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Case report

Recurrent motor paralysis in a young patient with hypertension

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Abstract: We present a case of a young patient with severe myopathy and repeated episodes of lower limb paralysis. The patient was diagnosed as having primary hypertension and because of severely increased blood pressure he received four hypo-tensive drugs including a diuretic. Laboratory analyses indicated severe hypokalemia and increased creatin kinase. The association of resistant hypertension with hypokalemia suggested primary hyperaldosteronism (PA) confirmed by the increased values of aldosterone and suppression of renin. Abdominal computed tomography identified an adrenal ade-noma as being the cause of increased aldosterone concentration. Even though muscle weakness is frequently associated in PA, paralytic hypokalemic myopathy with rhabdomyolysis is a rare finding, imposing a differential diagnosis with other causes of myositis. In our patient, muscular symptoms have remitted after hypokalemia correction. Surgical re-moval of adrenal adenoma significantly reduced arterial pressure values and persistently normalized serum potassium. Conclusion. In patients with arterial hypertension, muscle weakness may be a sign of hypokalemic myopathy suggest-ing PA. The administration of diuretics in these patients may aggravate hypokalemia and its unfavorable clinical con-sequences including severe rhabdomyolysis.

Keywords: hypokalemic paralytic rhabdomyolysis, secondary hypertension, primary hyperaldosteronism

1. Introduction

Primary hyperaldosteronism (PA) is characterized by increased and uncontrolled aldosterone secretion caused by either an adrenal tumor, usually benign, or by hyperplasia of the adrenal glands. This syndrome has been described for the first time by Conn in 1955, in a patient with severe hypertension and hypokalemia due to an aldosterone secreting adrenal tumor [1].

Hyperaldosteronism is one of the main causes of secondary hypertension, identified in 10-20% of patients with resistant hypertension [2,3], and in 5 and 13% of all patients with hypertension [4]. Nevertheless, PA is still markedly undiagnosed [5].

Increased aldosterone induces excessive renal sodium retention with circulating volume overload and abnormal high arterial blood pressure. Concomitantly, it also causes excessive potassium secretion and hypokalemia [4]. Even though muscle weakness is often associated with PA, myopathy, documented by biological and/or histopathological examination, is not common [6]. Nevertheless, although rare, cases with extremely low serum potassium are at risk of rhabdomyolysis and muscle paralysis [7].

Here we present the medical history of a young patient with severe hypertension, treated with an association of four hypotensive drugs, including a diuretic, who

had suffered from recurrent episodes of inferior limb muscle paralysis for three years. On admission, he had marked hypokalemia.

Case presentation

Presenting symptoms:

Our patient is a 35-year-old male who presented in the emergency room for a severe and persistent episode of inferior limb paralysis.

Medical history:

The patient has a 3-year history of increased blood pressure. He has been diagnosed as having essential hypertension and has been treated during this period of time, with an association of three or four antihypertensive drugs, including a diuretic (thiazide or loop diuretic) because of severely increased blood pressure.

Despite this intensive treatment, hypertension was usually uncontrolled. During the previous 2 years, the patient noted repeated episodes of inferior limb muscle paralysis, of various intensities, spontaneously reversible after several hours of rest. Muscle weakness was frequently induced by exercise. His medical history is free of any significant disease. There was no familial history of any neurological or cardiovascular disease. On admission his medication consisted of an angiotensin converting enzyme inhibitor (Perindopril 10 mg/day, a beta blocker (betaxolol 10 mg/day), a calcium channel blocker (amlodipine 10 mg/day) and a loop diuretic (furosemide 40 mg/day), taken during the last three months.

Physical examination revealed an increased body mass index (BMI=32 m^2/kg) and an abdominal distribution of the adipose tissue. On admission, he had proximal muscle weakness of both legs, but neurological examination found normal muscle tone and mass as well as normal cranial nerves function. He had no modification of cutaneous reflexes, while osteotendinous reflexes were diminished. The cardiovascular examination found normal heart rhythm at a frequency of 70 beats/min, without murmurs, brachial arterial pressure was 170/100 mmHg while peripheral pulse waves were normal. The abdominal physical examination revealed mild hepatomegaly. The abdomen was not painful and there were no abdominal arterial murmurs.

