CASE REPORT

Development of chronic heart failure in a young woman with hypertension associated with renal artery stenosis with preserved renal function

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SUMMARY

A 33-year-old woman with presumed essential hypertension and symptoms equivalent to New York Heart Association class II was suspected of heart failure and referred to echocardiography. The patient's ECG showed a left bundle branch block. Electrolytes, serum creatinine and estimated-glomerular filtration rate as well as urine test for protein were all normal. The patient had no peripheral oedema. The transthoracic echocardiography confirmed systolic and diastolic dysfunction and an ejection fraction of 25% and left ventricular hypertrophy. Ultrasound of renal arteries and renal CT angiography (renal CTA) revealed a significant stenosis and an aneurysm corresponding to the right renal artery with challenges to traditional interventions.

BACKGROUND

Renal artery stenosis is an uncommon but important cause of hypertension and early diagnosis and treatment are important to prevent organ damage. The American College of Cardiology Foundation/American Heart Association guidelines for management of patients with peripheral artery disease list a variety of clinical clues that can lead to the diagnosis of renal artery stenosis—among these are: onset of hypertension before the age of 30 years; accelerated, resistant or malignant hypertension; sudden, unexplained pulmonary oedema; and unexplained congestive heart failure.¹ Chronic heart failure is a very rare complication. Chronic heart failure, however, was the clue that helped in diagnosing this patient. By writing this case report the link between chronic heart failure and renal artery stenosis, as well as the importance of optimal treatment of hypertension in young participants will be brought to attention.

CASE PRESENTATION

A 33-year-old woman presenting with exertional dyspnoea was referred by her general practitioner to echocardiography due to suspicion of heart failure. The ECG showed sinus rhythm with a left bundle branch block.

Fourteen years earlier the patient was diagnosed with having essential hypertension with a blood pressure of 170/105 with no evident hereditary predisposition. Echocardiography showed normal left ventricular ejection fraction (LVEF) and the septum measured 11 mm. The isotope renography showed no signs of unilateral renal disease at that time; accordingly, it was assessed that a

renovascular cause was unlikely. No further tests were performed and the patient started receiving antihypertensive treatment consisting of ramipril 2.5 mg×2, which reduced the blood pressure to 135/80. The ECG subsequently showed left bundle branch block. The patient received ramipril until 2007, but her antihypertensive treatment was changed to methyldopa 250 mg×3 during her two successive pregnancies in 2007 and 2009. In 2007, home blood pressure under treatment was 140–150/70–80 and in 2009 home blood pressure was 150–160/85 under treatment. No complications occurred during or after the two pregnancies, particularly no pre-eclampsia. At the time of referral due to heart failure symptoms, the patient had not received any antihypertensive treatment for the last 4 years.

At the first visit to the heart failure clinic the patient presented with exertional dyspnoea equivalent to NYHA class II (New York Heart Association classification system of symptom severity I–IV), fatigue and dizziness. She had no orthopnoea or angina pectoris and no signs of clinical decompensation (ie, no peripheral oedema and no jugular vein congestion). Her blood pressure was 152/107 mm Hg and pulse was 64 bpm. Blood samples including serum creatinine and estimated-glomerular filtration rate (eGFR) were normal and there was no trace of protein in the urine sample. Echocardiography showed systolic and diastolic dysfunction, LVEF of 25% and a sign of left ventricular hypertrophy (interventricular septal diameter 14 mm, LV posterior wall diameter 12 mm; [figure 1](#)). Thus, the patient still had unexplained hypertension and newly diagnosed chronic heart failure, without any familial predisposition, at the young age of 33 years. Further investigations were necessary due to the suspicion of a secondary cause for hypertension and/or heart failure.

INVESTIGATIONS

In order to exclude coronary artery disease, especially congenital coronary artery disease and aortic disease, a coronary CT angiography (CTA) including the aorta was performed. The coronary CTA scan showed a single small non-obstructive calcified plaque in the left anterior descending artery. Otherwise, no sign of coarctation of the aorta or other abnormalities was found.

