Panacea Journal of Medical Sciences 2022;12(3):714–716

Content available at: https://www.ipinnovative.com/open-access-journals

Panacea Journal of Medical Sciences

Journal homepage: http://www.pjms.in/

Case Report

Ventricular fibrillation – reverted in the settings of acute myocardial infarction and thrombolysis – A case report

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ARTICLE INFO

Article history:
Received 20-07-2021
Accepted 02-12-2021
Available online 28-11-2022

Keywords:
ventricular fibrillation
STelevation myocardial infarction
Acute Myocardial Infarction

ABSTRACT

Ventricular fibrillation (VF) may be coarse or fine rapid, fibrillary movements of the ventricular muscle, Factors that commonly precipitate cardiac arrhythmias include hypoxia, electrolyte disturbance, myocardial ischemia and certain drugs. Symptoms during arrhythmia may be syncope, palpitation, breathlessness and sudden loss of consciousness. Approximately 10% of patients with AMI develop VT that degenerates into VF[2]. This may occur in the settings of acute myocardial infarction (AMI) in thrombolytic patients. We present 3 cases of VF during and immediately after thrombolysis, which are reverted to normal with prompt cardioversion.

1. Introduction

The management of acute ST-Elevation Myocardial Infarction (STEMI) has rapidly evolved worldwide during the last two decades better to understand the need for early reperfusion and protocol-based pharmacotherapy. When primary percutaneous coronary intervention (PCI) cannot be performed in a timely way in patients with ST-segment myocardial infarction (STEMI), fibrinolytic treatment is used as a therapeutic option.[1] Fibrinolytic therapy can help restore coronary artery blood flow, which is helpful in myocardial salvage, reduced mortality, and better cardiovascular outcomes. The effectiveness of fibrinolytic therapy is time-dependent, with the highest results occurring within the first two hours after the onset of the symptom, and the benefits rapidly reduce after three hours.[2] Streptokinase is a 415–amino acid bacterial protein. Streptokinase, the least expensive fibrinolytic and still widely used globally, it is administered by short-term infusions.[3] Ventricular tachycardia (VT) and ventricular fibrillation (VF) can occur in acute myocardial infarction due to total blockage or reperfusion. Ischemic arrhythmias are defined as arrhythmias that occur as a result of decreased myocardial Perfusion, whereas reperfusion arrhythmias are defined as arrhythmias that occur during reperfusion.[4] During 15 months from January 2014 to the end of March 2015, we had 87 patients with AMI. Of the 87 cases, 74 were ST-elevation MI (STEMI); out of this 74, only 36 were eligible for thrombolysis & were thrombolysed. Unfortunately, VF is a rapidly fatal rhythm which never terminates spontaneously and CPR must be initiated promptly.Prompt defibrillationduring ventricular fibrillation could revert the rhythm to normal with 100 percent success.

2. Case Report 1

A 58-year-old male patient was admitted to the emergency department early in the morning with severe chest pain for 1 hour associated with sweating. The patient was not a known hypertensive hyperlipidemic or diabetic, with a smoking history of 20 cigarettes for 30 years. Physical examination

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https://doi.org/10.18231/j.pjms.2022.132

of the patient showed blood pressure 110/78 mmHg, facial appearance of acute illness, no jugular engorgement, clear lungs, no added breathing sounds, non-expanded heart boundary, heart rate was 78 bpm, regular in rhythm, hearts sounds were soft, no murmurs. No pedal edema. No crackles over in lungs fields and his SpO2 at 4L of O2 was 94 percent. ECG revealed sinus rhythm, low voltage; ST elevation in leads V1 to V4 was made.

A diagnosis of acute anterior wall myocardial infarction was made. Within 30 minutes of arrival, thrombolytic therapy was started with 1.5 million units of streptokinase infusion. During thrombolysis, the patient developed ventricular fibrillation 25 minutes after starting the infusion. He was defibrillated immediately after the second shock of 360-joule, the rhythm was reverted to normal, the patient was conscious, and the SpO2 was 92 percent with 6L of O2, heart rate of 90 bpm, and a respiratory rate of 26 breaths per minute. Thrombolysis was completed on time, his heart rate was 84 bpm, and his SpO2 was 90% with 4L of oxygen. ST-segment elevation also returned to normal. The patient was given Enoxaparin 60mg twice a day for 5 days and additional cardiac drugs. The patient was stable and discharged on day 5 and advised to attend cardiologist for CAG.

