

Aldosteronism with mild hypokalemia presenting as life-threatening ventricular arrhythmias

A case report

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Abstract

Rationale: Primary aldosteronism (PA) with hypokalemia increases the risk of life-threatening ventricular arrhythmias. Cases of PA with malignant arrhythmia as the first symptom have been reported. The role of severe hypokalemia in triggering malignant ventricular arrhythmia is well documented. However, few cases of PA with mild hypokalemia that presented with life-threatening ventricular tachycardia have been reported.

Patient concerns: A 74-year-old man was admitted to our hospital 25 hours after suffering from syncope caused by ventricular tachycardia without chest pain. Electrocardiogram showed ST segment depression and T wave inversion in leads III, avF, V4–V6. Mild QT prolongation was observed during sinus rhythm. Blood tests showed mild hypokalemia and elevated plasma aldosterone level. Abdominal computed tomography showed a nodule in the left adrenal gland. Coronary angiography revealed stenosis in the right coronary artery.

Diagnosis: Prolonged QT interval, hypokalemia, high level plasma aldosterone, a nodule in the left adrenal gland and right coronary artery stenosis led to a diagnosis of aldosterone hyperplasia and adrenal nodule with ischemic heart disease.

Intervention: Intravenous potassium and magnesium were administered to correct hypokalemia and a stent was implanted in the right coronary artery for vascularization. A prescription aldosterone receptor antagonist, spironolactone, was prescribed for hyperaldosteronemia.

Outcomes: During 6 months of follow-up, no episodes of ventricular tachycardia or syncope occurred, and serum potassium level remained normal.

Lessons: In patients with ventricular tachycardia and mild hypokalemia, physicians need to consider that PA and ischemia heart disease may be one of the possible causes of electrical storm.

Abbreviations: CAD = coronary artery disease, PA = primary aldosteronism, RCA = right coronary artery, TIMI = thrombolysis in myocardial infarction.

Keywords: hypokalemia, primary aldosteronism, ventricular tachycardia

1. Introduction

Primary aldosteronism (PA) is usually associated with several cardiovascular diseases. The most common phenotype of PA is

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endocrine hypertension characterized by autonomous aldosterone production, hypertension, hypokalemia, and suppressed plasma renin activity.^[1] On the other hand, PA with high levels of circulating aldosterone increases the risk of cardiovascular events, including myocardial infarction, stroke, and arrhythmias.^[2] PA may also induce hypokalemia which manifests as prolongation of QT-interval on electrocardiogram. The role of severe hypokalemia in triggering malignant ventricular arrhythmia is well documented.^[3] However, these arrhythmias are atypical signs of PA even with severe hypokalemia. In this case report, we describe a case of PA with mild hypokalemia that led to life-threatening ventricular tachycardia.

2. Case report

The case report was approved by the Institutional Review Board of the Hospital of Jilin University. Informed consent has been obtained from the patient. A 74-year-old man was hospitalized in the emergency department 25 hours after suffering from syncope without chest pain. The patient had a history of hypertension for 20 years (highest recorded blood pressure: 180/90 mmHg) and intermittent treatment with oral nifedipine controlled-release tablets. The blood pressure was controlled at 140/70 mmHg. He

