

Patent Foramen Ovale: An Unusual Cause of Refractory Hypoxemia

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Case Report

We report the case of a 70-year-old woman with a history of cryptogenic ischemic stroke for which she underwent intravenous fibrinolysis with no neurological sequelae. She had no known history of pulmonary or heart disease.

Approximately 1.5 years later, she presented to the Emergency Department (ED) with a one-week history of cough, fatigue, and shortness of breath. She denied fever, chest pain, orthopnea, or paroxysmal nocturnal dyspnea.

Upon admission, she was normotensive with a heart rate of 90 bpm, eupneic despite a peripheral O₂ saturation level of 90%, and afebrile. The remaining physical examination was unremarkable, except for basal crackles on pulmonary auscultation. Initial arterial blood gas (ABG) on O₂ at 1L/min via nasal cannula showed a pH of 7.46, pCO₂ of 36 mmHg, HCO₃ of 26 mmHg, pO₂ of 61 mmHg, SaO₂ of 91%, and lactate of 1.3 mmol/L.

Initial blood work showed no anemia as well as raised troponin, N-terminal pro-B-type natriuretic peptide, and inflammatory biomarkers. A broad respiratory pathogens polymerase chain reaction panel was negative for SARS-CoV-2, but parainfluenza virus was detected. Chest X-ray showed no pleural effusion or consolidations. Chest CT angiography ruled out pulmonary embolism. However, it showed mild ground-glass opacities with central distribution and atelectasis of the lung bases.

Her type 1 respiratory failure progressively worsened during her 24-hour stay in the ED, with no objective cause — an ABG showing a pO₂ of 54 mmHg on a 60% Venturi mask (P/F ratio = 90). However, she had no significant signs of increased work of breathing. At that time, she was admitted to an intensive care unit (ICU).

She was started on high-flow nasal cannula (HFNC) oxygen therapy in the ICU and titrated to 90%/50L. She showed gradual improvement, allowing weaning from the HFNC, but still had significant oxygen requirements.

No other microorganisms were isolated (urinary antigen tests, blood cultures), and serologic tests for autoimmune diseases were negative.

A high-resolution chest CT scan was performed at 72 hours (Figure 1). It showed areas of consolidation in the lower lobes, particularly at the bases, with air bronchogram. However, there was no clinical evidence of bacterial infection.

Because of the cryptogenic cause of respiratory failure and chest CT findings that were not proportional to the severity of hypoxemia, a transthoracic echocardiogram bubble study was performed (Video 1). It showed non-dilated cardiac cavities, mild left ventricular septal hypertrophy, preserved left and right systolic function, no significant valvular regurgitation, and no apparent macroscopic defect of the interatrial septum. However, the passage of many contrast bubbles (> 30) shortly after entering the right atrium raised the suspicion of a patent foramen ovale (PFO).

A transesophageal echocardiogram (Videos 2 and 3) showed detachment and tunneling of the fossa ovalis membrane with an almost continuous right-to-left shunt and a very significant, immediate, continuous bubble contrast passage after injection of agitated saline. There were no other obvious defects of the interatrial septum.

A Swan-Ganz right heart catheterization (RHC) performed 10 days after ICU admission documented the absence of pulmonary hypertension (mean pulmonary artery pressure of 15 mmHg) with normal right and left filling pressures. Cardiac output was preserved. The Qp/Qs ratio was 1.5.

Patient underwent transcatheter closure of PFO with a CeraFlex™ PFO Occluder (25 × 30 mm) (Video 4). She was on a 60% Venturi mask at the time. Subsequent echocardiographic follow-up showed no residual shunt. After the procedure, her respiratory failure improved significantly. She was discharged home with no need for ambulatory oxygen therapy.

Although we can conclude the primary cause of severe persistent hypoxemia was the open PFO with right-to-left shunt, the acute viral respiratory infection also contributed, not only as a mechanism for the shunt but also as a factor contributing to hypoxemia.

