Case Reports

Late Bacterial Endocarditis in an Intravenous Drug User With an Amplatzer Septal Occluder

Michael S. La Sala, DO, MS
Hajir Zohourian, DO
Joseph McKeown, DO
Samuel Snyder, DO

Infective endocarditis of a fully endothelialized cardiac prosthesis, and especially the late presentation of endocarditis, challenges our current understanding of device-related complications. Late bacterial endocarditis associated with the Amplatzer Septal Occluder, a device frequently used to close atrial septal defects, has been documented only rarely. We report the case of an intravenous drug user who had late infective endocarditis associated with his Amplatzer Septal Occluder, secondary to methicillin-sensitive Staphylococcus aureus *bacteremia nearly 14 years after device insertion. The patient recovered after surgical excision and débridement of the vegetative mass, which may be the first time that a surgical approach has been taken to treat this condition. This report corroborates the need for late screening of high-risk patients who have septal occluder devices.* ***(Tex Heart Inst J 2020;47(4):311-4)***

Key words: *Cardiac surgical procedures; endocarditis, bacterial/complications/diagnosis/etiology; heart septal defects, atrial/pathology; prostheses and implants; prosthesis-related infections/diagnosis/surgery; risk factors; septal occluder devices/adverse effects; treatment outcome*

From: *College of Osteopathic Medicine (Drs. La Sala and Snyder), Nova Southeastern University, Davie, Florida 33328; and Departments of Cardiology (Dr. Zohourian) and Internal Medicine (Dr. McKeown), Graduate Medical Education, Broward Health Medical Center, Fort Lauderdale, Florida 33316*

Address for reprints: *Michael S. La Sala, DO, Graduate Medical Education, Broward Health Medical Center, 1600 S. Andrews Ave., Fort Lauderdale, FL 33316*

E-mail: *BHMCcasereport@gmail.com*

The Amplatzer Septal Occluder (ASO) (St. Jude Medical, an Abbott company) is chief among medical advances for repairing atrial septal defects (ASD) in the ostium secundum.[1] As this device approaches its 3rd decade on the market, sequelae associated with its use are appearing. Infective endocarditis (IE) is an extremely rare sequela of an ASD closure device, especially later than 6 months after implantation.[2] Incomplete device endothelialization is thought to be responsible[3-5]; however, this has not been fully substantiated, because of few reported cases and no long-term screening methods. In addition, intravenous drug use (IVDU) is the greatest risk factor for IE in patients who have heart devices.[6] The role of IVDU in ASO-related complications has not been studied. We report our experience with treating an intravenous drug user whose late IE was associated with an ASO.

Case Report

In February 2018, a 26-year-old man presented at our emergency department with a 2-day history of left peripatellar pain and swelling. At age 6 years, he had undergone percutaneous closure of a secundum ASD with use of the buttoned device.[7] Seven years later, a transesophageal echocardiogram (TEE) revealed that the fabric of the buttoned device had torn, leaving a 6-mm central gap that created a left-to-right cardiac shunt. The buttoned device was percutaneously replaced with an ASO. The new defect was sized with a balloon, and an ASO (waist size, 8 mm) was introduced through the center of the old device. The 2 occluder discs engulfed and enclosed the existing device, leaving no residual shunt.

When the patient was admitted to our institution almost 14 years later, he was tachycardic but had no fever or leukocytosis. He had no splinter hemorrhages, Janeway lesions, or audible murmurs. He had been taking drugs intravenously for one year, and his urine toxicology results were positive for benzodiazepine and cocaine. Polymerase chain reaction tests detected methicillin-sensitive *Staphylococcus aureus* in joint aspirate and blood cultures.

A TEE showed a freely oscillating, windsock-like vegetative mass (area, 2.25 cm^2) on the left atrial septal wall, attached by a stalk to the peripheral wiring of the patient's ASO (Fig. 1A–B). No vegetation on the valve was noted. We prescribed 2 weeks of antibiotic therapy with rifampin, gentamicin, and nafcillin. Three days after therapy began, blood cultures showed no microorganism growth, and the regimen

was changed to nafcillin alone for 6 weeks. A TEE then showed that the mass had shrunk to 1 cm^2 but had not resolved (Fig. 1C), so surgical removal was scheduled.

