Diagnosis and Treatment of Abdominal Aortic Aneurysm

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Disclosure

Educational grants from:
W.L. Gore and Associates
Cook Medical
Definition

- Localized dilatations at least 1.5 times the diameter of the normal artery
Background

- Saccular or fusiform
- May cause Rupture or Atheroembolism
- The most common site of arterial aneurysm
- Most often occurs between the renal and inferior mesenteric arteries
- 5%~15% involve the renal or visceral arteries
- 15,000 deaths in the US from AAA rupture, 13th leading cause of death
Concomitant aneurysm
- Iliac 50%
Background

Concomitant aneurysm

- Iliac 50%
- Thoracic 12%
Background

Concomitant aneurysm
- Iliac 50%
- Thoracic 12%
- Peripheral 15%
Epidemiology

- Prevalence
  - ~9% of >65 yrs
  - 4:1 male:female

- Asymptomatic for many years

- If untreated, about one third will cause death from rupture
Risk Factors

- **Age**
  - Negligible <60 yrs
  - Increases dramatically with age

- **Smoking**
  - In a VA study, most strongly associated (OR 5.1)
  - Increases with #years and decreases with #years after the cessation
  - Promotes the rate of aneurysm growth
Familial Clustering

- First-degree relatives of a patient with an AAA have a 12 fold increased risk of aneurysm
- Brothers of a patient with an AAA have an 18 fold increased risk
- Women with AAA are much more likely to have affected relatives than men with AAA
Risk Factors

- Hypertension
  - relatively small effect
- PVOD
  - 5% of patients with CAD
  - 10% of those with arteriosclerosis obliterans
- Peripheral aneurysm
  - 41% incidence of AAA in pts with popliteal aneurysm
  - 66% incidence of AAA in pts with femoral aneurysm
Risk Factors for Rupture

- Aneurysm size
- Hypertension
- COPD
- Women

Mortality rate:
- >90% for all comers
- ~50% after repair
Pathophysiology

- Infection: salmonella, syphilis
- Cystic medial necrosis
- Collagen vascular disorders
  - Marfan’s syndrome
  - Ehlers-Danlos syndrome
- Elastin and collagen degradation by proteases
Pathophysiology

- AAA is characterized by breakdown of the structural component of the vascular wall associated with disruption and degradation of the media.
- Increased proteolytic activities including plasminogen activators and metalloproteinases in atherosclerotic wall may contribute to remodeling of extracellular matrix leading to physical weakening and expansion of AAA.
AAA and Inflammation

- Large amounts of the tissue-degrading proteolytic enzymes present in AAA are derived from neutrophilic granulocytes
- The neutrophils are mainly located within the ILT, which develops at the interface between the circulating blood and the aneurysmal wall.
- The ILT is a laminated neotissue comprising a red blood cell-rich luminal layer at the interface with the flowing blood, an intermediate zone, and a brown fibrinolyzed abluminal layer, which lines the aneurysmal wall
The presence of intraluminal thrombus (ILT) in AAA: 75%
Characterized by neutrophil trapping & platelet activation, fibrin formation, and binding of plasminogen
Reduces O2 diffusion to the AAA wall; cause local hypoxia and wall weakening
Causes oxidative and proteolytic injury of the arterial wall
AAA and ILT / Calcification

- Thinner AAA wall adjacent to ILT with more macrophages and other inflammatory cells than in walls without ILT
- Computational analyses showed that ILT reduces peak wall stress
- Calcification increases wall stress in AAA due to its stiffness and adverse stress distribution

Li JVasc Surg 2008;47:928
AAA and Inflammation

- The progressive dilation of the aortic wall in AAA is not only associated with extracellular matrix breakdown and smooth muscle cell disappearance, but it is also characterized by the infiltration and activation of inflammatory cells.
- A reduced number of neutrophils within the ILT, which can be obtained through platelet inhibition, limits AAA enlargement.
LTB₄ Involvement in AAA

