



Modern Clinical Retrospective on Ventricular Rhythm Disturbances in Acute Myocardial Infarction: Latest Treatment Mode According to the Valid Recommendations

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SUMMARY

Ventricular arrhythmias are the most common cause of mortality in patients with acute myocardial infarction. Arrhythmias in acute coronary syndrome occur because of unbalanced function of the autonomic nervous system, because of electrolyte status disturbances (hypokalemia, hypomagnesaemia, intracellular hypercalcemia) and because of slow conduction in ischemical myocardium zones. Arrhythmias contribute to acidosis and release of free oxygen radicals occurring during reperfusion of ischemic myocardium. Ventricular arrhythmias in acute myocardial infarction indicate the presence of extensive myocardial damage at a reduced left ventricular ejection fraction, and not infrequently in the formation of an aneurysm of the left ventricle. In patients with acute coronary syndrome, the most common ventricular arrhythmias are premature ventricular beats, ventricular tachycardia, ventricular flutter and fibrillation and accelerated idioventricular rhythm. Ventricular fibrillation, still the leading cause of sudden death in patients with acute coronary syndrome within the first four hours of myocardial infarction, occurs in 80 % of all ventricular fibrillations. The use of beta-blockers in the first 24 hours of acute myocardial infarction in patients with early ventricular fibrillation and tachycardia does not affect the degree of worsening heart failure and is directly associated with reduced rates of early mortality in these patients. Quick and adequate treatment of ventricular arrhythmias is a major advancement in the treatment of acute myocardial infarction.

Keywords: acute myocardial infarction, ventricular arrhythmias, treatment

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INTRODUCTION

Acute coronary syndrome (ACS) is the most common cause of hospitalization of patients in industrialized countries. The most common cause of sudden cardiac death is a coronary heart disease (80% of patients), followed by cardiomyopathy (10% -15 %), and in 5% of patients with sudden cardiac death its cause remains unknown until the end. The most common causes of ACS mortality are malignant heart rhythm disorders. As most authors believe, the main electrophysiological mechanism of arrhythmias in the acute phase of coronary occlusion is micro reentry, and the most common cause of occurrence of the disturbances in electrolyte status (hypokalaemia, hypomagnesaemia, hypocalcaemia) and slow implementation in the areas of ischemic myocardium.

Ventricular arrhythmias in acute myocardial infarction can be monitored hemodynamically in a stable condition of the patient, and the patient is often asymptomatic or has minimal symptoms, such as palpitations. On the other hand, ventricular arrhythmias in patients with acute myocardial infarction may lead to hemodynamic instability and cause syncope, sudden cardiac arrest and death [1]. Due to the reduced systolic function, patients with acute myocardial infarction have been reduced, but a relatively stable stroke volume and depending on changes in heart frequency change the value of cardiac output. Heart rate is a major determinant of myocardial oxygen consumption and the higher the heart rate, myocardial energy requirements are increased to a level that can adversely affect myocardial ischemia. Hemodynamic instability during certain arrhythmias contributes to reducing atrial contributions to ventricular preload. Absence of atrial function decreases cardiac output of the left ventricle by 15-20 %.

Occurrence of ventricular rhythm disturbances in acute myocardial infarction usually indicates the presence of extensive myocardial damage at a reduced left ventricular ejection fraction, and not infrequently in the formation of an aneurysm of the left ventricle [2]. The results of the study before the era of primary percutaneous intervention in acute myocardial infarction indicate poor prognosis in patients with ventricular arrhythmias occurred more than 48 hours from the start of acute myocardial infarction [2,3,4,5].

Ventricular arrhythmias were more frequently occurred earlier, when the use of thrombolytic therapy was the only reperfusion strategy in the treatment of acute myocardial infarction than now when primary percutaneous intervention is most often used for revascularization of the infarct artery.

The main objective of this paper is to show the frequency and significance of the occurrence of ventricular arrhythmias in patients with acute myocardial infarction and present the latest in their treatment modalities. The work is based on the recommendations of the European Society of Cardiology for the treatment of ventricular arrhythmias in acute myocardial infarction with ST-segment elevation that were published in 2012. Recommendations clearly specified gradation in the treatment of some ventricular arrhythmias, class of recommendations and level of evidence of the use of certain procedures or medications.