Discussion

PFO occurs in 25–30% of the general population, with a higher prevalence in patients with cryptogenic stroke.¹ Noninvasive diagnosis can be done by an echocardiogram (transthoracic/transesophageal) bubble contrast study.²

PFO-mediated hypoxemia occurs when deoxygenated venous blood from the right atrium enters and mixes with oxygenated arterial blood in the left atrium. Patients with

Keywords

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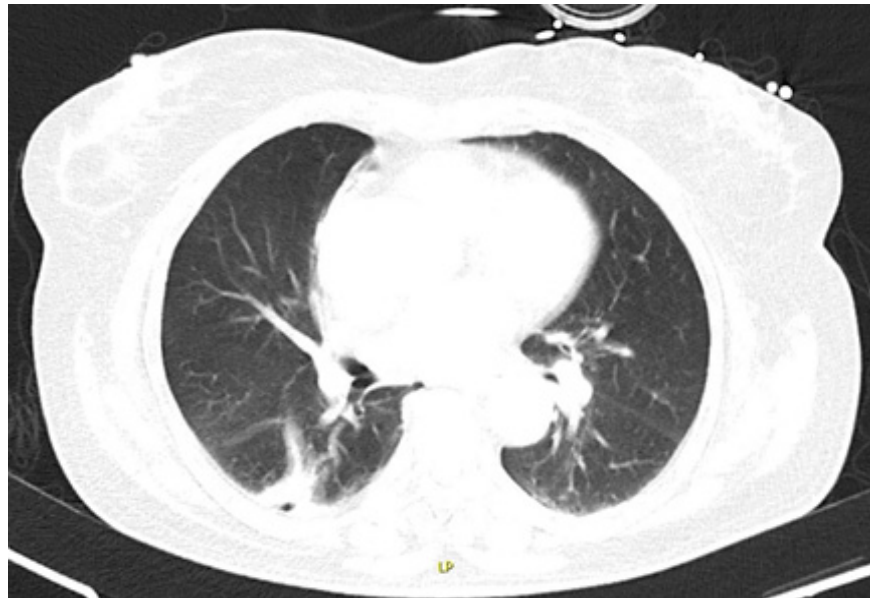
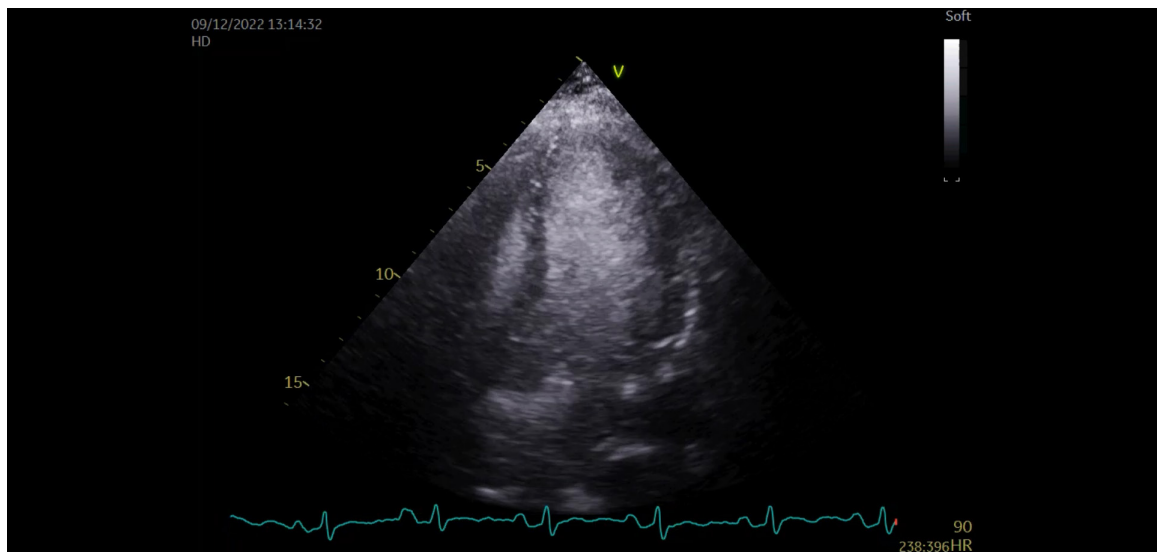


Figure 1 – Chest CT scan (transversal plane): areas of consolidation with air bronchogram in lower lobes



Video 1 – Link: http://abcimaging.org/supplementary-material/2024/3702/2024-0030-video_01.mp4