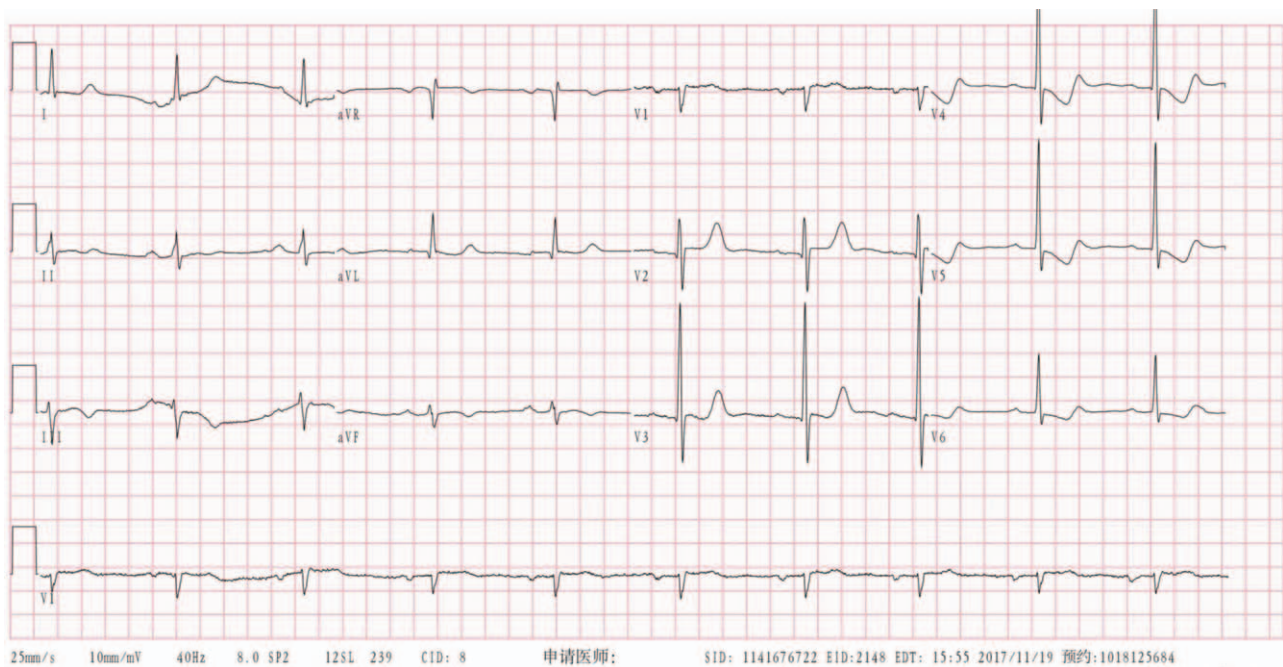


Figure 1. Electrocardiogram showing T-wave inversion in leads II, III, avF, and ST-T changes in leads V4 to V6. QT interval: 462 ms, corrected QT interval: 453 ms.

also had a history of cerebral hemorrhage 25 years ago, right eye glaucoma leading to near blindness 2 years ago, left eye central artery occlusion 2 years ago, and history of smoking for >50 years.

At admission, his blood pressure was 153/90 mmHg. His heart beats were irregular and the electrocardiogram showed ST segment depression and T wave inversion in leads III, avF, V4–V6 (Fig. 1). Mild QT prolongation was observed (QT = 462 ms, corrected QT interval = 453 ms) during sinus rhythm. Laboratory examination revealed mild hypokalemia (K^+ 3.44 mmol/L) and increased B-type natriuretic peptide (271.0 pg/mL). Markers of cardiac injury were normal (CK-MB < 1.0 ng/mL; myoglobin 62.00 ng/mL; troponin I 0.05 ng/mL) (Table 1). Echocardiography indicated a thickened ventricular septum and reduced movement of the inferior wall of the left ventricle. Coronary angiography showed localized stenosis (up to 95%) of the distal portion of right coronary artery (forward blood flow: thrombolysis in myocardial infarction [TIMI] class 3). Subsequently a stent was implanted (Fig. 2A and B). Although intravenous potassium and magnesium were administered on the first day, he sustained 3 episodes of ventricular tachycardia and ventricular fibrillation after admission (Fig. 3). However, it was difficult to maintain his serum potassium concentration >4.5 mmol/L. Twenty-four hour urinary potassium was normal (97.0 mmol/L, reference range: 51–102 mmol/L) in the presence of serum potassium level of 4.44 mmol/L. No recurrence of ventricular tachycardia or ventricular fibrillation occurred. PA was suspected and he was referred for further investigation of the renin-aldosterone axis. Plasma renin activity (0.99 ng/mL/h, reference range: 0.05–0.79 ng/mL/h) and plasma aldosterone level (0.30 ng/mL, reference range: 0.06–0.174 ng/mL) were elevated, and the ratio of plasma aldosterone and renin activity was 30.3. Concentrations of glucocorticoids, serum, and urinary catecholamines (epinephrine, norepinephrine, metanephrines, and vanillylmandelic acid) were within the respective reference range. Abdominal CT showed a nodule (7 × 10 mm) in

the left adrenal gland (Fig. 4). Based on the hormone levels and adrenal CT findings, PA with adrenal nodule was diagnosed. Hypokalemia was corrected by aldosterone receptor antagonist. On urological consultation, follow-up of the patient with CT

Table 1

Results of hematologic and biochemical investigations.