3. Case Report 2

A 37-year-old male notice 8/10 retrosternal chest pain with shoulder discomfort for 2 hours, reported to the Emergency department in the early morning. The patient described the current pain as severe and sharp, in addition, the patient also complained of nausea and shortness of breath. The patient's vital signs were HR: 110 BP: 150/90 RR: 30 Temp. 97.6 O2 Sat: 92% on 2L of O2 nasal cannula, EKG shows ST-segment elevation in leads II, III and AVf. This patient is a chronic alcoholic. ECG on arrival showed ST-segment elevation, inferior Wall myocardial Infarction.

Treatment was initiated with 5mg of isosorbide dinitrate sublingually; his blood pressure dropped to 80/60mmHg suddenly. Heart rate decreased to 40/bpm was administered 3amps of 0.6mg atropine within a span of 10min. An intravenous bolus of 750ml of normal saline was started & his BP was raised to 160/100mmhg. Dopamine infusion was discontinued. Inj. Pethidine along with inj. Phenergan was administered for his chest pain.

Patient treatment was started with thrombolysis with 1.5 million units of streptokinase was started around 10.40 am and completed by 11.20 am. He was restless and in agitation during thrombolysis and after. He was administered 2mg of IV midazolam to control his restlessness. It was ineffective. He was then given an inj. haloperidol 2mg IV, which quietened him. However, within few minutes, he had ventricular fibrillation and was reverted to normal by immediate defibrillation with 360 joules electric shock. The patient was stable at discharged with the advice to continue cardiac drugs.

4. Case Report 3

A 35-year-old heavily built male was brought to ICU in the evening with a history of chest pain radiating to his back for 2 hours. In addition, the pain radiated to the right shoulder and arm associated with sweating. He denied palpitations, dizziness, or syncope; he had an episode of nausea before presenting to the emergency department. Within 10 to 15 minutes of his arrival, the pain was severe. There was no significant family history of atherosclerotic coronary artery disease (CAD). ECG showed ST elevation in the precordial leads V1 to V4 defines the presentation of anteroseptal myocardial infarction.

He was given a loading dose of aspirin, clopidogrel, and atorvastatin stat. For his chest pain inj. Morphine 3mg and 2mg at an interval of 5mins along with 25 mg of inj. Phenergan.

Thrombolysis was started with 1.5 million units of streptokinase infusion. The patient was restless throughout and he was sedated with 5mg of diazepam. Streptokinase infusion was completed in 45 minutes. Heart sounds were normal. Heart rate was 100b/mt; blood pressure was 130/100mmhg, SpO_2 98% in room air. Lung fields were clear. After an hour of thrombolysis, the patient complained of chest pain and suddenly lost consciousness.

Patient ECG monitor showed features of ventricular fibrillation; cardiac resuscitation was started at once with CPR, epinephrine and shock in cycles. After the 3^{rd}electrical shock with 360 joules, the patient regained consciousness in 20 minutes. Epinephrine is the presser agent most frequently used during CPR that converts fine VF into more easily converted coarse VF[5]. Dopamine was on flow. In about 10mins the patient was conscious, heart rate was 140/bpm, blood pressure was 100/70mmHg SpO_2 86% with 6 liters of O_2. To maintain O_2 saturation anesthetist opinion was sought. Arterial blood gas analysis showed metabolic acidosis. The anesthetist was of the opinion to intubate and to ventilate to maintain saturation. The patient was referred for a coronary angiogram in a specialty center. After few days, he was discharged from the referral hospital in a stable status.

5. Discussion

VF is not a rare occurrence in thrombolytic AMI patients. VF in the settings of AMI with early cardiac resuscitation and defibrillation can revert the rhythm to normal. The time delay between VF and defibrillation is an important factor in determining survival.[5] In case 1, the time delay is less ~ 1 min and in case 2, less than 2 min and in case 3~3min.

