

Favorable Neurological Outcome Following Cardiac Arrest in the Emergency Department: A Three-Patient Case Series

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ABSTRACT

Introduction:

Cardiac arrest with an initial non-shockable rhythm (pulseless electrical activity [PEA] or asystole) carries a substantially lower rate of neurologically favorable survival than shockable rhythms, and asystole in particular carries a worse prognosis than PEA.

Case presentation:

We report three adult patients who developed cardiac arrest in the emergency department, each with an initial non-shockable rhythm, who achieved return of spontaneous circulation (ROSC) and survived to hospital discharge with a favorable Cerebral Performance Category (CPC 1–2). Case 1 presented already in PEA arrest; cardiopulmonary resuscitation (CPR) was continued for 20 minutes before ROSC, followed by a 10-day critical care admission and discharge with CPC 1. Case 2, a 70-year-old man, presented with severe abdominal pain and profound shock (pH 7.01, bicarbonate 3.5 mmol/L, lactate 14.1 mmol/L), was treated with fluid resuscitation, noradrenaline, and sodium bicarbonate, then developed asystolic arrest; ROSC was achieved after 10 minutes of CPR, and he was discharged after 18 days of critical care with CPC 2. Case 3, a 28-year-old woman with diabetic ketoacidosis, developed PEA arrest during treatment; ROSC was achieved after approximately 30 minutes of CPR, and she was discharged after 6 days with CPC 2.

Conclusion:

These cases demonstrate that favorable neurological survival is possible after emergency department cardiac arrest with an initially non-shockable rhythm, including after prolonged resuscitation, when high-quality CPR, systematic evaluation of reversible causes, and appropriate post-arrest care are provided.

Keywords: Cardiac arrest; Cardiopulmonary resuscitation; Emergency department; Return of spontaneous circulation; Pulseless electrical activity; Asystole; Neurological outcome; Cerebral Performance Category; Utstein style.

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Introduction

Cardiac arrest is among the most time-critical presentations encountered in the emergency department (ED), and outcome depends on rapid recognition, immediate high-quality cardiopulmonary resuscitation (CPR), rhythm-appropriate intervention, systematic identification and correction of reversible causes, and structured post-cardiac-arrest care.[1] Return of spontaneous circulation (ROSC) is the immediate resuscitation goal, but survival with meaningful neurological recovery is the outcome that matters to patients and is now an explicit component of post-arrest care guidance.[2][3] Neurological status is conventionally graded using the Cerebral Performance Category (CPC) scale, a 5-point scale first described by Safar[4] and operationalised in the Brain Resuscitation

Clinical Trial,[5] where CPC 1–2 (good function or moderate disability with independence) is considered a favorable outcome.

Non-shockable initial rhythms — pulseless electrical activity (PEA) and asystole — present a particular prognostic and management challenge, since defibrillation has no role unless the rhythm subsequently becomes shockable, and outcome instead depends on high-quality compressions, appropriately timed epinephrine, and correction of the underlying cause.[6] The two rhythms are not prognostically equivalent: in a national Japanese registry of over 200,000 out-of-hospital arrests, neurologically favorable survival was 1.4% with PEA versus 0.2% with asystole (adjusted odds ratio for PEA

7.86, 95% CI 6.81–9.07).[7] Comparable large-scale in-hospital data specific to the ED are less abundant, which increases the value of well-documented ED case material. We report three adult patients who developed cardiac arrest in the ED, each with an initial non-shockable rhythm and each associated with a distinct precipitating clinical picture, who achieved ROSC and survived to discharge with CPC 1–2. This report is written in accordance with the CARE guidelines for case reports,[8] and, because cardiac-arrest reporting has its own field-specific reporting standard, we additionally structure the resuscitation data against the Utstein in-hospital cardiac arrest template[9][10] — a framework that also makes explicit which standard data elements are not available in the material provided for this report.

