

Role of late surgical explantation of device from perimembranous ventricular septal defect for left bundle branch block and left ventricular dysfunction



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Introduction

Device closures of perimembranous ventricular septal defects (pm VSD) have been carried out for over 2 decades now and are considered an accepted alternative to surgical closure in selected cases.¹ While the development of heart block or aortic regurgitation had been major concerns in the past, the occurrence of complete left bundle branch block (LBBB) after device closure of pm VSD is quite rare, with few published reports.^{2,3} Development of LBBB is nevertheless significant, leading to septal dyssynchrony and progressive deterioration of left ventricular (LV) function. We report a case of resolution of LBBB after surgical explantation of Amplatzer duct occluder, several months after transcatheter pm VSD closure.

Case report

A 6-year-old, asymptomatic boy weighing 18 kg underwent device closure of a moderate-sized pm VSD with septal aneurysm using 12/10 Amplatzer duct occluder (Abbott, Santa Clara, CA) in April 2018. The left ventriculogram showed 2 separate jets on the LV aspect and opacification of the right ventricle and right atrium through several openings in the ventricular septal aneurysm ([Supplementary Video 1](#)). The combined diameter of all defects on the right ventricular aspect was around 15 mm. Device closure of the defect was considered owing to dilated left-sided chambers on echocardiography (LV end-diastolic diameter z-score of +2.0) and a cardiothoracic ratio > 0.5 on chest radiography. The preprocedural electrocardiogram (ECG) was normal, except for prominent “q” waves in left precordial leads, suggestive of LV volume overload ([Figure 1A](#)). The procedure was uneventful. Postprocedural electrocardiogram at 24 and 48

KEY TEACHING POINTS

- Left bundle branch block (LBBB) is a less recognized complication of transcatheter closure of perimembranous ventricular septal defects and is more likely to occur in defects that extend into the trabecular septum.
- Device-induced LBBB can lead to progressive ventricular dysfunction due to septal dyssynchrony.
- Fascicular blocks, complete bundle branch blocks, or complete heart block after placement of a device may be late complications, and long-term follow-up is mandatory after device closure of perimembranous ventricular septal defects.
- While cardiac resynchronization therapy has been conventionally used to improve left ventricular function in this setting, surgical removal of the device (even if carried out much later) could lead to spontaneous resolution of LBBB with normalization of ventricular function.

hours after the procedure ([Figure 1B](#)) showed normal sinus rhythm with 1:1 atrioventricular (AV) conduction and normal QRS duration (QRSd) of 40 ms. He was advised follow-up at 1 month post procedure. The ECG at 1 month follow-up showed normal sinus rhythm with complete LBBB and QRSd of 130 ms ([Figure 1C](#)). The echocardiogram showed the device in position, no residual shunt, no aortic regurgitation, and normal biventricular function. Conservative management was preferred at this time, after which he was lost to follow-up for 6 months, when he presented with mild exertional dyspnea and increased precordial activity. An ECG at this point showed sinus rhythm with 1:1 AV conduction and persistent LBBB with QRSd of 130 ms ([Figure 1D](#)). The echocardiogram showed ventricular septal

KEYWORDS Device; Left bundle branch block; Perimembranous ventricular septal defect; Septal dyssynchrony; Surgical explantation ([Heart Rhythm Case Reports 2020;6:178–182](#))