The time delay between VF and defibrillation is an important factor in determining survival.[5] In case 1, the time delay is less than ~ 1 min and in case 2, less than 2 mins, and

in case 3, approximately 3mins. In the 1st case, VF occurred during thrombolysis. According to a report in one study, all cases received the identical 360-joule shock that the likely hood of VF on thrombolysis or placebo is similar.[6,7]

In our study, age did not play any significant role in recovery. In case 1, the patient was 58 years and in cases 2, 3, it was 37 years and 35 years, respectively, but all were recovered soon with timely defibrillation. Out of the 36 thrombolysed patients, there were 2 deaths, and 3 cases of VF were reverted. Thus, approximately 15% of thrombolysed patients had suffered VF in this small study.

The factor that increases the tendency of the heart to fibrillate is an increase of extracellular potassium concentration resulting from loss of potassium from the ischemic cardiac myocytes; currant of injuries from the infarct; increased irritability of cardiac muscle resulting from sympathetic reflexes, circum movements because of increased path length for impulse conduction.[8]

VF may be due to multiple factors, hypoxia, ischemia, necrosis and reperfusion. VF may also occur due to pro-arrhythmic drugs. In an animal study, haloperidol significantly increased VFT (making it more difficult to induce ventricular fibrillation). In addition, haloperidol significantly decreased MAP but did not substantially affect mean HR or duration of the QRS or QTc intervals on surface ECG. Haloperidol increased VFT in all animals studies the administration of haloperidol has been associated with prolongation of QT interval on surface ECG.[9]

In a study, VF occurring within 24 hours of thrombolysis is said to be an epiphenomenon that does not require long-term antiarrhythmic treatment· In addition, it is said that VF is a non-invasive indicator of

VF in the settings of STEMI and thrombolysis is not a rare occurrence. In the 1st 12 hours of post thrombolytic period, we should be watchful and expect such arrhythmias. Successful resuscitation is the rule if cardiac resuscitation and defibrillation are attempted early.

6. Source of Funding

None.

7. Conflicts of Interest

There is no conflict of interest.

References

1. Reddy K, Khaliq A, Henning RJ. Recent advances in the diagnosis and treatment of acute myocardial infarction. *World J Cardiol*. 2015;7(5):243–76. doi:10.4330/wjc.v7.i5.243.
2. Dalal J, Sahoo PK, Singh RK, Dhall A, Kapoor R, Krishnamurthy A, et al. Role of thrombolysis in reperfusion therapy for management of AMI: Indian scenario. *Indian Heart J*. 2013;65(5):566–85.
3. Kumar A, Cannon CP. Acute coronary syndromes: Diagnosis and management, part II. *Mayo Clin Proc*. 2009;84(11):1021–36. doi:10.1016/S0025-6196(11)60674-5.
4. Bhar-Amato J, Davies W, Agarwal S. Ventricular Arrhythmia after Acute Myocardial Infarction: 'The Perfect Storm'. *Arrhythm Electrophysiol Rev*. 2017;6(3):134–9. doi:10.15420/aer.2017.24.1.
5. Nollain K. The ICU Book. *Sultan Qaboos Univ Med J*. 2008;8(1):95–6.
6. Solomon SD, Ridker PM, Antman EM. Ventricular arrhythmias in trials of thrombolytic therapy for acute myocardial infarction. A meta-analysis. *Circulation*. 1993;88(6):2575–81. doi:10.1161/01.cir.88.6.2575.
7. Khonsary SA. Guyton and Hall: textbook of medical physiology. *Surg Neurol Int* . 2017;8:275. doi:10.4103/sni.sni_327_17.
8. Hall J. Guyton and Hall: Textbook of Medical Physiology. *Surg Neurol Int*. 2011;8:275. doi:10.4103/sni.sni_327_17.
9. Bett JH, Holt GW. Malignant ventricular tachyarrhythmia and haloperidol. *Br Med J*. 1983;287(6401):1264.

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Cite this article: Jayaraj R, Archana S, Venkataraman DD. Ventricular fibrillation – reverted in the settings of acute myocardial infarction and thrombolysis – A case report. *Panacea J Med Sci* 2022;12(3):714-716.