Right-to-Left Shunt Through a Patent Foramen Ovale in Acute Right Ventricular Infarction

Two Case Reports and a Proposal for Management

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Right-to-left shunting through a foramen ovale complicating acute right ventricular infarction and resulting in severe arterial hypoxemia has been described eight times before. Treatment strategies have often aimed at reducing the shunt. Four patients died. Less attention has been paid to attempts at revascularization and, despite a high incidence of atrioventricular conduction disturbances, to temporary dual-chamber pacing. We describe herein two patients with postcardiac surgical right ventricular infarction complicated by severe right-to-left intratrial shunting. Treatment strategy was aimed at improving right ventricular function, and right-to-left shunting ceased. All efforts should be directed at treating right ventricular dysfunction, which is the cause of the clinical picture, and not at reducing the shunt, which is a secondary phenomenon.

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AV=atrioventricular; PEEP=positive end-expiratory pressure; PTCA=percutaneous transluminal coronary angioplasty; SaO2=arterial oxygen saturation

Key words: aortic dissection; cardiac surgery; coronary artery disease; foramen ovale; hypoxemia; intracardiac shunt; right ventricular infarction

A right-to-left shunt through a foramen ovale complicating acute right ventricular infarction has been described eight times.1-8 We present two additional cases and review the literature, proposing a strategy for patient management.

Case Reports

Case 1

A 55-year-old man underwent coronary angiography because of unstable angina under treatment with nitrates and nifedipine. A 90% stenosis of the proximal left anterior descending artery and a 40% stenosis in the mid portion of the right coronary artery were found. Percutaneous transluminal coronary angioplasty (PTCA) of the left anterior descending artery led to thrombotic occlusion with severe intermittent anterior wall ischemia. The patient underwent uncomplicated emergency coronary bypass surgery. Blood gas determinations during mechanical and spontaneous ventilation were normal. Eighteen hours postoperatively, acute inferior and right ventricular infarction occurred. This was complicated by ventricular fibrillation, high-degree atrioventricular (AV) block requiring AV sequential pacing, cardiogenic shock, and arterial hypoxemia to a minimum transcutaneously measured oxygen saturation of 60% while breathing 100% oxygen.

Pulmonary arterial systolic pressure increased to 50 mm Hg and right atrial pressure rose from 13 to 30 mm Hg. The right coronary artery was successfully recanalized, after which O2 saturation increased to about 90 to 92% while the patient was still breathing 100% oxygen.

Transsthoracic echocardiography showed good left but poor right ventricular contraction. Contrast echocardiography showed right-to-left intratrial shunting. For the first 8 h after PTCA, right atrial pressure remained about 6 mm Hg higher than left atrial pressure, after which right atrial pressure decreased. Further dips in arterial oxygen saturation (SaO2) to minimally 76%, temporally related to atrial fibrillation, administration of verapamil, and positive pressure ventilation with positive end-expiratory pressure (PEEP) occurred over the next 36 h. Under isotropic support, normalization of hemodynamic parameters and oxygenation occurred over 72 h. No right-to-left shunting could be demonstrated on the 13th postoperative day.

Case 2

A 35-year-old woman with a history of hypertension and chronic renal failure (creatinine=2 mg/dl) due to polycystic disease was hospitalized in shock and respiratory distress after she had complained of severe chest pain. A diagnosis of type A aortic dissection with severe aortic regurgitation was made, and the patient underwent urgent replacement of the ascending aorta and aortic valve by a composite graft, including a 25-mm prosthesis (St.-Jude Medical). The coronary arteries were reimplanted into the graft. Postoperatively, a combination of epinephrine (Adrenalin), norepinephrine (Noradrenaline), milrinone, dabutanne, and dopamine was required to maintain cardiac output and blood pressure. Oxygenation was poor and reached a nadir 4 days after the operation when the SaO2 under a forced expiratory oxygen of 80% and 100% was 61% and 73%, respectively. Administration of PEEP was associated with deterioration of arterial oxygenation. New inferior Q waves developed on the ECG. Transesophageal echocardiography showed good left ventricular function but very poor right ventricular function. In
addition color-coded Doppler examination demonstrated a turbulent jet from right to left through the interatrial septum (Fig 1). The patient's condition improved slowly over the following days, and inotropic therapy was stopped on the 15th day. After recovery, except for some refractory atelectasis of the left lower lobe, the patient attained an O₂ saturation of 92% while breathing room air. Transesophageal echocardiography demonstrated improved right ventricular function and no interatrial shunting.

**DISCUSSION**

An open foramen ovale, necessary during fetal development, occurs in up to 30% of postmortem examinations and more frequently in patients who have suffered ischemic stroke. Normally, right-to-left shunting occurs only when right ventricular pressure exceeds left atrial pressure as in right ventricular infarction and other situations. The resulting arterial hypoxemia adds to the morbidity and mortality. At least six attempts at surgical or nonsurgical occlusion have been made, five in right ventricular infarction and one in ARDS. Three of the patients with right ventricular infarction survived after occlusion procedures. Treatment with inotropic drugs has also been found to be useful.

