

Flash pulmonary edema in the cardiac catheterization laboratory: A case report

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Abstract

We report a 51-year-old female with a history of a degenerated aortic bioprosthesis and severe valvular dysfunction who was admitted for redo valve surgery and unexpectedly developed acute flash pulmonary edema, a dramatic form of cardiogenic pulmonary edema before coronary angiography and was managed by timely and proper treatment.

Introduction

Flash pulmonary edema (FPE) is a remarkable and life-threatening condition caused by an imbalance in the fluid homeostasis in the pulmonary vascular bed, which results from elevated left-sided cardiac filling pressures along with endothelial dysfunction and increased permeability of pulmonary capillaries.^(1, 2)

Common causes of FPE are myocardial ischemia, hypertensive emergencies, tachyarrhythmias, and acute mitral or aortic valve regurgitation. Bilateral atheromatous renovascular disease and pheochromocytoma are also other predisposing factors.⁽³⁾

FPE can occur during cardiac catheterization procedures at any time. The immediate diagnosis and management of this critical condition are imperative to stabilize the patient as soon as possible and prevent unfortunate consequences.

This report presents a 51-year-old female with a history of hypertension, degenerated bioprosthetic aortic valve, and severe valvular dysfunction who was a candidate for redo valve surgery and developed acute FPE before coronary angiography.

Case Presentation

The patient was a 51-year-old female with a history of hypertension and biological aortic valve replacement surgery 21 years ago who was referred to our hospital for dyspnea. Electrocardiogram revealed sinus rhythm and incomplete left bundle branch block. Transthoracic echocardiography demonstrated normal left ventricular (LV) systolic function and severe LV diastolic dysfunction. Mild LV hypertrophy was also observed. There was a flail bioprosthetic leaflet due to the degeneration of aortic bioprosthesis resulting in severe aortic regurgitation and severe aortic stenosis (Video S1, Video S2, Video S3). Furthermore, severe posteriorly directed mitral regurgitation was detected. The patient was admitted for redo aortic valve replacement and mitral valve surgery, and she was submitted to coronary angiography before the surgery due to chest discomfort and electrocardiographic changes. In the catheterization laboratory, she had a blood pressure of about 160 over 90 mmHg after placing the sheath in the right femoral artery. Prior to the procedure, the patient gradually developed dyspnea and severe orthopnea. Her blood pressure rose to 270 over 130 mmHg (Figure 1), and she had a respiratory rate of 30 per minute, a heart rate of 120 per minute, and an oxygen saturation of 89 percent in the room air. Diffuse rales were heard in both lungs. Her situation suddenly exacerbated, and clinical evidence of FPE was apparent. She was placed in a semi-setting position, and

supplemental oxygen therapy with a mask was started. Resuscitation equipment was prepared. ECG was obtained, but it did not show any new changes. Intravenous nitroglycerine and furosemide were soon administered. Nitroglycerin infusion was started at a rate of 30 micrograms per minute and the dose doubled every 15 minutes up to 90 micrograms per minute. She also received three doses of 40 milligrams of furosemide at 20-minute intervals. After about an hour with the continuation of the vasodilator, loop diuretic therapy, and respiratory support under hemodynamic monitoring, the patient's condition improved clinically, and she was stabilized. Coronary angiography was performed and showed no significant lesion. Two days later, she underwent aortic and mitral valve replacement surgery. The timeline of the events is shown in Table 1.

Discussion

We discussed the case of a middle-aged female with acute respiratory distress and elevation of blood pressure in the cardiac catheterization laboratory in favor of FPE.

Acute cardiogenic pulmonary edema is a common and critical condition caused by increased LV end-diastolic and left atrial (LA) pressures, which are retrogradely transmitted to the pulmonary venous and the capillary system. It consequently increases the hydrostatic pressure across pulmonary capillaries, resulting in fluid accumulation within the lung interstitium and alveoli.⁽¹⁾ FPE is a life-threatening and extreme form of cardiogenic pulmonary edema which develops within minutes. It is related to a sudden rise in cardiac filling pressures as well as endothelial dysfunction and increased permeability of pulmonary capillaries, which are the key features to distinguish FPE from cardiogenic pulmonary edema where we solely have elevated filling pressures.⁽²⁾