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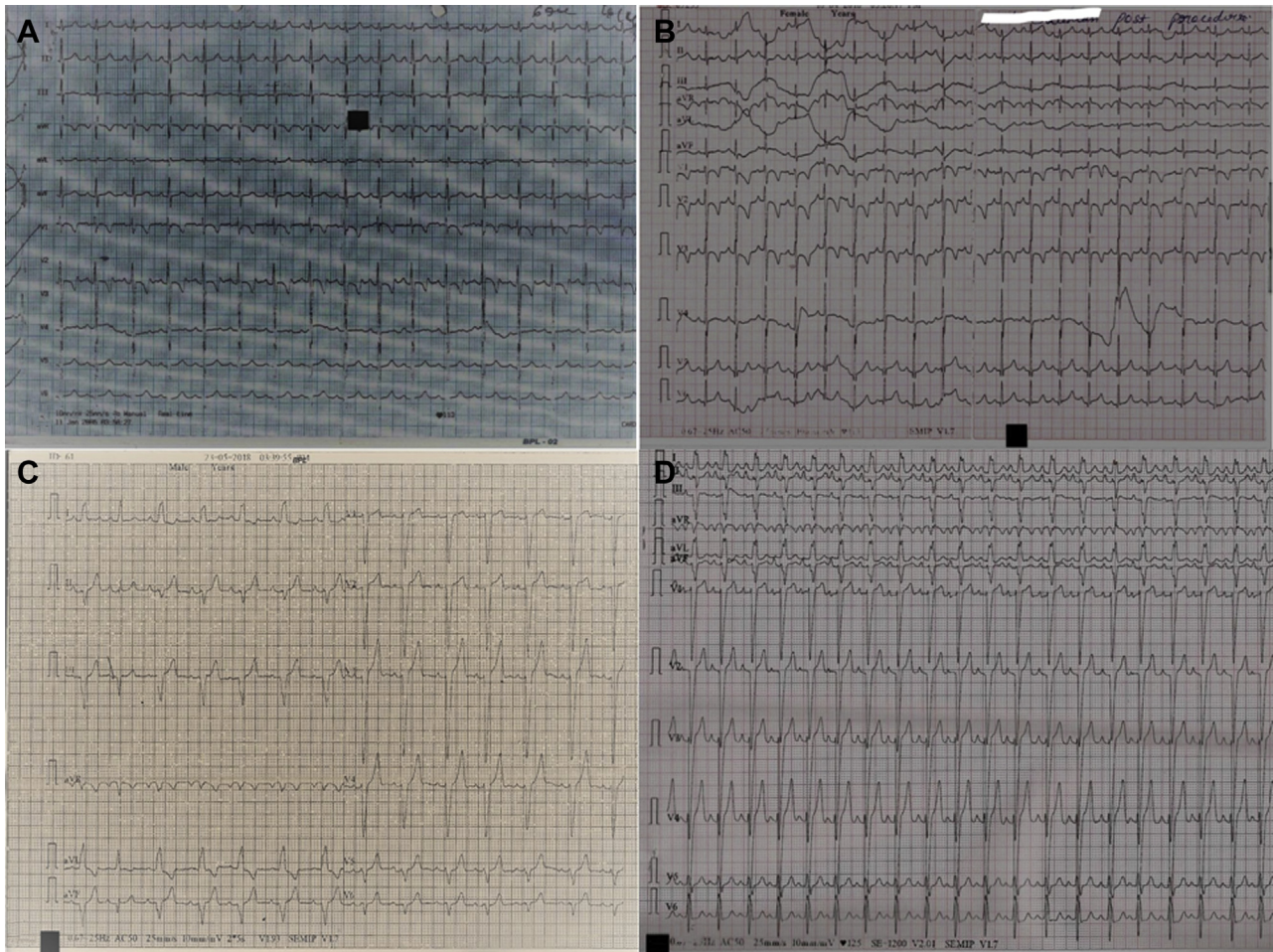


Figure 1 Electrocardiograms. **A:** Preprocedure; **B:** immediately postprocedure; **C:** at 1 month after perimembranous ventricular septal defect (pm VSD) device closure; and **D:** 6 months after pm VSD device closure. Note left bundle branch block in the latter 2 images.

dyssynchrony, dilated left ventricle, and LV dysfunction (LV ejection fraction [LVEF] of 30%). There was no history to suggest an alternative cause for the sudden deterioration in ventricular function. Cardiac magnetic resonance imaging (CMR) showed the device in the perimembranous location with dyssynchronous contraction of the ventricular septum, mild LV dilation (indexed LV end-diastolic volume: 102 mL/m²; indexed LV end-systolic volume: 76 mL/m²), and severe LV dysfunction (LVEF: 24%) (Supplementary Videos 2 and 3). There was no myocardial edema or late gadolinium enhancement (LGE) to suggest inflammation or scarring in the area around the device and there were no findings to suggest an alternate etiology for LV dysfunction (Figure 2A–D). A 24-hour Holter recording showed persistent LBBB, whereupon angiotensin-converting enzyme inhibitors and diuretics were started for symptomatic benefit.

A multidisciplinary team consisting of pediatric cardiologists, cardiac surgeons, electrophysiologists, and a cardiac imaging specialist debated on the further course of action. A decision to attempt device retrieval was taken in preference to implantation of a cardiac resynchronization therapy (CRT) device, given the child's age (potential need for multiple pack changes and lead replacement), the nitinol make of the device

(persistent tendency to expand post implantation), and the absence of LGE in the peri-device area on CMR (barring the area of susceptibility artifact due to the device), which excluded overt focal fibrosis, thus suggesting possible recovery of LBBB. The decision was also guided by our experience with a similar case, wherein the child succumbed to LV dysfunction several months after the development of LBBB, following pm VSD closure using an Amplatzer duct occluder II device.

