Original Research Article

Sustained ventricular tachycardia (VT) in coronary artery disease (CAD): A study from tertiary care center in north India

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Abstract

Background: Ventricular Tachycardia (VT) constitutes an important manifestation of coronary artery disease (CAD). VT can occur in the immediate acute myocardial infarction (MI) period, further complicating the management. VT also occurs after long duration of acute coronary syndrome (ACS) in the healed MI.

Aim: The aim of our study was to evaluate the epidemiology, clinical presentation, hemodynamic status, treatment received and finally the outcome of CAD patients manifesting as sustained VT.

Materials and methods: This prospective study was conducted at Sher I Kashmir Institute of Medical Sciences (SKIMS), a tertiary care center in Srinagar, Jammu and Kashmir, India, between August 2013 to May 2016. All the cases of definite sustained VT already admitted in the hospital or

presenting in the emergency department including those who developed VT during the course of acute MI were evaluated.

Results: In our study, a total of 35 patients of CAD manifesting as sustained VT were observed. Majority of these patients were males. The most common presenting symptom was chest pain seen in a total of 14 patients. A total of 23 patients (66%) were hemodynamically stable at the time of VT. A decreased Left Ventricular Ejection Fraction (LVEF <50%) was seen in 18 patients (51%). Monomorphic VT was seen in a total of 28 patients (80%) and the rest of 7 patients showed polymorphic VT. Mortality was seen in 8 patients (23%).

Conclusion: Polymorphic pattern of sustained VT, hemodynamic instability at the time of VT and a decreased LVEF are associated with increased mortality in patients of CAD manifesting as VT.

Key words

Coronary artery disease (CAD), Ventricular tachycardia (VT), Coronary angiography (CAG), Left ventricular ejection fraction (LVEF), Arrhythmogenic right ventricular cardiomyopathy/ dysplasia (ARVC/D), Myocardial infarction (MI), Acute coronary syndrome (ACS).

Introduction

Significant improvements in the prevention, diagnosis, and treatment of coronary artery disease (CAD) have resulted in higher rates of survival following acute myocardial infarction (MI) [1]. As a result of rapid reperfusion, increased implementation of hemodynamic support devices, and adherence to antiplatelet and neurohormonal therapies, an increasing number of patients with healed myocardial infarcts require electrophysiological management for the treatment and prevention of ventricular arrhythmias [2]. Ventricular arrhythmias are among the most feared complications of coronary artery disease (CAD). Ventricular fibrillation (VF) accounts for the majority of deaths occurring in the acute phase of an ischaemic event [3], and can be the first manifestation of the disease in more than half of all cases. Sustained, monomorphic ventricular tachycardia (VT) occurs most frequently in the setting of healed MI, and may appear in the subacute phase or long after the acute ischemic injury [4]. Sustained, monomorphic VT usually develops in patients with more extensive MI who also have lower Left Ventricular ejection fraction (LVEF). The overall incidence of sustained VT following MI was classically established at about 3% to 5%, but has been estimated to decline to 1% in recent years due to major advances in MI management, resulting in smaller infarct scars. The VT risk in the overall population, however, has been fairly stable and could in fact be increasing, on account of an improved post-MI survival and the possibility of VT occurrence years after the initial MI, along with a progressively aging population. Arrhythmogenesis early in the course of an acute coronary syndrome (ACS) often manifests as polymorphic VT [5].

Clinical presentation of patients with CAD who have ventricular arrhythmias is variable. Patients with ventricular arrhythmias complicating acute ischemia might experience palpitations in addition to chest pain if the arrhythmia is stable and clinically tolerated, but more often present with syncope and sudden cardiac death (SCD) as a result of hemodynamically unstable VT or VF. In the case of ventricular arrhythmias related to an old MI, patients might be asymptomatic when the arrhythmia is slow and stable, but palpitations, dyspnea, or chest discomfort are common symptoms.

