Emergency Bulletin
Hong Kong College of Emergency Medicine

## Breaking the ventricular tachycardia storm in the ED

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A 40-year-old man presented to our emergency department for chest pain and dizziness. During the ambulance ride, he was noted to have wide complex tachycardia with a heart rate of 250bpm on the cardiac monitor, with BP of 70/40mmHg.

Upon arrival, the cardiac monitor showed monomorphic ventricular tachycardia (VT) with a heart rate of 250bpm. He had a palpable pulse and BP 84/58mmHg. He was afebrile. The oxygen saturation was 99% on room air. The Glasgow Coma Scale was 15/15.

Upon examination, he was alert without respiratory distress. The chest and abdominal exams were unremarkable. There was no lower limb edema. His point-of-care blood tests revealed a glucose level of 7.7 mol/L and a hemoglobin level of 14.7 g/dL. ECG was performed, revealing a monomorphic VT at 255bpm, as shown in Fig.1.

A brief history was taken, which was corroborated by the patient's wife. This was the patient's first episode of chest pain with dizziness. He had no recent febrile illness, no history of arrhythmias or syncope, and denied any drug overdose. He had good past health.

As the patient was hemodynamically unstable, cardioversion was required. He was given IV morphine 1mg for analgesia and IV midazolam 2mg for sedation.

Biphasic synchronized cardioversion with 120J using the Zoll defibrillator was then performed. Subsequently, the patient regained a sinus rhythm of 90bpm with BP 99/59mmHg. His oxygen saturation was noted to be 92% on room air, and he was given 2L/min of oxygen via nasal cannula, which improved his oxygen saturation to 99%. Chest X-ray was performed and was unremarkable. Cardiac Care Unit (CCU) was not available in our hospital, so the CCU doctor of another hospital was consulted, and they agreed to admit the patient to the unit.

However, monomorphic VT soon recurred, with a rate of 244bpm and BP 75/50mmHg. The patient then received synchronized biphasic cardioversions of 120J, 150J, and then 200J, with an eventual conversion to a sinus rhythm of 105bpm and BP 105/66mmHg.

