The effect of Ramadan fasting on circadian variation of Turkish patients with acute myocardial infarction

Wpływ postu w ramadanie na dobową zmienność występowania ostrego zawału serca u tureckich pacjentów

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Abstract

Introduction: The mechanisms of circadian variation in cardiovascular events are not clear.

Aim: Evaluation of the effect of Ramadan fasting on circadian variation of acute ST-elevation myocardial infarction (STEMI) in Turkish patients.

Material and methods: This comparative cross-sectional study included 151 consecutive patients with acute STEMI. The time of onset of STEMI was determined by the attending physician on the basis of patients' self-reports. The patients were divided into two group based on the history of fasting. The standard hourly profile of the onset of STEMI was obtained over a 24-h period. Patients were then categorized into four 6-hour increments according to the time that the symptoms began (00:00–05:59; 06:00–11:59; 12:00–17:59 and 18:00–23:59 h).

Results: In all study participants, the highest incidence of STEMI was between 12.00 and 18.00; the odds ratio was 2.77 (95% CI: 2.63–2.92, $p < 0.001$). In the non-fasting group, the highest incidence of STEMI occurred between 06:00 and 12:00 A.M. The highest incidence of AMI occurred between 12.00 A.M. and 18.00 in patients with fasting; the odds ratio was 2.70 (95% CI: 2.51–2.91, $p < 0.001$). There were significant differences between fasting and non-fasting groups regarding circadian variation of STEMI ($p < 0.001$).

Conclusions: There are significant changes in the circadian variation of patients with STEMI in relation to Ramadan fasting.

Key words: circadian rhythm, myocardial infarction, Ramadan fasting.

Streszczenie

Wstęp: Dobowa zmienność występowania zdarzeń sercowo-naczyniowych jest niewyjaśniona.

Cel: Ocena wpływu postu w ramadanie na dobową zmienność występowania ostrego zespołu wieńcowego z uniesieniem odcinka ST (*ST-elevation myocardial infarction* – STEMI) u tureckich pacjentów

Materiał i metody: W porównawczym badaniu przekrojowym wzięło udział 151 kolejnych pacjentów ze STEMI. Czas wystąpienia STEMI był określany przez lekarza prowadzącego na podstawie relacji pacjenta. Pacjentów podzielono na dwie grupy w zależności od wywiadu stosowania postu. Określono godzinowy profil występowania STEMI w trakcie pełnych 24 godzin. Następnie pacjentów przydzielono do czterech grup w zależności od czasu, w którym wystąpiły objawy, według 6-godzinnych przedziałów (00:00–5:59; 6:00–11:59; 12:00–17:59 i 18:00–23:59).

Wyniki: U wszystkich uczestników badania odnotowano największą częstość wystąpienia STEMI w godzinach 12:00–18:00; iloraz szans wynosił 2,77 (95% CI: 2,63–2,92; $p < 0,001$). Największą częstość wystąpienia STEMI u chorych nieposzczących obserwowano w godzinach 6:00–12:00, a u pacjentów poszczących między 12:00 a 18:00; iloraz szans wynosił 2,70 (95% CI: 2,51–2,91; $p < 0,001$). Odnotowano istotne różnice w dobowej zmienności występowania STEMI u chorych poszczących i nieposzczących ($p < 0,001$).

Wnioski: Istnieje istotna zmienność dobowa występowania STEMI zależna od stosowania postu w ramadanie.

Słowa kluczowe: rytm dobowy, zawał serca, post w ramadanie

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Introduction

It has been previously reported that there is circadian periodicity of acute coronary events, with the highest rate between 8:00 A.M. and noon [1, 2]. One large-scale multicenter study has revealed that acute ST wave elevation myocardial infarction (STEMI) is 1.28 times more likely to begin between 6:00 A.M. and 12:00 P.M. than during the other three 6-hour intervals of the day [2]. The mechanisms of this predisposition are not clear [3]. Several factors have been proposed such as changes in catecholamine levels, fibrinolytic activity, blood pressure, platelet aggregability, coronary tone and endothelial function [4, 5]. A secondary peak in the circadian variation of acute thrombotic events has also been demonstrated [6]. This peak may be related to the ingestion of the main daily meal, in conjunction with post-prandial physical inactivity, irrespective of sleep [6].

