Case Report

Vol. 5, No. 2; 2021; pp 102–107
DOI: 10.26676/jevtm.v5i2.196

REBOA-Assisted Resuscitation in Non-Traumatic Cardiac Arrest due to Massive Pulmonary Embolism: A Case Report with Physiological and Practical Reflections

Philippe Rola[1], Philippe St-Arnaud[2], Timur Karimov[3] and Jostein Rødseth Brede[4-7]

[1]Chief of Service, Intensive Care Unit, Santa Cabrini Hospital, Montreal, Quebec, Canada
[2]Intensive Care Unit, Santa Cabrini Hospital, Montreal, Canada
[3]Intensive Care Unit, Department of General Medicine, Honore Mercier Hospital, St-Hyacinthe, Canada
[4]Department of Emergency Medicine and Pre-hospital Services, St. Olavs University, Hospital, Trondheim, Norway
[5]Department of Anaesthesiology and Intensive care, St.Olavs University Hospital, Trondheim, Norway
[6]Department of Research, Norwegian Air Ambulance Foundation, Oslo, Norway
[7]Department of Circulation and Medical Imaging, Faculty of Medicine and Health Sciences, Norwegian University of Science and Technology (NTNU), Trondheim, Norway

We present the case of a 36-year-old woman who suffered from and ultimately did not survive a non-traumatic out-of-hospital cardiac arrest (NTCA) likely due to massive pulmonary embolism. The resuscitation attempt included the use of a resuscitative endovascular balloon occlusion of the aorta (REBOA) catheter which resulted in a return of spontaneous circulation and distinct improvements in arterial blood pressure, end-tidal CO_2 and cerebral oximetry values. This suggests that the use of REBOA has the physiological basis and potential to improve the rate of both survival and favorable neurologic outcome and warrants further study.

Keywords: *REBOA; Cardiac Arrest; Resuscitation; Endovascular Techniques*

Received: 2 April 2021; Accepted: 25 April 2021

A 36-year-old woman who sustained a non-traumatic out-of-hospital cardiac arrest presented to the emergency department of an urban community hospital. There was a considerable delay before transfer to hospital, due to the stretcher not fitting in the elevator at the patient's apartment complex. Upon arrival to the emergency department, she had already received cardiopulmonary resuscitation (CPR) for approximately 50 minutes, including an initial 10 minutes of chest compressions from her husband as instructed via telephone by the emergency operator.

The patient's cardiac rhythm was asystole and a mechanical chest compression device (MCCD) was used (Lucas2, Lund, Sweden). Focused bedside ultrasound ruled out cardiac tamponade, pneumothorax or a ruptured ectopic pregnancy, and revealed an extensively thrombosed right femoral vein. The patient was endotracheally intubated by the emergency physician, while a critical care physician inserted a resuscitative endovascular balloon occlusion of the aorta (REBOA) catheter in retrograde fashion via the left femoral artery under direct ultrasound guidance. Simultaneously, given the likely diagnosis of acute pulmonary embolism, the patient was given intravenous thrombolysis with 40 mg of Tenecteplase. Real-time monitoring of intra-arrest hemodynamic variables included arterial pressure measured from the intra-aortic catheter tip, end-tidal CO_2 ($EtCO_2$) from the endotracheal tube and cerebral tissue

Corresponding author:
Philippe Rola, Intensive Care Unit, Santa Cabrini Hospital, 5655 Rue St-Zotique, H1T1P7, Montreal, Quebec, Canada.
Email: philipperola@gmail.com

oxygen saturation ($SCTO_2$), which was measured with an Invos Somanetics device (Medtronic).

Resuscitative trans-esophageal echocardiography (TEE) was performed by a critical care physician to adjust chest compression position and confirm aortic zone 1 placement of the balloon (Figure 1).

TEE initially showed a lack of significant opening of the aortic valve during CPR both in mid-esophageal long axis and mid-esophageal aortic valve short axis views. The Lucas2 device was repositioned to improve the aortic valve opening during the rest of the resuscitation effort.

