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SHORT REPORT

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Long-term outcome of an infant resuscitated from iatrogenic potassium intoxication with a serum level of 17.7 mmol/l

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Acute potassium intoxication is a life-threatening event requiring aggressive therapy and a delay in diagnosis may be fatal. We report on the long-term outcome of a 6-week-old infant with severe iatrogenic potassium intoxication during post-operative care after cardiac surgery.

The infant was admitted to the paediatric intensive care unit after correction of a truncus arteriosus. Her clinical course was complicated by excessive chylothorax, ascites and two episodes of sepsis. At the time of intoxication, she required inotropic support and external cardiac pacing due to atrio-ventricular block.

On the 34th post-operative day, the infant suddenly developed severe arrhythmia. Intra-arterial blood pressure monitoring revealed circulatory arrest. Advanced cardiopulmonary resuscitation was initiated immediately with ventilation with FiO_2 = 1.0, chest compressions and bolus injections of epinephrine (total 60 µg/kg). Chest compressions re-established effective circulation (mean arterial pressure >40 mm Hg), but refractory ventricular arrhythmia persisted. A chest-X-ray film ruled out tension pneumothorax. Arterial blood sampling revealed a potassium level of 17.7 mmol/l. All intravenous infusions were halted. Under continued manual chest compression and ventilation, i.v. calcium chloride (total 0.23 mmol/kg), sodium bicarbonate (total 5 mmol/kg), infusion of glucose (total 0.7 g/kg) and insulin (total 2 U/kg), repeated furosemide boluses (total 2.7 mg/kg) and continuous inhalation with salbutamol (total 8 mg/kg), potassium levels gradually declined (Table 1). At a level of 8 mmol/l and 45 min after cardiac arrest, spontaneous pulse-generating cardiac rhythm returned with a mean arterial pressure of 40 mm Hg. External pacing was restarted and the patient returned to a stable circulatory status under continuing inotropic support with dopamine (20 µg/kg per min) and dobutamine (6 µg/kg per min). Shortly thereafter, the infant opened her eyes and was fully awake. Recently we have seen the girl at 4 years of age. She has achieved all developmental milestones and shows no neurological deficit except for severe iatrogenic hearing impairment.

Subsequent investigations revealed that a short-term co-trimoxazole infusion had been erroneously prepared in the unit using a 15% potassium chloride solution with a total of 30 mmol potassium chloride instead of a 5% glucose solution. The company producing both the potassium chloride and the glucose solution was informed about the incident. Since then, the two vials now differ both in size and colour.

There is no reported survival of patients with potassium levels exceeding 12 mmol/l [1]. Several authors have emphasised early initiation of either peritoneal or haemodialysis, stating that conventional procedures to lower serum potassium levels are not sufficient [4, 5, 6]. In this tiny infant (body weight 2.95 kg) with extensive thromboses in major venous vessels, neither peritoneal nor haemodialysis was an immediately available option. The key to the successful resuscitation of this infant was (1) the monitoring in place including arterial access [3], (2) the immediate start of conventional measures to shift potassium from the extracellular into the intracellular compartment [2] and (3) the data-guided (arterial blood analysis and blood pressure) advanced cardiac life support performed by highly skilled staff. This child possibly survived under conventional measures because the acutely infused amounts of potassium had not yet been distributed into the extravascular space, unlike the more gradual onset of potassium intoxication from enteral sources. Considering the present clinical status of this patient, the dramatic combination of potassium intoxication and post-surgical severe cardiac failure was endured with good outcome.

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Table 1 Timing of events, serum potassium levels and arterial blood analyses

Time	Potassium (mmol/l)	pH	pCO_2 (kPa)	pO_2 (kPa)	BE (mmol/l)	SaO_2 (%)
6.00 p.m.	3.6	7.35	5.6		–2.9	93
8.55 p.m.	Acute arrhythmia followed by circulatory arrest					
8.59 p.m.	Advanced cardiopulmonary resuscitation					
9.10 p.m.	17.7 [a]	7.37	3.7	6.15	–7.8	81
9.28 p.m.	10.4	7.63	4.4	16.1	+12.9	93
9.35 p.m.	8.6	7.50	3.8	50.3	+1.3	98
9.41 p.m.	8.1	7.31	6.8	19.8	–1.9	97
9.43 p.m.	Spontaneous circulation restored					
10.00 p.m.	7.7	7.33	6.6	11.7	–0.5	95
10.20 p.m.	6.6	7.44	5.2	37.3	+2.5	99

[a]Confirmed by second arterial blood analysis on a different analyser. Results in the column are shown for the first analyser used for subsequent measurements

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