The surgeons elected to use a standard midline sternotomy and aorto-bicaval cannulation for cardiopulmonary bypass, and cardioplegia was achieved using Del Nido cardioplegic solution at 30° Celsius. The aorta and right atrium were opened. The device was seen adherent to the septal leaflet of the tricuspid valve and its chordae. A fibrous capsule had formed all around the device and the retention skirt of the duct occluder was well apposed to the LV surface of the pm VSD. The screwing end of the device was isolated and the delivery cable was screwed on to the device for possible exteriorization into a loader sheath after collapsing the device. Because of endothelialization within and around the device, the device could not be slenderized within the sheath. The device was teased away from the surrounding

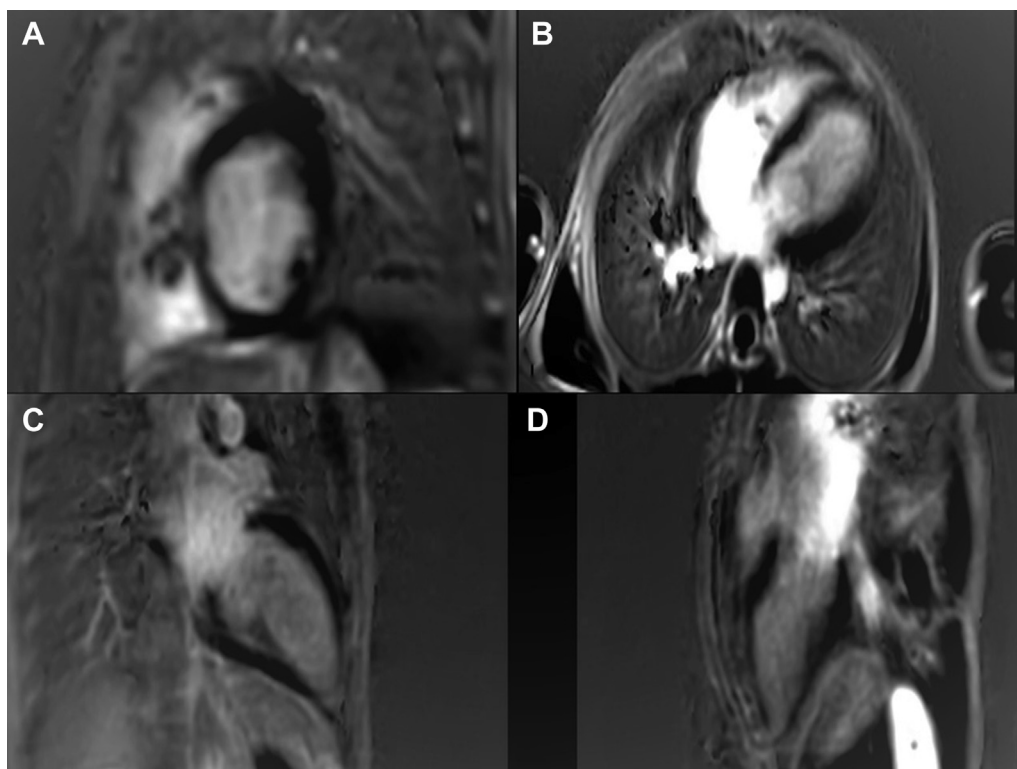


Figure 2 Post-contrast phase-sensitive inversion recovery images in ventricular short axis (A), 4-chamber (B), 2-chamber (C), and 3-chamber (D) views demonstrating absence of late gadolinium enhancement in the ventricles.

fibrous capsule on both sides of the septum by meticulous dissection and extracted. The VSD was closed using a Sauvage patch (Bard Inc, Tempe, AZ) with interrupted prolene sutures. The aortic cusps were inspected and found to be intact. Post VSD closure, intraoperative pulmonary artery pressures were one-third of systemic with no step up on oximetry run. Two steroid-eluting, bipolar pacing leads (CapSure Sense 4968 – 35 cm and CapSure 4968 – 60 cm, Medtronic Inc, Minneapolis, MN) were placed epicardially on the lateral aspect of the left ventricle between 2 obtuse marginal vessels, at a distance of 2 cm from each other and tunneled to a pocket created in the left infraclavicular area, anticipating a need for CRT in the future. The patient came off bypass smoothly, with transient 2:1 AV block, which reverted to sinus rhythm with 1:1 AV conduction with persistence of LBBB and QRSd of 130 ms (Figure 3A). He was discharged home a week after surgery on appropriate doses of anticongestive medications. A month after device explantation, his LBBB had recovered, with a normal QRSd of 40 ms (Figure 3B). Echocardiogram (Supplementary Video 4) showed lesser degree of septal dyssynchrony and improved LV dimensions and systolic function (LV fractional shortening of 24% and LVEF of 50%). This resolution continued to be maintained at 6 months follow-up and 24-hour Holter recording confirmed resolution of LBBB. Guideline-mandated anti-heart failure therapy continues, with anticipated 2-year duration of therapy. Although there has been no recurrence of LBBB over the last 6 months, the pacing leads placed on the LV epicardial surface have not been removed.