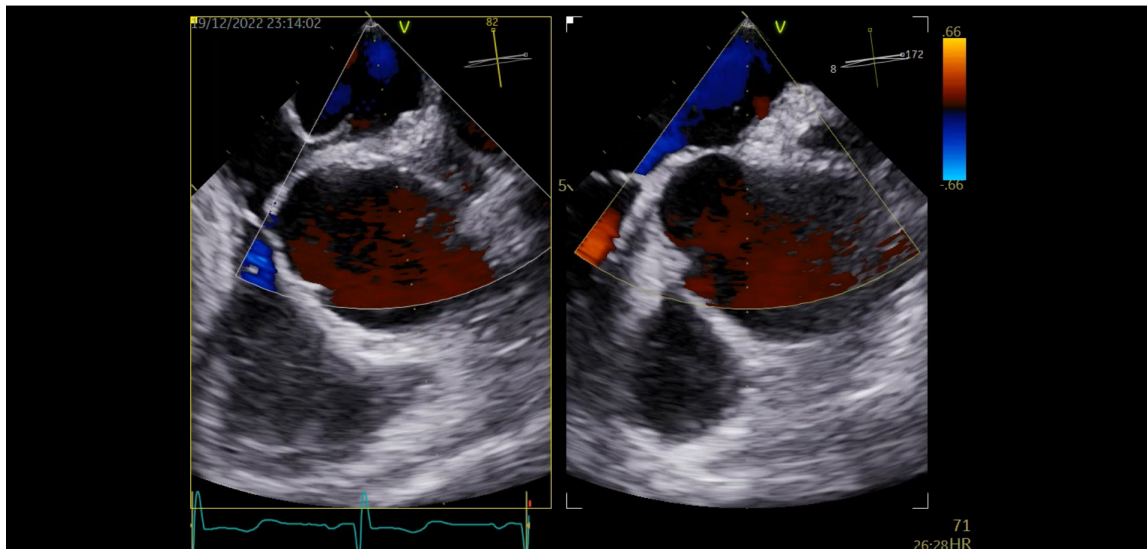
an intracardiac right-to-left shunt may experience intense hypoxemia disproportionate to the underlying primary lung disease.^{3,4}

Hypoxemia associated with PFO usually occurs secondary to right-to-left shunt in the presence of pulmonary hypertension. However, hypoxemia in the absence of pulmonary hypertension is less common. This condition has been reported in the medical literature and is thought to be because of abnormal anatomic features that promote right-to-

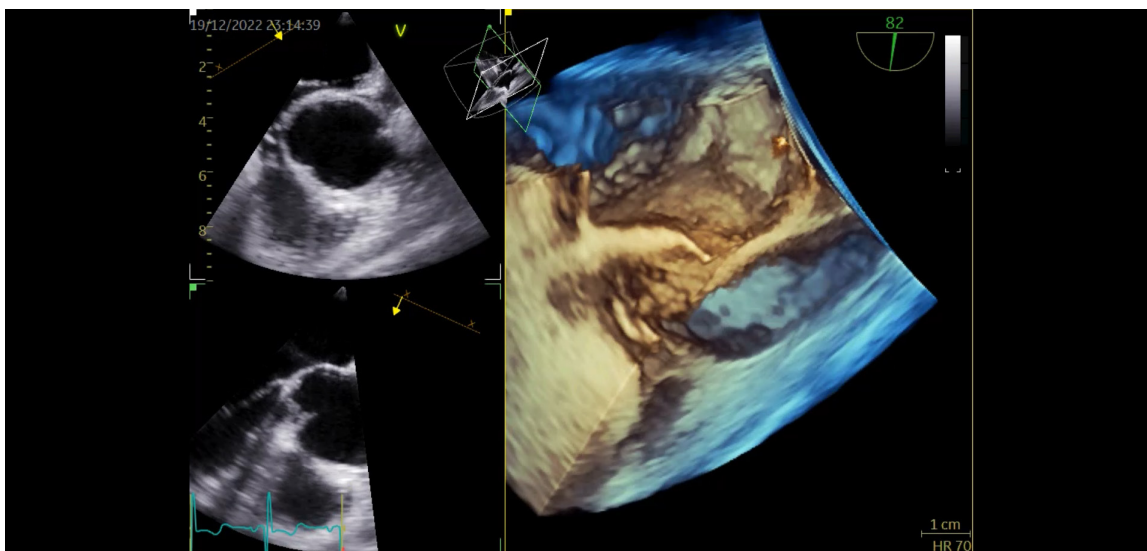
left shunt through the atrial septal defect, such as prominent Eustachian valve, Ebstein anomaly, or a right atrial mass.³⁻⁵

