Hypoxemia Secondary to Right-to-Left Interatrial Shunt Through a Patent Foramen Ovale in a Patient With an Elevated Right Hemidiaphragm

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Though uncommon, right-to-left shunt through a patent foramen ovale with normal right-side pressure and with a normal interatrial pressure gradient has been reported. The speculated pathophysiology is attributed to directional blood flow streaming from the vena cava to the left atrium. Hypoxemia secondary to right-to-left shunt with normal pulmonary artery pressure has been extensively documented after right pneumonectomy. Five prior cases have documented hypoxemia secondary to a right-to-left shunt through a patent foramen ovale in the presence of an elevated right hemidiaphragm. This is the sixth documented case of right-to-left shunt through a patent foramen ovale in the presence of an elevated right hemidiaphragm with a similar presentation in which closure of the patent foramen ovale resulted in resolution of hypoxemia. *Key words: cardiac shunt, septal occlusion device, diaphragm paralysis, refractory hypoxemia, dyspnea, patent foramen ovale*. [Respir Care 2008;53(4):462–465. © 2008 Daedalus Enterprises]

Introduction

A patent foramen ovale is a common finding on echocardiogram and on autopsy. Patent foramen ovale persists in approximately 25–30% of adults.[1,2] Though patent foramen ovale is normally asymptomatic, it is a potential source for a right-to-left shunt in the face of increased pulmonary artery and right heart pressure. We report a case of hypoxemia secondary to a right-to-left interatrial shunt through a patent foramen ovale in a patient with an elevated right hemidiaphragm.

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The authors report no conflict of interest in the content of this case report.

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Case Summary

A 73-year-old white woman was transferred from an outside facility after initially presenting secondary to the complaint of dyspnea on exertion. She was in her usual state of health until 2 weeks prior to presentation, when she noted shortness of breath with exertion after a car trip. She denied any lower-extremity swelling or any history of thromboembolic disease. Her medical history was notable only for osteoarthritis, hypothyroidism, osteopenia, and gastroesophageal reflux disease. Her medications included aspirin, celecoxib, levothyroxine, risedronate, and omeprazole. She denied any occupational or environmental exposures and had a negative tobacco history. At the outside facility her blood oxygen saturation (measured via pulse oximetry) was 80% on room air.

After she was placed on a nonrebreather mask with a 15 L/min oxygen bleed-in, her blood oxygen saturation increased to 93%. Her arterial blood gas values while receiving 15 L/min of oxygen were pH 7.39, P_{CO_2} 41 mm Hg, and P_{aO_2} 65 mm Hg. Interestingly, her chest radiograph revealed an elevated right hemidiaphragm in the absence of any other cardiopulmonary abnormalities. A chest radiograph 6 months prior did not reveal any diaphragmatic elevation. Because of her chest radiograph findings she underwent a sniff test that revealed right hemidiaphragm paralysis, with paradoxical movement.

In the work-up of her hypoxemia, a ventilation-perfusion scan was performed and was read as a low-probability scan. Doppler imaging of her lower extremities was negative for deep venous thrombosis. Her D-dimer level was elevated. A spiral computed tomogram of the chest for pulmonary embolism protocol was negative. Bronchoscopy revealed no endobronchial lesions or other abnormalities. A 2-dimensional echocardiogram revealed an ejection fraction of 50–55%, without any evidence of pulmonary hypertension. The patient was then transferred to our facility for further evaluation.

On arrival to our institution, her blood oxygen saturation was 88% while receiving 15 L/min of oxygen via nonrebreather mask. Her arterial blood gas values were pH 7.43, P_{CO_2} 34 mm Hg, and P_{aO_2} 50 mm Hg while receiving 15 L/min of oxygen, when her blood oxygen saturation was 86%. The outside radiographs were reviewed and confirmed by the pulmonologists and radiologists at our facility. Because of a high clinical suspicion of pulmonary embolism, a pulmonary angiogram was performed. The angiogram was negative. Pulmonary function tests revealed a forced expiratory volume in the first second (FEV_1) of 0.99 L (49% of predicted), a forced vital capacity (FVC) of 1.42 L (44% of predicted), an FEV_1/FVC of 70%, and a diffusing capacity for carbon monoxide of 10.1 mL/min/mm Hg (44% of predicted).[3] There was no significant difference between the supine and seated spirometry values, which suggested that her elevated hemidiaphragm was not substantially affecting her ventilation.

