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Robotic-Assisted Radical Cystectomy and Acute Intraoperative Hyperkalemia: A Case Series

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ABSTRACT

Taking into consideration the effects of pneumoperitoneum on kidney function, anesthesiologists should diligently monitor potassium levels during robotic-assisted radical cystectomy, particularly during prolonged ureter clamping, in the presence of chronic kidney disease, administration of renin-angiotensin-aldosterone system-blocking medications, or when there is a high risk of rhabdomyolysis.

1 | Introduction

Potassium plays a crucial role in cellular functions, and its serum concentration must be precisely maintained between 3.5 and 5.5 mEq/L [1]. Hyperkalemia, found in 7.3% of hospitalized patients, may range from mild asymptomatic to life-threatening [2]. Radical cystectomy is the gold standard treatment for patients with muscle-invasive or high-risk non-muscle-invasive urinary carcinoma of the bladder, with robot-assisted surgery offering benefits such as reduced blood loss and better postoperative recovery [3]. Though rarely reported [4], acute intra-operative hyperkalemia can arise during robotic radical cystectomy, as illustrated by the following two cases that were successfully managed.

This case series aims to describe the occurrence and management of intraoperative hyperkalemia without EKG changes in two cases of robotic radical cystectomy and to analyze the potential contributing factors to the development of hyperkalemia. This case series was written in compliance with the CARE Checklist for case reports (File S1). Both patients provided written informed consent to publish this report in accordance with the journal's patient consent policy.

2 | Case History

2.1 | Case 1

A 72-year-old man, American Society of Anesthesiologists (ASA) classification physical status II, body mass index (BMI) 30 kg/m^2, with a history of colon cancer treated with laparoscopic sigmoidectomy and childhood asthma, was diagnosed with high-grade muscle-invasive urothelial carcinoma of the bladder and scheduled for robotic-assisted cystectomy with intracorporeal neobladder creation and radical lymphadenectomy. General anesthesia was induced with intravenous injection of 100 mg of lidocaine, 100 mcg of fentanyl, 180 mg of propofol, and 100 mg of rocuronium. General anesthesia was maintained with a mixture of sevoflurane for a target minimum alveolar concentration (MAC) of 0.8–1, rocuronium boluses to maintain neuromuscular blockade, and remifentanil

Abbreviations: ABG, arterial blood gas; AKI, acute kidney injury; ASA, American Society of Anesthesiologists; BMI, body mass index; CK, creatine kinase; GFR, glomerular filtration rate; MAC, minimum alveolar concentration; RAAS, renin-angiotensin-aldosterone system; TOF, train of four.

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infusion ranging from 0.1 to 0.2 mcg/kg/min. The patient was monitored with EKG leads, pulse oximetry, waveform capnography, neuromuscular monitoring for train of four (TOF) ratio, esophageal temperature probe, and a radial arterial line. After incisions were made and trocars were placed, the robot was docked and position was changed from supine to steep Trendelenburg. The procedure began with the release of extensive left colonic adhesions resulting from the previous sigmoidectomy. After the induction of anesthesia, arterial blood gas (ABG) analysis showed a normal potassium level of 4.2 mEq/L. Five hours into the surgery, and after the ureters had been clamped for 3.5 h, the arterial potassium level was found to be 5.9 mEq/L.

2.2 | Case 2

A 64-year-old man, smoker, ASA classification physical status of III, BMI of 32 kg/m^2, known to have epilepsy controlled with valproate and clonazepam, diabetes mellitus type II managed with linagliptin and long-acting insulin, untreated hypertension, dyslipidemia on fenofibrate, chronic kidney disease with a baseline creatinine of 1.5 mg/dL, benign prostatic hypertrophy, and right colon cancer treated with right colectomy, was diagnosed with high-grade muscle invasive urothelial carcinoma of the bladder and scheduled for robotic-assisted cystectomy with intracorporeal neobladder creation and bilateral lymph node dissection. General anesthesia was induced with intravenous injection of 80 mg of lidocaine, 100 mcg of fentanyl, 150 mg of propofol, and 100 mg of rocuronium. General anesthesia was maintained with a mixture of sevoflurane for a target MAC of 0.8, rocuronium as needed to maintain neuromuscular blockade, and remifentanil infusion ranging from 0.05 to 0.1 mcg/kg/min. The patient was monitored with EKG leads, pulse oximetry, waveform capnography, neuromuscular monitoring for TOF ratio, esophageal temperature probe, and a radial arterial line. After incisions were made and trocars were placed, the robot was docked and position was changed from supine to steep Trendelenburg. The procedure began with the release of extensive small bowel adhesions resulting from the previous colectomy. After the induction of anesthesia, ABG showed a normal potassium level of 5 mEq/L. Four hours into the procedure, and after the ureters had been clamped for 2 h, the arterial potassium level was found to be 6.8 mEq/L.

