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CASE REPORT

Primary aldosteronism presenting as embolic myocardial infarction

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Abstract

INTRODUCTION: Primary aldosteronism is now recognized as the most common cause of secondary hypertension. Increasing evidence has demonstrated increased cardiovascular events in primary aldosteronism patients. Heart failure and atrial fibrillation are the most common cardiovascular complications occurring in these patients, and a few cases of coronary artery disease have been reported. Herein, we report a rare case of primary aldosteronism in a patient who presented with myocardial infarction associated with coronary embolism.

CASE REPORT: A 52-year-old woman was admitted to our hospital because of chest pain. ST-segment elevation was observed on an electrocardiogram. Although no significant stenosis was observed, embolization of the far distal left anterior descending artery was noticed on angiography. Blood test results revealed hypokalemia and increased aldosterone-renin ratio. Abdominal computed tomography revealed an adenoma in the left adrenal gland. After adrenalectomy, the serum potassium level normalized, and blood pressure was well controlled.

CONCLUSION: Primary aldosteronism must be considered in patients who have had various cardiovascular diseases, including embolisms and situations in which the discrimination of secondary hypertension is necessary

Abbreviations:

AF	- Atrial fibrillation
CAD	- Coronary artery disease
CE	- Coronary embolism
ER	- Emergency room
MI	- Myocardial infarction
PA	- Primary aldosteronism
HTN	- Hypertension
ECG	- Electrocardiogram
ARR	- Aldosterone-renin ratio
STEMI	- ST-segment elevation myocardial infarction

INTRODUCTION

Primary aldosteronism (PA) is a condition resulting from the excessive autonomous production of aldosterone that escapes regulation from angiotensin or plasma potassium concentrations (Meng *et al.* 2020). Such inappropriate production of aldosterone causes hypertension (HTN), sodium retention, plasma renin suppression, and increased potassium excretion that may lead to hypokalemia (Funder *et al.* 2016). PA is one of the most frequent forms of secondary HTN,

with an estimated prevalence ranging between 3.2% and 12.7% in primary care and reaching up to 30% in patients with resistant hypertension (Kayser SC *et al.* 2016). Increasing evidence have also demonstrated an increased cardiovascular risk in patients with PA compared to those with essential HTN (Meng *et al.* 2020). Heart failure and atrial fibrillation (AF) are the most common cardiovascular complications occurring in these patients, and a few cases of coronary artery disease (CAD) have also been reported (Milliez *et al.* 2005; Nishimura *et al.* 1999; Byun *et al.* 2013). Furthermore, the majority of CAD in PA patients is associated with atherosclerosis. Herein, we report a rare case of a PA patient who presented with myocardial infarction (MI) associated with coronary embolism (CE) in the absence of coronary artery atherosclerosis.

CASE REPORT

A 52-year-old woman was transferred to our emergency room (ER) with sudden onset chest pain and an abnormal electrocardiogram (ECG). The pain occurred in the morning and radiated from the chest to the shoulder area. An ECG from another hospital showed ST-segment elevation in the precordial lead (Fig. 1A). She previously had never experienced chest discomfort. She had never smoked and had no cardiovascular risk factors except HTN. She was diagnosed as having HTN 4 years ago and was taking four antihypertensive drugs: amlodipine 10 mg, olmesartan 40 mg, hydrochlorothiazide 12.5 mg, and carvedilol 25 mg. In the ER, her blood pressure was 170/90 mmHg; pulse rate, 62 beats/minute; respiration rate, 18 breaths/minute; and body temperature, 37.4°C. Unlike her prior examination, no ST elevation was observed on the ECG performed in our ER at 3-hour interval; instead, a prominent U wave in the precordial lead and T wave inversion in V3 to V5 were observed (Fig. 1B). Cardiac enzyme test results were as follows: troponin-T, 0.177 (<0.1) ng/mL; creatine-kinase-MB, 23.58 (<0.288) ng/mL; and pro-BNP, 224 (<113) pg/mL. Echocardiography revealed akinesia of the left ventricular apex with a normal ejection fraction (61%). Acute MI was suspected. After intravenous heparin and nitrate applications, high dose antiplatelet