Variable	At admission	Reference range, adults
Hematocrit, L/L	0.433	0.400–0.500
Hemoglobin, g/L	140	130–175
Total leukocyte count, $\times 10^9/L$	8.10	3.50–9.50
Differential leukocyte count (%)		
Neutrophils	0.88	0.40–0.75
Lymphocytes	0.08	0.20–0.50
Monocytes	0.04	0.03–0.10
Eosinophils	0.00	0.004–0.08
Platelet count, $\times 10^9/L$	122	125–350
Mean corpuscular volume, fL	97.3	82–100
Glucose, mmol/L	6.65	4.1–5.9
Sodium, mmol/L	141.7	137–147
Potassium, mmol/L	3.44	3.5–5.3
Blood urea nitrogen, mmol/L	4.10	3.6–9.5
Creatinine, $\mu\text{mol/L}$	98.3	57–111
Calcium, mmol/L	2.11	2.11–2.52
Arterial blood gas		
pH	7.42	7.35–7.45
PCO ₂ , mmHg	42	35–48
PO ₂ , mmHg	71	83–108
HCO ₃ ⁻ , mmol/L	27.2	18–23
Aspartate aminotransferase, U/L	48.5	15–40
Alanine aminotransferase, U/L	66.3	9–50
B-type natriuretic peptide, pg/mL	271	0–100
D-dimer, ng/mL	636	100–600
Myoglobin, ng/mL	62	0–107
Troponin, ng/mL	0.05	0–0.05
Creatine kinase isoenzymes, ng/mL	<1.0	0.0–4.3

