Case Report – Παρουσίαση Περιστατικού

Successful Anesthesia Management in a Patient with Refractory Hypokalemia and Hypertension Due to Conn Syndrome Undergoing Laparoscopic Adrenalectomy: A Case Report

Gülin Köseoğlu[1*], Denada Haka[1], Murathan Ekent[2], Nedim Çekmen[2]

[1]MD, Anesthesiology

[2]MD, PhD, Anesthesiology

[1]Department of Anesthesiology and Intensive Care Unit, Baskent University, Faculty of Medicine Ankara, Turkey

**Correspondence: Baskent University, Faculty of Medicine, Department of Anesthesiology and Intensive Care Unit, Ankara, Turkey, Email: glnksgl@gmail.com, GSM:+905304573934, Tel: 0312203 68 68-4867*

ABSTRACT

Successful Anesthesia Management in a Patient with Refractory Hypokalemia and Hypertension Due to Conn Syndrome Undergoing Laparoscopic Adrenalectomy: A Case Report

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Conn's syndrome is characterized by excessive aldosterone secretion, which can also occur due to an adrenal adenoma or adrenal hyperplasia. Conn's syndrome is a prevalent cause of secondary hypertension characterized by excessive aldosterone secretion, resulting in Na^+ retention, hypokalemia, and resistant hypertension. We aimed to present the successful anesthesia management of a 40-year-old female patient with severe hypokalemia, hypertension, and muscle cramps who underwent laparoscopic adrenalectomy due to Conn's syndrome, due to a left adrenal adenoma. We emphasize the importance of comprehensive preoperative evaluation, a multidisciplinary approach, personalized patient management, and ensuring electrolyte imbalance and hemodynamic stability in the perioperative period.

Keywords: Conn's syndrome, resistant hypokalemia, anesthesia, perioperative management

INTRODUCTION

Primary hyperaldosteronism, also known as Conn syndrome, is characterized by autonomous aldosterone secretion from the adrenal cortex and is usually due to an adrenal

adenoma or bilateral adrenal hyperplasia, resulting in suppression of plasma renin activity[1-3]. While Conn syndrome is seen in 5-10% of all hypertensive patients, this rate is around 20-25% in those with resistant hypertension[3]. Increased aldosterone secretion results in Na^+ and water retention by the kidneys and increased excretion of potassium (K^+) and H^+ ions. These pathophysiological mechanisms lead to hypertension, hypervolemia, hypokalemia, and metabolic alkalosis, especially manifesting clinically as hypertension and hypokalemia. Suppressed plasma renin activity is a hallmark diagnostic feature[4,5]. Surgical intervention is generally indicated in cases of an aldosterone-producing adenoma[4]. Anesthetic management in Conn's syndrome presents considerable challenges due to persistent hypertension, the risk of arrhythmias induced by hypokalemia, and altered responses to neuromuscular blocking agents[6]. The mechanism and treatment of Conn syndrome are outlined in Table 1.

Aldosterone is **released** in response to RAAS activation, hyperkalemia hyponatremia

↓

Kidney conserves H_2O and Na^+ and excretes K^+ and H^+

HYPERALDOSTERONISM

Excessive H_2O and Na^+ retention

Hypokalemia (K^+ excretion)

Metabolic alkalosis (H^+ excretion)

↓

Often **caused by** aldosteronoma, pheocromocytoma, overactive adrenal gland (Long term licorice ingestion resembles hyperaldosteronism)

↓

Treatment

Fix primary problem (removal of aldosterone secreting tumor)

Aldosterone antagonists (i.e., spironolactone)

Correct electrolytes: K+ supplements and fluid/Na+ restriction

Table 1. The Mechanism and Treatment of Conn Syndrome

Herein, in our case, we present the successful anesthetic management of a 40-year-old woman who underwent laparoscopic adrenalectomy due to Conn syndrome secondary to a left adrenal adenoma and presented with severe hypokalemia, hypertension, and muscle cramps.

