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## CASE REPORT

# A CASE REPORT OF RESISTANT HYPERTENSION DUE TO RENAL ARTERY STENOSIS: LONG TERM SUFFERINGS OF A MIDDLE-AGED GENTLEMAN

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**Abstract:**

*Atherosclerotic renal artery stenosis is the leading cause of reno-vascular hypertension and is prevalent in elderly patients (over 65 years) and those with resistant hypertension. This article presents a case report of a 60-year-old gentleman who presented with resistant hypertension resulting in heart failure and significant morbidity. The patient's medical history included frequent hospitalizations with uncontrolled hypertension, flash pulmonary edema and heart failure. Despite multiple hospitalizations, the underlying cause of his condition remained unrecognized until further investigation revealed left sided renal artery stenosis as the culprit lesion. Successful renal artery angioplasty resulted in the resolution of the patient's high blood pressure and improvement of general well-being. This case highlights the potential for resistant hypertension due to renal artery stenosis to be overlooked, particularly in patients with multiple comorbidity. It is important to consider the possibility of renal artery stenosis in patients with uncontrolled blood pressure with recurrent pulmonary oedema, not responding to multiple anti-hypertensive drugs in the highest possible dose.*

***Key words:*** Resistant hypertension, Renal artery stenosis, Flash pulmonary edema, Renal angioplasty

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**Introduction:**

Renal Artery Stenosis (RAS) is a pathological condition that results in restricted blood flow to the kidneys due to the narrowing of the renal artery. Atherosclerotic renal artery stenosis (ARAS) is the most common type of RAS, caused by the buildup of plaque in the renal artery, resulting in a decrease of more than 60% in luminal diameter. ARAS is the leading cause of renovascular hypertension and is prevalent in elderly patients (over 65 years) about 6.8%; more than 14% in patients with another atherosclerotic lesion, and 24% in patients with resistant hypertension (RHTN)[1].

RAS is found in patients with high blood pressure, chronic kidney disease, congestive heart failure, myocardial infarction, and stroke. RAS can lead to Reno-vascular hypertension, resulting in activation of the Renin-Angiotensin-Aldosterone (RAAS) system, which leads to sodium retention, vascular contraction, and secondary hyperaldosteronism.

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Bilateral or unilateral RAS can cause renal and cardiac failure due to sodium and liquid retention in the kidneys[2]. Ischemic nephropathy is a potential cause of irreversible renal failure that may lead to high blood pressure and chronic kidney disease. RAS may destabilize patients with heart failure or acute coronary syndromes due to uncontrolled blood pressure and volume retention[3].

Here, we have reported a case of 60-year-old gentleman presented with recurrent severe breathlessness due to acute pulmonary oedema with uncontrolled hypertension. All groups of anti-hypertensive drugs in highest possible dosage along with diuretics failed to control his blood pressure below 140/90 mmHg. Detailed investigations were done to find out any secondary cause of hypertension and later revealed the presence of left sided renal artery stenosis. Successful renal artery angioplasty with the implantation of a drug-eluting stent resulted in the resolution of the patient's high blood pressure and improvement of general well-being. This case highlights the potential for resistant hypertension due to renal artery stenosis to be overlooked.

**Case Report:**

A 60-year-old gentleman came to the emergency department in a wheelchair with severe breathlessness. He was unable to walk or lie down due to dyspnoea. There was no fever or cough. Examination revealed he was conscious, oriented, dyspnoeic, and anemic, and his blood pressure was 260/ 140 mm Hg, pulse 84 /min, temperature normal, respiratory rate 30 breaths/ minute, Spo2 95% in room air. Auscultation of the chest revealed bilateral fine basal crepitations up to midzone with widespread rhonchi. Fundoscopy revealed grade 4 hypertensive retinopathy. He was immediately hospitalized as a patient of hypertensive emergency. He was managed with intravenous frusemide, other anti-hypertensive medications & other supportive treatments for acute left ventricular failure in the ICU setting.

After some improvement, he mentioned his long story of sufferings. He has suffered from hypertension for the last 20 years with regular medications & follow-ups. However, his BP has remained high for the last 1 year despite regular anti-hypertensive medication intake. Different combinations of anti-hypertensive drugs were prescribed by internists & cardiologists with the highest possible dosage along with diuretics and centrally acting anti-hypertensive drug, but most of the time, his BP was uncontrolled; average BP was > 160/100 mmHg. He used to suffer from exertional breathlessness, unable to perform his daily activities. There was sleep disturbance due to orthopnoea. He was non-smoker, non alcoholic, and used to lead a healthy lifestyle. On routine check-ups, his renal function was found to be deteriorating; serum creatinine became > 2mg/dl. He was gradually becoming anemic.