Patient Information, Clinical Findings, and Diagnostic Assessment

Three adult patients with cardiac arrest in the emergency department are included in this series. Table 1 summarises the clinical timeline of all three cases, and Table 2 summarises the available Utstein in-hospital cardiac arrest data elements.[10]

Case 1

A 63-year-old male presented to the Emergency Department in a gasping state with no palpable central pulse. Cardiac monitoring showed pulseless electrical activity (PEA), a non-shockable rhythm. Defibrillation was therefore not indicated, and CPR was started immediately.

Resuscitation was carried out according to the advanced life support algorithm, with intravenous epinephrine administered during CPR. Arterial blood gas analysis demonstrated severe hypercapnic respiratory acidosis, with a PaCO₂ of approximately 125 mmHg, pH around 6.09, and HCO₃⁻ approximately 30 mmol/L, consistent with severe ventilatory failure. Hypoxia was identified as a reversible cause and was addressed during resuscitation. CPR was continued for approximately 20 minutes, after which return of spontaneous circulation (ROSC) was achieved.

The patient was a known case of chronic obstructive pulmonary disease (COPD). Arterial blood gas analysis demonstrated severe hypercapnia, with a PaCO₂ of 125 mmHg. The patient was subsequently admitted to the intensive care unit for further evaluation and care.

Table 1. Case 1 — arterial blood gas and biochemistry on presentation

Parameter	Result	Reference range
pH	6.09	7.35–7.45
PaCO ₂	125 mmHg	35–45
HCO ₃ ⁻	30 mmol/L	22–26
Lactate	7.1 mmol/L	0.5–2.2

Parameter	Result	Reference range
Sodium	134 mmol/L	135–145
Potassium	3.7 mmol/L	3.5–5.1
Chloride	98mmol/L	96–106
Glucose	254 mg/dL	70–140

Case 2

A 70-year-old man presented to the Emergency Department with severe abdominal pain and nausea, reportedly beginning at approximately 06:00 hours. On initial assessment, the airway was patent. He was tachypnoeic, with a respiratory rate of 34/min, tachycardic, with a pulse rate of 140/min, and profoundly hypotensive, with a blood pressure of 60/40 mmHg. His oxygen saturation was 80% on room air and improved to 94% following oxygen administration by face mask.

Initial arterial blood gas analysis showed severe acidemia, with a pH of 7.01, PaCO₂ of 7 mmHg, and bicarbonate of 3.5 mmol/L. Lactate was markedly elevated at 14.1 mmol/L, consistent with severe lactic acidosis in the setting of profound shock. The measured PaCO₂ was lower than expected for compensation alone. According to Winter's formula, the expected PaCO₂ was approximately 13 ± 2 mmHg, suggesting an additional respiratory alkalosis.

The reported anion gap was 25 mmol/L. However, based on the reported sodium, chloride, and bicarbonate values, the calculated anion gap was 46.5 mmol/L [152 – (102 + 3.5)]. Blood ketones were negative. Serum glucose was 254 mg/dL, creatinine was 1.21 mg/dL, and urea was 80 mg/dL. Overall, the biochemical findings were consistent with severe high-anion-gap metabolic acidosis with marked hyperlactataemia.

The patient was managed for profound shock and severe metabolic acidosis in the Emergency Department. Intravenous fluid resuscitation was initiated, and noradrenaline was started for persistent hemodynamic instability. Sodium bicarbonate was administered in view of the severity of the metabolic acidosis. Given the severe respiratory distress and hypoxemia, the airway was subsequently secured with endotracheal intubation and mechanical ventilation was initiated. Despite ongoing resuscitative measures, the patient subsequently developed an asystolic cardiac arrest. Cardiopulmonary resuscitation was initiated immediately, and return of spontaneous circulation was achieved after approximately 10 minutes of CPR.

Following ROSC, the patient was transferred for continued critical care and supportive management.. The underlying cause of the abdominal pain and profound shock could not be established from the available clinical information.

Table 3. Case 2 — arterial blood gas and biochemistry on presentation, with anion gap recalculation.

