POTASSIUM AND MASSIVE BLOOD TRANSFUSION

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ABSTRACT

Sixty patients who received massive blood transfusion intraoperatively and/or in the immediate post-operative period were analysed. Six patients had hypokalemia and two had hyperkalemia. The multifactorial changes leading to electrolyte disturbances especially involving potassium are discussed in relation to hypotension, hypothermia, acidosis, pH, and release of catecholamine. Potassium changes in relation to anaesthesia are discussed. The danger of routine administration of calcium during massive blood transfusion is stressed.

Keywords: Potassium, massive blood transfusion, anaesthesia

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INTRODUCTION

With the advent of safer anaesthetic techniques, massive blood transfusion is becoming common practice. Massive blood transfusion is defined as the administration of 10 or more units of blood within a 24 hour period[1]. It is widely accepted that massive blood transfusion can lead to electrolyte disturbances especially potassium[1,2]. Most are of the view that hypokalemia is more common than hyperkalemia[2-6]. The changes in serum potassium may be primary as a result of acid base disorder, haemorrhagic shock, hypotension, catecholamine release, secondary to hypothermia or the rate of transfusion[2,3,5-8]. The purpose of this study was to analyse potassium abnormalities both intraoperatively and post-operatively.

MATERIAL AND METHOD

A total of 1,865 cases who had received more than one unit of blood during surgery in Hospital Universiti Sains Malaysia from 1985 - 88 were retrospectively reviewed. Out of these, 60 patients had received massive blood transfusion during surgery (Table I). Patients who were less than 18 years of age, and those who did not receive a minimum of 10 units within 24 hours were excluded. Patients suffering from medical disorders were included so as to compare their outcome with those without such ailments. There were 27 males and 33 females. The mean age was 29 years (range 21-72). There were 14 elective and 46 emergency cases.

RESULTS

Shock and Resuscitation

Twenty-one patients were in hypovolemic shock prior to surgery. They were resuscitated with blood ranging between 1 to 3 units in addition to Ringer's lactate ranging between 1 to 2 L.

Table I
Indications for massive blood transfusion

Obstetrics	n = 30	Surgery	n = 21
CPD	1	Intra abdominal Injury	6
PP	16	Carcinoma Thyroid	2
PPH	9	Liver Injury	5
Ruptured Uterus	2	BPH	6
Retained Placenta	2	Poly Trauma	1
		Renal Stone	1
Orthopaedics	**n = 5**	**Neuro Surgery**	**n = 4**
Fractured Femur	3	Head Injury	3
Fractured Pelvis	2	Intra cranial Tumour	1

CPD = Cephalo Pelvic Disproportion
PP = Placenta Praevia
PPH = Post Partum Haemorrhage
BPH = Benign Prostatic Hypertrophy

Investigations

Fifty-two patients had potassium within normal limits (range 3.5-5.0 mmol/l). Six patients were hypokalemic (<3.5 mmol/l) and 2 were hyperkalemic (>5.5 mmol/l). Pre-operative blood gas was not routinely done. None of the patients were hypo or hyperthermic. Fourteen patients had medical problems which were well controlled (diabetes, hypertension, B. asthma, COPD).

Intraoperative Results

The minimum value of blood transfused was 5L and the maximum was 14 L with an average of 5.2 L. Blood and crystalloids were routinely warmed using portex blood warmer waterbath. The duration of blood transfusion varied from 2 to 20 hours with an average of 5 hours. All the patients received Ringer's lactate as resuscitative fluid in addition to blood. The volume of Ringer's lactate varied from 2.5L to 16 L with an average of 6 L per patient. Two patients received 1 L of gelatin (haemaccele) and 5 received fresh frozen plasma (F.F.P) varying from 1 to 3 units to correct coagulation defects.

Intraoperatively 9 patients had hypovolemic shock. Intraoperative potassium was not available in 4 patients and 48 patients had normal range of potassium. Hypokalemia was noted ranging from 2.7 mmol/l to 3.4 mmol/l in 6 (10%) patients and 2 (3.33%) had significant hyperkalemia (6.1 and 7.2 mmol/l). The blood pH ranged from 7.30 to 7.56. Five patients were

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acidotic and three were alkalotic (Table II). Urine output ranged between 1-1.5 ml/kg/hr. None of the patients received diuretics or mannitol during surgery.

