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Review Article

Acute Type A Aortic Dissection: for Further Improvement of Outcomes

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Despite improved outcomes of acute type A aortic dissection (AAAD), many patients die at the moment of onset, and hospital mortality is still high. This article reviews the latest literature to seek the best possible way to optimize outcomes. Delayed diagnosis is caused by variation in or absence of typical symptoms, especially in patients with neurological symptoms. Misdiagnosis as acute myocardial infarction is another problem. Improved awareness by physicians is needed. On arrival, quick admission to the OR is desirable, followed by assessment with transesophageal echocardiography, and malperfusion already exists or newly develops in the OR; thus, timely diagnosis without delay with multimodality assessment is important. Although endovascular therapy is promising, careful introduction is mandatory so as not to cause complications. While various routes are used for the systemic perfusion, not a single route is perfect, and careful monitoring is essential. Surgical treatment on octogenarians is increasingly performed and produces better outcomes than conservative therapy. Complications are not rare, and consent from the family is essential. Prevention of AAAD is another important issue because more patients die at its onset than in the following treatment. In addition to hereditary diseases, including bicuspid aortic valve disease, the management of blood pressure is important.

Keywords: aortic dissection, malperfusion, complication, echocardiography

Acute type A aortic dissection (AAAD) not only causes sudden death at the time of onset but leads to various life-threatening complications. In spite of improved diagnostic imaging and surgical strategies, in-hospital mortality is still high, and patients who have survived often suffer from sequelae that make subsequent quality of life poor. This article is aimed to review the latest literature and ideas to seek the best possible way to minimize undesirable results.

Various complications of AAAD caused by separation of the aortic wall to outer and inner layers are summarized as three categories: 1) aortic rupture due to disruption of the outer layer; 2) malperfusion caused by the inner layer, that is, the intimal flap; and 3) aortic regurgitation by a deformed aortic valve, leading to acute heart failure. Approximately 20% of patients instantaneously die due to aortic rupture,[1] and 1 to 2% of the remaining patients die every hour unless appropriately treated.[2] Although surgical outcomes are gradually improving, in-hospital mortality of surgical patients (repair on the aortic root to the arch) in 2009 was as high as around 10%, according to the Japanese database.[3] For the further improvement of outcomes, problems should be solved in each of pre-onset, pre-hospital, and in-hospital stages. In this review, the latter two stages are discussed first, since most of the literature focuses on these stages. After that, the issue of pre-onset stage, i.e., prevention is discussed.

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Delayed Diagnosis

In consideration of the natural history of AAAD, the "chain of onset-to-surgery" should be as short

as possible. Delayed diagnosis is the initial pitfall. Harris et al. reported that variables associated with delayed diagnosis included an initial visit to a non-tertiary care hospital,[4] indicating that awareness of general physicians is to be improved. Even recently, there are not a few reports of misdiagnosis at this stage.

Despite that chest to back pain is a typical symptom of AAAD, it can be masked in the presence of neurological symptoms. Gaul et al. reported that chest pain was apparent in 94% of 72 AAAD patients without neurological symptoms but was only apparent in two thirds of 30 patients with symptoms.[5] Similarly in Japan, Imamura et al. found that 16 of 98 patients (17%) had no pain.[6] The painless group more frequently had consciousness disturbance or other neurological symptoms and had more unfavorable outcomes. When patients present with neurological symptoms, such as paraparesis[7] or seizure,[8] they may be regarded as a primary neurological disorder. When a focal neurologic deficit such as hemiplegia is manifest, thrombolytic therapy can be started as the initial treatment for cerebral infarction. If AAAD causes this symptom, however, it leads to a catastrophic outcome. Chiang et al. reported a case of manifestation of cerebral ischemia, in which thrombolytic treatment was interrupted just in time, based on the duplex finding of dissection in the carotid artery.[9]

