Management of Ventricular Tachyarrhythmia Early After Myocardial Infraction: The Role of Implantable Cardioverter Defibrillator and Ablation

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Abstract

Secondary prevention implantable cardioverter defibrillator (ICD) implantation after 48 hours of myocardial infarction (MI) is well documented for many years, but approach to patients with early ventricular tachycardia/ventricular fibrillation (VT/VF) during the first 48 hours of MI, in the term of ICD implantation is the area of some uncertainty. The current paper reviewed a few existing studies on this subject, and follow the guideline recommendations briefly.

Keywords: Early Post-Myocardial Infarction, Ventricular Tachycardia

1. Context

Sustained ventricular tachycardia (VT) or ventricular fibrillations (VF) are considered as the subgroup of malignant ventricular arrhythmias (VA). Patients suffering from untreated acute coronary syndromes (ACS) commonly showed sudden cardiac death (SCD) due to sustained ventricular tachycardia. The incidence of VA depends on the size of jeopardized myocardium, presence of preexisting LV dysfunction, and autonomic instability. Patients with transmural myocardial infarction have a 4-fold higher risk for VA than patients with non-ST-elevation myocardial infarction (NSTEMI). The highest (90%) incidence of VA is the first 48 hours after myocardial infarction, whereas 60% of VA in patients with non-transmural myocardial infarction occurred after 48 hours (1, 2). Incidence is further increased by inherited cardiomyopathies such as long-QT-syndrome, short QT syndrome, Wolff-Parkinson-White (WPW) syndrome, Brugada syndrome, arrhythmogenic right ventricular cardiomyopathy, hypertrophic cardiomyopathy, catecholaminergic polymorphic ventricular tachycardia and other genetic variants.

The incidence of ventricular arrhythmia induced by ACS is difficult to determine, since a great number of patients do not arrive alive in hospitals (3, 4). In patients with ACS who arrive alive in hospital VA is common during the early hours. According to reports from implanted cardiac device, the incidence of non-sustained VT is 13%, sustained VT is 3%, and VF is 3% in the early after MI period (5). Sustained VA reported in 6% of the cases with ACS in retrospective analysis of two randomized trials (GUSTO IIB and GUSTO III) (6).

Urgent revascularization and neurohormonal modulators after myocardial infarction has reduced VA in recent decade (7). In the current setting of coronary care units, patients with sustained VA following an acute myocardial infarction are usually located in the below categories:

- Patients with large myocardial infarction and cardiogenic shock.
- Patients who come very late to the emergency room and delayed reperfusion therapy was performed.
- Patients with incomplete revascularization due to difficult anatomy or technical problems.
- Patients with underlying left ventricular dysfunction (LVD) and previous myocardial scar due to old myocardial infarction.

All of the aforementioned patients are characterized by the presence of severe and/or prolonged ischemia or arrhythmogenic substrates prior to the acute coronary event.

2. Mechanisms of Ischemia-Related Ventricular Arrhythmias

Ventricular arrhythmia early in the course of an acute myocardial ischemia, often manifested as polymorphic VT
or VF, is observed in a minority of patients with acute ischemia and is usually associated with genetic predisposition (8).

Acute myocardial ischemia leads to ATP deficiency, acidosis, increase in extracellular potassium (K+), and lysophosphatidylcholine accumulation. The aforementioned structure of proceedings makes the electrophysiological changes and cellular transmembrane ionic imbalance as below in summary; (a) smaller period of the action potential (b) a reduced amount of negative resting membrane potential (c) fewer contractile force; and (d) and reduced conduction velocity. Myocardial reperfusion may cause many electrophysiological changes, based on the prior duration of ischemia. VT occurs more frequently with increasing duration of ischemia, but later in the course of an ACS, and with extensive myocardial damages, the incidence of VT declines. The important changes in ion channels and cardiac enzyme involved in arrhythmogenesis are the Na+/Ca2+ exchange pump, the slowly activating delayed rectifier K+ current and phosphorylation of sarcoplasmic reticulum proteins by calcium and calmodulin-dependent protein kinase II (CAMKII) (9). The intracellular Ca2+ overload causes early and delayed after depolarization and triggers PVCs. In addition, dispersion of repolarization and prolongation of action potential duration results in unidirectional block and initiation of reentrant ventricular arrhythmia.

