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

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Abstract: Flash pulmonary edema is a potentially fatal condition that can suddenly deteriorate a patient's status in a variety of settings, including the catheterization laboratory. We describe a 51-year-old woman with a history of hypertension who was admitted for a second valve operation for degenerated aortic bioprosthesis. Before undergoing coronary angiography, she looked a little worried, she experienced respiratory distress and a significant increase in blood pressure in favor of acute flash pulmonary edema, which was immediately and successfully managed by respiratory support and administration of high-dose intravenous nitroglycerine and loop diuretic therapy. The present scenario highlights the significance of being aware of the warning signs of acute flash pulmonary edema to make a prompt diagnosis and initiate the appropriate treatment to prevent catastrophic consequences.

Keywords: Aortic Valve Insufficiency, Bioprosthesis, Case Reports, Coronary Angiography, Pulmonary Edema

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1. Introduction

Flash pulmonary edema (FPE) is a life-threatening condition caused by an imbalance in the fluid homeostasis in the pulmonary vascular bed, resulting from elevated 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 (3).

In addition to the aforementioned causes, flash pulmonary edema can be caused by anxiety and severe stress (4).

FPE can occur in different situations. Known and effective treatments for pulmonary edema, along with anti-anxiety and stress treatments can significantly help manage this disastrous condition. The immediate diagnosis and management of this critical condition are imperative to stabilize the patient 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.

2. 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 with dyspnea. The electrocardiogram (ECG) 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 (Figure 1). Furthermore, severe 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 seemed a bit worried at first but calmed down with some talking and reassurance. She had a blood pressure of about 160 mmHg over 90 mmHg, after placing the sheath in the right femoral artery. Before the procedure, the patient gradually developed dyspnea and severe orthopnea. Her blood pressure increased to 270 mmHg over 130 mmHg, which was also noticeable on her femoral artery pressure tracing (Figure 2). She had a respiratory rate of 30 per minute, a heart rate of 120 beats per minute, and an oxygen saturation of 89% 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. 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 (mg) of furosemide at 20-minute intervals. Considering the significant role of extreme stress in the

occurrence of this serious complication, 3 mg of intravenous morphine sulfate was also injected: both as an adjunctive treatment for pulmonary edema and as an anxiolytic. After about an hour, her condition improved clinically, and she was stabilized. Coronary angiography 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.

3. Discussion

FPE is a life-threatening and extreme form of cardiogenic pulmonary edema which develops within minutes. It is related to a sudden rise in left-sided cardiac filling pressures as well as endothelial dysfunction and increased permeability of pulmonary capillaries (2).

Exaggerated sympathetic and renin-angiotensin-aldosterone system (RAAS) activities 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 elevation of left atrium and pulmonary venous pressure (5). Both sympathetic hyperactivity and activation of RAAS raise systemic vascular resistance and precipitate acute elevation of blood pressure (2).

Endothelial dysfunction and excessive permeability of pulmonary capillaries are other consequences of catecholamines (2,5).

FPE was reported in some other clinical conditions. Several reports discussed FPE associated with renal artery stenosis and excessive RAAS activity (6,7). 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 starting intravenous nitrate in minutes (5). In a case report by 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).

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.

The diagnosis of FPE is based on clinical evaluation including history and physical examination (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, orthopnea, tachypnea, and diaphoresis. 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 (8-10).

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. 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 impact of loop diuretics is mostly due to their vasodilator instead of diuretic activity (2). 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).

4. Conclusion

Flash pulmonary edema can occur at any point during the coronary angiography, even before the procedure, especially in the presence of severe anxiety and stress and if it is not instantly recognized, it can be lethal. This case illustrates the value of being aware of warning signs of flash pulmonary edema and prompt, appropriate therapy commencement for a favorable outcome.

5. Declarations

5.1. Acknowledgement

None.

5.2. Authors' contribution

The authors meet all criteria for authorship based on the recommendations of the International Committee of Medical Journal Editors (ICMJE).

5.3. Conflict of interest

None.

5.4. Funding

No funding was received for this study.

5.5. Patient consent

Written informed consent was obtained from the patient to publish this report.

5.6. Data availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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Table 1 Timeline of clinical events

Time	Events
Day 0	Hospital admission
Day 1-10:00 AM	Transfer to the cardiac catheterization laboratory
Day 1-10:15 AM	Preparation for the procedure and obtaining femoral artery access
Day 1-10:20 AM	Occurrence of dyspnea and orthopnea and elevation of blood pressure to 270 mmHg over 130 mmHg
Day 1-10:30 AM	Clinical evidence of flash pulmonary edema, initiation of supplemental oxygen therapy, nitroglycerine infusion, intravenous furosemide, and also intravenous morphine sulfate
Day 1-11:00 AM	Improvement in the patient's condition with the continuation of the above treatments
Day 1-11:30 AM	The patient's condition was stabilized
Day 1-12:00 PM	Coronary angiography was performed
Day 3	Aortic and mitral valve replacement surgery