Table II
Correlation of potassium to Blood, pH and shock

Age (years)	Diagnosis	Blood/RL Units	K mmol/l	pH	Hypo-tension*
26	PPH	10/12	2.7	7.30	Yes
35	Liver Injury	15/15	3.1	7.50	No
34	MVA Poly trauma	26/32	3.0	7.00	Yes
45	Head Injury	10/12	2.7	7.56	No
39	Ruptured Uterus	13/15	3.0	7.31	Yes
35	PPH	18/20	3.1	7.50	No
32	Liver Injury	10/12	7.2	7.20	No
20	Neurofibroma	13/16	6.1	7.32	No

K = Potassium
RL = Ringer's lactate
* = Systolic blood pressure below 90 mm Hg

Postoperative Management

Twenty-five patients were admitted to intensive care for ventilatory support and monitoring. Patients who were hypokalemic during massive blood transfusion received potassium supplements. The 4 patients in whom potassium was not determined in the intraoperative period had normal range of potassium in the post operative period. On the 1st post operative day, 12 patients were hypokalemic, ranging from 2.2 to 3.4 mmol/l with an average of 2.7 mmol/l. On the 2nd post operative day hypokalemia ranging from 2.9 to 3.4 mmol/l was seen in 7 patients with an average of 3.1 mmol/l. Hyperkalemia was noted in one patient. Out of 14 patients who had associated medical illness 2 (14.28%) patients had hypokalemia intraoperatively. Out of 3 patients who died postoperatively 1 was hypokalemic and 1 was hyperkalemic.

DISCUSSION

In the management of massive blood transfusion much attention has been directed to the electrolyte changes, notably potassium. Potassium is of unique concern to the anaesthesiologist since there may be a marked, sometimes fatal rise in serum potassium after the administration of succinylcholine to patients who have sustained major trauma, burns, spinal cord injuries and neuromuscular disease. Hypokalemia may worsen digitalis toxicity potentiate the activity of non-depolarising muscle relaxants, respiratory insufficiency, cardiac arrhythmias and post-operative paralytic ileus[9,10]. Intravenous calcium injections which are often recommended during massive blood transfusion may worsen the hypokalemic effect[11].

The occurrence of hypokalemia during massive blood transfusion is found to be of multifactorial in origin. Illner and Shrines[7] have shown that in haemorrhagic shock there is an inhibition of sodium and potassium pump in the cell membrane, with a preferential increase in the concentration of potassium in the interstitial compartment. Carmichael[3] found 14 patients in hypotension and 8 were hypokalemic. Shin[12], in a study of 212 cases, found 56 patients in shock and 185 in hypokalemia. In our series we had 30 (50%) patients in shock, out of which 3 (10%) were hypokalemic.

Simmons[5] found that the potassium level corresponded to the pH of the blood rather than to the causative factor namely, respiratory or metabolic alkalosis. Wilson[1] noted a pH of 7.51 was associated with a decrease of 0.5 mmol/l in the plasma potassium concentration. Wilson et al[4] reported that out of 31 patients receiving massive blood transfusion, 9 (29%) had alkalosis and 6 were hypokalemic. Carmichael[3] found 8 out of 15 patients (53%) were alkalotic, among them 3 (38%) were hypokalemic. In our study 21 (32.30%) were alkalotic and hypokalemia was seen in 3 (14.28%) patients.

Vincent[6] suggested a rise in plasma catecholamine secretion secondary to stress, pain or beta 1 agonists may cause a decrease in serum potassium concentration by driving potassium into the cell. Although epinephrine levels were not measured in our study, it is likely that epinephrine levels might be high.

Hypothermia may cause hypokalemia by reactivation of the pump shifting potassium into the cell[4]. Even though we have used blood warmers it may be difficult to exclude hypothermia as a cause of hypokalemia since core temperature was not monitored.

Many factors have been incriminated in the causation of hyperkalemia. Even carefully stored blood has a plasma potassium concentration of about 17 mmol/l after 21 days storage. Linko[2] found transient hyperkalemia in 11 patients out of 21 receiving massive blood transfusion. This was attributed to rate of transfusion, and/or acidosis of respiratory of metabolic origin.

Other factors which are incriminated in the causation of hyperkalemia include the rapid potassium loss from damaged anoxic tissues, and the reduced potassium excretion associated with oliguria. Linko[2] and Wilson[4] reported hyperkalemia in 52.36% and 12.1% respectively. In our series the incidence of hyperkalemia was 3.07%.

Massive infusion of Ringer's lactate per se should not give rise to hypokalemia as its potassium content is same as that of plasma. We were unable to correlate with other studies as no data was available.

Underlying medical illness did not increase the risk of hypo or hyperkalemia. In our study 11.8% without medical history were hypokalemic compared to 13.5% of hypokalemia with medical illness. None of the patients with medical problems exhibited hyperkalemia.

The wide difference in the incidence of potassium changes is to be expected in view of multifactorial origin[8].

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