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Abstract | Figures/Tables

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Activation of cardiac progenitor cells through paracrine effects of mesenchymal stem cells

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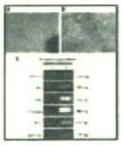

Fig. 1. Morphological features and gene expression of CPC derived from neonatal rat heart. (A,B) Representative photographs of CPC isolated by explant method. White arrows indicate CPC and black arrow indicates fibroblast-like cells. (A) Bar: 200 μm. (B) Bar: 20 μm. (C) Gene expression profile of CPC by RT-PCR.

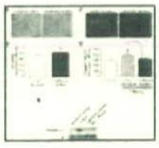

Fig. 2. Proliferative and antiapoptotic effect of MSC-derived conditioned medium on CPC. (A) Representative photographs of CPC incubated in basal culture medium (serum starvation) and MSC-derived conditioned medium for two days. Bar: 200 μm. (B) MTS assay of CPC. $^{*}p < 0.05$ vs serum starvation. (C) TUNEL staining of CPC. TUNEL-positive apoptotic CPC are stained green. Nuclei are stained with DAPI (blue). White arrows indicate TUNEL/DAPI double-positive cells. Bar: 50 μm. (D) Caspase-3 activity of cultured CPC. $^{*}p < 0.05$ vs control, $^{\dagger}p < 0.05$ vs serum starvation. (E) Western blot analysis. MSC-derived conditioned medium as well as 50 ng/mL HGF phosphorylated Akt compared to basal culture medium (standard medium).

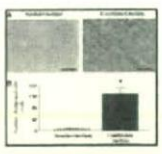

Fig. 3. Migration of CPC induced by MSC-derived conditioned medium. (A) Representative photographs of migrated CPC incubated in basal culture medium (standard medium) and MSC-derived conditioned medium. White arrows indicate migrated CPC (standard medium). Bars: 100 μm. (B) Quantitative analysis of migrated CSC. $^{*}p < 0.05$ vs standard medium.

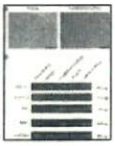

Fig. 4. Cardiomyogenesis of CPC induced by MSC-derived conditioned medium. (A) Morphological features of CPC after incubation in complete culture medium (Vehicle) and MSC-derived conditioned medium for two weeks. Cultured CPC did not beat spontaneously. (A) Bar: 50 μm. (B) RT-PCR analysis of CPC after induction of cardiomyogenesis. Adult rat heart extract was used as positive control.

Table 1.

Primer pairs for RT-PCR

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ORIGINAL ARTICLE

Competition Between Native Flow and Graft Flow After Coronary Artery Bypass Grafting. Impact on Indications for Coronary Artery Bypass Grafting for Localized Stenosis with Giant Aneurysms Due to Kawasaki Disease

Etsuko Tsuda · Hideki Fujita · Toshikatsu Yagihara · Osamu Yamada · Shigeyuki Echigo · Soichiro Kitamura

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Abstract We report the postoperative course of native and graft flow after coronary artery bypass grafting (CABG) in two patients with giant aneurysms and localized stenosis due to Kawasaki disease (KD). Although both patients had undergone CABG to the left anterior descending artery (LAD) with the left internal thoracic artery (ITA), at 5 and 10 years old, respectively, the ITA grafts were occluded 1 month postsurgery. However, when the two patients suffered complete occlusion of the native LAD more than 10 years after surgery, angiograms showed that the ITA grafts had reopened. We believe that this postoperative course reflects competition between the native artery flow and graft flow after CABG. CABG in patients with severely delayed coronary flows or recurrence of thrombus in giant aneurysms was ineffective in preventing myocardial infarction or damage. We conclude that CABG in giant aneurysm without significant localized stenosis should be avoided.

Keywords Kawasaki disease · Coronary artery aneurysm · Intracoronary thrombus · Intracoronary thrombolysis · Coronary artery bypass grafting

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Giant aneurysm is one coronary artery lesion that characterizes Kawasaki disease (KD) [6, 7] and, after the acute episode, may lead to thrombotic coronary artery occlusion or coronary artery stenosis and eventually progress to ischemic heart disease [2, 10]. Myocardial revascularization by coronary artery bypass grafting (CABG) to prevent myocardial infarction or myocardial damage is useful for stenotic lesions due to KD [4, 11], but determining its optimal timing is difficult. We sometimes encounter recurrent thrombus in a giant aneurysm or severely delayed flow in a giant aneurysm with ischemic signs, and we discuss the decision whether or not to recommend CABG. We report unusual latent occlusion of the internal thoracic artery (ITA) graft more than 10 years postsurgery in two patients with giant aneurysms and localized stenosis. This experience might provide some basis for determining the indications for CABG in patients with giant aneurysms caused by KD.

Case Reports

Case 1

One patient had acute KD when 4 years old and was treated with flurbiprofen and steroid. Bilateral giant aneurysms were detected by two-dimensional echocardiography (2DE) 12 days after the onset of KD. She underwent cardiac catheterization and selective coronary angiograms (CAG) 2 months later, when the maximum diameter of both the right coronary artery (RCA) and the left coronary artery (LCA) was 12 mm. Aspirin was started to prevent thrombotic occlusion. Four months after the onset of KD, the patient had an acute inferior myocardial infarction. When she was 5 years old CAG revealed segmental

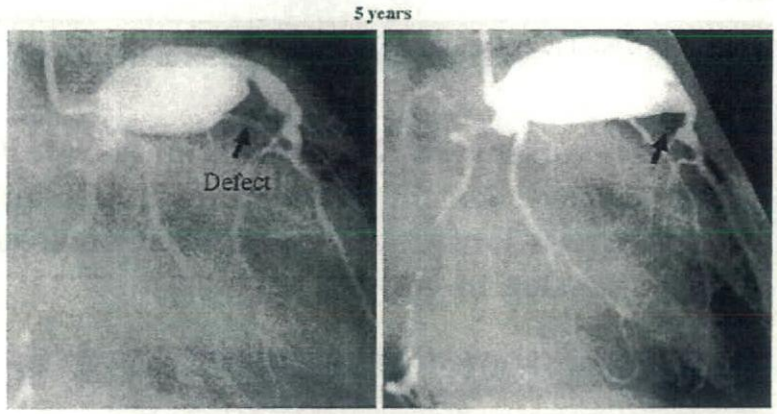

Fig. 1 Left coronary angiograms (Case 1). **Left**: The left coronary angiogram revealed a defect due to thrombus in a giant aneurysm. **Right**: The defect due to thrombus in the giant aneurysm disappeared after intracoronary thrombolysis

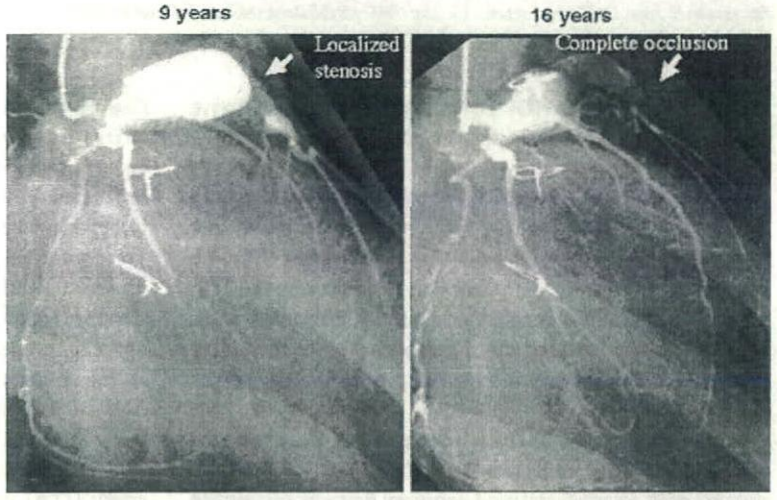

Fig. 2 Left coronary angiograms (Case 1). **Left**: The left coronary angiogram revealed segmental stenosis of the left anterior descending artery at the age of 9 years. **Right**: The left anterior descending artery was occluded at the age of 16 years

stenosis of the RCA. One week after the examination, she became pale and felt generally unwell. After 2 months, she was referred to our hospital. A thrombus in the aneurysm of the LCA was detected by 2DE, a perfusion defect was observed in the inferior and anterolateral wall of the left ventricle by dipyridamole-loaded ^{201}Tl myocardial imaging, and redistribution was detected in the anterolateral wall of the left ventricle. CAG confirmed a giant aneurysm of the LCA with a defect due to thrombus (Fig. 1, left). The left ventricular ejection fraction (LVEF) was 50%. Blood flow in the left anterior descending artery (LAD) was delayed. Intracoronary thrombolysis (ICT) by tissue plasminogen activator was performed for the LCA, and the thrombus dissolved (Fig. 1, right). There was no stenosis of LAD beyond the giant aneurysm. However, the thrombus reoccurred within 2 months despite the administration of urokinase and heparin intravenously. Warfarin was started in addition to aspirin. After 2 months, CABG to the LAD with the left internal thoracic artery (ITA) was performed at the age of 5 years 8 months. One month after surgery, angiography showed that the ITA graft was completely occluded and flow in the native LAD was detected. When the patient was 9 and 13 years old, CAGs showed localized stenosis of the LAD (Fig. 2, left). ITA graft flow was not detected in the follow-up angiograms (Fig. 3, left). Depression of ST-T segments in the left precordial leads after exercise was detected from the age of 13 years. Perfusion defects in the anterior wall, inferior wall, and apex of the left ventricle were demonstrated by exercised ^{99m}Tc myocardial imaging. The patient had remained symptom-free since surgery. When she was 16 years old, she had abdominal discomfort; CAG at that time revealed complete occlusion of the native LAD (Fig. 2, right). At that angiogram, the ITA graft, although narrow, had reopened (Fig. 3, right). The left ventricular end-diastolic volume index (LVEDVI) and the LVEF were 112 ml/m^2 and 50%, respectively. The ITA graft was patent on angiography at the age of 21 years.

