Acute Respiratory Distress Syndrome After Early Successful Primary Percutaneous Coronary Intervention Therapy in Acute Myocardial Infarction: A Case Report

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Acute respiratory distress syndrome (ARDS) is characterized by acute-onset dyspnea, diffuse bilateral pulmonary infiltration, low pulmonary capillary wedge pressure (PCWP), and an arterial oxygen tension/inspired oxygen fraction (PaO_2/FiO_2) ratio of less than 200 mmHg. Acute myocardial infarction (AMI), whether complicated by circulatory arrest, cardiogenic shock, and hypotension or not, was reported as an etiologic factor in the development of ARDS in the prethrombolytic era. In the thrombolytic era, two cases of AMI complicated with ARDS have been reported. ARDS in these two patients resulted from anaphylactic reaction to the thrombolytic agent and not from the hemodynamic consequences of AMI. Development of ARDS during the AMI period has not been reported after early successful primary percutaneous coronary intervention (PCI). Herein, we report a 61-year-old male patient with persistent chest pain who was diagnosed with Killip II anterior ST-segment elevation AMI. He was treated successfully with primary PCI 2.5 hours after the onset of chest pain. Unfortunately, on the third hospital day, acute-onset dyspnea (respiratory rate, 33 beats/min), fever (38.5°C), leukocytosis (white blood cell count, 18,360/μL), and diffuse bilateral pulmonary infiltration were noted. ARDS was diagnosed from the low PCWP (8 mmHg) and a PaO_2/FiO_2 of less than 200 mmHg (160 mmHg). No usual causes of ARDS such as infection, aspiration, trauma, shock, or drug reactions were noted. We assumed that, in this particular patient, the systemic inflammatory response syndrome frequently induced by AMI might have caused this episode of ARDS. This may imply that AMI itself is a possible etiology of ARDS.

Key Words: acute respiratory distress syndrome, acute myocardial infarction, primary percutaneous coronary intervention
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Acute respiratory distress syndrome (ARDS) was first described in 1967 by Ashbaugh et al in a heterogeneous group of patients who presented with severe respiratory failure [1]. Etiologies of ARDS can be divided into those associated with infection, such as sepsis, and those not linked to infection, including burns, hemorrhagic shock, aspiration, drug reaction, and acute pancreatitis [2–6]. Another rare etiology that has been reported is acute myocardial infarction (AMI) accompanied by circulatory arrest, cardiogenic shock, or hypotension [7]. In the prethrombolytic era, Yoshida et al reported a patient with uncomplicated AMI who developed ARDS [8]. In the thrombolytic era, two cases of AMI complicated with ARDS have been reported, in which the ARDS resulted from anaphylactic reaction to the thrombolytic agent and not

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from the hemodynamic consequence of AMI [9,10]. ARDS developing during AMI is rare and the etiology is still not clear. ARDS developing in patients with AMI after successful treatment with primary percutaneous coronary intervention (PCI) has not been reported. Herein, we report a 61-year-old male patient with Killip II ST-segment elevation AMI (STEMI) who received early successful primary PCI treatment but developed ARDS on the third day after AMI.

Case Presentation

A 61-year-old male with persistent chest pain and cold sweats was sent to our emergency room 1 hour after the onset of typical chest pain. A 12-lead electrocardiogram (ECG) showed ST-segment elevation in leads I, aVL, and V1–V6 with reciprocal ST-segment depression in leads II, III, and aVF. The pulse rate was 76 beats/min, blood pressure was 108/74 mmHg, and physical examination revealed a bilateral lower lung inspiratory crackle. These signs and symptoms suggested Killip II anterior wall STEMI. An anteroposterior view chest roentgenogram showed mild cardiomegaly with pulmonary congestion (Figure 1A). The white blood cell count (WBC) was 14,790/μL, hemoglobin concentration (Hb) was 16.1 g/dL, platelet count (PLT) was 197,000/μL, aspartate aminotransferase (AST) was 25 U/L, glutamate-pyruvate transaminase (GPT) was 21 U/L, blood urea nitrogen (BUN) was 20 mg/dL, serum creatinine (Cr) was 1.2 mg/dL, random sugar level was 192 mg/dL, concentrations of Na, K, and Cl were 136, 4.2, and 102 mmol/L, prothrombin time (patient/control) was 11.4/11.6 seconds (International Normalized Ratio, 0.95), partial thromboplastin time (patient/control) was 26.4/29.6 seconds, concentrations of serum creatine kinase (CK) and myocardial bound CK (CK-MB) were 101 and 4.4 U/L, troponin-I concentration was 0.024 ng/mL, C-reactive protein (CRP) was 75.7 mg/dL, cholesterol was 246 mg/dL, low-density lipoprotein cholesterol (LDL-C) was 154 mg/dL, high-density lipoprotein cholesterol (HDL-C) was 58 mg/dL, and triglyceride was 82 mg/dL.