Blood | Intraluminal thrombus | Wall

Intraluminal thrombus:
- Luminal
- Deeper
- Media
- Adventitia

Blood:
- Neutrophil
- Chemotaxis
- BLT
- 5-LO
- LTA₄
- LTB₄

Intraluminal thrombus:
- Elastase
- MPO
- MMP-9
- Wall injury

Endothelium:
- Media

Adventitia:
- Macrophage
- 5-LO
- LTA₄
- LTB₄
- BLT

T-lymphocyte

Proteolysis
Inflammatory and immune responses
AAA and Coagulopathy

- AAA is associated with increased plasma levels
  - Fibrinogen, D-dimer, TAT
  - Thrombin generation
  - Thrombin activity
  - Fibrin turnover/fibrinolysis
- Increased fibrin turnover within the thrombus
  - Positive correlation between the extent of hemostatic derangement and AAA size and ILT
AAA and Coagulopathy

- Linear association between D-dimer concentration and aneurysm diameter
- Higher fibrinogen and D-dimer concentration in patients with AAA
- D-dimer stimulates the release of proinflammatory cytokines and proteolytic enzymes
- D-dimers levels are higher in AAA pts as compared with PAD pts
- The coagulation cascade involves a complex series of enzymatic reactions that culminate in thrombin generation and the deposition of insoluble fibrin.
AAA and Coagulopathy

- Open repair
  - Increased thrombin generation and activity
  - Increased fibrin turnover
  - Attenuation of prothrombotic diathesis by 3 months, but TAT and D-dimer remain elevated
  - Atherosclerotic disease also contributes to hemostatic derangement
AAA and Coagulopathy

- EVAR
  - Activation of the coagulation and fibrinolytic pathways and Platelet consumption
    - Thrombin generation may be higher than Open: Mechanical injury by instrumentation
    - Contrast agent injures endothelium and causes platelet activation
    - Larger prosthetic surface area
  - Proinflammatory properties of residual thrombus in the aneurysm sac
    - Type II endoleak: proinflammatory mediators in contact with systemic circulation
  - Improved by 3 months, but not normalized
Normal growth rate is 3~4 mm/year
AAA expand more rapidly as they get larger

Higher rate in smokers
Slower rate in DM or PVOD

Risk of Rupture

United Kingdom Small Aneurysm Trial

- < 4 cm: 0.3% for AAAs
- 4~4.9 cm: 1.5% for AAAs
- 5~5.9 cm: 6.5% for AAAs

- Not accurate for Women (17% of the trial)
  - had a 4.5-fold higher risk for rupture than men did.
- An underestimate of actual annual rupture risk because some underwent repair.

## Rupture Risk

<table>
<thead>
<tr>
<th>AAA diameter</th>
<th>Rupture Risk (%/yr)</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>
Diagnosis

- Physical Examination
- Ultrasound
- CT
- **NOT** Aortogram
Screening

- 4 Randomized Controlled Trials
  - Involved more than 125,000 men
  - Each trial observed a reduction in AAA-related mortality ranging from 21% to 68%. (statistically significant in 2 trials)
  - Only one trial included women: found no benefit.
  - If an initial screening study is negative, No need to repeat
    - Additional small AAA were detected in 2% to 4% at 4 to 12 years of follow-up
    - Almost all were <4 cm and non were likely to require repair during the life of the patient
Screening: US Preventive Services Task Force Recommendation 2005

- One time screening in men aged 65 to 75 who have ever smoked (100 cigarettes).
  - This encompasses 69% of men aged 65 to 75 in the US. This is a “grade B” recommendation (“at least fair evidence ... improves important health outcomes and concludes that benefits outweigh harms”)
- No recommendation for or against screening for AAA in men aged 65 to 75 who have never smoked
- Against screening in women
  - It failed to take into account specific risk factors that may make screening cost-effective in selected groups.
Medicare will pay for screening for once as “Welcome to Medicare” preventive visit.

