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CASE REPORT

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Respiratory failure and rhabdomyolysis caused by severe hypokalemia in a young female with hypertension: a rare critical condition in primary aldosteronism

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Abstract

Background The two classic manifestations of primary aldosteronism are hypertension and hypokalemia. However, acute respiratory failure due to hypokalemia in primary hyperaldosteronism is rare.

Case presentation The patient was a 27-year-old female who presented with drowsiness and weakness in all extremities. She had been diagnosed with hypertension three years prior, with irregular follow-up, and had a history of preeclampsia one year later. She exhibited high blood pressure and severe hypokalemia (2 mEq/L), leading to respiratory depression and impending respiratory arrest. Consequently, the patient was intubated and transferred to the intensive care unit (ICU). She also developed rhabdomyolysis. Blood pressure tests, including hormonal tests (aldosterone: 13.2 ng/dL, plasma renin activity: 0.32 ng/mL/h), were conducted. Due to the high aldosterone-renin ratio, an abdominopelvic computed tomography (CT) scan was performed. The CT scan revealed a 14 × 12 mm round mass with a washout value above 60%, consistent with an adrenal adenoma, leading to a diagnosis of primary aldosteronism. The patient was discharged after stabilization, and one and a half months after ICU admission, a laparoscopic left adrenalectomy was successfully performed without post-operative complications. Histopathology showed encapsulated hypertrophy of the adrenal cortex with a predominance of large clear cells, confirming the diagnosis of adrenal adenoma. At the most recent follow-up, the patient had normal potassium levels, was normotensive without any medications, and exhibited no alarming signs or symptoms.

Conclusion Respiratory depression to the extent of impending respiratory failure and rhabdomyolysis as a result of hypokalemia in primary aldosteronism are extremely rare. In this patient, who developed respiratory depression due to resistant hypokalemia, timely investigation of secondary causes and diagnosis of adrenal adenoma were crucial. The surgery provided definitive treatment for the patient's blood pressure and prevented the recurrence of life-threatening complications.

Keywords Adrenal adenoma, Conn's syndrome, Hyperaldosteronism, Hypertension, Hypokalemia, Laparoscopy

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Background

Data from the United States and England show the prevalence of hypertension was about 29% in 2007 and 30% in 2006, respectively [1–3]. Approximately 35% of hypertensive patients in general, and higher rates in patients with drug-resistant hypertension, have secondary hypertension [4]. General characteristics suggestive of secondary hypertension include early onset of hypertension, resistant or severe hypertension, hypertensive emergency, sudden increase in blood pressure, target organ damage, and non-dipping or reverse dipping during 24-hour ambulatory blood pressure monitoring [5]. These patients should be screened to exclude the most common causes of secondary hypertension, including obstructive sleep apnea, renal parenchymal and vascular diseases, primary aldosteronism, thyroid diseases, Cushing's syndrome, pheochromocytoma, and coarctation of the aorta [5].

Concomitant hypokalemia and hypertension narrow the differential diagnoses. The most probable causes of hypertension and hypokalemia include primary aldosteronism, Cushing's syndrome, renovascular diseases, and other causes of secondary hyperaldosteronism and non-aldosterone mineralocorticoid excess. Hypokalemia in hypertensive patients must be evaluated for secondary hypertension due to hyperaldosteronism. Case detection is recommended in the following patients: sustained blood pressure above 150/100 on each of three measurements obtained on different days, hypertension (blood pressure > 140/90) resistant to three conventional antihypertensive drugs (including a diuretic), controlled blood pressure (blood pressure < 140/90) on four or more antihypertensive drugs, hypertension and spontaneous or diuretic-induced hypokalemia, hypertension and adrenal incidentaloma, hypertension and sleep apnea, hypertension and a family history of early onset hypertension or cerebrovascular accident at an age below 40 years, and all hypertensive first-degree relatives of patients with primary aldosteronism [6]. Acute respiratory failure due to hypokalemia is a rare and life-threatening but treatable presentation of hypokalemia [7], and acute respiratory failure due to hypokalemia in primary aldosteronism is extremely rare.