Voluntary fasting, or the abstinence from food or drink or both, is practiced in many of the world's religions and cultures. Some of the changes associated with Ramadan may interfere with the circadian rhythm and biological clock of individuals during that month, which is manifested as a change in the circadian pattern of body temperature, a decrease in the amplitude of the melatonin rhythm and a nocturnal increase in cortisol level [7]. No interference of Ramadan with acute coronary syndromes was reported [8, 9]. However, Al Suwaidi *et al.* found significant changes in the circadian rhythm of acute heart events in fasting patients in Qatar [10]. Differences in the circadian variation of STEMI have also been reported in different regions of the world and ethnic groups [11, 12].

Aim

The aim of this study was to evaluate the effect of Ramadan fasting on circadian variation of STEMI in Turkish patients.

Material and methods

Subjects

This comparative cross-sectional study included 151 consecutive patients with acute STEMI who were admitted to the Coronary Care Unit of our centers (Duzce and Isparta) between 11th August 2010 and 8th September 2010 and also 1st August 2011 – 29th August 2011. Duzce is located on the plateau of the West Black Sea coast and its population is 338 000 (2010). Isparta is a city in the south-west of Turkey and its population is 407 463 (2010). Patients with STEMI could be treated only in our centers in each city. The diagnosis of STEMI was determined from the presence of ≥ 30 min of continuous chest pain, ST-segment elevation > 2.0 mm in ≥ 2 contiguous electrocardiographic leads, and more than a two-fold elevation of creatine kinase-MB (CK-MB) level. The time of onset of AMI was determined by the attending physician on the basis of patients' self-reports. Patients without chest pain and those who could not recall the time of the onset of chest pain, patients with STEMI and unstable angina pectoris, and patients experiencing STEMI after coronary bypass grafting or invasive cardiac procedures, who partly had a break in fasting were excluded from the study.

All the patients were treated according to the latest published guidelines and clinical practice. Primary percutaneous coronary intervention (PCI) is performed only in one center (Duzce), whereas thrombolytic treatment was used as reperfusion therapy in the other center (Isparta). The study protocol was approved by the Ethics Committee of Duzce University and every subject signed a written consent form. The patients were divided into two groups based on the history of fasting. The standard hourly profile of the onset of STEMI was obtained over a 24-h period. Patients were then categorized into four 6-hour increments according to the time that the symptoms began (00:00–05:59; 06:00–11:59; 12:00–17:59 and 18:00–23:59 h). Clinical characteristics of the population in each group were assessed: previous history of coronary artery disease (CAD), family history of CAD, prevalence of male gender, obesity, diabetes mellitus (DM), hypertension, smoking, and current medication. In each subject, blood pressure (BP) was measured on at least three separate days after 15 min of comfortably sitting and averaged. Individuals who had systolic BP ≥ 140 mm Hg and/or a diastolic BP ≥ 90 mm Hg or prior use of antihypertensive agents were diagnosed as hypertensive. Smoking was defined as current regular use of antihypertensive therapy. Obesity was defined as a body mass index (BMI) ≥ 30 kg/m^2. Diabetes mellitus was defined by the patient's self-report of such history, use of insulin or hypoglycemic agents or a fasting plasma glucose level measured on three separate days in a week > 126 mg/dl (7.0 mmol/l) or impaired oral glucose tolerance test: fasting plasma glucose < 126 mg/dl (7.0 mmol/l) but 2-h plasma glucose after a 75-g oral glucose challenge > 140 mg/dl (7.8 mmol/l). In addition major adverse cardiac events (MACE) (re-angina, heart failure after MI, re-MI, death) of patients were recorded in hospital and at 30 days after the onset of chest pain.

Echocardiography

In all subjects, transthoracic M-mode, two-dimensional, pulsed-wave, continuous-wave and color and Doppler echocardiographic examinations were performed using Vingmed System 5 (GE, Horten, Norway) and Vingmed System 7 (Vivid 7, GE, Horten, Norway). Left atrial and ventricular dimensions and left ventricular ejection fraction were measured by M-mode echocardiography in the parasternal long axis view by using the American Echocardiography Society M-mode technique [13]. The degree of native valvular regurgitation and stenosis were assessed by the method recommend by current guidelines [14].

Blood sampling

Blood Sample Collection and Hematological Assays Blood samples were drawn at initial presentation from the antecubital vein. Whole blood count and routine biochemical tests including cardiac enzymes were performed.

