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Double Peripheral Venous and Arterial Cannulation for Extracorporeal Membrane Oxygenation in Combined Septic and Cardiogenic Shock

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Patient: Female, 15
Final Diagnosis: Septic shock
Symptoms: Hypotension • lactic acidosis • respiratory insufficiency • tachycardia
Medication: Linezolid
Clinical Procedure: Veno-arterial extracorporeal membrane oxygenation
Specialty: Critical Care Medicine

Objective: Unusual clinical course

Background: The use of venoarterial extracorporeal membrane oxygenation (va-ECMO) via peripheral cannulation for septic shock is limited by blood flow and increased afterload for the left ventricle.

Case Report: A 15-year-old girl with acute myelogenous leukemia, suffering from severe septic and cardiogenic shock, was treated by venoarterial extracorporeal membrane oxygenation (va-ECMO). Sufficient extracorporeal blood flow matching the required oxygen demand could only be achieved by peripheral cannulation of both femoral arteries. Venous drainage was performed with a bicaval cannula inserted via the left V. femoralis. To accomplish left ventricular unloading, an additional drainage cannula was placed in the left atrium via percutaneous atrioseptostomy (va-va-ECMO). Cardiac function recovered and the girl was weaned from the ECMO on day 6. Successful allogenic stem cell transplantation took place 2 months later.

Conclusions: In patients with vasoplegic septic shock and impaired cardiac contractility, double peripheral venoarterial extracorporeal membrane oxygenation (va-va-ECMO) with transseptal left atrial venting can by a lifesaving option.

MeSH Keywords: Extracorporeal Membrane Oxygenation • Leukemia, Myeloid, Acute • Shock, Cardiogenic • Shock, Septic

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Background

Venoarterial extracorporeal membrane oxygenation (va-ECMO) is a promising therapeutic option in septic shock, especially in the presence of septic cardiomyopathy and low cardiac output [1,2]. However, in states of septic shock with preserved left ventricular function, so-called distributive or vasodilatory shock, the use of peripheral va-ECMO has been less encouraging [3]. In these circumstances, an already increased cardiac output might not suffice to achieve sufficient organ perfusion, and peripheral va-ECMO blood flow may not adequately support the heart. This phenomenon is further exaggerated in patients that can only be equipped with small-bore ECMO cannulas due to small blood vessels. The current case report describes the successful treatment of combined septic and cardiogenic shock utilizing a double peripheral va-ECMO (va-va-ECMO) with left atrial discharge.

Case Report

A 15-year-old girl (168 cm, 57 kg) with acute myelogenous leukemia (AML) received standard induction treatment consisting of 2 cycles of chemotherapy according to AML-BFM guidelines. After the second cycle of chemotherapy, she developed pancytopenia. She received a broad anti-infective treatment for an infection with unknown focus with ciprofloxacin, linezolid, meropenem, tobramycin, and liposomal amphotericin. After 3 weeks of treatment and declining signs of infection, she developed tachycardia (140/min) and hypotension (75/41 torr), as well as lactic acidosis. She was therefore admitted to the pediatric intensive care unit.

Transthoracic echocardiography revealed a severely impaired left ventricular ejection fraction (LVEF) of 12% without dilatation of the left ventricle (LVIDd 38 mm). The electrocardiogram showed sinus tachycardia with incomplete right bundle branch block. N-terminal prohormone of brain natriuretic peptide (NT-proBNP) was 19 384 pg/ml (normal range 0–153 pg/ml) and high-sensitive troponin T 53.7 pg/ml (normal range 0–14 pg/ml). In addition to fluid therapy, noradrenaline, dobutamine, and milrinone were started. Deep sedation and mechanical ventilation had to be initiated due to respiratory insufficiency. Lactate acidosis worsened (16 mmol/l, pH 6.91) and she remained hypotonic (50/30 torr) with a heart rate of 160/min. Therefore, va-ECMO was initiated via cannulation of the left femoral artery and vein (13 Fr/15 cm arterial and 21 Fr/55 cm venous HLS Cannula, Maquet GmbH, Rastatt, Germany) per Seldinger technique under sonographic guidance. A 9 Fr sheath (Arrow, Reading, PA, USA) was placed into the left superficial femoral artery to ensure distal leg perfusion since the diameter of the artery (about 3–4 mm) was very small. Va-ECMO (Cardiohelp System with a 7.0 HLS Set Advanced Bioline Coating (Maquet GmbH)) was applied at a blood flow of 3.3 l/min at 3800 rpm. Extracorporeal blood flow (ECBF) could not be increased further, since pre-oxygenator membrane pressure was above 350 torr. Mean arterial pressure (MAP) was at about 45 torr, with a concomitant administration of noradrenaline (80 µg/min), vasopressin (2 units/h), levosimendan (0.5 mg/h), and hydrocortisone (200 mg/d). Residual LVEF remained at about 10–15%. Continuous veno-venous hemodiafiltration (CVVHDF) was started because of anuric acute kidney injury. Anti-infective therapy was continued with dose-adjustment of meropenem, ciprofloxacin, metronidazole, cotrimoxazole, liposomal amphotericin B, and acyclovir. Linezolid was replaced by vancomycin. Procalcitonin (PCT) was 2.37 ng/ml (normal range 0–0.5 ng/ml) and C-reactive protein (CRP) 2.98 mg/dl (normal range 0–0.5 mg/dl).

