

Case Report

Late Surgical Explantation of an Amplatzer Duct Occluder-II (ADO II) in an Adolescent with Persistent Residual Perimembranous Ventricular Septal Defect and Tricuspid Septal Leaflet Adherence: A Case Report

Zakiur Rehman Ansari¹, Zainul Abedein Hamdulay², Azizullah Khan³, Sanjesh Jain⁴, Meher Hamdulay⁴

¹Department of Clinical Research in Cardiovascular Surgery and Critical Care Medicine, Masina Heart Institute & GHC Hospitals, Mumbai, Maharashtra, India

²Department of Cardiothoracic Surgery, Masina Heart Institute & GHC Hospitals, Mumbai, Maharashtra, India

³Department of Critical Care Medicine, Masina Heart Institute & GHC Hospitals, Mumbai, Maharashtra, India

⁴Department of Cardiac Anaesthesia, Masina Heart Institute & GHC Hospitals, Mumbai, Maharashtra, India

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Corresponding author's email:

dr.zaki9821@gmail.com

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ABSTRACT

Background: Transcatheter closure is an established treatment option for selected perimembranous ventricular septal defects (VSDs). Because of the close anatomical relationship of the perimembranous septum to the tricuspid valve, aortic valve, and conduction system, residual shunting and device–valve interactions remain important considerations during follow-up.

Case Presentation: A 14-year-old boy was referred in 2026 with progressive exertional dyspnea and occasional chest discomfort. Four years earlier, on February 5, 2022, he had undergone transcatheter closure of a perimembranous VSD at another institution using a 6/6-mm Amplatzer Duct Occluder II (ADO II). The available procedural record documented catheter-based VSD crossing, device deployment, and immediate transesophageal echocardiographic assessment before release, with satisfactory device position reported. The original catheterization laboratory fluoroscopic, angiographic, and echocardiographic images were unavailable for retrospective review. Follow-up echocardiography in 2023 and 2024 demonstrated a persistent residual VSD of approximately 5 mm without significant tricuspid regurgitation (TR) or pulmonary hypertension, and surgical closure had been advised. In 2025, the patient developed mild exertional dyspnea, which progressed to New York Heart Association (NYHA) functional class II symptoms by 2026. Preoperative transthoracic echocardiography demonstrated a 6–7-mm residual perimembranous VSD inferior to the device, with predominantly left-to-right flow and a peak Doppler gradient of approximately 87 mmHg. Ventricular systolic function was preserved, without significant TR or pulmonary hypertension. Following multidisciplinary assessment, surgery was undertaken because of the persistent residual defect, previous recommendations for definitive closure, evolving symptoms, and the anatomical relationship of the device to the tricuspid septal leaflet, with repeat transcatheter intervention considered limited and of uncertain feasibility. Surgical exploration demonstrated the ADO II between the tricuspid septal leaflet and the right-ventricular aspect of the interventricular septum, with adherence to the septal leaflet and localized leaflet distortion. The leaflet was structurally intact. The device was carefully explanted, the residual VSD was directly closed, and the distorted tricuspid septal leaflet was repaired. Intraoperative transesophageal echocardiography demonstrated complete VSD closure and satisfactory tricuspid leaflet coaptation with mild TR. At 6-month follow-up, the patient had no dyspnea or breathing difficulty, with persistent VSD closure, preserved ventricular function, and stable mild TR.

Conclusion: This case documents a late operative device–tricuspid anatomical relationship following ADO II closure of a perimembranous VSD. The principal finding was the location of the device between the tricuspid septal leaflet and the right-ventricular aspect of the interventricular septum, with leaflet adherence and localized distortion despite the absence of significant preoperative TR. Because the original implantation imaging was unavailable, the timing and mechanism of this relationship cannot be established. Furthermore, the restrictive residual VSD, preserved ventricular function, and absence of pulmonary hypertension or major ventricular volume loading do not establish the residual shunt as the direct cause of the patient's exertional symptoms. Surgical exploration permitted simultaneous device explantation, definitive VSD closure, and restoration of tricuspid leaflet coaptation, with satisfactory short-term outcome.

Keywords: Ventricular Septal Defect; Amplatzer Duct Occluder II; Residual VSD; Transcatheter Closure; Tricuspid Septal Leaflet; Device Adherence; Surgical Explantation; Congenital Heart Disease

Introduction

Perimembranous ventricular septal defects (VSDs) are anatomically challenging lesions because of their close relationship to the atrioventricular conduction system, aortic valve, and tricuspid valve. Although surgical closure remains an established treatment, transcatheter closure has become an option for selected patients with suitable defect morphology. Amplatzer Duct Occluders (ADOs), including the Amplatzer Duct Occluder II (ADO II), have been used in selected perimembranous VSDs, with favorable procedural success and acceptable short- and intermediate-term outcomes. Nevertheless, residual shunting and device interaction with adjacent cardiac structures remain important considerations. [1–4]

The perimembranous location is particularly relevant to the tricuspid valve because the septal leaflet and its supporting structures are anatomically adjacent to the right-sided aspect of the interventricular septum. Device-related tricuspid valve injury and late tricuspid regurgitation have been reported following transcatheter VSD closure. [5,6] Persistent residual defects may also remain during longitudinal follow-up and require continued echocardiographic assessment. [7,8]

Late surgical explantation of a VSD occlusion device is uncommon but has been described in association with clinically significant device-related complications. Kumar et al. reported late surgical

removal of an ADO in a patient who developed left bundle branch block and left ventricular dysfunction, with operative evidence of device adherence to the tricuspid septal leaflet and supporting structures. [9] Longer-term pediatric data have also demonstrated the importance of continued surveillance for tricuspid regurgitation following transcatheter closure of perimembranous VSDs. [10] Long-term experience with ADO II has similarly supported continued follow-up after device implantation. [11]

The present case describes the longitudinal persistence of a residual perimembranous VSD following implantation of a 6/6-mm ADO II and the late operative identification of the device between the tricuspid septal leaflet and the right-ventricular (RV) aspect of the interventricular septum, with adherence and localized leaflet distortion despite no significant preoperative TR. Surgical management consisted of device explantation, definitive VSD closure, and tricuspid leaflet repair. Because the original catheterization laboratory fluoroscopic, angiographic, and transesophageal echocardiographic images from the 2022 implantation were unavailable, this report does not attempt to establish whether the observed anatomical relationship was present at implantation or developed subsequently. The findings are therefore presented descriptively rather than as evidence of a specific mechanism.

