

Unilateral Pulmonary Edema: A Rare Initial Presentation of Cardiogenic Shock due to Acute Myocardial Infarction

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Cardiogenic unilateral pulmonary edema (UPE) is a rare clinical entity that is often misdiagnosed at first. Most cases of cardiogenic UPE occur in the right upper lobe and are caused by severe mitral regurgitation (MR). We present an unusual case of right-sided UPE in a patient with cardiogenic shock due to acute myocardial infarction (AMI) without severe MR. The patient was successfully treated by percutaneous coronary intervention and medical therapy for heart failure. Follow-up chest Radiography showed complete resolution of the UPE. This case reminds us that AMI can present as UPE even in patients without severe MR or any preexisting pulmonary disease affecting the vasculature or parenchyma of the lung.

Key Words: Unilateral Pulmonary Edema; Acute Myocardial Infarction; Heart Failure; Mitral Regurgitation

INTRODUCTION

Cardiogenic unilateral pulmonary edema (UPE) is a rare clinical entity that presents diagnostic challenges and is associated with an independent increased risk of mortality. It should therefore be promptly recognized to avoid delay in treatment (1). Most cases of UPE reported in the literature occur on the upper right side and are caused by severe mitral regurgitation (MR) (2). Here we describe the case of a 79-yr-old man who, after presenting with cardiogenic shock due to extensive acute myocardial infarction (AMI) resulting from left main coronary artery disease, developed acute right-sided UPE with associated transient mild ischemic MR. The patient was successfully treated by percutaneous coronary intervention (PCI) and medical therapy for heart failure.

CASE DESCRIPTION

A 79-yr-old man, a current smoker and hypertensive, presented to the Emergency Department with altered mentality on December 4, 2010. For the previous hour, he had severe chest pain. His medical history was remarkable for hypertension and benign prostatic hyperplasia. On examination, the patient was

found to be in drowsy state, pale, diaphoretic, and cyanotic. His blood pressure was 65/40 mmHg, pulse 77 beats per minute, body temperature 36.0°C. Heart sounds were unremarkable. His initial oxygen saturation checked by pulse oximetry was 50% in room air. An electrocardiogram showed ST depression in V4-6 and II, III, aVF leads, QS pattern in V1-3 leads, ST elevation in aVR, and VPC bigeminy (Fig. 1). Chest X-ray revealed alveolar-interstitial infiltrates limited to the right lung (Fig. 2A). Laboratory tests gave white blood cell count 18,500/ μ L with 73% neutrophils, hemoglobin 13.8 g/dL, creatinine 1.3 mg/dL, sodium 140 mEq/L, potassium 5.0 mEq/L, B-natriuretic peptide 591 pg/mL, C-reactive protein (CRP) 7.56 mg/dL, myoglobin 119.4 ng/mL, creatine kinase isoenzyme MB 4.1 ng/mL and troponin I 0.11 ng/mL. Peak troponin I was 208.2 ng/mL. The patient was intubated and given ventilator support. Echocardiography at admission disclosed an ejection fraction of approximately 40% with global hypokinesia and mild MR (Fig. 3A). Anteroposterior left atrium (LA) diameter measured from the parasternal long axis view was 4.46 cm, and LA volume using the prolate ellipse method was 54 mL. With a diagnosis of cardiogenic shock due to AMI, the patient underwent emergency coronary angiography. This revealed significant stenosis from the distal left main coronary artery (LM) to the mid left an-

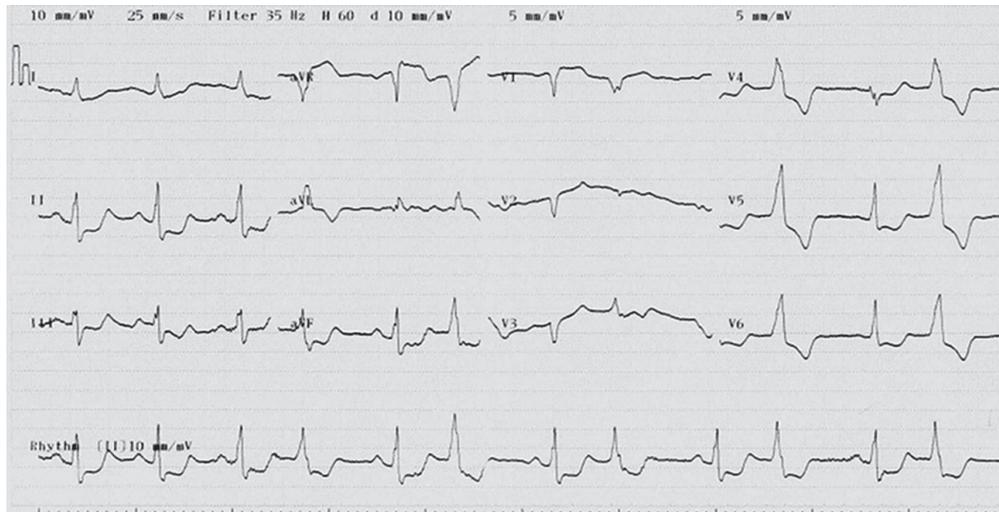


Fig. 1. Initial 12-lead electrocardiogram showing: ST depression in V4-6 and II, III, aVF leads, QS pattern in V1-3 leads, ST elevation in aVR and VPC bigeminy.

