Case Report

Lactate clearance for initiating and weaning off extracorporeal membrane oxygenation in a child with regressed left ventricle after arterial switch operation

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ABSTRACT

We hereby report a child with transposition of great arteries and regressed ventricle who underwent arterial switch operation (ASO) with the aid of cardiopulmonary bypass and "integrated" extracorporeal membrane oxygenation (ECMO) circuit. The significance of lactate clearance as a guide to initiate and terminate veno-arterial ECMO in a post ASO child with regressed left ventricle is discussed.

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INTRODUCTION

After cardiac surgery, an increase in the level of lactate indicates a mismatch between oxygen demand and supply. An increase in lactate levels for a prolonged period of time has been associated with poor prognosis and mortality. The lactate level reflects the balance between production and clearance of lactate. Lactate is cleared by the liver, skeletal and cardiac myocytes, and proximal tubule cells in the kidneys. Patients who are diagnosed with sepsis, but are hemodynamically stable develop hyperlactatemia because of a decrease in lactate clearance rather than increased production of lactate due to tissue hypoxia. Similarly, in patients who are on Extracorporeal Membrane Oxygenation (ECMO) and have achieved hemodynamic stability, lactate clearance becomes more important compared with absolute levels of lactate. We hereby report the case of a child with transposition of great arteries and regressed ventricle who underwent arterial switch operation with the aid of cardiopulmonary bypass and "integrated" ECMO circuit.

CASE REPORT

A 10-month-old boy (weight 7.5 kg and height 67 cm) was admitted to our institute with a history of cyanosis since 2 months. His transthoracic echocardiography showed an enlarged right atrium and right ventricle (RV), a "D" shaped normally contracting left ventricle (LV), an atrial septal defect (ASD), intact interventricular septum, aorta arising from RV, pulmonary artery arising from LV, and mild right ventricular dysfunction. The branch pulmonary arteries were 5 mm each.

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Table 1: The hemodynamic parameters, arterial blood gas (ABG) analysis including lactate clearance (LC) and blood transfusion during ICU stay

Time point in ICU (h)	T1 (0)	T2 (6)	T3 (12)	T4 (24)	T5 (36)	T6 (48)	T7 (72)	T8 (96)	T9 (120)
Arterial blood gas analysis									
pH	7.44	7.42	7.36	7.52	7.42	7.46	7.42	7.44	7.34
PCO_2 (mmHg)	28	31	37	25	30	31	32	33	39
PO_2 (FiO_2) (mmHg)	329 (1)	264 (0.6)	270 (0.4)	317 (0.9)	290 (0.9)	235 (0.6)	150 (0.6)	106 (0.6)	108 (0.6)
BE (mmol/L)	−3.8	−4.9	−3.8	−1.6	−4.1	−1.4	−2.5	−1.5	−0.2
HCO_3 (mmol/L)	18.7	18.2	21	19.4	19	21.2	20.9	22	26
HCT (%)	38	34	42	36	38	31	34	33	38
Na^+ (mmol/L)	138	144	144	146	148	144	154	150	147
K^+ (mmol/L)	4.5	3.6	4	3.7	3.5	3.1	3.4	3.6	3.5
SaO_2 (%)	100	100	100	100	100	100	100	100	100
ACT (s)	182	225	246	196	230	190			
Blood sugar (mg/dL)	190	162	184	112	142	154	102	130	120
Hemodynamic parameters									
Heart rate (/min)	140	190	162	182	175	157	154	146	132
Blood pressure (mmHg)	51/30	45/20	50	55/28	57/28	58/34	66/40	58/36	60/35
Mean arterial pressure (mmHg)	37	32	50	37	38	42	49	43	43
Right atrial pressure (mmHg)	5	7	4	4	9	9	7	10	8
SPO_2 (%)	100	100	100	100	100	100	100	99	99
Peripheral temperature (°C)	27.9	30.1	30.3	31.9	34.3	35.4	32.3	33	31
Core temperature (°F)	97.8	99	96.8	97.2	98.4	99	98.4	97.6	98.4
Lactate levels and clearance									
Lactate (mmol/L)	2.4	7.5	5	2.6	2.1	2.3	2.2	1.7	1.5
Lactate clearance (%)		−212	−108	−8.30	12.50	10	8.30	29	37
Mediastinal chest tube drainage (mL)	530								
Blood transfusion									
Packed red blood cells (mL)	420								
Platelet concentrate (mL)	60								
Fresh frozen plasma (mL)	150								
Cryoprecipitate (mL)	90								

Time points - T1: Immediately after arriving in ICU; T2–T9: 6, 12, 24, 36, 48, 72, 96 and 120 h after arrival in ICU. ICU: Intensive Care Unit

The posterior wall thickness of LV was 3 mm, and end systolic and end diastolic dimensions were 9 and 14 mm, respectively. The patient underwent an arterial switch operation and ASD closure with the aid of cardiopulmonary bypass (CPB) and "integrated" extracorporeal membrane oxygenation (ECMO) circuit.[1,2] The patient was successfully weaned off CPB and was shifted to Intensive Care Unit (ICU) with aortic and venous cannulae *in situ* connected to ECMO circuit, which was put on recirculation. The patient was on intravenous infusions of adrenaline (0.15 mcg/kg/min) and nitroglycerine (1 mcg/kg/min). The hemodynamics, arterial blood gas (ABG) analysis and lactate clearance (LC), after shifting to ICU are depicted in Table 1.

