

Rapid Left Ventricular Recovery After Correction of a Secundum Atrial Septal Defect: Understanding the Hemodynamics

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ABSTRACT: Closure of an atrial septal defect (ASD) may lead to a change in the function of both ventricles. Although right ventricular function typically improves, the left ventricle (LV) may behave in different ways. This has been a matter of much debate, with some authors reporting a decline in LV function after ASD closure and others reporting delayed improvement or no identifiable change. We report the case of a 41-year-old female with a large left-to-right ASD shunt (Qp:Qs 2.3:1 and shunt volume 3.6 L/min) who presented with biventricular systolic dysfunction that improved within 24 hours of ASD closure. We also attempt to explain the underlying hemodynamics responsible for LV failure and recovery in this patient.

BACKGROUND

Atrial septal defects (ASDs) account for 13% of congenital heart diseases worldwide.¹ The basic hemodynamic disturbance in patients with ASDs is a left-to-right shunt that leads to an imbalance in blood flow to the pulmonary and systemic circuits. This causes right ventricular (RV) overload and subsequent RV failure. However, there are multiple reports of patients with ASDs who exhibit concomitant left ventricular (LV) dysfunction or failure as well.²⁻⁴ These patients may have normal LV end-diastolic pressures relative to decreased end-diastolic volumes, which indicates LV dysfunction.⁴

The hemodynamic abnormalities underlying ASD-associated LV failure are poorly understood. Proposed hypotheses implicate either intrinsic problems with the LV or problems related to the RV. Intrinsic LV problems include decreased distensibility, underfilling of the LV, or the presence of coexisting conditions unrelated to the ASD that cause LV dysfunction. Left ventricular dysfunction related to the RV could occur due to ventricular interdependence.³⁻⁵ After ASD closure, some patients may develop worsening LV function,⁶ whereas other patients may show improvement in LV volumes, delayed improvement, or no significant improvement of LV ejection fraction (LVEF).⁷⁻¹¹

We describe the case of a patient with biventricular heart failure and a concomitant large ASD who showed rapid improvement of her LVEF within 24 hours after ASD closure. To the best of our knowledge, a similar case has not been previously reported.

CASE DESCRIPTION

A 41-year-old African-American female without significant past medical history presented to us with new-onset dyspnea on

exertion and bilateral pedal edema that started 2 days prior to admission and was worsening. She had never experienced similar symptoms prior to this. At baseline, she was able to ambulate comfortably. However, during the week prior to admission, her functional status had gradually declined to NYHA class III.

She was afebrile at admission with a blood pressure of 177/117 mm Hg, pulse of 89 per minute, respiratory rate of 18 breaths per minute, and oxygen saturation of 93% on room air. Her weight at admission was 148.9 pounds, which was approximately 10 pounds over her dry weight. Her physical examination was suggestive of right-sided congestive heart failure with jugular venous distension and bilateral pitting pedal edema. On auscultation, a soft 2/6 systolic murmur was heard, most prominently over the left second intercostal space.

An initial echocardiogram showed an LVEF of 30% to 34%, severely depressed RV function, normal atrial septum, and an estimated pulmonary artery systolic pressure of 65 to 70 mm Hg. Due to her depressed LVEF, cardiac magnetic resonance imaging (CMR) was performed to help determine a cause of her cardiomyopathy. This revealed a large secundum ASD measuring 4 cm x 2.4 cm (Figure 1). Due to the large size of the ASD, there was no color Doppler flow seen across the ASD on the initial echocardiogram; the defect seen on the subcostal view was therefore considered to be a dropout, and the interatrial septum was at first considered normal. CMR revealed a predominantly left-to-right shunt with a small bidirectional component. The net shunt volume was 3.6 L/min and the Qp:Qs ratio was 2.3:1. The patient also had global hypokinesia with an RVEF of 28% and LVEF of 37%. The Qp:Qs ratio calculated from right heart catheterization was 3.2:1. Baseline pulmonary vascular resistance (PVR) was approximately 1.9

