Developments in AAA / TAA Repair

Innovation in Care
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Aneurysm Definition

• The abnormal enlargement or bulging of an artery caused by an injury or weakness in the blood vessel wall

• A localized dilatation of the aorta with an increase in diameter of >1.5 times its normal diameter
AAA Facts

- Over the last three decades, incidence has tripled\(^1\)
- 1.5 million people in the US have AAAs\(^2\)
- Men present 4:1 over women\(^3\)
- Risk increases for men by 40% every 5 years after age 65\(^4\)
- 15,000 deaths annually due to ruptured AAAs in the US – 13\(^{th}\) leading cause of death\(^2\)
AAA Facts

• Most AAAs are infrarenal – patients often have other aneurysms, including iliac (41%) and femoropopliteal (15%) lesions

• Hypertension (30 – 40%)⁴

• Smoking (6:1)⁴,⁵

• > 50,000 procedures per year for AAA repair⁶
• Incidence of aortic aneurysms

Each year, physicians diagnose approximately 200,000 people in the United States with AAA.

(www.vascularweb.org/contribution_pages/patient_information)
Clinical Challenge

- The majority of patients are asymptomatic

- Approximately 40% of patients with ruptured AAAs die prior to presentation to the emergency department

- Only 10% to 25% of individuals with ruptured AAAs survive until hospital discharge
Major Risk Factors

- Current or former smoker
  - A history of smoking has been associated with a 3- to 5-fold increase in AAA prevalence across all age groups
- Family history of AAA
- Age – over 60
- Gender
  - Abdominal aneurysms are four times more common in men than women
    - 5% of US males over 60 are estimated to have a AAA
Lesser Risk Factors

- Hypertension
- Atherosclerosis
- CAD
Prevalence of AAAs

• AAAs encountered more frequently in contemporary practice due to:
  – Better diagnosis
  – True increase in prevalence

• Prevalence as well as rupture risk increase sharply with age\(^7\)

• Population over the age of 65 will grow from 34 million to 50 million by 2020\(^8\)
• AAA is often called a “silent killer” because there are no obvious symptoms of the disease (www.SIRweb.org)
  – Three out of four aneurysms show no symptoms at the time they are diagnosed (www.SIRweb.org)

• Possible symptoms may include:
  – Abdominal pain
  – Pain in the lower back that may extend to the buttocks, groin or legs
  – Pulsating sensation in the abdomen

• Symptoms indicating a rupture may include:
  – Sudden onset of severe back or abdominal pain
  – Nausea
  – Dizziness, fainting and/or sudden weakness
Diagnostic Methods

- **Physical exam, palpation and auscultation**

- **Abdominal Ultrasound**
  - Commonly used as a primary screening tool
  - Provides details of the vessel wall and plaque

- **Computed Tomography Arteriography (CTA)**
  - Most accurate test to determine size and location
  - Readily available
  - Eliminates the need for invasive angiography but requires IV contrast
Diagnostic Methods

• Arteriogram
  - Less useful modality
  - Unable to accurately delineate AAA due to thrombus lining the flow lumen
  - Provides images of associated arterial occlusive disease
When to Treat

Elective repair of AAA is recommended when the maximal aneurysm diameter is 5.5 cm or more. Aneurysms that have a saccular morphology may be considered for repair even if < 5 cm in diameter. Aneurysms that have a fusiform morphology may be considered for repair if they are painful, have caused distal embolization, or are rapidly enlarging (>0.5 cm/year).
AAA Treatment Options

• Medical Management / Monitor
  – Wait, watch and control hypertension
  – Typically reserved for aneurysms < 5 cm that are not rapidly expanding or causing symptoms
  – Most commonly monitored with regular CT scans or ultrasound examinations
### AAA Treatment Options

<table>
<thead>
<tr>
<th>Risk Diameter (cm)</th>
<th>Rupture (% per year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 4</td>
<td>0%</td>
</tr>
<tr>
<td>4 - 5</td>
<td>0.5 - 5%</td>
</tr>
<tr>
<td>5 - 6</td>
<td>3 - 15%</td>
</tr>
<tr>
<td>6 - 7</td>
<td>10 - 20%</td>
</tr>
<tr>
<td>7 - 8</td>
<td>20 - 40%</td>
</tr>
<tr>
<td>&gt; 8</td>
<td>30 - 50%</td>
</tr>
</tbody>
</table>

