

Inter-hospital extracorporeal life support

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ABSTRACT

A 60-year-old man with history of hypertension and unspecified left ventricular dysfunction had chest pain at home at 9 am. At 1 pm he was transported to a peripheral hospital and treated for acute myocardial infarction. At 4.30 pm, despite pharmacological and intra aortic balloon pump support, the extreme hemodynamic instability and the echocardiographic signs forced the doctors in charge to contact the “extracorporeal membrane oxygenation team” of our Intensive Care Unit. The team, that in our hospital is composed of an intensivist, a cardiac surgeon, a perfusionist and a nurse, reached the hospital at 5.15 pm and performed a percutaneous cannulation of right femoral artery and left femoral vein connecting the patient to the extracorporeal membrane oxygenation circuit. At 6.30 pm the patient on extracorporeal membrane oxygenation was transferred by ambulance to the Cardiac Surgery Intensive Care Unit of San Gerardo Hospital in Monza. On day 20 he was transferred back to the original hospital without neurological deficits, with normal renal function and normal blood gas analysis.

Keywords: *extracorporeal membrane oxygenation, cardiogenic shock, cardiac arrest.*

INTRODUCTION

Cardiogenic shock is the leading cause of death for patients with acute myocardial infarction who reach the hospital alive. It affects about 6 to 8% of patients with acute myocardial infarction. (1) Despite the advantages of early intra-aortic balloon pump (IABP), fibrinolysis, percutaneous coronary intervention or coronary arterial bypass graft, once shock is diagnosed, the mortality remains high (about 50%) with half of death occurring during the first 48 hours. (2).

Some studies suggested that haemodynamic and metabolic parameters could be more effectively reversed by ventricular assist de-

vice than by standard treatment with IABP (3).

In this investigation we report our experience of using extracorporeal membrane oxygenation (ECMO) to resuscitate a 60-year-old man with critical left main coronary artery disease complicated by acute myocardial infarction (AMI) and cardiogenic shock. The connection of patient to ECMO circuit was performed in an hospital other than ours and the patient was then transported to our hospital on ECMO with an Advanced Care Mobile Unit.

CASE REPORT

A 60-year-old man with history of hypertension and unspecified left ventricular dysfunction started to have chest pain at home at 9 am. His medications included beta-blockers, a sartanic and a tiazidic di-

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uretic. He waited more than four hours at home before calling for help. At 1.30 pm he alerted the emergency department (118) and the advanced life support team found him awake, hypotensive, symptomatic and with ECG signs of extensive antero-lateral AMI. Morphine and apirine were administered, dopamine infusion started and the patient was transferred to the nearest peripheral hospital having a coronary care unit, ten kilometres from our hospital.

At 2 pm during the angiographic study he developed severe cardiogenic shock unresponsive to catecholamine (dopamine 20 mcg/kg/min) and requiring IABP support (1:1). The angiographic study showed distal occlusion of left main coronary artery and the cardiologist performed angioplasty on left anterior descending and circumflex coronary artery and positioned a bare metal stent on left main and left anterior descending arteries administering heparin bolus 7500 IU and continuous infusion of reopro.

The procedure was successful and obtained a good revascularization, but the TIMI score was 1 (the patient had severe hypotension despite high-dose norepinephrine). At 3.30 pm the patient had a bradi-asystolic cardiac arrest treated with 20 minutes advanced life support algorithms including tracheal intubation and transvenous pacing.

After the resuscitation manoeuvres, the patient was still in cardiogenic shock refractory to high amines dosage (dopamine 16 mcg/kg/min plus adrenaline 0.14 mcg/kg/min), IABP, pacing, mechanical ventilation: systolic blood pressure was 90 mmHg, heart rate 100 beats per minute, anuria, paO₂ 53 mmHg, paCO₂ 60 mmHg, pH 7.04, lactates > 6 mmol/L and base excess -14 mmol/L. An echocardiography examination showed the presence of contractile reserve (i.e. temporary improvement of ejection fraction and arterial pressure during epinephrine

bolus). At 4.30 pm the doctors in charge decided to call our extracorporeal membrane oxygenation (ECMO) team, composed of four figures (intensivist, cardiac surgeon, perfusionist and nurse by cardiac surgical care unit). At 5.15 pm we reached the peripheral hospital and, being the patient hemodynamically unstable, we decided for ECMO implantation. Percutaneous cannulation of right femoral artery with a 17 French cannula and of left femoral vein with a 21 French cannula was performed. The perfusionist prepared and primed the circuit and at 5.40 pm the patient was connected to ECMO with blood flow 3.5 L/min, gas flow 3 L/min and FiO₂ 0.6 with rapid hemodynamic stabilization, reduction of amines, recovery of diuresis and normalization of blood gas parameters (paO₂ 457 mmHg, paCO₂ 29 mmHg, pH 7.48, lactates 1,9 mmol/L, BE -1,2 mmol/L). The reopro infusion was interrupted at the beginning of the manoeuvre and we had no hemorrhagic complications. At 6.30 pm the patient on ECMO was transferred by ambulance to the cardiac surgery intensive care unit of San Gerardo Hospital in Monza. The transport required 20 minutes for a distance of about 10 kilometres and was uneventful. The patient was maintained sedated, with continuous perfusion of muscle relaxant and, for the first 24 hours therapeutic hypothermia was applied for brain protection. With total ECMO support he was hemodynamically stable without catecholamine support. Monitoring included arterial, central venous and pulmonary artery catheter. Hemodynamic data were: arterial blood pressure 150/60 mmHg, heart rate 80 beats, sinus rhythm, central venous pressure 12 mmHg, wedge pressure 15 mmHg, pulmonary artery pressure 29/18 mmHg, cardiac output 2,5 L/min, venous oxygen saturation 72%. A transesophageal echocardiography showed an ejection fraction of 10% under full ECMO blood flow. Dobutamine

