

Case Reports

Platypnea-Orthodeoxia and Patent Foramen Ovale in a Patient in the Setting of COVID-19

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Abstract

Platypnea-orthodeoxia syndrome is a rare condition characterized by positional dyspnea and hypoxemia, with symptoms presenting in the upright position and improving when recumbent. Hypoxemia in platypnea-orthodeoxia syndrome is defined as a drop in PaO_2 of more than 4 mm Hg or oxygen saturation greater than 5% when moving from a supine to an upright position. Etiologic mechanisms include intracardiac or pulmonary shunts and ventilation perfusion ratio mismatch. Platypnea-orthodeoxia syndrome has been described as part of COVID-19 pneumonia sequelae, with differing physiologic mechanisms from cardiac pathologies. This report presents the case of a patient with platypnea-orthodeoxia syndrome of multifactorial etiology: intracardiac shunting secondary to a patent foramen ovale and ventilation perfusion ratio mismatch as a sequela of COVID-19 pneumonia.

Keywords: Heart septal defects, atrial; dyspnea; foramen ovale, patent; platypnea orthodeoxia syndrome; supine position

Case Report

Presentation and Physical Examination

A 69-year-old female patient was referred to the reporting institution for evaluation of patent foramen ovale (PFO) closure. The patient initially presented to a community hospital with left facial weakness, disorientation, and behavioral changes and was found to have had an ischemic stroke in the area of the right anterior cerebral artery.

Medical History

The patient's medical history included hypertension and recent COVID-19 pneumonia with residual hypoxemia in the prior 2 weeks.

Investigations

As part of the stroke workup, a transthoracic echocardiogram with bubble study was performed that revealed an intracardiac shunt with more than 100 bubbles within 2 cardiac cycles and a mobile intra-atrial septum. The right atrium and right ventricle were mildly dilated with normal pulmonary pressures. A lower extremity Doppler image was positive for a deep vein thrombosis extending from the left common femoral artery to the popliteal vein. Given the concern for paradoxical emboli as the etiology of the stroke, she was transferred for evaluation.

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On admission to the reporting institution, the patient was found to be confused and disoriented. Physical examination was relevant for positional dyspnea and hypoxemia, with an oxygen saturation of 97% on high-flow nasal cannulas (30 L/80%) while supine and a drop to 82% while sitting up, a constellation of findings suggestive of platypnea-orthodeoxia syndrome. A repeat lower extremity Doppler image again demonstrated left common femoral arterial and popliteal deep vein thrombosis; computed tomographic pulmonary angiography ruled out pulmonary embolism but showed lung parenchymal findings secondary to a subacute infectious process (Fig. 1). Because of concern for a vascular shunt, a ventilation perfusion ratio (\dot{V}/\dot{Q}) scan was performed that was positive for \dot{V}/\dot{Q} mismatch secondary to a right to left shunt (Fig. 2). As part of

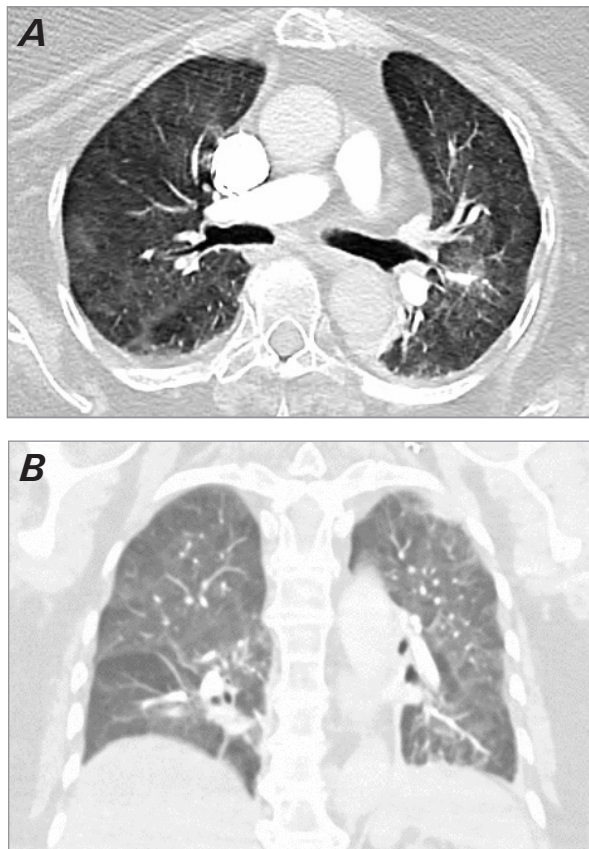


Fig. 1 Contrast-enhanced computed tomographic image of the chest (pulmonary embolism protocol) shows (A) the axial view at the level of the mainstem bronchi, with diffuse areas of patchy ground-glass opacities consistent with a mosaic attenuation pattern, and (B) the coronal view, which demonstrates the extent of diffuse lung parenchymal involvement. Overall findings are strongly suggestive of subacute infection in the setting of recent COVID-19 pneumonia.