Case Presentation

A 14-year-old boy was referred to our cardiovascular and cardiothoracic surgical center in 2026 for definitive management of a persistent residual VSD following previous transcatheter device closure.

The patient had undergone transcatheter closure of a perimembranous VSD on February 5, 2022, at another institution. The available contemporaneous procedural documentation recorded right femoral arterial and venous access, angiographic identification and catheter-based crossing of the VSD, and deployment of a 6/6-mm Amplatzer Duct Occluder II. Device position was assessed by fluoroscopy and transesophageal echocardiography (TEE) before release and was reported as satisfactory. The available documentation did not clearly establish the presence or absence of a residual interventricular shunt immediately after device deployment.

However, the original catheterization laboratory fluoroscopic, angiographic, and TEE images were unavailable for retrospective review. Therefore, the original VSD dimensions, morphology, detailed relationship to the aortic and tricuspid valves, and precise three-dimensional relationship between the

device and surrounding structures at the time of implantation could not be independently assessed. Consequently, the timing of onset of the subsequently documented residual VSD and the mechanism of its persistence cannot be determined.

Longitudinal Follow-up

Follow-up echocardiography in 2023 demonstrated a persistent residual VSD measuring approximately 5 mm. The residual shunt was not associated with significant TR or pulmonary hypertension. Surgical closure was advised, but the family initially elected continued observation.

Repeat echocardiography in 2024 continued to demonstrate an approximately 5-mm residual VSD. Tricuspid valve function remained satisfactory without significant regurgitation, and there was no evidence of pulmonary hypertension. Surgical intervention was again advised, and observation was continued.

In 2025, the patient developed mild exertional dyspnea, initially corresponding to NYHA functional class I. The symptoms subsequently progressed to NYHA class II by 2026. Occasional chest discomfort was also reported. He was not receiving any regular

medications. On examination, vital signs were stable, and a cardiac murmur was present. Electrocardiography showed normal sinus rhythm. Laboratory investigations revealed hemoglobin 14.5 g/dL, white blood cell count 9,800/ μ L, platelet count 250,000/ μ L, serum creatinine 0.60 mg/dL, total bilirubin 0.9 mg/dL, aspartate aminotransferase 23 U/L, alanine aminotransferase 22 U/L, and alkaline phosphatase 78 U/L. Chest radiography was normal.

Preoperative Assessment

At our institution, two-dimensional transthoracic echocardiography (2D-TTE)

demonstrated the previously implanted 6/6-mm ADO II in the perimembranous region. A persistent residual VSD measuring approximately 6–7 mm was identified inferior to the device. Color Doppler demonstrated predominantly left-to-right flow, with a peak Doppler gradient of approximately 87 mmHg, consistent with restrictive physiology (Figure 1).

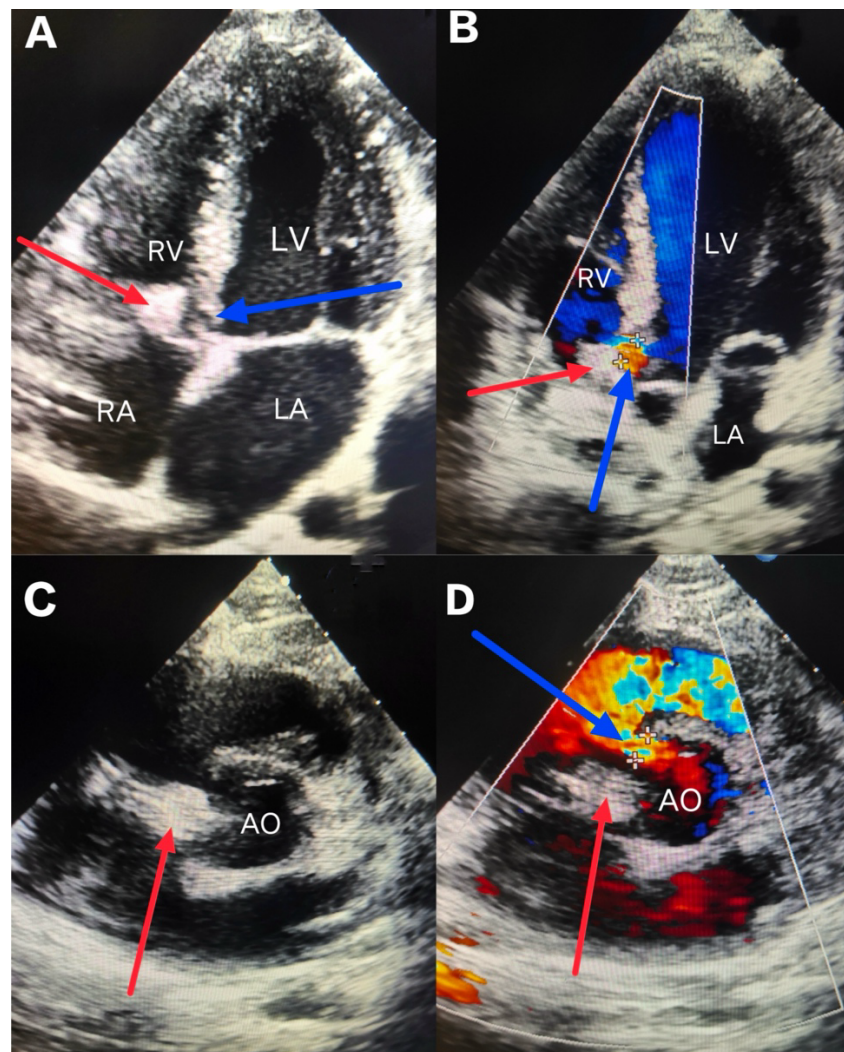


Figure 1. Preoperative transthoracic echocardiographic assessment.

