CASE REPORT

Rising From The Dead!

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SUMMARY

A report of a patient with Lazarus phenomenon (the return of spontaneous circulation after cardiopulmonary resuscitation) following cardiac arrest (myocardium ischemia) is presented. A 65 year patient was found unconscious at home. He taken to the emergency department On arrival he was unconscious, his pupils fixed and dilated bilaterally. Resuscitation proceeded for 55 minutes. He was then pronounced dead. Forty minutes later spontaneous breathing was noted and his blood pressure was 110/48 and heart rate 90bpm. He survived a further 13 days in the coronary care unit. The implications for management of cardiac arrest in the emergency and medical department are discussed.

KEY WORDS:
Lazarus phenomenon

INTRODUCTION

The Lazarus phenomenon is described as delayed return of spontaneous circulation (ROSC) after cessation of cardiopulmonary resuscitation (CPR). It is uncommon and was first described by Linko *et al* in 1982. So far 38 cases of delayed ROSC have been published in the medical literature. The term "Lazarus phenomenon" was coined by Bray[1] to describe this situation after the biblical character Lazarus who Jesus reputedly raised to life.

It is also probably under reported [1] due to the reluctance to report these cases because of concern regarding lack of complete documentation or monitoring of the event, its medicolegal ramifications and the fear of being criticized for negligence. Lack of a satisfying scientific explanation for the events and a doctor's disbelief of his or her own observations are added factors. It is undeniable that the Lazarus phenomenon is exists although there is lack of good scientific explanations. Nevertheless, several mechanisms have been postulated for this phenomenon.

CASE

A case of Lazarus phenomenon associated with myocardial infarction seen in the medical department is presented in Seberang Jaya Hospital.

A 65 year old, chronic male smoker was found unconscious at home at 11.00a.m. He had underlying hypertension. He was brought in by ambulance to the emergency department (ED). On arrival at ED, he was unconscious, his pupils fixed and dilated bilaterally. His blood pressure was unrecordable and pulse was not palpable.

Cardiopulmonary resuscitation (CPR) was commenced and he was intubated. Resuscitation proceeded for 55 minutes. During the CPR, he developed ventricular fibrillation (VF) and ventricular tachycardia (VT) which reverted with defibrillation. Adrenaline (total of IV 8 mg) and amiodarone (IV 300 mg push) were given intravenously (peripheral branula) during the CPR. Despite the resuscitation, the patient did not respond and ECG showed asystole. He was pronounced dead at 12.20 noon.

After cessation of the resuscitation, medications were discontinued. The monitors were turned off, and the ventilator support was disconnected. The patient was kept at the body holding area in ED. At 1.00 p.m, 40 min after being pronounced dead, the emergency team noted that the patient regained spontaneous breathing. The patient was immediately reintubated and the cardiac monitor was reattached. His blood pressure was 110/48 mm Hg (heart rate: 90 bpm). A 12 lead ECG showed sinus rhythm with deep Q wave over leads V1-V3. Cardiac enzymes were raised. The patient was reviewed by medical team and diagnosed to have acute anteroseptal myocardial infarction. He was admitted to coronary care unit (CCU) for medical therapy.

Post resuscitation, the patient was maintained on a mechanical ventilation for 13 days in the CCU. He was initially supported by inotropes which were discontinued on day 2. He developed acute kidney injury requiring renal replacement therapy. Patient's GCS level remained low (GCS – E1M1Vt) throughout the CCU stay. Serial CT brain imaging which was initially normal on Day 1 subsequently showed features of hypoxic ischaemic encephalopathy with bilateral basal ganglia haemorrhage. The patient deteriorated and was pronounced dead on Day 13 after admission.

DISCUSSION

The exact mechanism for Lazarus phenomenon remains unclear and it is possible that more than one mechanism is involved. One of the proposed mechanisms is dynamic hyperinflation of the lung during CPR causing increased positive end expiratory pressure (PEEP). This occurs most commonly in patients with known COPD or asthma, or after mechanical ventilation. CPR leads to gas trapping and an increase in the end-expiratory pressure leading to delayed venous return, low cardiac output and even cardiac arrest [3]. With cessation of CPR, the PEEP will be lowered and may contribute to ROSC.

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High dose adrenaline has also been thought to contribute to unexpected ROSC after cessation of CPR. Delayed action of drugs administered during CPR is another postulated mechanism for the Lazarus phenomenon. This is particularly true when medication is administered via a peripheral branula during CPR. Medication injected through a peripheral vein is inadequately delivered centrally due to impaired venous return, and when venous return improves after cessation of CPR, delivery of medications could contribute to the phenomenon.

Following myocardial ischemia, prolonged myocardial dysfunction can occur up to several hours before normal function returns. Asystole or PEA (Pulseless Electric Activity) following counter-shock of prolonged VF is common and occurs in around 60% of patients [2]. However, the prognosis normally is poor: only 0–3% are discharged alive. It is possible that asystole or PEA after countershock is transient before a perfusable rhythm restores circulation. However, transient asystole would not explain delayed ROSC in the majority of patients, including our patient similarly is this patient as the duration of asystole was prolonged much longer. (40 minutes for this patient)

There are few cases of Lazarus phenomenon occurring in the presence of hyperkalaemia. It is explained by the fact that hyperkalaemia prevents adequate efflux of potassium that is necessary for repolarization of myocardium. This prevents myocardium to be able to respond to the next action potential. As a result, hyperkalaemia will result in a diastolic depolarized electromechanical myocardial arrest as the cardiac myocytes are refractory to further stimulation. However, this was unlikely for this patient as the initial potassium was 3.0 mmol/dL.

In conclusion, we present a case of spontaneous recovery after failed CPR. Despite the available literature that suggest observing patients for 10 minutes post cessation of resuscitation [3], this appeared to be inadequate in this patient as he developed ROSC after 40 minutes. Therefore, we suggest that the duration of observation post-CPR should be prolonged. More importantly, all patients with Lazarus phenomenon should be reported so that more data can be collected for better understanding of the phenomenon and for assisting in further management.

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