

# THORACOABDOMINAL ANEURYSM REPAIR: A CASE REPORT

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*Aneurysms that extend from the descending thoracic aorta into the abdomen, which can also involve the visceral segments of the upper abdominal aorta, are traditionally classified as thoracoabdominal. Thoracoabdominal aortic aneurysm reconstruction is very complex vascular surgery associated with high postoperative morbidity and mortality and related postoperative complications. In addition to the surgical complexity associated with repair of these aneurysms, the temporary interruption of blood flow distal to the clamp introduces multiple considerations for the anesthetic practitioner to consider for the reduction of potential complications.*

*This case report involves an 80-year-old female presenting for elective repair of a type IV thoracoabdominal aortic*

*aneurysm, with utilization of standard invasive hemodynamic monitoring and cerebrospinal perfusion pressure monitoring as a neuroprotective measure. Hemodynamic stability was maintained via vasoactive agents, as well as compensatory vascular repletion guided by invasive monitoring. A definitive anesthetic plan based on thorough preoperative assessment, insistent intraoperative management techniques to minimize potential complications, and postoperative management of this patient allowed for successful vascular reconstruction, resulting in a positive patient outcome.*

**Key words:** Aneurysm, spinal cord perfusion, thoracoabdominal aortic aneurysm.

**A**n aneurysm is an abnormal enlargement of a portion of a blood vessel. This type of enlargement causes weakening of the wall of the vessel and has the potential for rupture or clot formation. Aneurysms commonly occur in the aorta and are classified in 1 of 2 categories; “true” aneurysms that are caused by factors such as atherosclerosis, syphilis, and some degenerative diseases, or “false” aneurysms occurring as a result of trauma or nonaneurysmal aortic lesions.<sup>1</sup> In a “true” aneurysm, all 3 layers of the aortic wall are involved in a destructive cycle of degeneration, stretching, and increased tension that increases the chance of rupture. This may be caused by a basic defect in elastin production or function that leads to elastin overload, followed by collagen overload and dilation of the aorta combined with an excessive force or pressure load.<sup>1</sup>

The patient in this case study presented for repair of a thoracoabdominal aortic aneurysm. These aneurysms are typically incidental findings diagnosed from routine chest radiographs, computed tomography scans, or magnetic resonance imaging performed because of other symptoms not related to the aneurysm.<sup>2</sup> The lack of remarkable symptoms can be

ominous for the patient. The aneurysm may be critical before symptoms appear. When patients are fortunate enough to have early diagnosis, commonly an antihypertensive drug treatment is employed. Despite medical management, research reports have shown that aneurysms continue to grow in size with increased risk of rupture. Once aneurysms reach a critical size of 5 cm to 6 cm or show an accelerated growth of 1 cm per year, surgery is indicated.<sup>3</sup> Until recently, surgical treatment of thoracoabdominal aortic aneurysms (TAAAs) has been associated with a very high operative mortality and morbidity rate. However, recent advances in anesthetic techniques, monitoring capability, and perfusion technology that reduce both ischemic injury and reperfusion apoptosis have collectively contributed to improved patient outcomes.<sup>4</sup>

Despite recent advances, the risks involved and challenges associated with the surgical treatment of a TAAA are great. The focus of anesthetic management of patients with TAAA is to preserve perfusion of the spinal cord, kidneys, and mesenteric viscera and minimize ischemia that may occur during the cross-clamping of the aorta during the procedure.<sup>5</sup> It has been documented that cessation of blood flow to these

organs can be safely tolerated for up to 20 to 30 minutes; however, the surgical procedure may last 3 to 4 hours, and effective application of circulatory management techniques is necessary.<sup>6</sup>

## Case report

An 80-year-old ASA class 3 female presented for repair of a type IV descending TAAA discovered on a routine physical examination by her primary care physician. She was referred to a vascular surgeon following an aortic angiogram that revealed a 6.5 cm TAAA. The patient's past medical history was significant for coronary artery disease, hypertension, hyperlipidemia, renal insufficiency, atrial fibrillation, and degenerative joint disease. Her surgical history included a 3-vessel coronary artery bypass grafting in 1997, bilateral cataract removal, and a hysterectomy. Anesthetic history was insignificant. The patient reported no known allergies, and current medications included aspirin, ramipril, diltiazem, losol, nitroglycerin transderm, metoprolol, zocor, and a multivitamin. Aspirin and the multivitamin were stopped 9 days prior to surgery.

