Electrocardiographic features in critical pulmonary embolism. Results from baseline and continuous electrocardiographic monitoring

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Key words:
Electrocardiography; Pulmonary embolism.

Background. **Pulmonary embolism (PE) afflicts millions of individuals worldwide. Electrocardiography along with chest X-ray and arterial blood gas analysis represent the basic examinations to reinforce the clinical suspicion of PE. We describe the electrocardiographic (ECG) features in a series of patients with PE and a critical clinical presentation.**

Methods. **We report the ECG findings registered at baseline, 48 hours after admission and on continuous ECG monitoring in 51 patients with PE and critical clinical conditions.**

Results. **At admission, the following parameters were recorded: an S1Q3 pattern in 34 patients, a "septal embolic pattern" in 27, anterior lead T-wave inversion in 8, and a new right bundle branch block in 7. At 48 hours after admission a trend toward a regression of the S1Q3 and "septal embolic" patterns was noted together with evident T-wave inversion in the anterior leads. During continuous ECG monitoring no major arrhythmias were recorded, even in case of cardiopulmonary arrest.**

Conclusions. **Critical PE induces transient ECG abnormalities reflecting right ventricular overload and/or strain. The patient's clinical status is usually not complicated by major ventricular arrhythmias, not even in case of cardiopulmonary arrest.**

(Ital Heart J 2004; 5 (3): 214-216)

Received December 9, 2003; accepted January 15, 2004.

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Introduction

Pulmonary embolism (PE) is a major international health problem with a mortality of 2-8% in adequately treated (anticoagulants) patients[1,2]. Due to the non-specific nature of the symptoms and signs, the diagnosis is often difficult to make and is frequently missed. Electrocardiography along with chest X-ray and arterial blood gas analysis represent the basic examinations to reinforce the clinical suspicion of PE[3]. The purpose of the present report was to describe the electrocardiographic (ECG) findings in a series of patients with PE and in critical clinical conditions.

Methods

The study population consisted of 51 patients (20 males, 31 females, mean age 73.6 ± 8.4 years) with PE and a critical clinical presentation[4] admitted to the coronary care units (CCU) of the Tortona (from 1986 to 1996) and Galatina General Hospitals (from 1997 until 2002). The majority of cases (38/51, 74.5%) were outpatients, admitted via emergency room. In the remaining cases, the PE occurred during hospitalization for surgical or medical indications.

Each patient underwent clinical evaluation, baseline standard 12-lead electrocardiogram and continuous ECG monitoring (from admission until discharge or death) by means of a radiotelemetric system and two-dimensional transthoracic echocardiographic color Doppler exam. Serial serum enzyme analysis (lactate dehydrogenase, aspartate aminotransferase, creatine kinase, creatine kinase-MB) was also performed (at CCU admission and every 12 hours until the fourth day of hospitalization). Furthermore the levels of plasma D-dimer were assayed by the ELISA method.

All electrocardiograms were analyzed at baseline and 48 hours after admission in order to assess the presence of one or more of the following parameters: a) S1Q3 pattern; b) "septal embolic pattern" (defined

by the presence of at least two of the following findings: i) incomplete right bundle branch block; ii) ST-segment elevation in lead V_1; iii) T-wave inversion in lead V_1 or V_1-V_2 or V_1-V_3); c) inferior pseudo-infarction pattern (Q wave and ST-segment elevation in at least one of the inferior leads); d) complete, not previously diagnosed, right bundle branch block.

Any complex cardiac arrhythmia was automatically recorded on paper. If reversible or irreversible circulatory arrest occurred, the cardiac rhythm was recorded on paper during the resuscitation maneuvers. All data were collected by an experienced cardiologist (MC) who worked during the respective years at the above-mentioned hospitals.

Results

The majority of patients (51%) had a positive medical history for deep venous thrombosis. Severe acute dyspnea was the most common presenting symptom (94%); it was associated with chest pain, syncope and cough in respectively 19, 10 and 6 cases. Clinical signs of right heart failure (neck vein enlargement and/or hepatic enlargement and/or a systolic murmur indicative of tricuspid regurgitation) were present in 16 patients (Table I). Seventeen (33%) patients died during hospitalization. Of these 7 (41%) died within the first 48 hours.

On admission, 34 (67%) and 27 patients (53%) presented with an S1Q3 and a "septal embolic" pattern respectively. A trend toward a regression of the S1Q3 and "septal embolic" patterns together with evident T-wave inversion in the anterior leads were noted 48 hours later (Table II). On continuous ECG monitoring most patients (80%) had persistent sinus tachycardia; no one developed major ventricular arrhythmias (Table III). Twenty episodes of cardiopulmonary arrest (CPA) were observed. In 15 cases CPA complicated the clinical course of a first episode of PE. In the remaining 5 cases, CPA developed in relation to an intrahospital relapse of PE. Only 3 of these 20 patients (15%) were successfully resuscitated. All episodes of CPA were characterized by pulseless electrical activity. In 19/20 cases, sinus rhythm was observed at the beginning of the pulseless electrical activity episodes (regular in 3 cases; tachycardic in 16 cases). In one case, a high-grade sino-atrial block (or sinus arrest) with a slow junctional rhythm was registered. Major ventricular arrhythmias such as ventricular flutter/fibrillation or ventricular tachycardia were never detected.

