Cardiology in the Young

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Transient biventricular collapse after atrial septal defect closure in an adolescent: a diastolic dysfunction-driven paradox

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Case Report

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Abstract

While transcatheter atrial septal defect closure is routinely performed, acute biventricular failure is an extraordinary complication in adolescents, with only anecdotal reports in the literature. We present a 16-year-old male with borderline left ventricular systolic function and biventricular diastolic dysfunction who developed transient severe biventricular failure immediately following Amplatzer Septal Occluder (Abbott, Plymouth, MN, USA) deployment. Preprocedural echocardiography revealed right ventricular dilation, a D-shaped septum in diastole. Intraprocedural haemodynamic assessment demonstrated elevated right ventricular diastolic pressures (right ventricular minimal diastolic pressure: 11 mmHg; end-diastolic pressure: 17 mmHg). Haemodynamic collapse occurred within minutes of device release, necessitating emergent dopamine infusion and urgent coronary angiography to rule out device-related complications. Remarkably, ventricular function normalised within 2 hours, enabling extubation the same day. Reports of transient biventricular failure following atrial septal defect closure in adolescents without comorbidities are exceedingly rare, underscoring the critical role of preexisting diastolic dysfunction in precipitating acute decompensation. This case advocates for preprocedural balloon occlusion testing and vigilant haemodynamic monitoring in adolescents with impaired ventricular compliance to mitigate catastrophic outcomes.

Introduction

Atrial septal defect, a common congenital cardiac malformation characterised by abnormal communication between the systemic and pulmonary circulations, results in equilibrated atrial pressures in large defects, with shunt magnitude determined by ventricular compliance and defect size.[1,2] Chronic volume overload in large atrial septal defects leads to right-sided cardiac chamber dilation, causing geometric distortion of the left ventricle and subsequent diastolic dysfunction—a phenomenon termed reverse Bernheim effect.[3] Studies have demonstrated that left ventricular end-diastolic pressure is elevated in atrial septal defect patients compared to healthy controls.[4,5] In adults, left ventricle diastolic dysfunction—manifested as increased stiffness or impaired relaxation—is a well-documented complication following atrial septal defect closure. This is attributed to abrupt haemodynamic changes, including a sudden rise in left ventricle diastolic pressure post-closure, which may precipitate heart failure.[6] In contrast, paediatric populations exhibit preserved left ventricular passive stiffness, though subtle relaxation abnormalities have been reported; to date, heart failure following atrial septal defect closure remains exceedingly rare in children, with only isolated cases documented in the setting of comorbidities.[7,8]

Also, prolonged left-to-right shunting in atrial septal defects induces chronic right ventricular volume overload, which over time leads to myocardial injury from increased oxygen demand in the dilated right ventricle, thereby exacerbating diastolic dysfunction.[9]

In this study, we present the case of an adolescent with borderline left ventricular systolic function and biventricular diastolic dysfunction who developed transient biventricular failure following transcatheter atrial septal defect closure. To our knowledge, this represents one of the very few reported paediatric cases of transient heart failure secondary to atrial septal defect closure, highlighting the critical interplay between ventricular compliance and post-procedural haemodynamic adaptation.

Case report

A 16-year-old male patient was admitted to the catheterisation laboratory for transcatheter atrial septal defect closure. Preprocedural evaluation revealed severe right ventricular dilation and mild impairment of biventricular diastolic function, demonstrated by mitral and tricuspid

