

VENTRICULAR TACHYCARDIA ELECTRICAL STORM

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Abstract

The term “electrical storm” (ES) indicates a life-threatening clinical condition characterized by the recurrence of hemodynamically unstable ventricular tachycardia (VT) and/or ventricular fibrillation (VF). We are presenting a case of ES in hemodynamically unstable VT treated at pre-hospital and hospital level.

Seventy-one-year-old patient with a previous history of hypertension, insulin-dependent diabetes, congestive stroke and myocardial infarction, severe three-vessel coronary artery disease and myocardial revascularization, diabetic nephropathy, chronic kidney failure, and chronic ischemic cardiomyopathy, collapses at home. After examination and diagnostic procedures, the emergency medical physician (EMS) diagnoses hemodynamically unstable VT, which he treats with medication and gets the patient urgently transported to a hospital. In the next 24 h, during hospitalization, there are five more separate episodes of VT with pulse treated with DC shock and antiarrhythmics. On the twelfth day of hospitalization, an implantable cardioverter-defibrillator is implanted to prevent sudden cardiac death. On the seventeenth day, the patient is discharged from hospital in stable condition.

The question arises of further best pharmacological and non-pharmacological therapy of ES in hemodynamically unstable VT. It is necessary to educate EMS physicians in the practical application of synchronized cardioversion at the prehospital level.

Key words: electrical storm, unstable, ventricular tachycardia

Introduction. The term “electrical storm” (ES) was first used in 1990 to define the state of electrical heart instability as multiple and potentially lethal ventricular arrhythmia (VA) occurring over a short period of time [1]. The clinical definition of ES is varied, somewhat arbitrary, and a reason for frequent scientific debate. According to the 2017 AHA/ACC/HRS Guideline [2], VT/VF storm (electrical storm or arrhythmic storm) refers to a state of cardiac electrical instability that is defined by ≥ 3 episodes of sustained VT, VF, or appropriate shocks from an ICD within 24 h. Incidents of sustained VT/VF that occur 5 min apart are considered separate episodes. ES is associated with increased mortality and requires immediate medical attention [3,4]. Arrhythmias can be self-terminating but most are terminated by the use of antiarrhythmic drugs or device-related therapy (cardioversion, defibrillation or antitachycardia pacing) [5]. Preventive implantation of an implantable cardioverter-defibrillator (ICD) could be effective therapy to reduce the risk of sudden cardiac death (SCD) and improve the prognosis for some patients [6]. Survival rate of patients with ES is very poor with mortality of 40% within a year [3].

We are reporting a successful joint prehospital and hospital treatment of ES with hemodynamically unstable VT, the first such described in our region.

Case report. An emergency medical team (EMS) intervened on a 71-year-old man who suddenly lost consciousness. Fifteen minutes earlier he had complained of choking and chest discomfort. According to heteroanamnestic data obtained from his wife, he is a person with a previous history of arterial hypertension, type 2 diabetes on insulin therapy, with a stroke and left-sided hemiparesis from three years ago, with a mild myocardial infarction with an inferoposterior localization two years ago, with severe three-vessel coronary disease and myocardial revascularization a year ago, with diabetic nephropathy and chronic renal failure, and chronic ischemic cardiomyopathy.

On examination – unconscious, normal colouration of the skin and visible mucous membranes, afebrile, anicteric, bradypnoic (5 respirations/min), with palpable carotid pulse, obese. No visible injuries. The head and neck of common configuration. Neck veins are not distended. A scar on the chest from recent surgery. On auscultation of the lungs – normal respiratory noise, rare inspiratory cracks basal right. Heart rate rhythmic, tachycardic (Sf 160 bpm) tones quiet, systolic murmur at the top of the heart, normotensive (TA 115/70 mmHg on both arms). Abdomen above the chest level, peristalsis audible, soft, insensitive on palpation, liver and spleen not palpable to be enlarged. Extremities: mild pretibial and perimaleolar edema. On ECG monitor, VT 160–220 bpm (Fig. 1).

