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Cardiac troponin I release in acute pulmonary embolism in relation to the duration of symptoms

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Abstract

Purpose: To evaluate the release of cardiac troponin I in normotensive patients with acute pulmonary embolism in relation to the duration of symptoms. *Methods:* Fifty-seven normotensive patients with acute pulmonary embolism were included in the study. Patients were divided into two groups based on the duration of symptoms at presentation: symptoms of ≤72 h, group A; symptoms of >72 h, group B. Serum cardiac troponin I levels were measured at presentation. *Results:* Mean age was 63 ± 18 years and 23 (40%) patients were males. Thirty-three (58%) patients had symptoms of ≤72 h (group A) and 24 (42%) had symptoms of >72 h (group B). Both groups had similar prevalence of right ventricular dysfunction on echocardiography (55% [$n=18$] in group A vs. 42% [$n=10$] in group B, $p=$NS). Sixteen patients had elevated serum cardiac troponin I (mean ± S.D. 3.3 ± 2.3 ng/ml, range 0.6–8.3 ng/ml). Elevated serum cardiac troponin I was strongly associated with right ventricular dysfunction ($p=0.015$). All patients with elevated serum cardiac troponin I ($n=16$) were in group A ($p<0.0001$). Twelve of 18 (67%) patients with ($p=0.0005$) and 4 of 15 (27%) patients without ($p=$NS) right ventricular dysfunction had elevated serum cardiac troponin I. Thirteen of 16 (81%) patients with elevated serum cardiac troponin I had duration of symptoms ≤24 h at presentation. *Conclusions:* The dynamics of cardiac troponin I release in acute pulmonary embolism in patients who present with symptoms of ≤72 h duration could be different from those who present with longer duration of symptoms. Therefore, the use of cardiac troponin I in risk stratification of acute pulmonary embolism might be limited to the patients presenting within 72 h of the onset of symptoms.

Keywords: Cardiac troponin I; Pulmonary embolism; Risk stratification; Cardiac markers; Pulmonary circulation; Right ventricular dysfunction

1. Introduction

Cardiac troponins are highly sensitive and specific markers of myocardial injury, and their role in acute coronary syndrome is well established [1–4]. Increased serum levels of cardiac troponins in patients with pulmonary embolism identify patients with right ventricular dysfunction, which is of paramount importance, as right ventricular dysfunction correlates with increased risk of death [5–10]. Cardiac troponin I have been identified to have independent prognostic value in acute pulmonary embolism [11,12]. The release of cardiac troponin I in relation to duration of symptoms is well known in acute coronary syndrome [1,13,14]. To use cardiac troponin I in risk stratification of acute pulmonary embolism, it is imperative to know its release in relation to the duration of symptom in acute pulmonary embolism. This study was performed to evaluate relation of the duration of symptoms and the serum cardiac troponin I elevation at the time of clinical presentation in patients with acute pulmonary embolism.

2. Patients and methods

2.1. Study population

The study population included 57 consecutive patients with confirmed diagnosis of acute pulmonary embolism admitted in Long Island College Hospital. Diagnosis of pulmonary embolism was confirmed by high probability ventilation–perfusion scan, high-resolution computerized

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Table 1
Baseline characteristics

Characteristics	Number of patients
Study population	57
Males	23 (40%)
Presenting symptoms	
Shortness of breath	42 (74%)
Chest pain	20 (35%)
Syncope	7 (12%)
Cardiovascular risk factors	
Hypertension	33 (58%)
Diabetes mellitus	11 (19%)
Smoking	9 (16%)
Thromboembolic risk factors	
Malignancy	12 (21%)
Recent surgery	5 (9%)
Immobilization	3 (5%)
Obesity	10 (17%)
Diagnostic work up	
Ventilation perfusion scan	36 (63%)
Computerized tomography of lungs	36 (63%)
Venous Duplex of lower extremities	57 (100%)
Echocardiography	57 (100%)
Right ventricular dysfunction	28 (49%)
Deep vein thrombosis	7 (12%)

tomography of chest or both. All patients underwent transthoracic echocardiography and bilateral venous Duplex study of the lower extremities on presentation or within 24 h. Data were collected on symptoms and thromboembolic and cardiovascular risk factors. No follow-up data on outcome was collected. The exclusion factors were persistent systemic hypotension or cardiogenic shock, duration of symptoms more than 14 days, concomitant sepsis, chronic stable angina pectoris and history of myocardial infarction or percutaneous coronary intervention or coronary artery bypass grafting.