For detection of a possible renal artery stenosis, an ultrasound of renal arteries was performed. The ultrasound showed a considerable renal artery

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Figure 1 Echocardiography. (A) Four-chamber image. (B) Left ventricle.

stenosis with 70% occlusion on the right side. Therefore, a renal CTA was performed showing an aneurysm 1.7 cm in diameter on the right renal artery surrounding a significant arterial

Figure 2 Red arrow: aneurysm on the right renal artery. White arrow: departure of the right renal artery from the abdominal aorta.

stenosis (figures 2 and 3). The stenotic renal artery provided two-thirds of the blood supply to the right kidney. Isotope renography demonstrated that the right kidney provided 39% of global renal function. A captopril test showed no significant change in the renal function of the right kidney compared with the left. The right kidney provided 37% of the global renal function. The eGFR of the right kidney was estimated at 40 mL/min without captopril and 25 mL/min with captopril while the eGFR of the left kidney was 63 mL/min with and 43 mL/min without captopril, respectively. Hence, treatment of the hypertension with ACE inhibitor was still possible.

DIFFERENTIAL DIAGNOSIS

In this case, chronic heart failure could also be due to peripartum, inflammatory cardiomyopathy or myocardial dysfunction due to pre-eclampsia. However, the patient did not at any point suffer from pre-eclampsia nor did she have any symptoms of heart failure prior to the symptoms that led her to the heart failure clinic in 2013. Additionally, the slight septal hypertrophy already seen in 1999 at the first echocardiography was deteriorated at the examination in 2013, indicating increased pressure and remodelling as in hypertensive cardiac disease.

TREATMENT

Initially, medical treatment was chosen due to the relatively small size of the aneurysm and the fact that hypertension can be treated adequately with medication.¹ The patient was treated for heart failure and hypertension with an ACE inhibitor (enalapril 10 mg×2), a β -blocker (carvedilol 6.25 mg×2) and a calcium antagonist (amlodipin 10 mg×1). Owing to the nature of the anatomical challenges, it has been assessed that a percutaneous renal artery revascularisation is technically not possible, but a surgical renovascular bypass to restore blood flow and resect the aneurysm would be the appropriate treatment.

OUTCOME AND FOLLOW-UP

LVEF after optimal medical treatment was improved and estimated at 35–40%. Mean blood pressure during 24 h blood pressure monitoring was 130/77. The patient is now less tired, her dyspnoea has improved and she has resumed jogging and cross-fit training.

Figure 3 Red arrow: aneurysm on the right renal artery. White arrow: stenosis of the right renal artery.

DISCUSSION

Renal artery stenosis is a common and progressive disease in patients with atherosclerosis but a relatively uncommon cause of hypertension. In young individuals who seldom develop hypertension, renal artery stenosis is a more prevalent cause of hypertension. Therefore, appropriate diagnostic studies to identify clinically significant renal artery stenosis are indicated in patients with early onset of hypertension before the age of 30 years.¹ Nevertheless, it remains unclear whether atherosclerotic renovascular disease defined as renal artery stenosis >50% might be associated with worsening of cardiac function. Emans *et al*² found that renal artery stenosis is more prevalent in patients with combined chronic heart failure and chronic kidney disease, but it is not correlated with worsening in LVEF, left ventricular volumes and mass or myocardial fibrosis. In a study by de Silva *et al*³ renal artery stenosis was associated with an increase in morbidity and mortality in patients with chronic heart failure. All studies were based on older individuals. In our case the renal artery aneurismal changes may be attributed to fibromuscular dysplasia.

Cases of cardiac dysfunction associated with untreated hypertension have previously been reported, particularly in the era before effective drug therapy, and cardiac dysfunction was at that time a major cause of morbidity and mortality.^{4 5} These

cases describe symptoms of heart failure due to malignant hypertension. Currently, because of effective drug therapy, heart failure due to hypertension is less frequently observed. Nevertheless, it is still important to take into account that chronic heart failure seldom can occur due to untreated hypertension in younger patients.

In this case report, the patient had developed symptomatic chronic heart failure with reduced LVEF as well as diastolic dysfunction and left ventricular hypertrophy without signs of coronary artery disease and with preserved renal function. Most likely the patient developed heart failure on the basis of inadequately treated hypertension due to the delayed diagnosis of renal artery stenosis.

Contributors CB was involved in concept, draft preparation, figures, patient contact and submission. JA contributed to the concept, draft preparation, general and linguistic review.

Competing interests None.

Patient consent Obtained.

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Learning points

- ▶ Chronic heart failure combined with renal artery stenosis is seen in older individuals as well as in younger patients with preserved renal function.
- ▶ Newly diagnosed chronic heart failure in a young patient can be a clinical clue to the diagnosis of renal artery stenosis.
- ▶ Early diagnosis of the cause of hypertension is important to avoid potential complications such as acute congenital heart failure and also to avoid development of chronic heart failure.

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