Blood glucose values on the glucometer were greater than 30 mmol/l. Airway patency was assured by using oropharyngeal airway and assisted ventilation was applied by ambu balloon. The venous route was established, and 80 mg of 2% lidocaine in continuous infusion of 0.9% NaCl 1000 ml was administered. Twelve (12) units of crystalline insulin s.c. were administered (patient’s therapy).

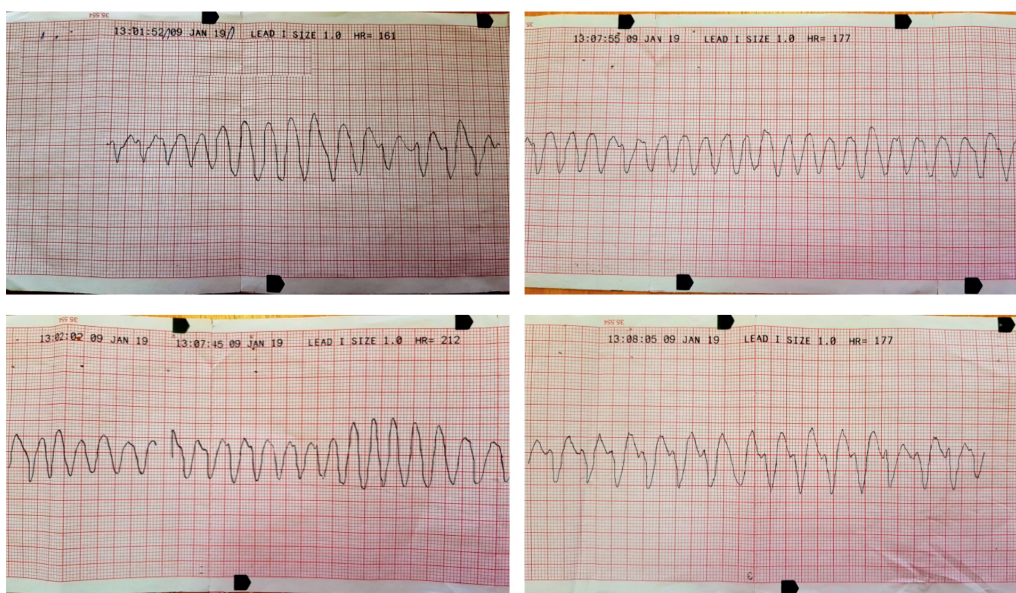


Fig. 1. ECG reading of VT at pre-hospital level

The sinus rhythm Sf 78 bpm was established, radial pulse palpated, the patient breathed spontaneously (did not tolerate airway). The patient was transported to the hospital.

During the transport, the patient was conscious but incorrectly oriented in time and space, eupneic (16 respirations/min), normotensive. The ECG monitor detected a polymorphic VT, which was converted to a sinus rhythm with a DC shock of 360 J. Amiodaron 300 mg in 20 ml of 5% glucose was administered in bolus. The patient was given O₂ 6 l/min (SatO₂ at 80% pulse oximeter). He was handed over to an internist on duty.

On admission, the patient was conscious, disoriented, eupneic, hemodynamically stable.

ECG reading: atrial fibrillation with a normal ventricular response (ventricular rate: 82/min), levogram, rS in inferior walls, left anterior hemiblock, QS waves from V1-V2, anterior R waves in V3-V4, ST segment elevation in leads AVR and V1, negative T wave in V1, AVL and V6.

ECG at discharge: sinus rhythm frequency of 56/min, QS complex in lead D3.

Table 1 shows the findings of the diagnostic procedures used during hospitalization.

