Rapid onset severe hyperkalemia during robotic radical cystectomy: a case report

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Radical cystectomy is a preferred treatment for muscle-invasive bladder cancer. Despite known complications, rapid onset, severe hyperkalemia necessitating abortion of surgery has not been reported. In this case report, a patient with end stage renal disease (ESRD) undergoing attempted cystectomy developed severe intraoperative hyperkalemia and acidosis that led to abortion of surgery and transfer to the medical intensive care unit for emergent hemodialysis. The multifactorial etiology was related to respiratory acidosis, ESRD, patient positioning, clipping of ureters, and body habitus, as well as an idiopathic element. Knowledge of hyperkalemia etiologies can assist in diagnosis and treatment of this serious condition.

Key Words: hyperkalemia, bladder cancer, cystectomy, end stage renal disease, intraoperative complication

Introduction

Rapid onset, severe hyperkalemia is a medical emergency that must be promptly treated to avoid myocardiocyte instability that could lead to cardiac arrythmias and possible death. While some cases of moderate hyperkalemia have been reported during cystectomy, these were not well characterized and were not noted to have rapid onset of severe hyperkalemia.[1] A recent case report described a thoracic surgical case utilizing one-lung ventilation where severe hyperkalemia was incidentally observed, although the case was able to proceed.[2] Rare, rapid onset, severe hyperkalemia poses a distinct threat to patients. This report describes such a case and provides a brief overview of common etiologies of intraoperative hyperkalemia.

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Case presentation

A 55-year-old man with hypertension, obesity (BMI 36), and a 2-year history of end stage renal disease (ESRD) on daily peritoneal dialysis (making 1 L of urine per day) was undergoing work up for transplantation when a bladder tumor was discovered on CT imaging, Figure 1. His medications included calcitriol, calcium acetate, sevelamer, cinacalcet, carvedilol, clonidine, and losartan. He underwent a TURBT without issue and the pathology showed invasive moderately-differentiated adenocarcinoma of the bladder along with Gleason 6 acinar focal adenocarcinoma of the prostate. He was scheduled for radical cystoprostatectomy. Preoperative labs were significant for a potassium of 5.3 mmol/L [reference range: 3.5-5.0 mmol/L] and creatinine of 17.4 mg/dL [reference range 0.7-1.3 mg/dL]. Two days prior to the surgery the patient had a tunneled catheter placed for hemodialysis. His last dose of losartan was the day prior to surgery. On the morning of his surgery, his potassium was 4.8 mmol/L.

Anesthesia induction was performed using propofol, rocuronium was used for muscle relaxation,

