CASE REPORT

Severe ischaemic cardiogenic shock with cardiac arrest and prolonged asystole: a case report

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Background	Extracorporeal life support (ECLS) by veno-arterial extracorporeal membrane oxygenation (VA-ECMO) is a valuable treatment option during severe cardiogenic shock and during cardiac arrest unresponsive to conventional management. It is applied to bridge the first critical days until the patient recovers or a destination therapy is established.[1] Prolonged episodes without cardiac electrical activity during VA-ECMO are a major problem, as they may cause pulmonary oedema and severe left ventricular (LV) thrombosis.[2] Here, we report a case of a 50-year-old man who presented with a 30-h episode of complete absence of electromechanical activity during ECLS and finally recovered with favourable neurological outcome.
Case summary	A 50-year-old man with out-of-hospital cardiac arrest was transferred to a peripheral hospital after initial successful cardiopulmonary resuscitation (CPR). In the emergency room, he presented with ST-segment elevation myocardial infarction and cardiogenic shock with third-degree atrioventricular block. After immediate insertion of a temporary pacemaker, he received percutaneous coronary intervention of the left anterior descending artery and the circumflex artery. Due to worsening cardiogenic shock, ECLS with VA-ECMO and an Impella® pump was established. Cumulative time of CPR (out of hospital and in hospital) was 41 min. After transfer to our institution's intensive care unit, both the heart's mechanical and electrical activity ceased for more than 24 h and recovered slowly thereafter. After showing promising neurological outcome, epicardial pacemaker leads, an implantable cardioverter-defibrillator, and finally, a LV assist device were implanted. He was dismissed into rehabilitation with only minor neurological residua 6 weeks later.
Discussion	Impella® implantation on top of VA-ECMO may be considered beneficial in the therapy of prolonged cardiac arrest.[3] While VA-ECMO ensures oxygenation and organ perfusion, Impella® vents the left ventricle and enhances coronary perfusion. In the presented case, a favourable outcome was reached despite an 'untreated' prolonged absence of cardiac electromechanical activity. Under specific circumstances during ECLS with extracorporeal membrane oxygenation and Impella®, waiving of temporary pacing may be considered in absent cardiac electromechanical activity to avoid further complications.
Keywords	ST-elevation myocardial infarction • Cardiogenic shock • Extracorporeal life support • Extracorporeal membrane oxygenation • Impella • Left ventricular assist device • Case report

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Learning points

- In the setting of extracorporeal life support with Impella® on top of veno-arterial extracorporeal membrane oxygenation in ischaemic cardiogenic shock, a favourable outcome can be reached despite a prolonged complete absence of cardiac electromechanical activity.
- It should be further discussed and investigated whether under specific circumstances, the waiving of myocardial pacing in asystole due to cardiogenic shock may even be beneficial and contribute to electromechanical recovery.

Timeline

Presentation and initial management	Out of hospital cardiac arrest with a total of 41 min of cardiopulmonary resuscitation. Due to ST-elevation myocardial infarction (STEMI) and third degree atrioventricular block, a transfemoral pacemaker is inserted, and percutaneous coronary intervention of the left anterior descending artery and circumflex artery are performed. In cardiogenic shock, Impella CP® (Abiomed, Danvers/USA) and veno-arterial extracorporeal membrane oxygenation (VA-ECMO) therapy are established.
First 24 h	After transfer to our Heart Center, the patient requires expansive fluid volume substitution and vasoactive therapy in 'post-cardiac arrest syndrome'. Mild therapeutic hypothermia (33°C) is applied. Echocardiography shows no visible ventricular contraction, and the patient develops asystole refractory to transcutaneous pacing.
Day 2	Fasciotomy is performed due to compartment syndrome of the left lower leg. Continuous renal replacement therapy is initiated in acute renal failure.
Day 3	Episodes of electrical activity and mechanical contractions of the left ventricular return after approximately 30 h of asystole, and the haemodynamic situation improves. In an assessment of responsiveness, the patient displays basic neurologic reactions.
Day 10	An epicardial pacemaker is inserted and pericardial implantable cardioverter-defibrillator implantation is performed via a minimally-invasive subxyphoid approach.
Day 15	The Impella CP® is removed and an Impella® 5.0 implanted via the right subclavian artery as a 'second bridge to decision' approach.
Day 20	Left ventricular assist device (LVAD) implantation takes place. Both VA-ECMO and Impella® 5.0 are explanted during the surgery.
Day 61	The patient is dismissed into rehabilitation with only minor neurological residua.

