Reimplantation of a tricuspid valve bioprosthesis due to its thrombosis and recurrence of infective endocarditis

Anna Olasińska-Wiśniewska, Bartłomiej Perek, Sławomir Katarzyński, Hanna Wachowiak-Baszyńska, Paweł Marcinkowski, Marcin Ligowski, Marek Jemielity

Department of Cardiac Surgery and Transplantology, Poznan University of Medical Sciences, Poznań, Poland

A 21-year-old woman, a former drug addict, was admitted for urgent cardiac surgery, presenting with severe heart failure (New York Heart Association functional class IV) due to tricuspid bioprosthesis dysfunction. Two months earlier in another hospital, she had undergone mitral valve replacement with a 25-mm Edwards Perimount valve (Edwards Lifesciences Corp., Irvine, California, United States) and tricuspid valve (TV) replacement with a 29-mm Edwards Perimount valve (Edwards Lifesciences Corp.) due to methicillin-sensitive *Staphylococcus aureus* infective endocarditis. She was adequately treated with intravenous antibiotic therapy for 5 weeks following the surgery. International normalized ratio (INR) values were poorly controlled owing to lack of regular pharmacotherapy. Although the patient was treated with intravenous heparin, progressive deterioration was observed. Transthoracic (TTE) and transesophageal echocardiography demonstrated mitral periprosthetic leak and tricuspid prosthesis obstruction with a large thrombus impairing the motion of the leaflets (FIGURE 1A–1C; Supplementary material, *Videos S1* and *S2*). Intraoperatively, debridement of the tricuspid bioprosthesis turned out to be impossible; therefore, bioprosthesis replacement was performed. Unfortunately, signs of persistent infection were also found. Macroscopically, tissues adjacent to the tricuspid valve were not completely healed. Moreover, an emptied abscess cavity was noted and debrided. A mitral paravalvular leak without local signs of active infection was sealed with 2 pledgetted 2-0 Ticron sutures. Antibiotic therapy was initiated. Microbiological cultures of the explanted prothesis yielded negative results. In the postoperative period, a significant clinical improvement was achieved. However, on postoperative TTE, a small ventricular septal defect with an insignificant left-to-right shunt was detected and then confirmed by cardiac magnetic resonance imaging. The patient was discharged and referred to the rehabilitation hospital with a recommendation to present for regular checkups, which she unfortunately missed. The woman reported low INR values despite the use of increased oral anticoagulant doses, which most probably resulted from noncompliance to pharmacotherapy. However, she refused further hospitalization and was lost to contact.

Cardiac surgery is a rare option for the treatment of TV endocarditis. If necessary, repair is a preferred treatment of choice. Although several authors reported similar long-term survival rates after TV repair and replacement, longer freedom from recurrent IE and reoperation encourages the repair.[1,2] It is strongly recommended that surgeons remove a vegetation and repair the valve whenever possible instead of implanting any artificial materials into the actively infected tissues. Additionally, thrombosis of TV prostheses occurs in 20% of cases[3] and may result from their thrombogenic surface, inadequate anticoagulation, numerous turbulences, and lower pressures on the right side, with slower transprosthetic flow.[1,3] Of note, any kind of prosthesis should be particularly avoided when a history of drug abuse and labile INR is known, since recurrence is often observed.[1,4,5] In our

