Delayed visceral malperfusion after Bentall procedure for type A acute aortic dissection

Satoshi Yamashiro*, Yukio Kuniyoshi, Yuya Kise and Ryoko Arakaki

Division of Thoracic and Cardiovascular Surgery, Ryukyu University Hospital, Okinawa, Japan

* Corresponding author. Division of Thoracic and Cardiovascular Surgery, Ryukyu University Hospital, 207 Uehara, Nishihara, Okinawa 903-0215, Japan. Tel: +81-98-8951168; fax: +81-98-8951422; e-mail: y3104@med.u-ryukyu.ac.jp (S. Yamashiro).

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Abstract

Rapid restoration of flow into the true lumen and obliteration of a false lumen is considered the optimal approach to treating malperfusion syndrome due to acute aortic dissection. However, organ malperfusion can occasionally persist after proximal aortic graft replacement despite redirecting blood flow into the true lumen. A 35-year old man underwent the modified Bentall procedure for Stanford type A acute aortic dissection without organ malperfusion. Ischaemia of the visceral and lower extremities developed on postoperative day 8. Enhanced computed tomography (CT) revealed a thrombus in the false lumen interfering with the true lumen above the celiac trunk. We immediately performed a left axillary-to-bilateral femoral artery bypass. The patient recovered uneventfully and was discharged on postoperative day 28. Although organ malperfusion persisting after proximal aortic graft replacement despite redirecting blood flow into the true lumen is rare, close observation remains imperative after central repair of type A dissection.

Keywords: Type A aortic dissection • Cardiovascular diseases • Visceral ischaemia • Extra-anatomical bypass

INTRODUCTION

The natural course of type A acute aortic dissection is rapidly lethal, mainly as a result of aortic rupture or from organ malperfusion [1]. Significant postoperative complications such as perioperative myocardial infarction, coma, delirium, sepsis, acute renal failure, need for dialysis, acute limb ischaemia and multisystem organ failure are associated with malperfusion [2]. The rapid restoration of flow into the true lumen and obliteration of a false lumen is considered the best approach to treating malperfusion syndrome. However, organ malperfusion can occasionally persist after proximal aortic graft replacement despite redirecting blood flow into the true lumen.

CASE REPORT

A 35-year old man who suddenly developed severe chest and back pains was transported to a nearby hospital by ambulance. Chest CT disclosed Stanford type A acute aortic dissection extending from the ascending aorta to the bilateral common iliac arteries without organ malperfusion. He was transferred to our hospital for surgery. An initial evaluation revealed a blood pressure of 100/60 mmHg without a significant difference between the right and left upper extremities and a regular pulse of 62 beats per min. Transthoracic echocardiography revealed severe aortic regurgitation with a dilated aortic root.

Cardiopulmonary bypass was established with arterial cannulation from the right axillary and right femoral arteries, and venous cannulation from the right atrium. We usually use the femoral and axillary arteries for arterial cannulation during aortic dissection to prevent organ malperfusion and ensure adequate flow.

The modified Bentall operation with ascending aortic replacement proceeded under deep hypothermic circulatory arrest with antegrade selective cerebral perfusion. An intimal tear was identified in the ascending aorta immediately above the left coronary arterial orifice.

The postoperative course was initially uneventful, but abdominal fullness and bilateral leg pain developed, together with a significantly weakened femoral arterial pulse on postoperative day 8 (POD) 8. Enhanced CT disclosed a compression and significant narrowing of the true lumen by a thrombosed pseudo-lumen above the celiac trunk with a left renal arterial occlusion (Fig. 1a and b). Since malperfusion had apparently progressed to include the visceral and lower extremities, we performed an immediate left axillary-to-bilateral femoral artery bypass. The patient recovered uneventfully from this procedure, although acute renal failure required continuous hemodialysis for one week. The true lumen expanded after these procedures and visceral malperfusion disappeared (Fig. 1c and d). A postoperative follow-up CT revealed a well-preserved, expanded true lumen and extra-anatomical bypass flow (Fig. 2). The patient was free of organic dysfunction at the time of discharge on POD 28.

