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Frequency of Ventricular Premature Beats and Ventricular Tachycardia in STEMI Treated with Fibrinolytics

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A B S T R A C T

To determine in acute myocardial infarction with an ST elevation (STEMI) treated with fibrinolytics frequency of ventricular premature beats (VPBs) and ventricular tachycardia (VT) according to the damaged area and residual cardiac function. With anterolateral infarction with ejection fraction (EF)<45%, incidence of VPBs<10/h was statistically significantly reduced ($p<0.001$) while incidence of VPBs≥10/h as well as VPBs in a pair and VT was increased ($p<0.001$). With anteroseptal infarction with EF<45%, incidence of VPBs<10/h was statistically reduced ($p=0.06$) and incidence of VPBs>10/h, VPBs in a pair and VT was increased ($p=0.06$). With inferior and inferoposterior infarction with EF<45%, incidence of VPBs<10/h was reduced and incidence of VPBs≥10/h, VPBs in a pair and VT was increased. However, such difference was not statistically significant. Along with reduced residual cardiac function, one can also expect increase in frequency of VPBs and VT in all forms of STEMI regardless the area of damage. Such frequency is significant with all forms of anterior infarction, that is to say, slightly more with anterolateral infarction in relation to anteroseptal one. However, with inferior and inferoposterior infarction this frequency of VPBs i VT is not significant.

***Key words**: STEMI, ventricular premature beats, ventricular tachycardia, ejection fraction, Karlovac, Croatia*

Introduction

Ventricular premature beats (VPBs) is among the most common arrhythmias and it affects patients with and without heart disease. In people who do not suffer from heart disease, incidence of VPBs is not connected with increased morbidity or mortality[1]. VPBs and ventricular tachycardia (VT) can be caused by the well-known mechanism: increased automatism, re-entry and trigger activity[2,3]. That is the most frequent and almost always present form of arrhythmias in patients suffering from acute myocardial infarction (AMI)[4]. As a consequence of infarction, there are in myocardium non-homogeneous areas of necrosis which surround the islets of still viable myocardium. Due to weak connection among these islets, which still have normal electrophysiological qualities, one can expect heterogeneous speed of implementation and duration of refractoriness which is the basic element of circular arrhythmias. It is considered today that VPBs and VT do not have a long-term prognostic importance if they occur in the course of 24–72 hours after discomfort has begun. Thus today, routinely prophylactic antiarrhythmic treatment is not recommended except in case of stable or symptomatic arrhythmias[5,6]. The arrhythmias which occur after this period of time have a tendency of later recurrence and thus worse prognosis, and they refer to more serious damage of ventricles and they are connected with increased mortality[7–9]. Researches show that, while frequent complex ventricular ectopia is an independent risk factor, damaged ventricular function is a significantly bigger risk factor[10,11]. While in the past, frequent multifocal or early diastolic extrasystoles were routinely treated, today pharmacotherapy is meant only for the patients suffering from stable or symptomatic arrhythmias. Today in the era of invasive cardiology, there are also not enough clear data about the influence of reduced blood flow in coronary arteries upon the incidence of such arrhythmias[12–14]. Higher incidence of VPBs and VT with a segmental incident of contracility in the course of post-infarction period was discovered by Siogas et al. but with increased arterial blood pressure[15]. Although the highest incidence of VPBs and VT is during

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the first hours of AMI[16], due to higher prognostic importance of later arrhythmias[17], the period after seven days of hospitalization was analyzed in this study.

The aim of this study is to re-evaluate, in patients suffering from AMI with an ST elevation (STEMI), frequency of significant VPBs and VT regarding the damaged area and residual cardiac function after the first week of hospitalization.

