Case Report

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Resuscitative endovascular balloon occlusion of the aorta during non-ST elevation myocardial infarction: A case report

Eleanor E Curtis[1,2], Rachel M Russo[1], Eric Nordsieck[3], Michael Austin Johnson[4], Timothy K Williams[2], Lucas P Neff[2,5], Lisa Hile[6], Joseph M Galante[1] and Joseph J Dubose[1,2,7]

Abstract

Resuscitative endovascular balloon occlusion of the aorta (REBOA) is a hemorrhage control technique that is increasingly being adopted for the management of noncompressible bleeding. In addition to limiting hemorrhage, REBOA increases blood flow to the heart, lungs, and brain. A small number of case reports and animal studies describe the use of REBOA to increase coronary perfusion during cardiopulmonary resuscitation. We report a case in which REBOA may have reversed ST-segment abnormalities during a Type II non-ST elevation myocardial infarction (NSTEMI) in a patient with previous trauma. We describe the presentation, course, and decision making that contributed to the use of REBOA in this case. Additionally, we will present a review of the literature on the effects of REBOA on coronary perfusion.

Keywords

REBOA, cardiac arrest, ACLS, aortic balloon

Introduction

Resuscitative endovascular balloon occlusion of the aorta (REBOA) is a growing technique for the salvage of exsanguinating trauma patients. In addition to controlling distal hemorrhage, animal models demonstrate that REBOA increases blood flow to the heart, lungs, and brain, and improves coronary circulation,[1] but limited literature exists regarding the effect of REBOA on coronary perfusion in humans. We report a case of REBOA usage and hypothesize increased coronary perfusion in a trauma patient undergoing a Type II non-ST elevation myocardial infarction (NSTEMI).

Case report

A 71-year-old woman was brought to the emergency department by ambulance from the scene of a head-on motor vehicle collision. She was hypotensive on arrival with a blood pressure (BP) of 65/48 mmHg, yet responsive to fluids with an increase in BP to 185/89 mmHg after 1L of plasmalyte. Focused assessment with sonography for trauma (FAST) was nondiagnostic. As the source of her initial instability and prompt fluid responsiveness was not clear, she was prepared for CT of the

[1]Department of Surgery, University of California Davis Medical Center, Sacramento, CA, USA
[2]Department of Vascular and Endovascular Surgery, David Grant Medical Center, Fairfield, CA, USA
[3]Department of Cardiology, University of California Davis Medical Center, Sacramento, CA, USA
[4]Department of Emergency Medicine, University of California Davis Medical Center, Sacramento, CA, USA
[5]Department of Pediatric Surgery, Emory University School of Medicine, Atlanta, GA, USA
[6]Department of Emergency Medicine, Johns Hopkins University, Baltimore, MD, USA
[7]Department of Vascular Surgery, University of California Davis Medical Center, Sacramento, CA, USA

Corresponding author:
Eleanor E Curtis, Department of General Surgery, Harborview Medical Center, 325 9th Ave, Seattle, WA 98104, USA.
Email: eleanorc@uw.edu

head through pelvis. As a precaution, due to her difficult airway secondary to her body habitus, she was intubated using Rocuronium and Etomidate but developed profound hypotension followed by immediate pulseless cardiac arrest (PEA). Cardiopulmonary resuscitation was initiated and blood transfusion including two units of packed red blood cells (PRBCs), one unit of fresh frozen plasma (FFP), and one unit of pooled platelets were delivered through a Belmont rapid infuser (Belmont Instruments, Billerica, MA). Simultaneously, bilateral tube thoracostomies were performed without return of blood or significant air from either side. Arterial access was obtained by placing a 5F micropuncture sheath (Boston Scientific Corporation, Natick, MA) in the right common femoral artery under ultrasound guidance in anticipation of possible REBOA placement. After 2 min of CPR and 1 mg of epinephrine the patient had return of spontaneous circulation (ROSC) with blood pressure of 144/56 by arterial line. Hemoglobin demonstrated an appropriate rise from 9.4 g/dL on admission, to 11.4 g/dL post code, initial troponin was 0.97 ng/mL; repeat FAST was nondiagnositic. The patient's blood pressure was stable and the decision was made to transport to CT, which revealed small bilateral pneumothoraces, free intrabdominal air without free fluid, a mesenteric hematoma, and multiple fractures of bilateral upper and lower extremities (Figure 1).

