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Endovascular repair as a rescue strategy to restoring the extracorporeal membrane oxygenation flow

Filip Depta[1]*, Dušan Rybár[1], Ivan Kopolovets[2], Matej Moščovič[3], Tomáš Grendel[1]

Departments of [1]Critical Care, [2]Vascular Surgery and [3]Cardiology, East Slovak Institute of Cardiovascular Diseases, Košice, Slovakia
**Corresponding author*

Abstract:

Ventricular septal defect (VSD) is a known complication after myocardial infarction associated with high mortality. Extracorporeal membrane oxygenation (ECMO) is being successfully used in patients with VSD as a bridge to definitive surgical repair. Although often the only possibility to stabilize hemodynamics and oxygenation, ECMO has many potential complications, carrying significant morbidity and mortality. Here, the patient presented with a postinfarct VSD on peripheral venoarterial ECMO who developed a dissection of the common iliac artery (CIA) on the 5th day after ECMO implantation. As a result, a sudden drop in ECMO flow has become evident along with high pressures in the arterial cannula. After a definitive diagnosis of a CIA lesion obstructing the blood flow was made, trans-ECMO endovascular repair of CIA was performed. Four days after endovascular repair, we encountered the same problem of decreased blood flow associated with stent kinking and were approached with another endovascular repair to re-establishing full ECMO flow.

Keywords:

Common iliac artery, endovascular repair, extracorporeal membrane oxygenation, ventricular septal defect

Introduction

Ventricular septal defect (VSD) is a well-recognized mechanical complication of acute myocardial infarction with a mortality rate over 90%.[1] Although both the percutaneous approach and surgical repair have been described as a mode of definitive treatment, the appropriate timing remains controversial.[2] Venoarterial extracorporeal membrane oxygenation (VA ECMO) as a bridge to definitive surgical repair of VSD has been well described, although it has many potential complications, including significant morbidity and mortality.[3] However, to the best of our knowledge, a case report describing endovascular repair of arterial obstruction and thus restoring sufficient ECMO flow has not been published to date.

Case Report

A 69-year-old obese female (body mass index 37.4) presented to the emergency department due to acute onset chest pain suggestive of ST-elevation myocardial infarction. She was transported to the cath-lab where percutaneous coronary intervention (PCI) was performed, and a drug-eluting stent was inserted into the occluded right posterior descending artery. After PCI, her vitals were stable, and she was transferred to the coronary unit for

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ORCID:
FD: 0000-0002-2854-0450
DR: 0000-0003-0803-3213
IK: 0000-0002-2721-6114
MM: 0000-0001-7134-2049
TG: 0000-0002-7583-9789

Address for correspondence:
Dr. Filip Depta, Department of Critical Care, East Slovak Institute of Cardiovascular Diseases, Košice, Slovakia.
E-mail: fdepta@protonmail.com

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further management and observation. At admission, transthoracic echocardiography (TTE) revealed a preserved left ventricular ejection fraction of 50% and hypokinesis of the basal half of the inferior wall with no significant valvular abnormalities.

The same day at night, she suddenly developed dyspnea (respiratory rate 40 breaths per minute), hypoxemia (oxygen saturation of 80% on 10 liters per minute [lpm] O2 mask) and lactate of 8.7 mmol/l. Hypotension (blood pressure 60/40 mmHg) required norepinephrine (0.25 ug/kg/min) to maintain mean arterial pressure of 65 mmHg. Emergency TTE was performed visualizing VSD with the size of 27 mm. TTE revealed no fluid in pericardial space. Due to the development of cardiogenic shock, ultrasound-guided insertion of peripheral VA ECMO (Cardiohelp, Maquet, Getinge AB, Sweden) was performed percutaneously by experienced intensivists on the first attempt. A 23Fr venous cannula was inserted through the right femoral vein ending at the cavoatrial junction. A 17Fr arterial cannula was inserted into the left femoral artery (total length of insertion was 23 cm from the skin puncture), with the tip of the cannula ending at the border of common iliac artery (CIA) and external iliac artery. ECMO flow was smoothly initiated with a flow of 4 lpm at 3600 rotations per minute (rpm) with anticoagulation initiated and maintained with unfractionated heparin to target activated partial thromboplastin time of 50–70 s. Somatic oximeter sensor pads (INVOS, Medtronic, USA) were placed on both legs to monitor perfusion of the lower extremities as per the local protocol.

