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CURRENT TOPICS REVIEW ARTICLE

Clinical dilemma in the surgical treatment of organ malperfusion caused by acute type A aortic dissection

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Abstract The malperfusion syndrome associated with acute aortic dissection draws attention because the clinical picture is very dramatic, and patients with malperfusion have poorer clinical outcomes. To improve surgical outcomes, the ischemic damages associated with organ malperfusion should be minimized by restoring perfusion as early as possible, which occasionally can hardly coexist with central repair. This paper reviews the current evidence, problems, and dilemma related to the diagnosis and treatment of the malperfusion syndrome caused by acute type A aortic dissection.

Keywords Aortic dissection · Malperfusion syndrome · Hybrid operating room

Introduction

Malperfusion syndrome associated with acute aortic dissection draws attention because clinical picture is very dramatic, and those with malperfusion are associated with poorer clinical outcome [1–3]. To improve the surgical outcomes, ischemic damages of malperfused organs should be minimized by restoring perfusion as early as possible, which occasionally can hardly coexist with central repair. This paper reviews current status, problems and dilemma with diagnosis and treatment of malperfusion syndrome with acute type A aortic dissection.

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What is malperfusion syndrome

Malperfusion syndrome was defined as the presence of signs and symptoms due to a altered blood flow in an organ system with clinical evidence of a lack of blood flow, resulting in ischemia with organ dysfunction [4]; (Fig. 1). Incidence of malperfusion has been reported ~20–40 %, and it varies according to the affected organs. Depending on the organ system, the malperfusion syndromes were classified as cerebral(3–13 %:stroke or transient ischemic attack), cardiac [5–11 %: cardiac dysfunction concordant with malperfused coronary vessel(s)], limb (25–60 %: loss of pulse, sensory or motor function with clinical signs of limb malperfusion), mesenteric(10–20 %:abdominal tenderness, bowel paralysis or necrosis, lactate acidosis with depression of liver and pancreatic function), renal (23–75 %: creatinine elevation, lack of urine output) and spinal (2–9 %:transient/permanent paraparesis/paraplegia) [5].

Diagnosis of malperfusion syndrome

Preoperatively, physical examination, enhanced CT scan and ultrasonography are the gold standard for diagnosing malperfusion not only by detecting the presence of aortic dissection, but also by assessing the severity of clinical symptoms and the degree of branch stenosis. CT scan is also important to elucidate the mechanism of malperfusion. However, malperfusion may develop not only preoperatively but also intraoperatively, especially right after the initiation of cardiopulmonary bypass. This may be due to the shift of intimal flap due to retrograde arterial perfusion (i.e., femoral artery cannulation) or changing mode of perfusion. Near-infrared spectroscopy and intraoperative

