Aortic Aneurysms

Objectives

- Abdominal aortic aneurysms
- Demographics
- Pathophysiology
- Symptoms
- Diagnosis
- Treatment

Aneurysma

- “A widening”
- Ectasia - < 50% diameter increase
- Arteriomegaly - diffuse ectasia
- Aneurysmosis
- Aneurysm - > 50% diameter increase

<table>
<thead>
<tr>
<th>AORTA</th>
<th>DIAMETER</th>
<th>GENDER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Root</td>
<td>3.50–3.72</td>
<td>Female</td>
</tr>
<tr>
<td></td>
<td>3.63–3.91</td>
<td>Male</td>
</tr>
<tr>
<td>Ascending</td>
<td>2.86</td>
<td>Female/male</td>
</tr>
<tr>
<td>Descending</td>
<td>2.45–2.64</td>
<td>Female</td>
</tr>
<tr>
<td></td>
<td>2.39–2.98</td>
<td>Male</td>
</tr>
<tr>
<td>At diaphragm</td>
<td>2.40–2.44</td>
<td>Female</td>
</tr>
<tr>
<td></td>
<td>2.43–2.69</td>
<td>Male</td>
</tr>
<tr>
<td>Infrarenal</td>
<td>1.5–1.7</td>
<td>Female</td>
</tr>
<tr>
<td></td>
<td>1.7–1.9</td>
<td>Male</td>
</tr>
</tbody>
</table>

Aneurysm

- Pathological dilatation of the aorta involving one or several segments
- A permanent localized dilatation having a diameter at least twice the normal diameter of that segment
## Demographics

- 200,000 patients diagnosed with non-ruptured AAA each year
- 1.5 to 2 million are estimated to have an undiagnosed AAA
- 50% of patients with untreated aneurysms > 5.5 cm will die of rupture within five years
- 15,000 deaths each year

## Inexorable Progression to Rupture

- Average rate of growth
  - 0.4 cm/ year
  - ~10% per year

## Growth Rate of AAA

<table>
<thead>
<tr>
<th>Initial size (cm)</th>
<th>Mean growth rate (cm/yr)</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.0 - 3.9</td>
<td>0.39</td>
<td>0.20-0.57</td>
</tr>
<tr>
<td>4.0 - 4.9</td>
<td>0.36</td>
<td>0.21-0.50</td>
</tr>
<tr>
<td>5.0 - 5.9</td>
<td>0.43</td>
<td>0.27-0.60</td>
</tr>
<tr>
<td>6.0 - 6.9</td>
<td>0.64</td>
<td>0.16-1.10</td>
</tr>
</tbody>
</table>


## Risk of Rupture

- Mortality = 35 - 75%
  - unchanged over past 4 decades
  - higher with COPD, multiple co-morbidities

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

*J Vasc Surg 2003; 37: 1106-17*
Rupture

- Approximately 40% of patients with ruptured AAAs die prior to presentation to the emergency department
- Only 10% to 25% of individuals with ruptured AAA survive until hospital discharge
- Prevent rupture!

Classification

- True Aneurysms
  - saccular
  - fusiform
- False Aneurysms
  - not all layers of the arterial wall (intima, media, adventitia) are present
  - one or more layers of the arterial wall have been disrupted

Etiology: Medial Degeneration

- Aging
- Atherosclerosis
- Infection
- Inflammation
- Trauma
- Congenital anomalies
- Smoking
- Genetic predisposition
Genetics and Pathophysiology

• Most of what we know about AAA is descriptive
• 20% of AAA patients will have a first degree relative with AAA
• OR of AAA in first degree relative of index patient 9.7
• Thought to be autosomal dominant based on family studies

Risk Factors

• Males
  – account for 80% of AAA
  – 5% of men over 60 have AAA
• Age > 55
• COPD / smoking
  – > 100 packs smoked confers 7x greater risk of AAA
• Caucasians
• High blood pressure
• Diabetes
• Hypercholesterolemia

Risk Factors

• Atherosclerosis
  – not a risk factor?
  – localizes to infrarenal aorta
  – plaque is present in AAA wall
  – shares RF with AAA (smoking, HTN, hypercholesterolemia)
• Squirrel monkeys fed an atherogenic diet
  – all developed ASD
  – only 1.6% developed AAA
• Theories
  – related to atherosclerotic injury response
  – related to ASD plaque regression
**Inflammation**