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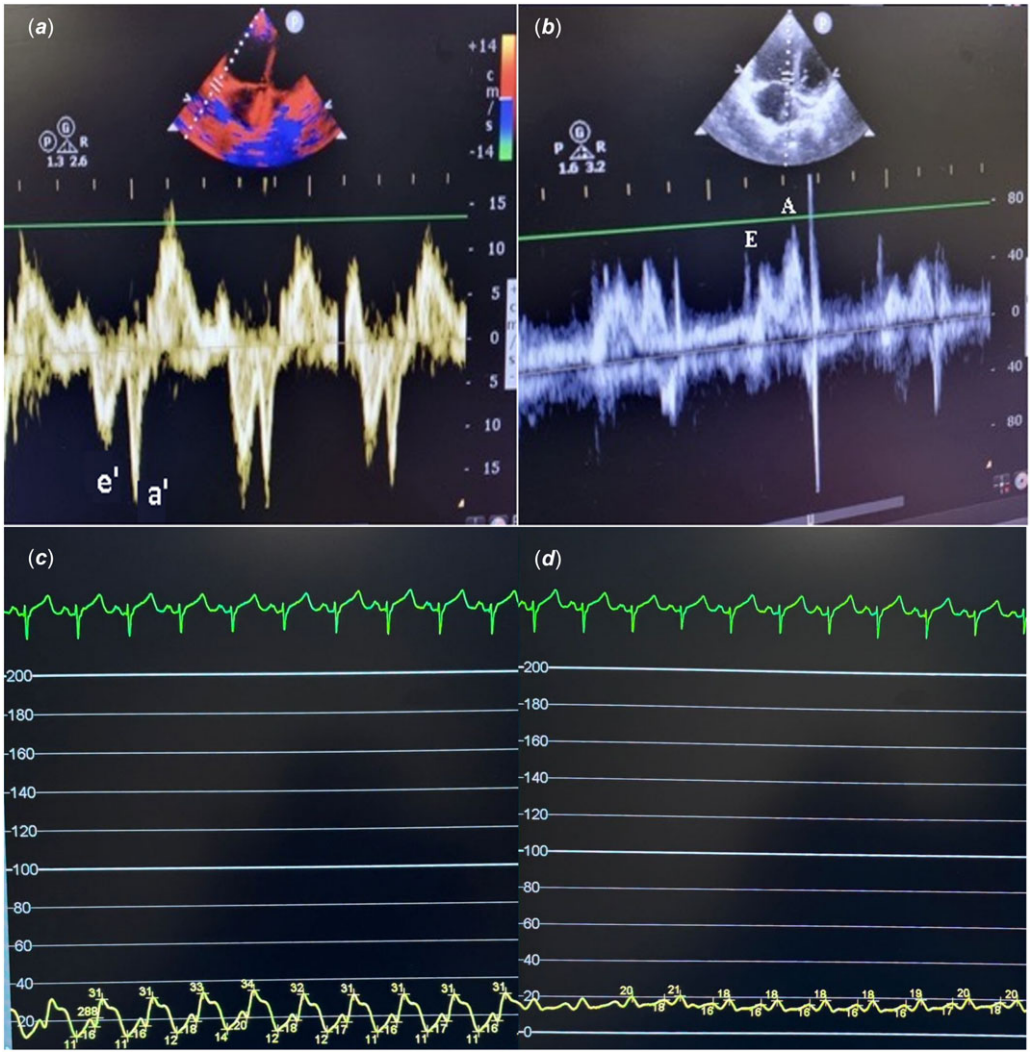


Figure 1. Imaging and haemodynamic parameters prior to atrial septal defect closure, demonstrating biventricular diastolic dysfunction. (***a***): Conventional Doppler interrogation of the tricuspid inflow, showing diastolic flow velocities with E-wave velocity lower than A-wave velocity (early-to-late diastolic velocity ratio <1), indicative of impaired right ventricular relaxation. (***b***): Tissue Doppler imaging of the tricuspid valve, demonstrating lower early diastolic tricuspid annular velocity (e′) than late diastolic tricuspid annular velocity (a′) consistent with impaired right ventricular relaxation. (***c***): Right ventricular pressure tracing during angiography, revealing elevated minimal diastolic (11 mmHg) and end-diastolic (17 mmHg) pressures. (***d***): Left atrial pressure tracing, demonstrating increased mean left atrial pressure (16 mmHg).

inflow early-to-late diastolic velocity ratios <1 on pulsed Doppler and by early-to-late diastolic myocardial velocity ratios <1 on tissue Doppler imaging (consistent with impaired relaxation), and a D-shaped interventricular septum during diastole, and borderline left ventricular systolic function (ejection fraction: 57%) (Figure 1a and b). Transthoracic apical four-chamber view demonstrated an atrial septal defect measuring 21 mm at its widest diameter (Supplementary Video 1). A 24-hour Holter monitor prior to the procedure demonstrated rare ventricular extrasystoles, as it was performed because a single ventricular extrasystole had been noted on the baseline electrocardiogram. Due to borderline left ventricular systolic function, treatment with enalapril maleate 5 mg daily was initiated before the procedure.

Intraprocedural haemodynamic assessment showed elevated right ventricular pressures (minimal diastolic pressure: 11 mmHg; end-diastolic pressure: 17 mmHg) (Figure 1c). Left atrial pressure was also mildly elevated, with a mean of 16 mmHg. Under transesophageal echocardiography guidance, balloon sizing was performed with a 34 mm Amplatzer sizing balloon, and the large atrial septal defect (24 mm) was successfully closed using a 24 mm Amplatzer Septal Occluder. The device was released after appropriate positioning was confirmed by transesophageal echocardiography.

Within minutes of device release, follow-up transthoracic echocardiography revealed severe biventricular systolic dysfunction (Supplementary Video 2). Immediate intervention with dopamine infusion (10 mcg/kg/min) was initiated. Given the unprecedented nature of this complication at our institution, selective coronary angiography was performed to exclude device-related coronary artery compression, which was ruled out (Figure 2). The surgical team was consulted for potential device retrieval or extracorporeal membrane oxygenation support.

Serial echocardiograms demonstrated gradual improvement in ventricular function. The patient was transferred to the ICU under inotropic support. Two hours post-procedure, biventricular function returned to preprocedural baseline (Supplementary Video 3), and the patient was extubated the same evening. Dopamine infusion was discontinued the following day, and the patient was transferred to the paediatric cardiology ward. Treatment with lisinopril dihydrate 2.5 mg once daily was initiated, and he was discharged 3 days later under this therapy.