Eligibility
- Have a family history of AAA
- Males 65 to 75 years of age who have ever smoked (at least 100 cigarettes).
Reasonable for men > 60 years who would be candidates for at least EVAR
Beneficial for women if they have other risk factors for AAA (smoking or a family history)
Screen at earlier age (50 ~ 55 years) for those with high risk factors
Repeat screening may be reasonable in patients with family history who were screened at a young age
Smoking cessation

- The single most important modifiable risk factor for both the development and expansion of AAA
- UKSAT: only factor linked to increased growth rates
- PAD & DM: significantly slower growth rate: Age, gender, hypertension, and cholesterol level had no impact
Beta Blocker

- Effective in reducing growth rate in animal studies
- In PRC trials, No effect on AAA growth
- Not well tolerated, worse quality of life
- In pts who tolerated it well: No reduction in AAA expansion rate
Antibiotics

- *Chlamydia pneumoniae*
- Not due to antibiotic property
  - No correlation b/w chlamydia titer and expansion suppression
- Roxithromycin 30 mg/d
- Doxycycline 150 mg/d
  - Suppress expression of MMP in human AAAs and reduce aneurysm formation in animals
  - Growth rate halved at 18 mos after Rx for 3 mos
Statins have been shown to decrease MMP-9 within the aneurysm wall. No randomized prospective studies related to statins and aneurysms.
Treatment Options

- Open Surgical Repair
  - Charles Dubost in 1951: First open repair of AAA with a homograft

- Endovascular Repair
  - Juan Parodi in 1991: First endovascular repair of AAA
Open Surgical Repair

- Sac is opened
- All feeding vessels within sac are ligated
- Graft is hand-sewn
- Sac is closed around the graft
- Technically demanding
- Morbidity can be significant
  Technically demanding
- Proven durability
Open Surgical Repair

- Evolution and perfection of surgical techniques since 1951
- Measure of success: Patient Survival and rupture prevention
- The Standard of Care
Open Surgical Repair

- Mortality rate: <5%
- Without CAD, Renal insuff. & COPD: <2%
- With all 3: ~50%
Open Surgical Repair

- Big incision
- Postoperative pain
- Longer recovery: ~3 mos to return to baseline
- Not everybody is a candidate
# Open Surgical Repair

### Morbidity rates

<table>
<thead>
<tr>
<th>Condition</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>All cardiac</td>
<td>15%</td>
</tr>
<tr>
<td>MI</td>
<td>2-8%</td>
</tr>
<tr>
<td>All pulmonary</td>
<td>8-12%</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>5%</td>
</tr>
<tr>
<td>Worsening renal</td>
<td>5-12%</td>
</tr>
<tr>
<td>Stroke</td>
<td>1%</td>
</tr>
<tr>
<td>Leg ischemia</td>
<td>1-4%</td>
</tr>
<tr>
<td>Colon ischemia</td>
<td>1%</td>
</tr>
<tr>
<td>Spinal cord ischemia</td>
<td>0.25%</td>
</tr>
</tbody>
</table>
Patient Apprehension
First EVAR 1991

J.C. Parodi

Parodi’s initial EVAR device in 1990

First English language-publication by Juan Parodi et al in 1991

Transfemoral Intraluminal Graft Implantation for Abdominal Aortic Aneurysms

J.C. Parodi, MD*, J.C. Palmaz, MD†, H.D. Barone, PhD, Buenos Aires, Argentina, and San Antonio, Texas

*Ann Vasc Surg 1991;5:491–499
Currently Available FDA-Approved EVAR Devices

Excluder, Zenith, Talent, Powerlink, Endurant, Ovation
Endovascular Repair

- Blood flow and the pressure is re-routed via the endoluminal graft
- Fixation is dependent on friction and hooks
- Feeding vessels are allowed/hoped to thrombose
- Avoids big incision
- Fast recovery
Is EVAR better than Open repair?
UK EVAR-1 Trial

- Randomized comparison of EVAR vs OSR for AAA ≥ 5.5 cm in patients > 60 Years old
- GOOD RISK CANDIDATES Suitable for both
- 1082 patients from 1999 to 2003
- 41 Hospitals

DREAM Trial

- Randomized comparison of EVAR vs OSR for AAA ≥ 5.0 cm
- GOOD RISK CANDIDATES Suitable for both
- 351 patients from 11/2000 to 12/2003
- 28 Hospitals in Holland and Belgium
- AAA Target recruitment (400) not reached

