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Defying Death: A Case Report of the Lazarus Phenomenon

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1. Introduction

The Lazarus Phenomenon, also referred to as autoresuscitation, describes the spontaneous return of circulation (ROSC) after cardiopulmonary resuscitation (CPR) has ceased. First documented in the medical literature [1], the term "Lazarus" was coined by Bray in 1993 [2], drawing inspiration from the biblical story of Lazarus's resurrection [3,4]. Although traditionally considered rare, the Lazarus Phenomenon may be underreported, with estimates suggesting that 37–50% of emergency physicians have encountered it in both out-of-hospital (OHCA) and in-hospital cardiac arrest (IHCA) settings [5].Despite advancements in CPR techniques and monitoring technologies, the exact mechanisms behind the Lazarus Phenomenon remain elusive. Several theories have been proposed, such as delayed effects of resuscitation drugs, lung hyperinflation, hyperkalemia, and hypoperfusion, though none provide a conclusive explanation [6,7]. This unpredictability raises critical concerns regarding medical-legal implications and the challenges it poses to existing death-determination protocols [8].A thorough understanding of autoresuscitation is vital for healthcare professionals as it impacts clinical practice and challenges established guidelines for resuscitation efforts and the determination of death. This report presents a case of a patient who exhibited signs of life an hour after being declared dead, with a focus on contributing factors such as hyperkalemia and hypothermia.

2. Case Report

A 70-year-old woman with a history of ischemic stroke, without residual effects, was admitted to the intensive care unit (ICU) on May 11th after suffering thermal burns to 65% of her total body surface area (TBSA) following a gas cylinder explosion. The distribution of burns included:

- Face: 1st-degree burns across the entire surface.
- Upper Limbs: Superficial and deep 2nd-degree burns in a mosaic pattern on both arms.
- Back: Deep 2nd-degree and 3rd-degree burns, also in a mosaic pattern.
- Perineum: 3rd-degree burns.
- Lower Limbs: Deep 2nd-degree and 3rd-degree burns, primarily affecting the thighs and posterolateral legs.

Upon admission, the patient was alert and oriented with a Glasgow Coma Scale (GCS) score of 15. However, her initial vital signs indicated systemic distress:

- Heart Rate: 120 bpm (tachycardia)
- Blood Pressure: 90/50 mmHg (hypotension)
- Respiratory Rate: 36 breaths per minute (tachypnea) with SpO_2 at 97% on nasal cannula
- Coarse breath sounds bilaterally

3. Initial Management

The patient was stabilized using fluid resuscitation based on the Parkland formula. Central venous access was established via a right internal jugular central line, and an arterial line was placed for continuous blood pressure monitoring. A Foley catheter was also inserted to monitor urine output. Despite aggressive fluid management, the patient remained hypotensive, prompting the initiation of norepinephrine infusion at 0.4 µg/kg/min to maintain blood pressure. Additionally, albumin was administered to optimize intravascular volume expansion and improve hemodynamic stability.

4. Operative Course

On May 13th at 21:30, the patient was scheduled for wound debridement and dressing in the operating room (OR). However, logistical delays left her waiting in the corridor for over an hour, likely contributing to significant hypothermia. The 45-minute procedure involved cleansing the burns with saline and soap, followed by the application of Flamazine on the limbs and trunk, and MEBO on the face. The patient was returned to the ICU at 23:21.

5. ICU Course and Asystole

On the night of May 13th, the patient's condition deteriorated significantly, marked by bradycardia and an increased norepinephrine requirement of 0.8 µg/kg/min. Her blood pressure dropped to a critical level of 72/40 mmHg, and she subsequently suffered a cardiac arrest characterized by asystole.Advanced Life Support (ALS) protocols were immediately initiated. High-quality chest compressions were performed, the airway was secured with manual ventilation, and resuscitative medications were administered, including 10 mg of epinephrine. Despite these efforts, the patient alternated between pulseless electrical activity (PEA) and asystole, ultimately remaining in asystole. To confirm the absence of cardiac output, the team double-checked the cardiac monitor leads and performed a bedside ultrasound, which confirmed the absence of any cardiac activity. During resuscitation, an arterial blood gas (ABG) analysis revealed a critically deranged metabolic state. Findings included severe hyperkalemia (potassium levels >7 mEq/L), profound metabolic acidosis (pH 6.8), and significantly elevated lactate levels, indicative of systemic hypoperfusion and cellular hypoxia. Recognizing hyperkalemia as a reversible cause of cardiac arrest, the team promptly administered calcium gluconate to stabilize the myocardial membrane, insulin with glucose to drive potassium intracellularly, and a diuretic (Lasix) to enhance potassium excretion. Sodium bicarbonate was also administered to correct the severe metabolic acidosis and further assist in driving potassium intracellularly.Despite aggressive adherence to ALS protocols and targeted therapeutic interventions, the patient's condition did not improve, and circulation could not be restored. After 30 minutes of CPR, the patient was pronounced dead at 00:17.