CAD embraces a broad spectrum of clinical scenarios where all arrhythmia mechanisms (enhanced automaticity, triggered activity, and reentry) can converge. Whereas the VT associated with MI scarring constitutes the clinical paradigm of reentry, focal activation by abnormal automaticity is the main mechanism

involved in the VT arising from the ischemic border zone during acute ischemia [6]. Focal discharge by calcium overload and triggered activity in the form of delayed or early after-depolarizations is also likely a mechanism of VT initiation during ischemia, but this has not been proven experimentally thus far [7].

Even in highly developed healthcare systems, only 70-80% of patients who present with ST-elevation myocardial infarction (STEMI) receive reperfusion therapy [8]. Patients at high risk in the setting of an ACS, easily assessed by clinical risk scores such as GRACE or TIMI [9-13], are likely to suffer from larger myocardial damage, especially when the ECG shows prominent ischemic changes, the patient presents with persistent symptoms after initiation of therapy, or creatine kinase (CK) /troponin release patterns suggest extensive myocardial damage. These patients are at increased risk of sustained VT [14].

Prompt and adequate revascularization therapy, usually by interventional reopening of occluded vessels and stabilization of the culprit lesion with a stent, combined with initiation of adequate secondary prevention therapies (statin, dual antiplatelet therapy, beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers) aimed at preventing subsequent acute coronary events [15], have markedly reduced these life-threatening events in modern coronary care unit.

Materials and methods

Aim

To evaluate the epidemiology, clinical presentation, hemodynamic status, treatment received and finally the outcome of CAD patients manifesting as sustained VT.

Study design

This prospective study was conducted at Sher I Kashmir Institute of Medical Sciences (SKIMS), a tertiary care center in Srinagar, Jammu and Kashmir, India between August 2013 to May 2016.

Study population

Inclusion criteria: All the cases of definite sustained VT (lasting more than 30 seconds or terminated by intervention) already admitted in the hospital or presenting in the emergency department including those who developed VT during the course of acute MI were evaluated . Sustained (VT) was diagnosed using the "Brugada algorithm" [16] and/or The "Avr Vereckei algorithm" [17].

A total of 88 patients of sustained VT were evaluated and out of these, 35 patients were diagnosed having CAD as a cause of sustained VT.

Exclusion criteria: The following patients were excluded from the study

- Patients in whom the etiology of sustained VT was other than the coronary artery disease (CAD). Other etiologies of sustained VT were in the form of Arrhythmogenic right ventricular cardiomyopathy /dysplasia (ARVC/D), Non ischemic dilated cardiomyopathy (DCMP) and other type of cardiomyopathies among other causes of sustained VT.
- Patients with non-sustained VT.
- Patients without known etiology who died early after hospitalization thereby precluding further evaluation.
- Patients who refused for further evaluation

Consent: An informed consent was obtained from all the subjects.

Ethical clearance: The study was cleared by the institutional Ethics Committee.

Evaluation

Eighty eight patients of sustained VT were enrolled in total. This included patients presenting to emergency with sustained VT and patients already admitted in hospital who developed sustained VT during the course of hospital stay. All the patients were attached to non-invasive monitor for continuous ECG, blood

pressure, and pulse oximetry monitoring. We defined two groups of patients based on their hemodynamic status on presentation. Hemodynamically stable and Hemodynamically unstable patients. Hemodynamic instability was defined as need for immediate cardioversion due to loss of consciousness, hypotension, shock or occurrence of congestive heart failure. Otherwise the patients were defined as hemodynamically stable. The etiological evaluation was done in all the patients. ECG characteristics during VT and in resting stage were observed. 2-D echocardiography was done in all the patients. Exercise test and 24 hours Holter monitoring was done in selective cases. Coronary angiography (CAG) was done in patients with definitive or suspected MI and those patients with intermediate to high risk of CAD derived from Framingham risk calculator (based on Wilson , D, Agostiono, Levy, et al. "Prediction of coronary heart disease using risk factor categories) [18]. Also the patients with baseline ECG suggestive of previous MI were subjected to coronary angiography CAG. Coronary Artery Disease (CAD) as a cause of sustained VT was defined as following

- Patients with acute myocardial infarction as evidenced by current ECG and or CAG.
- Patients with age indeterminate myocardial infarction (presence of significant Q waves on baseline ECG, Regional Wall Motion Abnormalities (RWMA) on echocardiography and or myocardial transmural scar on late gadolinium enhancement cardiac Magnetic Resonance Imaging (MRI).