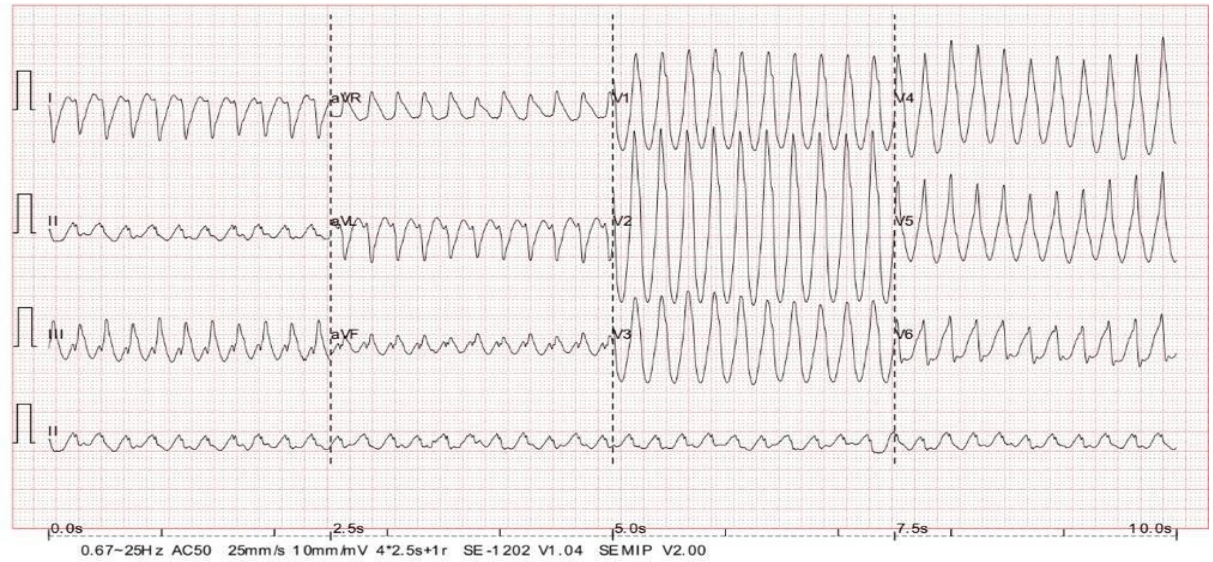


Fig.1 ECG of the patient upon arrival to the ED

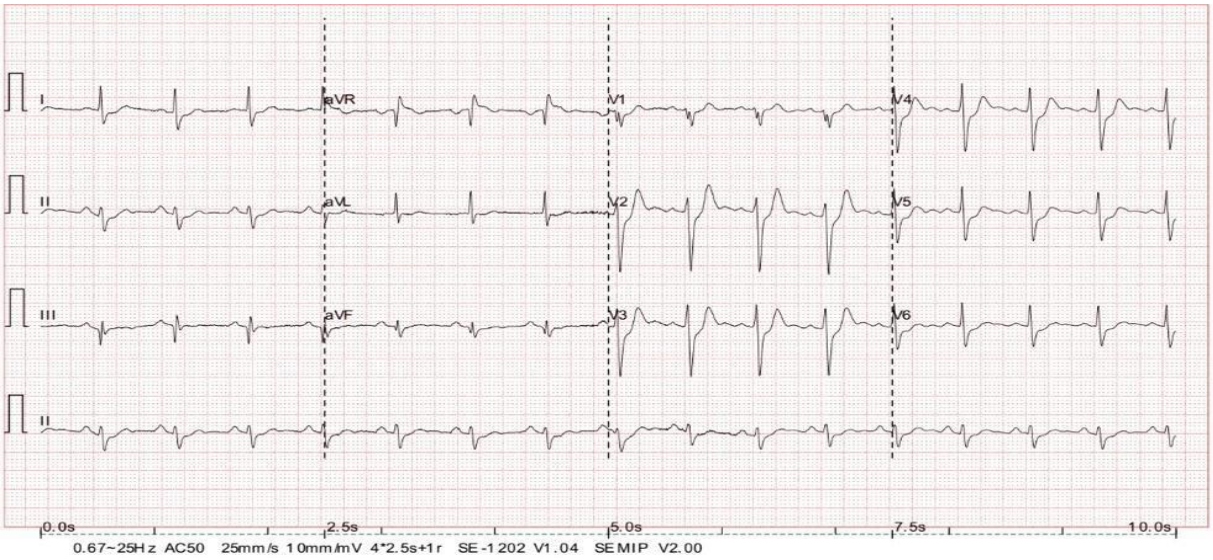


Fig.2. ECG of the patient after cardioversion

ECG was repeated, revealing a sinus rhythm of 95bpm with ST elevation in lead aVR and diffuse ST depression over leads V2-6, (shown in Fig.2). As there was concern for underlying ischemia, the patient was then started on lignocaine infusion with 1.5mg/kg IV bolus as a loading dose, then 4 mg/min as a maintenance dose.

However, before transport, the patient developed a recurrence of VT. Amiodarone infusion was started, with 150mg IV bolus as a loading dose, then 1 mg/min as a maintenance dose.

The CCU doctor was informed, and the patient was then transported to CCU with a doctor and nurse escort via ambulance. During the 22-minute transport, the patient continued to be in monomorphic VT of around 230bpm, but his BP was maintained above 90/60mmHg. The patient maintained full consciousness throughout with a

| Category | Etiology |
|---|---|
| Acute ischemia | Acute coronary syndrome, reperfusion arrhythmia (e.g after PCI / thrombolysis) |
| Structural heart disease | Ischemic cardiomyopathy (myocardial scar from old infarct)<br>Non-ischemic cardiomyopathy (dilated, hypertrophic, arrhythmogenic RV)<br>Myocarditis (giant cell, viral, lymphocytic)<br>Infiltrative diseases (SLE, RA, hemochromatosis, sarcoidosis, amyloidosis, Chagas disease)<br>Congenital heart disease (repaired tetralogy of Fallot) |
| Channelopathies | Long/short QT syndrome<br>Brugada syndrome<br>Catecholaminergic polymorphic VT (CPVT)<br>Malignant early repolarization syndrome |
| Electrolyte imbalance | Hypokalemia<br>Hypomagnesemia<br>Hypocalcemia |
| Metabolic | Acidosis (diabetic ketoacidosis, renal failure)<br>Thyrotoxicosis<br>Pheochromocytoma |
| Drug Toxicity | QT prolonging agents (anti-arrhythmics, antipsychotics, antibiotics)<br>Sympathomimetics (cocaine, methamphetamine)<br>Digoxin |
| Iatrogenic | Pacemaker-mediated<br>ICD malfunction |
| Others | Idiopathic<br>Neurological injury (subarachnoid hemorrhage) |