On the 5th day after ECMO insertion, a marked drop in flow was observed (flow 2.3 lpm, 3600 rpm) along with high pressures in the arterial cannula (360 mmHg). Hemodynamics worsened in terms of tachycardia, the norepinephrine requirements doubled (0.2 ug/kg/min) to maintain a mean arterial pressure of 65 mmHg, and she developed dyspnea. With the sudden loss of sufficient ECMO output along with high arterial cannula pressures, the differential diagnosis included malfunction (kinking or thrombosis) of the arterial cannula, clogged ECMO oxygenator, or ECMO pump head. Since there was a normal pressure difference between internal and arterial pressures and proper anticoagulation, oxygenator problems or clogged pump head became very unlikely, and therefore, a decision to replace the arterial cannula was made. The patient was transferred to the operation room and subjected to general anesthesia. First, a guidewire to the right femoral artery was inserted for safety reasons if any problem occurred during the replacement of the arterial cannula. Second, under the assumption of a kinked arterial cannula and to prevent complete ECMO arrest, a guidewire was inserted through the plastic side of an arterial ECMO cannula. Surprisingly, the arterial cannula was patent without occlusion, but the obstruction became evident deeper at 35 cm from the skin entry. Therefore, angiography was performed through the arterial cannula, revealing focal dissection of the left CIA with thrombus apposition. The dissection was confined to the left CIA only, without progression proximally or distally with an arterial ECMO cannula positioned in the true lumen.

As a result, the CIA became subtotally occluded, with almost the entire ECMO flow being recirculated through the internal iliac artery back to the left leg [Figure 1a]. This explained the sudden drop in ECMO flow and high arterial cannula pressures. At the same time, INVOS monitoring on the left leg showed supranormal levels of 92%, while the right leg displayed constant values of 58%, supporting the hypothesis of recirculation in the left lower extremity.

Due to hemodynamic instability, we decided to perform an endovascular repair of the CIA. A 6Fr sheath was inserted from the plastic side of the arterial cannula [Figure 1b] near the puncture site, and the guidewire was introduced. Then, stenting (Sinus-SuperFlex-535 10 mm × 40 mm, Optimed, Germany) of the CIA was performed. Ultimately, a brief ECMO stop was necessary to replace the actual arterial cannula, which was damaged by the inserted sheath. Finally, full ECMO flow was restored (flow 4.2 lpm, 3600 rpm), and the pressure in the arterial cannula was normalized to 240 mmHg.

Four days after endovascular repair, we registered the same problem of decreased ECMO flow with high pressure on the arterial line, raising the suspicion of stent malfunction. Unfortunately, stent kinking was visualized with angiography, occluding the left CIA [Figure 2a]. Then, endovascular repair with a wider and longer stent (Vena Zilver 14 mm × 60 mm, Cook Medical, Denmark) was performed via the left brachial artery. At the time of intervention, multiple brief ECMO flow stops were necessary to allow angiographic visualization using native circulation. Successful stent insertion

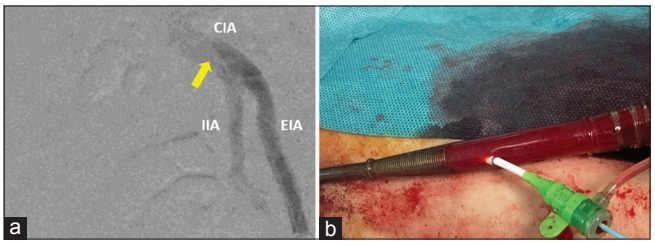

Figure 1: (a) perioperative angiography showing focal dissection (arrow) and occlusion of the left CIA and subsequent recirculation of blood in the left leg, (b) "trans-ECMO" approach with 6Fr sheath inserted in the arterial ECMO cannula, CIA – common iliac artery, IIA – internal iliac artery, EIA – external iliac artery. ECMO = Extracorporeal membrane oxygenation

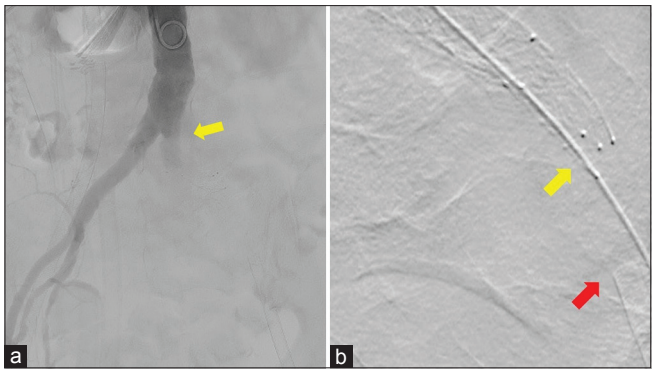

Figure 2: (a) Native circulation angiography through left brachial artery confirming left CIA occlusion (arrow) due to stent malfunction, (b) stent insertion through left brachial artery to the left CIA, yellow arrow – distal end of the stent, red arrow – end of the arterial ECMO cannula (distance between the arrows is 3 cm), CIA: common iliac artery, ECMO = Extracorporeal membrane oxygenation

was confirmed [Figure 2b], and full ECMO flow with normalized pressures was restored once again.

