DISSECTING ANEURYSMS OF THE AORTA
or
AORTIC DISSECTION

CLASSIFICATION

DeBakey classified aortic dissections into types I, II, and III:

- **Type I dissection** the tear site originates in the ascending aorta, usually just above the left main coronary artery, and the dissection continues distally into the descending or abdominal aorta.
- **Type II dissection** the tear site is in a similar location in the ascending aorta, but the dissection stops distally at the innominate artery.
- **Type III dissection** the tear site originates in the upper descending thoracic aorta, just distal to the subclavian artery; in type IIIA the dissection is localized in the thoracic aorta, and in type IIIB the dissection proceeds into the abdominal aorta.

The Stanford classification proposed by Miller is based on the clinical course and the surgical significance of the dissection.

- **Stanford type A** dissection includes any dissection involving the ascending aorta (DeBakey types I and II)
- **Stanford type B** dissection involves only the descending aorta (DeBakey type III).

Borst and associates advocated a more simplified, descriptive classification. In this system

- **Proximal aortic segment** (ascending and transverse arch)
- **Distal aortic segment** (descending thoracic and thoracoabdominal)

Aortic dissection also is categorized based on the time elapsed since the initial event.

- **Acute** within the first 14 days following the initial tear.
- **Subacute** period from day 15 through 60 after the initial event.
- **Chronic** phase after day 60 (some consider chronic >14 days without subacute phase).
Etiology
Aortic dissection begins as a tear in the intima, with entry of blood and separation of the media for a variable distance, resulting in blood flow down a “false lumen.” A localized aneurysm may develop immediately, or months or years later where the aortic wall has become weakened and enlarged from the original dissection. The predisposing factors to aortic dissection are:

- Male sex: The disease is 3 to 4 times more common in males than in females.
- Age: Predominantly in older patients.
- Marfan’s syndrome
- Congenital heart disease, such as coarctation of the aorta or bicuspid aortic valve disease.
- Aortic injury during catheterization or surgery is a common cause of iatrogenic dissection.
- Cocaine and amphetamine abuse.
- Combination of hypertension and degenerative connective tissue disease. A history of hypertension is present in 80% to 90% of patients.
- Cystic medial necrosis, which may be idiopathic (Erdheim’s cystic medial necrosis) or secondary to a known connective tissue disease, such as Marfan syndrome or Ehlers-Danlos syndrome.
- An atherosclerotic plaque or traumatic injury serves as the initiating tear site for aortic dissection.
- Epinephrine-induced dissection, and endocrine factors may cause medial necrosis. The epinephrine effect that produces a hyperkinetic heart may be important even when hypertension is not present.
- Pregnancy may incite the hypertension and hyperkinetic heart, and these responses may represent dissections in these patients.
- Trauma.

Pathology
The major initiating pathologic event is a tear in the intima and media, usually involving half the circumference of the aorta. The intimal tear permits blood to enter the media and dissect distally. The aortic wall progressively separates (“dissects”) with an inner lumen composed of intima and an outer false lumen composed of the media and adventitia. Once the dissection begins, it usually extends rapidly through the thoracic and abdominal aorta into the peripheral arteries.

A “reentry” tear can be identified in most patients, located in the aorta in about one-half of the patients, and in a peripheral artery in the others. Multiple entry and re-entry points may be seen throughout the aorta.

As the dissection progresses, the pathology involves:

**Proximally**

- The coronary arteries may be involved (most often the RCA).
- Often one or more aortic valve commissures are detached, creating aortic insufficiency.

**Distally**

- Innominate or carotid artery involvement may produce neurologic injury.
- Obstruction of a subclavian artery may produce arm ischemia and a differential pressure between the two arms.
- Occlusion of intercostal arteries may cause spinal cord injury with paraparesis or paraplegia.
- Dissection of renal arteries may produce renal insufficiency, hematuria, oliguria, or anuria.
- Acute obstruction of the iliac or femoral arteries may cause leg ischemia, manifested with pain, sensory loss, or even gangrene.
The dissection may result in a fatal complication at any time.

- If the dissection involves a coronary artery, sudden death is likely as a result of myocardial ischemia.
- Rupture into the pericardial cavity with cardiac tamponade is the most common fatal complication.
- Blood may enter into the mediastinum, which may lead to sudden death.
- Rupture into the left pleural cavity or the retroperitoneum occurs less commonly.

Clinical Manifestations

- **Pain** is the most frequent symptom of patients presenting with acute aortic dissection. It is best described as catastrophic chest pain, so severe that the individual almost immediately seeks medical attention. After the initial episode, the pain may be confined to the chest or may be substernal, in the back, along the route of the aorta, in the abdomen, or in a combination of these sites. Another characteristic is the tendency for the pain to migrate into different areas as the dissection extends distally.
- A neurologic deficit was present in 30%, and 25% had nausea and vomiting. Dyspnea is not uncommon, and pulmonary edema may be present. Many patients may be normotensive or hypotensive in the aftermath of the acute episode, or this finding may be the result of pericardial tamponade.
- Other presenting symptoms include congestive heart failure, tamponade, syncope, stroke, peripheral neurologic injury, leg or arm ischemia, paraplegia, gastrointestinal hemorrhage, hematuria or anuria, hoarseness, dysphagia, superior vena cava syndrome, and aortic insufficiency.

Physical Examination

*Frequently, the seriously ill patients present with*

- **shock** despite previously known hypertension.
- **differential blood pressure and pulse** in the upper extremities or diminished or differential pulses in the legs. The patient who presents with both catastrophic chest pain and diminished pulses must be immediately considered to have had an aortic dissection.
- **Neurologic findings** are clearly related to the degree of dissection and to involvement of the arch vessels involved in the cerebral blood supply.

