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Case Report

A case of primary aldosteronism who experienced cardiopulmonary arrest, was resuscitated and cured

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ABSTRACT

A 45-year-old female went into cardiopulmonary arrest. She was in ventricular fibrillation (VF) and was defibrillated using an automated external defibrillator. After arrival at our hospital, electrocardiography monitoring showed QT prolongation. Serum potassium was low at 2.2 mEq/L, and hypokalemia-induced long QT syndrome was considered to be the cause of this patient's VF. An intravenous infusion of potassium and magnesium sulphate was started, which normalized her serum potassium and QTc interval, with no recurrence of ventricular arrhythmias. Endocrinological investigations showed a plasma renin activity of <0.1 ng/(mL h) and a plasma aldosterone concentration 258 pg/mL. Computed tomography scanning revealed a low signal area 16 mm × 20 mm in size of the right adrenal gland. From the above findings, this patient was diagnosed with a right adrenal tumor and primary aldosteronism. We concluded that the right adrenal tumor was excreting excess amounts of aldosterone from adrenal vein sampling, and performed laparascopic right adrenalectomy. Serum potassium levels rose immediately to normal levels postoperatively. We were able to withdraw her antihypertensive medication 3 months after adrenalectomy. We report a case of primary aldosteronism who experienced cardiopulmonary arrest, was resuscitated, and cured.

<**Learning objective:** When you come across ventricular fibrillation, please consider one of the reasons is caused by hypokalemia due to primary aldosteronism. After an appropriate resuscitation, both hypokalemia and hypertension are completely curable by removing the adrenal tumor.>

Case report

Patient: 45-year-old female.

Chief complaint: cardiopulmonary arrest.

Present history of presenting condition: underwent annual health checks until the age of 37, with no abnormalities identified, after which she did not undergo any health checks. At the age of 44, she was diagnosed with moderate hypertension, with a blood pressure of 170/100 mmHg, and commenced on antihypertensive medication (telmisartan 40 mg daily). On 16 September 2011, she was administered a bowel preparation for colonoscopy, scheduled for investigation of anemia. This resulted immediately in diarrhea and limb weakness, for which she was given intravenous therapy and recovered after several days. Two weeks later, she collapsed suddenly during a meal, holding her chest. She went into cardiopulmonary arrest, for which a bystander administered cardiac massage. When ambulance officers arrived (14 min later), she was in ventricular fibrillation (VF). She was defibrillated using an automated external defibrillator (AED) three times, and a spontaneous rhythm was re-established 27 min after the onset of cardiopulmonary arrest. However, even after arrival at this hospital she remained in an impaired conscious state – 3 points on the Glasgow coma scale (GCS; E1 V1 M1), and was admitted as an emergency patient following intubation to maintain her airway.

Her past history included microcytic anemia since her 20s and as her family history her mother and grandmother were hypertensive. Her social history showed that she was a non-smoker and had taken no alcohol nor herbal supplements. She had irregular periods.

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Findings on admission

Height 160 cm. Weight 43 kg. Consciousness level 3 points on GCS (E1 V1 M1). Blood pressure 147/93 mmHg. Heart rate 63 beats/min, regular. Body temperature 35.8 °C.

Heart sounds S1→, S2→, S3(−), S4(−), no murmurs. Breath sounds no rales.

Blood testing (Table 1): hypokalemia, metabolic acidosis with respiratory compensation.

Chest plain radiograph: cardiothoracic ratio 63%, enlarged left 4th arch.

Electrocardiograph (Fig. 1): normal sinus rhythm, ventricular premature contractions (bigeminy), left ventricular hypertrophy, prolonged QTc interval (0.55 ms).

Echocardiography: normal wall motion, no left ventricular wall thickening.

Head computed tomography (CT) scan: no significant abnormalities seen.

Coronary angiography: no coronary arterial stenosis found.

Table 1
Laboratory findings on admission.

WBC ($\times 10^3/\mu L$)	9.1 (3.5–9)
Hb (g/dL)	10.5 (11.1–15.1)
Plt ($\times 10^4/\mu L$)	31.5 (13.2–36.8)
Alb (g/dL)	4.3 (4–5)
AST (IU/L)	108 (13–33)
ALT (IU/L)	101 (6–30)
LDH (IU/L)	422 (119–229)
CK (IU/L)	121 (45–163)
BUN (mg/dL)	16 (8–22)
Cr (mg/dL)	0.8 (0.4–0.7)
Na (mmol/L)	141 (138–146)
K (mmol/L)	2.2 (3.6–4.9)
Cl (mmol/L)	97 (99–109)
Ca (mg/dL)	9.2 (8.7–10.3)
BS (mg/dL)	303 (69–109)
HbA1c (%)	4.7 (4.3–5.8)
LDL-C (mg/dL)	130 (<140)
UA (mg/dL)	6.6 (2.3–7)
CRP (mg/dL)	0.02 (<0.03)
D-Dimer (μg/mL)	1.4 (<1.0)
Troponin T (ng/mL)	<0.03 (<0.10)
TSH (μIU/mL)	5.10 (0.43–4.83)
FT4 (ng/dL)	1.62 (0.87–1.72)
pH	7.39
pO_2	109.6 mmHg
pCO_2	30.3 mmHg
HCO_3^-	17.7 mmol/L
B.E.	−5.9 mmol/L