At surgery, the ASO was grossly intact without areas of incomplete endothelialization. The mass, attached to the peripheral portion of the occluder, was excised at the lowest possible point, and the device was preserved. On gross examination, the mass was pink with a thin stalk and a smooth external surface. Histologic analysis revealed a dense thrombotic formation of mixed chronic and acute inflammatory cell infiltrate surrounded by endothelium (Fig. 2). Nafcillin was continued for 6

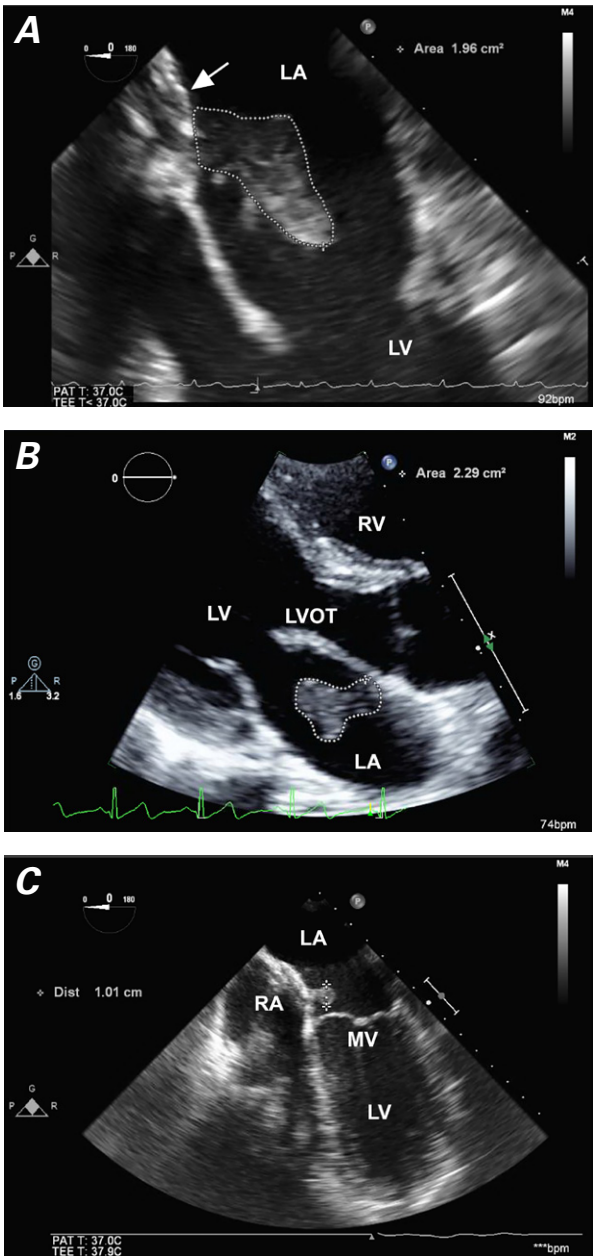

Fig. 1 *At presentation, a large vegetative mass (outlined) was seen on* ***A****) transesophageal and* ***B****) transthoracic echocardiograms. In* ***A****), the Amplatzer Septal Occluder (arrow) is shown.* ***C****) After 6 weeks of antibiotic therapy, a transesophageal echocardiogram shows a pedunculated mass remnant.*

LA = left atrium; LV = left ventricle; LVOT= left ventricular outflow tract; MV = mitral valve; RA = right atrium; RV = right ventricle