Representative two-dimensional and color Doppler TTE images demonstrating the previously implanted 6/6-mm ADO II and persistent residual perimembranous VSD. The residual defect is located inferior to the implanted device and demonstrates predominantly left-to-right flow with restrictive physiology. The peak Doppler gradient across the residual VSD was reported as approximately 87 mmHg.

RV, right ventricle; LV, left ventricle; RA, right atrium; LA, left atrium; AO, aorta.

Left ventricular ejection fraction (LVEF) was approximately 60%, with preserved left ventricular (LV) systolic function. The right ventricle was normal in size with preserved systolic function. There was no significant TR or echocardiographic evidence of significant pulmonary hypertension. No left ventricular

outflow tract obstruction, intracardiac thrombus, vegetation, or pericardial effusion was identified.

The high Doppler gradient was consistent with a restrictive residual VSD rather than a large nonrestrictive defect. There was no significant pulmonary hypertension, ventricular systolic dysfunction, or documented major ventricular volume loading. Therefore, the available echocardiographic findings did not establish that the residual VSD alone was responsible for the patient's exertional symptoms.

The patient was referred to our center for surgical assessment for complete VSD closure and device explantation. The decision to proceed with surgery was made following multidisciplinary cardiology and cardiovascular surgery assessment. The team considered the persistence of the residual defect over several years, previous recommendations for definitive closure, the patient's evolving symptoms, the anatomical relationship of the implanted device to the adjacent tricuspid septal leaflet, and the limitations and uncertain feasibility of repeat transcatheter intervention in the established device–valve configuration.

Thus, the indication for surgery was not based on evidence that the restrictive residual VSD was hemodynamically dangerous. Rather, the persistent defect and evolving clinical status were considered together with the anatomical concern regarding the implanted device and adjacent valvular structures and the limitations of further catheter-based management. Additional factors supporting operative management included the ongoing risk of device-related vegetation and infective endocarditis, as well as the potential for progressive damage to the tricuspid and aortic valves given the subaortic location of the residual defect.

Surgical Management

The patient underwent surgical intervention through median sternotomy under general anesthesia. Pre-repair intraoperative TEE was performed to delineate the residual perimembranous VSD and its relationship to the implanted device and surrounding structures (Figure 2).

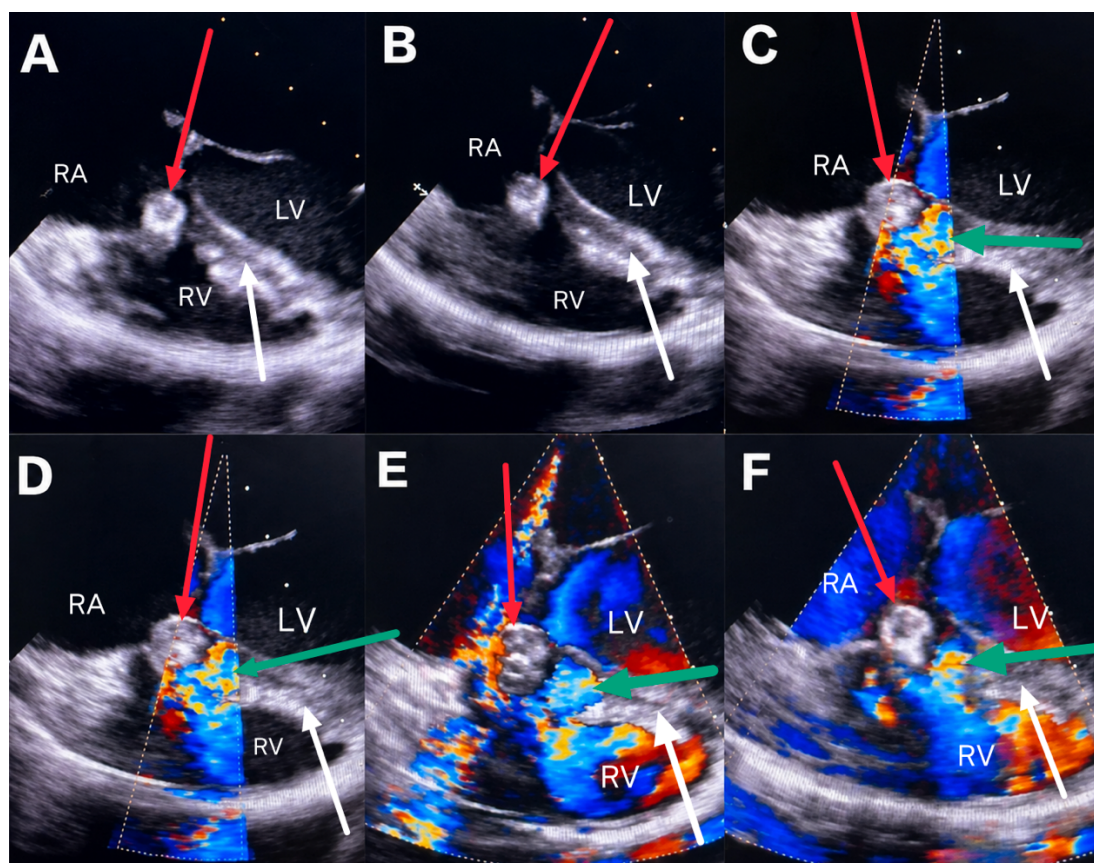


Figure 2. Intraoperative transesophageal echocardiographic assessment before surgical intervention. Intraoperative TEE images (A–F) demonstrating the previously implanted VSD occlusion device (red arrows), residual VSD adjacent to the device (green arrows), and the interventricular septum (IVS; white arrows). Panels A and B show two-dimensional TEE images demonstrating the device in relation to the IVS. Panels C–F demonstrate color Doppler flow across the residual VSD, confirming persistent left-to-right shunting around the device. RA, right atrium; RV, right ventricle; LV, left ventricle; IVS, interventricular septum; TEE, transesophageal echocardiography.