Discussion

The proximity of the AV node in relation to a membranous VSD is well known, and surgical and device closures have an inherent risk of developing complete heart block. This most commonly occurs either intraoperatively or immediately post procedure. The bundle of His traverses a distance before piercing the central fibrous body before bifurcation. In perimembranous defects, the bundle traverses inferoposterior to the VSD and usually bifurcates along its inferior margin, where the muscular septum starts.⁴ While the right bundle branch is well defined and descends along the right ventricular aspect of the septum, the extensive left bundle fans out into anterior, septal, and posterior fascicles on the LV aspect of the septum. The specialized cells that form the left bundle, although interconnected, are more widely distributed on the LV aspect of the interventricular septum.⁵ Not surprisingly, right bundle branch block and left fascicular blocks have been described more often following pm VSD device closure or even during sheath and catheter manipulation across the defect.^{2,6} The left bundle branch would be more vulnerable in defects that extend inferiorly into the muscular septum, rather than true membranous defects. However, all perimembranous defects extend variably into the trabecular portion of the septum and have been closed using devices, with low risk of postprocedural complications. Oversized devices may predispose to conduction disturbances. As there were multiple openings on the right ventricular aspect of the aneurysm, along with an indirect Gerbode defect, complete closure of the VSD depended on the

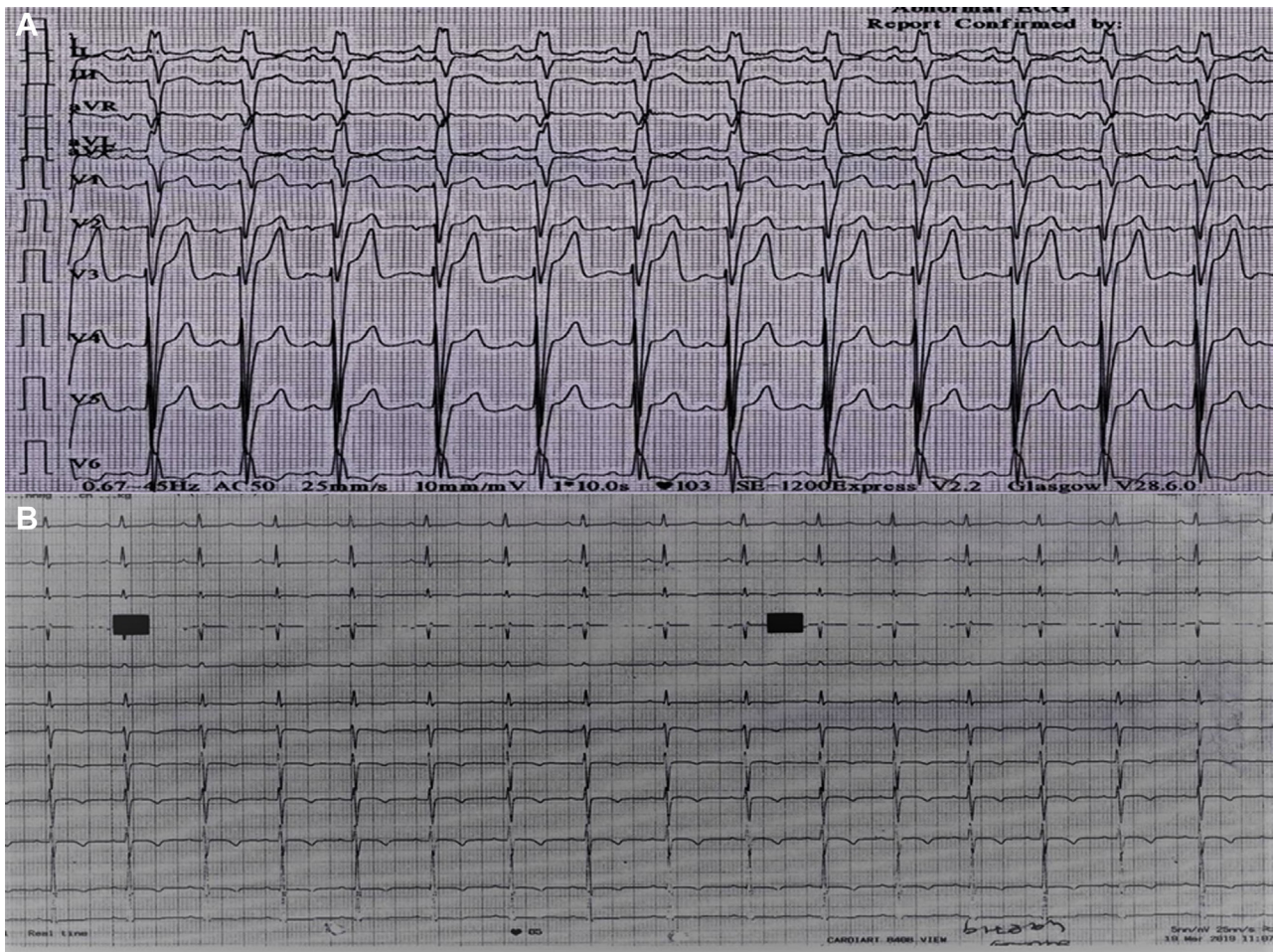


Figure 3 Electrocardiogram immediately after surgical device retrieval (A) and 1 month post surgery (B), with resolution of left bundle branch block.

retention skirt, rather than the waist of the device, which would have occluded only the opening where it was deployed. The 12/10 mm Amplatzer duct occluder has a retention skirt measuring 18 mm in diameter. The septal aneurysm is prone to stretch, and placing the device entirely within the aneurysm may have resulted in small residual leaks. Intra-aneurysmal device deployment is not feasible in all pm VSDs. Hence, the device was placed with the retention skirt apposed to the LV aspect of the septum ([Supplementary Video 5](#)). Whether downsizing the device could have prevented the complication while still achieving defect closure is speculative, but the larger device may have played a part. Variations in anatomy and branching pattern of the bundle might be a possibility in the few case reports describing complete LBBB. Peri-procedural LBBB has been erroneously classified as a minor complication in literature. Steroids have been used anecdotally to reduce edema and thereby improve conduction.⁷ We do not know if commencement of steroids a month after the procedure would have altered the bundle branch pattern. We postulate mechanical compression of the left bundle branches by the retention skirt and persistent tendency of the nitinol material in the closure device to expand as potential reasons for the delayed appear-

