MedCrave
Step into the World of Research

Journal of Cardiology & Current Research

Case Report

Open Access

CrossMark

Effectiveness of amiodarone and lidocaine combination in termination of monomorphic ventricular tachycardia electrical storm in patient with dilated cardiomyopathy and severe left ventricular dysfunction

Abstract

Electrical storm is life threatening condition that is defined by ≥3 episodes of sustained ventricular tachycardia (VT), Ventricular fibrillation, or appropriate shocks from an ICD within 24h. In hospitals lacking electrophysiology service, the therapy includes rapid recognition of the condition, treatment of the reversible causes, immediate start of antiarrhythmic drugs and sedation. Catheter ablation should be considered in drug refractory electrical storm.we present a case of dilated cardiomyopathy with severe left ventricular dysfunction came with electrical storm of monomorphic ventricular tachycardia. He was found to have hyperkalemia and metabolic acidosis as precipitating factors which were aggressively treated. He received total of 43 electrical cardioversion shocks for hemodynamic unstable VT. Concomitant administration of intravenous infusion of amiodarone and lidocaine was an effective approach to control the electrical storm within24 h of starting both.

Keywords: electrical storm, recurrent ventricular tachycardia

Volume 12 Issue 1 - 2019

Ahmed AlKalbani, Najib AlRawahi, Rashid AlMuqbali
Ibri and National Heart Center, Royal Hospital, Oman

Correspondence: Ahmed AlKalbani, Ibri and National Heart Center, Royal Hospital, Oman, Tel 0096899272387, Email ask3@yahoo.com

Received: December 31, 2018 | **Published:** January 18, 2019

Introduction

Electrical storm is life threatening condition that is defined by ≥3 episodes of sustained ventricular tachycardia, ventricular fibrillation, or appropriate shocks from an ICD within 24h.[1] According to the above-mentioned definition, the incidence of electrical storm in patients who have an ICD inserted for secondary prevention of sudden cardiac death, is about 10-20%.[2] It is lower when ICDs are placed for primary prevention with an incidence of 4%.[3] Monomorphic VT is the most common form of electrical storm with an incidence of 86-97%.[4] Typically leads to a poor outcome and its management is challenging. The mortality is high as 82%.[5] Also increased mortality has been documented in patients experiencing electrical storm in the AVID, MADIT II trials.[2,3]

Case presentation

This is 34-year-old male patient presented to our hospital with 2 days history of dizziness and myalgia. Initially he denied any past medical illnesses. He is working at a company in the desert. Up on arrival his BP was 80/50, HR 160/min, ECG showed monomorphic ventricular tachycardia (Figure 1). While preparing for cardioversion, he received adenosine 6mg then 12 mg IV, then IV 150 mg of amiodarone but no response noted. He was sedated and first cardioversion was given. His rhythm converted to sinus (Figure 2). Due to the hypotension, he was resuscitated with IV normal saline. Then he developed dyspnea and desaturated in room air, required intubation and ventilation. He was put on amiodarone infusion. His initial work up showed the following Table 1.