- Both AAA and ASD are characterized by:
  - inflammatory cells that elaborate
  - proteolytic enzymes
  - cytokines that upregulate proteolysis
  - infiltration of macrophages and lymphocytes into plaque, intima, and adventitia

**Autoimmunity**

- Aneurysm wall IgG is an autoantigen that has homology with elastin microfibrils, stimulating an anti-elastin immune response
- AAA is associated with an MHC III locus related to RA

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**Inflammation**

- ASD
  - primarily a T lymphocyte infiltrate
  - late adventitial inflammation
- AAA
  - T and B lymphocyte infiltrate
  - consistent adventitial involvement
  - immunoglobulins
  - complement
  - “inflammatory” aneurysm – an extreme on the continuum of AAA inflammation

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- 141 of 4404 (4.2%) had AAA
- 7.7% of pts w/COPD had AAA (OR 1.59)
- Association of aneurysms with COPD may be related to medication and coexisting disease

<table>
<thead>
<tr>
<th></th>
<th>Yearly Expansion Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>COPD</td>
<td>2.74 mm</td>
</tr>
<tr>
<td>No COPD</td>
<td>2.72 mm</td>
</tr>
<tr>
<td>Steroids</td>
<td>4.7 mm</td>
</tr>
<tr>
<td>No Steroids</td>
<td>2.6 mm</td>
</tr>
</tbody>
</table>

Symptoms

- Most nonruptured AAA patients are asymptomatic at diagnosis
- Vague abdominal pain with back pain is the most common complaint
  - constant or throbbing
  - rapid expansion may cause intense pain
- GI symptoms (uncommon)
  - Early satiety, nausea, weight loss may indicate intestinal compression

Diagnosis

- History
- Physical exam
  - pulsatile, tender abdominal mass
  - bruit
- Ultrasound
  - Good Screening Test
  - > 80% accurate
- CT
- Angiography – not good for diagnosis
Physical Exam

- n = 198
- 48% of AAA were diagnosed clinically
- Physical exam missed 38% of cases detected radiographically

<table>
<thead>
<tr>
<th>Aneurysm diameter</th>
<th>Sensitivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.0-3.9 cm</td>
<td>29%</td>
</tr>
<tr>
<td>4.0-4.9 cm</td>
<td>50%</td>
</tr>
<tr>
<td>≥ 5.0 cm</td>
<td>76%</td>
</tr>
</tbody>
</table>

Ultrasound

- Sensitivity 82% to 99%
- Approaches 100% in cases with a pulsatile mass
- In a small proportion of patients, visualization of the aorta is inadequate because of obesity, bowel gas, or periaortic disease

Screening

- 1 / 1000 in adults less than 60
- 7 / 1000 in adults in 60's
- 3 / 1000 in adults older than 70
- 5 – 10% of men over 60 have AAA, most are small
- Prevalence of AAA is 6x lower in women than men
Screening

Table V. Prevalence of small and medium AAAs among 73,451 US military veterans between 50 to 79 years

<table>
<thead>
<tr>
<th>Race</th>
<th>Gender</th>
<th>Smoking status</th>
<th>AAA ≥3 cm (%)</th>
<th>AAA ≥4 cm (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td>Male</td>
<td>Smoker</td>
<td>5.9</td>
<td>1.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nonsmoker</td>
<td>1.9</td>
<td>0.4</td>
</tr>
<tr>
<td>White</td>
<td>Female</td>
<td>Smoker</td>
<td>1.9</td>
<td>0.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nonsmoker</td>
<td>0.6</td>
<td>0.0</td>
</tr>
<tr>
<td>Black</td>
<td>Male</td>
<td>Smoker</td>
<td>3.2</td>
<td>0.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nonsmoker</td>
<td>1.4</td>
<td>0.1</td>
</tr>
</tbody>
</table>

- 70,495 men 65 – 74 years old
- Randomized to ultrasound screening or no screening
- In the screened group
  - > 3 cm were rescreened
  - < 3 cm were not rescreened
- Endpoints of AAA-related mortality and overall mortality
MASS

- 50% reduction in AAA related death at 7 years
- Cost effective
- Women?

