Prevention and Management of Arrhythmias in Acute Myocardial Infarction

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ABSTRACT

Different types of arrhythmia (both brady- and tachyarrhythmia) may occur in myocardial infarction patients which may be silent or symptomatic (incidence around 75%). Many arrhythmias are fatal requiring urgent treatment and increases mortality while others are benign and do not affect prognosis of patients. Pathophysiology of arrhythmias is different in early versus late arrhythmia, also treatment modalities. Various factors contribute to arrhythmogenesis in acute myocardial infarction (AMI) like metabolic changes, electrophysiological changes, increased sympathetic activity, vagal stimulation, left ventricular (LV) ejection fraction and scar formation. Prevention and prompt treatment of these arrhythmias are important in decreasing mortality in AMI both peri-infarct period and follow-up.

Keywords: Prevention, Arrhythmias, Acute Myocardial Infarction

INTRODUCTION

It is known that ischemia and infarction leads to metabolic and electrophysiological changes that may cause silent and symptomatic life-threatening arrhythmia. At least 75% of patients with acute myocardial infarction (AMI) have an arrhythmia during the peri-infarct period.[1] Sudden cardiac death (SCD) is most often attributed to this pathophysiology and around one-half of death occurs before patient reach hospital. Cause of death in AMI before hospitalization is most often ventricular tachycardia/ventricular fibrillation (VT/VF). Both atrial and ventricular arrhythmia may occur in setting of acute coronary syndrome (ACS) including ventricular tachyarrhythmia which may cause circulatory collapse and hence need immediate treatment. Atrial fibrillation (AF) may also warrant urgent treatment when a fast ventricular rate is associated with hemodynamic deterioration. The management of other arrhythmia is also based largely on symptoms. Prophylactic antiarrhythmic management strategies have largely been discouraged. Improvement in medical care, early relief of ischemia, use of beta-blocker, angiotensin-converting enzyme inhibitor (ACE-1) have declined incidence of arrhythmia, still it remain major cause of mortality in these patients. Use of implantable cardioverter-defibrillator (ICD) has promising effect in primary and secondary prevention of ventricular arrhythmia (VA) in ACS patients.

DIFFERENT TYPES OF ARRHYTHMIA AND THEIR INCIDENCE IN AMI

Different types of arrhythmia (both brady and tachy) can occur as shown in Table 1. A higher incidence of bradyarrhythmia is associated with inferior and posterior AMI, compared to anterior and lateral AMI. According to one study, around 3.7% of patients with inferior or posterior AMI developed complete heart block, only 1% of those with anterior or lateral AMI developed complete heart block.[2] Similarly, diagnosis of first-degree atrioventricular (AV) block (1.1% vs 0.6%), second- degree AV block types I (1.1% vs 0.4%) and II (0.2% vs 0.0%) were more common in the context of inferior or posterior compared to anterior or lateral AMI. There was no difference in the incidence of VT by AMI location (7.3% in inferior or posterior AMI versus 7.9% in anterior or lateral AMI, HR = 0.89, p = 0.064) while VF was marginally more frequent among patients with an anterior or a lateral AMI (9.0% vs 8.1%, HR = 0.65, p = 0.023). Table 2 shows incidence of different types of arrhythmia according to location of AMI. Patients with anterior or lateral AMI were more likely to die prior to hospital discharge than patients with an inferior or posterior AMI (11.3% vs 7.7%).

ETIOPATHOGENESIS OF DIFFERENT ARRHYTHMIA

According to various studies, about 30% of patients experience sinus tachycardia, especially those with anterior location. Mechanism of sinus tachycardia is due to physiologic response to left ventricular (LV) dysfunction or stimulation and overactivity of sympathetic nervous system due to various factors like pain, anxiety, persistent pain, epinephrine, or dopamine; rarely, it occurs in patients with atrial infarction.

Although the reported incidence of atrial tachyarrhythmia, they are uncommon during early phase of myocardial infarction (MI).[3] These are often transient. Pathophysiologic mechanisms of development of these arrhythmia are they are augmented sympathetic stimulation of the atria and often occur in patients with LV failure, pulmonary emboli in which the arrhythmia intensifies hemodynamic deterioration, or atrial infarction, pericarditis. In AF, worsening of hemodynamics is due to fast ventricular rate, loss of atrial contribution to cardiac output.

