Ann Med Res 2021;28(4):850-7

Annals of Medical Research

DOI: 10.5455/annalsmedres.2020.03.273

Review Article

Arrhythmia complications in acute coronary syndrome: Focused on tachyarrhythmias

Eka Prasetya Budi Mulia[1], Andrianto[2]

[1]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Airlangga - Dr. Soetomo General Hospital, Surabaya, Indonesia
[2]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Airlangga - Dr. Soetomo General Hospital, Surabaya, Indonesia

Abstract

Arrhythmias may occur as complications in acute coronary syndrome (ACS) patients. It includes tachyarrhythmia, which may be asymptomatic or symptomatic. At least around 75% of myocardial infarction patients develop arrhythmia during the peri-infarction period. Pathophysiology pathways differ in each type of arrhythmias. . It also contributes to different treatment modalities. Arrhythmogenesis in ACS patients includes various factors: electrophysiological changes, metabolic changes, increased sympathetic activity, vagal stimulation, reduced left ventricular ejection fraction (LVEF), and scar formation. Myocardial reperfusion also may result in complex electrophysiological changes, depending on previous ischemia duration. Ventricular arrhythmia is more common with increased ischemia duration. At present, the anti-arrhythmic prophylactic management strategy has mostly been abandoned. Although the primary therapy for arrhythmias is anti-arrhythmic drugs (AADs), especially amiodarone and sodium channel inhibitors, their utilization now has declined, since the emergence of clinical evidence with inconclusive results in the use of these AADs. Besides, therapies for ACS and their arrhythmic management are increasingly based on invasive approaches. Some tachyarrhythmias are malignant and may increase death risk, which requires immediate treatment, while some are benign and do not alter the outcome of patients. Understanding the mechanism and adequate treatment of these tachyarrhythmias is essential in reducing mortality in ACS patients during the acute phase and follow-up.

Keywords: Acute coronary syndrome; arrhythmia; myocardial infarction; tachycardia

INTRODUCTION

Myocardial ischemia and infarction can cause electrophysiological and metabolic changes causing asymptomatic or even life-threatening arrhythmias. About 75% of acute myocardial infarction (AMI) patients experience arrhythmias during the peri-infarction period (1). CARISMA trial mentioned that arrhythmia incidence in AMI amounted to 28% of new-onset atrial fibrillation, 13% of non-sustained ventricular tachycardia, 10% of high-degree atrioventricular (AV) block (≤30 bpm for ≥8 seconds), 7% sinus bradycardia (≤30 bpm for ≥8 seconds), 5% sinus arrest (≥5 seconds), 3% sustained ventricular tachycardia, and 3% ventricular fibrillation (2). Sudden cardiac death (SCD) is most frequently associated with this arrhythmia, and about half of the deaths occur before the patients reach the hospital. The most common cause of death in patient with AMI in pre-hospitalization is ventricular tachycardia/ventricular fibrillation (VT/VF) (1). Both atrial and ventricular arrhythmias can develop in ACS, including ventricular tachyarrhythmias, which can lead to a collapse of the circulatory system and require immediate therapy. Atrial fibrillation (AF) can also require immediate therapy when rapid ventricular response causes hemodynamic declines. The management of other arrhythmias is also primarily based on symptoms to prevent progression to more severe artistry. At present, the anti-arrhythmic prophylactic management strategy has mostly been abandoned (1,3).

Although the primary therapy for arrhythmias is anti-arrhythmic drugs (AADs), especially amiodarone and sodium channel inhibitors, their utilization now has declined, since the emergence of clinical evidence with inconclusive results in the use of these AADs. Besides, therapies for ACS and their arrhythmic management are increasingly based on invasive approaches (3). Improvements in medical services, early recovery of ischemia, beta-blockers, and angiotensin-converting enzyme inhibitors (ACE-I) have also reduced the incidence of arrhythmias, although arrhythmias remain the leading cause of death in ACS patients. The use of implantable cardioverter-defibrillator (ICD) also has a potential effect

Received: 28.03.2020 **Accepted:** 10.06.2020 **Available online:** 26.04.2021
Corresponding Author: Andrianto, Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Airlangga-Dr. Soetomo General Hospital, Surabaya, Indonesia **E-mail:** andrianto@fk.unair.ac.id

on the prevention of primary and secondary ventricular arrhythmias (VA) in patients with ACS (1). This literature review aims to gain a thorough understanding of the complications of tachyarrhythmias and their management in patients with ACS.