Laboratory investigations:

The biochemical analysis indicated severe hypokalemia (K=1.50 mmol/l), normal natremia (Na=140 mmol/l), metabolic alkalosis (blood PH=7.53, pCO2=47 mmHg, standard bicarbonate=33 mmol/l), normal renal function tests, hyperglycemia (176 mg/dl, 150 mg/dl) and glycosuria.

The total creatin kinase was extremely elevated (22 894 U/l) compared to normal values (<171U/l) indicating rhabdomyolysis. The aspartate amino-transferase (ASAT) and the lactic dehydrogenase (LDH) were also elevated while other liver function tests including alanine amino-transferase, gamma glutamyl transpeptidase and alkaline phosphatase were normal, suggesting muscular origin of ASAT and LDH.

Since our patient declared low alcohol consumption (one low alcohol drink once or twice weekly) and denied statin treatment we excluded alcohol or statin induced rhabdomyolysis. We also excluded hypothyroid myopathy based on normal thyroid stimulating hormone and free T4. Inflammatory markers (CRP, ESR) and antinuclear antibodies were within normal values ruling out inflammatory myositis.

The exploration of lipid metabolism showed normal LDL-cholesterol and HDL-cholesterol and increased triglycerides (348 mg/dl).

Electrocardiography showed a normal sinus rhythm, ST depression and T wave flattening in all precordial leads explained by severe hypokalemia.

Echocardiography did not identify any significant anomaly.

Marked hypokalemia and metabolic alkalosis in a young patient with severe arterial hypertension were strongly suggestive for PA. The screening tests were performed without stopping antihypertensive medication because hypertension was severe. Plasma aldosterone and renin concentrations were measured in supine position. The aldosterone level was elevated 53.7 ng/dl (reference range 1.17-23.6 ng/dl), direct renin concentration was suppressed < 0.3 ng/l (low limit 3.6 ng/l) and aldosterone/renin concentration ratio was extremely elevated > 1790. These laboratory modifications supported the diagnosis of PA.

We performed further imaging examinations of the adrenal glands. Abdominal ultrasonography failed to identify any suprarenal pathologic modification, but the examination conditions were not appropriate due to increased periadrenal adipose tissue. Nevertheless, contrast-enhanced computed tomography of both adrenal glands showed an adenoma located in the body and medial limb of the right adrenal gland (Figure 1). Both abdominal ultrasonography and computed tomography showed hepatic steatosis.

We withdrew loop diuretics, replacing them with an anti-aldosterone drug spironolactone and we supplemented potassium via intravenous route. Intravenous hydration was added. Progressively, serum potassium values increased, muscular weakness remitted, and muscular enzymes normalized. The resolution of muscular symptoms and normalization of muscular enzymes after potassium substitution supported our diagnosis of hypokalemic myopathy with rhabdomyolysis.

