Severe Hypoxemia Due to Shunting Through a Patent Foramen Ovale: A Correctable Complication of Right Ventricular Infarction

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A patient with recent inferior myocardial infarction with right ventricular involvement developed severe hypoxemia unresponsive to 100% oxygen. Contrast two-dimensional echocardiography revealed right to left shunting through an aneurysmal fossa ovalis with a patent foramen ovale. This was confirmed by cardiac catheterization. Surgical closure of the defect was probably lifesaving. This case report illustrates that right to left shunting through a foramen ovale should be considered in the differential diagnosis of hypoxemia in patients presenting with inferior myocardial infarction.

Case Report

A 71 year old white woman was hospitalized on February 2, 1983 with recent onset of severe substernal pain. She was a nonsmoker and had no prior history of cardiac or pulmonary disease. Serial electrocardiograms and cardiac enzyme determinations documented an acute inferior myocardial infarction. Persistent chest pain and new T wave electrocardiographic changes in the anterolateral leads led to her transfer to our institution on February 4.

Physical findings. On admission to our hospital, the patient reported moderate chest pain but was in no respiratory distress. Her blood pressure was 160/90 mm Hg, pulse 72/min and respirations 18/min. Jugular venous pressure was considered normal and the lungs were clear. A fourth heart sound was noted. No murmurs were detected.

Initial hospital course. The patient received intravenous morphine sulfate for chest pain and was given a topical nitrate, hydrochlorothiazide, 50 mg daily; nifedipine, 10 mg every 8 hours and heparin, 5,000 units subcutaneously every 12 hours. The electrocardiogram revealed an inferior wall myocardial infarction with ST elevation in lead V1 (Fig. 1), suggesting right ventricular involvement. Serum creatine kinase (CK) increased to 3,440 units/liter and 12% MB fraction, with no evidence of a secondary peak in the CK curve. A blood gas determination with the patient breathing room air revealed an arterial oxygen tension (Po2) of 61 mm Hg with a saturation of 91%. The next morning (February 5), she developed atioventricular (AV) dissociation, bradycardia with transient hypotension and chest pain. A temporary pacemaker was placed by way of the right internal jugular vein. A left radial arterial line was also inserted.

On the evening of February 6, the patient became oliguric and hypertensive, with a blood pressure of 90/50 mm Hg. A Swan-Ganz catheter was positioned in the right pulmonary artery through the left subclavian vein. Pressures were consistent with right ventricular infarction, with mean right atrial and pulmonary artery wedge pressures of 10 mm Hg and a pulmonary artery pressure of 18/10 mm Hg. Hypertension and oliguria improved with intravenous fluids and low dose dopamine infusion. Later the same evening, she was noted to be in respiratory distress. An arterial blood gas determination with the patient receiving nasal oxygen at 2 liters/min showed a Po2 of 38 mm Hg, oxygen saturation

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of 75.7%, arterial carbon dioxide tension ($\text{PCO}_2$) of 25 mm Hg and pH of 7.47. Hypoxemia ($\text{PO}_2$ 53 mm Hg) persisted despite an increase in the inspired oxygen concentration ($\text{FiO}_2$) to 100%.

Early in the morning of February 7, endotracheal intubation was performed and the patient was placed on assisted mechanical ventilation with an $\text{FiO}_2$ of 100%. Over the next several hours, the $\text{PO}_2$ averaged in the mid 50s despite continued administration of 100% oxygen. Addition of a positive end-expiratory pressure of 10 cm water resulted in no immediate improvement in oxygenation. Approximately 20 hours after endotracheal intubation, oxygenation improved transiently, even though no associated change in treatment or hemodynamics was recognized. Serial arterial blood gas determinations obtained at 11, 20 and 24 hours after intubation revealed $\text{PO}_2$ values of 52, 181 and 253 mm Hg, respectively. However, this period of improved oxygenation lasted for less than 6 hours, after which oxygenation deteriorated for no apparent reason. Thirty-six hours after intubation, the $\text{PO}_2$ was 48 mm Hg, with an $\text{FiO}_2$ of 80% and it increased slightly to 56 mm Hg with an $\text{FiO}_2$ of 100%.

