Effective Cardiopulmonary Resuscitation - How Long Is Not Long Enough?

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A 45 year old 75 kg diabetic male, chronic tobacco chewer, was scheduled for wide local resection along with radical neck dissection for carcinoma lip. His diabetic status was controlled on oral hypoglycemic agents but he was shifted to plain insulin (22U/day) one week before surgery. His preoperative investigations were all within normal limits (Hb-9.6gm%, TLC-8800/cu.mm, platelets-110000/cu.mm, Na/K-141/4.1 meq/l, blood urea-35mg%, s.creatinine-1mg%, s.bilirubin-1.1mg%, ALT/AST/ALP-19/28/48 IU/l, total proteins-6.8gm%, blood sugar fasting-108mg%, post prandial 146mg%, HbAIc - 6.3gm%). His chest X ray and ECG did not reveal any abnormality. Airway examination revealed mouth opening to be less than one finger breadth. Neck movements and thyromental distance were within normal limits. Rest of the systemic examination was normal.

An awake fibreoptic intubation was performed and the trachea was intubated nasally. Intraoperatively Alberti's regime was used to control the blood glucose levels. The intraoperative period was uneventful. Blood and fluids were adequately replaced. The surgery lasted for eight hours and the patient was shifted to the intensive care unit (ICU) for elective ventilation postoperatively. The patient was put on antibiotics and steroids in the ICU.

On postoperative day three it was decided to extubate the trachea of the patient with an ENT team on the stand by for an emergency tracheostomy. Prior to extubation, additional steroids, bronchodilators and anti-aspiration prophylaxis were administered along with reversal agents. Within 1-2 minutes, patient went into acute stridor with falling oxygen saturation (81%). He did not respond to adrenaline nebulisation (for airway edema) and oxygen supplementation. Bag and mask proved to be ineffective due to a grossly distorted facial anatomy and an element of laryngospasm (SpO2-64%). His ABG showed acute respiratory acidosis with hypoxemia. It was decided to reintubate his trachea. Direct laryngoscopy could not be performed at this time due to a limited mouth opening and suturing of the buccal mucosa with the mucosa on the undersurface of the tongue. Since fibreoptic bronchoscope was not available in our ICU for intubation, it was decided to do an emergency tracheostomy.

While tracheostomy was being done, the patient went into pulseless ventricular tachycardia (VT). Cardiopulmonary resuscitation (CPR) was immediately started and he was defibrillated with 200 J energy (biphasic). This was followed by 5 cycles of CPR. The rhythm still showed VT, CPR was continued and a second biphasic shock of 200 J (biphasic) was delivered followed by immediate resumption of CPR. Epinephrine 1 mg was also administered intravenously (IV). Rhythm check again showed VT and a third shock of 200 J was delivered. Still the rhythm was VT, CPR was continued. Since the patient had developed shock resistant VT, lidocaine 1.5mg/kg was administered IV as per the AHA protocol as amiodarone was not immediately available to us in the ICU. When the VT did not revert, repeated cycles of CPR and shocks along with lidocaine (three doses) were given. By the time amiodarone became available, we had given 22 shocks to the patient. Since the rhythm had still not reverted, a 300 mg bolus of amiodarone was given IV followed by resumption of CPR. This was followed by a second dose of amiodarone 150 mg IV and 2 more cycles of CPR plus defibrillation when the rhythm finally reverted back to normal sinus rhythm with palpable pulses, after approximately 55 minutes of CPR. An amiodarone infusion was subsequently started at the rate of $1mg^{-1}$minute for the next 6 hours, reduced to $0.5mg^{-1}$minute for the subsequent 18 hours. His pupils, at this time were fixed and dilated. He was given neuromuscular blocking agent, sedation, inotropic support and ventilated with 100% oxygen. Mannitol and phenytoin were given for cerebral protection.[1,2] He was cooled to a target temperature of 34°C for 12 hours using ice packs, gastric and bladder lavage with cold saline and convective heat loss through alcohol evaporation.[3] A repeat ABG showed uncompensated metabolic acidosis (pH- 6.9, bicarbonate - 12 meq l^{-1}), which was corrected with administration of sodium bicarbonate. The blood sugar increased to 500 mg% and was controlled with intravenous insulin bolus.

Unfortunately after two hours, the patient again went into pulseless VT. CPR was immediately started and repeated cycles of CPR and shocks were delivered along with injection adrenaline 1 mg as per the AHA protocol. A

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repeat bolus of amiodarone 300 mg was also administered IV as the patient had developed a pulseless VT with the amiodarone infusion running at 1mg/minute. He was defibrillated with 200 J (biphasic) six times before his ECG reverted back to normal sinus rhythm. His pupils, at this time, were fixed and dilated, though over the next two hours they started showing response to a light stimulus.