The patient is treated with intensive administration of Henle loop diuretics, acetylsalicylic acid, amiodarone, low beta-blocker doses with anticoagulant therapy with unfractionated heparin with the use of ACE inhibitors, trimethazidine, nitrates, as well as statins with gastroprotection with proton pump inhibitors and

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The findings of the used diagnostic procedures

Lungs and heart X-ray	Heart-vessel shadow globally enlarged more to the side of LV, aortic button calcified, congested hila with perihilar congestion of lung parenchyma in pulmonary edema type. Costophrenic sinuses of a mildly decreased transparency, the condition after median sternotomy.
Heart echo	LV normal dimensions. Akinesis of the inferoposterior and inferoseptum walls, with severe LV global systolic dysfunction. LVEF 30%. Posterior mitral cusp calcification. MR trace in mildly enlarged left atrium. Normal aortic diameter, Ao V max 1.8 m/s. Pressure gradient 13 mmHg. Right heart cavities normal dimensions. TR up to 1+, RVSP 45 mmHg. No pericardial effusion.
24-hour Holter ECG	Sinus rhythm, max frequency about 72/min, minimum 33/min, average 52/min, about 14 individual polymorphic PVCs and two PVC pairs were registered.
Laboratory analysis	CRP 52 mg/l (RV < 5 mg/l), BNP 888 µg/l (RV to 67.6 µg/l, INR 1.72 (RV 0.9–1.3), APTV 21.56 s (RV 25–42 s), D-dimer 5.15, Glucose 21.9 mmol/l (RV 4.1–6.1 mmol/l), BUN 20.4 mmol/l (RV 2.5–8.3 mmol/l), creatinine 235 mg/dl (74–124 mg/dl), AST 58 U/l (RV 10–50 U/l), ALT 59 U/l (RV 10–55 U/l), troponin 0.01 µg/l (RV < 0.01 µg/l). Other laboratory findings: SE, TSH, FT4, WBC, RBC, Hgb, Ht, MCV, MCH, MCHC, PC, Na, K, Cl, Mg, phosphates, total protein, albumin, total bilirubin, CK, MB, LDH were within reference values.

Legend: LV – left ventricle; LVEF – left ventricular ejection fraction; MR – mitral regurgitation; Ao V max – aortic peak instantaneous velocity; TR – tricuspid regurgitation; RVSP – right ventricular systolic pressure; RV – reference value; CRP – C-reactive protein; BNP – B-type natriuretic peptide; INR – International Normalized Ratio; APTV – activated partial thromboplastin time; BUN – blood urea nitrogen; OPP – oxidation protein products; AST – aspartate aminotransferase test; ALT – alanine aminotransferase test.

glycoregulation by subcutaneous administration of crystalline insulin according to the daily glycemic profile. Two hours after admission, excellent diuresis (2600 ml) is achieved.

During the first 24 h of hospitalization, five more episodes of monomorphic VT with loss of consciousness were recorded, treated with 200 J synchronous DC shock and with amiodarone and lidocaine.

The next day, a tachyarrhythmia was recorded which, with the use of amiodarone and beta blockers, was converted to a sinus rhythm at frequency 50–55 bmp. No VT episodes were recorded after that.

Upon stabilization, implantation of dual-chamber implantable cardioverter-defibrillator device (ICD) as part of secondary prevention of sudden cardiac death (SCD) was indicated.

It was done 12 days later. After two weeks of hospitalization he was discharged with recommended daily oral therapy: furosemid, cordarone 200 mg, metoprolol 50 mg, fosinopril 10 mg, isosorbide dinitrate 40 mg, trimetazidine 70 mg, rosuvastatin 20 mg, acetylsalicylic acid 75 mg, pantoprazole 40 mg, probiotics, benzodiazepine 1.5 mg and subcutaneous injection short-acting human insulin at a dose 34 ij/day.

Discussion. According to both European [2] and American [3] current guidelines our patient met the criteria for being diagnosed with ES. The case is all the more interesting because, in contrast to the available papers on ES in sustained VT, this is a rare representation of ES in unstable VT and a hemodynamically unstable patient. There could be different causes of instability, such as hypovolemia, heart attack, shock, GI bleeding, ruptured abdominal aortic aneurysm, increased intracranial pressure, VT (as in our patient), atrial fibrillation or flutter, heart blocks. According to the AHA guide, a patient is hemodynamically unstable when VT is with pulse and VT is the cause of one or more of the following conditions: altered mental status, shock, hypotension, pulmonary edema and ischemic chest discomfort. The presented patient was in a coma, with X-ray signs of pulmonary edema, ECHO signs of decompensated cardiomyopathy, and renal insufficiency (elevated urea and creatinine values). VT may be considered unstable when the patient presents with at least two of the following: respiratory distress, discomfort or chest pain, hypotension, signs of shock, pulmonary edema and inadequate perfusion. This is most often associated with mental status changes or a loss of consciousness [7,8].