**Diagnostic procedures.** A chest X-ray film revealed haziness at the left lung base with good position of the endotracheal tube. A perfusion lung scan was negative for pulmonary embolism. Bronchoscopic examination performed on the second day after endotracheal intubation was

**Figure 1.** Twelve lead electrocardiogram showing sinus rhythm and acute inferior and lateral myocardial infarction. RSR' and mild ST elevation are noted in lead V1, which may indicate right ventricular infarction.

**Figure 2.** Two-dimensional echocardiogram, apical four chamber view, displayed with the apex pointing down before (A) and during (B) contrast injection into the right atrium. Contrast agent (arrows) is seen in the left atrium (LA) and left ventricle (LV). Real time studies localized the shunt through an aneurysmal fossa ovalis. The drawings illustrate the redundant and aneurysmal fossa ovalis. I = inferior; L = left; MV = mitral valve; R = right; RA = right atrium; RV = right ventricle; S = superior; TV = tricuspid valve.
Figure 3. Pressure tracings from the right atrium (RA) (A), left atrium (LA) (B), simultaneous left (LV) and right (RV) ventricles (C) and pulmonary artery (PA) (D), showing equalization of mean right atrial, mean left atrial, right ventricular end-diastolic, left ventricular end-diastolic and pulmonary artery diastolic pressures. A prominent Y descent is noted in both atria. Inspiratory (Insp) decline in pressures is present in the left atrium. Simultaneous left and right ventricular pressure tracings show the "square root" pattern. High grade AV block with junctional rhythm is seen in the electrocardiogram of panel C.

Figure 4. Indicator dye-dilution curve with injection into the inferior vena cava (IVC) and sampling from the aorta (curve 1) shows a prominent early appearance deflection on the upstroke of the curve, indicating a right to left intracardiac shunt. With injection in the right ventricle (RV) and sampling from the aorta (curve 2), there is no evidence of an intracardiac shunt.

Cardiac catheterization was then performed by way of the right femoral artery and vein while the endotracheal tube was in place and ventilation was unassisted. The left atrium was readily entered through a patent foramen ovale. Intracardiac pressure tracings were typical of right ventricular infarction with equalization of diastolic pressures (Fig. 3). The indicator-dilution curve obtained with sampling from the aorta demonstrated an early appearance deflection with injection of indocyanine green into the inferior vena cava, but not with injection into the right ventricle (Fig. 4). This finding localized the shunt at the atrial level. Oximetric measurements (Table 1) revealed right to left shunting at the atrial level with no evidence of left to right shunting. The calculated right to left shunt was 28% of systemic flow. Coronary arteriography demonstrated total proximal occlu-
sion of an unusually large, dominant right coronary artery and mild disease of the left coronary vessels. The right ventricular angiogram showed akinesia of the inferior and apical segments (Fig. 5). Left ventricular angiography was not performed.

Surgical findings. With the persistent hypoxemia and high risk of pulmonary oxygen toxicity, the patient was taken for surgery after cardiac catheterization. Surgical inspection revealed infarction of the inferior walls of the right and left ventricles. The septum primum (membrane lining the fossa ovalis) was redundant, aneurysmal and incompetent. The defect was closed with a Dacron patch. An arterial blood gas determination 6 hours after operation with an \( F_{O_2} \) of 88\% showed a \( P_{O_2} \) of 154 mm Hg. Twenty-two hours after surgery, the \( P_{O_2} \) was 84 mm Hg with an \( F_{O_2} \) of 45\%. The patient was subsequently weaned from oxygen and made an uneventful recovery.