Key Points

- **Assess all probable causes.** It is important to evaluate all potential causes of platypnea-orthodeoxia syndrome in patients presenting with positional hypoxemia, especially when a PFO and \dot{V}/\dot{Q} mismatch are involved.
- **Impact of COVID-19.** Recent COVID-19 subacute lung injury can contribute to the symptoms of platypnea-orthodeoxia syndrome, which may not fully resolve after PFO closure.
- **Targeted differential diagnosis.** Given the multifactorial nature of platypnea-orthodeoxia syndrome, a targeted differential diagnosis is essential for effective treatment.

Abbreviations

PFO, patent foramen ovale
 \dot{V}/\dot{Q} , ventilation perfusion ratio

Supplementary Materials

For supplemental materials, please see the online version of this paper.

the preoperative workup, a transesophageal echocardiogram with intravenous saline injection was performed that confirmed a PFO with substantial shunt flow and aneurysmal dilatation of the atrial septum (Fig. 3).

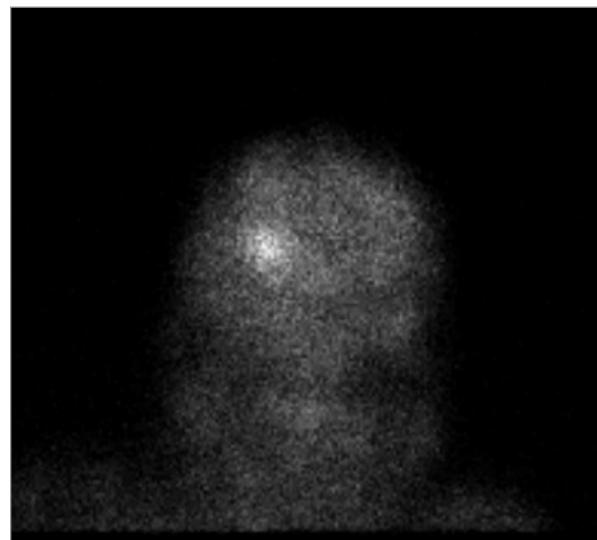


Fig. 2 Perfusion-only lung scan shows an anterior view of the brain with clinically significant tracer uptake in the region of interest. Findings are strongly suggestive of a right-to-left shunt.

