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Case report

Use of resuscitative endovascular balloon occlusion of the aorta (REBOA) and ultrasound-guided left stellate ganglion block to rescue out of hospital cardiac arrest due to refractory ventricular fibrillation: A case report

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Abstract
Out of hospital cardiac arrest from shockable rhythms that is refractory to standard treatment is a unique challenge. Such patients can achieve neurological recovery even with long low-flow times if perfusion can somehow be restored to the heart and brain. Extracorporeal cardiopulmonary resuscitation is an effective treatment for refractory cardiac arrest if applied early and accurately, but often cannot be directly implemented by frontline providers and has strict inclusion/exclusion criteria. We present the case of a novel treatment strategy for out of hospital cardiac arrest due to refractory ventricular fibrillation utilizing Resuscitative Endovascular Balloon Occlusion of the Aorta-assisted cardiopulmonary resuscitation and intra-arrest left stellate ganglion blockade to achieve return of spontaneous circulation and eventual good neurological outcome after 101 minutes of downtime.
Keywords: Cardiac arrest, Resuscitative endovascular balloon occlusion of the aorta, Stellate ganglion block, Refractory ventricular tachycardia/fibrillation

Introduction

Out of hospital cardiac arrest (OHCA) due to ventricular tachycardia/ventricular fibrillation (VT/VF) that is refractory to advanced cardiac life support (ACLS) occurs ~25,000–36,000 times per year in the United States.[1,2] Recent clinical trials have shown that extracorporeal cardiopulmonary resuscitation (E-CPR) incorporating intra-arrest venoarterial extracorporeal membrane oxygenation (VA ECMO) is effective in treating OHCA from refractory VT/VF (rVT/VF) if implemented early and efficiently.[3,4] However, the inclusion/exclusion criteria and significant expertise/resources required for E-CPR leave room for other novel treatment strategies in this population. Resuscitative endovascular balloon occlusion of the aorta (REBOA) and left stellate ganglion block (SGB) have both been reported to treat OHCA from rVT/VF.[5–8] However, their successful combined use with resultant good neurological outcome has not been described previously.

Case report

A previously healthy 17-year-old male was witnessed by his family to suddenly collapse while mowing the lawn. Upon EMS arrival, he was unresponsive with a palpable pulse. Pulses were lost during the preliminary assessment and the initial rhythm was ventricular fibrillation (VF). Fig. 1. displays the resuscitation time-course. ACLS was performed with bag-valve mask ventilations and eventual placement of a supraglottic airway (SGA), continuous manual and then mechanical chest compressions, repeated manual defibrillation (6*200 J),

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https://doi.org/10.1016/j.resplu.2023.100524
Received 25 October 2023; Received in revised form 20 November 2023; Accepted 23 November 2023
Available online xxxx

Location	Scene				Transport		Emergency Department			
Airway	BVM			BVM/SGA	SGA		Endotracheal Tube			
CPR	Manual	LUCAS				LUCAS/Manual	Manual			
Rhythm	VF				VF/Asystole	Asystole/PEA	VF/Asystole	Asystole/PEA	PEA/Sinus	VF	Sinus
Defibrillation	5				1		1			3	
Medications	Epi 2mg, Mg 1g	Epi 1mg, Mg 1g, Midaz 2.5mg	Epi 1mg, Mg 1g		Epi 1mg, Amio 300mg			Epi 2mg	Epi 1mg, Bicarb 150mEQ	Lido 200mg, Mg 4g, Bicarb 50mEQ	Calcium 2g, Epi + Norepi gtt
Procedures	IO	IV						Intubation, CVC, Cutdown	REBOA	REBOA, SGB	REBOA
Time (min)	0 – 10	11 – 20	21 – 30	31 – 40	41 – 50	51 – 60	61 – 70	71 – 80	81 – 90	91 – 100	101 – 110

Fig. 1 – Resuscitation time-course. CPR = cardiopulmonary resuscitation, BVM = bag-valve mask, VF = ventricular fibrillation, IO = intraosseous, IV = intravenous, SGA = supraglottic airway, PEA = pulseless electrical activity, CVC = central venous catheter, REBOA = resuscitative endovascular balloon occlusion of the aorta, SGB = stellate ganglion block, gtt = infusion.

and administration of epinephrine (5 mg) and amiodarone (300 mg). Magnesium sulfate (3 g) was also given due to concern for polymorphic ventricular tachycardia (PMVT). During resuscitation, the patient exhibited CPR-induced consciousness (eye opening, jaw clenching, spontaneous movement) during chest compressions, but became unresponsive during pulse checks, for which he received 2.5 mg of midazolam to facilitate airway management. Repeated defibrillation was also hampered by significant diaphoresis that caused the adhesive pads to fall off and need to be replaced. After 45 minutes without return of spontaneous circulation (ROSC), the patient was transported from the scene with ongoing mechanical CPR.