3. Specific Features of Patients With Myocardial Infarction and Sustained Ventricular Arrhythmias

Even in the best healthcare systems, only 70% - 80% of patients with transmural myocardial infarction receive reperfusion therapy (10). VF or syncope may be the first presentation of the acute myocardial ischemia, especially in patients who come very late to hospital.

Clues in history and para-clinical investigations of delayed presenter with VA are as follows: higher maximal creatine kinase (CK) values, well-developed Q-waves on the electrocardiography (ECG), markedly reduced LV function, and a long history of symptoms attributable to the coronary event. Patients at high risk of sustained VA in the setting of an acute ischemic event, can be assessed by clinical risk scores such as GRACE or thrombolysis in myocardial infarction (TIMI) (11-15), are likely to have extensive myocardial damage, especially when the ECG shows prominent ischemic changes, the patient presents persistent symptoms after initiation of therapy, or CK/troponin release patterns that suggest extensive myocardial damage (16).

4. Incomplete Revascularization and Cardiac Arrhythmia

Revascularization of the culprit lesion is not possible in the specific settings such as in highly calcified lesion or technically difficult (e.g., bifurcated) lesions. Furthermore, significant stenosis in the non-culprit vessel presents in about 30% - 40% of patients with ST-elevation MI (STEMI) and 70% - 80% of those with STEMI complicated by cardiogenic shock (17). Such patients remain at risk for recurrent ischemic events and ventricular arrhythmia event after acute incomplete revascularization (17). A careful review of the coronary anatomy can identify such patients. Data suggest that early completion of revascularization may be beneficial, but further data are needed (1).

5. Pre-Existing Myocardial Scare

Atherosclerosis is a progressive cardiac disease and many patients experience the second coronary event. Myocardial scare presents in patient with old myocardial damage. Therefore, patients with pre-existing LVD and an acute coronary event are at increased risk for sustained ventricular tachyarrhythmia in the acute and sub-acute phase of MI. ECG and 12-lead ECG are helpful to identify old MI. Furthermore, patients with sympathetic overactivity, or the ones in cardiogenic shock state are at increased risk of sustained ventricular arrhythmia in the setting of a recurrent acute myocardial insult. Patients with genetic predisposition such as inherited cardiomyopathies or channelopathies are prone to sustained VA after ACS (8,18).

6. Management of Ventricular Arrhythmias in Patients With ACS

Treatment approaches of the ACS are published and all information about the diagnosis and treatment modalities of ACS, NSTEMI, or STEMI are provided in details (19, 20). The study focuses on the specific role of reperfusion and/or revascularization to prevent and treat VT/VF in patients with ACS.

At present, there are increasing number of aborted sudden cardiac death out of hospital. Urgent coronary angiography and revascularization should be considered in patients with persistent ST segment elevation after cardiopulmonary resuscitation (21). However, the absence of ST-segment elevation does not exclude obstructive or even thrombotic coronary culprit lesions, which may be present in 25% - 58% of patients (21, 22). In the setting of ACS and recurrent VT/VF, immediate coronary revascularization is essential to further arrhythmia prevention (19, 20).
7. General Considerations in Anti-Arrhythmic Drugs Prescription in the Setting of Acute Coronary Syndromes

Cardiac shock (cardioversion or defibrillation) is the treatment of choice in patients with VAs in the setting of ACS (10). Early intravenous administration of beta blockers can help to prevent recurrent arrhythmias (I, B) (10, 23). Treatment with intravenous amiodarone should be considered in case of recurrent monomorphic VT refractory to cardioversion or defibrillation (I, C) (10). Intravenous lidocaine may be considered for recurrent sustained VT/VF refractory for treatment with beta-blockers or amiodarone or the case of contraindications to amiodarone (IIb, C). Catheter ablation should be considered in patients with recurrent VT/VF triggered by premature ventricular complex (PVC) originating from ischemic Purkinje fibers (IIa, C) (24-28).

8. Premature Ventricular Complexes in ACS Setting

PVCs, alone or in association with non-sustained ventricular tachycardia (NSVT), are not infrequent in patients with ACS. They are more frequent after reperfusion of coronary arteries. PVCs require cardiac monitoring and antiarrhythmic drugs are not recommended. high count PVCs can be a sign of myocardial ischemia and revascularization may be recommended (10, 20).

Frequent symptomatic NSVT with effects on cardiac hemodynamic should be treated with intravenous amiodarone (10).