Mechanism of ischemic-related arrhythmia

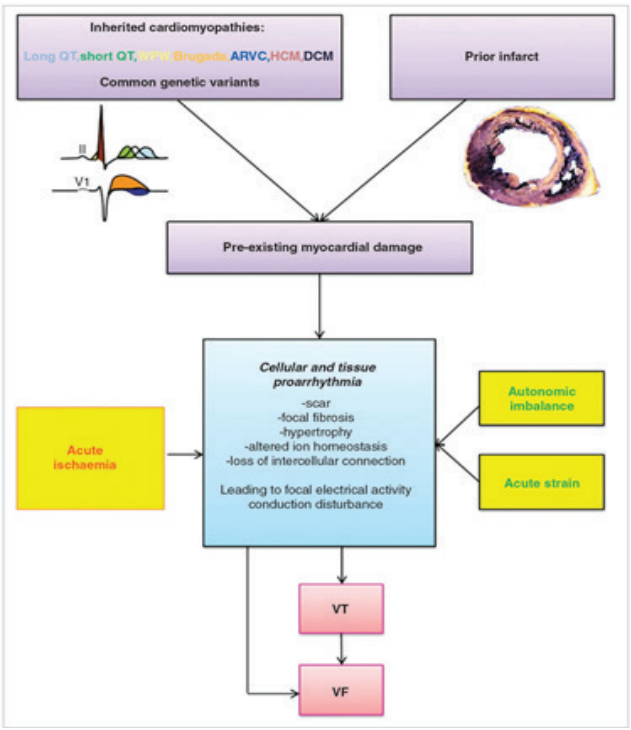
In ischemia, various factors play a role in arrhythmogenesis. Hypoxia, changes in ion-current, and electrolyte imbalance allow the development of arrhythmias. In addition, the increased automaticity of the purkinje systems and myocardium is triggered by autonomic cardiac dysfunction. Increased efferent sympathetic activity, increased catecholamine concentration, and local catecholamine release from nerve endings in the myocardium itself is involved. Further, transmural infarction can disrupt the afferents and efferents of the sympathetic nerve that innervates the distal myocardium of the infarct area. This autonomic imbalance triggers the development of arrhythmias. Other mechanisms that can influence include increased levels of free fatty acids and free radicals derived from oxygen, which may also play a role in the development of arrhythmia in ACS (4,5). There are several types of tachyarrhythmia which may complicate ACS such as ventricular and supraventricular tachyarrhythmia (Table 1).

Table 1. Types of tachyarrhythmias on ACS [1]

Category	Arrhythmia
Ventricular tachyarrhythmia	Ventricular premature beat
	Ventricular tachycardia
	Ventricular fibrillation
Supraventricular tachyarrhythmia	Sinus tachycardia
	Paroxysmal supraventricular tachycardia
	Atrial fibrillation
	Accelerated junctional tachycardia

Ventricular arrhythmias often occur earlier after the onset of AMI. The pathomechanism of arrhythmia is multifactorial, including ongoing ischemia, hemodynamic instability, and electrolyte imbalance (hypomagnesemia, hypokalemia), metabolic disorders (acidosis, hypoxia), re-entry, and increased automaticity (1). The formation of ventricular action potentials by ion-dependent voltage and current substrates are the cornerstone for each myocardial cell contraction in each person. This ionic equilibrium can be disrupted by the presence of disorders, such as ischemia/reperfusion. Arrhythmogenesis at the beginning of ACS course, which manifests as polymorphic VT or VF, occurs in a small proportion of patients with acute ischemia and is frequently related to a genetic predilection (6). The incidence of death in the hospital, owing to VT/VF or acute heart failure (HF), has decreased with the wide-ranging utilization of reperfusion strategies (3).

Myocardial reperfusion also may result in complex electrophysiological changes, depending on previous ischemia duration. VT is more common with increased ischemia duration, but when there is widespread of myocardial injury, VT incidence decreases. Significant contributors involved in the development of arrhythmia are the delayed activated K+rectifier current, Na+/Ca2+ exchange pump, and sarcoplasmic reticulum proteins phosphorylation by Ca2+/calmodulin dependent protein kinase type II (CAMKII). An excess of intracellular Ca2+ (partly caused by oxidative stress) will cause spontaneous Ca2+oscillation (excess calcium paradox) that triggers initial and delayed depolarization, which induces ectopic pulses. Also, these will contribute to temporal and spatial repolarization dispersions (variations in duration of the action potential), and reentrant arrhythmias build upon one-way conduction blocks, cellular electronic fractionation programs, and short duration of action potentials. The important arrhythmogenic mechanism is the current flowing from the ischemic/reperfusion area to the non-ischemic zone (3,7).



