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Primary aldosteronism with hypokalemic rhabdomyolysis: a case report and review of the literature

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Abstract

Background Hypokalemic rhabdomyolysis is a rare clinical manifestation of primary aldosteronism, making its diagnosis challenging, particularly when it becomes the primary presenting symptom. Herein, we present a case of primary aldosteronism with hypokalemic rhabdomyolysis and conduct a related literature review.

Case presentation We report the case of a 54-year-old Chinese male patient who presented with intermittent weakness over the past year and was admitted with sudden limb paralysis for 2 days. The final diagnosis was primary aldosteronism accompanied by hypokalemic rhabdomyolysis syndrome. By reviewing the related Chinese and English literature, we noticed that only a few cases were published since 1978. After excluding irrelevant literatures, we summarized and analyzed 43 patients of with primary aldosteronism accompanied by hypokalemic rhabdomyolysis syndrome. All patients showed good recovery, with normalized blood potassium levels, and a majority achieved normalized blood pressure. Some patients still required medication for blood pressure control.

Conclusions Primary aldosteronism rarely causes rhabdomyolysis; the occurrence of severe hypokalemia and rhabdomyolysis should prompt consideration of primary aldosteronism in the differential diagnosis. Early detection and treatment are crucial for determining patient prognosis.

Keywords Primary aldosteronism, Rhabdomyolysis, Hypokalemia, Literature analysis

Background

Primary aldosteronism (PA), commonly known as Conn's syndrome, is the most common cause of secondary hypertension. Approximately 5–10% of adults with hypertension are estimated to have primary aldosteronism [1]. Its main characteristic is excessive secretion of aldosterone, which leads to increased tubular reabsorption of sodium and enhanced excretion of potassium in the kidneys. Consequently, elevated blood pressure and hypokalemia ensue, elevating the risk of cardiovascular events and target organ damage. Although hypokalemia is a common manifestation of primary aldosteronism, hypokalemic rhabdomyolysis represents a rare presentation of this disease. Rhabdomyolysis (RM) is a syndrome characterized by the breakdown of skeletal muscle, leading to the release of electrolytes, myoglobin, creatine kinase, and other substances. This can result in electrolyte imbalances, metabolic acidosis, renal failure, and other complications [2]. Rhabdomyolysis can lead to hyperkalemia, but overall, potassium deficiency can occur. However, the mechanism of rhabdomyolysis induced by hypokalemia has not been determined.

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We report a case of primary aldosteronism combined with hypokalemic rhabdomyolysis and summarize the relevant literature to enhance clinicians' understanding of this condition. Early diagnosis and treatment are crucial for improving patient outcomes.

Case presentation

A 54-year-old Chinese male who was engaged in physical labor and admitted to the hospital due to intermittent fatigue for 1 year and sudden onset of paraplegia for 2 days. The patient experienced intermittent fatigue without apparent triggers for the past year, with improvement after rest. A total of 3 days prior, after exertion, he once again experienced fatigue accompanied by pain in the limbs and lower back. Family members provided massage to the lumbar and leg areas, resulting in bruising on the left lower limb. Symptoms improved with rest, and the patient had not previously sought medical attention. A total of 2 days prior, while working, he experienced heightened fatigue and developed paraplegia. He presented to our emergency department, where a head magnetic resonance imaging (MRI) scan showed no significant abnormalities, while a lumbar spine MRI revealed disc protrusion at L3–L4.

The electrolyte results revealed a blood potassium level of 1.83 mmol/L and a blood sodium level of 151.4 mmol/L, and liver function indicators revealed a total bilirubin level of 29.2 g/L and an aspartate aminotransferase (AST) level of 49.6 U/L. Potassium supplements were administered, and subsequent potassium monitoring revealed levels maintained between 2.0 and 2.3 mmol/L. Owing to the necessity for further treatment of hypokalemia, the patient was admitted to our department. Since the onset of symptoms, the patient's diet and sleep have been normal, with no significant changes in urine or stool. The patient had a history of hypertension for 3 years, with the highest recorded blood pressure being 160/100 mmHg. Currently, the patient is receiving a daily regimen of 5 mg amlodipine and 150 mg/12.5 mg valsartan/hydrochlorothiazide, maintaining blood pressure levels of approximately 120–130/80–85 mmHg. No family history of hypertension was reported, and the individual had no significant personal medical history. On admission, the patient's examination revealed the following: a temperature of 36.5 °C, heart rate of 100 beats of minute, respiratory rate of 19 breaths per minute, and blood pressure of 150/90 mmHg. The patient was alert with clear speech. Tenderness was noted in the soft tissues on the inner side of the left thigh, with a visible contusion measuring 20 cm × 15 cm. The thyroid was not palpably enlarged and no abnormalities were detected during the physical examination of the heart, lungs, or abdomen. Muscle strength and tone did not significantly differ among the four limbs. Neurological examination revealed no obvious abnormalities, and the lower extremities exhibited no edema. After admission, comprehensive laboratory investigations were conducted, including tests for adrenocorticotropic hormone (ACTH), cortisol (COR), thyroid function, renal function (repeatedly checked), coagulation profile, complete blood count, urinalysis, stool analysis, lipid profile, and blood glucose; all of these tests showed no abnormalities. Table 1 presents electrolyte levels, liver function, myocardial enzymes, urinalysis, and 24-hour urine potassium levels. The changes in blood potassium and creatine kinase levels at different timepoints after patient admission were presented (Fig. 1). The renin, angiotensin II, and aldosterone levels are detailed in Table 2. In this case, since the patient did not discontinue the valsartan/hydrochlorothiazide tablets for 2 weeks according to the standard posture test of the renin–angiotensin–aldosterone system, the positive results obtained supported the diagnosis. Blood gas analysis revealed a pH of 7.441, a HCO^{3-} concentration of 30.4 mmol/L, a standard base excess of 6.2 mmol/L, and a blood potassium concentration of 1.8 mmol/L. The electrocardiogram showed sinus rhythm with a prolonged QT interval.