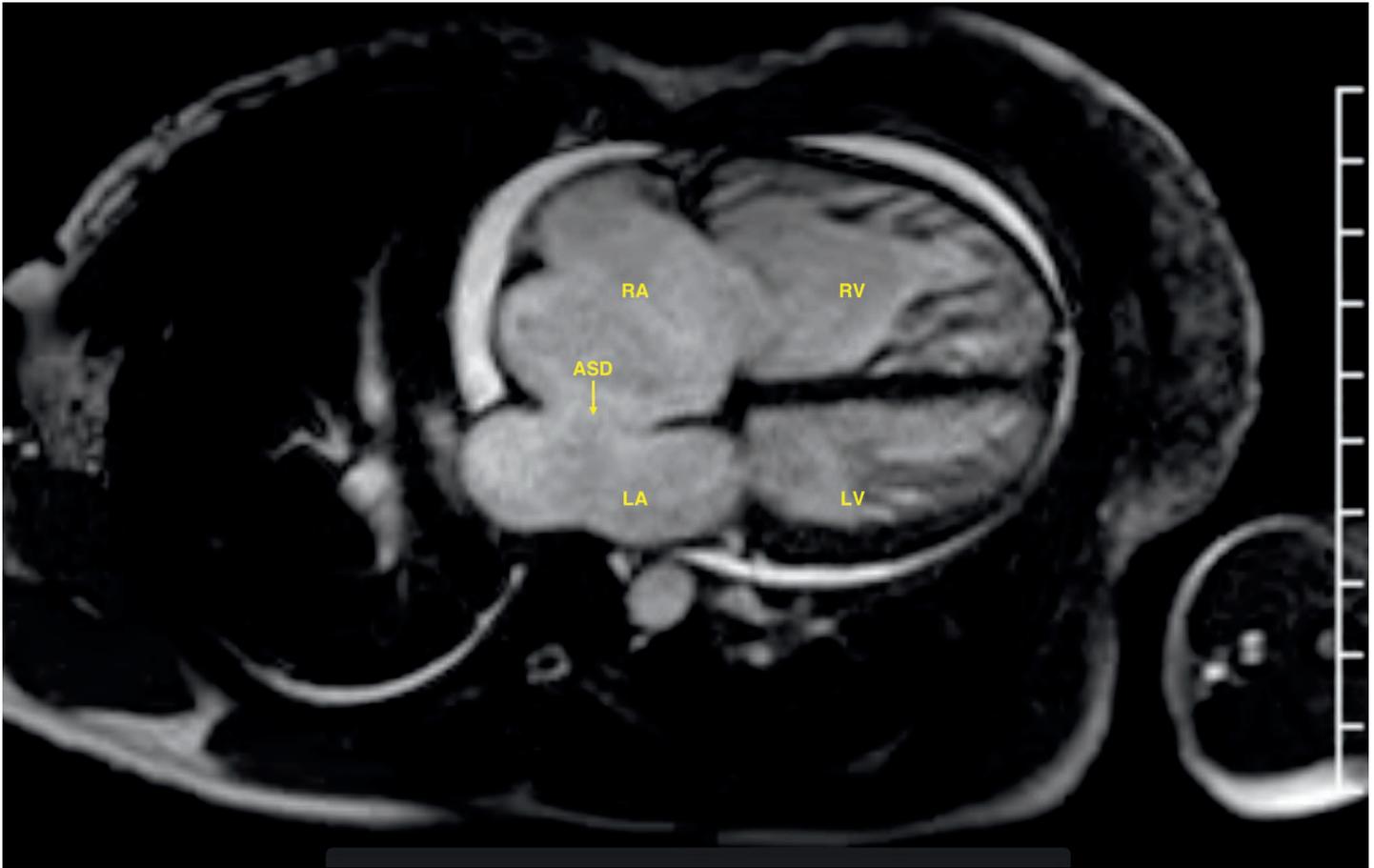


Figure 1. Four-chamber view on cardiac magnetic resonance imaging showed a large 4-cm x 2.4-cm secundum atrial septal defect (ASD) with resultant severe right ventricular enlargement. RA: right atrium; LA: left atrium; RV: right ventricle; LV: left ventricle

	PRECLOSURE			POSTCLOSURE		
	SYSTOLIC (MM HG)	DIASTOLIC (MM HG)	MEAN (MM HG)	SYSTOLIC (MM HG)	DIASTOLIC (MM HG)	MEAN (MM HG)
Right atrium	17	16	14	14	12	10
Right pulmonary artery	40	20	27	50	20	30
Right pulmonary wedge pressure	12			12		
Left ventricle	70	12	-	111	12	-
Aortic	76	54	61	116	57	78

Table 1. Intracardiac pressures measured on right heart catheterization.

