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Clinical Case Report

Cardiac arrest due to acute massive aortic root thrombosis after pericardial bioprosthetic aortic valve replacement☆

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ABSTRACT

Acute aortic root thrombosis extended to coronary ostia is a rare but potentially life-threatening complication of aortic valve replacement with bioprosthetic substitutes. We aimed to present the case of a 72-year-old woman with symptomatic rheumatic valve disease and associated atrial fibrillation who underwent conventional mitroaortic valve replacement with two stented bioprostheses (pericardial and porcine, respectively). Eight days after surgery, she had cardiac arrest due to ventricular fibrillation, requiring immediate cardiopulmonary resuscitation. Left ventricle akinesia by echocardiography and troponin levels up to 35,000 ng/L pointed to coronary ischemia. Emergent coronary angiography showed a subocclusion of the left main trunk, with the suspicion of aortic root thrombosis. The patient was immediately reoperated, fresh thrombi were removed from the aortic root, and the aortic Magna Ease 21-mm bioprosthesis was replaced with a stentless Solo Smart 21-mm bioprosthesis. The patient died of septic complications.

1. Introduction

In older patients with valvular disease, bioprostheses are preferred over mechanical valves because of the advantage of avoiding chronic oral anticoagulation and its related complications. However, available biological substitutes can degenerate or develop late prosthetic thrombosis, which may lead to the need for anticoagulation or reoperation [1]. Acute massive thrombosis is a rare finding, but it is a life-threating condition, requiring immediate clinical suspicion and subsequent emergent treatment.

2. Case presentation

We describe the case of a 72-year-old woman (body mass index 26.7 kg/m^2) with a known history of mitroaortic rheumatic disease, with associated persistent atrial fibrillation on oral anticoagulation therapy. In the last year before hospital admission, the patient developed worsening dyspnea (NYHA II–III) due to valve function deterioration. Despite a mitral valve percutaneous valvuloplasty, she was finally scheduled for surgical replacement because of severe mitroaortic stenosis. The preoperative diagnostic examinations revealed a biventricular preserved function with no coronary artery lesions.

Through a full sternotomy, under bicaval-aortic cardiopulmonary bypass (CPB) at 32°C, the patient underwent mitral and aortic valve replacement with Biocor Epic 31-mm prosthesis (St. Jude Medical, Minneapolis, MN, USA) and Magna Ease 21-mm prosthesis (Edwards Lifesciences, Irvine, CA, USA), respectively, and left atrial appendage closure.

The immediate postoperative course was uneventful except for two isolated episodes of tonic–clonic seizures on postoperative day (POD) #0 without evidence of ischemic and/or hemorrhagic lesions at the head computed tomography. She was extubated on POD #3, and she was transferred to the floor on POD #5, where she got well in the first following 3 days except for high-grade fever (up to 38°C) and inadequate rate control of her known atrial fibrillation (for which she was anticoagulated by means of subcutaneous heparin). The postoperative echocardiogram showed a normal function of both bioprostheses, mild pericardial effusion, and a no significant subaortic dynamic gradient.

On POD #8, she suddenly fainted. Ventricular fibrillation was detected at the immediate electrocardiogram monitoring, and cardiopulmonary resuscitation maneuvers were started. The patient was intubated, and despite immediate starting of pharmacologic inotropic support, the clinical evidence of a low cardiac output syndrome imposed emergent percutaneous femorofemoral venoarterial extracorporeal membrane oxygenation (ECMO) assistance. The transesophageal echocardiogram (TEE) performed immediately after cardiac arrest showed severe left ventricle

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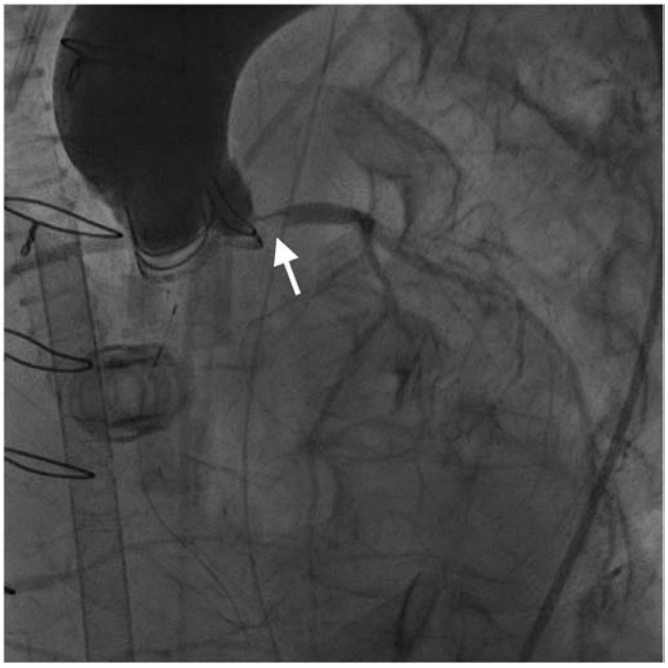

Fig. 1. Emergency coronary angiography performed soon after cardiac arrest showing the left main trunk occlusion (white arrow).

