Polymorphic Ventricular Arrhythmia Successfully Managed By Electrical Defibrillation: A Case Report And Review Of Literature

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Abstract

Cardiac arrest is a common occurrence on the medical ward and the rate of complete physiologic recovery following in-hospital arrest is poor. Previous reports on the survival of patients who have had in-hospital cardiac arrest showed that only about 20% are alive to be discharged home. Most of the survivors of in-hospital arrest often have a witnessed ventricular tachycardia (VT) or fibrillation (VF) and are defibrillated urgently. A critical assessment of events preceding many cardiac arrests may reveal abnormalities of the airway, breathing and circulation. Hospital processes such as availability of defibrillators, emergency medical team response and state of intensive care unit facilities may have significant effects on patient outcome. The case of a 41 year old man with pre-existing QTc prolongation, who developed polymorphic ventricular arrhythmia and hemodynamic collapse while on admission in the hospital, is discussed. He was successfully managed and discharged.

Key words: QTc prolongation, torsade de pointes, electrical defibrillation.

Introduction

Cardiac arrest is the cessation of cardiac mechanical activity as confirmed by the absence of signs of circulation. Outcome following cardiac arrest and cardiopulmonary resuscitation is dependent on critical interventions, particularly early defibrillation, effective chest compression and advanced life support[1]. Survival from cardiac arrest decreases by 10% to 7% each minute defibrillation is delayed[2]. The incidence of in-hospital cardiac arrest in Europe and America is between one to five events per 1000 hospital admissions and the survival rate is between 10-20%[1-2]. Most adult survivors of in-hospital arrest have a witnessed ventricular tachycardia (VT) or fibrillation (VF) and are defibrillated urgently[2]. *Torsade de pointes* is a form of polymorphic VT which usually occurs in the setting of QT interval prolongation, and it may degenerate into VF, cardiac arrest and sudden cardiac death (SCD). Early recognition and stabilization of patients at risk, prompt in-hospital resuscitation and early defibrillation improve patient outcome[3-4]. Often, medical and nursing staffs do not possess acute-care knowledge and skills, and may lack confidence when dealing with acute-care problems[4]. In addition, hospital processes and policies may delay emergency response and early defibrillation of patient which often lead to poor outcome. The establishment of medical emergency teams in hospitals has been reported to improve patients' survival[3-4]. We report a case of a 41 year old man with alcoholic cardiomyopathy complicated by QTc prolongation and polymorphic ventricular tachycardia, who was successfully managed by electrical defibrillation. Factors that may affect patients' survival adversely are discussed.

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Case

Mr OE, a 41 year old "beer seller" was admitted to the medical ward of the Obafemi Awolowo University Teaching Hospital Complex Ile-Ife on the 15th of October, 2003 on account of two week history of progressive effort intolerance, cough productive of whitish sputum, paroxysmal nocturnal dyspnoea, orthopnoea and leg swelling. He was not a known hypertensive or diabetic but he took about 240 grams of ethanol per day for 15 years. He did not smoke and was married with 4 children.

Physical examination revealed a young man, who was dyspnoeic at rest. He was not pale, anicteric, acyanosed, afebrile but had bilateral pitting pedal edema. Cardiovascular examination showed pulse rate of 105 beats per minutes, regular but with occasional missed beats and the blood pressure was120/80 mmHg. The jugular venous pressure was raised to the angle of the jaw. The apex beat was located at the 6th intercostal space, anterior axillary line. Auscultation

