

The value of ECG parameters in estimating myocardial injury and establishing prognosis in patients with acute pulmonary embolism

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Abstract

Background: The electrocardiogram (ECG) is characterised by little sensitivity and specificity in the diagnostic evaluation of acute pulmonary embolism (APE).

Aim: To assess the significance of ECG changes in predicting myocardial injury and prognosis in patients with APE.

Methods: The study group consisted of 225 patients (137 women and 88 men), mean age: 66.0 ± 15.2 years, in whom the diagnosis of APE was made, mostly based on computed tomography (n = 206, 92%).

Results: We observed 26 in-hospital deaths (mortality rate: 11.5%) and complications occurred in 58 (25.7%) patients. Elevated levels of troponin were observed in 103 (46%) patients. Logistic regression analysis showed that in-hospital mortality was associated with: coronary chest pain (0.06–0.53, OR 0.18), systolic blood pressure below 100 mm Hg (2.3–13.64, OR 5.61), heart rate above 100 bpm (1.17–15.11, OR 4.21), the S1Q3T3 sign (1.31–6.99, OR 3.02), QR in V₁ (1.60–12.32, OR 4.45), ST-segment depression in V₄–V₆ (0.99–5.40, OR 2.31), ST-segment elevation in III (0.99–6.96, OR 2.64), ST-segment elevation in V₁ (1.74–9.49, OR 4.07); borderline (1.51–16.07, OR 4.93), moderate (1.42–17.74, OR 5.01) and severe troponin elevation (2.88–36.38, OR 10.24). In patients with cTnT(+), compared to patients with normal troponin levels, the following ECG changes were significantly more common: the S1Q3T3 sign (43 vs 21%, p = 0.003), negative T waves in V₂–V₄ (57 vs 27%, p = 0.0001), ST-segment depression in V₄–V₆ (40 vs 14%, p = 0.001), ST-segment elevation in III (22 vs 7%, p = 0.0006), V₁ and V₂ (43 vs 10%, p = 0.0001) and QR in V₁ (16 vs 5%, p = 0.007).

Conclusions: ECG parameters are useful in predicting myocardial injury and assessing prognosis in patients with APE.

Key words: acute pulmonary embolism, ECG, troponin, complications

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INTRODUCTION

According to the current European Society of Cardiology (ESC) recommendations, a prognostic assessment necessary for risk stratification and decision making should be performed in patients being diagnosed with acute pulmonary embolism (APE). Risk stratification is achieved in a stepwise fashion from the

assessment of the haemodynamic status to the assessment of right ventricular (RV) function and laboratory tests (troponin, BNP, NT-proBNP) [1]. Markers of myocardial injury (troponins) have become a standard parameter assessed in patients with APE. In normotensive patients with APE, the presence of manifestations of RV dysfunction (RVD) and/or myocardial injury

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identifies patients as the moderate risk group. On the other hand, the absence of RVD and/or myocardial injury manifestations in normotensive patients makes it possible to classify them to the low risk group [1]. Recent studies have unequivocally showed that elevated troponin levels are associated with adverse prognosis not only in the entire APE population but also in the subgroup of normotensive patients [2].

The electrocardiogram (ECG) in patients with APE may present a wide variety of manifestations: from a completely normal tracing to arrhythmias (supraventricular and ventricular arrhythmias), conduction abnormalities (right bundle branch block [RBBB]), changes in the electrical axis of the heart (left axis deviation, right axis deviation), changes in the morphology of the P wave (the so-called P pulmonale) changes in the QRS complex size (low amplitude of the QRS wave) or various changes of the repolarisation period (changes in T wave polarisation, ST-segment depression and elevation) along with QT prolongation [3]. Electrocardiography does not offer any specific or sensitive criteria or manifestations to detect APE. However, a correctly interpreted ECG suggestive of APE may contribute to an appropriate and rapid diagnostic evaluation leading to a correct diagnosis, which in turn affects the prognosis and determines the correct treatment.

The aim of the study was to assess the significance of ECG changes in predicting myocardial injury and prognosis in patients with a confirmed diagnosis of APE.

