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SECONDARY HYPERTENSION

Secondary Causes of Hypertension: Illustrative Cases

Sreenivas K Arramraju

ABSTRACT

Secondary hypertension is defined as increased systemic blood pressure due to an identifiable cause. The incidence of secondary hypertension varies from 5 to 10%. The most common etiology of secondary hypertension is due to renovascular causes. In this article, we briefly discuss as to when and how to suspect this pathology and give two illustrative real-world case examples with follow-up.

Keywords: Renal artery stenosis, Renovascular hypertension, Secondary hypertension.

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INTRODUCTION

The most common cause of secondary hypertension is renovascular hypertension.[1] We briefly present to you when and how to suspect this pathology and give two illustrative real-world case examples with follow-up. This we feel will help us to understand the importance of proper evaluation, management of the condition, and also highlight the role of interventional management in preventing the progression of disease and onset of end-stage renal disease (ESRD) in young patients.

The commonest causes of renovascular hypertension are:

Etiology:

- Nonspecific aortoarteritis
- Atherosclerosis
- Fibromuscular dysplasia
- Other – aortic/renal dissection, Thrombotic/cholesterol emboli, neurofibromatosis, posttransplant stenosis, postradiation stenosis.

Renovascular hypertension should be suspected by the following history and examination signs:

History:

- Onset of hypertension age <30 years or >55 years
- Sudden-onset uncontrolled hypertension in previously well-controlled patient
- Accelerated/malignant hypertension
- Intermittent pulmonary edema with normal left ventricular (LV) function.

Physical examination/lab investigation:

- Epigastric bruit, particularly systolic/diastolic
- Azotemia induced by angiotensin-converting enzyme inhibitors (ACEI).
- Unilateral small kidney.

Renovascular hypertension – Clinical

- History
- Onset hypertension age <30 or >55 years
- Sudden-onset uncontrolled hypertension in previously well-controlled patient
- Accelerated/malignant hypertension
- Intermittent pulmonary edema with normal LV function

Physical examination/lab investigation

- Epigastric bruit, particular systolic/diastolic
- Azotemia induced by ACEI
- Unilateral small kidney

The two most common pathologies which are most common are:

Atherosclerotic renal artery stenosis (RAS)

- 75 to 90% of RAS
- Usually men, age >55 years, other atherosclerotic disease
- Progression of stenosis is 51% at 5 years, 3 to 16% to occlusion, with renal atrophy noted in 21% of RAS lesions >60%
- End-stage renal disease in 11% (higher risk if >60% baseline renal insufficiency, systemic blood pressure (SBP) > 160)

Treatment

- Percutaneous transluminal renal angioplasty (PTRA) success 60 to 80% with restenosis 10 to 47%
- Stent success 94 to 100% with restenosis 11 to 23% (1 year)
- "Cure" of renovascular hypertension <30%

Fibromuscular Dysplasia

- 10 to 25% of all RAS
- Young female, aged 15 to 40 years
- 90% is medial disease often involves distal renal arteries
- 30% progressively worsen but total occlusion is rare

Chief

Department of Cardiology, Citizens Hospital, Hyderabad Telangana, India

Corresponding Author: Sreenivas K Arramraju, Chief Department of Cardiology, Citizens Hospital, Hyderabad Telangana, India, Phone: +919848046785, e-mail: arramraj@yahoo.com

Treatment – Percutaneous Transluminal Renal Angioplasty

- Successful in 82 to 100% of patients
- Restenosis in 5 to 11%
- "Cure" of hypertension in 60%

Case Example 1 – Case of Fibromuscular Dysplasia

A 19-year-old female presented with a history of headache and giddiness of 2 weeks duration. She had a blood pressure (BP) of 180/120 mm Hg without any significant difference in all four limbs. Her pulse rate was 85/minute. Systemic examination was normal but for an abdominal bruit. Ultrasound abdomen revealed contracted kidney on right with suspicion of left RAS. Her baseline creatinine was 1.5 gm/dL. Her angiogram revealed small contracted nonright kidney with tight RAS in distal portion of her left renal artery (Fig. 1). She was planned for left PTRA and stenting. A 6 × 18 mm Palmaz Stuart stent was deployed (Fig. 2). She had a 20% residual stenosis in spite of high-pressure postdilatation. Her renal parameters improved – serum creatinine (S. creatinine) decreased to 0.9 mg/dL and her BP was 120/80 mm Hg only with 2.5 mg Nebivolol. She was also given dual antiplatelet drugs (aspirin 150 mg once daily and clopidogrel 75 mg twice daily initially for 6 weeks which was later decreased to once daily) along with atorvastatin 40 mg per day.