A 2-dimensional echocardiogram with bubble study was performed to evaluate for a cardiac shunt. Agitated saline bubbles appeared in the left heart within 1 to 2 cardiac cycles. The estimated right-ventricular systolic pressure was normal, and she had mild right-ventricular hypertrophy. Cardiology was consulted for a transesophageal echocardiogram, which revealed a 5-mm patent foramen ovale with bi-directional shunting, seen via color Doppler imaging (Figs. 1 and 2). The right atrium was noted to be small, and the right ventricle had right-ventricular hypertrophy. Right-ventricular systolic pressure was again estimated to be normal. All the pulmonary veins were in normal anatomical position. Additional saline injections were performed, with directed evaluation of the pulmonary veins. It was concluded that there was not an intrapulmonary shunt, based on the absence of the early appearance of bubbles in the pulmonary veins.

At cardiac catheterization, a multi-purpose catheter was placed across the patent foramen ovale into the left upper pulmonary vein, which revealed pulmonary venous oxygen saturation of 97%. Despite the finding of right-ventricular hypertrophy on echocardiogram, the patient's hemodynamic measurements were mean right atrial pressure 5 mm Hg, mean right-ventricular pressure 9 mm Hg, mean left atrial pressure 4 mm Hg, and mean pulmonary artery

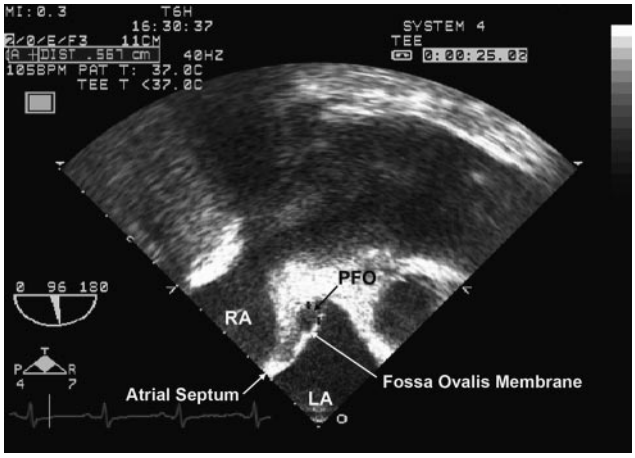

Fig. 1. Transesophageal echocardiogram showing a 0.5-cm patent foramen ovale (PFO).

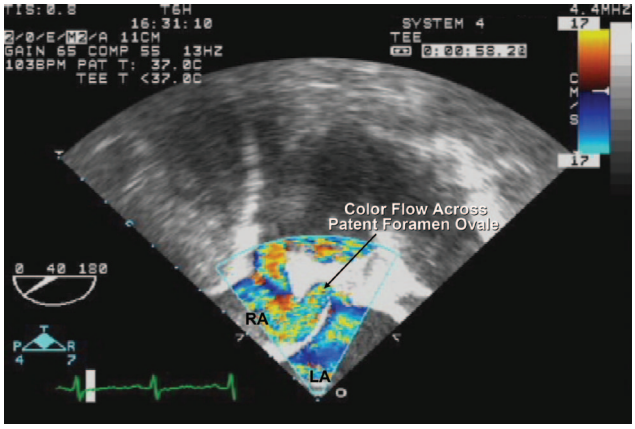

Fig. 2. Transesophageal echocardiogram with color Doppler showing bidirectional flow across the patent foramen ovale (PFO). RA = right atrium. LA = left atrium.

pressure 14 mm Hg. On room air her oxygen saturation was 84%. She was placed on mechanical ventilation for the transesophageal echocardiogram, and on a fraction of inspired oxygen (F_{IO_2}) of 0.8 her oxygen saturation (measured via pulse oximetry) was 97%. The F_{IO_2} was gradually decreased to 0.4, with progressive decline in pulse-oximetry-measured saturation, to 82%. When a sizing balloon (Amplatzer 24 mm sizing balloon, AGA Medical, Plymouth, Minnesota) was placed across the patent foramen ovale and inflated to occlude the patent foramen ovale, her oxygen saturation increased to 97% while on an F_{IO_2} of 0.4 (Fig. 3).

A septal occluder device (Amplatzer septal occluder, AGA Medical, Plymouth, Minnesota) was placed across the patent foramen ovale, which resulted in an oxygen saturation of 95% on room air (Fig. 4). Transesophageal echocardiogram confirmed placement of the septal occluder. After the procedure, her oxygen saturation remained

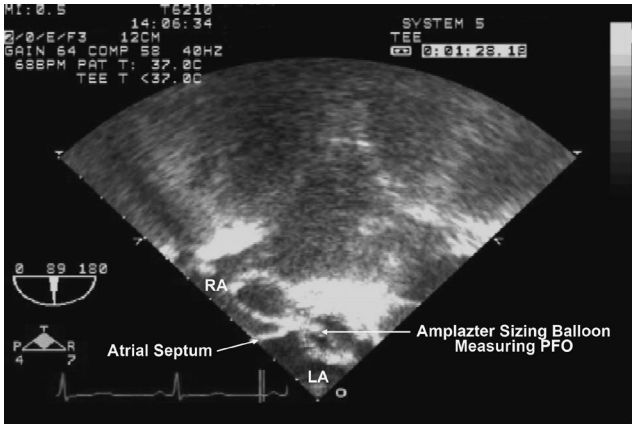

Fig. 3. A 24-mm septal occluder balloon is placed across the patent foramen ovale to occlude right-to-left flow and evaluate the patient's oxygen saturation. RA = right atrium. LA = left atrium.

