Management of visceral malperfusion complicated with acute type A aortic dissection

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Abstract

OBJECTIVES: The extent of visceral malperfusion due to acute type A aortic dissection remains difficult to assess in view of the clinical signs that typically present at a late stage. We suspected that visceral malperfusion can persist after proximal aortic graft replacement despite redirecting blood flow into the true lumen. We therefore evaluated the operative outcomes of visceral malperfusion complicated with acute type A aortic dissection.

METHODS: Among 121 patients with acute type A aortic dissection treated at our hospital between January 2000 and December 2014, 10 (8.2%) were preoperatively complicated with visceral malperfusion. Eight of them had been treated by visceral arterial branch bypass followed by central repair, and 2 with circulatory instability had undergone central repair followed by laparotomy.

RESULTS: The 2 patients who underwent initial central repair required extensive intestinal resection due to necrosis and died of multiple organ failure related to visceral necrosis in hospital (hospital mortality rate, 20.0%). The ischaemic time (interval between the onset of dissection and visceral arterial revascularization) was significantly longer for patients who initially underwent central repair compared with those who were initially treated by visceral arterial revascularization. However, base excess and lactate levels did not significantly differ between the two groups.

CONCLUSIONS: We believe that if visceral ischaemia is severe and extensive in patients with type A aortic dissection, abdominal surgery should proceed before the aorta is surgically approached to avoid further irreversible ischaemic damage caused by circulatory arrest in organs with compromised perfusion.

Keywords: Visceral malperfusion • Aortic dissection • Superior mesenteric artery ischaemia

INTRODUCTION

About 25% of aortic dissections have evidence of peripheral malperfusion at presentation. However, the optimal treatment for complicated acute type A aortic dissection remains controversial. The mortality rates for patients with such complications are significant, particularly when mesenteric ischaemia (that results in multiorgan failure) renders surgical repair difficult. The extent of visceral malperfusion is difficult to assess in view of poor clinical signs that typically present at a late stage. Many surgeons believe that the rapid restoration of flow into the true lumen and obliteration of the false lumen is the optimal strategy for treating malperfusion syndrome. Nevertheless, we suspected that visceral malperfusion can persist after proximal aortic graft replacement despite redirecting blood flow into the true lumen, and that the surgical techniques required for central repair might affect the incidence of visceral malperfusion.
branch bypass followed by central repair. The patients were endotracheally intubated and then placed on an operating table in the supine position under general anaesthesia. Before the visceral bypass procedure, we infused 100 U/kg of heparin intravenously to maintain an active coagulation time (ACT) >200 s. Blood flow was disordered in the SMA and/or CA but intestinal necrosis was not evident at laparotomy. Therefore, an emergency saphenous vein bypass proceeded from the right common iliac artery to the SMA with or without the CA, where it was free from dissection. All patients who had initially been treated by visceral arterial revascularization had also undergone second-look re-laparotomy immediately after central repair to evaluate visceral circulation. None of the patients required additional treatment for visceral problems. Central repair was immediately followed by laparotomy in 2 patients with late diagnosis.

The central operation proceeded as follows. Cardiopulmonary bypass (CPB) was established with right axillary and femoral arterial cannulation for arterial inflow and right atrial drainage. More heparin (200 U/kg) was injected immediately before central repair to maintain ACT at >400 s during CPB. Hypothermia was produced by core cooling during CPB. Systemic circulation was arrested when the rectal temperature of the patient reached 20°C and the aorta was opened. The patient was placed in the Trendelenburg position and then 12-Fr malleable cerebral perfusion catheters (Fuji System, Tokyo, Japan) connected to the oxygenator with a separate single-roller pump head were inserted into the left common carotid and left subclavian arteries through the aortic lumen. A cannula was inserted into the right axillary artery for the right antegrade selective cerebral perfusion (SCP) with proximal brachiocephalic artery clamping using another single-roller pump. Cerebral perfusion was started at a rate of 10–15 ml/min/kg and adjusted to maintain left radial and bilateral carotid arterial stump pressure between 40 and 60 mmHg. Perioperative blood flow through the middle cerebral arteries was continuously monitored using bilateral transcranial Doppler ultrasound (Viasys, Inc., Conshohocken, PA, USA) and cerebral oxygen saturation was monitored using an Invos® cerebral oximeter (Somanetics Corp., Troy, MI, USA). Distal repair proceeded via an open distal anastomosis with a single- or triple-branched Dacron graft. Both hypothermic circulatory arrest (HCA) and SCP were terminated after reconstructing the distal aorta with antegrade perfusion from the graft branch and then proximal repair was completed during rewarming.