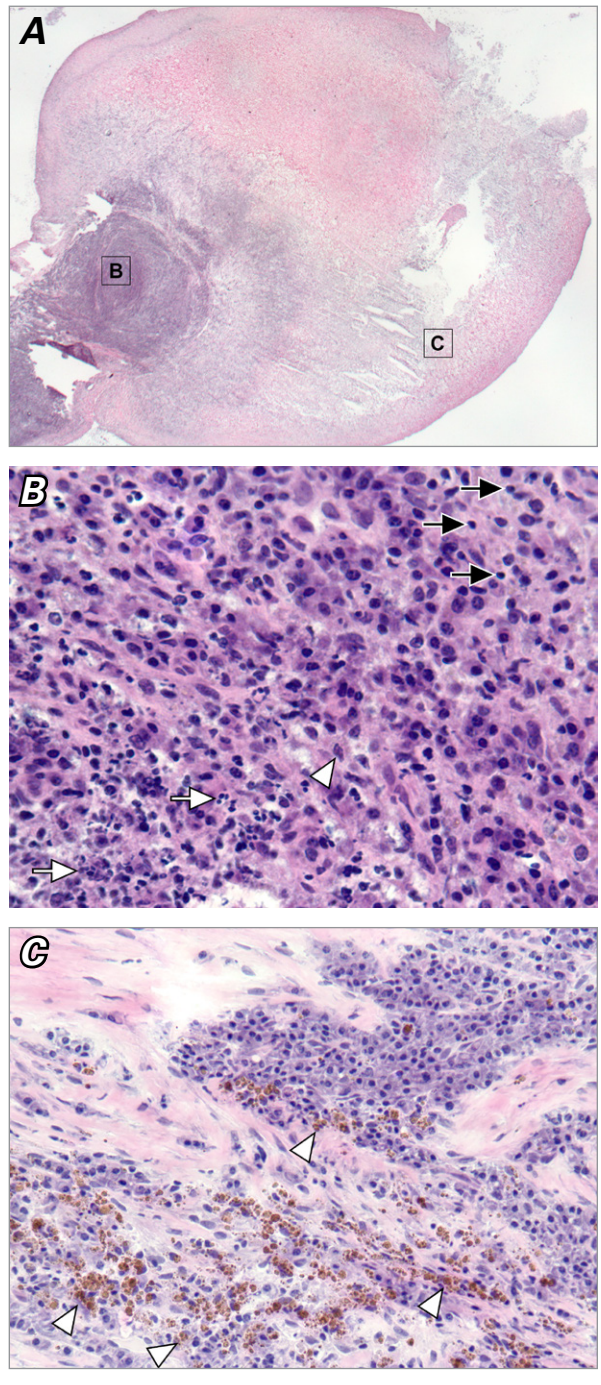

Fig. 2 *Photomicrographs.* ***A****) Cross-sectional image shows a pedunculated vegetative mass (H & E, orig. ×2.5). Labels B–C correspond to the regions magnified in* ***B****)* and ***C****).* ***B****) The core of the mass contains abundant dense fibrin lymphocytes (black arrows), neutrophils (white arrows), and scarce histiocytes (arrowhead) (H & E, orig. ×40).* ***C****) The periphery of the mass shows dense fibrin and golden-brown hemosiderin-laden macrophages throughout (arrowheads) (H & E, orig. ×20).*

weeks, and the patient recovered uneventfully before his discharge from the hospital. A transthoracic echocardiogram at his one-month follow-up appointment showed no residual mass or new growth (Fig. 3). Blood cultures were free of organisms, as were an acid-fast bacilli smear and fungal and bacterial cultures of the excised vegetation. The patient was lost to follow-up thereafter.

Discussion

To our knowledge, 14 years is the longest interval between ASO implantation and diagnosis of IE. Ours is also the first report of using surgical excision and débridement to treat an ASO-associated vegetation caused by bacterial endocarditis.

The ASO has been used to repair various hole defects, such as a ruptured sinus of Valsalva or Fontan fenestrations.[8] Given the location and size of our patient's residual shunt, the use of an ASO to replace the failed device was highly effective. However, whether the replacement ASO increased his risk of IE is not clear.

Our literature review revealed 24 reports of ASD-related bacterial endocarditis, 18 of which involved an ASO.[9,10] Late bacterial endocarditis is defined as infection occurring after implantation and at least 6 months after prophylactic antibiotic therapy has been completed.[10-12] In preliminary studies in pigs, 100% endothelial coverage was observed within this time frame.[13] Presumably, complete device endothelialization prevents future endocarditis; however, incomplete or no endothelialization may persist.[3-5] Therefore, the extent of endothelial coverage can vary among patients. No studies or established screening methods are available to determine what percentage of complete endothelialization effectively protects against infection.

Full endothelialization was reported in 3 of the 18 previous cases of ASO-associated IE.[14,15] Krantz and Lawton[15] described IE affecting a patient's endothelialized ASO after a prolonged urinary tract infection from disseminated methicillin-resistant *S. aureus*. Gross and histologic examination of our patient's ASO revealed endothelial tissue extending over the mass and device and continuing along the septum. Given our patient's history and clinical course, his recent systemic methicillin-sensitive *S. aureus* infection probably caused the endocarditis. This suggests that possible bacterial seeding of a completely endothelialized device should be considered in cases of ASO-associated IE.

The preferred treatment for ASO-associated IE is to remove the device and replace it with a homologous patch.[4,5,9,10,12,14,15] Other treatments have also been suggested. Aruni and colleagues[11] showed that, absent device failure, antibiotic therapy alone was sufficient to resolve a vegetative mass. In our patient, prolonged antibiotic therapy was not completely effective. His IVDU led us to débride the ASO surgically. He recovered uneventfully, and no vegetation was evident on follow-up.