She was doing well till September 2016, when she again started developing higher BP with worsening of S. creatinine to 1.5 mg/dL. She was suspected to have developed restenosis in the stent and was taken up for repeat renal angiography which revealed a tight stenosis again within the stent at the site of original lesion (Fig. 3). This time (October 2016) she was stented with drug-eluting stent – Promus Element 4 × 15 mm stent was postdilated with 5 mm NC balloon (Fig. 4). Her S. creatinine again improved and BP control is better with two drugs.

Case Example 2 – Atherosclerotic RAS

A 65-year-old man was admitted with complaints of sudden-onset chest pain, breathlessness, and sweating

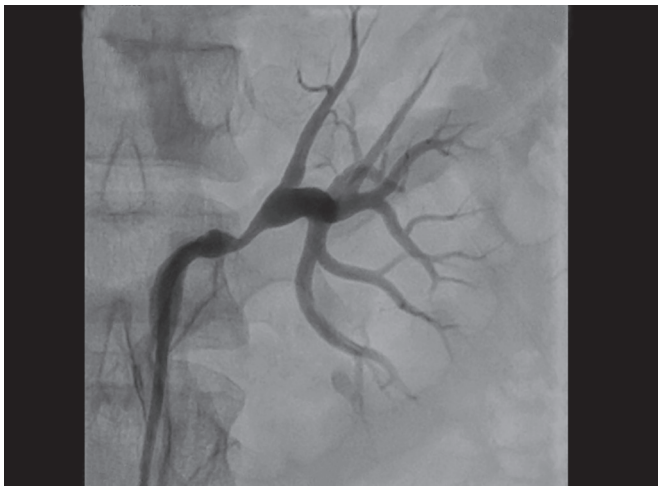

Fig. 1: Renal angiogram of left renal artery showing critical stenosis

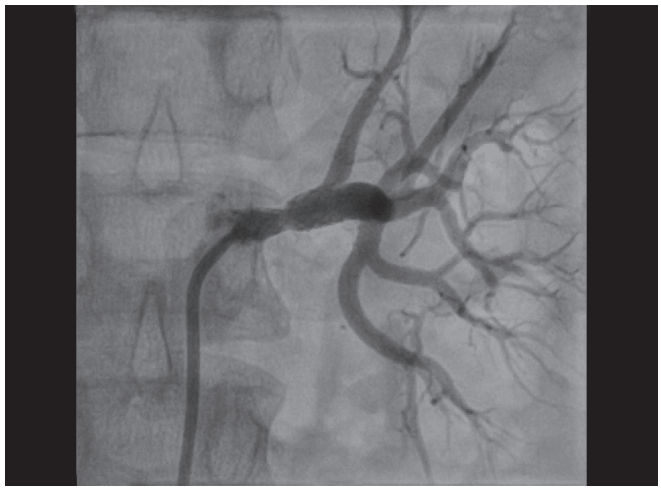

Fig. 2: Renal angiogram postrenal angioplasty and stenting of left renal artery (2013)

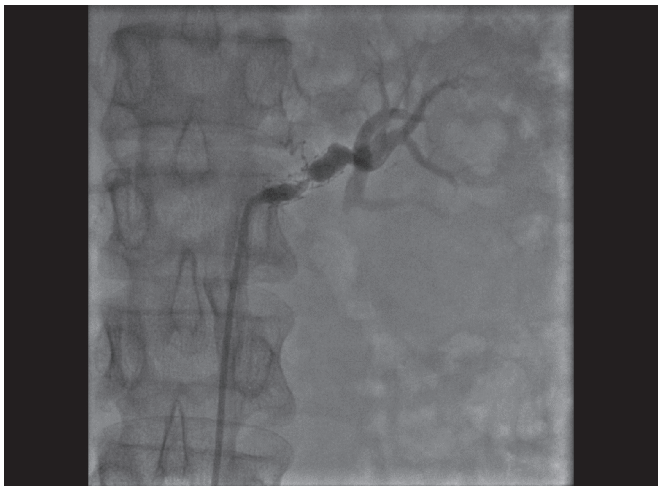

Fig. 3: Renal angiogram showing restenosis of left renal artery stent (2016)