In all patients, two-dimensional echo revealed a dilated right atrium and ventricle and hypokinesia of the right ventricle with an abnormal septal motion as for right ventricular pressure overload. No inspiratory collapse of the inferior vena cava was noted. Color Doppler analysis showed ≥ moderate tricuspid regurgitation with a pulmonary artery systolic pressure ≥ 60 mmHg. Right atrial thrombi in transit were detected in only 2 patients. The left ventricle was relatively small with a preserved global and segmental systolic function. No major left heart cardiac abnormalities were noted.

In 49 patients the maximal lactate dehydrogenase values were above the normal range. Plasma D-dimer levels were found > 500 ng/ml.

Table I. Demographic and clinical characteristics.

Age (years)	73.6 ± 8.4
Sex (M/F)	20/31
Associated conditions	
Deep vein thrombosis	26 (51%)
Cancer	10 (20%)
Diabetes	2 (4%)
Cerebrovascular accident	2 (4%)
Presenting symptoms	
Dyspnea	48 (94%)
Chest pain	22 (43%)
Syncope	13 (25%)
Cough	6 (11%)
Presenting signs of right heart failure	16 (31%)

Table II. Electrocardiographic (ECG) patterns on admission and 48 hours later.

ECG pattern	At admission (n=51)	48 hours later* (n=44)
S1Q3	34 (67%)	10 (20%)
Septal embolic pattern	27 (53%)	9 (18%)
T-wave inversion in anterior leads	8 (16%)	30 (59%)
New right bundle branch block	7 (14%)	1 (2%)
Inferior infarct pattern	2 (4%)	0

* 7 patients (6 with an S1Q3 pattern and/or a septal embolic pattern and 1 with new right bundle branch block) died within 48 hours of admission.

Table III. Arrhythmias on continuous electrocardiographic monitoring.

Persistent sinus tachycardia	41 (80%)
Frequent premature supraventricular beats	8 (16%)
Frequent premature ventricular beats	6 (12%)
Paroxysmal atrial fibrillation	6 (12%)
Junctional rhythm	2 (4%)
Paroxysmal atrial flutter	1 (2%)
Accelerated idioventricular rhythm	1 (2%)

Discussion

PE afflicts millions of individuals worldwide with at least 60 000 new cases per year in Italy[1]. Due to the wide range of clinical presentations, a high index of clinical suspicion is required in order to avoid missing the diagnosis. Along with a careful clinical evaluation, chest X-ray and blood gas analysis, electrocardiogra-

phy is considered to be a first-line diagnostic test in order to assess the clinical probability of PE and the overall conditions of the patient[1].

In our series we have confirmed that in patients with PE and critical clinical conditions the baseline electrocardiogram at the time of hospital admission shows signs reflecting right ventricular overload and/or strain[5-9]. An acute pathologic increase in the pulmonary pressures and the consequent right ventricular dilation in combination with hypoxemia may be considered determinant causative factors of the ECG abnormalities observed[10]. It was remarkable that the electrocardiogram appeared within normal limits in 3/51 cases (5.9%) underlying the fact that the absence of ECG changes does not rule out critical PE. In a later phase, we have also observed a time-dependent ECG evolution characterized mainly by a trend toward a gradual regression of the S1Q3 pattern, a disappearance of the "septal embolic pattern", and a high incidence of gradual T-wave inversion in the anterior leads (Table III). Considering the transient and changing nature of the ECG abnormalities, in PE serial ECG recordings are recommended[7,11]. Thus, a single electrocardiogram should be interpreted as a "frame in a movie". Additionally, in our study population no major arrhythmias were registered during continuous ECG monitoring. In fact, the most frequent arrhythmia observed was sinus tachycardia with only 6 patients presenting with frequent isolated premature ventricular beats. Furthermore, in all cases CPA never occurred as a consequence of severe cardiac arrhythmias. More studies are warranted for a better understanding of the pathophysiologic processes affecting right-sided cardiac conduction and repolarization in the presence of an acute elevation of the right ventricular pressure and right ventricular subendocardial ischemia[12-16]. Finally, we evaluated only a limited number of highly selected cases. As such our findings cannot be considered as being applicable to all PE patients.

In conclusion, critical PE induces transient ECG abnormalities reflecting right ventricular overload or strain. The patient's clinical status is usually not complicated by major ventricular arrhythmias. This holds true even in case of CPA.

Acknowledgments

We are grateful to Catherine Klersy and Marcello Chimienti for their precious suggestions and support.

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