On the second day with va-ECMO, the mean arterial blood pressure (MAP) declined to 40 torr despite increasing doses of vasopressors (noradrenaline 144 µg/min, vasopressin 4 IE/h). The LVEF further decreased below 10%. Levosimendan infusion was stopped after 21 h without any positive effect. Furthermore, acute ischemic injury of the non-cannulated leg developed. The right femoral artery was dissected in the groin. This revealed no embolic event, but there was insufficient blood inflow due to low cardiac output, causing the leg ischemia. In addition, lactate levels rose to 25 mmol/l. Therefore, a Dacron® conduit was sewed on the right femoral artery and a second arterial cannula (13 Fr/15 cm) introduced through the conduit was connected to the ECMO circuit per Y-connector to support perfusion of the right leg as well as to augment the ECBF. With 2 arterial ECMO lines, ECBF could be enhanced to 4–5 l/min, with tolerable ECMO inflow pressures below 300 torr. With rising ECMO blood flow, the MAP could be maintained at 40–50 torr. The ECBF was now limited by venous drainage pressures below –100 mmHg. Sufficient preload within the right atrium and vena cava was frequently verified by echocardiography. Inspiratory oxygen fraction of the respirator was increased up to 0.55 to prevent harlequin syndrome because of impaired lung function. The arterial oxygen partial pressure in blood drawn from the right radial artery was normal.

PCT levels rose to 42.5 ng/ml and the Hickman catheter was explanted as a potential septic focus. However, no causal organism was identified.

Broad-complex tachycardia occurred, and was treated with esmolol and metoprolol. Left ventricular function further decreased, and the aortic valve ceased to open. To prevent left ventricular overdistension, pulmonary edema, and even intracardiac clot formation, a second venous cannula (21 Fr/55 cm) was now placed through the right femoral vein into the left atrium via percutaneous atrioseptostomy in the catheter lab (Figure 1). The second venous cannula was connected to