Abbreviations: WBC, white blood cell; Hb, hemoglobin; Plt, platelet; Alb, albumin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; CK, creatine kinase; BUN, blood urea nitrogen; Cr, creatinine; Na, natrium; K, potassium; Cl, chlorine; Ca, calcium; BS, blood sugar; HbA1c, hemoglobin A1c; LDL-C, low density lipoprotein-cholesterol; UA, urinary acid; CRP, C-reactive protein; TSH, thyroid-stimulating hormone; FT4, free T4; B.E., base excess.
( ), normal range

Progress

Initially a cardiac condition was suspected, so angiography was performed as soon as the patient's condition was stabilized following resuscitation. No coronary arterial lesions were found, therefore hypokalemia-induced long QT syndrome was considered to be the cause of this patient's VF and cardiopulmonary arrest. An intravenous infusion of potassium and magnesium sulphate was started, which normalized her serum potassium levels and QTc interval, with no recurrence of ventricular arrhythmias. Fortunately, CT scanning showed no evidence of hypoxic encephalopathy, her conscious state gradually improved, and she could be extubated on Day 4 of the admission. She then gradually recovered her activities of daily living, and returned to her pre-arrest state.

Endocrinological investigations were performed to determine the cause of her hypokalemia, from 14 days after cessation of telmisartan (Table 2). The results showed a plasma renin activity (PRA) of <0.1 ng/(mL h) (normal range 0.3–2.9 ng/(mL h)), a plasma aldosterone concentration (PAC) 258 pg/mL (29.9–159 pg/mL), and an aldosterone-to-renin ratio (ARR) ≥ 2580 ng/dL per ng/(mL h), exceeding 200 ng/dL per ng/(mL h). Other adrenocortical and adrenomedullary hormone levels were within normal limits. CT scanning revealed a low signal area 16 mm × 20 mm in size of the right adrenal gland (Fig. 2). Captopril challenge testing revealed an abnormal response, with an ARR ≥ 200 ng/dL per ng/(mL h) 60 (and 90) min after administration of captopril, demonstrating marked suppression of PRA. From the above findings, this patient was diagnosed with a right adrenal tumor and primary aldosteronism. Her blood pressure was well controlled at around 120/80 mmHg with salt restriction (<6 g/day), nifedipine CR 20 mg daily, and

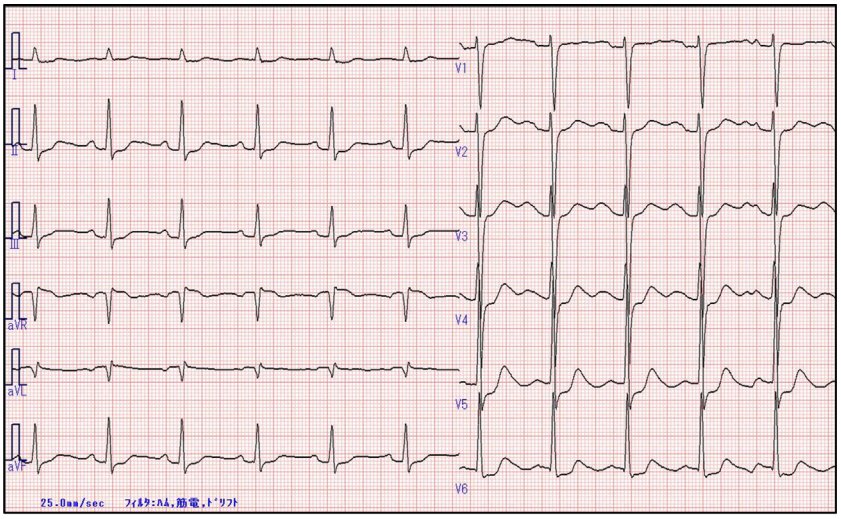

Fig. 1. Electrocardiogram. NSR, normal sinus rhythm; AV, atrioventricular; LVH, left ventricular hypertrophy.