Return of spontaneous circulation (ROSC) was achieved within 10 minutes of aortic balloon occlusion, approximately 20 minutes after arrival in the emergency room. The MCCD was paused and adequacy of circulation assessed. The arterial line tracing revealed a systolic blood pressure of 105 mmHg and TEE demonstrated cardiac contractions. The aortic balloon was deflated over 10–15 seconds and an epinephrine infusion started. Clinicians subsequently noted a decrease in systolic blood pressure to approximately 40 mmHg, diastolic blood pressure levels at approximately zero mmHg, $EtCO_2$ from 27 mmHg to 15 mmHg and the onset of progressive bradycardia. In order to maintain perfusion, chest compressions were resumed with a resultant compression phase blood pressure of approximately 60 mmHg. The aortic balloon was then re-inflated with immediate improvement of compression-phase blood pressure to 115–125 mmHg, decompression-phase blood pressure at approximately 20 mmHg and return of $EtCO_2$ to 24–26 mmHg (Figure 2).

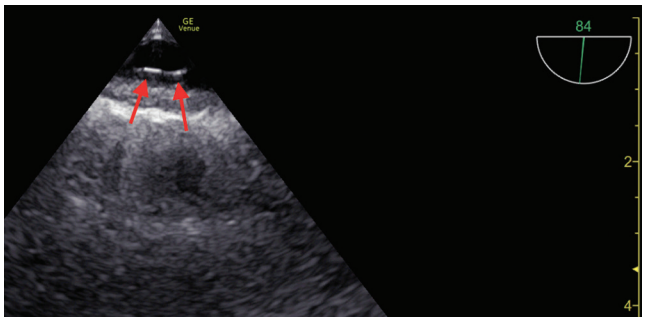

Figure 1 Tip of REBOA catheter (red arrows) in descending thoracic long axis view of the aorta. REBOA, resuscitative endovascular balloon occlusion of the aorta.

At this point, centers with capability to perform extracorporeal membrane oxygenation (ECMO) were contacted. However, because the patient's resuscitation efforts had lasted more than 70 minutes, combined with an initial s-lactate above 15 mmol/l, transfer was declined unless clinical improvement occurred. The clinicians at the bedside decided that due to the patient's young age, a positive outcome was still possible, and briefly conferred amongst themselves to optimize their resuscitation strategy. They determined that augmenting cardio-cerebral perfusion in the hopes of reaching sustainable cardiac performance would ideally lead to improved s-lactate measurements and sufficient clinical improvement to permit transfer to an ECMO center. Resuscitation efforts continued with the MCCD and aortic occlusion. A more detailed TEE examination revealed a dilated right ventricle and atrium as well as large mass on the tricuspid valve (Figure 3). In the clinical context, this was assumed to be a clot-in-transit, an echocardiographic finding that is near diagnostic for pulmonary embolism.

In an effort to prevent irreversible damage to splanchnic organs, the balloon was slowly deflated approximately every 10 minutes for about 1 minute, or until systolic blood pressure dropped below 50 mmHg, a level at which it was assumed that coronary perfusion pressure would be incompatible with maintaining cardiac activity and would likely devolve to asystole [1].