Prinssen et al. *NEJM* 2004
OVER Trial

- Randomized comparison of EVAR vs OSR
- GOOD RISK CANDIDATES Suitable for both
- From 10/2002 to 04/2008
- 881 patients
- 42 VA Hospitals in the USA
- Designed to have 80% power to detect a 25% relative reduction in death after 9 years

# UK EVAR-1 Trial

<table>
<thead>
<tr>
<th>30-Day</th>
<th>Open N=539</th>
<th>EVAR N=543</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Survival rate</td>
<td>4.7%</td>
<td>1.7%</td>
<td>0.01</td>
</tr>
<tr>
<td>Freedom from reintervention</td>
<td>5.8%</td>
<td>9.8%</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Unequivocal Proof: EVAR Reduces mortality of AAA Repair

## DREAM Trial

<table>
<thead>
<tr>
<th></th>
<th>Open N=174</th>
<th>EVAR n=171</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>30-day Mortality</td>
<td>4.6%</td>
<td>1.2%</td>
<td>0.1</td>
</tr>
<tr>
<td>Death + Serious Morbidity</td>
<td>9.8%</td>
<td>4.7%</td>
<td>0.1</td>
</tr>
<tr>
<td>Moderate + Severe Comp.</td>
<td>26.4%</td>
<td>11.7%</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

**EVAR is Preferable over Open Repair**

Prinssen et al. *NEJM* 2004
## Early Results

|                | Open  
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>n=437</td>
<td></td>
</tr>
<tr>
<td>Survival rate</td>
<td>3.0%</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>
|                | EVAR  
| n=444          |       |
|                | 0.5%  |
|                |       |
|                | P     |
|                | 0.004 |

Prospective Randomized Trials: Early Results

- Superior Early Safety and Efficacy of EVAR compared to Open Repair
### EVAR-1 Trial - Long-term

<table>
<thead>
<tr>
<th>At 8 Years</th>
<th>Open N=626</th>
<th>EVAR N=626</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any Death</td>
<td>264/626</td>
<td>260/626</td>
<td>0.72</td>
</tr>
<tr>
<td>AAA-related Death</td>
<td>40/3626</td>
<td>36/626</td>
<td>0.73</td>
</tr>
<tr>
<td>Complications</td>
<td>2.5%</td>
<td>12.6%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Reintervention</td>
<td>1.7%</td>
<td>5.1%</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*NEJM 2010*
EVAR-1 Trial - Longterm

FREEDOM FROM COMP.

- Endovascular repair, 48% (95% CI, 43–52)
- Open repair, 85% (95% CI, 81–88)

No. at Risk
- Endovascular repair: 626, 378, 280, 174, 58
- Open repair: 626, 496, 413, 259, 91

FREEDOM FROM REINTERVENTION

- Endovascular repair, 72% (95% CI, 67–76)
- Open repair, 90% (95% CI, 87–93)

No. at Risk
- Endovascular repair: 626, 470, 377, 243, 83
- Open repair: 626, 503, 428, 271, 97

NEJM 2010
At median follow-up of 6.4 years, the majority of late deaths were not aneurysm- or cardiovascular-related.

<table>
<thead>
<tr>
<th></th>
<th>Open n=178</th>
<th>EVAR n=173</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Survival rate</td>
<td>69.9%</td>
<td>68.9%</td>
<td>0.97</td>
</tr>
<tr>
<td>Freedom from reintervention</td>
<td>81.9%</td>
<td>70.4%</td>
<td>0.03</td>
</tr>
</tbody>
</table>
DREAM Trial-Longterm

De Bruin et al. NEJM 2010
Long-Term Comparison of Endovascular and Open Repair of Abdominal Aortic Aneurysm

Frank A. Lederle, M.D., Julie A. Freischlag, M.D., Tassos C. Kyriakides, Ph.D., Jon S. Matsumura, M.D., Frank T. Padberg, Jr., M.D., Ted R. Kohler, M.D., Panagiotis Kougias, M.D., Jessie M. Jean-Claude, M.D., Dolores F. Cikrit, M.D., and Kathleen M. Swanson, M.S., R.Ph., for the OVER Veterans Affairs Cooperative Study Group*
OVER Trial Freedom from Death