Table 1: Differential diagnoses of ventricular tachycardia (PCI: Percutaneous Coronary intervention; RV: right ventricle; SLE: Systemic Lupus Erythematous; RA: Rheumatoid arthritis; ICD: Implantable Cardioverter Defibrillator)

Glasgow Coma Scale of 15/15. He was continued on lignocaine and amiodarone maintenance infusion. The patient was then admitted to the CCU for further management.

**What are the differential diagnoses of ventricular tachycardia?**

Determining the underlying etiology is important in guiding the management of VT. Table 1 outlines the common differential diagnoses we need to consider. Notably, sustained monomorphic VT most commonly results from a myocardial scar from an old infarct (no acute infarct), whereas if the ischemia is acute, ventricular fibrillation is more likely to result instead.[1]

**Clinical progress of the patient**

The patient continued to have multiple episodes of VT in the CCU, with a brief episode of pulseless VT. Multiple cardioversion attempts were done, and lignocaine and amiodarone infusion were continued, but the ventricular tachycardia persisted. Eventually, the intensive care unit (ICU) doctor was consulted after the 7th episode of VT. The patient received rapid sequence induction, was intubated, and put into deep sedation with midazolam and fentanyl infusion. The tachycardia aborted, and sedation was weaned off on the next day, approximately 14 hours later.

The patient was found to be Flu A positive

and started on Tamiflu. Blood, urine, and sputum cultures were negative. Repeated ECGs revealed a normal sinus rhythm with no ST elevation, normal PR interval, no delta wave, or left ventricular hypertrophy features. Blood tests including complete blood count, liver and renal function, and thyroid function test were grossly normal. Troponin-I level was initially 10.5ng/L, then elevated to 3830ng/L, and eventually peaked at 20450ng/L.

Echocardiography was done, revealing normal heart chamber sizes, LVEF 55-60%, no regional wall movement abnormalities, borderline increase in left ventricular wall thickness, mild mitral regurgitation, no pericardial effusion, and the IVC was not engorged. Coronary angiogram was performed with normal findings. Antithrombotic and anticoagulation therapy that had been empirically given were stopped.

Electrophysiology study with cardiac mapping was done with the impression of possible left ventricular substrate (electrical or structural) problem or acute myocarditis. Cardiac Magnetic Resonance Imaging was performed, revealing subepicardial late gadolinium myocardial enhancement along the free lateral and inferior wall of the left ventricle, and mild myocardial hyperemia. There was no definite myocardial edema nor increase in T2 value on parametric mapping. Possible differential diagnoses included myocarditis and infiltrative or genetic cardiomyopathy (NDLVC phenotype). He was assessed for genetic cardiomyopathy with the impression of possible left ventricular noncompaction cardiomyopathy, and is pending further genetic testing.

The patient eventually received implantation of a transvenous automated implantable cardioverter defibrillator AICD. He has remained asymptomatic at the writing of this report.

**Ventricular tachycardia (VT) storm**

Electrical storm is defined as ≥3 episodes of sustained ventricular tachycardia (VT) or ventricular fibrillation (VF) or appropriate ICD shocks within 24 hours.[4] The term "electrical storm" was first coined by Credner et al. in 1998[5], and is often used interchangeably with the term "VT storm". However, "electrical storm" is an umbrella term that consists of VT (86-97%), VF alone (1-21%), mixed VT/VF (3-14%), and polymorphic VT (2-8%).[6]

**Pathophysiology of VT storm**

It is important to recognize VT storm as a distinct arrhythmia, as it has an underlying mechanism that requires early aggressive intervention that differs from how we normally treat VT.