Parameter	Result	Reference range
pH	7.01	7.35–7.45
PaCO ₂	7 mmHg	35–45
HCO ₃ ⁻	3.5 mmol/L	22–26
Lactate	14.1 mmol/L	0.5–2.2
Sodium	152 mmol/L	135–145
Potassium	3.5 mmol/L	3.5–5.1
Chloride	102 mmol/L	96–106
Glucose	254 mg/dL	70–140
Creatinine	1.21 mg/dL	0.6–1.3
Urea	80 mg/dL	15–40
Ketones	Negative	Negative
Anion gap, as reported in the record	25	8–16
Anion gap, recalculated [Na–(Cl+HCO ₃)]	46.5	8–16
Anion gap, recalculated including K ⁺ [(Na+K)–(Cl+HCO ₃)]	50.0	—

Case 3

A 28-year-old woman presented to the Emergency Department with palpitations and nausea that had started at approximately 06:00 hours. On arrival, her airway was patent. She was tachypneic with a respiratory rate of 34/min, oxygen saturation was 94% on room air, pulse rate was 140/min, and blood pressure was 160/90 mmHg. Her random blood glucose was markedly elevated at 556 mg/dL. Initial arterial blood gas analysis showed metabolic acidosis with a bicarbonate level of 13.5 mmol/L, and urine ketones were positive. Her initial serum potassium was 3.1 mmol/L. The overall clinical and biochemical findings were consistent with diabetic ketoacidosis (DKA) with hypokalemia.

Initial management included intravenous fluid resuscitation and potassium replacement, followed by human regular insulin infusion with close monitoring of blood glucose, serum electrolytes, and acid–base status. During the course of treatment, the patient developed bradycardia followed by pulseless electrical activity (PEA) cardiac arrest. CPR was initiated immediately, and epinephrine was administered according to advanced life

support protocols. Reversible causes were assessed and addressed during resuscitation. An arterial blood gas obtained during CPR showed a potassium level of 3.3 mmol/L. CPR was continued for approximately 30 minutes, after which return of spontaneous circulation (ROSC) was achieved.

Following ROSC, the patient was transferred for continued critical care and post-cardiac-arrest management. Treatment for DKA was continued with close monitoring and correction of her metabolic and electrolyte abnormalities.

Timeline*Table 1. Clinical timeline, all three cases.*

Clinical event	Case 1	Case 2	Case 3
ED presentation	Gasping, pulseless	Abdominal pain, shock, metabolic acidosis	Nausea, palpitations, DKA
Initial rhythm	PEA	Asystole	PEA
CPR initiation	Immediate (timing not further specified)	Immediate (timing not further specified)	Immediate (timing not further specified)
Epinephrine	Administered (dose/timing not specified)	Administered (dose/timing not specified)	Administered (dose/timing not specified)
Defibrillation	Not indicated (non-shockable)	Not indicated (non-shockable)	Not indicated (non-shockable)
CPR duration to ROSC	20 min	10 min	30 min
Post-ROSC care	Critical care	Critical care	Critical care
Targeted temperature management	Not specified	Not specified	Not specified
ICU duration	10 days	18 days	6 days
Hospital discharge	Yes	Yes	Yes
CPC at discharge	1	2	2

Table 2. Cross-walk against Utstein in-hospital cardiac arrest reporting elements,[10] with data gaps stated explicitly.

Utstein in-hospital element	Case 1	Case 2	Case 3
Age	63 years	70 years	28 years
Sex	Male	Male	Female
Location of arrest	Emergency department	Emergency department	Emergency department
Witnessed arrest	Not specified	Not specified	Not specified
Monitored at time of arrest	attached	attached	attached
First documented rhythm	PEA	Asystole	PEA
Time from collapse to CPR	immediate	immediate	immediate
Time to first epinephrine dose	Immediately after cpr started	Immediately after cpr started	Immediately after cpr started
Presumed cause of arrest	TYPE 2 RESPIRATORY FAILURE	Severe shock with HAGMA; etiology not established	DKA-associated; mechanism not established
Total CPR duration	20 min	10 min	30 min
Sustained ROSC	Yes	Yes	Yes
ICU length of stay	10 days	18 days	6 days
CPC at hospital discharge	1	2	2
CPC at longer-term follow-up	Not available	Not available	Not available

Therapeutic Intervention

Case 1

CPR was initiated immediately on identification of PEA arrest, with advanced life support measures including epinephrine administration, following approximately 20 minutes of resuscitation before ROSC.[6] The patient was subsequently transferred to critical care for post-cardiac-arrest management.

