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Right Ventricle Systolic Dysfunction: A Rare Cause of Right-To-Left Interatrial Shunt with Normal Pulmonary Artery Pressure

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Abstract

There is little information about right-to-left shunting with normal pulmonary artery pressure, which is produced as a consequence of right ventricle systolic dysfunction. We presented a case of unexpected transesophageal echocardiography findings of right-to-left shunting through an atrial septal defect despite normal pulmonary artery pressure, related to right ventricle systolic dysfunction in a 27-year-old young woman. Transesophageal echocardiography imaging revealed right ventricular systolic dysfunction and normal pulmonary artery pressure.

Keywords

Atrial Septal Defect, Right to Left Shunt, Pulmonary Hypertension, Transesophageal Echocardiography, Right Ventricular Dysfunction

Introduction

The occurrence of right-to-left (R-L) shunting in patients with atrial septal defect (ASD) is uncommon and is usually an ominous sign, heralding irreversible pulmonary hypertension (PH) [1]. There is little information about similar shunts in patients without PH, and the majority of these patients have an anatomic anomaly that favors this type of shunt. However, we present a patient with an unusual R-L shunt owing to right ventricular dysfunction in the absence of PH, without an anatomic anomaly. Written consent was obtained from the patient's relatives for the publication of this case report.

Case Presentation

A 27-year-old woman presented to our hospital with a complaint of mild chest discomfort without dyspnea during exercise. Physical examination revealed a systolic murmur over the second left intercostal space. There were no other abnormalities on cardiovascular or respiratory examination. A transthoracic echocardiogram (TTE) was performed, revealing multiple dropouts in the atrial septum, a suspicious low-speed shunt, and enlargement of the right atrium (RA) and right ventricle (RV) (54mm and 33mm, respectively). Additionally, mild tricuspid regurgitation and normal left ventricular (LV) systolic function were observed (ejection fraction was 69%). The electrocardiogram showed a normal sinus rhythm of 64 beats per minute with an incomplete left bundle branch block and left axis deviation. Her hemoglobin

level was 18.5 g/dl. Preoperative pulmonary angiography showed normal pulmonary artery pressure and anatomy. She was scheduled for atrial septal defect (ASD) repair surgery.

In the operating room, the patient was found to be hypoxemic, with a pulse oximetry reading of 84% on room air, which increased to 92–95% with 100% oxygen through a nonrebreather mask. Arterial blood gas analyses were performed (with air: pH 7.389, $PaCO_2$ 33.3 mm Hg, PaO_2 53.3 mm Hg vs with 100% O_2: pH 7.42, $PaCO_2$ 28.4 mm Hg, PaO_2 70.2 mm Hg). The central venous pressure reading was 17 mm Hg (current fluid intake is 300 ml).

After uneventful induction, we examined the heart using an adult Transesophageal echocardiography (TEE) probe to confirm the defect by an experienced and skillful sonographer. In the mid-esophageal four-chamber view, TEE revealed an unexpected right-to-left (R-L) shunt flow at the level of the atria, which was visualized by color-flow Doppler (**Fig-1A**). This finding can explain the preoperative hypoxemia. Then, we measured the peak tricuspid regurgitant velocity, which was 1.24m/s, and the pressure gradient was 23 mm Hg (**Fig-1B**). This suggests that the R-L shunt may not be secondary to pulmonary artery hypertension.

There were no echocardiographic signs of evident valve structural abnormalities or any abnormal venous connections. Nevertheless, the surgeon decided to repair the ASD. Surgical exploration confirmed the presence of ASD and the absence of anatomical distortion, myxomas, or pericardial adhesions. The surgery lasted approximately three hours, and the whole procedure was smooth and uneventful. Post-CPB TEE imaging revealed no evidence of residual shunt. Postsurgical arterial blood gas showed the following values: pH 7.33, pCO_2 36.6 mm Hg, pO_2 189.5 mm Hg, and oxygen saturation 99%.

Postoperative TTE showed no residual shunting, while LV systolic function was preserved (ejection fraction 47%), and RV systolic function was significantly lower. Her postoperative recovery was uneventful, and she was discharged in good condition. At the six-month follow-up, the patient's TTE showed normal left ventricular systolic function (ejection fraction 60%) and a mild tricuspid regurgitation, with no apparent discomfort.

Discussion

ASD with a right-to-left (R-L) shunt without Eisenmenger physiology is rarely reported and may be underestimated due to the difficulty in its diagnosis [2]. Common reasons for right-to-left shunting with normal pulmonary pressure in ASD include inter-atrial pressure gradient and heart anatomical distortion. The former can be observed as a result of early atrial systole or during early ventricular systole, right atrial myxomas [3], adult respiratory distress syndrome [4], obstructive sleep apnea [5], lung resection [6], and

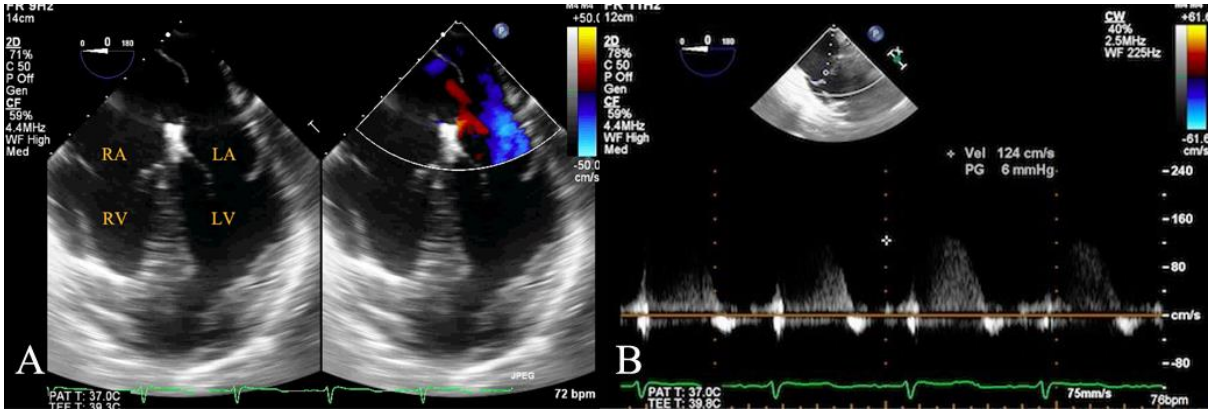

Fig-1:

