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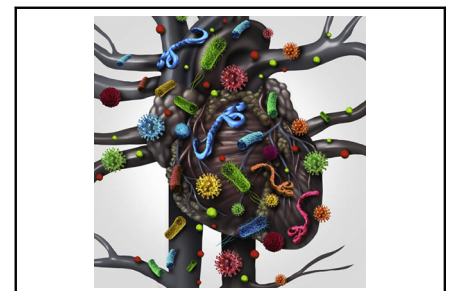


Commentary: Don't leave patients to their own devices: Consider long-term complications after percutaneous atrial septal defect closure

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Percutaneous device closure of atrial septal defects (ASDs) was introduced in the late 1990s as a feasible alternative to surgical repair.¹ Since then, several studies have shown that short-term outcomes and complication rates after percutaneous ASD closure are noninferior to those after surgery.^{2,3} However, an important consideration with percutaneous ASD closure is the prevalence of late complications that have come to light after its widespread adoption. Some of these, including cardiac erosion, thromboembolism, and infective endocarditis, carry significant morbidity and mortality. Kitamura and colleagues⁴ present a case report of a 20-year-old man with atopic dermatitis and a secundum ASD who was treated with Amplatzer device (Abbott, Abbott Park, Ill) closure and developed endocarditis with large mobile vegetations 3 years later. The authors concluded that ASD closure devices are associated with risks of thrombotic and infectious complications over the long-term.⁴

Kitamura and colleagues⁴ should be commended for publishing this interesting report. They provide context for their work and discuss a complication which holds relevance for



Infective endocarditis is an important late complication after ASD device closure.

CENTRAL MESSAGE

Patients undergoing percutaneous closure of atrial septal defects require close monitoring for late complications such as infective endocarditis, cardiac erosion, and valve damage.

primary care providers, interventional cardiologists, and cardiovascular surgeons alike. In addition, the authors describe well the preoperative findings and operative techniques used to address the lesion. However, additional details about the medical and social history of the patient, such as comorbidities and history of intravenous or inhaled substance use, could possibly elucidate additional characteristics of the high-risk patient. Furthermore, the authors do not quantify from either their personal experience or literature review the prevalence of ASD device complications.

It is intriguing that several case reports describe the same complication of late endocarditis after ASD device deployment.⁵⁻⁷ As Kitamura and colleagues⁴ describe, a likely explanation for this pathology is poor endothelialization of the device and resultant exposure of bare prothrombotic metal that could also provide a surface for bacterial adhesion. Although endocarditis is relatively rare after ASD device placement, with approximately 20 cases reported in the literature to date, it carries significant morbidity and mortality because more than 85% of these patients required surgery and 11% experienced operative mortality.⁸ Other analyses have reported several other long-term complications after ASD device placement, such as cardiac erosion, nickel hypersensitivity, and valve damage, all of which often present suddenly and are potentially lethal.⁹ Although there are no differences in short-term complication rates between surgical and device ASD

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closure,³ similar sudden late complications after surgery are relatively rare.

Although percutaneous ASD closure has become the preferred treatment at many centers due to the shorter recovery and less invasiveness, it is important to carefully follow patients long-term after ASD device closure. This is especially important for those vulnerable patients with underlying immunocompromise, syndromic comorbidities, or metabolic derangements. In addition, patients with risk factors for endocarditis certainly may be better served with a traditional surgical repair, which remains the gold standard for ASD closure.

Kitamura and colleagues⁴ should be commended for their contribution to the literature on a late complication of percutaneous ASD closure. Indeed, after undergoing this procedure, patients should not be left to their own devices!

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