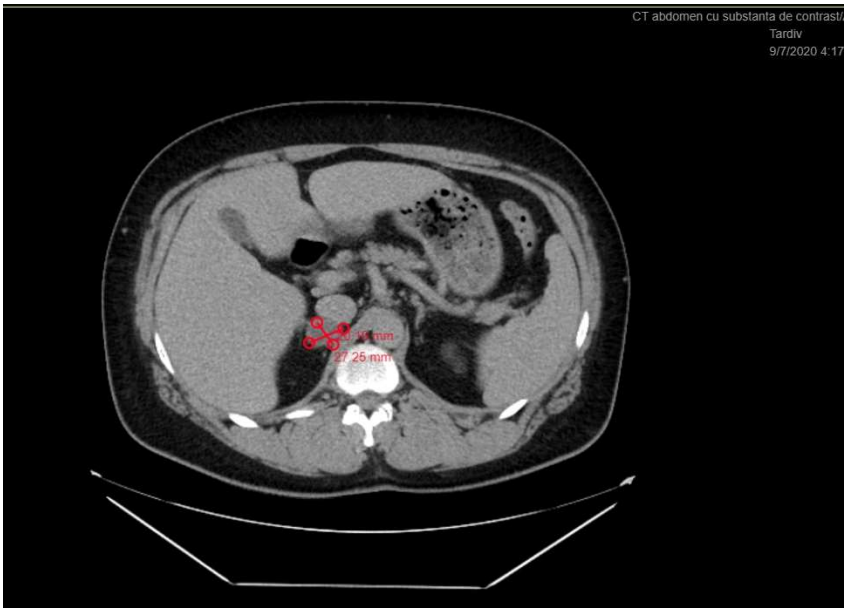
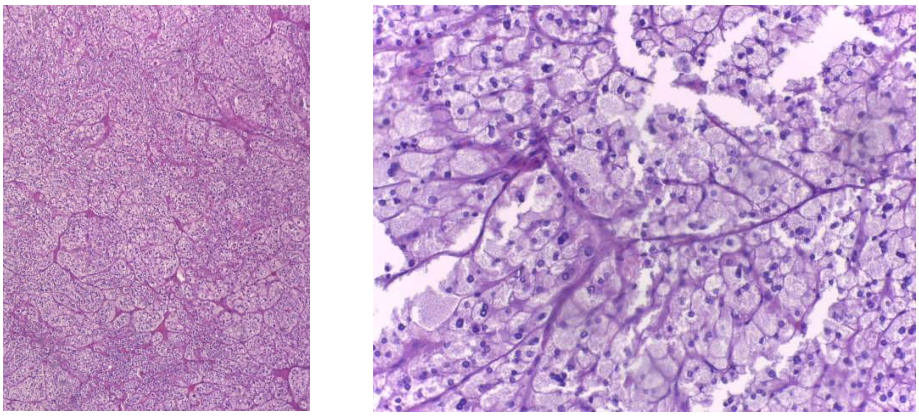


Figure 1. Contrast-enhanced computed tomography scan of the adrenal glands showed a hypodense oval lesion, 27/20 mm, affecting the body and the medial limb of the right adrenal gland. The left adrenal gland was normal.

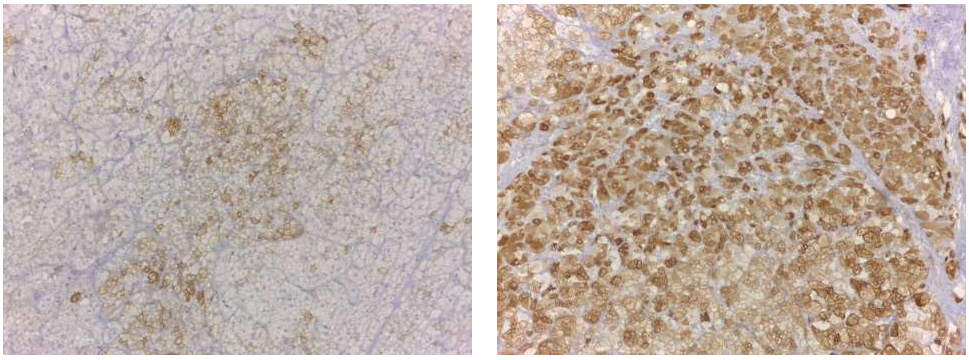
A laparoscopic adrenalectomy of the right adrenal gland was subsequently performed. A nodular, brown-orange tumor surrounded by a fine fibrous pseudocapsule was identified macroscopically. Histological examination confirmed adrenal cortical adenoma. Hematoxylin-eosin staining (Figure 2, a and b) revealed cellular proliferation disposed in nests, separated by fine conjunctive-vascular septa. The predominant cells were large, with pale cytoplasm and round, uniform nucleus with normally distributed chromatin. There were no areas of necrosis or cell mitoses.