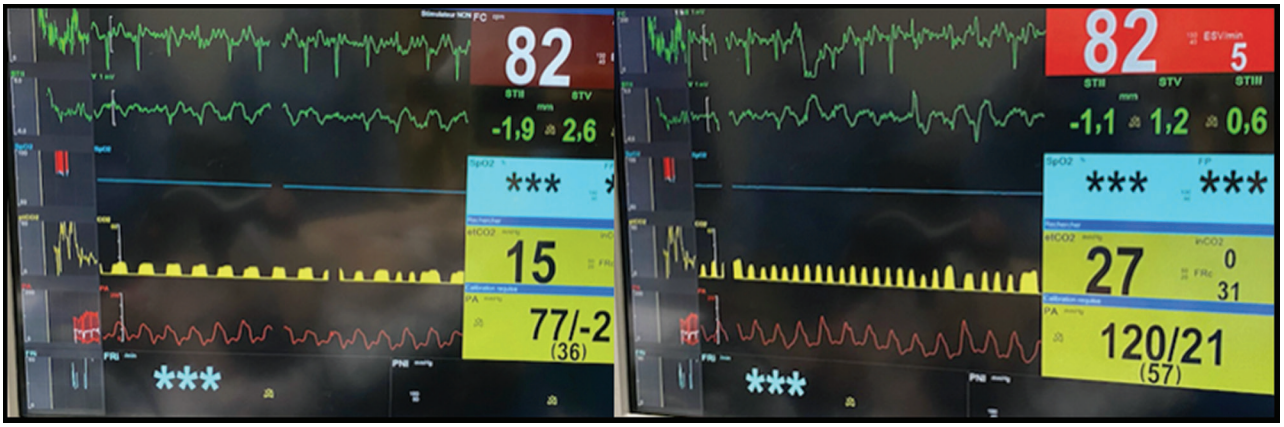

Figure 2 $EtCO_2$ and blood pressure values after REBOA deflation (left) and following re-inflation (right). $EtCO_2$, end tidal CO_2. REBOA, resuscitative endovascular balloon occlusion of the aorta.

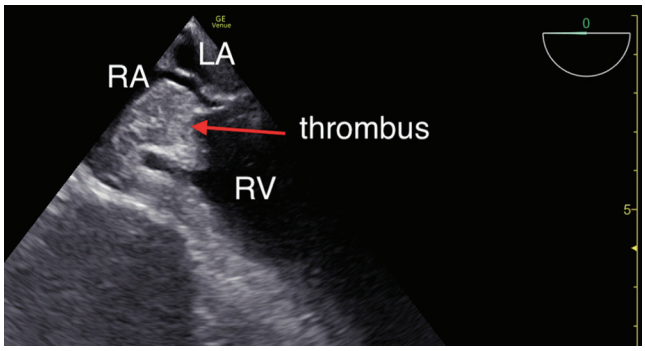

Figure 3 TEE view in mid-esophageal 4 chamber with rotation towards right sided chambers showing a large (>2 cm) mass attached to the tricuspid valve. TEE, trans-esophageal echocardiography.

In the ensuing 40 minutes, the 10:1-minute inflation:deflation cycles were repeated with serial reassessments of cardiac function by TEE, combined with a pause in chest compressions. In each of these deflation cycles, the blood pressure and $EtCO_2$ dropped and then responded to balloon reinflation (Figure 2). $SCTO_2$ dropped from 50–55% to 35–40% and responded to re-inflation. The s-lactate did not improve and remained above 15 mmol/l.

Resuscitative efforts were stopped after a total of approximately 110 minutes, in view of perceived futility and non-availability of ECMO support, and the patient passed away.

Ethical Approval and Informed Consent

The patient's next of kin provided informed consent for this case, including patient data and imaging, to be used for the purpose of science and teaching.

Given the nature of the case and extreme urgency, the critical care resuscitation physician decided that benefit outweighed risk and took the decision to perform REBOA cannulation. This was explained to the next of kin after the unsuccessful resuscitation, who was appreciative of the efforts made.

DISCUSSION

The last decade has seen a steady progress in ECMO-CPR (E-CPR) as an advanced adjunct to cardiac arrest resuscitation. As an example, the recent publication of the ARREST Trial by Yannopoulos et al. [2] has set a new standard for advanced resuscitation all over the world. Most importantly, the study highlights the possibility of better cardiac arrest outcomes, which have seen little improvement in the last two or three decades. However, there are many hurdles to overcome before E-CPR becomes widespread and available to most cardiac arrest patients.

Other interventions may significantly improve the quality of cerebral and coronary perfusion during resuscitation efforts. Optimizing circulation in these two vascular beds is the primary challenge in terms of both obtaining ROSC and a favorable neurologic outcome. Thoracic aortic occlusion with or without proximal perfusion have been the subject of several animal studies and human case reports and series, with human trials currently underway [3–6].