MI: Myocardial infarction; VA: Ventricular arrhythmia; VT: Ventricular tachycardia; VF: Ventricular fibrillation; WPW: Wolf-Parkinson-White Syndrome; ARVC: Arrhythmogenic right ventricular cardiomyopathy; HCM: hypertrophic cardiomyopathy; DCM: dilated cardiomyopathy (3).

Figure 1. Arrhythmogenesis in ACS. Pre-existing substrates for VA, both secondary to long-standing MI, inherited cardiomyopathy, or genetic predilection to VA, and interacting with acute ischemia, autonomic imbalance, and acute LV strains to result in triggered activity and VA

The mechanism accountable for the induction of VA may differ according to underlying condition (Figure 1): re-entry (intramural) in the ischemic process, while triggered activity seems to be a primary mechanism in reperfusion. Moreover, when the mechanisms accountable for the occurrence of VT, the role of these mechanisms may be different but joined to play a role in both arrhythmia severity and SCD (3).

Ventricular Tachyarrhythmia

Predictor and Outcome of Ventricular Arrhythmia in ACS

Sudden cardiac death due to sustained VA often occurs in untreated MI patients (3). Early VT events (<48 hours) can be found in up to 6% of ACS patients. Although early VT worsens the early course of ACS, studies had not invariably exhibited an association with poor long-term survival. Further, appropriate management in early VT patients in ACS is controversial. Based on a statement in some consensus, ICD implantation in early VT patients was appropriate only in LVEF ≤35% and obstructive coronary artery disease (CAD) irreparable with revascularization, or LVEF <50% with non-sustained VT and positive electrophysiology study (8,9).

Although there is an apparent decrease in the prevalence of SCD with better reperfusion and CAD prevention through statin therapy and smoking cessation, ACS and advanced arrhythmias after AMI remain the cause of SCD. A large number of SCD events take place in the pre-hospital ACS phase, underscoring the importance of the role of the screening program in identifying patients at risk. The VA incidence in the hospital phase has also reduced recently, mostly due to early revascularization, and adequate initial pharmacological management. Still, up to 6% of patients with ACS can develop VT or VF within the first 48 hours after the onset of MI, frequently before or during revascularization (8).

VA that appears in the first few days of MI can be caused by the following things, summarized in Figure 1:

(1) Late presenter i.e., patients whose reperfusion therapy is delayed due to inadequate care chain from the onset of symptoms to the acute coronary care center or delays related to the patient himself ('severe acute ischemia' in Figure 1).

(2) Patients whose revascularization is unsuccessful or only partially successful due to technical difficulties ('severe acute ischemia' in Figure 1).

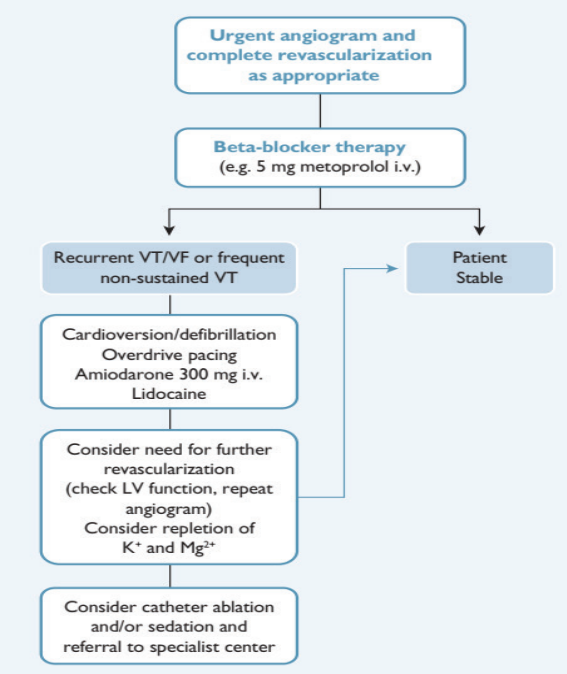
(3) Patients who have an arrhythmogenic substrate before the event, either due to previous infarction or due to a tendency to electrophysiological instability ('pre-existing myocardial damage' in Figure 1).