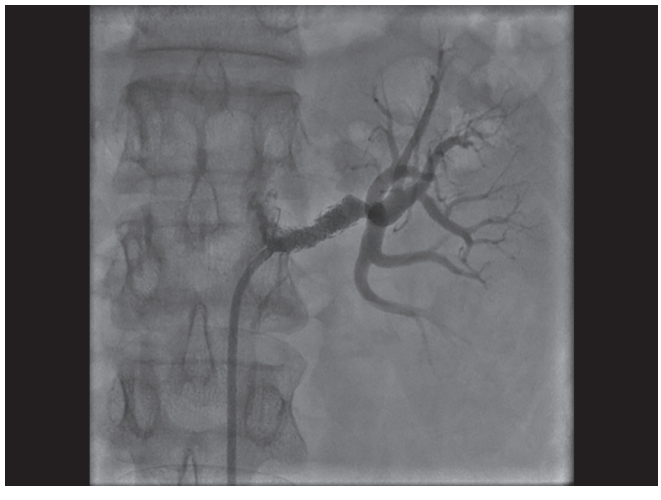

Fig. 4: Renal angiogram postrenal angioplasty and stenting of left renal artery stent restenosis

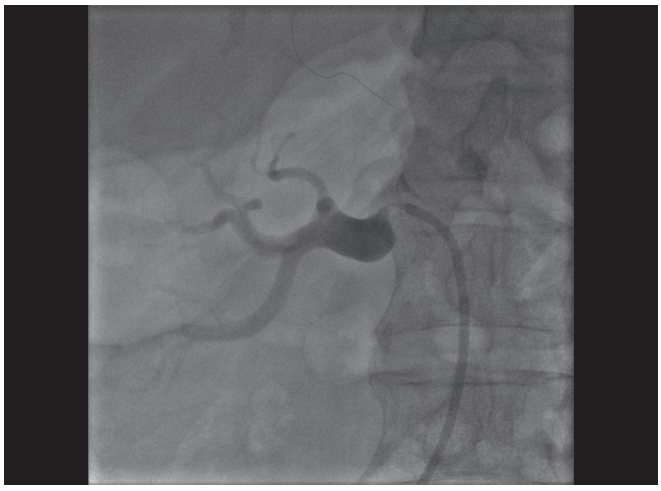

Fig. 5: Renal angiogram showing atherosclerotic right renal artery stenosis

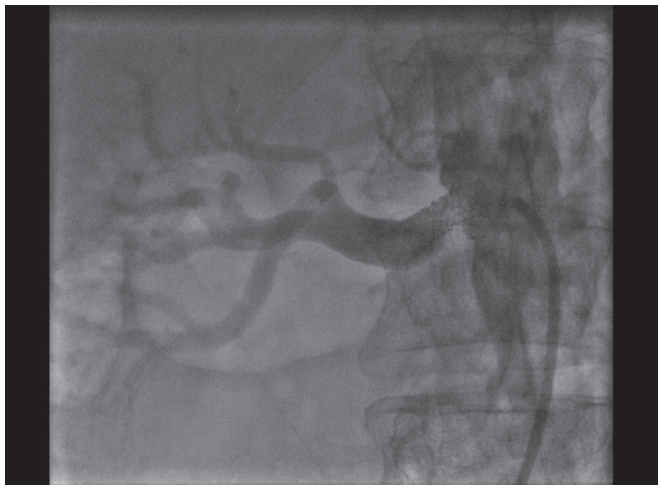

Fig. 6: Renal angiogram postrenal angioplasty and stenting of right renal artery

of 1 day duration. He also had recent history of bilateral leg swelling.

He was hypertensive and diabetic. He also had a history of coronary artery disease and had undergone percutaneous coronary intervention in 1999 and 2007 and coronary artery bypass grafting in 2008. On examination, patient was conscious, alert, and well-oriented afebrile, he had bilateral pedal edema. His pulse rate was 74/minute and BP was 200/90 mm Hg. Investigations revealed a raised creatinine (1.99 mg/dL). His two-dimensional echocardiogram revealed moderate LV dysfunction. His cath study revealed patent grafts but both renal arteries had critical stenosis (Fig. 5) with left-sided contracted kidney. He was subjected to right renal artery stenting with 6 × 18 mm stent. Poststenting, the renal perfusion improved with no residual stenosis (Fig. 6) and S. creatinine improved to 1.2 mg/dL after 3 days and BP was better controlled with two drugs.

REFERENCE

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