9. Sustained VT and VF

Incomplete revascularization and myocardial ischemia can present with refractory polymorphic VT and VF. Urgent coronary angiography should be considered (I, C) (10, 20). Frequent episodes of polymorphic VT degenerating into VF can be treated with beta blockers (I, B). In addition, deep sedation may be helpful to reduce episodes of VT/VF. Intravenous amiodarone should be considered to acutely suppress recurrent hemodynamically relevant VAs (I, C). Class I (la and lc) antiarrhythmic drugs are not recommended for medical treatment of ventricular arrhythmia after acute MI (III) (10, 23).

10. Catheter Ablation and Recurrent Sustained Ventricular Tachyarrhythmias

Radiofrequency catheter ablation is recommended in patients with recurrent VT/VF despite PCI or coronary artery bypass graft and medical management (IIa, C). repeated VF episodes may be initiated by PVCs arising from ischemic Purkinje fibers or ischemia and/or reperfusion related ventricular myocardium injury.

The arrhythmogenic substrate can be accessed from the endocardium in most patients. An epicardial approach is rarely needed. cardiac mapping and ablation of focal arrhythmia (PVC) triggering VF, or modification of substrate sustaining arrhythmia, is a sophisticated and time-consuming procedure. Thus, patients with refractory Ventricular tachyarrhythmia should be referred to a specialized VT ablation center (24-28).

In patients with recurrent VT/VF that cannot be managed with the aforementioned treatments, implantation of left ventricular assist devices or extracorporeal life support should be considered for hemodynamic stabilization (IIa, B). Such treatment modalities may provide opportunities to perform coronary intervention in hemodynamic instability due to VT/VF. recurrence of ventricular arrhythmia is high despite initial hemodynamic support by ventricular assist devices (29).

11. The Prognostic Role of Early Ventricular Fibrillation

In-hospital, mortality increases up to 5-fold after VF occurring within 48 hours during ACS (30). It may be associated with increased longer-term mortality. All of the death before arriving to the hospital are not sudden, and risk factors other than VT/VF are needed to recommend defibrillator therapy (IIb, C) (30, 31).

Based on data from the last guideline (European society of cardiology guideline 2015, regarding SCD prevention), ICD implantation or temporary use of a wearable cardioverter defibrillator (WCD) may be considered during the first 40 days after MI in selected patients. These high risk patients have the following features: (IIb, C).

- Incomplete revascularization that means failure to treat the culprit lesion or the presence of non-culprit lesions, which cannot be treated.
- Pre-existing LVD.
- Occurrence of arrhythmia > 48 hours after the onset of ACS.
- Polymorphic VT/VF.

12. Sustained Monomorphic VT and Dilemma of ICD Treatment in Early After MI Period (< 48 Hours)

In the recent guidelines implantation of ICDs for survivors of VF during the First 48 hrs of STEMI do not support. Nevertheless, there are a few studies in a real-life setting with long-term follow-up in this issue. A higher in-hospital
mortality (12% vs. 2%), with no more long-term mortality in whom discharged alive (4%) was showed in STEMI patients (32). In another investigation it was showed, in-hospital mortality rate was significantly higher in VF patients (OR 7.38, P < 0.001), however no increase in 5-years mortality in patients with acute phase VF (HR 0.78, P = 0.21) was seen. SCD mortality was the same for both groups (13%VF vs. 12.9% non-VF). ICD implant was very low in both groups (1.2%) (30); although it rarely occurs, sustained monomorphic VT may complicate patients with MI in the first 48 hours (1.9% in one study) (33). It was an independent predictor of in-hospital mortality, compared with patients with VF, they had more extensive MI with higher Killip class (Killip class > 1, 63% vs. 30%), creatine kinase type M/B (CK-MB) (P < 0.01) and arrhythmia recurrence rate (31% vs. 4%); during the follow-up period 42% died from cardiac causes and 17% had recurrence of VT (33). Another question that should be answered is the effect of mechanical revascularization on outcome improvement in the patients with sustained VT/VF in acute phase of MI. In a study by Piccini et al., in-hospital mortality in VF group was 16.3% vs. 3.7%, successful percutaneous coronary intervention (PCI) was associated with reduction in mortality from 41% to 14% (P < 0.001) (32), but they had greater in-hospital mortality, even after PCI, than patients without VT/VF (P < 0.001). Independent predictors of VT/VF included cardiogenic shock, heart failure, CKD and first six hours after presentation (32). Controversial evidence, regarding VT/VF in acute phase of MI and its effect on medium and long-term prognosis in patients with ACS makes it very difficult to decide on ICD implantation. Furthermore, there are various scenarios in patients with VT/VF in the first 48 hours of MI that portend higher risk of arrhythmia recurrence in later time.