Ventricular Arrhythmia Management in ACS

Ventricular arrhythmias are associated with worsening hemodynamics that occurs during the acute, sub-acute, or chronic phase of ACS and must be treated with DC cardioversion immediately. Strategies for treating recurrent arrhythmias in ACS involved reperfusion, pharmacological therapy, intravenous sedation, over-drive pacing, and catheter ablation. PRAMI studies show better outcomes with complete revascularization, compared to only ST-elevation myocardial infarction (STEMI) culprit artery (10). Incomplete revascularization can encourage ongoing VA in ACS patients, although data are currently limited to support this hypothesis. European Society of Cardiology (ESC) guidelines recommend "prompt and complete revascularization" is performed in treating myocardial ischemia with recurrent VT and VF (11).

Recent retrospective trials mentioned VA occur in up to 6% in the initial phase of AMI and show the importance of VT / VF in this condition. The role of anti-arrhythmic drug therapy (AAD) in treating sustained VT / VF in the ACS is still very debatable. There are limited data from clinical studies that mention the use of AADs in ACS. Randomized Control Trial (RCT) comparing several different AADs in ACS is still lacking (3).

Early use of beta-blockers in ACS reduces the incidence of VA and mortality (Figure 2). Likewise, hypomagnesemia and hypokalemia correction are recommended due to the possible role of electrolyte imbalance in VA. Treatment with statin decreases death in CAD and includes recommended routine treatment. Intensive and early statin treatment had been published to decrease the occurrence of ventricular premature beat (VPB) and non-sustained VA (3).



VA: Ventricular arrhythmia; ACS: Acute Coronary Syndrome; VT: Ventricular tachycardia; VF: Ventricular fibrillation; K+: Potassium; Mg2+: Magnesium (8)

Figure 2. Diagnostic studies in patients with sustained VA and ACS

AADs are broadly utilized but have efficacy that is not too good and has significant side effects. Some data show the potential dangers of most AAD therapies for ischemic heart disease patients. However, even with advances in non-pharmacological therapy alternatives such as catheter ablation, short-term or long-term AAD therapy may still be regarded for refractory VA treatment. Early efforts to overcome VT/VF in ACS, even if repeated, cardioversion (CV) / defibrillation must be carried out. AAD therapy should be considered if VT / VF episodes are frequent, and sequential CV/defibrillation cannot control it. Catheter ablation has proven to be very effective and should be considered in recurrent VT / VF that are triggered by VPB coming up from injury to some Purkinje fibers (3).

Lidocaine is AAD class I, which can decrease the VA incidence associated with myocardial ischemia, although there are no beneficial effects on early death. Because of the potential for pro-arrhythmias, with a high tendency for mortality, prophylactic treatment with lidocaine is mostly not recommended (8). Otherwise, successful prophylactic therapy using lidocaine after cardiac arrest and resuscitation has displayed significantly favorable results for recurrent VA as well as survival. Nevertheless, other reports mention a lower risk of mortality in the first 24 hours post-thrombolysis and use of lidocaine prophylactic; and an indistinctive effect on all-cause mortality. Based on the efficacy and potential risk, lidocaine must be accounted for intravenous AAD in recurrent VT/VF acute management in ACS (3).

Amiodarone is AAD class III with several anti-arrhythmic properties. Amiodarone may hold an equal efficacy and risk profile in patients with severe structural heart disease (SHD). Two clinical trials (the Canadian Amiodarone Myocardial Infarction Trial the and European Amiodarone Myocardial Infarction Trial) evaluates amiodarone as primary prevention in patients recovered from MI, cardiac mortality and mortality due to arrhythmia, or cardiac arrest that is resuscitated, was significantly lower in the amiodarone group, compared to placebo, if the patient also received beta-blockers. There appears to be no advantage to amiodarone compared to placebo in patients who do not receive beta-blockers (3).

Amiodarone therapy results in high mortality if used in long-term in advanced HF patients. However, amiodarone considered includes types of AADs that is absent or having a small effect on long-term outcomes when used in severe SHD and/or extensive MI patients. In ACS, therapy using amiodarone - compared to lidocaine - has exhibited an increase in short-term and long-term mortality retrospectively. Besides that, in cardiac arrest outside the hospital, amiodarone is associated with better admission rates and survival compared to lidocaine in patients with shock-refractory VF. Amiodarone should be accounted for prevention (oral) and suppression (oral or intravenous) of recurrent arrhythmias together with beta-blockers (3). Flecainide, ajmaline, propafenone, azimilide, dofetilide, and ranolazine are among anti-arrhythmic drugs that have been mainly deserted or not recommended in ACS setting due to its risk of increased mortality and lack of evidence (3).