Two hours after ROSC, while awaiting OR availability and after massive transfusion products had been returned to the blood bank, the patient again became hypotensive with a systolic blood pressure of 45 mmHg on arterial line monitor. Cardiac tracing on the bedside monitor demonstrated ST segment depressions, which were confirmed with electrocardiogram, as was a significant ST segment elevation in aVR. (Figure 1). At this time, the decision was made to use a REBOA catheter to limit any ongoing blood loss from her lower extremity injuries, which were too proximal for adequate control with tourniquets. The previously placed micropuncture sheath was exchanged for a 7Fr × 13 cm introducer sheath (Boston Scientific Corporation, Natick, MA) using Seldinger technique and a low-profile REBOA catheter (ER-REBOA,

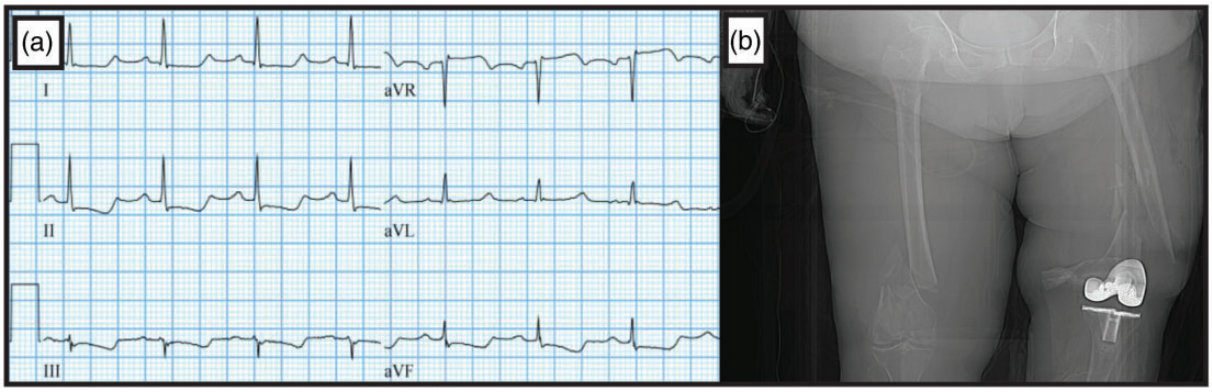

Figure 1. ECG of NSTEMI event and scout film of lower extremity injuries.

