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Endothelial dysfunction evaluated by peripheral arterial tonometry is related with peak TnI values in patients with ST elevation myocardial infarction treated with primary angioplasty

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ABSTRACT

Purpose: The role of endothelial-dependent function in patients with acute ST elevation myocardial infarction (STEMI) is not clear. Endothelial dysfunction may contribute to the pathophysiological processes occurring after STEMI and influence the extension of myocardial necrosis. Endothelial-dependent dysfunction evaluated by peripheral arterial tonometry (PAT) has already showed to be correlated with microvascular coronary endothelial dysfunction. Our purpose was to evaluate the impact of endothelial dysfunction on peak Troponin I (TnI) values, as a surrogate for the extension of myocardial infarction, in patients with STEMI treated with primary angioplasty (P-PCI).

Methods: 58 patients with STEMI treated with P-PCI (mean age 59.0 ± 14.0 years, 46 males) were included. Endothelial function was assessed by reactive hyperaemia index (RHI) determined by PAT. Patients were divided in two groups according to the previously reported RHI threshold for high risk (1.67). The extension of myocardial necrosis was evaluated by peak TnI levels.

Results: RHI median value was 1.78 (IQR 0.74); 25 patients had endothelial dysfunction (RHI < 1.67). The two groups had no significant differences in age, gender, main risk factors and pain-to-balloon time. Patients with an RHI < 1.67 had significant larger infarcts: TnI 73.5 ng/mL (IQR 114.42 ng/mL) versus TnI 33.2 ng/mL (IQR 65.2 ng/mL); $p = 0.028$. On multivariate analysis, the presence of an RHI < 1.67 kept significant impact on TnI peak values ($p = 0.02$).

Conclusions: The presence of endothelial-dependent dysfunction, assessed by PAT, is related with higher peak TnI values in STEMI patients treated with P-PCI. These results strength the possibility that endothelial-dependent dysfunction may be a marker of poor prognosis and eventually a therapeutic target in patients with STEMI.

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1. Introduction

Primary percutaneous coronary intervention (P-PCI) is an established reperfusion strategy in the treatment of acute myocardial infarction with ST-segment elevation (STEMI) (Steg et al., 2012; PT et al., 2013). Despite achieving normal epicardial coronary artery flow after P-PCI, a significant proportion of patients (ranging between 20% and 40%) have a poor outcome because of microvascular coronary damage (Bekkers et al., 2010). The extent of microvascular dysfunction has been shown to be an important and independent contributor to subsequent changes in left ventricular geometry and performance (Bolognese et al., 2004). Patients with impaired microvascular perfusion have larger infarcts, as evaluated by CK and troponin release, less electrocardiographic ST elevation resolution, larger long-term left ventricular wall motion abnormalities and lower left ventricular ejection fraction (Van't Hof et al., 1997; Amaya et al., 2011; Desmet et al., 2004; Ito et al., 1996). As a consequence of these, myocardial malperfusion is associated with higher event rates, risk of progression to heart failure and mortality (Schröder et al., 2001; Jaffe et al., 2010).

The precise mechanisms underlying myocardial malperfusion after the restoration of epicardial blood flow are not known and probably are multifactorial: endothelium-independent microvascular dysfunction (caused by thrombi, debris, embolization, ventricular hypertrophy, myocardial, and vascular oedema and smooth muscle dysfunction) is one of the mechanisms involved (Luscher and LA, 2012; Lerman et al., 2007). The contribution of endothelial-dependent dysfunction, on the other hand, is less well established, even knowing that it plays a crucial role in vascular tone and blood flow regulation (Gutiérrez et al., 2013), which are main determinants of myocardial infarction extension. Several techniques were recently developed that allow the evaluation of endothelial-dependent dysfunction. Peripheral arterial tonometry

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(PAT), a simple and reproducible technique (CE et al., 2012) based on the change in finger pulse wave amplitude in response to reactive hyperaemia, is closely correlated with coronary artery endothelial function (Bonetti et al., 2004), allowing a non-invasive evaluation of endothelial-dependent dysfunction.

We hypothesized that endothelial-dependent dysfunction (evaluated with PAT), as a determinant of coronary microvascular flow, could have an impact on the extension of myocardial infarction, measured by peak Troponin I (TnI) values.

2. Methods

2.1. Study population

A total of 92 consecutive patients with acute ST elevation myocardial infarction submitted to primary angioplasty admitted in a single center between March and October 2011 were prospectively screened for inclusion in the study. After excluding patients that died (n = 6), patients with residual ischemia (including patients still needing nitrates for pain relief) and hemodynamic or rhythm instability in the first 24 h (n = 6), patients admitted during weekend days (in which performing the endothelial evaluation was not possible, due to logistical reasons; n = 10) and patients not willing to participate or not able to consent (n = 8), 62 patients were considered for endothelial function evaluation. Of these, 4 were excluded, due to low quality or technical problems in the endothelial function evaluation (Fig. 1). Fifty eight patients were finally included in the study protocol (68% of the screened patients). The institutional ethical committee approved the study, which was conducted in compliance with the Declaration of Helsinki.

