ORIGINAL ARTICLE

Prediction of new onset atrial fibrillation in patients with acute pulmonary embolism: The role of sPESI Score

Akut pulmoner emboli hastalarında yeni gelişen atriyal fibrilasyon öngörücülüğü: sPESI Skoru'nun rolü

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ABSTRACT

Objective: Acute pulmonary embolism (APE) is a serious clinical situation and atrial fibrillation (AF) is the most common arrhytmia in clinical practice. The Pulmonary Embolism Severity Index (PESI) is an accepted risk stratification tool used to predict short term mortality in APE. The aim of this study was to evaluate the relationship between the PESI score and new-onset AF in patients with APE.

Methods: The records of 869 APE patients admitted between May 2012 and December 2015 were evaluated retrospectively. The PESI score was calculated for every patient. Clinical variables associated with new-onset AF in APE were assessed after the exclusion of patients with hypertension, coronary or hemodynamically significant valvular heart disease, hepatic or renal dysfunction, chronic obstructive pulmonary disease, thyroid dysfunction, diabetes mellitus, sleep apnea, any history of inflammatory or infectious disease, or recent trauma. New-onset AF was detected in 42 (4.8%) patients.

Results: Age, gender, systolic and diastolic blood pressure, heart rate, fasting glucose level, serum creatinine, left ventricle ejection fraction, tricuspid annular plane systolic excursion value, and pulmonary artery systolic pressure measures were not significantly different between patients with and without AF. New-AF patients demonstrated larger LVEDD and LAD dimensions ($p < 0.001$ for both). The PESI score was higher in the new-onset AF group (93±23 vs.75±17; $p < 0.001$). LVEDD, LAD, levels of uric acid, bilirubin, albumin, and troponin, and PESI score were univariate predictors of new-onset AF.

Conclusion: In patients with APE, the PESI score was positively correlated with new-onset AF. A PESI score greater than 82.50 may be useful to predict new-onset AF in these patients.

ÖZET

Amaç: Akut pulmoner emboli (APE) ciddi bir klinik durumdur ve atriyal fibrilasyon (AF) klinik pratikte en sık görülen aritmidir. Pulmoner Emboli Ciddiyet İndeksi (PESI) APE'de kısa dönem mortaliteyi öngören skorlama sistemidir. Bu çalışmada, APE'li hastalarda PESI skoru ile yeni gelişen AF ilişkisini bulmayı amaçladık.

Yöntemler: Mayıs 2012 ile Aralık 2015 arasında başvuran 869 hastanın bilgileri geriye dönük olarak değerlendirildi. Tüm hastaların PESI skorları hesaplandı. Hipertansiyon, koroner veya yapısal kalp hastalığı, hepatik veya böbrek disfonksiyon, kronik obstrüktif akciğer hastalığı, tiroit disfonksiyon, diyabet veya uyku apne, enflamatuvar veya enfeksiyon hastalığı öyküsü, yeni travma veya cerrahi, steroid veya nonsteroid enflamatuvar ilaç kullanımı bulunan hastalar dışlandı. Kırk iki (%4.8) yeni-AF'li hasta dahil edildi.

Bulgular: Yaş, cinsiyet, sistolik ve diyastolik kan basıncı, kalp hızı, açlık glikoz düzeyi, serum kreatinin, sol ventrikül ejeksiyon fraksiyonu, trikuspit anüler düzlem hareket değeri ve pulmoner arter sistolik basınç ölçümleri AF olan ve olmayan hastalar arasında anlamlı farklılık göstermedi ($p > 0.05$). Yeni-AF hastalarında daha uzun LVEDD ve LAD değerleri mevcuttu ($p < 0.05$). Yeni-AF grubunda PESI skoru daha yüksekti (93±23 ve 75±17; $p < 0.001$). LVEDD, LAD, ürik asit, bilirubin, albumin, troponin değerleri ve PESI skoru Yeni-AF için tek değişkenli öngörücüler olarak bulundu.