ance of LBBB. Over a period of time, this might lead to irreversible damage and fibrosis. CMR in our patient did not show any LGE to suggest fibrosis in any portion of the left ventricle.⁸ We assumed a fair chance of recovery, if the device could be safely removed without causing further damage to the surrounding conduction tissue during surgery. As the device was presumed to have endothelialized, surgical dissection during device retrieval had to be meticulous and carried out with finesse to avoid the development of complete heart block.

It is rather tempting to attribute such ECG changes to other nonspecific etiologies, such as myocarditis. Unless proven otherwise, bundle branch blocks, nonspecific intraventricular conduction disturbances, and complete heart block should be considered post procedural complications. The fact that LBBB was seen a month after the procedure, when the child still had normal ventricular function and absence of any other changes on CMR to suggest myocarditis,⁹ only confirmed our suspicion of device-induced LBBB. LBBB in LV dysfunction secondary to myocarditis or idiopathic dilated cardiomyopathy is usually a late finding, portends poor prognosis, and does not precede the development of ventricular dysfunction. The LBBB in our patient was thus

postprocedural and LV dysfunction was a result of chronic interventricular dyssynchrony.⁹ CRT has been tried in an elderly woman who developed LBBB.¹⁰ As mentioned earlier, we did not want CRT alone to be a treatment option. While preparing for it, we believed in giving a chance at spontaneous recovery. One cannot overemphasize the advantage of surgical closure of ventricular septal defects, where the patch is placed only on the right side of the septum, virtually eliminating this risk. Current device designs invariably depend on the left-sided disc for device anchor. Device designs need to be reviewed, keeping such possibilities of delayed conduction tissue trauma in mind, and device construction with alternate material needs to be explored.

Conclusions

To the best of our knowledge, this is the first report of late VSD device explantation from a perimembranous location for LBBB and resultant ventricular dysfunction. While transcatheter device closure of pm VSD seems attractive, with lesser morbidity, the procedure is not free of significant complications. Development of complete LBBB after pm VSD device closure is extremely rare and is probably more common with defects extending into the muscular septum or with abnormal bundle branch anatomy. There are currently no electrophysiologic tools to identify who might be at risk for such events. Our experience, though limited to this case, seems to suggest that late device explantation might still reverse LBBB and LV dysfunction, favorably altering the clinical course.

Appendix A Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.hrcr.2019.12.006>.

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