In patients with preserved LV function that present VT/VF during 48 hours of MI, there are no conclusive data that the risk is negligible in future (2, 34); it may depend on the LV function and other unproven factors (e.g. heart rate variability, microvolt T-wave alternans, NSVT based on Holter monitoring and programmed ventricular stimulation).

At the time of patient discharge from hospital, WCD can recognize and terminate VT and VF. In a report by Chung et al., the first shock was successful in nearly 100%, in another report by Epstein et al., 91% of patients resuscitated from VA (35, 36). Usefulness of WCD to prevent SCD in the hospital stay period in the patient population was unclear (IIb, C), but it may be a solution for some patients in the future (37, 38).

In non-ST-segment elevation acute coronary syndrome (EARLY ACS) trials that compare patients with NSTEMI, with early < 48 hours and > 48 hours VT/VF, risk of event was high even after one year in < 48 hours group especially in patients with low left ventricular ejection fraction (LVEF) and recommendation for such patients was a more aggressive strategy (39). The role of programmed ventricular stimulation (PVS) in this subgroup of patients is unknown and some data support early PVS in MI survivors (within 10 days) (IIb, B) (39). Until the availability of well-designed and powerful meta-analysis and systematic review and further proof from RCTs, the current practice is to neglect VT/VF at this time window (first 48 hours) to make decision on appropriateness of ICD implantation in this subgroup of patients addressed by appropriate use criteria (AUC) in ICD and cardiac resynchronization therapy (CRT) in 2013 (40).

Table 1 and Figure 1 present the summery of aforementioned recommendations.

### Table 1. Coronary Artery Disease: Ventricular Fibrillation or Hemodynamically Unstable Ventricle Tachycardia Associated Acute (< 48 Hours) Myocardial Infarction

<table>
<thead>
<tr>
<th>Indication</th>
<th>Appropriate Use Score (1-9)</th>
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<tbody>
<tr>
<td>Total Revascularization Completed After Arrest</td>
<td>LVEF</td>
</tr>
<tr>
<td>≥ 50%</td>
<td>16% to 45%</td>
</tr>
<tr>
<td>1. Single episode VF or polymorphic VT during acute (&lt; 48 hours)/MI</td>
<td>R (2)</td>
</tr>
<tr>
<td>2. Recurrent VF or polymorphic VT during acute (&lt; 48 hours)/MI</td>
<td>R (3)</td>
</tr>
<tr>
<td>3. VT or polymorphic VT during acute (&lt; 48 hours)/MI</td>
<td>R (5)</td>
</tr>
<tr>
<td>NSVT 4 days post-MI</td>
<td></td>
</tr>
<tr>
<td>Inducible VT/VF at EPS ≥ 4 days after revascularization</td>
<td></td>
</tr>
<tr>
<td>No Revascularization Indicated (1e, No Significant CAD)</td>
<td>LVEF</td>
</tr>
<tr>
<td>≥ 50%</td>
<td>16% to 45%</td>
</tr>
<tr>
<td>4. Single episode VF or polymorphic VT during acute (&lt; 48 hours)/MI</td>
<td>R (2)</td>
</tr>
<tr>
<td>5. Recurrent VF or polymorphic VT during acute (&lt; 48 hours)/MI</td>
<td>R (2)</td>
</tr>
<tr>
<td>Obstructive With Coronary Anatomy Not Amenable Revascularization</td>
<td>LVEF</td>
</tr>
<tr>
<td>≥ 50%</td>
<td>16% to 45%</td>
</tr>
<tr>
<td>6. VT or polymorphic VT during acute (&lt; 48 hours)/MI</td>
<td>M (5)</td>
</tr>
<tr>
<td>No EPS done</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: A, appropriate; CAD, coronary artery disease; EPS, electrophysiological study; LVEF, left ventricular ejection fraction; M, may be appropriated; MI, myocardial infarction; NSVT, non-sustained ventricular tachycardia; R, rarely appropriated; VF, ventricular fibrillation; VT, ventricular tachycardia; courtesy of Andrea M Russo et al. (40).


References


30. Peichl P, Chikah R, Kozeluhova M, Wichterle D, Vancura V, Kautzner J. Catheter ablation of arrhythmic storm triggered by monomor-