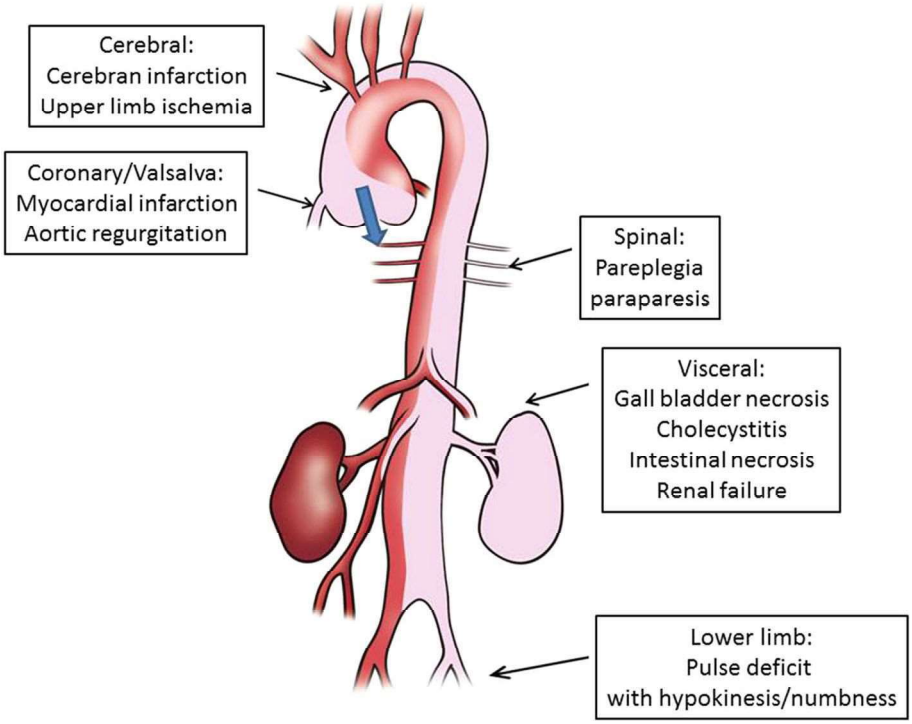

Fig. 1 Distribution and clinical impact of malperfusion associated with acute type A aortic dissection

transcutaneous carotid ultrasonography either from surgical field or from anesthesiologist side are easy and reliable tool to monitor cerebral perfusion [6, 7]. Visceral ischemia can be frequently overlooked because it often lacks clear preoperative symptom, and may exacerbate occultly during operation, and TEE can be the real-time monitoring tool with experienced hands [8].

Recently, hybrid operating room is reported to be useful in terms of exact diagnosis of coronary status and downstream malperfusion which enables flexible and appropriate design of surgical and/or endovascular treatment with no time delay and negligible additional risk [9].

How do we define malperfusion? Clinically or radiographically?

Currently, in majority of reports with significant patient size, the definition of malperfusion is clinical (presence of symptom due to lack/decrease of organ), not radiological (vessel narrowing or occlusion due to aortic dissection according imaging modalities such as CT scan or echo), because the prognostic value of malperfusion syndrome is primarily determined by the severity of organ ischemia [10]. In addition, current gold standard for the evaluation of malperfusion syndrome is CT scan, and its accuracy of evaluation is still affected various factors such as patient weight, contrast volume, the timing of scanning, target vessel diameter. Moreover, in cases of severe malperfusion of mesenteric artery, its direction at origin is often perpendicular to the scanning axis, which may be another source of measurement error. Surgeons may often encounter the patients with total occlusion of innominate artery due to newly developed aortic dissection who have no neurological deficits and clear consciousness presumably due to good collateral network. Concerning the mesenteric malperfusion, it has been shown that 40 % of patients with mesenteric ischemia did not have abdominal pain, whereas ~20 % of patients without mesenteric malperfusion had abdominal pain [11, 12].

However, it has been shown that although patient with symptomatic malperfusion is associated with worse in hospital mortality than those with asymptomatic malperfusion, long-term survival was in fact worse with those with asymptomatic malperfusion [3], which indicates that surgeons should not underestimate the negative impact of radiologically overt malperfusion even though ischemia was not very apparent during hospitalization.

Pathophysiology and classification of malperfusion syndrome

Several classification systems of malperfusion syndrome have been proposed focusing on anatomy, dynamism of malperfusion [13–16], and it is classified either due to compressed true lumen with expanded false lumen (aortic type, dynamic obstruction) or due to avulsion of branch's

orifice or dissection extended into branch itself (branch type, static obstruction) or combination of both. Those classification systems can be useful if it can guide surgeons to judge whether the malperfusion can be fixed with central repair or they need additional revascularization procedure. So far no classification system has reached this standard.

Malperfusion syndrome and its impact on cannulation site of cardiopulmonary bypass

Historically, femoral artery is the choice of cannulation site for arterial return for cardiopulmonary bypass in treating type A aortic dissection because of its ease in exposure. Axillary artery has gained popularity because of its ease in perfusing true lumen, particularly in cases of cerebral malperfusion, thus avoiding organ malperfusion. In cases of severe cerebral malperfusion, direct innominate cannulation in the operating room [17], or early reperfusion with roller pump in the emergency room [18] was reported with favorable outcome although the number of the patients treated with these methods was limited.