When a patient has a sudden onset of chest pain, with laboratory data and ECG findings compatible with acute myocardial infarction (AMI), the patient can be taken to have catheterization, or even tissue plasminogen activator may be given before AAAD is ruled out. Needless to say, the latter leads to catastrophic results.[10] Hirata et al. reported that ECG was normal, only in 27.0% of AAAD patients: ST elevation was closely related to direct coronary involvement, while ST depression or T wave changes were often noted in shock and cardiac tamponade.[11] Rapezzi et al. found that troponin positivity and acute coronary syndrome-like ECG was one of the pre- and in-hospital risk factors for the diagnostic delay of AAAD.[12] Luo et al. reported that in 11 of 239 AAAD cases that appeared to be AMI, 6 patients died[13]; all 6 had undergone CAG without the diagnosis of AAAD, but eccentric AR or elevation of D-dimer could have been used as a clue to suspect AAAD earlier. Courand et al. presented a case of LMT occlusion, in which CAG was done, based on the ST elevation in $aV_{R,}$ but the LMT could not be catheterized.[14] Although subsequent angiography and TEE revealed AAAD, the patient died. Unawareness of cardiologists remains an unsolved problem at the present moment.

Besides ECG, chest X-ray is another diagnostic tool used in non-tertiary care hospitals. Although a widened mediastinum in a chest roentgenogram is an important finding, which suggests the presence of AAAD,[15] not a few patients present with normal mediastinum shadow; thus, aortic dissection cannot be ruled out, only with chest X-ray findings.[16]

Among blood examinations, D-dimer can be useful for quickly suspecting or ruling out aortic dissection. Negative D-dimer (<500 ng/mL) in patients with chest pain makes the presence of AAAD or pulmonary embolism unlikely.[17,18] Sbarouni et al. reported that a cutoff level of 700 ng/mL was useful, not only for suspecting acute aortic dissection, but also for ruling out chronic aortic aneurysm.[19]

Transthoracic echocardiography can be a promising modality, which potentially provides the definite diagnosis of AAAD at bed side.[20,21] Cecconi reported that the sensitivity, specificity, and positive and negative predictive values for diagnosing AAAD were 87%, 91%, 75%, and 95%, respectively. These were much higher in cases that had good image quality: 97%, 100%, 100%, and 99%, respectively.[22] The recently developed smartphone-sized echo device may further increase the chance of echo assessment in patients with chest symptoms, even prior to ECG or chest X-ray. Although it may not be easy to detect an intimal flap or rule out AAAD only with echo,[23] unusual pericardial effusion, eccentric aortic regurgitation, or asynergy, or presence of flap in the peripheral arteries may be an important clue for making the early diagnosis of AAAD.

Use of TEE for the initial diagnosis of AAAD was started in the 1980s because of its excellent resolution, compared to the CT imaging of those days.[24] Kyo et al. reported that intraoperative TEE was useful for detecting flap and false lumen (detection rate 97.6%) as well as for detecting coronary lesion and malperfusion in the descending aorta.[25] Since TEE is rather invasive for AAAD patients, it is better used under general anesthesia.

Tremendously improved CT performance has enabled the definite diagnosis of AAAD by providing abundant information on morphology and hemodynamics.[26] The 256 or more multidetector row CT has reduced the time for visualization to several seconds[27] and even enables identification of an entry.[28] Since CT has become available in the majority of emergency department or hospitals, it is currently the first choice examination when aortic dissection is suspected.

Aortic rupture is unpredictable and often brings an end to elaborate treatment. While delayed diagnosis should be avoided, pre-hospital management needs to be optimized. Marek et al. reported a case of aortic rupture while the patient was informed of a life-threatening disease.[29] Winsor et al. pointed out that hemodynamic management before transport was often inadequate in patients with suspected AAAD.[30] It is of primary importance to consider the treatment goal every moment and exclude every obstacle against it. Chavanon et al. reported direct admission to the OR of patients with highly suspected AAAD.[31] The definite diagnosis of AAAD was then made by means of TEE under anesthesia, and AAAD was immediately operated on in 197 of 245 patients (80.4%), after direct admission to the OR. Although this may be an extreme strategy, one should keep in mind that every preoperative assessment is done in exchange of the risk of unpredictable rupture, which potentially leads to the finale or subsequent sequelae. Once the diagnosis of AAAD is confirmed, the patient should be taken to the OR, and all other information is obtained with TEE.[32,33]

However, many surgeons are reluctant to rely on TEE because it necessitates expertise, or they believe that it is not the task of surgeons, but of anesthesiologists or cardiologists. It is the other way around. If it effectively improves surgical outcomes, surgeons should willingly be innovative or strongly urge co-working anesthesiologists to learn it, since the aim of every effort is to enhance the efficacy of treatment. In the following sections, specific applications are described.