We usually prevented infection under such emergency situations by administering antibiotics (cefazolin and vancomycin) for 3 or 4 days. Postoperative visceral vascularization was evaluated by CT and angiography in all survivors. Postoperative graft patency and the visceral arterial blood supply were evaluated annually by CT in all survivors during long-term follow-up.

### Statistical analysis

Continuous data are expressed as means ± standard deviation, and categorical variables are expressed as ratios (%). Characteristics between the two groups were compared using Student’s t-test and StatView 5.0. Aortic event-free survival was estimated using the Kaplan–Meier product-limit method. P < 0.05 was considered to indicate statistical significance.
RESULTS

One of 7 patients with visceral ischaemia treated with an SMA branch bypass required a simultaneous CA branch bypass (Fig. 1). The coeliac trunk of this patient was dissected, the true lumen was compressed by a thrombosed false lumen and the SMA arose from the false lumen without thrombosis. A saphenous vein bypass graft proceeded from the right common iliac artery to the SMA and a CA branch (gastroduodenal artery) beyond dissection (Fig. 2). Thereafter, total arch replacement proceeded using a 24-mm Hemashield arch graft via a CPB with concomitant SCP. The intraoperative flow rates measured in the SMA and CA grafts immediately after central repair using an MFV-3200 electromagnetic flowmeter (Nihon Kohden, Tokyo, Japan) were 400 and 420 ml min⁻¹, respectively. Flow rates and pressure in the target vessels were increased: flow in the SMA increased from 120 ml min⁻¹

Figure 1: CT findings. (A) Coeliac trunk (arrow) is dissected and lumen is compressed by thrombosed false lumen. (B) SMA (dotted arrow) arises from false lumen without thrombosis. SMA: superior mesenteric artery.

Figure 2: Operative findings and schema of visceral bypass. Saphenous vein bypass grafting (to CA and SMA, white and black dotted arrows, respectively) performed from the right common iliac artery to the CA branch (gastroduodenal artery) (white arrow) (A and B) and the SMA (black arrow) (C and D) beyond dissection. SMA: superior mesenteric artery; CA: coeliac artery.
when the graft was closed, to 160 ml min$^{-1}$ when it was open and that in the CA increased from 130 to 380 ml min$^{-1}$ upon opening the graft. At a systemic blood pressure of 112/58 mmHg, pressure in the SMA and CA was 95/50 and 93/55 mmHg, respectively, with the grafts open, and 78/48 and 56/44 mmHg, respectively, with the grafts clamped. Arterial pulsation in the mesentery recovered. Postoperative angiography revealed good perfusion of the vein grafts from the right common iliac artery to the SMA and gastroduodenal artery (Fig. 3).

We immediately performed an initial left axillary-to-bilateral femoral artery bypass in one patient, since malperfusion had apparently progressed to include the visceral and lower extremities. Enhanced CT disclosed dissection extending into the visceral arteries as well as a compressed and significantly narrowed true lumen caused by a thrombosed pseudo lumen above the coeliac trunk with left renal arterial occlusion (Fig. 4). A modified Bentall operation then proceeded with ascending aortic replacement under deep HCA and SCP. Postoperative follow-up CT revealed a well-preserved, expanded true lumen and extra-anatomical bypass flow (Fig. 5).

The mean interval from the onset of dissection to surgery in all patients was 4.8 ± 1.0 h, and this did not significantly differ between patients initially treated by visceral arterial revascularization or central repair (4.5 ± 0.9 vs 6.0 ± 0.1 h, $P > 0.05$). However, the ischaemic time was significantly longer for patients who were initially treated with central repair than with visceral arterial revascularization (13.0 ± 1.4 vs 5.5 ± 0.9 h; $P < 0.001$). Two patients who were initially treated with central repair and required extensive intestinal resection due to necrosis died in hospital (hospital mortality, 20.0%) as a result of multorgan failure arising related to the necrosis. Immediately after weaning them from CPB, their haemodynamics collapsed with significant acidosis, and then laparotomy revealed necrosis of the gall bladder and entire small intestine. Only one of the patients who were initially treated with visceral arterial revascularization required short-segment intestinal resection for necrosis without other complications. The postoperative course of all those initially treated by visceral arterial revascularization was uneventful. Two (66.7%) of 3 patients who required intestinal resection died.