At 3-month follow-up, echocardiography demonstrated an improvement in left ventricular systolic function (ejection fraction: 60%). Right heart chambers remained dilated, although smaller compared to pre-procedural measurements. Importantly, diastolic

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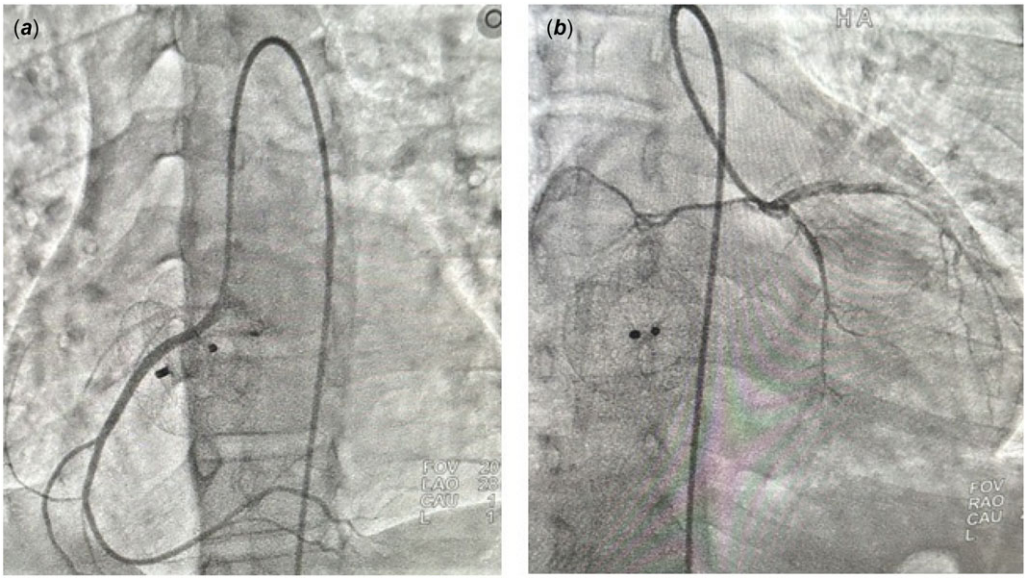


Figure 2. Selective coronary angiograms performed during acute haemodynamic collapse following Amplatzer occluder placement. (***a***): Right coronary artery angiogram: demonstrates patent right coronary artery with no evidence of stenosis or external compression from the atrial septal defect occluder. (***b***): Left coronary artery angiogram: shows normal left main, left anterior descending, and circumflex arteries, excluding device-related coronary compromise.

function had normalised, with mitral and tricuspid Doppler inflow early-to-late diastolic velocity ratios both above 1.

Discussion

As the current gold standard for atrial septal defect management, transcatheter closure correlates with reversal of preprocedural biventricular dysfunction in paediatric cohorts, marked by restored ventricular geometry and systolic/diastolic efficiency.[10,11] While abnormal ventricular relaxation may occur in paediatric patients, transient left ventricular dysfunction following atrial septal defect closure is rare in this population. In contrast, post-procedural left ventricular dysfunction secondary to impaired ventricular stiffness is a well-documented complication in adults—a phenomenon rarely observed in paediatric populations.[6] A notable exception is the case reported by Vehbi et al., in which an 8-year-old patient with thiamine-responsive megaloblastic anaemia developed transient heart failure after transcatheter atrial septal defect closure, resolving with thiamine and dopamine therapy.[8]

To our knowledge, this represents one of the very few reported cases of transient biventricular failure following transcatheter atrial septal defect closure in adolescents without underlying comorbidities. In adults, chronic right ventricular volume overload secondary to atrial septal defect can induce myocardial remodelling, leading to increased left ventricular diastolic stiffness and impaired relaxation. These alterations elevate left ventricular diastolic pressures, predisposing to transient ventricular dysfunction post-closure. In our patient, the haemodynamic profile was striking: the right ventricular minimal diastolic pressure (normally near 0 mmHg) was elevated to 11 mmHg, with elevated biventricular end-diastolic pressures.

This case underscores that catastrophic, albeit rare, biventricular failure may occur even in adolescents with preexisting diastolic dysfunction. We propose that balloon occlusion testing during atrial septal defect closure, coupled with close echocardiographic monitoring of ventricular pressures and function, should be considered in adolescents with borderline systolic function or diastolic impairment. Such measures may help identify high-risk patients and guide timely intervention to mitigate haemodynamic collapse.

Conclusion

This case highlights transient biventricular failure following atrial septal defect closure in an adolescent, likely triggered by preexisting diastolic dysfunction and elevated ventricular filling pressures. Rapid recovery with inotropic support underscores the reversibility of this rare complication. Preprocedural assessment of diastolic function and ventricular pressures, alongside intra-procedural balloon occlusion testing, may identify high-risk adolescents. Clinicians should remain vigilant for acute haemodynamic collapse, even in structurally uncomplicated atrial septal defect closures.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S1047951125110524

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