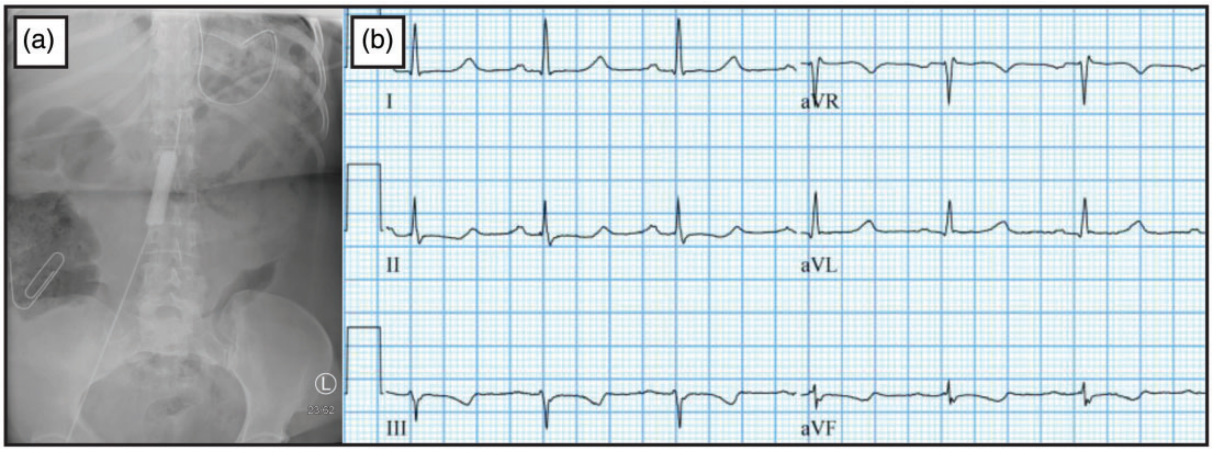

Figure 2. Inflated REBOA and ECG showing resolution of changes.

Prytime Medical, Boerne, TX) was positioned in the infra-renal aorta (zone 3) using external anatomic landmarks[2] and the location confirmed with an abdominal X-ray (Figure 2). The placement shows the balloon at L2 and when compared with patient's prior abdominal CT, confirmed a zone 3 placement. Proximal and distal aortic blood pressures were transduced through the central lumen of the REBOA catheter and the side port of the 7Fr sheath. The balloon was inflated until the distal arterial waveform was lost, to provide complete occlusion. Proximal blood pressure improved to 94/47 and remained at that level or higher throughout REBOA. A repeat ECG demonstrated resolution of the ST depressions (Figure 2).

Empiric transfusion of two units of PRBCs and one unit of FFP was initiated approximately 5 min after the onset of REBOA, when the products became available. After 10 min blood pressure had improved to 128/83 and blood product administration was underway. The REBOA balloon was partially deflated until a distal arterial waveform was present in order to theoretically facilitate gradual washout of ischemic metabolites and avoid further cardiac instability. Throughout this 2-min period of partial REBOA, systolic blood pressure was sustained above 100 mmHg with no further evidence of ST segment abnormalities on cardiac monitoring.

The balloon was then completely deflated over 2 min, but remained in position until the patient arrived to the operating room. She underwent exploratory laparotomy for perforated viscous as well as external fixation of her orthopedic injuries without the need for fasciotomy or amputation. Prior to the removal of the 7Fr sheath, an intraoperative angiogram of both lower extremities confirmed no evidence of arterial injury or thrombosis. The sheath was then removed and pressure was held for 30 min. Twenty-eight hours after her Type II NSTEMI, Troponin-I measurements peaked at 14 ng/mL. The patient was extubated on post-injury day 7 and transferred to another hospital to complete her recovery on post-injury day 14. At the time of transfer she was hemodynamically stable and neurologically intact, awaiting cardiac catheterization. Records from the receiving facility were unavailable at the time of this publication.

Discussion

This is the first case report to capture reversal of cardiac ischemia illustrated by ST-segment depressions and an isolated ST-elevation in aVR by ECG during the application of REBOA, in a patient diagnosed with a type II NSTEMI. Myocardial injury was confirmed by elevated troponins in a rise and fall pattern with associated ST changes[3] (ST depression in leads II, III, aVF, and ST elevation in aVR) (Figures 1 and 2). Upon inflation of REBOA the patient's ECG returned to normal and remained stable after balloon deflation. ST elevation in lead aVR is of particular interest as this has been associated with increased 90-day mortality in STEMI patients.[4] The cause of the NSTEMI in this case was thought to be secondary to traumatic hemorrhage leading to demand ischemia. As evidenced by elevated troponin upon admission, the patient may have been experiencing myocardial ischemia prior to arrival or may have had a component of cardiac contusion. Although it is impossible to determine the contribution of each factor to the total ischemia represented by the peak troponin, the reversal of the ECG changes with REBOA may suggest a significant contribution from inadequate coronary perfusion. Direct injury to the myocardium may contribute to troponinemia, but would not cause ischemic changes on an ECG. It is important to note that the indication for REBOA in this instance was to avoid impending cardiac arrest due to presumed hemorrhagic shock, rather than for the explicit purpose of augmenting coronary perfusion pressure and reversing myocardial ischemia due to NSTEMI.