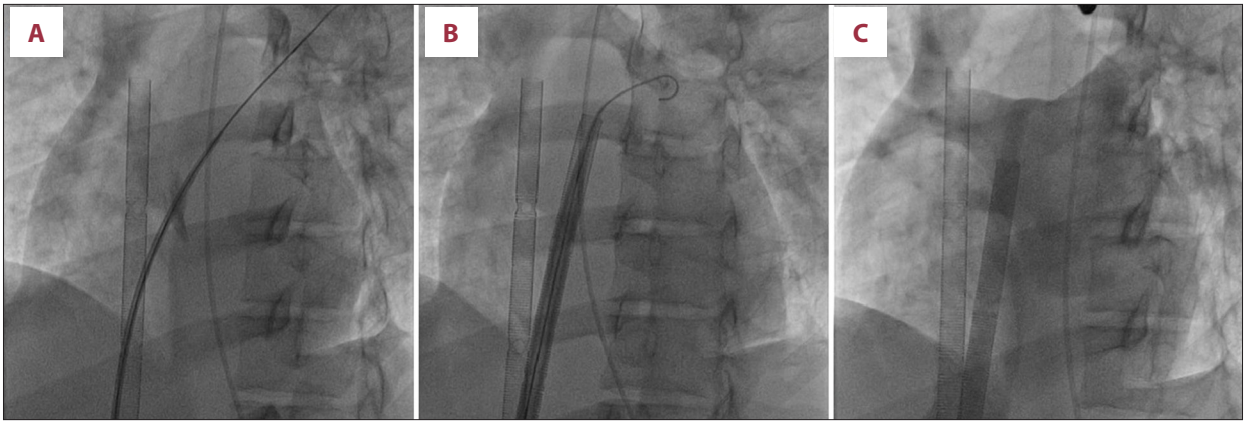

Figure 1. (**A–C**) Venting of the left heart. Fluoroscopy-assisted placement of a second venous cannula into the left atrium by catheter-based atrioseptostomy. (**A**) Transseptal puncture with insertion of a guidewire into the upper left pulmonary vein; (**B**) Positioning of the transeptal venous cannula; (**C**) Confirmation of placement in of the left atrium by contrast agent injection.

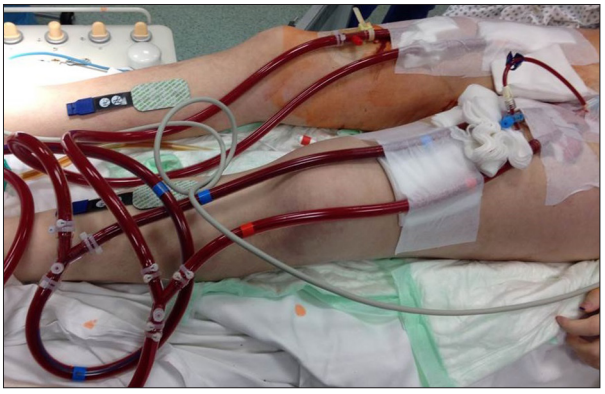

Figure 2. Peripheral va-va-ECMO. Venous drainage from the vena cava and the right atrium (left femoral vein) and the left atrium (right femoral vein). Arterial return by the left iliac artery (left femoral artery) and by the right femoral artery (cannula in a Dacron® conduit in the right femoral artery). Distal perfusion of the left leg (left superficial femoral artery).

the ECMO circuit by another Y-connector (Figure 2). ECBF now reached 5–6 l/min at 3850–4200 rpm according to improved venous drainage. Lactate levels peaked at 29 mmol/l, but fell instantly and the MAP rose from a nadir of 30 torr after acidosis leveled out. However, cerebral oximetry by near infrared spectroscopy (INVOS™, Covidien, Boulder, CO, USA) showed sufficient regional oxygen saturation of about 60–80% after the initiation of va-ECMO therapy. In echocardiography, the left ventricle was unloaded with no residual left ventricular function. Therapeutic anticoagulation was ensured by unfractionated heparin with a target partial thrombin time of 60–80 s. During the first 3 days, PCT peaked with 80.4 ng/ml and CRP with 7.23 mg/dl. Leukocytes remained low at 300/µl. High-sensitive troponin T increased to 343 pg/ml and creatine kinase MB to 178 U/l (total creatine kinase 577 U/l). All microbial specimens collected during this period remained negative. Viral myocarditis was excluded by a myocardial biopsy.

Throughout the following days, cardiac systolic function gradually recovered. On day 5, the ECMO cannulas in the left femoral vessels were removed. On day 7, the ECMO was completely removed in the operating theater after LVEF had recovered to approximately 30%, with mean arterial pressures of 60–70 torr. After removal of the transseptal cannula, no relevant communication between the left and right atrium could be observed. CVVHDF could be stopped due to normal renal function. Leucocyte count recovered to 1000/µl on day 13 and normal values were reached spontaneously on day 30. Respirator weaning was reached on day 33. Successful allogenic stem cell transplantation was performed 6 weeks later. Another 5 months later, the patient was discharged to a rehabilitation facility, still suffering from a severe critical illness, polyneuropathy. The LVEF recovered to approximately 40% without ventricular dilatation.