Ventricular arrhythmias

Rare, premature ventricular depolarization occurs in almost all patients with acute myocardial infarction and the most common causes of this occurrence are: ischemia, elevated catecholamines and hypokalaemia. Before the widespread use of reperfusion therapy in the treatment of acute myocardial infarction with ST-segment elevation (STEMI) was considered to frequent ventricular extra systoles (VES) (more than 5/min), multifocal VES, VES with R/T, preceded by atrial chamber so that such arrhythmias called "warning arrhythmias" [6]. Prophylactic antiarrhythmic drugs to suppress reporting "warning arrhythmias" (initially intravenous lidocaine and later oral antiarrhythmic agents) are no longer recommended because its use may be associated with an increased risk of fatal bradycardia and asystole [7]. Instead of the routine application of antiarrhythmic drugs it is advised to prevent re-myocardial ischemia and correction of electrolyte and metabolic imbalance. A correction of hypokalaemia and hypomagnesaemia with a tendency to serum potassium level is about 4.5 mEq/L and magnesium approximately 0.4 mEq/L is indicated. Ventricular arrhythmias occur in the framework of reperfusion arrhythmias after administration of thrombolytic therapy (lysis of thrombus) or after plaque reduction after percutaneous intervention (PCI). The drugs most effectively interrupt

ventricular ectopic activity as beta-blockers, and it is shown that their application is associated with a reduction in the incidence of ventricular fibrillation in patients with myocardial infarction [8, 9].

Beta blockers are particularly indicated in patients with anterior myocardial infarction localization in patients with elevated sympathetic activity and hypertension.

While the occurrence of ventricular premature beats in people with healthy hearts often a benign phenomenon in patients with acute myocardial infarction frequent premature ventricular leads to a decrease in cardiac output, precipitating anginal symptoms and causes of cardiac insufficiency.

The occurrence of frequent ventricular infarction in the period was closely associated with the sudden death of the patient. Patients who have a coronary disease manifest signs and symptoms of heart failure and ejection fraction (EF) of the left ventricle of less than 30%, and in which the Holter ECG monitoring registers more than 10 VES/hour, have an increased mortality rate in the three-year follow-up period.

The occurrence of single ventricular premature beats does not require specific antiarrhythmic therapy. Corrections of electrolyte disturbances are necessary (hypokalaemia and hypomagnesaemia), and possibly the use of beta blockers. The first line therapy in the treatment of repetitive ventricular (more than 10 VES/hour, polymorphic VES, VES with the phenomenon of "R on T") is a correction of electrolyte imbalance and use of beta blockers. The second line therapy includes intravenous lidocaine (at virologic failure with beta-blockers or when they are contraindicated), procainamide, amiodarone.

Ventricular tachycardia

Ventricular tachycardia (VT) in patients with acute myocardial infarction occurs in the form of asymptomatic episodes of VT to episodes

that are accompanied by hemodynamic collapse and sudden death. The anatomical substrate for the occurrence of VT usually stems from significant scarring of the heart muscle resulting from myocardial infarction. The mechanism of VT in most cases is the re-entry phenomenon. Ventricular tachycardia in acute myocardial infarction may be long-term (continuous, "sustained") or short-term (discontinuous, "non-sustained") VT. Sustained ventricular tachycardia is defined as a VT that lasts longer than 30 seconds or as ventricular tachycardia that is necessary to stop due to hemodynamic collapse. In the first 11 days of acute myocardial infarction the incidence of long-term VT is 3% [9, 10]. "Sustained" VT is almost always symptomatic and is often accompanied by hemodynamic disturbances and need immediate suppressive therapy [9,11,12]. It leads to hypotension, pulmonary edema, cerebral hypoperfusion, cardiopulmonary arrest and quickly degenerates into ventricular flutter/fibrillation. Non-sustained VT (≥ 6 VES in the trace, the maximum duration to 30 seconds) occurs both in people with heart disease and in patients without heart disease. Although "non-sustained" VT usually causes no symptoms and is not considered a predictive marker for the occurrence of early ventricular fibrillation, more prolonged episodes of "non-sustained" VT may cause hypotension, heart failure and progress into ventricular fibrillation [9,13]. VT that occurs in myocardial infarction may be uniform (monomorphic) or can be varied from impact to impact (polymorphic) (Figure 1, 2). Bidirectional ventricular tachycardia is VT with alternative amplitude and axis of the QRS complex. For this tachycardia is typically QRS complex has a shape like a bundle branch block with an alternating electric axle up (to the left) and downwards (to the right). Torsade de pointes is VT with polymorphic QRS complexes with a variable amplitude and the cycle length, which are shown in the form of oscillations around the baseline.

