SEVERE HYPERKALEMIA FOLLOWING ADRENALECTOMY FOR ALDOSTERONE-PRODUCING ADENOMA: A CASE REPORT

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ABSTRACT

Severe hyperkalemia after adrenalectomy for aldosterone-producing adenoma is an uncommon but potentially life-threatening complication if not promptly recognized and managed. This condition may develop several weeks after surgery, particularly in elderly patients with long-standing hypertension and underlying chronic kidney disease (CKD), emphasizing the need for vigilant postoperative surveillance. We herein report a case of severe hyperkalemia after adrenalectomy for aldosterone-producing adenoma, with a focus on its pathophysiology, postoperative monitoring, and therapeutic management. This case highlights the critical importance of early and serial assessment of serum potassium levels and renal function in high-risk patients following adrenalectomy.

Keywords: hyperkalemia, aldosterone-producing adenoma, fludrocortisone

I. INTRODUCTION

Primary aldosteronism (PA) is the most common endocrine cause of secondary hypertension, accounting for approximately 5–14% of hypertensive patients in primary care settings and up to 30% in specialized referral centers [1]. The two most common etiologies are aldosterone-producing adenoma (APA) and idiopathic adrenal hyperplasia, which together account for more than 95% of cases.

For patients with unilateral aldosterone-producing adenoma, adrenalectomy is considered the treatment of choice and is recommended by international endocrine guidelines [1]. Surgical removal eliminates the source of excessive aldosterone production, improves blood pressure control, normalizes potassium levels, and reduces the risk of cardiovascular and renal complications.

Although most patients experience significant clinical improvement following surgery, delayed postoperative hyperkalemia may occur in a small subset of patients. This complication is relatively rare and has mainly been described in case reports and small case series. In severe cases, hyperkalemia may become life-threatening if not promptly detected and treated. [2],[3],[4]

Despite increasing recognition of this complication, postoperative monitoring strategies for high-risk patients remain poorly standardized. Delayed hyperkalemia may be under-recognized because serum potassium levels are often normal immediately after surgery, leading to delayed diagnosis and treatment. Elderly patients with chronic kidney disease and long-standing hypertension appear particularly vulnerable to this complication [2][3].

Here, we report a case of severe hyperkalemia occurring one month after unilateral adrenalectomy in an elderly patient with chronic kidney disease, highlighting the underlying pathophysiology and therapeutic considerations

II. CASE PRESENTATION

A 75-year-old woman was admitted to the emergency department with complaints of fatigue and nausea. Laboratory evaluation revealed serum potassium of 6.3 mmol/L, creatinine 207 µmol/L, and urea 26.6 mmol/L. Electrocardiography demonstrated peaked T waves. The patient had undergone left adrenalectomy for an aldosterone-producing adenoma one month prior to admission and was subsequently referred to the Endocrinology Department for further management.

Her medical history included 20 years of hypertension, treated with amlodipine 5 mg daily, with poor blood pressure control (170/100 mmHg at clinic visits). She had not been receiving diuretics or renin–angiotensin system inhibitors. Six months prior, she had been diagnosed with stage III chronic kidney disease, with a baseline creatinine level of 130 µmol/L (**estimated glomerular filtration rate [eGFR] approximately 38 mL/min/1.73 m²**).

An adrenal lesion had been incidentally detected on thoracic computed tomography (CT) performed during evaluation for chronic cough. Subsequent abdominal CT imaging revealed a 14 × 12 mm left adrenal mass with an absolute washout of 73%,

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suggestive of adrenal adenoma. Twenty-four-hour urinary metanephrine and normetanephrine levels were within normal limits. Plasma aldosterone concentration was 30.5 ng/dL, and renin level was 1.47 µIU/mL, consistent with primary aldosteronism due to unilateral aldosterone-producing adenoma. However, prior to surgery, adrenal venous sampling (AVS) was not performed.

Other laboratory findings included creatinine 136 µmol/L (eGFR 36 mL/min/1.73 m²) without proteinuria, and a normal complete blood count. Electrocardiography showed no ST-T abnormalities or evidence of left ventricular hypertrophy. Echocardiography revealed normal cardiac function. Renal ultrasound demonstrated increased echogenicity of the renal parenchyma with reduced corticomedullary differentiation.

The patient underwent retroperitoneoscopic left adrenalectomy in April 2025. Postoperatively, blood pressure improved significantly (110/60–130/80 mmHg), and antihypertensive and mineralocorticoid antagonist medications were discontinued. Serum potassium remained within the normal range (4.1–4.3 mmol/L), and postoperative creatinine was 119 µmol/L (eGFR approximately 43 mL/min/1.73 m²). The patient was discharged without additional medications.