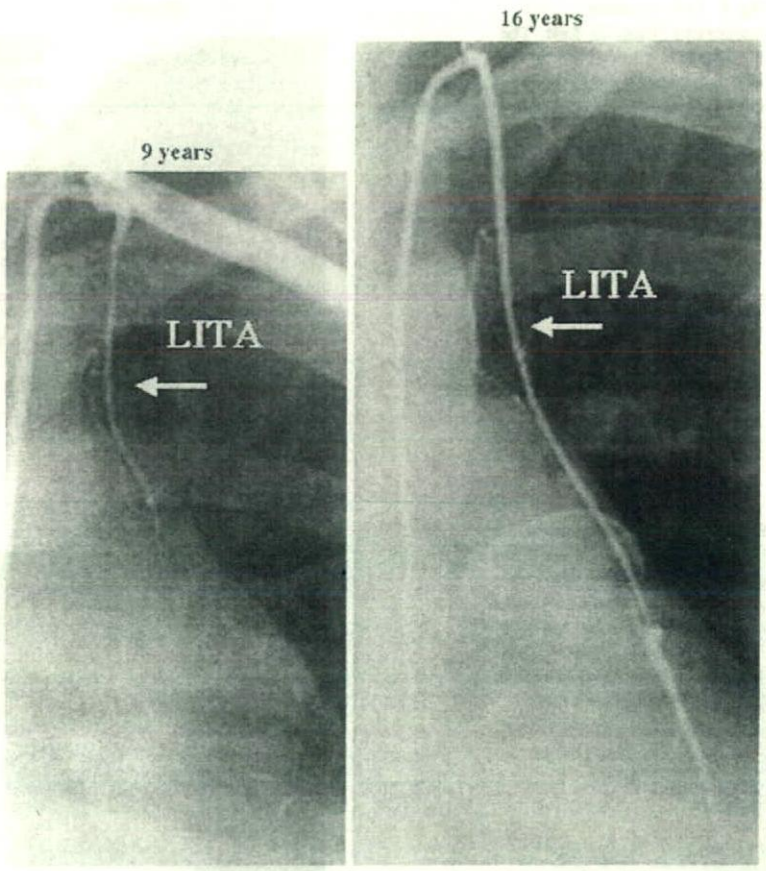

Fig. 3 Left internal thoracic angiograms (Case 1). **Left**: The left internal thoracic graft was occluded at the age of 9 years. **Right**: Flow in the left anterior descending artery was detected through the left internal thoracic graft at the age of 16 years. The stenosis at anastomosis was detected

Case 2

The second patient had acute KD when 8 years old, and she received flurbiprofen and intravenous immunogloblin within 7 days after the onset. Bilateral giant aneurysms were detected by 2DE 14 days after the onset of KD. She underwent cardiac catheterization and CAG after 52 days. At CAG, the maximum diameter of the right coronary artery (RCA) was 10 mm and that of the LAD was 11 mm. Aspirin and dipyridamole were started. CAG at 1 year after the onset of KD revealed localized stenosis of the LAD with severely delayed flow. A perfusion defect in the interventricular septum was detected by ^{201}Tl myocardial imaging after dipyridamole and isoproterenol loading. Two months after the CAG, the patient had a CABG to the LAD using the left ITA at the age of 10 years. One month after surgery, angiography showed that the ITA graft was occluded and the delayed flow in the native LAD persisted. When she was 17 years old, the patient had an acute anterior myocardial infarction. ICT by tissue plasminogen activator for the LAD was initially successful, but she had a reinfarction the next day, and ICT was repeated. The interventricular septum was akinetic by 2DE and perfusion defects were detected in the anteroseptal wall and apex of the left ventricle. The LVEF was 35% and the creatinine phosphokinase was elevated to 8415mg/dl. Warfarin and angiotensin converting enzyme were started. The patient had been asymptomatic since her previous myocardial infarction. CAGs, 3 and 7 years after myocardial infarction, revealed severe delayed flow in the LAD, although the ITA graft was occluded (Fig. 4, left, and Fig. 5, left). Although a localized stenosis of the RCA was detected by CAG when the patient was 20 years old, there was no ST-T change on treadmill testing. When she was 26 years old, CAG revealed complete occlusion of the RCA and the LAD; at that time distal LAD flow through the ITA graft was detected (Fig. 4, right, and Fig. 5, right). The LVEDVI and the LVEF were 131 ml/m^2 and 37%, respectively.

Discussion

Thrombus is often detected in giant aneurysms and presents a treatment challenge. Usually warfarin and an antiplatelet agent are administered, and if either chest pain or ischemic signs on examination occur, ICT by tissue

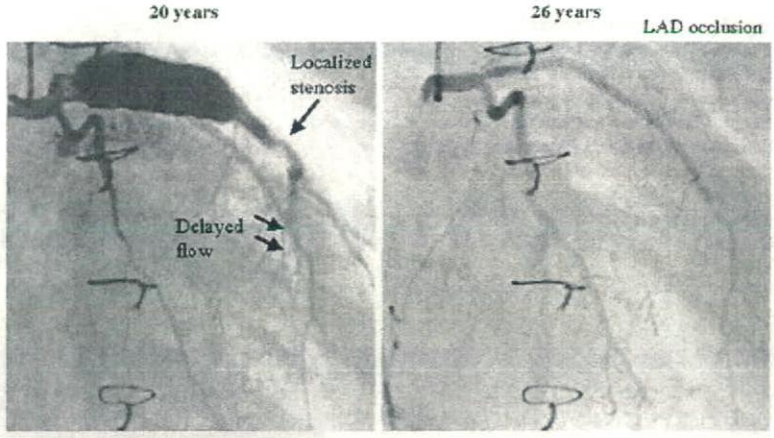

Fig. 4 Left coronary angiograms (Case 2). **Left**: The left coronary angiogram revealed severely delayed flow and localized stenosis of the left anterior descending artery at the age of 20 years. **Right**: The left anterior descending artery was completely occluded at the age of 26 years

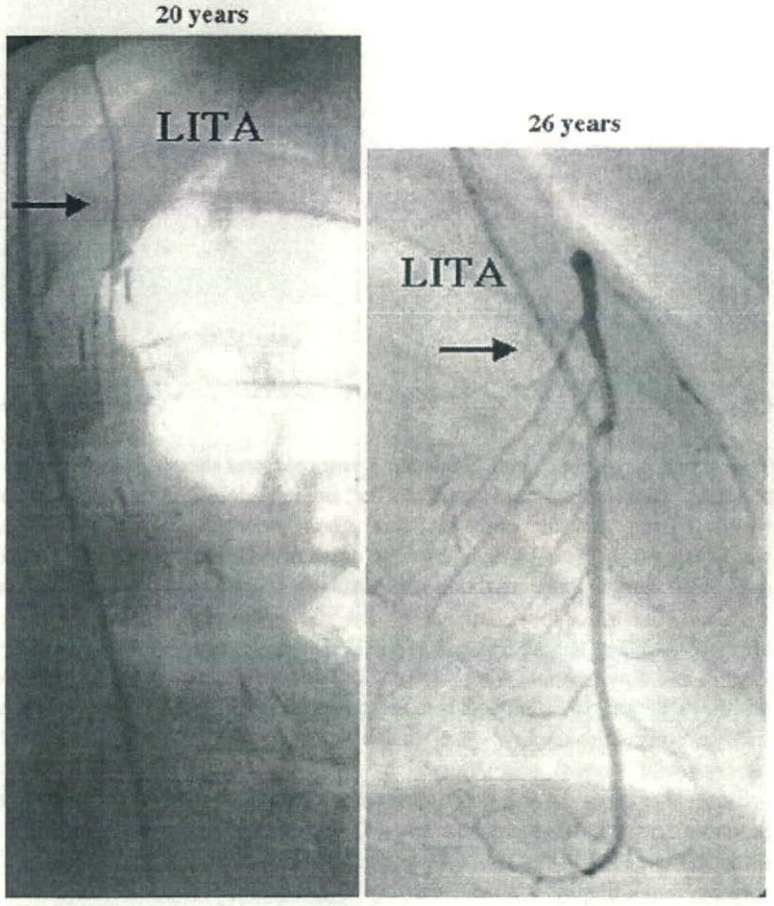

Fig. 5 Left internal thoracic angiograms (Case 2). **Left**: The left internal thoracic graft was occluded at the age of 20 years. **Right**: Flow in the left anterior descending artery was detected through the left internal thoracic graft at the age of 26 years