Case presentation

The management of cardiogenic shock complicating acute myocardial infarction has long been and is still a challenging issue. In the last decades, advancements in pharmacotherapy, the establishment of coronary reperfusion therapy and, most recently, mechanical circulatory support (MCS) in refractory shock have led to profound improvements in survival.[4,5] However, despite these efforts, the in-hospital mortality remains substantial, with figures ranging between approximately 34% and 51%.[6–9] In particular, the management of patients receiving MCS is often complex and requires individualized decisions in a multidisciplinary approach, as specific protocols for defined clinical conditions are still lacking.[1]

Here, we present the case of a 50-year-old man with severe ischaemic cardiogenic shock who recovered with favourable neurological outcome despite a prolonged absence of cardiac electromechanical activity.

A previously healthy 50-year-old man collapsed at his workplace. His co-workers performed lay cardiopulmonary resuscitation (CPR) including chest compressions, and he regained consciousness after approximately 1 min. An automated external defibrillator was applied but no shock was advised.

Upon arrival, the emergency physician detected third-degree atrioventricular (AV) block that quickly converted into sinus rhythm, where electrocardiogram (ECG) showed ST-segment elevation over the anterior wall (in leads I and aVL). Five thousand I.U. of unfractionated heparin and 300 mg of aspirin were administered intravenously. The patient was transferred to the emergency room of a peripheral hospital with spontaneous circulation, fully conscious and responsive, but agitated and disoriented. On physical exam, he had a pulse of 60 b.p.m. with few arrhythmic beats but no heart murmurs. He was tachypnoeic and auscultation revealed wet rales in both lungs. The peripheral oxygen saturation showed 91%. Blood pressure was not measurable. The skin was cold and the patient sweating heavily. The pupils were isocor, round and reacted to light bilaterally. Arterial blood gas analysis showed the following values: pO_2 72 mmHg, pCO_2 59 mmHg, pH 6.87, $pHCO_3$ 10.8 mmol/L, BE -24.4 mmol/L, Na^+ 132 mmol/L, K^+ 4.6 mmol/L, and lactate 16.8 mmol/L. According to his relatives, symptoms suspicious of myocardial infarction (retrosternal pain with radiation in the jaw and left upper arm) had persisted since the previous day. Besides being a smoker, there was no pre-existing condition or medical history beyond that.

While still in the ER, total AV block recurred and the patient was again in need of CPR. After the administration of 2 mg of epinephrine and endotracheal intubation, spontaneous circulation was re-established in the ER after 10 min. Catecholamine therapy was started with epinephrine (25 mg/50 mL, 16 mL/h), norepinephrine (50 mg/50 mL, 30 mL/h), and dobutamine (250 mg/50 mL, 10 mL/h). The transthoracic echocardiogram showed a severely reduced left ventricular (LV) function with merely minor contractility of the posterior wall, but no LV or right ventricular (RV) dilatation or relevant valve abnormalities. The patient was consecutively transferred to the cath lab, where at first a temporary pacemaker was inserted under fluoroscopic guidance via the right femoral vein. Due to poor LV function, cardiogenic shock with high catecholamine demand and signs of pulmonary oedema, an Impella CP® (Abiomed, Danvers/ USA) was then placed in the left ventricle via the left femoral artery.

Systolic blood pressure then stabilized at 80–90 mmHg during continued catecholamine support. Coronary angiography was performed and revealed an ostial thrombotic occlusion of the left anterior descending artery (LAD), while the circumflex artery (CFx) and right coronary artery (RCA) showed only wall irregularities without significant stenoses. Percutaneous coronary intervention of the LAD was performed with implantation of one drug-eluting stent (DES). Despite a therapeutic activated clotting time (ACT), the proximal circumflex artery (CFx) occluded thrombotically during procedure. After thrombus aspiration of the CFx, TIMI III flow was established on both vessels. The loading dose of ticagrelor (180 mg via a nasogastric tube) was administered.