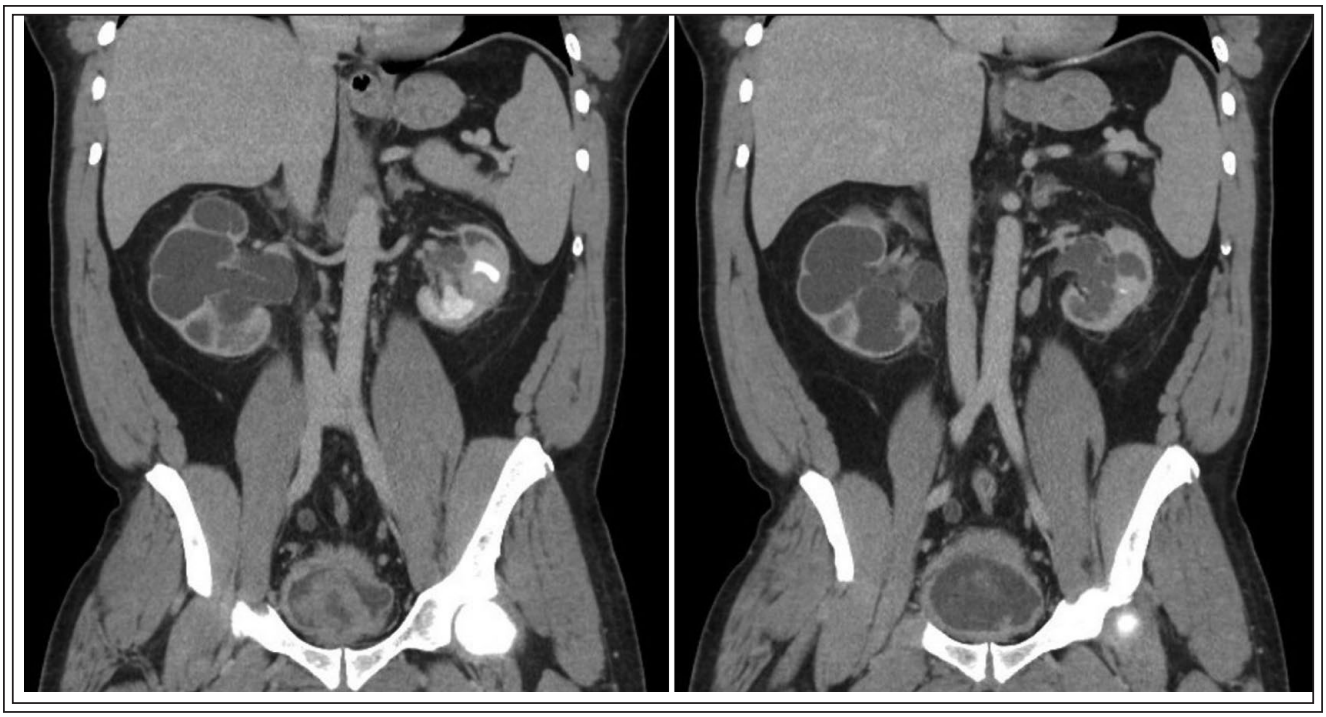

Figure 1. Coronal abdominal CT scan showing bladder tumor and severe bilateral hydronephrosis.

sevoflurane was used for maintenance anesthesia, and a total of 0.6 L normal saline was given for maintenance fluids throughout the procedure. The robot was docked, the patient was placed in the Trendelenburg position, and pneumoperitoneum was established. The patient's habitus was notable for a large, tight abdominal cavity. He was mechanically ventilated on pressure control settings with tidal volumes in the 300 mL range due to high peak inspiratory pressures between 27-43 cmH_20.

Intraoperative findings were significant for bowel distention, and a peritoneal lesion adjacent to the bladder which was found to be cancer on rapid frozen section, Figure 2, consistent with a locally invasive disease. Due to lack of good systemic options the decision was made to proceed with surgery and the ureters were clipped and transected. Two hours after the start of the surgery the patient was noted to have a potassium of 8.0 mmol/L with a pH 7.1 on arterial blood gas. The potassium value was initially suspected to be spurious. Venous blood gas at 2 hours and 15 minutes after surgery was 8.6 mmol/L. Given the confirmed severe hyperkalemia and the planned surgery still needing multiple hours for completion the decision was made to abort the surgery. The patient was given insulin (15 units), 8 puffs of albuterol, 2000 mg calcium chloride for cardiac stabilization, and 100 mg sodium bicarbonate for treatment of the acidosis. The patient was taken out of Trendelenburg position, the incisions were quickly closed and the surgery terminated. A repeat blood draw 3 hours