DISCUSSION

Aortic rupture and organ ischaemia are the two main causes of early death in patients with type A aortic dissection. Aortic...
rupture leads to the unpredictable abrupt onset of circulatory derangement with systemic malperfusion. Organ ischaemia is found in 20 to 40% of patients with acute aortic dissection accompanied by various symptoms [1]. Organ ischaemia starts at the onset of dissection or later in the subsequent course and is progressive unless adequate perfusion is restored.

Figure 1: Postoperative findings of enhanced computed tomography. The thrombosed pseudo-lumen above the celiac trunk has compressed and significantly narrowed the true lumen with left renal arterial occlusion (a and b). The true lumen has expanded after extra-anatomical bypass (c and d).

Figure 2: Postoperative follow-up computed tomography findings at discharge. Image shows the well-preserved, expanded true lumen (a-d) and extra-anatomical bypass flow (e). The thrombosed pseudo-lumen above the celiac trunk has disappeared (b-d).
Surgical intervention to treat the aortic pathology often aims to either improve or stabilize malperfusion syndromes such as neurological complications, renal failure or extremity ischaemia. However, when extant systemic or visceral malperfusion complicates repair, a strategy is needed to address abdominal complications before aortic repair can proceed. In some situations such as associated mesenteric ischaemia the conventional strategy of initially addressing the aortic pathology might result in intestinal necrosis and septic complications [2]. Mesenteric malperfusion is associated with a 33.3% in-hospital mortality [3]. Adverse outcomes are mainly associated with a delayed diagnosis and intestinal ischaemia that is already irreversible at the time of surgical exploration. A primary goal of early surgery with graft replacement of the diseased aorta is to minimize morbidity and mortality by preventing or reserving end-organ malperfusion.

The general belief among surgeons is that the rapid restoration of flow into the true lumen and obliteration of a false lumen is the best approach to treating malperfusion syndrome. Fann et al. showed that up to 92% of patients with a peripheral vascular perfusion compromised by acute dissection can expect spontaneous resolution after a proximal aortic replacement [1]. They reported that malperfusion persisted only after central repair of type A dissection. However, we suspected that the obliteration of the primary tear site with the restoration of flow in the true aortic lumen results in the decreased revascularization of the malperfused organ systems. Panneton et al. reported similar findings [4]. Organ malperfusion can persist after proximal aortic graft replacement despite redirecting blood flow into the true lumen, and surgical techniques for central repair might affect the incidence of malperfusion. Persistent or de novo malperfusion could require surgical options such as aortic branch vessel implantation, aortic origin or extra-anatomical bypass grafts.

Orihashi has described the following types of visceral artery perfusion [5]:

(i) No dissection extending into a branch artery with detectable blood flow.
(ii) Dissection extending into a branch artery with a patent and dominant true lumen associated with a small nonperfused or perfused false lumen.
(iii) Dissection extending into a branch artery with a narrowed true lumen compressed by a false lumen without detectable blood flow.
(iv) No dissection in the branch artery, but the orifice is obstructed by an intimal flap in the aorta.

The condition of our patient seemed to fit the dissection with organ malperfusion due to a collapsed true lumen in the aorta. Catheter intervention including aortic fenestration has recently been suggested as a strategy to treat organ malperfusion complicated by aortic dissection. However, we did not consider this option because our patient had a thrombosed occlusion of the pseudo-lumen. The purpose of endovascular intervention is to create pressure in both aortic channels. We believe that surgical revascularization of the visceral arteries remains a safe and straightforward alternative. The extra-anatomical bypass graft thus performed on our patient recovered the perfusion of the distal, true lumen. This experience revealed that the true lumen might not always recover despite being perfused. A thrombus in a false lumen can interfere with the recovery of the true lumen because it occupies space in the vessel and does not dissolve within a short period. A postoperative follow-up CT revealed a well-preserved, re-expanded true lumen in our patient.

Although organ malperfusion persisting after proximal aortic graft replacement despite redirecting blood flow into the true lumen is rare, various mechanisms of malperfusion should be carefully considered. Adverse outcomes are mainly associated with a delayed diagnosis and irreversible organ ischaemia. Early diagnosis and immediate intervention are imperative for patients with organ ischaemia due to aortic dissection, but reliable diagnostic markers or findings to predict the condition have not yet been identified. Close observation remains imperative after central repair of type A dissection.

Conflict of interest: none declared.

REFERENCES