Catheter ablation for VA in the acute phase is uncommon. The success rate in the acute phase is 70% and carries peri-procedural mortality of 3% in unstable patients and long-term mortality of 18% due to decompensated HF (7). Catheter ablation is usually used in recurrent VT/VF and/or electrical storm despite had been treated by revascularization, CV/defibrillation, and other pharmacological therapies (Figure 2). ESC issued a comprehensive recommendation for the prevention and management of SCD in ACS, including the use of revascularization, LV assist devices, and also pharmacological treatment (Table 2) (8).

Table 2. ESC recommendations for prevention and management of SCD in ACS (8)

Recommendations	Class[a]	Level[b]
Urgent reperfusion is recommended in patients with STEMI	I	A
Coronary revascularization is recommended in patients with NSTEMI or unstable angina according to the ESC NSTEMI guidelines	I	C
A coronary angiogram followed, if necessary, by coronary angioplasty within 2 h of hospital admission is recommended in patients with high-risk NSTEMI, which also includes life-threatening VA	I	C
Prompt and complete coronary revascularization is recommended to treat myocardial ischemia that may be present in patients with recurrent VT or VF	I	C
Beta-blocker treatment is recommended for recurrent polymorphic VT	I	B
Intravenous amiodarone is recommended for the treatment of polymorphic VT	I	C
Immediate electrical cardioversion or defibrillation is recommended in patients with sustained VT or VF	I	C

Urgent coronary angiography followed, when indicated, by revascularization is recommended in patients with recurrent VT or VF when myocardial ischemia cannot be excluded	I	C
Correction of electrolyte imbalances is recommended in patients with recurrent VT or VF	I	C
Oral treatment with beta-blockers should be considered during the hospital stay and continued thereafter in all ACS patients without contraindications	IIa	B
Radiofrequency catheter ablation at a specialized ablation centre followed by the implantation of an ICD should be considered in patients with recurrent VT, VF or electrical storms despite complete revascularization and optimal medical treatment	IIa	C
Transvenous catheter overdrive stimulation should be considered if VT is frequently recurrent despite use of anti-arrhythmic drugs and catheter ablation is not possible	IIa	C
Intravenous lidocaine may be considered for the treatment of recurrent sustained VT or VF not responding to beta-blockers or amiodarone or in the presence of contraindications to amiodarone	IIb	C
Prophylactic treatment with anti-arrhythmic drugs (other than beta-blockers) is not recommended	III	B

ESC: European Society of Cardiology; SCD: sudden cardiac death; ACS: acute coronary syndrome; STEMI: ST-segment elevation myocardial infarction; NSTEMI: non–ST-segment elevation myocardial infarction; VT: ventricular tachycardia; VF: ventricular fibrillation.
[a]Class of recommendation; [b]Level of evidence

Supraventricular Tachyarrhythmia

Atrial Fibrillation in ACS

Atrial fibrillation (AF), which is the most frequent clinical arrhythmia, also often occurs in acute MI. AF aggravates the course of AMI in 2.3-21% of admitted patients. In recent years, the wide-ranging use of early revascularization therapy, besides the use of beta-blockers, ACE-I, and angiotensin II receptor blockers, has caused a significant reduction in AF post-MI incidence. Nevertheless, the incidence of AF increases with age, so we can assume that AF still becomes AMI complication that needs attention (3).

Pre-existing atrial fibrillation attributes about one-third of all AF cases in AMI, while new-onset AF two-thirds of cases (12). When AF appears, there is usually a significant hemodynamic disorder due to irregular ventricular filling, high ventricular response, and/or absence of atrial contribution to cardiac output. Independent predictors of AF in AMI include old age, increased heart rate at admission, LV hypertrophy, pre-existing AF, symptoms of HF, and LV dysfunction. In addition, AF patients more often to have diabetes, hypertension, previous MI, multivessel CAD, high cardiac markers, and low TIMI flow after revascularization therapy (3).

AF increases the risk of the hospital, short-term (<30 days), medium-term (>30 days to 1 year), and long-term (>1 year) mortality in AMI patients, with at least an increase risk of 5.7%-40%, regardless of AF type (new-onset or pre-existing, symptomatic or asymptomatic). Including an increased risk of both the death due to SCD and non-SCD (13,14). This risk differs according to the time of the onset of AF, and the highest mortality occurs in AF that appears >30 days after MI (15). Recommendations for atrial fibrillation management in ACS are as follows (3,16,17):

• Rate control is recommended using beta-blockers or likely calcium channel blockers. In severe LV dysfunction/acute HF patients without hypotension, amiodarone is recommended. In severe LV dysfunction/acute HF patients with hypotension, it is recommended using digitalis.

