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ORIGINAL ARTICLE

# Circadian Rhythm of Infarct Size and Left Ventricular Function Evaluated with Tissue Doppler Echocardiography in ST Elevation Myocardial Infarction

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**Background** We aimed to investigate the circadian rhythm on left ventricular (LV) function and infarct size, according to the onset of ST elevation myocardial infarction (STEMI), with echocardiography in patients with first STEMI successfully revascularised with primary percutaneous coronary intervention (PCI).

**Methods** We conducted a retrospective analysis of 252 STEMI patients. Patients were divided into the four, six-hour periods of the day. Conventional and tissue Doppler imaging (TDI) echocardiography were performed within 48 hours after onset of chest pain. The average of peak systolic myocardial velocities (Sm) in each of the four myocardial segments and LV ejection fraction (LVEF) were calculated.

**Results** A negative linear correlation was shown between CK-MB levels and Sm (r= -0.209, p = 0.001). There was an oscillation between time of day and average of Sm. The lowest Sm and largest infarct size were in the period of 06:00-noon compared with period of noon-18:00 and 18:00-midnight (p = 0.029 and p = 0.031, respectively). A secondary analysis showed that both LVEF and Sm were lower in the midnight-noon group compared with the noon-midnight group (44.9 ± 7.3% versus 47.3 ± 7.9%, p = 0.018, and 7.6 ± 1.4 cm/s versus 8.2 ± 1.6 cm/s, p=0.003, respectively).

**Conclusions** This study has shown that there was a circadian rhythm of infarct size and LV function evaluated by echocardiography according to time of STEMI onset. The largest infarct size and poor LV function occurred in the midnight-noon period, in particular in the 06:00-noon period.

**Keywords** Circadian • Myocardial infarct size • Myocardial infarction • Tissue Doppler • Left ventricular function • Echocardiography

## Introduction

It is well known that circadian rhythm influences cardiovascular pathophysiology and events such as heart-rate, blood pressure, circulating hormones, stent thrombosis, and sudden cardiac death [1–4]. It is also known that clinical manifestations of acute myocardial infarction (AMI) have a peak incidence between 06:00-noon [5,6]. In recent years, a study

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demonstrated that the cardiomyocyte circadian clock mediates time of dependence for myocardial reperfusion injury tolerance. The authors observed that the largest myocardial injury occurred at sleep to wake time [7]. Retrospective analysis of previous cohorts of AMI patients provided contradictory results [8,9]. Suares et al. observed that infarct size was larger with onset of symptoms between 06:00-noon [8]. However, Reiter et al. showed that the greater myocardial injury occurred at a 01:00 am onset of ischaemia [9]. There are still not enough data about the effect of the circadian clock on infarct size and LV function measured by comprehensive echocardiographic parameters in patients with ST elevation AMI.

The aim of this study was to investigate the effect of circadian rhythm on LV function and infarct size according to the onset of STEMI with comprehensive echocardiographic parameters in patients with first STEMI successfully revascularised with primary PCI.

# Methods

The methodology was previously used to answer a different research question, and described in a previously published work [10].

## Study Population

A retrospective analysis was performed on all consecutive STEMI patients enrolled in a single-centre registry, without history of MI, heart failure, by-pass surgery and cardiac arrest or cardiopulmonary resuscitation, who were admitted to the coronary care unit of Selcuk University, Faculty of Medicine Hospital, Konya, Turkey, from 2008-2009. Patients' variables of 1) time onset of symptoms; 2) arrival at the emergency department; and 3) first balloon inflation time were analysed. Demographic, clinical, and angiographic variables were reviewed. Peak MB fraction of creatinine kinase (CK-MB) was also analysed. Patients who met the following criteria were excluded from the analysis: 1) time of symptom onset unknown; 2) first balloon inflation time unknown; 3) treated with thrombolytic therapy; 4) underwent facilitated PCI; 5) initial thrombolysis in myocardial infarction (TIMI) flow > 0; 6) antegrade or retrograde collateral flow presence; 7) TIMI < 3 flow after primary PCI; 8) ischaemic time more than six hours; 9) severe valvular heart disease; 10) Killip >2. The study was approved by the local ethics committee.

## Definitions

Acute MI is typical chest pain lasting for more than 30 min with ST segment elevation > 1 mm in 2 or more consecutive precordial or inferior leads [11].

There is a positive correlation between Sm and LVEF [12–14]. Furthermore, global strain and the average of Sm of each myocardial segment obtained by TDI is a significant predictor of post-MI LVEF and infarct size [15,16]. Therefore, the averages of Sm of each myocardial segment were used to define infarct size in successfully revascularised patients with STEMI.

The ischaemic times were defined as the time difference between the onset of chest pain to first balloon inflation during primary PCI.

Time of day of STEMI onset was divided into four, six-hour groups according to previous studies (group 1: midnight-06:00, group 2: 06:00-noon [dark-to-light transition], group 3: noon-18:00, group 4: 18:00-midnight) [1,5,17,18].

## Coronary Angiography and PCI

All angiographies and PCIs were performed by means of an angiography unit (Integris Allura 9, Philips, Eindhoven, Netherlands). Coronary angiography was performed by the Judkins technique through femoral arterial access. Thrombolysis in myocardial infarction flow of the infarct-related artery was measured before and after the procedure [19]. The severity of coronary artery disease was assessed using the Gensini score [20]. Pre-PCI antiaggregant treatment of patients was as follows: oral administration of 300 mg of acetyl salicylic acid and 300–600 mg loading dose of clopidogrel, an intravenous 70 U/kg bolus dose of unfractionated heparin, followed by 12–15 U/kg adjusted according to activated partial thromboplastin time (60–80 s). Alternatively, enoxaparin was used in most of the patients as an anticoagulant with a dose of 1 mg/kg bid subcutaneously. Tirofiban administration was at the discretion of the physician performing the procedure; however, it was encouraged in case of high thrombus burden. Tirofiban was infused in two stages: 0.4 mc/kg/min during the first 30 min, then 0.1 mc/kg/min over 24 h. A bare metal stent was implanted in all patients.

## Echocardiography

All patients were evaluated by two-dimensional, pulsed wave Doppler echocardiography and colour Doppler TDI. Echocardiographic examination was performed within 48 hours after onset of chest pain by two experienced echocardiographers. Patients were examined in the supine and left lateral decubitus position using a Philips Envisor C echocardiograph (Philips Medical Systems, Andover, MA, USA) using a 3.5 MHz transducer. Echocardiograph tracing was obtained during ECG examination. The heart was imaged through multiple acoustic windows (parasternal, longitudinal, and cross-sectional, apical four-chamber, and two-chamber views). Left ventricular ejection fraction was determined from apical two- and four-chamber views using the Simpson's biplane formula [21]. We calculated the averages of two- and four-chamber LVEF. The colour Doppler TDI records were obtained on the same echocardiography machine using a 2.5 MHz transducer with a mean frame rate of 105 frames/s (80–140 frames/s) in the left lateral decubitus position during shallow respiration or end-expiratory apnoea. Guided by the two-dimensional four-chamber view, a sample volume (2-mm) was placed at the septal and lateral corner of the mitral annulus from the apical four-chamber view. The TDI cursor was placed at the anterior and inferior

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