(**a**) (**b**)

Figure 2. Histological examination. a. Cell nets separated by thin connective tissue septa (HE, 25x). b. Histological details of cell nests consisting of great cells (HE, 200x).

Histochemistry (Figure 3) found focal positivity for synaptophysin (a) and diffuse positivity for inhibin alpha (b) confirming cortical adrenal origin of the tumor



(**a**) (**b**)

Figure 3. Histochemistry. a. Focal positivity for Synaptophysin. b. Diffuse positivity for inhibin alpha.

Post-surgical evolution was favorable with significant reduction of arterial pressure. The patient needed only one medication (candesartan 32 mg/d) for optimal blood pressure control. Sulfonylurea medication (gliclazide 60 mg/d) was indicated for his type 2 diabetes mellitus. At discharge he had no muscular symptoms and potassium serum values were normal.

4. Discussion

The clinical picture, dominated by severe, uncontrolled hypertension and recurrent episodes of muscle weakness, was highly suggestive for PA.

Aldosterone is an essential hormone in sodium-potassium homeostasis. It stimulates Na^+/K^+ ATP-ase in the basocellular membrane of the distal nephron, inducing sodium and water reabsorption as well as potassium and hydrogen ion secretion. Aldosterone also stimulates extrarenal potassium secretion by the salivary glands, airway epithelium and colonic mucosa [2]. Hyperaldosteronism causes hypertension mainly because of sodium and water excessive retention, hypokalemia and metabolic alkalosis.

Hypokalemia increases resting membrane potential and reduces muscle excitability and contractility. Even though muscle weakness is frequently seen in PA, rhabdomyolysis is rarely reported, usually associated with very low potassium levels (below 2.0 mmol/l) [7,8,6,9,10,11,12,13,14]. Moreover, it has been reported that hypokalemia is detected in less than half of patients with PA [4,5].

Hypokalemia is more pronounced in patients taking rich salt diets because increased aldosterone production stimulates potassium secretion if sodium concentration in the collecting tubes is high [8,25]. Asian populations have been reported to be more exposed to hypokalemic paralysis, which is rarely described in occidental populations [8]. Aggravation of hypokalemia in patients with PA may be caused by vomiting [10], diarrhea [15], or potassium spearing diuretic administration [11,12,14,15,16,17].

Chow et al. reported a case of PA with lumbar plexopathy with unprovoked severe hypokalemia and rhabdomyolysis [18]. Muscular weakness accompanied by severe spontaneous hypokalaemia has been also reported [13,19,20,21,22].

The mechanism involved in hypokalemic rhabdomyolysis is not completely elucidated. Impaired exercise vasodilation of muscle arterioles, reduced glycogen synthesis and storage, and alteration of cell membrane transport have been suggested [7,21,23]. Optical microscopic examination in hypokalemic myopathy with rhabdomyolysis revealed diffuse necrosis and muscle cells vacuolization. In electron microscopy, dissolution of microfilaments and sarcoplasmic reticulum in necrotized cells has been described [19,21].

In our patient we were able to exclude other more frequent causes of rhabdomyolysis including drug or alcohol consumption, hypothyroidy, statin use or other medications, viral and bacterial infections, autoimmune myositis, trauma or excessive exercise. Moreover, the correction of hypokalemia was accompanied by progressive decrease of total creatin kinase and complete remission of muscular symptoms, which supported our diagnosis of hypokalemic myopathy. Our patient correlated muscular symptoms with more intensive exercise, but he could not establish a cause-effect relationship with the use of diuretics. Nevertheless, the extremely low hypokalemia might have been aggravated by the prolonged administration of potassium spearing diuretics.

Screening for hyperaldosteronism is indicated in patients with resistant hypertension, as well as in patients with hypertension and spontaneous or diuretic induced hypokalemia. The recommended test is plasma aldosterone to renin ratio [6,24]. Suppression of renin activity and increased serum aldosterone with aldosterone/renin ratio exceeding 20/1 argues in favor of PA. Confirmatory tests such as oral sodium loading, saline infusion, fludrocortisone suppression, and the captopril challenge test aim to suppress aldosterone secretion. In PA aldosterone levels remain elevated [25].