Table 1 Dynamic changes in laboratory examination parameters at different timepoints during admission and after admission

Laboratory tests	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 8	Day 9	Day 10	Day 11	Day 12	References
K^+ (mmo/L)	1.83	2.0	1.9	1.97	2.79	3.5	2.89	2.99	3.26	3.44	3.19	3.5–5.5
CK (U/L)			3309	8008	15514	12879	3175		1066		254	50–310
CK-MB (U/L)			74.9	145	221.6	206	79.2		37.26		22	7–25
AST (U/L)	49.6		47.3	84.5	145.4	131.4	70.5		39.7		22.9	15–40
LDH (U/L)			475	639	932	652	362		322		255	120–250
Myo (ng/ml)						889.5	598.7		322.6		92.62	28–72
Urine occult blood					2 +							Negative
Urine potassium (mmol/24h)				33						108		25–125

CK, creatine kinase; CK-MB, creatine kinase isoenzyme; AST, sspartate transaminase; LDH, lactate dehydrogenase; Myo, myohaemoglobin

An adrenal computed tomography (CT) revealed a low-density mass in the medial limb of the left adrenal gland, with a CT value of approximately 0 Hounsfield unit (HU) and dimensions of approximately 22 mm ×16 mm (Fig. 2A). Enhanced CT demonstrated a circular lesion in the left adrenal gland, with a maximum cross-sectional area of approximately 22 mm ×16 mm, clear borders, and enhancement phases with CT values of approximately 51 HU, 64 HU, 45 HU, and 21 HU, respectively (Fig. 2B). Bilateral adrenal vein sampling yielded no successful results. Upon admission, the patient received oral and intravenous potassium supplementation, intravenous fluids, and symptomatic treatment for hypertension. After confirming the diagnosis of primary aldosteronism, the patient was prescribed spironolactone 20 mg twice a day in combination with sustained-release potassium chloride 2 g three times a day orally. Following the complete resolution of rhabdomyolysis, the patient underwent urological surgery at an external hospital one month later.

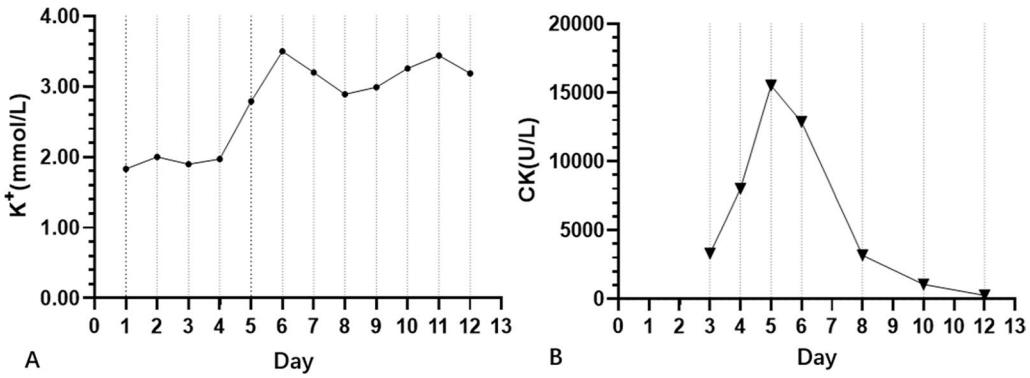

Fig. 1 Serum potassium and creatine kinase levels over time in patients after admission

Table 2 Preoperative standard posture test of the renin–angiotensin–aldosterone system