Case 2

Initial management included a 1-litre bolus of normal saline followed by a noradrenaline infusion for persistent

severe hypotension. In view of the severe metabolic acidosis, with a bicarbonate level of 3.5 mmol/L, 200 mEq of sodium bicarbonate was administered. The patient subsequently developed bradycardia followed by asystolic cardiac arrest. Cardiopulmonary resuscitation was initiated immediately according to advanced life support protocols, with administration of epinephrine. Return of spontaneous circulation (ROSC) was achieved after approximately 10 minutes of CPR.

Case 3

The patient was managed for diabetic ketoacidosis with intravenous fluid resuscitation using normal saline and potassium supplementation in view of the initial hypokalemia. Following initiation of potassium replacement, a human regular insulin infusion was commenced with close monitoring of blood glucose, serum potassium, other electrolyte levels, and serial acid-base parameters. The patient was monitored closely throughout treatment for correction of hyperglycemia, ketosis, metabolic acidosis, and electrolyte abnormalities. During the course of treatment, she developed bradycardia followed by pulseless electrical activity (PEA) cardiac arrest, for which cardiopulmonary resuscitation was initiated immediately according to advanced life support protocols.

Follow-up and Outcomes

Case 1: The patient achieved ROSC after approximately 20 minutes of CPR and subsequently received continued critical care. The patient was discharged after a 10-day ICU stay with a Cerebral Performance Category (CPC) score of 1, indicating good cerebral performance.

Case 2: ROSC was achieved after approximately 10 minutes of CPR following an asystolic cardiac arrest. The patient subsequently received continued critical care and supportive management and was discharged after 18 days of critical care with a CPC score of 2.

Case 3: ROSC was achieved after approximately 30 minutes of CPR following PEA cardiac arrest. The patient subsequently continued to receive critical care and treatment for DKA and its associated metabolic abnormalities. She improved clinically and was discharged after 6 days with a CPC score of 2.

Overall, all three patients survived to hospital discharge with a favorable neurological outcome, defined in this case series as a CPC score of 1–2. Longer-term neurological follow-up beyond hospital discharge was not available for these patients.

Figure

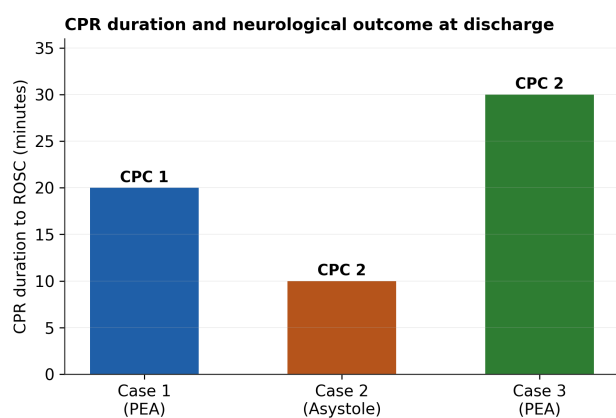


Figure 1. CPR duration to ROSC and Cerebral Performance Category (CPC) at hospital discharge, by case. All three patients achieved a favorable outcome (CPC 1–2) despite CPR durations ranging from 10 to 30 minutes.