Malperfusion

Malperfusion caused by AAAD poses several problems: 1) limited preoperative assessment, 2) surgery in the presence of organ damage, 3) new malperfusion under artificial perfusion, and 4) irreversible damage leading to sequelae. It is well documented that the presence of malperfusion increases mortality and postoperative complications.[34,35] Geirsson et al. examined 221 surgical patients for AAAD, including 59 patients with malperfusion, and reported that in-hospital mortality was significantly higher in patients with malperfusion than in those without it (30.5% vs. 6.2%, P <0.001).[34] Along with immediate, aortic repair for preventing rupture, perfusion in an ischemic organ needs to be restored as early as possible.

Coronary artery

Neri et al. reported that the coronary artery was involved in 24 of 211 AAAD cases (11.3%) that had a poor prognosis, with an in-hospital mortality of 20%, because of unsuccessful weaning from cardiopulmonary bypass or postoperative low cardiac output.[36] Coronary obstruction is caused mostly by compression of ostium or proximal portion of coronary artery due to an expanded false lumen[36,37] or uncommonly by intimal flap[38] or disruption of the intima at the orifice.[36]

Until recently, CAG has been the only measure for making the diagnosis of coronary artery involvement. Ramanath et al. retrospectively analyzed 1343 AAAD cases and reported that preoperative CAG did not significantly increase in-hospital mortality (25.6% with CAG vs. 20.6% without CAG, P = 0.14).[39] However, mortality was over 20% in both groups, probably because malperfusion in multiple organs was involved in both groups. Overall management of ischemia in every organ is needed.

Recently CT has enabled assessment of coronary involvement in AAAD.[40] Rosskoph et al. compared the CT angiography and CAG and reported the sensitivity, specificity, positive and negative predictive value was 33%, 99%, 50%, and 99%, respectively.[41] CT assessment has greatly reduced invasiveness and time delay before surgery. However, it is questionable whether additional CT angiography for assessment of coronary involvement is essential in patients with the definite diagnosis of AAAD.

TEE is useful for assessing coronary malperfusion, especially in patients who have been directly admitted to the OR. Because AAAD predominantly affects the ostium and proximal portion of the coronary artery, it is clearly visualized with TEE.[37,42] Asynergy of the left ventricle is an indirect but useful finding indicating significant coronary malperfusion. Furthermore, malperfusion can newly develop during surgery due to false lumen perfusion[43] or even after aortic repair.[44] TEE is helpful for identifying the mechanism of malperfusion, thus making an immediate and appropriate strategy, leading to survival of both patients without complications. An intraoperative event is beyond the capability of CT assessment.

Tominaga et al. reported two cases of LMT involvement.[45] Although both patients survived, left ventricular function remained depressed, which limited their QOL. They suggested that not only timely surgery but also preoperative placement of perfusion catheter could be advantageous. Catheter intervention to the LMT can

work as a bridge to surgery, although it takes time before surgery.[46)]

Cerebral malperfusion

Cerebral malperfusion causes neurological complications, which is related to increased mortality[47)] and also leads to deteriorated quality of life, even if the patient survives. Morimoto et al. reported that time to surgery was a predictor of lack of improvement.[48)] In this regard, pre-hospital factor as well as "door-to-OR" time is important.

Surgeons often hesitate to perform aortic repair in cases with significant neurologic findings, especially, the comatose state or apparent evidence of cerebral infarction. Tsukube et al. reported surgical outcomes of 27 cases with the comatose state among 181 AAAD patients, including 21 patients with immediate surgery before 5 hours from the onset and 6 patients without it.[49)] Brain protection during surgery was deep hypothermia with antegrade cerebral perfusion, followed by therapeutic hypothermia and magnesium treatment postoperatively. In the former group, hospital mortality was lower (14% versus 67%), and incidence of full consciousness recovery was higher (86% versus 17%). Other investigators stated that neurologic findings may be reversible, and immediate surgery should not be contraindicated.[50,51)] Broad cerebral infarction, however, may cause severe brain edema by cardiopulmonary bypass. In such cases, delayed surgery may be recommended.[52)]

Strategies for cerebral perfusion varies among institutions. After retrograde cerebral perfusion was attempted,[53)] antegrade cerebral perfusion has recently gained popularity.[54,55)] However, status in the skull varies among individual patients, and unexpected malperfusion may occur under artificial perfusion. It should be detected and corrected without delay. For this purpose, appropriate monitors for cerebral perfusion are essential.