- **AAA Expansion Rate**\(^\text{14}\)
  - Although a number of studies have found that small AAAs expand at approximately 0.5 cm in diameter per year, individual patients show considerable variation in aneurysm expansion rates.
AAA Treatment Options

• Surgical Treatment
  – Elective repair has a perioperative mortality rate of about 3 - 5%³
  – 22 - 30% morbidity
  – High risk surgical group includes: cardiac, renal, pulmonary disease, and morbid obesity⁹
  – Patients > 75 years of age have a higher perioperative mortality rate⁹
  – Average 7 to 10 days hospitalization¹⁰
  – Emergency repair: mortality 40 - 50%¹¹
  – Coronary events are the leading cause of death following repair of AAAs¹²
Open Surgical Repair

Surgical Treatment - Two Methods:

- Transabdominal
  - Most common approach
- Retroperitoneal
  - Reduced gastrointestinal impairment following operation
Results of Standard Open Repair

- Effective and durable…but
  - 4 - 5% mortality in population-based studies\textsuperscript{15,16,17}
  - Recovery 2 - 4 months\textsuperscript{18}
  - High risk patients often denied repair\textsuperscript{19}
  - 5-year survival rate of 46\%\textsuperscript{9}
Open Surgical Repair

- Complications
  - Infection
  - Limb thrombosis
  - Aortoenteric fistula (AEF)
  - Paraplegia
  - Pseudoaneurysm formation at anastomoses
Infection: Catastrophic event\(^1\)
- Mortality (25 - 88%)
- Incidence (1 - 2%)
- Mean time to onset (6 months - 4 years)
- 8 - 10% of all aneurysms yield positive bacterial cultures
Endovascular Repair

- **Principles of Endovascular Aortic Repair (EVAR)**
  - Anchoring and secure fixation of endoluminal device above and below AAA in normal arterial segments
  - Hemostatic seals exclude AAA from circulation
  - Exclusion and depressurization prevent AAA rupture
Endovascular Repair

- **Goal of EVAR**
  - Accurate placement and secure attachment of a sutureless prosthesis across an aneurysm, to effect repair quite similar to standard open surgical grafting but using only:
    - Small groin incision
    - Minimal anesthesia
• Potential Benefits of EVAR
  – Shorter hospital stay; 1 - 3 days vs. 5 - 13 days\textsuperscript{2,16}
  – Safer option for high risk patients: most have significant concomitant disease (e.g., CAD, COPD)\textsuperscript{2}
  – Anesthesia:\textsuperscript{15,16}
    • General = shorter time and less blood loss
    • Regional = epidural
    • Local = percutaneous (closure devices)
  – Overall lower morbidity
  – Lower mortality rate
  – Patient comfort\textsuperscript{14}
• **GORE EXCLUDER® AAA Endoprosthesis**

  - US Pivotal Trial results at 60 months (n = 235 patients enrolled)* demonstrate the GORE EXCLUDER® AAA Endoprosthesis is:

  - **Safe**
    - Significantly lower rate of major adverse events
    - Comparable survival

  - **Effective**
    - 0 ruptures
    - 1 post-procedure migration
    - 1 stent fracture
    - 0 graft tears
    - 0 post-operative limb disconnections
    - 0 deployment failures
    - 10% total endoleak rate**
      (0% Type I, 8% Type II, 0% Type III and IV, and 2% indeterminate)
    - 100% cumulative patency
    - Low post-procedure conversion rate of 4.3%

  * Site Reported Data Through October 3, 2005
  ** At 5 years

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Summary

- The annual rupture risk of 5 - 6 cm AAA is approximately 3 - 15%, and the average growth (expansion) rate is approximately 0.5 cm per year.
- Although open surgical repair is effective and durable, morbidity remains substantial and other limitations exist.
- In appropriate patients, EVAR offers a safe and effective alternative with many potential advantages.
TAA: Thoracic Aortic Aneurysm

• Location of TAAs
  ▪ Ascending thoracic aorta—40%
  ▪ Descending thoracic aorta—35%
  ▪ Upper abdomen ("thoracoabdominal aneurysms")—15%
  ▪ Aortic arch—10%