3 | Methods

3.1 | Case 1

No ECG changes were noted, and the patient was hemodynamically stable. To treat his hyperkalemia, the patient was given 2 g of calcium gluconate, 5 units of regular insulin, and 40 mL of D30 water. At the end of the surgery, and before extubation, arterial potassium increased again to 6 mEq/L, and the patient was treated with the same previous regimen. An hour after extubation and transfer to the postanesthesia care unit, blood chemistries showed an acute kidney injury (AKI) with creatinine of 1.36 mg/dL. The estimated blood loss was 400 mL, primarily occurring during the adhesiolysis phase.

3.2 | Case 2

No ECG changes were noted, and the patient was hemodynamically stable. To treat his hyperkalemia, the patient was given 2 g of calcium gluconate, 10 units of regular insulin, and 40 mL of D30 water. Three hours later, potassium levels increased again to 6.8 mEq/L, and the patient was given another 10 units of regular insulin with 40 mL of D30 water. The patient was successfully extubated and transferred to the post-anesthesia care unit. Blood chemistries were withdrawn and showed an AKI with a creatinine of 2.12 mg/dL and a potassium of 5.8 mEq/L. The estimated blood loss totaled 700 mL, mostly occurring during adhesiolysis.

The levels of sodium, potassium, and creatinine were thoroughly monitored postoperatively and treated accordingly. Details of the perioperative variations of sodium, potassium, and kidney function are shown in Table 1 and intraoperative metrics and outcomes are shown in Table 2.

4 | Conclusion and Results

For both cases, potassium levels returned to normal, creatinine levels gradually decreased to baseline (Table 1), and the rest of the hospital stay was uneventful.

Our case series highlights the potential occurrence of intraoperative hyperkalemia during robotic cystectomy, emphasizing the importance of prompt diagnosis and treatment to prevent its complications. Taking into consideration the effects of pneumoperitoneum on kidney function, anesthesiologists should diligently monitor potassium levels during this procedure, particularly during prolonged ureter clamping, in the presence of chronic kidney disease, administration of RAAS-blocking medications, or when there is a high risk of rhabdomyolysis.

5 | Discussion

This case series reports on two patients with normal preoperative potassium levels who developed sudden intraoperative hyperkalemia during robotic cystectomy without EKG changes. The hyperkalemia was incidentally detected in both cases during blood gas analysis, which was performed mid-surgery to assess hemoglobin levels, lactate, glycemia, and acid–base status.

Hyperkalemia was adequately treated; however, the patients subsequently developed AKI, which resolved after a few days. To our knowledge, only one case was documented in the literature by Srinivasaraghavan et al. [4] describing an incident of hyperkalemia arising during robotic cystectomy for invasive bladder carcinoma and was identified subsequent to the appearance of EKG changes. However, case reports of intraoperative hyperkalemia during laparoscopic radical nephrectomy in a patient with underlying renal insufficiency [5] and during laparoscopic pelvic surgery and prostatectomy [6] had been previously published.

Hyperkalemia during robotic cystectomy can be explained by a multitude of factors, including prolonged ureteral clamping

TABLE 1 | Perioperative levels of sodium, potassium, creatinine and eGFR.

Case 1							
	Preoperative	**Five hours into the surgery (ureters clamped for 3.5 h)**	**After first hyperkalemia treatment**	**Before emergence**	**In PACU**	**Ten hours postoperative**	**Day 3 postoperative**
Sodium[a] (mEq/L)	142	135	138	129	138	139	143
Potassium[b] (mEq/L)	4.9	5.9	5.2	6	4.7	4.2	3.9
Creatinine[c] (mg/dL)	1	NA	1.46	NA	1.36	1.02	0.8
eGFR[d] (mL/min)	73	NA	47	NA	52	73	89

Case 2									
	Preoperative	**Five hours into the OR (ureters clamped for 2 h)**	**After first hyperkalemia treatment**	**Three hours later**	**Before emergence**	**In PACU**	**Ten hours postoperative**	**Day 3 postoperative**	**Day 6 postoperative**
Sodium[a] (meq/L)	135	136	136	135	137	137	136	136	138
Potassium[b] (meq/L)	5.1	6.8	5.2	6.8	5.6	5.8	6.9	4.4	3.6
Creatinin[c] (mg/dL)	1.52	NA	NA	NA	NA	2.12	2.2	2.3	1.56
eGFR[d] (mL/min)	48	NA	NA	NA	NA	32	30	29	46

[a]Sodium normal range 135–145 mEq/L.
[b]Potassium normal range 3.5–5.1 mEq/L.
[c]Creatinine normal range 0.6–1.2 mg/dL.
[d]eGFR normal range > 60 mL/min/1.73 m^2.

