Evaluation of Interatrial Block in Patients Presented with Acute Pulmonary Embolism

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ABSTRACT

Introduction: Interatrial block (IAB), which is defined as a conduction delay between the right and left atrium, is characterized by the prolongation of P wave on the electrocardiography (ECG). In this study, we aimed to investigate the relationship between the presence of IAB and acute pulmonary embolism (APE) in patients admitted to emergency department with a preliminary diagnosis of APE.

Patients and Methods: In this retrospective case-control study, a total of 82 patients with a preliminary diagnosis of APE were enrolled. Of these patients, 42 patients were diagnosed with APE via pulmonary computed tomographic angiography. In all patients, the ECGs were recorded on admission.

Results: Our study findings revealed that P wave dispersion, P wave duration of ≥ 120 ms, and notched P wave were significantly higher in patients with APE ($p< 0.05$ for all). Also, elevated heart rate and lower systolic blood pressure were found in patients with APE ($p= 0.022$ and $p= 0.043$, respectively). In multivariable logistic regression analysis, P wave duration of ≥ 120 ms was found to be an independent predictor of APE (OR: 3.958; 95% CI: 1.095-14.308; $p= 0.036$). In a receiver operating characteristics curve analysis, IAB predicted the APE with a sensitivity of 40.5% and a specificity of 85%.

Conclusion: Prolonged P wave duration was observed more frequently in patients with APE. The study findings showed that IAB may be an important predictor of APE in patients with a preliminary diagnosis of APE.

Key Words: Interatrial block; pulmonary embolism; electrocardiography.

Akut Pulmoner Emboli ile Başvuran Hastalarda İnteratriyal Blok Varlığının Değerlendirilmesi

ÖZ

Giriş: Sağ ve sol atriyum arasında bir iletim gecikmesi olarak tanımlanan interatriyal blok (IAB), elektrokardiyografi (EKG)'deki P dalgasının uzaması ile karakterize edilir. Bu çalışmada, acil servise başvuran hastalarda akut pulmoner emboli (APE) ön tanısıyla IAB varlığı ile APE arasındaki ilişkinin araştırılması amaçlanmıştır.

Hastalar ve Yöntem: Bu retrospektif olgu kontrol çalışmasına, APE ön tanısı ile acil servisimize başvuran toplam 82 hasta dahil edilmiştir. Bu hastaların 42'sine APE tanısı pulmoner bilgisayarlı tomografik anjiyografi ile konulmuştur. Tüm hastaların başvuru sırasındaki EKG kayıtları alınmıştır.

Bulgular: Çalışma bulgularımız P dalga dispersiyonunun, P dalga süresinin ≥ 120 ms ve çentikli P dalgasının APE hastalarında anlamlı olarak yüksek olduğunu göstermiştir ($p< 0.05$). Ayrıca APE tanısı alan hastalarda kalp atım hızı daha yüksek ve sistolik kan basıncı daha düşük tespit edilmiştir (sırasıyla $p= 0.022$ ve $p= 0.043$). Çok değişkenli lojistik regresyon analizinde, P dalga süresinin ≥ 120 ms olması APE'nin bağımsız bir belirleyicisi olduğu bulunmuştur (OR: 3.958, %95 CI: 1.095-14.308, $p= 0.036$). Yapılan ROC eğri analizinde, IAB, APE'yi %40.5 duyarlılık ve %85 özgüllük ile öngörmüştür.

Sonuç: Uzamış P dalga süresi, APE tespit edilen hastalarda daha sık gözlenmiştir. Çalışma bulguları, IAB'nin APE ön tanısı alan hastalarda önemli bir APE belirleyicisi olabileceğini göstermiştir.

Anahtar Kelimeler: İnteratriyal blok; pulmoner embolizm; elektrokardiyografi.