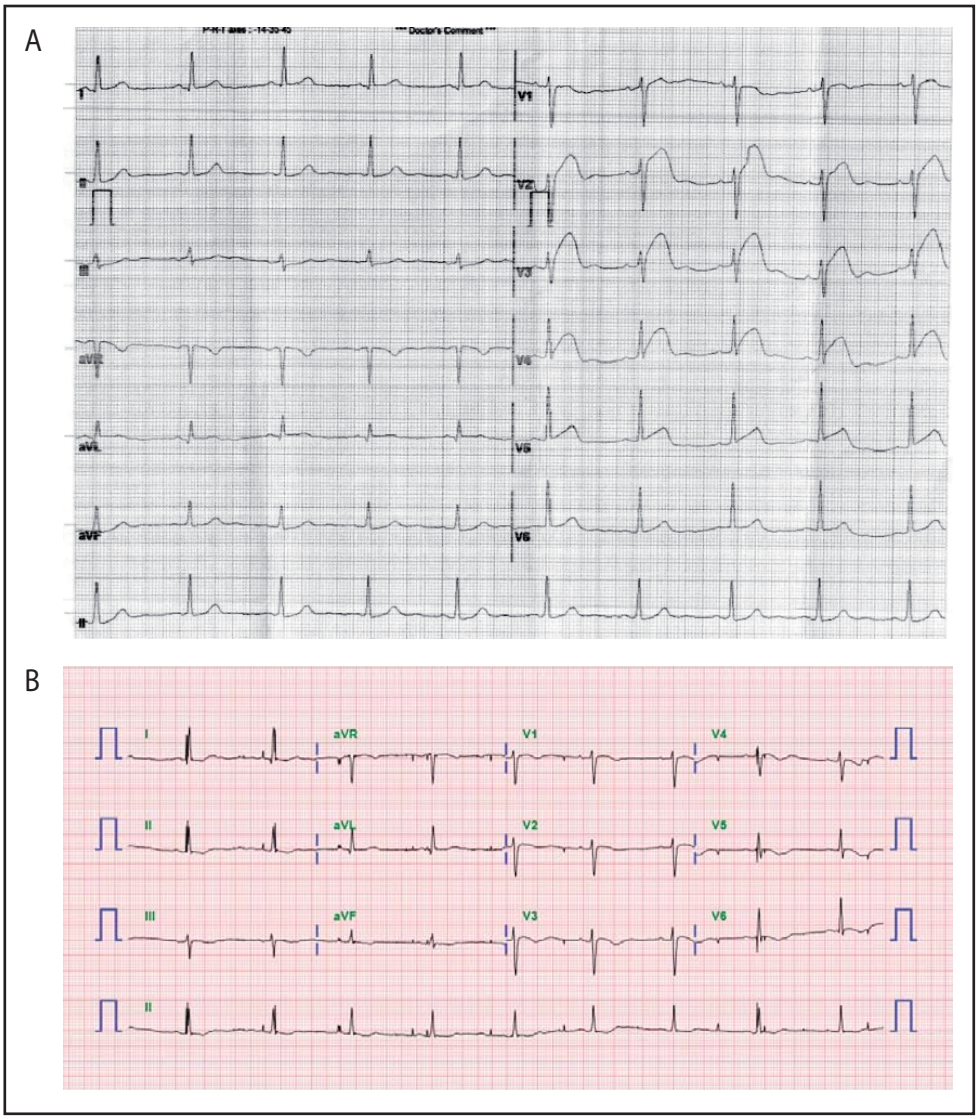

Fig. 1. Electrocardiogram of the patient. (A) Initial ECG showed ST-segment elevation in the precordial lead. (B) three hours later, ECG showed a prominent U wave in the precordial lead and T wave inversion in V3 to V5.