Repeated transthoracic echocardiography showed an increasing circular pericardial effusion (20 mm in diameter), which was considered haemodynamically relevant, as the right atrium (RA) and right ventricle (RV) were compressed. Echocardiography-guided percutaneuos pericardiocentesis with insertion of a pericardial tube was performed by a subxyphoid approach. However, only minimal amounts of blood (6 mL) were aspirated and the tube therefore quickly removed. The effusion did not progress any further in the following subsequent echocardiographic examinations.

Due to ongoing cardiogenic shock with unstable haemodynamics, unchanged high serum lactate concentration (of around 16 mmol/L) and rising catecholamine demand, our cardiac arrest team was requested for the initiation of extracorporeal life support (ECLS). Upon their arrival, the patient was again undergoing CPR, and veno-arterial extracorporeal membrane oxygenation (VA-ECMO) (CardioHelp®, Maquet, Rastatt, Germany) had to be implanted while mechanical chest compressions were being performed. Extracorporeal membrane oxygenation (ECMO) flow was finally established after another 30 min of CPR (venous cannulation of the left femoral vein, arterial cannulation of the right femoral artery, no antegrade perfusion cannulae). Finally, the patient was transferred to our University Heart Center after a total of 41 min of CPR.

Upon arrival at our intensive care unit, third-degree AV block recurred and ECG showed a ventricular escape beat of 30–40 b.p.m. After several hours of Impella® therapy, vesicular breathing sounds were heard on lung auscultation, with no signs of pulmonary oedema. SpO₂ on arrival was 100% and arterial blood gas analysis showed a pO₂ of 489 mmHg under mechanical ventilation with an FiO₂ of 1.0, which was then immediately reduced to avoid hyperoxygenation. Serum lactate was 19.0 mmol/L (pH 7.31). Invasively measured blood pressure was 70/50 mmHg and body temperature 34°C. Computed tomography (CT) scan of the head showed no signs of cerebral haemorrhage or ischaemia. Repeat transthoracic echocardiography showed no visible ventricular contraction, while the circumferential pericardial effusion was measured 15–20 mm in diastolic diameter. Laboratory tests indicated the development of multi-organ dysfunction with elevated renal parameters (creatinine 2.8 mg/dL, eGFR 25 mL/min) and liver enzymes (ASAT/GOT 1416 U/L and ALAT/GPT 697 U/L, total bilirubin 4.2 mg/dL).

Management at our intensive care unit was performed based on a goal-directed therapy protocol focusing on ECMO flow (target 5 L/min), serum lactate concentration, invasive mean arterial pressure (target 60 mmHg), and arterial oxygen saturation (target normoxia). The placement of a pulmonary artery catheter for advanced haemodynamic monitoring was waived due to the proximity to the venous ECMO cannula. While the ECMO initially had a stable flow of 4.3 L/min on average, the Impella® was not able to deliver more than 0.5 L/min due to 'sucking-up' phenomenon, i.e. Impella® blood flow was restricted due to recurring wall contact of the blood inlet area of the pump. This phenomenon may be more likely to occur in a 'vented' or 'empty' left ventricle of low volume when Impella® is used on top of VA-ECMO therapy.

While the patient had initially presented with signs of pulmonary oedema only a few hours earlier, he meanwhile required rising amounts of fluid substitution in combination with vasoactive therapy to overcome vasoplegia in manifest 'post-cardiac arrest syndrome'. Approximately 4 L of crystalloids were administered within the first hours, thus sparing exorbitant doses of catecholamines worsening microcirculation. Additionally, the patient received numerous blood and coagulation products to maintain a target haemoglobin level of at least 10 mg/dL and an aPTT of 50–60 s. Bleeding stigmata were not present. FiO₂ levels were quickly reduced to avoid hyperoxia. According to current guidelines,[10] mild therapeutic hypothermia (33°C) was quickly initiated and maintained for 24 h.