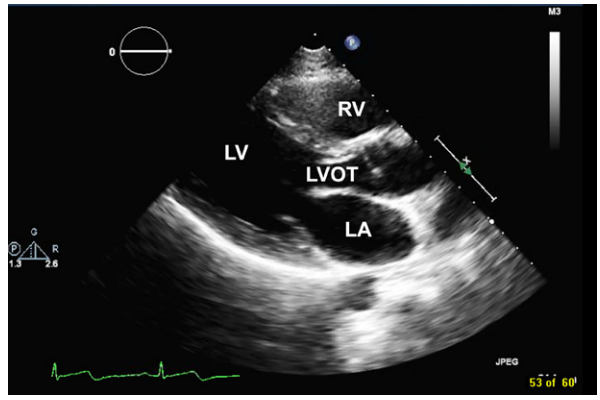

Fig. 3 *Transthoracic echocardiogram (parasternal long-axis view) one month after excision shows no mass.*

LA = left atrium; LV = left ventricle; LVOT= left ventricular outflow tract; RV = right ventricle

The American Heart Association recommends a 6-month course of prophylactic antibiotics to prevent endocarditis in patients who have ASD closure devices.[16] These guidelines presume complete endothelialization of the device after 6 months and give no recommendations for long-term follow-up care. Furthermore, guidance for managing incomplete endothelization is limited. On the basis of our case and recent reports, we recommend placing greater emphasis on preventive care for patients who have ASD closure devices. Performing TEE is warranted in the presence of bacteremia, and elective TEE should be considered in asymptomatic patients who exhibit high-risk behavior after device insertion.

References

1. Bissessor N. Current perspectives in percutaneous atrial septal defect closure devices. Med Devices (Auckl) 2015;8:297-303.
2. Jalal Z, Hascoet S, Baruteau AE, Iriart X, Kreitmann B, Boudjemline Y, Thambo JB. Long-term complications after transcatheter atrial septal defect closure: a review of the medical literature. Can J Cardiol 2016;32(11):1315.e11-e18.
3. Nguyen AK, Palafox BA, Starr JP, Gates RN, Berdjis F. Endocarditis and incomplete endothelialization 12 years after Amplatzer Septal Occluder deployment. Tex Heart Inst J 2016;43(3):227-31.
4. Bialkowski J, Pawlak S, Banaszak P. Incomplete endothelialisation of an Amplatzer Septal Occluder device followed by meningitis and late acute bacterial endocarditis. Cardiol Young 2016;26(4):808-10.
5. Divchev D, Podewski EK, Mengel M, Meyer GP, Drexler H, Schaefer A. Inflammatory, abscess-forming foreign body reaction mimics a thrombus formation on an atrial septal defect closure device: a commented case report. Eur J Echocardiogr 2007;8(4):298-302.
6. Pant S, Patel NJ, Deshmukh A, Golwala H, Patel N, Badheka A, et al. Trends in infective endocarditis incidence,

microbiology, and valve replacement in the United States from 2000 to 2011. J Am Coll Cardiol 2015;65(19):2070-6.
7. Sideris EB, Sideris SE, Thanopoulos BD, Ehly RL, Fowlkes JP. Transvenous atrial septal defect occlusion by the buttoned device. Am J Cardiol 1990;66(20):1524-6.
8. Awasthy N, Tomar M, Radhakrishnan S, Shrivastava S. Unconventional uses of septal occluder devices: our experience reviewed. Indian Heart J 2015;67(2):128-35.
9. Amedro P, Soulatges C, Fraisse A. Infective endocarditis after device closure of atrial septal defects: case report and review of the literature. Catheter Cardiovasc Interv 2017;89(2):324-34.
10. Toporcer T, Kolesar A, Ledecky M, Sabol F. Late infective endocarditis of an Amplatzer atrial septal device twelve years after implantation. Cor et Vasa 2018;60(2):e174-8.
11. Aruni B, Sharifian A, Eryazici P, Herrera CJ. Late bacterial endocarditis of an Amplatzer atrial septal device. Indian Heart J 2013;65(4):450-1.
12. Zahr F, Katz WE, Toyoda Y, Anderson WD. Late bacterial endocarditis of an Amplatzer atrial septal defect occluder device. Am J Cardiol 2010;105(2):279-80.
13. Han YM, Gu X, Titus JL, Rickers C, Bass JL, Urness M, Amplatz K. New self-expanding patent foramen ovale occlusion device. Catheter Cardiovasc Interv 1999;47(3):370-6.
14. Balasundaram RP, Anandaraja S, Juneja R, Choudhary SK. Infective endocarditis following implantation of Amplatzer atrial septal occluder. Indian Heart J 2005;57(2):167-9.
15. Krantz SB, Lawton JS. Subacute endocarditis of an atrial septal closure device in a patient with a patent foramen ovale. Ann Thorac Surg 2014;98(5):1821-3.
16. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP 3rd, Guyton RA, et al. 2014 AHA/ACC Guideline for the Management of Patients with Valvular Heart Disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines [published erratum appears in Circulation 2014;129(23):e650]. Circulation 2014;129(23):2440-92.