

# AN INTENSIVE CARE PATIENT HAVING RECURRENT FLASH PULMONARY EDEMA: DIAGNOSIS AND TREATMENT

Mustafa YILDIZ MD

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*Flash pulmonary edema is a term characterized by sudden and recurrent episodes of dyspnea at rest resulting from acute pulmonary venous congestion in the presence of normal or well-preserved left ventricle systolic functions. This is usually associated with bilateral renal artery stenosis. It was reported here an intensive care unit patient, 60-year-old man, having normal ejection fraction and bilateral renal artery stenosis at bedside echocardiography and ultrasonography, respectively; and presented with recurrent episodes of flash pulmonary edema. He was successfully treated with intensive medical treatment (such as diuretic, dopamine, nitrat) under cardiac and respiratory monitorization in intensive care unit. Key words: Intensive care unit, flash pulmonary edema, treatment*

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## INTRODUCTION

Flash pulmonary edema is a life-threatening clinical presentation of severe atherosclerotic renovascular disease characterized by sudden and recurrent episodes of dyspnea at rest resulting from acute pulmonary venous congestion in the presence of generally normal left ventricle systolic functions (1,2). This is usually associated with bilateral renal artery stenosis (1,2). In this case report, we aimed to better understand the pathophysiology and response to treatment of flash pulmonary edema causing by bilateral renal artery stenosis in the intensive care unit.

## CASE

A 60-year-old man with a history of a poorly controlled hypertension and recurrent dyspnea, especially in the night, was admitted with pulmonary edema in the intensive care unit. His medication list included metoprolol 5 mg, valsartan 160 mg, amlodipine 10 mg, and aspirin 100 mg. He was a previous smoker. Arterial blood pressure was 210/100 mmHg, heart rate was 120 beat/min. There was S3 on

### From:

Sakarya University Internal Medicine and Cardiology,  
Sakarya, Turkey

### Address for reprints

Mustafa YILDIZ, MD  
Bayar Cad, Gulbahar Sok, Emniyet Sitesi, A Blok, A  
Kapisi, D6, Kozyatagi  
Istanbul/TURKEY  
Telefon: +905323711701  
E-mail: mustafayilldiz@yahoo.com

the cardiac examination. Pulmonary exam was remarkable for bilateral rales 2/3<sup>rd</sup> up both lung fields. His electrocardiogram showed ST segment depressions in V5, V6. Serum urea, creatinine, thyroid function tests and complete blood count were normal. Transthoracic bedside echocardiography and ultrasonography, performed in the coronary intensive care unit, revealed normal ejection fraction and bilateral renal artery stenosis, respectively. Previously coronary angiography was normal. The patient was monitoring for cardiac, respiratory and urinary control. Nasal oxygen and glyceryl trinitrate infusion (25 microgram/min increasing in steps of 25 microgram/min at 5 minute intervals until the desired drop in blood pressure, 150/90 mmHg, is achieved) were initiated. Also, furosemide, dopamine (renal doses) and captopril 25 mg was initiated to levels of blood pressure. Two days later, the blood pressure was normal (130/80 mmHg) and dyspnea didn't occur. After intensive care unit period, he was referred for renal revascularization for the bilateral critical renal artery stenosis.

## DISCUSSION

This current case is most important for understanding and treatment the sudden and recurrent flash pulmonary edema under the intensive care unit conditions. Usually, the disease occurs in the presence of bilateral obstructive renal atherosclerosis leading to recurrent episodes of acute pulmonary edema associated with acute systemic hypertension (3,4). The rapid onset of pulmonary edema, usually during the night, is typical of the disease (4). The diagnosis is usually confirmed by the amelioration of the symptoms after renal revascularization. Before the revascularization, the patients were treated with intensive medical treatment such as diuretic, dopamine, nitrat under cardiac and respiratory monitorization in intensive care unit, as in our case. The underlying pathophysiological mechanisms of flash pulmonary edema are not well understood. This mechanism may be associated with severe renal artery stenosis and volume overload. In the presence of critical bilateral renal artery stenosis, volume overload occurs and may cause flash pulmonary edema (4). The dip in blood pressure during night may reduce renal

perfusion in the presence of renal artery stenosis and induce the renin-angiotensin system (4).

In conclusion, the intensive care specialist must be attentive for the diagnosis and treatment of flash pulmonary edema.

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