Spinal Cord Protection During Thoracoabdominal Aneurysm Repair

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Abstract

Spinal cord injury after thoracoabdominal aortic surgery remains a devastating and unpredictable complication, caused by clamping of the thoracoabdominal aorta, resulting in exclusion of blood flow in critical and essential intercostal arteries. Various protective methods against spinal cord ischemia have been proposed and performed clinically. These include preoperative spinal angiography, distal aortic perfusion, hypothermia, reattachment of the intercostal artery, cerebrospinal fluid drainage, administration of neuroprotective agents, and monitoring of somatosensory and motor-evoked potentials. The information to date suggests that multimodality approaches should be used to prevent spinal cord injury after thoracic and thoracoabdominal aneurysm repair.

Key words Spinal cord injury · Thoracoabdominal aneurysm · Multimodality approach

Introduction

The effective operative treatment of thoracoabdominal aneurysms remains one of the most formidable challenges in vascular surgery, although recent advances in surgical techniques, perfusion technology, transfusion technology, and critical care skills have resulted in remarkably improved morbidity and mortality. According to recent reports, the mortality is 3.0%–23.0%,1–18 and the incidence of spinal cord injury, the most devastating complication, is 3.0%–18.0%.1–18 Those patients whose postoperative outcome is complicated by paraplegia or paraparesis not only suffer severe physical disability, but their long-term survival is known to be shorter. In one reported series, the 5-year survival rate for patients with spinal cord injury was 44%, whereas it was 62% for those without spinal cord injury.19 Postoperative spinal cord injury is caused by several factors including ischemia, reperfusion injury, and postoperative hemodynamics. Many methods of protecting the spinal cord during thoracoabdominal aneurysm repair have been devised. These include preoperative spinal angiography, distal aortic perfusion, hypothermia, reattachment of the intercostal artery, cerebrospinal fluid (CSF) drainage, the administration of neuroprotective agents, and monitoring of somatosensory and motor-evoked potentials. We review these methods in this article.

Preoperative Identification of the Adamkiewicz Artery (Arteria Radicularis Magna, Great Radicular Artery)

Kieffer et al.20 performed preoperative spinal cord arteriography in patients with thoracic and thoracoabdominal aneurysms, and identified the arteria radicularis magna (ARM) in 85%. They reported that the risk of paraplegia was 5% if the ARM was identified preoperatively and reimplanted, whereas it was 50% if the ARM was not reattached. Williams et al.21 performed selective intercostal angiography in patients with thoracoabdominal aneurysm, and identified the ARM in 55%. By examining the angiographic localization of the spinal cord blood supply and its relationship to postoperative paraplegia, they concluded that selective intercostal angiography was safe and provided information to help understand the mechanisms and risks of spinal cord complications after thoracoabdominal aneurysm repair.

On the other hand, Minatoya et al.22 reported that postoperative paraplegia could not always be prevented, even in patients with an identified and reimplanted great radicular artery. Takase et al.23 identified
the artery of Adamkiewicz by using multidetector-row helical computed tomography in 90% of patients.

**Distal Perfusion**

Although the clamp and sew technique is used successfully in most cases,24,25 several studies confirm the need for an additional protective measure if the aortic cross-clamp time is longer than 30 min.14,18,27,28 Katz et al.29 reported a 71% incidence of spinal cord injury in patients with disease of the descending thoracic aorta and cross-clamp times longer than 30 min. Acher et al.13 performed cerebrospinal fluid (CSF) drainage and gave naloxone as additional protection, and reported a low incidence of spinal cord injury. Cambria et al.27 used the clamp and sew technique, with epidural perfusion cooling for protection of the spinal cord. Jex et al.30 reported that the risk of spinal cord injury decreased from 44% to 8% when distal aortic perfusing was used. Several techniques have been advocated to perfuse the distal aorta in a retrograde fashion.31,32 These methods include femorofemoral bypass, passive shunts, and left heart bypass. Femorofemoral bypass with roller pumps has been used for distal aortic perfusion since the 1960s. Partial or total cardiopulmonary bypass can be used to increase distal perfusion beyond the aortic clamps; however, the use of a roller pump requires systemic heparinization. In a series of patients undergoing cardiopulmonary bypass for dissection of the aorta, 20%–30% suffered complications related to hemorrhage and one third of the deaths resulted from bleeding.31–35

The Gott shunt tube has been used as a passive shunt;36 however, according to two published reports of traumatic tear of the thoracic aorta, the Gott shunt did not decrease the incidence of paraplegia from that associated with the clamp and sew technique.37,38 Moreover, to minimize spinal cord injury, the distal aortic perfusion pressure needs to be greater than or equal to 60 mmHg, which makes the Gott shunt less desirable than techniques that allow the flow to be actively maintained.