CASE PRESENTATION

A 40-year-old female (height 1.55 m, body weight 105 kg, and BMI 43.75 kg/m²) presented with hypertension, hypokalemia, fatigue, muscle cramps, and generalizedweakness. A computed tomography (CT) scan revealed a left adrenal adenoma (Figure 1), and she was planned for a left-sided laparoscopic adrenalectomy.

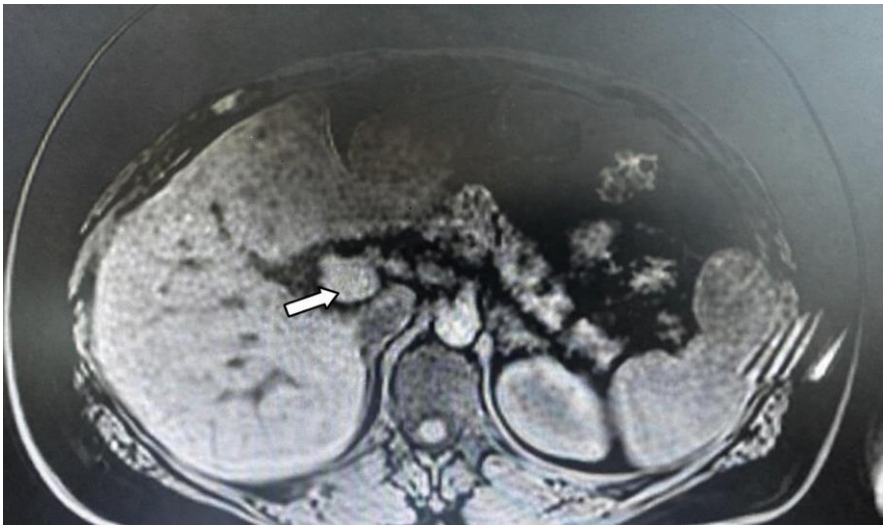

Figure 1. Patient's computed tomography (CT) scan demonstrating a left adrenal adenoma (arrow).

On physical examination, her blood pressure (BP) was 180/100 mmHg. Laboratory tests are detailed in Table 2. The electrocardiogram (ECG) showed a heart rate (HR) of 54 beats per minute. A transthoracic echocardiography showed normal left ventricular wall thickness and preserved diastolic function with an ejection fraction of 53%.

Laboratory Values	Value	Reference Range
Hemoglobin (g/dL)	11.1	12–16 (female)
Hematocrit (%)	34.2	36–46 (female)
Glucose (mg/dL)	207	70–110 (fasting)
Potassium (K^+)	2.6	3.5-4.5
Sodium (Na^+)(mEq/L)	143 mEq/L	135-145
Creatinine (mg/dL)	0.8 mg/dL	0.5-1
Blood urea nitrogen (mg/dL)	13 mg/dL	6-20
Plasma renin activity (ng/mL/hr)	0.1	0.1-6.56
Aldosterone (ng/mL)	472	35-300

Table 2. Laboratory Values

Abdominal ultrasonography identified hepatic steatosis, and both kidneys exhibited normal size, parenchymal thickness, and echogenicity. The right adrenal gland appeared normal, whereas a 21×15 mm lesion was noted in the left adrenal gland (Figure 1). The patient was on antihypertensive therapy including nifedipine 30 mg twice daily, doxazosin 4 mg once daily, and nebivolol 5 mg once daily. Potassium (K^+) supplementation was continued with tablets containing 2 grams of potassium bicarbonate and 2.17 grams of potassium citrate, administered twice daily. She had discontinued spironolactone treatment eight months prior. Venous aldosterone sampling was unsuccessful due to technical difficulties during catheterization. The 1 mg dexamethasone suppression test showed adequate cortisol suppression. Twenty-four-hour urine metanephrine and normetanephrine levels were normal. Aldosterone suppression was not observed after saline infusion testing. Based on these findings, a diagnosis of primary hyperaldosteronism was made, and surgical treatment was planned.