Figure 1. Monomorphic ventricular tachycardia

(taken over from the Internet, address: ventricular rhythm disorders pictures-Google search)

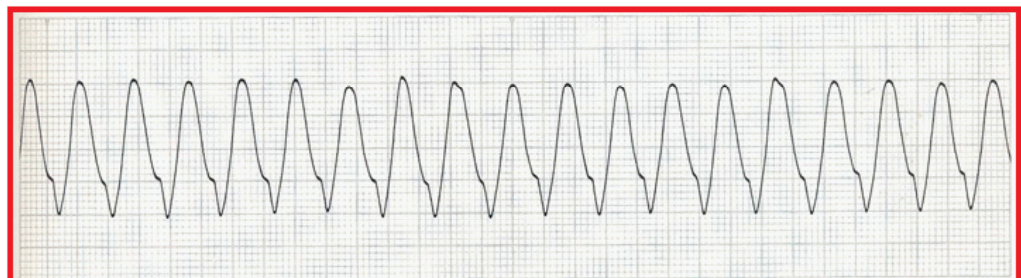




Figure 2. Polymorphic ventricular tachycardia

(taken over from the Internet, address: ventricular rhythm disorders pictures - Google search)

In the early hours of myocardial infarction VT is usually caused by ischemia, while VT that occurs later in the course of myocardial infarction is caused by reduced left ventricular function, residual ischemia, myocardial large aneurysm chamber or other serious complications. Most episodes of VT in STEMI patients occur within the first 48 hours. Studies have shown that the main predictors for the occurrence of early VT in patients with STEMI are age, the absence of anterograde epicardial flow in the infarct artery before PCI and larger infarction zone [2]. Other studies suggest that low systolic blood pressure after primary PCI is the main predictor of early VT/VF [2, 14]. When the “sustained” VT occurs later in STEMI (over 24 hours), it is associated with increased mortality in the first 30 days of follow-up compared with patients who did not have “sustained” VT [15].

The treatment of VT must be urgently initiated because the VT may progress to VF. Urgent diagnosis must be carried out, as well as treatment of reversible factors that lead to VT (heart failure, acute ischemia). Patients with clinically stable VT can be treated with antiarrhythmic drugs. Although previously considered “non-sustained” VT is rarely the cause of hemodynamic collapse and does not require acute treatment, it is considered today that it may indicate a high-risk arrhythmic substrate, and if it occurs 4 days after STEMI in patients with reduced left ventricular ejec-

tion fraction it may indicate the risk of sudden death.

As noted above, if a “non-sustained” VT is extremely fast despite a brief period of life, especially if repeated, this may lead to hemodynamic instability, and pharmacotherapy similar to that recommended for sustained VT should be introduced in these cases. In about 50 % to 80% of patients, the use of antiarrhythmic drugs reduces the incidence of non-sustained VT.

