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Diversion of the inferior vena cava following repair of atrial septal defect causing hypoxemia

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Abstract

Atrial septal defects (ASDs) are a common congenital abnormality, and operative repair is a routine, safe procedure. Diversion of the inferior vena cava (IVC) into the left atrium is an unusual complication following ASD closure. We report a case that illustrates the problem created by this right-to-left shunt. A middle-aged woman underwent ASD repair. She developed hypoxemia postoperatively. A transthoracic echocardiogram confirmed a right-to-left shunt, found only with agitated saline injected into the femoral vein, not into the basilic vein. Surgical reexploration revealed a residual ASD diverting IVC flow into the left atrium, which was repaired with a pericardial patch. Echocardiography with agitated saline injected from the femoral vein is an easy method to diagnose this uncommon complication.

Atrial septal defects (ASDs) are the third most common congenital cardiac malformations.¹ Repair of these defects is a fairly safe and routine procedure. Inadvertent diversion of the inferior vena caval flow into the left atrium (LA), although unusual, remains a cause of morbidity following repair of ASD. This leads to dyspnea and hypoxemia, which may present immediately postoperatively or several years later. We present a patient in whom hypoxemia and respiratory distress occurred immediately after surgery. Transesophageal echocardiogram (TEE) and transthoracic echocardiogram (TTE) contrast studies were able to show the mechanism of this defect.

Case Report

A 44-year-old white female presented to her primary care physician for evaluation of hypertension. On physical exam, she was found to have a 2/6 systolic ejection murmur with a fixed split S2. Chest X-ray was done, revealing cardiomegaly with clear lung fields. An echocardiogram revealed a secundum-type ASD. Cardiac catheterization was subsequently performed, which showed a significant ASD. The shunt fraction ($Q_p:Q_s$) was calculated to be 2.27:1; right ventricular pressure was mildly elevated at 41/14 mmHg.

The patient underwent elective ASD repair. The right atrium (RA), right ventricle, and pulmonary artery were moderately dilated. A secundum ASD approximately 1.0×1.5 cm in diameter was identified, lying low in the atrium, near the orifice of the IVC. The defect was closed in two layers with a running 4-0-prolene suture tied at either end. Noncontrasted TEE was performed by the anesthesiologist; no residual defect was demonstrated.

Postoperatively, while in the open-heart recovery room, she developed respiratory distress and hypoxemia. An ABG on room air revealed a PaO_2 of 46 and O_2 saturation 87%. On 100% FIO_2 , her PaO_2 was 61 and O_2 saturation was 93%. Chest X-ray showed no evidence of infiltrate or fluid overload.

A pulmonary arteriogram was performed and was within normal limits. An echocardiogram with agitated saline injected into the basilic vein revealed no shunt (Fig. 1). A TEE revealed a residual ASD inferiorly. The interatrial septum was distorted, causing it to override the IVC. Color flow Doppler demonstrated flow from the IVC to both the RA and LA (Fig. 2). Bulging of the septum primum was seen, caused by flow from the IVC into the LA. A TTE was performed, with

contrast injected into the left femoral vein (Fig. 3). Immediate appearance of contrast in the LA was seen, confirming a right-to-left shunt.

The patient returned to the operating room. The initial repair had left a small residual ASD at the entrance of the IVC into the RA. The defect was at the inferior angle, near the IVC perfusion cannula, which may have blocked the view of the residual ASD. An atrial septal aneurysm was also detected. The interatrial septum was overriding the IVC, which caused blood flow to be directed into the LA. The previous repair was taken down. The residual septum primum that constituted an atrial septal aneurysm was resected, and a pericardial patch was sutured over the defect. The patient did well postoperatively.

Discussion

In a recently reported surgical series of ASD repair, there were no operative deaths. The common complications were postoperative atrial arrhythmias, SA, and AV blocks necessitating pacemaker implantation, mediastinal bleeding, and TIAs or strokes.²

Inadvertent diversion of the inferior vena caval flow into the LA, although unusual, remains a cause of morbidity following repair of ASD. Reported cases have presented primarily with cyanosis and hypoxia, which can occur immediately postoperatively or months to years after operation. Exertional desaturation has also been reported,³ as has orthodeoxia.⁴ Reported factors associated with this complication include a large secundum defect or sinus venosus defect, and anomalous pulmonary return into the RA.⁵ This complication was more frequent prior to the use of cardiopulmonary bypass, due to time limitations imposed with only hypothermia and inflow occlusion.⁵ Previous case reports involve repair of a low-lying ASD proximal to the inferior vena cava (IVC). In most of the cases, a prominent Eustachian valve was included in the repair of the ASD or the inferior rim of the defect was not included in the closure stitch leading to incomplete closure of the defect.⁴ Transcatheter repair is becoming a more widely used and successful technique, which may avoid this complication.

