The dilemma of refractory hypoxemia after inferior wall myocardial infarction

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ABSTRACT

Patent foramen ovale (PFO) occurs in 25% of people. The decision on whether to close the PFO found after myocardial infarction and specifically right ventricular infarction is debated, with no solid guidelines addressing this subject. Here we present the case of a 59-year-old man who presented with a myocardial infarction and was found to have PFO. He was treated with revascularization of the culprit artery, followed by supportive care.

KEYWORDS Intracardiac shunt; myocardial infarction; patent foramen ovale

A patent foramen ovale (PFO) may allow right-to-left interatrial shunting when the pressure in the right atrium exceeds that of the left as in Valsalva maneuver.¹ Significant right-to-left shunting may be present in some patients upon positional changes, leading to the so-called platypnea-orthodeoxia syndrome.² A PFO may allow for paradoxical embolization, which may be the explanation for a proportion of cases of cryptogenic stroke.³ Refractory hypoxemia with an unremarkable chest radiograph or lung exam should always prompt suspicion for a right-to-left shunt. The occurrence of right ventricular infarction, which complicates 40% of posterior wall myocardial infarction (MI), may lead to right-to-left shunting, due to restrictive diastolic physiology of the ischemic right ventricle.^{4–12}

CASE PRESENTATION

A 59-year-old Hispanic man with no significant past medical history presented to the emergency department in Juarez, Mexico, with severe left-sided chest pain of 1-hour duration. His electrocardiogram confirmed an acute inferior ST elevation MI (*Figure 1*). Because percutaneous coronary intervention was not available locally, the patient was given thrombolytics. He developed worsening dyspnea, hypotension, and bradycardia. Thrombolytic infusion was stopped and he was transferred to our facility for further management. Upon arrival, his oxygen saturation was 82% despite a 15-L non-rebreathing mask. On examination, he had prominent jugular venous ingurgitation with a positive Kussmaul sign, an S4 gallop, and clear lungs to auscultation.

Figure 1. Electrocardiogram at initial presentation showing acute inferior ST segment elevation more prominent in lead III, suggesting the right coronary artery as the culprit vessel.

Figure 2. (a) Initial injection shows occlusion of the right coronary artery with a large thrombus. (b) After balloon angioplasty with stenting of the culprit lesion, flow was restored.

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Figure 3. (a) Mid-esophageal two-dimensional view shows a long, tunnel-like patent foramen ovale. (b) Mid-esophageal two-dimensional view with color Doppler confirms continuous right-to-left flow through the patent foramen ovale. (c) Transthoracic echo in 4-chamber view shows a large quantity of bubbles in the left atrium and ventricle within 3 beats of arrival of bubbles to the right heart. This is consistent with a right-to-left shunt at the atrial level.

Urgent coronary angiography revealed occlusion of the right coronary artery (*Figure 2a*). Successful rescue percutaneous coronary intervention was performed and an Everolimus drug-eluting stent was inserted (*Figure 2b*). Left ventricular end-diastolic pressure was 12 to 14 mm Hg. A transthoracic echocardiogram showed dilated right-sided chambers, depressed right ventricular systolic function, normal left atrium and left ventricular size with posterior wall akinesis, left ventricular ejection fraction of 50% to 55%, and paradoxical septal motion from right ventricular overload. Due to the persistent hypoxemia, an agitated saline contrast study was performed that demonstrated a right-to-left shunt during transthoracic echocardiography (*Figure 3c*). Transesophageal echocardiography confirmed the presence of PFO (*Figure 3a and b*). The patient was started on dual antiplatelet therapy with aspirin and clopidogrel. An eptifibatide infusion was continued for 12 hours after coronary intervention. He was given beta-blockers and an angiotensin-converting enzyme inhibitor and his oxygen demand steadily decreased. He was discharged after 10 days on home oxygen 2 L/min with an oxygen saturation > 90%.

DISCUSSION

The occurrence of right ventricular infarction may lead to right-to-left shunting, due to restrictive diastolic physiology.^{4–12} Right-to-left shunting can be considered after excluding more common causes of hypoxemia following MI, such as pulmonary edema from cardiogenic shock or ischemic mitral regurgitation, underlying pulmonary disease, or pulmonary embolism. The modality of choice to diagnose intracardiac right-to-left shunts is transesophageal echocardiography with agitated saline contrast. Transesophageal echocardiogram can be used when transthoracic echocardiogram image quality is poor or to guide closure of an intracardiac shunt if needed.^{6,7} Literature guiding management of right-to-left shunting through a PFO complicating right ventricular infarction is limited. Revascularization of the culprit artery is paramount, after which supportive care may suffice, as shunting decreases as the right ventricular function recovers.¹² Supplemental oxygen is often ineffective and positive pressure ventilation may worsen shunting¹³; many of these interventions are poorly tolerated in the setting of right ventricular failure.^{6,9,10} Other options to support a failing right ventricle refractory to medical

therapy may include venoarterial extracorporeal membrane oxygenation, atrial septostomy, atrial pacing in patients with bradycardia, and acute mechanical circulatory support devices.¹⁴

Persistent shunting after a trial of supportive measures may warrant closure of the PFO, which has been performed successfully using percutaneous techniques or surgery.^{8,10,11} However, because shunting allows for right ventricular decompression and improved left ventricular filling, shunt closure carries the risk of worsening right ventricular failure and compromised left ventricular preload.¹⁵

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