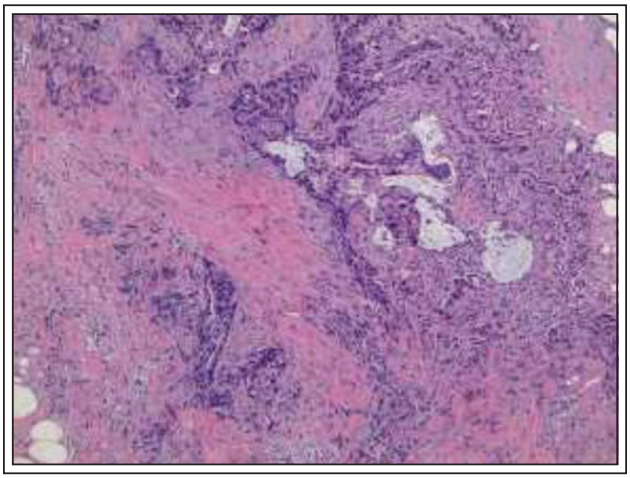

Figure 2. Metastatic moderately-differentiated adenocarcinoma with associated mucin, seen on Hematoxylin and Eosin stain of the rapid frozen section taken from the peritoneal lesion seen intraoperatively. Histology is similar to specimens obtained during TURBT.

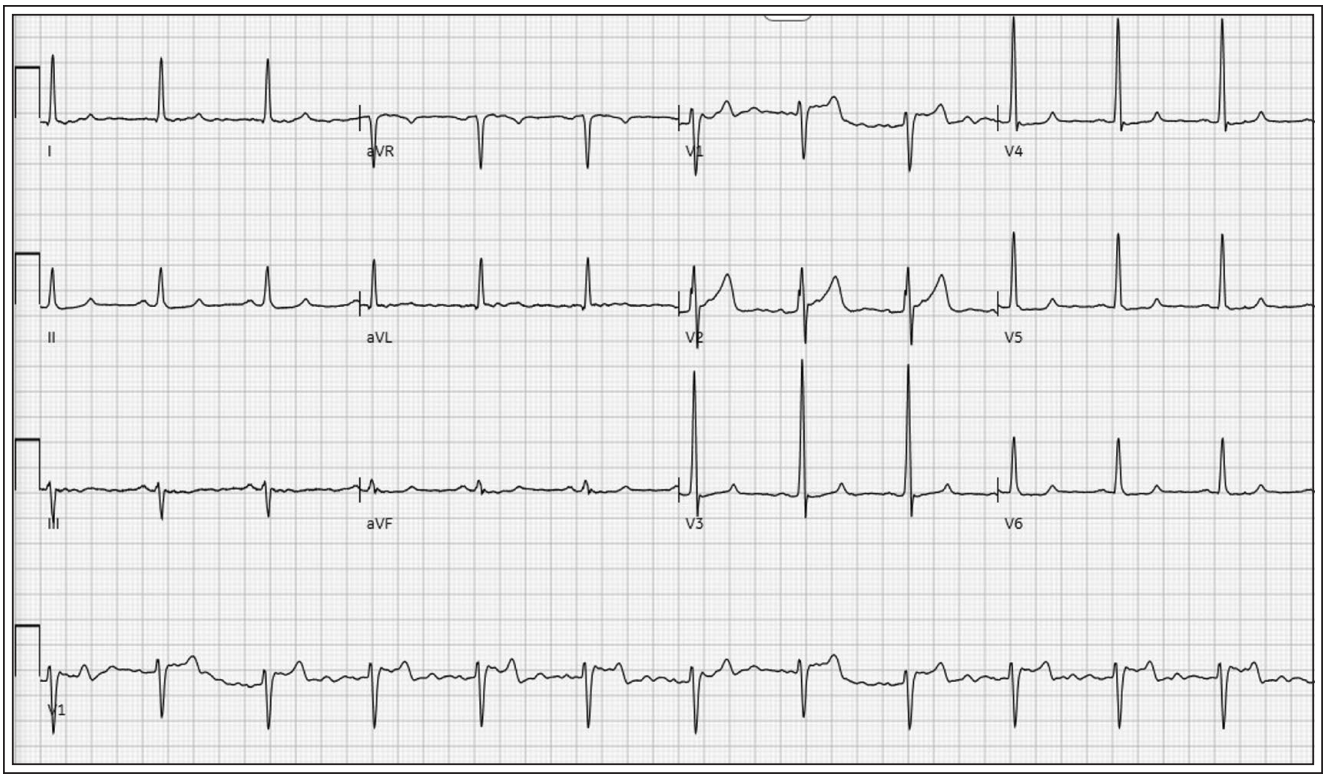

Figure 3. Electrocardiogram performed upon arrival to the post anesthesia care unit. No peaked T-waves are seen, although these are not necessarily always present in hyperkalemia.

