistical significance was maintained when multivariate analysis was performed for TAPSE in patients with APE who developed RVD [Table 2].

Thrombolytic therapy was given to three patients in Group 2 who had SPE and developed RVD. In total, 143 patients in Group 2 received thrombolytic therapy [Table 1].

R_2CHADS_2 score and CHA_2DS_2-VASc score were compared with ROC statistical analysis to show the development of RVD

Table 2: Predictors of right ventricular dysfunction in patients with acute pulmonary embolism

Variable	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*P*	OR (95% CI)	*P*
Smoking	1.024 (0.692-1.374)	0.874		
Systolic blood pressure	0.960 (0.952-1.024)	0.316		
Cardiac disease	2.390 (1.628-3.498)	<0.001	2.302 (0.786-6.721)	0.106
Prior DVT	1.386 (0.650-2.824)	0.321		
DVT	2.473 (1.702-3.610)	<0.001	3.554 (1.249-9.626)	0.006
Prior PTE	1.348 (0.588-3.152)	0.443		
eGFR	1.828 (1.204-5.571)	0.004	2.281 (2.124-3.221)	<0.001
Systolic PAP	1.224 (1.183-1.216)	<0.001	1.206 (1.154-1.262)	<0.001
TAPSE	1.416 (1.274-1.55)	<0.001	1.401 (1.253-1.569)	<0.001
sPESI	1.401 (1.063-1.846)	0.017	1.359 (1.019-1.814)	0.037
PaO_2	0.896 (0.895-0.932)	<0.001	0.988 (0.941-1.030)	0.697
O_2 saturation	0.897 (0.860-0.943)	<0.001	0.944 (0.885-1.028)	0.724
CHA_2DS_2-VASc score	1.266 (1.139-1.432)	<0.001	1.486 (1.024-2.162)	0.030
R_2CHADS_2 score	2.102 (1.986-2.224)	<0.001	2.225 (1.892-3.018)	0.002

CHA_2DS_2-VASc=Congestive heart failure, hypertension, age ≥75 years (doubled), diabetes mellitus, prior stroke or transient ischemic attack (doubled), and vascular disease, age 65-74 years, and sex category (female); DVT=Deep venous thrombosis; CI=Confidence interval; PAP=Pulmonary artery pressure; PaO_2=Partial pressure of oxygen; PTE=Pulmonary thromboembolism; $RCHA_2DS_2$: Renal failure addition to CHA_2DS_2; TAPSE=Tricuspid annular plane systolic excursion; OR=Odds ratio; sPESI=Simplified Pulmonary Embolism Severity Index; eGFR=Estimated glomerular filtration rate

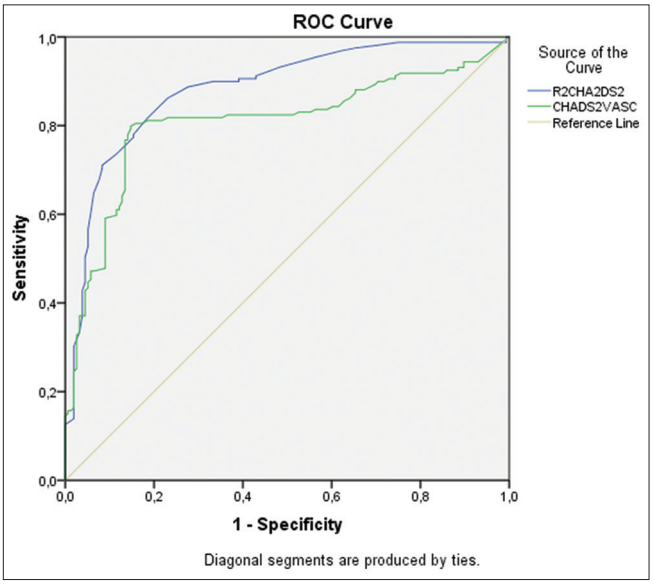

Figure 1: Receiver-operating characteristic curve of CHA_2DS_2-VASc and R_2CHADS_2

in patients with APE (AUC: 0.88, 95% CI: 0.844–0.921 AUC: 0.80, 95% CI: 0.756–0.859, respectively) [Figure 1].