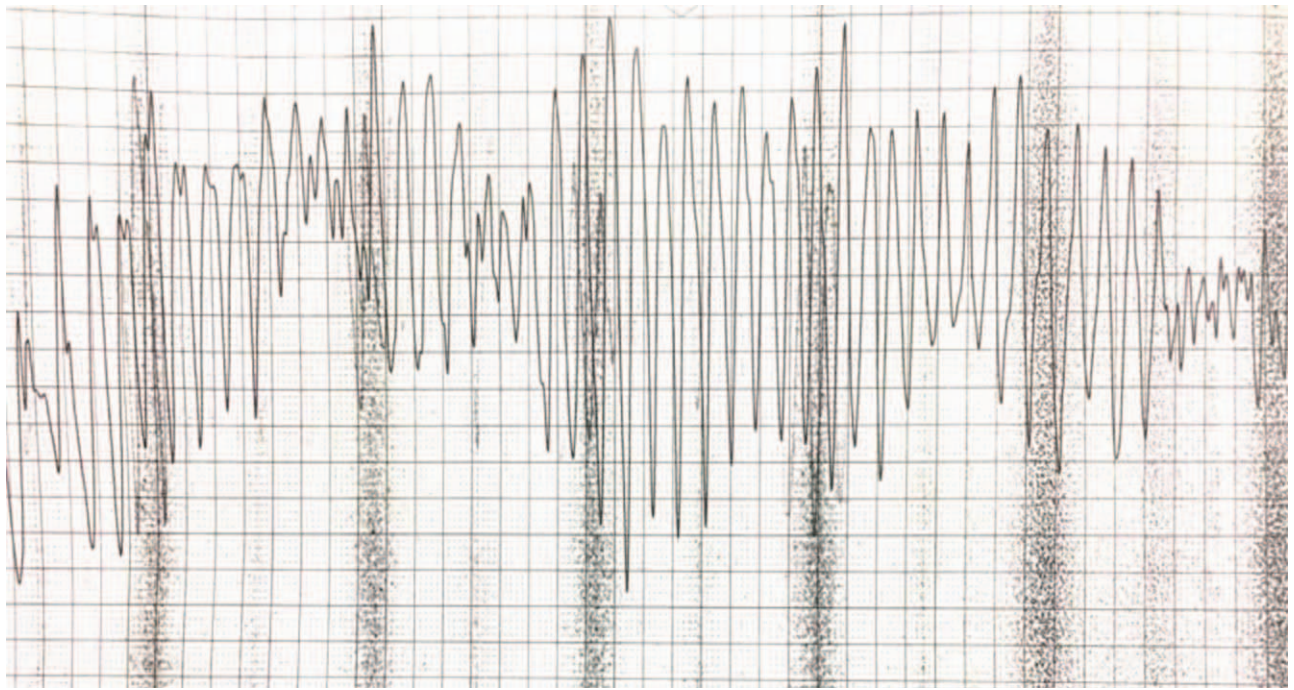


Figure 2. Monitor showing ventricular tachycardia.

scan of adrenal gland was suggested, with removal of adrenal nodules when the adrenal nodule is large $>10 \times 10$ mm. However, the patient refused our treatment with an implantable cardioverter defibrillator for secondary prevention of sudden cardiac death. As of 6 months after hospital discharge, no episodes of syncope or ventricular tachycardia occurred; his serum potassium level is normal and hypertension is well controlled with only aldosterone receptor antagonist.

3. Discussion

Multiple episodes of ventricular tachycardia is a rare presenting feature of patients with PA.^[4] Our patient had 3 episodes of ventricular tachycardia or ventricular fibrillation occurring within a 24-hours period, which was considered as electrical storm.^[5] Our patient had good control of hypertension with mild hypokalemia, mild prolongation of QT interval, and right coronary artery stenosis without chest pain. It is difficult to