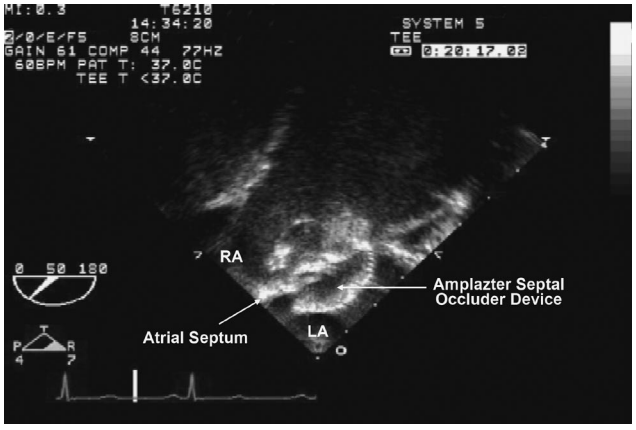

Fig. 4. Amplatzer septal occluder deployed across the patent foramen ovale. RA = right atrium. LA = left atrium.

above 94% during a 6-min walk. She was discharged home on room air. On follow-up in the pulmonary clinic her oxygen saturation was 97% at rest and 94% during exertion. She also reported decreased dyspnea, measured with the Borg dyspnea scale, from 10 (severe) down to 2 (slight).

Discussion

Diagnosis of hypoxemia from an atrial right-to-left shunt through a patent foramen ovale with normal right-side pressure in the absence of an interatrial pressure gradient is uncommon; however, this phenomenon has been documented in several cases after right pneumonectomy and has been reported in the literature as the platypnea-orthodeoxia syndrome.[4-7] In these cases, injection of contrast material in the vena cava at the junction with the right atrium has shown complete filling of the left atrium through the patent foramen ovale.

The pathophysiology of this mechanism has been described by Zanchetta et al as the "flow phenomenon."[8] Normally, blood flow in the right atrium follows the embryologic circulating pattern. The right atrium can be embryologically divided into an anterior and posterior portion. The superior vena cava orifice opens into the anterior portion of the right atrium and blood flows downward and forward through the tricuspid valve, whereas the inferior vena cava orifice arises from the posterior portion of the right atrium and blood flows upward and backward along the fossa ovalis. This creates a pattern of rotation that prevents collision of blood flow and directs the blood toward the tricuspid valve.

In the presence of an elevated right hemidiaphragm or after pneumonectomy, the inferior vena cava becomes more directly aligned with the foramen ovale due to horizontal positioning of the interatrial septum.[8] A shift in the anatomic relationship of the vena cava to the interatrial septum positions the atrial defect so it is in line with the blood flow directly from the vena cava at the entrance to the right atrium. This allows blood to stream through the patent foramen ovale, resulting in a right-to-left shunt with normal right-side pressure and without an interatrial pressure gradient.[8] Controlled tilting of the patient during transesophageal echocardiogram allows detection of the right-to-left shunt.[9] In addition, several case reports on post-pneumonectomy patients have documented resolution of hypoxemia after percutaneous closure of the patent foramen ovale.[4-6]

Our case report provides further support to the theory that a paralyzed hemidiaphragm can contribute to a right-to-left shunt through a patent foramen ovale. Though it is impossible to prove that this patient's right-diaphragmatic paralysis caused the right-to-left shunt through the patent foramen ovale, it is notable that she had a normal chest radiograph 6 months prior, no previous symptoms, and no prior hypoxemia during her endoscopy several years before this event.

There are various causes of unilateral diaphragmatic paralysis. The most common nontraumatic cause is malignant invasion of the phrenic nerve. The second most common etiology is idiopathic.[10] After extensive evaluation, including a detailed neurologic examination and cervical and thoracic spine imaging, it was concluded that our patient's unilateral diaphragmatic paralysis was idiopathic.

This case adds to the existing literature on 5 prior cases of right-to-left shunt via patent foramen ovale in patients with elevated right hemidiaphragm.[9,11-14] We emphasize the importance of including cardiac shunt in the differential diagnosis of hypoxemia, even in the presence of normal cardiac pressures, once other more common diagnoses have been excluded.

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