Near-infrared spectroscopy provides real-time information on oxygen debt in brain tissue. Sustained reduction of regional oxygen saturation in the brain tissue (rSO_2) is related to neurological outcomes.[56)] However, the rSO_2 drop is not specific to malperfusion but is also affected by impaired oxygenation, low hemoglobin level, or venous congestion. Since reduced rSO_2 does not identify its cause, it is necessary to examine whether malperfusion is present or not.

Although ultrasonography of neck vessels was employed in 1990s,[57)] it cannot rule out an obstruction in the internal carotid artery. Temporal artery pressure monitoring bears the same limitation. Transcranial doppler has been applied to assess blood flow in the skull.[58)] During cardiopulmonary bypass, however, it is often difficult to know whether undetectable flow indicates malperfusion or is due to a technical error. Thus, the author devised orbital Doppler to assess the blood flow in the central retinal artery using a transthoracic echo probe.[59)] When uni- or bilateral blood flow is undetectable, it indicates the presence of malperfusion in the internal carotid artery. The reason for cerebral malperfusion is often found in the arch or branch arteries. Coletti et al. assessed the arch flow with TEE.[60)] The TEE can visualize each of three arch branches and is helpful for identifying the mechanism of malperfusion.[61)]

Mesenteric ischemia

Mesenteric ischemia considerably deteriorates prognosis. Di Eusanio et al. reported that mesenteric ischemia was found in 3.7% of AAAD patients (68/1809) and was associated with high mortality: 63.2% with mesenteric ischemia compared with 2.38% without (P <0.001).[62)] Delayed diagnosis on mesenteric ischemia remains a problem. Abdominal pain is caused not only by bowel ischemia but also often by the aorta itself. As in chest pain, abdominal pain can be masked in patients with neurologic symptoms. Physical examination often fails to detect it until sign of peritonitis becomes apparent, that is too late for salvaging the intestine.

Angiography is useful for diagnosing mesenteric ischemia when the patient presents with symptoms suggesting ischemia such as melena.[63)] CT angiography enables noninvasive assessment of mesenteric perfusion. Kurimoto et al. reported that the superior mesenteric vein/artery ratio was helpful for detecting mesenteric ischemia.[64)] What makes diagnosis difficult, however, is that the SMA can be opacified in the presence of significant ischemia. Although dynamic CT potentially reveals hypoperfusion, it causes time delay before surgery. In contrary, CT is very helpful for ruling out an involvement of SMA in the morphological aspect.

A laparotomy or diagnostic laparoscopy enables direct inspection of intestines.[65)] It is not always easy, however, to select the case of this assessment because aortic repair or reperfusion to the brain or myocardium is prioritized. A small laparotomy or laparoscopy in the limited area may overlook segmental bowel necrosis.[66)]

When the patient is hemodynamically unstable or

directly taken to the OR, mesenteric perfusion needs to be assessed in the OR. TEE can visualize the visceral arteries and assess perfusion in SMA in real time, and thus is helpful not only for making the diagnosis of ischemia but also for detecting persistent malperfusion under systemic true lumen perfusion.[66,67]

As mesenteric ischemia is confirmed, it is necessary to make an appropriate strategy. If the bowel is necrotic already, bowel resection should be prioritized.[68] When ischemic damage is likely reversible, early reperfusion is achieved by using fenestration[69] or surgical revascularization[70,71] or recently, endovascular treatment such as vascular stenting.[72] However, in cases of concomitant ischemia in the brain or coronary artery, or risk of rupture, aortic repair should be done first. In such a situation, selective perfusion to the distal branch of SMA or concomitant revascularization may be helpful.[73]