For the etiological diagnosis of PA, contrast-enhanced computed tomography scan of the adrenal glands is recommended. Adrenal venous sampling is necessary to detect unilateral aldosterone secretion (adenoma or hyperplasia). However, according to recent diagnosis algorithms, in young patients (less than 35 years), with severe hypokalemia, markedly increased serum aldosterone and unilateral adrenal mass on CT scan, confirmatory tests and adrenal venous sampling are not required before surgical intervention [6].

In clinical practice, early diagnosis of PA is essential because patients with PA carry an excessive risk of cardiovascular complications which is independent of arterial pressure values [27,28]. This can be explained by the aldosterone's direct vascular and cardiac effects, including endothelial dysfunction, wall remodeling and fibrosis,

myocardial hypertrophy and fibrosis [28,29]. Moreover, aldosterone is involved in metabolic disturbances including insulin resistance, systemic inflammation and abdominal obesity which subsequently increase cardiovascular risk [30].

The treatment of choice in PA for patients with unilateral adenoma or unilateral adrenal hyperplasia is unilateral laparoscopic adrenalectomy. In patients with bilateral adrenal hyperplasia, medical treatment is indicated including a mineralocorticoid receptor antagonist, such as spironolactone or, as an alternative, eplerenone [24].

Our patient's arterial pressure significantly improved following laparoscopic unilateral adrenalectomy, and, on discharge, he received only one hypotensive medication (candesartan 32 mg/day) which normalized his arterial pressure. It has been reported that, after adrenalectomy, in 65% of cases, only moderate control of arterial pressure is obtained imposing the continuation of medical hypotensive medication. Predictive factors for complete remission of hypertension after adrenalectomy are the following: 2 or fewer antihypertensive medications, body mass index ≤25 kg/m^2, duration of hypertension ≤6 years, and female sex [31]. Our patient has at least three risk factors for incomplete resolution including male sex, more than 2 hypotensive drugs and increased body mass. Log time follow up will be necessary for the control of blood pressure, diabetes mellitus, obesity and serum potassium. Adenoma recurrence is possible, although exceptional, after complete unilateral adrenalectomy [32].

5. Conclusion

In young patients with severely increased blood pressure, a cause of secondary hypertension must be investigated. Repeated episodes of limb paralysis associated with resistant hypertension are indicative of PA. In such cases, investigations for the confirmation of PA are necessary because the administration of potassium sparing diuretics may aggravate muscular symptoms and induce severe rhabdomyolysis.

Author Contributions:

Conceptualization, A.A. and D.C.; methodology, A.A., A.N; software A.P.; validation, X.X., Y.Y. and Z.Z.; formal analysis, A.A. and A.N ; A.A. and D.C, ; resources, A.A., A.N; data curation, A.A; writing—A.A., A.N., A.P.; writing—A.A X.X.; A.A; supervision, D.C. All authors have read and agreed to the published version of the manuscript.