• Aortic dissection
  • Tear in the intimal layer of the aortic wall

DeBakey
Type I  Originates in the ascending aorta, propagates at least to the aortic arch and often beyond it distally
Type II  Originates in and is confined to the ascending aorta
Type III  Originates in the descending aorta and extends distally down the aorta or rarely retrograde into the aortic arch and ascending aorta

Stanford
Type A  All dissections involving the ascending aorta, regardless of the site of origin
Type B  All dissections not involving the ascending aorta

**TAA: Thoracic Aortic Aneurysm**

- Prevalence of up to 4.2% of the general population without hypertension\(^1\)
- Estimated incidence of 5.6-10.4 cases per 100,000 patient-years\(^1\)
- Average age at the time of diagnosis is 69 years\(^2\)
- Direct correlation between age and incidence is not sex specific\(^2\)

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TAA: Annual Rates of Rupture, Dissection, or Death Related to Aortic Size

Initial Aortic size (cm)

Rupture

<table>
<thead>
<tr>
<th>Initial Aortic size (cm)</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.5 - 3.9</td>
<td>0.3%</td>
</tr>
<tr>
<td>4.0 - 4.9</td>
<td></td>
</tr>
<tr>
<td>5.0 - 5.0</td>
<td></td>
</tr>
<tr>
<td>&gt; 6.0</td>
<td>3.6%</td>
</tr>
</tbody>
</table>

Dissection

<table>
<thead>
<tr>
<th>Initial Aortic size (cm)</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.5 - 3.9</td>
<td></td>
</tr>
<tr>
<td>4.0 - 4.9</td>
<td>1.5%</td>
</tr>
<tr>
<td>5.0 - 5.0</td>
<td></td>
</tr>
<tr>
<td>&gt; 6.0</td>
<td>3.7%</td>
</tr>
</tbody>
</table>

Death

<table>
<thead>
<tr>
<th>Initial Aortic size (cm)</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.5 - 3.9</td>
<td>4.6%</td>
</tr>
<tr>
<td>4.0 - 4.9</td>
<td></td>
</tr>
<tr>
<td>5.0 - 5.0</td>
<td></td>
</tr>
<tr>
<td>&gt; 6.0</td>
<td>10.8%</td>
</tr>
</tbody>
</table>

Rupture, Dissection, or Death

<table>
<thead>
<tr>
<th>Initial Aortic size (cm)</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5 - 3.9</td>
<td></td>
</tr>
<tr>
<td>4.0 - 4.9</td>
<td>5.3%</td>
</tr>
<tr>
<td>5.0 - 5.0</td>
<td></td>
</tr>
<tr>
<td>&gt; 6.0</td>
<td>14.1%</td>
</tr>
</tbody>
</table>

TAA: Pathogenesis

- Mechanisms of TAA formation overlap those of aortic dissection
  - Cystic medial necrosis
  - Focal degeneration of the elastic and muscle tissue
  - Aortic wall weakens and dilates as a result of high pressure of intraluminal blood flow
- Acquired and hereditary conditions can exacerbate medial necrosis
  - Bicuspid aortic valve is a common congenital heart abnormality, occurring in ~2% of the population

# TAA: Etiology

| Degenerative aneurysms | • Most common  
<table>
<thead>
<tr>
<th></th>
<th>• Associated with hypertension, age, smoking</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atherosclerotic</td>
<td>• More commonly involves the descending aorta and arch</td>
</tr>
</tbody>
</table>
| Genetically triggered aneurysm syndromes | • Marfan syndrome  
|                        | • Loeys-Dietz Syndrome  
|                        | • Bicuspid aortic valve  
|                        | • Turners syndrome  
|                        | • Familial non-syndromic TAA syndrome |
| Aortitis               | • Infectious: syphilis; salmonella; staphylococcal; mycobacterium  
|                        | • Noninfections/inflammatory:  
|                        | • More common: Giant cell and Takaysu arteritis  
|                        | • Less common: Behcets, Cogans syndrome, relapsing polychondritis  
|                        | • Rare: Rheumatoid arthritis, spondyloarthropathies |
| Trauma                 | • Typical location is at the aortic isthmus  
|                        | • Complications include rupture, pseudoaneurysm, chronic dissection with secondary aneurysm formation |
| Chronic aortic dissection | • Aneurysm due to growth and pressure differential of false lumen |
TAA: Other Risk Factors