TABLE 2 | Intraoperative metrics.

	Case 1	Case 2
Procedure duration (h)	10	9
Ureters clamping duration (h)	6	5
Total crystalloid intake (L)	3.2	4.2
Urine output (before ureteral clamping and after completion of the uretero-ileal anastomosis)	> 0.5 mL/kg/h	> 0.5 mL/kg/h
Estimated blood loss (mL)	400	700
Blood transfusions	None	None

time, the effects of pneumoperitoneum on kidney function, and the effect of prolonged surgery leading to rhabdomyolysis.

Prolonged clamping of the ureters can contribute to the development of hyperkalemia. The physiologic mechanisms for this phenomenon include increased intratubular pressure [7], and disruption of renal circulation secondary to obstruction and subsequent inflammation, leading to a decrease in glomerular filtration rate (GFR) [8]. In a retrospective study by Ishiyama et al. [3], ureteral clamping time was found to be associated with AKI, and a clamping time > 210 min was shown to be associated with a higher incidence of acute kidney injury. The authors concluded that ureteral clamping time should be minimized to avoid potential AKI [3]. In this case series, the duration of clamping of the ureters was 6 h (360 min) and 5 h (300 min) in case 1 and case 2, respectively. However, the first reading of hyperkalemia was detected only 3.5 h (210 min) and 2 h (120 min) into ureteral clamping, for case 1 and case 2, respectively, suggesting that factors beyond ureteral clamping alone significantly contributed to this condition.

Furthermore, pneumoperitoneum has devastating effects on renal function. Raised intra-abdominal pressure leads to increased renal vascular resistance and direct compression on the renal parenchyma. The cardiac output is also decreased during pneumoperitoneum, further decreasing renal perfusion pressure. Additionally, renal damage may be caused by pneumoperitoneum-associated ischemia–reperfusion, leading to tissue damage [9].

In addition, hyperkalemia can be caused by rhabdomyolysis [10]. Prolonged positioning in the Trendelenburg position during robotic pelvic surgery can lead to tissue injury and impaired renal function. Skeletal muscle disruption and the release of its constituents into the circulation can lead to renal vasoconstriction, intraluminal cast formation, and direct myoglobin toxicity [10]. Risk factors that have been linked to the development of rhabdomyolysis during surgery are high BMI, long operative time, extreme tilt decubitus positioning, drugs, and pulmonary or cardiovascular disease [11]. Rhabdomyolysis is diagnosed with an elevated serum creatine kinase (CK). In our case series, CK levels were not obtained; thus, rhabdomyolysis could not be ruled out as a potential cause of hyperkalemia, especially since the patients had multiple risk factors for rhabdomyolysis, such as prolonged surgery and extreme positioning.

Moreover, patients with chronic renal disease may have hyporeninemic hypoaldosteronism, leading to decreased potassium renal excretion [5]. In case 2, the patient's chronic renal disease could have contributed to hyperkalemia. Also, some drugs such as angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, direct renin inhibitors, nonsteroidal anti-inflammatory drugs, and beta-agonists are known to cause renin-angiotensin-aldosterone system (RAAS) blockade and may predispose to hyperkalemia [5].

This case series has some limitations. Firstly, the cause of hyperkalemia and AKI was not thoroughly investigated, particularly whether the AKI was pre-renal, intrinsic, or post-renal, as urine studies were not conducted. Secondly, CK levels were not obtained to rule out rhabdomyolysis as a potential cause.

Author Contributions

Amro Khalili: conceptualization, data curation, investigation, methodology, writing – original draft, writing – review and editing. **Christian K. Raphael:** conceptualization, data curation, investigation, methodology, writing – original draft, writing – review and editing. **Rasha Shreim:** methodology, writing – original draft, writing – review and editing. **Nancy Abou Nafeh:** conceptualization, methodology, project administration, writing – original draft, writing – review and editing. **Cynthia Karam:** conceptualization, project administration, writing – review and editing.

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The authors have nothing to report.

Consent

Both patients provided written informed consent.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

All data generated or analyzed during this study are included in this published article [and its Supporting Information file].

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.