ORIGINAL ARTICLE

Ventricular Tachycardia Storm: A Case Series and Literature Review

P L Chia, MRCP(UK), S Y Loh, MRCP(UK), D Foo, MRCP(UK)

Dept of Cardiology, Tan Tock Seng Hospital, 11 Jalan Tan Tock Seng, Singapore 308433

SUMMARY

Introduction: Ventricular tachycardia (VT) storm is an uncommon but life-threatening condition. We describe the incidence, causes and management of VT storm among patients admitted to the coronary care unit of a large tertiary hospital.

Materials and Methods: Between 1 November 2009 and 30 April 2010, 198 patients were admitted to the coronary care unit and 7 (3.5%) presented with VT storm. A retrospective review of their records was conducted. The mean follow-up period was 268 (196 to 345) days.

Results: The mean age was 67 years and 4 patients were male. One patient had a previous myocardial infarction. All had abnormal left ventricular ejection fraction, median of 30%. Acute myocardial infarction (4 patients) was the most common trigger, followed by decompensated heart failure (1), systemic inflammatory response syndrome on a background of non-ischemic dilated cardiomyopathy (1) and bradycardia-induced polymorphic VT (1). Three patients had polymorphic VT and the rest had monomorphic VT. Intravenous amiodarone, lignocaine, overdrive pacing and intra-aortic balloon pump counterpulsation were useful in arrhythmia control. Three patients underwent coronary revascularization, 3 patients received implantable cardioverter-defibrillators, 1 had a permanent cardiac pacemaker, 1 died during the acute episode. Five out of the 6 survivors were prescribed oral beta-blockers upon discharge. On follow-up, none of the patients had a recurrence of the tachyarrhythmia.

Conclusion: Acute myocardial infarction was the main trigger of VT storm in our patients. Intravenous amiodarone, lignocaine, overdrive pacing and intra-aortic balloon pump counterpulsation were useful at suppressing VT storm.

KEY WORDS:
Electric storm; Ventricular tachycardia; Arrhythmia

INTRODUCTION

Ventricular tachycardia (VT) storm refers to recurrent ventricular tachyarrhythmias requiring electrical cardioversion 3 or more times within 24 hours [1]. Patients repeatedly develop VT and receive serial electric shocks and antiarrhythmic agents in an attempt to cardiovert the arrhythmia. Despite best efforts, the mortality rate is high, especially in patients who had a recent myocardial infarction (MI) or ongoing myocardial ischemia [2,3].

We present our experience with the management of VT storm among patients who were admitted to the coronary care unit of a large tertiary hospital. We aim to report the incidence, causes and management of this uncommon but life-threatening condition.

MATERIALS AND METHODS

This was a single centre patient registry. Between 1 November 2009 and 30 April 2010, 198 patients were admitted to the coronary care unit of which 7 presented with VT storm. A retrospective review of both inpatient and outpatient case records was performed. Follow-up data was complete in all patients till 30 November 2010, with a mean follow-up period of 268 (196-345) days.

RESULTS

The mean age at presentation was 67 (48-80) years. There were 4 male and 3 female patients. One patient had diabetes mellitus, 6 had hypertension, 4 had hyperlipidemia and 1 was an active smoker. One patient had a previous MI. None had implantable cardioverter-defibrillator (ICD) or previous episode of arrhythmia.

The precipitating causes of VT storm were identified as follows: 4 cases of acute MI (2 ST-segment elevation and 2 non ST-segment elevation MI); 1 had decompensated heart failure secondary to dilated cardiomyopathy; 1 had systemic inflammatory response syndrome from community acquired pneumonia with a background history of dilated cardiomyopathy; the last presented with bradycardia-induced polymorphic VT. Three patients presented with polymorphic VT and the rest had monomorphic VT. None of the patients had significant serum electrolyte or acid/base imbalances. All 7 patients had left ventricular systolic dysfunction, with a median left ventricular ejection fraction of 30% (20% to 45%) on transthoracic echocardiography just prior to discharge.

