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. 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.

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 1 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
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    | -             | Overdrive pacing | Yes           | PCI                      | ICD           | Survived |
| 2  | 48  | Male   | No       | STEMI       | Polymorphic| 16           | Amiodarone    | Lignocaine    | -             | Overdrive pacing | Yes           | PCI                      | ICD           | Survived |
| 3  | 70  | Male   | Yes      | NSTEMI      | Monomorphic| 8            | Amiodarone    | Lignocaine    | -             | Overdrive pacing | -             | PCI                      | ICD           | Survived |
| 4  | 76  | Female | No       | NSTEMI      | Monomorphic| 4            | Amiodarone    | Lignocaine    | -             | Overdrive pacing | -             | PCI                      | ICD           | Survived |
| 5  | 48  | Male   | No       | Heart failure | Monomorphic| 3            | Amiodarone    | -             | -             | -             | -             | PCI                      | ICD           | Died     |
| 6  | 77  | Female | No       | SIRS        | Monomorphic| 4            | Amiodarone    | -             | -             | -             | -             | PCI                      | ICD           | Survived |
| 7  | 80  | Female | No       | Bradycardia | Polymorphic| 10           | Magnesium sulphate | -             | -             | Overdrive pacing | 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
IABP: Intra-aortic balloon pump counterpulsation
PCI: Percutaneous coronary intervention
CA: 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. The incidence is lower when ICDs are implanted for primary versus secondary prevention. 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. 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. 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. Other reported precipitants of VT storm include worsening heart failure, hypokalemia, hypomagnesemia, anti-arrhythmic drug therapy, hyperthyroidism, and infection or fever.

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, decompenated 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. 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. Congestive heart failure trials also report that beta-blockers significantly reduce sudden death that is presumed to be due to arrhythmias. 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. 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), patients on amiodarone who were also on beta-blockers had a significant reduction in primary outcome events compared to patients not on beta-blockers. 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.
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 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, 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