Sonuç: Çalışmamızda yüksek PESI skoru yeni gelişen AF ile ilişkili bulundu. APE hastalarında, 82.50'den büyük PESI skoru yeni-AF öngörücüsü olarak kullanılabilir.

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Acute pulmonary embolism (APE) is one of the most serious cardiovascular diseases encountered in the emergency department and is associated with a mortality rate of 15% to 20%.[1] The Pulmonary Embolism Severity Index (PESI) is an accepted clinical prognostic score to predict survival in patients with APE.[2] Jiménez et al.[3] developed a simplified version of the PESI (sPESI), based on a study in which they reported that APE patients classified with a low SPESI score had an in-hospital mortality of 1.1% compared to 8.9% in those classified with a high sPESI score.

Atrial fibrillation (AF) is the most commonly sustained cardiac arrhythmia, occurring in 3% of the general population aged 40 years and older.[4] Many clinical conditions, including aging, hypertension, heart failure, valvular heart diseases, cardiomyopathies, congenital cardiac disease, coronary artery disease, thyroid disorders, obesity, diabetes mellitus, chronic obstructive pulmonary disease, sleep apnea, and chronic renal disease, are associated with AF, which in turn may lead to stroke or other thromboembolic events, heart failure, cardiovascular hospitalization, impaired quality of life, or reduced exercise capacity, as well as an increased mortality rate.[5]

The potential effect of acute or chronic thromboembolism on the occurrence of AF is controversial and remains unclear. In recent pulmonary thromboembolism guidelines, the relationship between AF and pulmonary thromboembolism (PTE) and the development of AF in patients with PTE are insufficiently addressed. The aim of this study was to evaluate the effect of the severity of acute PTE on the occurrence of AF.

METHODS

Patients admitted to our clinic with suspected APE between May 2012 and December 2015 and had a confirmed diagnosis of APE based on a pulmonary computed tomography angiography or V/Q scintigraphy were enrolled to the study. The following conditions were excluded: hypertension, structural heart disease, hepatic or renal dysfunction, chronic obstructive pulmonary disease, thyroid dysfunction, diabetes mellitus, and sleep apnea. In addition, none of the participants had any history of inflammatory or infectious disease, recent (within the previous 4 weeks) trauma or surgery, or treatment with nonsteroidal anti-inflammatory or corticosteroids drugs. Individuals with missing data in their patient files were also excluded. PESI and sPESI scores were calculated for every patient. According to hospital procedure, echocardiography controls are performed every 3 months to determine any development of chronic thromboembolic pulmonary hypertension. An electrocardiogram (ECG) examination was performed before echocardiography in all patients. SPESI and PESI scores were calculated from the first medical contact data with emergency services.

Abbreviations:

AF	*Atrial fibrillation*
APE	*Acute pulmonary embolism*
ECG	*Electrocardiogram*
LAD	*Left atrial diameter*
LV	*Left ventricle*
LVEDD	*Left ventricle end diastolic diameter*
LVEF	*Left ventricle ejection fraction*
PASP	*Pulmonary artery systolic pressure*
PE	*Pulmonary embolism*
PESI	*Pulmonary Embolism Severity Index*
PTE	*Pulmonary thromboembolism*
ROC	*Receiver operating characteristic*
sPESI	*Simplified Pulmonary Embolism Severity Index*
TAPSE	*tricuspid annular plane systolic excursion*

This retrospective study was conducted in compliance with the principles outlined in the Declaration of Helsinki and the use of data for this retrospective study was allowed by the institutional committee.