plasminogen activator is considered [1]. Recurrence of thrombus despite aggressive anticoagulant therapy threatens possible myocardial infarction. We sometimes observe at CAG severe delays in flow due to giant aneurysms with localized stenosis, especially in the LAD. The degree of localized stenosis is difficult to quantify because of the severe delayed flow. If ischemic signs are present, a high risk of myocardial infarction is assumed. In the middle

1980s it was difficult to decide whether or not CABG should be performed for these patients. Our patients had no symptoms or evidence of severe ischemia on examination. Prophylactic CABG at the selected time in these two patients was ineffective in preventing myocardial damage.

In these patients, the ITA grafts became occluded immediately after surgery and antegrade native LAD flow persisted. When the antegrade flow in the native LAD disappeared, the ITA reopened and provided distal LAD flow. We believe the apparent occlusion was functional, not mechanical. Although ITA grafts in children are known to grow with physical development [3], it is striking that an apparently occluded ITA graft had grown more than 10 years after surgery despite absence of flow. This is an interesting example of a "living graft." Until recently, the results of ITA graft patency in small children with stenotic lesions caused by KD have been unsatisfactory [11]. Early graft occlusion after surgery often occurred because of anastomotic stenosis and competition with the native coronary flow [8]. Our experience showed that the ITA graft flow was compromised because of competition with the native LAD flow. Recently we have reported that percutaneous transluminal balloon angioplasty for the anastomotic site performed a few months after surgery helped prevent graft occlusion in patients with giant aneurysm and localized stenosis [5].

The appropriate decision regarding CABG is very important. The existence of severe ischemia on examination and severe localized stenosis or complete occlusion would be absolute indicators for CABG [9]. However, it is not easy to diagnosis clearly the existence of ischemia in cases with recurrent thrombus or severe delayed flow in giant aneurysms. Patients with recurrent thrombosis in giant aneurysms do not always have significant localized stenosis, and severe delayed flow in giant aneurysms occurs because of the hemodynamic effects due to large aneurysms. The thrombus formation and delayed flow often occur simultaneously, and these phenomena influence reciprocally. Further, it is also difficult to detect the degree of localized stenosis accurately in such cases. Flow in the graft will be compromised if the native artery flow is not decreased. In cases with recurrent thrombus in a giant aneurysm despite aggressive anticoagulation or with severe delayed flow in the giant aneurysm without significant localized stenosis, CABG is not necessarily effective, and the indication for CABG should be considered carefully. Our experience with these two patients suggests that CABG in patients with giant aneurysm and mild localized stenosis should be avoided.

Conclusion

We encountered two patients with early ITA graft occlusion after surgery in whom the grafts reopened more than 10 years after surgery with the disappearance of the native coronary artery flow. Prophylactic CABG in patients with severely delayed coronary flow or recurrent thrombus in giant aneurysms was ineffective in preventing myocardial infarction or damage. Our experience suggests that CABG in giant aneurysm without significant localized stenosis should be avoided.

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Interactive CardioVascular and Thoracic Surgery

Spinal cord malperfusion caused by using the segmental clamp technique during descending aortic repair for chronic type B aortic dissection

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Negative results - Vascular thoracic

Spinal cord malperfusion caused by using the segmental clamp technique during descending aortic repair for chronic type B aortic dissection

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Abstract

Several effective strategies for spinal cord protection have been advocated in descending and thoracoabdominal aortic repairs. The segmental clamp technique has been known as a useful adjunct to shorten the duration of spinal cord ischemia. However, we experienced two cases of spinal cord malperfusion during segmental aortic clamping in descending aortic repair for chronic type B aortic dissection. In these patients, the intercostal arteries including the Adamkiewicz artery had originated from the false lumen. In one patient, spinal cord ischemia was initially detected as decreased motor-evoked potentials. Transesophageal echocardiography simultaneously revealed blood flow congestion in the false lumen during segmental aortic clamping and spinal cord ischemia had developed due to malperfusion of the intercostal arteries branching from the false lumen. Segmental clamping in patients with aortic dissection may not always be useful for shortening the duration of spinal cord ischemia. Transesophageal echocardiography as well as motor-evoked potentials is a useful modality for obtaining the details of intraoperative blood flow in dissecting lumens and malperfusion of the intercostal arteries related to spinal cord injury.

Keywords: Spinal cord malperfusion; Aortic dissection; Segmental clamp technique

1. Introduction

The segmental clamp technique has been reported as a useful adjunct to shorten the duration of spinal cord ischemia in descending and thoracoabdominal aortic repairs [1]. However, we experienced two cases of spinal cord malperfusion during segmental aortic clamping.

1.1. Case 1

An 82-year-old male was diagnosed with chronic type B aortic dissection. Its maximum size was 58 mm in diameter at the level of the proximal descending aorta. Enhanced computed tomography (CT) revealed that the false lumen was not thrombosed and that the primary tear was located at the proximal descending aorta. Magnetic resonance angiography (MRA) revealed that the Adamkiewicz artery had originated from the 11th intercostal artery and the patent intercostal arteries had almost branched from the false lumen. Graft replacement of the entire descending aorta was performed. After establishing a partial cardiopulmonary bypass with a femoro-femoral circuit, the core temperature of the patient was reduced to 32 °C. The proximal aortic clamp was applied immediately distal to the left subclavian artery, and the second aortic clamp was applied at the level of the 6th intercostal space for the proximal aortic anastomosis. During aortic clamping, distal aortic perfusion at a pressure above 60 mmHg was maintained by partial cardiopulmonary bypass. The dissecting aneurysm was incised, and the primary tear with a diameter of 20 mm was detected between the clamped sites. Five minutes after the commencement of the proximal anastomosis, the amplitude of motor-evoked potentials (MEPs) disappeared. The double-barreled distal anastomosis as well as the proximal anastomosis was rapidly completed with the preservation of the 8th intercostal artery. The 3rd and 7th intercostal arteries were additively reattached to the graft because the MEP amplitude was not restored. Further, steroid and naloxone were administrated along with cerebrospinal fluid drainage. During surgery and the three days postoperative period, cerebrospinal fluid pressure was maintained at 10 mmHg or less, while cerebrospinal fluid was drained. However, paraplegia was an eventual complication occurring during the postoperative course.

1.2. Case 2

A 75-year-old female was referred for the treatment of an aneurysm of the descending thoracic aorta. Her CT revealed chronic type B aortic dissection associated with a

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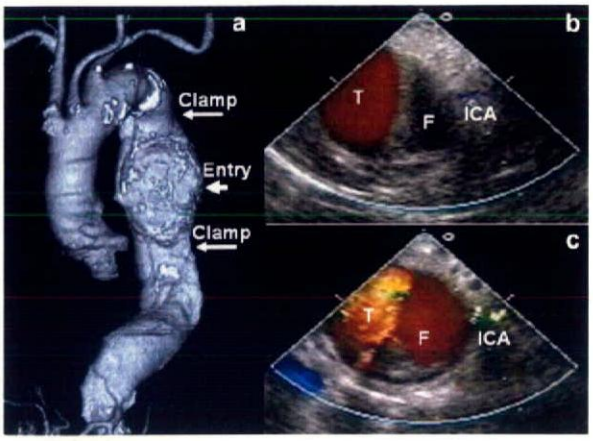

Fig. 1. A three-dimensional computed tomography image (a). The aortic dissection extends from the distal arch to just proximal to the superior mesenteric artery. The primary tear is located at the mid descending aorta. The mid descending aorta exhibits an aneurysmal change, and its maximum diameter is 61 mm. The false lumen is not completely thrombosed. Arrows indicate the segmental aortic clamping and the entry sites. A transesophageal echocardiography image (b, c). It shows blood flow congestion in the false lumen and decreased flow in the intercostal arteries originating from the false lumen during segmental aortic clamping (b). After releasing the segmental clamps, the blood flow in the false lumen and intercostal arteries recovered (c). T; true lumen, F; false lumen, ICA; intercostal artery.

