

Renal artery intervention for a patient with flash pulmonary edema accompanied by elevation of troponin levels due to bilateral renal artery stenosis and multivessel coronary disease: a case report

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Abstract

An 84-year-old woman complaining of acute-onset chest distress for 2 hours was referred to the Department of Cardiology, Guangzhou Red Cross Hospital, China. A physical examination showed signs of acute pulmonary edema with considerably elevated blood pressure of 186/120 mmHg. An electrocardiogram showed ST segment depression in leads I, II, and III, and from V4 to V6. A laboratory test showed markedly elevated creatine, high-sensitivity cardiac troponin T, and N-terminal pro-brain natriuretic peptide levels. Echocardiography showed a mildly enlarged left ventricle with an ejection fraction of 43%. The patient was diagnosed with acute coronary syndrome, non-ST segment elevation myocardial infarction, and Killip 3 grade heart function. The non-ST segment elevation myocardial infarction Global Registry of Acute Coronary Events score was 156. Emergency coronary angiography showed severe three-vessel disease with a global ejection fraction of 50% based on left ventricular angiography. Selective renal artery angiography was performed and major stenosis at the ostia in both renal arteries was found. We did not touch the coronary artery, but performed intervention of the renal artery by implanting two bare metal stents in both ostia of bilateral renal arteries. An unexpected clinical benefit was obtained.

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Keywords

Renal artery stenosis (RAS), flash pulmonary edema, acute coronary syndrome, ostium, ST segment depression, troponin

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Introduction

Refractory hypertension and chronic kidney failure are classic manifestations of bilateral renal artery stenosis (RAS). Acute heart failure, especially that unexplained by coronary artery disease or any other organic heart disease, is another clinical presentation of RAS that has been described as “flash pulmonary edema.” The morbidity rate of acute heart failure is approximately 12% in patients with RAS. In the older population, atherosclerosis plays a predominant role in the course of RAS, and atherosclerotic RAS is not as rare as previously found.² We report an older patient who presented with refractory hypertension and severe heart failure, even with signs of acute coronary syndrome. These symptoms could not be explained by coronary artery disease, and her heart function suggested narrowing of the renal artery.

Case report

An 84-year-old woman was admitted to the Department of Cardiology at Guangzhou Red Cross Hospital because of acute onset of chest distress and shortness of breath. The main findings of a physical examination were orthopnea with jugular varicosity, moist rales over both lung bases, edema in both lower limbs, and sinus tachycardia, with a heart rate of 110 beats/minute. Her blood pressure was 186/120 mmHg on admission. She was diagnosed with hypertension and coronary heart disease 14 years previously and was taking angiotensin-converting enzyme inhibitors, β -blockers,

calcium channel blockers, and aspirin. Her blood pressure was still refractory under the above-mentioned treatment. She had a history of cerebral infarction.

An electrocardiogram showed ST segment depression (0.1–0.2 mV) in leads I, II, and III, and from V4 to V6 (Figure 1). Laboratory test results showed elevated creatine (98.0 $\mu\text{mol/L}$), high-sensitivity cardiac troponin (0.550 $\mu\text{g/L}$), and N-terminal pro-brain natriuretic peptide (15,260 pg/mL) levels. An emergency echocardiography showed mild systolic dysfunction (left ventricular ejection fraction: 43%), left ventricular hypertrophy, left atrial amplification, regional wall motion abnormality at the basal segment of the ventricular septum and inferior segment, and mild mitral and aortic regurgitation.

The primary diagnosis was non-ST segment elevation myocardial infarction with a Global Registry of Acute Coronary Events score of 156 and grade 3 Killip heart function. Emergency coronary angiography from right radial artery access by using 6F JL4 and a JR4 catheters (Vista Brite Tip; Cordis, Santa Clara, CA, USA) showed three-vessel disease with total occlusion of the right coronary artery, focal stenosis at the distal and proximal regions of the left circumflex coronary artery, and long stenosis at the proximal segment of the left anterior descending coronary artery (Figure 2). We also found a global ejection fraction of 50% based on left ventricular angiography. We did not perform coronary angioplasty because systolic function of the heart was essentially normal, as evaluated by fluoroscopy. Additionally, blood flow of the left