Primary aldosteronism may be caused by unilateral adrenal involvement, including aldosterone-producing adenoma, and bilateral disease [8]. While patients with unilateral adrenal hypersecretion may be treated with unilateral adrenalectomy, cases with bilateral involvement are treated by mineralocorticoid receptor antagonists [8]. In unilateral involvement, unilateral adrenalectomy significantly reduces aldosterone output and almost always corrects hypokalemia [8]. Laparoscopic adrenalectomy is preferred over open surgery since the former has fewer complications and shortens the length of hospital stay. Hence, it is the method of choice [9].

Herein, we present a 27-year-old female with hypertension who was intubated due to impending respiratory arrest as a consequence of unexplained resistant hypokalemia. Hormonal examinations and abdominal imaging were performed, and finally, with the diagnosis of adrenal adenoma, she underwent successful laparoscopic adrenalectomy.

Case presentation

Clinical presentation, past medical history, and admission to hometown hospital

The patient was a 27-year-old female with a known case of hypertension. She presented to the emergency department of her hometown hospital with a chief complaint of abdominal pain, vomiting, diarrhea, malaise, drowsiness, and weakness in all extremities. Her blood pressure was documented as 140/90 mmHg upon admission. She had undergone an endoscopy prior to admission to her hometown hospital because of her gastrointestinal symptoms, which showed erosive gastropathy.

In her past medical history, she had hypertension diagnosed three years ago, and a flare-up one year later, when she was pregnant, with a primary diagnosis of preeclampsia. The condition required observation and follow-up for diagnosis confirmation. The only medication she was receiving for hypertension was amlodipine 5 mg once a day. Overall, the patient had irregular follow-up, and it was unknown whether her blood pressure was controlled. There was no other significant past medical or surgical history except for cesarean section and dilation and curettage (D&C).

A spiral non-contrast brain computed tomography (CT) was performed because of her drowsiness. However, this imaging did not explain her condition. Biochemical laboratory tests upon admission showed severe hypokalemia (serum potassium: 2 mEq/L) and normal blood gas (pH: 7.37, pCO_2: 40.8, HCO_3: 23.2). A potassium chloride (KCl) regimen was initiated both intravenously and orally upon admission. Unexpectedly the potassium level did not rise after KCl administration and instead decreased to 1.7 mEq/L after two days. As a result, the condition worsened and the patient developed respiratory depression alongside impending respiratory arrest, which led to intubation and transfer of the patient to a tertiary care center for evaluation by a nephrologist and endocrinologist to rule out hyperaldosteronism. Respiratory depression arising from hypokalemia, especially with that intensity, is uncommon [7]. Hence, an accurate approach to the reason for the patient's condition was required. The patient was admitted to the tertiary care center to evaluate the probable causes of hypertension and hypokalemia, including primary aldosteronism,

Cushing's syndrome, renovascular diseases, and other causes of secondary hyperaldosteronism and non-aldosterone mineralocorticoid excess.

Tertiary care center

The patient was transferred to the intensive care unit (ICU). She was afebrile with a blood pressure of 140/90 mmHg. No other significant findings were found in the physical examination. Laboratory results upon her admission are shown in Table 1. Laboratory tests revealed hypokalemia, hypernatremia, and high creatine phosphokinase (CPK) and creatinine. Continuous cardiac monitoring with regular electrocardiography was established. Electrocardiography changes on admission indicative of hypokalemia included ST depression, T wave flattening and inversion, and U waves in precordial leads.

Hormonal evaluation to rule out other similar conditions, such as pheochromocytoma, was carried out (24-hour urine metanephrine: 20.9 µg/24 h, 24-hour urine normetanephrine: 50.8 µg/24 h). The urine screening test for pheochromocytoma was negative. Hence, we proceeded to the next step of the workup by measuring the level of serum aldosterone and renin (aldosterone: 13.20 ng/dL, plasma renin activity: 0.32 ng/mL/h, 24-hour urine free cortisol: 4.8 µg/24 h, 24-hour urine volume: 1050 ml/24 h).