After systemic heparinization, cardiopulmonary bypass was established using standard aortic and bicaval cannulation. The patient was cooled, the aorta was cross-clamped, and cardioplegic arrest was achieved. A right atriotomy was performed to provide direct access to the tricuspid valve and interventricular septal region.

Direct surgical inspection demonstrated that the previously implanted 6/6-mm ADO II was located between the septal leaflet of the tricuspid valve and the right-ventricular aspect of the interventricular septum. The device was adherent to the tricuspid septal leaflet,

with localized leaflet distortion, while the leaflet remained structurally intact without gross tearing or disruption (Figure 3). The operative findings confirmed device–leaflet adherence and localized distortion but did not demonstrate a specific mechanism such as physical trapping, tissue incorporation, tethering, or embedding. Because the original implantation imaging was unavailable, the timing and mechanism of this anatomical relationship could not be determined, including whether it was present at implantation or developed during subsequent follow-up.

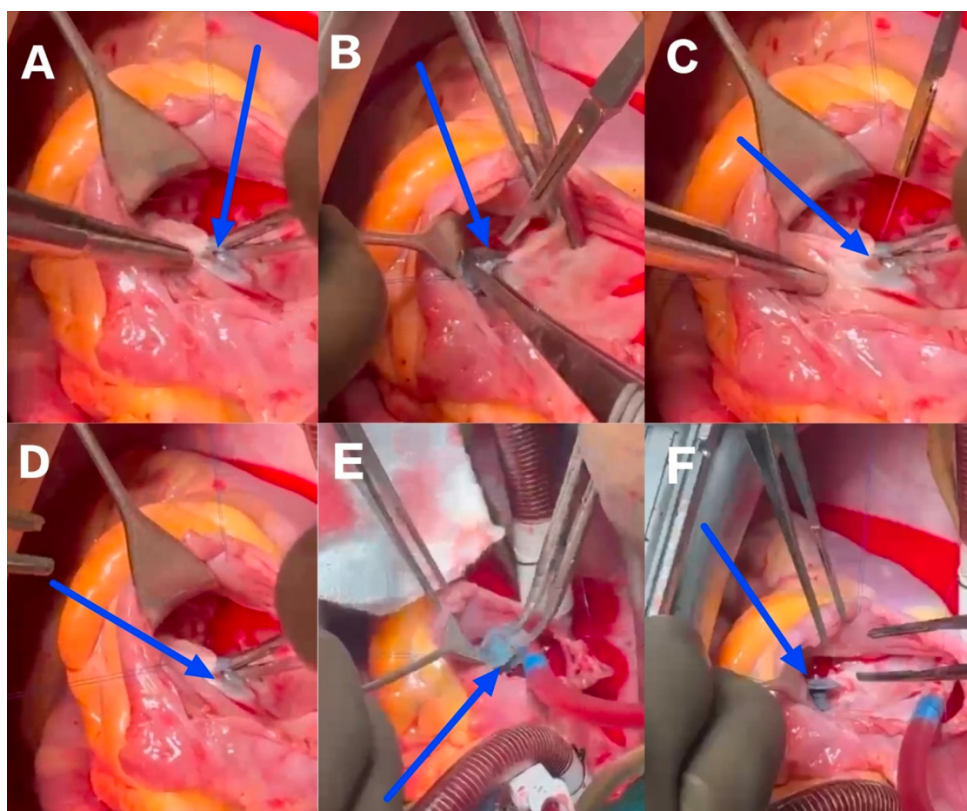


Figure 3. Intraoperative anatomical relationship of the VSD occlusion device and tricuspid septal leaflet before explantation. (A–F) Sequential surgical views demonstrating the 6/6-mm ADO II between the tricuspid septal leaflet and the right-ventricular aspect of the interventricular septum. The device is adherent to the tricuspid septal leaflet, which demonstrates localized distortion.

The device was carefully mobilized under direct vision and explanted, with attention to preservation of the adjacent tricuspid leaflet and septal structures. Following device removal, the residual perimembranous VSD was clearly identified and directly closed using pledgeted 5-0 polypropylene sutures.

Following device removal, the tricuspid septal leaflet was reassessed. Although structurally intact, the leaflet demonstrated persistent localized distortion. A polypropylene coaptation stitch was placed to restore leaflet configuration and achieve satisfactory coaptation. Intraoperative transesophageal

echocardiography confirmed good leaflet coaptation with mild tricuspid regurgitation. The explanted ADO II was inspected macroscopically and demonstrated adherent tissue on its surface (Figure 4).

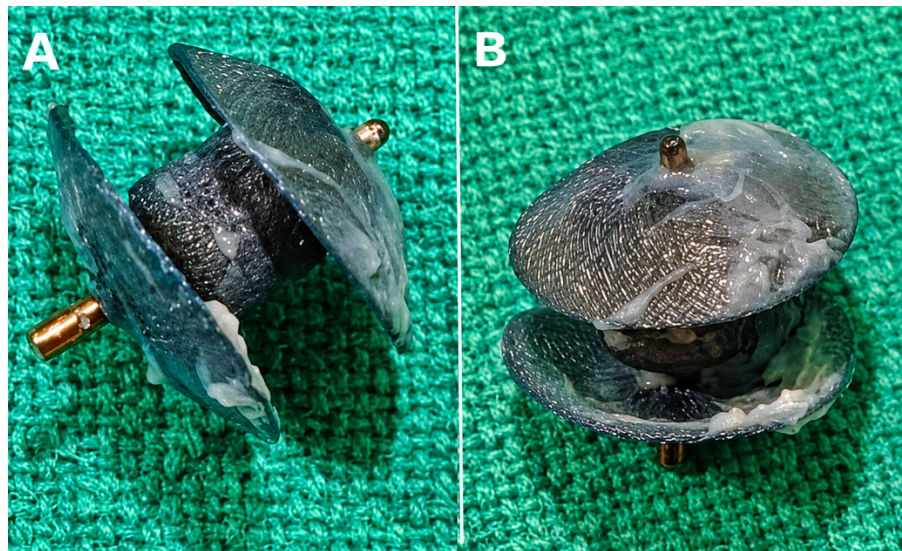


Figure 4. Explanted Amplatzer Duct Occluder II. (A) Lateral view of the explanted 6/6-mm ADO II demonstrating the dual-disc configuration and adherent tissue. (B) En-face/oblique view demonstrating the device and associated adherent tissue.

