

ORIGINAL RESEARCH



Electrocardiographic findings and risk factors for mortality in octogenarians with pulmonary thromboembolism

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Abstract

Background: This study aimed to investigate the electrocardiographic risk factors associated with mortality in elderly patients diagnosed with pulmonary thromboembolism (PTE). **Methods:** This multicenter, retrospective study analyzed electrocardiograms (ECGs) of patients aged 80 years and older at the time of their initial PTE diagnosis. Additionally, patient outcomes were assessed to determine whether death occurred due to PTE or related complications within a follow-up period of 180 days. The relationship between electrocardiographic findings and 30-day and 180-day mortality was examined. **Results:** A total of 241 patients were included, with a mean age of 83.5 ± 2.6 years; 143 (59.3%) were female. The most common electrocardiographic abnormality was sinus tachycardia, observed in 73.4% of patients. Mortality was recorded in 64 patients (26.5%) within 30 days and 88 patients (36.5%) within 180 days. The strongest predictor of 30-day mortality was ST segment (ST) depression in leads V4–6 (odds ratio (OR) 14.5; 95% confidence interval (CI) 5.1–41.0), while the most significant predictor of 180-day mortality was ST elevation in leads V1, augmented Vector Right (aVR) lead, or III (OR 15.8; 95% CI: 6.6–37.7). **Conclusions:** Pulmonary thromboembolism in octogenarians is associated with high mortality. Electrocardiographic evaluation at the time of diagnosis is crucial, as specific ECG abnormalities may serve as important prognostic indicators for 30-day and 180-day mortality.

Keywords

Electrocardiography; Pulmonary thromboembolism; Geriatrics; Mortality; Octogenarians

1. Introduction

Pulmonary thromboembolism (PTE) is characterized by the presence of a thrombus within the branches of the pulmonary artery. PTE has a broad clinical spectrum, ranging from asymptomatic cases to severe manifestations such as cardiogenic shock and sudden cardiac death. However, the majority of patients present to the emergency department with symptoms including shortness of breath, chest pain, syncope, hemoptysis, and a general deterioration in their condition. The diagnosis of PTE can be challenging, particularly in elderly patients who often have multiple comorbidities that may obscure or mimic its presentation [1–3].

Early diagnosis is crucial in reducing PTE-related mortality. Electrocardiographic (ECG) evaluation should be performed as quickly as possible in patients diagnosed with or suspected of having PTE in the preliminary diagnosis [1]. Numerous studies have investigated the ECG abnormalities associated with PTE, identifying changes such as sinus tachycardia, right bundle branch block, T-wave inversion, S1Q3T3 finding, and right axis deviation. Additionally, some of these ECG findings

have been directly linked to increased mortality [4].

However, research on the diagnostic and prognostic value of ECG findings in PTE has predominantly focused on younger populations, with limited studies examining elderly patients. Given the unique physiological characteristics and higher burden of comorbidities in this population, further investigation is imperative. Therefore, this study aimed to analyze ECG findings in elderly patients with PTE, compare the ECG differences between survivors and non-survivors, and evaluate the relationship between specific ECG abnormalities and mortality [5, 6].

2. Methods

This study was designed as a multicenter, retrospective investigation. The records of patients who presented to the emergency rooms of İstinye University Gaziosmanpaşa Medicalpark Hospital, İstinye University Bahçeşehir Liv Hospital, İstanbul Aydın University WM Medicalpark Hospital, WM Medicalpark Kocaeli Hospital, and BHT Klinik İstanbul Tema Hospital between 01 January 2019 to 30 September 2022 were

reviewed. Patients aged over 80 years who were diagnosed with PTE and had an ECG recorded at the time of emergency department admission were included in the study.

The ECGs of all patients were systematically analyzed for abnormalities associated with PTE, and these changes were documented. It was also investigated whether these ECG changes were indicators of mortality. PTE was diagnosed based on the presence of filling defects in the main pulmonary artery or its branches on contrast-enhanced computed tomography (CT), and only patients meeting this criterion were included in the study.