Figure 1. ***Abdominal computed tomography demonstrating a left adrenal adenoma measuring 14 × 12 mm.***

One month later, she presented with severe hyperkalemia. At admission, vital signs were stable, with blood pressure 140/70 mmHg and pulse 74 beats per minute. Physical examination revealed no signs of dehydration or fluid overload.

Initial treatment included intravenous insulin with glucose infusion, furosemide, polystyrene sulfonate, isotonic saline infusion, and sodium bicarbonate, with continuous electrocardiographic monitoring. After 48–72 hours of treatment, serum potassium normalized to 4.4 mmol/L, and creatinine decreased to 171 µmol/L.

However, hyperkalemia recurred two days later despite further improvement in renal function (creatinine 144 µmol/L, eGFR approximately 35 mL/min/1.73 m²). Subsequent laboratory testing performed four weeks after surgery showed supine aldosterone 3.07 ng/dL and renin 4.91 µIU/mL, suggesting relative hypoaldosteronism due to delayed recovery of the remaining adrenal gland. The combination of low aldosterone levels with non-suppressed renin supported impaired mineralocorticoid secretion. Persistent hyperkalemia despite improvement in renal function was also consistent with hypoaldosteronism.

Other potential causes of hyperkalemia were excluded. There was no history of potassium supplementation, renin–angiotensin system inhibitors, potassium-sparing diuretics, rhabdomyolysis, trauma, hemolysis, or hyperosmolar state and no significant tissue injury was identified.

Treatment with fludrocortisone 0.05 mg/day was initiated but failed to adequately control serum potassium levels. Increasing the dose to 0.1 mg/day resulted in stable potassium levels between 4.2–4.4 mmol/L, with stable renal function. Serum potassium normalized within approximately five days after dose escalation, without worsening hypertension, edema, or signs of fluid overload.

At one-month follow-up, serum potassium remained within the normal range (3.9–4.0 mmol/L), and creatinine was 165 µmol/L. After another month, repeat testing demonstrated recovery of adrenal function, with aldosterone 9.59 ng/dL and renin 13.68 µIU/mL. Fludrocortisone therapy was discontinued, and subsequent monitoring confirmed persistent normokalemia.

III. DISCUSSION

Primary aldosteronism (PA), also known as Conn's syndrome, is characterized by autonomous overproduction of aldosterone from the adrenal glands independent of the renin–angiotensin system. Excess aldosterone promotes sodium and water retention while increasing potassium excretion, leading to hypertension, hypokalemia,

and long-term cardiovascular, renal, and metabolic complications [1],[5].

Compared with patients with essential hypertension with similar blood pressure levels, individuals with PA have a significantly higher risk of cardiovascular events, including left ventricular hypertrophy, atrial fibrillation, stroke, myocardial infarction, heart failure, and chronic kidney disease. This increased risk is not solely attributable to elevated blood pressure but also to the direct deleterious effects of aldosterone on cardiovascular and renal tissues. Experimental and clinical studies have demonstrated that prolonged exposure to excess aldosterone promotes myocardial remodeling, vascular injury, and renal fibrosis through activation of mineralocorticoid receptors located in cardiomyocytes, vascular smooth muscle cells, renal tissue, and adipocytes. [1],[6]. Therefore, early detection and appropriate treatment of PA are essential for improving long-term clinical outcomes.

Aldosterone-producing adenoma (APA) is one of the two most common causes of PA, accounting for approximately 35% of cases [1]. These tumors are typically small, benign, and usually measure less than 2 cm in diameter. For patients with unilateral APA, adrenalectomy is considered the treatment of choice and is recommended by major international societies, including the Endocrine Society, the American Heart Association, and the European Society of Endocrinology. Several studies comparing surgical treatment with medical therapy using mineralocorticoid receptor antagonists have shown that adrenalectomy provides superior outcomes, including better blood pressure control and normalization of potassium levels. Furthermore, surgical treatment has been associated with a lower risk of all-cause mortality and major cardiovascular events compared with medical therapy alone.

Although most patients experience significant improvement in blood pressure and electrolyte balance after adrenalectomy, postoperative hyperkalemia may occur in a minority of cases. This complication is more likely in elderly patients and in those with pre-existing chronic kidney disease, as observed in the present case [2],[3],[4],[7].

Renal involvement in PA has distinct characteristics. In the early stages of the disease, excess aldosterone leads to increased sodium reabsorption, expanded extracellular volume, and suppression of tubuloglomerular feedback, resulting in glomerular hyperfiltration. Consequently, the glomerular filtration rate (GFR) may appear artificially elevated, and serum creatinine levels may remain deceptively low, thereby masking underlying renal damage. Over time, chronic aldosterone exposure can induce interstitial inflammation, fibrosis, glomerular injury, and microvascular damage, eventually leading to albuminuria and progressive kidney disease.