In ischemic heart disease in hemodynamically stable patients with monomorphic VT the use of beta-blockers and amiodarone is the safest. Both of these drugs reduce heart rate and thus the oxygen consumption of the heart muscle. Recommendations for STEMI patients to favour the application of amiodarone in case of patients with reduced left ventricular function (Class IIa recommendation, level of evidence C) [9,16,17]. The second drug of choice in these patients is lidocaine (Class IIa recommendation, level of evidence C) [9]. In patients with normal left ventricular EF is recommended to use a beta-blocker, procainamide (bolus of 15 mg/kg during 20 to 30 min; infusion of 1-4 mg/min), sotalol, amiodarone (bolus of 75-150 mg over 10-15 min, followed by infusion of 1.0 mg/min over 6 hours, and then 0.5 mg/min) and lidocaine (bolus 1-1.5 mg/kg; infusion of 20 to 50 µg/min) (Class IIa recommendations, the level of evidence C) [9].

Electrical cardioversion is indicated for the “sustained” VT which are accompanied by hemodynamic instability, pain in the chest and the development of heart failure [16]. In patients with hemodynamically unstable monomorphic VT is applied synchronous DC conversion (DC shock 100 J) (class of recommendation I, level of evidence C) [9]. If there are hemodynamically unstable VT monomor-

phic that is refractory to DC conversion in addition to drug therapy (lidocaine, amiodarone, beta blockers), the transvenous pacing catheter, radiofrequency ablation or implantation of antitachycardiac pacing are considered (Class IIa recommendation, level of evidence C) [9] (Table 1).

Treatment of polymorphous VT which is accompanied haemodynamic stability of patients, and that in the base electrocardiogram have a normal QT interval should begin treatment of ischemia, by correcting electrolyte disturbances, and apply beta blockers (class of recommendation I, level of evidence B), amiodarone (Class I recommendation, level of evidence C) or lidocaine (Class IIb recommendation, level of evidence C) [9]. In the case of patients with prolonged QT interval in the base electrocardiogram and having polymorphic hemodynamically stable VT, they need to correct electrolyte disorder and applied lidocaine (Class I recommendation, level of evidence C) [9]. Rapid, polymorphic VT, which is accompanied by hemodynamic instability should be considered similar to the ventricular fibrillation and be treated with the asynchronous electric-energy 200 J. After conversion to sinus rhythm all abnormalities such as hypoxia, hypotension, acid-base and electrolyte imbalances should be corrected (Table 2).

For patients with recurrent, refractory VT of implanting antitachycardiac pacemaker, radiofrequency ablation or surgical aneurysmectomy the possibility should be considered. Sometimes urgent revascularization will help in controlling refractory VT.

It is necessary to emphasize that each tachycardia with wide QRS complexes in patients with acute myocardial infarction (unless there is a pre VT branch block) should be considered as ventricular until proven otherwise (intracardiac electrophysiological study). All patients with acute myocardial infarction and complicated VT should be treated with beta-blockers unless these are contraindicated in cases of hypotension, bradycardia, or other clinical factors (vasospastic angina or disease of the airways). In patients with early VT the use of beta-blockers in the first 24 hours of myocardial infarction was directly associated with a reduced early mortality without worsening heart failure [18].

Table 1. Monomorphic VT treatment in acute myocardial infarction

* - i.v. sotalol, or other β-blockers not to be used if the EF is small
VT - ventricular tachycardia;
i.v. - intravenous

ESC RECOMMENDATIONS (2012)	Class	Level of evidence
Hemodynamically unstable monomorphic VT (Fr>150min)		
DC cardioversion (synchronous DC shock 100 J)	I	C
Hemodynamic unstable, sustained monomorphic VT (refractory to DC cardioversion)		
i.v. amiodarone	IIa	C
lidocaine or sotalol *	IIb	C
refractory to cardioversion and antiarrhythmic agents consider the application of transvenous pacing catheter, ablation, antitachycardiac pacemaker implantation	IIa	C
Repetitive symptomatic non/sustained monomorphic VT		
i.v. amiodarone, sotalol or other beta blocker	IIa	C

Table 2. Treatment of polymorphic VT in acute myocardial infarction

* - EF normal: beta-blockers, lidocaine, amiodarone, procainamide or sotalol
* - EF low: amiodarone (150 mg for 10 min) or lidocaine (0.5-0.75 mg/kg)
VT - ventricular tachycardia;
i.v. - intravenous