Endovascular Therapy

Endovascular therapy has become an option for solving malperfusion, not only for type B[74,75] but also for type A aortic dissection.[76,77] It can also be employed to malperfusion following aortic repair as well.[78] Furthermore, stent graft implantation to the aorta has been attempted for type B aortic dissection[79] or AAAD with retrograde extension from the descending aorta.[80,81] When malperfusion persists despite stent graft implantation, additional treatments are done.[79] However, O'Donnell et al. reported that endovascular therapy was associated with high morbidity (60.7%) such as renal failure, neurologic complications, cerebral infarction, or paraplegia.[79] They reported that 3 patients died during the procedure, due to rupture or cardiac tamponade. For the further refinement of endovascular therapy, it is mandatory to clarify the precise reason of those unexpected events and take an effective measure for prevention. One should be aware that the aortic wall and flap cannot be visualized with fluoroscopy. Despite the use of contrast agent, only a two-dimensional projection image is seen. The author experienced a case of endovascular fenestration, in which TEE revealed that the tip of needle catheter was located just a few millimeters from the aortic wall when it was about to penetrate the intimal flap. The procedure was immediately interrupted, and another puncture site was sought. Otherwise, this case would have passed away. The TEE may be helpful for shedding light on the blind area of fluoroscopy and thus minimizing trouble and optimizing the capability of endovascular therapy.

Perfusion Route

In many institutes, the femoral artery has been used as the preferred perfusion route because cardiopulmonary bypass is established quickly, in cases of hemodynamic instability. Despite acceptable results reported in the literature,[82,83] malperfusion occasionally occurs due to predominant perfusion into the false lumen leading to a compressed true lumen. When it takes place in the descending aorta, it causes sudden hypotension and is readily recognized. There are occasions, however, when malperfusion occurs without apparent changes in blood pressure.[43]

Instead, the axillary artery has been increasingly used as perfusion route because the length of retrograde perfusion is much shorter than in femoral arterial perfusion: between axillary artery and innominate artery. While Lee et al. reported that there was no significant difference in the incidence of cerebral complications between right axillary arterial perfusion and femoral arterial perfusion,[84] there are a number of reports on the superiority of the former.[43,85,86] The right axillary artery is mostly used[87,88] and also utilized for antegrade cerebral perfusion during circulatory arrest. Kano et al. have used left axillary arterial perfusion with fairly good results,[89] although a small sized artery may cause an inadequate perfusion rate in some cases. The axillary artery is directly cannulated,[90,91] or a vascular graft is anastomosed to the axillary artery for interposition.[89,92] In either manner, care should be taken on procedure-related limitation and complications. Schachner et al. experienced complications in 14% and needed to change the routes in 11% of cases.[93] Although direct cannulation is simple and quick, it may necessitate addition or switch of routes due to an insufficient flow rate or it can be associated with damage to the axillary artery or even dissection to the aorta.[93,94] Wong et al. reported weakness or numbness of the arm without arm ischemia,[95] potentially due to injury on the brachial plexus.

Other options include the ascending aorta, left ventricular apex, and other arteries. The aorta is directly cannulated, and the cannula tip is inserted by the Seldinger method and placed in the true lumen under the guidance of epiaortic echo or TEE.[96,97] The inner curvature is often punctured because this portion is intact in many cases. Left ventricular apex is also used: transapical approach.[98,99] Despite a high technical success rate of over 90%, one should pay attention to an entry to the left atrium, or conversion of route may be needed due to mal-

perfusion or aortic regurgitation. Rarely, the innominate artery[100] or carotid artery[101] is used for the perfusion route. However, these elaborate attempts do not necessarily ensure successful restoration of the true lumen.[66]

It is important that there is no perfect perfusion route. No matter which route is used, an adverse result should be carefully checked as soon as perfusion is started.