On day 9, there was a spontaneous rupture of the free wall of the right ventricle resulting in massive cardiac tamponade and virtually no ECMO flow which necessitated cardiopulmonary resuscitation, emergent sternotomy, and multiple transfusions. Despite eventually restoring ECMO flow, the patient remained unconscious after surgery, with computed tomography showing multiple areas of ischemia and hemorrhage in both hemispheres. The patient died of multiple organ failure on day 14 while still on ECMO. The patient has given a written consent approving the publication while alive.

Discussion

ECMO-related vascular complications are common, accounting for 7%–14% of all ECMO patients.[4,5] Although the Extracorporeal Life Support Organization has issued a various well-described guidelines for both, respiratory and circulatory support, clear guidelines on managing vascular complications are missing. Evidence of arterial or venous lesions treated by endovascular repair in patients with ECMO in the literature is scarce and mostly limited to case reports.[6-8]

Natural and supported flow counteract each other in most patients on ECMO, while in those with severely impaired cardiac performance, ECMO becomes the sole means of whole-body perfusion. Therefore, nonpulsatile retrograde ECMO flow presents a challenge for diagnostics (i.e. radiocontrast agent flow direction) as well as management (i.e. endovascular repair). During endovascular repair, every decrease of ECMO flow carries the risk of systemic hypoperfusion, and thus, endovascular repair while maintaining ECMO flow seems to be a potential treatment option.

We described a patient with repeated left CIA obstruction in whom endovascular repair using a stent was eventually successful to restore sufficient ECMO flow. We approached obstruction of left CIA by initially using "trans-ECMO" approach. We chose this approach because at first, there was a significant risk of complications due to the dissection of the left CIA if we approached CIA from the contralateral femoral artery. Second, we were assuming a kinked arterial cannula what was later found not to be the case. This approach ultimately damaged the arterial cannula that necessitated replacement. The possible advantage of this method was ECMO flow preservation during endovascular repair and reduction of the risk of hypoperfusion during the procedure.

Later, when decreased flow became evident again, the brachial approach was attempted. Such approach, requiring the native circulation for contrast visualization, carries the risk of severe global hypoperfusion as ECMO flow needs to be stopped. Patients having severely insufficient native circulation are therefore most at risk.

With repeated angiography, stent malfunction became evident, and we may only hypothesize the causes of stent kinking. Multiple factors were likely responsible, such as selection of the improper size or length of the stent, or the distance between the stent and outflow of the arterial cannula (in our case, it was 1, 5 cm). Furthermore, high blood velocity and pressure at the tip of the arterial cannula with proximity to the stent may also be responsible for its deformation. For the second intervention, we used a venous stent because the CIA diameter was larger than the biggest available arterial stent that we possessed. To the best of our knowledge, we could not identify any ECMO-related equipment (i.e. components or cannulas with integrated sheath), that would enable "trans-ECMO" endovascular repair of vascular lesions.

Conclusion

This case demonstrated that there are more options to restore insufficient ECMO flow if severe vascular lesions occur. Delayed-onset arterial occlusion should be a part of differential diagnosis of sudden unexpected low ECMO flow with high pressures in the ECMO. Somatic oximetry monitoring may support and aid the timely diagnosis. Trans-ECMO endovascular approach with adequate equipment and proper stent selection may become a therapeutic choice to restore sufficient ECMO flow with less risk of hypoperfusion when compared to the conventional approach.

Author contributions statement (CRediT author statement)
FD: Conceptualization, Resources, Visualization, Writing - original draft, Writing, Reviewing and Editing TG, DR, MM, IK: Resources,

Writing, Reviewing TG, DR, MM, IK: Writing – Extensive Review and Editing.

Conflicts of interest
None Declared.

Ethical approval
Independent Ethics Committee of East Slovak Institute for Cardiovascular diseases approved the publication (No. A1062022) on June 23, 2022.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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