(LV) dysfunction without any other abnormalities. The significant increase in troponin levels acutely after the event (>35,000 ng/L) was linked to coronary ischemia. Coronary angiography performed a few hours after the acute complication found a subocclusion of the left main trunk, with the suspicion of aortic root thrombosis, especially above the noncoronary and left coronary sinus (Fig. 1, Video 1). Consequently, the patient was emergently reoperated. Intraoperative TEE found aortic root thrombosis, with also evidence of thrombotic material in the LV and left atrium. After a resternotomy, under central CPB at 32°C, the previous aortotomy was reopened, and fresh thrombi were removed from the aortic root and from the left coronary ostium with thrombotic material attached to the aortic prosthesis. Removal of the remaining thrombi across the aortic valve and through the previous left atriotomy was also required. No signs of mitral bioprosthesis thrombosis were found intraoperatively. The thrombosed aortic prosthesis valve was replaced with a 21-mm size stentless Solo Smart 21-mm bioprosthesis (Sorin Group, Saluggia, Italy). ECMO was maintained, and a right pulmonary vein venting cannula was positioned to decompress the dysfunctional LV. Unfortunately, despite initial hemodynamic stability, the patient developed septic shock and died 14 days after the emergent reoperation. The explanted prosthetic valve showed macroscopic signs of thrombosis, especially upon the left coronary cusp (Fig. 2A–C). Microbiological cultures from material of the explanted bioprosthesis were negative.

3. Discussion

Even if aortic valve replacement with biological prostheses is the treatment of choice in older patients, there are still many concerns linked to disadvantages of this type of valve substitute. Leaflets degeneration is the most frequent complication. Symptomatic bioprosthetic thrombosis is an uncommon finding, and most of the available reports describe it as a late finding [2]. Early acute and symptomatic thrombosis is anecdotic, especially after aortic valve replacement with pericardial bioprostheses: in fact, there are a very limited number of reported cases both after transcatheter aortic valve implantation (TAVI) [3] and after conventional surgical replacement [4–7].

Although, in this report, the patient presented several risk factors for thromboembolic complications (e.g., atrial fibrillation, biatrial enlargement, postoperative hypercoagulability status, low international normalized ratio range), there are no predisposing factors universally recognized by international guidelines in order to prevent this sequelae. Moreover, immediate diagnosis of acute valve thrombosis is still challenging because of a lack of awareness of its existence, especially in this case where aortic thrombosis was suspected a few hours after the acute clinical emergency.

It is arguable (and not yet reported) if the positioning of a small-size stented prosthesis (with postoperative near-normal gradients) might have created abnormal flows in the aortic root, triggering the cascade of thrombus formation. According to the most recent evidences (especially in the subset of TAVI [8]), several factors (e.g., residual root calcifications, aortic root stiffness, prosthesis–patient mismatch) may promote abnormal flow patterns in the aortic sinuses. The subsequent wall shear stress above the cusps and the sinuses and associated blood stasis favor endothelial damage and consequent platelet adhesion, activation, and aggregation [9].

Precise mechanisms underlying increased thrombotic risk are not known yet, but it is likely that complex patient- and prosthetic-related factors are involved.

4. Conclusions

Acute pericardial bioprosthetic thrombosis in aortic position is an unexpected complication in the absence of well-established predisposing

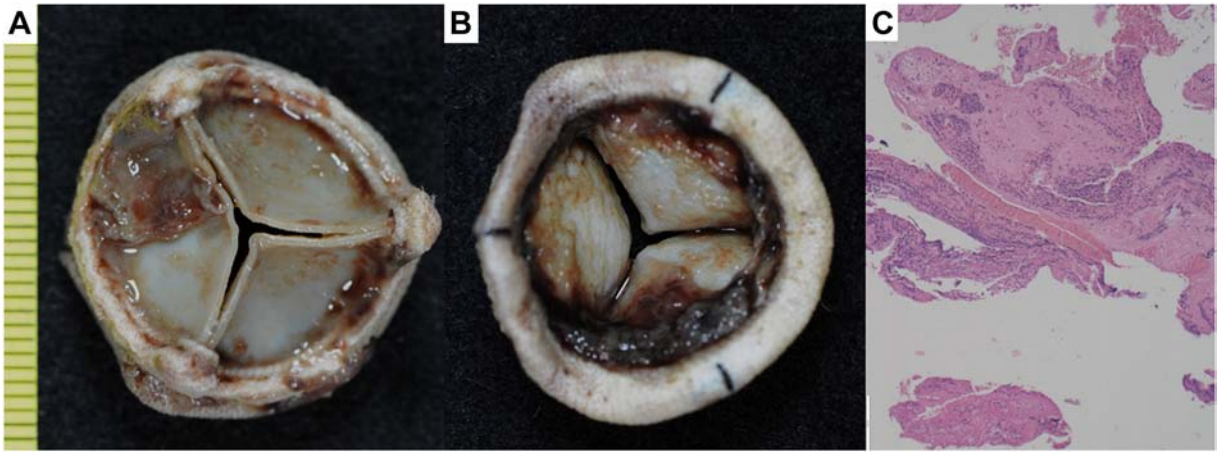

Fig. 2. Surgically removed Magna Ease 21-mm bioprosthesis. (A) Macroscopic view of the aortic side showing thrombosis, especially upon the left coronary cusp, (B) and of the ventricular side with circumferential thrombosis at the basal attachment of the three cusps. (C) Histology of the thrombosis confirming the fibrin network with entrapped blood cells in the absence of microorganisms (hematoxylin–eosin stain).

factors. When is correctly diagnosed and in the presence of hemodynamic instability and acute LV dysfunction, redo valve replacement and thrombectomy are mandatory even if at high risk of morbidity and mortality.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.carpath.2019.03.003.

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