Ventricular arrhythmias are common early after onset of AMI. The mechanism for these arrhythmias is multifactorial and includes ongoing ischemia, hemodynamic and electrolyte abnormalities (hypokalemia, hypomagnesemia),

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metabolic abnormalities (acidosis, hypoxia), re-entry, and enhanced automaticity. Acute myocardial ischemia leads to ATP deficiency, anaerobic glycolysis causing acidosis, elevation of extracellular potassium and lyso-phosphatidylcholine accumulation. This sequence of events results electrophysiologically in: (1) ionic imbalance, (2) less contractile force by events that culminate in mishandling of intracellular calcium and (3) reduced conduction velocity because of less functional gap junctions.[4] Ionic imbalance in turn leads to (a) shorter duration of the action potential by activation of the substrate-related potassium current: IK, ATP. Myocardial reperfusion may cause profound electrophysiological alterations,, depending on the prior duration of ischemia. I<+ current and phosphorylation of sarcoplasmic reticulum proteins by CAMKII (calcium and calmodulin-dependent protein kinase II).5 The intracellular Ca2+ overload (among others caused by reactive oxidative stress) will result in spontaneous Ca2+ oscillations (calcium overload) the ischemic/reperfused to the nonischemic (Figure l), 6 (intramural) re-entry in ischemia, whereas triggered activity appears to be the dominant mechanism in reperfusion. Arrhythmogenesis early in the course of an ACS, manifested as often polymorphic VT. Ventricular arrhythmia late after ACS is due to scar re-entry and incomplete revascularization.

PREDICTORS OF ARRHYTHMIA IN AMI

One should evaluate the risk of reoccurrence of a rhythm disturbance in AMI especially VT or VF as they are potentially lethal events. Arrhythmia risk evaluation includes, beside the careful reading of standard electrocardiogram (ECG), a series of useful more or less routine investigations (Table 3).[8] ECG features with significant arrhythmia risk are premature ventricular beats (PVBs) with profoundly altered morphology, polymorphic ventricular premature contraction (VPC), frequent ventricular couplets, triplets, nonsustained ventricular tachycardia (NSVT) episodes, QRS duration greater than 160 ms in patients with complete left bundle branch block (LBBB); alternation of T-wave amplitude (of microvolts); QT dispersion greater than 100 ms.

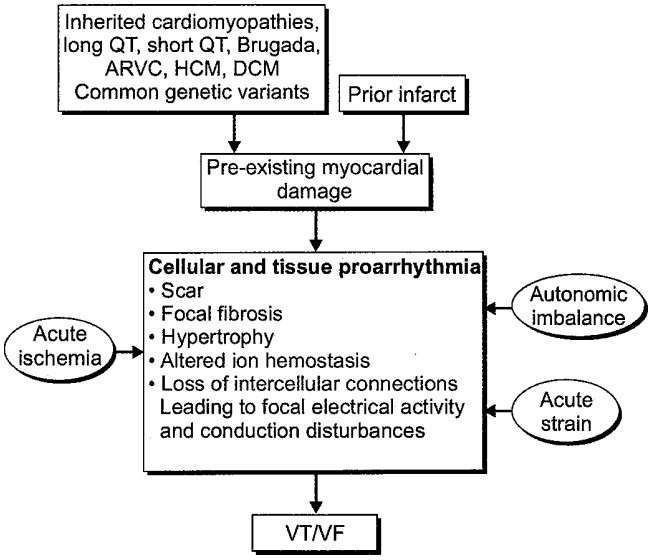

Figure-1: Mechanism of arrhythmia

Heart rate variability (HRV) can be analyzed in time and frequency domain using a 24-hour Holter ECG recording. post:MI and tends to return during the first year. The autonomous Tone and reflexes in acute myocardial infarction (ATRAMI)[9] study confirmed that HRV and baroreflex sensitivity (BRS) remains a significant predictor of cardiac mortality in patients after AMI (< 28 days). Also according to this study, the combination of low values of HRV and reduced left ventricular ejection fraction (LVEF) is useful for SCD risk stratification in patients after AMI.