On arrival to our emergency department (ED), the patient was in cardiac arrest for 60 minutes. Mechanical CPR was continued. The initial rhythm was VF. After a vector change from anterior-lateral to anterior-posterior pad placement, the patient was defibrillated (200J). The subsequent rhythm deteriorated into asystole. Simultaneously, ultrasound (US)-guided left common femoral arterial (CFA) access was attempted without pausing CPR. Blood was aspirated into the syringe, but a wire was unable to be thread into the vessel. Arterial blood gas (ABG) analysis revealed pH < 7.0, PCO2 45 mmHg, PO2 139 mmHg, lactate > 20 mmol/L, potassium 5.6 mmol/L. 6 minutes after ED arrival, the patient was evaluated for E-CPR by the cardiac surgical team. At that time, the rhythm was asystole and point-of-care US showed no cardiac motion. The decision was made not to proceed with ECMO cannulation.

Given the multiple positive prognostic aspects of the resuscitation, we proceeded to deploy a REBOA to augment CPR. Percutaneous left CFA access was attempted twice further using US but was unable to be obtained. Consequently, a left femoral cutdown was performed, without pausing CPR. An 18G × 15 cm arterial catheter was inserted and blood pressure (BP) was transduced with CPR. During the 8 minutes required for the cutdown, the SGA was exchanged for an endotracheal tube, a right femoral central venous catheter was placed, and an additional 2 mg of epinephrine were administered. Pulse checks revealed pulseless electrical activity (PEA).

78 minutes after collapse, BP during CPR was poor (Fig. 2a). Consequently, we immediately exchanged the arterial line for a 7F sheath and deployed an ER-REBOA catheter (Prytime Medical Devices Inc., Boerne, TX, USA) to Zone 1 (50 cm). The balloon was inflated with 28 cc of saline before tactile feedback of aortic occlusion, 24 minutes after ED arrival and 84 minutes after arrest onset. At the next pulse check, ROSC was achieved with an aortic

Fig. 2a – Initial invasive arterial blood pressure during cardiopulmonary resuscitation.

BP of 154/102 mmHg (Fig. 2b). Multiple doses of sodium bicarbonate (150 mEQ), epinephrine (0.5 mcg/kg/min), and norepinephrine (0.5 mcg/kg/min) infusions were begun to facilitate REBOA balloon deflation. However, 6 minutes after ROSC, and before the balloon

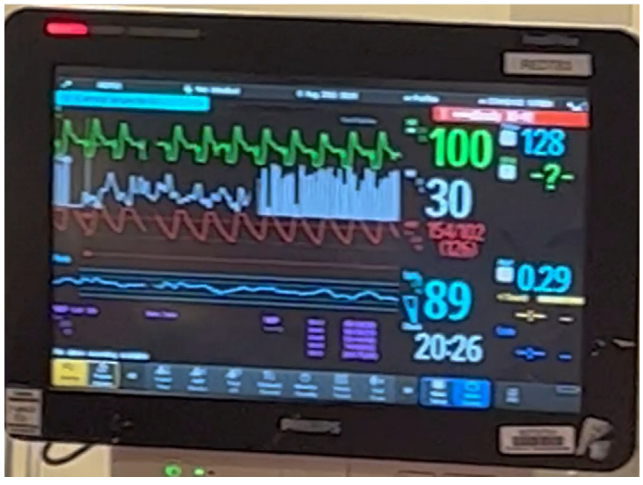

Fig. 2b – Transient return of spontaneous circulation after REBOA inflation.

could be deflated, the patient's rhythm returned to VF. CPR was restarted, defibrillation performed (2*200 J), an additional 4 g magnesium sulfate and 200 mg lidocaine were given out of concern for missed PMVT and/or recurrent VF, but the patient remained in VF.

Given the recurrence of VF despite adequate coronary perfusion with the REBOA, we focused subsequent treatment on the arrythmia itself. Having already employed multiple pharmacologic antiarrhythmics without success, we performed a left stellate ganglion block. Using dynamic US guidance with a 13-MHz linear transducer, we identified the region of the left anterior neck at the C6 level. Briefly pausing CPR, we inserted a 20G × 3.5-inch spinal needle in-plane posterior to the carotid artery, against the vertebrae, and injected 20 cc of 1% lidocaine. The SGB was completed 35 minutes after ED arrival and 95 minutes after the initial collapse. At the next pulse check, VF was defibrillated (200 J), with subsequent ROSC (101 minutes from arrest).