Statistical analysis

Continuous variables were expressed as mean ± SD and categorical variables were presented as percentages. Continuous variables were checked with Kolmogorov Smirnov test for normal distribution. Continuous variables were compared with Student *t* test for normally distributed values and with Mann-Whitney *U* test for abnormally distributed values. Categorical variables were compared with χ^2 test. The χ^2 was used to compare the time of onset of AMI within a 24-hour period (by a 6-h interval) between the groups. All the analyses were performed using SPSS (SPSS Inc. Chicago IL). A p value of < 0.05 (2-tailed) was considered significant.

Results

Clinical characteristics of the study population

One hundred fifty-one patients who were admitted with the diagnosis of STEMI during two Ramadan months (11th August 2010 – 8th September 2010 and 1st August 2011 – 29th August 2011) in our centers were included in the study. Of these, 55 patients (38.5%) were fasting on the day of presentation. The general characteristics, history and risk factors for CAD, hemodynamics, laboratory findings, and prior medications of the subjects in the two groups (fasting and not fasting) are presented in Table I. Age, gender, smoking status, body mass index and the frequency of hypertension, diabetes, heredity, and history of STEMI and prior PCI were similar between the groups. Additionally, heart rate, systolic and diastolic BPs, total cholesterol, LDL cholesterol, triglyceride, HDL cholesterol levels and peak CK-MB levels were similar between the groups. Also, there was no significant difference in the use of aspirin, β-blockers, and angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blocker (ARB) between the groups. Primary PCI and fibrinolytic therapies were similar in each group.

Comparison of the echocardiographic measurements, angiographic findings and clinical outcome

Echocardiographic findings were shown in Table II. Left ventricular ejection fraction (EF) was similar (42.9 ±5.9 vs. 41.5 ±8.5, p = 0.24) and there was no significant difference in the frequency of mitral regurgitation between the groups. There was no significant stenosis in the left main coronary artery in study patients. Also, other angiographic findings did not differ between the groups. Choices of treatment based on angiographic results were similar (Table II). Frequency of MACE (9% vs. 14%, p = 0.33) and heart failure were similar during hospitalization (7% vs. 7%, p = 0.99) and within 30 days (Table II). One patient died in fasting and three patients died in non-fasting group during hospitalization (p = 0.66) (Table II).

Analysis of circadian variation of acute myocardial infarction

In all study participants, the highest incidence of STEMI was between 12.00 and 18.00; the odds ratio was 2.77 (95 % CI: 2.63 to 2.92, p < 0.001). In the non-fasting group, the highest incidence of STEMI occurred between 06:00 and 12.00 A.M. The highest incidence of STEMI occurred between 12.00 A.M. and 18.00 in patients with fasting; the odds ratio was 2.70 (95% CI: 2.51-2.91, p < 0.001) (Figure 1). There were significant differences between fasting and non-fasting groups regarding circadian variation of STEMI (p < 0.001).

Discussion

The present study suggests that there was a circadian periodicity, with the highest rate of STEMI between 12.00 A.M. and 18.00 in patients with fasting.

The Multicenter Investigation of Limitation of Infarcted Size (MILIS) and Intravenous Streptokinase in Acute Myocardial Infarction (ISAM) studies have shown that circadian periodicity has been documented in all subgroups except patients taking β-blockers [2, 15] and aspirin [1]. However, Erdoğan *et al.* recently reported that prior use of statin did not impact circadian periodicity of STEMI. Although a morning peak of the onset of STEMI is very common, especially in western countries, several studies have reported a secondary peak at noon [16, 17]. Lunch is usually the main meal of the day in some countries including Turkey. Accordingly, Sari *et al.* recently reported that there was an afternoon predominance in circadian variation of AMI in a Turkish cohort. This study was done in Gaziantep, which is a province in south-central Turkey. Our centers are in the north and west of Turkey [11]. There are ethnic, cultural, and seasonal, temperature differences changes between these regions. These differences may have affected the circadian rhythm. Some studies have revealed that different circadian periodicity of STEMI results from not only ethnic differences but also patients' characteristics and lifestyle [17]. The presence of diabetes and other cardiovascular risk factors, smoking, eating habits, light and dark cycle, and sleep patterns may affect the circadian periodicity of cardiovascular events [16].