Following adrenalectomy, plasma aldosterone levels decline rapidly, and the previously masked renal dysfunction may become apparent as the GFR returns to its true baseline level. As a result, the underlying chronic kidney disease may be unmasked, increasing the risk of postoperative hyperkalemia [2],[4].

In the present case, the delayed onset of hyperkalemia approximately one month after surgery is clinically important because serum potassium levels remained normal immediately after adrenalectomy. This finding highlights the need for repeated postoperative biochemical monitoring, especially in elderly patients with chronic kidney disease.

Another important mechanism contributing to hyperkalemia after adrenalectomy is the prolonged suppression of the renin–angiotensin–aldosterone (RAA) axis before surgery. In patients with aldosterone-producing adenoma, chronic excessive aldosterone secretion suppresses renin release and leads to functional suppression and atrophy of the zona glomerulosa in the contralateral adrenal gland. After removal of the adenoma, the remaining adrenal gland may not immediately recover its ability to produce aldosterone. This transient or persistent hypoaldosteronism results in impaired potassium excretion in the distal nephron, particularly in the distal convoluted tubule and collecting duct, especially when renal function is already compromised [2],[3],[4].

Postoperative hyperkalemia is usually transient but may persist for weeks or even months, particularly in patients with advanced age, long-

standing hypertension, large tumors, or pre-existing renal impairment [8].

In the present case, other causes of hyperkalemia—including metabolic acidosis, rhabdomyolysis, trauma, and hyperosmolar states—were excluded. Although renal function initially deteriorated, it subsequently improved after appropriate medical management, while hyperkalemia persisted. Therefore, renal dysfunction alone was unlikely to fully explain the elevated potassium levels. Laboratory evaluation revealed abnormally low plasma aldosterone levels (3.07 ng/dL) with unsuppressed renin levels (4.91 µIU/mL), supporting the diagnosis of relative hypoaldosteronism due to delayed recovery of the remaining adrenal gland.

Our patient also presented with several risk factors for postoperative hyperkalemia that have been described in previous studies, including advanced age, chronic kidney disease, long-standing hypertension (20 years), and elevated preoperative aldosterone levels. This case highlights the importance of careful postoperative monitoring, particularly in high-risk patients. Early follow-up within the first weeks after surgery is essential for timely detection and management of potential complications.

Fischer et al. reported that suppression of the contralateral zona glomerulosa may persist for several weeks to months after adrenalectomy, resulting in prolonged hyperkalemia. In most cases, adrenal function gradually recovers through intrinsic regulatory mechanisms [2]. However, temporary mineralocorticoid replacement therapy should be considered when hyperkalemia persists or recurs, particularly when biochemical evidence of hypoaldosteronism is present.

Fludrocortisone is the preferred agent in this situation. It is a synthetic corticosteroid with potent mineralocorticoid activity that increases sodium reabsorption and potassium excretion in the distal nephron. Currently, no standardized treatment protocol exists, but the commonly recommended starting dose ranges from 0.05 to 0.1 mg per day [2]. Careful monitoring of serum potassium, blood pressure, and fluid status is required because excessive mineralocorticoid activity may lead to sodium retention, fluid overload, and hypertension.

In our patient, the initial dose of 0.05 mg/day did not adequately control serum potassium levels. After increasing the dose to 0.1 mg/day, potassium levels stabilized within the normal range without evidence of fluid overload or hypertension. After approximately two months of therapy, repeat laboratory tests showed recovery of aldosterone secretion and renin activity, indicating restoration of adrenal function. Fludrocortisone was subsequently discontinued, and serum potassium remained stable thereafter.

This report has several limitations. First, urinary potassium indices and transtubular potassium gradient were not measured. Second, adrenal venous sampling was not performed before surgery.

IV. CONCLUSION

Severe hyperkalemia is an uncommon but clinically significant complication following adrenalectomy for aldosterone-producing adenoma. Delayed onset several weeks after surgery may occur despite initially normal postoperative potassium levels. It is more likely to occur in elderly patients, those with chronic kidney disease, long-standing hypertension, or high preoperative aldosterone levels. Close postoperative monitoring of serum electrolytes and renal function is essential for early detection and management. Repeated monitoring during the first postoperative weeks should be considered in high-risk patients. Temporary mineralocorticoid replacement therapy with fludrocortisone may be required until adrenal function of the contralateral gland recovers.

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