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Case Report

A Case of Refractory Hypoxemia Secondary to Intracardiac Shunt Diagnosed in the Catheterization Laboratory

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An 80-year-old female patient presented to our institution with an inferior ST-elevation myocardial infarction, cerebral hypoxia, and refractory hypoxemia, without signs of pulmonary congestion. Invasive right-sided heart catheterization facilitated the diagnosis of a right-to-left (R-L) cardiac shunt. An unidentified R-L cardiac shunt is a rare cause of refractory hypoxemia in patients with a preexisting, unknown atrial septal defect or patent foramen ovale, particularly in cases presenting with right ventricular myocardial infarction (RVMI). A prompt diagnosis of this condition can guide the management of these patients and prevent the implementation of deleterious treatment measures. Herein, we detail the initial presentation, investigations, management, and clinical course of this patient.

Case Report

An 80-year-old female patient was brought to our institution by paramedics after being found on the floor of her senior residence with new-onset aphasia and right hemiparesis. The patient was previously known to have hypertension and anticoagulated atrial fibrillation. Upon the paramedics' arrival, the patient was severely hypotensive and had an oxygen saturation of 60% on room air, accompanied by cyanosis. Vital signs at arrival revealed a blood pressure of 94/54 mm Hg and a heart rate of 82 beats per minute. Notably, severe and persistent hypoxemia was present, with an oxygen saturation of 80%, despite the administration of 100% oxygen via a facemask. Physical examination did not reveal any cardiac murmur, and the lungs were clear. The patient was managed promptly with the activation of the stroke team. Urgent brain and neck computed tomography (CT) angiography showed no acute lesions, and her neurologic symptoms resolved spontaneously. Due to her unexplained hypoxemia, a chest CT without contrast was performed also, revealing no signs of lung congestion or pneumonia.

Despite the absence of any reported chest pain, an electrocardiogram was ordered, revealing a surprising 2-mm ST-segment elevation in the inferior leads, accompanied by small Q waves. The initial high-sensitivity troponin I level was 6197 ng/L, and the patient displayed a lactate level of 5.6 mmol/L. The clinical impression strongly suggested an evolving inferior ST-segment elevation myocardial infarction (STEMI) with cardiogenic shock. Given the persistent hypoxemia, despite high-flow oxygen, and indications of low cardiac output, the decision was made to transfer the patient to the cardiac catheterization laboratory for invasive management.

Coronary angiography revealed a significant ostial right coronary artery (RCA) stenosis, along with a completely occluded mid RCA, distal to the takeoff of the right ventricular branches. Nonsignificant disease was present in the left coronary artery. Left ventricular end-diastolic pressure (LVEDP) was measured at 16 mm Hg, and the left ventricular ejection fraction (LVEF) was recorded at 40% during ventriculography, with no indication of ventricular septal defect or severe mitral regurgitation. Urgent revascularization of the culprit artery was carried out, involving the implantation of 3 drug-eluting stents in the ostial and mid segments of the RCA.

Despite the culprit artery having been addressed, the patient remained severely hypotensive and hypoxemic, a condition disproportionate to the observed coronary and chest CT findings. During the same procedure, a pulmonary angiogram was conducted to rule out proximal pulmonary embolism. Additionally, a decision was made to perform a right heart catheterization, revealing a pulmonary artery

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See page 668 for disclosure information.

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Novel Teaching Points

- Refractory hypoxemia in acute myocardial infarction should be investigated with right heart catheterization.
- Intracardiac shunt should be suspected in cases of refractory hypoxemia associated with RVMI.
- Interventions that may increase preload or decrease afterload, in case of a shunt associated with RVMI, should be avoided.

pressure of 27/15 mm Hg, a right ventricular end-diastolic pressure of 13 mm Hg, and a right atrial (RA) pressure of 22 mm Hg, displaying a restrictive pattern. Low cardiac output was demonstrated, with a Fick cardiac output of 2.7 L/min. Given the elevated RA pressure compared to the LVEDP, suspicion arose regarding an underlying R-L atrial shunt. Consequently, we opted to probe the atrial septum using a regular J-curved .035 wire, which swiftly entered the left atrium (Video 1, view video online). The left atrial (LA) pressure measured 19 mm Hg, and blood drawn from the left superior pulmonary vein revealed an oxygen saturation of 100%, contrasting with the 82% found in the aorta. This finding confirmed the presence of an R-L atrial shunt as the cause of the refractory hypoxemia. The working hypothesis was that the patient experienced a right ventricular infarct, elevating right-sided heart pressure and leading to an R-L shunt through a possible preexisting atrial septal defect (ASD) or patent foramen ovale.