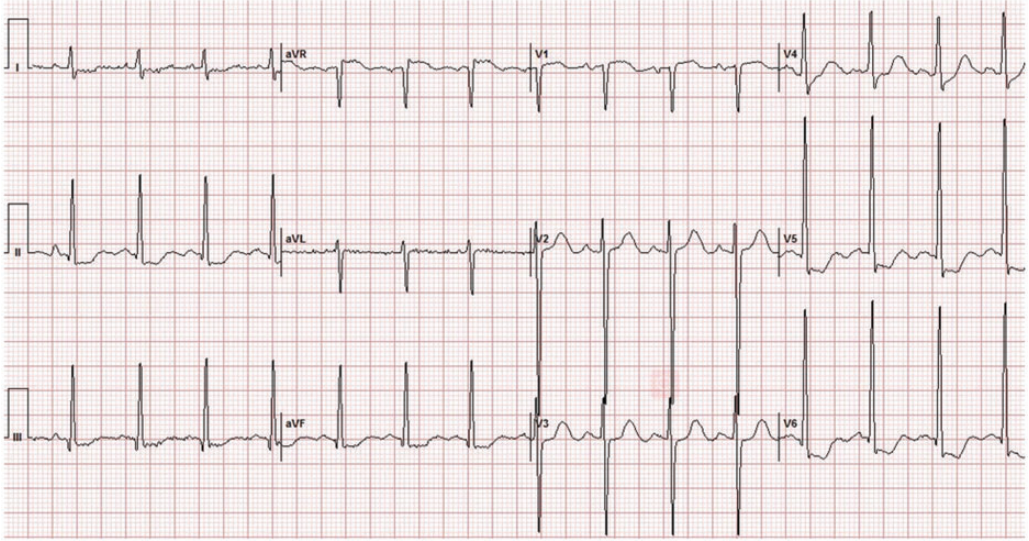


Figure 1. An electrocardiogram at admission shows ST segment depression (0.1–0.2 mV) in leads I, II, and III, and from V4 to V6.

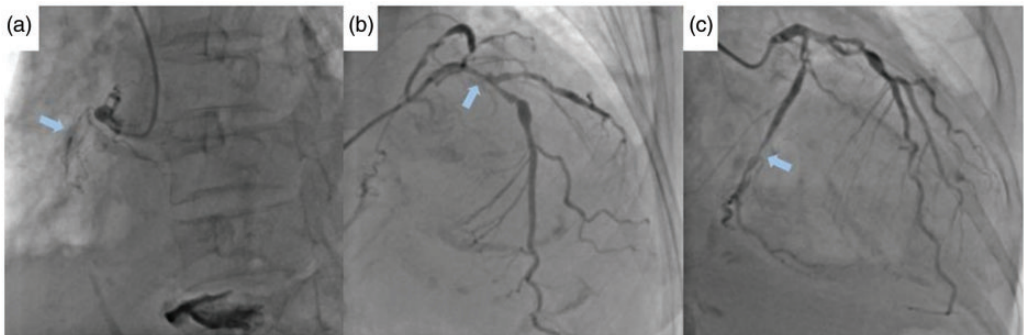


Figure 2. (a) Coronary angiography shows proximal total occlusion of the right coronary artery (blue arrow). (b) Narrowing by 80% in the left anterior descending coronary artery (blue arrow) can be seen. (c) Narrowing by 90% in the left circumflex coronary artery (blue arrow) can be seen.

anterior descending coronary artery, which had the greatest effect on cardiac function, was normal. These findings of a coronary artery lesion, left ventricular size, and contractive function were not consistent with her clinical features. Renal angiography was then performed from right femoral artery access to rule out the presence of renal artery stenosis by using a 6F JR4

angiographic catheter. Unsurprisingly, 95% stenosis at the ostium of the left renal artery was found and 80% stenosis of the right renal artery was observed from the right brachial artery access. The catheter could not enter the ostium of the right renal artery because of her severe tortuous abdominal artery (Figures 3, 4).

A 6F JR4 guiding catheter was then inserted into the right renal artery via right branchial access. We advanced a BMW guide wire (Balance Middleweight; Abbott Vascular, Santa Clara, CA, USA) into the distal right renal artery. Along the wire, a bare metal renal stent of 6.0×15 mm (Invatec; Medtronic, Minneapolis, MN, USA) was implanted at 10 atm after predilation with a coronary non-compliant balloon of 4.5×15 mm

(NC Trek; Abbott Vascular, Santa Clara, CA, USA) at 12 to 18 atm (Figure 3). Another bare metal stent of 6.0×15 mm was implanted at the ostium of the left renal artery with the same interventional procedure (Figure 4). Both stents expanded sufficiently without residual stenosis. A total of 5000 IU heparin was administered throughout the entire process.