The patient exhibited non-adherence to oral K^+ supplementation. Therefore, during preoperative optimization two days prior to the scheduled surgical procedure, initiation of intravenous (IV) K^+ replacement was necessary. Initial laboratory evaluation revealed a serum K^+ level of 2.6 mEq/L. To correct the hypokalemia, three ampoules of potassium chloride, each containing 0.75 g (≈10 mEq), totaling 30 mEq, were administered at an infusion rate of 10 mEq/hour, after which a control measurement was obtained. The serum K^+ level increased to 2.9 mEq/L following replacement. The following day, a similar total dose (30 mEq) of IV replacement was planned; however, pre-infusion assessment demonstrated a K^+ level of 2.7 mEq/L. After administration using the same protocol, the K^+ level rose only to 3.0 mEq/L. To correct refractory hypokalemia by addressing the underlying hypomagnesemia, the patient received magnesium sulfate replacement at 12 mEq, administered twice daily on the first and second days, for a total of 4 doses. The trend of the measured K^+ levels is presented in Table 3. Failure to achieve target levels despite consecutive and adequate IV K^+ replacement suggested refractory hypokalemia. Following these interventions, the patient's electrocardiogram showed no repolarization abnormalities or arrhythmic events attributable to hypokalemia. With hemodynamic parameters remaining stable and the limited response to electrolyte optimization, the decision was made to proceed with the surgical intervention.

The patient's American Society of Anesthesiologists physical classification is III, and the patient has a Mallampati score of II. The verbal and written consent was obtained from the patient for the publication of this case report.

Time Point	Serum K^+ Levels (mmol/L)
Preoperative Evaluation	2.6
2 days preoperatively (before replacement)	2.6
2 days preoperatively (after replacement)	2.9
1 day preoperatively (before replacement)	2.7
1 day preoperatively (after replacement)	3.0
At surgery onset	2.7
At surgery completion	3.1

Table 3. Serum K^+ Levels (mmol/L) during the Perioperative Period

In the operating room, the patient was connected to standard monitoring, including ECG, noninvasive blood pressure (NIBP), pulse oximetry (SpO_2), end-tidal carbon dioxide ($EtCO_2$), and a train-of-four (TOF) monitor for continuous neuromuscular monitoring. A central venous catheter was inserted, and a 16-gauge IV cannula was placed for volume replacement, and a left radial arterial catheter was inserted for invasive blood pressure monitoring. At the beginning of the procedure, the patient's HR was 75 bpm, NIBP was 175/100 mmHg, and SpO_2 was 98%. The patient was premedicated with 0.01 mg/kg midazolam IV. Anesthesia induction was carried out with propofol, fentanyl, and rocuronium bromide as a neuromuscular blocker. The patient was intubated with a 7.5 mm endotracheal tube without any complications. Anesthesia was maintained throughout the procedure using a 50% oxygen–50% air mixture, 2.2% sevoflurane, and a remifentanil infusion at 0.05–0.1 mcg/kg/min. 100 mg hydrocortisone was administered IV for its anti-inflammatory effects, to support cardiovascular stability, and to reduce the risk of adrenal insufficiency. The patient was in volume-controlled mode, and positive end-expiratory pressure (PEEP) was set at 4-6 cmH_2O. The patient's PEEP was maintained at a relatively high level to prevent complications related to pneumoperitoneum, which can occur during laparoscopic abdominal surgeries. During surgery, the patient's BP ranged between 130–180 mmHg systolic and 60–95 mmHg diastolic. Intravenous fluid therapy consisted of Plasmalyte and colloid solutions for preventing hypovolemia. The patient's blood gas results were analyzed and are presented in Table 4.

