Ultra-long-term histopathological workup of Amplatzer muscular septal defect occluders after surgical removal of the heart due to heart transplantation

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Information on the *in vivo* biocompatibility and tissue reaction to nitinol after applying Amplatzer occluders is limited. There is also lack of data on whether the endothelialization process of the device in the atria and ventricle is different. In the literature, we have not found descriptions of the long-term reaction of the cardiac endothelium after muscular device ventricular septum closure.

We present an interesting case of an 18-year-old woman who underwent pulmonary banding in infancy. This patient also had closure of multiple muscular ventricular septal defects (VSD) with two Amplatzer muscular occluders (AMO) in early childhood during 2 sessions (the second procedure was a hybrid procedure with surgical debanding performed at the age of two). Echocardiographic images have shown two AMOs closing VSDs in different parts of the interventricular septum. This patient was included in our analysis of long-term outcomes of transcatheter VSD closure, which were recently published [1].

In the last few years, this patient experienced increasing symptoms of heart failure related to cardiomyopathy. A magnetic resonance study (2022) confirmed the diagnosis of restrictive cardiomyopathy. The patient also suffered from atrial fibrillation, which was successfully treated with cardioversion and pharmacologically with propafenone. Unfortunately, her medical treatment was ineffective; therefore, she was qualified for the orthotopic heart transplantation. The operation was performed uneventfully in March 2024. The post-surgical period was also without any complications, and the patient remained in good, stable clinical condition (as of August 2024).

Visualization of AMO indicated complete neo-endothelialization of both devices without any inflammatory response and calcifications (Figure 1A–C). It should also be pointed out that the cross-section of the entire occluder was evaluated histologically. The photograph in Figure 1C shows a representative part of the occluder, as is accepted practice in histopathology.

The histopathological specimen of the explanted heart confirmed the clinical diagnosis of secondary restrictive cardiomyopathy due to endocardial fibrosis (Figure 1D).

Evaluation of device neo-endothelialization after transcatheter closure of atrial septal defect (ASD) is very rare [2]. The major consequence of incomplete neo-endothelialization of the device is the possibility of thrombus formation and/or bacterial endocarditis. We have found no publications describing the neo-endothelialization process in situations other than ASD in explanted human hearts. In our series of more than 2000 cases of percutaneous closures of interatrial communications, we observed one case of incomplete endothelialization after ASD, followed by late meningitis and acute bacterial endocarditis 2 years after the procedure. Consequently, this device was surgically removed [3]. Recently, incomplete neo-endothelialization was identified in 35% of ASD patients after 6 months of device implantation by cardiac computed angiography [4].

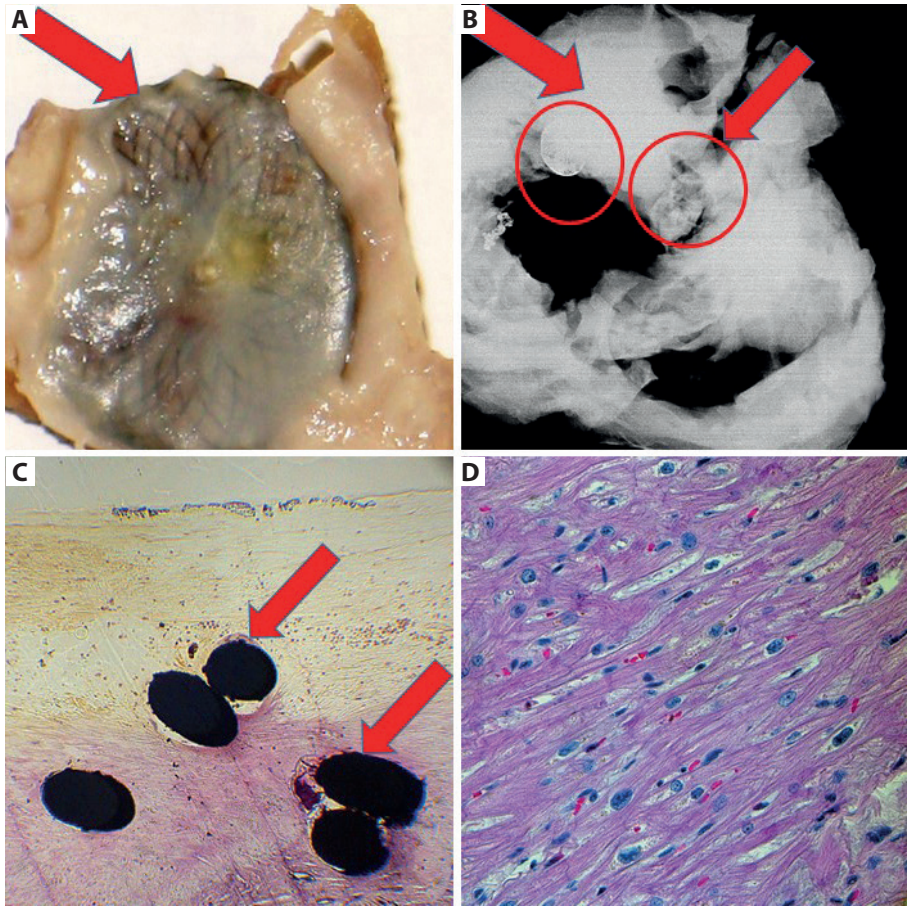

Figure 1. Amplatzer muscular septal occluders in the explanted heart. **A.** Excised device (red arrow) covered by thick endothelium. **B.** X-ray image with 2 implants (red circles and arrows). **C.** Histopathology of the endo- and myocardium — red arrows indicate nitinol struts of the device. **D.** Myocardium (histopathology examination, magnification × 150) with signs of hypertrophy and cardiomyopathy

We can speculate that percutaneous closure of multiple muscular VSD in early childhood in our patient protected her from developing pulmonary hypertension and was a bridge to successful heart transplantation.

In conclusion, this case demonstrates complete neo-endothelialization coverage of the surface and entire cellular organization of the tissue between the polyester fibers within an Amplatzer muscular ventricular septal occluder, without any thrombus, inflammatory reaction, or calcification 16 years after AMO implantation.

Article information

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