About 6 months back, he was admitted to CCU with severe breathlessness & was diagnosed with NSTEMI with acute left ventricular failure. His echocardiogram revealed ischemic heart disease with regional wall motion abnormality of the left ventricle (LV), dilated LV with mild LV systolic dysfunction (LVEF =43%), and concentric left ventricular hypertrophy. He was non diabetic. His serum creatinine was 2.8 mg/dl, eGFR 26. HRCT chest revealed healed lesion with fibrotic streaking in the left posterior segment. Coronary angiogram was done, which revealed non-critical coronary artery disease (30-40% stenosis in the proximal segment of the right coronary artery). He was discharged with optimum medical management.

Unfortunately, he needed hospitalization 3 more times with similar complaints within a short period of time. Each time, his blood pressure was very high with features of pulmonary oedema, which used to improve with intravenous frusemide in high doses.

When he was admitted for the fifth time within less than 6 months period, he requested his treating physician team earnestly to come to a solution to his recurrent problem. He & his family were drained physically, mentally & also financially. He was evaluated thoroughly again with the supervision of an internist, cardiologist, and nephrologist. His current anti-hypertensive regime included: Tab Atenolol 50 mg OD, Tab Nifedipine 20mg TDS, Tab Prazosin ER 5mg 2tab TDS, Tab Clonidine 0.1 mg TDS, Tab Frusemide 40 mg 5 tab daily in divided doses. ARB was avoided due to rising serum creatinine. With these 5 antihypertensive drugs in high doses, his blood pressure was still > 160/ 100 mmHg, pulse was 55-60/ min. He could not lie down or sleep; his life was miserable.

His investigations profile during this setting is shown in Table -01.

**Table-I :** *Investigations*

| Name of Investigation | Findings |
|---|---|
| Complete Blood Count | Haemoglobin- 8.7 mg/dl( MCV, MCH was low); total & differential count was normal, ESR 82 mm in 1st hour, |
| Serum creatinine | 2.14 mg/dl |
| Serum electrolytes | Na 134 , K 4.73, Cl 104, HCO3 20 mmol/L |
| Cardiac Troponin I | Normal |
| Name of Investigation | Findings |
| NT-pro BNP | >35,000.00 pg/ml ( normal < 125.00) |
| ECG | Sinus bradycardia with features of left ventricular hypertrophy |
| Chest X-ray | Cardiomegaly with features of pulmonary oedema |
| Echocardigram | Concentric LVH with moderate LV systolic dysfunction ( LVEF 37%), moderate pulmonary hypertension (PASP 67 mmHg) |
| Urin RE | Albumin Trace, otherwise normal |
| Liver function, serum calcium, inorganic phosphate, Blood sugar | Within Normal Limit |
| USG of KUB | Bilateral chronic renal parenchymal disease (right>left), prominent inferior venacava (2.2cm), mild enlargement of prostate, right sided mild pleural effusion |
| ABG | Metabolic acidosis |

We were searching for any secondary cause of his resistant hypertension. This time, a meticulous examination of the abdomen revealed the presence of renal bruit. Renal angiogram was performed, which revealed the left renal artery was visualized, which has got 90- 95% stenosis at its origin (Fig-1). Stenting of the left renal artery was done by an experienced vascular surgeon in the proper setting (Fig- 2), which finally brought the end of the long-term sufferings of this gentleman.

