CASE REPORT

Amplatzer Septal Occluder-induced Transient Complete Atrioventricular Block

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Percutaneous transcatheter atrial septal defect (ASD) closure is a widely used technique that has replaced open-heart surgical closure in many centers. The most common implant is the Amplatzer septal occluder which seems to be a highly effective and safe device. However, there are reports of complications associated with its implantation. We report a 9-year-old boy who presented with complete atrioventricular block after undergoing percutaneous closure of a large secundum ASD with an Amplatzer septal occluder. We treated the patient with oral prednisolone. The patient's atrioventricular conduction improved to second-degree Mobitz type 1 block on post-procedure day 24 and first-degree block on day 35. We conclude that patients with Amplatzer septal occluder-induced complete atrioventricular block generally have a good outcome, although it may take several weeks for improvement. [*J Formos Med Assoc* 2007;106(12):1052–1056]

Key Words: atrial septal defect, complication, heart block

Secundum atrial septal defect (ASD) occurs in 3.78 per 10,000 live births.[1] Surgical repair is a well established procedure with a low mortality rate.[2] Since its first introduction in 1976,[3] however, percutaneous closure of secundum ASD with an Amplatzer septal occluder (ASO) has gained wide acceptance as an effective alternative to open-heart surgery.[2–5] The major advantage of transcatheter closure is that it avoids a median sternotomy and cardiopulmonary bypass. In addition, the complication rate with the ASO, although not negligible, is lower than with surgery.[2,5,6] Reported complications include arrhythmias, thrombus, embolization, pericardial effusion, cardiac perforation with tamponade, transient ischemic attack, cerebral embolism, and sudden death.[7–10] The 2% incidence of arrhythmia after ASO implantation is considerably lower than the 30% incidence reported after surgical closure.[2] Electrocardiographic abnormalities reported after transcatheter closure are usually asymptomatic[11] and have included premature atrial contraction, atrial flutter, atrial fibrillation, supraventricular tachycardia, ST elevation, and atrioventricular (AV) block.[3] Complete AV block (CAVB) is reported to occur in 0.35–2.5% of cases and is usually benign and transient.[1,6,8,10,11] We report a patient who had CAVB for 24 days following ASO implantation.

Case Report

A 9-year-old boy was incidentally found to have ASD. He did not complain of fatigue or dyspnea. He was 123 cm tall and weighed 21 kg. On auscultation of the heart, there was a widely fixed split S2 and a grade 2/6 systolic ejection murmur at the left mid-sternal border. The patient did not have a bulging or hyperdynamic precordium, nor was there a right ventricular heave. Chest X-ray showed a dilated right atrium without increased pulmonary vascularity. Electrocardiography showed

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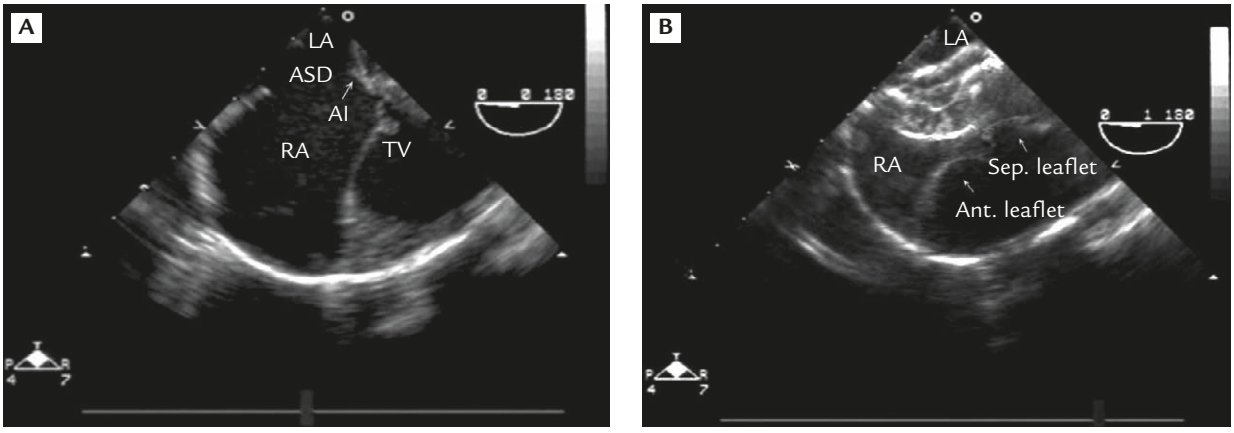

Figure 1. (A) Transesophageal echocardiography before Amplatzer septal occluder (ASO) implantation: apical four-chamber view shows a large secundum atrial septal defect with a limited anteroinferior rim measuring approximately 2 mm in length. (B) Although the right atrial disc of the ASO was seen touching the septal annulus of the tricuspid valve after occlusion, there was no significant tricuspid valve stenosis or insufficiency present. LA = left atrium; ASD = atrial septal defect; AI = anteroinferior rim; RA = right atrium; TV = tricuspid valve; Ant. = anterior; Sep. = septal.