In our patient, hypoxemia and respiratory distress occurred immediately after surgery. Other causes of postoperative hypoxemia, including heart failure, pulmonary infections, and pulmonary embolism, were excluded. Intraoperative TEE was performed without contrast. Postoperative TTE and TEE identified the anatomical defect created by the repair. Color flow Doppler demonstrated flow from the IVC to both RA and LA. Bulging of the septum primum was also demonstrated, which is caused by the flow from the IVC into the LA. A contrast study using agitated saline solution confirmed the shunting of inferior vena caval flow into the LA, but not the superior vena caval flow. The turbulent flow from the IVC into the RA prevented flow from the superior vena cava from crossing the defect, explaining the absence of contrast crossing into the LA with arm injection of the contrast solution.

Several methods have been employed for the detection of this complication. In previously reported cases, cardiac catheterization has been used, as has intraoperative dye dilution curves. Intraoperative TEE is generally performed after weaning from bypass, but before the removal of perfusion cannulas. Often, these are done with color flow Doppler only. In our case, agitated saline injected from below was necessary to diagnose this rare complication. With widespread

use of intraoperative TEE, this technique would not add significant cost. Therefore, agitated saline injected intravenously from below is a simple and inexpensive procedure and can potentially prevent reoperation. We propose this simple method for the diagnosis of this potential complication prior to closing the chest.

Conclusion

ASDs are a common congenital abnormality. Operative repair is a fairly routine procedure with relatively low morbidity and mortality. We present a case of secundum ASD, operatively repaired, which was complicated by diversion of IVC flow into the LA. The diagnosis was made by echocardiogram with agitated saline injected from the femoral vein. This is a simple and cost-effective method for early detection of this complication.

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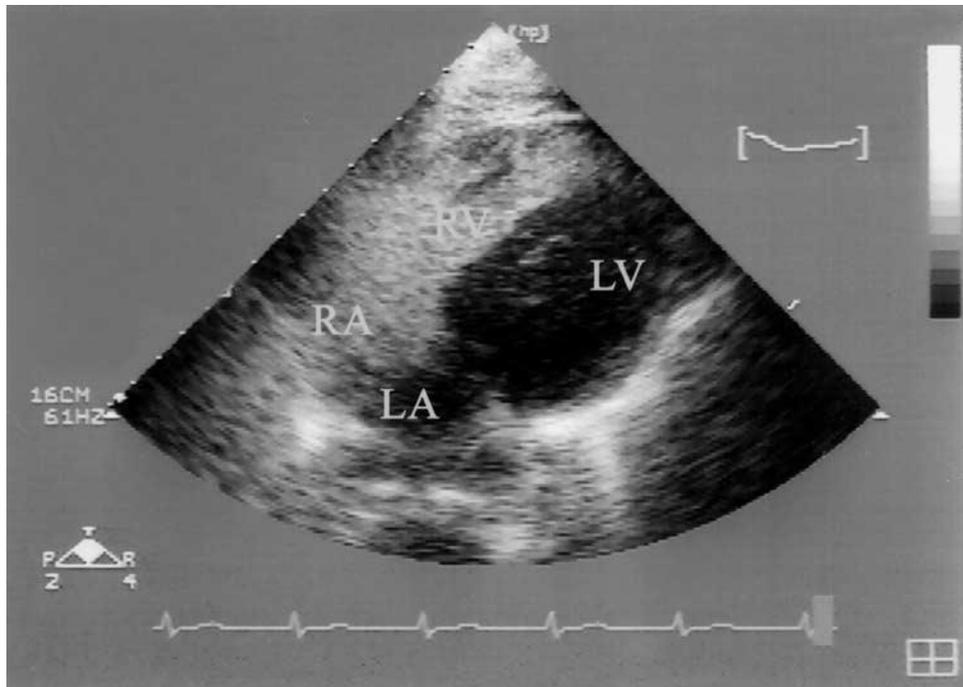


Figure 1. TTE with contrast injected into the basilic vein. There was no contrast observed in the left-sided chambers of the heart.