Table 1 The patient's laboratory results on admission to the tertiary center

Laboratory test	Patient's value	Reference range
WBC (/µL)	14×10^3	$4\times10^3-10\times10^3$
Hemoglobin (g/dL)	14.3	12–16
Platelet (/µL)	193×10^3	$150\times10^3-450\times10^3$
PTT (s)	34.8	25–35
PT (s)	21.0	13.8
INR	1.6	0.9-1.0
BUN (mg/dL)	30	8–20
Creatinine (mg/dL)	2.55	0.6–1.2
Sodium (mEq/L)	165	136–145
Potassium (mEq/L)	2.0 (minimum in hospitalization: 1.5)	3.5–5.5
Magnesium (mg/dL)	4.3	1.9–2.5
Calcium (mg/dL)	8.5	8.6–10.3
Phosphorate (mg/dL)	1.0	2.5–4.2
Uric acid (mg/dL)	5.5	2.6–6.0
Total protein (g/dL)	5.6	6.0–7.8
Albumin (g/dL)	3.2	3.5–5.2
Total bilirubin (mg/dL)	0.6	0.1–1.2
Direct bilirubin (mg/dL)	0.3	Equal or less than 0.3
AST (IU/L)	64	< 40
ALT (IU/L)	58	< 34
AlP (IU/L)	202	80–306
CPK (IU/L)	2,440 (maximum in hospitalization: 46,380)	24–174
LDH (IU/L)	1,358	< 480

ALP alkaline phosphatase, ALT alanine aminotransferase, AST aspartate aminotransferase, BUN blood urea nitrogen, CPK creatine phosphokinase, INR international normalized ration, LDH lactate dehydrogenase, PT prothrombin time, PTT partial thromboplastin time, WBC white blood cell

Imaging studies during that hospital course included abdominopelvic ultrasonography and CT scan, color Doppler ultrasonography of both renal vessels, brain CT, and high-resolution CT of both lungs for better evaluation. Both kidneys were normal in size in the abdominopelvic ultrasonography but with mild bilateral stasis and increased cortical parenchymal echogenicity. Additionally, color Doppler ultrasonography of both renal vessels showed no stenosis or vascular abnormalities. Spiral abdominopelvic CT with and without contrast was highly suggestive of lipid-rich adrenal adenoma as there was evidence of a homogeneously 14×12 mm round mass structure in the left adrenal gland associated with acceptable delayed washout. The radiodensity of the mass was as follows: Pre-contrast 30 Hounsfield units (HU), post-contrast 100 HU, and delayed 45 HU. Therefore, the washout was above 60%, consistent with an adenoma (Fig. 1). A high-resolution CT of the lungs was taken because of her severe respiratory depression, which detected evidence of sub-segmental collapse consolidation in the posterior basal aspect of both lung fields and the right upper lobe. Brain CT was performed for the second time (the first time was upon hometown hospital admission), which did not reveal any pathologic findings. Based on the imaging studies and laboratory investigations, the clinical diagnosis of primary hyperaldosteronism resulting from adrenal adenoma was suggested.

Central access was provided, and initial management and supportive therapy were administered for hypokalemia, rhabdomyolysis, and acute kidney injury, allowing the patient to be extubated and discharged after nine days of admission with stable vital signs, a Glasgow Coma Score (GCS) of 15, and without respiratory depression. Discharge medications were prescribed as amlodipine 5 mg and spironolactone 25 mg, both twice a day, and the patient was referred to us for urological assessment.

Admission for operation

One and a half months after ICU admission, laparoscopic left adrenalectomy was successfully performed as follows. Under general anesthesia and in a lateral position, a 10 mm trocar was placed lateral to the umbilicus, and three working ports (5 mm trocar subcostal, 10 mm trocar left anterior axillary line, 5 mm trocar left midclavicular line) were inserted. Transperitoneal laparoscopic exploration was performed, the left colon was medialized, and the inferior, posterior, and medial surfaces of the adrenal gland were released. Multiple adrenal arteries