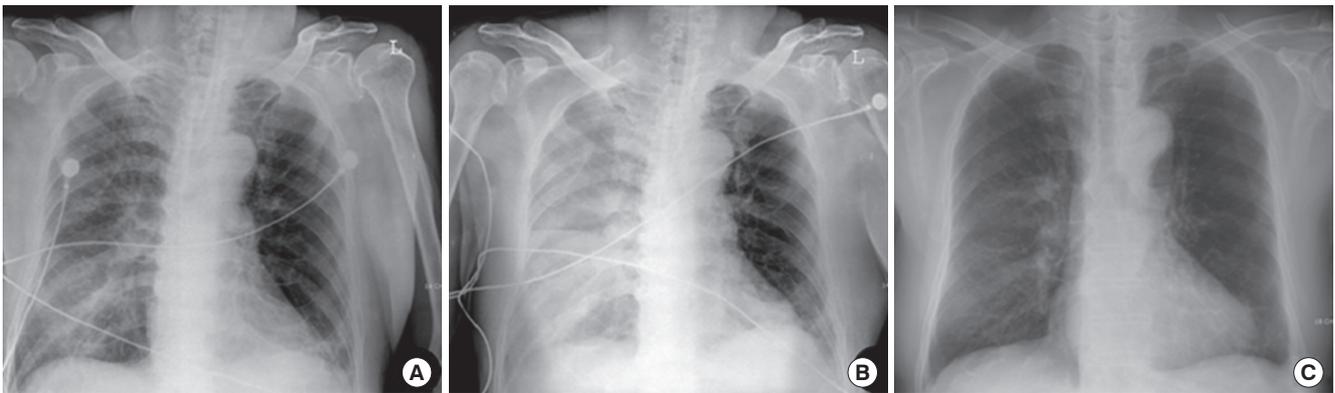


Fig. 2. Chest x-ray. (A) Anteroposterior radiograph of the chest at admission showing alveolar-interstitial infiltrates limited to the right lung. (B) Anteroposterior radiograph of the chest post-successful PCI showing a unilateral homogenous pulmonary opacity occupying the right lobe, and right pleural effusion. (C) Posteroanterior radiograph of the chest at discharge showing complete resolution of the unilateral pulmonary edema.

terior descending artery (LAD) with thrombolysis in myocardial infarction (TIMI) grade 2 flow and total occlusion at the ostium of the left circumflex artery (LCX) with TIMI grade 0 flow (Fig. 4A). PCI was performed using a 2.0×20 mm conventional balloon catheter followed by deployment of an Endeavor Resolute 2.75×18 mm stent at mid LAD, a Promus Element 3.5×28 mm stent at LM to proximal LAD, and an Endeavor Resolute 2.5×14 mm stent at proximal LCX with post-stent TIMI grade 3 flow (Fig. 4B). Dilation of the LAD and LCX by the complementary “kissing balloon” technique was also successfully performed. During PCI, mechanical support with intra-aortic balloon pump was not considered because blood pressure recovered and remained stable after hydration and catecholamine infusion with dopamine and dobutamine. Serial chest Radiography obtained for 3 days after the PCI demonstrated aggravation of a right-sided pulmonary edema (Fig. 2B). There was no evidence of renal failure or hypoalbuminemia. Although the patient’s temperature was normal, pneumonia could not be excluded in this el-

derly patient in the presence of a unilateral pulmonary infiltrate with effusion in combination with leukocytosis and an elevated CRP level, and treatment with intravenous antibiotics was initiated. A computed tomography scan performed on the third day after PCI revealed: a consolidation and ground-glass opacity in the dependent portion of the right lung, pleural effusion limited to the right lung, mild cardiomegaly with regurgitation of contrast material into the inferior vena cava, and no evidence of pulmonary artery and vein occlusion (Fig. 5). Diagnostic thoracentesis revealed transudate, and culture of pleural fluid was negative result.