Selection of perfusion route in cardiopulmonary bypass has been a topic of debate, and it has been reported that femoral artery has significantly higher complication rate such as cerebral emboli compared with axillary/subclavian artery [19, 20]; it has been reported that rate of intraoperative false lumen perfusion and that of stroke is 3.8 and 2.6 % with axillary/subclavian cannulation compared with 10.2 and 7.4 % with femoral cannulation, respectively [21, 22]. There are alternative sites and methods of cannulation such as through ascending aorta with echo-guided Seldinger technique (reported stroke rate: 4 %) [23] or with tracheal tube with balloon (0 %) [24], combination of axillary and femoral artery (5.7 %) [25] or through left ventricular apex (5.8 %) [26]. Although the stroke rate with the cannulation using tracheal tube appeared exceedingly good, the number of patients treated with this method is still limited, and reported mortality rate with aforementioned methods was similar between 12 and 18 %. There is so far no gold standard of cannulation sites in terms of preventing complication, and surgeon need to take every necessary change to improve perfusion once he/she encounters perfusion exacerbation. There is no clear-cut answer.

Surgical/hybrid treatment of malperfusion syndrome

The basic principle of the treatment of malperfusion syndrome is prioritized central repair if clinical status of aortic dissection is unstable, otherwise prioritize peripheral revascularization, either with bypass surgery or catheter-based intervention (i.e., stenting or fenestration) according to the severity of ischemia [27], because it is reported that in cases of severe metabolic disturbance due to malperfusion, temporary postponement in surgical repair while peripheral reperfusion re-establishment has been proved beneficial [28, 29]. The mere presence of radiological, not clinical, malperfusion usually does not lead to prioritizing peripheral revascularization [28]. Figure 2 illustrates the proposal of the decision-making tree of the treatment for the acute type A dissection with malperfusion. The damage caused by localized ischemia due to malperfusion is significantly enhanced by generalized ischemia caused by circulatory collapse such as cardiac tamponade [30], and pericardial drainage with pig-tail catheter in the emergency room may mitigate the damage caused by malperfusion, and can bridge the patient in more stable condition to the operating room [31]. Although basic treatment strategy of malperfusion syndrome may sound straightforward and simple, it is easier said than done.

We present our two cases of clinical dilemma associated with malperfusion syndrome

Case 1

A 60-year-old male underwent coronary artery bypass grafting with left internal mammary artery to left anterior descending artery, and free radial artery to circumflex artery with proximal anastomosis achieved side biting clamp on ascending aorta whose diameter was slightly larger than 40 mm. 3 months later, he was referred to us for acute type A aortic dissection with partially thrombosed false lumen of ascending aorta of 50 mm, and 90 % stenosis of superior mesenteric artery due to compression of thrombosed false lumen. His hemodynamics was stable with no cardiac tamponade and aortic regurgitation. His chest and back pain was refractory despite the optimal blood pressure control and analgesics. His abdomen was soft and flat without any pain. His lactate level was normal. We underwent emergency repair of ascending aorta. Immediately after weaning of cardiopulmonary bypass, his hemodynamics collapsed with significant acidosis, and laparotomy then revealed necrosis of gall bladder and entire small intestine. He passed away the next day.

Clinical dilemma Should we have prioritized revascularization of superior mesenteric artery, despite the lack of clinical evidence of mesenteric malperfusion?

Case 2

A 65-year-old male was referred to us because of severe back pain and difficulty of moving his right leg. Emergency CT scan revealed acute type A aortic dissection of Crawford type III retrograde with partially thrombosed