ESC RECOMMENDATIONS (2012)	Class	Level of evidence
Polymorphic VT (hemodynamically stable patients)		
* Basal QT normal (correct electrolytes, treat ischemia)		
i.v. sotalol or other β-blockers	I	B
i.v. amiodarone	I	C
i.v. lidocaine	IIb	C
Basal QT interval prolonged		
correction of electrolyte, magnesium checks, overdrive pacing, isoproterenol, or lidocaine	I	C
consider emergency angiography in patients with polymorphic VT in within myocardial ischemia	I	C
Polymorphic VT (hemodynamically unstable patients)		
Asynchronous DC shock (360 J (single-phase)/200 J (biphasic)		

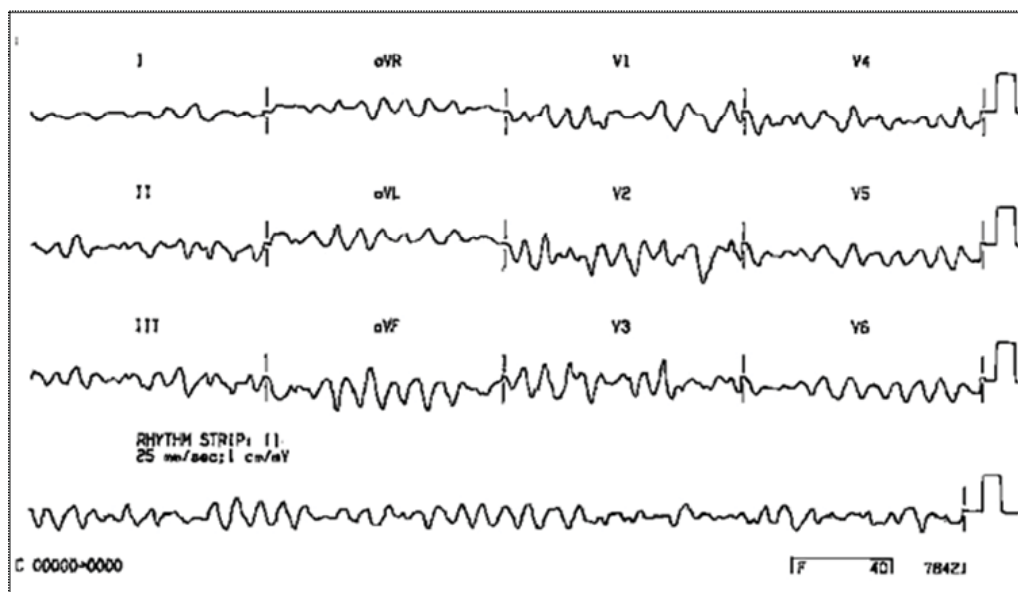


Figure 3. Ventricular fibrillation

(taken over from the Internet, address: ventricular rhythm disorders pictures - Google search)

Ventricular tempest

Frequent episodes of VT (> 2 for 24 h) requiring cardioversion represent a ventricular storm. The most common cause of ventricular tempest is heart disease, but it also occurs with Brugada syndrome, with an overdose of drugs and electrolytic imbalance. Treatment of ventricular tempest is based on the identification and exclusion of precipitating factors, medication application of amiodarone in acute myocardial ischemia, pacing or ablation, while in Brugada syndrome there are intravenously administered beta-blockers, isoproterenol (isoprenaline) or quinidine.

Ventricular flutter and ventricular fibrillation

Ventricular flutter represents a malignant ventricular tachyarrhythmia (Fr: 250-350/min) looking like a zig-zag path that leads to hemodynamic collapse and requires immediate cardioversion DC shock. Ventricular fibrillation (VF) is a completely disorganized, chaotic electrical activity of the ventricular myocardium, which has no coordination of contraction of myofibrils (Figure 3). Figure VF electrocardiogram shows a rapid, irregular rhythm with vaguely defined QRS complexes. The most common is the first hour after the occurrence of myocardial infarction, and then its incidence is gradually reduced.