a normal sinus rhythm with incomplete right bundle branch block. Transthoracic echocardiography showed a 2.74-cm secundum ASD and a bicuspid aortic valve. Left ventricular systolic function was borderline normal (ejection fraction, 0.53), and the right atrium, right ventricle, and main pulmonary artery were dilated. There was also mild pulmonary stenosis and moderate tricuspid regurgitation. Hemodynamic data obtained during cardiac catheterization revealed a step-up of O_2 saturation in the right atrium (91%) and main pulmonary artery (91%) compared to the superior (72%) and inferior (79%) vena cavae. Pulmonary arterial pressure was normal. The ratio of pulmonary to systemic blood flow (Qp/Qs) was 3.5. Contrast medium injected into the right upper pulmonary vein in the four-chamber view showed a large secundum ASD.

With consent from the patient's parents, we performed transesophageal echocardiography and implanted an ASO under general anesthesia with endotracheal intubation. The ASD had a limited anteroinferior rim (Figure 1A) and a deficient superoanterior rim, measuring 23 mm by echocardiography and 25 mm by Amplatzer balloon sizing. We therefore chose to implant a 28-mm ASO (AGA Medical Corp, Golden Valley, MN, USA), which was deployed under fluoroscopic and echocardiographic guidance. The left atrial disc was deployed in the left atrium and the waist in the defect itself. The right atrial disc was then deployed in the right atrium. Although the ASO, with the delivery cable still in place, was seen to be in the proper position, the electrocardiogram showed CAVB with an accelerated junctional rhythm of 80 bpm. We therefore retrieved the ASO, and the normal sinus rhythm returned. However, CAVB recurred after redeployment of the device. Although the right atrial disc of the ASO was seen to be touching the septal annulus of the tricuspid valve after occlusion, there was no significant tricuspid valve stenosis or insufficiency present (Figure 1B). We thought that a slightly smaller device (26-mm ASO) might be insufficient to close the ASD. In addition, the patient's junctional escape rhythm was around 75 bpm (Figure 2) and his CAVB might be transient, as has usually been reported. Therefore, we released the ASO.

After the procedure, however, he continued to have CAVB, which was initially associated with mild hypotension. Dopamine was therefore administered for the first 2 days. We thought the CAVB might have resulted from tissue edema and began oral prednisolone 40 hours after ASO implantation, starting with a dose of 1.4 mg/kg/day for 20 days, then tapered to 0.25 mg/kg/day for 7 days before discontinuation. The patient was also

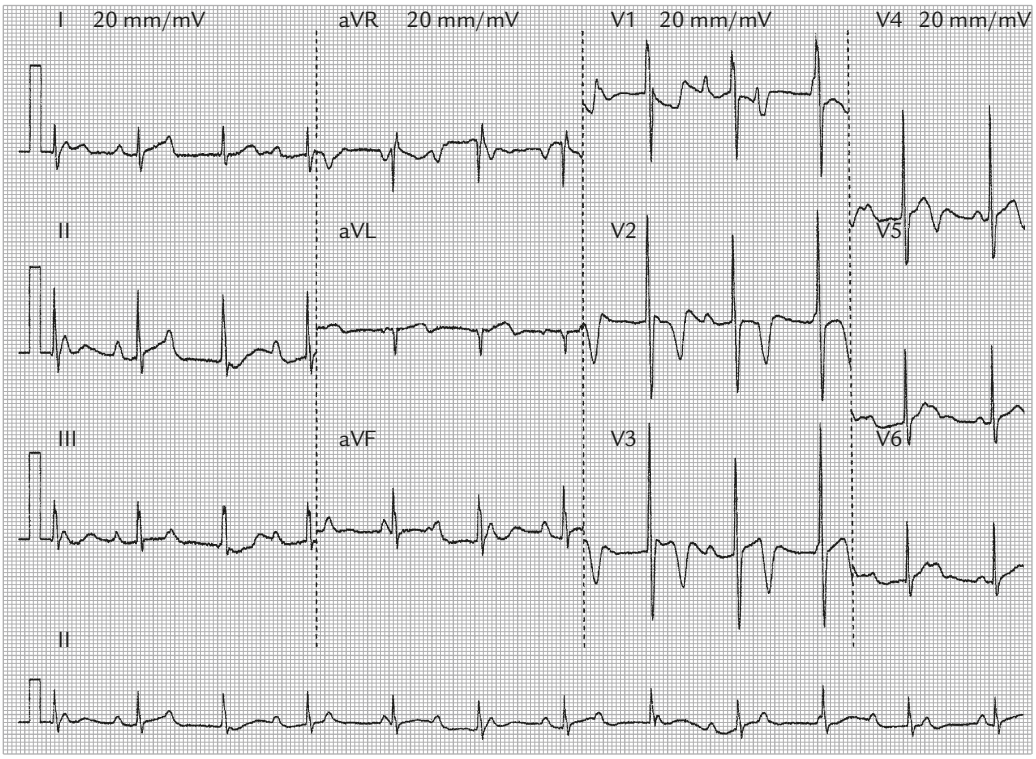

Figure 2. Electrocardiogram after the second deployment of the Amplatzer septal occluder shows atrioventricular dissociation with an atrial rate of 100 bpm and a ventricular rate of 75 bpm.

