Thoracic and Cardiovascular Surgery

Indications, Timing, and Prognosis of Operative Repair of Aortic Dissections

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Since the first description of aortic dissection in the 1700s, the understanding and treatment of this catastrophic disease has evolved. Aortic dissections are identified as a tear in the aortic intima and inner layer of the media that allows for blood flow within the aortic wall. The area of the vessel involved determines its classification. The classification, in turn, helps to predict outcomes, which allows for appropriate treatment planning. The goal of this article is to outline the operative indications and timing for Stanford type A and type B dissections, based on prior reported data and our own clinical experience with 176 patients treated surgically at the Yale Center for Thoracic Aortic Disease. With this data we will revisit the importance of looking at each patient individually to devise an appropriate operative plan, with the knowledge that treatment for type A dissections is operative and treatment for type B dissections is medical unless patients present with actual or impending rupture, malperfusion, or failure of medical management.
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Several authors have been credited with providing the earliest description of aortic dissection, including Fernelius, Vesalius, Sennertus, Nicholls, Laennec, Maunoir, and Morgagni.[1] Nicholls' report of the autopsy of King George II in 1761 may be the most famous early description,[2] but his and other early reports viewed it as an early stage of saccular aneurysm rather than a separate entity. Although Laennec is often credited with having first used the term "dissecting aneurysm" (*aneurysme dissequant*) in 1826, it may have been familiar to Maunoir as early as 1802.[1,3,4]

Unfortunately, despite these early postmortem descriptions, it was not until 1856 that Swaine and Latham made the first antemortem diagnosis, and by 1934 Shennan could only report on the antemortem diagnosis of dissection in 6 of 300 patients.[1,5] The first attempt at surgical repair was in 1935 by Gurin and associates.[6] This was a fenestration of a descending dissection into the iliac artery, and while perfusion to the limb was restored, the patient died of postoperative renal failure.[6]

DeBakey and colleagues published the first series of attempted repairs in 1955 with survival in four of six patients using fenestrations for ascending dissections and excision with graft replacement for descending dissections.[7] The use of extracorporeal circulation during clamping in descending aortic surgery was introduced by Cooley and associates in 1957.[8] Several subsequent advances in diagnostic, anesthetic, and surgical techniques would set the stage for the excellent results with aortic repair in the modern era.

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Anatomic and Clinical Considerations

Definition

Aortic dissection occurs when a tear in the aortic intima and media allows blood to course freely through a false lumen in the outer third of the media. The result is a dissection flap which separates the aorta into true and false lumina. The misnomer *dissecting aortic aneurysm* has frequently been applied to this condition, but dilation does not generally occur in the acute setting. Rather, should the patient survive, gradual dilation of the aorta may result.[9] Therefore we prefer the term *aortic dissection* and reserve the term *aortic aneurysm* for those situations in which dilation of the aorta occurs. Clinically, dissections identified within 2 weeks of the onset of symptoms are termed *acute*; subsequently they are classified as *chronic*.

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Classification Systems

The first widely used classification system for aortic dissection was published by DeBakey in 1965.[10] This system divided dissections into three types: type I included dissections arising in the ascending aorta and extending into the descending aorta; type II are limited to the ascending aorta, whereas type III are limited to the descending aorta (Fig. 1A). Subsequently, the simpler Stanford classification was proposed to guide therapy.[11] Under this classification, type A dissections involve the ascending aorta, whereas those that involve only the descending aorta are classified as type B (Fig. 1B).

Etiology, Pathology, and Pathogenesis

The acute pathogenic event that defines the onset of a dissection is a tear through the intima of the aorta with separation of the layers of the media, allowing blood within the aortic wall (Figs. 2B and 3). The tear progresses transversely and usually involves less than half the aortic circumference. Whether blood initially enters the wall through the tear or blood in the wall leads to a tear remains controversial. Blood in the wall, however, does cause an internal longitudinal discontinuity of the vessel layers.[12] Propagation of the injury can lead to catastrophic events including aortic rupture, tamponade, aortic regurgitation, branch vessel occlusion, and aneurysm formation.