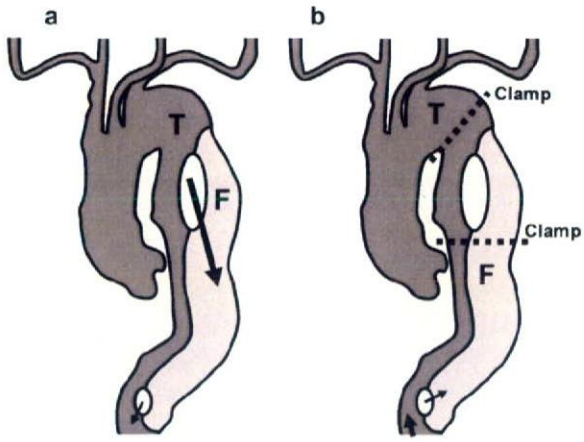

Fig. 2. Flow directions are indicated (arrows), when aorta is unclamped (a). With aortic clamping, the flow directions change as indicated (arrows, b). Dashed lines show the clamping sites. T; true lumen, F; false lumen.

true aneurysm at the mid descending aorta (Fig. 1a). The false lumen was not completely thrombosed, the primary tear existed in the true aneurysm, and the maximum diameter of the descending aorta was 61 mm. Moreover, the small reentry and three lumens were detected around the phrenic level. MRA revealed that the Adamkiewicz artery had originated from the left 10th intercostal artery that had branched from the false lumen. Under partial cardiopulmonary bypass with a core temperature of 32 °C, she underwent replacement of the entire descending aorta. Segmental clamping was put on the proximal and distal sites to the true aneurysm and this clamped segment included the primary tear. During aortic clamping, distal aortic perfusion pressure was maintained at 60–70 mmHg by partial cardiopulmonary bypass. Thereafter, the amplitude of the MEPs diminished, and transesophageal echocardiography (TEE) revealed blood flow congestion in the false lumen in the part distal to the segmental clamp, from where the intercostal arteries had originated. After the segmental clamps were released, the blood flow in the false lumen and intercostal arteries recovered (Fig. 1b,c). The core temperature of the patient was reduced to 20 °C for spinal cord protection, and the entire descending aorta was replaced with the reconstruction of the 10th, 11th, and 12th intercostal arteries under hypothermia circulatory arrest. The postoperative recovery was uneventful, and paraplegia did not occur.

2. Comment

Spinal cord injury is of a major concern in descending or thoracoabdominal aortic repairs. Various strategies for spinal cord protection have been advocated, such as distal perfusion with left heart bypass [2] or partial cardiopulmonary bypasss [3], preoperative identification of the Adamkiewicz artery and intraoperative monitoring of spinal cord ischemia with MEPs [4], reattachment of the intercostal or lumbar arteries [1, 5], cerebrospinal fluid drainage [6], and epidural cooling [7]. The segmental clamp technique has also been reported as a useful method for preventing spinal cord injury [1]. In Case 2 described above, spinal cord ischemia was initially represented as decreased MEPs. Simultaneously, TEE confirmed the malperfusion of the intercostal arteries due to segmental clamping that was related to spinal ischemia. When aorta was unclamped, antegrade and adequate flow from the heart perfused into the false lumen via the large entry (Fig. 2a). However, in our consideration, with proximal aortic clamping, reversal of the aortic flow via the small reentry from the femoral artery during the partial cardiopulmonary bypass might not be adequate for the false lumen perfusion to prevent from spinal cord ischemia (Fig. 2b). In Case 1, malperfusion of the intercostal arteries arising from the false lumen probably occurred because the clamped segment contained the primary tear. In conclusion, the segmental clamp technique in the surgery for aortic dissection may not always be helpful to shorten the duration of spinal cord ischemia during descending and thoracoabdominal aortic repairs. In particular, we should consider cases in which the intercostal arteries, including the Adamkiewicz artery, branch from the false lumen. TEE as well as MEPs is useful for obtaining the details about the blood flow in both the lumens and malperfusion of the intercostal arteries that may cause spinal cord injury.

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eComment: Features of the spinal cord collateral pathways in presence of pathology and opportunity of their usage during main stage of surgical procedure

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We would like to show our appreciation to the authors for this up-to-date publication, and also for the openness and determination to discuss the negative results in a wide press [1].

The problem of paraparesis and paraplegia following surgical procedures on thoracic and thoracoabdominal aorta could not be limited to discussion of methods of intra-operative spinal cord defense (assisted circulation; mild hypothermia; CSF drainage; medical defense). Besides well known anatomic features of spinal cord blood circulations (variety of arteria radicularis magna (Adamkewicz) origin; inconstancy and intermittence of anterior spinal artery, limited number of radiculo-medullaris arteries and terminal branches of anterior spinal artery in diameter and intermittence of its pathway) in such patients we should also be aware of individual features (number of radiculo-medullaris arteries and placement of their origins, opulence of collateral spinal cord circulation pathway, length of aortic lesion; critical aortic zone blood supply features; suspected time of aortic clamping in the zone of 'critical arteries' origin).

We completely share the author's opinion in analysis of spinal stroke development in the patient in the first case history. Adequacy rate of the reasons analysis we were able to note in positive results of operative intervention in the second case history, in which the previous negative experience was considered. But we should note that the method of hypothermic circulatory arrest suggested by the authors has limits and high risk of complication development, particularly in patients with diffuse thoracoabdominal aortic lesion.

We would like to congratulate the authors for the computed solution they found positive results in the second case history. Still it should be repeated that this problem could not be limited to discussion of the intraoperative methods of spinal cord defense. The more detailed analysis of anatomic and individual features of spinal cord blood circulation due to manifested possibilities of the preoperative diagnosis of spinal cord circulation by means of CT and MRT should be performed. Such a detailed analysis allows to evaluate the existence of spinal stroke risks, review features of the spinal cord collateral pathways in presence of pathology and opportunity of their usage during main stage of surgical procedure.

In connection with the above-stated information we are to come back to discussion of anatomic features of spinal cord blood circulation and to bring to your attention the review on the given subject.

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and colleagues [4] reporting no false-positive results and 99% to 100% sensitivities, specificities, positive/negative predictive vales, and accuracies, but our cases suggests otherwise. Magnetic resonance imaging has reported sensitivities and specificities of 95% to 100%. Transesophageal echocardiography, a quick bedside or intraoperative test, despite operator dependence, is also very sensitive (about 98%) and specific (63% to 96%); but clearly, none of these tests are 100% accurate [3].

Although CT scans are known to be extremely valuable, understanding the potential causes for false-positive results is vital. Abnormal venous structures, such as a low-lying left innominate or left pulmonary vein can mimic an aortic flap. A large contrast bolus at the time of the procedure or motion can result in imaging artifacts. Another potential for a false-positive result is a thickened pericardium closely adherent to the ascending aorta [5]. The incidence of false-positive scans that result in negative explorations is unknown and probably underreported. The cause of our initial false-positive scan probably reflects a combination of motion and contrast artifacts from pulmonary artery reflections.

Recognition of the limitations of current technology can present a diagnostic dilemma. Often patients are initially seen at smaller centers that may be less versed in understanding the evaluation of complex and maybe infrequently observed problems, which may lead to overcalling a diagnosis in fear of missing a fatal problem. Hence, these patients are transferred with a potentially erroneous diagnosis.

It is important to recognize that no single test is 100% perfect. Confirmatory studies, especially when clinical judgment questions initial test results, are critical in avoiding the potential for a negative aortic exploration.

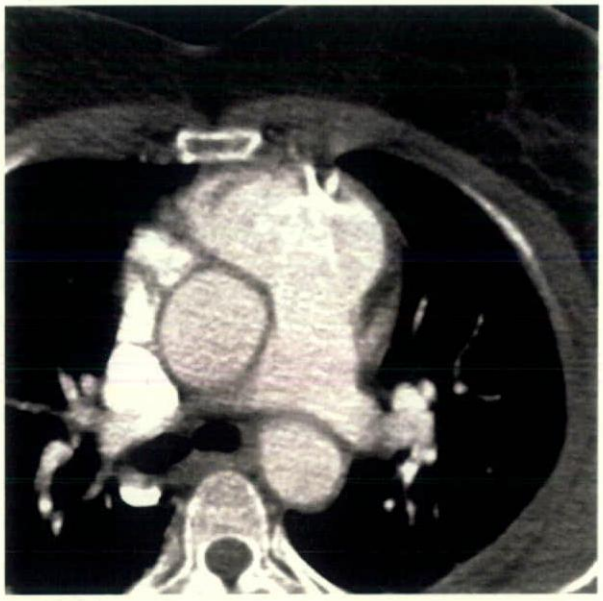

Fig 3. A repeat multidetector, contrast-enhanced, electrocardiograph-gated, computed tomography of the chest demonstrated an aorta of normal sized and caliber. The previously visualized flap was not seen.

