Adult Respiratory Distress Syndrome in the Course of Acute Myocardial Infarction*

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The diagnosis of adult respiratory distress syndrome (ARDS) has been made in our intensive coronary care unit in four patients during the course of acute myocardial infarction (AMI). In all four patients, the syndrome manifested itself either after resuscitation or after a transient hypotensive state. In two of the patients none of the conditions known to be possible etiologies of ARDS was present; in the third, smoke inhalation preceded; and in the fourth, aspiration followed the AMI. The clinical and x-ray pictures were indistinguishable from acute left heart failure, the PaO₂ levels were about 40 mm Hg, and a low pulmonary arterial wedge pressure was measured in all cases. Positive end-expiratory pressure was used successfully, combined with other therapeutic measures, and three patients recovered from the ARDS. The association of ARDS and AMI carries a grave risk in view of the additional damage that may be caused by the severe hypoxemia to the already compromised myocardium. The AMI, if complicated by circulatory arrest, cardiogenic shock, or hypotension, seems to be an etiologic factor in the development of ARDS and it should be added to the growing list of conditions that may give rise to this new syndrome.

Adult respiratory distress syndrome (ARDS) is a relatively new entity, induced by acute injury of the lungs, and is characterized by pulmonary edema accompanied by a low pulmonary artery wedge pressure (PAWP). The ARDS has been reported to follow chemically-induced lung injury, oxygen toxicity, chest trauma, disseminated intravascular coagulation, septic shock, and other situations. As far as we know, there are no previously reported patients in whom ARDS accompanied the acute stage of myocardial infarction (AMI). Low PAWP and pulmonary edema during the course of AMI were, however, described by DeLuiz et al. and Stein and co-workers in patients following crystalloid fluid overload; these authors suggested that this situation was consequent to the reduction in plasma colloid osmotic pressure. McHugh et al. found a good correlation between elevated PAWP and the clinical and radiologic signs of pulmonary edema in AMI, but in three patients with severe hypoxemia, shock, and severe pulmonary congestion, they could not find an explanation for the presence of a low PAWP.

We report on our experience with four patients with AMI who experienced clinical pulmonary edema; concomitantly low PAWP was measured and ARDS was diagnosed.

METHODS

Pulmonary arterial pressure (PAP) and PAWP were measured in all patients through a Swan-Ganz catheter and were recorded using Statham pressure transducers, zeroing done in all patients in the standard fashion. Cardiac outputs were calculated by the Fick formula. Blood pressure (BP) was monitored through a peripheral arterial catheter. Blood gas values were determined with the use of a blood gas analyzer. Volume-controlled ventilators (MA 1), with an average tidal volume of 10 ml/kg, were used. The positive end-expiratory pressure (PEEP) applied ranged from 5 to 12 cm H₂O. The criteria for its level being the blood pressure, PaO₂, PAWP, and cardiac output. The PaO₂/FIO₂ ratio was used for expressing change in arterial oxygen tension. To evaluate the arteriovenous shunting, the alveolar-arterial oxygen tension difference (D{(A-a)O₂}) was measured after 15 minutes of 100 percent O₂ breathing.

CASE REPORTS

CASE 1

A 83-year-old man was brought to the emergency room while in cardiorespiratory arrest (CRA), 24 hours after chest pain started. After a successful cardiorespiratory resuscitation (CRR), the diagnosis of AMI was made. The BP was gradually restored with dopamine, diluted in 400 ml of crystalloid fluids. There were no signs of aspiration. About two hours later, the clinical picture of pulmonary edema developed and chest x-ray film was also consistent with this diagnosis. A Swan-Ganz catheter was introduced and a high PAP (56/40 mm Hg) was measured; at that stage, PAWP could not be obtained for technical reasons. A decrease of PaO₂ to 60 mm Hg was noted. After the increase of FIO₂ to 0.4, the administration of diuretics, and restriction of fluids, the clinical picture improved and PaO₂ rose to 120 mm Hg. A few hours later, a second CRA was managed successfully, but pulmonary edema developed once again, and the PaO₂ dropped to 42 mm Hg. In spite of intubation and of the use of FIO₂ of 0.4 and 1, diuretics, and digitalis, no improvement in blood gas levels was achieved. At this stage, PAWP was 5 mm Hg, the pulmonary arterial diastolic pressure, 16 mm Hg; central venous pressure (CVP) was high, 19 cm H₂O; and the systolic BP, 100 mm Hg (Fig 1). In view of the persisting clinical picture of pulmonary edema associated with the low PAWP, the diagnosis of ARDS was made, and the patient was treated with corticosteroids, albumin, PEEP, and antibiotics. Fluid administration was carried out with strict supervision.
of PAWP, cardiac output, urinary output, etc. During the next hours, the PaO₂ rose to 60 mm Hg and the FIO₂ could be reduced to 0.4. Eight hours later, left heart failure gradually developed, and the PAWP rose to 25 mm Hg. Another CRA developed, and shortly thereafter, the patient died. The clinical and laboratory findings of this patient are summarized in Figure 2.

