

Open repair of chronic thoracic and thoracoabdominal aortic dissection using deep hypothermia and circulatory arrest



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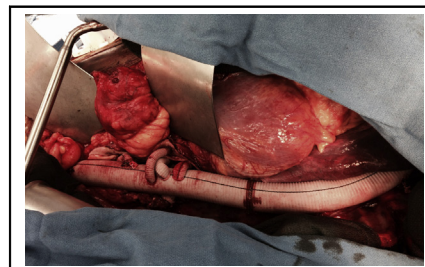
ABSTRACT

Background: Chronic dissection of the thoracic and thoracoabdominal aorta as sequela of a prior type A or B dissection is a challenging problem that requires close radiographic surveillance and prompt operative intervention in the presence of symptoms or aneurysm formation. Open repair of chronic thoracic and thoracoabdominal aortic dissection using deep hypothermia has been our preferred method to treat this complex pathology. The advantages of this technique include organ and spinal cord protection, the flexibility to extend the repair proximally into the arch, and the ability to limit ischemia to all vascular beds.

Methods: Open repair of arch by left thoracotomy and descending thoracic and thoracoabdominal aortic pathology using deep hypothermia was performed in 664 patients from 1995 to 2015. A subset of this cohort had chronic thoracoabdominal aortic dissection (n = 196). All nonemergency cases received coronary angiography and echocardiography preoperatively. Significant coronary artery disease or severe aortic insufficiency was addressed before repair of the chronic dissection. In recent years, lumbar drains were placed preoperatively in the most extensive repairs (extents II and III). Important intercostal arteries from T8 to L1 were revascularized with smaller-diameter looped grafts. Multibranched grafts for the visceral segment have been preferred in recent years.

Results: Mean age of patients was 58 ± 14 years. Men comprised 74% of the cohort. Aortopathy was confirmed in 18% of the cohort. Prior thoracic aortic repair occurred in 57% of patients, and prior abdominal aortic repair occurred in 14% of patients. Prior type A aortic dissection occurred in 44% of patients, and prior type B occurred in 56% of patients. Operative mortality was 3.6%, permanent spinal cord ischemia occurred in 2.6% of patients, permanent hemodialysis occurred in 0% of patients, and permanent stroke occurred in 1% of patients. Reexploration for bleeding was 5.1%, and respiratory failure requiring tracheostomy occurred in 2.6%. Postoperative length of stay was 11.9 ± 9.7 days. Reintervention for pseudoaneurysm or growth of a distal aneurysm was 6.9%. The 1-, 5-, and 10-year survivals were 93%, 79%, and 57%, respectively.

Conclusions: Open repair of chronic thoracic and thoracoabdominal aortic dissection using deep hypothermia and circulatory arrest has low morbidity and mortality. The need for reintervention is low, and long-term survival is excellent. We believe that open repair continues to be the gold standard in patients who are suitable candidates for surgery. (*J Thorac Cardiovasc Surg* 2017;154:389-95)



Completed repair of a chronic thoracoabdominal aortic dissection and aneurysm.

Central Message

Open repair of chronic thoracic and thoracoabdominal aortic dissection using deep hypothermia and circulatory arrest provides excellent early and late outcomes.

Perspective

For the suitable operative candidate, open repair of chronic thoracic and thoracoabdominal aortic dissection should remain the gold standard. All patients with connective tissue disorders should receive open repair. The open repair is definitive, rarely requires secondary procedures for the treated aortic segment, and has proven durability.

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Abbreviation and Acronym

IQR = interquartile range

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Chronic dissection of the thoracic and thoracoabdominal aorta as sequela of a prior type A or B dissection is a challenging problem that requires close radiographic surveillance and prompt operative intervention in the presence of symptoms or aneurysm formation. Every thoracoabdominal aortic dissection is unique in its combination of primary entry tear, reentry tears, fenestrations, extent of aortic involvement, involvement of branch vessels, flow distribution, and interplay between the true and false lumens. This complex pathology typically does not lend itself to a simple surgical solution.

Open repair of chronic thoracic and thoracoabdominal aortic dissection using deep hypothermia has been our preferred method to treat this complex pathology. The advantages of this technique include organ and spinal cord protection, the flexibility to extend the repair proximally into the arch, and the ability to limit ischemic injury to important vascular beds. The disadvantages include longer perfusion times and limited applicability to the ruptured aneurysm. The entire thoracoabdominal dissection can be addressed at once if the entire thoracoabdominal aorta is aneurysmal. Alternatively, only the aneurysmal portion needs to be replaced with fenestration of the dissection septum at the distal extent of the repair.

This study highlights our experience with open repair of arch by left thoracotomy and descending thoracic and thoracoabdominal aortic dissection using deep hypothermia and circulatory arrest.