	Preoperative supine (day 1)	Preoperative supine (day 7)	Reference (supine)	Preoperative upright (day 7)	Reference (upright)
PRA (pg/mL)	3.37	3.68	4–24	3.58	4–38
PAC (pg/mL)	333.12	855.27	10–160	729.52	40–310
ARR	116.65	232.41		203.78	

PRA, plasma renin activity; PAC, plasma aldosterone concentration; ARR, aldosterone–renin ratio

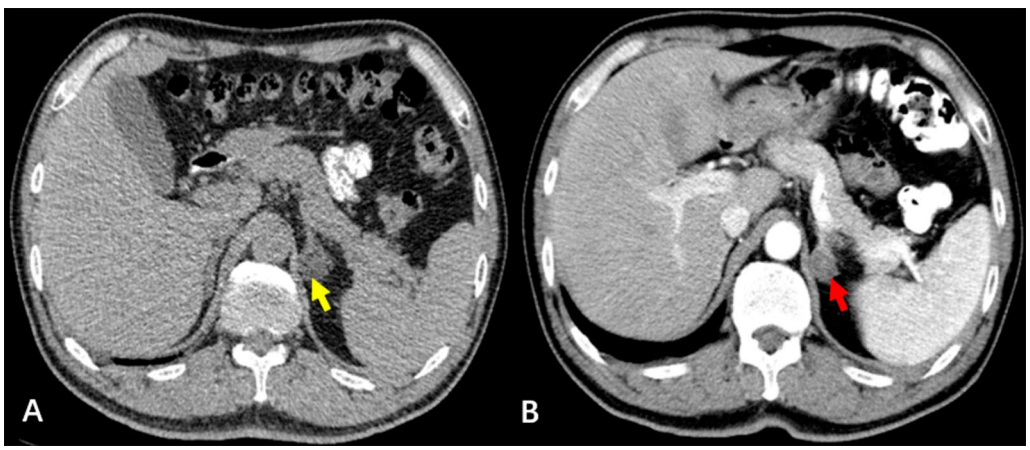

Fig. 2 **A** Plain renal CT image showing a low-density mass in the medial limb of the left adrenal gland, with a CT value of approximately 0 HU (yellow arrow). **B** Adrenal enhanced CT image demonstrating a round-like mass in the left adrenal gland, with a maximum cross-sectional area of approximately 22 mm ×16 mm, indicating mild enhancement during the enhanced scan (red arrow)

Pathology revealed nodular cortical hyperplasia in the left adrenal gland, with active local cell proliferation and focal medullary hyperplasia (Fig. 3). During follow-up at 1 month, 6 months, and 1 year, the patient reported relief of fatigue and muscle pain symptoms, with no recurrence of limb paralysis. Blood potassium levels returned to normal, and the patient required daily administration of 30 mg of nifedipine controlled-release tablets to maintain a blood pressure of approximately 120/80 mmHg.

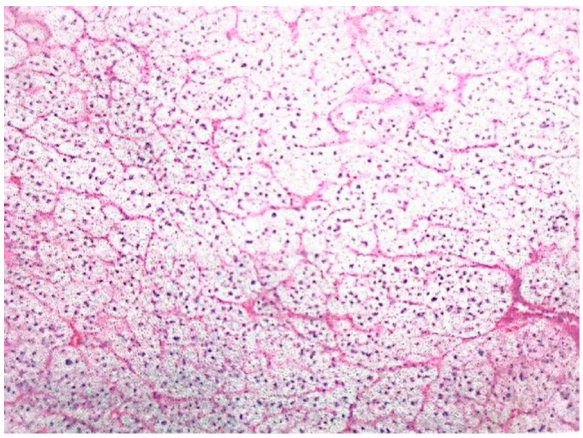

Fig. 3 Pathological findings suggestive of nodular hyperplasia with features of adrenal cortical adenoma were characterized by locally active cellular growth and focal medullary hyperplasia

Literature search and screening

Using the keywords "primary aldosteronism and rhabdomyolysis" or "primary aldosteronism and myopathy," a search was conducted on English databases (PubMed, Scopus, Web of science, and Embase) and Chinese databases, including China National Knowledge Infrastructure (CNKI), the Wanfang Medical Database, and the VIP Technology Journal Database. The search spanned from 1978 to 2023.

Two authors independently conducted searches in the Chinese and English databases using a predefined search strategy. After the literature was retrieved, duplicates were removed using EndNote X9 software. Subsequently, the two authors individually assessed the titles and abstracts of the remaining articles, excluding irrelevant articles. Relevant literature on primary aldosteronism combined with rhabdomyolysis was identified. In cases of disagreement between the two authors, decisions were made by the research team. Moreover, while reviewing the relevant literature, references were scrutinized, and citation tracking searches were conducted to address any omissions.

