Refractory Hypoxia in Right Ventricular Infarction

Dear Editor,

Acute myocardial infarction (MI) involving the right ventricle is a rare event.[1] Right ventricular myocardial infarction (RVMI) is associated with infarction of the inferior wall of the left ventricle, occurring in more than one third of such cases.[1-3] Patent foramen ovale (PFO) is an anatomic inter-atrial communication with the potential risk for shunting of blood. This usually closes within weeks to months after birth but in approximately 27% of adults, shunting through the PFO occurs. This defect is usually of no clinical consequence. However in the setting of right ventricular failure, high right ventricular diastolic pressure can cause a right-to-left shunting via the PFO, resulting in systemic hypoxaemia refractory to supplemental oxygen.

Here, we present a case of acute inferior myocardial infarction, complicated by refractory hypoxaemia due to the development of an acute right-to-left inter-atrial shunt through a previously dormant PFO.

Case Report

A 56-year-old Fillipino male, working as a seaman, presented to casualty department with acute chest pain, associated with shortness of breath and diaphoresis. This lasted for 20 minutes. His past medical history included Type 2 diabetes mellitus (on oral hypoglycaemic agents), hypertension and hyperlipidaemia.

An electrocardiogram (ECG) revealed ST elevation in leads II, III and AVF. A right sided ECG was performed and showed ST elevation in V4R-V6R. This was further complicated with intermittent 2:1 atrio-ventricular (AV) block, leading to complete heart block.

Emergency cardiac catheterisation was performed. This revealed 100% occlusion of the proximal right coronary artery (RCA), with collaterals from left coronary artery to right posterior descending artery (RPDA). The left coronary artery (left anterior descending artery and left circumflex) had minor irregularities. A temporary pacing wire was inserted. Thereafter, percutaneous coronary intervention (PCI) showed difficulty in passing the wire from proximal to mid RCA, as the clot burden appeared heavy. Several attempts were made to pass the wire into the true lumen. On one successful attempt of passing the wire to the distal RCA, the balloon failed to pass through the mid to distal RCA junction. Repeat angiogram revealed the heavy clot burden at the proximal to mid RCA and possibility of a false channel in the distal RCA.

The patient remained asymptomatic during the procedure and his haemodynamics remained stable. The decision was made to stop the primary angioplasty and to commence on anticoagulation and glycoprotein GP IIb/IIIa infusion. The patient was transferred to the coronary care unit. After the failed PCI attempt, his oxygen saturation dropped to 88%. He remained asymptomatic and was immediately placed on 100% oxygen. Despite being on fraction of inspired oxygen (FiO2) at 100%, his oxygen saturation remained approximately around 88% to 93%. He was placed on non-invasive ventilatory support (BiPAP). A respiratory consult was made. Methaemoglobin was 0.7% to 1.0%. A bedside trans-thoracic echocardiography with intravenous micro-bubbles revealed a presence of intra-cardiac, right-to-left shunt with hypokinetic segment in the inferior-basal wall of the left ventricle.

A formal trans-thoracic echocardiography (TTE) was performed and revealed left ventricular ejection fraction of 62%, with presence of regional wall motion abnormalities in the basal inferior and mid-inferior septal wall of the left ventricle. There was moderately dilated right ventricular cavity with mildly reduced right ventricular global systolic function. The right atrial cavity was moderately dilated. There was moderate tricuspid regurgitation. The tricuspid annulus was dilated at 4 cm. The pulmonary artery systolic pressure was normal (22 mmHg). The shunt was not demonstrable on the formal TTE.

A repeat coronary angiography was performed 2 days later, including a right heart catheterisation study. Another attempt of PCI to the proximal RCA (chronic total occlusion (CTO) with collaterals) was made and was unsuccessful. The results of the right heart catheterisation were presented in Table 1.

The findings in Table 1 revealed a patent foramen ovale (PFO) with a small intermittent right to left shunt (Qp:Qs ratio 1.1:1)

The case was discussed at the Cardiovascular Management Meeting with the cardiothoracic surgeons and the decision was made for medical therapy. There were no further need for interventions or trans-oesophageal echocardiogram, as the patient was stable, asymptomatic and the shunt was small.

