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Jaya Janadhyala, MD Thomas Jefferson University, jaya.janadhyala@jefferson.edu

Jonathan Foster, MD Thomas Jefferson University, jonathan.foster@jefferson.edu

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A Case of Patent Foramen Ovale as a Cause of Persistent Hypoxia

Jaya Janadhyala, MD1, Jonathan Foster, MD1

1. Department of Medicine, Thomas Jefferson University Hospital, Philadelphia, PA

INTRODUCTION

Patent foramen ovale (PFO) is a congenital cardiac variant caused by failure of the closure of a passage in the atrial septum. It is quite common, occurring in as much as 27% of the population based on autopsy studies.1 Most cases of PFOs are incidentally discovered or found during work-up of cryptogenic strokes as a potential cause of the stroke.¹ New research is being conducted on the role PFOs play in hypoxia from intracardiac right-to-left shunting, including in patients with co-existent cardiovascular and pulmonary disease.²

CASE PRESENTATION

A 52-year-old man with a past medical history of heart failure with reduced ejection fraction (EF 15%) from non-ischemic cardiomyopathy now status post single chamber ICD placement, atrial flutter, and chronic kidney disease presented for worsening hypoxia and bilateral lower extremity edema. Patient acknowledged worsening dyspnea on exertion, orthopnea, and lower extremity edema over the four weeks prior to admission. Other review of systems was negative. Vital signs upon presentation were significant for low blood pressures (90s/50s), tachypnea to mid-20s, and SaO2 88% on 6 L nasal cannula oxygen. Physical exam was significant for elevated JVP, bilateral pitting lower extremity edema, bibasilar crackles up to mid lung fields bilaterally, clubbing of fingernails, and a holosystolic 3/6 murmur. Labs were significant for an acute kidney injury with a creatinine of 2.06 (from baseline 1.7) and proBNP of 7444. Initial right heart catheterization showed elevated filling pressures (RA 15 and PCWP 22). Patient was aggressively diuresed with a furosemide drip with no improvement in hypoxia. Increasing the patient's nasal cannula from 6 L to higher levels also did not improve the hypoxia.

DIFFERENTIAL DIAGNOSIS

The differential could include hypoxia secondary to an exacerbation of the patient's heart failure and pneumonia.

However, the patient's hypoxia did not improve with aggressive diuresis, and the patient did not have any clinical symptoms of pneumonia. Work-up was initiated for patent foramen ovale as a cause of persistent hypoxia. Due to a concern for a shunt, right heart catheterization was repeated with a wedge saturation that did show some degree of shunting. A positional ABG showed a PO2 of 111 mmHg while lying and 72 mmHg while sitting upright. A TEE at that time showed a 6 mm PFO with significant R to L shunt. At this point, work-up was started for possible closure of PFO.

OUTCOME AND FOLLOW-UP

Due to a difficult social situation, including poor family/ social support and uninsured status, decision was made for patient to complete work-up for PFO closure after discharge with social work's assistance and follow-up at structural heart clinic. Patient is being followed in clinic after discharge for further evaluation regarding closure of PFO.

DISCUSSION

This case demonstrates how PFOs can be a contributing cause of hypoxia in patients with underlying cardiovascular disease. Patients with significant right to left shunting from PFOs usually present with hypoxia that does not improve with increasing oxygen requirement. Other symptoms include platypnea and orthodoxia, both of which our patient also demonstrated. Diagnosis of a PFO with shunting can be accomplished via a TEE with bubble study.1 After diagnosis, treatment depends on weighing the risks of cardiac surgery with the benefits of improvement in hypoxia and quality of life. Research is still being conducted on the definitive benefits of surgical repairs of PFOs in patients with hypoxia.2 The patient in this case continues to follow with structural heart after discharge and is being evaluated for surgical repair of his PFO, considering the severity of his shunt symptoms, in hope to improve his quality of life.

KEY POINTS

Patent foramen ovales (PFOs) can be a cause of persistent hypoxia in patients when there is significant right to left shunting. Research is still being conducted on the value of closure of PFOs as a treatment for persistent hypoxia.

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