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CASE REPORT

An uncommon Case of Resistant Hypertension: Stenosis of Renal Artery

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ABSTRACT

Hypertension is one of the most important risk factors for mortality and morbidity globally. It is the most common chronic cardiovascular disease that results in increased hospital admissions worldwide. As hypertension has a long list of primary as well as secondary causes, hypertension induced by renal artery stenosis (RAS) is a form of secondary hypertension caused by renin overproduction and it affects approximately 2 to 5% of hypertensive patients. We report a case of RAS as a cause of resistant hypertension. It is important to make a note that resistant hypertension is the blood pressure above a goal despite adherence to at least three optimally dosed antihypertensive medications of different classes, one of which is a diuretic. Besides, there are other categories of resistant hypertension or difficult-to-treat hypertension such as primary hyperaldosteronism, thyrotoxicosis, chronic kidney disease, drug–drug interactions as well steroids, nonsteroidal anti-inflammatory drugs, erythropoietin, and herbal preparations such as liquorice. Morbid obesity is also associated with resistant hypertension.

Keywords: Chronic kidney disease, Obstructive renal arterial disease, Renal artery stenosis, Resistant hypertension.

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INTRODUCTION

Resistant hypertension is defined as the blood pressure above a goal, that is, usually 140/90 mm Hg despite adherence to at least three optimally dosed antihypertensive medications of different classes, one of which essentially should be a diuretic.[1-4] We present the case of an elderly female who was admitted to the hospital with resistant hypertension and successfully treated. Optimal dose of medication is presumed to be a moderate dose but not necessarily the maximum dose. Patients requiring more than or equal to three antihypertensive medications (even if controlled) are classified as having resistant hypertension. In the general hypertensive population, renal artery stenosis is not an uncommon secondary cause accounting for 2 to 5% of hypertensive patients being affected by it, though not always detected.

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CASE REPORT

A 75-year-old female was admitted to our hospital with complaint of headache that was on and off, moderate in intensity, and temporarily relieved by medications for the past 2 months. She also had complaints of palpitations and dyspnea on minimal exertion for past one and a half month as well as a history of recurrent urinary tract infection. Her blood pressure was monitored at home, which showed levels up to 190/100 mm Hg despite being on four antihypertensive medications, namely tablet amlodipin 5 mg once daily, tablet telmisartan 20 mg once daily, tablet metoprolol 50 mg twice daily, and a combination of torsemide 40 mg daily and spironolactone 50 mg daily. She was a known case of hypertension for 30 years, diabetes mellitus for 25 years, and coronary artery disease since past 10 years. There is no history of any tuberculosis contact, jaundice, asthma, or thyroid disorder. Patient is a sedentary worker with no addictions and no history of chronic intake of NSAIDs, diet pills, decongestants, thyroid medications, or excessive salt intake. She was on oral antihypertensives, oral hypoglycaemic drugs, statins, and antiplatelet drugs. A family history of sudden death of mother possibly due to stroke could have been from hypertension. On physical examination, she was moderately obese in built, alert, and well oriented to time, place, and person. She had a pulse rate of 96/minute regular. Her blood pressure was 180/100 mm Hg in supine position. Respiratory rate was 22/minute and regular. There was mild bilateral pedal edema present that was pitting in nature. She had no icterus, cyanosis, clubbing, or lymphadenopathy. Jugular venous pressure of patient was not raised, no thyromegaly was noted, and skin and hair pattern was normal. On systemic examination, her central nervous system examination was within normal limits with all higher function, and cranial nerves normal with no sensory or motor deficit and no neck stiffness. Respiratory examination revealed shifting of

apical impulse down to 6th intercostal space from left five intercostal spaces in anterior axillary line, which is normal along with no tracheal deviation and normal bilateral respiratory expansion with normal tympanic resonance on percussion and bilateral wheezing present on auscultation. While doing cardiovascular examination, an added S3/third-heart sound was heard with apex beat shifting downwards and outwards from normal position. On abdominal examination, there was mild tenderness on epigastric region with a faint abdominal bruit.