INTRODUCTION

Acute pulmonary embolism (APE) is one of the life-threatening disease that may cause considerable morbidity and mortality. In addition, APE is a challenging emergency condition because the clinical presentation in patients with APE may range from mild chest pain to shock[1-3]. Even though the definitive diagnosis of APE is based on computerized tomographic pulmonary angiography (CTPA), some electrocardiographic (ECG) findings such as $S_1Q_3T_3$

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pattern, negative T-wave in precordial leads and atrial arrhythmias have been shown to have high specificity for APE[4-6].

Interatrial block (IAB), which is a conduction delay between right and left atrium, was defined as a positive P wave duration longer than 120 ms on the surface ECG, or manifested as P wave duration longer than 120 ms plus biphasic (±) morphology in leads II, III, and aVF[7]. Some previous studies concluded that IAB was related with the occurance of atrial fibrillation in acute coronary syndrome patients[8]. However, it has been unknown whether IAB is a marker of APE in patients presented with signs and symptoms of APE. Therefore, in our study, we aimed to evaluate the potential diagnostic utility of IAB in patients with a preliminary diagnosis of APE.

PATIENTS and METHODS

Study Patients

In this retrospective case-control study, the patients who presented to emergency department with signs and symptoms of APE from January 2016 to June 2018 and underwent CTPA were reviewed. The patients who had end-stage liver and renal disease, active infection, acute coronary syndrome, chronic inflammatory disease, and coagulopathy were not included in this study. In addition, the patients whose ECG morphology indicating atrial fibrillation or pace rhythm were also excluded from the study. After evaluation regarding with exclusion criteria, 82 patients were enrolled in our study. Of these patients, APE diagnosis was confirmed in 42 patients by CTPA. Control group consisted by remaining 40 patients with a final diagnosis other than APE. Among these patients, 10 patients had been treated as acute heart failure, four patients as pneumonia, and 16 patients as acute asthma or exacerbation of chronic obstructive pulmonary disease. In the remaining 10 patients, there was no detected organic pathology. All patients who had a diagnosis of APE were treated with the standard medical therapy in accordance with the recent guidelines. Our study protocol was confirmed by the local ethics committee of our hospital in accordance with the principle of the Declaration of Helsinki. Due to the retrospective design of the study, an informed consent was not needed.

Electrocardiographic Analysis

The standard 12-lead ECGs were achieved on a paper with speed of 25 mm/s, amplitude of 10 mm/mv, and a filter range between 0.5 to 150 Hz from whole patients on admission. The beginning of the P wave was defined as the initially detected upward or downward deflection calculated from baseline. The returning point of the P wave to the baseline was defined as the P wave offset. IAB was defined as the duration of P wave longer than 120 ms with or without presence of notching. The P wave maximum was calculated in each leads, and the algebraic difference between the two was accepted as the P wave dispersion. The ECGs were analyzed by two independent cardiologists who were blind to the patients' clinical data.

Echocardiographic and CTPA Examination

All echocardiographic examinations were obtained by an experienced cardiologist using an ultrasound machine (Vivid 7, GE Healthcare, USA) within 24 hours during hospitalization. The peak systolic pressure of pulmonary artery was calculated using the simplified Bernoulli equation. The left ventricular ejection fraction (LVEF) was calculated in accordance with the Simpson method.

In the present study, APE was diagnosed by CTPA with a standard APE protocol (which was a contrast material volume: 135 mL, contrast material injection rate: 4 mL/sec, field of view: 35 cm and section thickness: 3 mm). Two radiology specialists confirmed the diagnosis of APE if there was thrombus at the main pulmonary artery or its major branches.