Baseline demographic characteristics, the presence of any additional comorbid diseases (cancer, heart failure, chronic lung disease, chronic kidney disease, coronary artery disease, congenital heart disease, and chronic liver disease), vital signs, and oxygen saturation value at the time of admission were recorded. Laboratory parameters, including troponin, and D-dimer levels, were measured. In addition, the echocardiographic evaluation obtained during hospitalization, such as pulmonary artery pressure and evidence of right ventricular failure, were included in the analysis. To assess the severity of PTE, the pulmonary embolism severity index (PESI) scores were calculated for all patients. Based on PESI scores, the patients were divided into four groups: mild, moderate-mild, moderate-severe, and severe [1]. Based on the data, we determined whether the patients had died of PTE or PTE-related complications within the 30-day and 180-day follow-up periods. ECG differences between survivors and non-survivors were examined, and the prognostic significance of ECG findings in predicting 30-day and 180-day mortality was evaluated.

2.1 Electrocardiographic evaluation

The ECGs obtained at the time of the patients' initial emergency department admission were retrieved from medical records and analyzed. All ECGs were assessed by an experienced cardiologist. The study specifically examined the presence of ECG abnormalities previously identified in the literature as being associated with PTE. The ECG findings evaluated included: sinus tachycardia (heart rate >100 beats/min), right bundle branch block, right axis deviation (QRS complex (QRS) axis between +90 and +180), clockwise rotation (transition zone at V4 or higher), ST depression finding (≥ 0.05 mV) in leads V4–V6, S1Q3T3 finding (S wave in lead I >1.5 mm, Q in lead III >1.5 mm associated with a negative T-wave in lead III), newly developing atrial fibrillation or flutter, Qr in V1 (presence of a prominent Q wave of ≥ 0.2 mV and a ventricular depolarization <120 ms), T-wave inversions in V1 to V4, and ST elevation in V1, aVR, or III (≥ 1 mV) [4, 7–10].

2.2 Exclusion criteria

Patients were excluded from the study if they met any of the following criteria: PTE diagnosis could not be confirmed by computed tomography, absence of an ECG record at the time of admission, insufficient clinical information in their medical records, lack of an echocardiographic evaluation during hospitalization, voluntary refusal of treatment, discharge against

medical advice after hospital admission, patients under the age of 80, those who did not come back for follow-up in the 180 days post-discharge, and absence of follow-up records in their medical files.

2.3 Statistical analyses

Variables are presented as mean $\pm$ standard deviation (SD) or median (range, interquartile range (IQR)) for continuous data and proportion for categorical data. Categorical parameters were analyzed using the chi-square test, while continuous variables with normal distribution were analyzed with an unpaired *t*-test as appropriate. Kolmogorov-Smirnov test was used to identify whether continuous variables were normally distributed. Two-sided *p* values < 0.05 were considered statistically significant. Statistical analysis was performed using SPSS version 21.0 for Windows (SPSS, Inc., Chicago, IL, USA).

3. Results

A total of 241 patients were included in this study, with a mean age of 83.5 ± 2.6 and among them, 143 (59.3%) were women. The most common presenting symptom was dyspnea observed in 224 (92.9%) patients. Regarding risk stratification, 51 (21.1%) patients were classified as low-risk while 56 (23.3%) were categorized as high-risk group. The demographic, clinical, and laboratory parameters of the study population are shown in Table 1.

The most frequently observed ECG abnormality was sinus tachycardia, detected in 177 (73.4%) patients, whereas the least common finding was new-onset atrial fibrillation (AF) or flutter, observed in 16 (6.6%) patients. The ECG findings of the patients included in this study are summarized in Table 2.