The patient was evaluated in the preoperative holding area by the anesthesia team. Consents for general anesthesia, invasive monitoring, blood administration, and the possible use of cardiopulmonary bypass intraoperatively for TAAA repair were obtained. The patient's admission height and weight were 167 cm and 74 kg respectively. The airway was assessed as a Mallampati class II. Preoperative baseline vital signs were as follows: pulse, 67 beats per minute; respiratory rate, 18 breaths per minute; blood pressure, 166/72 mm Hg; and oxygen saturation on room air was 94%. Preoperative electrolytes, partial thromboplastin, and prothrombin times were within normal limits. The patient's hemoglobin and hematocrit were 11.7 g/dL and 35.7% respectively, platelets were 181,000, and creatinine was 1.0 mg/dL. Preoperative 12-lead electrocardiograms with comparative studies revealed resolved multifocal ST and T wave changes indicative of inferior lateral ischemia, and additional electrocardiograms reviewed from previous admissions revealed episodic atrial fibrillation following coronary artery bypass grafting. Preoperative chest radiography revealed normal postsurgical changes and was considered within normal limits.

The patient was transported to the operating room with an 18-gauge peripheral intravenous line in the left upper extremity with Ringer's lactate solution infusing. In the operating room, standard monitors were applied. Midazolam, 1 mg, was administered, and a 14-gauge left antecubital intravenous line was placed. A lumbar drain was inserted with the patient

in the lateral position. The lumbar drain was secured, and the transduced pressure was 14 mm Hg. The patient was then placed supine. A total of 50 µg of fentanyl was titrated in 25-µg increments for invasive line insertion. A right radial arterial line, left femoral arterial line, and right internal jugular 9 Fr introducer with pulmonary artery catheter were all placed. The procedures were tolerated well with minimal sedation. Vital signs remained stable throughout these procedures with systolic blood pressure ranging from 112 to 136 mm Hg.

Anesthesia was induced intravenously with fentanyl, 550 µg; midazolam, 4 mg; and pancuronium, 10 mg. A 37 Fr double lumen tube was inserted without difficulty under direct laryngoscopy. The double lumen endotracheal tube placement was confirmed with manual auscultation of individual lung ventilation and fiberoptic bronchoscopy. The patient was positioned supine with a slight lateral tilt and beanbag stabilization. Her arms were padded and tucked at her side. Her head and neck were maintained in a neutral position on a donut pillow. The patient was surgically prepped with sterile solutions that included prepping of bilateral femoral sites. One-lung ventilation was established shortly after induction of anesthesia with a minimal increase in peak inspiratory pressures and adequate tidal volumes. Vital signs remained stable throughout this period of time with a 20% to 25% reduction in systolic pressures that required the use of a phenylephrine, 80 µg bolus, immediately postinduction.

Anesthesia was maintained with 100% oxygen and titration of end tidal isoflurane 0.9% to 1.3%. No further administration of pancuronium was required. A total of 1,000 µg of fentanyl was administered throughout the case.

Prior to aortic cross-clamping, 5,000 units of heparin, 20 mg of furosemide, and small incremental boluses of phenylephrine were administered intravenously; a systolic blood pressure of 110 mm Hg was achieved before cross-clamping. With the establishment of aortic cross-clamping, the immediate rise in systolic pressure from 92 mm Hg to 144 mm Hg was treated with incremental boluses of nitroglycerin, 50 µg. Lumbar drain pressures increased from 8 mm Hg to 13 mm Hg, and 30 mL of cerebrospinal fluid (CSF) was passively drained into a cameo collection chamber to reduce the lumbar pressure to 9 mm Hg. An additional 30 mL of CSF was passively drained with a further decrease in the lumbar pressure to 5 to 6 mm Hg. Throughout the case, 20 to 30 mL increments of CSF were drained to maintain a lumbar pressure of 4 to 8 mm Hg, with a total of 120 mL of CSF drainage for the case.

Approximately 30 minutes after aortic incision, hemoglobin and hematocrit were measured at 9.9 g/dL and 29.0% via arterial blood gas sample, and 1 unit of packed red blood cells was transfused. After an additional blood loss of 300 mL, the patient was transfused with 450 mL of cell-saver blood. With continued blood loss, systolic blood pressure decreased, and incremental phenylephrine and ephedrine boluses were administered to maintain systolic pressures of 110 mm Hg. Calcium chloride, 500 mg, was administered, and a second unit of packed red blood cells was transfused, as well as 500 mL of 5% albumin. Serial arterial blood gases were obtained hourly. The paravisceral, as well as the descending thoracic aorta, were reconstructed with a 22 x 11 mm Hemashield (Boston Scientific Corporation, Natick, Mass) bifurcated graft. Circulatory flow to the mesenteric and renal systems was restored after 41 minutes. After proximal reconstruction, distal reconstructions were performed, and the right common iliac artery was anastomosed to the bifurcated graft.