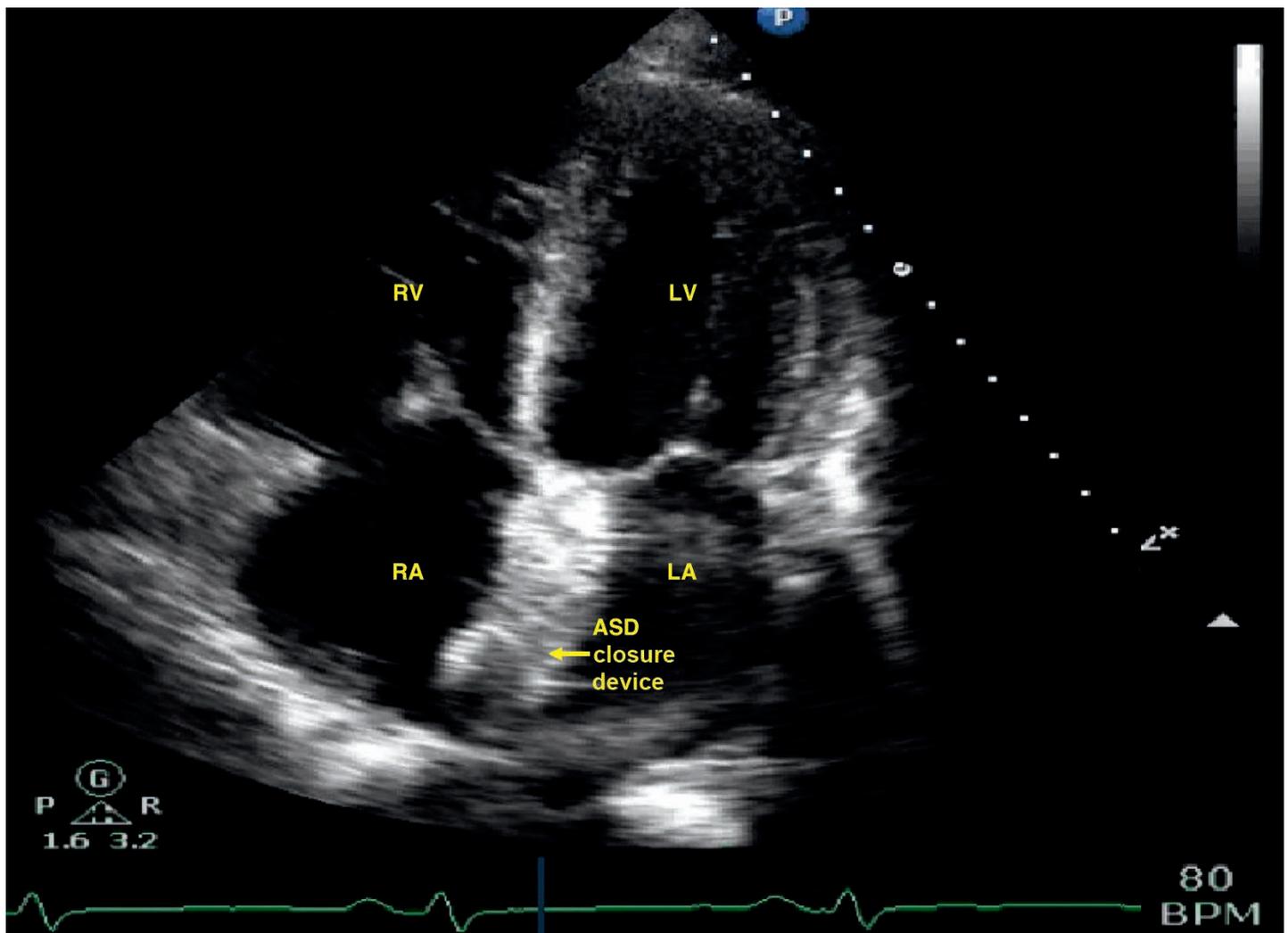


Figure 2.