Table I summarized the management strategy for all 7 patients. They received a mean of 7 (3 to 16) electric cardioversions. The patient with bradycardia-induced polymorphic VT received intravenous magnesium sulphate followed by cardiac pacing via a temporary transvenous pacing wire. The remaining 6 patients received intravenous

This article was accepted: 25 September 2012
Corresponding Author: P L Chia, Dept of Cardiology, Tan Tock Seng Hospital, 11 Jalan Tan Tock Seng, Singapore 308433
Email: powlichia@gmail.com

Table I: Management strategy for our series of 7 patients with ventricular tachycardia storm

No	Age	Gender	Prior MI	Cause of ES	Type of VT	No. of shocks	1st line drug	2nd line drug	Cardiac pacing	IABP support	Coronary revascularization	Cardiac devices	Outcome
1	72	Male	No	STEMI	Polymorphic	7	Amiodarone	Lignocaine	-	Yes	PCI	ICD	Survived
2	48	Male	No	STEMI	Monomorphic	16	Amiodarone	Lignocaine	Overdrive pacing	Yes	CABG	-	Survived
3	70	Male	Yes	NSTEMI	Monomorphic	8	Amiodarone	Lignocaine	-	-	PCI	ICD	Survived
4	76	Female	No	NSTEMI	Monomorphic	4	Amiodarone	-	-	-	-	-	Died
5	48	Male	No	Heart failure	Monomorphic	3	Amiodarone	Lignocaine	-	-	-	ICD	Survived
6	77	Female	No	SIRS	Polymorphic	4	Amiodarone		Overdrive pacing	-	-	-	Survived
7	80	Female	No	Bradycardia induced VT	Polymorphic	10	Magnesium sulphate	-	Temporary pacing wire	-	-	PPM	Survived

STEMI : ST-segment elevation myocardial infarction
NSTEMI : Non-ST segment elevation myocardial infarction
PPM : Permanent pacemaker
SIRS : Systemic inflammatory response syndrome
VT : Ventricular tachycardia
ICD : Implantable cardioverter-defibrillator
IABP : Intra-aortic balloon pump counterpulsation
PCI : Percutaneous coronary intervention
CABG : Coronary artery bypass grafting

amiodarone as first line drug therapy and intravenous lignocaine was added on as second line therapy in 4 of these patients. Two patients required overdrive cardiac pacing via a temporary transvenous pacing wires despite intravenous amiodarone and lignocaine. Intra-aortic balloon pump counterpulsation was initiated for hemodynamic support in the 2 patients with ST-segment elevation MI during emergency percutaneous coronary intervention.

Following successful arrhythmia suppression, 2 patients eventually underwent percutaneous coronary intervention, 1 had coronary artery bypass graft surgery, 3 had ICDs implanted. The patient with bradycardia-induced polymorphic VT received a dual chamber permanent cardiac pacemaker. One patient with non-ST elevation myocardial infarction died following degeneration of VT to asystole. With the exception of the patient with permanent cardiac pacemaker implant, the other 5 survivors were discharged with beta-blockers. The mean hospital stay for survivors was 5±5 days. On follow-up, none had arrhythmia recurrence.

DISCUSSION

The incidence of VT storm varies depending on the study populations. The condition occurs in 10% to 20% of ICD recipients [4]. The incidence is lower when ICDs are implanted for primary versus secondary prevention [5]. In our series, VT storm occurred in 7 out of 198 (3.5%) patients admitted to the coronary care unit. The lower incidence was expected as we evaluated a general population of patients in the coronary care unit and not just ICD patients per se. In-hospital mortality in our series was 14.3%.

Patients who present with VT storm often have a vulnerable anatomic substrate such as underlying structural heart disease and scarring post-MI [6]. Other risk factors for VT storm include advanced age, male gender, a low left ventricular ejection fraction, New York Heart Association functional class III or IV heart failure and chronic kidney disease [7]. In our study population, all patients had left ventricular systolic dysfunction and 1 had a previous MI.

The most common trigger of VT storm among our patients was acute MI. Studies have demonstrated that MI and myocardial ischemia influence the denervation of sympathetic-parasympathetic fibers, resulting in elevated sympathetic activity. This in turn contributes to a decreased threshold for ventricular tachyarrhythmias during coronary artery occlusion [8,9]. Other reported precipitants of VT storm include worsening heart failure, hypokalemia, hypomagnesemia, anti-arrhythmic drug therapy, hyperthyroidism, and infection or fever [10].

VT storm occurs when an arrhythmic substrate is affected by a precipitating event. It is therefore important to determine the causative factors and reverse them. Active ischemia, decompensated heart failure, and electrolyte imbalances should be remedied assiduously. In our series, intra-aortic balloon pump counterpulsation and coronary revascularization likely suppressed the arrhythmias through improvement in coronary perfusion. Bradycardia induced polymorphic VT was promptly controlled with pacing via a temporary transvenous pacing wire.

