Disturbance of Pulmonary Gas Exchange in Patients with Right Ventricular Infarction*

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To evaluate the difference in pulmonary gas exchange in patients with and without right ventricular infarction, 147 consecutive patients with their first inferior wall Q-wave acute myocardial infarction were studied. Thirty-nine patients (group 1) had electrocardiographic evidence of right ventricular infarction and it was absent in 108 patients (group 2). A significantly wider alveolar arterial oxygen difference and higher roentgenographic scores were observed in group 1 compared with group 2. Although there were no significant differences in pulmonary artery wedge pressure and colloid osmotic pressure between groups 1 and 2, mean right atrial pressure was significantly higher, while cardiac output and mixed venous oxygen saturation were lower in group 1 compared with group 2. Patients in group 1 had significantly more left ventricular segments with advanced asynergy and higher incidence of proximal right coronary artery lesions than those in group 2. Thus, our data suggest that disorder of pulmonary gas exchange in patients with right ventricular infarction may be explained by increased permeability of the alveolar capillary membrane secondary to larger extent of ischemic myocardium and by hemodynamic abnormalities associated with right ventricular infarction. (Chest 1992; 101:1489-93)

Methods

Patients

We studied 147 consecutive patients with inferior wall Q-wave acute myocardial infarction who fulfilled the following criteria: (1) admitted to the coronary care unit within 24 h from the onset of chest pain and in whom a Swan-Ganz catheter was inserted at the time of hospital admission; (2) survived the first three days after hospital admission; (3) no history of myocardial infarction and pulmonary diseases; (4) no anatomic shunts in the heart; and (5) sinus rhythm without intraventricular conduction defect at the time of hospital admission. All patients were examined by a physician and written informed consent was obtained before the Swan-Ganz catheter insertion.

Clinical Evaluation

The diagnosis of inferior myocardial infarction was made when the patients had an ST elevation with new Q waves (2, 3, and aVF) in serial electrocardiograms and at least a twice normal elevation in serum creatine kinase with MB isoenzyme ≥5 percent. At least 1 mm of ST-segment elevation and QS or QR in right precordial lead (V4) at the time of hospital admission were considered diagnostic of right ventricular infarction. Hemodynamic measurements, including cardiac output, pulmonary artery wedge pressure, and mean right atrial pressure were determined with a Swan-Ganz catheter within 3 h of hospital admission. Right ventricular infarction was diagnosed hemodynamically when mean right atrial to pulmonary artery wedge pressure ratio ≥0.8 and mean right atrial pressure was ≥10 mm Hg. Arterial oxygen tension and arterial carbon dioxide tension were measured at 37°C with the standard electrode technique using a system (Radiometer, Radiometer Company, Ohio ABL2) while the patient was breathing room air. Alveolar arterial oxygen difference was calculated by assuming a respiratory...
quotient equal to 0.8. Colloid osmotic pressure was obtained from the equation of Nitta et al.\(^a\) and Staub\(^b\) using the measured values for content of total protein, serum albumin, and serum globulin at the time of hospital admission: Colloid osmotic pressure = \(\sigma(2.8c + 0.12c^2 + 0.012c^3) + b(0.9c + 0.12c + 0.004c^3)\) where \(\sigma(\text{percent}) = \text{serum albumin; } b(\text{percent}) = \text{serum globulin; } c(\text{g/dl}) = \text{total protein.}\) After hospital admission, patients were treated conventionally according to their clinical and hemodynamic status, but hemodynamic stability was confirmed before the measurements were performed. During this study period, four patients had diuretics before the hemodynamic measurement and they were not included in this study. None of the patients had volume-loading therapy before the hemodynamic measurement.

**Echocardiography**

Two-dimensional and M-mode echocardiography were performed with a phased-array sector scanner (SSD 870, Aloka, Tokyo). All classic views were recorded on videotape for subsequent analysis by observers who were unaware of the patients’ clinical data. Regional left ventricular wall motion abnormalities were assessed by two-dimensional echocardiography obtained on the third day of hospitalization. Analysis of the left ventricular wall was performed with 11 segments obtained by long- and short-axis images.\(^a\) and the number of segments with advanced asynergy (akinesia or dyskinesia) was calculated by observer blinded to patients’ hemodynamic data. To further characterize the infract size in inferior infarction, echocardiographic scores were sub-grouped into two groups: small infract size (number of segments \(<3\)) and large infract size (number of segments \(\geq 3\)). The absence of anatomic shunt in the heart was confirmed by Doppler echocardiogram.