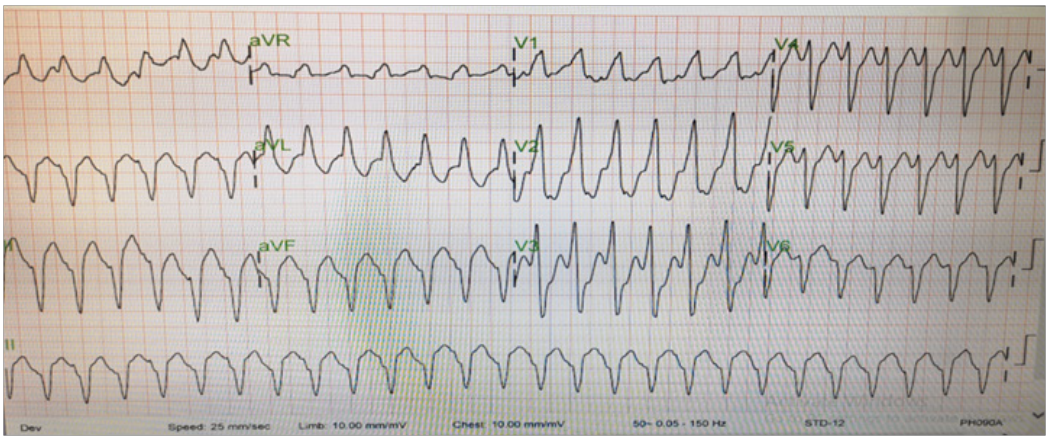

Figure 1 ECG: Monomorphic ventricular Tachycardia

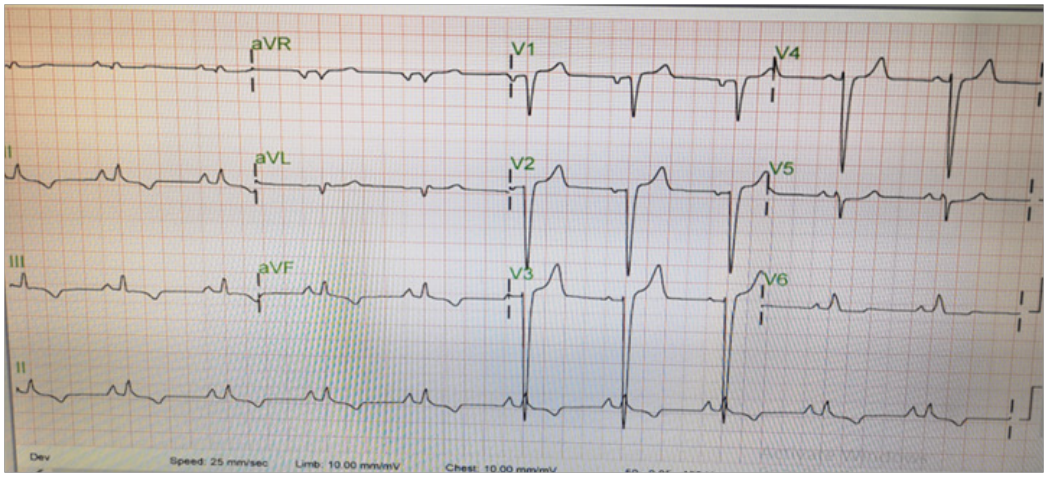

Figure 2 ECG: Normal sinus rythem post-cardiovertion

Table 1 Serum blood tests on admission

	On admission	Normal values
PH	7.16	7.35-7.45
HCO3	13	22-26 mmol/L
Lactate	4.7	mmol/L
Pco2	45	35-45 mm [Hg]
Hb	17	11.5-15.5 (g/dL)
Wbc	15.8	2.2–10 10*3/uL
Na	133	137–148 (mmol/)
K	5.6	3.5–5 mmol/L
Creatinine	134	45–100 umol/L
Urea	11	2.5-6.7 mmol/L
Random blood glucose	5.18	3-7.7 mmol/L
Total bilirubin	28	0–20 umol/L
ALT	595	0–40 U/L
ALP	69	40–150 U/L
AST	1600	5–35 U/L
Corrected Ca	2.4	2.07-2.6 mmol/L
Mg	1	0.65-1.1 mmol/L
Phosphorus	1.2	0.75-1.5 mmol/L
Troponin	219	0–14 pg/mL
CK	207	30–200 U/L

The chest x-ray showed cardiomegaly with pulmonary oedema. He was admitted to the ICU. An urgent echocardiography showed dilated left ventricle with severe LV systolic dysfunction, grade III diastolic dysfunction, moderate MR and normal RV function and size. He was kept on amiodarone infusion and IV fluids as maintenance. His repeated K was 6.7 mmol/L. Initial impression was metabolic acidosis/hyperkalemia with acute kidney injury secondary to hypovolemia and low cardiac output. The VT secondary to acidosis and hyperkalemia. He received anti-hyperkalemic measures and sodium bicarbonate to correct his acidosis and hyperkalemia. He was started empirically on ceftriaxone after sending septic workup. In view of hypotension he was started on nor-adrenaline and dobutamine. Unfortunately, he developed recurrent VT required multiple DC shocks and received total of 500 mg IV boluses of lidocaine and 2g of Magnesium sulfate along with amiodarone infusion.