Discussion

This study compares the association between CH_2ADS_2-VASc score and R_2CHADS_2 score about development of RVD in patients with APE. In the present study, we have determined that both scores are independent indicators of RVD.

Despite new diagnosis and treatment methods, APE still has significant morbidity and mortality rates. RVD is one of the most important causes of death in patients who die of PTE.[12] Hypoxia develops after thrombus-induced bronchoconstriction in APE, and this increases right ventricular pressure after a sudden rise in pulmonary artery pressure (PAP), causing right ventricular enlargement. The importance of RVD in determining the prognosis in patients with APE has led researchers to investigate this issue. Ates *et al*. showed that PLR (by dividing the number of platelets by the number of lymphocytes) was found to be associated with the clinical severity of the patients with PTE.[13] The role of platelet activity on embolic events is well known. Increased platelet activity in patients with APE is associated with increased severity of the clinical condition in pulmonary embolism.[14] Since detecting RVD in these patients or predicting its development during follow-up will trigger thrombolytic therapy option, the importance of RVD is more important in patients presenting with clinical signs and symptoms of submassive PTE rather than those presenting with massive PTE. RVD development in APE settings has been associated with certain specific clinical and laboratory variables, such as diabetes, advanced age, and female gender.[15,16] Risk scores containing these variables may be more accurate than those that are included alone. Both CHA_2DS_2-VASc and R_2CHADS_2 scores separately cover risk factors for RVD in patients with PTE.

Abnormal vascular function is recommended as a mediator of stroke.[17] Microvascular dysfunction plays a role in the pathogenesis of RVD. Although thrombus load and embolism constitute an important part of APE etiology, microvascular dysfunction and obstruction are seen in APE patients. The CH_2ADS_2-VASc score is recommended by current guidelines as a proven predictor of thromboembolic events in patients with nonvalvular AF.[4,18] The CHA_2DS_2-VASc score was matched with the results of various diseases such as PTE, AF, chronic obstructive pulmonary disease with or without

AF, decreased left ventricular ejection fraction, and coronary artery disease.[19-21] Recent studies have shown that the CHA_2DS_2-VASc score is independently associated with RVD development in patients with APE.[22] In our study, we also found that CHA_2DS_2-VASc score was an independent risk factor for predicting RVD in patients with APE.

Kidney failure has been shown to increase the risk of thromboembolism two-fold.[23] In International Cooperative Pulmonary Embolism Registration, high creatinine concentration (>177 mol/L) has been found to be a determinant for mortality at 3 months.[24] Similarly, in the large NIS database,[25] inhospital mortality in chronic kidney disease patients or patients with end-stage renal failure was doubled compared to patients with normal kidney function. On the other hand, some records have emphasized that the prevalence of PE or venous thromboembolism is higher in patients with chronic kidney disease.[25-27] In our study, we found an inverse relationship between eGFR and RVD, and we found that the R_2CHADS_2 score was significantly higher in the RVD group.

Once and for all, many risk factors in APE etiology overlap with risk factors for thromboembolism and endothelial and microvascular dysfunction. CHA_2DS_2-VASc score and R_2CHADS_2 also involve the risk factors related to atherosclerosis, vascular spasm, and microvascular dysfunction as the common risk factors. Both have a high predictive power of thromboembolic events and simultaneously contain common risk factors of APE and thromboembolism. They can be used as an exclusive risk estimation tool in APE. And also, according to our data, we found that both the R_2CHADS_2 score and CHA_2DS_2-VASc score are independent indicators of RVD and both scores can be used to predict RVD risk. Although the CHA_2DS_2-VASc score is an important helpful parameter in predicting APE, however, the R_2CHADS_2 score may also be considered as another auxiliary parameter in patients with renal dysfunction.

Study limitations

Our study has several limitations. It was an observational, retrospective, single-center study with a relatively small number of patients.

Conclusions

Our findings suggest CHA_2DS_2-VASc score and R_2CHADS_2 to be an independent predictor of RVD in patients with APE. Both scores can be very useful in this regard as an easily applicable instrument in prediction of high-risk patients. We recommend that clinicians may consider using more priority R_2CHADS_2 scores to predict RVD in patients with decreased renal function.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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