A diagnosis of AF and the subsequent treatment were performed according to the European Society of Cardiology guidelines.[5] An episode (defined with irregular RR intervals and distinct P waves) lasting at least 30 seconds on ECG was accepted for the diagnosis of AF. Atrial flutter was classified as part of the AF spectrum for the purpose of the current study. For the diagnosis of new-onset AF, patients who had been treated for AF before the APE diagnosis, and those with hypertension, structural heart disease, a prosthetic heart valve, hepatic or renal dysfunction, chronic obstructive pulmonary disease, thyroid dysfunction, diabetes mellitus, or sleep apnea were excluded. Left ventricle (LV) internal diameters, LV ejection fraction (LVEF; modified Simpson method), left atrial diameter (LAD), tricuspid annular plane systolic excursion (TAPSE), and pulmonary artery systolic pressure (PASP) were measured according to the guidelines of the American Society of Echocardiography.[6] Echocardiography examinations were performed within the first 3 days after diagnosis.

Continuous variables were expressed as mean±SD

and categorical variables were defined as percentages (%). A chi-square test was used for categorical variables. The data were tested for normal distribution using the Kolmogorov-Smirnov test. Continuous variables that were normally distributed were analyzed with an independent t-test, and continuous variables with non-normal distribution were analyzed with the Mann-Whitney U test. The Pearson correlation test was used for correlation analysis. Multiple logistic regression analysis was used with all of the prespecified factors with a p <0.25 in the univariate analysis. Statistical significance in the multivariate analysis was accepted at p <0.05. IBM SPSS Statistics for Windows, Version 20.0 (IBM Corp., Armonk, NY, USA) was used for all of the statistical calculations. Statistical significance was defined as a p value less than 0.05.

RESULTS

After exclusion of patients not meeting the required criteria, 42 new-onset AF and 107 non-AF patients were included (defined as New AF group and Non-AF group) in the study. The mean follow-up period was 823.6±278.2 days. The time interval between the diagnosis of APE and the diagnosis of AF was 647.7±341.4 days. In the New AF group, the mean age was 63±4 years, and 54.8% were male. In the Non-AF group, the mean age was 59±12 years, and 51.4% were male (Table 1). No significant differences were observed between the 2 groups regarding age, gender, systolic and diastolic blood pressure, heart rate, or level of fasting glucose or serum creatinine. Uric acid, C-reactive protein and erythrocyte sedimentation rate (p=0.087; p=0.085; p=0.058, respectively) levels were higher in the New-AF patients, but this difference was not statistically significant (Table 2). New-AF patients had a larger left ventricle end diastolic diameter (LVEDD) and LAD. There was no statistically significant difference between the 2 groups in LVEF, TAPSE, or PASP (p=0.090; p=0.089; p=0.860, respectively).

SPESI scores were higher in the New-AF group, and this difference was statistically significant (p=0.010) (Table 3). Regression analysis indicated that LVEDD, LAD, uric acid, albumin, total bilirubin, and troponin, as well as the PESI and sPESI scores, were univariate predictors of new onset-AF (Table 4). Multivariate stepwise logistic regression analysis showed that AF was associated with the PESI score, LAD, LVEDD, and levels of alkaline phosphatase and troponin (Table 4). The PESI score predicted AF with a sensitivity of 69.0% and specificity of 69.2% in receiver operating characteristic (ROC) curve analysis using a cut-off level of 82.50 (ROC area under curve: 0.721, 95% CI: 0.623–0.818; p<0.001) (Fig.1).

DISCUSSION

In this study, we found that the PESI score was as-

Table 1. Major demographic and echocardiographic features of the study population

Variable	New-atrial fibrillation group (n=42)	Non-atrial fibrillation group (n=107)	*p*
Age (years)	62.79±14.05	59.45±12.33	0.156
Gender			
Male	23 (54.8%)	55 (51.4%)	0.712
Female	19 (45.2%)	52 (48.6%)	
Systolic blood pressure (mm Hg)	109.64±15.75	112.48±13.86	0.282
Diastolic blood pressure (mm Hg)	71.67±10.63	71.87±7.38	0.895
Left ventricle ejection fraction (%)	56.10±6.43	58.43±7.88	0.090
Left ventricle end diastolic diameter (mm)	50.88±2.37	49.15±3.07	<0.001
Left atrial diameter (mm)	40.21±3.09	37.83±2.92	<0.001
Pulmonary artery systolic pressure (mm Hg)	35.33±10.63	35.03±8.97	0.860
Tricuspid annular plane systolic excursion (mm)	21.48±2.87	22.26±2.37	0.089
Pulmonary embolism severity index	93.02±23.41	75.24±17.20	<0.001