The physiology of aortic occlusion adjuncts is simple and elegant. It is well known that during optimal CPR, mechanical or manual, the cardiac output is only in the range of 25–40% of the pre-arrest values [7]. This low-flow state is often not sufficient to achieve adequate coronary and/or cerebral perfusion pressure. An increase in coronary perfusion pressure is associated with ROSC in humans [8]. The brain is susceptible to hypoxic injury – far more than rest of the body – and irreversible neurologic damage is hence the most common cause for post-resuscitation death [9]. Occlusion of the descending thoracic aorta decreases the vascular bed by more than two-thirds, thus increasing the perfusion of organs proximal to the occlusion, including heart, brain and upper extremities, via an increase in blood pressures in the aortic arch, coronary and cerebral arteries [10]. This is the rationale behind the use of REBOA to enhance CPR in non-traumatic cardiac arrest, as opposed to the original purpose and current indication, which is to control hemorrhagic shock. A recent case series by Brede et al. [4] showed that this procedure was feasible in the pre-hospital setting and resulted in an impressive rate of ROSC in patients with prolonged unsuccessful resuscitation efforts.

Another potential adjunct involves the infusion of oxygenated fluids, blood or saline, in a distally occluded aorta, termed the selective aortic arch perfusion technique [11]. This has been shown to work favorably in animal models but is not yet approved for use in humans.

The advantage of the REBOA approach is that it does not require the logistics and resources of E-CPR, nor the required level of training and experience in large gauge cannulation. Such competence is uncommon in community hospitals, where most cardiac arrests are managed. Physicians who regularly insert arterial lines or central vein catheters are familiar with the use of ultrasound and the Seldinger technique, and should be able to rapidly acquire the competence to insert a REBOA catheter. Structured training programs for this procedure are available [12].

Our institutional resuscitation team has been working towards E-CPR and advanced resuscitation techniques. While we do not routinely use REBOA in non-traumatic cardiac arrest, we decided to do so given the age of the patient and prolonged unsuccessful advanced cardiac life support (ACLS), in the hope of achieving ROSC.

While the outcome of this case was tragic, the observed dramatic improvement in physiological

parameters generated by a bedside technique that could be performed by most acute care physicians provides hope for the future. Our case achieved ROSC after balloon inflation despite a prolonged downtime, and demonstrated improvements in generated blood pressure and cerebral tissue saturation, as well as improved $EtCO_2$ similar to other studies [4].

This case also brings up some fascinating physiological questions and bedside clinical challenges. With the combination of MCCD and aortic occlusion, we obtained almost normal blood pressure. However, if the aortic balloon was deflated, ROSC was unable to be sustained, despite a high-dose epinephrine drip. While the team felt more comfortable with continuing the MCCD/REBOA approach despite ROSC, this would be at the cost of not perfusing the viscera and lower torso and limbs. One of the issues debated during this resuscitation was the feeling that since the liver was not perfused, lactate values were unlikely to improve. However, the clinicians determined that prioritizing coronary and cerebral flow to try to re-establish sustained cardiac performance took precedence over peripheral perfusion. The optimal balance between this can be debated. We empirically decided on a 10:1 time ratio for inflation:deflation, without real data to support this, only the assumption that cardiac perfusion had to take precedence, particularly given the diagnosis of pulmonary embolism.

REBOA-assisted circulation in massive pulmonary embolism has potential advantages, since coronary perfusion pressure is decreased by elevated right atrial pressure. Hence, a higher aortic pressure is required to maintain adequate right ventricular perfusion. The obstructive shock generated by massive pulmonary embolism occurs due to severe pulmonary hypertension and acute right ventricular failure. The combination of thrombolytics and REBOA may be a potential strategy to augment coronary perfusion pressure while thrombolysis, which is not instantaneous, relieves some of the obstruction and pulmonary hypertension. The optimal timing in relation to thrombolysis is unknown, but in our case happened almost simultaneously. We feel that either should not delay the other as thrombolysis is the only bedside therapy for massive pulmonary embolism, but that it should not be a contraindication for REBOA placement in arrest given the critical need for optimized aortic pressure due to the pathophysiology of pulmonary embolism.

At which point does the risk/benefit ratio tilt towards allowing distal perfusion at the cost of blood pressure and therefore coronary and cerebral perfusion pressures? The REBOA literature suggests 30–45 minutes as the upper margin of occlusion time, but can this easily translate to NTCA patients?