The centrifugal pump is used for left heart bypass,1,28,39 and provides the best means of maintaining distal aortic perfusion. By using centrifugal pumps flow can be regulated, there is less damage to the blood components than with roller pumps, minimal heparinization is required, and a reservoir can be used in the setting of trauma or a ruptured aneurysm.

Svenson and Loop40 reported that the risks of paraplegia were 2.2%, 2.3%, and 5.8% with bypass, passive shunts, and the clamp and sew technique, respectively. According to Kaplan et al.,41 active distal bypass perfusion achieved significantly greater distal aortic pressure than either the clamp and sew technique or passive shunting. However, the use of distal aortic perfusion cannot prevent spinal cord injury completely. Distal aortic perfusion increases blood flow to the distal aorta, but if the arteries supplying the anterior spinal artery arise from the excluded segment of the aorta, the spinal cord remains ischemic even if the distal aorta is perfused. The anatomical features of the anterior spinal artery must also be considered because there is a variation in size between the anterior spinal artery above and below the junction of the Adamkiewicz artery and the anterior spinal artery.40,42

**Hypothermia**

Hypothermia is the most reliable protective adjunct for the spinal cord, and is used by many surgeons. Hypothermia acts by decreasing oxygen consumption and inhibiting excitatory neurotransmitter release.43–46 Motoyoshi et al.47 reported that hypothermia also extended the production of heat shock protein in spinal cord motor neurons after reperfusion, and inhibited the level of apoptosis. There is a linear relationship between temperature and the duration of ischemia that may be tolerated by the spinal cord.48 There are two methods of cooling the spinal cord during thoracoabdominal aneurysm repair, namely, regional cooling and systemic cooling with cardiopulmonary bypass.

In 1961, Albin et al. demonstrated the effect and safety of regional spinal cord cooling.49 Around the same time, Negrin and Klauber50 reported using epidural catheters and perfusion with saline solution at a temperature of 7–15°C to achieve complete preservation of motor neurons after thoracic aortic cross-clamping. In 1993, Tabayashi et al.51 and Marsala et al.52 evaluated the effect of spinal cord cooling during spinal cord ischemia, and reported its usefulness in preventing ischemic spinal cord injury. Davison et al.53 devised and applied this method in eight patients undergoing thoracic or thoracoabdominal aneurysm repair, namely, regional cooling and systemic cooling with cardiopulmonary bypass.

Cardiopulmonary bypass may be used to lower the body temperature, thereby inducing systemic cooling. Some authors5,46,54,55 recommend profound hypothermic cardiopulmonary bypass and a period of circulatory arrest, whereas others recommend moderate hypothermic left heart or femorofemoral bypass. Kouchoukos et al.7 recommend profound hypothermia, based on the fact that there is no known association between the duration
of spinal cord ischemia and the development of paraplegia. Conversely, because this technique is associated with a high incidence of coagulopathy, pulmonary complications, and edema, some investigators recommend it only for patients who need concomitant repair of the transverse aortic arch, or if proximal control is hazardous because of rupture or the presence of atheromatous debris.56,57

Reattachment of the Intercostal and Lumbar Arteries

Ligation of patent T11 or T12 intercostal vessels was found to significantly increase the risk of neurological deficits after thoracoabdominal aneurysm repair when compared with vessel inclusion or pre-existing vessel occlusion from atherosclerotic occlusive disease.58 The rate of neurological deficits was 50% in the ligation group versus 12.9% in the reimplantation group and 9.9% in the pre-existing occlusion group. Reimplantation of the T9 and T10 intercostal arteries was associated with a significant reduction in delayed neurological events according to a multivariate analysis. Thus, reattachment of the intercostal arteries is important when deep hypothermic circulatory arrest is used as an adjunctive method during surgery, because of the frequently unstable postoperative hemodynamics.16

Some surgeons avoid intercostal reattachment.13,16 In fact, Acher et al.13 demonstrated excellent operative results without reattachment of intercostal arteries. They reported that quick oversewing of the intercostal arteries, with CSF drainage and naloxone administration, could help to reduce the incidence of spinal cord injury. Griepp et al.16 also showed that reattachment could be avoided by ligating the intercostal arteries before aortic cross-clamping while monitoring somatosensory evoked potential. These reports suggest that existing collateral vessels might improve the perfusion pressure.