The K^+ levels fluctuated between 2.7 and 3.1 mEq/L. The K^+ replacement was administered at 10 mEq/hour, totaling 20 mEq during the two-hour laparoscopic left adrenalectomy. Due to refractory hypokalemia, the patient was administered 12 mEq of magnesium sulfate twice.

However, there was no response to K^+ replacement, and serum K^+ levels could not be corrected to the desired range. Total surgical duration was 120 minutes. The procedure resulted in approximately 350 mL of blood loss, with no requirement for blood products. After postoperative hemostasis control, the surgery was completed without incident. The surgical specimen (Figure 2) was sent for pathologic examination**.**

Laboratory Values	Result	Result	Reference Range
pH	7.467	7.44	7.35–7.45
$PaCO_2$ (mmHg)	37	41	35–45
PaO_2 (mmHg)	81.5	84.3	75–100
SaO_2 (%)	96.4	97.7	94–98
Na^+ (mmol/L)	134	138	135–145
K^+ (mmol/L)	2.7	3.1	3.5–5.0
Ca^{2+} (mmol/L)	0.98	1.05	1.12–1.32
Lactate (mmol/L)	0.90	0.95	0.5–2.2
Glucose (mg/dL)	207	190	70–110 (fasting)
Hemoglobin (g/dL)	11.1	11.0	12–16 (female)
Hematocrit (%)	34.2	34	36–46 (female)
HCO_3^- (mmol/L)	26.4	26	22–26
Base Excess (BE) (mmol/L)	3.1	3.0	–2 to +2

Table 4. Periopetaive values of Arterial Blood Gas and Biochemical Parameters.

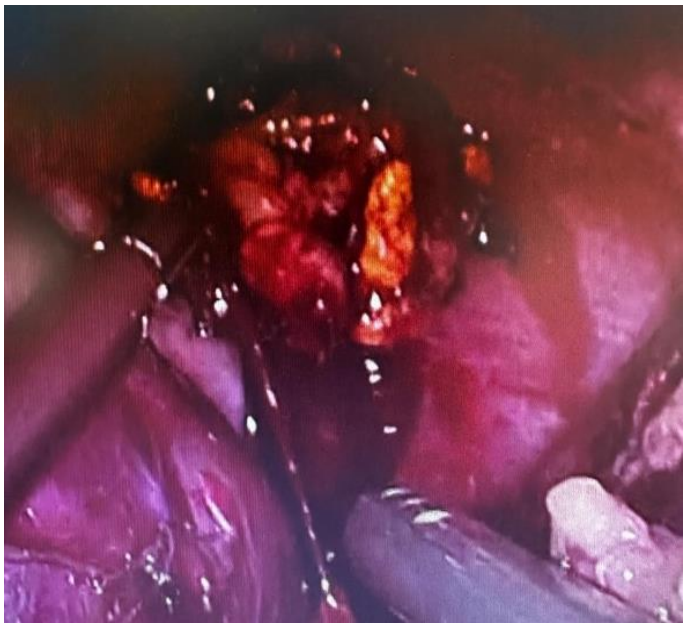

Figure 2. Laparoscopic view of left adrenal mass.

In the postoperative period, 1000 mg paracetamol was administered as an analgesic, and 3 mg granisetron was administered for nausea and vomiting. After 200 mg of sugammadex was administered for neuromuscular antagonism, the patient was extubated and transferred to the intensive care unit for close observation. On follow-up, the patient's potassium levels and clinical condition remained stable, and she was discharged 4 days after surgery.

DISCUSSION

In our case, we highlight the complex challenges anesthesiologists face in managing Conn's syndrome patients. In Conn's syndrome, excessive aldosterone secretion increases Na^+ reabsorption in the renal tubules, leading to water reabsorption, increased total blood volume, and refractory hypertension[1-3]. As Na^+ reabsorption increases, K^+ and H^+ are released, resulting in refractory hypokalemia (serum $K^+ < 3.5$ mEq/L) and alkalosis despite oral K^+ supplements[3-6]. Our patient had complaints of hypertension, hypokalemia (2.6 mEq/L), muscle cramps, fatigue, and generalized weakness.