Category	Arrhythmia
Ventricular tachy	Ventricular premature beats (VPC) Ventricular tachycardia/ventricular fibrillation (VT/VF), accelerated idioventricular rhythm (AIVR)
SVT	Sinus tachycardia Atrial fibrillation and/or atrial flutter Paroxysmal supraventricular tachycardia
Brady	Sinus bradycardia Junctional escape rhythm Atrioventricular block Intraventricular block

SVT, supraventricular tachycardia; AMI, acute myocardial infarction.

Table-1: Different types of tachy- and bradyarrhythmia in AMI

Conduction abnormalities	Inferior or posterior AMI	Anterior or lateral AMI
First degree AV block	1.10%	0.60%
Mobitz 1	1.10%	0.40%
Mobitz 2	0.20%	0.00%
Complete AV block	3.70%	1.00%
VT	7.30%	7.90%
VF	8.10%	9.00%
Death	7.70%	11.30%

AV, atrioventricular; VT, ventricular tachycardia; VF, ventricular fibrillation; AMI, acute myocardial infarction.

Table-2: Different types of arrhythmia by AMI location

Standard ECG
- Stress ECG, looking for:
  - Effort-induced arrhythmias
  - Premature beats, QT interval (adaptation to heart rate)
  - Heart rate adaptation to effort (maximum value, return to normal rhythm)
- Bedside ECG
- Ambulatory ECG monitoring (Holter), analyzing:
  - Eventual sustained or unsustained arrhythmias
  - Number of PVB, heart rate variability
  - Heart rhythm turbulence, QT variability
- Long-term monitoring with implantable devices
- Signal-averaged electrocardiography (SAECG)
- Evaluation of T-wave alternans (or MTWA—microvolt T-wave alternans)
- Evaluation of the baroreceptor reflex sensitivity
- Electrophysiological exploration (with programmed stimulation

ECG, electrocardiography; PVB, premature ventricular beat

Table-3: Evaluation for predictors of arrhythmia

Heart rate turbulence (HRT) is represented by variations in sinus rhythm cycle length determined by a VPC.

Signal-averaged electrocardiography (SAECG), allows the highlighting of small amplitude potentials, of the order of microvolts at the end of QRS complex. The presence of these conditions is labelled as late potential present/positive SAECG which are considered predisposing to re-entrant arrhythmias.

Signal-averaged electrocardiography and microvolt T-wave alternans (MTWA) have the negative predictive value of 97-99% is of practical importance.

PROGNOSIS OF ARRHYTHMIA IN AMI

Ventricular premature beats are almost universal on the first day of the acute phase and complex arrhythmias (multiform complexes, shortrunsortheR-on-Tphenomenon) are common.

An accelerated idioventricular rhythm (AIVR) typically occurs during the first 2 days, with about equal frequency in anterior and inferior infarctions. Most episodes are of short duration. Accelerated idioventricular rhythm is often observed shortly after successful reperfusion has been established with fibrinolytic therapy. However, the frequent occurrence of this rhythm in patients without reperfusion limits its reliability as a marker of the restoration of patency of the infarct-related coronary artery. In contrast to rapid VT, AIVR is thought not to affect prognosis, and routine treatment of AIVR is not indicated.

Ventricular tachycardia/ventricular fibrillation: Among patients who underwent fibrinolytic therapy in the (GUSTO-I) study, approximately 10% experienced VT/VF. In the Assessment of Pexelizumab in Acute Myocardial Infarction (APEX- AMI) study, which included patients treated with primary percutaneous coronary intervention (PCI), sustained VT/VF developed in 5.7%. Clinical outcomes were worse in patients with VT/VF than in those without VT/VF. Compared with patients without VT/VF, 90-day mortality risk was 2-fold higher for patients with early VT/VF and 5-fold higher in late VT/VF.12 Result of French Registry of Acute ST-Elevation or Non-ST Elevation Myocardial Infarction (FAST-MI) 2005 registry which was recently published, the overall in-hospital mortality was significantly higher among VF patients (25.0% vs 5.0% for non-VF patients). When considering VF timing, in-hospital mortality was higher in the late VF group as compared to the early VF group (33.3% vs 22.8%; $p < 0.001$). Also the investigators concluded that, while patients who develop VF during their index hospitalization for AMI are at significantly higher risk of in-hospital mortality, they are not at higher long-term mortality risk or SCD mortality.13