Cardiac MRI was done in cases where specific cardiomyopathy like that of ARVC/D or myocardial scarring post myocardial infarction were suspected.

After thoroughly evaluating all these 88 patients of sustained VT, CAD as a cause of VT was seen in 35 patients. We tried to evaluate the epidemiology, clinical presentation, hemodynamic status, treatment received and finally the outcome of these patients to broaden our existing knowledge about VT occurring as a manifestation of CAD.

Results

In our study, a total of 35 patients of CAD manifesting as sustained VT were observed. Majority of these patients were males (22 patients, 63%) and the remaining 13 patients (37%) were females with a male: female ratio of 1.7: 1. The minimum and maximum age at presentation was 26 years and 82 years respectively with a mean age at presentation of 50 years. The most common presenting symptom was chest pain seen in a total of 14 patients (40%). We divided the patients in to hemodynamically stable and unstable patients. A total of 23 patients (66%) were hemodynamically stable at the time of VT and 12 patients (34%) were in a state of hemodynamic instability. Monomorphic VT was seen in a total of 28 patients (80%) and the rest of 7 patients showed polymorphic VT. Right Bundle Branch Block (RBBB) was the most common morphology seen in 18 patients (51%) followed by Left Bundle Block Morphology (LBBB) which was seen in 10 patients (29 %). Remaining 7 patients (20%) were having polymorphic VT.

Echocardiography was done in all the patients. A decreased Left Ventricular Ejection Fraction (LVEF <50%) was seen in 18 patients (51%) whereas a normal LVEF (LVEF >50%) was present in the remaining 17 patients (49%). Coronary Angiography (CAG) was done in 28 patients (80%). Most common lesion seen was double vessel disease DVD) in 13 (46%) of these 25 patients, followed by single vessel disease (SVD) in 10 patients and triple vessel disease (TVD) in 5 patients. Cardiac MRI was done in five patients which showed myocardial transmural scar on late gadolinium enhancement in three of these patients.

Out of 35 patients in total, 13 patients (37%) presented to emergency as acute myocardial

infarction (MI). Out of these 13 patients presenting as acute MI, 8 patients had ST elevation MI (STEMI) and the rest 5 patients had Non ST elevation MI (NSTEMI). These 13 patients developed sustained VT during the course of their hospital stay in the immediate post MI period. Out of these 13 patients, 5 patients developed polymorphic VT and 8 Patients developed monomorphic VT. The remaining 22 patients (63%) presented with sustained VT rather than acute MI. These patients on evaluation were found to have CAD as the cause of sustained VT. Out of these 22 patients, 2 patients developed polymorphic VT and the rest 20 patients developed monomorphic VT.

DC cardioversion was given to all the 12 patients with hemodynamic instability. DC cardioversion restored sinus rhythm in all these patients. All the 23 patients who were hemodynamically stable at the time of VT received intravenous drugs initially, which reverted VT in 18 patients (78%). The remaining 5 patients were given DC cardioversion in addition, to revert VT.

Mortality was seen in 8 patients (23%). Among 23 hemodynamically stable patients, mortality was seen in 3 patients (13%) as compared to 5 patients (42%) dying among 12 hemodynamically unstable patients. Among 18 patients with decreased LVEF, 6 patients (33%) died whereas 2 patients (12%) died among 17 patients with normal LVEF. Among 28 patients with monomorphic VT, 7 patients (25%) were hemodynamically unstable whereas among 7 patients with polymorphic VT, 5 patients (71%) were hemodynamically unstable. Moreover among 28 patients of monomorphic VT, 3 patients (11%) died as compared to 5 patients (71%) dying among 7 patients with polymorphic VT. Both these results were hemodynamically significant ($p < 0.05$). Thus we concluded that polymorphic pattern of VT is associated with higher incidence of hemodynamic instability and overall mortality (**Table – 1 to 5**).

Table – 1: Distribution of various symptoms in CAD patients with sustained VT.

