Bristol Publishers

Case Reports and Case Series in Cardiology Journal

Case Report | Open Access | Volume: 01 Issue: 01 (April-June 2025) Pages: 06-10

ISSN: 3107-6416 (Online) | DOI: doi.org/10.64874/crcscj.v1i1.2025.002

Persistent Unexplained Desaturation After Myocardial Infarction

Haritha Kosanam ✉, Ram Manohar Talupula and Ramesh Babu Pothineni

Department of Cardiology, Aster Ramesh hospital, Vijayawada, Andhra Pradesh, India

Corresponding author: Dr. Haritha Kosanam, Department of Cardiology, Aster Ramesh hospital, Vijayawada, Andhra Pradesh, India. E-mail: harithakosanam@gmail.com.

Received: 17 January 2025; **Revised:** 22 March 2025; **Accepted:** 03 April 2025; **Published:** 05 June 2025

Academic Editor: Dr. Naga Sai Shravan Turaga

Abstract

Hypoxemia following myocardial infarction (MI) is frequently attributed to left ventricular dysfunction, valvular regurgitation, or pulmonary causes. However, intracardiac shunting, such as a patent foramen ovale (PFO), may also play a critical role in refractory cases. We present a 67-year-old woman with inferior wall MI and persistent desaturation despite successful revascularization and optimized medical management. Saline contrast echocardiography revealed a PFO with a right-to-left shunt. Conservative management with supportive care and improvement of right ventricular (RV) function led to the resolution of symptoms, underscoring the importance of comprehensive evaluation for unexplained hypoxemia in MI patients.

Case Presentation

A 67-year-old diabetic along with hypertensive woman presented within retrosternal chest pain lasting 12 hours and an episode of non-bilious vomiting. On admission, she was in cardiogenic shock, having a heart rate of 56 beats per minute and a blood pressure around 80/60 mmHg. Her blood gases remained normal, with the exception of a PaO2 of 55, whereas her oxygen saturation remained 90% on room air. The electrocardiogram showed reciprocal alterations in leads I and aVL and ST elevation across the inferior leads (II, III, and aVF), and 2:1 atrioventricular (AV) block with a prolonged PR interval (Figure 1).

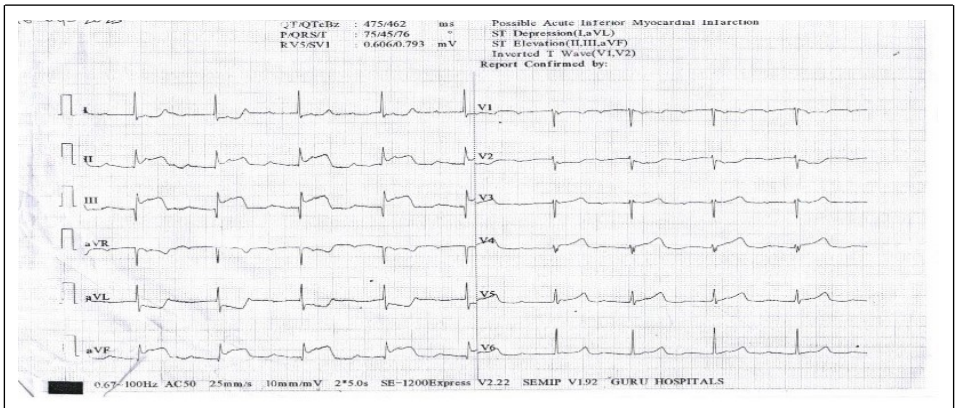

Figure 1: Electrocardiogram showing heart rate of 56/min, Leads II, III, and avF have ST segment elevations, while I and avL have ST depression.

Citation: Kosanam H, Talupula RM, Pothineni RB. Persistent Unexplained Desaturation After Myocardial Infarction. Case Rep Case Ser Cardiol J. April-June 2025; 01(01): 06-10. **DOI:** doi.org/10.64874/crcscj.v1i1.2025.002.

Echocardiography revealed moderate mitral regurgitation (MR), mild tricuspid regurgitation (TR), right ventricular (RV), hypokinesia in the basal and mid inferior wall, and an ejection fraction (EF) of 45%. dysfunction, and a mildly dilated RV (Figure2).

