Case Report

Renal artery stenosis as an etiology of secondary hypertension: A case report

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ABSTRACT

Secondary hypertension is often resistant to pharmacologic therapy alone and necessitates identification and management of the underlying cause. This report details a case of a 52-year-old middle-aged female with a history of resistant hypertension secondary to bilateral renal artery stenosis, managed successfully with percutaneous transluminal angioplasty. The case highlights the importance of recognizing renovascular hypertension and timely intervention to prevent complications such as heart failure and renal dysfunction.

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

Hypertension is one of the most common chronic illnesses worldwide, with a prevalence of 22.6% in India. It is impacting hundreds of millions of adults, and serves as a major risk factor for heart disease, stroke, and renal disease [1]. While most cases are due to essential (primary) hypertension, secondary hypertension accounts for a small but considerable number of cases, especially in younger individuals or those with difficult-to-control blood pressure (BP) [2]. Renal artery stenosis (RAS) is a significant cause of secondary hypertension. It occurs in patients who present with severe or resistant hypertension, abrupt worsening of BP control, sudden deterioration of renal function after starting an angiotensin-converting enzyme inhibitor or angiotensin receptor blocker, unexplained chronic kidney disease, or recurrent episodes of pulmonary edema that are difficult to explain otherwise [3,4]. RAS may be owing to atherosclerosis, especially in elderly persons with established vascular disease, or due to fibromuscular dysplasia (FMD), particularly in younger women. Other causes include vasculitis, congenital abnormalities, trauma, or external compression [1,2]. Pathophysiologically, RAS leads to diminished renal perfusion, which causes renin release. This activates the renin-angiotensin-aldosterone system, causing systemic vasoconstriction, salt and water retention, and ultimately hypertension [1]. Persistent hypoperfusion may also lead to ischemic nephropathy, scarring, and progressive kidney disease [5,6]. Early management may help preserve renal function and prevent cardiovascular and renal morbidity.

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CASE REPORT

A 52-year-old female presented to the emergency department with complaints of bilateral frontal throbbing headache and non-bilious vomiting (two episodes per day) for the past 10 days. She denied photophobia or hematemesis. Her medical history included hypertension for 10 years and hypothyroidism for 5 years. Two years prior, she had been diagnosed with bilateral RAS and an atrophic right kidney. Her regular medications included prazosin, metoprolol, and levothyroxine.

On examination, she was conscious and oriented. Her BP was significantly elevated at 240/130 mmHg in the right upper limb, 166/84 mmHg in the left upper limb, and 250 mmHg systolic in the right lower limb. Pulse oximetry showed SpO_2 of 97% on room air. Systemic examination was unremarkable. Electrocardiogram revealed T-wave inversions in leads I and aVL, and chest X-ray was unremarkable.

Laboratory investigations were largely normal except for hyponatremia (Na+ 129 mEq/L). Thyroid-stimulating hormone was 5.47 µIU/mL. Abdominal ultrasonography revealed an atrophic right kidney (5.4 × 2.8 × 0.6 cm) and a normal-sized left kidney (9.2 × 5.4 × 1.8 cm).

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Echocardiography indicated concentric left ventricular hypertrophy and mild pericardial effusion with preserved ejection fraction (60%). Initial management included intravenous furosemide, oral torsemide with spironolactone, amlodipine, metoprolol, prazosin, clonidine, and thyroxine, along with antiplatelet and statin therapy. BP decreased to 140/80 mmHg within 24 h.

However, the patient was readmitted 2 days later with acute dyspnea (modified medical research council grade IV), BP of 230/120 mmHg, SpO_2 of 88%, and clinical features of acute left ventricular failure. She was managed in the medical intensive care unit with nitroglycerin infusion and non-invasive ventilation (continuous positive airway pressure with FiO_2 40%), leading to improved oxygenation. Chest X-ray revealed pulmonary edema. Laboratories showed mild renal impairment (blood urea 50 mg/dL, serum creatinine 1.3 mg/dL) and electrolyte imbalances (Fig. 1).