Considering her age and the comorbidities, she was evaluated thoroughly. Her hemogram was within normal range. Acute-phase reactants were above normal such as C-reactive protein was 25.2 mg/l and erythrocyte sedimentation rate was 55 mm/hour. Serum creatinine and electrolytes were normal. Urine routine showed trace proteins and trace ketones. There was elevated HbA1c and hypovitaminosis B12. On echocardiography, 2D echo revealed left ventricular ejection fraction = 55%, left ventricular hypertrophy, and mild mitral regurgitation. Her sleep study was done for complaints of snoring and fragmented sleep, which was suggestive of sleep-disordered breathing leading to oxygen desaturation and sleep fragmentation with a good response to continuous positive airway pressure therapy. Ultrasound of abdomen revealed hepatomegaly with grade 1 fatty liver. Sonogram of kidneys revealed uneven size of kidneys with right kidney measuring 70 mm and left kidney of 90 mm. Her magnetic resonance aortography showed near occlusion in right renal artery, whereas left renal artery was normal (Fig. 1). Computed tomography (CT) angiography scan revealed occlusion in the abdominal aorta just below origin of renal artery and stenosis in right renal artery (Figs 2 and 3). All other aortic branches were normal. Renal angioplasty with stenting was done in the cath lab. A medicated stent was put in right renal artery and was stabilized. The procedure is done quite frequently in our institution *via* brachial artery. In this case, size of brachial artery is quite adequate and negotiable and therefore was decided that the same approach, that is, brachial artery approach may be followed. Following the procedure at 1-month follow-up, the blood pressure patient was controlled on 40 mg of telmisartan and she was advised to monitor her blood pressure regularly and visit again for a follow-up.