His repeated blood gas after maintenance of IV fluid and sodium bicarbonate infusion showed improvement of the acidosis (pH 7.27, pCO2 36 mm [Hg], HCO3 18 mEq/l), potassium 4.1 mmol/l, Lactate 1.6 mmol/L. However, the patient continued to have recurrent VT. External Electrophysiology consultation was done regarding the VT storm and the advice was to correct the acidosis, manage electrolytes imbalance, continue amiodarone infusion and start lidocaine infusion of 2mg/min. Over 24 hrs, the patient received 43 DC shocks. After the 24 hrs, he had 1 episode of VT which was aborted by it. Dobutamine was gradually tapered down as it is pro-arrhythmic and kept only on noradrenaline. The repeated blood gas after 24 hr showed pH 7.38, PCO2 47 mm [Hg], HCO3 28, potassium 3.5, lactate 1.7 mmol/L. On 3rd day of admission, the family brought a medical report, stated that the patient is known to have cardiomyopathy with left ventricular dysfunction diagnosed in September 2015. Coronary angiogram showed normal coronaries. He did Cardiac MRI on 10/2015 which showed moderate LV dysfunction with regional wall motion abnormalities involving inferior and lateral wall of LV along with transmural enhancement and features suggestive of amyloidosis. He did Cardiac PET scan 10/2015 which showed increased FDG uptake in inferior and lateral wall of LV suggestive of active inflammatory process. He was started empirically on immunosuppressant drugs and steroids. PET scan was repeated in 8/2016 and it was similar to 2015, hence immunosuppressants doses were increased. PET scan repeated in Jan/2018 showed persistent severe perfusion in lateral & major part of inferior part of LV and there was no improvement in extent & severity of cardiac inflammation, hence the immunosuppressant and steroid were stopped. He also did fat bad biopsy and bone marrow biopsy which were normal. Finally, he did cardiac endometrial biopsy 9/9/2016 (in September 2016) which showed fibrosis involving endometrium and interstium without features of amyloidosis or sarcoidosis. The final diagnosis was dilated cardiomyopathy probably post myocarditis. He was kept on losartan, metoprolol XL and eplerenone.

In view of background history of steroid use, hypotension, metabolic acidosis, hyponatremia and hyperkalemia, Addisonian crisis was to be ruled out. Hence, a short synacthen test was done then

Citation: AlKalbani A, AlRawahi N, AlMuqbali R. Effectiveness of amiodarone and lidocaine combination in termination of monomorphic ventricular tachycardia electrical storm in patient with dilated cardiomyopathy and severe left ventricular dysfunction. *J Cardiol Curr Res.* 2019;12(1):31–34.
DOI: 10.15406/jccr.2019.12.00425

patient was started on IV hydrocortisone empirically. It was noted that BP improved, and noradrenaline was gradually tapered down and stopped. Random cortisol at 1300 hrs was 350 nmol/. Serum ACTH level was not measured. The Serum cortisol levels of Short synacthen test (250 mcg) were as follows as 292nmol/L, before ACTH injection, 573nmol/Lat 30 minutes after ACTH injection and, 335nmol/L at 60 minutes after ACTH injection. The test was done while the patient was intubated and mechanically ventilated in ICU. Basal cortisol was lower than the cut-off expected in a critically sick patient. Adrenal cortisol responsiveness to 250 mcg of intravenous cosyntropin (ACTH) injection was satisfactory at 30 minutes. However, patient was treated with intravenous hydrocortisone injection as cover during acute stress. The addition of steroids along with other management measures, helped improve the patient's BP, improve acidosis, and ease electrolytes imbalance. Later on, and after patient was extubated and shifted to the general ward, he was shifted to smaller dose of intravenous hydrocortisone, then to oral steroids, that were later tapered off gradually. The patient remained stable off-steroids while still inpatient.