In the first four hours of myocardial infarction 80 % of VF occur. VF can occur in three forms in hospitalized STEMI patients. Primary VF (3-5 %) usually occurs in the first

four hours after STEMI (60 % in the first hour). It is usually caused by myocardial ischemia. In recent decades the primary VF occurred in 10% of hospitalized patients with STEMI, until recently its incidence is decreasing, probably due to an aggressive approach in reducing the size of the infarction zone, due to the rapid correction of electrolyte imbalance as well as the increasing use of beta-adrenergic blockers [19]. One-year mortality in patients with primary VF is about 15 %. Secondary VF occurs 4 to 48 hours from the onset of myocardial infarction symptoms. It is usually caused by left ventricular failure and cardiogenic shock, aneurysm and other serious complications of acute myocardial infarction. One-year mortality in patients with secondary VF is 85 %. VF that occurs during the first day of STEMI is a bad predictor of recurrent arrhythmias [20]. The so-called late VF occurs more than 48 hours after STEMI and usually, but not exclusively occurs in patients with large myocardial infarction and left ventricular dysfunction.

Despite the “warning arrhythmias” which may or may not be the VF forerunners, there are still no clear electrocardiographic signs to identify patients at risk of VF. In the early hours of myocardial infarction intravenous and then oral use of beta-blockers is associated with a lower percentage of early VF occurrence, whereas the previous routine use of lidocaine in order to prevent the occurrence of VF was abandoned [8]. The appropriate therapeutic mode of beta blockers application includes metoprolol intravenous administration of 5 mg every 2 minutes (total of 3 doses),

and continues for peroral administration (50 mg orally twice a day, at least 24 hours, and then increased to 100 mg twice daily) [1].

An alternative mode is the use of atenolol (5-10 mg intravenously for which goes 100 mg daily, orally) (Table 3). Clinical experience and data identified hypokalaemia and hypomagnesaemia as arrhythmogenic risk factors for VF.

Table 3. Intravenous doses of antiarrhythmic drugs in ventricular arrhythmias in acute myocardial infarction (ESC/AHA 2006)

Medicine	Bolus doses	Infusion application
Amiodarone	75-150 mg for 10 min	1 mg/min for 6 h and then 0.5 mg/min
	A bolus dose of 150 mg can be administered, for 10-30 min for recurrent arrhythmias, but is limited to 6-8 bolus over 24 h	
Lidocaine	1-1.5 mg/kg	20-50 µg/min
Esmolol	500 µg/kg for 1 min., followed by 50 µg/kg/min during 4 min	60 to 200 µg/kg/min
Metoprolol	2.5-5 mg during 2 min; a maximum of 3 doses	-
Atenolol	5-10 mg 1 mg/min	-
Propranolol	0.15 mg/kg	-
Sotalol	20-120 mg during 10 min (0.5-1.5 mg/kg)	-
	It can be repeated after 6 h (maximum 640 mg/24 h)	

Patients with VF are treated with emergency defibrillation. Successful defibrillation depends on the length of time until defibrillation (every minute until defibrillation reduces the chance of survival of 7-10 %), free energy, transthoracic impedance, the position of the shock electrode surface patch electrodes and the shock of the patient's metabolic status (acid-base and electrolyte). Ventricular fibrillation should be treated with asynchronous DC shock using DC shock of 200J. If the first shock is not established the heart function other than the shock 200-300J is required, and if needed a third shock of 360J is also required.

Biphasic waveform has an advantage over the single-phase, because less energy is required for defibrillation; the use less energy resulting in less post-shock ST segment abnormalities. If ventricular fibrillation is resistant to initial electric, its efficiency may be increased by administration of epinephrine (1 mg intravenously, or 10 ml of a 1:10,000 intracardiac), bretylium (bolus of 5 mg/kg) or amiodarone (75-150 mg bolus).