Table 2. Biochemical and hemogram parameters of the study population

Variables	New-atrial fibrillation (n=42) Mean±SD	Non-atrial fibrillation (n=107) Mean±SD	*p*
Glucose (mg/dL)	102.57±13.96	106.24±16.67	0.208
Creatinine (mg/dL)	0.93±0.47	0.88±0.23	0.361
Uric acid (mg/dL)	5.69±2.34	5.08±1.76	0.087
Albumin (g/dL)	3.76±0.52	4.01±0.45	0.006
Total bilirubin (mg/dL)	0.89±0.32	0.67±0.29	0.014
Aspartate aminotransferase (U/L)	38.02±15.15	22.50±10.26	0.065
Alanine aminotransferase (U/L)	33.57±25.72	23.05±12.89	0.068
Alkaline phosphatase (U/L)	91.36±32.64	71.88±19.64	0.003
Gamma-glutamyl transpeptidase (U/L)	46.21± 31.62	42.93±40.34	0.649
Lactate dehydrogenase (U/L)	239.10±164.46	234.38±65.20	0.800
Sodium (mmol/L)	138.09±3.24	138.84±3.61	0.245
Potassium (mmol/L)	4.29±0.55	4.31±0.43	0.757
Calcium (mmol/L)	9.17±0.48	9.21±0.43	0.653
C-reactive protein (mg/dL)	6.73±4.82	4.09±3.71	0.085
Erythrocyte sedimentation rate (mm/h)	40.95±23.61	33.9±22.06	0.058
D-dimer (ng/mL)	3110.38±2918.58	3797.94±3256.85	0.466
Troponin (pg/mL)	88.98±151.01	27.02±31.39	0.000
White blood cell count (10e3/μL)	9200±3615	9007± 3501	0.765
Neutrophil (10e3/μL)	6648±3434	6369±3138	0.636
Monocyte (10e3/μL)	604±344	546±270	0.281
Lymphocyte (10e3/μL)	1744±706	1983±974	0.151
Platelet (10e3/μL)	213±90	237±83	0.706
Hgb (g/dL)	13.58±1.95	13.67±1.37	0.756
Red cell distribution width (%)	16.01±2.79	16.15±2.71	0.775
Mean platelet volume (fL)	7.57±1.04	7.56±1.19	0.962

SD: Standard deviation.

Table 3. The relationship between AF and sPESI score

Variable	New-AF group (n=42)	Non-AF group (n=107)	*p*
sPESI <1	17 (40.5%)	68 (63.6%)	0.010
sPESI ≥1	25 (59.5%)	39 (36.4%)	

AF: Atrial fibrillation; sPESI: Simplified Pulmonary Embolism Severity Index.

sociated with new onset AF (New-AF group). A greater PESI score pointed to more serious clinical status in APE, and serious clinical presentation with pulmonary embolism (PE) was associated with new-onset AF. The univariate regression results showed that LVEDD, LAD, and uric acid level were predictors of new AF in the study patients. Impaired LV or left atrial function, and changes in the geometry of the LV and left atrium can trigger AF. This may be why patients with PE develop AF: PE may directly lead to cardiac dysfunctions that trigger AF. The results reported by Hald et al.[7] support this potential mechanism. An increased right atrial pressure may trigger the right atrial remodeling process, leading to the appearance of atrial arrhythmias. However, the findings of our study revealed no significant difference between PASP and TAPSE values between the 2 groups, which does not fully support the hypothesis of atrial arrhythmia due to right ventricular and atrial