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References

1. Conn J.W. Presidential address. Transl Res 1955; 45:3–17. Availa-ble: www.translationalres.com/article/0022214355900482/fulltext (accessed 2024 jan. 13).
2. Strauch B, Zelinka T, Hampf M, Bernhardt R, Widimsky J Jr. Prevalence of primary hyperaldosteronism in moderate to severe hypertension in the Central Europe region. J Hum Hypertens. 2003;17(5):349-352. doi: 10.1038/sj.jhh.1001554.
3. Douma S, Petidis K, Doumas M, Papaefthimiou P, Triantafyllou A, Kartali N, et al. Prevalence of primary hyperal-dosteronism in resistant hypertension: a retrospective observational study. Lancet. 2008;371(9628):1921-1926. doi: 10.1016/S0140-6736(08)60834-X. Erratum in: Lancet. 2008 Dec 13;372(9655):2022.
4. Young WF. Primary aldosteronism: renaissance of a syndrome. Clin Endocrinol (Oxf) 2007; 66(5): 607–618.
5. Mulatero P, Monticone S, Burrello J, Veglio F, Williams TA, Funder J. Guidelines for primary aldosteronism: uptake by primary care physicians in Europe. J Hypertens. 2016 Nov;34(11):2253-2257. doi: 10.1097/HJH.0000000000001088. PMID: 27607462.
6. Funder JW , Carey RM , Mantero F , Murad MH, Reincke M, Shibata H, et al. The management of primary aldoste-ronism: case detection, diagnosis, and treatment: an Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab. 2016; 101: 1889–1916. doi.org/10.1210/jc.2015-4061. Erratum in: J Clin Endocrinol Metab. 2025 Aug 13:dgaf445. doi: 10.1210/clinem/dgaf445.
7. Ma JT, Wang C, Lam KS, Yeung RT, Chan FL, Boey J, et al. Fifty cases of primary hyperaldosteronism in Hong Kong Chinese with a high frequency of periodic paralysis. Evaluation of techniques for tumour localization. Q J Med. 1986;61: 1021 1037. PMID: 3659246.
8. Huang YY, Hsu BR, Tsai JS. Paralytic myopathy--a leading clinical presentation for primary aldosteronism in Tai-wan. J Clin Endocrinol Metab. 1996 Nov;81(11):4038-41. doi: 10.1210/jcem.81.11.8923857. PMID: 8923857.
9. Tsai WT, Chen YL, Yang WS, Lin HD, Chien CC, Lin CL. Primary aldosteronism associated with severe hypokalemic rhabdomyolysis. Hormones (Athens) 2012; 11(4): 505-6. doi:10.14310/horm.2002.1385.
10. Cakir I, Senol S, Simsek Y, Karaca Z, Unluhizarci K, Fanrıverdi F. Primary hyperaldosteronism presenting with rhabdomyolysis in emergency room–Case report. JAD. 2016; 5(3): 264–266. doi.org/10.1016/j.joad.2016.03.020.
11. Lee JH, Kim E, Chon S. Hypokalemia-Induced Rhabdomyolysis by Primary Aldosteronism Coexistent With Sporad-ic Inclusion Body Myositis. Ann Rehabil Med. 2015;39(5):826-832. doi: 10.5535/arm.2015.39.5.826.
12. Wu C, Xin J, Xin M, Zou H, Jing L, Zhu C, Lei W. Hypokalemic myopathy in primary aldosteronism: A case report. Exp Ther Med. 2016;12(6):4064-4066. doi: 10.3892/etm.2016.3864.
13. 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 2: 6813, 2009. 5. doi 10.4076/1757-1626-2-6813.
14. 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(20):3910-3916. PMID: 26531278.
15. Goto A, Takahashi Y, Kishimoto M, Minowada S, Aibe H, Hasuo K, et al. Primary aldosteronism associated with se-vere rhabdomyolysis due to profound hypokalemia. Intern Med 2009; 48: 219-223. doi: 10.2169/internalmedicine.
16. Zaloznik AA, McCowen KD. Hypokalemic myopathy with serum enzyme elevations and failure of adrenal scinti-scan in a patient with adrenal adenoma: Case Report, Military Medicine, 1980, 145 (3), 186–187. doi.org/10.1093/milmed/145.3.186.
17. Cooray MS, Bulugahapitiya US, Peiris DN. Rhabdomyolysis: A rare presentation of aldosterone-producing adeno-ma. Indian J Endocrinol Metab. 2013 Oct;17(Suppl 1):S237-239. doi: 10.4103/2230-8210.119583. PMID: 24251171; PMCID: PMC3830317
18. Chow CP, Symonds CJ, Zochodne DW. Hyperglycemia, lumbar plexopathy and hypokalemic rhabdomyolysis com-plicating Conn's syndrome. Can J Neurol Sci 1997; 24(1): 67-69.
19. Sambrook MA, Heron JR, Aber GM. Myopathy in association with primary hyperaldosteronism. J Neurol Neurosurg Psychiatry. 1972;35(2):202-207. doi: 10.1136/jnnp.35.2.202. PMID: 5037033; PMCID: PMC494037.
20. Rauf FA, Pervaiz Z, Khan T, Swaminathan G, Trandafirescu T. Hypokalemia-Induced Rhabdomyolysis Secondary to Adrenal Adenoma: A Case Report. Cureus. 2024 Dec 3;16(12):e75063. doi: 10.7759/cureus.75063. PMID: 39759753; PMCID: PMC11698543.
21. Wen Z, Chuanwei L, Chunyu Z, Hui H, Weimin L. Rhabdomyolysis presenting with severe hypokalemia in hyper-tensive patients: a case series. BMC Res Notes 2013; 6: 155. doi: 10.1186/1756-0500-6-155.
22. Ohara N, Tani T, Terajima K, Ozawa T, Yoneoka Y, Shimada H, Nakamura Y, Hasegawa G, Nishiyama T. Primary Aldosteronism and Hypokalemia-induced Rhabdomyolysis in a Patient with Aldosterone-producing Adenoma: A Case Report and Literature Review. Intern Med. 2025;64(6):871-879. doi: 10.2169/internalmedicine.3629-24. Epub 2024 Aug 10. PMID: 39135257; PMCID: PMC11986306.
23. Knochel JP. Mechanisms of rhabdomyolysis. Curr Opin Rheumatol 1993; 5: 725-731. doi: 10.1097/00002281-199305060-00006.
24. Kline GA, Prebtani APH, Leung AA, Schiffrin EL. Primary aldosteronism: a common cause of resistant hypertension. CMAJ. 2017;189(22):E773-E778. doi: 10.1503/cmaj.161486.