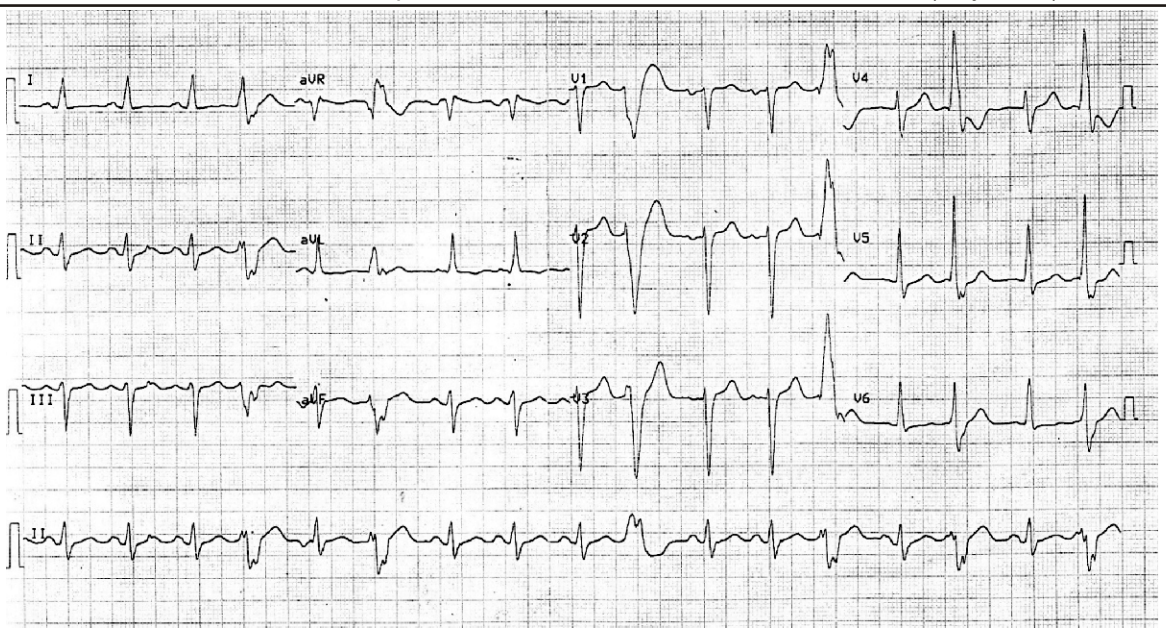
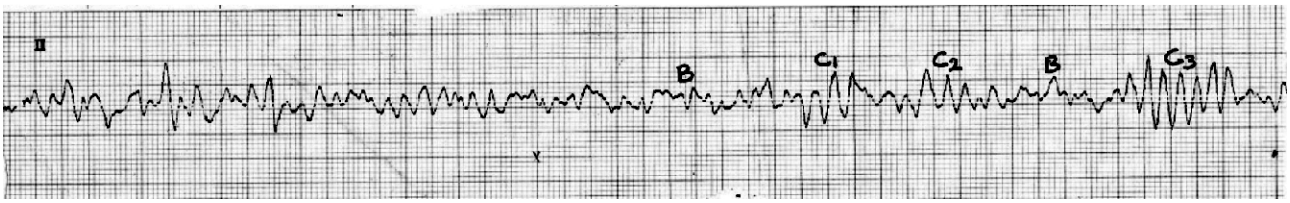


Figure 1: Shows baseline ECG of the patient which revealed LAE, LVH, QTc of 0.48 and frequent PVC's.

2a



2b

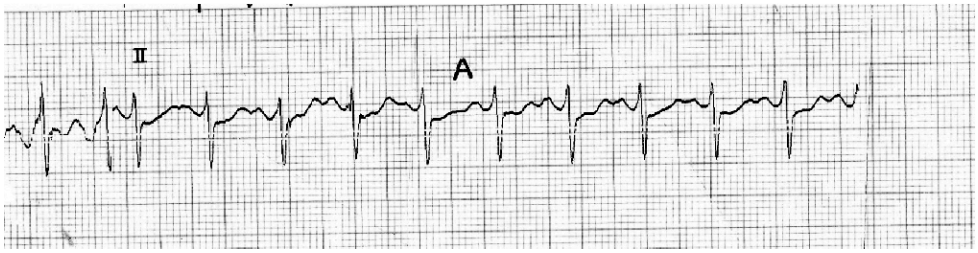


Figure 2: Upper panel (2A) is the first monitored rhythm following collapse showing *torsades de pointes* and paroxysmal ventricular fibrillation and lower panel (2B) is the rhythm strip after electrical defibrillation.

revealed S3 gallop rhythm with a grade 3 mitral regurgitant murmur. He had a tender hepatomegaly with a span of 16 cm and bilateral basal pulmonary rales.