The differential diagnosis in this group of patients includes:

- myocardial infarction
- rupture of the sinus of Valsalva
- cerebrovascular accident
- acute surgical abdomen
- pulmonary embolism
- arterial thrombosis or embolism of the aortic bifurcation
- occlusion of the peripheral arteries.

Diagnostic Studies

- **Chest radiograph** a widened mediastinum or a left pleural effusion from extravasation of blood frequently is seen. In some patients the chest radiograph is completely normal (10-16%). Calcium in the wall of the aorta, with obvious widening beyond the calcium, is also suggestive.
- **Electrocardiogram (ECG)** is of value in distinguishing a dissection from a myocardial infarction, but there are no characteristic features of aortic dissection. Occasionally the ECG is misinterpreted because of the presence of ST-segment elevation secondary to hypertension and ventricular strain. The most common abnormalities on the ECG are sinus tachycardia or left ventricular hypertrophy from the antecedent hypertension.
• **Transesophageal echocardiography (TEE)** is the initial diagnostic procedure of choice for most patients with suspected aortic dissection.
• **CT scan with contrast, MRI, or enhanced MR angiography** are useful.
• **Aortography** is highly accurate, but it is seldom necessary, it is usually indicated in cases with evidence of coexisting CAD. It may be contraindicated in cases of hemodynamic instability, aortic rupture, and pericardial effusion.

**Medical Treatment**

The goal was conversion of all acute aortic dissections to subacute or chronic status. Then, after careful evaluation, treatment by elective operation was reserved for any complications that arose.

Patients are monitored closely in the ICU with indwelling radial arterial catheters inserted in the arm with the best pulse and appropriate central venous lines are inserted for monitoring and drug administration.

**Nitroprusside**, **beta blockers**, **ACE inhibitors**, **calcium channel blockers** and **methylidopa** may be administered. These agents are used to achieve a heart rate between 60 and 80 beats/min., a systolic blood pressure between 100 and 110 mmHg, and a mean arterial blood pressure between 60 and 75 mmHg.

**Beta antagonists** are administered to all patients with acute aortic dissection, unless there are strong contraindications such as severe heart failure, bradyarrhythmia, high grade AV block, or bronchospastic disease.

In patients who cannot receive beta antagonists, **calcium channel blockers** such as diltiazem or nicardipine are an effective alternative.

**Nitroprusside**, a direct vasodilator, can be used once beta blockade is adequate.

**Nitroglycerine** is another vasodilator that can be used.

**Natural History and Operative Indications**

**Ascending Dissections**

Nearly all patients with ascending aortic dissection should be managed surgically immediately after the diagnosis is established.

However, specific patient groups may benefit from nonoperative management or delayed operation. Situation that warrant delayed repair include:

1. Patients presenting with acute stroke or mesenteric ischemia
2. Elderly patients with substantial comorbidity
3. Stable patients who may benefit from transfer to specialized centre
4. Patients who have undergone a cardiac operation in the remote past. As a caveat to the late situation, it must be emphasized that dissections occurring in the initial 3 weeks after cardiac surgery are at high risk for rupture and tamponade; these patients should undergo early operation.

The early causes of death in these patients are **pericardial tamponade**, rupture into the **mediastinum**, or **acute aortic insufficiency with cardiac and renal failure**. These are basically mechanical problems, and therefore surgical correction is mandatory.

The goal of the operation, then, is to

- *remove the intimal tear site*
- *correct the aortic insufficiency*, either with aortic valve **replacement** or more often with **resuspension** of the aortic valve
- *graft the ascending aorta directing the blood into the true lumen with obliteration of the false lumen*, thereby providing protection from rupture into the mediastinum or pericardium,
- *correct associated coronary ischemia.*
Descending Aortic Dissection

Dissections confined to the descending aorta can usually be carefully monitored. The life-threatening nature of descending aortic dissection is determined by:

- first, rupture into the pleural space
- second, involvement of the visceral vessels.

After the diagnosis has been confirmed and hypotensive therapy instituted, monitoring under close observation is imperative.

Indications for immediate operative intervention include:

1. failure to control hypertension,
2. continued pain,
3. expansion of the aneurysm
4. signs or symptoms of rupture such as pleural effusion
5. development of a neurologic deficit
6. evidence of compromise of major visceral vessels or arteries to the lower extremities
7. retrograde dissection with aortic valve involvement.

Approximately 10 percent of patients with acute type B dissection treated medically develop a serious complication within 2 weeks, and prompt surgery is indicated for these patients.

By use of a left lateral thoracotomy and either partial cardiopulmonary bypass or a heparin-bonded shunt, a graft is inserted into the thoracic aorta, obliterating the false lumen and redirecting blood flow into the true aortic lumen.

MORTALITY

The mortality of untreated aortic dissections is catastrophic, making immediate therapy of some type mandatory. Initially, antihypertensive pharmacologic therapy is generally employed.

The surgical mortality in acute ascending dissections should be between 10% and 20%, with long-term survival approaching 60%.

In patients with descending aortic dissections, the mortality for those in the surgical series and those managed with antihypertensive therapy is essentially the same. It is apparent that some patients with descending aortic dissections who are successfully managed with antihypertensive therapy will later be managed operatively for the reasons previously described, the most common indication being an expanding thoracic aneurysm. The operative mortality is less than 10%, and the long-term outlook is excellent.