Statistical Analysis

Categorical variables were stated as percentage (%), whereas continuous variables were stated as mean ± standard deviation or median. Kolmogorov-Smirnov test was used to test the normality distribution of continuous variables. Chi-square or Fisher exact test were used for comparison of categorical data. Correlation of continuous variables was analyzed by Pearson correlation test. Correlation of non-continuous variables was analyzed by Spearman test. Student t-test or Mann-Whitney U test was used to compare continuous variables between the two groups. In order to find the independent predictors of APE, a multivariate logistic regression analysis was performed. A 2-sided p value of < 0.05 was accepted as significant. The IAB value which predicted the best sensitivity and specificity of APE was calculated with receiver operating characteristic (ROC) curve analysis. The effect size (Cohen's d) and power value ($1–\beta$) for IAB, which was compared between patients with and without APE, were measured via G*Power software. The power value and effect size were 0.92 and 0.77, respectively. Statistical analysis were performed with SPSS version 21 (IBM Corp., Armonk, NY).

RESULTS

A total of 82 patients [43 females (52.4%), mean age 56 ± 18 years] were included in our study. The baseline clinical and laboratory characteristics of all patients are shown in Table 1. The level of heart rate, respiratory rate, systolic

Table 1. Baseline demographic characteristic and laboratory findings of all patients and patients with or without PTE

	All patients (n= 82)	PTE (-) (n= 40)	PTE (+) (n= 42)	p value
Age, years	56 ± 18	56 ± 17	56 ± 19	0.714
Female gender (%)	43 (52.4)	20 (50)	23 (54.8)	0.666
History				
Diabetes mellitus, n (%)	12 (14.6)	6 (15)	6 (14.6)	0.927
Hypertension, n (%)	20 (24.4)	7 (17.5)	13 (31)	0.156
COPD, n (%)	10 (12.2)	4 (10)	6 (14.3)	0.553
Coronary artery disease, n (%)	12 (14.6)	6 (15)	6 (14.3)	0.927
Chronic heart failure, n (%)	6 (7.3)	4 (10)	2 (4.8)	0.363
History of PTE, n (%)	2 (2.4)	1 (2.5)	1 (2.4)	0.972
Malignancy, n (%)	4 (4.9)	0 (0)	4 (9.5)	0.116
Deep vein thrombosis, n (%)	4 (4.9)	2 (5)	2 (4.8)	0.96
Smoking, n (%)	38 (46.3)	19 (47.5)	19 (45.2)	0.837
On admission				
Surgery or immobilization within past four weeks, n (%)	17 (20.7)	6 (15)	11 (26.2)	0.211
Hemoptysis, n (%)	8 (9.8)	3 (7.5)	5 (11.9)	0.503
DVT symptoms, n (%)	17 (20.7)	6 (15)	11 (26.2)	0.211
Heart rate (rate/min)	91 ± 22	87 ± 24	95 ± 19	0.022
Systolic blood pressure (mmHg)	126 ± 22	131 ± 23	121 ± 20	0.043
Body temperature (°C)	36.6 ± 0.6	36.6 ± 0.6	36.6 ± 0.6	0.985
Respiratory rate (rate/min)	22 ± 5	20 ± 4	23 ± 5	0.014
O_2 saturation (%)	93 (86-96)	94 (91-96)	91 (82-96)	0.011
Laboratory findings				
Glucose (mg/dL)	118 (101-146)	114 (95-153)	124 (106-142)	0.489
Creatinine (mg/dL)	0.8 ± 0.2	0.8 ± 0.2	0.8 ± 0.2	0.568
BUN (mmol/L)	36 (28-51)	36 (27-48)	35 (29-53)	0.849
ALT (U/L)	17 (13-30)	16 (13-31)	18 (13-30)	0.86
Potassium (mEq/L)	4.3 ± 0.5	4.3 ± 0.5	4.3 ± 0.5	0.97
Sodium (mEq/L)	137 ± 4.3	136 ± 4.6	138 ± 3.8	0.034
Calcium (mEq/L)	8.9 ± 0.6	8.9 ± 0.5	8.8 ± 0.6	0.173
WBC count ($x10^3/mm^3$)	9.5 ± 4.1	9.2 ± 4.6	9.8 ± 3.5	0.202
Hemoglobin (mg/dL)	13.8 ± 2.2	13.7 ± 1.9	13.8 ± 2.4	0.933
Platelets ($x10^3/mm^3$)	226 (173-298)	231 (195-278)	211 (165-312)	0.639
Positive D-Dimer test, n (%)	72 (87.8)	33 (82.5)	39 (92.9)	0.152
Positive troponin value, n (%)	32 (39)	11 (27.4)	21 (50)	0.037
Wells score	3 (3-5)	3 (3-4.5)	3 (3-6)	0.185
Geneva score	5 (3-8)	4 (1-6.5)	6 (4-8)	0.016