Discussion

Non-shockable rhythms are not a monolith

An important feature of this series is that all three patients had an initially non-shockable rhythm—PEA in Cases 1 and 3 and asystole in Case 2—and all three survived to hospital discharge with favorable neurological outcomes. However, these outcomes should not be interpreted as indicating that PEA and asystole carry equivalent prognoses. In the largest available registry analysis, neurologically favorable survival was seven- to eight-fold more likely among patients with PEA than among those with asystole.[7] Within this series, Case 2 is therefore particularly notable, given the survival with a CPC score of 2 following asystolic cardiac arrest in the setting of profound pre-arrest shock and severe acidemia. These cases highlight that an initially non-shockable rhythm does not uniformly preclude meaningful neurological recovery, particularly when potentially reversible causes can be identified and treated promptly.

Case 2: a very high anion gap in profound shock

The recalculated anion gap of 46.5–50.0 mmol/L, compared with the charted value of 25 mmol/L, is markedly elevated by either convention. The discrepancy between the reported and calculated values should be interpreted in the context of the original laboratory data. A systematic approach to severe high-anion-gap metabolic acidosis, such as the GOLDMARK framework (glycols, oxoproline, L-lactate, D-lactate, methanol, aspirin, renal failure, and ketoacidosis), may be useful in considering the differential diagnosis.[13,14] In this patient, blood ketones were reported as negative and creatinine was only mildly elevated, while lactate was markedly increased at 14.1 mmol/L in the setting of profound shock. Lactic acidosis therefore provides a plausible explanation for a substantial component of the elevated anion gap. However, the available clinical information does not exclude other contributing processes, including toxic or co-ingestion-

related causes. The underlying cause of the abdominal pain and profound shock could not be established from the available clinical information, and the available data do not permit a more specific etiological attribution.

Case 3: DKA, insulin therapy, and arrhythmic risk

Case 3 developed cardiac arrest during treatment for DKA, making the patient's metabolic and electrolyte status an important consideration. Insulin promotes intracellular potassium shift, and hypokalemia during treatment of DKA can increase the risk of cardiac arrhythmias. Current hyperglycemic-crisis management guidance recommends assessment of serum potassium before and during insulin therapy and potassium replacement when significant hypokalemia is present.[12] In this patient, the initial potassium level was 3.1 mmol/L, and potassium replacement was initiated before commencement of insulin therapy. An arterial blood gas obtained during resuscitation showed a potassium level of 3.3 mmol/L. Although these findings demonstrate the presence of hypokalemia during the clinical course, they do not establish that hypokalemia was the direct cause of the cardiac arrest. The precise mechanism of the arrest therefore remains uncertain.

Prolonged resuscitation does not preclude neurological recovery

The duration of CPR in this series ranged from 10 to 30 minutes, and all three patients achieved sustained ROSC and survived to hospital discharge with CPC scores of 1–2. These findings are consistent with evidence that prolonged resuscitation does not invariably result in poor neurological outcome. In a large in-hospital registry, resuscitation duration was longer among survivors from hospitals in the longest-duration quartile than in those from the shortest-duration quartile (adjusted RR for survival 1.12, 95% CI 1.02–1.23), although median resuscitation duration remained shorter among survivors than non-survivors (12 vs. 20 minutes).[15] In a review of prolonged CPR, defined as CPR lasting at least 20 minutes, 64 of 82 patients (78%) achieved full recovery or good cerebral performance.[16] A nationwide Japanese OHCA cohort similarly demonstrated that longer CPR duration can be compatible with favorable neurological outcomes, particularly when high-quality CPR and appropriate post-arrest care are provided.[17] The 30-minute resuscitation in Case 3, followed by survival with a CPC score of 2, further illustrates that CPR duration alone should not be used as an isolated determinant of futility, particularly in younger patients with potentially reversible causes.