The patient was transferred to the cardiac care unit for further medical management of her cardiogenic shock and intracardiac shunt. A transthoracic echocardiogram (TTE) revealed inferior wall left ventricular (LV) hypokinesis with a left ventricular ejection fraction (LVEF) of 45%, a dilated right ventricle (RV) with severely depressed function, and severe tricuspid regurgitation. The TTE also confirmed the presence of a large ASD measuring 13 mm, exhibiting a bidirectional shunt (Fig. 1; Video 2, view video online). Following milrinone perfusion and diuretics, her hemodynamic parameters rapidly improved, and her oxygen saturation increased to 95% after only 3 hours of therapy. She was successfully weaned off milrinone and oxygen therapy within 48 hours.

A repeat TTE on day 8 showed an improved LVEF of 50%, a still severely hypokinetic RV, and a shift in the ASD shunt from R-L to left-to-right (Fig. 2). Thus, given the improvement in the patient's hemodynamics and LV function, low-dose beta-blockers and an angiotensin-converting enzyme (ACE) inhibitor were introduced to address her coronary artery disease, per guideline-directed medical therapy.

The unusual and acute presentation of the patient led to the discovery, 4 days after her admission, that she had sustained a right femoral neck fracture following her fall. A hip replacement was performed on day 5 following admission. On day 9, the patient was considered stable enough to be transferred to the general cardiology ward. Although she remained hemodynamically stable for the rest of her hospitalization, she experienced delirium and deconditioning. Inpatient rehabilitation was initiated, and she was discharged to a rehabilitation facility on day 28.

After hospital discharge, she attended 3 outpatient visits, and was consistently stable, with no lingering angina or dyspnea symptoms. Adjustments are underway for her atrial fibrillation rate control. Currently, she awaits a follow-up TTE, the first since her hospitalization.

Discussion

Refractory hypoxemia, coupled with an unremarkable chest radiograph or lung examination in the context of acute myocardial infarction, should consistently raise suspicion for a right-to-left shunt. The incidence of RVMI ranges from 30% to 50%, and its occurrence can lead to R-L shunting in patients with a preexisting ASD, due to the acute increase in RA pressure.[1,2] The anticipated progression is that as the RV function improves, the R-L shunt should decrease. Patients with a patent

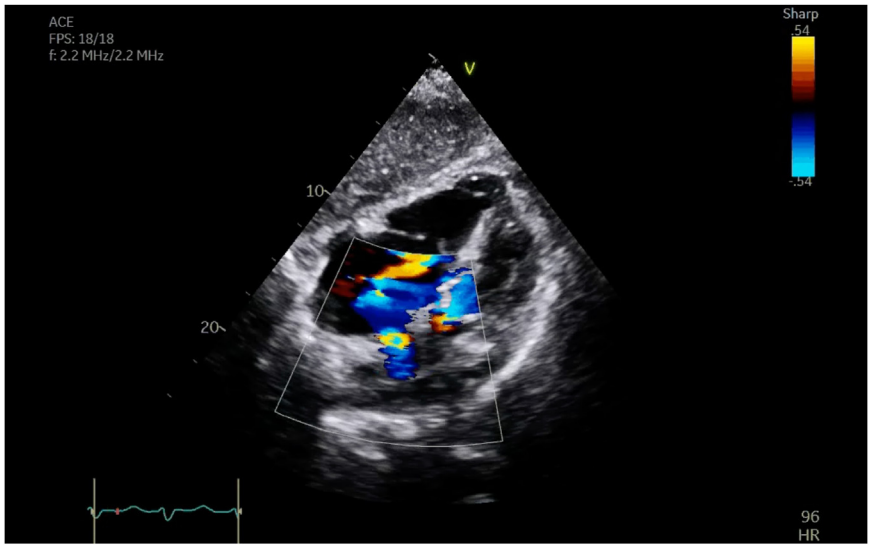

Figure 1. Initial echocardiography with bidirectional shunt.