• Cardioversion is required if the rate control cannot be achieved immediately using pharmacological therapy in patients with ongoing ischemia, severe hemodynamic instability, or HF.

• For sinus rhythm conversion, amiodarone can be considered, in addition to an electrical CV. Other AADs may be dangerous during AMI. Intravenous amiodarone is also indicated to reduce the risk of early recurrence of AF after electrical CV in new unstable onset AF patients.

• Appropriate anticoagulation, based on the individual risk of embolism and stroke, usually requires a period of combination treatment of antiplatelet and anticoagulants.

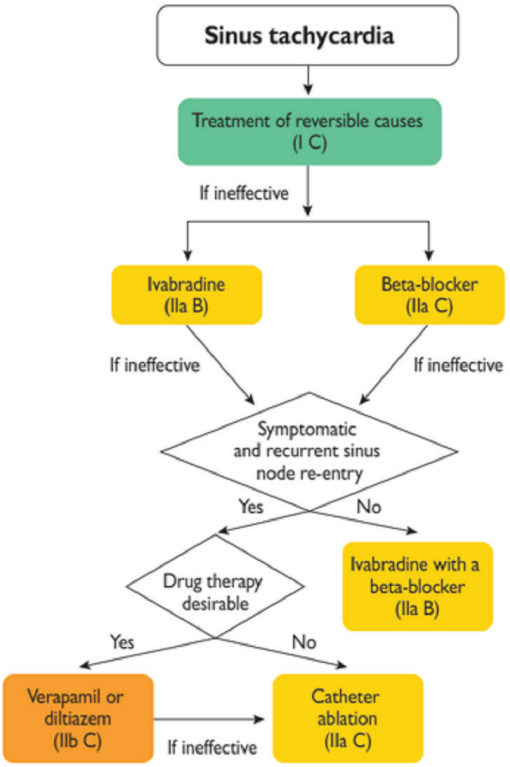
The topic of anticoagulation in AF has become controversial because AF may only occur temporarily during AMI. Patients with a CHA2DS2-VASc score of 0 or 1, instead, may not need anticoagulant and only treated with dual antiplatelet therapy (12). Current evidence suggests that patients with the first AF episode during MI have a 13-24% risk of having recurrent AF after 1037 days (median follow-up) compared to only 6% who remain sinus rhythm during MI (12,18).

Other Supraventricular Tachyarrhythmias

Sinus Tachycardia

Factors causing physiological sinus tachycardia are, by definition, physiological (stress, activity, or pregnancy), and may also arise secondary to medical conditions or other drugs, including myocardial infarction (19). Even though sinus tachycardia is a normal physiological response to stress, persistent sinus tachycardia after AMI carries an unfavorable prognosis with an increased risk of death.

Sinus tachycardia is related to increased sympathetic nervous system activity and may cause hypertension or transient hypotension. Increased heart rate increases myocardial oxygen demand while reduced diastolic time interferes with coronary blood flow and worsens myocardial ischemia. Persistent sinus tachycardia can be caused by ongoing pain, hypoxemia, HF, hypovolemia, anxiety, anemia, pulmonary embolism, pericarditis, or previous drug administration, such as epinephrine, dopamine, or atropine. Persistent sinus tachycardia in ACS patients requires appropriate examination and treatment, such as oxygen, adequate pain medication for ongoing pain, diuretics for HF, intravascular volume administration for hypovolemia, beta-blockers or nitroglycerin for ischemia, and anti-inflammatory agents for fever or pericarditis (1,4). The ESC guidelines generally recommend that the first step in the management of sinus tachycardia is the evaluation and treatment of reversible causes of sinus tachycardia. If it is not effective, other anti-arrhythmia can be used, such as beta-blockers, ivabradine, calcium blockers, or ablation (Figure 3) (19).



ESC: European Society of Cardiology (19)

Figure 3. ESC recommendation for management of sinus tachycardia

Paroxysmal Supraventricular Tachycardia

Paroxysmal supraventricular tachycardia (SVT) develops in no more than 10% of AMI patients but requires fast and aggressive therapy to reduce further ischemia caused by the rapid heart rate (4). Vagal maneuvers have low risk, such as valsava or carotid sinus massage, with careful examination of carotid bruits, which can sometimes restore sinus rhythm. The drug of choice for paroxysmal SVT in non-AMI patients is adenosine (6–12 mg rapid intravenous/IV) (19). Even though only a few data exist to guide adenosine use in AMI patients, many experts consider that adenosine can be given to AMI safely, provided there is no hypotension (systolic blood pressure <100 mm Hg) (4).