Coumel's triangle of arrhythmogenesis is the most commonly used framework to explain the complex pathophysiology of VT storm. It postulates that for VT storm to develop, it requires an arrhythmic substrate, a proarrhythmic trigger, and modulatory factors[7], of which autonomic imbalance is the main contributing factor.[8] All three aspects should be tackled when managing VT storm.

Arrhythmic substrates are conditions that predispose patients to developing VT storm. They can be anatomical or electrical. Anatomical substrates are often developed due to structural heart disease, where

remodeling and myocardial scarring promotes re-entry arrhythmias. Monomorphic VT are often a result of myocardial scarring from old ischemic infarcts or infiltrative diseases.[1] Electrical substrates are often developed from channelopathies that lead to proarrhythmic alterations in ion handling. Potential causes are listed in Table 1.

Proarrhythmic triggers are often external and reversible factors that trigger the VT storm. They commonly include acute myocardial ischemia, heart failure, electrolyte imbalances, and drug toxicity.[9] There are also many other causes, which are also listed in Table 1, such as sepsis, hormonal imbalance (thyrotoxicosis), fever (especially in Brugada syndrome), hypothermia (in malignant early repolarization syndrome), and physical exertion (in catecholaminergic polymorphic ventricular tachycardia). However, it is often difficult to identify a specific trigger, where a study found that only 13% of patients have a trigger identified.[10]

Autonomic imbalance plays a critical role in the initiation and perpetuation of VT storm. Cardiac injury or impaired cardiac function has been shown to stimulate autonomic neural remodeling to preserve systemic circulation, causing sympathetic overexcitation[11] and parasympathetic withdrawal.[12] The resulting sympathetic hyperactivity promotes the development of arrhythmic triggers, where changes to potassium handling promote re-entry, and changes to calcium handling promote early or delayed afterdepolarization.[13] Sympathetic hyperactivity also increases the arrhythmic susceptibility of anatomical substrates, where it amplifies the heterogeneity in repolarization in patients with myocardial ischemia.[14]

The resulting sympathetic overdrive ultimately leads to a vicious cycle: recurrent episodes of VT, shocks from cardioversion or ICD, along with the resulting pain and anxiety, stimulate the sympathetic nervous system, which in turn worsens the arrhythmia, which can induce or exaggerate cardiac ischemia, further promoting electrical instability.

Electrical storm can present with a broad spectrum of symptoms, from palpitations due to tolerated VT, to hemodynamically unstable VT/VF requiring advanced cardiovascular life support (ACLS), or even arrhythmic death.[15]

**Termination of VT storm**

*Electrical therapy*

The first step in managing VT storm is assessing the hemodynamic stability of the patient, and perform resuscitation promptly according to ACLS protocol. Synchronised electrical cardioversion should be initiated if the patient is hypotensive, or presenting with signs or symptoms of hemodynamic instability (e.g. angina, dyspnea, and altered mental status).[4] Initial cardioversion starts at 120-150J[16], with an increasing energy level if the initial shock failed. If the patient is conscious, sedation and anesthesia are advised before cardioversion. Even if the patient is hemodynamically stable, early termination of VT by cardioversion is advocated, given the high risk of sudden deterioration.[17] If the patient becomes pulseless, immediate unsynchronised defibrillation should be performed. In case of refractory VF, two alternative defibrillation strategies are shown to

improve survival.[18] These include vector-change (VC) defibrillation, where defibrillation pads are switched from an anterior-lateral (AL) to an anterior-posterior (AP) position, and double (or dual) sequential external defibrillation (DSED), where two defibrillators with pads placed in two planes (AL and AP position) provide sequential shocks.

*Anti-arrhythmic therapy*

It is essential to initiate anti-arrhythmic agents timely for VT storm. The 2017 AHA/ACC/HRS guideline recommends amiodarone as the 1st line antiarrhythmic because of its superior efficacy and safety profile.[4] Patients on chronic oral amiodarone should still be loaded with intravenous amiodarone.[20]

In cases where amiodarone is ineffective or contraindicated, lignocaine is recommended as a 2nd line antiarrhythmic by the 2022 ESC guidelines.[17] Existing evidence suggest that lignocaine is less effective than amiodarone in out-of-hospital cardiac arrests[21], though a recent study suggests that the reverse is true for in-hospital cardiac arrests.[22] Lignocaine has higher efficacy in ischemic tissue[23], and may be preferred by some clinicians especially in cases of suspected ischemic VT. Procainamide, an alternative anti-arrhythmic, is demonstrated to be more effective in terminating VT of unknown etiology compared to amiodarone.[24] However, its usage has fallen out of favour as it is contraindicated in acute ischemia, severe structural heart disease, decompensated heart failure, and advanced kidney disease, which are all commonly observed in patients with VT storm.