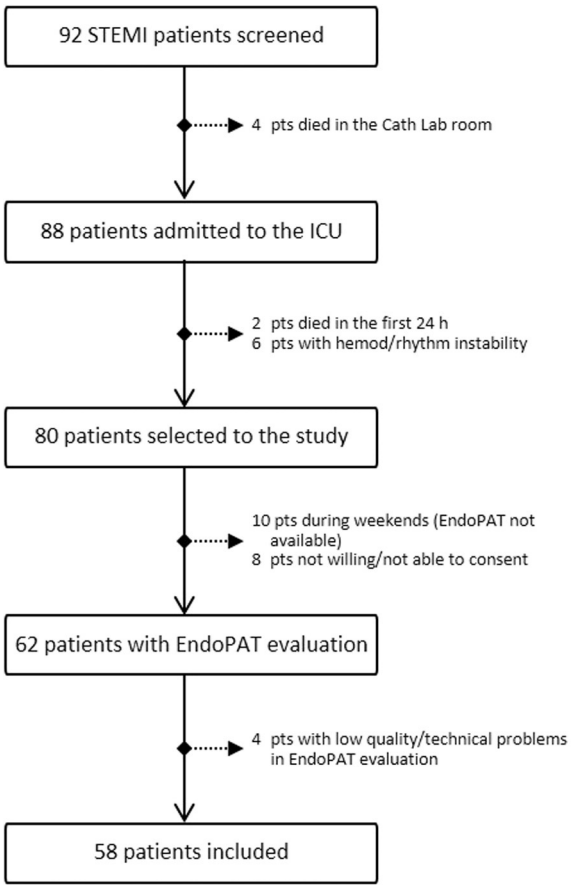

Fig. 1. Flow chart of patients included. (STEMI: ST elevation acute myocardial infarction; ICU: Intensive Care Unit; pts: patients.)

Clinical characteristics (age, gender, cardiovascular risk factors and clinical evolution), left ventricular ejection fraction (measured by echocardiography) and main angiographic characteristics (culprit coronary artery, number of vessels with disease) were assessed in all patients.

2.2. Endothelial function evaluation

Peripheral endothelial function was measured in the next morning after the acute event by digital pulse amplitude with the Endothelial Peripheral Arterial Tonometry (Endo-PAT2000, Itamar Medical, Caesarea, Israel), as previously described (Bonetti et al., 2004; Bonetti et al., 2003). Briefly, a blood pressure cuff was placed on 1 upper arm, while the contralateral arm served as a control. The peripheral arterial tonometric finger probe was placed on each index finger, and after a baseline period of 5 min, total occlusion was performed with a blood pressure cuff on the right arm for 5 min. The cuff was inflated to suprasystolic pressures to obtain complete occlusion. Thereafter, the cuff was deflated and the hyperemic response of the occluded right arm was recorded and compared to the nonoccluded left arm (control arm). The data were digitally analyzed (EndoPAT2000 software version 3.4.4) to obtain the reactive hyperemia index (RHI). RHI values below 1.67 were considered as endothelial dysfunction (ED), as previously reported (Bonetti et al., 2004).

2.3. Laboratory analysis

Blood samples were collected at the time of the primary angioplasty and 6, 12, 18, 24 and 48 h after the procedure, according to the usual Intensive Care Unit (ICU) protocol for acute myocardial infarction patients. Troponin I was measured by using sandwich chemiluminescent immunoassay based LOCI™ technology, with a Dimension Vista™ Intelligent Lab System (Siemens Healthcare Diagnostics™). The peak value for each patient was recorded. Creatin Kinase (CK) and its subfraction Mb (CK-Mb) were also measured. High sensitivity CRP, glucose, lipid profile and NT-pro-BNP levels were determined as part of the standard admission evaluation of these patients in the ICU.

2.4. Statistical analysis

Statistical analysis was performed using IBM® SPSS® Statistics 20.0. Data are presented as mean ± SD or median (interquartile range) for continuous variables and as number and frequencies for categorical variables. Distribution was tested with the Kolmogorove–Smirnov test. Continuous variables were compared using the Student t if they had a normal distribution and with the Mann–Whitney test if they didn't. Categorical variables were tested using the χ^2 test.

To evaluate the impact of different variables in troponin I values, a univariate statistical analysis was done. Variables that showed a significant impact on troponin I, as well as variables considered clinically relevant, were include in a linear multivariate regression model, in order to identify independent predictors of troponin I values. A 2-sided p value of <0.05 was considered statistically significant.

3. Results

3.1. Population characteristics and RHI values

Baseline characteristics are presented in Table 1. Forty-six patients (79.3%) were male and the mean age was 59.0 ± 14.0 years. The median value of RHI was 1.78 (IQR 0.74). Twenty-five (43%) patients had endothelial dysfunction (defined as a RHI below 1.67). Fig. 2 shows endoPAT evaluations in patients with (2B) and without (2A) endothelial dysfunction.

There were no significant differences between the groups of patients with and without endothelial dysfunction concerning age, gender, and traditional risk factors (although there was a trend for more patients

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