METHODS

Study group

A total of 225 patients (137 women and 88 men), from 17 to 89 years of age (mean age 66.0 ± 15.2 years), were included in the study. The mean duration of hospitalisation was 14.9 days (with a maximum duration of 46 days). Table 1 summarises the clinical characteristics of the patients.

The diagnosis of pulmonary embolism was based on the diagnostic evaluations listed in Table 2, in accordance with the current ESC recommendations [1]. In 9 patients with cardiogenic shock or marked hypotension, the diagnosis of APE was based on the signs of RV strain on echocardiography.

Criteria for right ventricular strain

The following criteria were adopted for the identification of RV strain on echocardiography: RV end diastolic dimension > 30 mm, peak tricuspid regurgitation jet velocity > 2.8 m/s, peak tricuspid regurgitation gradient > 30 mm Hg, RV outflow tract acceleration time < 90 ms, paradoxical interventricular septal motion, and the ratio of RV to left ventricular (LV) end diastolic dimension > 1 [1].

Markers of myocardial injury

The levels of troponin-T or troponin-I (depending on the centre) were determined on admission. The following commercial kits for determination of troponin were used (the values

Table 1. Demographic and clinical characteristics of the patients

| | |
|---|----------------------|
| Age [years] | 66.0 \pm 15.2 |
| Women/men | 137/88 (60.9%/39.1%) |
| Chest pain: | 105 (46.7%) |
| Coronary/pleural | 58/47 |
| Syncope | 67 (29.8%) |
| Obesity | 68 (30.2%) |
| Immobility | 61 (27.1%) |
| Leg phlebitis | 113 (50.2%) |
| Haemoptysis | 7 (3.1%) |
| Cancer | 17 (7.6%) |
| Hormone replacement therapy/contraception | 5 (2.2%) |
| NYHA class III/IV heart failure | 29 (12.9%) |
| Coronary artery disease | 43 (19.1%) |
| Chronic obstructive pulmonary disease | 20 (8.9%) |
| Low-grade fever | 25 (11.1%) |
| Duration of hospitalisation [days] | 14.9 |

Table 2. Diagnostic tests used in the evaluation of acute pulmonary embolism

| | |
|----------------------------|-------------|
| Computed tomography | 206 (91.5%) |
| Echocardiography: | 9 (4%) |
| Right ventricular strain | 6 |
| Embolitic material/thrombi | 3 |
| Venous Doppler | 6 (2.7%) |
| Scintigraphy | 2 (0.9%) |
| Autopsy | 2 (0.9%) |

in brackets are cut-off values for positive results): troponin-T ($0.03 \mu\text{g/L}$) — ECLIA (electrochemiluminescence immunoassay) from Roche Diagnostics; troponin-I ($0.4 \mu\text{g/L}$) — ECLIA from Roche Diagnostics; troponin-I ($< 0.1 \mu\text{g/L}$) from Abbott; troponin-I ($< 0.1 \mu\text{g/L}$) — enzyme-linked immunosorbent assay from bioMérieux.

Patients with abnormal troponin levels, depending on the concentration of troponin, were arbitrarily divided into three groups: borderline myocardial injury (troponin-T values from > 0.03 to $0.10 \mu\text{g/L}$; troponin-I values from > 0.1 to $0.5 \mu\text{g/L}$), moderate myocardial injury (troponin-T values from > 0.10 to $1.00 \mu\text{g/L}$; troponin-I values from > 0.51 to $1.00 \mu\text{g/L}$) and severe myocardial injury (troponin-T $> 1.00 \mu\text{g/L}$; troponin-I $> 1.00 \mu\text{g/L}$).

Electrocardiographic analysis

A 12-lead ECG was obtained in all the patients. The paper speed, depending on the centre, was 25 mm/s or 50 mm/s and the amplitude of the standard deflection was 10 mm/mV.

The first available ECG obtained in a given patient on admission or during hospitalisation was included in the analysis.

