Case Reports

Hypoxia due to Patent Foramen Ovale

in the Absence of Pulmonary Hypertension

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In most patients with a patent foramen ovale, blood flows from the left atrium to the right atrium in the absence of pulmonary hypertension. Our report describes a patient with a patent foramen ovale in whom flow occurred from the right atrium to the left atrium in the absence of pulmonary hypertension. We discuss hemodynamic findings and present a brief review of the pertinent medical literature regarding this phenomenon. We also discuss the role of transesophageal echocardiography in the diagnosis of this condition and in the elucidation of the underlying mechanisms, and we suggest several mechanisms that may explain the occurrence of this phenomenon in our patient. ***(Tex Heart Inst J 1999;26:306-8)***

In this report, we describe the case of a patient with cyanosis and a significant right-to-left shunt across a patent foramen ovale (PFO) in the absence of pulmonary hypertension. In most patients with a PFO, there is only transient blood flow across the interatrial opening, and this is due to the shift in transthoracic pressure during respiration. Reduced left ventricular compliance results in an increase in left atrial pressure, which decreases the degree of right-to-left flow across a PFO in the absence of pulmonary hypertension. We suggest several mechanisms that may explain the occurrence of significant right-to-left flow in our patient.

Key words: *Echocardiography, transesophageal; foramen ovale, patent; heart septal defects, atrial; hypoxia; shunts*

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Case Report

In January 1998, we evaluated a 74-year-old man for unexplained hypoxia that had recently been discovered during an upper gastrointestinal study for gastroesophageal reflux disease (GERD). His medical history was significant only for hypercholesterolemia, GERD, and a pulmonary contusion due to a motor vehicle accident that had occurred 2 years earlier. His medications included aspirin, simvastatin, omeprazole, and cisapride. Physical examination revealed that he was centrally cyanotic. His blood pressure was 140/70 mmHg and his pulse was 80 beats/minute and regular. His lungs were clear on auscultation, and cardiac examination was normal except for a soft 4th heart sound. The remainder of the physical examination was normal.

Electrocardiography revealed a 1st-degree atrioventricular block and a notched P wave. The patient's chest radiograph was unremarkable except for old fractures in some of his left ribs. Pulmonary function tests, ventilation-perfusion (V/Q) scan, and computed tomographic (CT) scan of his chest failed to elucidate the cause of his hypoxia. While he was on 6 liters of oxygen, the patient's arterial blood gases measured pH 7.42, pCO_2 32, and pO_2 55, and his hemoglobin oxygen saturation was only 93%. These findings suggested chronic compensated respiratory alkalosis secondary to hyperventilation. A transesophageal echocardiogram (TEE), which included multiple bubble studies as well as Doppler studies, revealed a significant right-to-left shunt across a PFO (Figs. 1 and 2) and deviation of the interatrial septum to the left atrial side, which was most pronounced after the P wave and during systole. We felt that this represented an atrial-septal aneurysm with PFO. We could not detect any left-to-right flow.

Hemodynamic findings included normal right heart chambers and pulmonary pressures: right atrial pressure of 4 mmHg, right ventricular pressure of 20/4 mmHg, pulmonary artery pressure of 20/10 mmHg, left ventricular end-diastolic

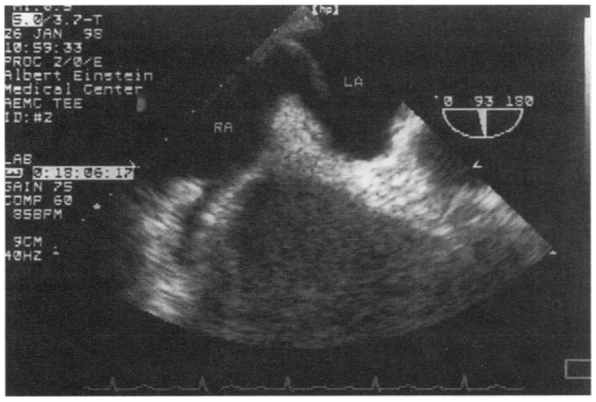

Fig. 1 *Transesophageal echocardiogram demonstrates patent foramen ovale.*

RA = right atrium; LA = left atrium

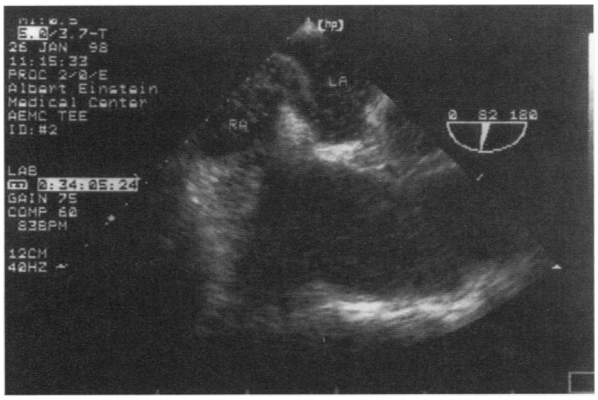

Fig. 2 *After the injection of microbubbles, transesophageal echocardiogram demonstrates flow of microbubbles from right atrium to left atrium.*

pressure of 5 mmHg, pulmonary capillary wedge pressure of 5 mmHg, and aortic pressure of 140/80 mmHg. The right and left atrial pressures were virtually identical. An oximetry run ruled out left-to-right shunting by revealing moderate desaturation at the left atrial cavity and no oxygen step-up at the right atrial level.