The base excess values for arterial blood gas and lactate levels immediately before initial visceral arterial revascularization

![Figure 3: Postoperative angiographic findings. Image shows good perfusion of vein grafts (arrow, to CA; dotted arrow to SMA) from right common iliac to gastroduodenal artery (white arrowhead) (A) and SMA (black arrowhead) (B). SMA: superior mesenteric artery; CA: coeliac artery.](image1)

![Figure 4: Preoperative enhanced CT findings. Dissection extends into visceral arteries and thrombosed pseudo lumen (dotted arrow) has compressed and significantly narrowed true lumen (arrow) of CA (A) and SMA (B). SMA: superior mesenteric artery; CA: coeliac artery; CT: computed tomography.](image2)
and immediately before initial central repair were $-2.8 \pm 1.2$ and $-4.0 \pm 2.8$ and $3.3 \pm 1.2$ and $5.0 \pm 1.4$, respectively. Intestinal resection and ischaemic time significantly differed between those initially treated by visceral arterial revascularization and those initially treated by central repair (Table 1).

Ischaemic time and intestinal necrosis were significant predictors of mortality. However, neither the base excess nor the lactate levels significantly differed regardless of the order of the two procedures.

The rate of aortic event-free survival in patients initially treated by visceral arterial revascularization at 5 years after surgery was 100%, and all patients were followed up for 115.8 ± 62.7 months.

**DISCUSSION**

That the natural course of type A acute aortic dissection is rapidly lethal, mainly as a result of aortic rupture or organ malperfusion, is widely accepted [1]. Organ ischaemia starts at the onset of dissection or later in the subsequent course and progresses unless perfusion is adequately restored. Organ or limb perfusion is usually restored by aortic grafting, aortic fenestration or an extra-anatomical bypass. However, a strategy is needed to address abdominal complications before aortic repair can proceed when extant systemic or visceral malperfusion complications occur. Acute aortic dissection is one of the most frequent causes of vascular acute abdomen.

This pathology can cause serious complications, such as aortic rupture with consequential immediate death, visceral ischaemic necrosis (bowel, liver and kidney) with possible mortal effects and ischaemia of the spinal cord leading to paraplegia [2].

In general, many surgeons believe that rapid restoration of flow into the true lumen and obliteration of the false lumen is the best way to treat malperfusion syndrome. In fact, aortic surgery usually proceeds first because of the high risk of aortic rupture, followed by visceral and peripheral surgery.

Fann et al. [1] found that up to 92% of patients with peripheral vascular compromise due to acute dissection can expect spontaneous resolution after proximal aortic replacement [1]. They reported that persistent malperfusion occurred only after central repair of type A dissection. However, we suspected that obliteration of the primary tear site with restoration of flow in the true aortic lumen results in the decreased revascularization of malperfused organ systems. Similar findings were reported by Panneton et al. [3]. In fact, both of our patients who were initially treated by central repair died of complications related to intestinal necrosis. Mesenteric malperfusion is associated with a 33.3% in-hospital mortality rate [4] and adverse outcomes are mainly associated with a delayed diagnosis and intestinal ischaemia that is already irreversible at the time of surgical exploration. Shimamoto and Komiya described that visceral malperfusion is the most lethal among all malignant phenomena associated with malperfusion syndrome, as it is associated with a poor prognosis [5].

A primary goal of early surgery with graft replacement of the diseased aorta is to minimize morbidity and mortality by preventing or resolving end-organ malperfusion. Even though replacement of the ascending aorta and arch repair does not always completely obliterate a false distal lumen, correcting the complex perfusion disturbance alleviates distal aortic branch malperfusion and can avoid the need for additional procedures to revascularize malperfused organ systems. A tailored multidisciplinary strategy is required to address the underlying risk in patients with more complex aortic dissections and achieve optimum perfusion and survival. We believe that abdominal surgery should precede aortic procedures if the aortic pathology is relatively stable haemodynamically, if hypertension is controlled well and if visceral ischaemia is severe and extensive, so as to avoid further irreversible ischaemic damage due to circulatory arrest in organs with compromised perfusion.

Uchida et al. proposed classifying organ malperfusion into two types [6]. One is dissection extending into branch arteries with a narrowed true lumen compressed by a false lumen without detectable blood flow and the other is dissection with organ malperfusion caused by a collapsed true lumen in the aorta. Such classifications can help surgeons to judge whether malperfusion could be addressed with central repair or by additional revascularization. The condition of our patients seemed to fit the first of the classifications proposed by Uchida et al. [6].