As patient continues to be off VT for 14 hrs, lidocaine was tapered down to 1 mg/min and stopped after 24 hrs. He was continued on amiodarone infusion 0.5 mg/min during the period of intubation. He was started also on bisoprolol once noradrenaline was stopped. He was extubated after 7 days from admission. He was started on amiodarone oral 400 mg tid after extubation. He was gradually restarted his antifailure medications including valsartan along with bisoprolol and frusemide. Later on, the patient did very well with no more VT and clinically he was not in heart failure. His lab works up on discharge showed improvement of his renal and liver profile with normal blood gases/acid base and electrolytes. He was transferred to another hospital where ICD was inserted and he was discharged home in a stable condition.

Discussion

Electrical storm is an emergency condition that requires prompt treatment. After proper diagnosis of VT, patient should be assessed immediately for hemodynamic stability and be treated according to advanced cardiac life support.[6] Reversible causes should be identified and treated aggressively. In most of the cases, the triggering cause cannot be identified and the reversible precipitating factors found in less than 10% of the patients.[7] The commonly reported precipitating conditions are acute myocardial infarction and ischemia, congestive cardiac failure decompensation, electrolyte abnormalities (Hypo/hyper-kalemia, Hypomagnesemia), hyperthyroidism and antiarrhythmic drug therapy (Vaughan-Williams Class IA, Class III).[8] In our patient the possible triggering factors of VT storm is acid-base imbalance (acidosis), electrolyte imbalance (hyperkalemia) and worsening heart failure. There are 2 possible scenarios of the acid-base and electrolytes imbalance in this patient. First dehydration with worsening of his renal profile leads to acidosis and hyperkalemia and subsequent VT storm. The other acceptable scenario is worsening of heart failure which trigger VT storm. Such High VT burden could compromise cardiac contractility and subsequent hypoperfusion of other organs including the kidneys. Along with treatment of reversible causes, administration of antiarrhythmic drugs should be started immediately. Electrical storm usually requires more than one antiarrhythmic medication. Although limited data exist, b-blockade in conjunction with amiodarone appears to be the most effective therapy for electrical storm.[9] Initially, this patient was put on amiodarone infusion. The b-blocker could not be given as he was hypotensive and required inotropic and vasopressor support. Amiodarone is considered the most effective antiarrhythmic drug and widely used in the treatment of electrical storm.[9] Unless presence of contraindications such as hyperthyroidism or QT prolongation, amiodarone can be given safely. It has a mixed antiarrhythmic class action with a prevalent class β action (potassium channel blocker).[7]

In view of recurrent VT that required several synchronized DC shock, hemodynamic instability and inability to give b-blocker, another anti-arrhythmic in conjunction to amiodarone was needed. The next available drug was lidocaine which was given in several boluses then continuous infusion. Combination of reversible cause's correction (hyperkalemia and acidosis) and dual antiarrhythmic infusion, the VT storm was controlled within 24 hr. Catheter ablation should be considered in drug refractory electrical storm.[1] lidocaine is class IB antiarrhythmic drug, acting as rapid sodium channel blockers binding to the receptor in a use-dependent fashion. Its effectiveness in terminating ventricular arrythmias is mostly in ischemia setting.[7] In conditions other than ischemia, the effectiveness of lidocaine in terminating ventricular arrythmias range from 8%-30% which is considered as relatively weak antiarrhythmic effect.[10] In retrospective study of 42 patients, lidocaine was effective in treating ventricular arrythmia in 26 patients (62%) and it was ineffective in 16 patients (38%). Out of the effective group, there were 11 patients (42%) already on amiodarone at the start of lidocaine. The remaining number of patients (15 patients) in the effective group was on lidocaine alone. In the ineffective group, there were also 15 patients on lidocaine alone. It was noted that left ventricular ejection fraction was significantly higher in the effective group who were on lidocaine alone compared to the other group (EF% 51+/- 16 vs 32+/-9). The results of this study suggest that lidocaine has favorable effect in patients with normal ejection fraction and combination with amiodarone can terminate most refractory arrhythmia.[11]