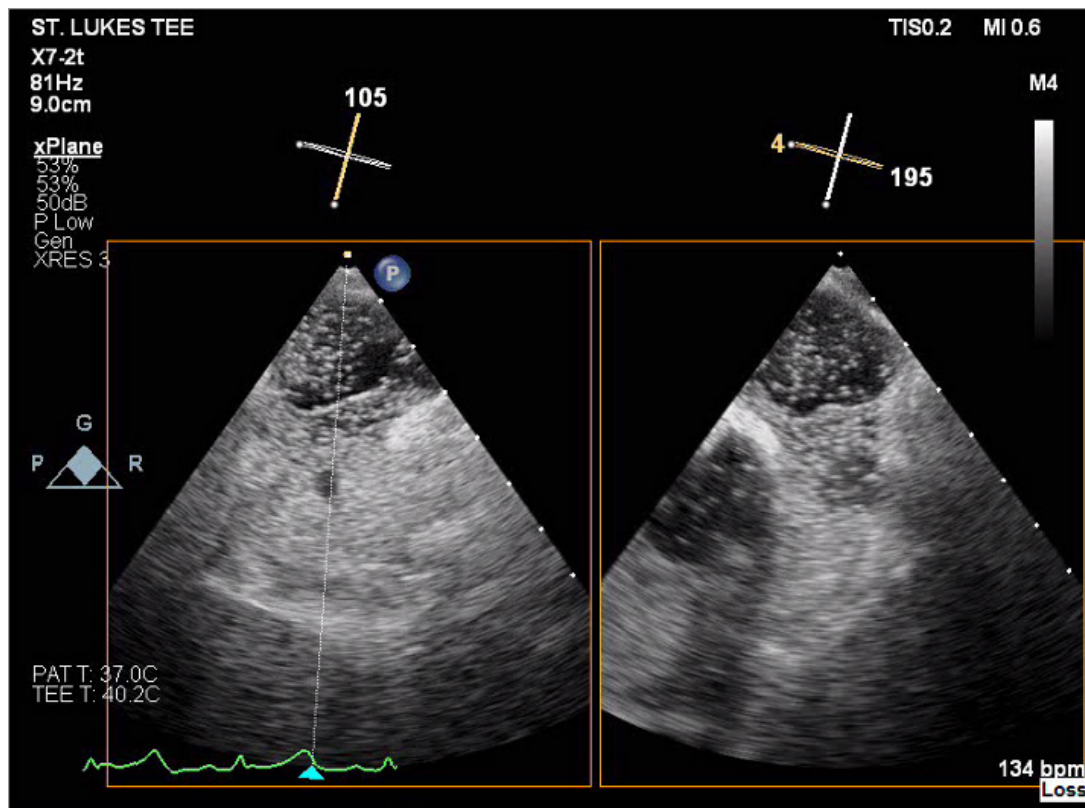


Fig. 3 Apical 2-chamber view on an transesophageal echocardiogram on admission shows substantial shunting during intravenous saline injection consistent with a patent foramen ovale.

Supplemental motion image is available for Figure 3.

Technique

The patient underwent transcatheter PFO closure with a 25-mm Amplatzer Talisman PFO Occluder device (Abbott Medical) without immediate complications. After the procedure, the patient was progressively weaned off high-flow nasal cannulas, achieving oxygen saturations ranging from 92% to 96% on 4 L oxygen. Postoperative transthoracic echocardiography revealed complete atrial septal occlusion with the patent closure device (Fig. 4).

Outcome

A week after the procedure, the patient continued to require oxygen, despite successful PFO occlusion, which was strongly suggestive of a multifactorial etiology or additional aggravating factors, most likely her subacute COVID-19 pneumonia. Her oxygen saturation on room air was 91% at rest and 88% with mild exertion, which increased to 97% and 94%, respectively, with 1 L oxygen via nasal cannula. The patient was then discharged with home oxygen to a skilled nursing facility to complete rehabilitation.

Discussion

Platypnea-orthodeoxia syndrome is a clinical syndrome with several etiologies, all of which have been well elucidated in the literature. This report presents a rare case of a patient with multifactorial etiology for platypnea-orthodeoxia syndrome as a result of her intracardiac shunt via a PFO, with COVID-19-mediated lung injury and an atrial septal aneurysm acting as exacerbating factors.

The pathophysiologic mechanism of platypnea-orthodeoxia syndrome in the setting of intracardiac shunts—in this case, a PFO—is associated with the right atrial stretch forces and the direction of blood flow within the atrium.^{1,2} While the individual is in the supine position, the stretch forces of the right atrium keep the atrial septum tense and therefore keep the PFO small or shut. At the same time, the direction of blood flow in the atrium tends to favor flow from the inferior vena cava into the atrial cavity. With the patient in the standing position, stretch forces in the right atrium conversely decrease, so the septum relaxes, leading to the aperture