Four-chamber view on a postprocedure transthoracic echocardiogram showed the atrial septal defect (ASD) closure device in the appropriate position. RV: right ventricle; RA: right atrium; LV: left ventricle; LA: left atrium

Wood units, and systemic vascular resistance was 24 Wood units. The PVR was considered satisfactory for ASD closure. Intracardiac pressure readings before and after ASD closure are presented in Table 1.

ASD measurement using a 34-mm sizing balloon revealed it to be between 34 and 36 mm in size. A small fenestration on the aortic side of the septum was also noted. A 38-mm AMPLATZER™ Septal Occluder (St. Jude Medical/Abbott) was deployed. Given the poor LV function, there was a concern that device closure would lead to acute left-sided overload and a subsequent increase in LV filling pressures with a worsening of LV function. Therefore, we measured right- and left-sided

pressures after device deployment. Since her LV pressures did not increase significantly, the device was released. Some residual shunt persisted through the fenestration on the aortic side of the atrial septum.

A postprocedure echocardiogram (Figure 2, Video 1) showed a significant improvement in systolic function, with an estimated LVEF of 50% to 54% and the RVEF returning to normal. Her LV cardiac output increased from 1.9 L/min to 4.7 L/min. There was also a significant change in the pulmonary artery systolic pressure from 70 mm Hg before the procedure to 39 mm Hg after ASD closure. There was no significant change in the LV end-diastolic diameter (preclosure 4.2 cm; postclosure 4.3 cm).

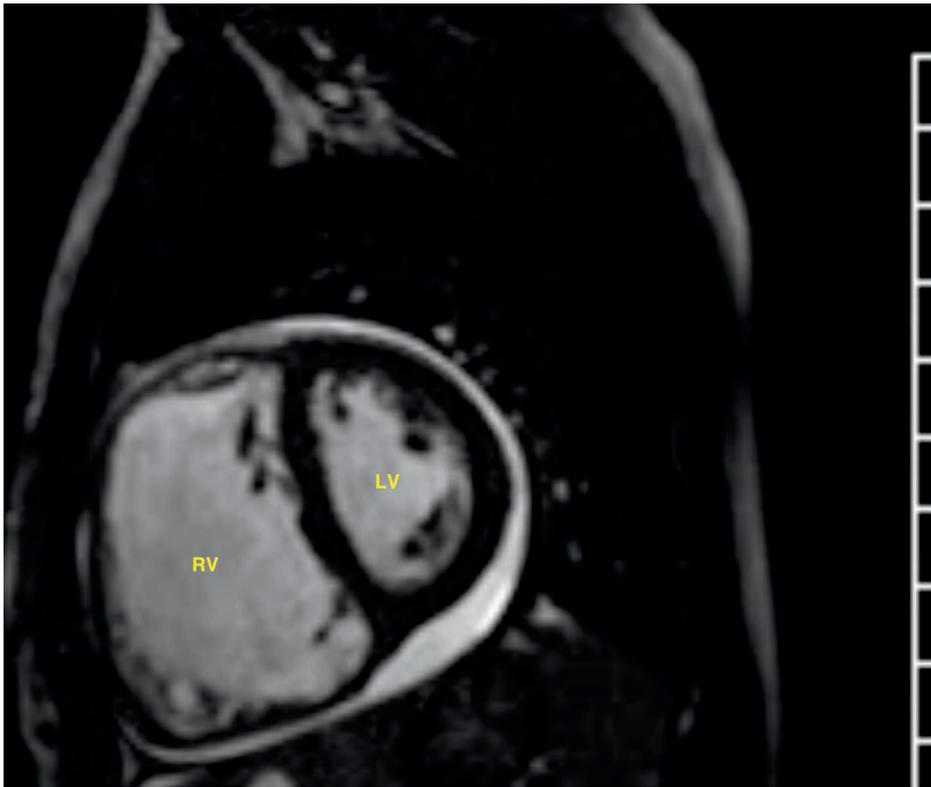


Figure 3.