Patients and Methods

This research was carried out in the Department of Cardiology of the General hospital in Karlovac (Croatia) between 1999 and 2005. This study included only the patients suffering from the first STEMI who were hospitalized within six hours after the pain had started. All the patients were exposed to a fibrinolytic treatment with streptokinase and in the period between the seventh and the tenth day of their hospitalization they were exposed to a 24-hour ECG (Holter) monitoring. In total 172 patients, 129 men and 43 women between the age of 42 and 76, with the average age 61, were studied. Ejection fraction (EF) of the left ventricle ≥45% was present among 114 patients and EF<45% among 58 patients. 29 patients suffered from anterolateral infarction, 51 patients suffered from anteroseptal infarction, 44 patients suffered from inferior infarction and 48 patients suffered from inferoposterior infarction. By means of Holter monitoring subjects with insignificant number of VPBs (<10/h) were verified in contrast with those subjects with significant VPBs (≥10/h or in a pair) and VT. Three or more consecutive VPBs were considered for VT. Patients with significant artefacts during Holter monitoring were excluded from the study. The patients were compared according to residual cardiac function (EF) which was determined by means of an ultrasound of the heart which was carried out in the period from the seventh until the tenth day of their hospitalization. Measurement of EF of left ventricle was carried out by an ultrasound (ALOKA-ProSound SSD 5500) in M-mode according to Teichholz's method. The control study included patients with EF≥45% and the case study included the patients with EF<45%. According to the regional damage of myocardium, patients suffering from anterolateral and anteroseptal infarction as areas for which the artery LAD (left anterior descending) and a greater part of the artery Cx (circumphlex artery) are responsible, were included in the study. The study also included the patients suffering from inferior and inferoposterior STEMI for which RCA (right coronary artery) and a lesser part of Cx are responsible. ECG criterium for acute anterolateral STEMI was ST elevation in leads V4–V6, for anteroseptal it was in leads V1–V3, for inferior in leads II, III aVF and for acute inferoposterior STEMI in leads II, III, aVF and ST depression and increase R wave amplitude in leads V1–V3. All types of infarction which did not completely satisfy these criteria or had criteria for other localizations (e.g. posterior, lateral, large anterior) were excluded from the study. The study included only those patients who did not have any other possible reasons for VPBs and VT such as hyperkalemia, hyperthyroidism, hypertrophy of the left ventricle, prolapse of mitral valve, febrility, therapy by digitalis and bronchodilatators as well as positive earlier anamnesis of VPBs and VT. Patients with chronic heart failure were also not included in the study. All the patients were treated with ß-blockers as the only antiarrhythmic drug, with acetylsalicylic acid (ASA), ACE-inhibitors and with statins. While testing, the test of proportions and Chi-square analysis with the level of reliability of 99% were used.

Results

With acute anterolateral STEMI with EF<45%, incidence of VPBs<10/h was statistically significantly reduced (p=0.001). However, incidence of VPBs≥10/h, VPBs in a pair and VT was increased (p<0.001).

TABLE 1
ANTEROLATERAL ACUTE STEMI

VPBs	EF≥45%		EF<45%		Z (p)
	N	%	N	%	
<10/h	21	91	3	50	2.38 (<0.001)
≥10/h, in a pair, VT	2	9	3	50	2.38 (<0.001)
Total	23	100	6	100	

STEMI – myocardial infarction with an ST elevation, EF – ejection fraction, VPBs – ventricular premature beats, VT – ventricular tachycardia

With acute anteroseptal STEMI with EF<45%, incidence of VPBs<10/h was reduced (p=0.06), while incidence of VPBs≥10/h, VPBs in a pair and VT was increased (p=0.06).

With acute inferior STEMI with EF<45%, incidence of VPBs<10/h was reduced but incidence of VPBs≥10/h, VPBs in a pair and VT was increased. However, such probability of occurrence was not statistically significant.

With acute inferoposterior STEMI with EF<45%, incidence of VPBs<10/h was reduced, but incidence of

TABLE 2
ANTEROSEPTAL ACUTE STEMI

VPBs	EF≥45%		EF<45%		Z (p)
	N	%	N	%	
<10/h	31	84	9	64	1.51 (0.06)
≥10/h, in a pair, VT	6	16	5	36	1.52 (0.06)
Total	37	100	14	100	

STEMI – myocardial infarction with an ST elevation, EF – ejection fraction, VPBs – ventricular premature beats, VT – ventricular tachycardia

TABLE 3
INFERIOR ACUTE STEMI

VPBs	EF≥45%		EF<45%		Z (p)
	N	%	N	%	
<10/h	28	76	5	71	0.24 (n.s.)
≥10/h, in a pair, VT	9	24	2	29	0.23 (n.s.)
Total	37	100	7	100	

STEMI – myocardial infarction with an ST elevation, EF – ejection fraction, VPBs – ventricular premature beats, VT – ventricular tachycardia

TABLE 4
INFEROPOSTERIOR ACUTE STEMI

VPBs	EF≥45%		EF<45%		Z (p)
	N	%	N	%	
<10/h	36	86	4	67	1.71 (n.s.)
≥10/h, in a pair, VT	6	14	2	33	1.72 (n.s.)
Total	42	100	6	100	

STEMI – myocardial infarction with an ST elevation, EF – ejection fraction, VPBs – ventricular premature beats, VT – ventricular tachycardia

VPBs≥10/h, VPBs in pair and VT was increased. However, such probability of occurrence was not statistically significant.