Thirty minutes later, 1.5 hours after the onset of pain, primary PCI was performed under heparin and glycoprotein IIb-IIIa receptor antagonist treatment. Coronary angiography revealed 50% discrete stenosis of the distal left main artery, total occlusion of the proximal segment of the left anterior descending artery (LAD), 80% discrete stenosis of the middle segment of the left circumflex artery (Figure 2A), 30% discrete stenosis of the proximal segment of the right coronary artery, and 50% discrete stenosis of the posterior descending artery. The proximal part of the LAD was designated as the infarct-related artery (IRA). The IRA lesion was successfully recanalized by balloon angioplasty (door to balloon time, 1.5 hours) and stent implantation (Figure 2B).

Cardiac enzymes after admission showed a series of changes with a peak CK/CK-MB of 7,955/352.5 U/L and a peak troponin-I of 145.77 ng/mL at the 12th hour after the onset of chest pain. Echocardiography showed anteroseptal

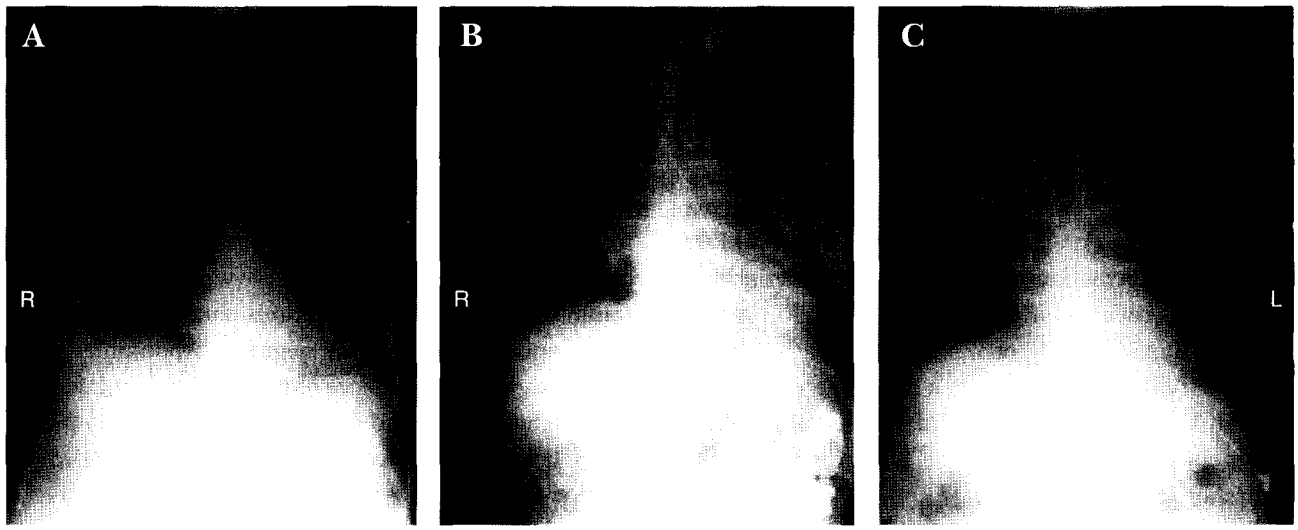


Figure 1. *Chest roentgenograms: (A) in the emergency room, showing mild cardiomegaly with pulmonary congestion; (B) on the 3rd hospital day, showing diffuse bilateral pulmonary infiltration with unchanged cardiac silhouette, clear costophrenic angle, no Kerley line, and no thickening of minor fissure; (C) on the 13th hospital day, showing partial resolution of diffuse bilateral pulmonary infiltration.*