As enhanced sympathetic activity contributes to VT storm, sympathetic blockade with beta-blockers have resulted in better mortality outcomes compared to treatment with Class I anti-arrhythmic agents such as lignocaine or procainamide [11]. This is consistent with evidence that the early administration of beta-blockers in post-MI patients reduces early mortality, predominantly due to the prevention of ventricular tachyarrhythmias [12,13]. Congestive heart failure trials also report that beta-blockers significantly reduce sudden death that is presumed to be due to arrhythmias [14,15]. In our patients, beta-blockers were not administered in the acute stage due to the possibility of asystole and hypotension. However, they proved effective at prevention of tachyarrhythmia recurrence.

Among the anti-arrhythmic medications, amiodarone is generally considered the first choice. Current literature supports the superiority of amiodarone over lignocaine in the control of ventricular tachyarrhythmias [16-18]. Combination of amiodarone with beta-blockers has also been shown to reduce mortality. In the Canadian Amiodarone Myocardial Infarction Arrhythmia Trial (CAMIAT) and the European Myocardial Infarct Amiodarone Trial (EMIAT) [19,20], patients on amiodarone who were also on beta-blockers had a significant reduction in primary outcome events compared to patients not on beta-blockers [21,22]. In our study, 4 patients required lignocaine in addition to initial amiodarone for arrhythmia suppression. Such drug combinations may be beneficial in the early phases of pharmacological arrhythmia suppression, until myocardial concentration of amiodarone has sufficient time to build up [23].

Other therapies that have been reported to be useful in the control of VT storm include sedation with propofol with its sympatholytic activity [24], overdrive pacing [25,26] and emergent catheter ablation [27,28]. Full hemodynamic support using intra-aortic balloon pump counterpulsation or ventricular assist device [10,29] is useful. These devices increase coronary perfusion pressure and can dramatically relieve the ischemic substrate.

Following arrhythmia suppression and coronary revascularization if indicated, ICD implantation may be considered. Under current guidelines [30], ICD therapy is indicated in survivors of cardiac arrest following correction of reversible inciting factors. In our series, 2 of the survivors declined ICD implantation and 1 had a permanent pacemaker implant as her VT storm was induced by bradycardia. The rest received an ICD for secondary prevention of sudden cardiac death.

CONCLUSION

Myocardial ischemia is the main trigger for VT storm. An individualized therapeutic approach is recommended with the aim to reverse any precipitating event. In the acute settings, the use of intravenous amiodarone and lignocaine are useful at terminating the ventricular tachyarrhythmias. Beta-blockers are effective at prevention of tachyarrhythmia recurrence.