Hazard ratio, 0.97 (95% CI, 0.77–1.22)
P = 0.81

No. at Risk
Open: 437 410 386 354 329 266 169 102 35
Endovascular: 444 423 410 381 347 265 159 94 34

NEJM November 22, 2012
OVER Trial: Freedom from death or reintervention

**Figure B**

Cumulative Probability of Death or Secondary Procedure

- **No. at Risk**
  - Open: 437, 385, 347, 314, 284, 222, 133, 79, 28
  - Endovascular: 444, 389, 366, 334, 292, 217, 123, 69, 23

- **Hazard ratio**: 1.06 (95% CI, 0.87–1.28)
- **P = 0.57**

*NEJM November 22, 2012*
Strikingly similar results between the three trials
Early benefit with EVAR is lost by 2-3 years
Similar late death rate
Higher graft-related complications and reinterventions with EVAR, and new complications continued to develop: In OVER trial, same reintervention rate (inc. Laparotomy-related complications)
Good medical therapy may forestall expected cardiovascular mortality
Old people do not benefit from EVAR
What is happening in the real world?
A large number of EVARs are being performed in large teaching hospitals as well as small community hospitals.
Real World Experience

Endovascular vs. Open Repair of Abdominal Aortic Aneurysms in the Medicare Population

Marc L. Schermerhorn, M.D., A. James O’Malley, Ph.D., Ami Jhaveri, M.D.,
Philip Cotterill, Ph.D., Frank Pomposelli, M.D., and Bruce E. Landon, M.D., M.B.A.

Real World Experience-Medicare Database

<table>
<thead>
<tr>
<th>Propensity Matched Medicare Population</th>
<th>30-day Mortality</th>
<th>Long-term Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>EVAR n=22,830</td>
<td>1.2%</td>
<td>34.0% at 5 yrs</td>
</tr>
<tr>
<td>Open n=22,830</td>
<td>4.8%</td>
<td>34.3% at 5 yrs</td>
</tr>
</tbody>
</table>

- Higher Reintervention rates after EVAR: 9.8% vs 1.7%
- Higher Surgery for Laparotomy-related complications after Open repair: 9.7% vs 4.1%

# Medicare Database Review

<table>
<thead>
<tr>
<th>Perioperative Outcome</th>
<th>Endovascular Repair (N = 22,830)</th>
<th>Open Repair (N = 22,830)</th>
<th>P Value</th>
<th>Relative Risk Associated with Open Repair (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical complications (% of patients)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>7.0</td>
<td>9.4</td>
<td>&lt;0.001</td>
<td>1.34 (1.26–1.42)</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>9.3</td>
<td>17.4</td>
<td>&lt;0.001</td>
<td>1.89 (1.79–1.98)</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>5.5</td>
<td>10.9</td>
<td>&lt;0.001</td>
<td>2.00 (1.87–2.14)</td>
</tr>
<tr>
<td>Renal failure requiring dialysis</td>
<td>0.4</td>
<td>0.5</td>
<td>0.047</td>
<td>1.33 (1.00–1.75)</td>
</tr>
<tr>
<td>Deep-vein thrombosis or pulmonary embolism</td>
<td>1.1</td>
<td>1.7</td>
<td>&lt;0.001</td>
<td>1.51 (1.29–1.76)</td>
</tr>
</tbody>
</table>

