Bilateral Renal Artery Stenosis Presenting as Flash Pulmonary Edema

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Abstract
Flash pulmonary edema is a condition characterized by sudden and recurrent episodes of dyspnea at rest resulting from acute pulmonary venous congestion in the presence of normal or well-preserved LV systolic function. This is usually associated with bilateral renal artery stenosis or stenosis of a single surviving kidney. We report here a case of a 58-year-old man, a patient of coronary artery disease who later developed bilateral renal artery stenosis and presented with recurrent episodes of flash pulmonary edema. He was successfully treated with stenting of both renal arteries.

INTRODUCTION
Acute recurrent pulmonary edema in association with renal artery stenosis was first described by Pickering et al. It differs from usual cases of left ventricular failure with pulmonary edema in that this condition is usually not associated with severe left ventricular systolic dysfunction. It is mostly nocturnal and comes rather suddenly. Although it is usually responsive to standard methods of treatment including diuretics occasionally it can be more serious and even may require ventilation. Characteristically it recurs after some time. In most cases the condition is associated with bilateral renal artery stenosis or stenosis of the renal artery of the single surviving kidney. There have been many reports ever since. We describe here a case of bilateral renal artery stenosis presenting as flash pulmonary edema and was successfully treated with angioplasty.

CASE SUMMARY
A 58-year-old man presented with recurrent episodes of breathlessness over one month. He had inferior myocardial infarction one year ago and underwent coronary angioplasty to proximal RCA with stent. He is a non-hypertensive and non-diabetic. He used to smoke 10 cigarettes a day for 30 years before myocardial infarction after which he stopped smoking. He has been on regular followup after his MI and was receiving all the usual cardiac medications including ACE inhibitors, beta-blockers, oral antiplatelet agents and statins. Patient was entirely free of symptoms and used to walk daily 2 kilometers to his place of work. He works as a junior level employee at the Metro Water Board and his work involves doing physical labour. He never experienced left ventricular failure during or after the myocardial infarction till he developed the present problem. The development of breathlessness used to be rather sudden and would respond to a few injections of intravenous furosemide and infusion of nitroglycerine. The patient would improve rather quickly and used to insist on going home next day. Altogether he had four admissions in one month. All these episodes were precipitated by straining at stools early in the morning and he would develop acute pulmonary edema by 7.00 AM and would reach to the hospital soon after that. The blood pressure at presentation used to be high in the...
range of 160-170(SBP)/100-110 mm Hg (DBP). Initially it was felt that the patient’s left ventricular dysfunction (due to old inferior MI) was responsible for the LVF. However he had no S3 at any time. The blood urea was 53 mg% and serum creatinine 1.7 mg%. In view of the fact that the development of pulmonary edema was sudden and the recovery rather quick coupled with mild elevation of renal parameters we suspected that the patient might be having renal artery stenosis. He was investigated for the same including ultrasound abdomen, Doppler of the renal arteries and coronary and renal angiography. The ultrasound examination of the abdomen and Doppler examination of the renal arteries were non-contributory. The angiography however was diagnostic. The left anterior descending artery showed a 30% discrete lesion at the origin of second diagonal which is the same as it was one year ago. The circumflex artery is normal. The RCA showed mild instent stenosis (30%). The LV angio showed mild LV dysfunction with ejection fraction of 30% and no MR. The renal angiography showed critical 99% lesion of left renal artery and 90% lesion of right renal artery at the ostia (Figs. 1& 5). In view of these findings we felt that the symptoms are due to renal artery stenosis. Renal angioplasty was done with stenting. The left renal artery stenosis was predilated with a 3.0 mm balloon and then it was stented with a 5.0x12 mm “Racer” stent.
(Medtronic Inc, Minneapolis, MN). The right renal artery was directly stented with 6.0x12 mm “Racer” stent with good result (Figs. 2,3,4,6 & 7). Patient recovered uneventfully after the procedure. He has been asymptomatic after renal angioplasty and completed three months of follow up. His renal parameters have improved and presently the blood urea is 31 mg% and serum creatinine 1.2 mg%. The left ventricular ejection fraction has increased from 30% (pre-renal stenting) to 40% at the end of 3 months post procedure. Patient did not develop any further episodes of breathlessness and is able to attend his work normally.

**DISCUSSION**

Pickering et al get the credit for highlighting the occurrence of acute recurrent pulmonary edema secondary to bilateral renal artery stenosis. In their first series they described 11 patients with history of multiple episodes of pulmonary edema, 7 of them had bilateral renal artery stenosis, 2 had stenosis of artery to a solitary kidney and 2 had unilateral stenosis. Successful revascularization improved blood pressure and renal function and virtually eliminated pulmonary edema. They propose that the cause of pulmonary edema in renal artery stenosis is due to reduced pressure natriuresis. An acute increase of renal artery pressure leads to decreased tubular sodium reabsorption, which would tend to counter the development of pulmonary edema but in patients with bilateral stenosis this effect would be reduced because the kidneys would not be exposed to the systemic pressure.

An association with coronary artery disease is not unusual and in fact rather common. All the patients involved in the original description by Pickering had significant coronary artery disease. In five of these 11 patients a rise in plasma creatinine on an ACE inhibitor had already occurred before the diagnosis of renovascular disease was made. However, it is only because the specific symptom of pulmonary edema was improved by angioplasty or renal artery bypass graft that the relationship between the renal artery narrowing and the pathophysiological condition was established.

The abrupt nature of the condition gives it its usual name ‘flash pulmonary oedema’. Planken and Ritveld report two cases where the precipitating factors were swimming and central venous catheterization. In our case the precipitating factor was straining while passing stools.

In our patient the development of renal artery stenosis with symptoms occurred one year after the myocardial infarction. Renal angiography was not done at the time of the original coronary angio/angioplasty. It is very unlikely that the patient had significant renal artery stenosis at that point of time, as that would have given rise to some symptoms. The use of ACE inhibitors was well tolerated and only shortly before the development of recent symptoms he developed rise in the renal parameters which once again reversed with stenting of the renal arteries.

The fact that the response to renal artery angioplasty has been excellent and patient became asymptomatic strongly supports the argument that the pulmonary edema was secondary to bilateral renal artery stenosis and not due to left ventricular dysfunction which in fact has improved following renal angioplasty.

A suspicion of renal artery stenosis is usually raised under the circumstances of uncontrolled hypertension despite several medications, recurrent pulmonary edema in the absence of or out of proportion to left ventricular
dysfunction, renal insufficiency, presence of renal artery bruit/peripheral vascular disease and rising renal parameters in serum in response to ACE inhibitors.

**CONCLUSION**

An important cause of sudden pulmonary edema is bilateral renal artery stenosis as exemplified by our case. Timely recognition and angioplasty with stent leads to a satisfactory result.

**Acknowledgements**

The authors hereby express their gratitude to Ms. Bharathi for secretarial assistance in preparing this manuscript.

**REFERENCES**


**Announcement**

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**RAPICON 2006**

Railway Chapter of Association of Physicians of India (RAPI) is organizing its 13th Annual Conference - RAPICON 2006 at NWR Headquarters Hospital, Jaipur.

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