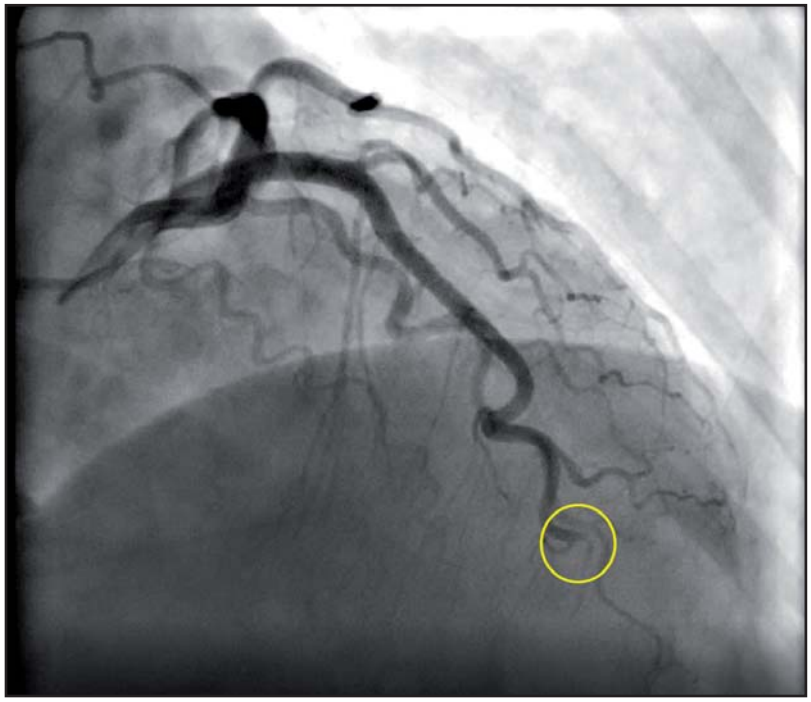

Fig. 2. Coronary angiography showing no significant stenosis, but embolization of the far distal left anterior descending artery is noticed.

drugs (300 mg aspirin and 180 mg ticagrelor) were loaded orally, and subsequently coronary angiography was performed. However, no significant stenosis was noted, although embolization of the far distal left anterior descending artery was observed (Fig. 2). Percutaneous coronary intervention was not performed, therefore intravenous heparin and nitrate were discontinued and medical treatment including aspirin 100 mg, ticagrelor 90 mg, rosuvastatin 10 mg, fimasartan 60 mg, and amlodipine 40 mg was maintained. Blood test results were reviewed, and additional tests were performed to determine the cause of coronary embolization. Because arrhythmia such as AF is a common cause of embolism, Holter monitoring was performed; however, no significant arrhythmia was observed other than atrial premature beats. Moreover, transesophageal echocardiography revealed no evidence of definite thrombi and intra-cardiac problems. The laboratory test results were as follows (shown in Table 1): serum sodium 143 mmol/L; potassium, 2.9 mmol/L; chloride, 101 mmol/L; blood urea nitrogen, 6 mg/dL; creatinine 0.38 mg/dL; arterial blood pH, 7.45; and HCO3, 34.9 mmol/L. Owing to her uncorrected hypokalemia and high blood pressure, endocrinological tests were performed. Her thyroid-stimulating hormone level was 1.22 μU/ mL; free T4, 16.97 ng/dL; plasma renin activity, 0.2 ng/ml/hr; aldosterone, 56.6 ng/dL; aldosterone-renin ratio (ARR), 283 (<30); adrenocorticotropic hormone, 6.22 pg/mL; cortisol, 10.2 μg/dL at 8:00 AM; metanephrine (plasma), 0.15 nmol/L; and normetanephrine, 0.39 nmol/L. PA was strongly suspected because of hypokalemia, elevated ARR, and high aldosterone levels. The confirmatory test could not be performed owing to high blood pressure and uncorrected hypokalemia. Abdominal computed tomography was performed, and a mass measuring approximately 1.8 × 1.5 cm in size was detected in the left adrenal gland (Fig. 3A).

After 4 months, when it was possible to discontinue the antiplatelet agents, laparoscopic adrenalectomy was performed. Histopathological findings were consistent with adrenocortical adenoma (Fig. 3B-C). After surgery, her serum potassium level normalized, and her aldosterone level was 7.2 ng/dL. Her blood pressure was well controlled by taking 40 mg of telmisartan only once a day.