Continuous variables are presented as mean ± SD or median; nominal variables presented as frequency. COPD: Chronic obstructive pulmonary disease, PTE: Pulmonary thromboembolism, DVT: Deep vein thrombosis, BUN: Blood urea nitrogen, ALT: Alanine aminotransferase, WBC: White blood cell.

blood pressure, oxygen saturation, sodium level, and positive troponin level were significantly different between the patients with and without APE (p< 0.05, for all). Comparing the echocardiographic findings showed that patients with APE had significantly higher prevalence of more than mild degree of tricuspid regurgitation, increased systolic pulmonary artery pressure, right ventricle dilatation, and increased right ventricle/left ventricle ratio (p< 0.05, for all) (Table 2). In terms of electrocardiographic parameters, the frequency of; increased P wave duration, P wave dispersion, P wave duration of > 120 ms, and P wave notching were significantly higher in patients with APE compared to controls (p< 0.05, for all). However; there was no significant difference in terms of negative T wave on the precordial leads, P wave maximum duration, and presence of fragmented QRS (p> 0.05, for all).

After the exclusion of patients who had chronic obstructive lung disease and chronic heart failure, electrocardiographic findings of patients with and without APE were compared and showed in Table 3. Heart rate, complete or incomplete right bundle brunch block, P wave duration, P wave dispersion, P wave duration of ≥ 120 ms, and P wave notching were remained different significantly between patients with and without APE (p< 0.05, for all).

In univariable regression analysis; right bundle branch block, heart rate, P wave dispersion, and P wave duration of ≥ 120 ms were found to be correlated with APE (Table 4). These parameters were entered to the multivariable logistic regression analysis. According to these analysis; heart rate (OR: 1.052, 95% CI: 1.021-1.084, p= 0.001) and P wave duration of ≥ 120 ms (OR: 3.958, 95% CI: 1.095-14.308, p= 0.036) were found to be independently associated with APE in the study population. ROC curve analysis showed that, IAB may predict the APE with sensitivity and specificity of 40.5% and 85% respectively.

Table 2. Comparison of echocardiographic and electrocardiographic findings of all patients and patients with or without PTE

	All patients (n= 82)	PTE (-) (n= 40)	PTE (+) (n= 42)	p value
Echocardiographic findings				
LVEF (%)	58 ± 8	58 ± 8	58 ± 7	0.809
TR more than mild degree, n (%)	34 (41.5)	9 (22.5)	25 (59.5)	0.001
Pulmonary artery pressure (mmHg)	39 ± 12	29 ± 5.5	48 ± 9.6	< 0.001
Right ventricle dilatation, n (%)	36 (43.9)	8 (20)	28 (66.7)	< 0.001
Right ventricle (mm)	37 ± 7.7	33 ± 5.5	41 ± 8.1	< 0.001
Left ventricle (mm)	38 ± 6.4	38 ± 5.9	37 ± 6.8	0.264
Right ventricle/Left ventricle ratio	1.0 ± 0.2	0.8 ± 0.2	1.1 ± 0.2	< 0.001
Electrocardiographic findings				
Right axis deviation, n (%)	10 (12.2)	2 (5)	8 (19)	0.089
Complete or incomplete RBBB, n (%)	18 (22)	5 (12.5)	13 (31)	0.044
Fragmented QRS, n (%)	20 (24.4)	9 (22.5)	11 (26.2)	0.697
T wave inversion on precordial leads, n (%)	33 (40.2)	14 (35)	19 (45.2)	0.345
ST segment depression, n (%)	16 (19.5)	7 (17.5)	9 (21.4)	0.654
ST segment elevation, n (%)	33 (40.2)	18 (45)	15 (35.7)	0.391
$S_1Q_3T_3$, n (%)	9 (11)	2 (5)	7 (16.7)	0.091
P wave duration (ms)	112 ± 48	100 ± 45	122 ± 52	0.004
P wave dispersion (ms)	106 ± 40	100 ± 34	116 ± 45	0.018
P wave duration of ≥ 120 ms, n (%)	23 (28)	6 (15)	17 (40.5)	0.001
P wave notching, n (%)	20 (24.4)	5 (12.5)	15 (35.7)	0.014
P wave maximum amplitude (mm)	1.39 ± 0.43	1.34 ± 0.34	1.43 ± 0.5	0.299

