Acute Type A Aortic Dissection: Surgical Intervention for All: PRO

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KEYWORDS

• Type A aortic dissection • Aortic surgery
• Tamponade • Malperfusion

Acute aortic dissection remains the most common of all aortic catastrophes and is associated with significant morbidity and mortality. The mortality for acute type A aortic dissection has been suggested to be 1% per hour in the first 48 hours, but in reality, as reported by Hirst, was 21% at 1 day, 37% at 2 days, 62% at 1 week, 74% at 2 weeks, and 93% at 1 year.[1] Without surgical intervention, early death occurs as a result of malperfusion syndromes (cerebrovascular, visceral, renal, or peripheral ischemia), cardiac complications (acute aortic insufficiency, coronary ischemia, cardiac tamponade), or free rupture.

Thus for most patients, expeditious surgical intervention for this devastating condition should be undertaken.[2,3] Certain conditions or situations such as acute stroke, previous cardiac surgery (PCS), and older age have been associated with poor outcomes and have led some to recommend nonoperative or medical management for acute type A aortic dissection. Every patient should be individualized, and there are certain cases such as neurologic devastation that should not undergo surgical intervention, but in general, most patients with acute type A aortic dissection should undergo expeditious repair. The authors describe a relatively uniform approach to acute type A aortic dissection and provide recommendations and details on how they deal with these less-favorable situations.

INITIAL PRESENTATION

Because most patients with acute aortic dissection (either type A or type B) initially present with hypertension, the immediate aim is to control blood pressure, alleviate pain, and halt the progression of dissection. Less frequent (<10%) are those that present with refractory hypotension; these patients should be considered for emergent transfer to the operating room even if the diagnosis has not been confirmed. In these cases, the use of transesophageal echocardiography becomes pivotal in differentiating proximal aortic involvement (type A) from distal aortic involvement (type B). But for most, admission to the cardiovascular intensive care unit (CVICU) for administration of anti-impulse therapy can be undertaken. Even if urgent surgery is decided on, transfer to CVICU is still performed because monitoring and administration of antihypertensive agents can be performed more expeditiously in an intensive care setting than in the emergency center.

Rupture remains the primary concern at initial presentation with acute type A aortic dissection, but, interestingly, few data exist regarding the effect of adequate anti-impulse therapy (mean arterial pressure <80 mm Hg, heart rate <60) on the risk of rupture in this setting. Although rare, cases of early rupture and death have occurred in the authors' experience only when anti-impulse

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therapy could not achieve the optimal hemodynamics. Thus, for the framework of the following discussion, when surgical intervention is discussed, it is assumed that optimal medical management (adequate anti-impulse therapy) has already been applied. The authors believe that medical management should be applied to all patients regardless of comorbidities; persons not candidates for surgical repair (very rare) are thus considered nonoperative.

SURGICAL APPROACH

In principle, like others, the authors[4] prefer a simplistic approach to repair that addresses the primary problems: prevention of rupture/tamponade with replacement of the ascending aorta, correction of aortic valvular insufficiency with resuspension and aortic root reconstruction, and avoidance of further malperfusion with excision of the tear and obliteration of the false lumen if possible. At present, it is rare that the aortic valve (<5%), aortic root (<5%), or transverse aortic arch (<5%) is replaced.

In patients managed surgically, repair is via a median sternotomy, using full heparinization, cardiopulmonary bypass, and profound hypothermic circulatory arrest with retrograde cerebral perfusion. Arterial cannulation is accessed most often via the femoral artery, with few occasions requiring axillary or aortic access. Open distal anastomosis is performed under circulatory arrest. The authors[4] agree that open distal reconstruction allows for complete excision of the ascending aorta and prevents anastomotic leaks. Cerebral oximetry, transcranial Doppler ultrasound, and systemic and nasopharyngeal temperatures aid in cerebral monitoring. Electroencephalograms are used to monitor cerebral function and determine the time to initiate circulatory arrest. For typical dissection, obliteration of the false channel is performed with resection of the tear if feasible. All patients are supplemented with retrograde cerebral perfusion. After completion of the distal anastomosis, antegrade systemic flow is reestablished via a commercially available side-armed branched graft. Abnormal aorta is resected to the sinotubular junction. The aortic valve is resuspended, and the aortic root is reconstructed with complete obliteration of the false lumen. This watertight root reconstruction prevents retrograde dissection, maintains valve competency, and prevents late root enlargement.[5]

CEREBRAL MALPERFUSION

Cerebral malperfusion can occur at any point in the operative period during repairs of acute type A aortic dissection, leading to neurologic complications. The incidence of stroke and temporary neurologic dysfunction can be as high as 40%, with devastating long-term consequences.[6,7]

It was previously hypothesized that identifying and correcting cerebral malperfusion would improve neurologic outcome during these repairs, and the authors demonstrated that during the course of operative repair, significant cerebral malperfusion occurred in 29% of patients.[8] Thus, the high incidence of cerebral malperfusion during repairs of acute type A aortic dissection remains a problem and may go undetected without any neurologic monitoring.

CEREBRAL MONITORING EQUALS CEREBRAL PROTECTION

The results of the study using power motion mode transcranial Doppler ultrasound (PM-TCD) to guide retrograde cerebral perfusion during repairs of the ascending and transverse aortic arch was previously reported.[9] In this study, it was identified that an ''opening'' retrograde cerebral perfusion (RCP) pressure was required to identify reversal of blood flow in the middle cerebral arteries. In addition, when using standard RCP flow and pressure (0.5 L/min and <25 mm Hg), reversal of middle cerebral artery (MCA) blood flow was identified in only 20% of cases, and 80% of cases required some modification of RCP flow. More recently, the use of PM-TCD during repairs of acute type A aortic dissection was examined. About 78.5% of patients required modification of RCP flow to identify reversal of MCA blood flow. In addition, the RCP pressures in this study were significantly higher than in the control group, 33.3 $\pm$ 7.1 versus 26.7 $\pm$ 10.6, $P = .008$. This higher pressure was again the opening pressure required to identify reversal of MCA blood flow (**Fig. 1**).[8]

The requirement of a higher opening pressure (as opposed to maintenance pressure) may be related to either an increase in cerebral venous resistance as a result of the conversion from antegrade to retrograde perfusion or the need to overcome competent venous valves.[10] The concept of an opening pressure has also been described for monitoring the adequacy of antegrade selective cerebral perfusion.[11] At any rate, standard RCP flows and pressures (0.5 L/min and 25 mm Hg) may not be adequate to achieve reversed cerebral perfusion during RCP.

Many controversies still remain regarding the optimal technique for cerebral protection (RCP vs selective antegrade cerebral perfusion) and the optimal cannulation approach for the