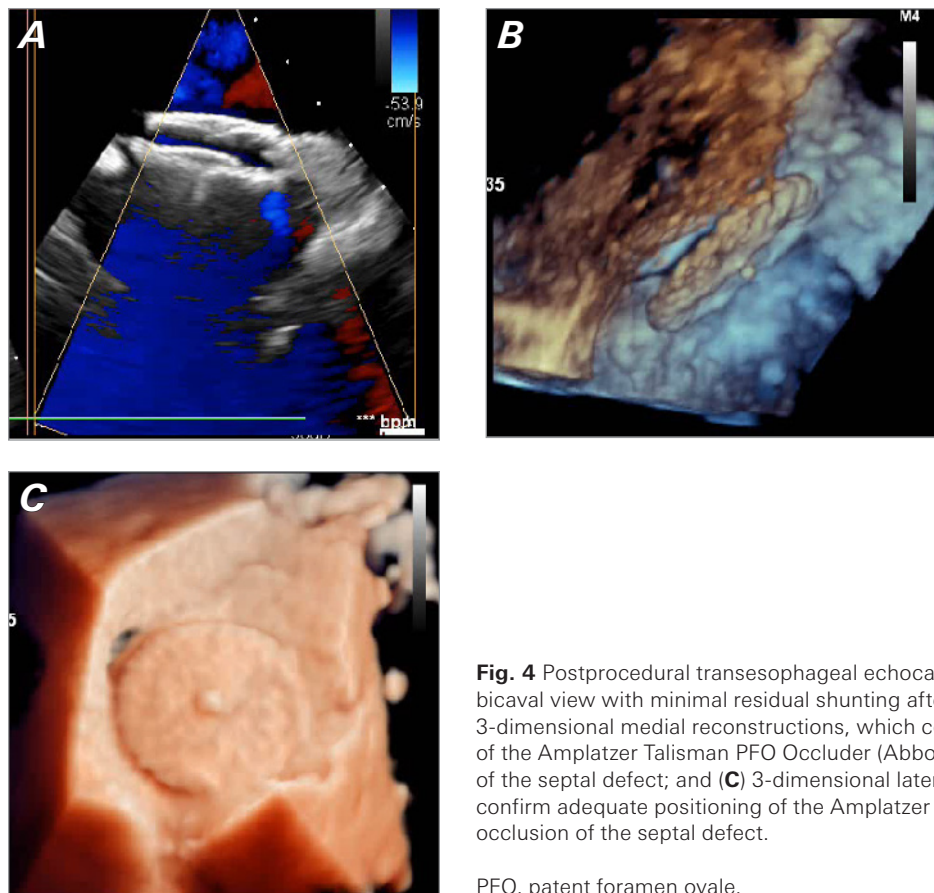


Fig. 4 Postprocedural transesophageal echocardiogram shows (A) a bicaval view with minimal residual shunting after device deployment; (B) 3-dimensional medial reconstructions, which confirm adequate positioning of the Amplatzer Talisman PFO Occluder (Abbot Medical), with occlusion of the septal defect; and (C) 3-dimensional lateral reconstructions, which confirm adequate positioning of the Amplatzer Talisman PFO Occluder, with occlusion of the septal defect.

PFO, patent foramen ovale.

of the PFO and increased shunting. Blood flow is directed from the inferior vena cava toward the shunt, exacerbating shunting. The patient in the current report presented with an atrial septal aneurysm that acted as a further aggravating factor for the shunt. Atrial septal aneurysms produce a back-and-forth swinging motion for approximately 10 mm, which pulls the interatrial communication open and worsens the shunt.²⁻⁴

The treatment for symptomatic PFOs involves interventional management to close the communicating defect. The patient's Risk of Paradoxical Embolism score was 5. Given the patient's underlying hypercoagulable state, the fact that she exhibited an atrial septal aneurysm was another indication for PFO closure. This procedure entails the percutaneous insertion of an occlusive device, usually umbrella shaped or metal mesh, using a catheter into the interatrial septum to occlude the interatrial communication.⁵

The residual oxygen requirements of the current patient, even after successful PFO closure, indicate a secondary underlying cause or exacerbating factor—in this case, her recent COVID-19 pneumonia. The pro-

posed mechanism of platypnea-orthodeoxia syndrome secondary to COVID-19 is related to the physiologic gravity-mediated shifting of pulmonary blood flow to the lower lung while sitting, resulting in an expected \dot{V}/\dot{Q} mismatch. In a nonpathologic scenario, this physiologic, positional \dot{V}/\dot{Q} mismatch is not clinically significant and occurs in everyone to a lesser or greater extent. Pathology affecting the alveolar-capillary unit of the lower lungs will, however, result in hypoxic vasoconstriction, worsening \dot{V}/\dot{Q} mismatch and ultimately causing an increased shunt fraction and hypoxia. Other factors include alveolar hypoventilation, reduced lung compliance, critical illness myopathy, and microangiopathy, all of which are present in COVID-19.⁶⁻⁸ The fact that COVID-19 has been well documented to cause both acute inflammation and lung injury that result in \dot{V}/\dot{Q} defects may mean it acts as a predisposing factor to platypnea-orthodeoxia syndrome.⁷ Other abnormalities, such as pulmonary arteriovenous malformations of the lower lungs, may exacerbate these deficits.

The definitive management of platypnea-orthodeoxia syndrome is the correction of the etiologic factor.⁵

Supportive measures, including verticalization training with supplemental oxygen, physical rehabilitation, respiratory techniques, and endurance training, have all been reported to have good outcomes in the symptomatic management of the syndrome.^{9,10}

Conclusion

The current case highlights the importance of assessing all probable causes of platypnea-orthodeoxia syndrome in a patient who presents with positional hypoxemia in the setting of a PFO and V/Q mismatch because of recent COVID-19 subacute lung injury in which the symptoms did not completely resolve after PFO closure. As a multifactorial entity, targeted differential diagnosis should be considered when treating these patients.

Article Information

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Conflict of Interest Disclosure: The authors have no conflicts of interest to declare.

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