The following were analysed: (1) heart rate; (2) presence of supraventricular and ventricular arrhythmias; (3) QRS axis; (4) presence of P pulmonale, if the amplitude of the P wave exceeded 0.25 mV in at least one limb lead corresponding to the inferior wall (II, III and aVF); (5) presence of a complete RBBB; (6) presence of the S1Q3T3 sign; (7) presence of negative T waves in III and aVF; (8) presence of negative T waves in V_2 - V_4 ; (9) presence of ST-segment depression in V_4 - V_6 ; (10) presence of ST-segment elevation in III and V_1 ; (11) presence of a notch in the R or S waves in V_1 ; (12) presence of Q(q)R complexes in V_1 ; (13) presence of dextrogyria, if the R-to-S wave amplitude in V_5 is 1 or less; (14) presence of low-voltage QRS (< 5 mm) in limb leads; (15) number of leads with negative T waves.

The following complications during the in-hospital follow-up were taken into account: death from any cause, cardiac arrest, cardiogenic shock on admission or during hospital stay and necessity to use ventilatory support.

Statistical analysis

Continuous variables with a normal distribution are expressed as means \pm SD. Qualitative variables are compared with the χ^2 test (with Yates' correction in the case of small sample sizes). Results with p values below 0.05 (two-sided) were considered statistically significant. The logistic regression model was used to assess the significance of the risk factors. The values of individual coefficients were calculated and their statistical significance was assessed ($p < 0.05$). In addition, in

order to assess the effects of each of the factors a unit odds ratio (OR) and its confidence interval (CI) were obtained. The statistical calculations were performed using Statistica PL v 6.1 (StatSoft, Inc.).

RESULTS

During hospitalisation a total of 26 deaths occurred (a mortality rate of 11.5%), including 24 deaths directly related to APE, 1 death due to haemorrhagic complications and 1 death due to cancer. Complications were observed in 58 (25.7%) patients.

In 103 (45.7%) patients elevated troponin levels were observed. Depending on the degree of troponin elevation, the groups with borderline, moderate and severe myocardial injury comprised 46 (44.7%), 34 (33.0%) and 23 (23.3%) patients, respectively. On admission, 38 (16.9%) patients were diagnosed with high-risk APE.

The ECG changes in the group of patients with elevated troponin levels compared to the group with normal troponin levels are summarised in Table 3. In the group of patients with elevated troponin levels compared to the group with normal troponin levels the following manifestations were more common: the S1Q3T3 sign, presence of negative T waves in V_2 - V_4 , ST-segment depression in V_4 - V_6 , ST-segment elevation in III and V_1 , and qR or QR in V_1 . In addition, in patients with elevated troponin levels, negative T waves were present in a higher number of leads (Table 3). No differences in the frequency of specific ECG parameters were observed depending on the degree of troponin level elevation (borderline, moderate or severe) (Table 4).

Table 3. Electrocardiographic parameters relative to elevated or normal levels of troponin

| ECG parameter | Troponin(+) (n = 103) | Troponin(-) (n = 122) | P |
|--|-----------------------|-----------------------|--------|
| Heart rate [bpm] | 107.1 \pm 23.5 | 98.8 \pm 28.4 | 0.02 |
| Atrial fibrillation | 20 (19.4%) | 27 (22.1%) | NS |
| Left axis deviation | 48 (46.6%) | 63 (51.6%) | NS |
| Right axis deviation | 16 (15.5%) | 15 (12.3%) | NS |
| S1Q3T3 sign | 44 (42.7%) | 25 (20.5%) | 0.0003 |
| Negative T wave in V_2 - V_4 | 59 (57.3%) | 33 (27.0%) | 0.0001 |
| Number of leads with negative T waves | 3.84 | 2.1 | 0.0001 |
| ST-segment depression in V_4 - V_6 | 41 (39.8%) | 17 (13.9%) | 0.0001 |
| ST-segment elevation in III | 23 (22.3%) | 8 (6.6%) | 0.0006 |
| Right bundle branch block | 15 (14.5%) | 12 (9.8%) | NS |
| qR in V_1 | 16 (15.5%) | 6 (4.9%) | 0.007 |
| ST-segment elevation in V_1 | 44 (42.7%) | 12 (9.8%) | 0.0001 |
| Dextrogyria | 62 (60.1%) | 72 (59.0%) | NS |
| Notched QRS in V_1 | 16 (15.5%) | 10 (8.2%) | NS |
| P pulmonale | 15 (14.5%) | 17 (13.9%) | NS |
| Low-voltage QRS | 5 (4.8%) | 8 (6.6%) | NS |

