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Intraoperative Hyperkalemia and Cardiac Arrests during Renal Transplantation in an Insulin-dependent Diabetic Patient

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Renal potassium excretion and cellular potassium uptake play vital roles in the body's defense against hyperkalemia.[1] Both mechanisms for regulating serum potassium are impaired in the diabetic patient who has chronic renal failure. Therefore, such patients may show dramatic elevations in serum potassium. An insulin-dependent diabetic patient in chronic renal failure, while undergoing two separate procedures for renal transplantation, 18 months apart, had cardiac arrests secondary to hyperkalemia.

REPORT OF A CASE

First Renal Transplantation. A 34-year-old woman who had juvenile-onset diabetes was scheduled for transplantation of a kidney from a living donor. Problems included hypertension and diabetic nephropathy with chronic renal failure, treated by hemodialysis. The patient's most recent dialysis had been performed two days prior to operation. Her usual dose of insulin had been given 24 hours preoperatively. She usually received 30 units of NPH and 5 units of regular insulin each morning, as well as alpha-methyldopa, 250 mg, *b.i.d.* Diabetic management had been difficult preoperatively, and blood glucose varied between 60 and 600 mg/dl. Blood glucose was 64 mg/dl at midnight, eight hours prior to operation. Blood urea nitrogen (BUN) was 47 mg/dl, creatinine 9.5 mg/dl, and arterial blood-gas analysis one day prior to operation revealed *p*H of 7.45, Pa_{O_2} 80 torr, Pa_{CO_2} 32 torr.

At 5 A.M. on the day of operation, blood glucose was 129 mg/dl, Na^+ 128 mEq/l, K^+ 5.2 mEq/l, Cl^- 94 mEq/l, and hematocrit (Hct) 22.5 per cent. Premedication included diazapam, 10 mg, *p.o.*, and methylprednisolone, 750 mg, iv. Insulin was not given. Five per cent dextrose in half-normal saline solution was started iv. An electrocardiogram revealed sinus rhythm with normal QRS and T waves. After preoxygenation, the patient received *d*-tubocurarine, 3 mg, iv, and anesthesia was induced with thiopental. Tracheal intubation was accomplished with difficulty after the patient had been given 120 mg succinylcholine. Anesthesia was then maintained with 50 per cent nitrous oxide in oxygen with 0.5 per cent halothane and intermittent doses of *d*-tubocurarine. Blood glucose, estimated by Dextrostix®, was 150 mg/dl one hour, 200 mg/dl two and a half hours, and 250 mg/dl four hours after induction. Insulin was not administered during this time. Blood pressure varied between 180/100 and 120/80 torr throughout. Temperature remained stable at 36 C. Five hours after induction, during surgical closure, widening of the QRS and peaking of the T waves was observed on the electrocardiogram. Potassium concentration in serum drawn at that time was later reported to be 7.9 mEq/l, and arterial blood-gas analysis revealed Pa_{O_2} 195 torr, Pa_{CO_2} 37 torr, and *p*H 7.21. Approximately 3 min later, cardiac arrest occurred. The patient was successfully resuscitated with external cardiac massage, ventilation with 100 per cent oxygen, and intravenous administration of sodium bicarbonate, insulin, calcium chloride, lidocaine and 50 per cent glucose. Serum potassium after resuscitation was 6.8 mEq/l, and arterial blood-gas analysis revealed Pa_{O_2} 210 torr, Pa_{CO_2} 34 torr, and *p*H 7.31. At the termination of anesthesia, six hours after induction, *p*H was 7.41, Pa_{O_2} 190 torr, Pa_{CO_2} 33 torr, and K^+ 6.1 mEq/l. The patient awakened, and the trachea was extubated; heart rate was 120/min and blood pressure, 180/90 torr.

Renal kidney function gradually deteriorated over the next year, and the patient underwent three surgical procedures with no difficulty: cystoscopy with spinal anesthesia; creation of an arteriovenous fistula with brachial plexus blockade; nephrectomy of the transplanted kidney with halothane anesthesia. In the latter procedure succinylcholine was used to aid intubation and electrolytes and arterial blood gases, measured several times during the four-hour procedure, were normal. The patient had received half her usual dose of lente insulin preoperatively and blood glucose during the operation was 250 mg/dl.