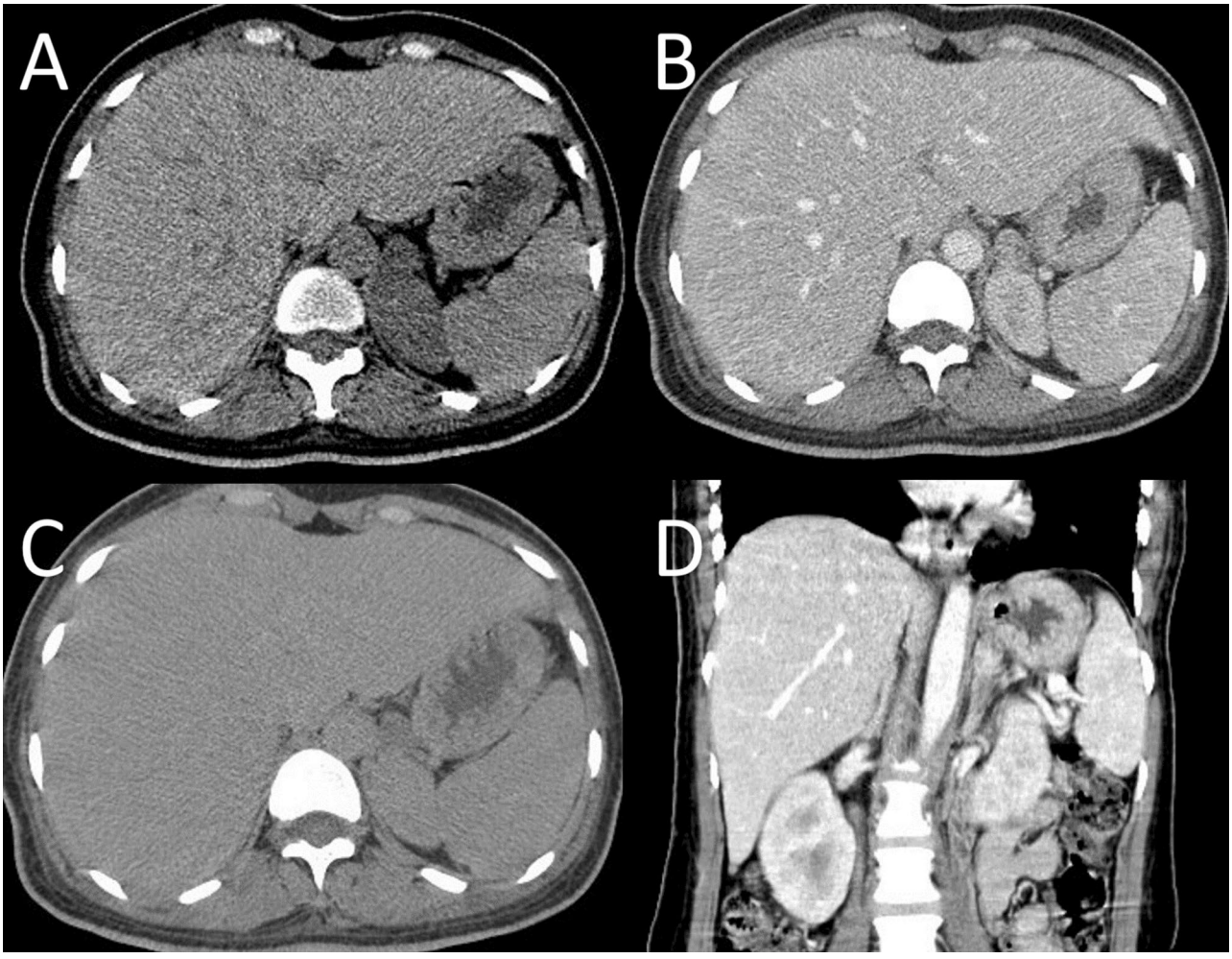

Fig. 1 There is a round soft tissue density structure in the left adrenal gland with enhancement in the portal phase and contrast washout in the delayed phase suggestive of adrenal adenoma. (**A**) Pre-contrast, (**B**) Post-contrast (portal phase), (**C**) Delayed post-contrast, (**D**) Post-contrast (portal phase)

and veins were dissected and ligated, and the mass was dissected from the spleen and upper pole of the kidney. After releasing the adrenal from the lateral abdominal wall, the specimen was removed via a Pfannenstiel abdominal incision. The incision and the trocar sites were then closed in layers. The specimen was submitted for surgical pathology analysis.

Pathology

Gross examination labeled the mass as a left adrenal mass, and the external surface was bosselated. The pathologic diagnosis was adrenocortical adenoma without necrosis and vascular invasion. The resected specimen was a variegated brown-mahogany color tissue measuring 5×2.5×1 cm and 12 g. Cut sections showed a hemorrhagic area. Four hematoxylin and eosin (H&E) slides were prepared. Light microscopic examination showed encapsulated hypertrophy of the adrenal cortex with a predominance of large clear cells (Fig. 2).

