CURRENT TREATMENT OF CHRONIC TBADs, ITS COMPLICATIONS & ANEURYSMS

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FACULTY DISCLOSURE
Gustavo S. Oderich MD
• Consulting, DSMB, CEC*
  Cook Medical Inc., WL Gore, Lombardi
• Honoraria
  WL Gore, Endologix
• Research grants*
  Cook Medical Inc., WL Gore, Atrium Maquet

SPECTRUM OF AORTIC DISEASE

Aneurysms
- Dissections
- IMH
- PAU
- Other rarities
  Trauma
  Vasculitides, coarctations, thrombus

SPECTRUM OF AORTIC DISEASE

TEVAR FOR TBCDs

<table>
<thead>
<tr>
<th></th>
<th>Acute Dissection (n=248)</th>
<th>Chronic Dissection (n=187)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>59±6</td>
<td>68±7</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Technical success</td>
<td>93±1%</td>
<td>96±1%</td>
<td>.57</td>
</tr>
<tr>
<td>30-day mortality</td>
<td>10±2%</td>
<td>3±1%</td>
<td>.015</td>
</tr>
<tr>
<td>Morbidity</td>
<td>22±3%</td>
<td>9±2%</td>
<td>.005</td>
</tr>
<tr>
<td>Retrograde type A</td>
<td>2±1%</td>
<td>3±1%</td>
<td>.55</td>
</tr>
<tr>
<td>Paraplegia</td>
<td>1±0.6%</td>
<td>0.5±0.5%</td>
<td>.79</td>
</tr>
<tr>
<td>1-yr survival</td>
<td>87±2%</td>
<td>93±2%</td>
<td>.08</td>
</tr>
</tbody>
</table>

*All consulting fees and grants paid to Mayo Clinic

Eggebrecht et al. Eur Heart J 2006

68M, fit, no genetic cause, DeBakey Ia s/p prior ascending repair (x2) with homograft.
45M, no genetic cause expansion of the abdominal aorta 8 weeks after acute DeBakey IIIb dissection

FACTORS AFFECTING CHOICE OF REPAIR

- Genetically triggered disease
- Age/clinical risk
- Prior arch/TAA repair
- Proximal landing zone
- Visceral artery targets
  - diameter, dissection, early bifurcation, origin from true/false lumen
- Distal landing zone
- Ilio-femoral access

OPTIONS FOR SAC GROWTH

- Open repair
- Distal false lumen exclusion
  - Closure of reentry sites
  - Embolization (coils, plugs…)
  - Candy plug, knickerbocker
- Completion eTAAA repair
  - Hybrid
  - Parallel stent-grafts
  - Fenestrated-branched endografts

Genetically Triggered Aortic Disease

Pomianowski P, Elefteriades JA. Am Cardiothorac Surg 2013

Classification Chromosome Gene Protein Frequency Inheritance

Syndromic

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Chromosome</th>
<th>Gene</th>
<th>Protein</th>
<th>Frequency</th>
<th>Inheritance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marfan</td>
<td>15q21.1</td>
<td>FBN1</td>
<td>Fibrillin 1</td>
<td>1:5,000-10,000</td>
<td>AD</td>
</tr>
<tr>
<td>Loeys-Dietz</td>
<td>3p24-25</td>
<td>TGFBR2</td>
<td>TGF-β-R2</td>
<td>Rare</td>
<td>AD</td>
</tr>
<tr>
<td>Ehlers-Danlos</td>
<td>2q24.3-31</td>
<td>COL3A1</td>
<td>Collagen III</td>
<td>1:10,000-25,000</td>
<td>AD</td>
</tr>
<tr>
<td>ATS</td>
<td>20q13.1</td>
<td>SLC2A10</td>
<td>GLUT10</td>
<td>Rare</td>
<td>AD</td>
</tr>
<tr>
<td>AOS</td>
<td>15q22.2-24.3</td>
<td>SLC2A10</td>
<td>GLUT10</td>
<td>Rare</td>
<td>AD</td>
</tr>
<tr>
<td>TAA</td>
<td>1q44</td>
<td>TGFBR2</td>
<td>TGF-β-R2</td>
<td>Rare</td>
<td>AD</td>
</tr>
</tbody>
</table>
| Non-Syndromic

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Chromosome</th>
<th>Gene</th>
<th>Protein</th>
<th>Frequency</th>
<th>Inheritance</th>
</tr>
</thead>
<tbody>
<tr>
<td>TAAD2</td>
<td>3p24.3</td>
<td>TGFBR2</td>
<td>TGF-β-R2</td>
<td>~3% of TAA</td>
<td>AD</td>
</tr>
<tr>
<td>TAAD4</td>
<td>16p13.12</td>
<td>ACTA2</td>
<td>SM actin</td>
<td>10-15% of TAA</td>
<td>AD</td>
</tr>
<tr>
<td>TAAD5</td>
<td>9q33-34</td>
<td>TGFBR1</td>
<td>TGF-β-R1</td>
<td>~2% of TAA</td>
<td>AD</td>
</tr>
<tr>
<td>TAAD-PDA</td>
<td>3p21.1</td>
<td>MLCK</td>
<td>MLCK</td>
<td>~1% of TAA</td>
<td>AD</td>
</tr>
</tbody>
</table>

