REVIEW ARTICLE

CEREBRAL MALPERFUSION IN ACUTE TYPE A AORTIC DISSECTION: SHOULD SURGERY PROCEED?

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BACKGROUND

Acute Type A Aortic Dissection (ATAAD) is defined by a tear in the inner layer of the ascending aorta, representing a critical disease with widely known out-of-hospital and in-hospital mortality, amounting to approximately 50% and 30% of mortality, respectively [1].

As a consequence of the formation of the false lumen, supra-aortic vessels and abdominal aortic visceral branches frequently suffer partial obstruction or complete occlusion of the true lumen, compromising distal perfusion and leading to brain and other visceral malperfusion. This may lead to ischemic damage in 16 to 34% of patients with ATAAAD [2].

The severity of ATAAD, apart from the anatomical location of the intimal tear and its extension, also depends on the presence of end-organ perfusion. It has been widely known that, in the setting of ATAAD, end-organ malperfusion is associated with an increased risk of mortality and post-operatory neurological damage[2]. It has been shown malperfusion syndrome not only increases perioperative mortality but is also a predictor of early and late mortality[3]. Cardiac malperfusion seems to have the highest risk of intraoperative mortality, also predicting 30-day and late mortality, as well as gastrointestinal, renal and peripheral malperfusion[3].

Cerebral malperfusion (CM) has been identified in 6% of patients in the International Registry of Acute Aortic Dissection[2], and varies from 3% to 21% of patients in different series[1]. Cerebral malperfusion increases the possibility of lasting neurological damage and many surgical teams refuse to operate on some patients with identified CM or clinical signs of neurological damage, since the surgical results of ATAAAD surgery in these patients appear dismal. Despite this, several groups have published satisfactory results of ATAAD surgery in patients with established CM, and techniques to ameliorate the prognosis of these very sick patients.

In this point-of-view, we review current evidence on the effect of pre-operatory cerebral malperfusion on mortality and neurological damage in the setting of ATAAD, and techniques for improving results when CM is present.

CAUSES OF CEREBRAL MALPERFUSION

The brain is one of the most sensitive human organs to hypoxia, and in situations of ischemia, prompt correction of ATAAD is the best approach to improve survival and reduce morbidity. In this view, early brain reperfusion is the best strategy to reduce neurologic complications.

Historically, malperfusion has been associated with propagation of the dissection flap into the branch vessels. However, recent imaging and basic science studies have enabled researchers to understand that the mechanism may be multifactorial and much more complex. There are three main causes of cerebral ischemia in ATAAD: First, obstruction/occlusion of the supra-aortic branches due to involvement by the dissection, through the natural progression of the flap, or by dynamic obstruction. Dynamic obstruction seems to be more relevant than static obstruction and is related to two distinct mechanisms: insufficient flow through the true lumen when perfusion of the vessel is maintained by the true lumen, and intimal flap mobility when the false lumen prolapses into a branch vessel ostium; Second, hypoxic encephalopathy due to diminished cardiac output (by tamponade, myocardial infarction or acute aortic insufficiency); Third, thromboembolism originating in the false lumen, due to the combination of the hypercoagulable state of the false lumen (attributable to the exposed adventitial and medial layers), and stasis. The combination of one or all of these factors

has the potential to create severe cerebral hypoperfusion that leads to neurological damage.

Additionally, cerebral hypoperfusion may be compounded by lung dysfunction and acidosis, fluid overload, and overall metabolic imbalance due to abdominal visceral ischemia, renal insufficiency and ARDS.

Importantly, reperfusion may exacerbate end-organ lesions. Reperfusion is associated with the up-regulation of several inflammatory mediators, including cytokines and immune cells, resulting in cellular and endothelial injury, increased vascular permeability and edema[4]. Cerebral edema may lead to increased intracranial pressure that may aggravate neurological damage in the setting of ATAAD, even before surgical correction. After surgical correction, with partial or complete restoration of cardiac output through the true lumen, reperfusion injury may cause additional neuronal damage, aggravating the inflammatory response and bursting the local inflammatory response into a systemic manifestation. The precise effect of reperfusion on neurological outcomes and mortality remains unknown, due to the extremely dynamic situation in critical patients, almost impossible to simulate in a controlled environment.

Moreover, aspects of surgical technique may also have a role in postoperative neurologic dysfunction. Prolonged cardiopulmonary bypass time is an intraoperative risk factor for novel postoperative neurologic dysfunction, although the surgical teams experience and medical and surgical strategies also have an important role[5]. The non-pulsatile flow at the initiation of cardiopulmonary bypass, Deep Hypothermic Circulatory Arrest (DHCA), and Selective Cerebral Perfusion (SCP) also contribute to neuronal lesions, although their role will be discussed further in this manuscript[6]. Heparin also facilitates local conversion of cerebral endothelial damage to full hemorrhagic cerebral lesions[7].