After a few hours however, with further decreasing body temperature, the heart rate slowed down and turned into complete asystole ('flatline' on the ECG). RV stimulation proved inefficacious (threshold > 12 V), even though there was no sign of a malpositioning in the X-ray. Cautiously, as lead perforation might have caused the pericardial effusion in the first place, several unsuccessful attempts to reposition the pacemaker lead were undertaken. Transcutaneuos pacing with an external defibrillator also merely resulted in stimulation of the phrenic nerve with contractions of the diaphragm. Simultaneously, haemodynamics had stabilized, catecholamine doses were reduced, there was spontaneous diuresis (at approximately 50 mL of urine output per hour) and a steady and strong decline in serum lactate concentration (from 19.0 to 4.1 mmol/L within the first 9 h). Therefore, in view of the possible risks and challenges of transjugular pacemaker insertion (e.g. RV perforation, difficulties in placement because of the proximity to the venous ECMO cannula at the junction between the inferior caval vein (IVC) and the RA, haemorrhage, pneumothorax), we decided against de novo lead placement and removed the transfemoral pacer. This resulted in a complete absence of both electrical and mechanical activity of the patient's heart.

Under target temperature management, sedation and catecholamine therapy, pupils were still narrow and isocor. The patient developed compartment syndrome of the left lower leg on the first day and underwent fasciotomy of the tibialis anterior compartment, resulting in diffuse haemorrhage requiring massive transfusion and numerous coagulation products. Intraoperatively, leg muscles appeared vital, so we decided against the insertion of an antegrade ECMO perfusion cannula. However, in beginning haemorrhagic shock, the patient was again haemodynamically unstable. ECMO flow decreased and even repeatedly halted for short episodes, but each time could immediately be re-established under continued major fluid administration. However, the patient developed acute renal failure with oliguria and hyperpotassaemia, and continuous renal replacement therapy was initiated in the form of continuous veno-venous haemofiltration (CVVH).

On the third day, after approximately 30 h of asystole, a return of short episodes of electrical activity (p-waves) was noted, followed by a slow ventricular escape beat (of 10 b.p.m.) and even intermittent proper AV conduction. At the same time, repeated echocardiograms showed some contraction of the interventricular septum, while the anterior and posterior LV wall were still akinetic. Invasive blood pressure monitoring and Impella® motor current waveforms showed tiny excursions with an amplitude of up to 5–10 mmHg. Eventually, sinus rhythm returned, first intermittently, then continuously, and LV contractions vaguely intensified.

Despite still under ECLS with ECMO and Impella®, the return of electromechanical coupling accounted for an improvement of the patient's haemodynamic situation, allowing for a weaning of norepinephrine substitution. Lactate concentration further decreased to normal range. With a rise in mean arterial pressure above the targeted 60 mmHg, we administered i.v. nitrate and i.v. urapidil to further reduce vascular resistance, afterload and left ventricular end-diastolic pressure (LVEDP) (in addition to 'mechanical' reduction through the Impella®), aiming at facilitating LV recovery.

To assess responsiveness, the patient's sedation was reduced by the end of day 3, and he opened his eyes, gradually moved both arms and legs and even at times reacted when spoken to. Low-dose sedation was continued to improve tolerance of the endotracheal tube.

Several episodes of supraventricular tachycardia (160 b.p.m.) occurred on day 4, so dobutamine was switched to milrinone. Levosimendan was subsequently administered 'on top' to further stimulate inotropy. Due to an increase in intra-abdominal pressure (to a maximum of 28 cmH$_2$O), the ultrafiltration rate of the CVVH was raised to up to 1000 mL/h to avoid abdominal compartment syndrome.

Communication with the patient eventually became possible over the following days. However, on day 8 and 9, ventricular tachyarrhythmias, ventricular flutter and even ventricular fibrillation occurred. The patient was defibrillated five times and i.v.-loading with amiodarone was initiated. As episodes of asystole and third-degree AV block recurred nevertheless, an epicardial pacemaker was inserted, and pericardial implantable cardioverter-defibrillator (ICD) implantation was performed via a minimally-invasive subxyphoid approach on day 10. The placement of epicardial leads was chosen over a transvenous approach due to the assumed persistent risk of lead perforation in a small ('unloaded') right ventricle, thus guaranteeing safe lead positioning (on the left ventricle). The patient developed acute pericardial tamponade shortly after the procedure nonetheless and had to undergo surgical drainage.