The patient's rhythm and BP remained adequate for the next several minutes as 2 g of calcium chloride and an isotonic bicarbonate infusion were administered, and the minute ventilation was increased. The REBOA balloon was then gradually deflated over 8 minutes. The patient tolerated the deflation with acceptable hemodynamics, remaining on epinephrine (0.1 mcg/kg/min) and norepinephrine (0.8 mcg/kg/min) after deflation. Total time of aortic occlusion was 35 minutes.

Initial neurologic examination revealed reactive pupils, absent corneal reflexes, absent cough/gag, with intact respiratory drive. ECG revealed sinus tachycardia with a right-bundle branch morphology and a pre-excitation delta wave consistent with Wolfe-Parkinson-White syndrome. QTc interval was 503 ms (Fig. 3). Immediately after sustained ROSC, pedal pulses were symmetric and palpable bilaterally. However, 30 minutes after ROSC, brisk bleeding began to emanate from the left CFA cutdown site and left pedal pulses were lost. With the 7F sheath still in place, arterial bleeding was noted from an inadvertent posterior CFA arteriotomy. Manual pressure was held, trauma and vascular surgery were emergently consulted, and the patient was taken to the operating room (OR) for vein patching of anterior and posterior CFA arteriotomies. Completion angiography showed intact proximal flow with 2-vessel runoff to the foot. En route to the ICU, whole-body CT scans were obtained. Imaging was notable for a head CT without cerebral edema, chest CT with findings of aspiration, and abdominal CT with diffuse small and large bowel edema and enhancement concerning for ischemic injury.

After stabilization in the ICU, sedation was paused and neurological examination revealed intact brainstem reflexes, spontaneous eye opening, and the ability to follow commands in all extremities except the left leg. Consequently, we initiated fever avoidance (≤37.5C) for 72 hours but did not implement further neurologic resuscitation. The patient's hospital course was complicated by hypoxemic respiratory failure, acute kidney injury requiring renal replacement therapy, rhabdomyolysis and left calf compartment syndrome requiring fasciotomy, ischemic colitis with gastrointestinal bleeding, pulmonary edema, and cardiogenic shock treated with vasoactive agents. He was extubated on post-arrest day 4 and transferred out of the ICU on day 8. He underwent radiofrequency ablation of his cardiac accessory pathway on post-arrest day 14. Pre-discharge neurocognitive testing revealed a Montreal Cognitive Assessment (MOCA) score of 28/30. He was discharged home on post-arrest day 36 with a cerebral performance category (CPC) score of 1 with some distal left lower extremity weakness. By post-arrest day 90, he was ambulating on his own and preparing to go back to school.

Discussion

Refractory OHCA from VT/VF represents a two-part challenge; successful restoration of cardiac function requires the reversal of both severe hemodynamic and electrical dysfunction. VA ECMO treats both aspects by directly providing circulatory support and coronary blood flow, which subsequently allows for revascularization (if needed) and medical/electrical antiarrhythmic interventions. However, current inclusion/exclusion criteria for E-CPR limit ECMO