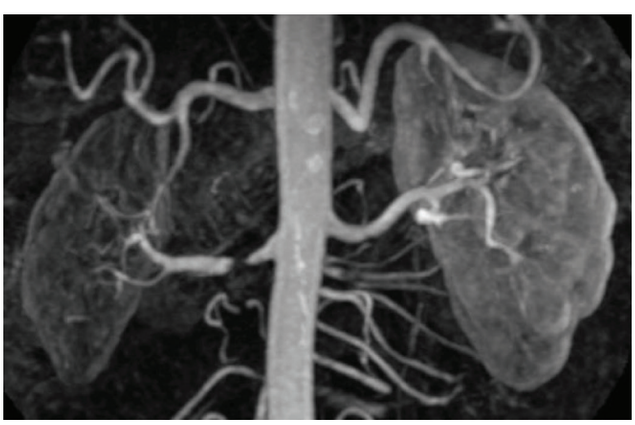

Fig. 1: Typical atherosclerotic stenosis—right renal artery

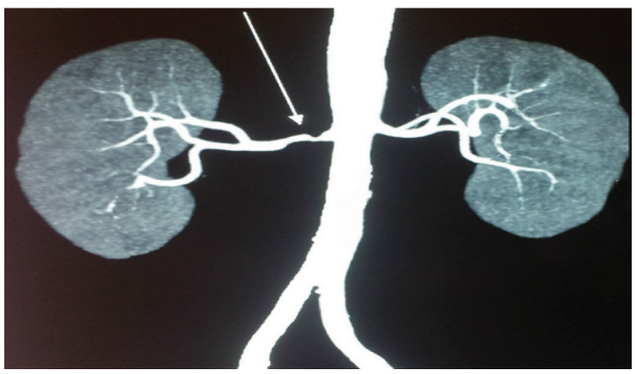

Fig. 2: Enhanced CT angiogram showing right renal artery stenosis

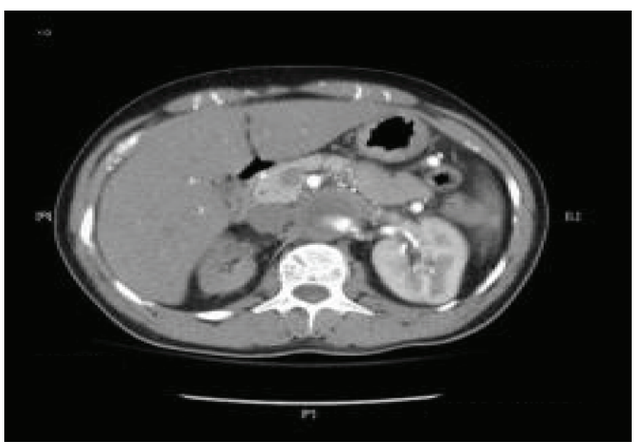

Fig. 3: Computed tomography of abdomen—right kidney is smaller than the left

DISCUSSION

Renal artery stenosis is the narrowing of one or both of the renal arteries, mostly caused by atherosclerosis or fibromuscular dysplasia. This narrowing of the renal artery can impede blood flow to the target kidney, resulting in renovascular hypertension – a secondary type of high blood pressure. Possible complications of renal artery stenosis are chronic kidney disease and associated coronary artery disease. Renal artery stenosis is most often caused by atherosclerosis that causes the renal arteries to harden and narrow due to the build-up of plaque. This accounts for about 90% of cases, rest due to fibromuscular dysplasia. Fibromuscular dysplasia is the predominant cause in young patients, usually for females under 40 years of age. A common cause of renin-angiotensin system (RAS) in the Indian subcontinent is Takayasu arteritis, an inflammatory panarteritis that involves the aorta and its major branches including renal

arteries. Resistant hypertension is common in obstructive renal arterial disease due to multiple factors such as low glomerular filtration rate (GFR), inappropriate activity of renin-angiotensin-aldosterone system (RAAS), and sympathetic nervous system (SNS). The cardiovascular and hemodynamic milieu in obstructive renal arterial disease is complex and governed by multiple hormonal and circulatory aberrations. Persistently high blood pressure can be a key contributor to stroke, heart failure, and other cardiovascular conditions. Resistant hypertension has several possible causes, including one or more other underlying medical conditions. In addition to treating resistant hypertension with medications, doctors typically investigate for secondary causes.

Renovascular hypertension is an uncommon but an important cause of secondary hypertension.[5,6] It should be understood that a mere anatomical/angiographic presence of renal artery stenosis is not synonymous with renovascular hypertension. One can have concomitant renal artery stenosis that may not be responsible for hypertension.[7,8] By definition, renovascular hypertension is a condition that is improved or cured by the correction of renal artery stenosis. Hence, the mere diagnosis of morphologic renal artery stenosis is not equal to renovascular hypertension. With widespread use of radiological imaging (ultrasound, CT, magnetic resonance imaging, and positron emission tomography scans, etc.), it is quite common to find 'incidental' renal artery stenosis. Therefore, much caution is advised in attributing hypertension to incidentally discovered renal artery stenosis. However, in cases of severe hypertension, resistant hypertension, complex hypertension, and sudden onset of new hypertension (in young and old), renovascular etiology should be considered. In our case, the patient presented with resistant hypertension that required proper assessment. Although possible obstruction sleep apnea (OSA) may have contributed to the patient's hypertension, the most likely explanation is renovascular hypertension. After the correction of renal artery stenosis, our patient's blood pressure was easily controlled just on one antihypertensive drug. Before the correction of renal artery stenosis, the patient was on five different antihypertensive drugs, but after renal angioplasty and stent, the patient's hypertension was well controlled on a single drug.

CONCLUSION

Resistant hypertension is commonly encountered in clinical practice. If the patient is complaint with optimal antihypertensive drug therapy and lifestyle changes and if the blood pressure still remains high, an underlying secondary cause should be suspected.[9] There are a number of secondary causes for hypertension. Work-up should be undertaken on the basis of clinical course, target organ damage, and biochemical abnormalities. In our patient, various factors including her age suggested possible renal artery stenosis and renovascular hypertension. The most common cause of renal artery stenosis is atherosclerosis. The other causes of renal artery stenosis include fibromuscular dysplasia, aortoarteritis, and vasculitis, etc. Renovascular hypertension should be suspected on clinical grounds and appropriate work-up should be initiated. Correction of renal artery stenosis may ameliorate hypertension in some patients with renovascular hypertension.[10]

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