The syndrome of right-to-left shunting through a patent foramen ovale in acute right ventricular infarction carries high morbidity and mortality (Table 1). It occurs so rarely that prospective study of differing treatment modalities is impossible. It is, however, possible to make some deductions from the cases described in the literature. First of all, one should consider the possibility of the anomaly when otherwise unexplained arterial hypoxemia occurs in the presence of right ventricular failure and especially when increasing PEEP worsens oxygenation. It may be argued that in our first patient, the high systolic pulmonary arterial pressure indicated a component of left ventricular failure in the context of inferior infarction. The basic problem, however, seems to be right ventricular systolic and diastolic failure, for when right ventricular function improves, it often does several days after acute right ventricular infarction. The right-to-left shunting diminishes and should cease. To bridge this period, positive inotropic drugs with pulmonary arterial dilating capacity should be given with careful monitoring of hemodynamics and SaO₂ while dual-chamber pacing should be performed when necessary to preserve a right atrial kick. PEEP should be avoided because it further reduces transpulmonary left ventricular filling. It is surprising that although seven of the ten patients described (including our case 1) had AV block or some form of junctional rhythm, only we, aided by intraoperatively placed pacing wires, performed AV pacing. Sterling et al. achieved significant improvement in blood pressure by ventricular pacing alone. Laham et al. recently described five patients with right atrial-to-left atrial shunting after myocardial infarction. Only three of these presented in the acute phase of their infarction, and two of the remaining three did not have SaO₂ below 90% so the procedure to occlude the interatrial defect was inappropriate. Only one of their patients was as ill as others described in the literature and appears in Table 1. Occluding the foramen ovale is probably not beneficial. Three of 5 patients undergoing such a procedure did survive, but it is not clear that survival was due to the procedure. In shock due to acute right heart failure, occlusion of a foramen ovale may further reduce left ventricular filling and increase already elevated right ventricular filling. Intraoperative creation of an atrial septal defect, in fact, has been described in a case of acute right ventricular dysfunction to improve left ventricular filling.

The great initial improvement seen in our first patient after PTCA of the right coronary artery finally suggests that aggressive attempts at revascularization of this vessel should be considered early. Thrombolysis is clearly contraindicated in a recently operated-on patient, but it may be helpful when it is not contraindicated. We do not know the coronary anatomy of our second patient but suspect that dissection into the origin of the right coronary artery rather than coronary artery disease caused the infarction.

**CONCLUSION**

The high mortality and morbidity of right-to-left interatrial shunting in acute right ventricular infarction seems to be related to right ventricular failure. Treatment should be directed at improving right ventricular function. Closure of the foramen ovale is unnecessary and contraindicated because left ventricular filling may become insufficient.
### Table 1—Overview of Reports on Right-to-Left Shunting Through a Foramen Ovale in Acute Right Ventricular Infarction*

<table>
<thead>
<tr>
<th>Source</th>
<th>Year of Article</th>
<th>Sex/Age, yrs</th>
<th>Attempt at Reperfusion</th>
<th>Shock</th>
<th>AV Block</th>
<th>Occlusion Procedure</th>
<th>Survived</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morris and Donen⁴</td>
<td>1978</td>
<td>M/63</td>
<td>No</td>
<td>+</td>
<td>JB</td>
<td>-</td>
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</tr>
<tr>
<td>Manno et al²</td>
<td>1983</td>
<td>M/49</td>
<td>fICTL</td>
<td>+</td>
<td>+</td>
<td>surgery</td>
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<tr>
<td>Rietveld et al³</td>
<td>1983</td>
<td>F/61</td>
<td>No</td>
<td>-</td>
<td>+</td>
<td>TVDO</td>
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<tr>
<td>Bansal et al⁴</td>
<td>1985</td>
<td>F/71</td>
<td>No</td>
<td>+</td>
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<td>TVDO</td>
<td>No</td>
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<tr>
<td>Uppstrom et al⁵</td>
<td>1988</td>
<td>F/65</td>
<td>psIVTL</td>
<td>+</td>
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<td>Broderick and Dillon⁶</td>
<td>1989</td>
<td>F/68</td>
<td>No</td>
<td>-</td>
<td>-</td>
<td>TVDO</td>
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<tr>
<td>Sterling et al⁷</td>
<td>1990</td>
<td>F/63</td>
<td>No</td>
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<td>Laham et al⁸</td>
<td>1994</td>
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<td>+</td>
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</tr>
</tbody>
</table>

*FICTL = failed intracoronary thrombolysis; JB = junctional bradycardia; JR = junctional rhythm; psIVTL = partly successful intravenous thrombolysis; sPTCA = successful PTCA; TVDO = transvascular device occlusion; plus sign = present; minus sign = absent or not done.

### References

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11. Moorthy SS, Losasso AM. Patency of the foramen ovale in the critically ill patient. Anesthesiology 1974; 41:405-07