MATERIALS AND METHODS

Institutional Review Board of Indiana University approval was obtained for the study. Individual patient consent was waived. From January 1995 to December 2015, 664 patients underwent open thoracic (by left thoracotomy) or thoracoabdominal aneurysm repair using deep hypothermia and circulatory arrest. The technique has been described.^{1,2} Briefly, anesthetic technique is composed of total intravenous anesthesia using propofol and remifentanyl to allow preservation of motor-evoked and somatosensory-evoked potentials for spinal cord monitoring. Placement of a double-lumen endotracheal tube (Covidien Mallinckrodt Endobronchial tube, left; Medtronic, Inc, Minneapolis, Minn) or single-lumen tube with a bronchial blocker (Arndt Endobronchial Blocker Set; Cook Medical Inc, Bloomington, Ind) is used for selective airway control. Transcranial

infrared oxygen sensors are placed on the left and right forehead (INVOS Cerebral/Somatic Oximeter; Somenetics Corporation, Troy, Mich). Motor-evoked and somatosensory-evoked potentials (Cadwell Cascade stimulator-detector, disposable subdermal needle electrodes; Cadwell Laboratories, Inc, Kennewick, Wash) are recorded after induction of anesthesia for baseline values and are assessed intraoperatively after separation from cardiopulmonary bypass.

The patient is placed on full cardiopulmonary bypass typically through the left common femoral artery and vein. The arterial inflow temperature is reduced to achieve 15°C. Once the heart fibrillates, decompression of the left ventricle is performed by placing a drainage catheter (Covidien Argyle, ventricular sump catheter, Medtronic, Inc, Minneapolis, Minn) through the left ventricular apex to low active suction. Once the patient has been cooled for 30 minutes with an arterial blood temperature of less than 20°C, circulatory arrest is performed. The left ventricular sump catheter is turned off, the venous line is clamped, and arterial flow is stopped. A crossclamp is placed distal to the proposed proximal anastomosis on the descending thoracic aorta. Hypothermic low flow (1-1.5 L/min) is started to the lower body while the proximal anastomosis is performed. If the entire transverse arch is to be replaced, perfusion catheters (Gundry Silicone RCSP cannula, Medtronic, Inc) are placed in the innominate and left common carotid arteries for bilateral selective antegrade cerebral perfusion at 10 mL/kg/min. After the arch is reconstructed or the proximal anastomosis performed, perfusion is restored to the upper body (1 to 1.5 L/min) and the repair progresses stepwise proximally to distally. Intercostal arteries, 3 to 4 levels, from T8 to L1 are revascularized. When the abdominal aortic segment is opened, perfusion catheters (9F Pruitt Irrigation Occlusion Catheter; LeMaitre Vascular, Inc, Burlington, Mass) are placed into the orifices of the visceral vessels, and low-flow hypothermic blood perfusion is instituted at 200 to 300 mL/min. After reconstruction of the abdominal visceral segment is completed and the abdominal viscera reperfused, rewarming can commence with commensurate increases in cardiopulmonary bypass flow. Finally, the distal anastomosis is performed at the infrarenal aorta at the bifurcation or prior abdominal aortic graft. If the common iliac arteries are aneurysmal, iliac artery reconstructions also are performed.

As the patient is being rewarmed, the heart is defibrillated when the arterial inflow temperature is greater than 30°C. Once rewarmed to tympanic and bladder temperatures greater than 35°C, the patient is weaned from cardiopulmonary bypass. Vasopressor support is used to achieve a mean arterial pressure of 70 to 90 mm Hg. If motor-evoked potentials are not present in the bilateral lower extremities, the mean pressure is elevated to 90 to 110 mm Hg, and a lumbar drain is placed before leaving the operating room if it was not placed at the beginning of the procedure. Immediate postoperative hemoglobin is maintained at greater than 8 mg/dL.

Recent technical changes in our surgical strategy for repair of all pathologies of thoracic and thoracoabdominal aneurysm have included the addition of arterial cannulation sites in the ascending aorta, distal aortic arch, proximal descending aorta, and left or right common carotid arteries to effort to avoid retrograde flow in the thoracoabdominal aorta. However, the majority (188/196) of the patients with chronic dissection had common femoral arterial cannulation for cardiopulmonary bypass. We also have championed the use of branched surgical grafts or smaller-diameter grafts to reconstruct the abdominal visceral arteries. In addition, revascularization of intercostal arteries is now performed using a small-diameter looped bypass graft.³ It is our thought that the use of smaller diameter grafts will reduce the likelihood of visceral or intercostal patch pseudoaneurysm.

Of 664 patients who underwent thoracic (by left thoracotomy) or thoracoabdominal aortic aneurysm repair, 196 patients, who comprise the current study, had chronic thoracoabdominal aortic dissection from prior type A or B dissection. Indications for repair included an overall maximal size of the aneurysmal aorta to be at least 50 to 55 mm in diameter or rapid growth of the aorta at a rate greater than or equal to 5 mm per year by computed tomography imaging (and since 2012 with center-line reconstruction). Patients with a known or suspected connective tissue disorder or a known

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