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Original Contribution

Predictors of early death in patients with acute pulmonary embolism ☆

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ABSTRACT

Aim: We aimed to determine the predictors of early death in the course of acute pulmonary embolism (APE).
Materials and methods: We included 206 patients who had been admitted to our hospital between January 2011 and April 2013 with the diagnosis of APE. We derived a new model including corrected QT interval dispersion (QTcd) and P wave dispersion (Pd), echocardiographic findings, laboratory markers, and blood cell count indices to predict early death in patients with APE.
Results: Thirty patients (14.5%) died; 176 patients (85.5%) lived after diagnosis of APE. Logistic regression (LR) analysis found that troponin I (odds ratio [OR], 1.084 [95% confidence interval {CI}, 1.009-1.165]), creatinine (OR, 4.153 [95% CI, 1.375-12.541]), mean platelet volume (OR, 1.991 [95% CI, 1.230-3.223]), neutrophil to lymphocyte ratio (NLR) (OR, 1.079 [95% CI, 1.005-1.160]), QTcd (OR, 1.084 [95% CI, 1.043-1.127]), Pd (OR, 1.049 [95% CI, 1.004-1.096]) were associated with early death in APE. New LR model (area under the curve [AUC], 0.970) performed better than the simplified pulmonary embolism severity index (sPESI) score (AUC, 0.859) in predicting early death in APE ($P = .021$).
The predictivity of the sPESI score significantly improved after its single combination with creatinine, QTcd, or troponin I. When the combined model was constructed together with these 6 independent variables and sPESI score, stepwise LR model automatically excluded Pd and NLR, and the AUC from the rest of the combined model was 0.976, which is significantly different from the AUC of sPESI (0.859) ($P = .0031$).
Conclusions: Creatinine, troponin I, and QTcd significantly improves sPESI score. A new model with troponin I, creatinine, mean platelet volume, NLR, QTcd, and Pd seems to have greater prognostic power than the sPESI scoring system.

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1. Introduction

Acute pulmonary embolism (APE) remains associated with high morbidity and mortality rates despite advanced therapeutic options. This may be rooted partially in deficient initial prognostic assessment of patients. Both European and US guidelines suggest more aggressive treatment, such as administration of thrombolytic agents, for those at high risk for early mortality [1]. Moreover, some studies indicate that thrombolytic therapy may have a place in the management of some moderate-risk patients [2]. Risk stratification of patients with APE is mandatory to allow assessment of the individual prognosis and guide therapeutic decision making. Interestingly, clinical scores have been developed and validated to predict short-term prognosis after APE [3-5]. The simplified pulmonary embolism severity index (sPESI) is the most extensively studied clinical score to date [3]. It includes 6 equally weighted variables: older than 80 years, history of cancer, history of chronic cardiopulmonary disease, heart rate greater than 110 beat per minute, systolic blood pressure less than 100 mm Hg, and arterial oxyhemoglobin saturation less than 90%. Other than these useful clinical scores, there is no clear consensus on the use of thrombolytic therapy, especially in intermediate-risk patients, defined as those without shock or hypotension but with adverse event predictors, such as elevated serum markers or right ventricular (RV) dysfunction by diagnosed by transthoracic echocardiography (TTE).

To highlight the problem, some studies have suggested that laboratory biomarkers, particularly cardiac troponins but also electrocardiographic (ECG) and echocardiographic parameters and some complete blood cell count indices, could be useful in identifying patients with an elevated risk of death and complications during the acute phase of pulmonary embolism (PE) [6-10]. In addition, data indicate that serum creatinine levels may have prognostic importance in PE [11]. A positive characteristic of these prognostic markers is that all can be achieved easily in the emergency department (ED).

To the author's best knowledge, there are no data in the literature about combined use of these prognostic markers in the course of APE. We aimed to determine the predictive abilities of ECG, TTE, laboratory

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markers, blood cell count indices, and associated clinical conditions regarding early death in the course of APE. In addition, we used logistic regression (LR) analyses of independent variables to derive a valuable model to predict those at high risk. We also tested the additive prognostic determination values of these independent variables when combining them to the sPESI score.

2. Materials and methods

2.1. Patients

This study was conducted in Adnan Menderes University Faculty of Medicine in Aydın, Turkey. We retrospectively reviewed archived data of 288 patients who had been hospitalized in Faculty Hospital between January 2011 and April 2013 with the diagnosis of PE and who received final diagnoses after exact demonstration of thrombus in pulmonary arteries via computed tomography (CT). The study included 206 patients who were in sinus rhythm, suitable for QT and P wave analyses, and who had accessible TTE data and laboratory test results. Eighty-two patients were excluded from the study (36 had incomplete TTE data, TTE was not performed on 12 of them, 11 had atrial fibrillation in the ECG, 9 had excessive noise in the ECG, and 14 patients had missing laboratory data) (Fig. 1).