An arterial blood gas was drawn shortly after right iliac reperfusion, and the results were as follows: pH, 7.40; PaO<sub>2</sub>, 304 mm Hg; PaCO<sub>2</sub>, 29 mm Hg; HCO<sub>3</sub><sup>-</sup>, 20.5 mEq; base deficit, -7.8; hemoglobin, 9.0 g/dL; hematocrit, 27.0%; sodium, 142 mEq/L; and potassium, 3.8 mEq/L. Based on these results, 25 mEq of sodium bicarbonate was administered and a third unit of packed red blood cells was administered. The left common iliac aneurysm was resected and reconstructed with an 11-mm Hemashield graft and anastomosed to the body of the graft. The remaining 25 mEq of sodium bicarbonate was administered before reperfusion of the left iliac artery.

After all reconstructions were complete, a considerable amount of time was dedicated to hemostasis at all anastomotic sites and suture lines with hemostatic agents. Protamine, 30 mg, was then administered intravenously. Before completion of the abdominal closure, an additional 405 mL of cell-saver blood and 2 units of fresh frozen plasma were administered. Dual lung ventilation was established after reconstruction of the diaphragmatic division, and as surgical closure concluded, systolic blood pressure increased to 130 mm Hg. Beta blockade was initiated by incremental administration of metoprolol resulting in a 10-mg total dosage over a 30-minute period. Systolic blood pressure was maintained at 115 to 120 mm Hg for transport to the cardiothoracic intensive care unit.

Total fluids administered to the patient included 3,600 mL of crystalloid, 750 mL of colloid, 3 units of packed red blood cells, 855 mL of autologous cell-

saver transfusion, and 463 mL of fresh frozen plasma. Total patient urine output was 680 mL, blood loss was estimated at 2,100 mL, and cerebrospinal fluid drainage totaled 120 mL.

Upon awakening from anesthesia, the patient followed all commands appropriately, and motor responses were equal bilaterally. A propofol drip was initiated and titrated between 20 and 40 µg/kg per minute for postoperative tolerance of the endotracheal tube. Subsequently, he was weaned from the ventilator and extubated 14 hours postoperatively. Immediate postoperative laboratory results were as follows: hemoglobin, 11.0 g/dL; hematocrit, 32.8%; platelets, 91,000; blood urea nitrogen (BUN), 20 mg/dL; and creatinine, 1.1 mg/dL. Postoperative urine output remained between 200 and 400 mL hourly for a total of 3,100 mL. Laboratory values on the first postoperative day revealed an increase in BUN and creatinine, 25 mg/dL and 1.6 mg/dL respectively. Platelets continued to decrease to 86,000; however, hemoglobin and hematocrit levels stabilized at 11.7 g/dL and 34.5%. The patient's postoperative platelet count continued to decrease until she received a dose-pack of platelets on the third postoperative day. The patient was discharged 7 days after surgery to a skilled nursing center, neurologically intact, and ambulating with minimal assistance. Her final laboratory values upon discharge included hemoglobin, 11.4 gm/dL; hematocrit, 34.5%; platelets, 203,000; BUN, 29 mg/dL; and creatinine, 1.8 mg/dL.

## Discussion

Patients undergoing surgery involving the aorta face dramatic changes in hemodynamic and cardiac function that can have profound systemic effects. Therefore, extensive invasive monitoring is required. Goals during induction of anesthesia include maintaining hemodynamic stability while completely blunting the sympathetic response to direct laryngoscopy and intubation. Combinations of short-acting vasodilators and beta blockers, as well as alpha agonists, are most commonly used to maintain cardiac stability.

Application of aortic cross-clamping on the descending aorta produces a dramatic and instantaneous increase in systemic vascular resistance. This can produce profound hypertension. Maintenance of baseline blood pressures should be established to prevent devastating complications such as intracranial hemorrhage, congestive heart failure, and myocardial complications.<sup>1</sup> Target blood pressure should be maintained within 20 mm Hg of precross-clamp values. Most often, vasodilators such as nitroglycerin, sodium nitroprusside, and short-acting beta-blockers,

such as esmolol, are employed to maintain baseline blood pressure.<sup>7</sup>

The period of greatest hemodynamic instability is following the release of the aortic cross-clamp, referred to as “release hypotension.” This results in an immediate decrease in afterload in combination with reperfusion acidosis (due to the release of lactic acid) from previously hypo-perfused tissue causing systemic hypotension. This effect, coupled with decreased preload and possible anastomotic bleeding, is an exceptionally challenging period for the anesthetist. If the anastomotic bleeding is profound, or blood pressure is unphysiologic and refractory to vasoactive support, it may be necessary to cross-clamp the aorta until fluid balance and hemostasis can be restored.