Results

A total of 39 articles were retrieved, comprising 9 in Chinese and 30 in English (Fig. 4). All the included articles were case reports, resulting in a total of 43 patients (Table 3). Three articles reported two patients each. A total of 19 of these patients were male and 24 were

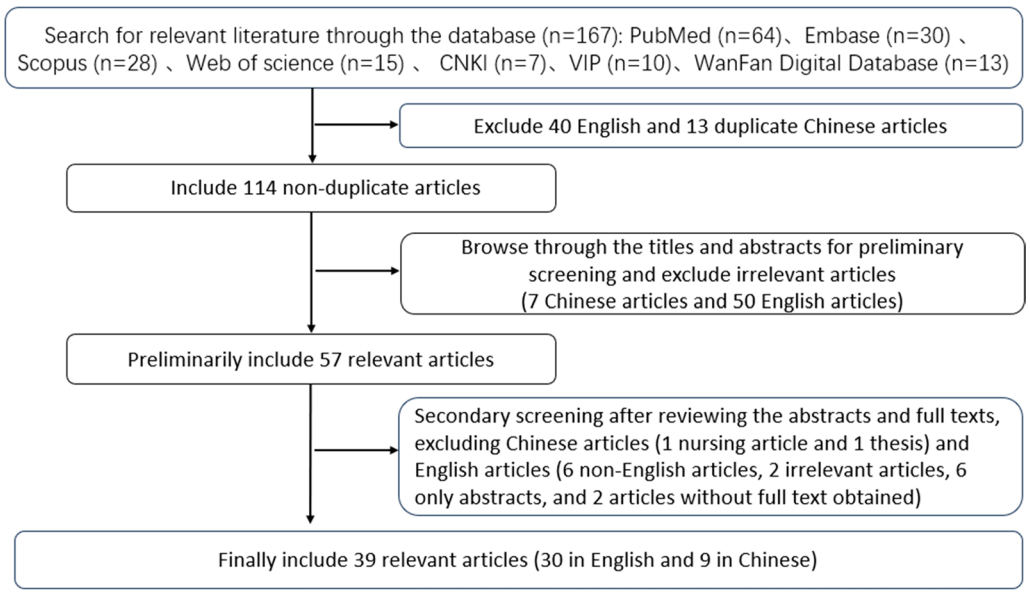

Fig. 4 Flowchart and results of the literature screening process. A total of 39 articles were included, including 30 in English and 9 in Chinese. CNKI, China National Knowledge Infrastructure; VIP, China Science and Technology Journal Database; Wanfan Digital Database, China Wanfang Data Knowledge Service Platform

Table 3 Summary of the clinical characteristics of patients with primary hyperaldosteronism combined with rhabdomyolysis in the literature and the present case

Variable	Category	Number of cases
Gender	Male	19
	Female	24
Age (years)	≤45	20
	>45	23
Clinical Symptom	Weakness	43
	Hypertension	42
Use of diuretics	Yes	9
K^+(mmol/L)	<2.0	33
	2.0–2.5	8
	2.5–3.5	2
CK (U/L)	<10000	31
	≥10000	12
ARR	<30	6
	≥30	29
	NA	8
AKI	Yes	8
Treatment	Surgery	32
	Medicine	11

CK, creatine kinase; ARR, aldosterone-renin ratio; AKI, acute kidney injury

female, with an age range of 14–73 years and an average age of 47.6 years. Muscle weakness was the common clinical symptom among all patients and was accompanied by additional manifestations such as muscle pain, muscle atrophy, and, in some cases, vomiting. All patients exhibited hypokalemia, with the lowest potassium level recorded being 1.04 mmol/L. Creatine kinase (CK) levels increased to varying degrees upon admission. Only one patient among all the patients did not have hypertension. Acute kidney injury occurred in eight patients, but they recovered to normal function after treatment. Nine patients, including the patient in the present case, were treated with diuretics before being diagnosed.

Among the 43 patients, 32 underwent surgical excision of the lesions, with pathology revealing adenomas in 29 patients and adrenal hyperplasia in 3 patients (Table 3). Additionally, 11 patients were treated with oral spironolactone (40–120 mg). All patients recovered well, with normal blood potassium levels observed in all patients and normalization of blood pressure in the majority of patients. A few patients still required medication for blood pressure control. A summary of clinical features, diagnosis data, and treatment of all cases with primary hyperaldosteronism combined with rhabdomyolysis in both the literature and the present case were summarized in the Supplementary Table.