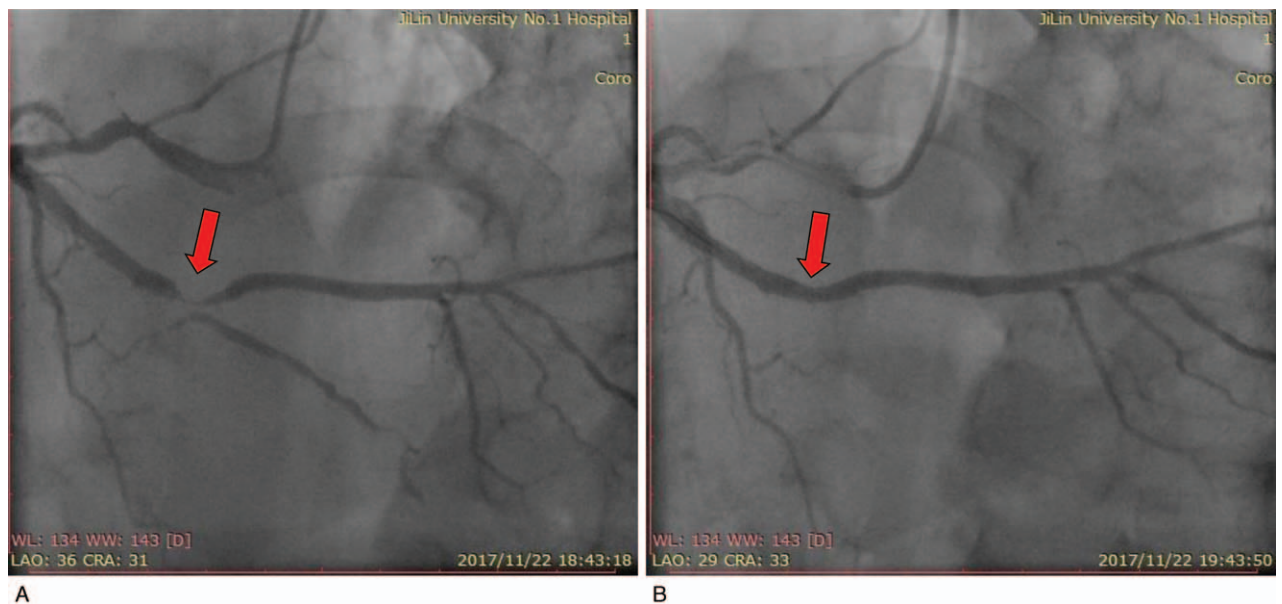


Figure 3. Coronary angiography and stent implantation (A) coronary angiography shows localized stenosis (up to 95%) of the distal portion of the right coronary artery (RCA); the posterior descending branch shows subtotal occlusion. The forward blood flow was TIMI2. The proximal localized stenosis of the left ventricular posterior branch is $>95\%$ and the forward blood flow was TIMI 3 (arrow). (B) RCA was stented (arrow). RCA=right coronary artery; TIMI=thrombolysis in myocardial infarction.

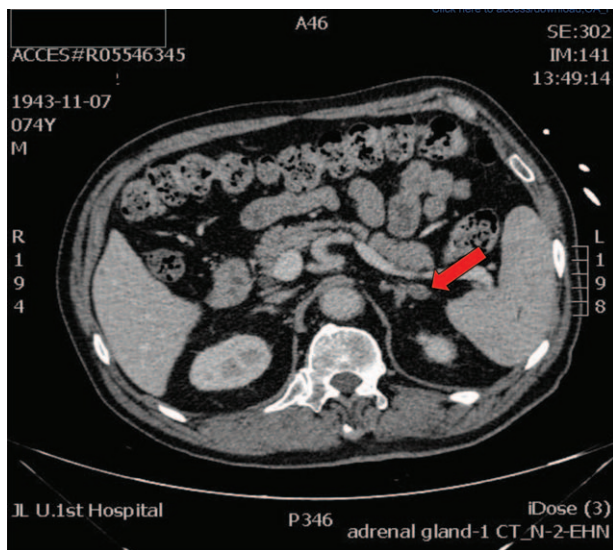


Figure 4. Abdominal computed tomography showing left adrenal adenoma (arrow). A nodular low-density shadow is seen in the left adrenal gland, about 0.7 to 1.0 cm in size (arrow).

identify the main reason of electrical storm. Coronary artery disease (CAD) is one of the most common causes of electrical storm.^[5] Physicians must pay particular attention to identify the possible reason of electrical storm. This case illustrates the importance of ruling out PA during diagnostic workup of patients with hypertension, especially in patients with hypokalemia. The possible reason of life-threatening arrhythmia in our patient was right coronary artery stenosis and mild hypokalemia induced by PA. Since only mild hypokalemia and mild prolongation of QT interval were observed in this patient, the diagnosis of PA was liable to be missed. After diagnosis, our treatment strategy is well corrected hypokalemia by aldosterone receptor antagonist.

A recent meta-analysis of 31 studies found that patients with PA have a significantly higher risk of cardiovascular and cerebrovascular events, target organ damage (left ventricular hypertrophy), metabolic syndrome, and diabetes, as compared with patients with essential hypertension.^[6] A few cases of CAD with concomitant PA have also been reported.^[7,8] However, the mechanism of development of CAD in the setting of PA is not clear. Studies have demonstrated that aldosterone and/or mineralocorticoid receptor activation may induce cardiac oxidative stress and vascular inflammation, which may contribute to increase cardiovascular morbidity and mortality.^[9]

PA also impairs the function of vascular smooth muscle cells which may lead to coronary constriction.^[10] Thus, PA probably induces coronary vasospasm and coronary microvascular vasoconstriction. The combined effects of these possible factors, that is, mild prolongation of QT interval induced by mild hypokalemia, coronary stenosis, and coronary vasospasm may have caused diffuse impairment of cardiac electrical activity

leading to electrical storm. The limitation of this case is that the patient refused an implantable cardioverter defibrillator for secondary prevention of sudden cardiac death. It makes the patient at risk of cardiac arrest at all times.

4. Conclusion

Physicians need to determine the underlying cause of life-threatening arrhythmia, especially in the presence of mild prolongation of QT interval. PA due to adrenal adenoma in patients with hypertension and ischemic heart disease may be a possible cause of electrical storm, and its detection can give them a chance to avoid life-threatening arrhythmia.

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Author contributions

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