Case report - Valves

**Thrombus in the distal aortic arch after apicoaortic conduit for severe aortic stenosis**

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Abstract

We report an uncommon case of thrombogenesis in the distal aortic arch after apicoaortic conduit (AAC) for severe aortic stenosis (AS). A 71-year-old woman underwent AAC with a bioprosthetic valve for severe AS because of heavy calcification of the ascending aorta. Although anticoagulant therapy with warfarin was performed, a postoperative computed tomographic (CT) scan revealed a thrombus in the distal aortic arch. Cine magnetic resonance imaging (MRI) revealed stagnation of the blood flow at that site. Administration of warfarin was continued. A follow-up CT-scan showed a marked reduction of the thrombus at six months after the surgery. A follow-up MRI revealed that the antegrade flow through the native aortic valve was decreased at one year after the surgery. We suggest that thrombogenesis may occur after AAC because of stagnation of the blood flow and that the distribution of the blood flow may change during the follow-up period. Therefore, we recommend that postoperative anticoagulant therapy should be initiated immediately, even when a bioprosthetic valve is used.

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1. Introduction

Recent reports have shown that apicoaortic conduit (AAC) is a feasible option for high-risk patients with aortic stenosis (AS), as such that caused by previous coronary artery bypass grafting or porcelain aorta. Concerning acquired AS, thromboembolic events have been shown to be uncommon after AAC [1–3]. Therefore, postoperative anticoagulant therapy remains controversial. We report a case of thrombogenesis in the distal aortic arch after AAC.

2. Case

A 71-year-old woman was admitted to our hospital with a complaint of dyspnea on rest (New York Heart Association (NYHA) class IV). She was 158 cm in height and 81 kg in weight with a body mass index of 32. Echocardiography revealed severe AS with an aortic valve area of 0.44 cm² and a peak aortic pressure gradient of 98 mmHg. The ejection fraction was 54%. Cardiac catheterization demonstrated 90% stenosis of the large diagonal branch. A chest computed tomographic (CT) scan showed heavy calcification from the ascending aorta to the aortic arch. It was difficult to carry out a conventional aortic valve replacement with aortic cannulation, aortic cross-clamp and aortotomy. To avoid manipulation of the ascending aorta, we decided to perform AAC on the patient.

A left thoracotomy was performed through the fifth intercostal space, and a left internal mammary artery (LIMA) graft was harvested. Cardiopulmonary bypass was established by arterial cannulation via both the right femoral artery and left axillary artery. First, the LIMA was anastomosed in an end-to-side fashion. Ventricular fibrillation was induced by a fibrillator under hypothermia of 28 °C and the proximal conduit was anastomosed to the diagonal branch under cardiopulmonary bypass. The valved conduit was constructed of a 24-mm Gelseal graft (Vascutek Ltd, Renfrewshire, Scotland) and a 19-mm Carpentier–Edwards Perimount pericardial bioprosthesis (Edwards Lifesciences, Irvine, CA). We usually used three components to perform AAC: a proximal conduit for apical-connection; a distal conduit for aortic-connection; and a valved conduit with a bioprosthetic valve. However, epiaortic echography revealed severe atheroma of the descending aorta to be anastomosed, and we considered that it was not a suitable site for distal anastomosis. Therefore, a short segment of the descending aorta was replaced with a 22-mm Gelseal graft and the distal conduit was anastomosed in an end-to-side fashion. Ventricular fibrillation was induced by a fibrillator under hypothermia of 28 °C and the proximal conduit was anastomosed to the apex. After defibrillation and rewarming, these two conduits were interposed with the valved conduit (Fig. 1).

The postoperative course was uneventful. Warfarin was initiated on postoperative day (POD) 3 when the patient was extubated. Although it required three weeks for the prothrombin time-international normalized ratio (PT-INR) value to exceed 2.0, it was controlled between 2.0 and 3.0 thereafter. A CT-scan on POD 18 revealed a thrombus in...
nately, continuation of the anticoagulant therapy reduced the thrombus and she did not suffer a stroke. The thrombus was probably caused by stagnation of the blood flow, which was revealed by cine MRI with to-and-fro of the bloodstream in the aortic arch. In addition, although it required three weeks until the prothrombin time was sufficiently extended (PT-INR > 2.0), no other anticoagulant therapy including heparin was carried out during the early postoperative period. These two factors would affect thrombogenesis in the distal aortic arch.

Few reports have referred to the distribution of the blood flow after AAC for acquired AS. Echocardiography, cardiac catheterization and cine MRI are usually used for evaluation of the hemodynamics after AAC [4, 5]. Gammie et al. [1] reported that a mean value of 28% of the cardiac output was ejected through the native aortic valve. They suggested that this distribution of the blood flow after AAC would protect the brain against thromboembolism because no retrograde flow from the conduit was derived to the brain. Balaras et al. [6] also pointed out that all the blood flow to the brain was completely supplied by antegrade flow across the aortic valve using computational modeling. In the present case, postoperative cine MRI demonstrated that 29% of the cardiac output occurred through the native aortic valve. However, a follow-up MRI revealed that the antegrade flow had decreased to 6% of the cardiac output at one year after the surgery. The distribution of the blood flow would, therefore, have changed during the follow-up period.

The necessity for anticoagulant therapy after AAC for acquired AS is controversial. Lockwandt [2] reported that 13 patients undergoing AAC for calcified AS were administered warfarin for three months after the surgery, and no thromboembolic events were encountered. Gammie et al. [1] reported that 31 patients were treated with 325 mg of aspirin alone after AAC for severe AS, and no stroke episodes were encountered during the follow-up. On the other hand, Takeda et al. [7] reported a case of a patient who had a thrombus formation in the aortic arch after AAC using a bioprosthetic valve. Warfarin was initiated immediately, but they did not describe the follow-up CT-scan.

We have performed AAC in seven patients to date. All of these patients were administered warfarin, and no thrombogenesis in the aortic arch has been encountered except for the present case. Four of the seven patients underwent a follow-up MRI at one year after the surgery to evaluate the distribution of the blood flow. In all of these patients, the antegrade flow was decreased. We suggest that thrombogenesis possibly occurs owing to stagnation of the blood flow after AAC, even if a bioprosthetic valve is used. In addition, although the site of the thrombogenesis depends on the distribution of the blood flow, we cannot predict how the distribution will be changed in the future and where a thrombus will occur in individual cases. Therefore, we recommend that extensive anticoagulant therapy should be initiated for all patients early after AAC.

3. Discussion

Although anticoagulant therapy with warfarin was carried out in the present case, a thrombus developed in the distal aortic arch after AAC using a bioprosthetic valve. Fortu-

References