The right atriotomy was closed, the heart was de-aired, and the aortic cross-clamp was released. The patient was successfully weaned from cardiopulmonary bypass. The total cardiopulmonary bypass time was 98 minutes, and the aortic cross-clamp time was 70 minutes.

Intraoperative TEE demonstrated complete closure of the VSD without residual interventricular shunting, preserved ventricular systolic function, and satisfactory tricuspid leaflet coaptation with mild TR.

Postoperative Course and Follow-up

The postoperative course was uncomplicated without major cardiovascular or surgical complications. Pre-discharge TTE demonstrated an intact interventricular septum without residual VSD or residual interventricular shunt. Biventricular systolic function remained preserved. Mild TR was present without significant pulmonary hypertension (Figure 5).

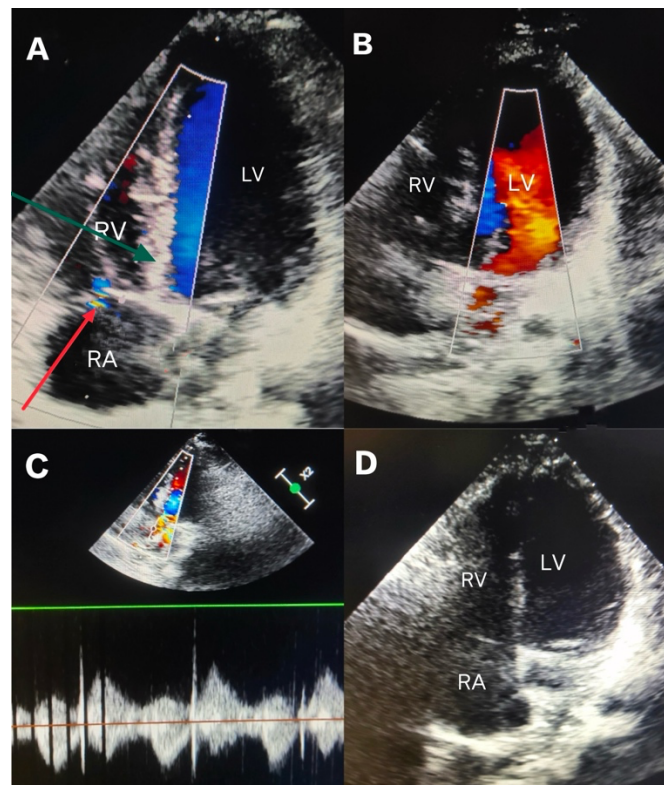


Figure 5. Postoperative transthoracic echocardiographic assessment after VSD device explantation and surgical closure. (A) Color Doppler demonstrating an intact interventricular septum without residual defect or shunt and mild tricuspid

regurgitation. (B) Color Doppler demonstrating no significant residual VSD shunt. (C) Color Doppler with spectral Doppler tracing confirming absence of residual shunt. (D) Two-dimensional four-chamber view demonstrating an intact interventricular septum with preserved ventricular function. RA, right atrium; RV, right ventricle; LV, left ventricle; IVS, interventricular septum; TR, tricuspid regurgitation.

The patient was subsequently followed clinically and echocardiographically at 7 days, 1 month, and 6 months after surgery. At 7 days, there was no residual VSD or residual interventricular shunt. Ventricular systolic function remained preserved, and mild TR was present without pulmonary hypertension.

At 1 month, the clinical and echocardiographic findings remained stable. At 6 months, no residual VSD or pulmonary hypertension was identified. Left and right ventricular systolic function remained preserved,

and mild TR persisted without evidence of progressive valvular dysfunction. Clinically, the patient reported no dyspnea or breathing difficulty. Because six months represents short-term follow-up, these findings do not establish long-term durability of the surgical repair or long-term tricuspid valve outcome.

The overall clinical sequence from initial ADO implantation through serial follow-up, development of symptoms, surgical intervention, and postoperative follow-up is summarized in [Table 1].

Table 1. Clinical sequence of events, diagnostic evaluation, surgical management, and follow-up.

Date/Period	Clinical event
February 5, 2022	Transcatheter closure of perimembranous VSD with a 6/6-mm Amplatzer Duct Occluder II (ADO II) at another institution. Device position was reported as satisfactory before release.
2023	Echocardiography demonstrated an approximately 5-mm residual VSD without significant TR or pulmonary hypertension. Surgical closure was advised; observation was continued.
2024	Persistent approximately 5-mm residual VSD without significant TR or pulmonary hypertension. Surgical intervention was again advised; observation continued.
2025	Mild exertional dyspnea developed and subsequently progressed to NYHA class II symptoms, with occasional chest discomfort.
2026	TTE demonstrated a 6–7-mm residual perimembranous VSD inferior to the ADO II, with predominantly left-to-right shunting and a reported peak Doppler gradient of approximately 87 mmHg. Biventricular systolic function was preserved without significant TR or pulmonary hypertension.
2026 – Surgery	Surgical exploration demonstrated ADO II adherence to the tricuspid septal leaflet with localized distortion. The device was explanted, the residual VSD was directly closed, and the leaflet was repaired.
Postoperative follow-up	Intraoperative TEE confirmed complete VSD closure and satisfactory tricuspid leaflet coaptation with mild TR. At 6 months, there was no residual VSD or pulmonary hypertension, ventricular function remained preserved, and mild TR was stable; the patient reported no dyspnea.

Discussion

Principal Observation

The principal finding in this case was the late surgical identification of a 6/6-mm ADO II positioned between the tricuspid septal leaflet and the right-ventricular aspect of the interventricular septum, with adherence and localized distortion of the tricuspid septal leaflet. This occurred in the setting of a persistent residual perimembranous VSD documented on serial echocardiography over several years following transcatheter closure.