The major changes in Ramadan fasting concern meal schedule and frequency [18]. Meals are exclusively nocturnal and less frequent [18, 19]. Body weight slightly decreases during this month [18-20]. Studies have shown that the total cholesterol and HDL levels increase while LDL levels and LDL/HDL ratio decrease during Ramadan [20-22]. An increase of apoprotein A1 and its ratio to

Table 1. Clinical characteristics, laboratory findings and prior medications of the study groups
Tabela 1. *Charakterystyka kliniczna, wyniki laboratoryjne i wcześniejsze leczenie w badanych grupach*

Variables	Fasting group (*n* = 55)	Non-fasting group (*n* = 96)	Value of *p**
Baseline characteristics and risk factors			
Age [years]	59.4 ±14.6	59.2 ±9.0	0.93
Male/female, *n* (%)	45 (82)/10 (18)	71 (74)/25 (26)	0.27
Body mass index [kg/m^2]	28.6 ±3.4	27.6 ±5.0	0.20
Hypertension, *n* (%)	32 (53)	50 (53)	0.47
Diabetes mellitus, *n* (%)	17 (31)	26 (27)	0.62
Cigarette smokers			0.09
Non-smokers, *n* (%)	21 (38)	43 (45)	
Current smokers, *n* (%)	17 (31)	38 (39)	
Ex-smokers, *n* (%)	17 (31)	15 (16)	
History of MI, *n* (%)	3 (10)	7 (16)	0.73
Prior PCI, n (%)	5 (9)	15 (16)	0.25
Baseline hemodynamics			
Systolic BP [mm Hg]	126.2 ±8.1	124.1 ±22.8	0.51
Diastolic BP [mm Hg]	80.3 ±6.7	78.6 ±9.9	0.24
Heart rate [beats/min]	86.8 ±16.0	83.9 ±25.9	0.46
Laboratory findings			
Creatinine [mg/dl]	0.91 ±0.16	0.92 ±0.12	0.97
Total cholesterol [mg/dl]	188.4 ±28.5	191.1 ±44.8	0.69
Triglyceride [mg/dl]	144.7 ±60.2	150.8 ±53.0	0.52
HDL cholesterol [mg/dl]	46.3 ±9.7	49.2 ±13.9	0.18
LDL cholesterol [mg/dl]	112.5 ±23.4	105.8 ±35.8	0.21
Hemoglobin [g/dl]	14.4 ±1.35	14.3 ±1.47	0.51
Peak CK-MB [IU/l]	152.5 ±127.6	166.5 ±112.5	0.49
Prior medications			
ACE inhibitors, *n* (%)	11 (20)	30 (31)	0.14
ARBs, *n* (%)	10 (18)	10 (10)	0.18
β-Blockers, *n* (%)	12 (22)	30 (31)	0.21
ASA, *n* (%)	26 (55)	47 (49)	0.87
Statin, *n* (%)	20 (37)	23 (24)	0.13
Fibrinolysis, *n* (%)	11 (20)	33 (34)	0.07
Streptokinase, *n* (%)	5 (9)	14 (25)	0.45
t-PA, n (%)	6 (11)	16 (17)	0.47
Primary PCI	42 (76)	63 (66)	0.20

*Values are mean ± SD or proportions (percentages). *Mann-Whitney U and χ^2 tests*

ASA – acetyl salicylic acid, ACE – angiotensin-converting enzyme, ARB – angiotensin receptor blocker, BP – blood pressure, CAD – coronary artery disease, CK-MB – creatine kinase MB fraction, HDL – high-density lipoprotein, LDL – low-density lipoprotein, PCI – percutaneous coronary intervention, t-PA – tissue plasminogen activator

apoprotein B and to HDL was observed in healthy and in hyperlipidemic fasting subjects [23, 24]. Saleh *et al.* showed that by the end of Ramadan fasting, there was a significant improvement in the mean levels of hemoglobin, total cholesterol, triglyceride (TG), HDL cholesterol, LDL cholesterol, total cholesterol/HDL cholesterol, LDL cholesterol/HDL cholesterol, lipoprotein (a), apoprotein A1, apoprotein B, prothrombin time and systolic and diastolic blood pressure that persisted for 4 weeks after fasting [25]. Also there was a significant difference between dietary intake, weight, BMI, percent body fat and waist, fibrinogen and factor VII activity [25]. In another study, markedly elevated levels

Table 2. Echocardiographic and angiographic findings of the study groups, and follow-up results
Tabela 2. *Wyniki echokardiografii i koronarografii w badanych grupach oraz wyniki w trakcie obserwacji*

