Case report - Cardiac general

Flash pulmonary edema in an orthotopic heart transplant recipient

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Abstract

Flash pulmonary edema (FPE) is a severe renovascular disease that leads to acute recurring pulmonary edema and acute systemic hypertension. Though rarely reported in the literature, its incidence is probably underestimated secondary to misdiagnosis, especially in patients with normal left ventricular function. We report the case of an orthotopic heart transplant recipient who presented with FPE despite having normal left ventricular function and no signs or symptoms of transplant rejection. Discovery of severe bilateral atherosclerotic renal artery stenosis in this patient led to emergency hepatorenal bypass surgery and a favorable postoperative course.

Keywords: Postoperative care; Transplantation, Heart; Atherosclerosis; Revascularization; Peripheral vascular disease

1. Introduction

First described by Pickering et al. in 1988, the flash pulmonary edema (FPE) syndrome is associated with acute recurrent pulmonary edema secondary to severe renovascular disease in patients with varying degrees of left ventricular or renal function [1]. This report describes the case of an orthotopic heart transplant recipient who presented with FPE and subsequently underwent successful revascularization of the renal arteries.

2. Case report

A 60-year-old man presented with recurrent acute dyspnea. His medical history included ischemic heart failure, asymptomatic peripheral arterial disease, and heart transplantation in 1996. Cardiovascular risk factors included hypertension and a history of smoking. Physical examination revealed hypertension (190/100 mmHg) and evidence of pulmonary edema. Laboratory tests revealed an elevated plasma creatinine level of 228 μmol/l; an elevated brain natriuretic peptide (BNP) level of 7523 ng/l; and normal levels of cardiac enzymes. Chest radiography suggested a pulmonary edema. Electrocardiography (ECG) results were normal. Echocardiography revealed normal left ventricular function. Right heart catheterization studies demonstrated normal pulmonary artery, central venous, and pulmonary capillary wedge pressures and a normal cardiac index. Myocardial biopsies revealed no evidence of allograft rejection. Coronary angiography revealed no significant coronary artery stenosis.

In hospital, the patient was given diuretics and nitrates but continued to experience episodic acute dyspnea, especially at night. His renal function deteriorated further over the next three days. Color Doppler ultrasonography revealed diffuse calcification of the abdominal aorta, bilateral renal artery occlusion, and occlusions of the inter- and infrarenal abdominal aorta and both iliac arteries. Only the right kidney appeared functional. These findings were confirmed by magnetic resonance angiography (MRA) (Fig. 1). Because FPE was considered one possible cause of the patient’s acute pulmonary edema, it was decided to perform emergency revascularization of the right renal artery. Moreover, because of the extensive aortic calcifications and occlusions of the abdominal aorta and iliac arteries, it was also decided to perform an extra-anatomic bypass. To this end, a saphenous vein graft was used to create a hepatorenal bypass between the common hepatic and distal right renal arteries.

The patient’s postoperative course was uneventful. By postoperative day 5, his plasma creatinine levels had fallen to 175 μmol/l and his blood pressure was being controlled. At two years of follow-up, the hepatorenal venous bypass graft remained patent (Fig. 2). After three years of follow-up, the patient remains asymptomatic, showing no signs or symptoms of FPE and having a plasma creatinine level of 145 μmol/l.

3. Discussion

To our knowledge, this is the first published report of FPE in a heart transplant recipient. Usually, the FPE syndrome occurs in the presence of unilateral or bilateral obstructive renal atherosclerosis leading to recurrent episodes of acute pulmonary edema associated with acute systemic hypertension [1–3]. The rapid onset of pulmonary edema, usually
Fig. 1. Magnetic resonance angiogram showing an occlusion of inter- and infrarenal aorta, occlusions of bilateral renal arteries and both iliac arteries before renal artery revascularization (arrow shows the right renal artery).

during the night, is typical of the syndrome [2]. The diagnosis is usually confirmed by the relief of the symptoms after renal revascularization [1–3]. FPE can be difficult to diagnose, especially in a heart transplant recipient who is already at risk of cardiac or renal impairment (or both). Thus, coronary artery disease must first be ruled out as a diagnosis because it may be associated with asymptomatic myocardial ischemia that triggers transient left ventricular dysfunction, increased left ventricular end-diastolic pressure, and acute pulmonary edema [4]. Our patient’s left ventricular function and coronary angiography were normal, and myocardial biopsies revealed no cardiac allograft rejection. Therefore, in the absence of a cardiac cause, renovascular disease appeared to be the most likely etiology.

Declining renal function after heart transplantation is common. In an estimated 2–8% of all heart transplant recipients, it becomes severe enough to warrant dialysis or renal transplantation [5]. Although the nephrotoxic effect of chronically administered cyclosporine plays a major role in this process, so do hypertension, hyperlipidemia, diabetes, pretransplantation peripheral vascular disease, and other immunosuppressive drugs, all of which can promote atherosclerotic renal artery stenosis [5, 6]. Decompensated coronary artery disease and heart failure may be induced in the presence of renal artery stenosis by peripheral arterial vasoconstriction, by the direct effect of angiotensin II on the myocardium, and by volume overload [3]. In fact, any diagnosis of FPE must assume a link between severe renal artery stenosis and volume overload. In the presence of severe renal artery stenosis, increased amounts of renin are secreted from the juxtaglomerular apparatus, which in turn induces the ipsilateral kidney to retain sodium and water [3]. Usually, the contralateral kidney counterbalances this effect by reducing renin secretion. However, in the presence of significant bilateral renal artery stenosis or a non-functioning contralateral kidney, volume overload occurs and may induce FPE [3]. The previously mentioned nocturnal predominance of FPE may be explained by the fact that a reduction in blood pressure during the night may reduce renal perfusion in the presence of renal artery stenosis and induce renin secretion [2].

Renal artery revascularization (percutaneous balloon angioplasty or surgical revascularization) is the treatment of choice for FPE [1, 7]. In the present case, extensive and diffuse renal artery lesions prevented any attempt at balloon angioplasty, and the rapid and severe renal impairment that was observed required emergency surgical revascularization. Moreover, in the face of the patient’s diffuse and heavy aortic calcifications, only an extra-anatomic procedure was considered. Simultaneous aortoiliac reconstruction was not considered because (a) it would likely have increased the risk of death and acute renal failure and (b) the iliac artery occlusions were asymptomatic.

In summary, in this unusual case of FPE in a heart transplant recipient, renal artery revascularization improved renal function and blood pressure and relieved acute recurrent pulmonary edema. It should be noted, however, that diagnosis of FPE in such cases must first rule out decompensated myocardial ischemia or heart failure (or both) and await definitive retrospective confirmation after adequate renal artery perfusion has been restored.

References


