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Case Report

Renal Artery Stenosis presenting as acute worsening of Heart Failure with Preserved Systolic Function in a Hypertensive Patient

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Abstract

We describe a case of 52-year-old female who presented with heart failure, uncontrolled hypertension and worsening renal function with progression of heart failure on starting an Angiotensin Converting Enzyme Inhibitor (ACE-I). Magnetic Resonance Angiography (MRA) revealed significant stenosis of the right renal artery. Her symptoms and blood pressure improved after renal artery stenting.

Introduction:

Renal artery stenosis is a rare cause of secondary hypertension occurring in less than 1% of cases. “Flash” pulmonary edema is one of its known associations especially after starting an Angiotensin Converting Enzyme Inhibitor (ACE-I). Our case represents an uncommon presentation with worsening heart failure and hypertension after starting ACE-I.

Case presentation:

A 52-year-old female with type-II Diabetes Mellitus, Dyslipidemia, Benign Intracranial Hypertension, anemia, and recent onset hypertension on treatment, presented with generalized body swelling, shortness of breath and chest tightness. On physical examination her blood pressure was 170/72mmHg, heart rate was 116 beats/minute; there were mild bi-basal crepitation and S4 on auscultation. There was generalized body and ankle edema. Her musculoskeletal system showed back tenderness and diminished bilateral peripheral pulses.

White blood count was 10,600 cells/uL, hemoglobin was 7.1 gm/dl and platelet count of 369,000/uL. Creatinine

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was 0.8. Potassium was 5.2mmol/L, sodium was 136mmol/L.

Her electrocardiogram showed normal sinus rhythm with left ventricular hypertrophy and strain. Her initial chest X-ray showed evidence of cardiomegaly with pulmonary edema that worsened after starting her on Perindopril (Figure 1).

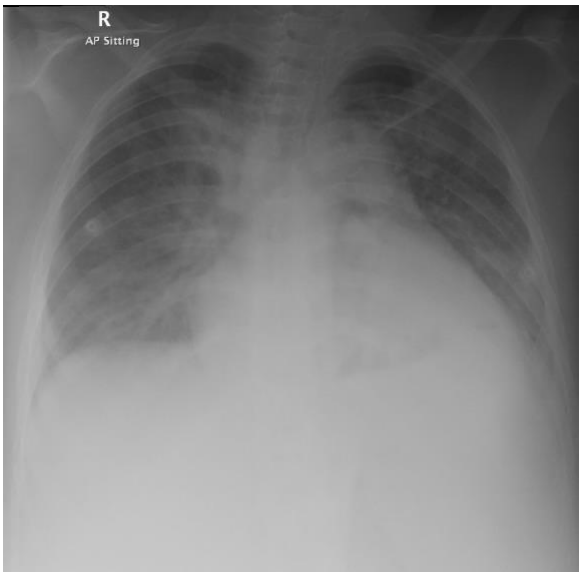

Figure 1: prominent pulmonary edema after commencement of Perindopril.

Echocardiography revealed normal left ventricular systolic function with ejection fraction (EF) of 76%, mild aortic valve stenosis, normal cardiac chambers, mild left ventricular hypertrophy, and grade 1 diastolic dysfunction. The patient was admitted to the cardiac care unit and started on intravenous diuretics and maintained on non-invasive ventilatory support. Her blood pressure was controlled with intravenous Nitroglycerin, Perindopril 5mg, Amlodipine 10mg, and Moxonidine 0.4mg. She was then noticed to have worsening shortness of breath and her blood pressure rose up to 190/100mmHg. Urea increased to 129mg/dl (from 68mg/dl), and Creatinine increased to 2.0 mg/dl (from 10.8 mg/dl). Deterioration in her clinical condition raised the suspicion of renal artery stenosis. Perindopril was stopped, hydralazine 50mg three times a day and spironolactone 25mg once daily were started. The patient condition's improved gradually after stopping Perindopril but her blood pressure remained elevated in spite of maximizing antihypertensive medication.

She underwent Magnetic Resonance Angiography (MRA) that showed severe right Renal Artery Stenosis (RAS) – Figure 2.