Despite escalation of antihypertensive therapy, including sodium nitroprusside infusion and intermittent labetalol, her BP remained uncontrolled, and urine output declined. Nephrology consultation was sought, and renal angiography was planned. The patient received N-acetylcysteine prophylaxis for contrast nephropathy. Renal angiography demonstrated 90–95% ostial stenosis of the left renal artery and non-visualization of the right renal artery, suggestive of complete occlusion (Fig. 2). Percutaneous balloon angioplasty of the left renal artery was performed successfully.

Post-procedure, BP improved to 140/90 mmHg without IV antihypertensives. Oral antihypertensive doses were reduced, renal function and urine output normalized, and the patient was weaned off ventilatory support. She was discharged in stable condition with optimized oral therapy. The patient was followed up after a month, and was found to be in good condition.

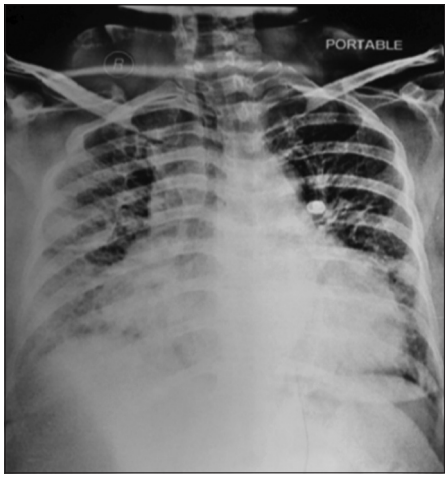

Figure 1: Portable chest X-ray suggestive of acute pulmonary edema

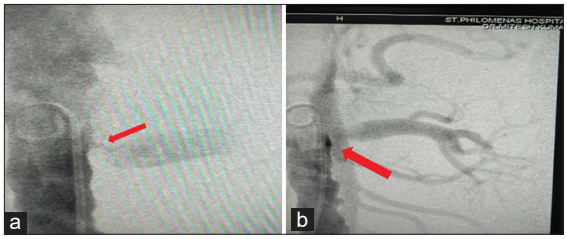

Figure 2: (a) Abdominal aortogram showed left renal artery ostial stenosis (90–95%). (b) Right renal artery not visualized (100% occluded from the ostium)

DISCUSSION

RAS is a recognized but often underdiagnosed cause of secondary hypertension. Atherosclerotic disease remains the predominant etiology in older adults, whereas FMD occurs more frequently in younger women and typically affects distal segments of the renal artery [7]. The clinical spectrum is wide, ranging from asymptomatic presentations to resistant hypertension, renal dysfunction, and recurrent flash pulmonary edema. High clinical suspicion is warranted in patients with unexplained renal atrophy, early- or late-onset hypertension, or worsening renal function following renin–angiotensin system blockade [8,9].

Diagnostic evaluation includes imaging modalities such as duplex ultrasonography, computed tomography (CT) angiography, and magnetic resonance angiography, with digital subtraction angiography remaining the gold standard. Timely recognition is crucial, as several reports highlight RAS as an underlying cause of recurrent pulmonary edema or cardiorenal syndromes [10]. These findings stress the importance of integrating both clinical and radiologic assessment for early diagnosis and management.

The therapeutic approach has been shaped by randomized controlled trials. The angioplasty and stenting for renal artery lesions and cardiovascular outcomes in renal atherosclerotic lesions trials reported no significant benefit of routine stenting compared with medical management in unselected patients [3,4]. However, these trials excluded or underrepresented high-risk subgroups, such as patients with bilateral disease, recurrent pulmonary edema, or rapidly declining renal function. Observational data and case-based evidence suggest that revascularization may offer significant clinical benefit in these select populations, with improvements in BP control, renal function stabilization, and resolution of heart failure symptoms [11,12].

Current consensus supports optimal medical therapy as first-line treatment while reserving revascularization for patients with hemodynamically significant RAS and high-risk clinical features [13]. The present case illustrates this selective benefit, highlighting that individualized assessment and timely intervention can prevent irreversible renal damage and improve long-term outcomes.

CONCLUSION

This case shows why it is so important to look for underlying causes when BP remains high despite treatment. In renovascular hypertension, catching it early

can protect the kidneys and heart from serious harm. With timely diagnosis and the right intervention, patients have a much better chance at healthier, longer lives.

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