Catheter intervention has recently been suggested as a strategy to treat organ malperfusion complicated with aortic dissection [7].
Endovascular treatment including fenestration and branch stenting during the same hospitalization halves the mortality rate [8]. Initial ascending aortic repair and post-repair laparotomy to treat advanced visceral malperfusion are associated with high mortality rates that could theoretically be reduced if the malperfusion is treated first. 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.

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 considered. Some patients did not have clinical signs of intestinal malperfusion, despite having significant peripheral ischemia. To diagnose mesenteric ischemia in the setting of an acute aortic dissection can be difficult, but any delay in treatment might be fatal. We experienced nine patients with visceral malperfusion who required emergency visceral bypass for type B acute aortic dissection within the same period [9]. Three of them had dissection extending to the visceral branch and two others died. As acidosis and lactate levels did not significantly differ between the surviving patients and those who died. The only significant difference was the amount of time that elapsed before they underwent surgery (survived vs died: 8.6 ± 2.3 vs 23.3 ± 10.6 h; P = 0.01). We performed a visceral bypass 6 h after branch dissection occurred in 1 patient who survived. This patient had significant intestinal oedema during surgery despite the short period from the onset of dissection.

Demir et al. noted that no single serum marker, including traditional serum lactate, can be regarded as reliable enough to diagnose acute mesenteric ischemia [10]. Another study found that lactate levels might be affected by several factors including ischaemia-related hepatic dysfunction [11].

How to prioritize central or peripheral repair, or even triage for a patient is a clinical dilemma. We considered that if the degree of visceral damage is uncertain, then laparotomy should be prioritized. The current gold standard for evaluating malperfusion syndrome is CT, but its diagnostic accuracy is affected by various factors such as patient weight, contrast volume, the timing of assessment and the target vessel diameter. However, we proceed with laparotomy first when dissection extending into both visceral arteries is suspected. We have recently started to perform a small laparotomy to identify visceral malperfusion before central repair even when the pathology of visceral malperfusion is a collapsed true lumen in the aorta of patients with stable haemodynamics. This strategy was applied to several patients within the same period.

Although a surgical duration of 10 h seems to impose a burden on such critically ill patients, malperfusion syndrome associated with acute type A aortic dissection is associated with poor clinical outcome, especially when the malperfusion is visceral. Accurate and timely preoperative, intraoperative and postoperative assessment, using imaging and monitoring modalities and condition-dependent treatment such as delayed central repair with prior peripheral revascularization or central repair with subsequent peripheral repair, is mandatory for decreasing mortality rates. Therefore, the current consensus should be that central aortic repair takes priority in the absence of ongoing or exacerbating severe visceral ischaemia with bowel necrosis [5]. We attempt to systematically diagnose and treat acute aortic syndromes within the shortest possible time frame. We determine whether patients have cardiac tamponade, massive aortic regurgitation or poorer contrast of visceral arteries on CT images from referring physicians. Visceral ischaemia can be overlooked because preoperative symptoms are often ambiguous, and may become exacerbated without discernible symptoms during surgery. Damage caused by localized ischaemia due to malperfusion is significantly enhanced by generalized ischaemia caused by circulatory collapse such as cardiac tamponade and coronary ischaemia. However, we have never experienced simultaneous visceral malperfusion and cardiac tamponade or coronary ischaemia.

Although preoperative CT clearly identified dissection extending into both visceral arteries in 2 patients, we decided to proceed with central repair first because we considered that their haemodynamics had become destabilized due to cardiac tamponade or massive AR. We felt that a diagnosis of visceral ischaemia was delayed in these 2 patients.

A clear-cut strategy for treating visceral malperfusion due to aortic dissection has not been established. Surgeons should not underestimate the negative impact of radiologically overt malperfusion even though ischaemia is not particularly obvious during hospitalization.

The operative mortality rate was even higher among patients who required abdominal exploration. We considered that surgical treatment initially be directed towards the peripheral vascular complication. Moreover, close observation remains imperative after central repair of type A dissection. This study is limited by the relatively small patient cohort. However, our findings might have practical implications for the ongoing evolution of treatment for visceral malperfusion complicated with acute type A aortic dissection.

Conflict of interest: none declared.

REFERENCES