CASE 2

A 63-year-old woman, who underwent hemicolectomy six months earlier, was admitted immediately after a successful CRR. A left pneumothorax was found, induced by the resuscitation. Signs of aspiration were also noted. The diagnosis was anterior wall AMI, and the patient was found to be clinically in shock. Her hemoglobin value was 5.4 gm/100 ml, and hematocrit, 18 percent. No rales were heard over the lungs except for the left base, where, several hours later, aspiration pneumonia was diagnosed. No signs of bleeding from the gastrointestinal tract were noticed. A Swan-Ganz catheter was immediately introduced, and the PAWP was found to be low (6 mm Hg). The PaO₂ was 140 mm Hg with FIO₂ of 0.65. The treatment during the next hours consisted of 500 ml of crystalline fluids, dopamine, and blood transfusion of 2 units of packed cells; diuretics were not given. The pneumothorax was treated with intercostal drainage. The BP gradually rose to 100/60 mm Hg. After eight hours, fulminant pulmonary edema suddenly developed with extreme respiratory distress, expectoration of huge amounts of white froth through the mouth and nose, cyanosis, and rales all over the lungs. Even now, the PAWP remained low and the average value was 8 mm Hg. X-ray film showed "white lungs." The diagnosis of ARDS was made. Treatment consisted of PEEP, fluid replacement, corticosteroids, albumin, antibiotics, and supportive measures. The patient's condition improved progressively. The use of PEEP did not complicate the therapy of the pneumothorax. Four days later, left ventricular failure developed and was treated accordingly. On the sixth day, the use of PEEP was stopped, and the patient was transferred to the general ward. About a week later, the patient developed severe Proteus morgagni pneumonia and died on the next day. The clinical and laboratory findings of this patient are summarized in Figure 3.

CASE 3

A 56-year-old man had syncope and CRA, and one half hour prior to admission, underwent CRR. AMI, inferior and true posterior wall, was diagnosed. The patient was in hypovolemic shock with a PAWP of 5 mm Hg and markedly decreased blood gas values, which were corrected by administration of O₂ through a Venturi mask. There were no signs of aspiration. Crystalloid fluids were administered under close monitoring of PAWP, maintaining a meticulous fluid balance. During the next days, the patient's condition improved gradually. On the fourth day, diuretics were given because of transient left heart failure (rise in PAWP).

During the next day, fluid balance was kept slightly nega-
tive, and the PAWP gradually decreased. No diuretics were administered on this day. On the sixth day, acute respiratory distress started and the diagnosis of pulmonary edema was clinically made, but the PAWP was low (5 mm Hg). The PaO₂ was 52 mm Hg, with FIO₂ of 0.8 and an extreme arteriovenous shunting through the lungs was measured. The diagnosis of ARDS was made and treatment with PEEP and other measures was applied. The clinical picture and the severe hypoxemia improved gradually. On the 11th day, the patient was breathing spontaneously, was discharged from the ICU a few days later, and left the hospital after one more week. The clinical and laboratory findings are summarized in Figure 4.