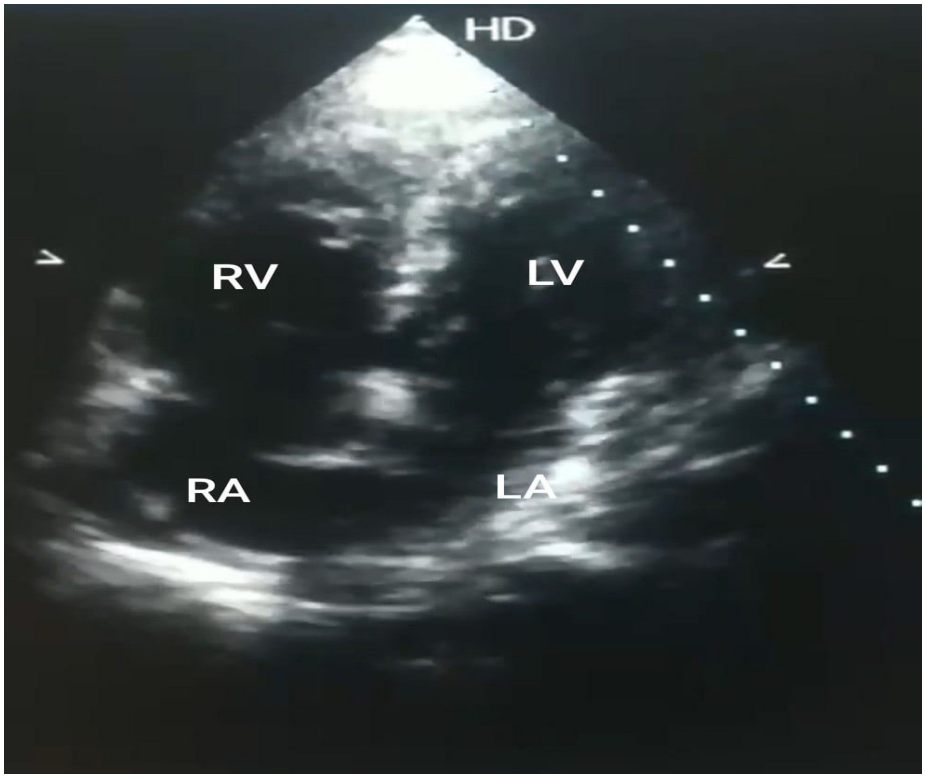

Figure 2: Echocardiogram showing dilated right ventricle in RV focused apical 4 chamber view.

The right coronary artery (RCA) was completely blocked, the left circumflex artery (LCX) had 90-95% stenosis, and the proximal left anterior descending artery (LAD) had 30-40% stenosis, according to emergency coronary angiography. Under intra-aortic balloon pump (IABP) assistance, Everolimus-eluting stent insertion was used for primary percutaneous coronary intervention (PCI) to the RCA (Figure 3a and 3b).

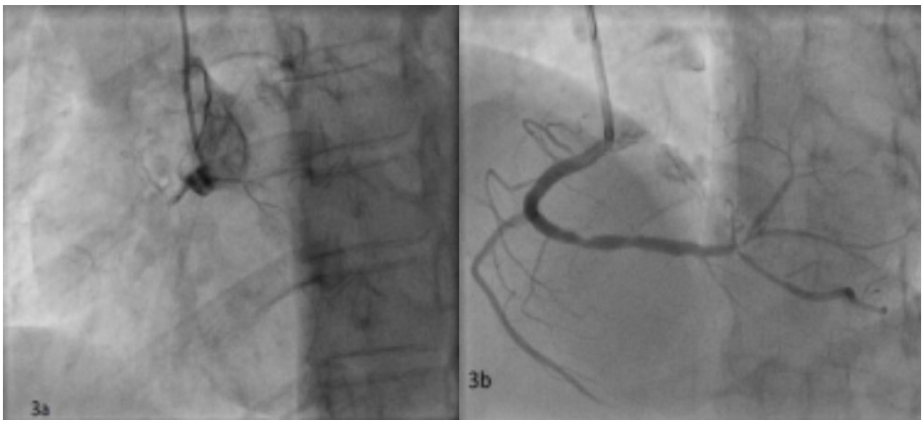

Figure 3a and 3b: 3a- LAO projection of RCA showing subtotal occlusion of RCA prior to stent deployment, **3b**- LAO projection of RCA showing TIMI 3 flow post stent deployment with residual mid to distal nonobstructive disease.

Citation: Kosanam H, Talupula RM, Pothineni RB. Persistent Unexplained Desaturation After Myocardial Infarction. Case Rep Case Ser Cardiol J. April-June 2025; 01(01): 06-10. **DOI:** doi.org/10.64874/crcscj.v1i1.2025.002.