Correspondence to:
Anna Olasińska-Wiśniewska, MD, PhD, Department of Cardiac Surgery and Transplantology, ul. Długa1/2, 61-848 Poznań, Poland, phone: +48 61 854 92 10, email: annaolasinska@ump.edu.pl
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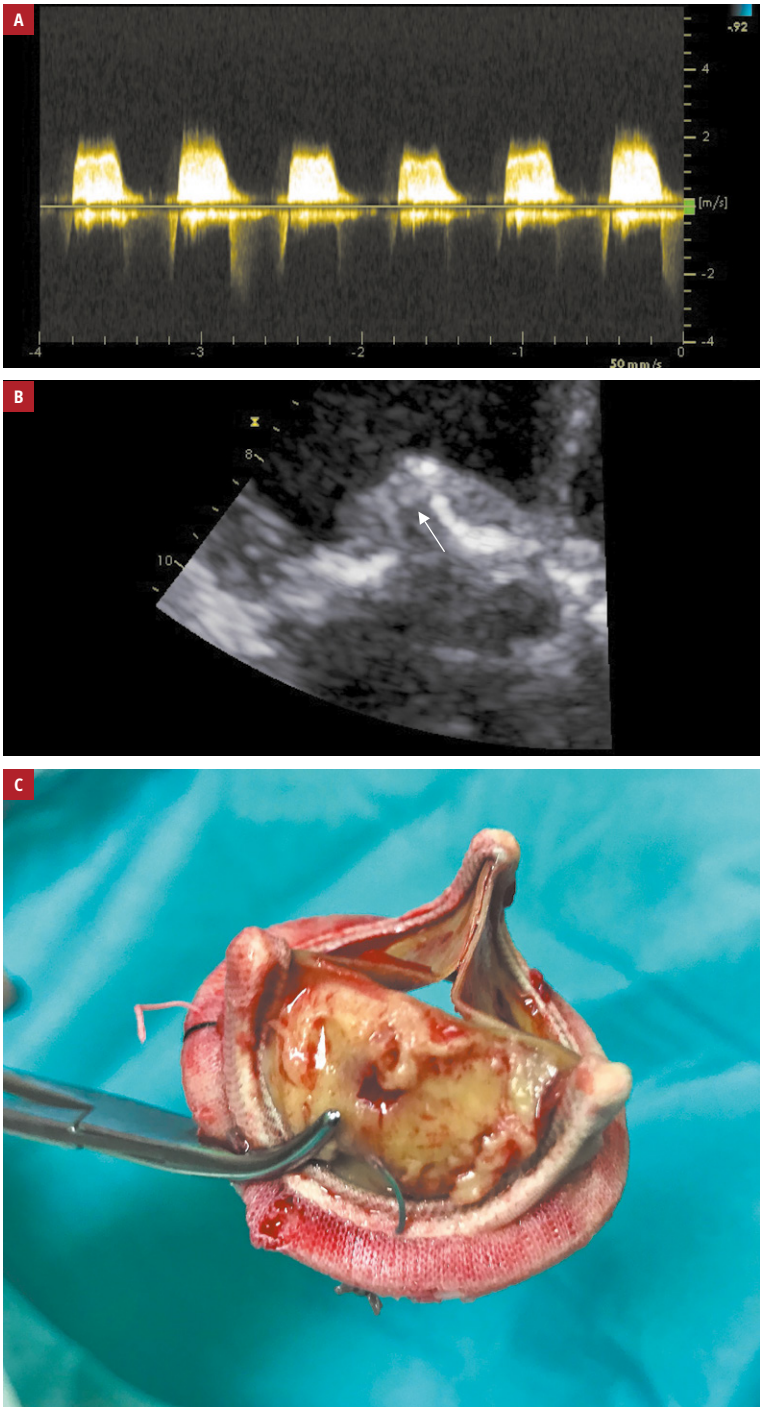

FIGURE 1 **A** – continuous transthoracic Doppler echocardiography showing a high transvalvular gradient caused by severe tricuspid prosthesis obstruction; **B** – transthoracic echocardiography demonstrating tricuspid prosthesis obstruction with a large thrombus (arrow) impairing the motion of the leaflets; **C** – intrasurgical view: the excised tricuspid valve bioprosthesis

patient's case, massive obstructive thrombosis resistant to high-dose heparin treatment prompted the decision to perform surgical reintervention. Moreover, previous valve replacement due to complete destruction of the remnants of the native TV forced the reimplantation of bioprosthesis during repeat surgery.

Ventricular septal defect found postoperatively in our patient was not visible on serial preoperative TTE. It could be caused by smoldering active infection (findings from reintervention) covered by one of the struts of the implanted, almost completely thrombosed bioprosthesis or hidden in turbulences of a severely stenotic TV. According to the current guidelines, patients with a history of endocarditis should undergo ventricular septal defect closure. In our patient, given the short time after the second cardiac surgery and the presence of an insignificant shunt, we decided to postpone the closure.

SUPPLEMENTARY MATERIAL

Supplementarwy material is available at www.mp.pl/kardiologiapolska.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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