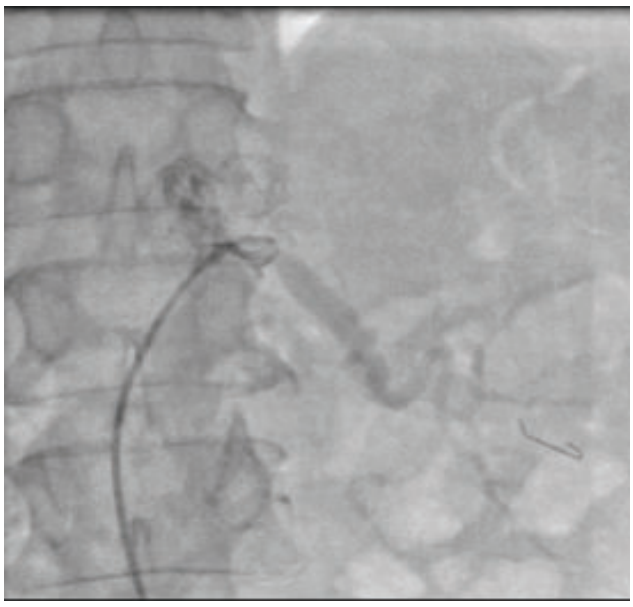

**Figure-1:** Renal Angiogram showing the stenosis and post stenotic dilatation

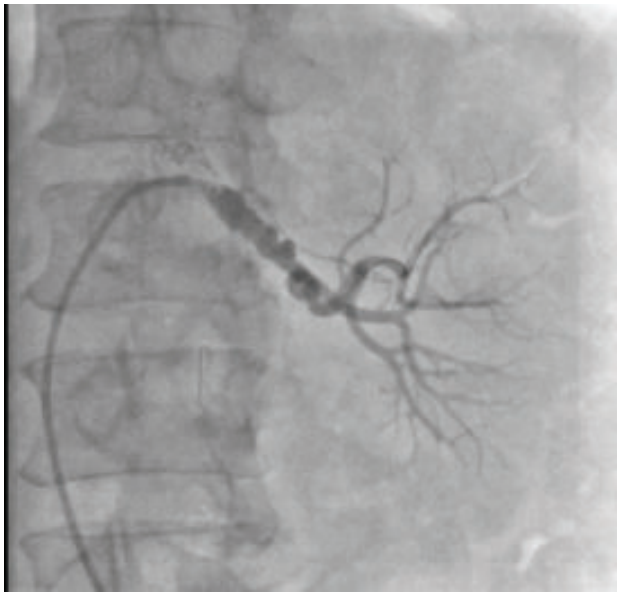

**Figure-2:** Renal Angioplasty with Stenting, showing the Nephrogram

In the follow-up visit after stenting, he came with a happy face without any features of dyspnoea. He does not have any physical complaints now. He can sleep now without an additional pillow. His blood pressure is maintained at 130/ 80 mm Hg with Tab Bisoprolol 10 mg, Tab Prazosin 5 mg BD, Tab Nifedipine 20 mg TDS, and Tab Clonidine 0.1 mg BD. His lung bases are clear.

**Discussion:**

Renal artery stenosis (RAS) is a condition characterized by the narrowing of one or both renal arteries and is a major cause of hypertension. The etiology of RAS can be attributed to atherosclerosis or fibromuscular dysplasia, with potential complications including chronic kidney disease and end-stage renal disease[4].

Detecting RAS is challenging because it is usually asymptomatic, and most cases occur with other diseases such as chronic kidney disease and diabetes. Screening for RAS can be done using Doppler ultrasound, computed tomography angiography, and magnetic resonance angiography[5]. The gold standard for diagnosis is renal angiogram. In this case, imaging modalities help us to make the diagnosis of left sided renal artery stenosis.

Treatment of RAS involves various medical measures, such as blood pressure control, lipid-reducing therapy, and antiplatelet agents. Lifestyle modifications such as dietary counseling, smoking cessation, and physical activity are also recommended[6]. Accurately correcting dyslipidemia, using drugs that block platelet aggregation, may require three or more different drugs to control blood pressure. Angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs) are preferably used for this purpose. Unfortunately, these two classes of drugs can also lead to an increase in serum creatinine and hyperkalemia and limit their use. In such a case, calcium channel blockers are a potential alternative[7]. Fat reduction therapy is widely accepted as an important treatment for all atherosclerotic vascular diseases. A statin is recommended to achieve LDL b 70 mg/dL. In a retrospective study, statin treatment was associated with a lower progression rate for renal failure (7.4% vs. 38.9%) and lower overall mortality (5.9% vs. 36.1%), $P<0.001$ for both[8]. The use of antiplatelet agents and smoking cessation in patients with ARAS has the same benefits as the other forms of atherosclerotic disease, including coronary and peripheral vascular disease[2].

Invasive procedures, such as renal artery stenting (PTRAS), are the preferred choice in most patients with high-grade RAS and cardiac instability symptoms. Stenting has been associated with improved kidney function and overall survival. Revascularization can also enable treatment with renin-angiotensin blockers, which may confer mortality benefits in patients with RAS who tolerate these medications[9]. Our patient had improvement in his blood pressure control within days after his procedure, and it has remained controlled in follow-up. He has had no further admissions with heart failure or pulmonary edema. Similar improvement of heart failure has been described by Alyamani M et al. after renal angioplasty in a patient with renal artery stenosis[10]. Also, Milewski et al. analyzed the clinical improvement of hypertension in 265 consecutive patients with ARAS treated with stenting[11].

In summary, here we presented a case study of a patient with unilateral RAS who experienced rapid improvement in blood pressure control within days of renal artery stenting, with sustained good results at follow-up visits. No adverse events were observed, and the patient's general condition remained good. These findings highlight the potential benefits of renal artery stenting in managing RAS and associated complications.

**Conclusion:**

Renal artery stenosis (RAS) leading to resistant hypertension can result in significant morbidity, which can be prevented with appropriate management. However, diagnosing and treating RAS can be challenging. A high level of suspicion is necessary for timely diagnosis. Revascularizations of the affected vessel are beneficial, particularly in sicker patients with recurrent hospitalizations due to hypertensive emergency and flash pulmonary edema.

**Conflict of Interest:**

The authors stated that there is no conflict of interest in this study

**Funding:**

This research received no external funding.

**Consent:**

For the purpose of publishing this case report and any related photos, the patient are written informed consent was acquired.

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