tion could occur because of the long-term use of anticoagulant therapy. Hence, in our patient, warfarin was responsible for the recanalization of the side branch. There is no case of recanalization of the unligated side branch of the LIMA after successful coil occlusion in patients who consumed warfarin owing to valve replacement.

The data is scarce regarding the treatment of recanalization of the side branch of LIMA in patients who consume warfarin. If the recanalization of the side branch was detected in these patients, different options may be considered such as vascular plugs, graft stents, gelatin sponge particles, combined drug-eluting and covered stents. As our patient’s LIMA diameter was relatively small for placing the vascular plug and graft stent, a coil reocclusion procedure was preferred. We used numerous large-sized coils to complete the occlusion of the vessel. The final angiographic view was satisfying with respect to total vessel occlusion.

Conclusion

Eventually after coil occlusion procedure, patients with admitted angina consuming warfarin should be considered in terms of recanalization. In cases when a coil occlusion procedure is preferred, the final result of procedure should be satisfactory in terms of total mechanical occlusion of the vessel using a coil rather than only stopping the flow.

References


Video 1. A large side branch originating from the left internal mammary artery and cessation of the side branch of left internal mammary artery flow with coil occlusion.

Video 2. Recanalization of the side branch of the left internal mammary artery, detachment of the proximal part of the coil attachment apparatus, implantation of the drug-eluting stent, and cessation of the side branch of left internal mammary artery flow with coil recocclusion.

Case Report

Transcatheter VSD closure was performed in the patient at the age of 3.5 years and weighed 15 kg. She had no significant medical problem other than VSD. Her ECG did not show any conduction abnormality. The size of the defect was measured to be 5.5 mm via transesophageal echocardiography (TEE). VSD was closed in a standard manner under the guidance of TEE and fluoroscopy. A 6-mm membranous VSD occluder (Amplatzer) was used. Hemodynamic measurements showed that the Qp/Qs ratio was 3 and the mean pulmonary artery pressure was 28 mm Hg. The intervention was uneventful, and there was only right bundle branch block (RBBB) without any atrioventricular conduction abnormality after the procedure. Transesophageal echocardiography (TEE) performed on the following day showed a complete closure of the defect with good device position (Fig. 1). Routine follow-ups were performed with ECG, TTE, and Holter monitoring at 1, 3, and 6 months as well as every 6 months after the procedure, thereafter. At her last follow-up visit, she was aged 7.5 years. Her ECG, TTE, and Holter monitoring did not show any abnormalities, except RBBB. She experienced a brief syncope episode at 51 months after the transcatheter VSD closure. She was urgently referred to our clinic because of significant bradycardia. Upon arrival, her ECG showed CAVB with a ventricular rate of 35/min (Fig. 2). Clinical studies showed no obvious reason for CAVB. Transvenous transient endocardial pacemaker was urgently placed and permanent endocardial pacemaker was implanted without any complication.

Discussion

A major concern for percutaneous perimembranous VSD closure is the risk of CAVB. The frequency of this alarming complication in
patient was reported to be 1%-5% (3-8). There may be an early occurrence of CAVB during the procedure. However, in most patients, CAVB develops days or even months after the intervention. The latest occurrence of CAVB in the published literature was 20 months after the procedure (5, 8). The development of CAVB is mainly related to the adjacency of the conduction system to the borders of the perimembranous VSD. Direct compression of the conduction system in early CAVB and fibrosis secondary to the inflammation provoked by the device in late CAVB has been suggested as possible mechanisms (5, 8). Although the VSD of the presented case was not perimembranous, it only had a muscular rim with a size of 2 mm separating it from the membranous septum. Therefore, the same mechanisms responsible for CAVB development may be accountable for CAVB development in this patient as well.

CAVB can also occur after surgical perimembranous VSD closure; however, its incidence in recent series is less than 1% in most centers (1, 9, 10). A recent multicenter study that involved the surgical perimembranous VSD closure of 4432 patients reported an incidence rate of 1.1% of CAVB requiring permanent pacemaker placement (9). Thus, the risk of surgical atrioventricular block is equal to or lower than the risk of transcatheter closure. Another important point that should be considered is that CAVB generally occurs early after operation in surgical patients; however, in percutaneously treated patients, the timing of CAVB development is completely unpredictable, and it is usually a late event (5, 8, 9).

Conclusion

The occurrence of CAVB after more than 4 years of intervention in our patient suggests that clinicians should be aware of the lifelong risk of CAVB development in patients undergoing percutaneous VSD closure, particularly those who had VSDs located near the membranous septum. The decision makers should think twice before percutaneous VSD closure, and the potential lifelong risk of CAVB should be considered when counseling families regarding options for VSD closure.

References