PTE: Pulmonary thromboembolism, LVEF: Left ventricle ejection fraction, TR: Tricuspid regurgitation, RBBB: Right bundle branch block.

Table 3. Comparison of electrocardiographic findings after exclusion of chronic obstructive lung disease

	All patients (n= 61)	PTE (-) (n= 31)	PTE (+) (n= 30)	p value
Heart rate (beat/min)	87 ± 20	79 ± 21	96 ± 16	0.001
Right axis deviation, n (%)	7 (11.5)	1 (3.2)	6 (20)	0.053
Complete or incomplete RBBB, n (%)	15 (24.6)	4 (12.9)	11 (36.7)	0.04
Fragmented QRS, n (%)	15 (24.6)	6 (19.4)	9 (30)	0.334
T wave inversion on precordial leads, n (%)	19 (31.1)	7 (22.6)	12 (40)	0.142
ST segment depression, n (%)	13 (21.3)	5 (16.1)	8 (26.7)	0.315
ST segment elevation, n (%)	25 (41)	14 (45.2)	11 (36.7)	0.5
S1Q3T3, n (%)	4 (6.6)	1 (3.2)	3 (10)	0.354
P wave duration (ms)	117 ± 28	105 ± 22	129 ± 28	< 0.001
P wave dispersion (ms)	110 ± 36	93 ± 30	126 ± 34	< 0.001
P wave duration of ≥ 120 ms, n (%)	16 (25.8)	3 (9.7)	13 (41.9)	0.004
P wave notching, n (%)	15 (24.2)	4 (12.9)	11 (35.5)	0.016
P wave maximum amplitude (mm)	1.38 ± 0.41	1.31 ± 0.33	1.44 ± 0.48	0.201

PTE: Pulmonary thromboembolism, RBBB: Right bundle branch block.

Table 4. Independent electrocardiographic predictors of pulmonary thromboembolism

	Univariable analysis			Multivariable analysis		
	OR	95% CI	p value	OR	95% CI	p value
Right bundle branch block	3.138	1.001-9.839	0.050	-	-	-
Heart rate	1.042	1.015-1.069	0.002	1.052	1.021-1.084	0.001
P wave dispersion	2.575	0.999-6.640	0.050	-	-	-
P wave duration of ≥ 120 ms	3.853	1.329-11.171	0.013	3.958	1.095-14.308	0.036

OR: Odds ratio, CI: Confidence interval.

DISCUSSION

Our study findings showed that the presence of IAB, namely P wave duration longer than 120 ms on the ECG, were significantly associated with APE, and it was found to be an independent predictor of APE. To the best of our knowledge, this was the first study demonstrating the potential effect of IAB for the presence of APE in patients with a preliminary diagnosis of APE.

The patients with APE may present with different clinical presentations, which may range from chest pain to cardiogenic shock[1,2]. Thus, it is a challenging disease based on the clinical presentations. Therefore, CTPA is performed in order to confirm or exclude the presence of APE. Similarly, the diagnosis of APE in our study was confirmed by CTPA in all patients.