Discussion

The research into the incidence of VPBs and VT in acute anterolateral, anteroseptal, inferior and inferoposterior STEMI was inspired by the notion that most researches into post-infarctions ventricular arrhythmias analyze their incidence according to only two damaged areas, anterior and inferior ones[18,19].

In this research, with acute anterolateral STEMI (Table 1) due to reduced EF<45%, incidence of VPBs<10/h statistically significantly decreased ($p<0.001$) while incidence of VPBs≥10/h, VPBs in a pair and VT increased ($p<0.001$). These data are in accordance with some other studies in which, according to Efre, ventricular arrhythmias more often occur in anterior infarction[20]. Also, Huikuri et al.[21] and Moss et al.[22] point out that reduced EF <35% causes significant increase in frequency of ventricular arrhythmias.

With acute anteroseptal STEMI (Table 2) due to reduced EF<45%, incidence of VPBs<10/h statistically significantly decreased, but at the level $p=0.06$ which is in accordance with the increase in incidence of VPBs ≥10/h, VPBs in a pair and VT ($p=0.06$). This indicates that even with anterior infarctions there are slight differences in significance of post-infarction VPBs incidence. Possibly this is so, because the anteroseptal area is irrigated mainly by LAD while the anterolateral area, besides LAD, is also irrigated by Cx. This causes necrosis which affects larger areas of myocardium and this is in accordance with some other author's data which say that damaged ventricular function also increases possibility of ventricular arrhythmias[10,11].

With acute inferior STEMI (Table 3) and inferoposterior STEMI (Table 4), there was no statistically significant difference between the incidence of VPBs and VT due to reduced EF <45%. Similar results about less frequent ventricular arrhythmias in inferior infarction in relation to anterior one were also obtained by Bluzaite et al.[18] and Stone et al.[19]. The difference is the fact that more frequent atrial arrhythmias occur in inferior infarction due to damage of sinoatrial node which is irrigated via RCA branch[20].

The weakness of this study is, due to simplicity of measurement of ejection fraction of left ventricle, the usage of Teichholz´s method in M-mode. More accurate estimate of hypokinetic areas would be obtained by measurement in 2 D-mode.

To conclude, along with reduced residual cardiac function in post-infarction period, one can also expect increase in frequency of VPBs and VT incidence in all forms of STEMI regardless the area of damage. Such frequency is statistically significant only in both forms of anterior infarction. It is a bit more significant in anterolateral infarction in relation to anteroseptal one. However, there is no statistically significant increase in frequency of VPBs and VT in inferior and inferoposterior infarction. This notion can help us anticipate in advance the danger of more frequent incidence of VPBs and VT in STEMI with regard to the area of damage.

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UČESTALOST VENTRIKULSKE EKSTRASISTOLIJE I VENTRIKULSKE TAHIKARDIJE U STEMI-u TRETIRANOM FIBRINOLITICIMA

S A Ž E T A K

Odrediti u akutnom infarktu miokarda sa ST elevacijom (STEMI) tretiranom sa fibrinoliticima učestalost ventrikulskih ekstrasistola (VES) i ventrikulske tahikardije (VT) prema zahvaćenoj regiji i rezidualnoj kardijalnoj funkciji. U anterolateralnom infarktu kod ejekcijske frakcije (EF)<45% statistički značajno se smanjuje incidencija VES<10/h (p<0,001), a povećava incidencija VES≥10/h, VES u paru i VT (p<0,001). U anteroseptalnom infarktu kod EF<45% statistički se smanjuje incidencija VES<10/h (p=0,06), a povećava incidencija VES≥10/h, VES u paru i VT (p=0,06). U inferiornom i inferoposteriornom infarktu kod EF<45% smanjuje se incidencija VES<10/h, a povećava incidencija VES ≥10/h, VES u paru i VT, no ta razlika nije statistički značajna. Smanjenjem rezidualne kardijalne funkcije povećava se učestalost VES i VT u svim oblicima STEMI-a bez obzira na regiju oštećenja. Ta je učestalost značajna kod oba oblika anteriornog infarkta i to nešto više kod anterolateralnog u odnosu na anteroseptalni infarkt, dok kod inferiornog i inferoposteriornog infarkta učestalost pojavljivanja VES i VT nije značajna.