Following the procedure, her blood pressure improved, and inotropes were weaned. Repeat echocardiography showed improvement in EF to 50%, persistent moderate MR, and mild RV dysfunction with a 12mm is the tricuspid annular plane systolic excursion (TAPSE). In spite of improved LV function, the patient remained dyspneic at rest, with persistent oxygen saturation of 90-91%. Suspecting MR as the cause of desaturation, PCI to the LCX was performed after four days (Figure 4a and 4b).

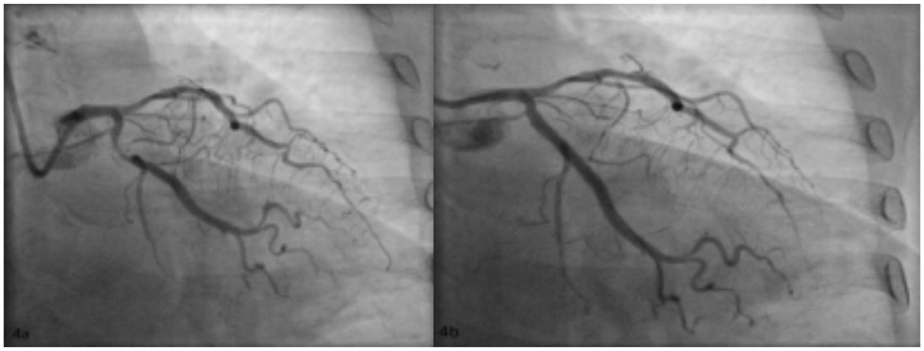

Figure 4a: RAO projection of the left system with a highgrade stenosis in proximal Lcx. **Figure 4b:** RAO projection of Lcx stenosis post stent deployment.

However, despite complete revascularization and use of high-dose loop diuretics, her desaturation persisted. A CT chest revealed scattered subpleural fibrotic bands in the basal segments but no significant lung parenchymal disease (Figure 5). High-flow oxygen therapy was ineffective.

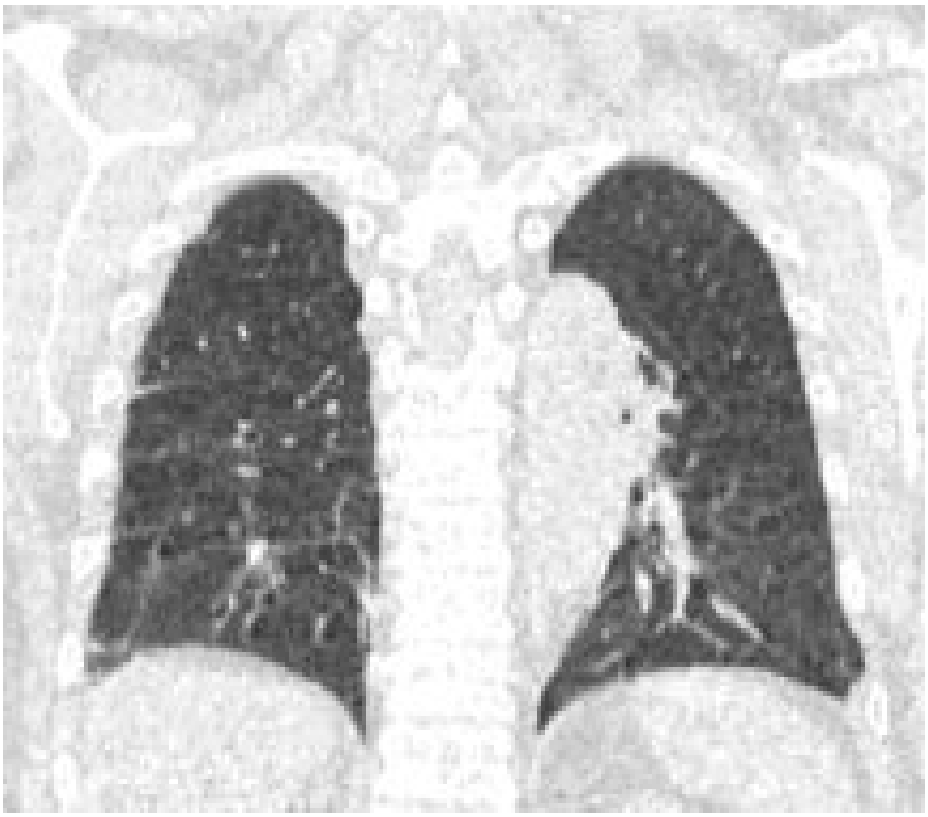

Figure 5: Computed tomography of chest showing subpleural fibrotic bands.