We recorded patients' baseline characteristics (sex, ages, etc), comorbidities, symptoms, hemodynamic conditions, all-cause mortality rates during hospitalization, total hospitalization times, radiographic test results, and laboratory findings, and we evaluated ECGs and TTEs obtained during admission (Table 1). Using collected baseline data at the time of PE diagnosis and the outcome data of this cohort, we retrospectively assessed sPESI scores for all patients. We excluded patients with atrial fibrillation during admission, excessive noise in ECG, or incomplete TTE data. The study protocol was approved by the Ethics Committee of the University of Adnan Menderes, and we followed all procedures in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008.

2.2. Diagnosis of PE

We diagnosed PE by spiral CT pulmonary angiography with the direct visualization of an intraluminal filling defect in the course of acute symptoms and signs suggesting PE.

2.3. Definitions

We defined early death as inhospital mortality. We defined major bleeding as (1) a fall in hemoglobin of 2 g/dL, (2) transfusion of 2 U or more of red blood cells, (3) symptoms in a critical organ (intracranial, intraspinal, intraocular, retroperitoneal, intraarticular or pericardial, or intramuscular with compartment syndrome), or (4) fatal bleeding. Otherwise, we defined bleeding as minor bleeding.

We defined the appropriate ECG to have at least 10 analyzable leads for the needed measurements. Otherwise, ECG was defined to have excessive noise.

If the TTE data included all needed measurements (such as RV and left ventricular [LV] diameters, ejection fraction, or pulmonary artery pressure), then we defined it to be complete; otherwise, we defined it to be incomplete data.

We defined hemodynamic instability as (1) PE causing hypotension (systolic blood pressure ≤90 mm Hg or a reduction of at least 40 mm Hg

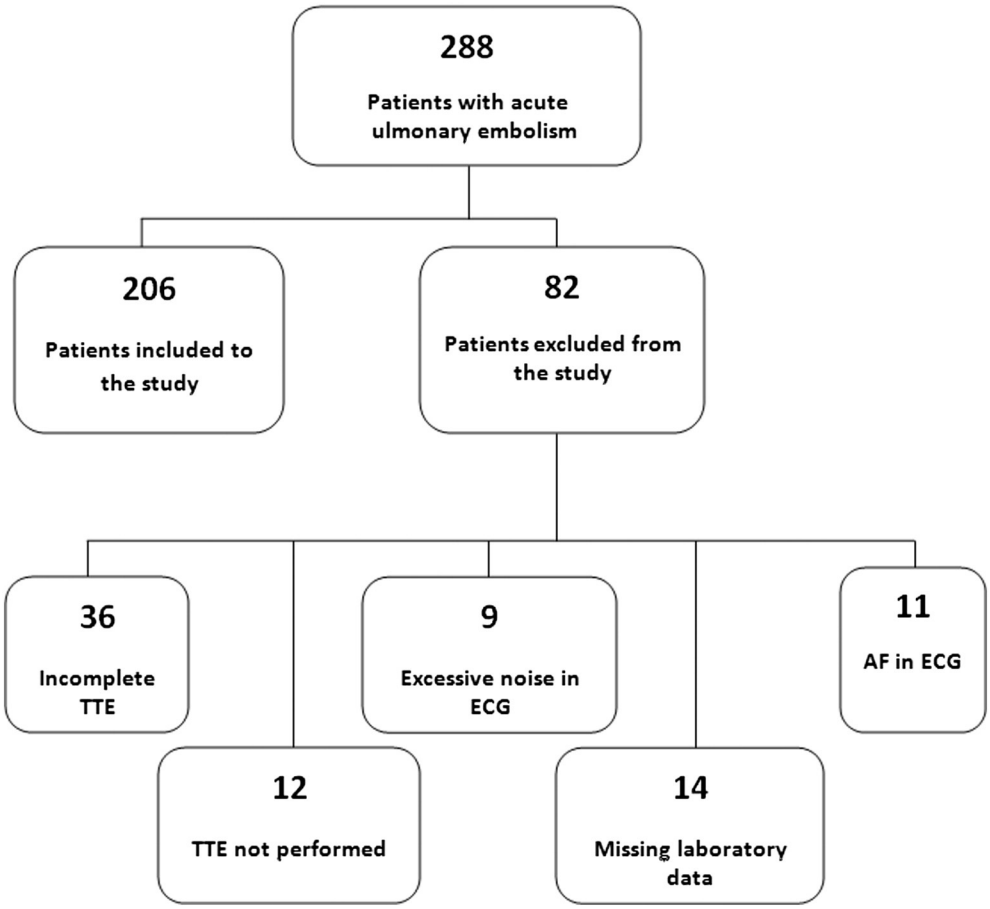

Fig. 1. A diagram depicting the inclusion phase of the patients. Abbreviation: *AF*, atrial fibrillation.

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