Surgical explantation of the device, direct closure of the residual VSD, and repair of the distorted

tricuspid leaflet resulted in complete VSD closure and satisfactory tricuspid valve function during 6 months of follow-up.

Device-related tricuspid valve interaction, residual shunting, and late surgical removal of VSD occlusion devices have previously been reported. [5,7,8,11] Therefore, this case should not be interpreted as demonstrating an entirely new complication. Its clinical contribution lies in the longitudinal documentation of persistent residual shunting followed by direct operative characterization of a late device–tricuspid leaflet relationship, with leaflet adherence and

localized distortion despite the absence of significant preoperative TR.

Transcatheter Closure of Perimembranous VSD

Transcatheter closure is an established treatment option for selected perimembranous VSDs with suitable anatomy. Studies evaluating ADOs, particularly ADO II, have demonstrated favorable procedural success and acceptable short- and intermediate-term outcomes in appropriately selected patients. [1,3,4] Nevertheless, residual shunting and interaction with adjacent cardiac structures remain recognized considerations, particularly because of the anatomical proximity of the perimembranous septum to the tricuspid valve, aortic valve, and conduction system. [5,6]

Device selection and deployment therefore require careful assessment of defect size, morphology, surrounding rims, and proximity to the tricuspid and aortic valves. Although many residual shunts decrease or resolve during follow-up, persistent residual defects may remain and warrant continued echocardiographic surveillance. [5,6]

In the present case, the contemporaneous procedural record documented catheter-based crossing of the VSD, deployment of a 6/6-mm ADO II, and assessment by TEE before device release. Device position was reported as satisfactory at the time of implantation. However, the original catheterization fluoroscopy, angiographic images, and TEE images were unavailable for retrospective review. Consequently, the original VSD morphology and precise three-dimensional relationship between the device, interventricular septum, and tricuspid valve cannot be independently reconstructed.

Persistent Residual VSD

The residual VSD was first documented approximately one year after device implantation and persisted on echocardiographic examinations in 2024 and 2026. It measured approximately 5 mm during earlier follow-up and approximately 6–7 mm in 2026, with predominantly left-to-right flow and a peak Doppler gradient of approximately 87 mmHg.

The high Doppler gradient is consistent with restrictive physiology rather than a large nonrestrictive VSD. The patient had preserved left and right ventricular systolic function, no significant pulmonary hypertension, and no documented major ventricular volume loading. Thus, the residual defect did not demonstrate the physiological features of a large hemodynamically significant VSD.

The persistence of the residual defect nevertheless remained clinically relevant because definitive surgical closure had been recommended on

previous assessments. Importantly, the available information does not allow differentiation between incomplete initial closure, characteristics of the original defect, subsequent tissue remodeling, or another mechanism. Therefore, no specific mechanism for persistence of the residual VSD can be established.

Device–Tricuspid Septal Leaflet Relationship

The close anatomical relationship between perimembranous VSD occlusion devices and the tricuspid valve is well recognized. Previous reports have described direct tricuspid leaflet injury following ADO implantation, including leaflet tears requiring surgical repair.[7] Late tricuspid regurgitation following transcatheter VSD closure has also been reported, and larger pediatric studies have evaluated the incidence and predictors of tricuspid regurgitation after perimembranous VSD device closure.[8,10]

In the present case, surgical exploration demonstrated a device–leaflet relationship characterized by the ADO II being located between the tricuspid septal leaflet and the right-ventricular aspect of the interventricular septum. The device was adherent to the septal leaflet, which showed localized distortion but remained structurally intact without gross tearing or disruption. Significant TR was not present preoperatively. Following device explantation, the persistent leaflet distortion was repaired with a polypropylene coaptation stitch to restore satisfactory leaflet configuration and coaptation. Intraoperative TEE confirmed satisfactory leaflet coaptation with mild residual TR.

The operative findings therefore documented device–leaflet adherence and localized distortion without evidence of leaflet rupture or a specific mechanism such as physical trapping, tissue incorporation, tethering, or embedding. Because the original implantation imaging was unavailable, it could not be determined whether this anatomical relationship was present at the time of device deployment or developed during subsequent follow-up. Accordingly, the findings should be interpreted as a documented late device–tricuspid leaflet relationship rather than evidence of a specific mechanism or timing of its development.

Interpretation of the Late Operative Anatomy

The late operative position of the device requires cautious interpretation. The ADO II was located between the tricuspid septal leaflet and the right-ventricular aspect of the interventricular septum at the time of surgery. However, without the original fluoroscopic, angiographic, and TEE images, it is impossible to determine whether this relationship was present immediately after implantation or developed progressively during follow-up.

The available procedural record documenting satisfactory device deployment cannot substitute for review of the original imaging. Accordingly, the present case does not establish device migration, displacement, malposition, erosion, or another specific implantation-related mechanism.

The operative findings describe the anatomical relationship present several years after implantation but do not establish its temporal evolution. This distinction is important because late operative anatomy should not be used retrospectively to infer a mechanism that cannot be demonstrated from the available longitudinal imaging.

Rationale for Tricuspid Leaflet Repair

The decision to repair the tricuspid septal leaflet was based on direct surgical assessment. Although the leaflet was structurally intact, localized distortion persisted after removal of the adherent ADO II. Restoration of the leaflet configuration and coaptation was therefore considered preferable to leaving the persistent distortion untreated.

The procedure permitted simultaneous management of the implanted device, persistent residual VSD, and tricuspid leaflet abnormality. Post-repair intraoperative TEE demonstrated complete VSD closure and satisfactory tricuspid valve coaptation with mild TR.

This experience illustrates the potential advantage of direct surgical visualization when definitive surgery is undertaken in the presence of a device closely related to the tricuspid valve. However, this single case does not establish that surgical leaflet repair is routinely required for similar device–tricuspid relationships.

