Cephalic arch and Central venous stenoses: best practices

AQ Urbanes, MD
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Disclosures

Vice-President, Lifeline Vascular Access
Consultant, Bard PV

Multifactorial causes of arch pathology

• shear stress
  – high flow
  – angulation of vein
• relatively immobile
  – tethered to fascia and muscle
  – impaired remodeling
• hypertrophied valves
Extent of the problem

- 15-40% of dysfunctional fistulae
- brachiocephalic >> radiocephalic
- non-DM > DM
- high-flow AVF
- Ca x iP product
- ESRD 2nd renovascular disease

Surgical options

What is the problem?

- resistant to high-pressure balloons
- non-durable results
- prone to rupture
- endovascular stents may limit future surgical options
  - translocation
  - use of basilic/axillary venous outflow

Non-surgical options

<table>
<thead>
<tr>
<th></th>
<th>6 mo. 1st patency</th>
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<th># procedures/patient year</th>
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<td>83 ± 8</td>
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Surgical outcomes

<table>
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<tr>
<th></th>
<th>Chen 2005</th>
<th>Kian 2008</th>
</tr>
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<tbody>
<tr>
<td>n</td>
<td>7</td>
<td>13</td>
</tr>
<tr>
<td>Immediate surgical success</td>
<td>86%</td>
<td>100%</td>
</tr>
<tr>
<td>Primary patency</td>
<td>15.7 ± 14.3 months (range = 0 to 39)</td>
<td>8%</td>
</tr>
<tr>
<td>6 mo. primary patency before revision</td>
<td>8%</td>
<td>69%</td>
</tr>
<tr>
<td>6 mo. primary patency after revision</td>
<td>0%</td>
<td>39%</td>
</tr>
<tr>
<td>12 mo. primary patency before revision</td>
<td>0%</td>
<td>39%</td>
</tr>
<tr>
<td>12 mo. primary patency after revision</td>
<td>0%</td>
<td>39%</td>
</tr>
<tr>
<td># interventions per patient year before surgery</td>
<td>0.54</td>
<td>3.5</td>
</tr>
<tr>
<td># interventions per patient year after surgery</td>
<td>1.0</td>
<td>1.0</td>
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Review

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Best practices

- Anticipate the problem
  - high-flow mature brachiocephalic AVF
- Treat the patient, not the lesion
  - clinical presentation
  - multi-segment stenoses
  - judicious angioplasty
- Treatment choices
  - PTA
  - then?
    - endovascular stenting vs. flow re-equilibration vs. both
    - vessel translocation

Central venous catheter & stenosis

- risk is related to
  - side (left 2x risk vs. right)
  - vein (subclavian 42% > IJ 10%)
  - multiple catheters
  - duration of catheter use
- no data relating risk to catheter caliber

Central venous stenosis

Etiology

- central venous catheters
- PICC
- cardiac rhythm devices (PPM/AICD)
- not associated with hardware

Catheter-related injury

- underlying venous pathology in CKD
- injury during or after insertion
- mechanical irritation
- inflammation from catheter-related infection
- irritation to endothelium during cardiac and respiratory motion

normal vs. CKD vein

Feinfeld 1999. AJKD. 34: 702.
Cephalic wall Δs in CKD

<table>
<thead>
<tr>
<th>Change</th>
<th>number</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>intimal hyperplasia</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>wall collagenosis</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>disruption/loss of internal elastic lamina</td>
<td>9</td>
<td>45%</td>
</tr>
<tr>
<td>disruption/loss of endothelial cell layer</td>
<td>6</td>
<td>30%</td>
</tr>
<tr>
<td>muscle loss or atrophy</td>
<td>6</td>
<td>30%</td>
</tr>
<tr>
<td>mucoid or myxoid degeneration</td>
<td>6</td>
<td>30%</td>
</tr>
<tr>
<td>inflammatory reaction – erythrocyte/histiocyte infiltration</td>
<td>5</td>
<td>25%</td>
</tr>
<tr>
<td>mural calcification</td>
<td>3</td>
<td>15%</td>
</tr>
<tr>
<td>telangiectasia</td>
<td>2</td>
<td>10%</td>
</tr>
</tbody>
</table>


Central venous stenosis

Review

• Vascular hardware use is the single most important modifiable factor in the genesis of CV disease.
• Changes consistent with neo-myointimal hyperplasia are present in the CKD patient even before access creation.
• Changes are mediated by neuro-humoral mechanisms which are more prominent in the uremic environment.

Central venous stenosis

Subclavian vein log-jamming

Clinical presentation

from G Beathard, MD

Brachiocephalic vein stenosis with collaterals

from G Beathard, MD
Prevalence of Central Venous disease

- in general population
  - not known

### Prevalence

<table>
<thead>
<tr>
<th>Group</th>
<th>Incidence of Central Venous disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>CKD (-)</td>
<td>4.4% asymptomatic 25-50% asymptomatic</td>
</tr>
<tr>
<td>CKD (+)</td>
<td>30-65% asymptomatic 41% asymptomatic</td>
</tr>
<tr>
<td>hardware (+) vascular access (-)</td>
<td>65% asymptomatic</td>
</tr>
</tbody>
</table>

**Group Incidence of Central Venous disease**

- CKD (-) hardware (+): 24%
- CKD (-) hardware (-): 15%
- CKD (+) hardware (-) vascular access (-): 72%
- CKD (+) hardware (-) vascular access (+): 88%
- CKD (+) hardware (+) vascular access (-): 74%
- CKD (+) hardware (+) vascular access (+): 0.50%/day


### When to treat

<table>
<thead>
<tr>
<th>Treated</th>
<th>Not treated</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>62</td>
</tr>
<tr>
<td>BC/SC</td>
<td>32/30</td>
</tr>
<tr>
<td>% stenosis progression</td>
<td>24</td>
</tr>
<tr>
<td>% stenosis progression</td>
<td>0.50% /day</td>
</tr>
</tbody>
</table>

- n prior cath use: 48%
- 1st 12 mo. patency: 68 ± 6
- 2nd 12 mo. patency: 89 ± 5

Stechler 2006; 238 (3): 1051.


