

# A Case Report of Successful Resuscitation of Prolonged Cardiac Arrest Due To Severe Hyperkalaemia, Hypovolemia and Severe Metabolic Acidosis

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## ABSTRACT

*Hyperkalemia, Hypokalemia, Hypovolemia, Acidosis, hypoxia, Hypothermia, thrombosis-coronary and pulmonary, Cardiac tamponade, toxins and tension pneumothorax are the reversible causes of cardiac arrest. We report a case of successful resuscitation of cardiac arrest due to severe hyperkalemia, hypovolemia and severe metabolic acidosis in a patient of Diabetic Keto Acidosis. Our patient is unique for the fact that he had multiple long standing co morbidities, in prolonged cardiac arrest for about 45 minutes with non-shockable cardiac rhythm with multiple reversible causes such as hyperkalemia, hypovolemia and severe metabolic acidosis and was successfully resuscitated with the complete neurological recovery.*

*Consent for Publication: Obtained From the Patient*

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## CASE REPORT

A 46 years old Caucasian male patient was brought to the Accident and Emergency department with the GCS of 6/15, laboured breathing and decreased responsiveness. He is a known case of Type1 Diabetes mellitus with poor drug compliance, IgA nephropathy and Hypertension. He was unwell for few days and not taking food and his medications. While attended by the paramedics he looked very unwell, cold, pale, obvious kussmaul breathing was noted. The patient's vital signs were pulse rate 57/min, respiratory rate 25 to 30 / min, blood pressure was initially not recordable but after 500ml of IV normal saline one reading of 114/73 was recorded. Multiple ventricular premature beats on ECG tracing were noted during the transfer.

On arrival to A&E his GCS was 6/15, patent airways, respiratory rate was 16/ min, equal air entry on both sides, cold peripheries, central pulse was palpable but radial pulse was very feeble. Capillary refill time was more than 3 seconds. As soon as the patient arrived blood sample was taken for routine all blood investigations and Arterial Blood gas analysis.

While finish taking the blood samples he went into ventricular asystole. Cardio pulmonary resuscitation was started at once according to UK Resuscitation Council ALS Guidelines. His Arterial Blood Gas analysis showed pH < 6.8, K<sup>+</sup>10.1 mmol/ L, Lactate 4.2 mmol/L, PO<sub>2</sub> 15.7 kpa, PCO<sub>2</sub> 2.9 kpa, Na<sup>+</sup>115 mmol/L, Ca<sup>2+</sup>1.21 mmol/L, Base excess-Incalculable, HCO<sub>3</sub>Incalculable.

Hence the reversible causes of cardiac arrest such as severe acidosis, hyperkalemia and hypovolemia were considered to be treated after carefully excluding others. Two IV boluses of 50 mls of 50% dextrose with 10 units of actrapid, 20 mls of calcium chloride, 2 litres of ringer lactate solution intravenous bolus, 200mls of sodium bicarbonate 8.4% and 11 doses of 1mg of 1:1000 adrenaline boluses were given during the course of CPR which was continued about 45 minutes. After the 21 cycles of Ventricular Asystole patient's cardiac rhythm turned into Ventricular fibrillation. At the end of 22<sup>nd</sup> cycle of Cardio Pulmonary Resuscitation he was shocked with 200 Joules biphasic to ROSC. At the end of CPR his Arterial Blood Gas results were noted as pH 7.12, K<sup>+</sup>6.1 mmol/L, Lactate 6 mmol/L, PO<sub>2</sub> 35.4 kpa, PCO<sub>2</sub> 3.5 kpa, Na<sup>+</sup>123 kpa, Base excess -27.8 mmol/L, HCO<sub>3</sub> 4.6 mmol/L.

Post resuscitation care plan was to transfer to Intensive care unit, continue DKA protocol, aggressive fluid resuscitation, inotropic support, and ventilator support. His fluid deficit was corrected with the help of central venous pressure monitoring. During his earlier post resuscitation period he was polyuric which was treated with Desmopressin 1mcg to 2mcg IV boluses. He was extubated 48 hours later without any neurological deficit. He was treated

further according to DKA protocol, antibiotics. His further stay in the hospital was uneventful.

## DISCUSSION

Prolonged cardiac arrest is generally associated with poor outcome. Systematic and effective team approach in doing the CPR, early recognition and treatment of reversible causes of cardiac arrest are vital in achieving the best outcome in the event of cardiac arrest. Failure in delivering this will result in poor outcome in many ways such as systemic inflammatory response and distributive shock, myocardial infarction, adrenal insufficiency, hypoxic brain injury, renal failure, gastric ulceration, aspiration pneumonia, respiratory failure due to aspiration, ARDS and pulmonary oedema, post cardiac arrest ischaemic hepatitis, ischaemia of the gut and bacterial translocation and immune dysfunction and sepsis. Unwitnessed, out of hospital cardiac arrests with non-shockable cardiac rhythm (Asystole and PEA) has the worst out come when compared to the witnessed, in the hospital cardiac arrests with shockable rhythm (VF and pulseless VT).

## CONCLUSION

In conclusion, the extension of poor neurological recovery and other systemic effects depend largely on the delay of initial treatment, the efficiency of resuscitation, and the time elapsed between the point of cardiac arrest and return of spontaneous circulation (ROSC) and the quality of post resuscitation care. This reinforce the need for training and certification of all health care workers ranging from paramedics, nursing to doctors on Basic and advanced life support and periodic retraining. The hospitals should be equipped with a dedicated rapid response team to attend cardiac arrests, prearranged equipments like airway equipments, emergency drugs and provisions for quick and accurate laboratory services to avoid any delay in commencing and delivering effective cardio pulmonary resuscitation.

**Conflicts of Interest:** None

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