Cerebrospinal Fluid Drainage

Blaisdell and Cooley33 and Miyamoto et al.59 reported that CSF drainage was beneficial for reducing the incidence of spinal cord injury in a dog model. It was also shown that the relative spinal cord perfusion pressure increased with the CSF drainage. This effect presumably increased the perfusion pressure of the spinal cord, resulting in a decrease in spinal cord injury. However, in pig and baboon models, the benefits of CSF drainage in reducing postischemic spinal cord injury were not proven.60,61 McCullough et al.62 also reported the protective effect of CSF drainage in reducing the incidence of paraplegia in a canine model. They62 then introduced CSF drainage for 24 patients undergoing surgery for a thoracoabdominal aneurysm, none of whom were complicated by postoperative spinal cord injury. Crawford et al.63 performed a prospective randomized study of the effectiveness of CSF drainage for preventing paraplegia, and reported that it was not beneficial in this regard. Conversely, a more recent prospective randomized study of CSF drainage by Coselli et al.,11 and a report by Safi et al.,10 showed that CSF drainage did help to prevent spinal cord injury.

Administration of Neuroprotective Agents

Several pharmacologic agents including free radical scavengers, barbiturates, corticosteroids, papaverine, cocaine-derived anesthetics, and opiate antagonists have been evaluated, both experimentally and in the clinical setting.60,64–75 to determine their protective effects against spinal cord injury. The main free radical scavengers tested were superoxide dismutase (SOD), allopurinol, and deferoxamine. Although SOD was found to be effective in preventing paraplegia within 30 min of spinal cord ischemia in dogs, it was not found to be effective with extended cross-clamp times. Moreover, Svensson et al.60 reported that allopurinol and SOD did not prevent paraplegia in a baboon model.

Barbiturates have been shown to protect the central nervous system from ischemia. Nylander et al.65 reported that barbiturates had a protective effect against spinal cord ischemia in a canine model, but Kirshner et al.66 reported that barbiturates did not have a protective effect when used alone.

The role of corticosteroids in the prevention of spinal cord injury is controversial. Laschinger et al.69 reported that pretreatment with steroids was protective against spinal cord ischemia in a canine model, and Bracken et al.68 reported that treatment with methylprednisolone improved neurologic recovery in patients with acute spinal cord injuries when the medication was given in the first 8 h. Conversely, another recent report showed that high-dose steroid therapy was not useful for patients with spinal cord traumatic injury.69 Fowl et al.70 found that 21-amino steroid helped to prevent postischemic spinal cord injury in a rabbit model. However, the protective effect of steroids could be related to stabilization of the cell membrane, modulation of the immune system, and free radical scavengers.

Svensson et al.60 showed that CSF drainage plus an intrathecal papaverine injection increased the spinal cord blood flow, inducing a protective effect against spinal cord injury. Furthermore, a preliminary trial of intrathecal papaverine in humans by these authors showed no adverse side effects, or any case of paraplegia.40
The effects of cocaine-derived anesthetics on rat brain cells were also studied. Lidocaine, one of the cocaine-derived anesthetics, enhanced the recovery of somatosensory evoked potentials after ischemia in an acute cerebral model.71 Robertson et al.72 evaluated the protective effect of lidocaine followed by aortic occlusion in a rabbit model. However, the protective effect of tetracaine is probably related to the suppression of cellular metabolism and stabilization of the neuronal cell membrane.

Investigators have found that levels of endogenous opiates rise in the CSF of dogs with spinal cord ischemia. Opiates seem to decrease cerebral blood flow, increase vascular resistance, and decrease central nervous system acetylcholine turnover. It has been reported that naloxone, an opiate antagonist, improves neurologic recovery from trauma and spinal cord ischemia.74,75 Acher et al.13 found that naloxone with CSF drainage effectively lowered the incidence of spinal cord injury in patients undergoing thoracic and thoracoabdominal aortic aneurysm repair.

Somatosensory Evoked Potentials and Motor-Evoked Potentials

Somatosensory evoked potentials (SSEP) are designed to monitor spinal cord perfusion.76 Cunningham et al.77 and Schepens et al.78 demonstrated the usefulness of SSEP in lowering the incidence of spinal cord injury in patients undergoing thoracic and thoracoabdominal aneurysm repair; however, SSEP tracing has several limitations. First, SSEP tracings are altered by some anesthetic agents, hypothermia, and neuromuscular blockade; second, SSEP only evaluates the function of the posterior and lateral columns of the spinal cord; and third, there are reports of false-positive and false-negative responses during the intraoperative monitoring of SSEP.79

Motor-evoked potentials (MEP) can evaluate the function of anterior columns of the spinal cord. Laschinger et al.80 evaluated the usefulness of MEP to monitor spinal cord perfusion, while van Dongen et al.81 reported that MEP monitoring was feasible during low-dose propofol, fentanyl/50% N₂O in O₂ anesthesia, and partial neuromuscular blockade.

In summary, despite remarkable advances in the surgical treatment of thoracoabdominal aneurysms, postoperative spinal cord injury remains a devastating complication. Several methods of preventing paraplegia have been proposed and evaluated. The information to date suggests that multimodality approaches should be used to prevent spinal cord injury after thoracic and thoracoabdominal aortic aneurysm repair.

References


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