Post-operation and follow-up

Ultimately, the efficacy of surgery was assessed by serum potassium concentrations, and blood pressure monitoring continued for five days post-operatively, which showed correction of hypokalemia (latest result: 4.3 mEq/L) and stabilized blood pressure. No post-operative complications were present at the time. Also, post-operative abdominopelvic ultrasonography was normal. The most recent follow-up session (2.5 years after the operation) with the patient's endocrinologist was uneventful, and the clinical condition of the patient remained stable, as evidenced by a potassium level of 4.5 mEq/L, a normotensive state without any medications, and no alarming signs or symptoms.

Discussion

Primary hyperaldosteronism, or Conn's syndrome, is a condition characterized by non-suppressible hypersecretion of aldosterone, which most frequently results in hypertension and hypokalemia. Previously, it was

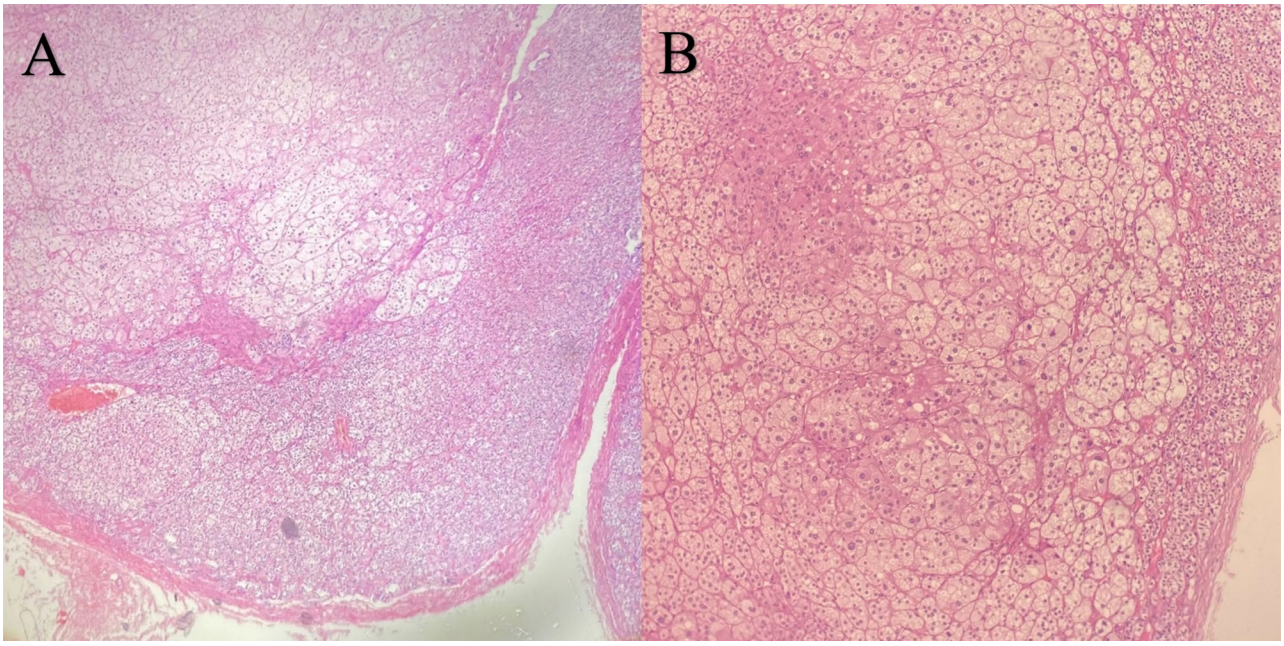

Fig. 2 Pathology images of the adrenocortical adenoma causing hyperaldosteronism. (**A**) Encapsulated adrenal gland cortical adenoma with a predominance of large clear cells (H&E staining, original magnification 40×), (**B**) Higher magnification of adrenal showing large clear cells arranged in sheaths and large nests (H&E staining, original magnification 100×)

estimated that approximately 1% of hypertensive patients were affected by primary hyperaldosteronism, but recent developments in screening methods, like the more extensive application of plasma aldosterone to renin ratio, have highlighted the actual prevalence to be up to 12% [10]. A low serum potassium concentration can lead to a variety of symptoms, including electrocardiogram abnormalities, fatigue, and muscle weakness, manifesting as constipation, exercise intolerance, and dyspnea. Additionally, acute respiratory failure is a rare but treatable presentation of this disorder [7]. Hypokalemia causing respiratory failure in primary aldosteronism is extremely rare. Herein, we present a 27-year-old woman with high blood pressure and severe hypokalemia, which caused respiratory failure. She finally underwent laparoscopic surgery after being diagnosed with adrenal adenoma as the cause of hypertension, and the patient recovered.