The patient's blood pressure decreased from 186/120 mmHg on admission to

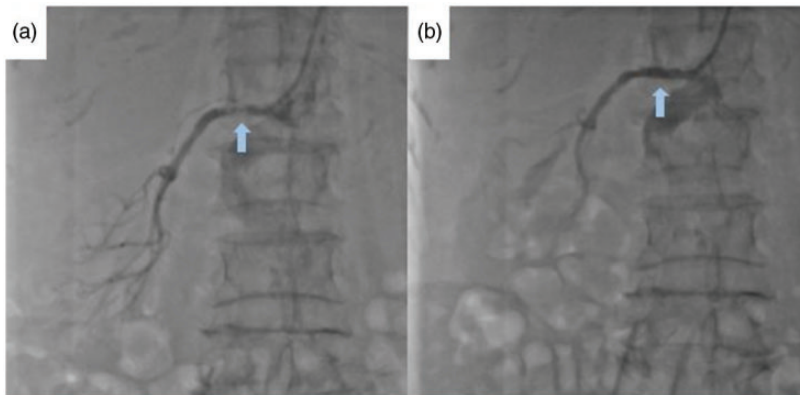


Figure 3. (a) Renal angiography shows 80% stenosis of the right renal artery (blue arrow). (b) A bare metal renal stent (6.0×15 mm) was implanted without residual stenosis after predilation of the narrowed area with a coronary balloon (blue arrow).

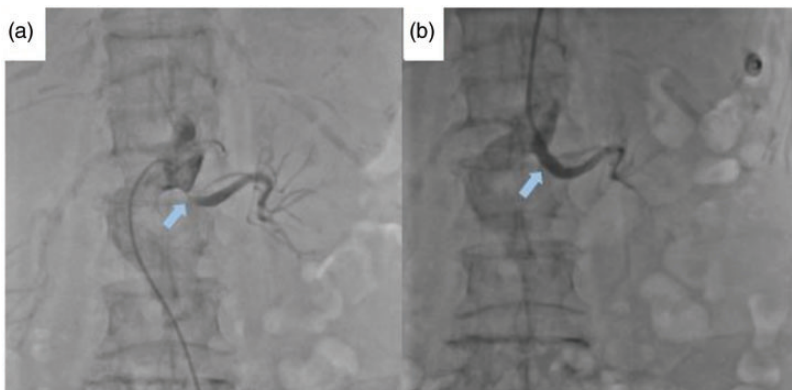


Figure 4. (a) Renal angiography shows 95% stenosis of the left renal artery (blue arrow). (b) A bare metal renal stent (6.0×15 mm) was implanted after predilation of the narrowed area with a coronary balloon (blue arrow).

120 to 140/70 to 80 mmHg immediately after the procedure, and the patient's symptoms were greatly reduced after stent implantation. On the third day after renal artery angioplasty, her creatine level dropped to a normal level, the high-sensitivity cardiac troponin level decreased from 0.550 ug/L to 0.171 ug/L, the N-terminal pro-brain natriuretic peptide level decreased from 15,260 pg/mL to 2078 pg/mL, and echocardiography showed normal systolic function (left ventricular ejection fraction: 56%). The depressed ST segment recovered almost completely with a sinus heart rate of 65 to 70 beats/minute (Figure 5). The patient was discharged 5 days later and was prescribed two antihypertensive drugs (β -blockers and calcium channel blockers), dual antiplatelet therapy (aspirin and clopidogrel), and statins. During follow-up, the patient maintained good exertional tolerance and had well-controlled blood pressure.

This study was approved by the Ethics Committee of Guangzhou Red Cross Hospital (approval number: 2019-233-01).

Written informed consent for publication was obtained from the patient.

Discussion

In this case report, we present an older female patient with severe ostial stenosis at bilateral renal arteries. She had frequently experienced heart failure in the past and was hospitalized for acute coronary syndrome accompanied by acute pulmonary edema, but had normal cardiac contractive function. Eventually, she underwent stent implantation in both renal arteries without touching the coronary artery, and the procedure was successful.