*Beta-blockade*

Antiarrhythmics such as amiodarone are often insufficient to terminate electrical storm due to sympathetic overdrive. Beta-blockers should be given in combination with amiodarone to reduce the impact of sympathetic overactivity in VT storm. The 2022 ESC guideline recommends propranolol, a nonselective beta blocker, over cardio-selective or beta-1 selective beta blockers, like metoprolol[17], as it is more effective, with a 2.7-fold reduction in arrhythmia.[25] This superiority is theorized to be related to ventricular remodeling in chronic heart failure, where beta-1 receptors are downregulated with relative sparing of beta-2 receptors, and that propranolol is lipophilic, which allows it to penetrate the central nervous system to block central and prejunctional receptors in addition to peripheral beta receptors.[26] A reasonable starting dose for propranolol would be 40mg orally, repeated every 6 hours[27], or an intravenous loading infusion of 0.15mg/kg followed by a maintenance infusion of 3-5mg every 6 hours.[28,29] If the patient is hemodynamically unstable, esmolol can be considered. Esmolol is short-acting and titratable but lacks efficacy at beta-2 receptors. A loading dose of 0.3-0.5 mg/kg followed by a maintenance infusion of 0.025-0.05 mg/kg/min titrating up to 0.25 mg/kg/min can be considered.[28,29]

*Deep sedation*

Sympathetic overactivity is a major driver in propagating VT storm. Sympathetic blockade is shown to be superior to standard anti-arrhythmic therapy used in ACLS[30], and often needed to terminate VT storm, as in our patient's case.

The 2017 AHA/ACC/HRS guideline recommends deep sedation as a treatment option to reduce sympathetic overactivity in electrical storm refractory to antiarrhythmics. Martins et al. has shown that termination of electrical storm occurred in 47.4% patients within 15 minutes[31], and up to 80% within hours in a study by Bundgaard et al[32]. Deep sedation with intubation and analgesia may also prepare the patient for further treatment such as catheter ablation.

General anaesthesia with propofol is emerging as the choice of agent.[33] For cases with hemodynamic concern, alternative agents such as midazolam and short-acting opioids (e.g. fentanyl) are effective choices.

*Stellate ganglion block*

In case of refractory electrical storm after anti-arrhythmic and deep sedation treatment, sympathetic neuromodulatory therapy can be considered as salvage therapy. Stellate ganglion block, a technique often done as an outpatient procedure for neuropathic pain, is increasingly utilized. It is relatively safe and can be done with bedside ultrasonography at the emergency department. It is shown to resolve ventricular arrhythmias in 67.5% of patients in a systematic study by Motazedian et al.[34], although the evidence is limited to small observational cohort studies.

The left stellate ganglion is more important in autonomic regulation of the heart, so unilateral left-sided blockade is often advocated. To perform the ganglion block, turn the patient's head to the contralateral side and identify the C6 transverse process of the operating side by palpating it adjacent to the trachea at the level of the cricoid membrane. This should be followed by using ultrasound to visualize the transverse process as well as the adjacent structures, such as the carotid artery, internal jugular vein, longs colli muscle as shown in Fig.3a. Then, with ultrasound in-plane technique, insert the needle at the lateral neck and point it medially as shown in Fig.3b. The needle tip should be directed towards the longus colli, which is in front of the anterior tubercle of the transverse process and lateral to the carotid artery, while avoiding important structures such as the esophagus, vertebral artery and inferior thyroid artery in the trajectory. Finally, inject local anesthetics (e.g. 10ml of 1% lidocaine) under the prevertebral fascia, which is located at the anterior surface of the longus colli muscle. Ipsilateral Horner's syndrome can be used as the evidence of a successful block.