D-shaped left ventricle (LV) and flattening of the interventricular septum on cardiac magnetic resonance imaging was consistent with right ventricular (RV) overload; significant RV overload likely led to LV failure due to ventricular interdependence.

Clinically, she rapidly became asymptomatic by the time of her discharge with improvement of her dyspnea.

DISCUSSION

Left ventricular performance before and after ASD repair is a matter of much debate. Our patient presented with significant biventricular failure that rapidly recovered in an atypical fashion within 24 hours of ASD closure. This was an unusual finding that raised two interesting questions: (1) What was the etiology underlying the depressed LVEF? (2) Why did the LVEF improve shortly after ASD closure?

Several authors have studied the LV in the setting of ASDs. Some have reported that patients with ASDs have normal EFs or may have subnormal LVEF responses to exercise, indicating an underlying LV dysfunction.^{3,4,12} Booth et al. postulated that patients with ASDs had stiffer LVs and a consequent predilection for heart failure.⁵

In 1982, Carabello et al. published data on hemodynamic parameters of patients with ASDs and signs of heart failure, and they compared this with data obtained from patients with ASDs but no heart failure as well as a group of normal subjects. They noticed that patients with signs of heart failure

tended to have higher LV end-diastolic pressures, but their LV cardiac indices and EFs still remained comparable to normal subjects.¹³ Our patient contradicted this finding given that her LVEF was 37% at presentation—a value markedly below normal range.

What may have caused this marked LV systolic impairment in our patient? Tikoff et al. postulated that in the natural history of an untreated ASD, LV failure constitutes the final stage (stage V).² They believed that the distensibility of the LV decreases with time, and as the shunt becomes bidirectional, the LV starts receiving a higher volume than it can accommodate, resulting in LV failure. However, if this were the case in our patient, then correcting the shunt and increasing preload to the LV should have caused a worsening of her LV function, not an improvement. We believe that our patient still had adequate LV reserve prior to ASD correction and had not yet reached stage V. Cardiac MR was not suggestive of other etiologies for her cardiomyopathy. Therefore, we propose the following explanation for LV failure in our patient: The large size of her left-to-right atrial shunt led to RV overload beyond its compensatory capacity. This was supported by findings on CMR that showed a flattened interventricular septum and a D-shaped LV (Figure 3, Video 2). This likely led to LV failure due to ventricular interdependence.

The rapid improvement of her LVEF subsequent to closure is also interesting. Studies in the past have shown that LVEF does improve after ASD closure; however, these patients initially had normal or near-normal LVEFs.^{11,14} On the other hand, there have also been reports about worsening LV function after ASD repair due to acute postclosure volume loading.^{6,15} For this reason, and the fact that our patient already had preexisting moderate-to-severe LV dysfunction, we were concerned that her LV systolic function would worsen after ASD occlusion. Contrary to our expectations, her LVEF improved. We hypothesize two reasons why this could have occurred. First, the patient received diuretics for several days prior to closure, and the improved volume status caused a lower preload that could be pumped more efficiently by the failing LV. Second, the patient had fenestrations in the anterior part of her atrial septum that we were unable to close, and they caused a persistent mild left-to-right shunt. It is possible that this mild shunt allowed for decompression of the left side, thus preventing the LV from being loaded beyond its filling capacity.

CONCLUSION

Left and right heart interaction is an important consideration in the closure of a large ASD. The mechanism of LV failure and subsequent recovery in this patient is still unclear. However, LV dysfunction at presentation did not preclude safe ASD closure.

Conflict of Interest Disclosure

The authors have completed and submitted the *Methodist DeBakey Cardiovascular Journal* Conflict of Interest Statement and none were reported.

Keywords

atrial septal defect, left ventricular dysfunction

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