Post-arrest care and neuro-prognostication

Return of spontaneous circulation represents the transition to post-cardiac-arrest care rather than the end of resuscitation. Current post-resuscitation care emphasizes haemodynamic optimization, controlled oxygenation and

ventilation, temperature control with active prevention of fever, identification and treatment of the underlying cause of arrest, seizure surveillance, and multimodal neurological prognostication.[3,18] In the present series, the available records do not provide sufficient information regarding several aspects of post-arrest management, including temperature-management strategies, coronary angiography, electroencephalography, or formal multimodal neuro-prognostication (Table 2). Therefore, the extent to which these interventions contributed to the observed neurological outcomes cannot be assessed from the available data. Nevertheless, the favorable CPC scores at hospital discharge demonstrate that meaningful neurological recovery was achieved despite initially non-shockable rhythms and, in Case 3, prolonged resuscitation.

Limitations

This study has several limitations. First, it is a small, retrospective, descriptive case series of only three patients and therefore cannot establish predictors of survival or favorable neurological outcome. The heterogeneity of the clinical presentations, underlying pathophysiological processes, initial non-shockable rhythms, and CPR durations also limits direct comparison between the cases and precludes any statistical inference.

Second, several important pre-arrest and resuscitation variables were not available in the records reviewed. These include some baseline clinical details in Case 1 and several standard Utstein in-hospital cardiac arrest reporting elements, such as witnessed status, time to initiation of CPR, time to first epinephrine, and other time-dependent resuscitation parameters (Table 2). The absence of these data limits more detailed assessment of the relationship between resuscitation characteristics and neurological outcome.

Third, the precise etiology of cardiac arrest could not be definitively established in all cases. In Case 2, there is a discrepancy between the reported and recalculated anion gap, although the available biochemical findings demonstrate severe high-anion-gap metabolic acidosis with marked hyperlactatemia. In Case 3, although DKA with hypokalemia was documented and potassium replacement was initiated before insulin therapy, the available potassium measurements do not establish hypokalemia as the direct cause of the subsequent cardiac arrest.

Finally, details of post-cardiac-arrest management were incompletely available, including specific temperature-management strategies, coronary angiography, electroencephalography, and formal multimodal neurological prognostication. Consequently, the contribution of individual post-arrest interventions to the observed neurological outcomes cannot be determined. Neurological outcomes were assessed at hospital discharge

using the CPC score, and longer-term neurological or functional follow-up beyond hospital discharge was not available.

Conclusion

These three cases demonstrate that favorable neurological survival is achievable after emergency department cardiac arrest with an initially non-shockable rhythm, including after prolonged resuscitation, when high-quality CPR, appropriate advanced life support, systematic evaluation of reversible causes are combined with structured post-arrest critical care. They also illustrate two of the most clinically important reversible-cause categories in emergency medicine — profound shock with severe metabolic acidosis, and DKA with its attendant risk of treatment-associated hypokalaemia — and underline that ROSC marks the beginning, not the end, of the resuscitation that determines neurological outcome.

Learning Points

- Favorable neurological survival can occur after emergency department cardiac arrest even when the initial rhythm is non-shockable, though PEA carries a substantially better prognosis than asystole and the two should not be treated as equivalent.[7]
- Immediate recognition of cardiac arrest and prompt initiation of high-quality CPR, with systematic identification and correction of reversible causes, remain the fundamental determinants of outcome in non-shockable rhythms.[1]
- A very high anion gap in a shocked patient warrants a systematic framework (e.g., GOLDMARK) rather than an assumed single cause, particularly when the reported and calculated values disagree.[13]
- Insulin therapy in diabetic ketoacidosis can precipitate a clinically important fall in serum potassium; potassium should be monitored closely during treatment, and insulin withheld or delayed if it falls below approximately 3.3 mEq/L.[12]
- Prolonged CPR (20–30 minutes or more) does not preclude favorable neurological recovery and should not, by itself, be used as a reason to terminate resuscitation in a patient with a potentially reversible cause.[15][16]
- ROSC is not the endpoint of resuscitation: structured post-cardiac-arrest care, including temperature control targeting normothermia, haemodynamic optimisation, and multimodal neuroprognostication, is central to the neurological outcome ultimately achieved.[3]

Patient Perspective

Not available.

Informed Consent

Written informed consent for publication was obtained from the patients or their legally authorised representatives, where applicable. The report was anonymised throughout.

Declarations

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