During follow-up, 64 (26.6%) patients died within the 30-day, while 88 (36.5%) patients died within 180-days due to PTE or PTE-related complications. When comparing the ECG findings of the patients who died versus those who survived within the 30-day follow-up period, no significant difference was found in the presence of or in V1 ($p = 0.856$), while all other ECG abnormalities were significantly different between the two groups ($p < 0.05$). Similarly, in the 180-day follow-up, the Qr in the V1 finding remained statistically insignificant ($p = 0.733$), whereas other ECG parameters significantly differed between the survivors and non-survivors ($p < 0.05$). The comparisons are summarized in Table 3. Among the ECG abnormalities, ST depression in leads V4–6 was identified as the strongest predictor of 30-day mortality (OR: 14.5, 95% CI: 5.1–41), while ST elevation in leads V1, aVR, or III had the highest predictive value for 180-day mortality (OR: 15.8, 95% CI: 6.6–37.7). The distribution of ECG findings in relation to 30-day and 180-day mortality is summarized in Table 4.

4. Discussion

The findings of our study suggest that ECG differences may exist between patients who survived and those who did not in both the 30-day and 180-day follow-up periods of elderly patients (aged over 80 years) diagnosed with PTE. Furthermore,

TABLE 1. Baseline clinic, demographic, and laboratory values of the study population.

Variables	Values
Age, yr	83.5 ± 2.6
Male gender, n (%)	98 (40.7)
SBP <100 mm Hg, n (%)	34 (14.1)
Heart rate ≥100 bpm, n (%)	177 (73.4)
Respiratory rate >30, breaths/min	46 (19.0)
Active cancer, n (%)	17 (7.0)
Heart failure, n (%)	30 (12.4)
Chronic lung disease, n (%)	29 (12.0)
Temperature <36 °C, n (%)	13 (5.3)
Altered mental status, n (%)	15 (6.2)
Arterial oxygen saturation <90, n (%)	39 (16.1)
Positive Troponin, n (%)	93 (38.5)
PESI points	104.2 ± 35.7
Pulmonary embolism severity	
Low risk, n (%)	51 (21.1)
Intermediate-low risk, n (%)	59 (24.4)
Intermediate-high risk, n (%)	75 (31.1)
High risk, n (%)	56 (23.2)
D-dimer (µg/L)	1730 (IQR = 910)
Ejection fraction (%)	49.4 ± 10.6
sPAP (mm Hg)	69.7 ± 21.3
Dyspnea, n (%)	224 (92.9)
Chest pain, n (%)	132 (54.7)
Cough, n (%)	53 (21.9)
Syncope, n (%)	29 (12.0)
Fatigue, n (%)	24 (9.9)
Vomiting, n (%)	22 (9.1)

IQR: Interquartile range; PESI: Pulmonary embolism severity index; SBP: Systolic blood pressure; sPAP: Systolic pulmonary artery pressure.

TABLE 2. Basal electrocardiographic findings of the study population.

ECG findings	n (%)
Sinus tachycardia	177 (73.4)
Right bundle branch block	128 (53.1)
Right axis deviation	27 (11.2)
Clockwise rotation	96 (39.8)
ST depression in V4–6 derivation	24 (10.0)
S1Q3T3	61 (25.3)
Atrial fibrillation or flutter	16 (6.6)
Qr in V1 derivation	24 (10.0)
T-wave inversions in V1–4 derivation	52 (21.6)
ST elevation in V1, aVR, or III	45 (18.7)

ST: ST segment; aVR: augmented vector right lead; ECG: Electrocardiography.

TABLE 3. The comparisons of ECG findings between the patients who died and those who survived at the 30-day and 180-day follow-ups.