- Patients with TAAs often have a history of hypertension, smoking, and COPD

- TAAs are more prevalent in males
  - Proportion of women with TAAs increases with age, and nearly equals that of males in the elderly

- Risk factors for rupture:
  - Increasing risk of rupture after the TAA is >5 cm in diameter
  - Risk of rupture nearly doubles with every 1-cm increment of aneurysmal diameter
  - Relative risk of rupture is increased by a factor of 2.6 for every decade of life

TAA: Symptoms and Complications

- TAAs often present with no symptoms and are typically detected on routine physical examination.
- Symptoms usually appear when complications develop:
  - Aortic regurgitation
  - Thromboembolism—may cause stroke, abdominal pain (due to mesenteric embolism), or extremity pain
  - Rupture—patients who do not immediately die of a ruptured TAA present with severe chest or back pain and hypotension or shock
  - Dissection—manifests with tearing pain, often radiating to the back

<table>
<thead>
<tr>
<th>Modality</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MDCT angiography</strong></td>
<td>• Rapid image acquisition (20-30 seconds)</td>
<td>• Need for iodinated contrast</td>
</tr>
<tr>
<td></td>
<td>• 3D reconstruction allows multiple views/orientations</td>
<td>• Radiation exposure (10-20 mSv) — of concern in young patients requiring serial imaging</td>
</tr>
<tr>
<td></td>
<td>• Ability for post image processing</td>
<td>• Image artifacts—esp. in aortic root</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Aortic size can be overestimated</td>
</tr>
<tr>
<td><strong>MRI/MR angiography</strong></td>
<td>• No radiation or iodinated contrast</td>
<td>• Caution with use of gadolinium in renal failure</td>
</tr>
<tr>
<td></td>
<td>• 3-D, multi planar and high resolution</td>
<td>• Need for breath hold</td>
</tr>
<tr>
<td></td>
<td>• Dynamic and functional information available</td>
<td>• Time consuming (10-30 minutes at minimum) depending on center</td>
</tr>
<tr>
<td></td>
<td>• May be appropriate for serial imaging over many years</td>
<td>• Not for use in unstable patients</td>
</tr>
<tr>
<td><strong>Transesophageal echocardiography</strong></td>
<td>• No radiation or iodinated contrast</td>
<td>• Cannot visualize entire aorta</td>
</tr>
<tr>
<td></td>
<td>• Can be performed at bedside</td>
<td>• May be limited by technical difficulties</td>
</tr>
<tr>
<td></td>
<td>• Immediate information availability</td>
<td>• Semi-invasive</td>
</tr>
<tr>
<td></td>
<td>• Excellent evaluation of valve function, pericardial effusion and left ventricular function—can visualize aorta from root to gastroesophageal junction</td>
<td>• Requires conscious sedation and patent/secure airway</td>
</tr>
</tbody>
</table>

Booher AM, Eagle KA. *Am Heart J.* 2011;162:38-46.e1
Aneurysms of Degenerative Origin

Figure 1.32E-H

- (E) The proximal anastomosis is made first using continuous #000 prolene sutures. Anastomotic bleeding is controlled when necessary by supplemental interrupted sutures.

- (F) The distal anastomosis is made in a similar manner. Prior to completion of the anastomosis, the head is lowered and both proximal and distal aortic segments are flushed by temporary loosening of the clamps; the graft is filled with blood to evacuate air, the distal clamp is removed, and finally, the proximal clamp is removed gradually as blood volume is replaced with the intravenous administration of blood and plasma to avoid hypotension.

- (G-H) Reconstruction is completed by suturing the aneurysmal wall tightly around the graft according to the method of incision.
There is an increasing risk of rupture after the TAA is >5cm in diameter. Relative risk of rupture is increased by a factor of 2.6 for every decade of life.

Although open surgical repair is effective and durable, morbidity remains substantial and other limitations exist.

In appropriate patients, TEVAR offers a safe and effective alternative with many potential advantages.


References


Thank You