In addition to being one of the first tests performed to the patients complaining of chest pain and shortness of breath, the ECG may aid in the diagnosis of APE when applied to the entire clinical situation. In previous studies, several ECG findings such as the $S_1Q_3T_3$ pattern, ST segment deviation on the precordial or inferior leads, T wave inversion on the precordial leads, and right bundle branch block have been shown as a marker for a main pulmonary trunk embolus[9,10]. In our study, we did not observe any significant difference in terms of $S_1Q_3T_3$ pattern, ST segment deviation on the precordial or inferior leads, and T wave inversion on the precordial leads between the groups even if we excluded the patients with chronic obstructive lung disease and chronic heart failure. These findings might be due to relatively small sample size of the study or low sensitivity of the above mentioned ECG parameters.

As possible explanations of the study findings, we thought that these ECG changes in APE are probably due to occlusion of the pulmonary artery by a massive embolus, which cause a rapid pressure overload to the right ventricle and the right atrium[11,12]. This rapid increase of pressure overload may lead to the dilatation and dysfunction of the right ventricle, thus resulting in decreased of blood flow through the right ventricle outflow tract, thereby reducing the left ventricle preload. Moreover, in an experimental study, Gold et al. demonstrated that acute pulmonary hypertension may cause right ventricle subendocardial ischemia, which may explain the disruption of the right-sided cardiac conduction and repolarization[13]. Furthermore, we hypothesized that decreased blood flow to the lung due to embolus may enhance the release of vasoconstrictive mediators such as histamine and catecholamines[10]. As a result, this release of vasoconstrictive mediators may cause ischemia of the right ventricle and right atrium as well as hypoxia of the Bachmann's bundle which is the equivalent of His-Purkinje system between the atria. Thus, the ischemic media can eventually result in deceleration of the conduction pathway of the right side of the heart, which may manifest itself as the prolongation of P wave on the ECG. In addition, P wave prolongation may reflect inhomogeneous atrial depolarization in response to various electrical and structural remodeling[8,14].

IAB, which is a newly introduced ECG parameter, is interpreted as the conduction time prolongation between the left and right atrium due to an impulse delay or blockage that is most often but not exclusively in the Bachmann's bundle. Several previous studies stated out that IAB had an association with the development of new-onset atrial fibrillation in patients with coronary artery disease and peripheral vascular disease[8,15,16]. Moreover, Senen et al. revealed that the left ventricular systolic dysfunction could lead to significant cardiac hemodynamic changes, which can alter the left atrium electrical properties resulting in a high prevalence of P wave dispersion and IAB in patients with dilated cardiomyopathy[17]. However, it is unknown whether there is any association between IAB and APE. This might be the first study to demonstrate that the prevalence of IAB, was significantly elevated in patients with APE. Moreover, the frequency of P wave dispersion was more common in patients with APE.

Our study findings may be useful and valuable in terms of clinical applicability. As a simple and easily obtained ECG parameter, IAB may have an additive diagnostic value in patients who present to emergency department due to APE signs and symptoms. However, as it was a retrospective case-control study, our study findings deserve further prospective and large scaled studies to clarify the exact role of IAB in patients with APE.

LIMITATIONS

Our study has some limitations. First of all, the sample size is relatively small. However, the power analysis of the study was found to be sufficient. Secondly, we were not able to observe whether there is any different in ECG findings during follow-up period. Finally, further studies with large sample size and longer follow-up time are required to increase the accuracy of the results.

CONCLUSION

In the present study, we demonstrated that IAB, namely P wave duration longer than 120 ms on the ECG, may be independently related with APE. As a simple, cheap, easy to obtain, and non-invasive ECG parameter, IAB may have an additive diagnostic value for predicting APE.

Ethics Committee Approval: The study was approved by the Kafkas University Faculty of Medicine Ethics Committee (Date: 30.01.2019; No: 80576354-050-99/31).

Informed Consent: Informed consent was obtained.

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