25. Cobb A, Leslie SW. Primary Hyperaldosteronism. [Updated 2025 May 4]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK539779/. (accessed 2025, September 12).
26. Aronova A, Fahey III TJ, Zarnegar R. Management of hypertension in primary aldosteronism. World J Cardiol. 2014; 6(5): 227-233 Available from: URL: http://www.wjgnet. com/1949-8462/full/v6/i5/227.htm doi: http://dx.doi. org/10.4330/wjc.v6.i5.227.
27. Rossi GP, Rossi FB, Guarnieri C, Rossitto G, Seccia TM. Clinical Management of Primary Aldosteronism: An Update. Hypertension. 2024;81(9):1845-1856. doi: 10.1161/HYPERTENSIONAHA.124.22642.
28. Marney AM, Brown NJ. Aldosterone and end-organ damage. Clin Sci (Lond). 2007; 113(6): 267-278. doi: 10.1042/CS20070123.
29. Prejbisz A, Warchoł-Celińska E, Lenders JW, Januszewicz A. Cardiovascular Risk in Primary Hyperaldosteronism. Horm Metab Res. 2015;47(13):973-980. doi: 10.1055/s-0035-1565124.
30. Otsuka H, Abe M, Kobayashi H. The Effect of Aldosterone on Cardiorenal and Metabolic Systems. International Jour-nal of Molecular Sciences. 2023; 24(6):5370. doi.org/10.3390/ijms24065370.
31. Benham JL, Eldoma M, Khokhar B, Roberts DJ, Rabi DM, Kline GA. Proportion of Patients with hypertension resolu-tion following adrenalectomy for primary aldosteronism: a systematic review and meta-analysis. J Clin Hypertens (Greenwich). 2016;18(12):1205-1212. doi: 10.1111/jch.12916.
32. Kong L, Zhang J, Dong L, Xu J, Gao P-J, Wang J-G and Zhu L Recurrence of Primary Aldosteronism 10 Years After Left Adrenalectomy for Aldosterone-Producing Adenoma: A Case Report. Front. Endocrinol. 2021;12:728595. doi: 10.3389/fendo.2021.728595.