According to ACLS (advanced cardiac life support) tachycardia algorithm for managing unstable tachycardia [9], prehospital diagnostic-therapeutic steps are recommended (Fig. 2), which were followed in the case presented here: 1. Check airway, breathing, and circulation; 2. Give oxygen if the oxygen saturation is less than 94% or the patient is short of breath; 3. Perform a 12 Lead ECG; 4. Identify rhythm; 5. Check blood pressure; 6. Identify and treat reversible causes if the rhythm is sinus tachycardia; 7. Start an IV.

If the signs and symptoms continue after you have given oxygen and supported the airway and circulation and if significant symptoms are due to the tachycardia, then the tachycardia is unstable and immediate cardioversion is indicated. A short time to direct current cardioversion is the major determinant of survival, and defibrillation should be performed as quickly as possible. ES is a clinical emergency, so in the case presented, the EMS physician made the decision for emergency transport and medicamentous conversion of the rhythm. Synchronized cardioversion was applied only in hospital settings [10]. The therapeutic protocol for VT recommends the use of antiarrhythmic agents (amiodarone and/or lidocaine) as the next therapeutic option [2]. Antiarrhythmic drugs may stabilize ventricular rhythm in many ES patients. In one study, amiodarone was more effective than lidocaine in terminating incessant VT with improved survival

