

Flash Pulmonary Edema Manifesting for the First Time after Cardiac Surgery: Case Report

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ABSTRACT

Flash pulmonary edema secondary to renal artery stenosis is an unrecognized complication following cardiac surgery. We report a case and discuss issues surrounding its diagnosis and management.

CASE REPORT

An 81-year-old hypertensive woman, known to suffer from intermittent claudication, was admitted with a 6-week history of angina of increasing frequency culminating in acute coronary syndrome. An angiogram revealed a tight left main stem stenosis and a blocked right coronary artery. A transthoracic echocardiogram showed moderate left ventricular dysfunction and moderate mitral regurgitation. Examination revealed bilateral carotid bruits, absent right and weak left femoral pulses, and no palpable distal pulses. Carotid Doppler studies confirmed an 80% to 90% stenosis of the right internal carotid artery (ICA) and 40% stenosis of the left ICA with a nonpatent anterior communicating artery. Therefore, as a preliminary procedure, a right carotid endarterectomy was performed while the patient was under local anesthetic. We considered performing a computed tomographic (CT) renal arteriogram to define the patient's renovascular anatomy prior to coronary artery bypass grafting (CABG). However, before this procedure could be performed, the patient suffered from recurring chest pain with electrocardiogram (ECG) changes and raised troponin T levels. An intraaortic balloon pump was inserted into the left femoral artery under direct vision and urgent CABG plus mitral valve repair was performed. A transesophageal echocardiogram confirmed the immediate preoperative transthoracic echo findings of moderate mitral

regurgitation. Postoperative recovery was slow, and an elective tracheostomy was performed. Ten days after surgery, while sitting in chair, the patient developed acute dyspnea associated with a blood pressure of 210/100 mm Hg without ECG changes. Chest x-ray revealed gross pulmonary edema. The pulmonary artery pressures were 28/15 mm Hg. The patient was reventilated, and frusemide infusion was commenced. A transthoracic echo showed good left ventricular contractility, no mitral regurgitation, and minimal pericardial collection. Serum creatinine level was within normal laboratory reference range. There was a rapid improvement in the patient's condition and complete resolution of pulmonary edema within the next few hours. Three further episodes with a similar sequence of events occurred in the next 2 weeks. ECG findings remained unchanged. Because the patient's left ventricular function did not show deterioration, renovascular etiology was considered as a cause of recurrent pulmonary edema. The patient's 24-hour urinary catecholamine levels were normal (noradrenaline, 334 nmol and adrenaline, <72 nmol). Serum renin levels were high. CT arteriogram of the renal arteries revealed a tight right-sided renal artery ostial stenosis and 50% stenosis of the left renal artery (Figure 1). A percutaneous balloon angioplasty with right renal artery stent insertion was carried out. Subsequently, the patient stayed free of symptoms for the next 20 days before having another minor episode of pulmonary edema, which resolved promptly. The patient made an unremarkable recovery thereafter and was transferred to another hospital for convalescence. However, she was readmitted after 6 weeks with another episode of pulmonary edema. Repeat renal arteriogram showed a patent right stent (Figure 2), and at this stage the left renal artery was stented as well (Figure 3). The patient was then discharged home, and no recurrence of pulmonary edema was observed at 6 months follow-up.

DISCUSSION

Renal artery stenosis is a rare cause of acute pulmonary edema. Pulmonary edema associated with unilateral or bilateral renal artery stenosis [Diamond 1993] may be sudden in onset, associated with poorly controlled systemic hypertension despite antihypertensive and diuretic therapy [Weatherford 1997], and disappear promptly with treatment. This classical picture is termed flash pulmonary edema (FPE).

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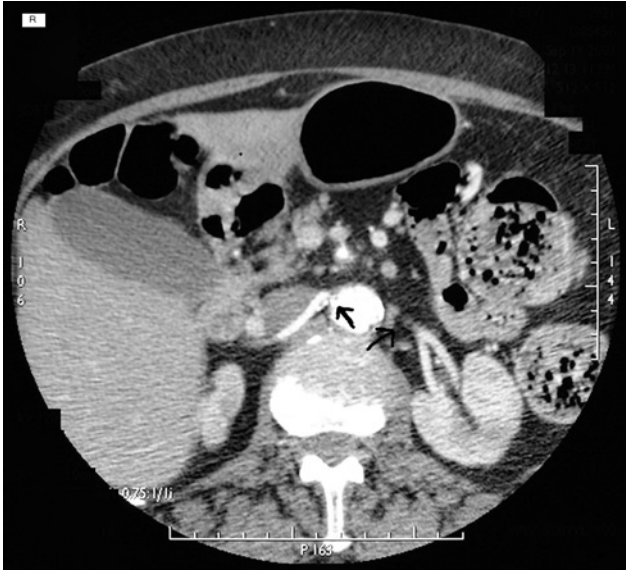


Figure 1. Computed tomographic renal arteriogram showing >90% stenosis of the right renal artery and approximately 50% stenosis of the left renal artery (black arrows).