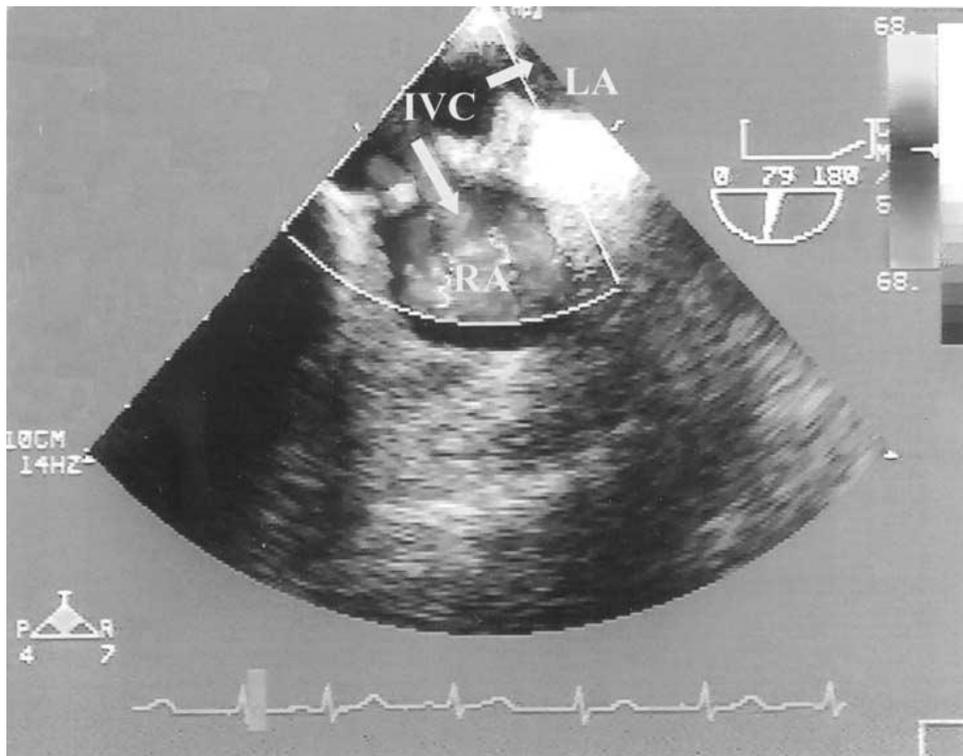


Figure 2. TEE of the interatrial septum with color flow Doppler. Flow from the IVC to both the RA and LA is observed.

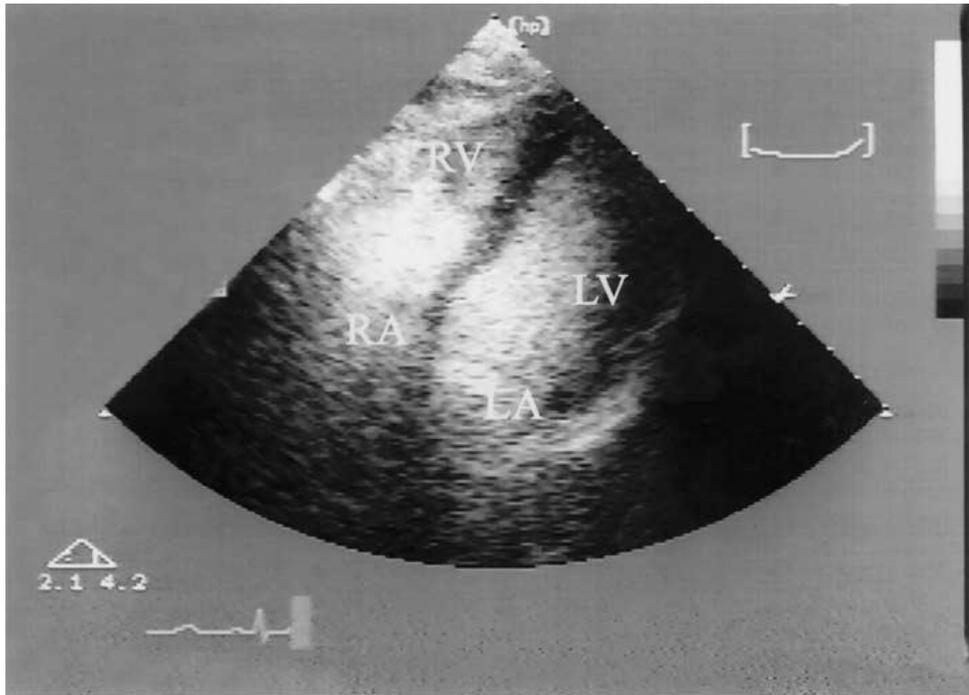


Figure 3. TTE with contrast injected into femoral vein. There was immediate appearance of the contrast in the LA, suggesting a right-to-left shunt.