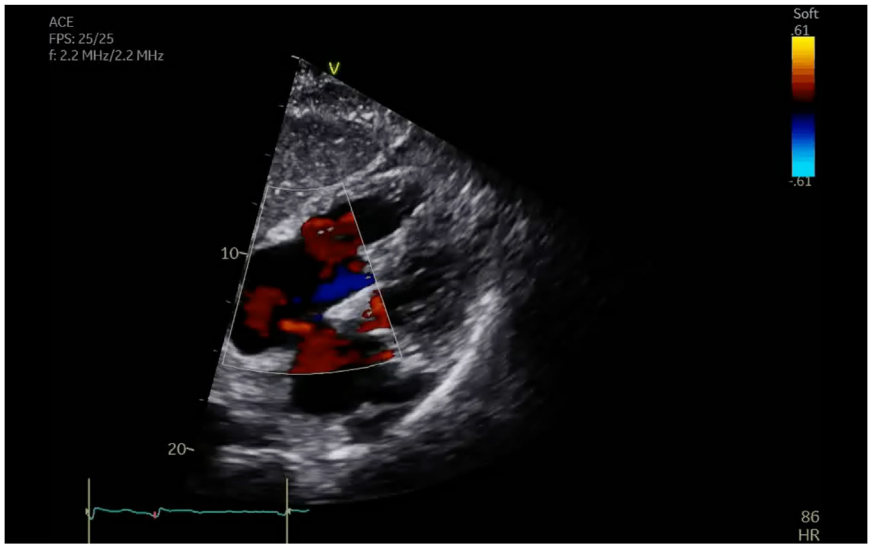

Figure 2. Predischarge echocardiography with left-to-right shunt.

foramen ovale or undiagnosed ASD may remain asymptomatic, and the shunt may manifest itself for the first time as refractory hypoxemia in the context of RVMI, especially when the RA pressure exceeds the LA pressure. This pathophysiological mechanism was thoroughly documented in our report, with the patient's RA pressure of 22 mm Hg surpassing her LA pressure of 19 mm Hg and her LVEDP of 16 mm Hg.

Early bedside echocardiography remains the gold standard for diagnosing R-L shunting during inferior myocardial infarction. However, in the unpredictable and emergent context of acute myocardial infarction, especially when an expert cardiac imaging specialist is not readily available before the patient is transported to the catheterization lab, interventionists should not hesitate to perform right heart catheterization. This procedure is particularly relevant if uncertainty persists regarding the origin of hypoxia even after echocardiography. In our case, the patient was promptly diagnosed with an R-L shunt during her primary percutaneous coronary intervention, enabling us to avoid medications or interventions that could exacerbate the shunt.

In RVMI without a suspected R-L shunt, hypotension is typically managed by maintaining adequate preload through fluid boluses, and refractory hypoxemia is addressed with oxygen therapy and positive pressure ventilation. However, boluses increase RV preload, and positive pressure ventilation increases transpulmonary pressure and decreases LV preload and afterload. In the specific context of an ongoing R-L shunt, these interventions elevate right heart cavity pressure and decrease left heart cavity pressure, exacerbating shunting and worsening hypoxia. Also, closing this shunt in the acute setting of RVMI carries the risk of worsening the cardiac output, as a significant portion of the LA preload depends on the shunt bypassing the severely depressed RV.[3]

Inotropes, in addition to diuretics, should be initiated early in the medical management of this condition, to enhance RV function and reduce RV preload, aiming to revert the shunt back to left-to-right and alleviate hypoxia. In our case, the patient responded rapidly to this combined therapy.

According to the guidelines, achieving revascularization of the culprit lesion in acute myocardial infarction is the cornerstone of treatment, as reperfusion significantly improves RV function. In this case, the main presentation involved neurologic symptoms attributed to hypoxia and low cardiac output. Although the diagnostic cascade was deemed appropriate, given the patient's unusual presentation, these neurologic symptoms prompted an additional stroke evaluation, introducing some delays in the management of her ST-elevation myocardial infarction (STEMI).

Closure of an ASD is indicated when a sufficiently large left-to-right shunt is present that leads to RA and RV enlargement or dysfunction, provided that pulmonary pressure and/or resistance are not elevated excessively.[3,4]

Conclusion

Refractory hypoxemia in the absence of pulmonary edema in patients with myocardial infarction should raise suspicion of an intracardiac shunt. A prompt diagnosis can be lifesaving, and the role of right heart catheterization during primary angioplasty is essential in these cases.

Ethics Statement

This case report has adhered to relevant local ethical guidelines.

Patient Consent

The authors confirm that a patient consent form has been obtained for this article.

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Disclosures

The authors have no conflicts of interest to disclose.

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Supplementary Material

To access the supplementary material accompanying this article, visit *CJC Open* at https://www.cjcopen.ca/ and at https://doi.org/10.1016/j.cjco.2024.01.004.