As the reversal of ECG changes in the present case was seen after REBOA was initiated but prior to blood product transfusion, there is evidence to suggest that coronary circulation was directly improved by REBOA through a variety of potential mechanisms: balloon inflation reduced the volume of distribution of circulating blood, which may have shunted more blood volume to the coronary arteries; increased aortic afterload during occlusion may have improved retrograde coronary filling during diastole. The balloon was inflated in an attempt to control further hemorrhage from her lower extremity fractures and limiting ongoing blood loss may reduce anemia and hypovolemia-related coronary malperfusion. The administration of blood products as they became available supported coronary perfusion, allowing the controlled cessation of REBOA without return of myocardial ischemia. These findings are consistent with animal studies that demonstrate REBOA increases coronary perfusion, and when instituted concurrently with blood transfusion, may increase blood flow to proximal tissue beds faster than blood product administration alone.[5]

Despite growing evidence that REBOA may improve coronary perfusion during cardiac arrest,[6] it has not previously been used as an adjunct to augment proximal pressure during critical care. One reason that it has not been used to augment coronary perfusion in patients without active hemorrhage, is that occlusion of the descending thoracic aorta can induce significant ischemia to distal tissues.[7,8] Most studies on the effect of intra-aortic balloon occlusion on coronary perfusion examine occlusion of the descending thoracic

aorta (zone 1), which may lead to prohibitive distal ischemia.[9–11] However, there is growing clinical evidence that infra-renal, zone 3, aortic occlusion, as was used in this case, may provide some degree of cardiac support with less distal ischemia.[12] Other methods of increasing blood flow to the heart with REBOA while decreasing distal ischemia have been studied in animal models, including intermittent and partial REBOA.[7] Our experience in this single case suggests that zone 3 aortic occlusion alone or in combination with partial REBOA may confer cardiovascular support in humans as well; however, no comparative studies exist to compare the various methods of aortic occlusion to standard means of coronary support in humans. In the United States, Brenner et al.[13] has described the largest experience using REBOA for traumatic arrest. As all of these patients received zone 1 aortic occlusion, supra-celiac balloon positioning is recommended over infra-renal occlusion when using REBOA for coronary support of patients in arrest or with impending arrest. Our experience in the case may suggest that zone 3 occlusion could be a reasonable option to provide some coronary support when zone 1 occlusion results in excessive after load or intolerable distal ischemia. Similar to the Japanese experience, a reasonable protocol may be to perform zone 1 occlusion followed by zone 3 occlusion if there is an undesirably exaggerated response for zone 1 occlusion.

We chose to follow zone 3 occlusion with a period of partial REBOA. The transition from complete to partial occlusion allowed for gradual reperfusion and alterations to circulation while blood products were concurrently administered. Theoretically, this technique may avoid drastic fluctuation in blood flow and hemodynamics, and decrease the circulating concentration of ischemic metabolites, thereby reducing the risk of myocardial dysfunction.[14] At present these benefits are only theoretical; however, this hypothesis is an area of ongoing research.

Conclusion

This is the first case of documented ECG waveform improvement during the use of a zone 3 REBOA as part of an overall resuscitation strategy for an elderly patient with a NSTEMI after blunt traumatic injury. While an interesting finding, further investigation should be undertaken to determine if there is a role for REBOA when augmentation of coronary perfusion is required.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article. The views expressed in this material are those of the authors, and do not reflect the official policy or position of the U.S. Government, the Department of Defense, the Department of the Air Force, or the University of California Davis.

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Informed consent

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Ethical approval

University of California Davis does not require ethical approval for reporting individual cases or case series

Guarantor

EC

Contributorship

EC and RR performed the literature review, wrote and maintained the article through multiple iterations. EN and LH provided cardiology discussion references and reviewed the article. MJ created figures. JD suggested study design and was part of all reviews and editing and response to reviewers of the article. JG, LN, and TW edited and provided input on multiple revisions and approved the final article.

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