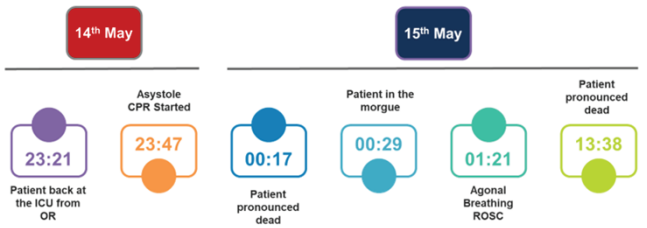
6. The Lazarus Phenomenon

Approximately one hour after being declared dead, at 01:21, the patient, now in the morgue, exhibited slow agonal breathing and

Laboratory Findings

The following table presents the patient's laboratory results upon admission and on the day of her cardiac arrest:

Test	Admission (May 11th)	Day of Arrest (May 13th)
Hemoglobin (HGB)	14.7 g/dL	12.6 g/dL
White Blood Cells (WBC)	35.4 x 10^9/L	28.2 x 10^9/L
Platelet Count (PLT)	355 x 10^3/L	250 x 10^3/L
Sodium (Na)	145 mEq/L	151 mEq/L
Potassium (K)	3.6 mEq/L	6.1 mEq/L
BUN	0.55 g/L	0.87 g/L
Creatinine (Creat)	12.3 mg/dL	12.1 mg/dL
Creatine Kinase	111 U/L	204 U/L
C-ReactiveProtein (CRP)	24.6 mg/L	
Chloride (Cl)	108 mEq/L	129 mEq/L
Alkaline Reserve	-	11
Lactate Dehydrogenase (LDH)	-	1612 U/L



Timeline

spontaneous cardiac activity (HR: 52 bpm, BP: 68/41 mmHg). She was readmitted to the ICU, intubated, and placed on mechanical ventilation. Despite ongoing vasopressor support, she remained in refractory shock and suffered a second cardiac arrest 12 hours later, leading to her final demise. No formal brain death assessment was conducted after her ICU readmission.

7. Discussion

The Lazarus Phenomenon, defined as the spontaneous return of circulation after cessation of cardiopulmonary resuscitation (CPR), is increasingly seen as underreported rather than rare4. Delayed effects of interventions, such as drug administration and metabolic corrections, often linked to factors like hypoperfusion or delayed drug metabolism, are thought to contribute to its occurrence [3,6,7]. In this case, hyperkalemia played a key role. Elevated potassium, a well-known cause of cardiac arrest, can decrease myocardial excitability and cause asystole9,10. Despite treatment, delayed action due to poor perfusion may explain the late return of spontaneous circulation (ROSC). Hypothermia, which reduces metabolic demands and prolongs tissue viability, was also significant. The patient's hypothermic state, exacerbated by delays and rewarming, likely contributed to cardiac arrest and ROSC [7,9,11]. Moreover, mild hypothermia has been shown to improve defibrillation outcomes in ventricular fibrillation [12].Other possible contributors include auto-PEEP resolution, delayed drug efficacy, and spontaneous coronary perfusion recovery [5,9]. These factors, individually or combined, may explain delayed ROSC in complex cases like this. Hornby et al. [13]. Documented 32 cases of autoresuscitation, with ROSC occurring between seconds and 33 minutes post-CPR [3,9]. However the longest period before ROSC reported in the literature was 180min13. In our case, ROSC occurred after nearly an hour, likely influenced by hyperkalemia and hypothermia. While more common in non-traumatic arrests, autoresuscitation can also occur following trauma, underscoring the importance of remaining vigilant in all cardiac arrest cases [14].Delayed ROSC has significant medico-legal and ethical implications, especially if a patient declared dead later shows signs of life or sustains neurological deficits [9]. The 2021 ERC guidelines recommend terminating resuscitation after 20 minutes of asystole without reversible causes, but factors like hyperkalemia and hypothermia may necessitate extending this timeframe [15].The pathophysiology of the Lazarus Phenomenon is not well understood. Proposed mechanisms include myocardial recovery after temporary dysfunction and the resolution of arrhythmias post-resuscitation efforts6. Hyperkalemia may cause temporary myocardial refractoriness, leading to a delayed response to resuscitation [16].The emotional and ethical challenges posed by the Lazarus Phenomenon are significant [17]. For healthcare providers, witnessing a return of signs of life after declaring a patient dead can lead to self-doubt and professional anxiety, particularly in high-stress environments like emergency or critical care departments. Such events may cause clinicians to question the accuracy of their clinical decisions. Additionally, for families who have been informed of a patient's death, the resurgence of life can provoke shock, confusion, and mistrust toward the medical team18. In some instances, they may seek clarification or question whether every possible effort was made, complicating the delicate nature of end-of-life care12,19. These scenarios also raise broader ethical issues about resuscitation protocols, the timing of death pronouncements, and the need for transparent communication during such critical moments [6,17]. Based on observations from Linko et al. [18], it is recommended that patients remain under close clinical observation with continuous ECG monitoring for at least 10 minutes after CPR has been stopped [1,9]. This window is critical as it coincides with the period during which the Lazarus Phenomenon is most likely to occur [20]. Vigilant monitoring during this time helps ensure that any delayed return of spontaneous circulation is promptly recognized and managed accordingly.

8. Conclusion

The Lazarus Phenomenon, though rare, highlights the need for a deeper understanding of factors that delay ROSC, particularly in cases involving hyperkalemia and hypothermia. It raises important ethical and scientific questions about protocols for terminating resuscitation efforts and the declaration of death. Thorough documentation and continued study of such cases are essential to developing evidence-based guidelines for CPR termination and improving the determination of death [21].

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