Complications in Open Repair of the Aortic Arch: Tips for Prevention and Tricks for Management

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Aortic arch replacement represents one of the most challenging and complex procedure for the cardiovascular surgeon and despite the progressive improvement in the last 15 - 20 years of the surgical and anesthesiological techniques with consequent improvement of the outcome of patients after aortic arch surgery, brain injury and neurological complications continues to be the most feared complication and frequent cause of death. For this reason the knowledge of cerebrovascular complications after open surgery of the aortic arch and the tips and tricks for their prevention and management become of primary importance.

There are 2 basic mechanism for ischemic cerebral injury during aortic arch surgery, that needs temporary exclusion of the cerebral circulation [1]. Global ischemia caused by interrupted or inadequate flow leads to subtle brain injury that manifests itself as Temporary Neurologic Dysfunction (TND), a direct consequence of inadequate cerebral protection that can result in anoxic brain injury. This condition, usually represented by lethargy, agitation, and confusion with complete resolution before discharge and negative brain imaging for local/diffuse damage is, commonly, self-limited and benign [2]. The second mechanism is represented by ischemic infarcts that potentially can cause devastating consequences as localized stroke or coma and defined as Permanent Neurologic Dysfunction (PND). These infarcts are usually due to embolic events. Stroke has a devastating impact on survival, causing death in almost of 30% of those affected and leaving only 28% alive at two years. Moreover, hospital morbidity is dramatically increased in stroke survivors, including ICU stay, duration of intubation and postoperative hospital stay [2].

In the light of this, prevention and management of these cerebrovascular complications become of primary importance both for surgeon and for anesthesiologist.

The first step in preventing neurologic injury in aortic arch surgery is to identify those patients who are at highest risk in order to assess the operative risk and to plan the neuromonitoring.

From the surgical point of view, the arterial cannulation site for cardiopulmonary bypass is crucial for an effective brain protection. In recent decades, the use of central cannulation sites (ascending aorta, axillary/subclavian artery and brachiocephalic trunk) has been proposed in order to reduce perioperative embolic risk [3-5]. In fact, the cannulation of the femoral artery generates a retrograde flow in the thoracoabdominal aorta, where atherosclerotic plaques, thrombi and calcifications are often localized and mobilized by the retrograde blood flow with consequent cerebral embolization. Moreover, in the case of aortic dissection, the central cannulation, allowing an antegrade flow in the thoracic aorta, can be effective in reducing the risk of cerebral and splanchnic malperfusion which can instead occur, with the cannulation of the femoral artery, by inversion of the flap intimal and/or by expansion of the false lumen [6]. However, the femoral artery remains a viable option especially in patients with acute aortic dissection with cardiac tamponade, where extracorporeal circulation is required in emergency conditions, or in cases where other cannulation sites cannot be used for the presence of parietal calcifications or because of dissection. Standard techniques of neuromonitoring include electroencephalogram, somatosensory evoked potentials, near infrared-spectroscopy (NIRS) and transcranial doppler.

The location and the length as well as the exact anatomy of the aortic lesion obtained by the angio-CT scan, MRI and angiography may be of fundamental importance in order to evaluate risk and type of monitoring required for specific procedures. We understand how “key points” in this type of surgery are represented by the methods of brain protection and neuromonitoring.

In fact, an accurate neurophysiologic monitoring can identify malperfusion or evidence of ischemic injury to the brain and spinal cord intraoperatively, so that interventions can be undertaken to reduce or reverse neurologic injury. Current methods of brain protection include Deep Hypothermic Circulatory Arrest (DHCA), Retrograde Cerebral Perfusion (RCP) and Antegrade Selective Cerebral Perfusion (ASCP). The main hypothesis at the base of cerebral protection methods is that hypothermia (18°C - 22°C) reduces the cerebral metabolism enough to allow a safe period of total circulatory arrest sufficient to the surgeon to perform the arch reconstruction without the occurrence of detectable functional or organ derangements. However there is still controversy about basic aspects of DHCA. In fact, DHCA is a simple and valid method but it has the principal disadvantage of a limited “safe” time of circulatory arrest (< 40 minutes at 18°C). The limited duration of safe circulatory arrest seems to be a significant drawback of this technique especially for extended aortic arch reconstructions that, in the hands of most surgeons, requires duration of circulatory arrest longer than 30 minutes. RCP through the superior vena cava and ASCP were introduced as an adjunct to DHCA to extend the “safe” duration of circulatory arrest obtained with DHCA alone. Unfortunately, contradictory results have been obtained with RCP. ASCP is instituted by means of endoluminal cannulation and selective perfusion of the innominate artery and left common carotid artery. Due to the fact that ASCP can safely extend the duration of circulatory arrest up to 90 minutes without increasing the risk of death and adverse neurologic outcome, it is generally considered as the best method to protect the brain during extended aortic arch reconstructions. Several studies demonstrate superior outcomes with ASCP as compared to both DHCA alone and DHCA with RCP [7-9].

Regarding postoperative management of cerebrovascular injury, aortic arch repair requires aggressive postoperative ICU care. A proper neurologic evaluation and frequent neurochecks are mandatory even if patients who have periprocedural stroke have limited therapeutic options. The only pharmacologic therapy demonstrated to be effective for acute ischemic stroke is intravenous tissue plasminogen activator. However, despite these potential stroke therapies, postoperative stroke care is largely supportive.

Moreover, there is no data suggesting the beneficial effect of the anticoagulation therapy in acute stroke patients. In fact, although intravenous heparin modestly reduce the risk of stroke recurrence there is a commensurate increase in the risk of hemorrhagic conversion and thus, there is no benefit. However, the ratio risk-benefit may favour anticoagulation if the primary stroke is small, and the risk of recurrent emboli is deemed to be high.

In conclusion, neurological injuries remain the most feared complications in thoracic aortic surgery and in aortic arch surgery in particular. In order to prevent them and to minimize the risk of neurological complications, a thorough knowledge of the pathophysiological mechanisms of the neurological injuries and of anesthesiological and surgical methods is necessary to prevent them.

Bibliography


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