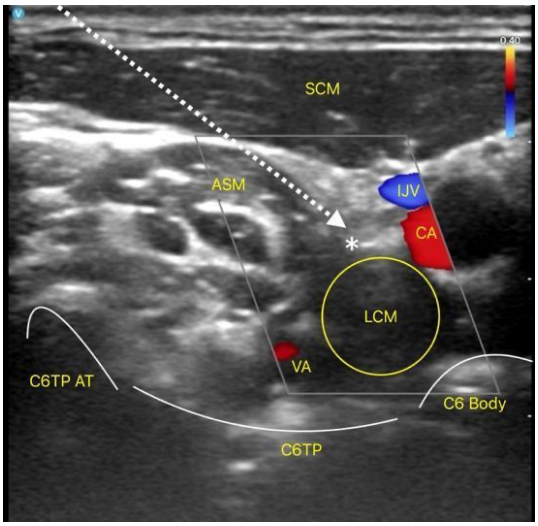


Fig.3a Ultrasound guided stellate ganglion block with needle trajectory (⇢) and injection site (*) as shown (ASM: anterior scalene muscle; AT: anterior tubercle; CA: carotid artery; IJV: internal jugular vein; LCM: longus colli muscle; SCM: sternocleidomastoid muscle; TP: transverse process; VA: vertebral artery)

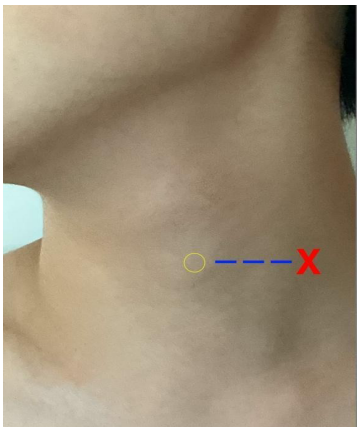

Fig.3b Landmarks of ultrasound probe placement and needle insertion (O: cricoid cartilage; ---: high frequency linear ultrasound probe; X: needle insertion point)

**Hemodynamic support**

Anti-arrhythmics and sedative agents used for electrical storm often have negative inotropic and chronotropic effects that can cause hypotension. Diastolic blood pressure should be sustained to prevent myocardial ischemia, which can be arrhythmogenic. However, there are many limitations to the choice of inotropes or vasopressors. Common inotropes such as epinephrine and dopamine are proarrhythmic and should be avoided, whilst pure vasoconstrictors such as phenylephrine may reduce cardiac output, limiting their use in cardiogenic shock.[35] Norepinephrine, while having a slight risk of pro-arrhythmia, has a better hemodynamic profile and can be considered instead, with a usual dose range of 0.01-0.3 microgram/kg/min.[35]

In cases where vasopressors fail to restore hemodynamic stability or cause arrhythmia, mechanical circulatory support should be initiated, which may include intra-aortic balloon pump, ventricular assist devices, and extracorporeal membrane oxygenation.

**Evaluation and further management**

The underlying etiologies or triggers of VT storm should be identified at the same time while the patient is being stabilised. A focused history typically includes cardiac symptoms such as palpitation, chest pain, shortness of breath, and syncope. These questions, while important, often only aid in assessing hemodynamic stability. To seek the underlying etiology, we also need to explore the patient's past medical history of cardiac disease, family history of sudden cardiac death or genetic arrhythmias, recent cardiac procedures, medications or substance use, and identify potential precipitating factors such as prior exercise, fever, dieting, or emotional stress.

A twelve-lead electrocardiogram (ECG) is essential, which may reveal the causative arrhythmic substrate (e.g. structural heart disease or channelopathy), or pro-arrhythmic trigger (e.g. cardiac ischemia, electrolyte imbalance, drug toxicity).

Blood tests of serum electrolytes (Na+, K+, Mg2+, Ca2+) and thyroid function are standard for evaluating proarrhythmic triggers.[19] A complete blood count and inflammatory markers can also point towards possible infection or inflammation. Liver and renal function tests, if impaired, may increase vigilance for drug toxicity. Toxicology screening may be indicated in cases of suspected abuse. Serial serum troponin levels should also be monitored with correlation of anginal symptoms and ECG changes to look out for acute coronary syndrome, which can both be a trigger and a result of ventricular arrhythmia, especially if poorly hemodynamically tolerated.