ATS, Aortic Tortuosity Syndrome; AOS, Aneurysm Osteoarthritis Syndrome; TAA, Thoracic Aortic Aneurysm/Dissection.
Genetically triggered disease in young, fit patients treated by open repair

- 26M with Loeys-Dietz Syndrome
- 50M with TAADs (TGFBR1)
- 49M with Marfans

Courtesy of Tom Bower and Alberto Pochettino

OPEN REPAIR FOR CHRONIC TRADs

<table>
<thead>
<tr>
<th>Study period</th>
<th>n</th>
<th>Mean age (years)</th>
<th>Genetically triggered</th>
<th>In-hospital mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>1987-2005</td>
<td>73</td>
<td>66 ± 13</td>
<td>65% (Marfan 15%)</td>
<td>11%</td>
</tr>
<tr>
<td>2005-2013</td>
<td>198</td>
<td>58 (46 – 62)</td>
<td>7% (Marfan/LDS 7%)</td>
<td>5%</td>
</tr>
<tr>
<td>1991-2011</td>
<td>519</td>
<td>59 (19 – 87)</td>
<td></td>
<td>9%</td>
</tr>
</tbody>
</table>

Outcomes of open distal aortic aneurysm repair in patients with chronic DeBakey type I dissection

Joseph S. Coselli, MD,
Susan Y. Green, MPH,
Samantha Zarda, MS,
Courtney C. Nalty, MSPH,
Matt D. Price, MS,
Michael S. Hughes, BM,
Ourania Preventza, MD,
Kim I. de la Cruz, MD,
Scott A. LeMaire, MD

Conrad/ Cambria et al
J Vasc Surg 2011
Estrera/Safi et al
Ann Thorac Surg 2015
Coselli et al
J Thorac Cardiovasc Surg 2014
Conrad/M Cambria et al
J Vasc Surg 2011
Coselli et al
J Thorac Cardiovasc Surg 2014
Estrera/Safi et al
Ann Thorac Surg 2015

In-hospital mortality
- 11%
- 7%
- 9%

Stroke
- 3%
- 5%
- 2%

Spinal Cord Injury
- 16%
- 6%
- 3%

Tracheostomy
- ?
- 11%
- 4%

Dialysis
- ?
- 7%
- 5%

COMPLETION eTAAA REPAIR
Parallel SGs
Fenestrated branched SGs
Hybrid repair
Gib Upham Jr MD Staged Hybrid repair
Armando Lobato MD PhD Parallel SGs for TAAA

69F with type II TAAA, chronic dissection and severe COPD

78M with prior hemi-arch/TEVAR, severe CKD, CHF

69M with total arch on dialysis and asymptomatic expanding Type II TAAA
**F-BEVAR FOR CHRONIC TBADs**

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>n</th>
<th>Mean age</th>
<th>Technical success (%)</th>
<th>30-d Mortality (%)</th>
<th>Re-intervention (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oikonomou K, Verhoeven E et al. Ann Cardiothorac Surg 2014</td>
<td>17</td>
<td>65±8</td>
<td>100</td>
<td>12</td>
<td>20</td>
</tr>
<tr>
<td>Kitagawa A, Greenberg R et al. J Vasc Surg 2013</td>
<td>30</td>
<td>64±8</td>
<td>100</td>
<td>0</td>
<td>27</td>
</tr>
<tr>
<td>Mayo Clinic Experience</td>
<td>6</td>
<td>71±3</td>
<td>100</td>
<td>0</td>
<td>33</td>
</tr>
<tr>
<td>Case reports</td>
<td>2</td>
<td>73±3</td>
<td>100</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

**Oour Approach to Chronic TBADs**

Based on most common presentations

<table>
<thead>
<tr>
<th>Presentation</th>
<th>Genetic Cause</th>
<th>No Genetic Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elective, good landing zones</td>
<td>Open Repair</td>
<td>TEVAR</td>
</tr>
<tr>
<td>Rupture</td>
<td>TEVAR Open Repair</td>
<td>TEVAR</td>
</tr>
<tr>
<td>Arch involvement</td>
<td>Total arch ± Open Repair</td>
<td>Total arch ± Frozen ET Debranch/ Branch &amp; TEVAR</td>
</tr>
<tr>
<td>TAAA degeneration</td>
<td>Open Repair</td>
<td>F-BEVAR</td>
</tr>
</tbody>
</table>

**DIRECT FALSE LUMEN OCCLUSION**

78M, DeBakey Type I, prior ET arch repair, enlarging TAAA

58M, fit, no genetic cause and DeBakey IIIb dissection treated by TEVAR with late endoleak and DIC

**MAYO CLINIC 150 Years SERVING HUMANITY**
Prior to exclusion

After to exclusion by plugs

ACUTE, RUPTURED DISSECTION OR SAC GROWTH AFTER TEVAR

DISEASE PROGRESSION

- Aortic enlargement in >80% of patients
- False lumen growth averages 3mm/year
- 50% of patients rupture or require repair within 4 years
- Repair indicated in patients with size >5.5-6cm, symptoms, recurrent events or organ malperfusion

Fattori et al. JACC 2013