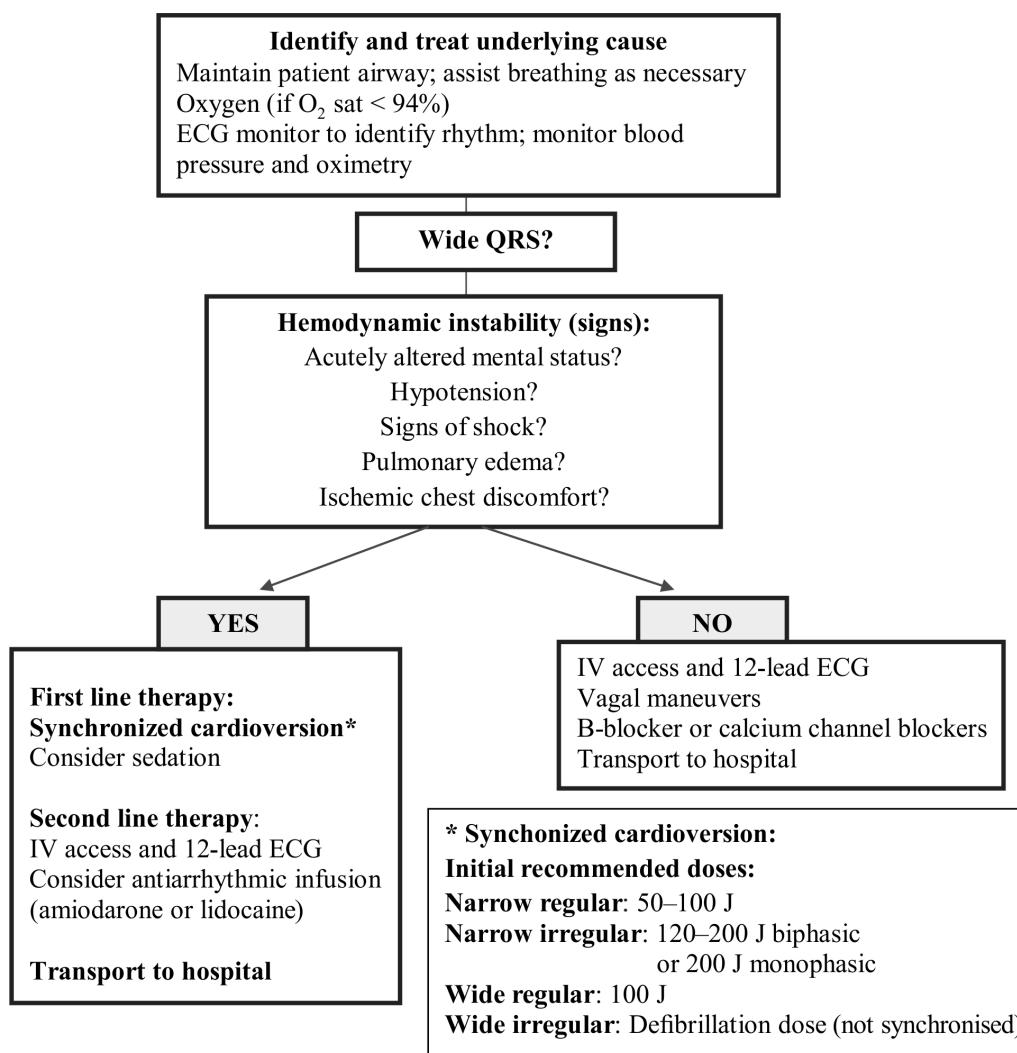


Fig. 2. Prehospital algorithm for managing of ventricular tachycardia (prepared according to ACLS tachycardia with a pulse algorithm available at: <https://www.acls.net/images/algo-tachycardia.pdf>)

at 24 h [11]. Occasional studies with amiodarone have shown positive results, but this is not a consistent finding [12]. In 2016, an interesting paper entitled “The end of amiodarone” [13] was published, in which it is stated that the hype following the introduction of amiodarone pushed procainamide and other antiarrhythmics aside despite compelling evidence. Two recent trials threaten to finally topple its dominance. A recent trial comparing these agents against placebo showed neither improved survival nor neurologic function [14]. Then the PROCAMIO trial tested amiodarone directly against procainamide in hemodynamically stable ventricu-

lar tachycardia [15]. This was a prospective trial of 62 randomized patients who received procainamide or amiodarone. The termination of VT was much more successful with procainamide (67%) compared with amiodarone (38%). Only 9% of procainamide-treated patients suffered a major cardiac adverse event as compared with 41% who received amiodarone, with the most common event being hypotension requiring electrical cardioversion.

Patients with ES undergo an increase of the sympathetic tone and this can provoke further recurrent ventricular arrhythmias. Due to their profile of safety and effectiveness in the treatment of VA and reduced risk of SCD, beta blockers are often first-line antiarrhythmic therapy [11]. With the exception of beta-blockers, currently available antiarrhythmics in randomized clinical studies have not demonstrated efficacy in the primary treatment of patients with life-threatening VA or in the prevention of SCD. ES storm causes significant morbidity and is associated with increased mortality. Therefore, according to the recommendations for secondary prevention of SCD (followed in our case), implantation of ICD (class I, level of evidence B) is recommended in patients with hemodynamically unstable VT, which significantly increases the one-year survival rate of these patients [11].

Left ventricular ejection fraction (LVEF) is also a powerful predictor of cardiac mortality. In clinical practice, LVEF has become the primary criterion used for defibrillator placement. The MADIT II (Second Multicenter Automated Defibrillator Implantation) trial [16] has demonstrated a significant reduction in SCD after ICD placement as in our patients with LVEF of less than 30%. LVEF now occupies a central position in guidelines for the use of ICD when recommended for primary prevention of sudden cardiac death [17].

In the OPTIC trial, a combination of beta-blockers and amiodarone has been shown to reduce the number of ICD shocks. However, side effects may result in discontinuation of therapy in a significant number of patients [18].

A recent meta-analysis of 5912 patients (857 with electrical storm) compiled from 13 studies, found that ES is a strong mortality risk factor and it is associated with an increased combined risk of death (RR 3.15; 95% IC 2.22–4.48), heart transplantation and hospitalization for acute heart failure (RR 3.39; 95% IC 2.31–4.97) [19].

Future research should focus on finding the best pharmacological and non-pharmacological therapy of ES in hemodynamically unstable VT. Practical training of EMS physicians in the application of cardioversion at the prehospital level is also required. This would improve the survival rate of patients with hemodynamically unstable VT whose care begins at this level.

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