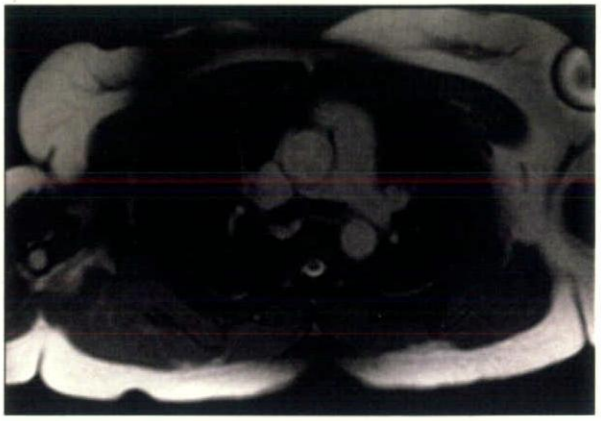

Fig 4. Magnetic resonance imaging (MRI) shows no evidence of intraaortic pathology from the proximal ascending aorta (shown) to the aortic bifurcation.

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Hemolytic Anemia After Operation for Aortic Dissection Using Teflon Felt Strips

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We report three cases of hemolytic anemia caused by anastomotic stenosis after surgical treatment for aortic dissection in which internal and external Teflon (DuPont, Wilmington, DE) felt strips were used for reinforcement of the aortic stump. To detect this complication, laboratory findings typical of red cell fragmentation syndrome

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as well as appropriate imaging modalities are necessary. As a precaution, it is necessary to be meticulous when stitching the internal felt strip.

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Operative survival for aortic dissection has recently improved with advances in surgical techniques and perioperative management; however, there are still several potential early and long-term postsurgical complications. Since 1986, we have used internal and external Teflon (DuPont, Wilmington, DE) felt strips for the reinforcement of the aortic stump. Here, we report three cases of hemolytic anemia caused by anastomotic stenosis after surgical treatment for aortic dissection using felt strips for the reinforcement of the aortic stump.

Case Reports

Patient 1

A 48-year-old man underwent emergency ascending and total arch replacement with an elephant trunk technique for acute type A dissection at the National Cardiovascular Center. We reinforced the proximal stump using internal and external felt strips and gelatin-resorcin-formalin glue. The patient's postoperative course was uneventful. One month later, he presented with high-grade fever, anemia, and systolic murmur of Levine 3/6. The hemoglobin level was 7.3 g/dL, the lactate dehydrogenase (LDH) level was 650 IU/L, the reticulocyte percentage was 6.2%, and schistocytes appeared in the peripheral blood.

Two-dimensional computed tomographic (CT) scans failed to demonstrate any abnormalities at the anastomoses. The transesophageal echocardiography (TEE) showed an abnormal projection in the aortic lumen at the proximal anastomosis. Color Doppler echocardiography demonstrated an acceleration in flow, with a peak velocity of 3.4 m/s.

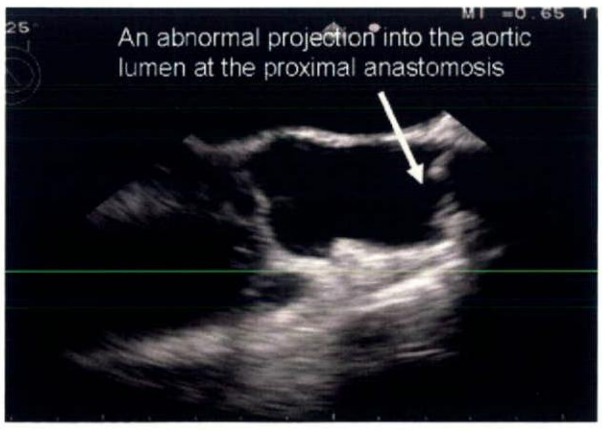

Fig 1. Transesophageal echocardiography image demonstrates an abnormal projection into the aortic lumen at the proximal anastomosis (arrow).

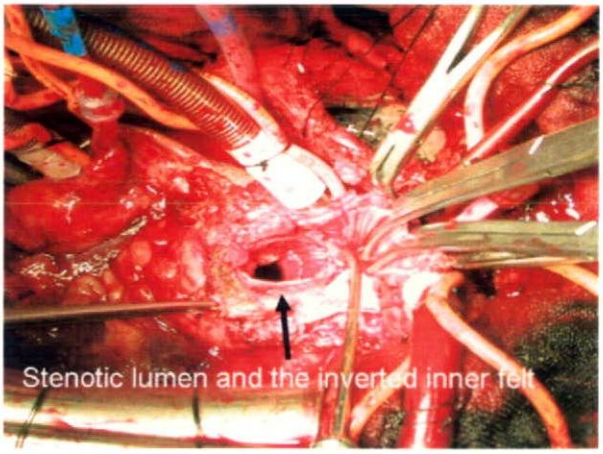

Fig 2. Intraoperative finding shows stenotic aortic lumen (arrow) caused by the circumferentially inverted internal felt strip.

During reoperation, we found the internal felt strip at the proximal anastomosis was turned upward, which reduced the inner diameter to 13 mm. We completely removed the internal felt strip and reanastomosed the previous graft to the ascending aorta. The LDH level decreased to 133 IU/L, and the patient was discharged at postoperative day 21.

According to the pathologic examination, inflammatory cells, including eosinophils, macrophages, and lymphocytes, infiltrated the aorta near the felt strip. During the follow-up for 6 years, laboratory findings have shown no sign of hemolytic anemia, and the latest LDH value was 175 IU/L.

Patient 2

A 60-year-old man underwent emergency ascending and total arch replacement for acute type A dissection at another hospital. Two months later, the hemoglobin level was 9.4 g/dL, the LDH level was 578 IU/L, and schistocytes appeared in the peripheral blood. Two years later, hemoglobin level was 8.7 g/dL, the LDH level was 1535 IU/L, and the reticulocyte percentage was 2.6%. A TEE showed an abnormal projection into the aortic lumen at the proximal anastomosis, which caused an acceleration in the flow, with a peak velocity of 4.2 m/s (Fig 1).

During reoperation, we found that the internal felt strip at the proximal anastomosis was stiff and turned upward, which reduced the inner diameter to 11 mm (Fig 2). We replaced the ascending aorta with a new graft and removed the internal felt strip. The patient's hemolytic anemia was cured, with a reduced LDH level that reached 251 IU/L at discharge. The patient has been followed-up for 1 year, and the LDH level is 167 IU/L now. The latest CT scans showed no sign of anastomotic stenosis.

Patient 3

A 79-year-old woman underwent emergency graft replacement of the descending aorta for acute type B

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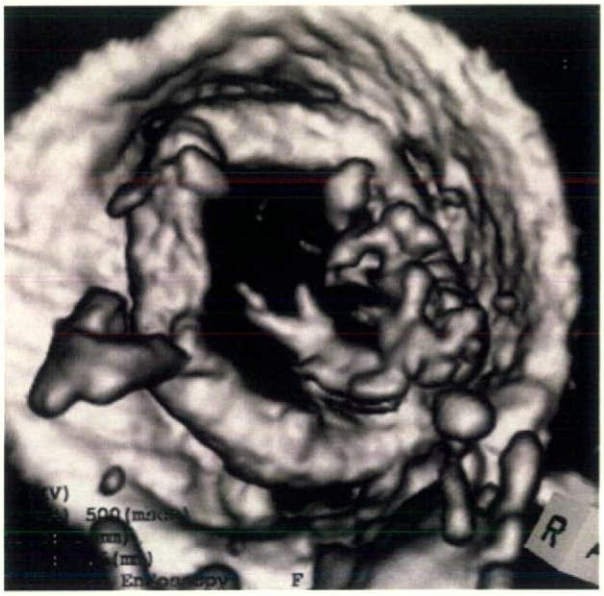

Fig 3. Computed tomographic scan demonstrates stenotic aortic lumen caused by an abnormal projection into the aortic lumen at the proximal anastomosis.

dissection at our hospital. We reinforced the proximal stump using internal and external felt strips and gelatin-resorcin-formalin glue. Two years later, she had dyspnea on exertion. Levels were hemoglobin, 6.6 g/dL, LDH, 1913 IU/L; reticulocyte percentage, 8.4%; and platelet count, 87000/μL. Computed tomography scans (Fig 3) and TEE revealed an abnormal projection in the aortic lumen at the proximal anastomosis, which caused the flow to accelerate. The estimated pressure gradient was 100 mm Hg. Indicator levels in further laboratory examination were fibrinogen, 109 mg/dL; fibrinogen degradation product, 51 μg/mL; thrombin-antithrombin complex, 46 μg/L; and D-dimmer, 23.7 μg/mL.