Anesthetic management in patients with Conn's syndrome undergoing adrenalectomy requires careful preoperative optimization of persistent hypokalemia, labile BP, and metabolic alkalosis to reduce the risk of perioperative cardiovascular complications[5,6]. Hypokalemia prolongs the effects of non-depolarizing neuromuscular blocking agents[6], and therefore, we used TOF. Hypokalemia can lead to arrhythmias because it depresses baroreceptor tone, and therefore, hypokalemia should be treated aggressively in the perioperative period[6]. Hypertension treatment requires a combination of several drugs to regulate BP, depending on comorbidities and complications. The first-line therapeutic agent is spironolactone, a K^+-sparing diuretic administered (100–400 mg/day). Alternatively, amiloride or nifedipine (30–90 mg/day), 5–15 mg/day, and angiotensin-converting enzyme (ACE) inhibitors (captopril) and angiotensin receptor inhibitors (losartan-Na^+) can also be used[2,4,6]. In our case, maintaining normokalemia despite aggressive K^+ replacement and maintaining BP control despite multiple antihypertensive agents (Esmolol and nitrate) proved challenging.

Hypovolemia, which develops as a result of hypokalemia suppressing baroreceptor tone and a combination of other factors such as antihypertensives (ACE inhibitors) and diuretics, anesthetic drugs, positive pressure ventilation, and laparoscopic insufflation, should be treated aggressively[4,6]. In our patient, the antihypertensive drugs were stopped on the morning of surgery, and the patient's hemodynamics were monitored with invasive monitoring and stabilized by maintaining adequate depth of anesthesia with anesthetic

agents such as sevoflurane and remifentanil, volume replacement with IV crystalloids and colloidal fluids during the operation. During surgery, hemodynamic fluctuations can occur frequently due to sudden changes in circulating aldosterone and catecholamine levels, which are often triggered by surgical manipulations. Agents such as esmolol, phentolamine, nitroglycerin, and norepinephrine were available throughout the surgery to manage sudden hemodynamic fluctuations. In our case, minimal hemodynamic fluctuations were noted during surgical manipulation.

In laparoscopic surgeries, venous return and lung compliance are reduced by raised intra-abdominal pressure (IAP) and hypercarbia induced by pneumoperitoneum, leading to significant changes in ventilation, oxygenation, and hemodynamics[7]. Therefore, we tried to keep our patient's IAP at 14 mmHg, the patient's position, and avoid hypercarbia.

Ravi R et al.[8] reported that a 36-year-old patient with hypokalemia, hypertension, and muscle cramps who underwent right-sided laparoscopic adrenalectomy developed severe hypotension following pneumoperitoneum, requiring fluid replacement and vasopressors. Kharat PA et al.[9] reported that the perioperative course of a 37-year-old patient with right adrenal adenoma, quadriparesis, malignant hypertension, hypokalemia, metabolic alkalosis, rhabdomyolysis, and high aldosterone levels who underwent laparoscopic adrenalectomy under general anesthesia was uneventful. Latha YS et al.[10] reported that no problems other than a hypotension episode were encountered during laparoscopic adrenalectomy surgery in a 51-year-old patient with Conn syndrome who had hypertension, generalized muscle weakness, and hypokalemia. Our patient's hemodynamics remained stable, but the most important intraoperative problem encountered in our patient was persistent hypokalemia resistant to treatment.

CONCLUSION

Patients with Conn's syndrome present a significant challenge for anesth-esiologists due to persistent hypertension and hypokalemia. Careful and vigilant periope-rative hemodynamic monitoring and correction of electrolyte and metabolic abnormalities are crucial to their successful management. A multidisciplinary team approach, including anesthesiologists, endocrinologists, and surgeons, is essential for meticulous perioperative monitoring.

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