A clinical assessment of alcoholic cardiomyopathy was made and he was commenced on intra-venous frusemide, 40mg 8hourly, oral lisinopril, 2.5mg daily, oral slow K, 600mg daily and low dose aspirin, 75mg daily. Electrolyte, urea and creatinine, full blood count, electrocardiography (ECG) and echocardiography (Echo) were carried out as initial investigations in the patient.

ECG (Figure 1) showed left atrial enlargement, left ventricular hypertrophy, QTc prolongation (QTc=0.48) and premature ventricular contractions (PVCs). The echocardiography revealed dilated heart chambers with poor systolic function (ejection fraction=24%) and restrictive pattern (grade 3) of diastolic dysfunction. There was no intra-mural thrombus seen. The serum level of sodium was 139mmol/L, potassium was 4.2mmol/L, urea was 9.6mmol/L and creatinine was 108µmol/L. The full blood count was within normal limit

He made satisfactory progress until the 5th day of hospital admission when he suddenly collapsed with loss of consciousness. Examination showed absent peripheral pulses and the blood pressure was un-recordable. The first monitored rhythm using defibrillator revealed torsade de pointes with paroxysmal ventricular fibrillation (figure 2A). He was urgently resuscitated and defibrillated using 100J x 2. Post defibrillation electrocardiography (figure 2B) showed conversion of the arrhythmia to supra-ventricular tachycardia. The heart beat returned within 3 minutes of defibrillation and he regained

consciousness in 5 minutes. He subsequently did well on treatment and was discharged on 10th admission day and was followedup in medical out-patient department. He had no residual neurological deficit.

Discussion

Our patient had a witnessed cardiac arrest due to polymorphic ventricular tachycardia (*torsade de pointes, figure 2A*) and was defibrillated urgently within 5 minutes of ictus. This really contributed significantly to this patient's survival and good neurological outcome[5]. Some factors that may delay defibrillation in patients with cardiac arrests include lack of cardiac monitors in medical wards, lack of defibrillators, inexperience of medical and nursing staff in the use of defibrillators, and lack of or slow response by the medical emergency teams. All these, contribute to high mortality in such patients. Ventricular arrhythmias are common in patients with cardiac diseases and are often associated with sudden cardiac death[6-7]. Cardiac arrest due to VT or VF (shockable rhythm) is associated with favorable outcome as compared with pulseless electrical activity (PEA) or asystole[1]. Markers of arrhythmic risk are closely linked to the level of ventricular function and their presence may indicate the presence of advanced disease[8-10]. QTc prolongation is a risk factor for the development of early after-depolarizations and *torsade de pointes*. The patient presented had a pre- existing QTc prolongation mostly likely from long standing left ventricular disease[11]. Thiamin deficiency is also common in alcoholics and may contribute to QT interval lengthening in our patient[12]. Other factors that may cause QTc prolongation include electrolyte abnormalities such as hypokalaemia, hypocalcaemia and hypomagnesaemia, and drugs such as quinidine, amiodarone, probuccol, clarithromycin, ketoconazole and haloperidol[14]. This patient had normal serum electrolytes and was not on drugs that increase QT interval.

Reduction of QT interval in patients with QTc prolongation has been found to be associated with reduced risk of mortality especially in patients with chronic heart failure (CHF). Long-term administration of angiotensin converting enzyme (ACE) inhibitors is associated with reduction in QT interval in patients with QTc prolongation [6,14-15]. There are indications that this may be one of the possible mechanisms of the beneficial effects of treatment with ACE inhibitors. Modest elevation of serum potassium in patients with CHF may also normalize potentially arrhythmogenic QT abnormalities. Choy et al[16] had demonstrated normalization of acquired QT prolongation following infusion with potassium of 12 healthy subjects treated with quinidine sulfate, placebo and 8 patients with CHF. This may have survival benefit.

In conclusion, regular screening for QT abnormalities, correction of electrolyte derangement and early treatment with ACE inhibitors may reduce the risk of development of *torsade de pointes* and cardiac arrest in patients with QTc prolongation. Careful assessment of QT interval is also very important before drugs that lengthen QT interval are prescribed to patients with left ventricular disease. Provision of cardiac monitors, defibrillators, staff training in emergency care and establishment of medical emergency teams will reduce mortality following cardiac arrest in medical wards.

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