To reduce the stenosis at the proximal anastomosis, and considering her age and coagulopathy, we performed a palliative axillobilateral femoral bypass. The postoperative echocardiography revealed that the pressure gradient through the stenosis decreased to 30 mm Hg. The LDH level at discharge declined to 459 IU/L, but the bleeding tendency caused by coagulopathy continued. The patient was lost to follow-up after 9 months. The latest LDH level was 455 IU/L.

Comment

Teflon felt has been widely used in cardiovascular operations to reinforce the anastomoses; however, several reports have described complications related to the usage of felt strips. First, it is a foreign body that is subject to infection. Second, it may cause distal embolism. Sogawa and colleagues [1] reported a 60-year-old woman who had multiple cerebral infarctions after an operation for acute type A dissection caused by a mobile thrombus on the internal felt strip. Bedetti and colleagues [2] have also reported a patient presenting with coronary embolism of a felt used in the placement of a Bjork-Shiley aortic valve. Third, like in our patients, the use of felt strips may cause hemolytic anemia. Moreira Neto and colleagues [3] reported 2 patients presenting with hemolytic anemia after mitral valve plasty with the use of Teflon felt strip for posterior annuloplasty. Shingu and colleagues [4] have also reported a similar case to ours, although they did not mention the postoperative follow-up.

In 2 of the 3 patients presented in this report, the proximal part of the internal felt strip was turned upward circumferentially and had stiffened to form stenosis at the anastomotic site. It is unknown whether red cell fragmentation syndrome was caused by the stenosis or by the turbulence caused by the collision of blood with the internal felt strip that was turned upward. In the third patient, the pressure gradient through the stenosis decreased from 100 to 30 mm Hg by means of a palliative axillobilateral femoral bypass, but there still remained a mechanical destruction of red blood cells.

In terms of diagnosis of this complication, the clues are systolic ejection murmur at the anastomotic site, laboratory findings compatible with red cell fragmentation syndrome, including anemia, elevated LDH level and reticulocyte percentage, and schistocytes. Imaging studies are also needed for the diagnosis. In our series, TEE, which clearly demonstrated an abnormal projection in the aortic lumen at the anastomotic site, was more useful than CT scans. The degree of stenosis can be evaluated by a color Doppler technique. In addition, Garcia and colleagues [5] have advocated the efficacy of magnetic resonance angiography for the evaluation of complications in surgically treated aortic dissection.

Laboratory findings in our 3 patients had already shown red cell fragmentation syndrome within a few months after the first operation. We speculated that the syndrome had developed as the internal felt strip became stiffer over time. To prevent this complication, the internal felt strip should be narrow, and stitches should be put in the more proximal portion of the felt strip so that the proximal part will not turn upward.

In summary, hemolytic anemia caused by anastomotic stenosis after surgical treatment for aortic dissection using Teflon felt strips for reinforcement is a rare complication. To detect this complication, laboratory findings typical of red cell fragmentation syndrome as well as appropriate imaging modalities, such as TEE or CT scans, are needed, and necessary precautions should be exercised while stitching the internal felt strip.

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Delayed Improvement in Valve Hemodynamic Performance After Percutaneous Pulmonary Valve Implantation

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We report the case of a 21-year-old woman with a severely stenotic pulmonary homograft who underwent percutaneous pulmonary valve implantation, with no significant change in transvalvular gradient within the 24 hours after the procedure. Major improvement in hemodynamic valve performance of more than 60% decrease in transvalvular gradient and more than 30% increase in pulmonary valve area was observed 3 months after the procedure, showing that hemodynamic improvement can occur late after pulmonary valve implantation. An echocardiogram after 3 months should be done before concluding the procedure failed and that reintervention is necessary.

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Percutaneous pulmonary valve implantation (PVI) has been recently proposed as an alternative to surgical repair for the treatment of diseased right ventricle-to-pulmonary artery conduits [1]. Pulmonary valve implantation generally leads to an immediate and significant improvement of transvalvular gradient in most patients with predominantly stenotic conduits [2]. The present case demonstrates that hemodynamic improvement can occur late after PVI implantation.

A 21-year-old woman with a history of bicuspid aortic valve leading to severe aortic stenosis and insufficiency underwent a Ross procedure at the age of 10 years, with implantation of a 22-mm homograft in the pulmonary position. She presented with exertional dyspnea 11 years after this operation, and the chest roentgenogram showed the presence of significant calcification at the level of the pulmonary homograft. A Doppler echocardiographic examination revealed a peak gradient of 75 mm Hg and mean gradient of 46 mm Hg across the pulmonary homograft, a pulmonary valve effective orifice area of 0.65 cm^2, and moderate pulmonary insufficiency. The patient underwent maximum exercise stress test. The peak workload was 150 W, and oxygen consumption was 32 mL/kg/min. The transvalvular gradients increased markedly during exercise (peak, 110 mm Hg; mean, 74 mm Hg). The decision was made to implant a percutaneous pulmonary valve, and written informed consent was obtained from the patient.

The procedure was performed by femoral approach, under general anesthesia. Right systolic ventricular pressure was 60 mm Hg, with a peak systolic gradient across the pulmonary homograft of 42 mm Hg. Angiography performed at the level of the pulmonary homograft allowed the calculation of a minimal lumen diameter of 16 mm at the mid portion of the homograft.

A Melody valve (Medtronic, Minneapolis, MN), which consists of a bovine jugular valve sutured within a platinum-iridium stent, was mounted in a 22-mm balloon delivery system and inserted through an extra-stiff guidewire up to the pulmonary homograft. After valve implantation, balloon dilation was performed with a 20-mm Mullins high-pressure balloon (Numed Canada Inc, Cornwall, Ontario, Canada).

At the end of the procedure, the right systolic ventricular pressure was 56 mm Hg, and the peak transvalvular gradient was 39 mm Hg. Pulmonary angiography showed the absence of pulmonary regurgitation (Fig 1). The day after the procedure, Doppler echocardiography showed no improvement in the peak transvalvular gradient (75 mm Hg) and only slight improvement in the mean transvalvular gradient (37 mm Hg) compared with values before the procedure. There was, nonetheless, a moderate improvement in valve effective orifice area (0.96 cm^2). No right ventricular outflow tract obstruction was observed at anytime. A chest roentgenogram showed the correct position of the valve in the pulmonary homograft and the absence of any stent fracture.

The possibility of immediate reintervention (surgical homograft replacement or homograft stent implantation) was considered, but after a meeting with the physician responsible for the patient and the interventional team, it was decided that close clinical follow-up rather than immediate reintervention would be undertaken.

In the weeks after intervention, the patient reported a progressive improvement of symptoms and was completely asymptomatic at the 3-month follow-up. At that time, the chest roentgenogram showed the correct position of the valve, the absence of any stent fracture, and no significant change in stent diameters compared with those obtained 24 hours after the procedure. Doppler echocardiography revealed significant improvement in the transvalvular gradient (peak, 26 mm Hg; mean, 16 mm Hg), further improvement in the effective orifice area

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Evolving arch surgery using integrated antegrade selective cerebral perfusion: Impact of axillary artery perfusion

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Objective: The study objective was to determine the impact of integrated antegrade selective cerebral perfusion with right axillary artery perfusion during arch surgery.

Methods: All surgeries were performed through a median sternotomy. Direct cannulation of the right axillary artery in the axilla was used for cardiopulmonary bypass and antegrade selective cerebral perfusion under hypothermia. In addition, ascending aortic or femoral artery perfusion was used. The clinical records of 531 patients (median age, 72 years) between 1999 and 2006 were reviewed, of whom 137 patients (25.8%) underwent emergency surgery. There were 164 dissecting and 367 nondissecting aortic lesions. The surgeries included total arch replacement in 431 patients, partial arch replacement in 9 patients, and hemiarch replacement in 91 patients.

Results: The early mortality rate was 4.0% (2.3% of 30-day mortality and 1.7% of in-hospital mortality). The incidence of permanent neurologic dysfunction was 2.9% in all (3.3% in total arch replacement and 1.0% in hemiarch or partial arch replacement). The incidence of temporary dysfunction was 9.9% in all (10.6% in total arch replacement and 7.0% in hemiarch or partial arch replacement). Multivariate analysis demonstrated that the risk factors for early mortality were chronic renal failure, ruptured nondissecting aneurysm, and prolonged surgery. The midterm survival was 87.2% ± 1.7% at 3 years and 80.5% ± 2.6% at 5 years.

Conclusion: Right axillary artery perfusion is an advantageous adjunct to cardiopulmonary bypass and antegrade selective cerebral perfusion in arch surgery.

