CASE REPORT

The Lazarus phenomenon: spontaneous cardioversion after termination of resuscitation in a Scottish hospital

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SUMMARY

A 66-year-old man suffered a ventricular fibrillation cardiac arrest after an episode of chest pain. Despite advanced life support, his condition deteriorated; the situation was considered irrecoverable and resuscitation was discontinued. The patient was observed for about 5 min with irregular agonal gasping respirations evident but no palpable pulse. A gradual increase in the regularity of breathing pattern heralded the return of spontaneous circulation. An inferior myocardial infarction was diagnosed. Primary percutaneous coronary intervention was performed. The patient was discharged from hospital after 9 days with no neurological sequelae. There are several possible mechanisms to explain the Lazarus phenomenon including auto-positive end expiratory pressure and delayed drug actions, but cases are rarely reported in the medical literature. After discontinued resuscitation, we suggest consideration should be given to a period of continued monitoring. However, evidence is limited and prospective studies would be welcome to allow firmer recommendations.

BACKGROUND

The Lazarus phenomenon describes the spontaneous return of circulation (or auto-cardioversion) subsequent to the termination of resuscitation efforts in a patient suffering from cardiac arrest. It was first described in 1982,¹ received its eponymous title in 1993² and, as the name suggests, it is a rare occurrence.

Case presentation

A generally fit 66-year-old man with a history of well-controlled hypertension and type 2 diabetes collapsed with chest pain. Paramedics who arrived at the scene 5 min later confirmed cardiac arrest and a primary rhythm of ventricular fibrillation (VF). Cardiopulmonary resuscitation (CPR) was commenced, biphasic direct current (DC) shocks were delivered and adrenaline was administered as per advanced life support (ALS) guidance. Prehospital insertion of a laryngeal mask airway (LMA) was done to facilitate ventilation. Despite a brief return of spontaneous circulation (ROSC) during transfer, the patient had remained in refractory VF and continued to undergo full resuscitation measures as he arrived at the emergency department (ED), 18 min after arrest.

Differential diagnosis

A working diagnosis of cardiac arrest due to myocardial infarction was made.

Treatment

Endotracheal intubation was performed; initial end-tidal CO₂ was 2.2 kPa. Resuscitation continued as per ALS guidance (including amiodarone, magnesium and stacked DC shocks), but the patient remained in refractory VF. At a point 45 min postarrest, although the patient remained in VF, his condition was clinically clearly deteriorating (pH 6.96 and lactate 16 mmol/L) with no evidence of response to treatment. A collective team decision (including interventional cardiology) was made to discontinue resuscitation. The endotracheal tube was removed as it appeared distressing to the relatives, and the patient developed pulseless electrical activity before monitoring was disconnected.

At this stage, there were agonal brain stem gasps at a rate of about two to three per minute, but there was no pulse or other signs of ROSC, and it looked clear that death would be imminent. This situation remained unchanged over the next 5 min after which the respiratory rate surprisingly began to rise. Shortly after a carotid pulse was palpable and within minutes there was a rate of about 100 beats per minute with regular runs of ventricular tachycardia, a recordable blood pressure and 12-lead ECG was consistent with an inferior ST elevation myocardial infarction.

The cardiology team was recalled, a rapid sequence induction was performed, the patient was reintubated and transferred to the Catheter Laboratory. Primary percutaneous coronary intervention (PPCI) performed via the right radial artery confirmed proximal occlusion of the right coronary artery which was treated with three drug eluting stents.

Post-PPCI the patient was taken to the intensive care unit for systemic cooling and further management.

Outcome and follow-up

The patient improved quickly, allowing extubation the following day and proceeded to discharge from hospital 9 days after admission. At follow-up, he had made a full neurological recovery, played golf regularly and had married his long-term partner.

DISCUSSION

Since the first description of the Lazarus phenomenon, case reports and letters have appeared sporadically in

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the literature across a large age range,^{3–6} although only three reports describe this phenomenon occurring in the context of refractory VF.^{7–9} Despite its recognition as an entity, under-reporting is believed to exist due to physician embarrassment, surprise and fear of the legal ramifications.¹⁰ However, there is evidence that the reporting of such events is on the increase, including in the UK,¹¹ attributed to improving recognition and greater transparency.⁵ A systematic review in 2014 found that 63% of patients with the Lazarus phenomenon died prior to discharge.⁵ Of the three cases identified with VF, two were discharged with neurological sequelae and the other died 3 months postdischarge.^{7–9} In contrast, the patient described here was discharged with full neurological recovery and went on to make an excellent physical recovery. We were unable to clearly identify a reason for this difference, but the prompt commencement of CPR may be important.⁸

Since the number of cases reported is small, there is a paucity of data to review and explanations of aetiology are very much theoretical. Some have suggested that during resuscitation increased positive end-expiratory pressure (PEEP) may develop in the airways due to inadequate time given for complete exhalation.^{4 12} That patients are often overventilated during CPR, even if delivered by experienced personnel, is well recognised,¹³ leading to increased intrathoracic pressure, decreased venous return and subsequently reduced cardiac output.^{5 12} Our patient was managed as per ALS guidelines from the time he was attended to by paramedics and then by experienced ED staff. Ventilation was performed prehospital via an LMA and this was converted to an endotracheal tube shortly after arrival in the ED. The pCO₂ on the first venous sample obtained in hospital was 3.43 kPa, which may represent hyperventilation (including auto-PEEP) but could also be influenced by reduced cardiac output.

Others have postulated that an auto-PEEP-induced reduction in cardiac output may also delay the effects of resuscitation medications,^{4 14} allowing adrenaline to accumulate in the circulation in high doses that reach the heart if auto-PEEP is reduced (when resuscitation ceases), resulting in beneficial effects on cardiac physiology.⁴ Given our patient's prolonged resuscitation (with multiple doses of adrenaline), it is theoretically possible that such a phenomenon played a part in his auto-cardioversion. Conversely, since his final dose of adrenaline was given 13 min prior to ROSC, the reduced influence of this drug on the pulmonary artery and right heart function may have been beneficial.¹⁵

Lastly, we are aware that increasingly there are facilities to take selected patients in VF arrest to the Catheter Laboratory, often using mechanical CPR devices. Such a pathway did not exist in our institution at the time.

Learning points

- ▶ The Lazarus phenomenon or auto-cardioversion does exist and emergency physicians should be aware of it.
- ▶ After discontinued resuscitation attempts, consideration should be given to a period of continued monitoring.
- ▶ However, evidence is limited and prospective studies would be welcome to allow firmer recommendations.
- ▶ Perhaps, hyperventilation should be the fifth 'H' in the list of potentially reversible causes of cardiac arrest.

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