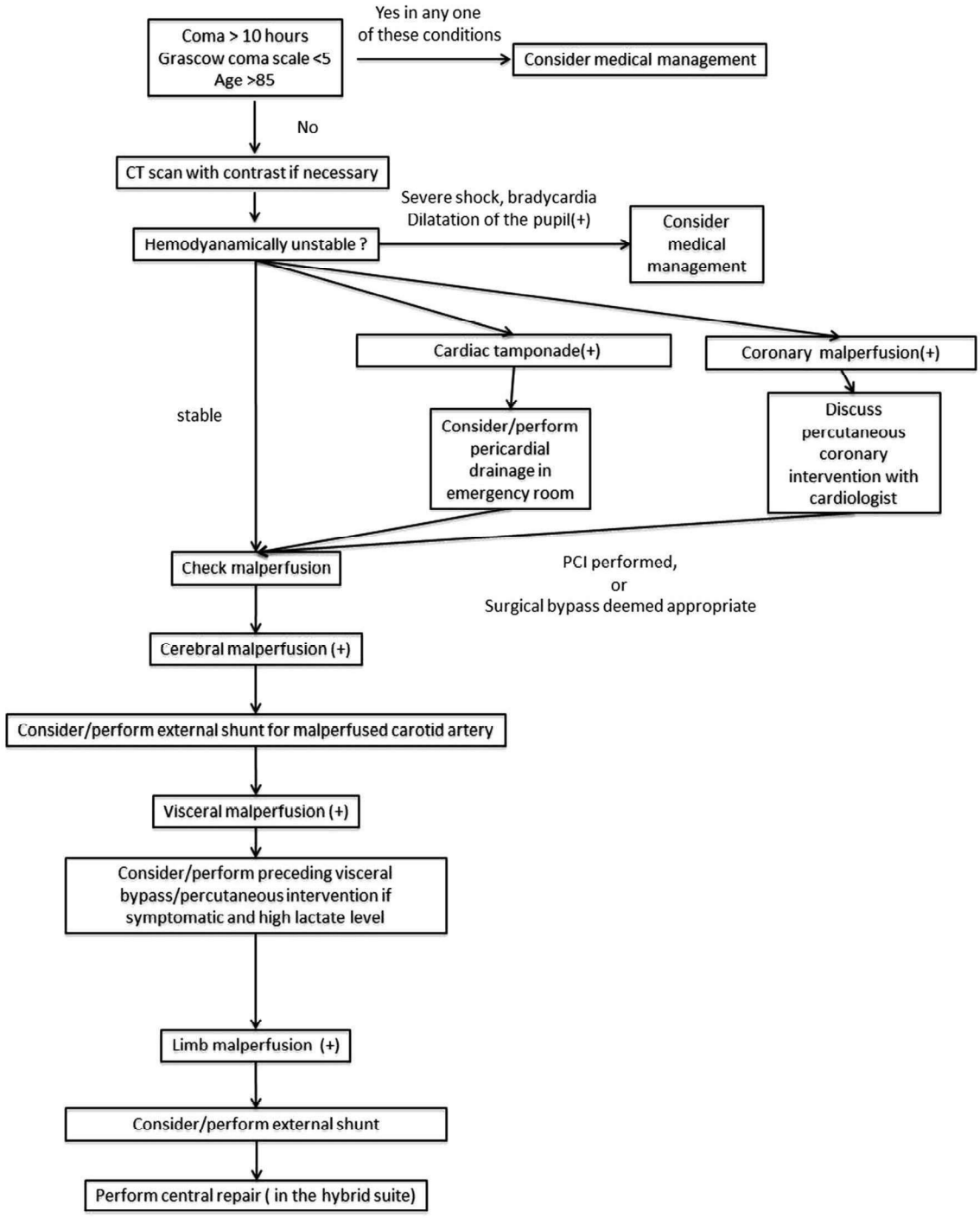

Fig. 2 Proposal of the decision-making tree in the treatment of acute type A aortic dissection complicate with malperfusion

false lumen. His hemodynamics was unstable due to cardiac tamponade with good cardiac wall motion, and his ascending aorta was 40 mm in diameter which was almost thrombosed with very small ulcer-like lesion. Immediately after shooting enhanced CT scan, his paraparesis of right leg was recovered. We underwent emergency repair of ascending aorta. His postoperative course was uneventful except he developed paraplegia, which did not recover despite the optimal control of blood pressure, spinal drainage and intravenous corticosteroids.

Clinical dilemma For his better quality of life with viable foot, should we have only resolved cardiac tamponade with minimal anesthesia, and take him back to intensive care unit for medical management?