Age and Indication for Surgical Treatment

In the last decades, indication for surgical treatment in aged patients was often discussed with septuagenarians.[102,103] With improved surgical outcomes, however, the issue has recently shifted to surgeries on AAAD patients over 80 years old.[104–106] Hata et al. have devised "less invasive quick replacement (LIQR)" using distal anastomosis at a rectal temperature of 28 degrees without cerebral perfusion, followed by rapid warming with a heating mat and circulating blood at 40 degrees, achieving improved outcomes in elderly patients.[107] Rylski et al. surveyed 381 septuagenarians and 83 octogenarians or older and reported that the former had acceptable mortality (30-day mortality of 16%) but the latter had higher mortality (35%).[108] Though mortality in octogenarians is still high, the outcomes of nonsurgical treatment are inferior to those of surgical treatment.[108,109] In aged patients, however, there is a wide variation among individual cases. Hata reported a case presentation of pulseless shock on arrival, but a good course with conservative treatment.[110] Even a 93 year-old patient could survive surgery.[111] Nonetheless, since surgery on octogenarians is often associated with sequelae,[106] it is important to inform the assumable range of outcomes to the family for discussion on the treatment strategy. Comprehension, consent, and support of the family on either good or poor outcomes is essential.

Prevention

In-hospital mortality of AAAD patients has recently been reduced to lower than 10%. At this moment, it should be noted that a larger number of patients die at the onset of AAAD. Therefore, prevention is an equally or more important issue in the treatment of AAAD. Predisposing factors and triggers of onset are to be considered.

Hereditary diseases as the predisposing factor include Marfan syndrome, Loeys-Dietz syndrome, Erlers-Danlos syndrome, and Turner syndrome. The aortic size and its increase need to be carefully followed up so as not to miss the chance of surgical treatment before the onset of an aortic dissection.

Bicuspid aortic valve has been found to be associated with tissue fragility of the ascending aorta.[112,113] This anomaly is found in 1 to 2% of the population[114] and causes aortic stenosis or regurgitation at a younger age than the atherosclerotic aortic valve. When these are surgically treated, the dilated aorta is occasionally found. Surgical repair of an aorta larger than 45 mm is indicated to prevent an occurrence of aortic dissection.[115] It may be needed to survey the previous medical records of patients who underwent aortic valve replacement. If the patient proves to have had a bicuspid aortic valve, the aortic size should be monitored regularly. The guideline for thoracic aortic disease recommends that first-degree relatives of patients with bicuspid aortic valve should be evaluated for the presence of this anomaly and asymptomatic thoracic aortic disease.[116] Screening of bicuspid aortic valve in the public population may be discussed.

There are several triggers on the onset of aortic dissection. Benouaich et al. examined the relationship of meteorological factor to the onset of aortic dissection and found that a decrease in atmospheric temperature, especially during the preceding three days, was closely related to the onset of aortic dissection.[117] Hatzaras et al. pointed out the relationship between aortic dissection and weight lifting, in which systolic blood pressure is markedly elevated.[118] An abrupt elevation of blood pressure may lead to a tear of intima, although there has been no literature which clearly demonstrated a direct relationship of blood pressure variation and incidence of aortic dissection. Hatzaras reported that 65 of 90 patients with aortic dissection (72%) could recall specific inciting events, exertional or emotional, immediately prior to the onset of dissection.[119] Twenty-four of these 65 patients had a history of thoracic aortic aneurysm. Such information is to be informed not only to the postoperative patients in the follow-up period but also to the general public because an improved lifestyle habit may be helpful for reducing the number of new AAAD patients. An "AAAD warning" based on a continued low temperature may be helpful as well, for those patients with hypertension and/or thoracic aortic aneurysm. The author has experienced not a few patients with hypertension, which had been known prior to the onset of AAAD but had not been appropriately managed by physicians under the self-judgment as "mild." In other patients with medication, the systolic blood pressure was below 130 mmHg at the clinic but varied largely, occasionally up to 200 mmHg. Some measures

to decrease the peak blood pressure might be effective to reduce the incidence of AAAD.

These pre-onset factors are usually deemed to be out of the territory of cardiovascular surgeons. However, contribution by surgeons based on wealth of experience and not a few painful lessons may change the fate of up to 20% of AAAD patients.

Conclusions

Through review of the latest literature on management of AAAD, the outcomes of in-hospital management are approaching a level that is high enough, and it is getting hard to determine the statistically significant superiority among strategies. Further refinement may be achieved by eliminating every unexpected result based on accurate analyses using various diagnostic modalities. On the other hand, management of the pre-hospital stage, including prevention, needs to be developed considerably, to further improve the outcomes of treatment of the entire stage of AAAD.

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