In this clinical report, we assume that the acute viral respiratory infection may have transiently elevated pulmonary artery pressures (both directly and through hypoxemia), reversing the right-to-left shunt through the large PFO, leading to disproportionately severe hypoxemia. Had the RHC been performed earlier, the hemodynamic results may have been different.⁶⁻⁷

Case Report



Video 2 – Link: http://abcimaging.org/supplementary-material/2024/3702/2024-0030-video_02.mp4



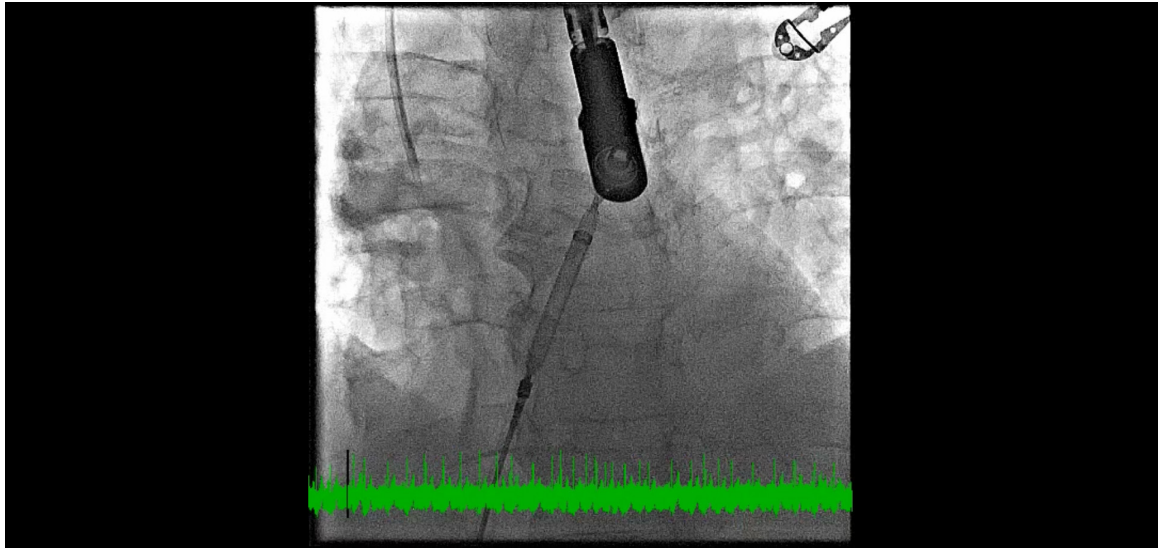
Video 3 – Link: http://abcimaging.org/supplementary-material/2024/3702/2024-0030-video_03.mp4

PFO closure for systemic hypoxemia remains controversial. To date, only small studies have been conducted, with conflicting results regarding hypoxemia and functional improvement.⁸ Successful closure in these cases requires not only acceptable periprocedural and long-term risk (known as “procedural success”) but also complete reversal of the shunt and resolution of hypoxemia (known as “clinical success”).⁹ Therefore, the risk-benefit ratio and physiologic considerations must be carefully considered before choosing percutaneous device closure as a treatment modality for hypoxemia.¹⁰ In a subset of patients, particularly in the absence of severe

pulmonary hypertension, percutaneous PFO closure may result in a significant improvement of dyspnea and hypoxemia.^{4,11}

Conclusion

Physicians should be aware of the association between right-to-left shunting and hypoxemia, as removal of the shunt may result in decreased oxygen requirements. Although removal of the shunt appeared to improve blood oxygenation in this patient, further research is needed to inform practice in this clinical setting.



Video 4 – http://abcimaging.org/supplementary-material/2024/3702/2024-0030-video_04.mp4

Author Contributions

Conception and design of the research and analysis and interpretation of the data: Miranda I, Strong C, Póvoa P; acquisition of data and writing of the manuscript: Miranda I, Strong C; critical revision of the manuscript for intellectual content: Strong C, Póvoa P.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This article does not contain any studies with human participants or animals performed by any of the authors.

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