	30 days mortality			180 days mortality		
	Mortality (64)	No mortality (177)	<i>p</i>	Mortality (88)	No mortality (153)	<i>p</i>
Tachycardia, n (%)	59 (92.2)	118 (66.7)	<0.001	74 (84.1)	103 (67.3)	0.005
RBBB, n (%)	51 (79.7)	77 (43.5)	<0.001	70 (79.5)	58 (37.9)	<0.001
Right axis deviation, n (%)	17 (26.6)	10 (5.6)	<0.001	22 (25.0)	5 (3.2)	<0.001
Clockwise rotation, n (%)	44 (68.8)	52 (29.4)	<0.001	54 (61.4)	42 (27.5)	<0.001
ST depression in V4–6, n (%)	19 (29.7)	5 (2.8)	<0.001	19 (21.6)	5 (3.3)	<0.001
S1Q3T3, n (%)	37 (57.8)	24 (13.6)	<0.001	43 (48.8)	18 (11.7)	<0.001
AF or flutter, n (%)	10 (15.6)	6 (3.4)	0.001	11 (12.5)	5 (3.3)	0.006
Qr in V1, n (%)	6 (9.4)	18 (10.2)	0.856	8 (9.1)	16 (10.5)	0.733
T-wave inversions in V1–4, n (%)	23 (35.9)	29 (16.5)	0.001	41 (46.6)	11 (7.2)	<0.001
ST elevation in V1, aVR, or III, n (%)	27 (42.7)	18 (10.2)	<0.001	38 (43.2)	7 (4.6)	<0.001

AF: Atrial fibrillation; ECG: Electrocardiography; RBBB: Right bundle branch block; ST: ST segment; aVR: augmented vector right lead.

TABLE 4. The association of ECG findings with 30-day and 180-day mortality.

	30 days mortality			180 days mortality		
	OR	95% CI		OR	95% CI	
Tachycardia	5.9	2.2	15.4	2.5	1.3	4.9
RBBB	5.0	2.5	10.0	6.3	3.4	11.7
Right axis deviation	6.0	2.5	14.0	9.8	3.5	27.1
Clockwise rotation	5.2	2.8	9.8	4.1	2.4	7.3
ST depression in V4–6	14.5	5.1	41.0	8.1	2.9	22.7
S1Q3T3	8.7	4.5	16.8	6.4	3.4	12.1
AF or flutter	5.2	1.8	15.1	4.2	1.4	12.6
Qr in V1	0.9	0.3	2.4	0.8	0.3	2.0
T-wave inversions in V1–4	2.8	1.4	5.4	11.1	5.3	23.5
ST elevation in V1, aVR, or III	6.4	3.2	12.9	15.8	6.6	37.7

AF: Atrial fibrillation; CI: Confidence interval; ECG: Electrocardiography; RBBB: Right bundle branch block; OR: Odds ratio; ST: ST segment; aVR: augmented vector right lead.

certain ECG abnormalities observed at the time of emergency department admission may serve as a predictor of mortality.

There is a limited number of studies in the literature that have specifically investigated ECG abnormalities in patients with PTE. In a study involving 390 patients aged over 65 years, the presence of at least one classic right ventricular strain pattern on ECG, including S1Q3T3, right bundle branch block, or T-wave inversion in leads V1–4, were associated with a higher risk of adverse events during follow-up [4]. Similarly, in another study involving 386 patients with PTE, it was shown that patients with any of the three classic signs of right ventricular strain had an increased risk of death or adverse events in the hospital (hazard ratio 2.6) [11, 12]. Most studies focusing on non-elderly patients have reported that S1Q3T3, Right bundle branch block (RBBB), or T-wave inversion in leads V1–4 are linked to adverse outcomes in patients with PTE, which aligns with our findings. However,

our study further indicates that ST depression in leads V4–6 is particularly a strong predictor of 30-day mortality in the elderly patient population, emphasizing its prognostic significance in this age group [13].

The pathophysiology of PTE and the ECG changes it induces are not yet clearly understood. It is believed that these changes result from a combination of anatomical, hemodynamic, metabolic, and autonomic alterations in the cardiac tissue following PTE [14]. Acute PTE leads to obstruction of the main pulmonary artery or its branches, causing an increase in right ventricular pressure. As a result, right heart dilation and alterations in the electrical axis orientation may occur. In addition, right ventricular overload can result in decreased myocardial perfusion and subsequent myocardial ischemia [15, 16]. Some ECG findings may serve as an indicator of the occurrence of right ventricular ischemia. Hypoxia triggered by PTE leads to an increased release of chemical mediators,