The fact that type A and type B dissections have different predisposing factors suggests more than one process is responsible for the formation of a dissection. However the presence of hypertension in greater than 67% of patients with both type A and type B dissections stresses the role of blood pressure in initiating and propagating the process.[13,14]

Another frequent comorbidity in patients with aortic dissections is connective tissue disorders. Ten percent of patients seen at Yale over the past 20 years with type A dissections have associated connective tissue disorders including Marfan or Elhers–Danlos syndromes compared with 6.8% with type B dissections. Typically in this population there is degeneration of the elastic tissue histologically, which is thought to contribute to weakening of the aortic wall. An additional 12.7% of patients with type A dissections have a family history of thoracic aortic disease compared with 18.1% of patients with type B dissections. Other factors that predispose patients to type A dissections include bicuspid aortic valves, aortic coarctation, increased aortic diameter, pregnancy, trauma, and surgical manipulation.[15-17]

Clinical Presentation

Unlike many other fatal diseases treated in medicine, the timing of the diagnosis of an aortic dissection is essential and can be the difference between life and death. Therefore, accurate and rapid recognition of the signs and symptoms of a dissection are vital and clinical suspicion must remain high.

To define the presentations of patients with type A and type B dissections, the University of Michigan on behalf of the International Registry of Acute Aortic Dissections (IRAD) has evaluated 944 patients from multiple institutions. The majority of patients are white males and have a known history of hypertension.[1,13,14] Chest or back pain was the most consistently reported symptom and was present in over 84% of patients.[1,13,14] Ninety-one percent of type A patients described the pain as abrupt in onset. Other descriptions included agonizing, ripping, or tearing pain. Pain often started in the chest and migrated to the back or epigastrium. Syncopal episodes reported or witnessed were often secondary to pain or to cardiac or neurological sequela.

Physical examination frequently demonstrates signs of distress or shock. Patients are often diaphoretic and pale. Blood pressure is normal or high 80% of the time. Hypotension found in 27% of type A patients and 3% of type B patients is often correlated with rupture into the chest, pericardium, or secondary to coronary artery compromise.[13,14] Other less common findings include pulse deficits, newly diagnosed diastolic murmurs, pericardial friction rubs, distention of neck veins, and neurological deficits, including strokes and altered level of consciousness.

Pathologic Variants of Aortic Dissection

Two other pathologic entities similar to, but distinct from, aortic dissection merit discussion as well: penetrating atherosclerotic ulcers (PAU) and intramural hematomas (IMH). In PAU, atheromatous plaques ulcerate and penetrate through the internal elastic lamina and thence into the media (Fig. 2C).[18-20] While this may result in a region of intramedial dissection, its extent is limited by areas of severe calcification.[21-23] IMH was historically referred to as "dissection without intimal tear" and is thought to result from rupture of the vaso vasorum, resulting in an intramural hematoma discontiguous with the lumen of the aorta (Fig. 2D).[21] Patients with these two entities are distinctly older than those with classic aortic dissection.[21] While these are both more localized processes than classic dissection which may propagate for the entire extent of the aorta, their behavior is highly malignant with rupture rates between 30 and 40% in our series.[21] A full discussion of these entities and their appropriate operative treatment is beyond the scope of this article, but they merit vigilant attention with serial imaging and early operative intervention (especially in the ascending aorta) to prevent complications and death.

The Natural History of Aortic Dissection

Type A Dissections

Acute dissection of the ascending aorta may lead to several potentially fatal complications including aortic rupture, aortic valve and coronary artery compromise, and occlusion of aortic branch vessels. Accordingly, the mortality of acute ascending dissection (Stanford type A) approaches 1 to 2% per hour.[1,5] Early series suggested that mortality within 24 hours of symptom onset ranged from 21 to 65% with 2-week mortality approaching 75%.[1,5] Consolidating 963 patients from six series, Anagnostopoulos reported 48-hour mortality of 50%, and 1-week survival of only 30%.[24] More recent expe-

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