The patient’s condition stabilized with adjustment of medical therapy for heart failure including diuretics, beta-blocker and angiotensin receptor blocker. Repeat chest Radiography showed complete resolution of the unilateral edema (Fig. 2C). Follow up echocardiography revealed a mildly improved left ventricular ejection fraction of 49% and improvement of mitral valve leaflet motion. MR was completely resolved (Fig. 3B). Antero-

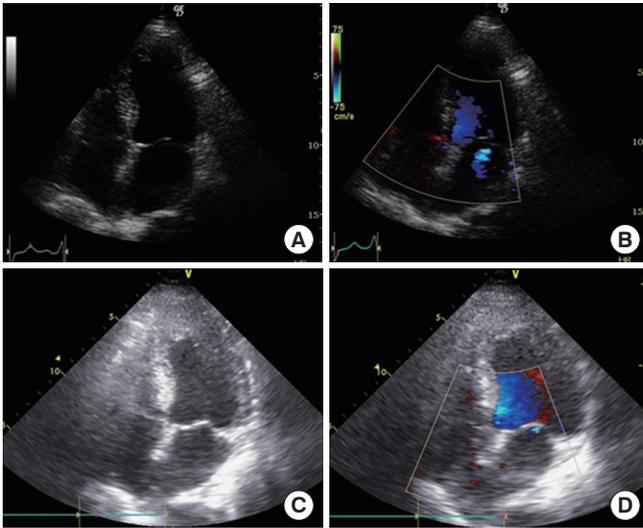


Fig. 3. Transthoracic echocardiography on admission and four days later after percutaneous coronary intervention (PCI). Echocardiography on admission showing global hypokinesia, mild mitral regurgitation (MR) and left atrium (LA) enlargement (LA volume, 54 mL) (A, B). Four days later after PCI, left ventricular contraction and LA enlargement have improved (LA volume, 34 mL), and MR has resolved (C, D).

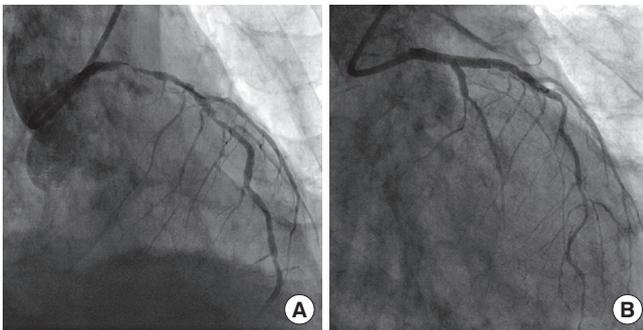


Fig. 4. Coronary artery angiography during the primary percutaneous coronary intervention. (A) Left coronary angiography showing significant stenosis from the distal left main coronary artery to the mid left anterior descending artery with thrombolysis in myocardial infarction (TIMI) 2 flow and total occlusion at the ostium of the left circumflex artery with TIMI 0 flow. (B) Final coronary angiography showing successful revascularization without periprocedural complications.

posterior LA diameter measured from the parasternal long axis view was 3.80 cm and LA volume was 34 mL. The patient was discharged uneventfully after 14 days of hospitalization.

DISCUSSION

Acute cardiogenic pulmonary edema is a critical condition associated with high mortality, and may be caused by a variety of cardiac diseases, including coronary artery disease (3). The usual radiographic findings in acute cardiogenic pulmonary edema are bilateral symmetrical opacities in the central zones of the lungs, resulting in the classic “butterfly shadow”. UPE is a rare entity that can be mistaken for other causes of unilateral infiltrate on chest Radiography, especially pneumonia. It has

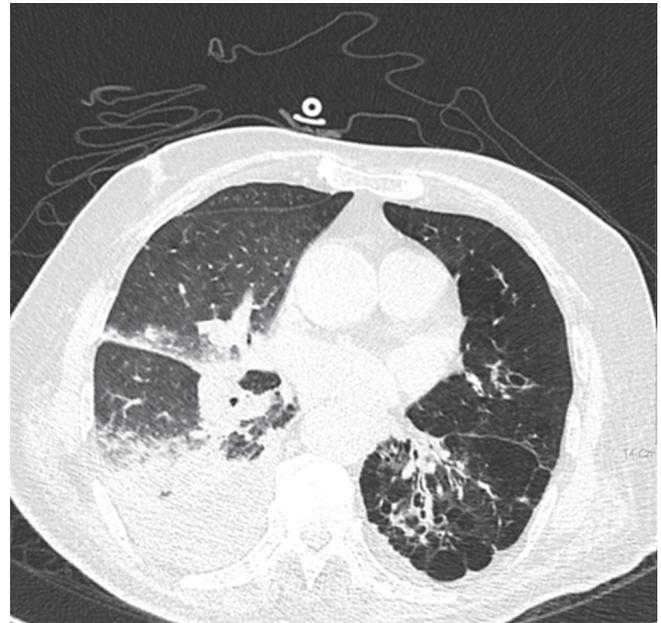


Fig. 5. Computed tomography scan revealing consolidation and ground-glass opacity in the dependent portion of the right lung, with pleural effusion limited to the right lung.