Comparison With Previous Reports

Late surgical explantation of VSD occlusion devices is uncommon but has been described in patients with clinically important device-related complications. Kumar et al. reported late ADO explantation in a patient who developed left bundle branch block and left ventricular dysfunction, with operative evidence of device adherence to the tricuspid septal leaflet and supporting structures. [9]

The present case shares the features of delayed device removal and a device–tricuspid relationship but differs in its clinical presentation. There was no documented conduction abnormality, significant ventricular dysfunction, or clinically important preoperative TR. Instead, the principal findings were persistent residual VSD and late adherence and distortion of an otherwise structurally intact tricuspid septal leaflet.

These differences indicate that late surgical explantation may occur in different clinical settings.

However, the present case does not provide sufficient evidence to define a new mechanism, risk factor, or causal pathway for device-related tricuspid interaction.

Literature Gap and Novelty

Previous reports have established that ADO-based closure of selected perimembranous VSDs can be effective and that residual shunting, tricuspid valve interaction, and late device-related surgical intervention are recognized clinical events. However, reports providing longitudinal documentation of a persistent residual VSD followed several years later by direct operative characterization of the spatial relationship between an implanted ADO II, the tricuspid septal leaflet, and the right-ventricular septal surface are limited.

The specific contribution of this case is the documented combination of four findings: (1) persistent residual VSD on serial follow-up, (2) late operative identification of the ADO II positioned between the tricuspid septal leaflet and the right-ventricular aspect of the interventricular septum, (3) tricuspid septal leaflet adherence and localized distortion despite no significant preoperative tricuspid regurgitation, and (4) successful simultaneous device explantation, residual VSD closure, and tricuspid leaflet repair.

The novelty therefore lies in the longitudinal clinical–echocardiographic follow-up and subsequent direct operative anatomical correlation, rather than in proposing a previously unrecognized device complication or mechanism. Because the original implantation fluoroscopy, angiography, and TEE images were unavailable, the timing and mechanism of the device–leaflet relationship cannot be determined. This case should consequently be interpreted as an uncommon late anatomical observation that expands the descriptive literature on delayed device–tricuspid valve relationships after ADO II closure of perimembranous VSD.

Exertional Symptoms and Clinical Interpretation

The patient's progressive exertional dyspnea occurred during the period in which the residual VSD persisted. However, the residual defect demonstrated restrictive physiology with a high Doppler gradient and was not associated with significant pulmonary hypertension, ventricular systolic dysfunction, or documented major ventricular volume loading.

Therefore, the available findings do not establish the residual VSD as the cause of the patient's symptoms. Other potential contributors, including deconditioning or non-cardiac causes, were not systematically evaluated. The temporal association between symptoms and the residual defect was

considered in the overall clinical assessment but should not be interpreted as evidence of causality.

The postoperative absence of dyspnea is clinically reassuring, but improvement after surgery does not prove that the residual VSD was the direct cause of the preoperative symptoms.

The decision to proceed with surgery was individualized, taking into account the persistent residual defect, previous recommendations for definitive closure, evolving symptoms, the anatomical relationship of the device to the tricuspid septal leaflet, and the limitations and uncertain feasibility of repeat transcatheter intervention.

Importance of Longitudinal Surveillance

Long-term follow-up remains important after transcatheter closure of perimembranous VSDs because residual shunting and changes involving adjacent cardiac structures may persist or become apparent during follow-up. [5,10,11]

In this patient, serial echocardiography demonstrated persistence of the residual VSD and provided evidence that significant TR and pulmonary hypertension were not present during earlier surveillance. This longitudinal information was useful in interpreting the later operative findings.

The case also emphasizes the importance of preserving complete procedural imaging. Availability of the original fluoroscopy, angiography, and TEE images would have permitted direct comparison between the initial device position and the late surgical anatomy. In their absence, the temporal evolution of the device–tricuspid relationship remains uncertain.

Surgical Implications

When surgery is selected for persistent residual VSD after device closure, direct surgical inspection can provide anatomical information that may not be completely characterized by preoperative imaging. In this patient, surgical exposure allowed direct assessment of the ADO II, residual VSD, interventricular septum, and tricuspid septal leaflet.

The ADO II was carefully explanted, the residual VSD was closed directly, and the distorted tricuspid septal leaflet was repaired. Intraoperative TEE confirmed complete VSD closure and satisfactory leaflet coaptation. Follow-up at 7 days, 1 month, and 6 months demonstrated persistent VSD closure, preserved ventricular function, absence of pulmonary hypertension, and stable mild TR.

These findings demonstrate the feasibility of combined surgical management in this individual patient. They should not, however, be generalized to all patients with residual VSD after device closure. Management should remain individualized according to residual defect characteristics, hemodynamic

significance, device anatomy, tricuspid valve involvement, ventricular response, symptoms, and the relative feasibility and risks of catheter-based and surgical approaches.

Limitations

This case has several limitations. The original fluoroscopic, angiographic, and transesophageal echocardiographic images from the 2022 device-closure procedure were unavailable, limiting assessment of the initial VSD anatomy and device–tricuspid relationship and precluding determination of the timing or mechanism of the late device–leaflet interaction. The original VSD dimensions and detailed hemodynamic characteristics could therefore not be independently reconstructed. Although the residual VSD was restrictive, with preserved ventricular function and no pulmonary hypertension or major ventricular volume loading, its direct contribution to the patient's exertional dyspnea cannot be established. In addition, the original spectral Doppler tracing corresponding to the reported peak gradient of approximately 87 mmHg was unavailable; this value was obtained from the contemporaneous echocardiography report and could not be independently verified. Finally, the single-case design and 6-month postoperative follow-up limit conclusions regarding the frequency, mechanism, and long-term outcomes of similar device–tricuspid leaflet interactions.

Long-term clinical and echocardiographic surveillance, including annual TTE when clinically appropriate, may help monitor delayed tricuspid regurgitation, right ventricular remodeling, and recurrent residual shunting, with follow-up frequency individualized according to clinical status and echocardiographic findings.

Clinical Implications

The present case emphasizes the value of continued echocardiographic surveillance after perimembranous VSD device closure, particularly when residual shunting persists. Assessment should include residual flow, ventricular response, pulmonary pressure, and adjacent valvular function.