Citation: Kosanam H, Talupula RM, Pothineni RB. Persistent Unexplained Desaturation After Myocardial Infarction. Case Rep Case Ser Cardiol J. April-June 2025; 01(01): 06-10. **DOI:** doi.org/10.64874/crcscj.v1i1.2025.002.

A saline contrast echocardiogram revealed a PFO with a right-to-left shunt (Figure 6a and 6b). Considering the improvement in RV function following RCA PCI, the heart team opted for conservative management. The patient's oxygen requirement gradually decreased, and she was discharged on home oxygen therapy (2 L/min) with a saturation of 94-95%. At follow-up, her RV function improved, oxygen saturation normalized to 95% on room air, and dyspnea resolved.

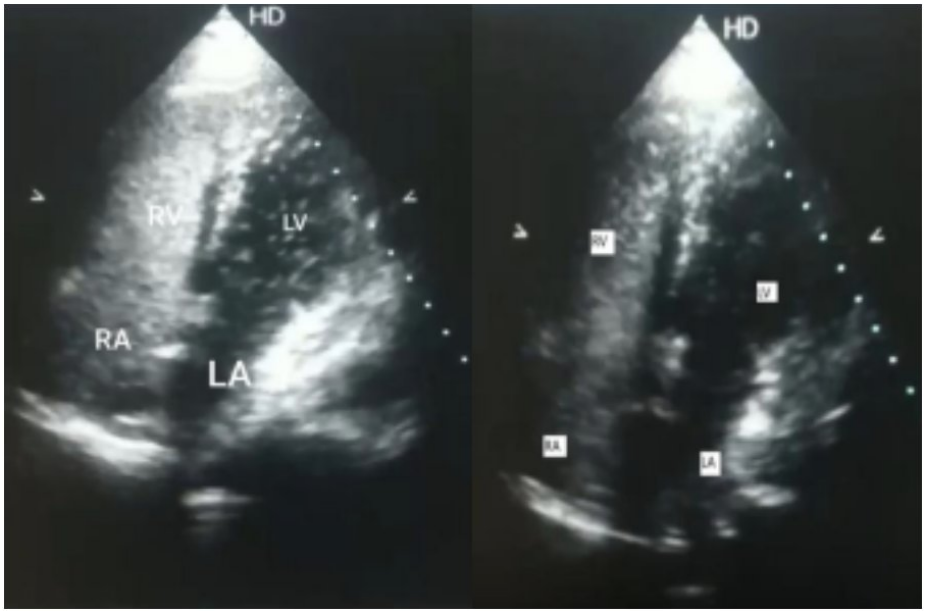

Figure 6a and 6b: Agitated saline contrast administration showing right heart opacification with early crossing of saline bubbles to the left heart suggestive of intracardiac shunt (patent foramen ovale in our case).

Discussion

Hypoxemia following myocardial infarction (MI) is commonly attributed to pulmonary edema, left ventricular (LV) dysfunction, or valvular abnormalities such as mitral regurgitation. However, when hypoxemia persists despite addressing these common causes, less frequent etiologies, including intracardiac shunting through a patent foramen ovale (PFO), should be considered. This case illustrates the significant role of PFO in causing refractory hypoxemia after MI, particularly in the setting of right ventricular (RV) infarction.

PFO is a congenital defect present in approximately 25% of the population. Under normal physiological conditions, the foramen flap remains closed, preventing blood flow between the atria. However, certain hemodynamic alterations, such as increased right atrial (RA) pressure secondary to RV infarction, can lead to right-to-left shunting [1]. The inferior wall MI in this patient extended to the RV, resulting in RV systolic dysfunction and diastolic restrictive physiology, which increased RA pressure [2,3]. This hemodynamic disturbance facilitated persistent shunting of deoxygenated blood from the RA to the left atrium (LA), bypassing the pulmonary circulation and causing hypoxemia.

Persistent hypoxemia in a post-MI patient, despite optimized management of LV function and valvular regurgitation, warrants further evaluation for a shunt lesion [4]. Saline bubble contrast echocardiography is the diagnostic modality of choice, as it can identify intracardiac shunting with high sensitivity. In this case, transthoracic echocardiography with saline contrast successfully demonstrated a PFO with a right-to-left shunt, confirming the diagnosis. While transesophageal echocardiography (TEE) is considered the gold standard for identifying PFO, high-quality transthoracic imaging may suffice in certain clinical settings [3].