Surgery for various aortic arch pathologies, including acute aortic dissection or ruptured atherosclerotic aneurysms, still features high mortality and morbidity.[1-5] In particular, postoperative cerebral morbidities remain prevalent despite recent great advances in intraoperative brain protection.[1-5] We have routinely used antegrade selective cerebral perfusion (SCP) under right axillary artery (RAxA) perfusion in conjunction with deep or moderate hypothermia.[6] The aim of this retrospective study is to determine the early and midterm outcome of arch surgery using integrated SCP with RAxA perfusion and to analyze relevant risk factors for early mortality and cerebral morbidity.

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Patients and Methods

Patients

We reviewed the clinical records of 531 patients who underwent various aortic arch surgeries between 1999 and 2006 at the National Cardiovascular Center, Japan (Table 1). The median age was 72 years (19–89 years). Routine clinical history and physical examination, including evaluation of brain, coronary, and peripheral ischemia, were done preoperatively. Particularly in regard to cerebral ischemia, brain computed tomographic scanning and carotid ultrasound were routinely performed. With any positive findings, further magnetic resonance imaging/angiography and Diamox loading cerebral flow scintigraphy followed, together with consultation

Abbreviations and Acronyms
CPB = cardiopulmonary bypass
RAxA = right axillary artery
SCP = selective cerebral perfusion

with neurophysicians or neurosurgeons. In 3 patients with carotid artery stenosis, carotid artery endarterectomy in 1 patient and carotid artery stenting in 2 patients were performed before the arch surgery. For coronary ischemia, dipyridamole loading Thallium perfusion scintigraphy was a routine examination in the early series. Positive signs warranted coronary angiography. Since 2004, coronary angiography has been a routine examination. When coronary artery disease was detected, simultaneous coronary artery bypass grafting was carried out in almost all of the patients, or percutaneous catheter intervention was performed for some limited patients by cardiologists. On an emergency basis, including acute dissection or ruptured nondissecting aneurysm, these complete examinations were not feasible.

Surgical Techniques and Brain Protection

Bilateral temporal arterial lines were placed to monitor cerebral perfusion pressure. All aneurysms were approached through a median sternotomy.

1. Cardiopulmonary bypass (CPB) establishment with RAxA perfusion: RAxA perfusion was routinely used to establish CPB and SCP.[6] The RAxA was exposed quickly through a 5 to 7-cm skin incision in the axilla (Figure 1). After full heparinization, a 10F to 16F straight thin-walled cannula (Medtronic, Minneapolis, Minn) was inserted into the RAxA, depending on the artery size. Empirically, even a 12F cannula allowed a flow of up to 1.5 L/min. In addition, the ascending aorta or femoral artery was cannulated for a total CPB flow of 1 to 2 L/min through the RAxA and 2.5 to 3.5 L/min via the ascending aorta or femoral artery. In the early series, femoral perfusion was routinely chosen. However, from 2003 to 2006, ascending aortic perfusion was predominantly used, although femoral perfusion was still an alternative for patients with a severely atherosclerotic ascending aorta. With severely atheromatous changes in the descending aorta, femoral perfusion was used to flush out debris that dislodged during the distal anastomosis. Bicaval venous drainage with left ventricular venting was performed. For patients with acute dissection, with standard femoral cannulation, RAxA perfusion was used to avoid the collapse of the true channel caused by retrograde femoral perfusion. In 4 patients whose left subclavian or innominate artery was occluded, left axillary perfusion was also used. In addition to RAxA perfusion, the ascending aorta was used in 141 patients, the ascending aorta and femoral artery were used in 57 patients, and the femoral artery was used in 333 patients.
2. Brain protection using SCP with RAxA perfusion (Figure 2, *A*): Alpha-stat management was used during hypothermia. We routinely used SCP combined with RAxA perfusion for brain protection.[6,7] Immediately after the induction of hypothermic circulatory arrest by discontinuation of the ascending or femoral perfusion, SCP with the constant flow through RAxA perfusion was easily commenced by clamping the innominate artery. The left common carotid and left subclavian arteries were also clamped. By means of this simple maneuver, sufficient perfusion of the right hemisphere with partial circulation of the left hemisphere through collateral vessels was quickly established. The ascending aorta was usually clamped, and cardioplegic solution was antegradely or retrogradely infused to attain cardiac arrest.

In the early series, the lowest bladder and nasopharyngeal temperatures were 20°C to 22°C. Both temperatures have been increased in stages up to the current lowest temperature of 28°C, except for high-risk patients with carotid or intracranial artery disease, or for patients with renal failure. For these patients, deep hypothermia was still used. The lowest temperature was 20°C in 151 patients, 22°C in 81 patients, 25°C in 141 patients, and 28°C in 158 patients.

3. Total arch replacement (Figure 2, *B*): The ascending aorta and transverse arch were opened, and a 12F or 14F SCP

TABLE 1. Patient profile (n = 531)

Variable	No	%
Gender		
Male	363	68.4
Female	168	31.6
Aortic pathology		
Dissecting	164	30.9
Nondissecting (+ dissecting)	367 (39)	69.1
Cause		
Atherosclerosis	357	67.2
Dissection	151	28.4
Marfan syndrome	11	2.1
Aortitis	9	1.7
Infection	2	0.4
Urgency		
Elective	394	74.2
Emergency	137	25.8
Acute dissection	108	20.3
Ruptured nondissecting	21	4.0
Others[a]	8	1.5
Reoperation	42	7.9
Cerebrovascular disease		
History of cerebrovascular accident	120	22.6
Carotid disease	54	10.2
Intracranial artery disease	52	9.8
Coexisting disease		
Coronary artery disease	145	27.3
Chronic renal failure	53	10.0
Chronic obstructive pulmonary disease	99	4.0
Smoking	267	50.2
Diabetes mellitus	71	13.4
Lowest core temperature		
Deep hypothermia (20°C–22°C)	232	43.7
Moderate hypothermia (25°C–28°C)	299	56.3

[a]Others: unstable aneurysm (eg, symptomatic nondissecting aneurysm, infected aneurysm).

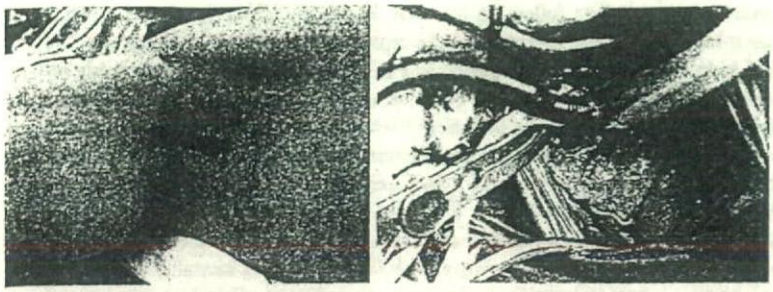

Figure 1. RAxA perfusion in the axilla. The distal part of the RAxA was exposed through a 5- to 7-cm skin incision in the axilla. A 10F to 16F straight thin-walled cannula was inserted depending on the size of the artery.

balloon-tipped cannula was inserted from within the aorta into the left common carotid artery. Between 1999 and 2002, SCP was instituted by RAxA and left common carotid artery perfusion, with the left subclavian artery clamped, at 20°C to 22°C. During SCP, the pressures of the bilateral superficial temporal artery or the balloon tips were maintained in the range of 30 to 50 mm Hg. Subsequently, SCP flows of 10 to 12 mL/kg/min were generated by a single roller pump separate from the systemic circulation. In 2003, left subclavian perfusion using another balloon-tip cannula was added, and the lowest temperature has been gradually increased from 25°C to 28°C. At 28°C, the SCP flow was also increased up to approximately 15 mL/kg/min to maintain perfusion pressure between 50 and 70 mm Hg. Through the aneurysm, the descending aorta distal to the aneurysm was transected from the inside using an electrical cautery, avoiding nerve and lung injury. Open distal anastomosis was performed during hypothermic circulatory arrest of the lower half body. "Stepwise distal anastomosis," with a short-length tube graft interposition, was frequently used for an easy and secure anastomosis. The details have been described.[6,7] An invaginated tube graft of 7 to 12 cm in length, composed of the multibranched Dacron graft, was inserted into the descending aorta. The proximal end was anastomosed to the descending aorta. The distal end of the inserted graft was then extracted proximally. The multibranched arch graft was connected to this interposed graft. Systemic circulation was resumed using a branch graft. The left subclavian artery was reconstructed, and the patient was rewarmed to 30°C to 32°C with an SCP flow of 1 to 1.5 L/min and a branch graft flow of 3 to 4 L/min. Then, the proximal aortic anastomosis above the sinotubular junction was made. Coronary circulation was initiated by unclamping. The other arch vessels were reconstructed, and the patient was fully rewarmed with a slightly higher total flow of 4 to 6 L/min. In patients with a risk of cerebral morbidity because of carotid or intracranial artery lesions, arch-vessel reconstruction was preferentially performed before the proximal aortic anastomosis. For 26 patients with extensive aneurysm involving the arch, 2-stage surgery was performed with stage I total arch replacement with an elephant trunk procedure. It was followed by stage II descending aortic replacement in 12 patients and by endoluminal stent grafting in 6 older patients with respiratory dysfunction.