Table 4. Electrocardiographic parameters relative to the degree of elevation of troponin levels

| ECG parameter | Borderline elevation of troponin levels (n = 46) | Moderate elevation of troponin levels (n = 34) | Severer elevation of troponin levels (n = 23) | P |
|---|--|--|---|----|
| Atrial fibrillation | 8 (17.4%) | 8 (23.5%) | 3 (13%) | NS |
| Left axis deviation | 22 (47.8%) | 14 (41.2%) | 13 (56.5%) | NS |
| Right axis deviation | 8 (17.4%) | 5 (14.7%) | 3 (13%) | NS |
| S1Q3T3 sign | 18 (39.1%) | 19 (55.9%) | 7 (30.4%) | NS |
| Negative T wave in V ₂ -V ₄ | 26 (56.5%) | 17 (50%) | 12 (52.2%) | NS |
| Number of leads with negative T waves | 3.9 | 4.1 | 3.3 | NS |
| ST-segment depression in V ₄ -V ₆ | 19 (41.3%) | 12 (35.3%) | 10 (43.5%) | NS |
| ST-segment elevation in III | 9 (19.6%) | 9 (26.5%) | 5 (21.7%) | NS |
| Right bundle branch block | 8 (17.4%) | 3 (8.8%) | 4 (17.4%) | NS |
| qR in V ₁ | 6 (13%) | 6 (17.6%) | 4 (17.4%) | NS |
| ST-segment elevation in V ₁ | 18 (39.1%) | 18 (52.9%) | 9 (39.1%) | NS |
| Dextrogyria | 31 (67.4%) | 19 (55.9%) | 12 (52.2%) | NS |
| Notched QRS in V ₁ | 7 (15.2%) | 7 (20.6%) | 2 (8.7%) | NS |
| P pulmonale | 3 (6.5%) | 7 (20.6%) | 5 (21.7%) | NS |
| Low-voltage QRS | 2 (4.3%) | 1 (2.9%) | 2 (8.7%) | NS |

Table 5. Logistic regression analysis: the effects of selected clinical and electrocardiographic parameters on in-hospital mortality

| | Odds ratio | 95% confidence interval | P |
|---|------------|-------------------------|--------|
| Age > 75 years | 2.05 | 0.98–4.69 | 0.088 |
| Syncope | 0.68 | 0.29–1.78 | 0.42 |
| Coronary chest pain | 0.18 | 0.06–0.53 | 0.002 |
| Systolic blood pressure < 100 mm Hg | 5.61 | 2.3–13.64 | 0.0001 |
| Heart rate > 100 bpm | 4.21 | 1.17–15.11 | 0.026 |
| S1Q3T3 sign | 3.03 | 1.31–6.99 | 0.008 |
| ST-segment depression in V ₄ -V ₆ | 2.31 | 0.99–5.40 | 0.05 |
| Atrial fibrillation | 0.89 | 0.32–2.51 | 0.82 |
| Negative T wave in III and aVF | 2.27 | 0.96–5.35 | 0.06 |
| Negative T wave in V ₂ -V ₄ | 1.69 | 0.74–3.86 | 0.21 |
| Number of leads with negative T waves | 1.16 | 0.99–1.35 | 0.054 |
| ST-segment elevation in III | 2.64 | 0.99–6.96 | 0.048 |
| Right bundle branch block | 2.52 | 0.91–7.04 | 0.07 |
| QR in V ₁ | 4.45 | 1.60–12.32 | 0.0039 |
| ST-segment elevation in V ₁ | 4.07 | 1.74–9.49 | 0.001 |
| ST-segment elevation in aVR | 0.99 | 0.91–1.08 | 0.84 |
| P pulmonale | 1.36 | 0.47–3.96 | 0.56 |
| Low-voltage QRS | 1.35 | 0.28–6.51 | 0.7 |
| Borderline elevation of troponin levels | 4.93 | 1.51–16.07 | 0.007 |
| Moderate elevation of troponin levels | 5.01 | 1.42–17.74 | 0.011 |
| Severe elevation of troponin levels | 10.24 | 2.88–36.38 | 0.0003 |

