EXTREME INTRAOPERATIVE HYPERKALEMIA
IN A NON DIALYSIS PATIENT UNDERGOING KIDNEY TRANSPLANTATION

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Abstract
The majority of patients who present for kidney transplantation have end stage renal disease and are on dialysis. Those patients are known to be at risk for the development of hyperkalemia. A patient who has not required dialysis, and with stable potassium levels would not be expected to acutely develop intraoperative hyperkalemia. Presented here is an unusual case in which a 61-year-old man with chronic renal disease but no history of dialysis developed severe intraoperative hyperkalemia during a renal transplant.

Case Report
A 61-year-old man with end stage renal disease secondary to polycystic kidney disease presented for right sided living unrelated renal transplant. Past medical history was also significant for hypertension and hyperlipidemia, and past surgical history revealed bilateral cataract repair. The patient had no history of dialysis. Home medications included atenolol, enalapril, and simvastatin. Physical examination revealed a well developed, well nourished man weighing 78 kg and measuring 176 cm; it was otherwise unremarkable. Preoperative EKG was normal. Preoperative comprehensive metabolic panel revealed a BUN of 70 mg/dl, a creatinine of 5.3 mg/dl, and a potassium of 5.1 mEq/L. Review of past potassium levels revealed a level of 5.4 mEq/L 5 days prior to surgery, and an earlier level of 5.5mEq/L. He had nothing per orem (NPO) after midnight on the night prior to surgery.

In the operating room, standard American Society of Anesthesiologists monitors were applied to the patient. Initial vital signs included heart rate of 52 beats/min with normal sinus rhythm and blood pressure of 135/88 mmHg. Following induction of general anesthesia with 2 mg of midazolam, 200 mg of propofol, 200 mcg of fentanyl, and 10 mg of vecuronium, an endotracheal was placed and anesthesia was maintained with desflurane. An arterial line was placed following the induction of anesthesia, and an arterial blood gas (ABG) shortly after the start of surgery revealed pH 7.28, paCO2 39 mmHg, paO2 284 mmHg, bicarbonate 18 mEq/L, glucose 94 mg/dl, and potassium 7.5 mEq/L. The electrocardiogram (ECG) revealed peak T waves. Repeat arterial blood gas confirmed elevated potassium at 7.4 mEq/L. Fifty mEq of sodium bicarbonate was given, followed by 25 g of dextrose, 1000 mg of CaCl2, and 10 units of regular insulin. When a subsequent ABG showed potassium 6.9 mEq/L and 244 mg/dL of glucose, 1000 mg of CaCl2 and 5 units of regular insulin were given. The next ABG taken 30 minutes later showed potassium = 5.2 mEq/L and glucose = 87 mg/dL, after which 12.5 g of dextrose was infused. Subsequent ABGs during the procedure revealed potassium levels <5 mEq/L and glucose levels in the 50s to 80s,
prompting a total of 37.5 g of dextrose to be given. The peaked T waves resolved as the potassium level became normalized. Two L of .9% NaCl, and 4 L of 45% NaCl, each with 75 mEq of bicarbonate, were administered intravenously (IV). Urine output was 2800cc.

The kidney was transplanted successfully into the right iliac fossa with minimal blood loss. Total surgery time was 5 hours 9 minutes. Postoperative potassium level was 3.8 mEq/L. For the remainder of the hospital stay, it remained <5 mEq/L. The patient made an uneventful recovery, the transplanted kidney functioned well with consistently adequate urine output, and he was discharged home on POD 3.

Discussion

This case is of particular interest in that the patient had no history of dialysis and had a record of past stable potassium levels in the range of low to mid 5 mEqs/L. Cases of intraoperative hyperkalemia may be attributable to RBC transfusions, extensive muscle dissection, metabolic acidosis, use of angiotensin receptor blockers (ARBs) or angiotensin converting enzyme (ACE) inhibitors, use of succinylcholine, and propofol infusion syndrome (1-3). In our patient with chronic renal disease, there is not a clear explanation for the acute and dramatic rise in potassium during this procedure. He did not receive any blood during the procedure, there was limited muscle dissection, and there was no propofol infusion. There was no potassium in the intravenous fluid at any time. Possible causes for his hyperkalemia include consequence of intraoperative metabolic acidosis which was apparent by the low bicarbonate level, administration of enalapril prior to surgery, and coincidental worsening of renal failure. It could have possibly been related to the ingestion of foods containing potassium, and then subsequent dehydration from the standard maintenance of NPO status preoperatively.

Patients with a history of chronic renal failure, especially those on ACEIs/ARBs, should be routinely monitored for potassium and creatinine levels. Raebel et al2 analyzed the characteristics of patients on ACEIs/ARBs that were predictive of compliance with routine potassium and creatinine monitoring. They found that risk factors of older age, comorbid chronic renal disease, heart failure, or diabetes, recent hospitalization, higher number of outpatient visits, and therapy with diuretics, potassium supplements, or digoxin increased the likelihood of monitoring.

Intraoperative treatment relied on administration of medications to actively lower potassium; this entailed giving sodium bicarbonate, insulin, and calcium. In addition, switch of fluids to 45% NaCl with sodium bicarbonate after diagnosis of hyperkalemia sought to prevent further rise in potassium by providing alkaline IV fluid instead acidic fluid. With continual insulin boluses, glucose levels were monitored to avoid hypoglycemia. In all kidney transplantation cases, the electrolyte levels should be assessed on the day of surgery. An arterial line should be placed and levels sampled intraoperatively in order to assess for acute changes and monitor treatments.

References