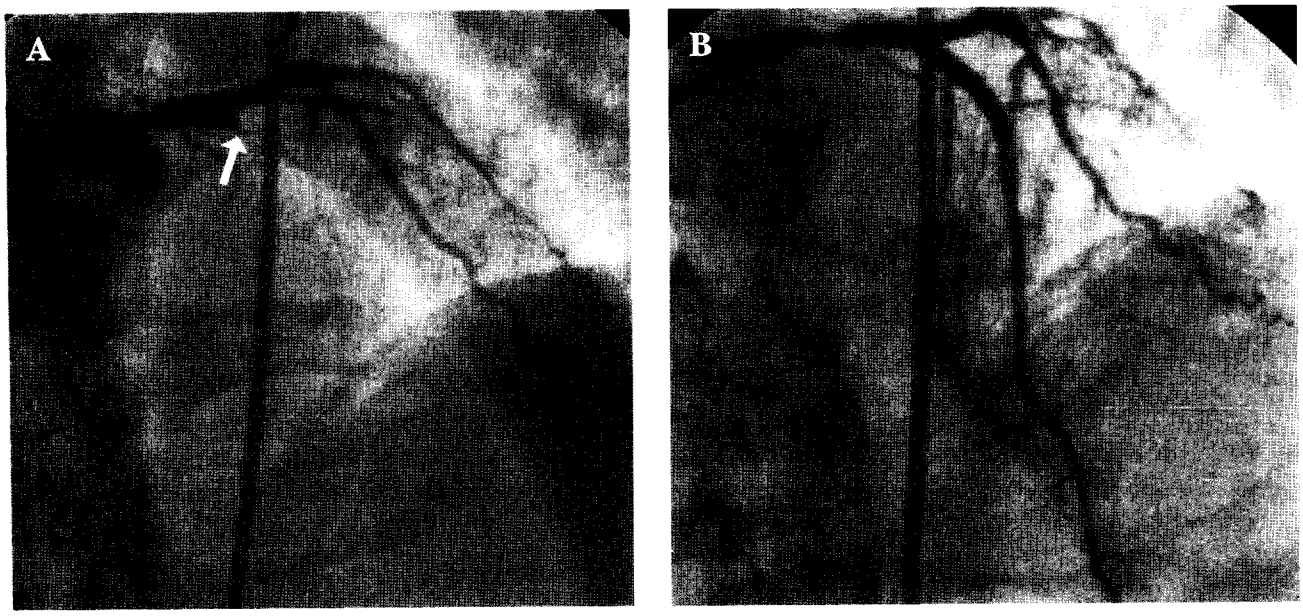


Figure 2. *Coronary angiograms: (A) left selective coronary angiography reveals 50% discrete stenosis of the distal left main artery, total occlusion of the proximal segment of the left anterior descending artery (LAD) (arrow), and 80% discrete stenosis of the middle segment of the left circumflex artery; (B) after successful primary percutaneous coronary intervention, left selective coronary angiography reveals no residual stenosis over the proximal segment of the LAD.*

wall hypokinesia and mild mitral and tricuspid regurgitation with fraction shortening of 28%. Unfortunately, on the third hospital day, cough with a little white sputum and respiratory distress with dyspnea (respiratory rate, 33 beats/min) were noted. Pulse rate was 110 beats/min, blood pressure was 102/68 mmHg, and body temperature was 38.5°C. Cardiac examination showed no new pathologic murmur, no jugular vein engorgement, and no S3 gallop, but moist rales could be heard over the entire lung field. Laboratory tests revealed the following: WBC, 18,360/μL; Hb, 15.5 g/dL; PLT, 178,000/μL; CRP, 107.7 mg/dL; BUN/Cr, 30/0.9 mg/dL; CK/CK-MB, 2,642/71.6 U/L; and troponin-I, 71.95 ng/mL (continuous decrease). Oxygen saturation (SaO_2) was less than 90% under 6 L nasal oxygen inhalation. A non-rebreathing mask (100%, 15 L) was used to maintain the SaO_2 at more than 90%. Arterial blood gas analysis revealed a pH of 7.434, a partial pressure of carbon dioxide (PCO_2) of 37.5 mmHg, a partial oxygen pressure (PaO_2) of 144.2 mmHg, a bicarbonate concentration (HCO_3) of 24.5 mmol/L, and SaO_2 of 99.3%. Chest roentgenogram showed diffuse bilateral pulmonary infiltration (Figure 1B).