The combination of all these aspects complicates outcomes in already critically patients, and in patients with previous neurological damage, DHCA and the need for Total Arch Replacement (TAR) seriously compound the risk for mortality and lasting neurological damage. On the other hand, the extent of the surgical correction needed (arch, aortic valve, aortic root, etc) is frequently only determined during the surgery and after cross-clamping and aortic arch inspection, and surgical teams face multiple challenges and difficult decisions in ATAAD patient with neurological damage.

FREQUENCY OF CM

The IRAAD[8] is the largest worldwide registry of ATAAD patients, with data originating from over 51 centers in 12 countries, and has amassed a considerable amount of valuable data. In 2013, IRAAD published the outcomes of the patients with neurological damage out of their 1873 patients with ATAAD. They found 4,7% had a preoperative stroke and 2,9% of patients presented in a coma. They also found that neurological damage increased the probability that the patients were not accepted for corrective surgery (which almost always translates into life-saving surgery) - patients without neurological damage were refused surgery in 11% of cases, patients with stroke were refused 24,1%, and patients in coma were not offered surgery 33% of times[8]. In a subanalysis of IRAAD, Angleitner et al found that in patients over 70 years, the pre-operative stroke rate was 7,6%, similar to the overall IRAAD population[1]. More recently, in 2019, the IRAAD found an incidence of 15% in their database, probably reflecting a growing acceptance of patients for ATAAD surgery despite the presence of CM[2]. Estrera et al published their findings on a series of 151 consecutive ATAAD patients, of whom 10,6% presented with a pre-operative stroke[9]. Morimoto et al found, in their similar series from Kobe University, found cerebral malperfusion in 41 of 157 patients (26,1%)[10]. Another series of 775 patients with ATAAD from the USA described a 10% frequency of CM[11].

ANATOMY OF CM

In the IRAAD registry, the right hemisphere is more frequently affected (81% of patients), vs the left (13%) or both hemispheres (6%). This is probably explained by the natural distal progression of dissections originating in the ascending aorta, that first extends to the brachiocephalic arterial trunk before involving the left carotid artery[9]. In this regard, Di Eusanio et al have also found that arch involvement is more frequent in patients with CM, seen in 61.8% of patients with stroke, 43.6% of patients with coma, and 36.1% of patients without neurological signs ($P<0.001$)[8]. The identification of arch involvement is clinically important since arch involvement on pre-operative imaging is the greatest predictor of stroke[12]. Patients with CM also had higher rate of abdominal artery involvement. Interestingly, the frequency of dissection flap (entry tear) being in the arch or on other locations is similar in patients with and without CM (5.9% vs 3.9%, $P=0.210$)[8].

The type of arch involvement has also been studied, with occlusion or severe stenosis (over 90% of lumen) of carotid arteries being associated with persistent neurological deficits, contrary to the patients in which dissection of the carotid arteries is not associated with severe stenosis or occlusion[12].

The type of involvement of the supra-aortic vessels by the dissection may also have an important role in managing and prediction on the neurologic outcome. The occlusion of Internal Carotid Artery (ICA) alone seems to be a clinical surrogate marker to predict massive cerebral edema with herniation syndrome. Fukuhara et al. have shown that, in their experience of 80 patients with CM, ICA occlusion was associated with 100% of cerebral herniation and mortality, while Common Carotid Artery (CCA) occlusion or non-complete obstruction was associated with in-hospital 21% mortality[11]. These findings are consistent with current findings in Neurology, as occlusion of the distal ICA is known as a predictor of fatal hemispheric swelling in acute stroke[13,14]. In

this regard pre-operative neck CT scan is becoming crucial in ATAAD patients to establish prognosis and identify in whom the surgery is beneficial and in whom it is futile.

CLINICAL CHARACTERISTICS OF PATIENTS WITH CM

Patients with pre-operative neurological deficits usually present more rapidly to a referring or tertiary hospital, mainly because they experience less thoracic or abdominal pain at the onset of symptoms, and have more frequently hypotension/shock/tamponade (no neurological deficit 25.2%; stroke, 40.2%; coma, 59.3%; P<0.001) and syncope (no brain injury, 15.3%; CVA, 43.2%; coma, 56.5%; P<0.001)[1,2,8]. In retrospective analysis, neurological deficits were also more frequently associated with renal failure (no cerebral injury, 7.9%; stroke, 13.4%; coma, 21.2%; p<0.002), myocardial ischemia or infarction (no brain injury, 10.2%; stroke, 13.4%; coma, 25.0%; p<.002), and limb ischemia (no cerebral injury, 9.7%; stroke, 18.3%; coma, 15.4%; p<.019)[8].