Second Renal Transplantation. A year and seven months after her first transplant, the patient was admitted to the hospital for renal transplantation with a cadaver kidney. Her medications included lente insulin, 30 units per day, and hydralazine, 50 mg, *t.i.d.* She had undergone dialysis at home on the day of admission. On the following morning, blood glucose was 56 mg/dl and serum K^+ was 4.2 mEq/l. Lente insulin, 15 units, and methylprednisolone, 750 mg, were given at 7 A.M., and an intravenous infusion was started with 10 per cent dextrose in water. The patient received no premedication, and was taken to the operating room at 2 P.M. Blood pressure was 180/100 torr. *d*-Tubocurarine, 3 mg, was given prior to induction of anesthesia with thiopental. Tracheal intubation was difficult, but was accomplished after the administration of three doses of succinylcholine, totaling 160 mg. Anesthesia was maintained with 50 per cent nitrous oxide in oxygen, 0.8 per cent halothane, and intermittent doses of pancuronium. An electrocardiogram showed a sinus rhythm with normal QRS and T waves. Blood glucose, estimated by Dextrostix, was 150–175 mg/dl intraoperatively. Two hours, 30 min after induction, the renal-artery clamps were released. Immediately thereafter the QRS complex on the ECG widened, the T waves became slightly more prominent, and within seconds ventricular fibrillation developed. The patient was resuscitated with external cardiac massage, intravenous administration of sodium bicarbonate, lidocaine, insulin, 50 per cent dextrose, intracardiac epinephrine, and 3 DC shocks, which resulted in slow sinus rhythm. After intravenous administration of atropine, normal heart rate and blood pressure were restored. Blood-gas analysis, after conversion to sinus rhythm, revealed *p*H 7.12, Pa_{O_2} 240 torr, and Pa_{CO_2} 44 torr; serum K^+ was 5.2 mEq/l. Additional bicarbonate was given. The patient awakened promptly and her trachea was extubated the following day. She was discharged from the hospital two weeks later.

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Discussion

The first cardiac arrest was due to well-documented hyperkalemia. The factors responsible for this include the patient's underlying disease—diabetes mellitus, with decreased circulating insulin and decreased renal function, repeated doses of succinylcholine, a massive dose of methylprednisolone, and metabolic acidosis. Insulinopenia results in diminished ability to transfer potassium into cells. Moreover, hyperglycemia results in a movement of potassium out of cells. Hyperglycemia may be aggravated by the administration of a massive dose of glucocorticoid. Insulin was not given on the morning of the operation. This patient's insulin requirements were difficult to predict, and she was relatively hypoglycemic the evening prior to operation. Intraoperatively, blood glucose levels were rising but did not exceed 250 mg/dl, and the patient received no insulin prior to cardiac arrest.

Serum potassium increases 0.5–0.7 mEq/l following the injection of succinylcholine in both normal and uremic patients.[2,3] This small increase did not precipitate the cardiac arrest in our patient, although it could have been a factor contributing to a gradual rise in serum potassium.

Aldosterone deficiency may contribute to the development of hyperkalemia even in the absence of renal function by altering the non-renal cellular uptake of potassium.[4] Serum aldosterone levels were not measured in our patient.

While the above-mentioned factors may have contributed to a gradual increase in serum potassium, the major factor that led to hyperkalemia and cardiac arrest during the first renal transplantation was untreated metabolic acidosis. Immediately prior to the first cardiac arrest arterial blood *p*H was 7.21, with Pa_{CO_2} 37 torr. The intracellular potassium concentration is 25 times the extracellular potassium concentration. Therefore, shifts of a relatively small fraction of the total intracellular potassium to the extracellular compartment will produce major changes in serum potassium concentration.[1] Possible causes of metabolic acidosis in our patient included her chronic renal failure, diabetic ketoacidosis, and influx of lactic acid following restoration of circulation to the hypoperfused limb following completion of the vascular anastomosis.

The second cardiac arrest immediately followed the release of the vascular clamps connecting the cadaver kidney to the patient's circulation. At that time the patient could well have received a large amount of potassium from the kidney, which had been perfused with Collins' solution, a preservative containing 115 mEq/l potassium.[5] Although most of this solution was drained from the kidney prior to completion of the vascular anastomosis, it is likely that some of this solution remained in the kidney and entered the circulation as a bolus when the vascular clamps were released. It has been shown that bolus doses of 0.1 mEq/kg potassium can result in transient peak plasma potassium levels of 10 mEq/l.[6] Moreover, Soulillou *et al.*[7] showed right atrial serum potassium concentration increase of as much as 5.3 mEq/l 5 sec after release of the renal vascular clamps in eight patients receiving kidneys perfused with Collins' solution. Their study was prompted by a hyperkalemic cardiac arrest in a patient receiving a kidney perfused with Collins' solution following release of the vascular clamps.

In summary, it appears that hyperkalemia was definitely the cause of the first cardiac arrest, and probably the cause of the second arrest. Different factors may have been responsible for the hyperkalemia during each transplantation. The two cardiac arrests experienced by this patient emphasize the importance of proper preoperative management of both diabetes and renal failure, and the importance of intraoperative prevention of acidosis and hyperkalemia. The transplantation of cadaver kidneys filled with Collins' solution should alert the anesthesiologist to the possible hazard of acute hyperkalemia upon release of the vascular clamps.

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