Exaggerated sympathetic activity and increased activity of the renin-angiotensin-aldosterone system (RAAS) are etiologies known to play essential roles in developing FPE.⁽²⁾ Catecholamines increase heart rate and reduce diastolic filling time of the LV, which notably in patients with diastolic dysfunction results in impaired diastolic filling and therefore elevation of LA and pulmonary venous pressures.⁽⁴⁾ Both sympathetic hyperactivity and activation of RAAS caused by catecholamines raise systemic vascular resistance and precipitate acute elevation of blood pressure. The augmented afterload results in increased LV wall stress and the imbalance between oxygen demand and supply, leading to even more diastolic dysfunction and elevation of filling pressures.⁽²⁾ Endothelial dysfunction and excessive permeability of the pulmonary capillaries are other consequences of catecholamines.^(2, 4) Besides systemic RAAS, activation of intrapulmonary RAAS contributes to fluid accumulation within pulmonary interstitial and alveolar areas by increasing pulmonary capillary permeability.⁽²⁾

FPE was reported in some other clinical conditions. Several reports discussed FPE associated with renal artery stenosis and excessive renin-angiotensin-aldosterone system activity. Symptoms were improved after revascularization of the renal artery in these cases.⁽⁵⁻⁷⁾ Naman Agrawal et al. in 2016 described sympathetic crashing acute pulmonary edema as the most severe entity of acute pulmonary edema, which must be treated with noninvasive ventilation and intravenous nitrate started in minutes.⁽⁴⁾ In a case report by Catrina Patrício et al. in 2014, a 64-year-old woman was reported who was admitted to the emergency department with FPE due to acute severe aortic insufficiency.⁽⁸⁾

FPE may develop in the cardiac catheterization laboratory at any time during the procedure. The diagnosis of FPE is based on clinical evaluation. Rapid progression of dyspnea and hypoxemia indicates the necessity of prompt diagnosis and management to avoid the need for mechanical ventilation and adverse outcomes.

It seems that activation of the sympathetic system secondary to extreme anxiety in combination with severe valvular dysfunction and severe LV diastolic dysfunction predisposed our patient to FPE.

History and physical examination are the most valuable tools for better determination of this event.⁽²⁾ Patients may have a history of LV diastolic dysfunction, systemic hypertension, or severe valvular dysfunction. The presence of bilateral renal artery stenosis or pheochromocytoma must be noted. Moreover, acute events such as tachyarrhythmias, acute mitral or aortic valve regurgitation, myocardial ischemia, and hypertensive crisis commonly predispose patients to FPE.^(2, 3) Patients usually present with respiratory distress, ortho-

pnea, tachypnea, and diaphoresis. Assessment of blood pressure, heart rate, and oxygen saturation must be done. Elevated blood pressure, tachycardia, and hypoxemia are common. Auscultation may reveal rales, S3 gallop, and valvular murmurs. Jugular venous pressure may be elevated, although it may be hard to measure. 12-lead ECG and bedside echocardiography are also helpful.^(3, 9)

Immediate initiation of high-dose intravenous nitroglycerine must be done in the first step because of its vasodilatory effect on the arterial system. The aim is to reduce the afterload and cut the vicious cycle of sympathetic and RAAS hyperactivity as soon as possible.⁽⁴⁾ Intravenous loop diuretics may also be helpful. Since FPE is basically caused by fluid redistribution rather than volume overload, and the diuretic effect of furosemide starts in 30 minutes at the earliest, the effect of loop diuretics is mostly due to their vasodilator instead of diuretic activity.^(2, 4) Supplemental oxygen therapy should be administered for hypoxemia as well. Once the patient is stabilized, further evaluation and determination of the cause of FPE must be performed.⁽²⁾

Conclusion

Flash pulmonary edema is not a rare clinical condition and may happen in critical situations, especially in the catheterization laboratory. This case highlights the value of urgent diagnosis and initiation of appropriate treatment in order to avoid catastrophic consequences.

Author Contributions:

Maryam Mehrpooya: Conceptualization; data curation; supervision; validation; writing – review and editing.

Mohammad Reza Eftekhari: Software; data curation; writing – review and editing.

Tara Moghaddasfar: Conceptualization; methodology; writing – original draft; resources; project administration; writing – review and editing.

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Figure Legend

Figure 1

Femoral arterial pressure tracing when the patient's condition started to exacerbate, showing a pressure of about 270 over 130 mmHg.

Table 1

Timeline of clinical events.

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Table 1.docx available at <https://authorea.com/users/521252/articles/620254-flash-pulmonary-edema-in-the-cardiac-catheterization-laboratory-a-case-report>