REFERENCES

1. Exner DV, Pinski SL, Wyse DG, *et al;* AVID Investigators. Antiarrhythmics Versus Implantable Defibrillators. Electrical storm presages nonsudden death. The antiarrhythmics versus implantable defibrillators (AVID) trial. Circulation 2001; 103: 2066-71.
2. Wolfe CL, Nibley C, Bhandari A, Chatterjee K, Scheinmen M. Polymorphous ventricular tachycardia associated with acute myocardial infarction. Circulation 1991; 84: 1543-51.
3. Dorian P, Cass D. An overview of the management of electrical storm. Can J Cardiol. 1997; 13: 13A-17A.
4. Emkanjoo Z, Alihasani N, Alizadeh A, *et al.* Electrical storm in patients with implantable cardioverter-defibrillators: can it be forecast? Tex Heart Inst J 2009; 36(6): 563-7
5. Sesselberg HW, Moss AJ, McNitt S, *et al.* Ventricular arrhythmia storms in postinfarction patients with implantable def ibrillators for primary prevention indications: a MADIT-II substudy. Heart Rhythm 2007; 4(11): 1395-402.
6. Villacastin J, Almendral J, Arenal A, *et al.* Incidence and clinical significance of multiple consecutive, appropriate, high-energy discharges in patients with implanted cardioverter-defibrillators. Circulation 1996; 93(4): 753-62.
7. Gatzoulis KA, Andrikopoulos GK, Apostolopoulos T, *et al.* Electrical storm is an independent predictor of adverse long-term outcome in the era of implantable defibrillator therapy. Europace 2005; 7(2): 184-92.
8. Zipes DP. Influence of myocardial ischemia and infarction on autonomic innervation of heart. Circulation 1990 ;82: 1095-105.
9. Lombardi F, Verrier RL, Lown B. Relationship between sympathetic neural activity, coronary dynamics and vulnerability to ventricular fibrillation induced by myocardial ischemia and reperfusion. Am Heart J 1983; 105: 958-65.
10. European Heart Rhythm Association; Heart Rhythm Society, Zipes DP, Camm AJ, Borggrefe M, *et al.* ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death). J Am Coll Cardiol 2006; 48(5): e247-346
11. Nademanee K, Taylor R, Bailey WE, Rieders DE, Kosar EM. Treating electrical storm: sympathetic blockade versus advanced cardiac life support-guided therapy. Circulation 2000; 102: 742-7.
12. B-Blocker Heart Attack Trial Research Group. A randomized trial of propranolol in patients with acute myocardial infarction. Mortality results. JAMA 1982; 247: 1707-14
13. Hjalmarson A, Elmfeldt D, Herlitz J, *et al.* Effect on mortality of metoprolol in acute myocardial infarction. A double blind randomized trial. Lancet 1981; 17: 823-7.
14. Ryden L, Ariniego R, Amman K, *et al.* A double blind trial of metoprolol in acute myocardial infarction. Effects on ventricular tachyarrhythmias. N Engl J Med 1983; 308: 614-8
15. Teerlink JR, Massie BM. Beta-adrenergic blocker mortality trials in congestive heart failure. Am J Cardiol 1999; 84(9A): 94R-102R.
16. Credner SC, Klingenheben T, Mauss O, Sticherling C, Hohnloser SH. Electrical storm in patients with transvenous implantable cardioverter-defibrillators: incidence, management and prognostic implications. J Am Coll Cardiol. 1998; 32: 1909-15.
17. Kudenchuk PJ, Cobb LA, Copass MK, *et al.* Amiodarone for resuscitation after out-of-hospital cardiac arrest due to ventricular fibrillation. N Engl J Med. 1999; 341: 871-8.
18. Dorian P, Cass D, Schwartz B, Cooper R, Gelaznikas R, Barr A. Amiodarone as compared with lidocaine for shock resistant ventricular fibrillation. N Engl J Med 2002; 346: 884-90.
19. Boutitie F, Boissel J, Connolly SJ, *et al.* Amiodarone interaction with beta-blockers: Analysis of the merged EMIAT and CAMIAT databases. Circulation 1999; 99: 2268-75.
20. Ogunyankin KO, Singh BN. Mortality reduction by anti-adrenergic modulation of arrhythmogenic substrate: Significance of combining beta-blockers and amiodarone. Am J Cardiol 1999; 84: 76R-82R.
21. Cairns JA, Connolly SJ, Gent M, Roberts R. Randomized trial of outcome after myocardial infarction in patients with frequent repetitive ventricular premature depolarizations. CAMIAT. Lancet 1997; 349: 672-82.
22. Julian DG, Camm AJ, Frangin G, *et al.* Randomized trial of effect of amiodarone on mortality in patients with left ventricular dysfunction after myocardial infarction: EMIAT. Lancet 1997; 349: 667-74
23. Kowey PR, Bharucha DB, Rials SJ, Marinchak RA. Intravenous antiarrhythmic therapy for high-risk patients. Eur Heart J 1999; 1: C36-40.
24. Burjorjee JE, Milne B. Propofol for electrical storm; a case report of cardioversion and suppression of ventricular tachycardia by propofol. Can J Anaesth 2002; 49: 973-7.
25. Tanabe Y, Chinushi M, Washizuka T, *et al.* Suppression of electrical storm by biventricular pacing in a patient with idiopathic dilated cardiomyopathy and ventricular tachycardia. Pacing Clin Electrophysiol 2003; 26: 101-2.
26. Kurisu S, Inoue I, Kawagoe T, *et al.* Temporary overdriving pacing as an adjunct to antiarrhythmic drug therapy for electrical storm in acute myocardial infarction. Circ J. 2005; 69: 613-6.
27. Bansch D, Oyang F, Antz M, *et al.* Successful catheter ablation of electrical storm after myocardial infarction. Circulation 2003; 108: 3011-6.
28. Schreieck J, Zrenner B, Deisenhofer I, Schmitt C. Rescue ablation of electrical storm in patients with ischemic cardiomyopathy: a potential-guided ablation approach by modifying substrate of intractable, unmappable ventricular tachycardias. Heart Rhythm 2005; 2: 10-4.
29. Windecker S. Percutaneous left ventricular assist devices for treatment of patients with cardiogenic shock. Curr Opin Crit Care 2007; 13: 521-7.
30. Epstein AE, Dimarco JP, Ellenbogen KA, *et al.* ACC/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities. Heart Rhythm 2008; 5: e1-62.