The logistic regression analysis showed a significant effect of the following parameters on in-hospital death rates: coronary chest pain, systolic blood pressure < 100 mm Hg, heart rate > 100 bpm, the S1Q3T3 sign, QR in V₁, ST-segment depression in V₄-V₆, ST-segment elevation in III, ST-segment elevation in V₁, and borderline, moderate and severe elevation of troponin levels (Table 5).

DISCUSSION

We showed that negative T waves in V₂-V₄, ST-segment depression in V₄-V₆, ST-segment elevation in III and V₁ and the presence of negative T waves in a higher number of leads occurred significantly more commonly in patients with elevated troponin levels than in patients with normal troponin levels.

The ECG parameters related to the risk of death during in-hospital follow-up in the logistic regression model included: the S1Q3T3 sign, QR in V₁, ST-segment depression in V₄-V₆, ST-segment elevation in III and ST-segment elevation in V₁.

Elevated troponin levels were observed in 45.7% of our patients. The most recent metaanalysis by Becattini et al. [2] showed that elevated troponin levels were a predictor of death in short-term observation not only in the entire population of APE patients, but also in the population of normotensive patients.

An ECG is one of the first assessments carried out in a patient presenting with chest pain or dyspnoea. It should be emphasised that there are no sensitive or specific ECG signs of APE. While the ECG is not intended to establish the diagnosis of APE, it may be helpful in the differential diagnosis between APE and other acute cardiovascular conditions. The role of ECG in risk stratification is currently not taken into account. Only a few reports have so far addressed ECG changes and their relation to the markers of myocardial injury (troponin). Kostrubiec et al. [4] showed that negative T waves and ST-segment depression were significantly more common in patients with APE and elevated troponin levels than in patients with normal troponin levels (97% vs 75%).

In our study, ST-segment depression in V₄-V₆ was observed in 25.8% of the patients, similarly to the study by Kaczyńska et al. [5], in which ST-segment depression was observed in 24% of the patients. In that study, these changes were significantly more common in the subgroup with elevated troponin T levels compared to patients without elevated enzymatic markers of myocardial injury (41.4% vs 0%, $p = 0.004$). The ST-segment depression also showed a significant association with an increased risk of death or in-hospital complications [5].

The parameters associated with in-hospitalisation mortality in the logistic regression analysis in our study included: coronary chest pain, systolic blood pressure < 100 mm Hg and heart rate > 100 bpm. In the prognostic scale proposed by Aujesky et al. [6], included in the current ESC 2008 guidelines, systolic blood pressure < 100 mm Hg and tachycardia > 110 bpm were the clinical predictors of 30-day mortality in the course of APE [1].

The S1Q3T3 sign and QR in V₁ are the ECG markers of RV strain/dysfunction. The RVD is associated with increased mortality, stratifying patients to the group of moderate risk according to the current ESC 2008 guidelines (mortality rate: 3–15%) [1]. The ECG changes in the course of APE typical of myocardial ischaemia, such as ST-segment depression in V₄-V₆ or ST-segment elevation in III and/or V₁ were also associated with the risk of death in our study. This phenomenon may be associated with the more frequent occurrence of elevated troponin levels in this group of patients, and — as is widely recognised — elevated troponin levels are associated with poor prognosis. Patients with elevated troponin levels belong to the group of moderate risk of death (mortality rate: 3–15%) [1]. In addition, changes of the ST-segment elevation/depression type may erroneously suggest acute coronary syndrome, delay the correct diagnosis and delay initiation of appropriate anticoagulant rather than antiplatelet treatment.