given aspirin, 100 mg once daily. He had no chest tightness or dyspnea at any time after the procedure. A 24-hour Holter study 4 days after the procedure showed CAVB with a mean junctional escape rate of 57 bpm (atrial rate, 93–136 bpm).

After discharge, the patient had serial electrocardiograms that showed gradual improvement in his AV conduction. On post-procedure day 24, he had Mobitz type I (Wenckebach) AV block (Figure 3) and on day 35, first-degree AV block with incomplete right bundle branch block (Figure 4). A 24-hour Holter study 6 months after the procedure showed recovery of sinus rhythm with first-degree AV block. Twelve months after the procedure, he still had first-degree AV block.

Discussion

In most reports, the CAVB following ASO implantation has resolved within 3 days. The longest previously reported duration of block was about 2 weeks. Our patient had CAVB for about 24 days, after which the block gradually improved over the next several months.

Precise measurement of the ASD so as to avoid placing an oversized device is important to minimize the risk of CAVB. In our patient, the balloon stretch diameter of the ASD was 25 mm, so we chose a 28-mm ASO to maximize the chance of ASD closure. Du et al recommended choosing an ASO equal in size or up to 2 mm larger than the balloon stretch diameter.[12] However, if the balloon stretch diameter is greater than 25 mm, an ASO 2–4 mm larger than the diameter has been reported to produce closure rates equivalent to those for smaller ASD occluded by a proportionately smaller ASO.[10] Chessa et al reported a patient with CAVB after implantation of an 18-mm ASO, with reversion to sinus rhythm when the ASO was replaced with a 14-mm device.[1] In a series of 197 patients reported by Wang and colleagues, only one patient developed CAVB for 3 days after ASO implantation. They used devices equal or only slightly larger (within 2 mm) than the ASD balloon stretch diameters.[8]

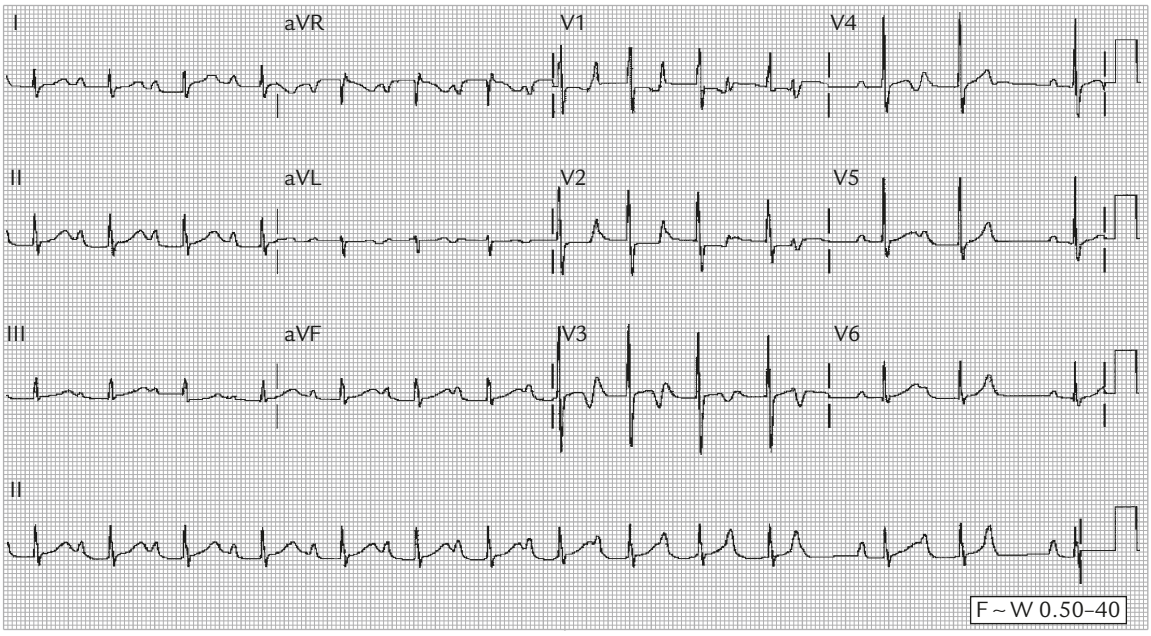

Figure 3. Electrocardiogram on post-procedure day 24 shows Mobitz type I (Wenckebach) atrioventricular block.

