ADULT RESPIRATORY DISTRESS SYNDROME IN A PATIENT
WITH UNCOMPLICATED ACUTE
MYOCARDIAL INFARCTION

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A 53-year-old woman was admitted with a diagnosis of acute extensive anterior myocardial infarction. Pulmonary capillary wedge pressure was 17 mmHg, and cardiac index 2.4 l/min-m². The patient was successfully treated with diuretics and nitrates but on the fifth hospital day moist rales were noted over the entire lung field. A chest roentgenogram showed diffuse bilateral pulmonary infiltration with unchanged cardiac silhouette. Arterial oxygen partial pressure was reduced to 45 mmHg under 3 liters of nasal oxygen inhalation. Pulmonary capillary wedge pressure was 8 mmHg. Diffuse infiltration was not ameliorated by intravenous injection of diuretics. The diagnosis of adult respiratory distress syndrome was finally established by successful treatment with steroids.

(Jpn Circ J 1992; 56: 1253—1256)

ADULT respiratory distress syndrome usually occurs in patients with circulatory shock. Acute myocardial infarction associated with cardiogenic shock is occasionally accompanied by this syndrome. However, to our knowledge, there are no reports of patients with uncomplicated acute myocardial infarction developing adult respiratory distress syndrome. We report a patient with acute myocardial infarction, in whom no conditions known to be possible etiologies of adult respiratory distress syndrome were present.

CASE REPORT

A 53-year-old woman without preinfarction angina pectoris was admitted with a 1 h history of anterior chest oppression. A 12-lead electrocardiogram showed ST-segment elevation in leads I, aVL, and V2-5 (Fig. 1). The pulse was 80 beats per min, the blood pressure 122/80 mmHg, and physical examination was normal except for the presence of xanthisms of the bilateral eyelids. Laboratory data on admission revealed a white blood cell count of 9,900/mm³ and total cholesterol level of 230 mg/dl. Other laboratory data were within normal limits. The time of a peak creatine kinase activity of 2747 IU/l was 18 h after the onset of symptoms of acute myocardial infarction. The chest roentgenogram was normal (Fig. 2). A Swan-Ganz catheter was introduced and a slightly elevated pulmonary capillary wedge pressure of 17 mmHg was obtained along with a normal cardiac index of 2.4 l/min-m². The patient was treated with diuretics and

Key words:
Adult respiratory distress syndrome
Acute myocardial infarction

(Received August 2, 1991; accepted May 7, 1992)
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nitrates and the clinical course was stable in Forrester’s subset I.

On the fifth hospital day she complained of respiratory distress with dyspnea and tachypnea. Moist rales were noted over the entire lung field. A chest roentgenogram showed diffuse bilateral pulmonary infiltration (Fig. 2) and blood gas analysis revealed severe hypoxemia with a $\text{P}_{\text{a}}\text{O}_2$ of 45 mmHg under 3 liters of nasal oxygen inhalation. Left ventricular failure was suspected from these laboratory data. However, the Swan-Ganz catheter revealed that the pulmonary capillary wedge pressure remained at 8 mmHg, the chest roentgenogram showed a normal cardiac silhouette, no increased pulmonary markings, and no Kerley-B lines. The diffuse infiltration was not ameliorated by the administration of catecholamines and diuretics, leading to a diagnosis of adult respiratory distress syndrome. The patient was prescribed methylprednisolone 0.5 g per day but despite the initiation of treatment there was no improvement in hypoxemia and diffuse pulmonary infiltration over subsequent days. The dosage of methylprednisolone was therefore increased to 2 g per day (Fig. 3). On the 14th hospital day, chest roentgenogram showed no infiltration. Cardiac catheterization was performed 2 months after the onset of acute myocardial infarction. Hemodynamic measurements were normal, pulmonary capillary wedge pressure was 12 mmHg and cardiac index was 2.5 l/min·m$^2$. A left ventriculogram obtained in the 30° right anterior oblique view showed akinesis of anterolateral and apical walls. The left ventricular ejection fraction was 38%. Coronary arteriography was performed by Judkins technique after a sublingual administration of nitroglycerin. The proximal part of the left anterior descending coronary artery had been recanalized with a residual stenosis of 25%.

DISCUSSION

In 1967, Ashbaugh and coworkers described for the first time acute respiratory distress in 12 patients who had a variety of manifestations, such as severe dyspnea, tachypnea, hypoxemia, loss of lung compliance and diffuse alveolar infiltrations that were refractory to respiratory therapy. Adult respiratory distress syndrome is attributable to variable etiologies. Cardiogenic shock, hypotension and/or multiple nonpulmonary organ failure in patients with acute myocardial infarction can lead to adult respiratory distress syndrome.

The most crucial thing in diagnosing adult respiratory distress syndrome is to exclude the possibility of association of heart failure. First, elevated pulmonary capillary wedge pressure on preceding days may have led to pulmonary congestion as a late effect. Second, the slightly decreased cardiac index on the fifth hospital day could have caused the documented hypoxemia. Furthermore, because it is reported that steroids may improve cardiac function through a mechanism of up-regulation of $\beta$-receptors of myocardium, the beneficial effect of the drug may be attributable to the improvement of heart failure. Thus, it appears to be difficult to exclude completely the possibility of

the hypoxemia secondary to heart failure. However, it may be logically concluded that the hypoxemia is not due to heart failure, because the amelioration of pulmonary infiltration and hypoxemia was caused not by the administration of catecholamines and diuretics but by the injection of steroids.

Some predisposing conditions resulting in the adult respiratory distress syndrome deserve comment. First, the possibility of pulmonary embolism must be considered. On the fifth hospital day, there were no electrocardiographic findings indicative of right ventricular overload and the pulmonary artery pressure was 27/6 mmHg. Thus, although a pulmonary perfusion study was not performed, it is unlikely that our patient had pulmonary embolism. Second, drug-in-
duced allergic response is considered to be an alternative etiology of the syndrome. However, the possible causative drugs such as nitrofurantoin, lidocaine and thiazide were not prescribed to our patient. Her manifestations and laboratory findings also precluded allergic response to drugs.

The clinical course of our patient was relatively mild compared to that of most patients with this syndrome. Although the efficacy of steroids in this syndrome is still controversial, prompt treatment with such agents might, at least in part, explain the good prognosis of our patient.

In our case, adult respiratory distress syndrome was accompanied by uncomplicated acute myocardial infarction. Although the etiology in our patient is unknown as mentioned above, early diagnosis and prompt treatment with steroids might have been effective. The direct measurement of the pulmonary capillary wedge pressure appears to be crucial in differentiating left ventricular failure from adult respiratory distress syndrome. Unless this syndrome is recognized by the physician, the symptoms and signs may be thought to be the sequela of acute myocardial infarction.

REFERENCES