The mechanism of antiarrhythmic effect of lidocaine and amiodarone combination therapy is that mainly due concomitant blockade of potassium and sodium channels. amiodarone has mainly potassium channel blockade effect with weak blocking effect on sodium channel. By adding the strong sodium channel blockade effect of lidocaine, the amiodarone antiarrhythmic effect is reinforced. In addition, lidocaine also provides blockade of inactivated sodium channels. The effectiveness of combination therapy was observed during both the acute and chronic phrases of amiodarone therapy.[11]

Conclusion

Electrical storm is an emergency condition that requires prompts treatment. In hospitals lacking electrophysiology service, the therapy includes rapid recognition, treatment of the reversible causes, immediate start of antiarrhythmic drugs and sedation. Aggressive correction of electrolytes imbalance and acidosis with concomitant administration of intravenous infusion of amiodarone with lidocaine is an effective approach in treating electrical storm in patients with dilated cardiomyopathy and severe left ventricular dysfunction.

Acknowledgments

None.

Conflicts of interest

Author declares that there is no conflict of interest.

Citation: AlKalbani A, AlRawahi N, AlMuqbali R. Effectiveness of amiodarone and lidocaine combination in termination of monomorphic ventricular tachycardia electrical storm in patient with dilated cardiomyopathy and severe left ventricular dysfunction. *J Cardiol Curr Res.* 2019;12(1):31–34.
DOI: 10.15406/jccr.2019.12.00425

References

1. Al-Khatib SM, Stevenson WG, Ackerman MJ, et al. AHA/ACC/HRS Guideline 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 on Clinical Practice Guidelines and the Heart Rhythm Society. *J Am Coll Cardiol.* 2017;138(13):e210-e271.
2. Exner DV, Pinski SL, Wyse DG, et al. Electrical storm presages non-sudden death: the antiarrhythmics versus implantable defibrillators (AVID) trial. *Circulation.* 2001;103(16):2066–2071.
3. Sesselberg HW, Moss AJ, Mc Nitt S, et al: Ventricular arrhythmia storms in postinfarction patients with implantable defibrillators for primary prevention indications: a MADIT-II substudy. *Heart Rhythm.* 2007;4(11):1395−1402.
4. Antonio Sagone. Electrical Storm: Incidence, Prognosis and Therapy: review article. *J Atr Fibrillation.* 2015;8(4):1150.
5. Nademanee K, Taylor R, Bailey WE, et al. Treating Electrical Storm Sympathetic Blockade Versus Advanced Cardiac Life Support–Guided Therapy. *Circulation.* 2000;102(7):742−747.
6. Link MS, Berkow LC, Kudenchuk PJ, et al. Adult Advanced Cardiovascular Life Support: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2015;132(18 Suppl 2):S444−S464.
7. Muser D, Santangeli P1, Liang JJ. Management of ventricular tachycardia storm in patients with structural heart disease. *World J Cardiol.* 201726;9(6):521−530.
8. Sorajja D, Munger TM, Shen WK. Optimal antiarrhythmic drug therapy for electrical storm (Review Article). *J Biomed Res.* 2015;29(1):20−34.
9. Vassallo P, Trohman RG. Prescribing amiodarone: an evidence-based review of clinical indications. *JAMA.* 2007;298(11):1312−1322.
10. Eifling M, Razavi M, Massumi A. The Evaluation and Management of Electrical Storm: review. *Tex Heart Inst J.* 2011;38(2):111−121.
11. Yoshie K, Tomita T, Takeuchi T, et al. Renewed impact of lidocaine on refractory ventricular arrhythmias in the amiodarone era. *Int J Cardiol.* 2014;176(3):936–940.

Citation: AlKalbani A, AlRawahi N, AlMuqbali R. Effectiveness of amiodarone and lidocaine combination in termination of monomorphic ventricular tachycardia electrical storm in patient with dilated cardiomyopathy and severe left ventricular dysfunction. *J Cardiol Curr Res.* 2019;12(1):31–34.
DOI: 10.15406/jccr.2019.12.00425