4. Hemiarch or partial arch replacement: The proximal arch was beveled proximal to the innominate artery. SCP balloon-tipped cannulae were inserted from within the aorta into the left common carotid artery with or without the left subclavian artery perfusion. A 22 to 26-mm single-branched Dacron graft was anastomosed with an open aortic technique. Distal perfusion was commenced using a branch-graft, and the patient was rewarmed. Then, the proximal anastomosis was made. In case of partial arch replacement, the innominate artery was reconstructed using an 8 to 10-mm branch graft.
5. Acute type A dissection: Hemiarch repair was the procedure principally used for our tear-oriented surgery. The false channel was closed with inside-outside Teflon felt strips. Extended total arch replacement was attempted in the following settings: 1) tear in the arch or proximal descending aorta, 2) Marfan syndrome, 3) arch aneurysm or dilatation, 4) atheromatous arch, 5) massive arch dissection, and 6) relatively

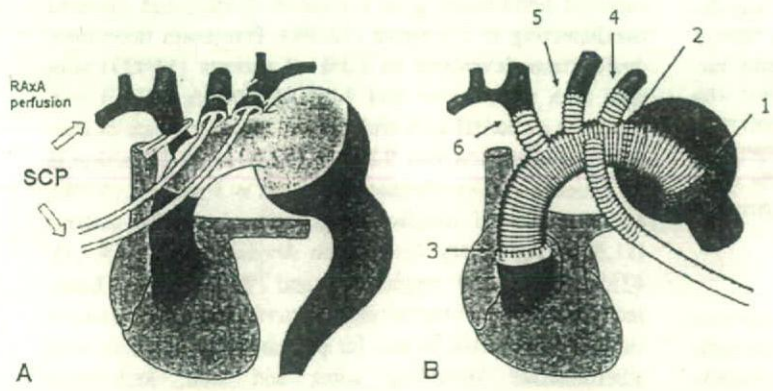

Figure 2. Total arch replacement using SCP with RAxA perfusion. A, Antegrade SCP with the perfusion through the RAxA, left common carotid artery, and left subclavian artery. B, Total arch replacement with a stepwise distal anastomosis using a multibranched Dacron graft. The numbers 1 to 6 show the turn of anastomosis. *RAxA*, Right axillary artery; *SCP*, selective cerebral perfusion.

young age (<70 years). The descending aorta was transected distal to the left subclavian artery. A modified elephant trunk technique was used for a secure anastomosis and early thrombosed closure of the distal false channel. A prosthetic graft of 16 to 24 mm in diameter was inserted into the distal true channel. The proximal end was attached to the descending aorta with the reinforcement with an external Teflon felt strip. Another multibranched arch graft was anastomosed to this aortic stump. Antegrade distal aortic perfusion was commenced with a branch graft. The left subclavian artery was reconstructed. At the proximal site, the ascending aorta was transected just around the sinotubular junction. The proximal false channel was closed with internal and external Teflon felt strips. In most, the proximal false channel was fixed using Gelatin-Resorcin-Formal glue (Cardial, Sainte-Etienne, France). The main graft was anastomosed to this end. Finally, the other arch vessels were reconstructed using branch grafts under SCP.

Arch repairs included total arch replacement in 431 patients, partial arch in 9 patients, and hemiarch in 91 patients. Concomitantly, coronary artery bypass (n = 95), composite root repair (n = 21), valve-sparing surgery (n = 6), sinotubular junction plication (n = 27), aortic valve replacement (n = 29), mitral valve repair (n = 3)/replacement (n = 3), tricuspid annuloplasty (n = 6), atrial septal defect closure (n = 1), arch-vessel reconstruction for the anomaly (n = 9), and peripheral bypass (n = 2) were performed. At the end, the cannula was removed from the RAxA, and the cannulation site was closed using a 6-0 polypropylene suture.

Definition of Neurologic Deficits and Other Variables

Permanent neurologic dysfunction was defined as the presence of permanent deficits persisting at discharge. Transient dysfunction was defined as the occurrence of confusion, agitation, obtundation, or delay of full awakening. It was difficult to distinguish between transient neurologic dysfunction and frequently occurring delirium in the elderly. When delirium was severe, it was also included in transient neurologic dysfunction. Cerebrovascular event included old cerebral infarction (including asymptomatic one) in 116 patients and cerebral hemorrhage in 4 patients. Carotid artery disease was defined as the presence of more than 50% stenosis or multiple plaques on ultrasound examination. Intracranial artery disease was defined as more than 75% stenosis of intracranial vessels on magnetic resonance angiography or as hypoperfusion on Diamox loading cerebral flow scintigraphy. Chronic renal failure was defined as a serum creatinine level of more than 1.5 mg/dL or the requirement of hemodialysis. Chronic obstructive pulmonary disease was defined as forced expiratory volume in 1 second less than 70% of the normal value. Reoperation was defined as re-sternotomy surgery after cardiac or aortic root to arch surgery. The patients who had previously undergone proximal descending replacement through left thoracotomy were also included among the reoperation cases, because it was sometimes troublesome to make the distal anastomosis to the prosthetic graft of the previous proximal descending aortic replacement.

Data Collection and Statistical Analysis

All of the surgeries were identified from the Registry of Cardiovascular Surgery in the National Cardiovascular Center. The data in the registry were approved for use by the institutional ethical committee. The follow-up rate was 99.6% (529/531), because 2 patients who underwent total arch replacement were lost to follow-up. The mean follow-up period was 2.7 ± 1.8 years. We retrospectively reviewed the overall outcome and investigated the risk factors for early mortality, including 30-day and in-hospital mortality, and permanent neurologic dysfunction. Statistical analysis was carried out using StatView 5.0 (SAS Institute, Cary, NC) software. Values were expressed as the mean ± standard deviation or medians (range). Univariate and multivariate logistic regression analyses were used to investigate risk factors for early mortality and permanent neurologic dysfunction. According to clinical importance and the result of univariate analysis, advanced age, ruptured nondissecting aneurysm, coronary artery disease, chronic renal failure, diabetic mellitus, concomitant surgery, prolonged surgery, and deep hypothermia (20°C–22°C) were involved in the following multivariate analysis for early mortality. For permanent neurologic dysfunction, multivariate analysis was not performed because of a low event rate and strong association of atheromatous ascending aorta and arch. Kaplan–Meier estimate was used to calculate the survival.

Results

The hypothermic circulatory arrest for open distal anastomosis, myocardial ischemia, SCP, CPB, and surgery lasted 57.0 ± 19.9 minutes, 129.7 ± 45.8 minutes, 134.6 ± 62.3 minutes, 233.3 ± 106.3 minutes, and 483.3 ± 189.7 minutes, respectively. The early mortality rate was 4.0% (21/531). Within 30 days, 12 patients (2.3%) died: acute dissection in 3 patients (2.7%), chronic dissection in 1 patient (1.9%), nonruptured nondissecting aneurysm in 6 patients (1.8%), and ruptured nondissecting in 2 patients (2.7%). The in-hospital mortality was 1.7% (9/519): acute dissection in 1 patient (0.9%) and nonruptured nondissecting aneurysm in 8 patients (2.4%). Table 2 shows the result of univariate and multivariate analyses of risk factors for early mortality. The multivariate analysis demonstrated that the risk factors for early mortality were chronic renal failure, ruptured nondissecting aneurysm, and prolonged surgery.

The presence of cerebral complications could not be assessed in 8 patients who died immediately after the surgery without awakening. The incidence of permanent neurologic dysfunction was 2.9% (15/523): acute dissection in 4 patients (3.6%), chronic dissection in 1 patient (1.9%), nonruptured nondissecting in 7 patients (2.1%), and ruptured nondissecting in 3 patients (12.0%). Permanent neurologic dysfunction developed in 3.3% of patients (14/423) with total arch replacement and 1.0% of patients (1/100) with hemiarch or partial arch replacement. The incidence of temporary dysfunction was 9.9% (52/523): acute dissection in 10 patients (9.1%), chronic dissection in 3 patients (5.7%), and nonruptured nondissecting aneurysm in 39 patients (11.6%). Temporary dysfunction developed in 10.6% (45/423) with total arch replacement and 7% (7/100) with hemiarch or partial arch replacement. Univariate analysis demonstrated that the risk factors for permanent dysfunction were atheromatous ascending aorta and arch, arch-vessel