2.2. Echocardiographic data

Patients with ≥ 1 of the following were considered to have right ventricular dysfunction: (1) right ventricular dilatation (end-diastolic diameter >30 mm or right ventricular/left ventricular end-diastolic diameter ratio >1 in four-chamber view), (2) paradoxical septal systolic motion and (3) pulmonary hypertension (pulmonary acceleration time <90 ms or presence of a right ventricular/right atrial gradient >30 mm Hg). However, these signs of right ventricular dysfunction were not considered acute in the presence of right ventricular wall hypertrophy.

2.3. Laboratory data

At clinical presentation specimens were collected in serum separator tubes without any additive for cardiac troponin I, creatine kinase and MB-fraction of creatine kinase. Analysis was carried out within 1–2 h of collection. Cardiac troponin I was measured by the Stratus II fluorometric enzyme immunoassay (Dade, Miami, FL) for cardiac troponin I by technologists unaware of the clinical data. This assay uses two monoclonal antibodies that recognize two different epitopes on the cardiac troponin I molecule. No cross-reactivity is seen with troponin I found in human skeletal muscle. In serum specimens from healthy persons without evidence of cardiac disease, the cardiac troponin I concentration is below the minimal concentration detectable by the assay (the smallest concentration that can be distinguished from zero), or 0.35 ng of cardiac troponin I per milliliter. The interassay imprecision for cardiac troponin I was $<10\%$. Creatine kinase was measured on Hitachi 747 Analyzer (Hitachi, Indianapolis, IN) by using enzymatic method according to manufacturer recommended protocol and creatine kinase-MB was measured on Abbott AXSYM System (Abbott Laboratories, Abbott Park, IL) by using microparticle enzyme immunoassay (MEIA). The serum levels of cardiac markers above following values were considered as elevated: cardiac troponin-I 0.4 ng/ml, creatine kinase 250 U/l and creatine kinase-MB 5 ng/ml.

2.4. Statistics

Data were expressed as means $\pm$ S.D. Continuous variables were analyzed by using Student's unpaired *t*-test. Dichotomous variables were expressed as percent and analyzed using the chi-square statistics. A *P*-value of 0.05 or less was considered significant. All statistical analyses were performed using statistical software MINITAB (Minitab, College Station, PA, USA).

3. Results

The baseline characteristics of the study population are given in Table 1. Mean age was 63 ± 18 years and 23 (40%) patients were males. Forty-two (74%) patients presented with shortness of breath, 20 (35%) with chest pain and 7 (12%) with syncope. Malignancy (21%) and obesity (17%) were two most common risk factors for

Table 2
Comparison based on the duration of symptoms

Characteristics	Symptoms $\leq$72 h ($n=33$)	Symptoms >72 h ($n=24$)	*P*-value
Age (years)	63 ± 17	64 ± 19	NS
Males	14 (42%)	9 (38%)	NS
Hypertension	20 (61%)	13 (54%)	NS
Diabetes mellitus	6 (18%)	5 (21%)	NS
Smoking	5 (15%)	4 (17%)	NS
Right ventricular dysfunction	18 (55%)	10 (42%)	NS
Normal right ventricular function	15 (45%)	14 (58%)	NS
Elevated cardiac troponin I	16 (48%)	None	<0.0001
Elevated cardiac troponin I with right ventricular dysfunction	12 (67%)	None	0.0005
Elevated cardiac troponin I with normal right ventricular function	4 (27%)	None	NS

All values are number of patients except for age.