In patients with no significant LV dysfunction, IV diltiazem (15-20 mg), metoprolol (5-15 mg), or verapamil (5-10 mg) can be an alternative therapy. Beta-blockers IV, combined with verapamil IV, must be avoided because the administration of these combinations may cause severe hypotension or high degree atrioventricular (AV) blocks. In patients who experience congestive heart failure (CHF) or severe hypotension, synchronized cardioversion is indicated with an initial dose of 50 J (Figure 4) (4,19).

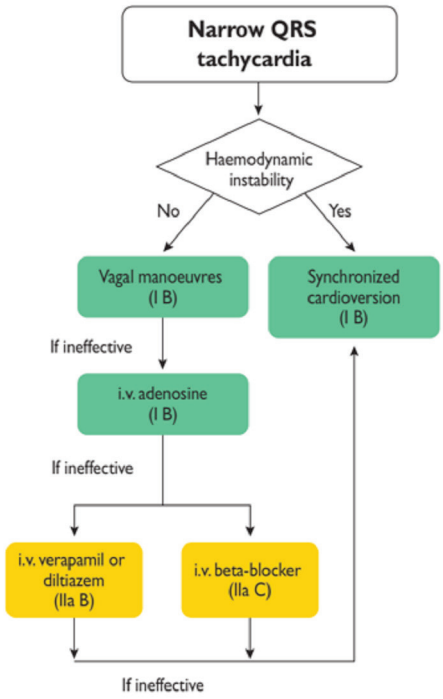
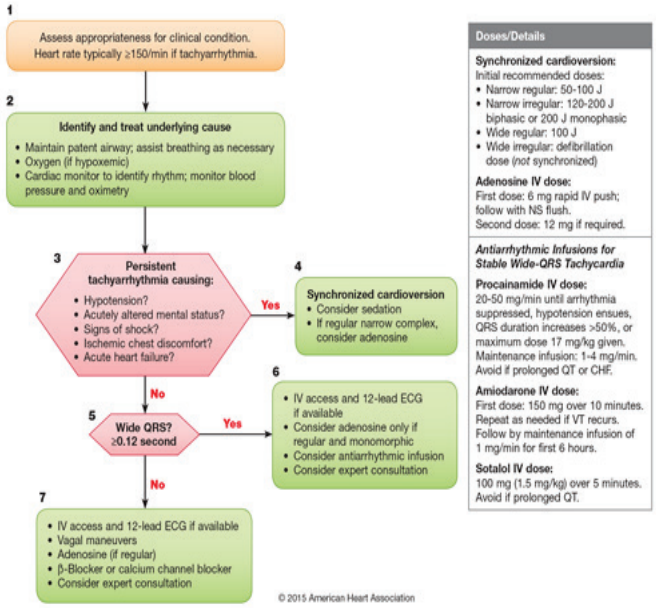


Figure 4. Acute therapy of narrow QRS tachycardia before definitive diagnosis (19)

Tachyarrhythmia in Acute Cardiac Life Support

Tachyarrhythmia in ACS can manifest in several forms and require different approaches to treatment. The most common cause of death at AMI before hospitalization is due to VT/VF. Atrial and ventricular arrhythmias may occur at ACS, including ventricular tachyarrhythmias that can cause the collapse of the circulation system and hence need immediate treatment. AF also requires immediate treatment if the rapid ventricular rate causes hemodynamic disturbances. Generally, in the acute phase of tachycardic patients in the presence of unstable signs or symptoms (for example, decreased level of consciousness, ischemic chest pain, acute heart failure, hypotension, and other signs of shock despite adequate airway and breathing), cardioversion is indicated primarily in: 1. unstable SVT, 2. unstable atrial fibrillation, 3. unstable atrial flutter, and 4. unstable monomorphic (regular) VT. However, if the

patient is stable, further evaluation and management are indicated by referring to the Adult cardiac life support (ACLS) guidelines by American Heart Association (AHA) (Figure 5) (20,21).



VT: ventricular tachycardia (21)

Figure 5. Adult tachycardia with a pulse algorithm

CONCLUSIONS

Ischemia and infarction can cause changes in metabolism and electrophysiology, which subsequently cause both asymptomatic and also life-threatening arrhythmias. In an emergency setting, management of tachyarrhythmia may refer to ACLS guidelines. Prophylaxis anti-arrhythmic is not recommended for ACS patients except beta-blockers. Tachyarrhythmias as the complication of ACS may worsen the outcome of ACS, hence appropriate management of tachyarrhythmias will expectedly improve the outcome of ACS.