It is of great importance that primary hyperaldosteronism is considered a leading cause of secondary hypertension, with a prevalence of 10% among the general hypertensive population and 20% in patients with resistant hypertension [11]. Therefore, hypokalemia in hypertensive patients, especially young ones, should be assessed for secondary hypertension due to hyperaldosteronism. It is worth mentioning that only 9–37% of patients with primary aldosteronism have hypokalemia [6]. Thus, the recommendation for case detection has been determined beyond only hypokalemia. In a study by Ruhle et al. [12], only 2.7% of patients among 37,000 with hypertension and hypokalemia were screened for primary aldosteronism. This shows how much this condition may be underdiagnosed even in tertiary centers. Therefore, an optimal screening strategy is needed in this regard.

Severe hypokalemia in our patient led to respiratory failure, extremity weakness, and rhabdomyolysis. These presentations, especially respiratory arrest, as a consequence of hypokalemia are extremely rare in primary aldosteronism. There are, however, a few reports in the literature regarding hypokalemic-induced respiratory failure in other conditions, including renal tubular acidosis [13, 14]. Low potassium levels can interfere with the transmission of signals between nerves and muscles, hindering muscle contractions. In severe cases of hypokalemia, this can result in paralysis, especially when potassium levels are extremely low. This paralysis can impact the muscles involved in breathing, potentially causing respiratory failure [15]. Additionally, severe hypokalemia may cause rhabdomyolysis, leading to kidney injury, similar to our patient [15]. Hypokalemic-induced rhabdomyolysis is also rare, but has been previously reported in a few case reports of primary aldosteronism [16–18]. Although we expected ileus and constipation due to severe hypokalemia in our patient, she had diarrhea. We assume that her diarrhea was not related to her electrolyte imbalance and was associated with other causes leading to her gastrointestinal problems. However, it is worth mentioning that, on the other hand, diarrhea may have worsened the hypokalemia in our case.

Considering the severe signs and symptoms of our patient, including respiratory failure and extremity weakness, one would expect very high serum aldosterone values (at least more than 20 or 30 ng/dL). The lower-than-expected value of serum aldosterone in our case might be due to severe hypokalemia (serum potassium: 2.1 mEq/L) at the time of aldosterone and renin estimation.

The first step to identifying the subtype and ruling out adrenal carcinoma is an adrenal CT scan [6]. Although a CT scan is very useful for diagnosis and treatment decisions, it has some limitations. In a study by Young et al. [19], 21.7% of patients would have been wrongly excluded from being considered for adrenalectomy, and 24.7% of patients might have undergone unnecessary or inappropriate adrenalectomy. In a systematic review by Kempers et al. [20], CT/MRI misdiagnosed the cause of primary aldosteronism in 37.8% of patients compared to adrenal venous sampling as the standard test. Therefore, adrenal venous sampling has been suggested as an essential diagnostic tool for patients with a potential surgical therapeutic plan [6]. Notably, based on Dekkers et al. [21], there is no significant difference between the therapeutic outcomes of CT-based and adrenal venous sampling-based diagnoses. Still, this subject is controversial and needs further assessment. On the other hand, magnetic resonance imaging (MRI) has no advantage over CT in subtype evaluation [6]. Moreover, MRI is more expensive and has less spatial resolution than a CT scan [6]. Unfortunately, we did not have the resources to perform adrenal venous sampling in our patient, which was a limitation of our report.

The new WHO classification supports the terminology of the HISTALDO classification, which employs CYP11B2 immunohistochemistry to pinpoint functional sites of aldosterone production, aiding in predicting the risk of bilateral disease in primary aldosteronism. Aldosterone-producing adrenal cortical carcinoma (APACC) and adenoma (APA) are solitary lesions that are clearly visible with routine H&E and immunohistochemical staining for CYP11B2. Sub-centimeter solitary lesions visible with H&E and immunohistochemical staining are called aldosterone-producing nodules (APNs). Their counterparts, which may be difficult to distinguish with H&E but are always visible with immunohistochemical staining, are known as aldosterone-producing micronodules (APMs). When multifocal, they are termed "multiple APN" (MAPN) and "multiple APM" (MAPM), respectively. Lastly, aldosterone-producing diffuse hyperplasia is characterized by continuous CYP11B2 staining along the zona glomerulosa [22, 23].