Pulmonary edema was suspected and intravenous diuretic and nitrate were administered, but the dyspnea persisted and diffuse infiltration on roentgenogram was not ameliorated. In addition, the amount of urine was maintained at more than 50 mL/hour, blood pressure was more than 90/60 mmHg, and orthopnea, frothy sputum, and paroxysmal nocturnal dyspnea were absent. Furthermore, acute mitral regurgitation and ventricular septal rupture were excluded by echocardiography. Therefore, pulmonary edema secondary to low cardiac output caused by AMI was unlikely. A balloon-tipped, flow-directed pulmonary arterial catheter (Swan-Ganz) was introduced and revealed a cardiac index of 2.84 L/min/m^2, pulmonary capillary wedge pressure (PCWP) of only 8 mmHg, and a systemic vascular resistance index of 2,204 dynes/sec/cm^{-5}/m^2. ARDS was highly suspected from the clinical presentation including low PCWP, a PaO_2/inspired oxygen fraction (FiO_2) of 160 mmHg, diffuse bilateral pulmonary infiltration, and acute onset of dyspnea. Possible etiologies causing ARDS were surveyed. There were no aspiration or trauma episodes. Multiple cultures obtained from blood, urine, and sputum were all negative. Abdominal sonography showed no evidence of intra-abdominal infection. Although there was no evidence of infection, empirical antibiotics were used because of a high risk of superinfection. Intravenous steroid was temporarily used for 2 days. Oxygen therapy with a non-rebreathing mask (15 L, 100%) was used for 6 days followed by a venturi mask (40%, 8 L) for 4 days, before finally shifting to nasal cannula (2 L) use. Follow-up chest roentgenogram on the 13th hospital day showed partial

resolution of diffuse bilateral pulmonary infiltration (Figure 1C). He was weaned from the oxygen supply on the 16th hospital day and discharged without event on day 17.

Discussion

ARDS can occur with various disorders, divided into those with direct injury to the lung and those causing indirect injury in the setting of a systemic disease. Direct causes include aspiration pneumonia, pulmonary contusion, toxic inhalation, and near drowning. Indirect causes include sepsis, shock, severe extrathoracic trauma, multiple fractures, drug overdose, multiple transfusions, cardiopulmonary bypass, eclampsia, burns, disseminated intravascular coagulation, acute pancreatitis, and air or amniotic fluid emboli [2–6]. As shown in the Table, there are rare case reports suggesting that ARDS may occur during AMI. Possible etiologies of ARDS were reported in six of these cases while no specific etiology was found in one case. In our case, there was no sepsis, trauma, gastric aspiration, or blood transfusion during the admission period. Multiple cultures obtained from sputum, urine, and blood were negative. All these data indicated that the usual etiologies of ARDS were not present in this patient. Our patient had undergone primary PCI within the golden time so that the degree of cardiac injury caused by AMI might have been less than that without reperfusion therapy. The benefit of this early intervention on cardiac function was supported by the lower normal level of the cardiac index and echocardiographic fraction shortening and normal PCWP. However, ARDS occurred under this normal hemodynamic status. Since there was no evidence of cardiogenic shock or other apparent etiologies, the ARDS in this patient may be attributed to the AMI disease process itself.

Inflammatory response leading to organ dysfunction and failure continues to be a major problem after tissue injury in many clinical conditions such as sepsis, acute pancreatitis, hemorrhagic shock, trauma, and severe burns. The systemic inflammatory response syndrome (SIRS) is an entirely normal response to injury. Systemic leukocyte activation is a direct consequence of SIRS and, if extensive, can lead to distant organ damage and multiple organ dysfunction syndrome (MODS) [11–13]. Acute lung injury seen clinically as ARDS is a major manifestation of MODS of various etiologies. Inflammatory mediators such as interleukin-1 (IL-1), IL-6, tumor necrosis factor-α (TNF-α), and intercellular adhesion molecule-1 (ICAM-1) play a key role in the pathogenesis of ARDS [14]. A growing body of experimental evidence suggests that inflammation is involved in the pathogenesis of acute coronary syndromes (ACS) and influences their clinical evolution. In patients with ACS, the following systemic signs of inflammatory reaction have been widely reported: activated circulating inflammatory cells (neutrophil, monocytes, and lymphocytes) and increased concentrations of inflammatory mediators such as IL-1, IL-6, TNF-α, ICAM-1, and CRP [15–19]. In our case, although the true etiology of the ARDS was undetermined, we assumed that SIRS induced by AMI might have caused systemic leukocyte activation, which in turn released extensive inflammatory mediators and caused the ARDS.

