CASE REPORT

Transient right-to-left intracardiac shunt following a right ventricular myocardial infarction

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SUMMARY

A 56-year-old smoker presented with a 3-day history of intermittent chest pain. Cardiovascular examination on admission was normal. ECG showed minimal (<1 mm) ST elevation in lead III, and right-sided leads revealed similar mild ST-elevation. Troponin I was elevated at 10.91. He was managed as a delayed-presentation ST-elevation myocardial infarction case. 12 h following admission, he developed oxygen-resistant hypoxia and hypotension. There were no clinical or radiological signs of pulmonary congestion. CT pulmonary angiogram revealed no pulmonary embolus. A bubble-contrast echocardiogram confirmed an active interatrial right-to-left shunt without Valsalva provocation. His hypoxia steadily improved over the following 24 h and he did not require any shunt closure. A repeat bubble contrast echocardiogram showed that the shunt was now only active following a Valsalva manoeuvre. Transoesophageal echocardiography confirmed a patent foramen ovale. This is a rare but important cause of resistant hypoxia following a right-heart infarct.

BACKGROUND

This was a very interesting case because while right-ventricular infarcts are common, an intracardiac shunt following these infarcts is a rare but serious complication. The important learning point in this case was that intracardiac shunts should be considered in patients with resistant hypoxia with no pulmonary oedema or significant lung pathology.

CASE PRESENTATION

A 56-year-old smoker with a background of cluster headaches and renal calculi was admitted with a 3-day history of intermittent chest pain. His pain had been constant over the previous 18 h.

On initial assessment, his blood pressure was 130/70, heart rate was 80 bpm, jugular venous pressure (JVP) was not elevated, heart sounds were normal and chest was clear and all peripheral pulses were palpable. The abdomen was soft with no tenderness or organomegaly. Neurological exam showed no focal neurological deficit.

Troponin I was elevated at 10.91. ECG showed <1 mm ST elevation in lead III with small inferior Q waves suggestive of recent inferior myocardial infarction. Right-sided chest leads showed some mild ST elevation. He did not undergo primary percutaneous coronary intervention (PCI) given his late presentation and minimal ST elevation. He was therefore managed conservatively with aspirin, clopidogrel, fondaparinux and low-dose intravenous nitrates. His chest pain resolved rapidly.

Twelve hours following his admission, he was noted to be hypoxic with SpO₂ of 89% in room air. Arterial blood gas revealed a pO₂ of 7.88, pCO₂ of 4.29, pH of 7.46 on 4 litres of oxygen. His blood pressure had fallen to 95/41 mm Hg. Clinically, there were no signs of pulmonary congestion or bronchospasm. His respiratory rate measured 25 and his JVP was elevated. His hypotension was attributed to some right ventricular (RV) involvement of his infarct, responding to slow intravenous fluids. He remained hypoxic.

Chest x-ray showed no pulmonary oedema. A transthoracic echocardiogram revealed right heart dilation with reduced RV wall contractility and mild left ventricular inferior hypokinesis.

A CT pulmonary angiogram showed no evidence of a pulmonary embolus and revealed mild emphysematous changes.

His saturations failed to improve over the next 8 h despite continuous up-titration of oxygen. His pO₂ measured 7.42 on 10 litres of high flow oxygen.

A subsequent bedside transthoracic echocardiogram with agitated saline demonstrated the immediate passage of a large amount of bubbles across the atrial septum into the left atrium without Valsalva provocation.

His hypoxia was deemed secondary to the development of a right to left shunt of desaturated blood via a PFO as a consequence of acute changes in RV pressures following his myocardial infarction (MI). His hypoxia proved resistant to oxygen therapy, consistent with this hypothesis, and he was therefore transferred across to the regional interventional centre for consideration of transoesophageal echocardiogram (TOE) and possible atrial septal defect/patent foramen ovale (PFO) closure if his hypoxia persisted.

Over the next 24 h, his hypoxia steadily improved and no interventional procedure was performed.

A repeat departmental bubble study 4 days post-MI showed an aneurysmal atrial septum with the passage of bubbles into the left heart on Valsalva manoeuvre (figure 1). A TOE confirmed a PFO and an aneurysmal interatrial septum with evidence of intermittent left-to-right shunting on a colour flow Doppler.

Cardiac catheterisation following resolution of the hypoxia revealed a right coronary artery (dominant) proximal occlusion (figure 2) with collateral filling from the left coronary artery. At this stage, the patient was pain-free and thus did not undergo PCI to the right coronary artery lesion.

The patient made an uneventful recovery and was discharged home 10 days post MI. He was seen again in the clinic 6 weeks and 4 months later

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Figure 1 Transthoracic echocardiogram showing passage of bubbles from the right heart to the left.

and was clinically well with no hypoxia. Right heart dimensions on an echo performed 4 months post MI were normal, with just mild inferobasal hypokinesis within the left ventricle.

DISCUSSION

We report a case of transient right-to-left interatrial shunting manifesting as profound hypoxia resistant to oxygen therapy, following MI. This is a rare but important complication that should be recognised early. There are 20 previous case reports of this phenomenon with varying severities and outcomes.^{1–18}

RV infarctions occur in approximately a quarter of the transmural posteroinferior MIs.¹⁹ The clinical incidence of patent foramen ovale in otherwise normal patients was found to be approximately 27% in a large autopsy study published in 1984.²⁰ Studies have shown that a raised RV systolic pressure could cause an increase in RA pressures.^{21–22} This, in effect, causes a right-to-left shunt across a previously silent PFO.

On review of the literature, we identified 17 reported cases of post MI intracardiac shunting secondary to a PFO. Sharif *et al*¹⁵ published a literature review in 1999 describing 15 such cases, including 3 of their own, and there have been two case reports since that time.^{17–18} Overall, of all these reported cases,

Figure 2 Coronary angiography showing a blocked right coronary artery.

eight underwent PFO closure and nine were managed conservatively.^{15–17–18}

This patient's case was interesting as he improved without intervention to close the PFO. His symptomatic improvement correlated directly with his improvement in cardiac function following myocardial stunning after his MI.

Early recognition of this condition is important as early closure of the PFO minimises end-organ damage and infarct extension secondary to arterial hypoxaemia.

Learning points

- ▶ Transient right-to-left shunting across a usually dormant patent foramen ovale/atrial septal defect could occur following changes in right-heart pressures—in this case, following a right ventricular infarct.
- ▶ Resistant hypoxia in a patient with a clinically and radiologically unremarkable chest could represent intracardiac shunting.
- ▶ It is important to investigate and manage this promptly to prevent end-organ damage.

Competing interests None.

Patient consent Obtained.

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