In the case series by Brede et al., the protocol mandated deflation when ROSC occurred, so as not to provide an increased afterload to the left ventricle and hinder cardiac output. However, if the cardiac output after ROSC is unable to maintain adequate arterial blood pressure, there may be an advantage in continuing aortic occlusion to maintain cardio-cerebral perfusion. This is a double-edged sword, with potentially improved myocardial perfusion due to increased aortic and subsequent coronary perfusion pressure on the one side, and increased afterload under which a stunned heart likely will succumb and lead to re-arrest on the other side. The best practice is currently not known. In our case, since TEE showed little difference in left ventricular function whether the REBOA was inflated or deflated, we assumed the issue was intrinsic rather than afterload related. The advent of REBOA catheters that allow for partial inflation may offer advantages and will certainly be the subject of further study, and whether a partial flow will extend the "safe" occlusion time is yet to be determined.

POSITION IN THE CHAIN OF RESUSCITATION

The recent development of endovascular resuscitation adjuncts raises organizational issues and challenges as well. The most important one is likely where they should fit in the chain of resuscitation.

In our opinion, the physiological benefit from REBOA-enhanced CPR would theoretically suggest it should be used in all non-traumatic cardiac arrest cases, as long as there is no delay to possible E-CPR treatment. REBOA-enhanced CPR may provide a bridge to a more definitive circulatory support such as ECMO or intra-aortic balloon pump (IABP) [3], or the introducer could easily be used to perform percutaneous coronary intervention, if appropriate. We propose a conceptual algorithm on how to introduce and position these interventions (Figure 4).

This assumes a patient who is a candidate for aggressive resuscitation including E-CPR but could also include other circulatory support strategies such as IABP or Impella-type devices, depending on availability and experience.

CONCLUSION

This case report describes the use of an adjunct treatment (REBOA) to cardiopulmonary resuscitation, which likely led to both ROSC and augmented objective parameters of resuscitation effect. E-CPR is not commonly available worldwide, even though it represents the highest quality of resuscitation and the best chance of survival, and REBOA-enhanced ACLS may provide the additional perfusion to obtain ROSC, or possibly a bridge to ECMO.

This case report additionally supports that more research is needed to answer many physiological and practical questions concerning the use of endovascular resuscitation in non-traumatic cardiac arrest.

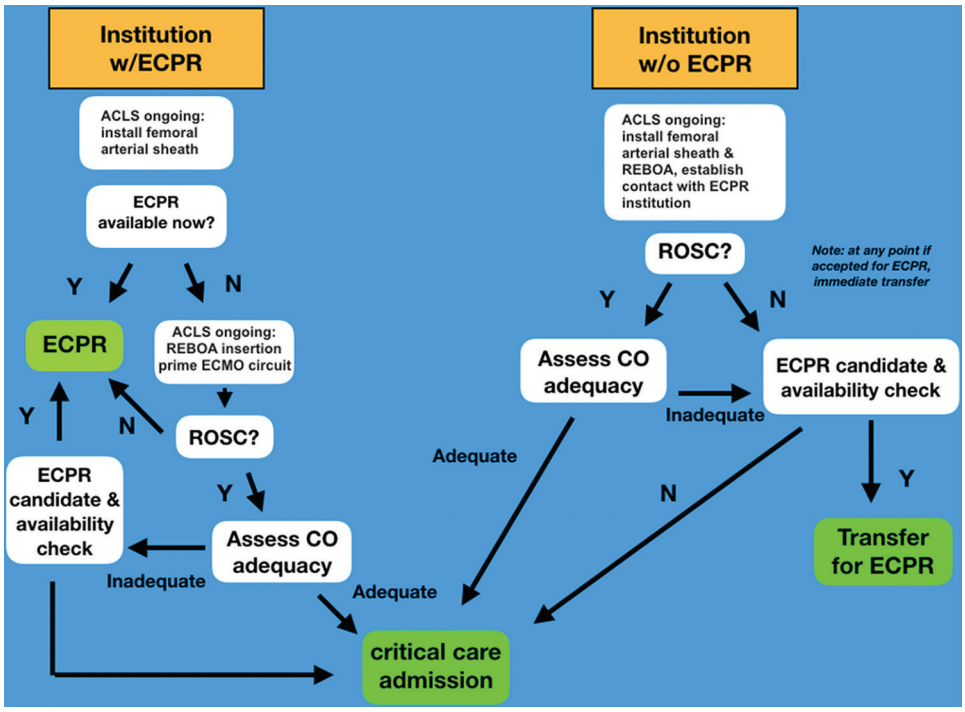

Figure 4 A proposed algorithm including REBOA-enhanced resuscitation. REBOA, resuscitative endovascular balloon occlusion of the aorta.