**Case 4**

A 48-year-old man had stayed in a burning room for some minutes. Shortly thereafter, he “fainted.” He did not vomit, and no aspiration was noted. First aid was delivered to him by his lay friends on the way to the hospital, where the diagnosis of anterior AMI was made. His BP was normal and his treatment consisted of O₂ administration and only minute amounts of fluids were given, to keep the IV line open. No diuretics were given. A few hours later, he developed a systolic BP of 80 mm Hg and the CPV was 10 cm H₂O. Following fluid administration, BP rose to 100 mm Hg and general condition improved. Eight hours later, BP fell again, he turned cyanotic, severely hypoperfused, and with an FIO₂ of 1, his PaO₂ was 50 mm Hg. The CVP was moderately elevated (15 cm H₂O and the PAWP was 4 mm Hg). The diagnosis of ARDS was made, and the patient was treated according to the principles mentioned previously. The PEEP was stopped after six days, he was extubated after another two days, and left the hospital 28 days after admission in a good clinical condition. The clinical and laboratory findings are summarized in Figure 5.

**Discussion**

The clinical manifestations of ARDS are similar to pulmonary edema induced by acute left heart failure, and this may be the reason that when acute dyspnea, cyanosis, rales over the lungs, expectoration of froth, etc, occur soon after an AMI, left heart failure is usually diagnosed. However, one hemodynamic feature differentiates left heart failure from ARDS, i.e., the high PAWP in the former and the low or normal pressure in the latter. A less striking difference is the hypoxemia, which in ARDS is much more refractory to or less correctable by O₂, and the entire clinical picture in ARDS is much more persistent and slower to clear.

The CVP is a disappointing parameter in both AMI and ARDS, as it expresses the functional state of the right ventricle only, which may be in disparity with left ventricular function. X-ray differentiation between left heart failure and ARDS is also impossible. Furthermore, the possibility of a “therapeutic phase-lag,” i.e., the persistence of a...
still congested appearance of the lungs on the x-ray film at a time when diuretics given for the heart failure had already lowered the PAWP,\textsuperscript{18} should also be considered. Such a situation could be suspected only in case 1 in our series as this was the only one who received diuretics prior to the ARDS. However, this possibility is not seriously suspected even in this case, since the typical white-lungs in the x-ray film (Fig 1) and low PAWP (Fig 2) appeared with the clinical picture of pulmonary edema and a sharp decline in the PaO\textsubscript{2} as well.

In two of the patients presented here, etiologies previously described for ARDS were associated with the AMI and shock, \textit{i.e.}, aspiration in case 2 and smoke inhalation in case 4. However, in the other two patients, no such additional conditions were present, and thus, one can certainly assume that in these two patients and possibly in the two others as well, the cardiogenic or hypovolemic shock of the AMI and/or the CRR alone resulted in injury to the alveologcapillary membrane, leading to ARDS. This is not entirely surprising, as it has already been shown that both hypovolemic and hypovolemic states may lead to ARDS.\textsuperscript{2,11,12}

The use of PEEP in all our patients was beneficial and no significant decrease in cardiac output was observed with pressures up to 12 cmH\textsubscript{2}O. Apparently, at these levels of PEEP, cardiac output is less influenced as long as a good filling pressure of the left ventricle is maintained.\textsuperscript{14,19} Although two of our patients developed acute left heart failure while on PEEP, we tend to connect this to a deterioration in left ventricular function in patients who are already in a delicate myocardial functional state. Following the treatment for heart failure, the clinical condition of case 4 improved in spite of continuation of PEEP, while in case 1, rapidly progressing heart failure culminated in death, in spite of trials to reduce PEEP.

All our patients had low PAWP, normal or low BP, and reduced cardiac output while ARDS was diagnosed. These parameters pointed to a hypovolemic state, probably induced by the accumulation of extravasated fluids in the lungs, and therefore, volume load was immediately started. Although Ashbaugh and other authors\textsuperscript{1,4} advised extreme caution against overhydration in ARDS, we had to use large amounts of fluid in order to restore the left ventricular filling pressure to the point where cardiac output became adequate. After restoring PAWP, BP, and cardiac output to levels compatible with life, the amount of fluids was restricted to replacement only.

All above mentioned therapeutic measures, such as PEEP, volume load, high molecular weight substances, etc, may have a deleterious effect on left ventricular function and may precipitate failure in patients with AMI. However, the decrease of the PaO\textsubscript{2} to critical values in such patients carries excessive danger, in view of the vital role of a satisfactory oxygenation to the acutely compromised myocardium. Therefore, the importance of early diagnosis of any process that may interfere with the PaO\textsubscript{2} level of patients with AMI is obvious, and immediate treatment is essential.

\textbf{REFERENCES}