If VF is repeated, the determination of its etiology may be useful in risk stratification and prevention of arrhythmias, since patients with "reversible" VF causes could constantly be at risk of future occurrence of VF. In patients with recurrent, persistent VF, the use of implantable cardioverter defibrillator (ICD) would significantly increase the outpatient survival. Among the survivors of VF or "sustained" VT ICD therapy was associated with a significant reduction in mortality compared with antiarrhythmic drugs [21]. With the exception of beta-blockers, antiarrhythmic drugs have not been shown to be effective in patients with life-threatening ventricular arrhythmias and should not be used as prevention of sudden cardiac death [9]. ICD is recommended as secondary prevention in reducing mortality in patients with severe left ventricular dysfunction who present with hemodynamically unstable "sustained" VT or have a VF after 48 h of acute myocardial infarction [22]. Electrophysiological evaluation should be done to all patients before deciding to ICD implantation with the aim of secondary prevention of sudden cardiac death [12, 21]. If there is a reduced EF ($\leq 30\%$) at least one month after STEMI and 3 months after myocardial infarction revascularization, it is reasonable to incorporate ICD without previous diagnostic electrophysiological study. Recommendations ESC/ACC/AHA do not recommend ICD implantation in patients who have "sustained" VT or VF occurred in the first 24-48 hours of myocardial infarction [20].

Accelerated idioventricular rhythm

This heart rhythm disorder is usually harmless and occurs most often in the context of reperfusion and is characterized by slow ventricular tachycardia frequencies of 60-120/min. These ventricular arrhythmias are not a significant predictive marker of early VF and are usually accompanied by hemodynamic stability and

do not require prophylactic antiarrhythmic therapy or treatment.

CONCLUSION

Patients with acute coronary syndrome should reach the nearest hospitals as soon as possible, as most deaths caused by ventricular arrhythmias occur during the first hours of acute myocardial infarction. Despite advances in the treatment of acute myocardial infarction, malignant ventricular heart rhythm disorders occur in a smaller but still significant percentage, their recognition, prompt and adequate treatment represents a major advance in the treatment of acute coronary syndromes. The quality and speed of treatment of ventricular rhythm disturbances depends on the knowledge of the pharmacokinetics of antiarrhythmic agents, because the dosage varies considerably depending on patient's age, body weight, and functional status of the liver and kidneys.

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Savremeni klinički osvrt na ventrikularne poremećaje ritma u akutnom infarktu miokarda: najnoviji modaliteti lečenja u skladu sa važećim preporukama

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KRATAK SADRŽAJ

Ventrikularne aritmije su najčešći uzrok mortaliteta u bolesnika sa akutnim infarktom miokarda. Aritmije u akutnom koronarnom sindromu se javljaju zbog neuravnotežene funkcije autonomnog nervnog sistema, zbog poremećaja u elektrolitnom statusu (hipokalijemija, hipomagnezijemija, intracelularna hiperkalcemija) i zbog usporenog provođenja u zonama ishemijskog miokarda. Pojavi aritmija doprinosi acidoza i oslobađanje slobodnih kiseoničkih radikala koji nastaju u toku reperfuzije ishemijskog miokarda. Javljanje ventrikularnih aritmija u akutnom infarktu miokarda ukazuje na prisustvo ekstenzivnog miokardnog oštećenja, na redukovanu sistolnu funkciju leve komore i ne retko na formiranje aneurizme leve komore. U bolesnika sa akutnim koronarnim sindromom najčešći ventrikularni poremećaji ritma su: ventrikularna ekstrasistolija, ventrikularna tahikardija, ventrikularni flater i fibrilacija i ubrzani idioventrikularni ritam. Ventrikularna fibrilacija je i dalje vodeći uzrok nagle smrti u bolesnika sa akutnim koronarnim sindromom, a u prvih 4 časa infarkta miokarda dešava se 80% svih ventrikularnih fibrilacija. Primena beta blokatora u prvih 24 časa akutnog infarkta miokarda kod bolesnika sa ranom ventrikularnom fibrilacijom i tahikardijom ne utiče na pogoršanje stepena srčane insuficijencije a direktno je povezana sa smanjenjem stope ranog mortaliteta u tih bolesnika. Brzo i adekvatno lečenje ventrikularnih aritmija predstavlja veliki napredak u lečenju akutnog infarkta miokarda.

Ključne reči: akutni infarkt miokarda, ventrikularni poremećaji ritma, lečenje

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