Table 4. Univariate and multivariate regression analysis for the predictors of new-AF in acute pulmonary embolism

	Univariate regression analysis			Multivariate regression analysis		
Variable	Odds ratio	CI 95%	*p*	Odds ratio	CI 95%	*p*
PESI	1.047	1.024–1.069	<0.001	1.072	1.021–1.125	0.005
sPESI	2.564	1.234–5.327	0.012	3.054	0.617–15.122	0.171
Age	1.021	0.992–1.051	0.156	0.950	0.888–1.016	0.134
Gender	0.874	0.427–1.788	0.712			
Systolic blood pressure	0.986	0.960–1.012	0.281			
Diastolic blood pressure	0.997	0.995–1.041	0.894			
Left ventricle ejection fraction	0.962	0.920–1.007	0.095	1.075	0.948–1.220	0.260
Left ventricle end diastolic diameter	1.220	1.073–1.386	0.002	1.372	1.003–1.878	0.048
Left atrial diameter	1.339	1.155–1.552	<0.001	1.481	1.175–1.866	0.001
PASP	1.003	0.966–1.042	0.859			
TAPSE	0.883	0.765–1.020	0.091	1.298	0.972–1.733	0.077
Glucose	1.009	0.997–1.021	0.130	1.019	1.000–1.038	0.051
Creatinine	1.619	0.559–4.688	0.374			
Uric acid	0.173	0.974–1.394	<0.001	1.190	0.789–1.795	0.407
Albumin	0.361	0.170–0.769	0.008	1.009	0.160–6.356	0.993
T.bilirubin	3.097	1.032–9.290	0.044	3.193	.395–25.783	0.276
Alkaline phosphatase	1.033	1.015–1.052	<0.001	1.071	1.027–1.117	0.001
Aspartate aminotransferase	1.020	0.989–1.053	0.213	1.054	0.943–1.178	0.357
Alanine aminotransferase	1.013	0.993–1.033	0.205	0.968	0.892–1.052	0.446
Gamma-glutamyl transpeptidase	1.002	0.993–1.011	0.648			
Lactate dehydrogenase	1.000	0.997–1.004	0.799			
Sodium	.942	0.853–1.041	0.245	1.152	0.895–1.482	0.272
Potassium	.885	0.409–1.911	0.755			
Calcium	.832	0.375–1.844	0.650			
C-reactive protein	1.044	0.998–1.092	0.060	0.963	0.873–1.062	0.452
Erythrocyte sedimentation rate	1.015	0.999–1.030	0.063	1.014	0.984–1.046	0.356
White blood cell count	1.016	0.918–1.124	0.764			
Neutrophil	1.027	0.920–1.146	0.633			
Monocyte	1.943	0.582–6.488	0.280			
Lymphocyte	0.720	0.460–1.126	0.150	0.426	0.181–1.002	0.051
Hgb	0.964	0.766–1.212	0.754			
Red cell distribution width	0.980	0.857–1.121	0.773			
Platelet	1.001	0.997–1.005	0.704			
Mean platelet volume	1.008	0.739–1.374	0.962			
D-dimer	1.000	1.000–1.000	0.466			
Troponin	1.010	1.000–1.018	0.007	1.017	1.002–1.032	0.026

AF: Atrial fibrillation; PASP: Pulmonary artery systolic pressure; sPESI: Simplfied Pulmonary Embolism Severity Index; TAPSE: Tricuspid annular systolic excursion; CI: Confidence interval.

dysfunction. In our study, a dilated LA and LV were associated with new-onset AF. Zhang et al.[8] found an association between serum uric acid level and AF, and Kawasoe et al.[9] also found that uric acid level