Regarding neurologic presentation, patients show signs and symptoms of cerebral ischemia, presenting with coma with or without seizures, stupor, hemiplegia, hemiparesis and hemianopsia. Presentation and extension depend on the location and severity of neurologic lesions.

CLINICAL IMPACT OF CM

Up until 1990, it was generally believed that pre-operative stroke was a contra-indication for immediate correction of ATAAD, due to the risk of reperfusion injury and heparin contributing to a worse prognosis. In 1989, an initial experience from Stanford published by Fans et all showed that, in 7 patients with pre-operative stroke out of 272 patients, survival at 1-year post-op was 57% and long-term survival was 43%, raising the possibility that these patients could be offered surgery in selected cases[15]. As we have previously mentioned here, IRAAD data reported that the presence of pre-operative neurological damage was associated with higher rates of surgical refusal, up to 33% in patients with coma[8].

Ever since this initial report, many groups have attempted to offer life-saving surgery to these patients. A recent sub-analysis of IRAAD focusing on CM showed that mortality of patients with CM was in fact higher when compared to patients without CM [25.7% vs. 12.0% (p<0,001)]. However, this difference in survival appears immediately after surgery and stays constant after 4 weeks post-operatively, suggesting that the initial hazard does not influence long-term results[2]. Recent evidence has even suggested that post-operative neurologic dysfunction might be more influenced by surgical management than by preoperative cerebral malperfusion and that only a relatively small percentage of in-hospital deaths are attributed to neurological causes[5,11]. Moreover, patients with CM had a reasonable survival rate, with an in-hospital survival rate of 75% and 1-year of 62.6%[2]. A similar result was found in the IRAAD subgroup analysis of patients over 70-years old[1].

Another important fact is that mortality is significantly higher (3 to 4 fold) in patients with medical management of stroke and coma[8,16]. In another retrospective analysis, medical treatment was associated with 100% mortality in patients with coma, and 76.2% in those with cerebrovascular events. On the other hand, surgery led to an in-hospital survival rate of 55.6% in patients presenting with coma and 49.6% in patients with cerebrovascular events. Surgery seems to be protective against mortality in patients with CM, and surgical benefits on survival are sustained at 5 years[8].

Mortality should not be the only outcome to evaluate the benefits of surgery, especially in the field of neurologic outcomes, since neurologic deficits can negatively and significantly impact a patient's quality of life. Besides surgical benefits on survival, surgery also facilitates neurological recovery. In an analysis from the German registry of AADA, Cozelmann et al reported that neurologic deficits resolved in 62.1% of patients, more specifically hemiparesis in 70.5%, paraparesis in 78.8%, aphasia in 80.6% and unconscious/comatose status in 70.9%[5]. Estrera et al reported a neurologic complete recovery in 14% of the patients, with 43% improving neurologic status and 43% remaining the same[9]. Recovery of consciousness and neurological function is especially achieved with immediate aortic repair, as reported by Tsukube et al, with full recovery of consciousness in 86% of patients and a significant improvement in postoperative National Institutes of Health Stroke Scale score (NIHSS) and independence in daily activities in 52% of the patients[17].

Curiously, cerebral malperfusion is not an independent predictor of late mortality, contrary to myocardial, renal or gastrointestinal malperfusion[3].

TIMING OF ATAAD REPAIR AND INFLUENCE ON OUTCOMES

Since brain ischemia is very time-sensitive, it is expected that time from neurological signs onset to surgical correction of ATAAD (and hopefully restoration of acceptable cerebral blood flow), influences neurological outcomes. In fact, evidence seems to point in that direction, since patients who undergo surgical correction of ATAAD in the first 10 hours more frequently show improvement in neurological status, while patients operated after 10 hours have elapsed shown no improvement in the NIHSS score[9].

In several surgical centers, intentional delay of aortic repair until neurologic recovery is a therapeutic option for ATAAD with CM. Although current evidence is not strong enough to advise against this clinical practice, it is known that rapid restitution of cerebral perfusion is essential to minimize neurologic damage. In fact, time to surgery is a predictor of poor neurologic recovery and is associated with the risk of permanent deficits. Patients with early surgical repair have better neurological neurologic outcomes, while surgical repair over 9.1h after the onset of symptoms has a reduced chance of neurologic improvement[10,17]. Similar results

have been found by the Kobe University group in Graduate School of Medicine: in comatose patients, immediate correction (mean 222 minutes from symptoms to OR) compared to medical therapy initially (mean 2129 minutes) improves neurological outcomes and early and long-term mortality[18].