Our study points out to a much more common presence of certain ECG changes in APE patients with elevated troponin levels. These ECG changes, similarly to elevated troponin levels, are associated with a poorer prognosis in these patients.

CONCLUSIONS

1. The following changes were significantly more common in ECG from APE patients with elevated troponin levels compared to patients with normal troponin levels: the S1Q3T3 sign, presence of negative T waves in V₂-V₄, ST-segment depression in V₄-V₆, ST-segment elevation in III and V₁, QR in V₁ and a higher number of leads with negative T waves.
2. The ECG parameters suggesting a higher risk of in-hospital death in patients with APE included: the S1Q3T3 sign, QR in V₁, ST-segment depression in V₄-V₆, ST-segment elevation in III and ST-segment elevation in V₁.

Conflict of interest: none declared

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Znaczenie zmian elektrokardiograficznych w prognozowaniu uszkodzenia miokardium i rokowaniu u chorych z ostrym zatorem tętnicy płucnej

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Streszczenie

Wstęp: Badanie elektrokardiograficzne (EKG) nie ma ani swoistych, ani czułych kryteriów i objawów służących do rozpoznania ostrego zatoru tętnicy płucnej (OZTP).

Cel: Celem pracy była ocena znaczenia zmian EKG w prognozowaniu uszkodzenia miokardium i rokowania chorych z OZTP.

Metody: Do badania włączono 225 chorych (137 kobiety, 8 mężczyzn) w wieku 17–89 lat (śr. wiek 66,0 ± 15,2 roku), u których rozpoznano OZTP [przede wszystkim na podstawie tomografii komputerowej — u 206 (92%) badanych].

Wyniki: W trakcie hospitalizacji wystąpiło 26 zgonów (śmiertelność 11.5%), u 58 (25.7%) chorych zaobserwowano powikłania. U 103 (46%) badanych stwierdzono podwyższone stężenie troponiny. Analiza regresji logistycznej wykazała istotny wpływ na występowanie zgonu w trakcie obserwacji wewnątrzszpitalnej następujących parametrów: skurczowe ciśnienie tętnicze < 100 mm Hg (2,3–13,64; OR 5,61), częstotliwość rytmu serca > 100/min (1,17–15,11; OR 4,21), objaw SI-QIII-TIII (1,31–6,99; OR 3,02), objaw QR w odprowadzeniu V₁ (1,60–12,32; OR 4,45), obniżenie odcinka ST w odprowadzeniach V₄–V₆ (0,99–5,40; OR 2,31), uniesienie odcinka ST w III (0,99–6,96; OR 2,64), uniesienie odcinka ST w V₁ (1,74–9,49; OR 4,07) oraz graniczny (1,51–16,07; OR 4,93), umiarkowany (1,42–17,74; OR 5,01) i znaczny wzrost (2,88–36,38; OR 10,24) stężenia troponiny. W EKG u chorych z podwyższonym stężeniem troponiny w porównaniu z osobami z prawidłowym jej stężeniem znamienne statystycznie częściej obserwowano: objaw McGinnea-White'a (43 v. 21%; p = 0,0003), obecność ujemnych załamek T w odprowadzeniach V₂–V₄ (57 v. 27%; p = 0,0001), obniżenie odcinka T w odprowadzeniach V₄–V₆ (40 v. 14%; p = 0,0001), uniesienie odcinka ST w odprowadzeniach: III (22 v. 7%; p = 0,0006) i V₁ (43 v. 10%; p = 0,0001), objaw Kuchera (16 v. 5%; p = 0,007) oraz więcej odprowadzeń z ujemnymi załamekami T (4 v. 2%; p < 0,001).

Wnioski: U chorych z OZTP parametry elektrokardiograficzne są pomocne w prognozowaniu uszkodzenia miokardium i określeniu rokowania w okresie wewnątrzszpitalnym.

Słowa kluczowe: zator tętnicy płucnej, EKG, troponina, powikłania

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