Variables	Fasting group (n = 55)	Non-fasting group (n = 96)	Value of p*
Echocardiographic findings			
Left ventricular EF [%]	42.9 ±5.9	41.5 ±8.5	0.24
LA [mm]	36.9 ±4.5	38.0 ±3.2	0.07
LVH, *n* (%)	15 (27)	28 (29)	0.65
Mitral regurgitation, *n* (%)	31 (56)	48 (50)	0.50
Mild, *n* (%)	18 (32)	18 (19)	
Moderate, *n* (%)	10 (18)	28 (29)	
Severe, *n* (%)	3 (5)	2 (2)	
Follow-up			
MACE during hospitalization, *n* (%)	5 (9)	14 (15)	0.33
MACE within 30 days, *n* (%)	6 (11)	18 (19)	0.21
Death in hospitalization, *n* (%)	1 (2)	3 (3)	0.63
Death at 30 days, *n* (%)	2 (3.6)	5 (5)	0.66
Heart failure during hospitalization, *n* (%)	4 (7)	7 (7)	0.99
Heart failure within 30 days, *n* (%)	5 (9)	9 (9.3)	0.95
Angiographic findings	**(n = 52)**	**(n = 81)**	
Presence of LMCA lesion, *n* (%)	–	–	
Multi-vessel disease, *n* (%)	12 (23)	26 (32)	0.16
Presence of LAD lesion, *n* (%)	27 (52)	54 (67)	0.09
Angiographic decision			
Medical treatment	7 (13)	26 (32)	0.08
PCI	41 (79)	59 (73)	0.44
CABG	4 (8)	5 (5)	0.72

*Values are mean ± SD or proportions (percentages). *Mann-Whitney U and χ^2 tests*

CABG – coronary artery bypass graft surgery, CAD – coronary artery disease, CK-MB – creatine kinase MB fraction, EF – ejection fraction, LA – left atrial diameter, LAD – left anterior descending artery, LMCA – left main coronary artery, LVH – left ventricular hypertrophy, MACE – major cardiac event, PCI – percutaneous coronary intervention

of plasminogen activator inhibitor (PAI) activity were noted at 9.00 A.M. in Ramadan and at 4.00 A.M. on a non-fasting day. However, the circadian variation in PAI activity levels are influenced more by sleep pattern rather than food intake or the normal activities of a working day [26].

Several investigators have documented reduction of cardiac events during fasting [27-29]. Fasting has been associated with catecholamine inhibition and reduced venous return, causing a decrease in the sympathetic tone, which leads to a fall in blood pressure, heart rate and cardiac output [29]. A reduction in heart rate was also shown [27].

It is known that seasonal changes and temperature differences affect the circadian rhythm [30]. In our study, there were no significant differences of demographic, clinical and echocardiographic characteristics of patients with fasting and non-fasting groups. Although a circadian shift in STEMI was detected in fasting patients, frequency of MACE and heart failure were similar during hospitalization and within 30 days.

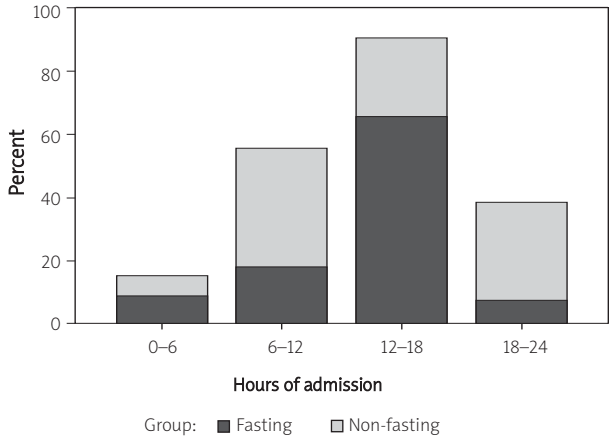

Fig. 1. Circadian periodicity in the onset of chest pain in fasting and non-fasting patients with myocardial infarction
Ryc. 1. *Częstość dobowa występowania bólu w klatce piersiowej u poszczących i nieposzczących pacjentów z zawałem serca*

The study cohort was relatively small and has a cross-sectional design. Laboratory data of patients before Ramadan were absent.

Conclusions

There are significant changes in the circadian variation of patients with STEMI in relation to Ramadan fasting. Further studies are required to determine the pathophysiological mechanisms underlying these changes.

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