Discussion and conclusions

The causes of primary aldosteronism (PA) include aldosterone-producing adenoma (APA), idiopathic hyperaldosteronism (also referred to as idiopathic aldosteronism), primary adrenal cortical hyperplasia (also termed unilateral adrenal hyperplasia), familial hyperaldosteronism, adrenal cortical carcinoma secreting aldosterone, and ectopic aldosterone-secreting tumors [3]. The various etiologies of primary aldosteronism necessitate distinct treatment approaches. For aldosterone-producing adenomas, unilateral adrenal hyperplasia, and adrenal cortical carcinoma secreting aldosterone, surgery is the preferred treatment. However, for idiopathic hyperaldosteronism and glucocorticoid-remediable aldosteronism, medication is the preferred treatment. Currently, adrenal vein sampling (AVS) is recognized as the gold standard for diagnosing and subtyping primary aldosteronism. According to literature review, a total of nine patients underwent successful adrenal vein sampling (AVS) [4]. Among them, one patient reported bilateral adrenal masses, and AVS suggested unilateral dominance, leading to surgical excision [5]. In a case reported by Zavatto, imaging indicated bilateral adrenal nodules, and comprehensive AVS confirmed the diagnosis of idiopathic hyperaldosteronism and was managed with oral medication [6]. Another patient showed no imaging abnormalities in the adrenal glands, but AVS detected increased aldosterone secretion from the right adrenal gland, prompting treatment with oral spironolactone [7]. Unfortunately, AVS was unsuccessful in the present patient. Pathology indicated nodular adrenal cortical hyperplasia, not suggesting aldosterone-producing adenoma. A patient reported by Kotsaftis also presented with a unilateral nodule, without AVS, and the pathology revealed adrenal hyperplasia [8]. These cases underscore the importance of AVS.

The International Consensus on the Pathological Diagnosis of Unilateral Primary Aldosteronism emphasizes the pivotal role of aldosterone synthase (CYP11B2) immunohistochemical staining in the pathological diagnosis and subtyping of unilateral primary aldosteronism [9]. Therefore, for patients with unilateral primary aldosteronism who are unable to undergo surgery or who have failed adrenal vein sampling (AVS), performing CYP11B2 immunohistochemical staining on pathological specimens is recommended. Among the patients described in this article, a total of 32 patients underwent surgical treatment [5, 6, 8, 10–34], with three of whom exhibited adrenal hyperplasia [8, 33]. Additionally, 11 patients received oral spironolactone (40–120 mg) [4, 7, 35–43]. None of the 32 surgically treated patients exhibited CYP11B2 immunohistochemical staining in the pathological reports.

Rhabdomyolysis (RM) can result from various underlying causes, such as trauma, ischemia, metabolic disorders, infection, drugs, and electrolyte imbalances [44]. In this particular case, low potassium levels constitute the primary reason for RM. Furthermore, the patient in this instance was taking diuretics, which potentially aggravated her hypokalemia. A literature review in this context included nine patients treated with diuretics [4, 6, 7, 19, 20, 37, 41]. Rhabdomyolysis-induced muscle breakdown releases a substantial amount of potassium into the circulation. However, when clinical syndrome manifests as both hypokalemia and rhabdomyolysis, the absolute concentration of potassium falls below detectable levels. Hence, substantial potassium supplementation and intravenous fluid administration are imperative. Reports in the literature highlight a markedly elevated risk of rhabdomyolysis when blood potassium levels fall below 2.0 mmol/L [21]. Additionally, reports indicate that severe hypokalemia, defined as a blood potassium concentration < 2.5 mmol/L, often leads to severe muscle weakness or RM [17]. According to literature review, 33 patients had initial blood potassium levels < 2.0 mmol/L, constituting approximately 76.7% of the total. There were 41 patients with blood potassium levels ≤ 2.5 mmol/L, representing 95.3% of the population, thereby affirming the previously drawn conclusion. However, the mechanisms by which hypokalemia leads to RM remain unclear. Severe hypokalemia (serum potassium < 2.5 mmol/L) may crucially contribute to muscle injury for three reasons [4, 19]: (1) microvascular constriction leading to reduced muscle blood supply and subsequent muscle cell dissolution, (2) inhibition of glycogen synthesis and storage, and (3) disruption of cell membrane ion transport.

Additionally, it is crucial to consider the relationship between serum transaminases and RM. Several studies have shown a correlation between changes in serum AST and alanine transaminase (ALT) and serum creatine kinase (CK) levels, suggesting that the increase in transaminase levels in patients with rhabdomyolysis may be a marker of skeletal muscle damage rather than liver cell damage [45, 46]. Of course, it is essential to inquire about the patient's history of liver disease, and if this information is missing, elevated transaminase levels may be indicative of skeletal muscle injury. In this literature review, 20 patients provided AST and ALT values, and these patients had no history of liver disease, yet transaminase values showed varying degrees of elevation.