Competing interests: The authors declare that they have no competing interest.

Financial Disclosure: There are no financial supports.

REFERENCES

1. Das B, Mishra TK. Prevention and management of arrhythmias in acute myocardial infarction. Int J Contemp Med Res 2016;3:1401-5.
2. Bloch Thomsen PE, Jons C, Pekka Raatikainen MJ, et al. Long-term recording of cardiac arrhythmias with an implantable cardiac monitor in patients with reduced ejection fraction after acute myocardial infarction: The Cardiac Arrhythmias and Risk Stratification after Acute Myocardial Infarction (CARISMA) study. Circulation. 2010;122:1258-64.
3. Gorenek B, Blomström Lundqvist C, Brugada Terradellas J, et al. Cardiac arrhythmias in acute coronary syndromes: position paper from the joint EHRA, ACCA, and EAPCI task force. EP Eur [Internet]. 2014;16:1655-73.
4. Perron AD, Sweeney T. Arrhythmic complications of acute coronary syndromes. Vol. 23, Emergency Medicine Clinics of North America. W.B. Saunders 2005:1065-82.
5. Panth N, Paudel KR, Parajuli K. Reactive Oxygen Species: A Key Hallmark of Cardiovascular Disease. Adv Med 2016;2016.
6. Bezzina CR, Pazoki R, Bardai A, et al. Genome-wide association study identifies a susceptibility locus at 21q21 for ventricular fibrillation in acute myocardial infarction. Nat Genet 2010;42:688-91.
7. Bhar-Amato J, Davies W, Agarwal S. Ventricular arrhythmia after acute myocardial infarction: "The perfect storm." Arrhythmia Electrophysiol Rev 2017;6:134-9.
8. Priori SG, Blomstrom-Lundqvist C, Mazzanti A, et al. 2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death the Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC) Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC). European Heart Journal. Oxford University Press; 2015;36:2793-8671.
9. Hai JJ, Tam E, Chan PH, et al. Incidence and predictors of sudden arrhythmic death or ventricular tachyarrhythmias after acute coronary syndrome: An asian perspective. Hear Rhythm 2017;14:81-7.
10. Wald DS, Morris JK, Wald NJ, et al. Randomized Trial of Preventive Angioplasty in Myocardial Infarction. N Engl J Med [Internet] 2020;369:1115-23.
11. Thomas DE, Jex N, Thornley AR. Ventricular arrhythmias in acute coronary syndromes-mechanisms and management. Contin Cardiol Educ [Internet]. 2017;3:22-9.
12. Kalarus Z, Svendsen JH, Capodanno D, et al. Cardiac arrhythmias in the emergency settings of acute coronary syndrome and revascularization: An European Heart Rhythm Association (EHRA) consensus document, endorsed by the European Association of Percutaneous Cardiovascular Interventions (EAPCI), and European Acute Cardiovascular Care Association (ACCA). Europace. 2019;21:1603-4.
13. Jabre P, Roger VL, Murad MH, et al. Mortality associated with atrial fibrillation in patients with myocardial infarction: A systematic review and meta-analysis. Circulation 2011;123:1587-93.
14. Stamboul K, Fauchier L, Gudjoncik A, et al. New insights into symptomatic or silent atrial fibrillation complicating acute myocardial infarction. Archives of Cardiovascular Diseases. Elsevier Masson SAS 2015;108:598-605.
15. Jabre P, Jouven X, Adnet F, et al. Atrial fibrillation and death after myocardial infarction: A community study. Circulation 2011;123:2094-100.

16. Kirchhof P, Benussi S, Kotecha D, et al. 2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. Eur Heart J [Internet]. 2016;37:2893-962.
17. Ibanez B, James S, Agewall S, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J [Internet]. 2018;39:119-77.
18. Guenancia C, Toucas C, Fauchier L, et al. High rate of recurrence at long-term follow-up after new-onset atrial fibrillation during acute myocardial infarction. Europace 2018;20:179-88.
19. Brugada J, Katritsis DG, Arbelo E, et al. 2019 ESC Guidelines for the management of patients with supraventricular tachycardiaThe Task Force for the management of patients with supraventricular tachycardia of the European Society of Cardiology (ESC). Eur Heart J 2019.
20. Neumar RW, Otto CW, Link MS, et al. Part 8: Adult advanced cardiovascular life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Circulation 2010.
21. Link MS, Berkow LC, Kudenchuk PJ, et al. Part 7: Adult advanced cardiovascular life support: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation. 2015;132:444-64.