Discussion

The imbalance of oxygen supply and demand is a hallmark of septic shock leading to global tissue hypoxia. Even in a hyperdynamic state, the heart may not be able to match the increased oxygen demand. Septic cardiomyopathy worsens this problem to the point of cardiogenic shock during the course of disease. ECMO support may become necessary, which for some time has been proven useful in children with refractory septic shock [4]. In adults, studies indicate increased survival as well, and ECMO is rapidly growing in usage [1,5]. However, in functional cardiac arrest combined with vasoplegic septic shock and massive acidosis, the usual ECBF of about 2 l/min/m^2 is insufficient to maintain adequate mean arterial pressures and oxygen delivery. The current case highlights

this common problem and shows that this limitation may be successfully overcome with double peripheral va-ECMO (va-va-ECMO) cannulation. In our case, the arterial femoral vessel size of 3–4 mm allowed only a cannulation with a very small cannula of 13 Fr. Due to the low cardiac output, lactate levels rose and leg ischemia of the non-cannulated limb occurred. However, the high flow resistance and the risk of blood traumatization did not allow a further enhancement of the ECBF. Thus, a second cannula was surgically inserted to increase the ECBF and maintain sufficient leg perfusion. With the vaaECMO-setting, a sufficient ECBF >5 l/min with pre-oxygenator membrane pressures below 300 torr could be maintained. With the second venous drainage, venting the left ventricle could be achieved. In addition, venous drainage pressures could be reduced. A similar approach was first described by Litmathe and Dapunt to treat septic shock in an adult ARDS patient [6]. In contrast to our patient, they cannulated upper and lower body vessels and used 2 extracorporeal circuits to reach a supra-normal ECBF flow. However, in our setting, venous drainage and pre-oxygenator pressures allowed an ECBF up to 6 l/min with a single ECMO system designed for an ECBF up to 7 l/min. Therefore, a second ECMO circuit was not necessary.

Alternatively, open chest central ECMO cannulation can successfully be used in children with septic shock [4]. However, peripheral va-ECMO is the prime mode of cannulation in emergency situations. In addition, central cannulation would bear a high risk for infectious and bleeding complications in this juvenile girl with pancytopenia. Moreover, we had to consider that stem cell transplantation for curative treatment of leukemia was required within a limited time period.

The current case further illustrates the importance of closely monitoring the ability of the left ventricle to open the aortic valve. Increased afterload due to high va-ECMO blood flow may result in left ventricular overdistension, pulmonary edema, or intracavity clot formation during severe left ventricular dysfunction. Left ventricular unloading must therefore be applied during peripheral va-ECMO to prevent these serious adverse events [7]. This could be done by either direct surgical cannulation of the left ventricle or a pulmonary vein. Other possibilities are percutaneous venting by a transaortic catheter or a venous cannula in the pulmonary artery [8]. Alternatively, a microaxial pump can be used (Impella®, Abiomed Inc., Danvers, MA, USA) [9].

In the current case, we chose a less invasive approach due to the specific risks mentioned above, and inserted an additional venous cannula into the left atrium after catheter-based atrioseptostomy. With this cannula, sufficient drainage of the left heart could be achieved, as previously described [10,11]. A transaortic microaxial pump was not established due to the small diameters of the femoral arteries.

The significance of the current case may be limited by the fact that the source of the combined septic and cardiogenic shock could not be revealed. High PCT levels do not prove an infection, since PCT can be elevated in shock and multiple organ failure of any reason. PCT was reported elevated up to 10–20 ng/ml in single patients with cardiogenic shock complicated by multiple organ failure [12]. An alternative explanation for lactate acidosis, multiple organ failure, and refractory shock, besides a severe septic shock, may have been mitochondrial toxicity caused by linezolid. Fatalities have been reported [13]. However, considering the presented clinical and diagnostic evidence and time response, we believe that an infectious cause of shock during pancytopenia is the most likely diagnosis.

Conclusions

Venoarterial extracorporeal membrane oxygenation (va-ECMO) can by a lifesaving option in patients with septic shock. If severe vasodilation is present and cardiac contractility is impaired, a high extracorporeal blood flow and even venting of the left heart can be necessary. Blood vessel diameters eventually do not allow insertion of large-bore cannulae to minimize extracorporeal in- and outflow pressures. In these cases, the double peripheral approach for cannulation of the blood vessels combined with transseptal left atrial venting is a valuable alternative to central cannulation for ECMO.

Conflicts of interests

Markus Kredel, Philipp M. Lepper and Ralf Muellenbach received lecture honoraria from Maquet GmbH, Rastatt, Germany.

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