Electrical storm requires specialized care and has a high risk of deterioration. Cardiologists should be involved early and patients should be admitted to the CCU for further management. Further investigation and management depends on the underlying triggers and etiologies, and are discussed below.

Electrolyte abnormalities are arrhythmogenic and should be corrected. Magnesium sulphate can be considered as an adjunct therapy even if the magnesium level is normal, especially in cases of polymorphic VT.[36] Potassium levels are also aimed to be high normal (>=4.5mmol/L) as it reduces myocardial excitability.[37]

Echocardiography should be performed to review for cardiac function and identify structural abnormalities. Coronary angiogram should be considered for patients suspicious of myocardial ischemia.[38] Notably, monomorphic VT is rarely caused by ischemia. Ischemic testing and revascularization does not seem to reduce VT recurrence nor affect mortality, but data is limited to small, retrospective, non-blinded studies.[39] Exclusion of coronary artery disease is considered reasonable before catheter ablation.[17]

Catheter ablation is indicated for monomorphic VT refractory to drug therapy. An electrophysiology study is done to map for conduction abnormalities, where identified treatable areas are then ablated. It has higher efficacy in patients with ischemic cardiomyopathies.[40] In these patients, catheter ablation is more beneficial than escalation of anti-arrhythmic drug therapy, and it has recently been shown to be more effective as a first-line therapy than antiarrhythmic drugs.[41]

In patients who continue to have refractory electrical storm, salvage therapies such as cardiac transplantation can be considered.

**Subsequent management after VT storm termination**

After termination of electrical storm, further investigation into the underlying etiology can be performed. Specifically, cardiac magnetic resonance imaging can be done to look for structural heart disease, and positron emission tomography can be used to look for inflammation in cases of myocarditis or sarcoidosis. Genetic testing for channelopathies should also be considered.

Long-term treatment of electrical storm is tailored to the underlying etiology. Generally, pro-arrhythmic triggers should be addressed. Guideline-directed medical therapy should be optimized to treat heart failure. Patients should be advised against pro-arrhythmic drugs and educated on a balanced diet to avoid electrolyte imbalances. Patients with myocardial ischemia who have undergone revascularization should receive antithrombotic and anti-ischemic medications.

Chronic anti-arrhythmic drug therapies for patients with structural heart disease or channelopathies are also shown to be effective. Specifically, amiodarone usage was associated with a 70% relative risk reduction for VT recurrence[42], and is recommended for patients with electrical storm caused by monomorphic VT or repeated ICD discharges.[17] However, there is also a higher rate of medication-related adverse events.[43] Anti-arrhythmic drug

therapy should be adjusted on an individualized basis.

ICD implantation is recommended as secondary prevention of sudden cardiac death in the absence of reversible causes.[17] If the patient already has an ICD, it should be interrogated for any inappropriate shocks and reprogrammed. The psychosocial impact of ICD implantation should also be addressed, as almost 20% of patients developed depressive and anxiety disorders post-implant, which is associated with a greater risk of mortality.[44,45]

**Prognosis**

VT storm is a life-threatening medical emergency with high mortality. It has a higher risk of recurrence and rehospitalization compared to isolated sustained ventricular arrhythmias.[46] It is an independent risk factor for cardiac death (relative risk 2.4), where the risk of death is greatest within three months, then gradually diminishes.[47] VT storm recurrence at one year was found to be 11% and 33% for patients who underwent catheter ablation and medical therapy, respectively.[48] The one-year mortality was found to be 34%, and is mostly driven by heart failure.[49] The 10-year all-cause mortality rate was found to be 39.4%.[50]

**Lessons to learn:**

1. **Electrical storm (VT storm) is defined as ≥3 episodes of sustained VT within 24 hours**
2. **Sympathetic overdrive plays an important role in the pathophysiology of VT storm**
3. **Double sequential external defibrillation should be considered for refractory VT**
4. **Management of VT storm includes early initiation of anti-arrhythmic agents, with the combination of amiodarone and beta-blocker as the first choice**
5. **Refractory VT storm should receive intubation and deep sedation therapy**
6. **For patients with persistent VT storm after sedation, ultrasound-guided stellate ganglion block can be performed**
7. **It is also important to evaluate and treat the underlying etiology, and to transfer the patient to an appropriate facility early for the assessment and management by a cardiologist**

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