PREVENTION AND MANAGEMENT OF ARRHYTHMIA IN AMI

Sinus Tachycardia

As it represents physiologic response to LV dysfunction and overactivity of sympathetic system, treatment includes optimizing hemodynamics and oxygenation, correction of anemia, electrolyte and acid-base disorder, pain control,

Recommendations	Class	Level
Direct current cardioversion is indicated for sustained VT and VF	1	C
Sustained monomorphic VT that is recurrent or refractory to direct current cardioversion: should be considered to be treated with IV amiodarone	IIa	C
May be treated with IV lidocaine or sotalol	IIb	C
Transvenous catheter pace termination should be considered if VT is refractory to cardioversion or frequently recurrent despite antiarrhythmic medication	IIa	C
Repetitive symptomatic salvoes of nonsustained monomorphic VT should be considered for either conservative management (watchful waiting) or treated with IV beta-blocker, or sotalol, or amiodarone	IIa	C
Polymorphic VT		
Must be treated by IV beta-blocker	I	B
Or IV amiodarone	I	C
Urgent angiography must be performed when myocardial ischemia cannot be excluded	I	C
May be treated with IV lidocaine	IIb	C
Prompt assessment and correction of electrolyte disturbances, consider magnesium	I	C
Should be treated with overdrive pacing using a temporary transvenous right ventricular lead or isoproterenol infusion	IIa	C
Management of ventricular arrhythmias and risk evaluation for sudden death on long-term		
Specialized electrophysiological evaluation of ICD implantation for secondary prevention of sudden cardiac death is indicated in patients with significant LV dysfunction, who suffer from hemodynamically unstable sustained VT or who are resuscitated from VF occurring beyond the initial acute phase	1	A
Secondary preventive ICD therapy is indicated to reduce mortality in patients with significant LV dysfunction, and hemodynamically unstable sustained VT or survived VF, not occurring within the initial acute phase	1	A
Risk evaluation for sudden cardiac death should be performed to assess indication for primary preventive ICD therapy by assessing LVEF (from echocardiography) at least 40 days after the acute event in patients with LVEF < 40%	1	A
VT, ventricular tachycardia; VF, ventricular fibrillation; ICD, implantable cardioverter-defibrillator; LV, left ventricle; LVEF, left ventricular ejection fraction; ESC, European Society of Cardiology; STEMI, ST-elevation myocardial infarction; AMI, acute myocardial infarction.		

Table-4: ESC 2012 STEMI guidelines for management of arrhythmia in AMI

anti-anxiety drugs. Beta-blockers are indicated for patients without evidence of significant LV dysfunction.

Atrial Tachyarrhythmia

Atrial fibrillation has been reported to complicate 2.3-21% of acute AMI patients.[14] Pre-existing AF observed in patients with AMI, and new-onset AF for the remaining two-thirds. ACE-Is and angiotensin II inhibitors has led to a substantial decline in the incidence of post-MI AF. (2.5-5 mg every 2-5 minutes to a total of 15 mg) or atenolol after heart failure and underlying conduction disease have been excluded. When there are absolute contraindications to beta first- line therapy for management of acute AF, but it plays role in patients with heart failure or LV dysfunction. Urgent direct current cardioversion (DCCV) is recommended for patients with severe hemodynamic instability or intractable ischemia, In addition to electrical cardioversion (CV), amiodarone can be used for restoration of sinus rhythm when the hemodynamic situation is stable.drugs (AADs) because of its limited negative ionotropic effect. Benefit of triple antithrombotic regimen should be weighed against risk of bleeding and accordingly type of stent [bare-metal stent (BMS) or drug-eluting stent (DES)] should be chosen. In patients with ACS, receiving triple antithrombotic therapy HAS-BLED score was found to have diagnostic value in prediction of bleeding. Necessary15 which is usually 1 month after BMSs and 3 to 6 month after DES. ("dual" therapy) for up to 12 months, and then by anticoagulant monotherapy lifelong.