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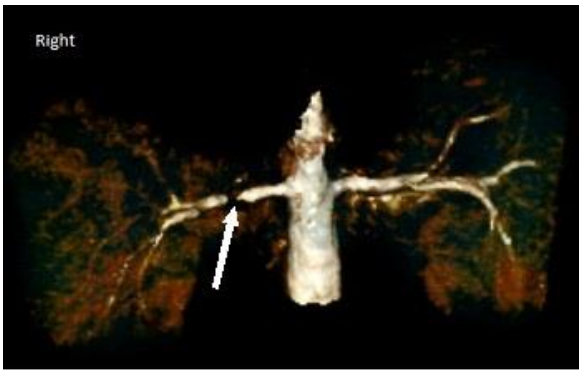

Figure 2: 3-D MRA illustrating severity of the right renal artery (arrow)

After confirmation of the diagnosis of right RAS by MRA, right Renal Artery Angioplasty with stent placement was performed (Figure 3). Her condition improved, blood pressure dropped to 125/78 mmHg and was discharged home in good condition. In the outpatient office her BP dropped further to 90/50 and her antihypertensive medication was reduced to 2 medications (from 4).

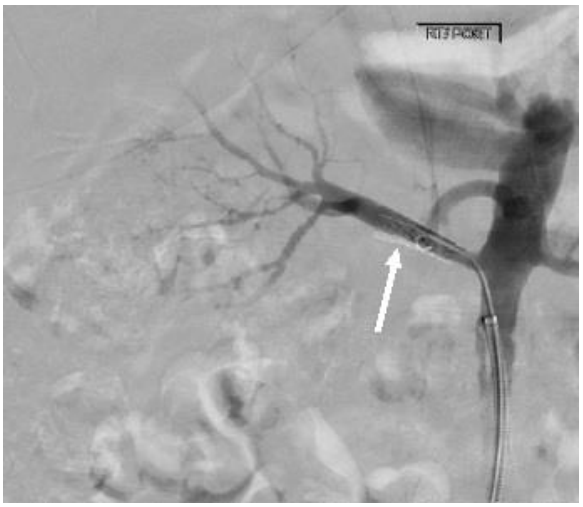

Figure 3: Renal angiography showing right renal artery stenting (arrow).

Discussion:

Hypertension in presence of RAS is found in less than 1% of patients with mild hypertension and as high as 10-40% in patients with acute severe hypertension (1,2). In patients with history of myocardial infarction, the prevalence of RAS is 12%; 6-19% in patients undergoing coronary artery angiography and 22- 59% in presence of peripheral vascular disease (3,4)

Diagnosis of RAS requires a high index of clinical suspicion especially in patients with several cardiovascular risk factors. The diagnostic accuracy can be enhanced by using novel modalities e.g. 3-D reconstructed MRA that can have a sensitivity and specificity approaching 100% (5).

Duplex ultrasound imaging can be of great help; however, it is operator dependent and has low sensitivity. If experts in renal duplex ultrasound are not available, the preferred imaging modality is either CT-arteriography or MRA especially in patients without renal insufficiency (6).

The commonest cause of RAS is atherosclerosis, seen in 90% of cases. Fibromuscular dysplasia is a cause of

RAS in 10% of cases specifically those below the age of 45 (7).

The treatment of choice of severe RAS in patients with uncontrolled hypertension, “flash” pulmonary edema, or severe acute hypertension is revascularization. Stent placement has a primary success rate of 88% in comparison to renal angioplasty, which carries a success rate of 57% (8). Surgical management has 80-95% cure rate when there are multiple small renal arteries, early branching of main renal artery, or if there is a need for aortic reconstruction.

Medical management is directed towards controlling hypertension and treatment of associated heart failure or renal impairment. ACE-I or Angiotensinogen Receptor blockers (ARB) can precipitate “flash” pulmonary edema as shown in our case. Thiazide diuretics, calcium channel blockers, mineralocorticoid blocker or beta-blockers can be used as add-on therapy (9).

Take Home Points

- A high index of suspicion is required for diagnosis of renal artery stenosis (RAS) as the condition does not commonly present with heart failure in hypertensive patients.
- MRA has high sensitivity in the diagnosis of RAS.
- Stenting of severe unilateral renal artery stenosis can lead to improved blood pressure control.

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