Hyperkalaemia following blood transfusion

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A 35-year-old man weighing 63 kg was presented for elective repair of an atherosclerotic thoraco-abdominal aortic aneurysm. He was a known hypertensive on oral nifedipine 20 mg 12 hourly and propranolol 20 mg 8 hourly. He had no known allergies. Routine biochemistry (Na^+ 135 mmol/l, K^+ 3.6 mmol/l, urea 5.1 mmol/l, creatinine 56 $\mu\text{mol/l}$) and haematological investigations were within normal limits. Echocardiography suggested normal left ventricular function and the electrocardiogram (ECG) was normal. An aortogram demonstrated a fusiform aneurysm extending from the descending thoracic aorta (distal to the left subclavian artery) to just distal to the renal arteries.

After anaesthetising the patient, the aneurysm was exposed using a thoraco-abdominal incision. Mannitol 0.5 g/kg and heparin 1 mg/kg were given before aortic cross-clamping. No temporary arterial shunts were used during the aortic cross-clamping and the total cross-clamped time was 65 min. On declamping, the patient developed severe hypotension (a systolic pressure of 40 mmHg), which was treated with rapid transfusion of 1 litre of crystalloids, 1 litre of colloids, 4 units of warmed CPD-A (citrate-phosphate-dextrose-adenine) stored whole blood and 10 ml of 10% calcium gluconate. Despite this, the hypotension persisted; therefore the aorta was re-clamped (supra-coeliac) to maintain the blood pressure and coronary perfusion. This was immediately followed by bradycardia, widening of QRS complexes and ventricular fibrillation (VF). The VF did not respond to two internal DC shocks of 20 J each and one DC shock of 30 J. Internal cardiac massage was started; the heart was found to be very flabby. Intravenous lignocaine (2 mg/kg) and additional doses of calcium gluconate (10 ml of 10%) and 7.5% sodium bicarbonate 100 ml produced no improvement. In view of the VF resistant to DC shock and a 'flabby' heart, hyperkalaemia was suspected. Blood transfusion was discontinued, even though the haemoglobin was 7 g/dl and the hypovolaemia was treated by infusing crystalloid and colloid solutions only. The patient's blood was analysed for serum potassium, which was 7.0 mmol/l. The blood used for transfusion was noted to be 16 days old and biochemical analysis of the unwarmed bag blood showed a pH= 6.803, standard bicarbonate content 7.8 mmol/l, base deficit 20.1 mmol and potassium 16.6 mmol/l.

The hyperkalaemia was treated with 100 ml of 50% dextrose and 20 units of actrapid insulin infused over 15 min along with frusemide 100 mg. Following this the heart recovered its tone and reverted to sinus rhythm with a systolic blood pressure of 80 mmHg. Dopamine 10 $\mu\text{g/kg/min}$ was started to augment the blood pressure with successful declamping of the aorta. At this stage the serum potassium was found to be 5.5 mmol/l, falling to 3.3 mmol/l one hour later. Subsequently only fresh blood was given. Unfortunately, the patient died of multiple organ failure 3 days after the operation in the intensive care unit.

Questions

- 1 What are the causes of hyperkalaemia during surgery?
- 2 What are the causes of hyperkalaemia during blood transfusion?

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Answers

QUESTION 1

The various causes of hyperkalaemia during surgery are listed in box 1.

Causes of hyperkalaemia during surgery

Shift of potassium from tissues/cells due to:

- drugs: eg, suxamethonium
- acidosis
- tissue damage: muscle injury (crush/ischaemic injury); haemolysis; internal bleeding; catabolic states; malignant hyperpyrexia
- blood transfusion
- impaired excretion: acute renal failure

Box 1

QUESTION 2

Following massive blood transfusion of stored blood, complications such as hyperkalaemia and citrate toxicity are well recognised but fortunately very rare. Normally, plasma contains 4–5 mmol/l of potassium whereas red blood cells contain 100–105 mmol/l. During storage there is a slow, but constant leakage of potassium from the cells into surrounding plasma as a result of sodium/potassium ATPase pump failure. The plasma level of potassium may increase by 0.5–1 mmol/l per day of refrigerator storage.¹ Accordingly, the total amount of extracellular potassium in a unit of whole blood stored for 35 days is about 8.2 mmol and that of red cell concentrate is 5.5 mmol.² On collection into a blood bag containing CPD or CPDA-1 solution (pH 5.5), the pH of blood decreases to approximately 7.0. The pH continues to decline further and may be as low as 6.6 after 21–35 days of storage. This is not only due to citrate in the anticoagulant, but also due to the metabolic processes in the red blood cells, resulting in accumulation of fixed acids and CO₂.³ The potassium level of the recipient is determined by the factors summarised in box 2.