A significant unilateral stenosis increases renin secretion from the juxtaglomerular apparatus, causing sodium and water retention by the ipsilateral kidney. A normal contralateral kidney suppresses its renin secretion and natriuresis occurs, restoring intravascular volume. This process may not occur if the contralateral kidney is abnormal, and pulmonary edema may result [Koch 1968]. Almost all patients with FPE have evidence of systemic atherosclerosis [Weatherford 1997] including ischemic heart disease, so a cardiac etiology is sought first, inevitably leading to a delay in the diagnosis. Moreover, FPE can also occur despite a normal left ventricular ejection fraction, a characteristic that differentiates FPE patients from patients with pulmonary edema secondary to poor cardiac contractility [Pickering 1988].

For our patient, based on carotid duplex results, we performed a right carotid endarterectomy. However, before a

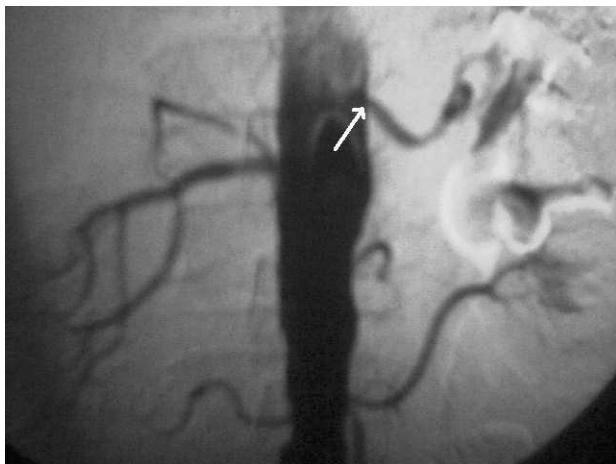


Figure 2. Selective renal angiogram showing patent right renal artery stent and approximately 50% stenosis of the left renal artery (white arrow).



Figure 3. Selective renal angiogram showing patent right and left renal arteries after stent insertion (white arrows).

CT renal arteriogram could be done, the patient developed acute coronary syndrome and required urgent surgery. Post-operatively, the following conditions probably contributed to the delay in the final diagnosis: First, after the primary episode of pulmonary edema, our attention was diverted toward left ventricular dysfunction, mitral valve incompetence, and possible cardiac tamponade. These concerns were not unreasonable considering the patient had undergone recent CABG and mitral valve surgery. Nevertheless, serial echocardiograms did not reveal clinically significant cardiac pathology, and there was no angina. Second, although FPE is usually associated with impaired renal function, the serum creatinine levels were within the normal reference range, and urine output was adequate for the patient's weight. Third, on each occasion, it could not be established with certainty whether pulmonary edema was preceded by hypertension (renal origin) or vice-versa (cardiac origin). On readmission the occurrence of angina was not associated with impaired contractility or valve dysfunction on transthoracic echocardiography.

In this case, FPE as a result of renal artery stenosis manifested itself for the first time after cardiac surgery in an 81-year-old woman. Although systemic hypertension following CABG has been reported to occur in 15% to 80% of cases, it has been found that plasma renin activity and levels of angiotensin II and aldosterone return to normal within a few hours after cardiopulmonary bypass in patients with normal renovascular anatomy [Weinstein 1987]. Kaul et al [1990] have shown that after cardiopulmonary bypass there is a greater activation of the renin-angiotensin system in patients who have undergone CABG combined with mitral valve surgery than with CABG alone. However, the degree of activation of the renin-angiotensin system after cardiac surgery in patients with renal artery stenosis still remains a matter of debate.

Poor cardiac contractility remains the main cause of pulmonary edema following cardiac surgery. However, FPE due to renal artery disease is a rare syndrome that should be considered when transient episodes of pulmonary edema cannot be adequately explained by the patient's cardiac status.

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