***A**: The midesophageal four-chamber view seen here permits imaging of the RA, LA, RV, LV, and Color Doppler echocardiogram demonstrating R-L shunt flow at the level of the atria. Notably, the flow occurs primarily during systole as shown on the corresponding electrocardiogram tracing.*

***B**: Continuous wave Doppler recording of the peak tricuspid regurgitation velocity.*

mechanical ventilation, particularly with increased pulmonary end-expiratory pressure. This pressure gradient can be exacerbated by changes in posture, inspiration, and the Valsalva maneuver [7]. The latter can be seen as a result of abnormal venous connections [1,8], Ebstein's malformation [9], pulmonary stenosis, loculated pericardial effusion [10], kyphoscoliosis, or dilated aortic root [11,12], and so on.

In our patient, we encountered no anatomical distortion, no myxomas, no pericardial effusion, no adult respiratory distress syndrome, no obstructive sleep apnea, and no history of lung resection, among other factors. However, the preoperative TTE showed enlargement of the right atrium (RA) and right ventricle (RV). The preoperative central venous pressure was 17 mm Hg, and elevated central venous pressure is indicative of right ventricular dysfunction (RVD) and/or fluid retention [13]. Additionally, we calculated the RV fractional area change (FAC), and the systolic myocardial velocity of excursion (S') was 27% and 9 cm/s, respectively [14]. An s' velocity <10 cm/s is considered RV systolic dysfunction; Fractional area change <35% indicates RV systolic dysfunction (**Fig-2A** and **Fig-2D**).

On the basis of these findings, we can conclude that the R-L shunt in the absence of PH is secondary to RV systolic dysfunction. Unfortunately, for financial reasons, cardiac MRI [15], which is considered the noninvasive gold standard for assessing cardiac function, has not been undergone.

TTE is an essential non-invasive diagnostic tool for CHD, but recent advances in TEE technology have led to its increasing importance in the care of both children and adults with CHD. This case specifically highlights that even though TTE is excellent at detecting CHD, scrutiny during the pre-CPB examination by TEE is necessary to confirm the preoperative TTE findings and also identify possible additional pathologic conditions [16]. Additionally, TEE has been shown to provide additional diagnostic information over TTE for specific structural cardiac anomalies and, in the perioperative setting, offers the opportunity for confirmation of preoperative diagnoses and influence the surgical plan if new or different pathology is identified [17,18].

Several points from this case deserve special emphasis. First, the occurrence of R-L shunting at the atrial level does not necessarily indicate severe pulmonary hypertension. Second, the occurrence of right heart enlargement is not always accompanied by left-to-right shunting in ASD. Third, R-L shunt may manifest in patients with ASD despite normal PA pressures, with potentially treatable conditions.

Conflict of Interest

The author has read and approved the final version of the manuscript. The author has no conflicts of interest to declare.

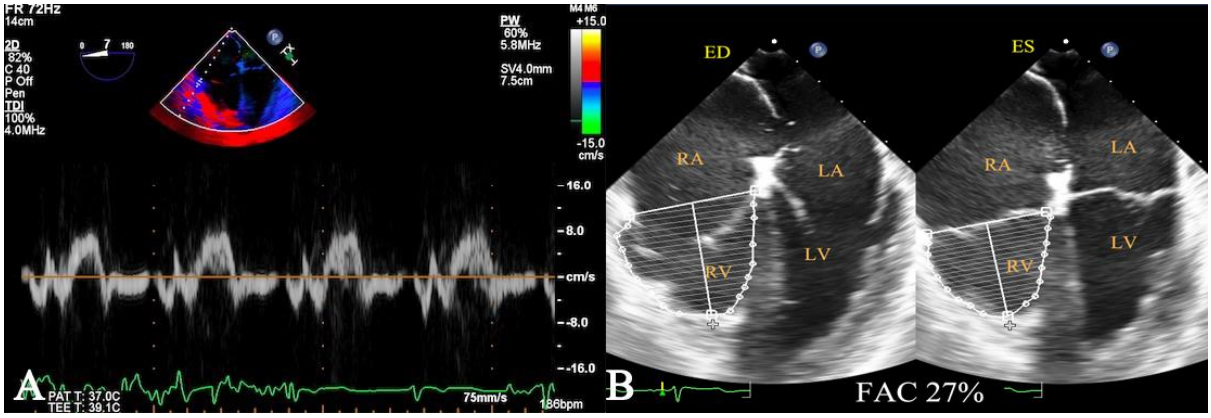

Fig-2:

***A**: Pulsed wave tissue Doppler Imaging from the apical 4 chamber view sampling from the tricuspid valve annulus.*

***B**: Right ventricular fractional area change (FAC). Percentage FAC = 100 x end-diastolic area (Area ED) –end-systolic area (Area ES)/end-diastolic area, FAC—here, from 14 cm^2 to 10.2 cm^2 = 27%.*

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