Extracorporeal cardiopulmonary resuscitation with low pump flow for blocked modified Blalock–Taussig shunt followed by spontaneous recanalization

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Running title: E-CPR with low pump flow after blocked mBT shunt

Abstract

A two-week-old male newborn with a double inlet left ventricle developed a cardiac arrest following modified Blalock-Taussig anastomosis in Pediatric intensive care unit (PICU). Probable causes of the arrest were hemodynamic instability and thrombosed shunt, which was later recanalized on extracorporeal membrane oxygenation (ECMO) therapy, which was successfully used with a pump flow lower than recommended in these patients; without the shunt clip, but without any complications.

Keywords: mBT shunt, low cardiac output, resuscitation, ECMO, E-CPR

Introduction:

The goal of palliative surgery for a single ventricle physiology is to convert a parallel to a serial circulation. If there is normal systemic flow and pulmonary flow is missing, the lung circulation is usually provided with patent arterial duct or by making systemic-to-pulmonary shunt.

Case report:

A male newborn with a complex congenital heart disease formed of double inlet left ventricle with D-transposition of the great arteries and pulmonary atresia was transferred with continuous prostaglandin infusion to our hospital on eighth day of life, weighing 3.8 kg. Five days later, an uneventful surgery was performed through median sterntomy to place a 3.5 mm modified Blalock-Taussig (mBT) shunt on the left side due to right aortic arch.He was admitted to the PICU intubated, mechanically ventilated, hemodynamically stable (blood pressure (BP) 102/42 mmHg, SpO2 90%, FiO2 50%, pulse 151/min) and with normal blood gas analysis (pH 7.28, pO2 5.7 kPa, pCO2 7.2 kPa, BE -1.3).The diuresis in the early period was 13 ml/kg/h. Four hours after the admission to the PICU he became hypotensive (BP 55/20 mmHg), bradycardic (pulse 60/min), with a low oxygen saturation of 40%. Although the fluid and medical treatment with high doses of adrenalin (1 mcg/kg/min) and dopamine (8 mcg/kg/min) were immediately started, hemodynamic instability progressed to cardiac arrest. The effective closed-chest cardiopulmonary resuscitation (CPR) was started. Immediate pre-CPR lactate level was 13.7 mmol/l with pH 7.12,pCO2 of 8.3 kPa, pO2 1.7 kPa and BE of -9.1. After two hours of continuous cardiopulmonary resuscitation, patient received a Maquet PLS ECPR system through a median sternotomy. An 8 French aortic cannula was set into the aortic arch, and the 14 French venous cannula into the right atrium. During cannulation, a loading dose of 1100

international units of unfractionated heparin was given. The initial ECMO flow of 77 ml/kg/min was achieved by a rotaflow pump speed of 2245 rotations per minute (RPM) and with the fraction of oxygen on the ECMO blender (FSO2) of 50%. Immediately after the beginning of ECMO, heart ultrasound revealed a significantly decreased systolic heart function with no flow through the mBT shunt. Anticoagulation therapy started 12 hours of ECMO flow and conducted with a continuous heparin treatment, with desired activated thromboplastin time (aPTT) value of 50-70 seconds and activated clotting time (ACT) value of 180-200 seconds. In the first six hours of the ECMO procedure both ACT and aPTT were immeasurably high. The ventilation parameters on ECMO were standard rest lung ventilation parameters (positive end expiratory pressure of 5, peak inspiratory pressure of 20, respiratory rate set at 10), there was no hemodynamic support and the blood gas analysis as well as vital signs were within the normal range. After 36 hours of ECMO, the ultrasound showed a better contractility with normal flow through mBT shunt. With an aim to start the weaning, ECMO flow was decreased to 50 ml/kg/min with FiO2 of 30%, but hypotension and low oxygen saturation occurred. The patient was treated with fluids, continuous noradrenalin infusion and ECMO flow was put back to 77 ml/kg/min. On the third postoperative day, a CT scan on ECMO was performed showing a normal flow through the pulmonary arteries and the mBT shunt. The patient was decanulated after 68 hours of ECMO run. Higher ventilation parameters were needed to achieve normal blood gas levels. Catheterization performed immediately after the ECMO treatment showed a normal function of the mBT shunt. Fifteen days after the decanulation he was successfully extubated. The total PICU stay was 21 day with in-hospital stay of 32 days. Five months later, the partial cavopulmonary anastomosis was performed, with no complications.

Discussion

Modified Blalock-Taussig shunt is usually the first treatment option in a single ventricle physiology. Although simple in concept, it may be associated with significant morbidity and mortality. Major problems are over-shunting or shunt thrombosis.[1] In the literature, there are few cases of successful extracorporeal cardiopulmonary resuscitation (E-CPR) after mBT shunt procedure.[1-5] The goal of E-CPR in those patients is providing an adequate organ perfusion with early ECMO set up, preferably within 45 minutes,[6] which is less than in our patient. Due to organizational and technical circumstances, the E-CPR procedure in our case was started with a delay. In the historic beginning of pediatric ECMO use, shunt patients were not suitable for ECMO because of shunt runoff on systemic perfusion.[5] The idea to overcome this problem was to clip the shunt, but there were no survivors in the group of four patients in the study published by Sherwin et al.[4] On the other hand, only one patient out of five died after leaving

the shunt patent but increasing the ECMO flow to compensate for the shunt runoff.[1] In the study published by Botha et al.,[5] 51 shunt patients were treated with ECMO support; 16 of them due to cardiovascular instability, and 19 of them after a cardiac arrest. Overall survival was 48%. In other similar reports, the patent shunt was left and higher pump flow (>150 ml/kg/min) was used with survival rate of 47% in one,[2] and 39% in the other study.[7] In the study published by Allan and colleagues,[8] in 17 out of 44 patients there was a progressive lactacic acidosis, and one or more surgical clips were placed to reduce runoff. In all the cases, significantly higher ECMO flows were maintained in the shunt group.

The initial flow we used (77ml/kg/min) was a half of the usually recommended flow. After the ECMO setup, no flow through mBT shunt was seen on the ultrasound, so clipping the shunt did not seem a reasonable option as it did not seem likely that runoff would occur. Excessive diuresis in early postoperative period had probably caused hypovolemia which led to hemodynamic instability and might have contributed to the occurrence of the shunt thrombosis. Therefore, the increased urination with excessive negative fluid balance should be avoided in this situation. We assume that the spontaneous recanalization of the shunt occurred after the start of the ECMO procedure and an adequate volume and anticoagulation treatment. Our intention was to modify the ECMO flow depending on the hemodynamic situation. Lactate level normalized 24 hours after the beginning of the ECMO support, which was the sign that there was no runoff. The heart ultrasound after lactate stabilization revealed a better contractility with a flow seen through mBT shunt. An adequate fluid balance ws easily achieved by tolerating lower ECMO flow. Although a low ECMO flow run poses a higher risk for development of thrombosis, this was not the case in our patient. One day later he was successfully decanulated. The duration of ECMO was a bit shorter than in other published reports.[2,5]

In conclusion, the fact that adequate hemodynamics were obtained at 77 ml/kg/min of ECMO flow with poor ventricular function suggests a poor pulmonary blood flow due to the shunt thrombosis. Although the standard approach to a blocked shunt implies its revision, the dynamics of the patient's condition in the first hours on ECMO can suggest an observational approach without operation because recanalization might be expected on continuous heparin treatment. Signs and markers of poor perfusion and end-organ injury should be monitored to change the strategy if necessary.

Consent for publication

Informed parental consent was obtained for publication.

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