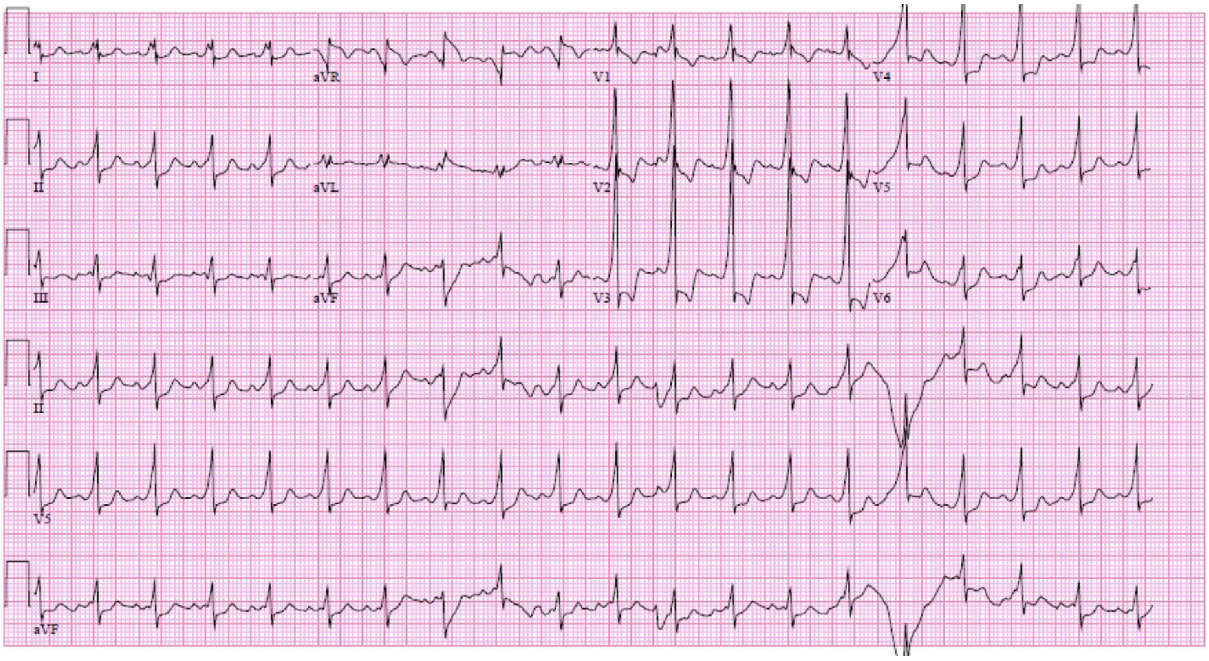

Fig. 3 – ECG after sustained return of spontaneous circulation.

usage based upon the predicted amount of pre-implementation cerebral ischemia, to avoid creating scenarios with cardiac but not neurological recovery.[3,9] At the time of consideration for E-CPR, our patient met several common exclusion criteria (lactate > 18 mmol/L, low-flow time > 60 minutes, asystolic rhythm). However, the patient simultaneously exhibited positive prognostic factors, such as young age and signs of life during resuscitation.[10] This case highlights the complex challenges in selecting patients for E-CPR.

Similar to VA ECMO, temporary aortic occlusion with REBOA can increase coronary perfusion and promote ROSC during CPR for rVT/VF.[11,5,6] While preliminary studies have demonstrated the feasibility and immediate hemodynamic effects of REBOA, there have been no neurologically-intact hospital survivors reported until now. Unlike with VA ECMO, the sub-diaphragmatic ischemia that occurs during Zone 1 REBOA can result in hemodynamic collapse or subsequent irreversible multiorgan failure after balloon deflation. No standard technique for balloon deflation after ROSC has been described in the literature. We employed pre-emptive adapted-alkalinization, vasopressor support, and gradual deflation based on our prior experiences in trauma and cardiac arrest patients.[12–14] Further studies should examine other methods to minimize visceral ischemia and achieve safe balloon deflation with REBOA. While ideally placed with percutaneous US guidance, arterial sheath insertion required open cutdown in our patient secondary to a constricted and collapsed femoral artery in the setting of prolonged low-flow time. This has also been described in REBOA use among young trauma patients.[14] While our patient suffered a complication as a result of the difficulty of arterial access, this may have been unavoidable whether REBOA or ECMO was employed.

Sympathetic blockade to treat refractory ventricular arrhythmias has an experimental and preliminary clinical foundation, primarily in patients with electrical storm but also in OHCA from rVT/VF.[15,16] While this first involves intravenous beta blockade, refractory cases can be treated with temporary or permanent cardiac sympathetic denervation (CSD) via percutaneous, neuraxial or operative techniques. In the case of OHCA from rVT/VF, US-guided SGB is the only feasible means of CSD that can be performed during CPR. Our case contributes to the burgeoning literature on this novel treatment, which can be performed by emergency care providers.[7,8] The effectiveness of beta blockade or SGB in relation to standard antiarrhythmics or defibrillation modifications for treatment of rVT/VF requires formal investigation.

We present the successful combined use of two novel, advanced resuscitation techniques, not supported by current ACLS guidelines, to rescue a unique case of OHCA due to rVT/VF. These were performed by ED physicians with formal critical care training. Expansion of this career path will hopefully help drive the development of similar innovative treatment strategies for life-threatening diseases not reversed by current methods.[17] Regardless of the specialized techniques employed, the patient's eventual good neurological outcome could not have been possible without prompt, continuous, and effective prehospital CPR, which remains the foundation of successful cardiac arrest care.

CRediT authorship contribution statement

Byron C. Drumheller: Conceptualization, Project administration, Writing – original draft. **Jonathan Tam:** Conceptualization, Project administration, Writing – review & editing. **Kimberly W. Schatz:** Conceptualization, Project administration, Writing – review & editing. **Ankur A. Doshi:** Project administration, Supervision.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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