ECMO and cytokine removal for bridging to surgery in a patient with ischemic ventricular septal defect - a case report


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This case study reports on a 64-year-old man who had suffered a myocardial infarction.

**Case presentation**
- The patient had no significant medical history, no known diabetes or hypertension, and no oral medication. Cardiovascular risk factors were obesity and long standing high nicotine consumption.
- On arrival at the local hospital the patient was awake and in a stable condition, with normal renal function (glomerular filtration rate >60 mL/min).
- Echocardiography and coronary angiography showed 2-vessel coronary disease. Emergency percutaneous coronary intervention (PCI) failed and the patient was transferred to a tertiary center sedated, intubated and in cardiogenic shock, requiring vasopressor support (norepinephrine 0.6 mcg/kg/min). Eventually, PCI was successful and the right coronary artery (RCA) was able to be revascularized. However, the patient remained in cardiogenic shock.
- A temporary pacemaker and intra-aortic balloon pump (IABP) where placed and the patient managed in the intensive care unit (ICU), where he remained hemodynamically unstable. The patient developed decompensated shock, acute kidney injury with metabolic acidosis (pH 6.9, bicarbonate <10 mmol/L, lactate >15 mmol/L) so continuous hemodialfiltration (HDF) was started. The patient developed ventricular fibrillation and underwent cardiopulmonary resuscitation for 25 mins. Post resuscitation the patient was on norepinephrine up to 1 mcg/kg/min, dobutamine 10 mcg/kg/min and vasopressin 3 IE/h.
- Due to the refractory shock, the echocardiogram was repeated, and ventricular septal defect (VSD) detected. Cardiac surgery was delayed due to the high risk of intraoperative death. The patient developed leg ischemia where the IABP was placed so the patient was put on veno-arterial Extracorporeal Membrane Oxygenation (vaECMO) as a bridge to surgery.
- As cardiogenic shock was aggravated by severe systemic inflammatory response syndrome (SIRS), a Cytosorb adsorber was inserted into the circuit.

**Treatment**
- Four treatments with Cytosorb for 24 hours each
- Cytosorb was added to the CRRT circuit downstream of the hemofilter
- Blood flow rate: 120 ml/min
- Anticoagulation: heparin and citrate

**Measurements**
- Demand for catecholamines
- Hemodynamic parameters
Results

- The patient’s condition began to stabilize a few hours after vaECMO and CytoSorb therapy, so that catecholamines could be significantly reduced within the first 36 hours.
- Norepinephrine was more than halved (0.86 to 0.38 mcg/kg/min), Dobutamine was reduced from 13.2 to 9.6 mcg/kg/min and vasopressin therapy was terminated (Fig. 1).
- Vital signs improved - systolic arterial pressure (SAP) from 55 mmHg up to 75 mmHg and MAP from 50 to 70 mmHg.
- After 4 days the patient was stable enough to go to surgery where he successfully underwent coronary arterial bypass surgery and VSD patch repair.

Patient Follow-Up

- vaECMO was continued for a further 17 days until the patient was stable enough to tolerate the start of respiratory weaning. The patient tolerated the removal of vaECMO well and remained stable within the following days with satisfactory cardiac function.
- Unfortunately, during the following ICU stay the patient developed fungal sepsis and eventually died of refractory septic shock.

Figure 1: Trend of dobutamine and noradrenaline (A), and lactate and vasopressin (B)

CONCLUSIONS

- This is the first successful use of ECMO combined with CytoSorb as a bridging technique for cardiac surgery patients with VSD requiring further interventions.
- ECMO combined with cytokine removal could be a promising technique in these unstable high-risk patients with SIRS.
- The authors describe the combination of ECMO and CytoSorb as a tool for buying time for surgery in patients with ischemic VSD.