## Medicare Database Review

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Year 4</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Endovascular Repair</td>
<td>Open Repair</td>
<td>Endovascular Repair</td>
<td>Open Repair</td>
<td>Endovascular Repair</td>
</tr>
<tr>
<td>Rupture</td>
<td>0.3</td>
<td>0.2</td>
<td>0.7</td>
<td>0.3</td>
<td>1.3</td>
</tr>
<tr>
<td>Any aneurysm-related reintervention</td>
<td>2.7</td>
<td>0.5</td>
<td>4.8</td>
<td>0.8</td>
<td>7.0</td>
</tr>
<tr>
<td>Major reintervention</td>
<td>0.4</td>
<td>0.2</td>
<td>0.7</td>
<td>0.3</td>
<td>1.2</td>
</tr>
<tr>
<td>Conversion to open repair</td>
<td>0.1</td>
<td>0.2</td>
<td>0.3</td>
<td>0.2</td>
<td>0.3</td>
</tr>
<tr>
<td>Open aneurysm repair</td>
<td>0.3</td>
<td>0.1</td>
<td>0.5</td>
<td>0.1</td>
<td>0.9</td>
</tr>
<tr>
<td>Repeat aneurysm repair or aortobifemoral bypass</td>
<td>0.1</td>
<td>0.1</td>
<td>0.4</td>
<td>0.1</td>
<td>0.7</td>
</tr>
<tr>
<td>Axillofemoral or axillobifemoral bypass</td>
<td>0.1</td>
<td>0.1</td>
<td>0.2</td>
<td>0.1</td>
<td>0.2</td>
</tr>
<tr>
<td>Repair of infected graft or graft–enteric fistula</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.2</td>
<td>0.2</td>
</tr>
</tbody>
</table>
As compared with open repair, endovascular repair of abdominal aortic aneurysm is associated with lower short-term rates of death and complications. The survival advantage is more durable among older patients. Late reinterventions related to abdominal aortic aneurysm are more common after endovascular repair but are balanced by an increase in laparotomy-related reinterventions and hospitalizations after open surgery.
EVAR Complications

- Endoleak
- Migration
- Material fatigue
- Sac growth
- Rupture
- Iatrogenic
Endoleak
## Endoleak Types

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Attachment leak</td>
</tr>
<tr>
<td>II</td>
<td>Branch flow</td>
</tr>
<tr>
<td>III</td>
<td>Defect in graft or modular disconnection</td>
</tr>
<tr>
<td>IV</td>
<td>Fabric porosity</td>
</tr>
</tbody>
</table>
Type II Endoleaks

- Incidence: ~10% to 30% of patients
- Transient: resolves within 6 mos
- Persistent: present beyond 6 mos
- Most are innocuous, but when associated with sac growth, carry adverse outcomes
- Most are IMA and Lumbars (38% each)
- Treated by coil embolization, Onyx (ethylene vinyl alcohol in dimethyl sulfoxide) glue, ligation (laparoscopic & open), sac plication & explantation

Schanzer Circulation 2011
Gallagher et al JEVT 2012
Sarac et al JVS 2012
Type II Endoleaks

- Overall complication rate with Rx: 8.6%
- Most require multiple reinterventions with the initial success rate of only 17%
- Onyx more effective than others
  - 40% recurrence rate
- After 5 yrs,
  - 20% require secondary procedure
  - 38% continued sac growth
  - 8.4% explant or open repair

Gallagher et al JEVTS 2012
Abularrage JVS 2012
Sarac et al JVS 2012
Abularrage JVS 2012
Modular Disconnection

April 2000

December 2000
Metal Fatigue

Aug 02

Dec 02

Fractures
Device Migration and limb disconnection after sac shrinkage
Suture Miniholes

Courtesy of K. Ouriel
AAA Sac: Majority...Stable

preop

12 months

24 months

Cho JVS 2004 June
Shrinkage

preop
6 months
12 months
24 months

Cho JVS 2004 June
Sac Increase

Cho JVS 2004 June
Excluder: Sac Re-Expansion

1 month: 59.1mm
6 months: 47.9mm
4 years: 54.9mm
Late abdominal aortic aneurysm enlargement after endovascular repair with the Excluder device

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Excluder Sac Size: Comparison to Baseline

- Percentage of increase, decrease, and no change over time.
- 37% highlighted with a red circle.
Fabric Failure: Hygroma

Highly Viscous Fluid or Gel in Sac

Courtesy of M Mehta, MD
Excluder Hygroma

Overlap zone free

Protein Rich Coagulum
Sac behavior after aneurysm treatment with the Gore Excluder low-permeability aortic endoprosthesis: 12-month comparison to the original Excluder device

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J Vasc Surg 2006;44:694-700
Sac Size Change ≥ 5mm at 1 Year (UPMC + Northwestern)
Long-term sac behavior after endovascular abdominal aortic aneurysm repair with the Excluder low-permeability endoprosthesis

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Predictors of Abdominal Aortic Aneurysm Sac Enlargement After Endovascular Repair