A small lesion (<40 mm) with a regular shape and well-defined margins along with hypodense and homogenous content suggests an adrenal adenoma [24]. The first choice of treatment is surgical resection. Hence, laparoscopic adrenalectomy is a reasonable option for any individual with an aldosterone-producing adenoma, offering fewer complications and similar blood pressure control and hypokalemia correction compared to open adrenalectomy [8, 25]. Laparoscopic surgery with transperitoneal or retroperitoneal approaches can be utilized. A meta-analysis indicated that the body of evidence for the comparison between these two approaches is limited. In this meta-analysis, evidence of very low quality suggests that for relatively small lesions (under 6–7 cm), the retroperitoneal approach might reduce late morbidity. Additionally, some post-operative parameters, such as the time to oral fluid or food intake and the time to ambulation, might favor the laparoscopic retroperitoneal adrenalectomy technique [26]. However, this subject needs further investigation. Furthermore, comparing total and partial adrenalectomy requires future studies to determine whether partial adrenalectomy avoids adrenal insufficiency without increasing the risk of persistent or recurrent aldosteronism [8]. Regarding the clinical outcome, based on a retrospective study of 168 cases diagnosed with primary aldosteronism who underwent adrenalectomy, 77% of patients with a unilateral adenoma had their hypertension resolved or under control [27]. When unilateral disease is clearly confirmed, adrenalectomy corrects hypokalemia, if it was present before surgery, in nearly all patients [8]. Hypertension is cured in approximately 30–60% of cases, and in the remaining patients, a significant improvement in blood pressure is observed [8].

The Primary Aldosteronism Surgery Outcome (PASO) criteria have been introduced to measure the outcome after adrenalectomy for unilateral primary aldosteronism [28]. According to the PASO criteria, our patient achieved complete clinical success as she had normal blood pressure in the last follow-up without any medications. However, we cannot determine the biochemical success due to the lack of an aldosterone-renin ratio after the surgery because the patient was not willing to undergo post-operative hormonal tests. Nevertheless, the serum potassium level was normal in the follow-up. Our follow-up duration was also acceptable as, according to the PASO criteria, the final outcome should be assessed at 6–12 months with annual reassessments.

This report of our patient with primary aldosteronism has limitations. CYP11B2 immunostaining, a useful pathologic tool to diagnose aldosterone overproduction in primary aldosteronism and to determine its subtype, was not utilized in our patient. Additionally, urinary cortisol was requested for the patient instead of dexamethasone-suppressed cortisol, which may be superior for adrenal masses. According to the Endocrine Society guidelines, one or more confirmatory tests are suggested

to confirm the diagnosis of primary aldosteronism. The guidelines state that there may be no need for confirmatory tests in the setting of spontaneous hypokalemia, plasma renin below detection levels plus plasma aldosterone concentration > 20 ng/dL [6]. Another limitation of our report is not using confirmatory tests and adrenal venous sampling in our patient. Furthermore, in the follow-up, the patient had normal serum potassium values and controlled blood pressure without any medication, but post-operative aldosterone and renin levels were not measured.

Conclusion

The main objective of this paper is to highlight two important points:

(1) Respiratory depression to the extent of impending respiratory failure and rhabdomyolysis as a result of hypokalemia in primary aldosteronism are rarely observed. In our patient, a life-threatening condition was encountered due to her irregular follow-up; despite having high blood pressure at such a young age, the patient had irregular follow-up.
(2) An important issue in our case with aldosteronism and critical conditions was the short period (one and a half months) between discharge from ICU and the surgical procedure to prevent further critical conditions.

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Author contributions
A.H. designed the study. M.A. and H.K. collected the data. H.B. and M.N. drafted the manuscript. B.S.Y. and H.B. provided the pathology images. M.G.J. provided the radiology images. H.K., A.H., M.S., M.E., and K.K.A. revised and proofread the manuscript. All authors read and approved the final version of the manuscript.

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Data availability
All information of the patient is presented in the manuscript.

Declarations

Ethics approval and consent to participate
Written informed consent was obtained from the patient.

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Competing interests
The authors declare no competing interests.

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