**Chest Roentgenography**

All chest roentgenograms were obtained with patients in the supine position within 1 h of hemodynamic measurements using routine mobile unit roentgenography. Roentgenograms were interpreted by observers blinded to patients’ clinical status. Lung field of the roentgenogram was graded on a four-point scale: 0, no acute infiltrate; 1, pulmonary vascular redistribution and/or mild diffuse interstitial infiltrate; 2, moderate diffuse interstitial infiltrate; 3, diffuse interstitial and focal alveolar infiltrate; and 4, diffuse alveolar infiltrate.\(^a\) As all patients with a roentgenographic score \(\geq 2\) had increased extravascular lung water (\(>7\) ml/kg) in a previous report,\(^a\) roentgenograms were classified into two groups: low score, scores 0 and 1; high score, scores 2, 3, and 4.

**Mixed Venous Sample**

Mixed venous oxygen saturation was determined in 43 patients who were breathing room air at the time of hemodynamic measurement. Mixed venous blood samples were drawn from Swan-Ganz catheter and mixed venous oxygen saturation was measured (Co-Oximeter, Radiometer Company, OSM2).

**Coronary Angiography**

A coronary arteriogram was performed before each patient was discharged from the hospital and coronary artery lesions with \(\geq 70\) percent reduction in diameter were considered to be obstructive. Each patient was classified as having 1-, 2-, or 3-vessel disease. Proximal right coronary artery lesions were defined as located before the acute marginal branch.

**Statistical Analysis**

Results are reported as mean \(\pm\) standard deviation. Student’s \(t\) test was used for quantitative data and \(x^2\) analysis was used for qualitative data. The least squares linear regression analysis was used to evaluate the correlation of the two variables. A \(p\) value below 0.05 was considered significant.

### Results

**Incidence of Right Ventricular Infarction**

Of the 147 patients with inferior wall Q-wave acute myocardial infarction, electrocardiographic evidence of right ventricular infarction was found in 39 patients (group 1) and was absent in 108 patients (group 2). When the hemodynamic criteria were used for the diagnosis of right ventricular infarction, 12 of 39 patients in group 1 fulfilled the hemodynamic criteria for right ventricular infarction.

**Clinical Characteristics**

Patients were treated with sublingual nitrate or morphine for chest pain before hospital admission and/or at the time of hospital admission (90 percent in group 1 and 88 percent in group 2; difference not significant), but none of the patients had chest pain at the time of hemodynamic measurement and patients treated with intravenous nitroglycerin was not included in this study. A significantly wider alveolar arterial oxygen difference was observed in group 1 compared with group 2 (Table 1). There was no significant difference in the age distribution between groups 1 and 2. Although there were no significant differences in pulmonary artery wedge pressure, mean pulmonary artery pressure, and colloid osmotic pressure between groups 1 and 2, cardiac output was significantly lower and mean right atrial pressure was significantly higher in group 1 compared with group 2. Patients in group 1 had significantly more left ventricular segments with advanced asynergy and higher incidence of high roentgenographic score compared with those in group 2.

### Table 1 — Clinical Characteristics of Patients with Electrocardiographic Right Ventricular Infarction*

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n = 39)</th>
<th>Group 2 (n = 108)</th>
<th>(p) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>P(A-a)O(_2), mm Hg</td>
<td>40 ± 11</td>
<td>34 ± 11</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Chest roentgenogram</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low score</td>
<td>20</td>
<td>79</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>High score</td>
<td>19</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>Age, yr</td>
<td>64 ± 12</td>
<td>65 ± 11</td>
<td>NS</td>
</tr>
<tr>
<td>CO, L/min</td>
<td>4.03 ± 0.92</td>
<td>4.81 ± 1.35</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>PCW, mm Hg</td>
<td>11 ± 4</td>
<td>10 ± 5</td>
<td>NS</td>
</tr>
<tr>
<td>PAm, mm Hg</td>
<td>17 ± 2</td>
<td>16 ± 2</td>
<td>NS</td>
</tr>
<tr>
<td>RA, mm Hg</td>
<td>7 ± 4</td>
<td>5 ± 3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>COP, mm Hg</td>
<td>24 ± 4</td>
<td>24 ± 3</td>
<td>NS</td>
</tr>
<tr>
<td>Advanced asynergy</td>
<td>2.9 ± 1.3</td>
<td>2.3 ± 1.6</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