Andres Schanzer, MD; Roy K. Greenberg, MD; Nathanael Hevelone, MPH; William P. Robinson, MD; Mohammad H. Eslami, MD; Robert J. Goldberg, PhD; Louis Messina, MD
Sac Enlargement

- Review of preoperative and postoperative imaging from M2S database 199-2008 in 10228 patients
- Data on Specific Device used were not available
- Most liberal and conservative IFU applied
- Sac size increase > 5 mm used

Schanzer et al Circulation 2011
Sac Enlargement

- 59% had a maximum AAA diameter < 55 mm
- 42% met anatomy meeting the most conservative definition of IFU
- 69% met anatomy meeting the most liberal definition of IFU
- 41% 5 year post-EVAR rate of AAA growth

Predictors of growth:
- age ≥ 80,
- aortic neck diameter ≥ 28 mm
- neck angle > 60°
- common iliac diameter > 20 mm

Schanzer et al Circulation 2011
Sac Enlargement

- 30% of sac growth after >3 yrs
- Higher growth rate outside of IFU
- Higher growth rate in recent years
Open Conversion
Delayed open conversions after endovascular abdominal aortic aneurysm repair

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Objective: Secondary interventions after endovascular aneurysm repair (EVAR) remain a concern. Most are simple catheter-based procedures, but in some instances, open conversions (OCs) are required and carry a worse outcome. We reviewed our experience to characterize these OCs.

Methods: A retrospective review was conducted of all patients who underwent an OC after a previous EVAR for an aneurysm-related indication from 2001 to 2010. Clinical outcomes are reported.

Results: Data were reviewed for 44 patients (77% men) with a mean age of 74 years (range, 55-90 years). The average time from EVAR to the first OC was 45 months (range, 2-190 months). In six patients (14%), the initial EVAR was at another institution. The endografts used were Ancure in 16, Excluder in 13, AneuRx in 8, Zenith in one, Renu in one, and undetermined in two. Twenty-two patients had previous endovascular reinterventions before their index OC. Indications for OC were aneurysm expansion in 15 (27%), and infection in four (9%). The endograft was preserved in situ in 10 patients (23%) or complete in 16 (41%). Endograft preservation was used for type II endoleak in ten cases. Ligation of the culprit arteries (lumbar in four, inferior mesenteric artery in five, and middle sacral in one). Proximal neck banding was performed in one type Ia endoleak. Overall morbidity was 55%, and mortality was 18%. No deaths occurred in a subgroup of patients who underwent endograft preservation with selective ligation of culprit vessels for type II endoleak. Intraoperative complications included bowel injury in two, bleeding in two, splenectomy in one, and ureteral injury in one. At a mean follow-up of 20 months, two patients underwent additional procedures after the index OC: one after endograft preservation and one after partial explantation. None of the patients who underwent elective OC with endograft preservation required subsequent endograft explantation.

Conclusions: Most OCs after EVAR are associated with significant morbidity and mortality, except when electively treating an isolated type II endoleak with ligation of branches and preservation of the endograft. (J Vasc Surg 2012;55:1562-9.)
Overall 30-day mortality was 17%, with an elective case mortality of 9.9%, nonelective case mortality of 37%, and 56% mortality for ruptures.
Rupture
AAA Rupture after EVAR
AAA rupture

- Although rare, Rupture after EVAR does occur with the annual cumulative risk of between 0.5 and 1.2%/patient/year
- Factors associated with Post-EVAR Rupture
  - Timing (within first 3 years)
  - Endoleaks
  - Migration
  - Sac Growth
  - Preoperative diam >6.5 cm
  - Follow-up noncompliance

Schlosser EJVS 2009;37:15
Coppi JVS 2009;49:582
Fransen EJVS 2003;26:487
May EJVS 1999;18:344
Prior endovascular abdominal aortic aneurysm repair provides no survival benefits when the aneurysm ruptures

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Objective: It has been proposed that prior endovascular abdominal aortic aneurysm (AAA) repair (EVAR) confers protective effects in the setting of ruptured AAA (rAAA). This study was conducted to compare outcomes of rAAA repairs in patients with and without prior EVAR.