**PTA only**

<table>
<thead>
<tr>
<th>Author</th>
<th>n</th>
<th>6 mo 1st patency</th>
<th>12 mo 1st patency</th>
<th>12 mo 2nd patency</th>
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<tbody>
<tr>
<td>Ozjer 2009</td>
<td>94</td>
<td>77</td>
<td></td>
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<tr>
<td>Bakken 2007</td>
<td>47</td>
<td>29</td>
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<tr>
<td>Maya 2007</td>
<td>32</td>
<td>20</td>
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<tr>
<td>Suwierc 2004</td>
<td>35</td>
<td>43</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quinn 1995</td>
<td>10</td>
<td>12</td>
<td></td>
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<tr>
<td>Kovalik 1994</td>
<td>30</td>
<td>50</td>
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<tr>
<td>Weisselink 1993</td>
<td>15</td>
<td>36</td>
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<tr>
<td>Beathard 1993</td>
<td>27</td>
<td>29</td>
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**Stent after failed PTA or recurrence**

<table>
<thead>
<tr>
<th>Author</th>
<th>Stent</th>
<th>n</th>
<th>6 mo. 1st patency</th>
<th>12 mo. 1st patency</th>
<th>12 mo. 2nd patency</th>
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<tbody>
<tr>
<td>Kundu 2011</td>
<td>PTFE</td>
<td>16</td>
<td>100</td>
<td>100</td>
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<tr>
<td>Jones 2011</td>
<td>PTFE</td>
<td>30</td>
<td>84</td>
<td>45</td>
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<td>Anaya-Ayala 2011</td>
<td>PTFE</td>
<td>25</td>
<td>56</td>
<td>100</td>
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<tr>
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<td>mix</td>
<td>43</td>
<td>68</td>
<td>33</td>
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<tr>
<td>Rajan 2007</td>
<td>nitinol</td>
<td>6</td>
<td>67</td>
<td>67</td>
<td>100</td>
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<tr>
<td>Maya 2007</td>
<td>mix</td>
<td>23</td>
<td>33</td>
<td>19</td>
<td>64</td>
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<tr>
<td>Spronche 2004</td>
<td>mix</td>
<td>29</td>
<td>50</td>
<td>100</td>
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<td>Vagel 2004</td>
<td>nitinol</td>
<td>16</td>
<td>74</td>
<td>67</td>
<td>55</td>
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<td>Aytekin 2004</td>
<td>mix</td>
<td>14</td>
<td>50</td>
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<td>55</td>
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<tr>
<td>Quinn 2003</td>
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<td>6</td>
<td>40</td>
<td>32</td>
<td>39</td>
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<tr>
<td>Oderich 2000</td>
<td>mix</td>
<td>37</td>
<td>41</td>
<td>18</td>
<td>59</td>
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<td>Versely 1998</td>
<td>Wallstent</td>
<td>20</td>
<td>42</td>
<td>25</td>
<td>56</td>
</tr>
<tr>
<td>Gray 1995</td>
<td>Wallstent</td>
<td>32</td>
<td>46</td>
<td>20</td>
<td>33</td>
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**Review**

- Incidence of CV disease in general population is not known.
- Vascular hardware increases incidence of CV disease in both CKD and non-CKD population.
- The contribution of a peripheral AV access to the development of CV disease is not well established. More likely symptomatic.
Flow re-equilibration

• Premise:
  – Central venous disease is not uncommon in CKD.
  – Central venous disease is not symptomatic until an access is created.
  – Presence of vascular access causes dysequilibrium of a previously compensated system.
  – Inflow reduction returns patient to equilibrium.

Flow re-equilibration

• Inflow reduction
  – 22 patients with symptomatic central venous hypertension
  – inflow reduction performed
    • Qa 1640 mL/min to 840 mL/min
  – 20/22 successful
    • 2/22: thrombosis; aneurysm formation
  – mean follow-up 8 months
  – patency data pending

Surgical options

• access ligation or reduction of flow
  – flow re-equilibration
  – allow just enough flow that the collateral circulation can handle
• extra-anatomic bypass
  – jugular vein turn-down
  – subclavian vein to EJ or IJ bypass
  – axillary to femoral vein bypass
  – high morbidity; uncommonly done
    • largest series in publication with n=13
Surgical option

• HeRO® graft

HeRO® patient candidates

• catheter-dependent or approaching catheter-dependency
• non-candidates for fistulae or grafts
• compromised central venous circulation

HeRO® (hemodialysis reliable outflow)

HeRO® components

HeRO® intended use

• HeRO® vascular access device is intended for use
  – in maintaining long-term vascular access
  – for chronic hemodialysis patients
  – exhausted peripheral venous access sites suitable for fistulae or grafts
• FDA classifies the HeRO® device as a graft

outflow (catheter) component
titanium connector

titanium connector

stenosis ≠ stenosis

Itkin. JVIR 2004: 15 (1); 51-56.
Effect of respiration
Best practices

• Prevention
• Treat the patient, not the lesion
  — anatomy, imaging, position, respiration
  — clinical presentation
  — judicious angioplasty
• Treatment choices
  — PTA
  — endovascular stenting
    • stent-graft vs. BMS
  — flow re-equilibration
  — HeRO® graft
  — surgical repair