When definitive surgery is required, direct inspection may identify device–leaflet relationships and permit simultaneous correction of the residual VSD and associated valve abnormality. The findings are best regarded as an uncommon descriptive observation that adds a longitudinal and operative anatomical perspective to the literature on late findings following transcatheter closure of perimembranous VSDs.

Conclusion

This case describes delayed surgical explantation of a 6/6-mm Amplatzer Duct Occluder II (ADO II) in an adolescent with a persistent residual perimembranous VSD. Serial echocardiography documented persistence of the residual defect without significant tricuspid regurgitation or pulmonary hypertension, while surgical exploration demonstrated device adherence to the tricuspid septal leaflet with localized distortion despite preserved leaflet integrity. The device was successfully explanted, the residual VSD was directly closed, and the tricuspid leaflet was repaired, with complete VSD closure and satisfactory valve function during 6 months of follow-up. This case highlights the value of longitudinal echocardiographic surveillance after perimembranous VSD device closure and demonstrates the importance of complete implantation and post-deployment imaging for assessing the evolution and timing of late device–tricuspid leaflet relationships.

Learning Points

1. Persistent residual VSD after transcatheter closure warrants longitudinal echocardiographic surveillance.
2. Follow-up should include assessment of residual shunting, ventricular response, pulmonary pressure, and tricuspid valve morphology and function.
3. A restrictive residual VSD with a high Doppler gradient does not, by itself, establish a cause for exertional symptoms.
4. Late surgical exploration may demonstrate a device–valve anatomical relationship despite the absence of significant preoperative tricuspid regurgitation.
5. When original implantation imaging is unavailable, late device–tissue relationships should be described anatomically without assuming malposition, migration, or another specific mechanism.

List of Abbreviations

ADO II – Amplatzer Duct Occluder II
TEE – Transesophageal echocardiography
TTE – Transthoracic echocardiography
VSD – Ventricular septal defect
TR – Tricuspid regurgitation
NYHA – New York Heart Association
LV – Left ventricle
LVEF – Left ventricular ejection fraction
RV – Right ventricle
IVS – Interventricular septum
POD – Postoperative day

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References

1. Ghaderian M, Sabri MR, Ahmadi A, Dehghan B, Mahdavi M. Safety and efficacy of using Amplatzer ductal occluder type I and II for peri-membranous ventricular septal defect closure: a systematic review. *ARYA Atheroscler.* 2024;20(6):54-64. doi:[10.48305/arya.2024.42641.2962](https://doi.org/10.48305/arya.2024.42641.2962)
2. Amoozgar H, Edraki MR, Ajami G, Mohammadi H, Mehdizadegan N, Amirghofran A. Short and mid-term outcomes of percutaneous perimembranous ventricular septal defect closure using Amplatzer duct occluders type II in children and adolescents. *Iran J Med Sci.* 2025;50(6):393-403. doi:[10.30476/ijms.2024.102819.3595](https://doi.org/10.30476/ijms.2024.102819.3595)
3. Liu S, Zhang W, Li J, Wang S, Qian M, Shi J. Transcatheter closure of perimembranous and intracristal ventricular septal defects using Amplatzer duct occluder II in children. *J Interv Cardiol.* 2021;2021:4091888. doi:[10.1155/2021/4091888](https://doi.org/10.1155/2021/4091888)
4. Cen H, Peng B, Li J, Chen S, Sun P. Efficacy and safety of the Amplatzer Duct Occluder II for ventricular septal defect closure: a meta-analysis. *Kardiologia Pol.* 2021;79(4):401-9. doi:[10.33963/KP.15762](https://doi.org/10.33963/KP.15762)
5. Elmarsafawy H, Hafez M, Alsawah GA, Bakr A, Rakha S. Long-term outcomes of percutaneous closure of ventricular septal defects in children using different devices: a single-centre experience from Egypt. *BMC Pediatr.* 2023;23:381. doi:[10.1186/s12887-023-04194-9](https://doi.org/10.1186/s12887-023-04194-9)
6. Haddad RN, Daou L, Saliba Z. Device closure of perimembranous ventricular septal defect: choosing between Amplatzer occluders. *Front Pediatr.* 2019;7:300. doi:[10.3389/fped.2019.00300](https://doi.org/10.3389/fped.2019.00300)
7. Kuwelker SM, Shetty DP, Dalvi B. Surgical repair of tricuspid valve leaflet tear following percutaneous closure of perimembranous ventricular septal defect using Amplatzer duct occluder I: report of two cases. *Ann Pediatr Cardiol.* 2017;10(1):61-4. doi:[10.4103/0974-2069.197052](https://doi.org/10.4103/0974-2069.197052)
8. Sarkislali K, Kalangos A. Late tricuspid regurgitation after percutaneous transcatheter closure of ventricular septal defect: an educational presentation. *Braz J Cardiovasc Surg.* 2021;36(2):253-6. doi:[10.21470/1678-9741-2020-0172](https://doi.org/10.21470/1678-9741-2020-0172)
9. Kumar HVJ, Mathew A, Subramanian A, Paraswanath BA, Padmanabhan D, Barthur A. Role of late surgical explantation of device from perimembranous ventricular septal defect for left bundle branch block and left ventricular dysfunction. *Heart Rhythm Case Rep.* 2020;6(4):178-82. doi:[10.1016/j.hrcr.2019.12.006](https://doi.org/10.1016/j.hrcr.2019.12.006)
10. Jiang D, Yi Y, Zhao L, Wang J, Wang Y, Lv J. Risk factors and long-term outcomes of tricuspid regurgitation after transcatheter closure of pediatric perimembranous ventricular septal defects. *J Am Heart Assoc.* 2025;14(6):e039443. doi:[10.1161/JAHA.124.039443](https://doi.org/10.1161/JAHA.124.039443)
11. Wang JM, Wang QG, Meng LL, Geng JS, Wang ZC, Xiao JW. Middle and long-term follow-up of effectiveness and safety of transcatheter closure of perimembranous ventricular septal defects using Amplatzer duct occluder II occluders in adults. *Chin Heart J.* 2023;35(2):150-5. <https://doi.org/10.32604/chd.2022.021855>