Potassium levels in transfusion recipients are determined by

- amount of extracellular potassium in the blood infused
- pre-existing level of potassium in the recipient
- rate of transfusion of blood
- renal excretion of potassium
- acidaemia/acidosis

Box 2

Discussion

Circulatory collapse on declamping the aorta is a frequent occurrence during aortic surgery.

The commonest cause as confirmed by two-dimensional transoesophageal echocardiography is severe hypovolaemia.⁴ If hypotension persists despite volume resuscitation for more than 4 min, re-clamping of the aorta below the renal arteries is recommended.⁴

In the present case, after unsuccessful volume resuscitation, the aorta was partially re-clamped (supra-coeliac) to increase the proximal arterial pressure and to maintain coronary perfusion. In the presence of a supra-coeliac aortic cross-clamp the coronary blood flow is increased by 43–46%.⁵ As the aorta was still partially clamped, the major quantity of citrated, hyperkalaemic, acidotic blood might have circulated in the upper part of the body, including the coronary arteries, producing widened QRS complexes, ventricular fibrillation and a 'flabby' heart. The impact of transfusion of stored blood on the potassium and acid-base status of the recipient is complex. It depends upon the rate of transfusion, the rate of citrate metabolism and the changing state of peripheral perfusion of the recipient.^{6,7} Although we did not measure plasma citrate concentration in our patient, citrate toxicity cannot be excluded, as massive blood transfusion can result in a plasma concentration of 2–4 mmol/l leading to myocardial depression and hypotension.⁸

The other possible cause of ventricular fibrillation is coronary ischaemia, which cannot be excluded, though there were no ST segment and T-wave changes in the ECG leads monitored prior to the events described.

The homeostatic disturbances which follow transfusion therapy are related to both the quality and quantity of blood infused. Weisel and co-workers have suggested that massive transfusion of blood with a low 2,3-diphosphoglycerate concentration may impair myocardial performance.⁹ Blood also contains important free radical scavenging systems, and with storage a decline in red cell¹⁰ as well as plasma¹¹ antioxidant activity has been reported. As associated coronary artery disease has been reported in patients with aortic aneurysm,¹² the use of fresher blood could be considered when such patients require massive transfusion. Although we did not use cell saver, it has an important role in minimising homologous blood transfusion in major surgeries.

In conclusion, potassium toxicity and citrate intoxication need to be considered during resuscitation of an acutely injured patient in the accident & emergency department or a patient bleeding in the operating theatre after aortic surgery. We believe that fresher blood may be beneficial in preventing these complications.

Final diagnosis

Hyperkalaemia following rapid blood transfusion during thoraco-abdominal aortic reconstructive surgery.

Keywords: aortic surgery; blood transfusion; hyperkalaemia; citrate toxicity

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A wheezy man with a bony abnormality

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A 56-year-old man presented to the chest clinic with worsening wheeze. His general practitioner had diagnosed asthma 3 years previously. At the time of presentation, he was taking inhaled terbutaline as required and budesonide 400 µg bid. Prednisolone 15 mg daily had been added following a recent exacerbation. He had been unable to work for 6 months owing to frequent attacks of asthma. He had stopped smoking 4 years previously but before this had had a 20 pack year history. On examination there was mild bilateral wheeze. Pulmonary function tests showed an obstructive pattern without significant reversibility. Chest X-ray showed hypertransradiancy of the left lung with normal appearance of the left pulmonary artery (figure 1). A computed tomography (CT) scan was performed (figure 2).

Figure 1 X-ray of the chest showing hypertransradiancy of the left lung

Figure 2 CT scan of the thorax at the level of the left main bronchus

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Questions

- 1 What does the CT scan show?
- 2 How would you further investigate and manage this patient?