been reported after congestive heart failure, prolonged rest on one side in patients with cardiac decompensation or receiving large amounts of fluids, in cases of rapid expansion of the lungs after pleural effusion and pneumothorax; it is also seen in the normal lung in patients with unilateral pulmonary disease such as MacLeod syndrome and unilateral pulmonary artery hypoplasia or agenesis, pulmonary artery compression from aortic dissection or left ventricular pseudoaneurysm and pulmonary venous obstruction from mediastinal fibrosis. However, it is mainly reported in association with severe MR (1-4).

Most cases of UPE associated with left heart failure affect the right lung. A possible explanation is the poorer lymphatic drainage of the right lung by the small-caliber right bronchomediastinal trunk in comparison with that of the left lung by the large-caliber thoracic duct (5). Another explanation relates to the left-sided cardiac enlargement that develops in most patients with heart failure and that may physically impede blood flow in the left pulmonary artery, thereby reducing capillary volume (6). Nevertheless, severe MR remains the main cause of UPE. An MR jet affecting predominantly the upper right pulmonary vein, can lead to a larger increase in mean capillary pressure on the right side and, consequently, a greater degree of right acute pulmonary edema. The main mechanism of MR in UPE is mitral leaflet prolapse, but functional MR may also be involved (1, 2).

During the early phase of AMI, transient ischemic MR is common and sometimes causes hemodynamic compromise. However, when several chordae tendineae or papillary muscle rupture occurs, this can lead to abrupt hemodynamic deterioration with cardiogenic shock. It is important to have a high in-

dex of suspicion for acute MR in any patient with acute pulmonary edema in the setting of AMI, especially if left ventricular systolic function is well preserved (7).

A unilateral radiography pattern may lead to a false diagnosis of pneumonia and so delay management. Although the induction of an acute phase reaction and an elevated peripheral leukocyte count, especially of neutrophils, in patients with AMI has been reported to be related to the extent of myocardial infarction and with prognosis, the association of unilateral pulmonary infiltrates with leukocytosis and/or acute respiratory distress often leads to antibiotic therapy, despite the absence of fever, especially in the elderly (8, 9). Furthermore, patients with UPE present with a higher risk of death than patients with bilateral pulmonary edema, and delay in adequate treatment of UPE may be one explanation for this increased mortality (1). The absence of fever, a history of sudden onset of dyspnea, and elevated levels of B-natriuretic peptide, may help to differentiate UPE from other diagnoses (10). A murmur on examination can be useful, especially for organic MR. However, the intensity of the murmur is low and correlates poorly with the degree of regurgitation in acute ischemic MR (11). Echocardiography is useful in determining the severity of MR and its cause. Cardiogenic UPE can easily be mistaken for pneumonia or some other pulmonary pathological condition. Therefore, additional parameters such as the physical examination findings and the clinical course should also be taken into account. UPE should be promptly recognized to avoid delays in treatment that affect prognosis.

In this case, the patient was diagnosed with pulmonary edema despite the unilateral pulmonary infiltrate with effusion on the basis of computed tomography scan, pleural fluid analysis by thoracentesis and response to treatment. Cardiogenic shock developed due to extensive AMI involving the left main coronary artery, but no severe MR caused by papillary muscle or chordae tendineae rupture was observed. Acute myocardial ischemia could have exacerbated diastolic dysfunction with after load mismatch and possibly with reduced systolic function as well as transient ischemic MR due to papillary muscle dysfunction. When regurgitant volume increases suddenly due to transient ischemic MR, the acute rise in LA pressure can be transmitted back to the pulmonary circulation, generating pulmonary edema. UPE can occur not only because the direction of the MR jet affects predominantly the upper right pulmonary vein, but also because of differences between the mechanisms controlling tissue osmotic pressure in the two sides of the lung. For example, in the case of a sudden increase of LA pressure, the right lung parenchyma can develop edema due to the low excretory capability of its lymphatic drainage compared to that of left lung, precipitating an event of right-sided UPE (2, 12). Re-

covery of the ischemic papillary muscle and the limited number of episodes of left atrial hypertension, which are the main determinants of pulmonary edema after AMI, may have contributed to the resolution of the pulmonary edema with treatment over a few days (13). The lesson of this case is that it is important to acknowledge that AMI can present as UPE even in patients without severe MR or any preexisting pulmonary disease affecting the vasculature or parenchyma of a lung.

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