Citation: Kosanam H, Talupula RM, Pothineni RB. Persistent Unexplained Desaturation After Myocardial Infarction. Case Rep Case Ser Cardiol J. April-June 2025; 01(01): 06-10. **DOI:** doi.org/10.64874/crcscj.v1i1.2025.002.

Management of PFO-related hypoxemia after MI is complex and centers on improving the underlying hemodynamics. In this case, revascularization of the RCA with stenting improved RV perfusion and systolic function, leading to a gradual decrease in shunting. Supportive medical treatment using angiotensin-converting enzyme inhibitors and beta-blockers further contributed to hemodynamic recovery. Device closure of the PFO was deferred, as improving RV function often reduces shunting without the need for invasive intervention. This conservative approach aligns with evidence suggesting that many cases of PFO-related hypoxemia resolve with optimized RV support and revascularization [5].

The role of device closure in PFO-related hypoxemia remains controversial and should be carefully individualized. Premature closure in the setting of unresolved RV infarction may exacerbate RV failure and reduce LV preload, potentially worsening hemodynamics. Device closure is typically reserved for cases where shunting persists despite optimized medical therapy or where hypoxemia significantly impairs quality of life [6,7]. Furthermore, positive pressure ventilation, often employed in refractory hypoxemia, is contraindicated in this setting. It can increase RA pressure, exacerbate shunting, and lead to hemodynamic deterioration by reducing RV preload and increasing afterload [8].

It emphasizes how crucial it is to keep a high level of skepticism. for PFO in patients with refractory hypoxemia after MI, especially those with RV involvement. A structured diagnostic approach, emphasizing the early use of contrast echocardiography, is essential for timely recognition of this condition. While most cases can be managed conservatively, the decision to close a PFO must consider the patient's clinical course and the risk-benefit profile of intervention. By focusing on revascularization, RV support, and individualized management strategies, clinicians can optimize outcomes in these complex cases.

This case highlights PFO as an underrecognized cause of persistent hypoxemia in post-MI patients and emphasizes the need for a thorough evaluation when common causes have been excluded. A conservative approach to management is often sufficient, with device closure reserved for refractory cases, ensuring effective treatment while minimizing risks.

REFERENCES

1. Mojadidi MK, Christia P, Salamon J. Patent Foramen Ovale: Unanswered Questions. Eur J Intern Med. 2015; 26: 743-751.
2. Hamid N, Keng F. Refractory Hypoxia in Right Ventricular Infarction. Ann Acad Med Singapore. 2011; 40: 325-326.
3. Franco T, Melández J, Malkin R, et al. Acute Right to Left Shunt Through Patent Foramen Ovale Presenting as Hypoxemia after Myocardial Infarction: A Case Report. Cases J. 2009; 2: 8878.
4. Cheng TO. Platypnea-Orthodeoxia Syndrome: Etiology, Differential Diagnosis, and Management. Catheter Cardiovasc Interv. 1999; 47: 64-66.
5. Krishnamurthy A, White H, Ahmed R. Transient Right-to-Left Intracardiac Shunt Following a Right Ventricular Myocardial Infarction. Bmj Case Rep. 2013; bcr2012008398.
6. Gudipati CV, Nagelhout DA, Serota H, et al. Transesophageal Echocardiographic Guidance for Balloon Catheter Occlusion of Patent Foramen Ovale Complicating Right Ventricular Infarction. Am Heart J. 1991; 121: 919-922.
7. Lin W, Yip JWL, Yeo TC. Refractory Hypoxaemia Following Inferior ST-Segment Elevation Myocardial Infarction: Case Report of an Unusual Complication and Review of Treatment Strategies. Heart Lung Circ. 2015; 24: 157-161.
8. Kernis SJ, Goldstein J, Yerkey M, et al. Percutaneous Atrial Septostomy for Urgent Palliative Treatment of Severe Refractory Cardiogenic Shock Due to Right Ventricular Infarction. Catheter Cardiovasc Interv. 2003; 59: 44-48.

Citation: Kosanam H, Talupula RM, Pothineni RB. Persistent Unexplained Desaturation After Myocardial Infarction. Case Rep Case Ser Cardiol J. April-June 2025; 01(01): 06-10. **DOI:** doi.org/10.64874/crcscj.v1i1.2025.002.