after surgery start showed the potassium continued to be 8.6 mmol/L. No peaked T-waves were seen on electrocardiogram, Figure 3. The patient was taken to the post-ambulatory care unit intubated, and the potassium had decreased to 6.1 mmol/L. He was subsequently transferred to the medical intensive care unit. The potassium rose again, reaching 7.9 mmol/L at around 12 hours from procedure start time. Nephrology consultation was obtained, and the patient started on hemodialysis which brought the potassium down to 4.4 mmol/L at the 18-hour mark. The patient was extubated without issue on postoperative day one and the peritoneal metastasis and prognosis was discussed. The decision was made not to return to the operating room to complete the surgery. He clinically improved and was discharged without further complications on postoperative day 4. Further discussion with anesthesiology and nephrology did not identify a clear etiology for his extreme hyperkalemia besides his acidosis and ESRD.

Discussion

Hyperkalemia is a complication which must be promptly treated. As potassium rises, extracellular potassium ions are present at higher concentrations and cause the myocardiocyte to be closer to its depolarization threshold, leading to heightened excitability. The increased ability to reach threshold increases the likelihood of inappropriate action potentials and arrythmias. The classic EKG finding is peaked T-waves, though these do not always occur. A further rise in hyperkalemia and the associated decrease in membrane potential can inactivate sodium channels and activate potassium channels, leading to a slowing of conduction and an increase in refractory period.[3] Subsequent cessation of cardiac contractility and cardiac arrest can occur.

Multiple factors can contribute to hyperkalemia with ESRD being a major risk factor. Patients with ESRD undergoing surgery have been shown to have increased mortality when hemodialysis is performed more than 1 day before surgery, with increasing risk each extra day.[4] This emphasizes the importance of performing hemodialysis close to the time of surgery in this patient population. Aside from ESRD, a number of other etiologies exist for hyperkalemia, some of which are known to occur in the intraoperative setting. These include blood transfusion,[5] propofol infusion syndrome,[6] acidosis,

intraabdominal infections, or administration of succinyl choline,[7] hyperaldosteronism, hyperosmolar states, or digoixin,[8] as well as ACEI's/ARB's and hypovolemia.[9] While easily forgotten, extended application of a tourniquet or hemolysis can lead to factious hyperkalemia and should be kept in the differential.[10] Hyperkalemia secondary to respiratory acidosis induced by hypoventilation has also been recently reported.[2]

In this case, the etiology was likely multifactorial. The combination of hypoventilation and pneumoperitoneum in an individual with a large body habitus likely led to poor ventilatory status and subsequent respiratory acidosis. The day prior to surgery he had taken losartan 50 mg, which is known to cause hyperkalemia due to its inhibition of angiotensin, lack of aldosterone release, and subsequent decrease in sodium absorption that leads to a decrease in potassium excretion. The losartan could have had a contributing effect. The patient did not receive succinylcholine and received an average infusion of 0.50 mg/kg/hr of propofol, which is only a sixth of a previously reported case of hyperkalemia secondary to propofol infusion syndrome, indicating this was not the likely hyperkalemia etiology. Infusion of normal saline is known to cause acidosis, although the limited fluids in this case likely did not contribute significantly. To compensate for acidosis, hydrogen ions move intracellularly in exchange for the extracellular shift of potassium, leading to a redistribution of potassium stores and a rise in blood potassium. The patient's ESRD and clipped ureters meant he was unable to process this acid in the absence of effective respiratory compensation.

These factors highlight the importance of intraoperative monitoring, close communication with anesthesiology, an awareness of risk factors for hyperkalemia and a knowledge of treatment methods. Prompt recognition and treatment can help prevent potentially fatal cardiac complications of hyperkalemia and aborting of surgical procedure for medical management should be an option. □

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