Over the next 24 hours we were able to decrease his ventilatory and inotropic support enabling gradual weaning from the ventilator. The patient was rewarmed passively. Over the next 48 hours, the patient steadily improved, became fully conscious, was following verbal commands, was off inotropic support and breathing spontaneously and adequately. Finally, 3 days after his cardiac arrest and shock resistant pulseless VT, the patient had recovered completely without any evidence of neurological deficit. Further follow ups have also not revealed any fresh episode of arrhythmia or any neurological sequelae.

DISCUSSION

Prolonged pulseless VT is generally not associated with a good outcome. The mortality in these cases is very high and the patients who survive are left with neurological sequelae. Case reports of complete neurological recovery after prolonged cardiac arrest are few and far in between. Our patient survived a prolonged pulseless shock resistant VT with complete neurological recovery. This is remarkable since there was a long delay before the return of spontaneous circulation (ROSC); and the severity of lactic acidosis (6.1meq/l), hemodynamic instability and initial coma score suggested a poor prognosis.

Shock resistant ventricular tachycardia is defined as VT which is resistant to 3 shocks from an external defibrillator, at least one dose of IV epinephrine and a fourth defibrillator shock.[4] Based on results from the Amiodarone for Resuscitation after Out-of-Hospital Cardiac Arrest due to Ventricular Fibrillation (ARREST) study[5], intravenous amiodarone has replaced lidocaine as the preferred antidysrrhythmic for the treatment of pulseless VT/VF in the 2000 guidelines.[7] It is recommended that amiodarone should be given as a bolus injection of 300 mg. A further dose of 150 mg can be given for recurrent or refractory VF/VT followed by an infusion of 900 mg over 24 hours.[7] Lidocaine 1-1.5mg/kg may be used as an alternative, but only if amiodarone is not available.

Recently a new drug, nifekalant hydrochloride is being used for treating resistant VF/VT. Takenaka et al reported that nifekalant is effective in treating malignant ventricular tachyarrhythmia following anterior wall myocardial infarction with severe ventricular dysfunction that is refractory to class Ia and Ib antiarrhythmic agents.[8] Igawa et al also showed that nifekalant could be an alternative drug for suppression of VT.[9] In another study, nifekalant was found to be more effective than lidocaine for termination of arrhythmia and for return of spontaneous circulation in patients with shock-resistant in-hospital VF or VT.[10]

Several studies have shown that moderate therapeutic hypothermia to a temperature of 32 to 33°C can reduce brain damage after cardiac arrest due to ventricular arrhythmia without significant side effects. The International Liaison Committee on Resuscitation (ILCOR) and the American Heart Association published an interim scientific statement with recommendations on the use of therapeutic hypothermia in comatose survivors of cardiac arrest.[3] This was followed, in 2005, by the American Heart Association Guidelines for CPR and Emergency Cardiovascular Care, which included the following treatment recommendations[11]: unconscious adult patients resuscitated after out of hospital cardiac arrest should be cooled to 32ºC to 34ºC (89.6ºF-93.2ºF) for 12 to 24 hours when the initial rhythm was ventricular fibrillation (Class IIa). Similar therapy may be beneficial for patients with in-hospital cardiac arrest or out-of-hospital arrest associated with an initial rhythm other than ventricular fibrillation (Class IIb). Based on these recommendations, we induced mild hypothermia in our patient after ROSC.

On reviewing literature we could find only one case report of complete neurological recovery after prolonged cardiac arrest due to pulseless VT. A 62 year old male patient who was a known case of coronary artery disease developed 15 episodes of sustained pulseless VT which were treated by electric shocks and continuous amiodarone infusion. Subsequently the patient developed another 90 episodes of pulseless VT which were resistant to deep sedation, continuous metoprolol and repeated lidocaine infusions and were treated by defibrillations.[12] Braque et al have reported a case of excellent neurological recovery after prolonged asystole requiring CPR for 90 minutes.[13] The patient had cardiac arrest after accidental intravascular injection of bupivacaine and lidocaine during lumbar plexus block. In another case report, a patient who was an intravenous drug abuser developed a cardiac arrest due to possible release of potassium and metabolic toxins from an acutely ischemic lower limb. He survived with excellent neurological recovery despite a total arrest time of 85 minutes.[14]

Duration of CPR that results in futility of care is unknown. Our patient goes to prove that effective CPR is the key to complete neurological recovery after prolonged cardiac arrest. We recommend early initiation and effective resuscitation along with defibrillation for in-hospital cardiac arrest in academic medical centers.

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We confirm that written informed consent for publication has been obtained from the patient described.

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