Table 1. Right Heart Catheterisation Study and Baseline Oximetry Results

Location	Pressures and Oximetry
Right Atrium (RA) pressure	20 mmHg
Right Ventricle (RV) pressure	Systolic 35 mmHg, Diastolic 14 mmHg
Pulmonary Artery (PA) pressure	Systolic 30 mmHg, Diastolic 18 mmHg
Left Atrium (LA) pressure	20 mmHg
Cardiac Output (Fick's equation)	6.72 L/min
Cardiac Index (Fick)	3.47 L/min/m^2
Body Surface Area	1.94 m^2
Systemic Vascular Resistance (SVR)	1071 dynes s/cm^2
Pulmonary Vascular Resistance (PVR)	98.48 dynes s/cm^2
Inferior Vena Cava (IVC)	71%
Superior Vena Cava (SVC)	65.2%
Mid RA	65.9%
Low RA	65.8%
RV	65.1%
Main PA	67.4%
Right PA	67.4%
Left PA	65%
LA	93.3%
Femoral Artery	94.2%
Pulmonary Vein	91.4%

The patient remained stable and his oxygen saturation improved over the next few days. He was successfully weaned off the BiPAP and was discharged 3 days later. He remained on dual anti-platelet therapy for 2 months, followed by long term aspirin therapy. He remained asymptomatic at the following 3 week visit prior to returning to Phillipines. He was advised to seek further follow-up in the Phillipines.

Discussion

When patients with MI present with persistent arterial hypoxaemia, one has to exclude causes such as left ventricular failure with pulmonary congestion, pre-existing lung diseases, pulmonary embolism and intra-cardiac shunt.[4]

A right-to-left interatrial shunt in the presence of MI carries a substantial risk of morbidity and mortality. Rapid diagnosis and treatment can significantly improve outcome. Efforts should be made to optimise the right ventricle function with pressors and volume expansion, in order to reduce the degree of shunting. There are no randomised controlled trials to guide the management and therapy of such cases as it is rare and one should take the approach on a case to case basis.

Revascularisation of the culprit lesion should be the first priority to maximise the right ventricular function. The use of pulmonary dilating drugs such as nitric oxide and positive inotropic agents such as dobutamine, have been shown to improve right ventricular function. In cases where there is haemodynamic compromise and bradyarrhythmias, AV pacing may be needed.[5]

Right ventricular function can improve after acute MI, and the non-surgical/non-interventional therapies described above may allow time for a sufficient recovery. However, if a clinically significant shunt persists or if the right ventricular function does not recover, then one must consider percutaneous or surgical closure of the PFO.

The clinical syndrome of acute right-to-left interatrial shunt through a PFO is under recognised. In conclusion, one must consider the diagnosis when there is persistent hypoxaemia in the setting of inferior myocardial infarction.

REFERENCES

1. Andersen HR, Falk E, Nielsen D. Right ventricular infarction: frequency, size and topography in coronary heart disease: a prospective study comprising 107 consecutive autopsies from a coronary care unit. J Am Coll Cardiol 1987;10:1223-32.
2. Goldstein JA. Pathophysiology and management of right heart ischemia. J Am Coll Cardiol 2002;40:841-53.
3. Isner JM, Roberts WC. Right ventricular infarction complicating left ventricular infarction secondary to coronary artery disease. Frequency, location, associated findings and significance from analysis of 236 necropsy patients with acute or healed myocardial infarction. Am J Cardiol 1978;42:885-94.
4. Fessler MB, Lepore JJ, Thompson BT, Semigran MJ. Right-to-left shunting through a patent foramen ovale in right ventricular infarction: improvement of hypoxemia and hemodynamics with inhaled nitric oxide. J Clin Anesth 2003; 15:371-4.
5. Nacht A, Kronzon I. Transesophageal echocardiography and innovative echocardiographic technology intracardiac shunts. Crit Care Clin 1996;12:295-314.

Nadira Hamid,[1] *MB BCh (Hons), MRCPI (Ireland), MRCP (UK),* Felix Keng,[1] *MBBS, M MED (Int Med), FRCP (London)*

[1]National Heart Centre Singapore

Address for Correspondence: Dr Nadira Hamid, National Heart Centre Singapore, Mistri Wing 17 Third Hospital Avenue Singapore 168752. Email: nadira.hamid@singhealth.com.sg