Methods: A retrospective review identified 18 patients with (group 1) and 233 patients without (group 2) antecedent EVAR who presented with rAAA from January 2001 to December 2008. Patient characteristics and perioperative variables were noted and the outcomes were compared. Multiple logistic regression was used to identify factors contributing to morbidity and mortality and Kaplan–Meier survival analysis was performed.

Results: Baseline characteristics were similar between groups. Median follow-up was 8 years in group 2 (P = .17). Men comprised 83.3% of patients in group 1 and 85.9% in group 2 (P = .50). Mean instability at rAAA was noted with similar frequency between groups (48.2% vs 46.7%; P = .53). Median time from EVAR to rAAA was 4.0 years and from last follow-up computed tomography to rAAA was 1.0 years. Endoleaks were Ancure (Guidant, Menlo Park, Calif) (9), AnuRx (Medtronics, Minneapolis, Minn), Excluder (W.L Gore, Flagstaff, Ariz) (1), and Excluder (Guidant, Menlo Park, Calif) (3), and Excluder (W.L Gore, Flagstaff, Ariz) (1). All but 1 patient had an endoleak at the time of rupture. Of 14 patients with droplets, 3 patients had a known increase in size (≥5 mm) and only 3 were known to have an endoleak. Fifteen patients were treated by a single intervention, whereas 3 patients underwent multiple procedures. In group 2, open repair was performed in 218 patients and EVAR in 15. Morbidity (66.7% vs 56.7%) and in-hospital mortality (38.9% vs 36.9%) were nearly identical between groups. One-year survival rates (27.8% vs 48.2%; P = .15) were also similar. The mortality rates for EVAR for primary rAAA was 20% as compared to 38.1% for open repair for rAAAs (P = .27).

Conclusion: rAAA remains a lethal problem in patients with and without prior EVAR alike. An existing endograft provides neither acute nor 1-year survival benefits after rAAA repairs. Prediction of patients at risk for rupture post-EVAR is difficult, as only a minority of patients had a known prior endoleak or sac enlargement. (J Vasc Surg 2010;52:1127-34.)
Conclusion

- Prior EVAR provides No Survival Benefits when the aneurysm ruptures.

- AAA sac after EVAR behaves in an unpredictable manner. Most endoleaks were new and sac were stable at last follow-up.
Beyond infrarenal AAA?
Chimney grafts

- Only for urgent / emergent or high risk patients!
- If AAA extends right up to the renal arteries
- Needs brachial approach
- Experienced endovascular skills required
- Can be used for endografts with infrarenal and suprarenal fixation
Chimney grafts
An unfavorable or short proximal aortic neck is the most common (up to 40%) factor limiting the applicability of EVAR.

A fenestrated graft extends the seal zone to the more stable para-visceral aorta while allowing perfusion of the visceral vessels through fenestrations in the stent graft.
FEVAR

- Each stentgraft is designed based on individual patient aneurysm and visceral vessel morphology.

- The fenestrations are oriented based on preoperative imaging and are reinforced with a nitinol ring.
FEVAR

A stent is flared in the visceral fenestrations to:

- Maintain alignment with artery
- Protect visceral arterial orifice
- Help with sealing against aorta
First FEVAR in Chicagoland Feb 2013

- 76 year old white male with juxtarenal AAA noted on evaluation for LLQ pain
- Inadequate neck for standard EVAR
- PMH – HTN, CAD, Heart attack
- PSH – None
- Considered for fenestrated EVAR
First FEVAR in Chicagoland Feb 2013
Conclusions

- AAA repair has come a long way!
- EVAR can be performed safely and effectively for the treatment infrarenal AAA with superior short-term outcome.
Conclusions

- Long-term performance is patient-and device-specific
- Life-long surveillance is mandatory
Questions

- Why do some AAA grow / rupture and some don’t?
- Why do some endoleaks resolve and some don’t?
- Why do some have intraluminal thrombus and some don’t?
- How can sac behavior be predicted?
Areas of Research

- Impact of EVAR on hemotologic derangements
- Utility of hemostasis, coagulation and inflammatory markers in
  - Prediction / identification of patients with AAA growth > 5 cm
  - Prediction / identification of patients with post-EVAR endoleaks and sac behavior
Thank you.