including catecholamines and histamine, which can contribute to coronary vasospasm. However, these changes are reversible most of the time. If a patient survives the acute phase and the thrombus is successfully dissolved through treatment, the associated ECG abnormalities and clinical symptoms generally resolve [17–19]. All these ECG changes are observed less frequently in younger patients, whereas they tend to be more common in elderly patients [19]. The higher prevalence of comorbid diseases (heart failure, coronary artery disease, hypertension, and chronic obstructive pulmonary disease) in the elderly population, combined with their reduced physiological tolerance to hypoxia and excessive pressure overload, may contribute to the increased likelihood of ECG changes in this population [20–22].

In a study conducted by Kukla *et al.* [10] on patients with PTE, ST segment depression in leads V1–4 was found to have an incidence of 51.6%, but this finding could not predict mortality. In another study by Geibel *et al.* [7], ST segment depression in V1–4 was found in 24.5% of all patients and 51.6% of high-risk patients. In another study by Kaczyńska *et al.* [23], ST depression was noted in 24% of the patients, and this finding was found to be significantly higher in the subgroup with high troponin values. In a study by Janata *et al.* [24], a mortality rate of 35.7% was found in high-risk patients with ST-segment depression, but a similar rate was also noted in low-risk patients. In our study, ST segment depression was detected in 10% of patients, and it emerged as a potential predictor of mortality. The observed lower incidence compared to previous studies is likely attributable to the advanced age group of our study population.

PTE can mimic acute myocardial infarction by causing ST elevation, particularly in leads DII, DIII, and aVF. In addition, it may lead to ST elevation in lead V1, which reflects the electrical activity of the right ventricular free wall, further resembling an acute myocardial infarction pattern. Another study found ST elevation in lead DIII in 12.5% of all PTE patients and 28.9% among high-risk patients [10]. The findings of our study are consistent with the literature, reinforcing the significance of ST elevation in V1, aVR, or III as a strong predictor of 180-day mortality in elderly PTE patients [25].

5. Study limitations

The primary limitation of our study is the lack of an evaluation of dynamic ECG changes over time. Since PTE is an emergency condition, pre-PTE ECG recordings were not available for comparison, making it difficult to determine whether some of the observed ECG abnormalities existed before the acute event. Additionally, there was no standardization in the timing between the onset of symptoms and the ECG examination, which may have introduced variability in the recorded findings. Furthermore, potential confounding factors, such as differences in anticoagulation therapy or thrombolysis, were not accounted for in the analysis. Finally, the findings should be validated in an independent cohort to confirm their reproducibility and generalizability to broader populations.

6. Conclusions

Our study indicates that ECG differences may help distinguish between survivors and non-survivors among patients over 80 years of age with PTE during the 30-day and 180-day follow-up periods. Certain ECG abnormalities detected at the time of emergency department admission may serve as valuable prognostic indicators of mortality. Among the observed ECG changes, sinus tachycardia was the most common finding. ST depression in leads V1–4 was identified as a significant predictor of 30-day mortality, while ST elevation in leads V1, aVR, or III was strongly associated with 180-day mortality. These findings suggest that specific ECG parameters could aid in early risk stratification for elderly PTE patients. However, larger prospective studies are needed to confirm their predictive value and enhance mortality risk assessment in this population.

AVAILABILITY OF DATA AND MATERIALS

The data used in this study are available from the corresponding author upon reasonable request.

AUTHOR CONTRIBUTIONS

TAG—conceptualization, methodology, data collection, writing—original draft. AS—formal analysis, data interpretation, writing—review & editing. AA—investigation, resources, writing—review & editing. TO—supervision, project administration, final approval of the manuscript. All authors have read and approved the final version of the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

All procedures performed in studies involving human participants were conducted in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards. This study was approved by the Istinye University Institutional Review Board and Ethics Committee (Decision: 19.01.2024, No: 23-294). Informed consent was obtained from all participants in accordance with ethical guidelines.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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