RAS is a disease of the large extra-renal arterial vessels and is caused by many factors. Atherosclerotic obstruction is the most frequent cause of RAS among older people. Previous studies have shown a strong relationship between the extent of coronary artery disease and the presence of RAS in elective consecutive patients (the number of coronary arteries involved is related to the prevalence of RAS).³ Another study

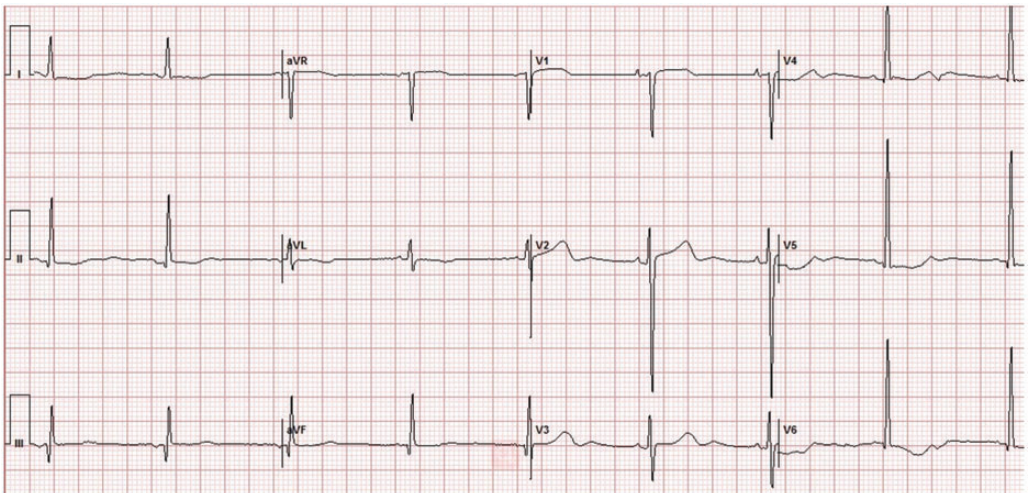


Figure 5. An electrocardiogram on the third day after renal artery angioplasty shows that the depressed ST segment has almost completely recovered.

showed an almost two times higher risk of recurrence of RAS after 1 year in patients with a previous history of an atherothrombotic event and multisite disease (vs. a single disease location).⁴ Furthermore, a systematic review published in 2009 described a significant increase in the prevalence of RAS with the number of coronary arteries involved (5.5% for one-vessel, 9.7% for two-vessel, and 15.1% for three-vessel disease, $p < 0.0001$).⁵ Recent studies showed that the prevalence of RAS in individuals with coronary artery stenosis was 14% to 29%, and this prevalence was 10% in individuals with normal coronary arteries.^{6,7} Additionally, the prevalence of atherosclerotic RAS increases with age, especially in patients with diabetes, hyperlipidemia, diffuse types of peripheral occlusive disease, and hyperlipidemia.^{7,8} A previous study showed that the frequency of RAS increased in proportion with the number of stenotic coronary arteries, and the incidence of RAS was 10%, 15.8%, and 18.1% in patients with single-vessel, two-vessel, and three-vessel coronary heart disease, respectively.⁹ Therefore, three-vessel coronary disease is a powerful and unique predictor of RAS, which is one reason for us choosing to perform renal arteriography immediately in our case.

Recent randomized, controlled trials reported that a reduction in New York Heart Association class was significantly greater in patients undergoing angioplasty than in patients who merely received medical therapy for RAS.¹⁰ Kane et al.¹⁰ also found a five-fold reduction in heart failure hospitalizations in 50 patients who were revascularized versus 50 sex-matched medically managed controls with heart failure and RAS. Another study showed that left ventricular hypertrophy on cardiac magnetic resonance imaging could be reduced after revascularization, which occurred along with a reduction in circulating angiotensin II levels.¹¹ Additionally, the advantageous

effects of revascularization of RAS as a therapy for heart failure was recently confirmed in a large observational study.¹² Distinguishing whether the renal artery or coronary artery is the culprit pathogenic factor for a certain clinical event is always a difficult, but critical, issue. Clinical manifestations that are strongly associated with RAS include refractory hypertension, unexplained elevation of serum creatinine levels, and acute heart failure, especially in those who do not conform to underlying heart disease. In our case, in addition to the characteristics mentioned above, considerable hypertension during pulmonary edema onset was another feature of RAS. In the current case, targeting RAS in the intervention rather than the coronary artery was an appropriate decision.

In conclusion, selective patients who present with multivessel coronary disease, refractory hypertension, unexplained heart failure, and renal dysfunction, even those with acute coronary syndrome, would benefit from RAS intervention rather than coronary artery intervention.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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