In ICU, strict asepsis was maintained. The Activated Coagulation Time (ACT) was maintained within 200–250 s with a heparin infusion of 10–15 U/kg/h. Phenoxybenzamine[2] was given slowly by intravenous route 8 mg, over 1 h, thrice daily. A nasogastric tube was placed, and enteral nutrition started at 20 mL every 3 hourly after 4 h of shifting to ICU. Continuous core and peripheral temperature monitoring were done. The mediastinal chest tube drainage was promptly replaced with packed red blood cells and fresh frozen plasma/platelet concentrate in a ratio of 3:2. Serum lactate levels were monitored and LC was calculated in relation to lactate level at T0 (immediately after shifting to ICU). LC was calculated as follows: 6 h LC % = ([serum lactate at 6 h − baseline serum lactate]/baseline serum lactate) ×100.

Six hours after shifting to ICU the child developed sinus tachycardia of 190/min and systolic blood pressure decreased to 45 mmHg. Since the PaO_2

was 264 mmHg (FiO_2 60%) and the base deficit was −4.9 mmol/l, there was an element of doubt about initiating ECMO. Therefore, the lactate level was measured and LC calculated. The LC was found to be −212% (T2) and hence the extracorporeal circulation was commenced. Thereafter, LC was calculated at different time points as shown in Table 1. The LC improved at 12 h to −108% and at 24 h postsurgery it became −8.30%. The child was weaned off ECMO support at 24 h after observing the hemodynamic parameters and ABG report. The LC remained more than 10% in the subsequent 24 h and the child was decannulated and sterum closed at 48 h postsurgery. On the 3rd postoperative day (POD) sildenafil, 5 mg thrice daily and enalapril, 1 mg twice daily, was started via nasogastric tube. Intravenous infusion of furosemide 0.5 mg/h was started and continued for next 3 days to decongest the lungs and prevent fluid overloading. For the next 3 days, the LC showed a positive trend and hemodynamics and blood gases remained stable.

At 6th POD, the child developed a clinical picture of ventilator-associated pneumonia. The endotracheal aspirate was sent for culture and, empirically, the child was started on injection colisitin 30,000 U/kg as per ICU protocol. On the 9th POD endotracheal aspirate turned positive for *Acinetobacter baumannii*, sensitive to colistin only. The decision of early tracheostomy, at 9th POD, was taken to expedite weaning from the ventilator. On the 12th POD, the child was successfully weaned from the ventilator and on 22nd day postsurgery the trachesotomy tube was removed.

DISCUSSION

LC may predict the prognosis of acute respiratory failure in patients on extracorporeal circulatory support.[3,4] It has also been correlated with survival, in postcardiac arrest children, after the return of spontaneous circulation with the help of ECMO.[5] In this case report, we establish LC as a guide to initiate, and wean off veno-arterial ECMO in a child (with transposition of great arteries and regressed LV) who has undergone an arterial switch operation. The oxygenation index (OI)[6,7] is commonly followed as a yardstick in most patients with respiratory failure to commence ECMO support. In our child, we anticipated difficult weaning from CPB, in view of regressed LV, therefore a veno-arterial ECMO was integrated in the CPB circuit beforehand.[2] The oxygenation and ventilation were never a problem in the postoperative period as reflected by high partial pressures of oxygen (PaO_2) in blood and persistence of mean airway pressures in the range of 10–12 cm H_2O. Usually, we try to keep PaO_2 in ranges of 150–200 mmHg on ECMO to provide a safety margin. Hence, OI was not considered as a parameter for decision making.

Early decrease in LC (6 h postbaseline) has been associated with mortality after respiratory failure, on ECMO support and following the return of spontaneous circulation after cardiac arrest.[3-5] Li *et al*.[8] observed that early change in lactate values (6 and 12 h) following the initiation of ECMO support can predict in-hospital mortality in postcardiac surgery patients. Early change in lactate values is also predictive of successful weaning from ECMO. In our patient, we measured lactate level and calculated LC at 6 and 18 h after initiation of ECMO. The improvement in LC, decreasing trend of lactate levels and improved hemodynamics encouraged us to wean the child off ECMO support.

The lactate levels may be considered as a marker of perfusion during ECPR in patients with refractory cardiac arrest on ECMO support.[9] Huang *et al*.[5] concluded in their study of 62 ECPR events that a serum lactate level of 3 mmol/l at 24 h post-ECPR is associated with good neurological outcome in children. In our patient, we followed LC and serum lactate levels. At 24 h postsurgery when the LC reached <−10% and serum lactate reached <3.0 mmol/l the child was weaned off ECMO. Similarly, when the LC remained within 10% of baseline and lactate levels remained <3.0 mmol/l the child was decannulated, 48 h after surgery, and the sternum closed.

In conclusion, serial estimation of lactate level and LC aids in both initiation and termination of ECMO support in a child with regressed ventricle who has undergone arterial switch operation.

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Conflicts of interest

There are no conflicts of interest.

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