5 mcg/kg/min was started to assure aortic valve opening.

Intravenous infusion of heparin had an activated clotting time of 180-200 seconds. Aspirin and clopidogrel were started without loading dose.

On day 1 echocardiography showed akinesia of apex and anterior wall, severe hypokinesia of septum and lateral wall, no valvular defects. Daily evaluations of legs perfusion were performed with clinical parameters and/or arterial doppler. We never found any problem of leg perfusion in this case.

On day 4, because of relative hypertension, we started infusion of calcium-sensitizer and weaning from ECMO gradually reducing blood flow at 2 L/min for 12 hours and then 1 L/min for others 12 hours while monitoring hemodynamic parameters and cardiac performance and dimensions with Swan-Ganz catheter and echocardiography. On day 5 the patient was weaned from ECMO with IABP (1:1) and dobutamine 10 mcg/kg/min.

The cannulae were removed with only external compression for hemostasis. After removal of ECMO cannulae the patient developed hyperdynamic shock (cardiac output 11 L/min, blood pressure 80/40 mmHg) requiring norepinephrine for 24 hours. We treated this hyperdynamic shock as a septic one, performing cultural examination and starting empiric antibiotic therapy with meropenem.

On day 6 we removed IABP and started nitrates. Echocardiography without ECMO and IABP support showed persisting akinesia of apex and anterior wall with good recovery of septal and lateral wall motion and an ejection fraction of 30%.

On day 7 linezolid was added to meropenem because of fever and elevation of leukocytes and inflammation indexes. The same day the patient had bronchial bleeding and deterioration of blood gas parameters, requir-

ing high PEEP and interruption of heparin and clopidogrel. Ecodoppler was performed to exclude the presence of thrombosis in the main vessels.

On day 8 the patient developed an hypertensive crisis with pulmonary oedema without changes in ECG or cardiac regional wall motion with high pulmonary pressures requiring mechanical ventilation with PEEP 15cmH₂O, high FiO₂ and inhalatory nitric oxide for 48 hours.

Negative water balance and infusion of calcium-sensitizer because of the evidence of diastolic dysfunction at echicardiography were performed. ACE-inhibitor and beta-blockers were introduced. On day 11 the patient was extubated and on day 13 we introduced digoxin for prevention/treatment of congestive heart failure (4).

On day 20 he was transferred back to the hospital he came from. At discharge from our unit the patient was awake, without neurological deficits, hemodynamically stable, in sinus rhythm, receiving ACE-inhibitor and beta-blocker, with normal renal function and normal blood gas analysis.

The equipment used during the transportation included: an usual Advanced Care Mobile Unit coordinated by our 118 department; a portable ECMO circuit (*Figure 1*) and a transport ventilator "Oxylog 3000 (Draeger, Lubeck, Germany). The portable ECMO circuit is composed by Rotaflow centrifugal blood pump, Rotaflow console and permanent life support system (Maquet, Jostra Medizintechnik AG, Hirrlingen, Germany); cannulae Bio-Medicus (Medtronic inc., Minneapolis, MN). The transport of ECMO_system was made with the "Mobile Heart-Lung Machine (HLM) holder HKH 8800 (Maquet, Jostra Medizintechnik AG, Hirrlingen, Germany)" (5). In our Unit we have an "ECMO-bag" containing devices for cannulation and emergency assistance of intensive patients ready for use.



Figure 1 - ECMO circuit with membrane oxygenator and centrifugal pump mounted on a specially designed multifunctional holder Steering and control unit including battery pack.

DISCUSSION

The details of our “ECMO project” are available online at and are inclusive of inclusion and exclusion criteria, flowcharts, duties of the emergency department (118), background and reasons that justify the project that offers to the surrounding hospitals that have no cardiac surgery and no expertise with ECMO the possibility of a prompt referral.

Within this project and till July 2009 the ECMO team was activated 7 times: in five cases no intervention was needed (we were on stand-by for elective dangerous procedures in catheter laboratories). The first patient with cardiogenic shock who received ECMO in the peripheral hospital and transport to our hospital died in our intensive care unit on day 2 for intestinal infarction due to important extracardiac arteriopathy. The second patient is described in details in this case report and had good recovery.

Overall, before July 2009, our experience with ECMO for cardiac assistance included 57 cases, 35% of them performed outside in the emergency department, in the catheterization laboratory or in other hospitals. and all of them with percutaneous cannu-

lation performed either by the cardiac surgeon or the intensive care specialist.

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