DISCUSSION

Here, we described an unusual case of a patient with PA who presented with MI associated with CE in the absence of coronary artery atherosclerosis and AF. To the best of our knowledge, this is the first case report of a PA patient presenting ST-segment elevation MI (STEMI) associated with CE. Although PA is a common cause of secondary hypertension, diagnosis is often delayed because of lack of any specific symptoms. This patient also had no symptoms during the period of taking blood pressure medication for 4 years before experiencing sudden chest pain. Fortunately, the observations of the presence of hypertension and hypokalemia assisted in timely diagnosis of PA. Since only a small percentage of PA patients (9–37%) have hypokalemia, it is very difficult to diagnose it in patients without hypokalemia unless associated with high index of suspicion (Funder *et al.* 2016).

Increasing evidence have demonstrated an increased cardiovascular risk in patients with PA compared with

Tab. 1. Laboratory test results of the patient

	Lab test	Results	Reference (unit)
Serum	Sodium	143	135-150 mmol/L
	Potassium	2.9	3.5-5.5 mmol/L
	Chloride	101	91-110 mmol/L
	BUN	6	8-23 mg/dL
	Creatinine	0.38	0.7-1.7 mg/dL
ABGA	pH	7.45	7.35-7.45
	HCO3	34.9	22-26 mmol/L
Endocrinological test	TSH	1.22	0.55-4.78 μU/ mL
	Free T4	16.97	11.5-22.7 ng/dL
	PRA	0.2	0.32-1.84 ng/ml/hr
	Aldosterone	56.6	4.2-20.9 ng/dL
	ARR	283	< 30
	Cortisol (morning)	10.2	> 5 μg/dL
	ACTH	6.22	5-60 pg/mL
	Plasma metanephrine	0.15	< 0.50 nmol/L
	Plasm normetanephrine	0.39	< 0.90 nmol/L

BUN: Blood urea nitrogen; TSH: thyroid-stimulating hormone; PRA: plasma renin activity; ARR: aldosterone-renin ratio; ACTH: adrenocorticotropic hormone

those with essential HTN, and it is thought that aldosterone causes organ damage, irrespective of their blood pressure (Milliez *et al.* 2005). This includes myocardial inflammation, excessive collagen deposition, fibrosis in myocardial tissue, vascular remodeling, and endothelial dysfunction (Higuchi *et al.* 2021; Lim *et al.* 2016; Monticone *et al.* 2018). Clinically, heart failure and AF are common, and the prevalence of atherosclerotic diseases such as stroke and CAD is also high (Milliez *et al.* 2005; Nishimura *et al.* 1999). Most cases of MI in PA patients are also associated with atherosclerotic CAD (Milliez *et al.* 2005; Byun *et al.* 2013).

Approximately 90% of MI patients have angiographic evidence of obstructive atherosclerotic CAD based on the registry studies (Gue *et al.* 2020). Approximately 10% of patients presenting MI are associated with non-obstructive coronary arteries such as vasospasm, thromboembolism, coronary dissection, and non-coronary causes such as myocarditis and stress cardiomyopathy. The prevalence of CE as a mechanism of STEMI is 3% according to previous studies (Gue *et al.* 2020; Popovic *et al.* 2018). CE has been associated with various clinical conditions such as valvular heart diseases, AF, endocarditis, intra-cardiac tumor, and diseases associated with the hypercoagulable condition and an increased risk of thromboembolism (Popovic *et al.* 2018). In this respect, PA may also be a causative condition. Excessive aldosterone causes endothelial dysfunction, vascular inflammation, and interstitial fibrosis of various organs, including the heart. This leads to atrial structural remodeling and in combination with AF, it increases the risk of thromboembolism (Pan *et al.* 2020). In this case study, AF was not noted on Holter monitoring, but paroxysmal AF events cannot be ruled out.

In conclusion, PA is often overlooked despite its high prevalence in clinical practice. However, it should be considered in patients who had various cardiovascular diseases, including embolisms, and situations under which discrimination from secondary hypertension is necessary.

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None.

DISCLOSURE

All authors have no potential conflicts of interest.

ETHICS STATEMENT

The present study protocol was reviewed and approved by the Institutional Review Board of the Jeonbuk National University Hospital (approval No. 2021-01-028). Informed consent was waived because of the retrospective nature of the study.

