Letter

How many times can we defibrillate a patient?

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Ventricular fibrillation (VF) is the cardiac rhythm disturbance which is the number one cause of cardiac arrest. Most common etiologic factor is the coronary heart disease [1]. VF may occur due to acute myocardial ischemia, infarction or a scar tissue formed previously because of a prior myocardial infarction (MI). Defibrillation is the first line therapy. Antiarrhythmic drugs should also be added, and the predisposing factors should be eliminated as well [2]. Sometimes multiple shocks are needed for resistant or recurrent VF. We have a case in whom we performed several times of defibrillation because of repeated VF attacks. This 38-year-old male who was resuscitated because of VF at a different center was transferred to our hospital with extensive anterior MI. Because of cardiogenic shock an intra-aortic balloon pump was placed in addition to positive inotropic therapy and mechanical ventilation. The patient was defibrillated twice in the 3rd and the 5th days of MI. The condition of the patient got worse and he was transferred to our unit in the 17th day of MI with respiratory failure, acute renal failure (BUN 76 mg/dL and creatinine 5.7 mg/dL) and frequent ventricular premature beats. Because of recurrent VFs he was under amiodarone infusion. Potassium and magnesium were replaced as well. Echocardiography revealed anterior IVS (interventricular septum) mid-apical segment, apex, and anterior wall were akinetic. We performed coronary angiography which showed that left anterior descending (LAD) artery was totally occluded at its ostium. Right coronary and circumflex coronary arteries were normal. LAD was opened and two bare metal stents were placed. Soon after the intervention VF attacks started. Lidocain infusion was started in addition to amiodarone infusion. Despite the antiarrhythmic infusions VF runs continued and he was defibrillated 50 times in 9 hours. ECG showed ST depression in leads D2-D3-aVF and ST elevations at precordial leads between the shocks. Reinfarction was thought and nitroglycerin and aspirin were added. At this period his blood testes was normal besides BUN and Cre. And his cardiac markers before and during attacks were CKMB: 1.4, 2.3, 11.7, 18.8-Myoglobin: 180, 229, 989, 1235- Troponin: 4.95, 4.81, 5.09, 4.86. At echocardiography 8 hours after attacks, apex was hipokinetic and other wall motions were normal. With all these findings reperfusion injury considered as the cause of these attacks. 30 hours after attacks patient was extubated, amiodarone infusion stopped gradually and metoprolol therapy was started. CKMB, myoglobin, troponin levels decreased, renal functions improved and the patient was discharged and referred to cardiology outpatient clinic.

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Discussion

VF is at the top of cardiac rhythm disturbances that cause cardiac arrest. There are many causes of VF but coronary heart disease is the most common etiologic factor among these. VF may occur due to acute myocardial ischemia/infarction or from a scar tissue because of a previous MI. Intracellular calcium accumulation, effects of free radicals, metabolic and autonomic variations are other mechanisms that lead to VF. Congenital anomalies like tetrology of fallot, long QT syndromes, cardiomyopathies are other causes that may trigger this lethal rhythm disturbance. VF attack is a condition that 2 or more times repeating ventricular tachycardia or ventricular fibrillation episodes with hemodynamic compromises [3].

Reperfusion injury occurs when an ischemic tissue is reperfused. Experimental studies showed that reperfusion injury may cause reperfusion arrhythmias, transient myocardial disfunction and cell death [4].

When a patient develops VF he had to be defibrillated and antiarrhythmic therapy should be started. Then, predisposing factors should be eliminated [2]. If the VF repeats frequently then multiple shocks are needed. It was shown experimentally that repeated defibrillations may lead to myocardial damage, conduction disturbances and decrease of myocardial functions after resuscitation. For this reason strategies to limit the number of defibrillation are needed. On the other hand in the present case nearly 50 times defibrillation in a short period did not cause cardiac damage and the patient survived. There are other cases in whom multiple defibrillations were applied successfully [5-7]. Reperfusion injury may be speculated as a cause of VF storm in the present case and this injury may cause fatal arrhythmias. Whatever the cause multiple shocks should be given and this approach may not cause major injury to the patient.

References