Acknowledgements

We wish to thank Dr. Hussein Fadlallah, cardiologist, Dr. Carol Zambrana, hospitalist, and Dr. Marie-Pier Talbot, emergency physician, for their assistance during this resuscitation.

Ethics Statement

(1) All the authors mentioned in the manuscript have agreed to authorship, read and approved the manuscript, and given consent for submission and subsequent publication of the manuscript.
(2) The authors declare that they have read and abided by the JEVTM statement of ethical standards including rules of informed consent and ethical committee approval as stated in the article.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

Author Contributions

All authors have substantially contributed to the article.

REFERENCES

[1] Stensaeth KH, Sovik E, Haig INY, Skomedal E, Jorgensen A. Fluoroscopy-free Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA) for controlling life threatening postpartum hemorrhage. PLoS ONE. 2017;12(3):e0174520.
[2] Yannopoulos D, Bartos J, Raveendran G, et al. Advanced reperfusion strategies for patients with out-of-hospital cardiac arrest and refractory ventricular fibrillation (ARREST): a phase 2, single centre, open-label, randomised controlled trial. The Lancet. 2020; 396(10265):1807–16.
[3] Coniglio C, Gamberini L, Lupi C, et al. Resuscitative endovascular balloon occlusion of the aorta for refractory out-of-hospital non-traumatic cardiac arrest–a case report. Prehosp Disaster Med. 2019;34(5):566–8.
[4] Brede J, Lafrenz T, Klepstad P, et al. Feasibility of pre-hospital resuscitative endovascular balloon occlusion of the aorta in non-traumatic out-of-hospital cardiac arrest. J Am Heart Assoc. 2019;8:e014394.
[5] Aslanger E, Golcuk E, Oflaz H, et al. Intraaortic balloon occlusion during refractory cardiac arrest. A case report. Resuscitation. 2009;80(2):281–3.
[6] Krüger AJ, Brede JR. The REBOARREST Trial: a randomised trial on resuscitative endovascular balloon occlusion of the aorta in non-traumatic out of hospital cardiac arrest. 2020. https://reboarrest.com/wp-content/uploads/2020/10/Signed-Study-protocol-REBOARREST-Trial-Version-1.1.pdf. Accessed 23 April 2021.
[7] Maier GW, Tyson Jr G, Olsen C, et al. The physiology of external cardiac massage: high-impulse cardiopulmonary resuscitation. Circulation. 1984;70(1):86–101.

[8] Paradis NA, Martin GB, Rivers EP, et al. Coronary perfusion pressure and the return of spontaneous circulation in human cardiopulmonary resuscitation. Jama. 1990;263(8):1106–13.
[9] Dragancea I, Rundgren M, Englund E, Friberg H, Cronberg T. The influence of induced hypothermia and delayed prognostication on the mode of death after cardiac arrest. Resuscitation. 2013;84(3):337–42.
[10] Daley J, Morrison JJ, Sather J, Hile L. The role of resuscitative endovascular balloon occlusion of the aorta (REBOA) as an adjunct to ACLS in non-traumatic cardiac arrest. Am J Emerg Med. 2017;35(5):731–6.
[11] Manning JE, Murphy Jr CA, Hertz CM, Perretta SG, Mueller RA, Norfleet EA. Selective aortic arch perfusion during cardiac arrest: a new resuscitation technique. Ann Emerg Med. 1992;21(9):1058–65.
[12] Brede JR, Lafrenz T, Krüger AJ, et al. Resuscitative endovascular balloon occlusion of the aorta (REBOA) in non-traumatic out-of-hospital cardiac arrest: evaluation of an educational programme. BMJ Open. 2019; 9(5):e027980.
[13] Deakin CD, Barron DJ. Haemodynamic effects of descending aortic occlusion during cardiopulmonary resuscitation. Resuscitation. 1996;33(1):49–52.