Discussion

Right ventricular infarction characteristically produces elevation of right-sided intracardiac diastolic pressures with equalization of right and left heart diastolic pressures (1,5,6). Constrictive pericarditis, restrictive myocardial disease and cardiac tamponade are other causes of diastolic pressure equalization (14,15). Of these entities, only right ventricular infarction and tamponade are likely to become manifest in the setting of acute myocardial infarction. The differentiating hemodynamic features have been reported previously (5,14,15). Pressure tracings in the present case revealed a "square root" pattern in the ventricular pressure tracings with equalization of diastolic pressures (Fig. 3). The right ventricular early diastolic pressure was elevated to 10 to 12 mm Hg, probably because of a noncompliant and infarcted right ventricle. The left atrial pressure tracing showed prominent respiratory variation in pressure, with a sharp decrease in pressure, apparently corresponding to inspiration (Fig. 3B). The right atrial pressure tracing had little respiratory variation (Fig. 3A). Thus, it is likely that both the increase

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**Table 1. Catheterization Data**

<table>
<thead>
<tr>
<th>Site</th>
<th>S/D Pressure (mm Hg)</th>
<th>Oxygen Saturation (%)</th>
<th>Oxygen Content (vol %)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SVC</td>
<td>—</td>
<td>61</td>
<td>9.3</td>
</tr>
<tr>
<td>RA</td>
<td>( v = 25 ), ( m = 19 )</td>
<td>51</td>
<td>7.7</td>
</tr>
<tr>
<td>RV</td>
<td>30/20</td>
<td>51</td>
<td>7.7</td>
</tr>
<tr>
<td>PA</td>
<td>28/20</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>LPV</td>
<td>—</td>
<td>98*</td>
<td>15.6</td>
</tr>
<tr>
<td>LA</td>
<td>( m = 18 )</td>
<td>88</td>
<td>13.3</td>
</tr>
<tr>
<td>LV</td>
<td>110/19</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Ao</td>
<td>110/55</td>
<td>89</td>
<td>13.4</td>
</tr>
</tbody>
</table>

Inspired oxygen concentration \( (F_{O_2}) \) = 88\%; hemoglobin = 11.0 g/100 ml; left to right shunt = none; right to left shunt = 28\%; left pulmonary vein (LPV) \( P_{O_2} \) = 327 mm Hg. \( Ao = \) aorta; \( D = \) diastolic; \( LA = \) left atrium; \( LV = \) left ventricle; \( m = \) mean; \( PA = \) pulmonary artery; \( RA = \) right atrium; \( RV = \) right ventricle; \( S = \) systolic; \( SVC = \) superior vena cava; \( v = \) v wave.

**Figure 5.** Diastolic (top) and systolic (bottom) frames of the right ventricular angiogram in the posteroanterior projection, showing akinetic inferior and apical segments (arrowheads). Mild tricuspid regurgitation (arrow) is noted. \( RV = \) right ventricle; \( RVOT = \) right ventricular outflow tract; \( TV = \) tricuspid valve.
in right atrial pressure due to right ventricular infarction and the inspiration-associated decrease in left atrial pressure created a pressure gradient favorable for right to left shunting through the patent foramen ovale.

This patient initially had adequate oxygenation, but became severely hypoxic 4 days after acute myocardial infarction. She then had transient improvement in oxygenation approximately 20 hours after recognition of severe hypoxemia. By the time right to left intracardiac shunting had been recognized, her condition was precarious and surgery was promptly performed. The inspiratory decrease in left atrial pressure apparently contributed to right to left shunting, and it is possible that interventions to minimize inspiratory effort would have reduced the shunting. Whether vasoactive drugs, changes in volume status or positive end-expiratory pressure affected shunting is not known.

Implications. A patent foramen ovale is a variation of normal and occurs in up to 27% of adults (16). Right ventricular infarction is commonly associated with inferior infarction, occurring in up to 24% of cases examined at necropsy (8). As awareness of the clinical entity increases, right to left shunting complicating right ventricular infarction may be recognized more frequently. Administration of high oxygen concentration may be required, but should be avoided if possible because oxygen is not particularly effective in correcting hypoxemia caused by intracardiac shunting. Spontaneous improvement in right ventricular function with the passage of time may reduce right to left shunting (17). However, with severe shunting and persistent hypoxemia, surgical closure of the defect can be life-saving.

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References