In the following days, the neurological condition further improved, while weaning of ECLS was hampered by continued haemorrhages from several sites (chest tubes, thoracotomy and catheter insertion sites, epistaxis) requiring transfusion of 4–6 packed red blood cells (300 mL per unit) per day. Moreover, recovery of the LV and RV function was still considered insufficient to either successfully wean ECMO therapy or successfully perform left ventricular assist device (LVAD) implantation. Therefore, on day 15, the Impella CP® was removed and an Impella® 5.0 implanted via the right subclavian artery as a 'second bridge to decision' approach. Both adequate ICD therapies for recurring ventricular fibrillation and several inadequate therapies decreased over the following days and eventually came to an

end. Elective tracheotomy was performed on day 16, and diffuse bleeding gradually ceased as well.

After regaining further ventricular contractility (the LV ejection fraction estimated at approximately 25%), the patient successfully underwent LVAD implantation (HVAD®, HeartWare, Framingham/USA) on day 20. Both VA-ECMO and Impella® 5.0 were explanted during the surgery. In the post-operative course, the patient tolerated a negative fluid balance of more than 10 L, while there were no signs of right heart failure under LVAD support. Mechanical ventilation was quickly weaned and no further significant complications occurred before the patient was moved out from the intensive care unit 21 days after LVAD implantation. He was dismissed into rehabilitation with only minor neurologic residua (memory problems) after a total of 61 days after initial admission. He still received haemodialysis three times a week upon dismissal.

Ten months later, reporting to our outpatient department, the patient is in a good physical condition and can climb three to four flights of stairs without breaks or major dyspnoea. There is no anginal pain or leg oedema. LV function is unchanged with an ejection fraction of approximately 25% under LVAD support. The performance in the 6-min walking test is stable, with distances repeatedly over 500 m. Renal function has recovered, and he has come off haemodialysis. With the exception of slightly elevated NT-proBNP levels, laboratory values show no pathological findings. There is sinus rhythm in the ECG and no episodes of VT or VF in the data storage of the ICD. He has stopped smoking and is happy to have regained weight ever since. The patient successfully underwent evaluation for heart transplantation and was placed on the Eurotransplant waiting list with the urgency status 'Transplantable (T)'.

Discussion

We report here on a case in which a favourable neurological outcome was reached after ECLS with VA-ECMO and Impella® in severe cardiogenic shock after more than 40 min of CPR. Assumingly, both rapid ECMO implantation, which has previously been shown to improve survival rate in out-of-hospital cardiac arrest (OHCA),[11] and Impella® implantation on top of VA-ECMO, which also has proven beneficial in this setting,[3] may have contributed to this positive result. Based on a brain CT scan without pathological findings after ECMO implantation (which has been described as an important predictor for good neurological outcome[11]), a regimen of differentiated catecholamine and inotropic therapy was applied according to current guideline recommendations.[10]

Cognitive impairments in general are common among survivors of OHCA and have been investigated in numerous studies, with figures ranging from 6% to 100%.[12] In the presented case, the serum concentrations of neuron-specific enolase (NSE), a highly specific indicator of poor neurologic outcome after cardiac arrest, were determined on days 1, 2, and 3. The concentrations measured were 68.9 μg/L on day 1, 48.0 on day 2, and 45.1 on day 3, while concentrations >90 μg/L 3 days after cardiac arrest have been identified as strong predictors of poor outcome in a similar patient group.[13]

During the clinical course, there was uncertainty regarding the management of absent cardiac electromechanical activity during therapeutic hypothermia under ECLS. To the best of the authors'

knowledge, no case of prolonged asystole under ECLS has been reported in literature so far. Despite the bradyarrhythmias and consecutive asystole, we decided to continue hypothermia therapy, given the evidence of its beneficial effect on outcome in non-VF initial rhythms.[14,15]

After removal of the femoral pacemaker, *de novo* lead insertion via the transjugular vein in asystole was discussed but eventually dismissed to avoid further complications (e.g. RV perforation). Besides, transvenous pacing had earlier proven inefficacious. In animal studies, inotropic therapy has shown to recruit a functional reserve in post-ischaemic stunned (atrial) myocardium, while electrical pacing did not augment myocardial contractility but rather had an adverse effect.[16] From this perspective, it should be discussed whether the waiving of myocardial pacing in asystole under specific circumstances may even be beneficial and contribute to electromechanical recovery.

The presented case demonstrates that the decision to leave the asystole 'untreated' under ECLS with ECMO and Impella®, resulting in a complete absence of cardiac electromechanical activity, can still result in a favourable outcome.

Acknowledgements

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