In our case, the patient's blood pressure did not return to normal after surgery. Considering the patient's middle-aged onset and short history of hypertension without a family history of hypertension, three different types of antihypertensive drugs were required preoperatively to control blood pressure. Postoperatively, only one antihypertensive drug was needed to control blood pressure. Combined with pathological findings suggesting adenomatous nodular hyperplasia, the possibility of a subtle lesion on the contralateral adrenal gland cannot be ruled out. Hypertension is considered to be secondary to aldosterone excess. The diagnosis and treatment of this case align with the 2020 expert consensus on the diagnosis and treatment of primary aldosteronism in China [47]. Unfortunately, the patient experienced AVS failure, and CYP11B2 immunohistochemical staining was not performed on the pathological specimen. Postoperative pathology indicates the disease is likely due to idiopathic hyperaldosteronism. Considering the possibility of recurrence of the disease, the levels of blood potassium and renin–angiotensin–aldosterone should be closely monitored after surgery.

Strengths of this case include clear diagnosis and timely treatment based on the patient's clinical presentation, laboratory results, and imaging examinations. Spironolactone was administered preoperatively to correct hypokalemia. Moreover, the diagnostic and treatment processes were standardized, aligning with the 2020 expert consensus on the diagnosis and treatment of primary aldosteronism in China [47]. However, some limitations of our presentation case merit consideration. Firstly, the patient's adrenal vein sampling (AVS) was unsuccessful, leading to insufficient grounds for surgical treatment. Secondly, postoperative pathological specimens did not undergo CYP11B2 immunohistochemical staining.

In this paper, 43 cases of primary aldosteronism with hypokalemic rhabdomyolysis were retrospectively analyzed to improve clinicians' understanding of the disease. We found that severe hypokalemia (potassium ≤ 2.5 mmol/L) may be a significant predictor of rhabdomyolysis. However, in the literature, the diagnosis and treatment of patients with primary aldosteronism complicated by hypokalemic rhabdomyolysis was not completely standardized. In this literature review, only nine cases underwent adrenal vein sampling (AVS) according to guidelines for the management of primary aldosteronism to determine the cause [48]. Pathological testing, including CYP11B2 immunohistochemical staining, is recommended for patients who have not had AVS or who have failed AVS according to the International Consensus on Pathological Diagnosis of Unilateral Primary Aldosteronism [9]. The literature did not identify case–control studies regarding the incidence rate of rhabdomyolysis associated with primary aldosteronism, such as stratifying by serum potassium levels (> 2.5 mmol/L and ≤ 2.5 mmol/L). Therefore, multicenter case-series control study will be our next research direction in the future.

Based on published cases, it can be concluded that hypokalemic rhabdomyolysis is a rare clinical manifestation of primary aldosteronism and is more commonly observed in middle-aged females (< 60 years old). Patients with hypertension and severe hypokalemia should be highly suspected. Early diagnosis and treatment can improve patient prognosis, reducing the frequency of complications such as rhabdomyolysis and acute kidney injury. Furthermore, for patients with PA, AVS should be conducted. In instances of unilateral PA, testing for CYP11B2 is essential for enhancing the accuracy of subtype diagnosis. Moreover, emphasis should be placed on the correlation between serum transaminase levels and RM.

Abbreviations

PA	Primary aldosteronism
RM	Rhabdomyolysis
AKI	Acute kidney injury
AVS	Adrenal vein sampling
CYP11B2	Aldosterone synthase
CT	Computed tomography

Supplementary Information

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Additional file 1.

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Author contributions
P.S. conceived, designed, and wrote the review. P.S. and Y.L. treated the patient and contributed to the data collection. C.W. and Y.L. commented and revised the manuscript. All the authors read and approved the final manuscript.

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Availability of data and materials
The datasets collected and analyzed during the current study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
The reported investigations did not require ethics approval since they were conducted as part of routine clinical care with therapeutic intent. Prior to undergoing any clinical procedures, the patient provided written informed consent.

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests
The authors declare that the article content was composed in the absence of any commercial or financial relationships that could be construed as a potential competing interest.

