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Proximal Renal Artery Stenosis Presenting as Uncontrolled Hypertension

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Abstract

Presentation
We present a case of 50-year-old lady who present with 1-month history of uncontrolled hypertension.

Diagnosis
Magnetic resonance angiography showed stenosis of the left renal artery just beyond the ostium extending over approximately 7mm in length. Computed tomography angiography show focal narrowing of the proximal renal artery just distal to vessel origin, approximately 60% stenosis.

Treatment
Anti-hypertensive medication was initiated in the ward. She was referred to vascular surgeon for renal bypass.

Discussion
Renal artery stenosis is common cause of hypertension but may go unrecognised. The focus of this case is on the evaluation and necessity for a complete evaluation of the patient who is presenting with uncontrolled hypertension. To rule out renal artery stenosis, patient should be examined using CT-angiography or, if possible, arteriography.

Introduction

Renal artery stenosis (RAS) is often associated with hypertension and ischemic nephropathy. A majority of renovascular lesions are attributed to atherosclerosis. Renovascular

hypertension secondary to renal artery stenosis is a frequent curable aetiology of secondary hypertension[1]. In this report we describe a lady with 1-month history of uncontrolled hypertension (HTN).

Case Report

A 50-year-old lady was referred by GP to the emergency department (ED) with 1-month history of high blood pressure. She was monitored by ABPM which the blood pressure was discovered to be 228/122. She was admitted for evaluation and management of her uncontrolled HTN. Prior to presentation she was not taking any antihypertensive medication and her past medical history include osteoarthritis and left knee arthroscopy.

On admission, her BP was 243/133 mmHg, while chest and cardiac examinations revealed no abnormalities or oedema. Neurological examination revealed a normal tone, power, and reflexes, while an ophthalmic examination was normal. Her electrocardiogram (ECG) show sinus rhythm. In ED, she was given a stat dose of amlodipine 5mg. The GP has prescribe her earlier Lercanidipine 10mg which was later on continued. Furthermore, her BP was monitored hourly which still remained high throughout the day. Patient was later on started on Nebivolol 5mg tice daily. Consequently, she received another dose of amlodipine on the admission day, however her BP readings remained high throughout the day.

Laboratory blood tests revealed normal haemoglobin (16.5 g/dL) and MCV levels (89.4 fL). Her liver function test and renal profile were normal. Her N-terminal pro B-type natriuretic peptide (NT-proBNP) was 160 ng/L and urine catecholamines and metabolites were normal. The level of aldosterone was 262 pmol/L (normal range 122-1179 pmol/L) and plasma renin and aldosterone levels were normal. Plasma normetanephrine, metanephrine and 3-methoxytyramine were normal which suggest no presence of pheochromocytoma.

To determine the cause of uncontrolled HTN, she underwent several imaging procedures including renal computed tomography angiography (CTA) and magnetic resonance angiography (MRA), renal ultrasonography (US) and echocardiography (ECHO). Renal US revealed both kidneys were normal cortical medullary differentiation with no evidence of hydronephrosis or scarring. The MRA revealed stenosis of the proximal left renal artery and renal CTA showed focal narrowing of the proximal left renal artery distal to vessel origin, approximately 60% stenosis (Figure 1). Echocardiogram revealed an EF of 55% with no valvular abnormality. Patient BP was optimised with lercanidipine (20mg OD), nebivolol (5mg OD), ramipril (10mg OD), hydrochlorothiazide (12.5mg OD), aspirin (75mg OD) and ezetimibe (10mg OD). She was referred to vascular surgeon for renal artery bypass.

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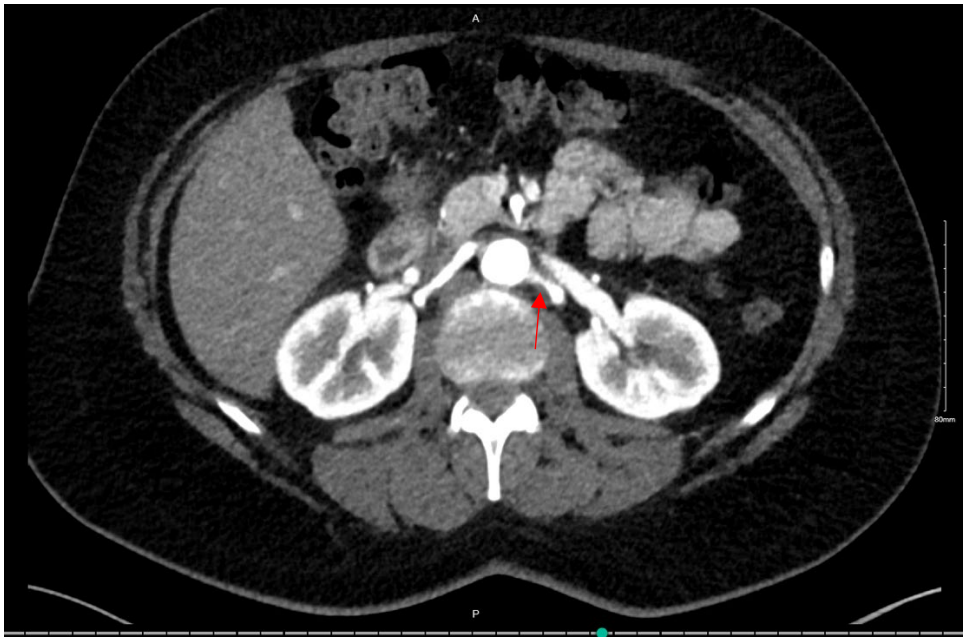
Figure 1: CTA documented narrowing of proximal left renal artery.



Discussion

Renal artery stenosis (RAS) is the most common secondary cause of hypertension and affects 1% to 5% of hypertensive patients, but the true prevalence of RAS is not known[2].

Atherosclerosis and fibromuscular dysplasia are the two most common causes of RAS. Atherosclerosis accounts for more than 90% of RAS lesions. Atherosclerotic renovascular lesions are common in the elderly, diabetics, patients with aortoiliac disease, hypertension, coronary artery disease, and peripheral artery disease[1,3].

Fibromuscular dysplasia accounts for less than 10% of RAS. Fibromuscular dysplasia typically presents in younger females with hypertension[4]. In this case, the underlying cause of our patient was challenging because she does not demonstrated any of above.

There are various opinions regarding the optimal diagnostic strategies. Angiotensin receptor blockers (ARB), angiotensin-converting enzyme inhibitors (ACEI), diuretics, calcium channel blockers(CCB), beta-blockers, and other antihypertensive agents are useful to manage hypertension in RAS[5]. ACEIs, ARBs, and calcium-channel blockers are recommended for the treatment of hypertension in unilateral RAS whereas β -blockers are recommended for the treatment of hypertension in patients with RAS in general[6]. In addition to BP control, aspirin

use, lipid lowering, and smoking cessation have been proposed for slowing the progression of RAS and preserving renal function[7].
Further treatment options beyond medical therapy involve renal artery revascularisation that can be accomplished surgically or percutaneously by balloon angioplasty or balloon angioplasty and stent placement[8].

In conclusion, the diagnosis of RAS should precipitate aggressive medical management in other to prevent secondary prevention. Timely diagnosis and management of RAS can lead to reduction in morbidity and mortality.

Declaration of Conflict of Interest:
None declared.

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