SURGICAL TECHNIQUE AND SPECIAL CONSIDERATIONS

Some concerns arise from the decision to proceed to surgery, especially regarding the use of anticoagulation and the possibility of worsening neurologic injuries. The rate of hemorrhagic conversion increases by approximately 7% with anticoagulation[19]. Estrera et al did not observe any case of hemorrhagic conversion in their series of patients with ATAAD with stroke, despite early use of full anticoagulation[9]. The use of anticoagulation in this setting was not associated with worsening of neurologic injuries.

Considering all cases of ATAAD, there are several cerebral protection techniques associated with cardiopulmonary bypass. When the use of hypothermic circulatory arrest with anterior cerebral perfusion (ACP) is necessary, we can find two major approaches: unilateral (u-ACP) or bilateral (b-ACP) perfusion. ACP is characterized by cerebral perfusion through the cerebral arterial circuit through one hemisphere (u-ACP) [cannulation performed through the right axillary artery] or both (b-ACP) [with the addition of a cannula in the left common carotid artery]. In the first case, the hemisphere contralateral to the cannulation is irrigated and dependent on the Willis polygon. Angleitner et al, in their retrospective study, showed that there were no statistically significant differences in outcomes after AAD surgery with b-ACP versus u-ACP under moderate to profound hypothermia. There were no significant differences regarding postoperative permanent neurologic deficits [18.7% vs 19.4%, P=0.753]. Ischemic stroke was the most common postoperative cerebral lesion, followed by hypoxic brain injury. Patients with b-ACP had higher rates of right-sided lesions, whereas the rate of left-sided lesions was higher in patients with u-ACP. Subgroup analyses demonstrated that b-ACP was associated with improved overall survival in patients with ACP of more than 50 minutes (log-rank p = 0.02), since death was less frequently caused by neurologic events than in the u-ACP group (33.3% vs 75%)[20]. Both u-ACP and b-ACP are validated strategies for cerebral protection strategies in aortic arch surgery, particularly in the treatment of ATAAD, although some authors recommend the use of u-ACP due to its simplicity and shorter manipulation of the supra-aortic vessels[21,22]. Besides the use of b-ACP, multiple centers have adopted more aggressive measures to limit cerebral malperfusion in ATAAD surgical repairs, including direct carotid perfusion and extra-anatomic revascularization strategies. Cannulation of the axillary artery may not restore the true lumen and may cause or aggravate cerebral hypoperfusion because of static malperfusion[23]. In fact, Gomibuchi et al have shown that extra-anatomic revascularization (the right common carotid artery is anastomosed to the aortic arch with an additional branched graft) in cases with CM may reduce the risk of neurological complications, but its use is not yet consensual and universally accepted[12].

The choice of the arterial cannulation site is another important factor. Regardless of the type of cerebral perfusion, it is known that the arterial cannulation site is a determining factor for hospital mortality, especially due to its impact on cerebral perfusion[24]. The ideal cannulation technique is controversial, and the need for more high-quality randomized studies on this topic is pressing. The discussion is mainly centered on two types of cannulation: axillary and femoral. Several studies around the world have shown that the results are similar between both approaches, pointing out that the selection depends on the medical conditions of the patients or the preference of the surgeons[25-28]. One of the main disadvantages of femoral cannulation is the possibility of malperfusion or athero-embolization in the elderly. On the other hand, cannulation of the axillary artery may be time-consuming and may need a graft[29]. In a recent meta-analysis by Hussain et al., axillary cannulation is associated with superior outcomes, especially in terms of mortality and neurological dysfunction[24]. The best evidence indicates that axillary cannulation may have benefits in the surgical treatment of ATAAD, with reduced overall mortality, neurological and malperfusion complications when compared to femoral cannulation. However, femoral cannulation remains a valid option in emergent situations when axillary cannulation cannot be performed[30].

PERSPECTIVES

To date, no consensus has been reached on how to manage and treat ATAAD with CM. CM is frequent and accounts for significant morbidity and in-hospital mortality. The impact on long-term survival is controversial, and several articles report conflicting results. However, patients presenting with neurologic symptoms may have complete reversal with surgical repair and surgery is indeed the best approach to protect against mortality and avoid definitive neurological defects. Surgical treatment provides acceptable results and outcomes, and benefits on survival are sustained for up to 5 years. Special surgical considerations should be taken in these cases, especially considering axillary cannulation, bilateral ACP and the eventual use of extra-anatomic conduct. We consider it extremely important to highlight the fact that multiple studies show that coma or the occurrence of a cerebrovascular accident does not prohibit surgery, and deficits may even be completely reversed after surgery. New insights in imaging and basic research can help to prioritize patients and establish better tools for prognosis, such as the presence of specific lesions in supra-aortic branches.

Concluding, the presence of CM does not prohibit surgical treatment in ATAAD and early reperfusion and extra-anatomic revascularization are the best approaches to improve prognosis.

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