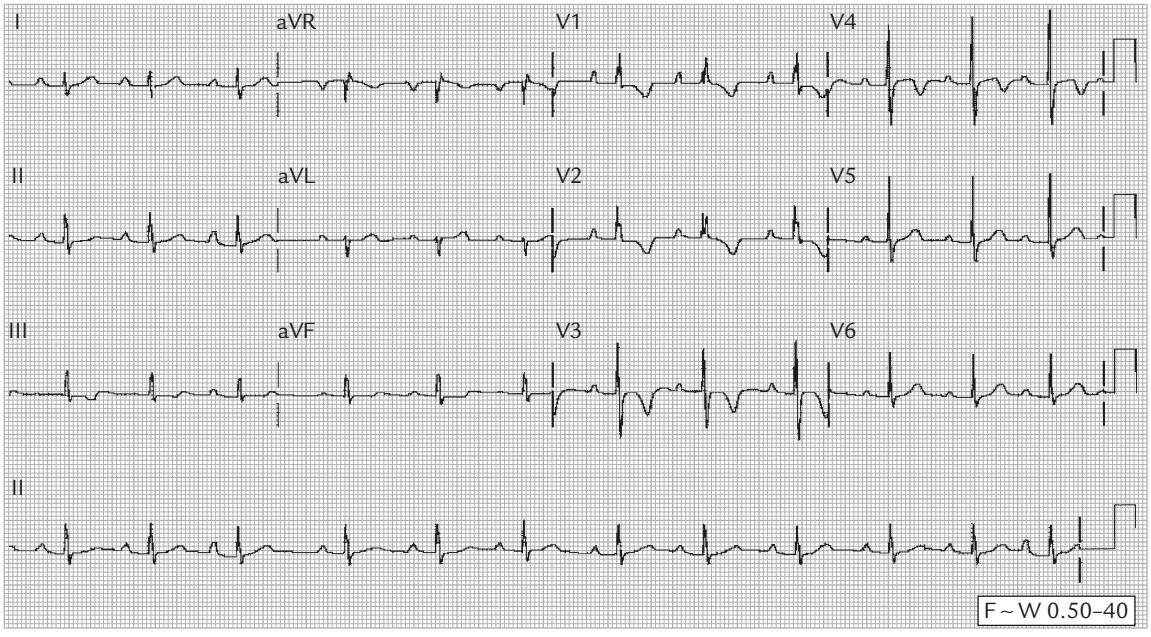

Figure 4. Electrocardiogram on post-procedure day 35 shows first-degree atrioventricular block, incomplete right bundle branch block and sinus tachycardia.

There may be factors other than just ASO size that are involved in the development of AV block. Suda et al reported that the device size, device/height ratio, and Qp/Qs were all significantly greater in patients who developed post-procedure AV block of any degree. In their study, patients with devices larger than 19 mm and a device/height ratio greater than 0.18 were at a higher risk of having procedure-associated CAVB.[4] Our patient had a high Qp/Qs and a device/height ratio of 0.22, which, by the results of Suda's group, put him at risk of developing AV block.

If second- or third-degree AV block does not spontaneously convert to 1:1 AV conduction within 30 minutes following implantation, it has been suggested that the device be retrieved and replaced

with a smaller ASO.[1,9] Our patient's CAVB resolved when we temporarily retrieved the device but recurred when it was reimplanted. However, he had a junctional escape rhythm with an adequate rate and we assumed that the CAVB would soon revert to normal. We therefore chose to leave the 28-mm ASO in place.

The mechanism causing conduction defects and rhythm disturbance after ASO implantation is unclear, although it is reasonable to hypothesize that it relates to the close proximity of the device to the AV node. A large ASO might compress the AV node, producing edema and inflammation of the adjacent cardiac tissue secondary to friction from the atrial discs. Assessment of the real spatial relationship between the atrial discs and the AV node by other methods, such as three-dimensional echocardiography, may yield more insight on this issue.[4]

There are no controlled studies supporting the use of corticosteroids to enhance AV node recovery after ASO implantation, so the treatment is empiric. High-dose intravenous steroids (2 mg/kg/day) followed by oral steroids for 3 weeks have been used empirically with apparently good results for AV block after transcatheter closure of a perimembranous ventricular septal defect.[13] In theory, high-dose steroid (2 mg/kg/day) and aspirin (100 mg/kg/day in 3 divided doses) ought to be effective in reducing inflammation and thereby normalizing conduction.[14] Our patient's response was not as rapid or complete as that in the aforementioned reports. His final outcome with only first-degree AV block, however, was good despite the time it took for improvement.

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