Symptoms	Frequency	%
Chest pain	14	40.0
Palpitations	8	22.9
Chest pain + Palpitations	10	28.6
Syncope	3	8.6
Total	35	100.0

Table – 2: Hemodynamic status with respect to morphology of VT.

VT morphology	Hemodynamic status		Total
	Stable	Unstable	
Monomorphic	21	7	28
Polymorphic	2	5	7
Total	23	12	35

Table – 3: Final outcome with respect to morphology of VT.

VT morphology	Outcome		Total
	Discharged	Died	
Monomorphic	25	3	28
Polymorphic	2	5	7
Total	27	8	35

Table – 4: Hemodynamic status with respect to LVEF.

LVEF	Hemodynamic status		Total
	Stable	Unstable	
< 50%	8	10	18
> 50%	15	2	17
Total	23	12	35

Table - 5: Final outcome with respect to LVEF.

LVEF	Outcome		Total
	Discharged	Died	
< 50%	12	6	18
> 50%	15	2	17
Total	27	8	35

Discussion

Ventricular arrhythmias are among the most feared complications of the CAD. Sustained VT is one of the most common type of arrhythmias occurring in the CAD patients. In our study, we observed the profile of CAD patients who developed sustained VT. The main observations of our study were as follows:

- Polymorphic VT occurs most commonly during the acute phase of myocardial infarction (MI) in contrast to monomorphic VT which develops more commonly during the subacute phase or long after acute ischemic injury.
- Polymorphic pattern of VT, hemodynamic instability and low LVEF are associated with higher mortality.

In our study, a total of 35 patients of CAD manifesting as sustained VT were observed. Majority of these patients were males with a male: female ratio of 1.7: 1. The mean age at presentation was 50 years. Thus majority of our patients were males in their sixth decade of life. Lemery, et al. reported that patients with idiopathic VT are younger and often hemodynamically stable than the ischemic VT patients [19]. Most common symptom was the chest pain seen in 14 patients (40%). Majority of the patients were hemodynamically stable (23 patients, 66%). The fact that 66 percent of sustained VT patients were hemodynamically stable shows that it is misleading to believe that VT always causes severe hemodynamic disturbance [20].

Monomorphic VT was seen in a total of 28 patients (80%). RBBB was the most common morphology seen in 18 patients (51%) followed by LBBB seen in 10 patients (29%). Out of 35 patients in total, 13 patients presented as acute MI. Among these 13 patients, 5 patients (38%) developed polymorphic VT whereas among other 22 patients, only 2 patients (9%) developed polymorphic VT. Thus it was seen in our study that polymorphic VT occurs more commonly in the immediate post MI period in contrast to monomorphic VT which occurs more commonly in the setting of healed MI and can manifest long after acute ischemic injury. This observation is consistent with the existing literature on the same topic.

A decreased Left Ventricular Ejection Fraction (LVEF < 50%) was seen in 18 patients (51%) whereas a normal LVEF (LVEF >50%) was present in the remaining 17 patients (49%). CAG was done in 28 patients (80%). Most common lesion seen was double vessel disease (DVD) in 13 patients (46%).

DC cardioversion was given to all the 12 patients with hemodynamic instability. DC cardioversion restored sinus rhythm in all these patients. All the 23 patients who were hemodynamically stable at the time of VT received intravenous drugs initially, which reverted VT in 18 patients (78%). The remaining 5 patients were given DC cardioversion in addition to revert VT.

Mortality was seen in 8 patients (23%). Fifty six percent of patients with decreased LVEF were hemodynamically unstable whereas 12% of patients with normal LVEF were hemodynamiocally unstable. Mortality was also more common in hemodynamically unstable patients This is consistent with several studies in the past which showed that overall mortality is more in patients of sustained VT with low LVEF [21, 22]. In our study hemodynamic instability and mortality were more common among the patients with polymorphic VT. Both these results were hemodynamically significant ($p < 0.05$). Thus we concluded that polymorphic pattern of VT is associated with higher incidence of hemodynamic instability and overall mortality.

Conclusion

Polymorphic pattern of sustained VT, hemodynamic instability at the time of VT and a decreased LVEF are associated with increased mortality in patients of CAD manifesting as VT.

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