*Group 1: patients with electrocardiographic evidence of right ventricular infarction; group 2: patients without electrocardiographic evidence of right ventricular infarction. P(A-a)O\(_2\) = alveolar arterial oxygen difference; CO = cardiac output; COP = colloid osmotic pressure; NS = not significant; PAm = mean pulmonary artery pressure; PCW = pulmonary artery wedge pressure; RA = mean right atrial pressure.
Table 2—Effect of Infarct Size on Pulmonary Gas Exchange

<table>
<thead>
<tr>
<th>No. of LV Segments with Asynergy</th>
<th>Segments &lt;3</th>
<th>Segments ≥3</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n = 72)</td>
<td>(n = 75)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P(A-a)O₂</td>
<td>33 ± 10</td>
<td>37 ± 11</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Chest roentgenogram score</td>
<td>0.9 ± 0.9</td>
<td>1.4 ± 0.9</td>
<td>&lt;0.005</td>
</tr>
</tbody>
</table>

*LV = left ventricular; others as in Table 1.

Effect of Infarct Size on Pulmonary Gas Exchange

When echocardiographic extent of infarct size was subgrouped into two groups, patients with large infarct size (number of asynergic segments ≥3) had significantly wider alveolar arterial oxygen difference and higher chest roentgenographic score compared with those with small infarct size (number of asynergic segments <3) (Table 2).

Relation between Alveolar Arterial Oxygen Difference and Clinical Variables

Although alveolar arterial oxygen difference had no significant correlation with any of the hemodynamic variables (cardiac output, pulmonary artery wedge pressure, and mean right atrial pressure) and colloid osmotic pressure, a fairly good correlation was observed with the chest roentgenographic score (r = 0.65, p<0.001).

Mixed Venous Oxygen Saturation

Mixed venous oxygen saturation was obtained from 14 patients in group 1 and from 29 patients in group 2. Mixed venous oxygen saturation was significantly lower in group 1 compared with groups 2 (54 ± 15 percent and 68 ± 7 percent, group 1 and 2, respectively, p<0.001).

Coronary Arteriography

A coronary arteriogram was obtained in 26 patients in group 1 and 68 patients in group 2. Thirteen patients in group 1 and 32 patients in group 2 had multivessel disease; the difference was not significant. Patients in group 1 had significantly higher incidence of proximal right coronary lesion than group 2 (85 percent and 43 percent, respectively, p<0.001).

Discussion

Right ventricular infarction is not rare in patients with inferior wall myocardial infarction and the advances in noninvasive cardiac diagnosis have increased the awareness of right ventricular infarction.6,7 However, not many studies have focused on pulmonary gas exchange and chest roentgenographic findings in patients with right ventricular infarction. Because hypoxemia can be compensated by hyperventilation, alveolar arterial oxygen difference was used as an indicator of pulmonary gas exchange. In this study, alveolar arterial oxygen difference and chest roentgenographic score were evaluated with seven other clinical variables that could affect the change in pulmonary gas exchange in patients with inferior infarction. As hemodynamically documented right ventricular infarction complicated by low cardiac output and high right atrial pressure has been estimated to occur in the minority of all myocardial infarction, we chose the presence of Q wave and at least 1 mm of ST-segment elevation in V₆₃ for the diagnosis of right ventricular infarction to maximize our diagnostic accuracy as much as possible. As a result, evidence of defective pulmonary gas exchange, as revealed by wider alveolar arterial oxygen difference, existed with the sign of increased extravascular lung water by chest roentgenograms in patients with electrocardiographic evidence of right ventricular infarction.