The patient's pulmonary venous oxygen saturation was normal (98.8%). In contrast, the left atrial oxygen saturation, which was obtained from the mid-left atrial cavity in the current of the right-to-left shunt, was significantly lower (88%). These results ruled out ventilation perfusion mismatch as a possible cause for systemic arterial oxygen desaturation. Due to the large size of the defect, catheter balloon occlusion of the defect with oximetry studies was not attempted.

The remainder of the oxygen saturation results were as follows (average of 3 samples): main, right, and left branches of the pulmonary artery, 65.8%; aorta, 92%; right ventricle, 66.3%; right ventricular outflow tract, 63.1%; right atrium (low), 68.3%; right atrium (mid), 66.8%; right atrium (high), 57.9%; superior vena cava (low), 56.6%; superior vena cava (high), 67.9%; and inferior vena cava, 69.4%.

Coronary angiography demonstrated a 90% stenosis of the patient's mid left anterior descending coronary artery (LAD), an 80% to 90% stenosis of the apical segment of his LAD, an 80% proximal stenosis of his 1st diagonal coronary artery, and a 60% proximal stenosis of his right coronary artery. These were incidental findings, since the patient had previously denied any symptoms of angina. He underwent repair of the PFO as well as coronary artery bypass surgery. At surgery, the PFO was examined and found to be a 1-cm^2 defect surrounded by redundant tissue. His postoperative course was uneventful. A follow-up echocardiogram demonstrated an acceptable result with normal left ventricular function. His pulse oximetry hemoglobin oxygen saturation at follow-up was 96% in room air, and he was not cyanotic.

Discussion

An anatomically patent foramen ovale may be detected in up to 20% of normal asymptomatic adults.[1] Cyanosis and right-to-left shunting in patients with atrial septal defects (ASD) usually occur in the setting of pulmonary hypertension. In the absence of pulmonary hypertension, however, other mechanisms that may be responsible for the shunting include anomalies of systemic venous return to the left atrium via the superior vena cava or the streaming of blood from the inferior vena cava (IVC) into the left atrium via an ASD. In these cases, the anatomy of the IVC, the Eustachian valve, or the septal defect may allow for marked right-to-left shunting in the absence of a pressure gradient or a mediastinal mechanical distortion.[2] A rare clinical disorder caused by right-to-left shunting of blood across a PFO is platypnea-orthodeoxia. In this syndrome, dyspnea and arterial desaturation become worse when the patient is upright but improve when he or she lies down.[3]

Transesophageal echocardiography provides high-resolution images of the interatrial septum, the fossa ovalis, and its flap valve, the septum primum, in the majority of patients studied. Langholz and associates[4] used saline contrast transesophageal echocardiography to demonstrate distinct mechanisms for right-to left-shunting across a PFO in the absence of pulmonary hypertension. Two such mechanisms are transient reversal of the pressure gradient between the right and left atria during each cardiac cycle and

displacement of the septum primum in accordance with the phase of respiration. It should be noted, however, that a negative air-contrast test does not exclude the presence of a PFO by TEE.[5]

The 1st mechanism, transient reversal of the pressure gradient between the right and left atria during each cardiac cycle, may occur as a result of the location of the sinoatrial node in the right atrium, which causes this chamber to be depolarized slightly earlier than the left atrium during each normal cardiac cycle. The resultant physiologic pressure differential between the right and left atria may be responsible for transient right-to-left shunting.

The 2nd mechanism of right-to-left shunting, displacement of the septum primum in accordance with the phase of respiration, is explained by the finding that the right-to-left pressure differentials vary during the different phases of respiration.[6] During inspiration, the septum primum deflects either against the limbus of the fossa ovalis, which prevents right-to-left shunting, or to the left, which causes marked right-to-left shunting. Respirometric recordings with simultaneous right atrial and wedge or left atrial pressures would be useful in providing these data; however, this was not performed in our patient.

It is possible that anatomic changes responsible for keeping the foramen ovale patent for a longer duration of the cardiac cycle are related to the patient's prior contusion to his chest. According to Gallaher and colleagues,[7] right-to-left shunting may occur following acute events that either reduce left ventricular afterload or decrease right ventricular compliance. Such mechanisms may explain the constant large volume of blood flow across the PFO observed in our patient. If so, the pulmonary contusion may have resulted in a short-term increase in pulmonary pressures that led to a permanent decrease in right ventricular compliance, which in turn caused persistent right-to-left shunt.

Right-to-left shunting is important since it is a potential cause of paradoxical embolism that may lead to cerebrovascular events. Surgical correction is indicated in patients who have persistent cyanosis, exercise intolerance, or known paradoxical embolism.

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