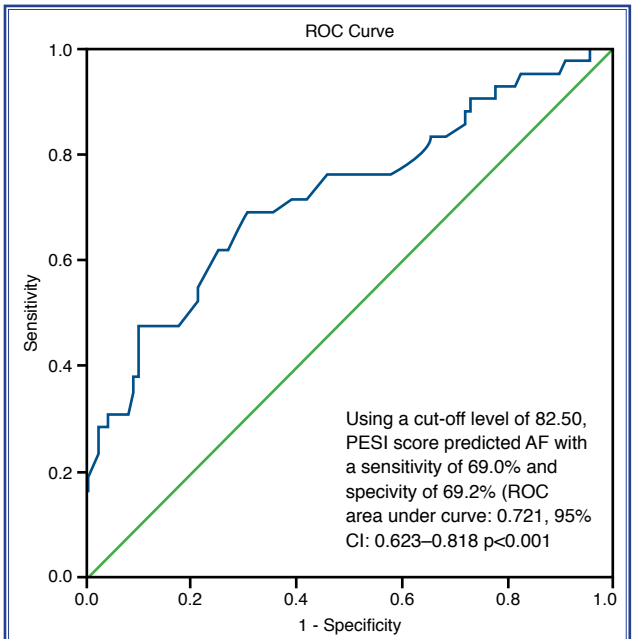

Figure 1. Receiver operating characteristic curve analysis. AF: Atrial fibrillation; CI: Confidence index; PESI: Pulmonary Embolism Severity Index; ROC: Receiver operating characteristic.

was significantly associated with AF, independent of other cardiovascular risk factors in 285,882 patients. The result of Kawasoe's study support our findings about a relationship between uric acid and new-onset AF. Uric acid level may be associated with oxidative stress and may be involved in the pathophysiology of AF. In one study, Ng et al.[10] found a prevalence of AF of 13.5% (126/935), but this study was not designed to analyze the prevalence and incidence of new-onset AF in patients with PE. Ng's study and our research determined a similar mean length of time from PE to subsequent AF (3.4±2.9 years; 647.7±341.4 days, respectively). In our study, since patients who had comorbidities that occurred between the time of diagnosis of APE and the diagnosis of AF were excluded, we can accept that new-onset AF was associated with APE. The biomarkers of myocardial injury (troponin) and coagulation activity (D-dimer) have been found to be associated with underlying pathophysiology, the effectiveness of treatment methods, and clinical outcomes in patients with AF.[11] In our study, troponin level was associated with new-onset AF but D-dimer level was not.

AF has been independently associated with an increased risk of all-cause mortality. In a review and meta-analysis, the results indicated that AF is a predictor of in-hospital mortality and 6-month mortality.[12] In our study, the mortality rate of the patients could not be fully assessed because follow-up procedures were performed by another clinic (data were collected from patients of pulmonary clinics).

There are a number of limitations to our study. As it was retrospective in design, data loss in patient follow-up and incomplete information about symptoms may have affected the study results. Obesity status and genetic predisposition of the study population could not be defined. Furthermore, silent AF could not be excluded. Some patients in the non-AF group may have had silent-AF, and these patients could not be identified and excluded. Periods of silent AF may also have affected the time between PTE diagnosis and AF diagnosis in the AF group of patients.

In conclusion, serious clinical status in APE (with a higher sPESI score) was associated with new-onset AF in our study. A PESI score greater than 82.50 may be useful to predict new-onset AF in these patients. The screening for AF after APE may be important to decrease morbidity and mortality specifically related to AF.

Ethics Committee Approval: This retrospective study was approved by the institutional EPK Committee of Atatürk Chest Disease and Thorasic Surgery Training and Research Hospital (approval date: 16.12.2015 approval no.: 512).

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Keywords: Acute pulmonary embolism; atrial fibrillation; Pulmonary Embolism Severity Index; Simplified Pulmonary Embolism Severity Index.

Anahtar sözcükler: Akut pulmoner emboli; atriyal fibrilasyon; pulmoner emboli ciddiyet indeksi; basitleştirilmiş pulmoner emboli ciddiyet indeksi skoru.