VENTRICULAR ARRHYTHMIA

Prophylaxis

Because hypokalemia can increase the risk for development of VT, low serum potassium levels should be identified quickly after admission for ST-elevation myocardial infarction (STEMI) and should be treated promptly.[16] Despite the lack of a consistent relationship between hypomagnesemia and VAs, magnesium deficits may still be linked to risk because patients with STEMI have reduced intracellular magnesium levels not adequately reflected by serum measurements. As noted earlier, magnesium should be repleted to achieve a serum level of 2 mEq/L. Early beta-blocker use has reduced VF and can be instituted in patients without contraindication. Lidocaine prophylaxis to prevent primary VF is no longer advised. Early revascularization reduces incidence of VA. Early and intensive statin therapy reduces incidence of premature ventricular contraction (PVC) and nonsustained VT.

Management

With widespread use of revascularization therapy limiting size of infarction and to increased use of beta-blocker, the incidence of sustained VT/VF has declined. Still it remains a major cause of mortality in ACS patients. Direct current CV/defibrillation is the treatment of choice in VT/VF. If ischemia is suspected to be responsible for arrhythmia, immediate reperfusion is of utmost importance. Early use of beta-blocker is done in absence of contraindication. Correction of any electrolyte abnormalities, if present is encouraged. Acidosis is not corrected by sodium bicarbonate infusion as it may increase hemodynamic burden, rather it is better to treat acidosis by hyperventilation.

Polymorphic ventricular tachycardia should be treated with DCCV, IV beta-blocker, IV amiodarone, urgent revas¬cularization, and correction of electrolyte abnormality (Table 4).

Recurrent VT/VF should be treated with repeat DCCV/DF. Antiarrhythmic drug treatment should be considered only if episodes of VT/VF are frequent and can no longer be controlled by successive CV/DF. IV amiodarone is antiarrhythmic drug of choice followed by iv lidocaine.[17] In patients with recurrent VT/VF triggered by VPC arising from partially injured Purkinje fibers, catheter ablation has been shown very effective and should be considered. VT/VF not responding to above measures, implantation of percutaneous left ventricular assist device (LVAD) and extracorporeal membrane oxygenation (ECMO) assisted primary PCI has been found promising as a bridge to recovery. ECMO-assisted PCI has been shown to significantly improved recovery and survival in patients with cardiogenic shock and refractory VT/VF. Proper hemodynamic support with inotropes and vasopressor is required and caution must be taken for dopamine use, as it increases risk of arrhythmia in patients with shock. Other inotropes are less arrhythmogenic. Sustained VT/VF developing beyond acute period (provided the arrhythmia is not due to reversible causes, like electrolyte imbalance, transient, ischemia, reinfarction) is liable to recur and is associated with high risk of death. Among survivors of VT/VF, ICD therapy is associated with significant mortality reduction compared to AADs. With exception of beta-blocker, AADs have not shown to be effective and should not be used for prevention of arrhythmia and death. An ICD should be recommended as a part of secondary prevention to reduce mortality before hospital discharge.

Implantable cardioverter-defibrillator implantation for primary prevention has been shown to reduce all-cause mortality in patients with reduced ejection fraction (< 40%) as a result of infarction that occurred at least 40 days earlier. ICD implantation should be deferred until at least 40 days after acute event to allow stunned myocardium to recover of function.

Ventricular Asystole and Electromechanical Dissociation

Asystole and electromechanical dissociation Other resuscitative measures, including chest compressions, atropine, vasopressin. Non- sustained VT are also seen commonly following successful reperfusion.[6,7] All these arrhythmias should be treated as mentioned previously.

Bradyarrhythmia

Atrioventricular do not affect prognosis. Vagal stimulation, ischemia and opioids often are responsible. It often requires no treatment. If accompanied by severe hypotension, sinus bradycardia should be treated by iv atropine, AV block is usually associated with inferior infarction and seldom causes adverse hemodynamic effects. Permanent pacemaker implantation is considered if bradyarrhythmia do not recover.

CONCLUSION

Recent improvement in treatment strategies, early use of beta-blocker, statins, ACE-I/angiotensin receptor blocker (ARB) has reduced the incidence of arrhythmia in MI, still

its prevalence is very high and it remains the most common cause of mortality in ACS. Early revascularization is helpful in both prevention and treatment of refractory early VA and also it prevents development of LV dysfunction and scar which is a substrate for development of late VT/VF. Use of ICD for both primary and secondary prevention of late VT/VF has revolutionized the management of arrhythmia and substantially decreased mortality in these patients.

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