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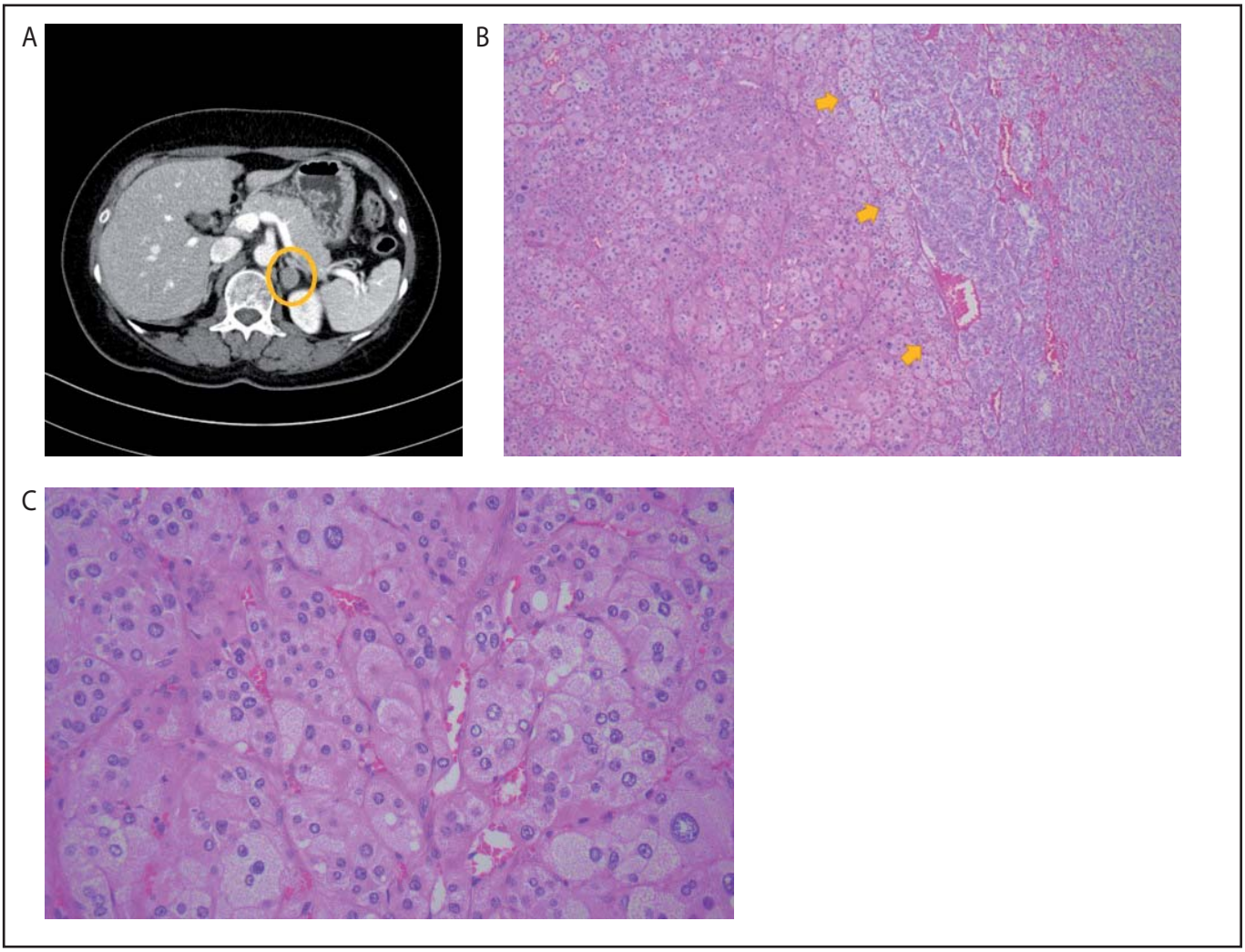

Fig. 3. A) Abdominal computed tomography showing an adrenal adenoma of the left adrenal gland measuring 1.8 × 1.5 cm in size. (B, C) Histopathology of an adrenal mass. This low-power view of an aldosterone-producing adenoma highlights normal compressed adrenal that is separated from the tumor (B, H&E, ×100). This high magnification of aldosterone-producing tumor cells arranged in a nesting pattern with moderate pleomorphism in the cell size (C, H&E, ×400).

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