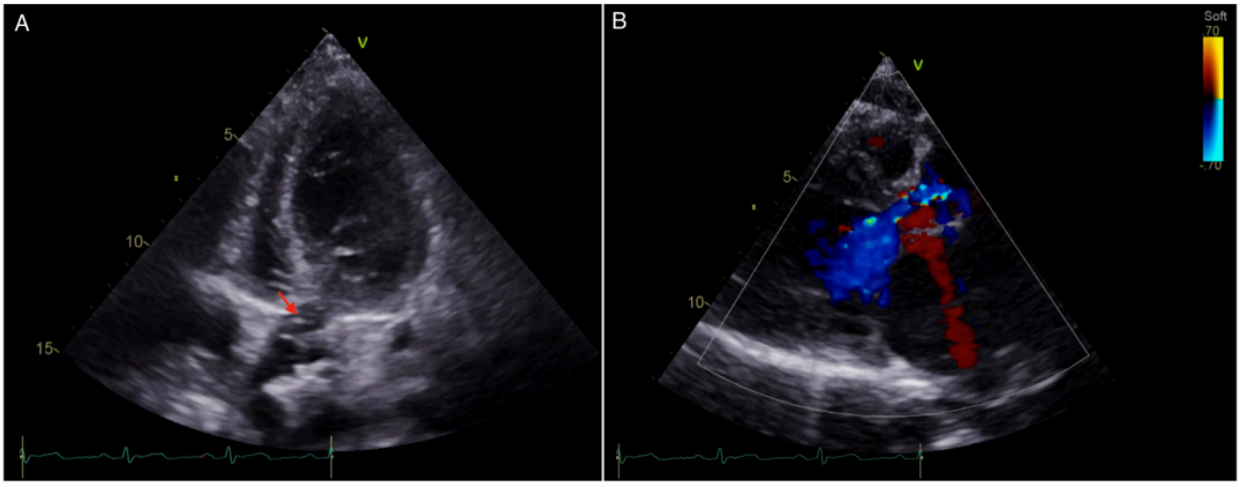

Figure 1 A: Transthoracic 2-dimensional echocardiography apical five-chamber view, arrow demonstrating flail posteromedial leaflet of the aortic bioprosthesis; B: Transthoracic echocardiography with color flow Doppler parasternal long axis view, showing severe regurgitation across the degenerated aortic bioprosthesis.

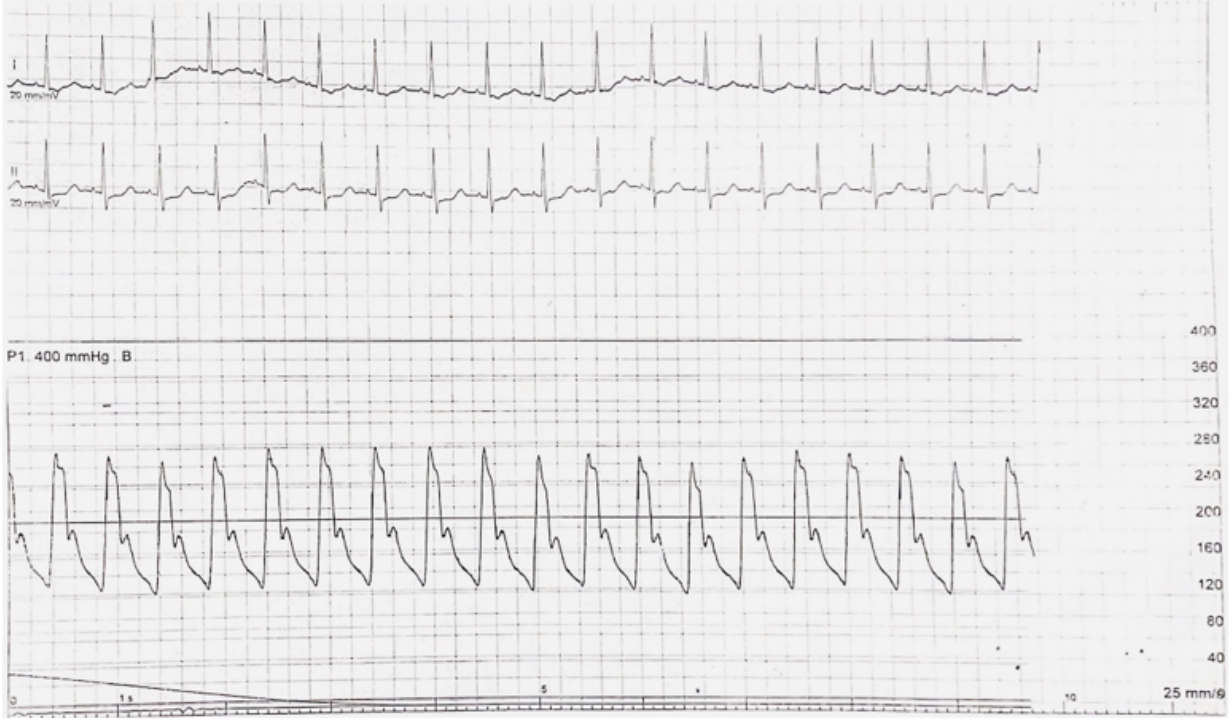

Figure 2 Femoral arterial pressure tracing when the patient's condition started to exacerbate, showing a pressure of about 270/130 mmHg.

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