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References

1. Funder JW, Carey RM. Primary aldosteronism: where are we now? where to from here? Hypertension. 2022;79:726–35.
2. Torres PA, Helmstetter JA, Kaye AM, Kaye AD. Rhabdomyolysis: pathogenesis, diagnosis, and treatment. Ochsner J. 2015;15:58–69.
3. Young WF Jr. Diagnosis and treatment of primary aldosteronism: practical clinical perspectives. J Intern Med. 2019;285:126–48.
4. Zavatto A, Concistrè A, Marinelli C, Zingaretti V, Umbro I, Fiacco F, *et al*. Hypokalemic rhabdomyolysis: a rare manifestation of primary aldosteronism. Eur Rev Med Pharmacol Sci. 2015;19:3910–6.
5. Eguchi T, Miyauchi S. A case of primary aldosteronism with secondary hyperparathyroidism and bilateral adrenal tumors. Endocrinol Diabetes Metab Case Rep. 2015. https://doi.org/10.1530/EDM-15-0029.
6. Pecnik P, Müller P, Vrabel S, Windpessl M. Two cases of hypokalaemic rhabdomyolysis: same but different. BMJ Case Rep. 2018. https://doi.org/10.1136/bcr-2017-223609.
7. Dominic JA, Koch M, Guthrie GP Jr, Galla JH. Primary aldosteronism presenting as myoglobinuric acute renal failure. Arch Intern Med. 1978;138:1433–4.
8. Kotsaftis P, Savopoulos C, Agapakis D, Ntaios G, Tzioufa V, Papadopoulos V, *et al*. Hypokalemia induced myopathy as first manifestation of primary hyperaldosteronism—an elderly patient with unilateral adrenal hyperplasia: a case report. Cases J. 2009;2:6813.
9. Williams TA, Gomez-Sanchez CE, Rainey WE, Giordano TJ, Lam AK, Marker A, *et al*. International histopathology consensus for unilateral primary aldosteronism. J Clin Endocrinol Metab. 2021;106:42–54.
10. Finsterer J, Lässer S. Severe hypokalemic paralysis as a manifestation of a mitochondrial disorder. Tohoku J Exp Med. 2013;231:9–12.
11. Martínez JJ, Oliveira CL, Meneses AL, Rodríguez SA, Corrales PP, López AH, *et al*. Rhabdomyolysis due to primary hyperaldosteronism. Endocrinol Nutr. 2009;56:431–4.
12. Goto A, Takahashi Y, Kishimoto M, Minowada S, Aibe H, Hasuo K, *et al*. Primary aldosteronism associated with severe rhabdomyolysis due to profound hypokalemia. Intern Med. 2009;48:219–23.
13. Chow CP, Symonds CJ, Zochodne DW. Hyperglycemia, lumbar plexopathy and hypokalemic rhabdomyolysis complicating Conn's syndrome. Can J Neurol Sci. 1997;24:67–9.
14. Díaz-López EJ, Villar-Taibo R, Rodriguez-Carnero G, Fernandez-Pombo A, Garcia-Peino R, Blanco-Freire MN, *et al*. Should we suspect primary aldosteronism in patients with hypokalaemic rhabdomyolysis? A systematic review. Front Endocrinol (Lausanne). 2023;14:1257078.
15. Han R, Jiang X. Hypokalemia-induced rhabdomyolysis as the first symptom of primary aldosteronism: a case report and literature review. Ann Palliat Med. 2022;11:2778–84.
16. Sirkeci O, Sirkeci EE, Kucukciloglu Y. Severe hypokalemia and rhabdomyolysis caused by Conn syndrome. Clin Ter. 2021;172:407–9.
17. Maung AC, Kerwen AK, Ching LP. Hypokalaemic rhabdomyolysis as initial presentation of primary aldosteronism. J R Coll Physicians Edinb. 2021;51:149–52.
18. Kollipara S, Ravindra S, Pai K, Shetty S. Quadriplegia and rhabdomyolysis as a presenting feature of Conn's syndrome. BMJ Case Rep. 2021. https://doi.org/10.1136/bcr-2020-234686.
19. Chen CT, Wang YC, Lin CM. Hypokalemia-induced rhabdomyolysis caused by adrenal tumor-related primary aldosteronism: a report of 2 cases. Am J Case Rep. 2021;22:e929758.
20. Lee JH, Kim E, Chon S. Hypokalemia-induced rhabdomyolysis by primary aldosteronism coexistent with sporadic inclusion body myositis. Ann Rehabil Med. 2015;39:826–32.
21. Wen Z, Chuanwei L, Chunyu Z, Hui H, Weimin L. Rhabdomyolysis presenting with severe hypokalemia in hypertensive patients: a case series. BMC Res Notes. 2013;6:155.
22. Cooray MS, Bulugahapitiya US, Peiris DN. Rhabdomyolysis: a rare presentation of aldosterone-producing adenoma. Indian J Endocrinol Metab. 2013;17:S237–9.
23. Karagüzel G, Bahat E, Imamoğlu M, Ahmetoğlu A, Yildiz K, Okten A. An unusual case of an aldosterone-producing adrenocortical