**Treatment Options**

- Watch and wait
  - AAA < 5cm, asymptomatic
  - surgical risks > risk of rupture
  - lifestyle changes cannot reduce the size of the AAA
- Open surgical repair
- Endovascular repair
- SVS guidelines…
**Prognosis of Patients Turned Down for Conventional AAA Repair**

- 106 patients turned down for elective repair
  - aneurysms > 5.5 cm
  - patient refusal, unfit for surgery, advanced age, cardiac disease, cancer, respiratory disease, dementia, paraplegia
- By the end of the study, 76 patients had died (median survival: 9 months)
  - 37 died from AAA rupture
  - 17% 3 year survival

*J Vasc Surg 2001; 33: 752-7*
Open Abdominal Aortic Aneurysm Repair

- Major surgical procedure
  - Mortality 3% to 8%
  - Other complications
    - Pseudoaneurysms (3%)
    - Erectile Dysfunction (20-30%)
    - Graft thrombosis (2%)
    - Graft infection (1-2%)
- Recovery time 2-4 months

Mayo Study on Open Repair, Early and Late Graft-Related Complications

- 307 patients underwent AAA repair
- Anastomotic aneurysm 9 (3.0%)
- Graft thrombosis 6 (2.0%)
- Graft-enteric erosion/fistula 5 (1.6%)
- Graft infection 4 (1.3%)
- Anastomotic hemorrhage 4 (1.3%)
- Colon ischemia 2 (0.7%)
- Tissue loss 1 (0.3%)
- Atheroembolism 1 (0.3%)

9.4% of patients had a major graft-related complication

Complications

- 15% non-aneurysm-related
  - cardiac
  - pulmonary
  - renal
### Long-Term Recovery

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assisted living facility</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Skilled nursing facility</td>
<td>9</td>
<td>6</td>
</tr>
<tr>
<td>Home</td>
<td>106</td>
<td>69</td>
</tr>
<tr>
<td>Hospital</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

33% felt they never fully recovered


### Early OSR vs. Watchful Waiting

- Combined ADAM and UKSAT trials
- Early/immediate repair vs. surveillance/delayed OSR
- AAA < 5.5 cm
- N = 2226

<table>
<thead>
<tr>
<th>Endpoint</th>
<th>Relative Risk</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>All cause mortality</td>
<td>1.01</td>
<td>0.77-1.32</td>
</tr>
<tr>
<td>Aneurysm-related mortality</td>
<td>0.78</td>
<td>0.56-1.10</td>
</tr>
</tbody>
</table>


### Transition to Endovascular Aneurysm Repair

- Total number of AAA repairs was not different (25,246 vs 25,850)
- Percentage of EVARS performed was significantly higher in 2007 (14,001 [55%] vs 19,471 [75%], P=0.001)


Francisco C. Albizures Jr, MD, Britz H. Tomasson, MD, Robert E. Noll Jr, MD, Gonzalo Cisneros, MD, Jason R. Kim, MD, and W. Charles Stevenborgh III, MD, New Orleans, Louisiana

Fig. 3. The percentage of endovascular aneurysm repair in the study period significantly increased in the second period, among others 77% between 2003 and 2008 (P < 0.05).
Conclusions

- Now over 80% of AAA are repaired with EVAR
- Overall mortality dropped from 4.9% to 1.8% over the 13 year study period
- Rupture mortality has significantly improved
- Open AAA repair has become more complex
EVAR 1 Trial

EVAR 1 Trial

EVAR 1 Trial
EVAR 1 Conclusions

- Compared with open repair
  - EVAR offers no advantage with respect to all-cause mortality
  - Is more expensive
  - Leads to a greater number of complications and reinterventions.
  - 3% better aneurysm-related survival.


EVAR 2 (Patients deemed unfit for Open Repair)

- EVAR had a considerable 30-day operative mortality in patients already unfit for open repair.
- EVAR did not improve survival over no intervention
- EVAR associated with a need for continued surveillance and reinterventions

EVAR 2 Trial

- EVAR had a considerable 30-day operative mortality in patients already unfit for open repair.
- EVAR did not improve survival over no intervention
- EVAR associated with a need for continued surveillance and reinterventions
Future Directions