Arterial hypoxemia in the patients with acute myocardial infarction is more likely to be due to one or more of the following mechanisms: right-to-left shunt, ventilation-perfusion inequalities, or a reduction in diffusing capacity. In our study, patients with right ventricular infarction (group 1) had lower cardiac output than those without right ventricular infarction (group 2). Furthermore, measurements of the mixed venous oxygen saturation revealed that patients in group 1 had significantly lower values compared with group 2. Mixed venous oxygen saturation is determined by both oxygen supply and demand.10,21 Therefore, one of the factors associated with more severe arterial hypoxemia in group 1 was due to lower mixed venous oxygen saturation that resulted from decreased cardiac output as well as increased oxygen consumption of whole-body tissues because of more severe myocardial infarction.21

A pulmonary artery pressure of 18 mm Hg has been shown to be critical for the onset of the radiologic sign of pulmonary congestion and hypoxemia was associated with elevated pulmonary artery diastolic pressure.1,2,5,8,23 In our patients, a fairly good correlation was found between alveolar arterial oxygen difference and chest roentgenographic score (r = 0.65) that implied that disturbance of pulmonary gas exchange was associated with increased extravascular lung water. Although patients in group 1 had higher mean right atrial pressure than those in group 2, pulmonary artery wedge pressure and mean pulmonary artery pressure in group 1 were not significantly higher than group 2 indicating that pulmonary venous congestion was not the major factor causing disturbance of pulmonary gas exchange. Elevation in venous pressure that induces a back pressure hindering lymph flow could also cause an increase in extravascular lung water,23 but our finding of a lack of good correlation
between alveolar arterial oxygen difference and hemodynamic indices suggests that these may not be the only factors causing increased extravascular lung water in group 1.

Two-dimensional echocardiography provides a noninvasive means for visualization of abnormal left ventricular wall motion, but this technique tends to exaggerate the infarct size.\(^2\) In this study, however, none of the patients had a history of prior myocardial infarction, and only advanced asynergy (akinisia and dyskinesia) was considered in estimation of the extent of infarction. Patients in group 1 were associated with more left ventricular segments with advanced asynergy and higher incidence of proximal right coronary artery lesions. Earlier studies have demonstrated that increased microvascular pressure is not a sole explanation for pulmonary edema after acute myocardial infarction and have suggested that some of the pulmonary edema without elevation in pulmonary artery wedge pressure is considered to be related to the increased permeability of the pulmonary vascular bed caused by pulmonary capillary disruption.\(^6-11,27,28\) In our previous study, we demonstrated that disturbance of pulmonary gas exchange, as revealed by high value of alveolar arterial oxygen difference, existed without left ventricular dysfunction in the early phase of myocardial infarction.\(^2\) Furthermore, extravascular lung water correlated positively with infarct size in patients with low pulmonary artery wedge pressure after acute myocardial infarction.\(^1\) Therefore, in addition to the hemodynamic abnormalities associated with right ventricular infarction, our results suggest that disturbance of pulmonary gas exchange and increase in extravascular lung water in patients with right ventricular infarction may be explained by an increase in permeability of the alveolar capillary membrane, probably resulting from larger extent of ischemic myocardium.

Two limitations of this study should be addressed. First, as the accuracy of electrocardiographic signs of right ventricular infarction is not complete, right ventricular involvement can exist even in the absence of electrocardiographic signs. However, the difference in right atrial pressure might have been larger if 100 percent of the patients in groups 1 and 2 had been correctly identified. Second, almost all the patients received nitrates for their chest pain, which might have decreased pulmonary artery wedge pressure. Nevertheless, there was no significant difference in the use of nitrates between the two groups and none of the patients was treated with intravenous nitroglycerin at the time of hemodynamic measurement. Therefore, an appraisal of our results in relation to results from other studies indicates that our conclusion would probably not have been altered by the use of nitrates.

In conclusion, hemodynamic and anatomic characteristics of right ventricular infarction may contribute to the disturbance of pulmonary gas exchange.

REFERENCES

23 Staub NC. Pulmonary edema. Physiol Rev 1974; 54:678-811

Plan to Attend ACCP’s

58th Annual Scientific Assembly
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