Treatment of ARDS includes respiratory support, underlying disease management, and therapy with pharmacologic agents. Four phases have been reported in the development of ARDS: the at-risk phase, acute

Table. Case reports of acute myocardial infarction (AMI) complicated with acute respiratory distress syndrome (ARDS)

Reference	Age/Sex	Type/Location of AMI	Mode of reperfusion	Possible etiology of ARDS
Keren et al [7]	83/M	NA/NA	No reperfusion therapy	Cardiac arrest
Keren et al [7]	63/F	NA/Anterior wall	No reperfusion therapy	Aspiration, cardiogenic shock
Keren et al [7]	56/M	NA/Inferoposterior wall	No reperfusion therapy	Hemorrhagic shock
Keren et al [7]	48/M	NA/Anterior wall	No reperfusion therapy	Smoke inhalation
Yoshida et al [8]	53/F	STEMI/Acute wall	No reperfusion therapy	No possible etiology
Le et al [9]	56/M	STEMI/Inferior and RV involvement	Thrombolytic therapy with APSAC	APSAC drug reaction
Tio et al [10]	54/M	STEMI/Anterior wall	Thrombolytic therapy with streptokinase	Streptokinase drug reaction

M = male; NA = not available; F = female; STEMI = ST-segment elevation myocardial infarction; RV = right ventricle; APSAC = anisoylated plasminogen streptokinase activator complex.

inflammatory phase, proliferative phase, and fibrotic phase. Drug therapy needs to be considered according to the phase of the disease progression [14,20]. Glucocorticoid may play a role in the acute inflammatory and fibroproliferative phases [21,22]. However, treatment with glucocorticoid in AMI patients may interfere with healing of the injured myocardium. Our patient temporarily received intravenous steroid therapy for 2 days and dyspnea was improved. In addition, aggressive oxygen therapy with non-rebreathing and venturic masks was given and standard treatment regimens for STEMI were administered. The patient recovered without event and was discharged after 17 days of management.

In conclusion, even though our patient had undergone successful primary PCI in the golden time of AMI, ARDS developed without apparent cause. We assumed that SIRS, frequently induced by AMI, might have caused this episode of ARDS, implying that AMI itself is a possible etiology of ARDS.

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急性心肌梗塞的病人在接受早期成功的緊急心導管介入術治療後發生急性呼吸窘迫徵候群

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急性呼吸窘迫徵候群的特徵就是喘、兩側瀰漫性的肺部浸潤、低的肺部楔形壓和血氧濃度比上給氧分壓小於 200 毫米汞柱。在血栓溶解治療前期，急性心肌梗塞不論是否併發心跳停止、心因性休克或低血壓均被報告過是急性呼吸窘迫徵候群的病因之一。在血栓溶解治療期，也有二篇急性心肌梗塞病人併發急性呼吸窘迫徵候群的報告，只不過其病因歸因於血栓溶解劑所引起的過敏反應而不是急性心肌梗塞所引發的血行動力學變化所致。在緊急心導管介入術的治療期，急性心肌梗塞的病人在接受早期成功的緊急心導管介入術治療後發生急性呼吸窘迫徵候群的案例，尚未被報告過。這裡我們報告一位 61 歲的男性病人，因持續性的胸痛而被診斷為 Killip 二級的 ST 節段上升的前壁急性心肌梗塞。他在胸痛 2.5 小時後接受緊急心導管介入術，並成功地打通其梗塞的血管。但不幸地，在住院的第三天，病人喘 (呼吸速率 33 下/分鐘)、發燒 (38.5°C)、白血球升高和胸部 X 光顯示為兩側瀰漫性的肺部浸潤。那時，肺部楔形壓只有 8 毫米汞柱再加上其血氧濃度比上給氧分壓也只有 160 毫米汞柱而被診斷為急性呼吸窘迫徵候群的病人。在這個病人身上，並沒有常見會發生急性呼吸窘迫徵候群的病因像感染、嗆到、外傷和藥物不良反應等被發現。我們推測急性心肌梗塞本身所引發的全身性的發炎反應是引發這次急性呼吸窘迫徵候群的主因。這也暗示急性心肌梗塞本身可能是引發急性呼吸窘迫徵候群的病因之一。

關鍵詞：急性呼吸窘迫徵候群，急性心肌梗塞，緊急心導管介入術

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