adenoma presenting with rhabdomyolysis. J Pediatr Endocrinol Metab. 2009;22:1087–90.
24. Mahdyoon H, Mermiges DN, Wisgerhof M. Conn's syndrome with rhabdomyolysis mimicking deep vein thrombophlebitis. South Med J. 1990;83:346–7.
25. Tan HY, Gong LL, Wang Y, Bai J, Ren W. Primary aldosteronism associated with rhabdomyolysis:a report of 2 cases and review of the literature. J Chongq Med Univ. 2018;6:763–7.
26. Gao PL, Yang ZM. A case of primary aldosteronism complicated by rhabdomyolysis and literature review. J Shanxi Med Univ. 2018;4:439–41.
27. Zhang J, Li H, Zhang XB, Guo LX. The first clinical manifestation of Rhabdomyolysis due to severe hypokalemic of a patient with primary aldosteronism. Int J Endocrinol Metabol. 2017;5:358–60.
28. Chen L, Han XM, Liu GX, Zang XP, Chen M. A case report of primary aldosteronism complicated by hypokalemic rhabdomyolysis. Chin J Urol. 2013;9:719–819.
29. Wang Y, Chen J, Yang K, Ren Q, Chen MH. Primary aldosteronism-induced profound hypokalemia and rhabdomyolysis: a case report. J Internal Intens Med. 2012;6:383–4.
30. Yao B, Qin Z, Tan Y, *et al*. Rhabdomyolysis in primary aldosteronism: a case report and review of the literature. AACE Clin Case Rep. 2015;1:e21–7.
31. Zhang L, Shi HJ. Primary aldosteronism with rhabdomyolysis: a case report. J Musculoskeletal Pain. 2014;22:102–5.
32. Demir K, Sonmez O, Kayrak M, *et al*. Severe hypokalemia-associated rhabdomyolise and unusual poliuria in patient with primary aldosteronism. Eur J Gen Med. 2012;9:211–3.
33. Gonerska-Szadkowska A, Wittych-Długosz J, Makarewicz J, *et al*. Rhabdomyolysis following severe hypokalemia caused by Conn's syndrome. Arch Med Sci. 2007;3(3):274–7.
34. Li RH, Cui XF, Wang XY. Primary aldosteronism characterized by habdomyolysis: a case report. J China-Japan Friendship Hospital. 2023;37:116–8.
35. Dyrmishi B, Olldashi T, Rista E, Fureraj T, Ylli D, Ylli A. Severe hypokalemia induced rhabdomyolysis by primary hyperaldosteronism coexistent with recurrent bilateral renal calculi. Acta Endocrinol (Buchar). 2017;13:228–31.
36. Tsai WT, Chen YL, Yang WS, Lin HD, Chien CC, Lin CL. Primary aldosteronism associated with severe hypokalemic rhabdomyolysis. Hormones (Athens). 2012;11:505–6.
37. Ozgür B, Kürsat S. Hypokalemic rhabdomyolysis aggravated by diuretics complicating Conn's syndrome without acute renal failure. Clin Nephrol. 2002;57:89–91.
38. Schady W, Yuill GM. Myopathy and primary hyperaldosteronism. Neurology. 1981;31:225–6.
39. Kang JJ, Su PX, Deng BM, Yang HJ, Wang ZC. A case of adenoma-associated primary aldosteronism complicated by rhabdomyolysis. J Diagnost Concepts Pract. 2019;18:583–4.
40. Zhang HM, Zou YB, Zhang YQ, Wu HY. A case of primary aldosteronism complicated by rhabdomyolysis. Chin Circulat J. 2006;6:477–577.
41. Petidis Anna K, Douma S, Aslanidis S, *et al*. Hypertension associated with rhabdomyolysis. J Clin Hypertens. 2007;9:60–2.
42. Cakir I, Senol S, Simsek Y, *et al*. Primary hyperaldosteronism presenting with rhabdomyolysis in emergency room—case report. J Acute Dis. 2016;5(3):264–6.
43. Ruan ZF, He WQ. Severe hypokalemia induced rhabdomyolysis: a case report. Chin J Difficult Complic Cases. 2012;6:474–574.
44. Stahl K, Rastelli E, Schoser B. A systematic review on the definition of rhabdomyolysis. J Neurol. 2020;267:877–82.
45. Jo KM, Heo NY, Park SH, Moon YS, Kim TO, Park J, *et al*. Serum aminotransferase level in rhabdomyolysis according to concurrent liver disease. Korean J Gastroenterol. 2019;74:205–11.
46. Weibrecht K, Dayno M, Darling C, Bird SB. Liver aminotransferases are elevated with rhabdomyolysis in the absence of significant liver injury. J Med Toxicol. 2010;6:294–300.
47. Chinese Society of Endocrinology. Expert consensus on the diagnosis and treatment of primary aldosteronism (2020). Chin J Endocrinol Metab. 2020;36:727–36.
48. Funder JW, Carey RM, Mantero F, *et al*. The management of primary aldosteronism: case detection, diagnosis, and treatment: an endocrine society clinical practice guideline. J Clin Endocrinol Metab. 2016;101:1889–916.

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