Renal failure following aortic surgery is 3% to 14% and is associated with prolonged cross-clamp time and hemodynamic instability.<sup>4</sup> Renal protection is an area of controversy, but commonly used protective strategies include fenoldopam infusion, mannitol, furosemide, mucomyst, and dopamine.<sup>7</sup> Furosemide was used in this case because of its ability to decrease metabolic oxygen demand and was administered at a dose of 20 mg before cross-clamping the aorta.

A major concern with reconstruction of TAAAs is postoperative paraparesis or paraplegia. Neurologic complications during aneurysmal repair can be as high as 40%.<sup>5</sup> These higher rates are associated with periods of cross-clamping lasting longer than 30 minutes, as well as extensive reconstructions. The majority of these complications can be traced to the physiologic response to altered blood flow in the anterior aspect of the spine. The anterior spinal artery supplies two thirds of the spinal tissue and tapers distally. The artery of Adamkiewicz joins the anterior spinal artery originating from T9 to L3 in 80% of the population.<sup>5</sup> This artery is compromised with aortic cross-clamping, causing a decrease in spinal perfusion. Opening the aneurysm causes further decrease in spinal perfusion as a blood steal is induced from the spinal arteries with higher resistance.<sup>6</sup> In an effort to lessen spinal cord ischemia and reduce postoperative paraplegia, a lumbar spinal catheter is placed to monitor spinal cord perfusion pressure (SCPP). SCPP is calculated by subtracting the central venous pressure or spinal CSF (whichever is higher) from the distal aortic pressure (mean arterial pressure). Failure to maintain a distal perfusion pressure above 60 mm Hg is associated with a high rate of paraplegia.<sup>8</sup> Cross-clamping of the aorta results in increases in CSF pressure, increased venous congestion of dural veins, and a reduction of venous capacitance.<sup>9</sup> Maintaining an SCPP greater than 30 mm Hg with a reduction of CSF pressure to a goal

pressure of 5 to 15 mm Hg increases blood flow in the anterior spinal artery.<sup>5</sup>

Current thoughts on neuroprotection are to employ a combination of strategies to optimize outcome. Monitoring somatosensory evoked potentials (SSEPs) and motor evoked potentials (MEPs) allows for early detection of neurological deficits.<sup>10</sup> These evoked potentials are the result of electrical stimulation and measure changes in the dorsal columns and ventral columns respectively. Spinal cord ischemia is indicated when amplitude of these potentials is diminished and latency (the time between stimulus initiation and detection) is increased. Normal amplitude and latency components of evoked potentials are maintained at cerebral blood flow levels of 20 mL/100 g per minute or above. Amplitude measures decline 50% with latency prolongations with cerebral blood flow levels of 16 mL/g per minute. Maintaining baseline SSEPs and MEPs waveforms improves good neurologic outcomes.<sup>11</sup>

Hypothermic techniques including epidural cooling with normal saline to achieve a rectal temperature of 34°C or passive cooling to 34°C has been used to increase ischemic tolerance.<sup>12</sup> Deep hypothermia at 20°C decreases metabolic needs by nearly 70% and allows for an open repair technique with a circulatory arrest time of 30 minutes.<sup>5</sup> The Gott shunt (Sherwood Medical Company, St Louis, Mo.) and the “clamp and go” are surgical techniques for maintaining blood flow to the spinal cord.<sup>8</sup> The Gott shunt is a heparinized tube that is placed to improve distal perfusion and to attenuate the hemodynamic response to aortic unclamping.<sup>11</sup> Cardiopulmonary bypass also may be used to maintain adequate oxygenation of tissues. There also are numerous pharmacologic interventions for neuroprotection that can possibly reduce the effects of aortic cross-clamping and subsequent spinal cord ischemia. Methylprednisone is considered to be one of the most effective pharmacologic agents at reducing inflammatory reaction.<sup>13</sup> It is often administered (7 mg/kg) at the onset of spinal cord ischemia (aortic cross-clamping). Allopurinol, a potent inhibitor of xanthine oxidase, reduces perfusion injury by limiting the deleterious effects of free oxygen radicals. Adenosine has neuroprotective effects by decreasing ischemia induced glutamate neurotoxicity, as well as controlling nitric oxide production.<sup>14</sup>

This case used SCPP only, although cardiopulmonary bypass was on standby in the operating room should the aortic cross-clamp time become extensive in light of the patient’s preexisting renal insufficiency.

TAAA repair is an extremely complex procedure. Perioperative management of these patients pose chal-

lenges that can only be met with proper planning and aggressive preoperative, intraoperative, and postoperative management. Anesthetic technique has to be modified according to the extent of the disease, surgical procedure, and many comorbid conditions and complications that are seen in these patients. Spinal cord and neuroprotective measures have to be an integral part of anesthetic management if devastating outcomes are to be avoided.

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