Carotid Revascularization and Hyper-perfusion Syndrome

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No disclosures related to this presentation
Cerebral Hyperperfusion Syndrome (CHS)

- CHS initially described by Sundt in 1981
- After a successful CEA the typical presentation
  - Increased arterial blood pressure
  - Ipsilateral migraine-like headache
  - Seizure
  - Transient focal neurologic deficits in the absence of cerebral ischemia

- Carotid stenosis can decrease the cerebrovascular reactivity
  - Inflow stenosis results in long standing status of maximally vasodilated cerebral arterioles to maximize tissue perfusion
  - Blunted reactivity of the arterioles in response to the increased perfusion after CEA or CAS results in persistent vasodilation and hyperperfusion
Clinical signs and symptoms

- Hypertension
- Headache
- Non specific neurologic complaints
- Seizures
- Intracranial bleeding
Incidence of CHS

• Relatively low incidence overall:
  • After CEA
    • 1.9% cerebral hyperperfusion syndrome
    • 0.4% intracranial hemorrhage
  • After CAS
    • 1.1% cerebral hyperperfusion syndrome
    • 0.8% intracranial hemorrhage

• High incidence when cerebral blood flow increases >100%
  • 16.7% to 28.6% incidence of cerebral hyperperfusion

Incidence of CHS

- Duration and intensity of cerebral hypoperfusion may impact the severity of microvascular autoregulation impairment
  - High grade ipsilateral carotid stenosis
  - contralateral carotid occlusion
  - poor collateral flow
  - Long standing HTN
- Prior neurologic event
  - Size and acuity add to risk
- Bilateral carotid revascularization
  - Increased risk of hyperperfusion
  - Should stage bilateral revascularization by more than 2-3 months
Single center review of CHS

- 841 CEAs
  - 2008 and 2010
- Uniform neurologic assessments
  - Post op and 30 days
- 14 (1.7%) patients with CHS
  - 5 (0.6%) with headache and/or mild neurologic symptoms
  - 5 (0.6%) with seizures
  - 4 (0.5%) with intracerebral hemorrhage

Maas et al, J of Neurol, Neurosurg & Psych. 84(5):569-72, 2013 May.
<table>
<thead>
<tr>
<th>Patient factor</th>
<th>CHS</th>
<th>No CHS</th>
<th>Univariate p value</th>
<th>Multivariate OR (95% CI), p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elective</td>
<td>35.7%</td>
<td>70.0%</td>
<td>0.01</td>
<td>0.32 (0.10 to 0.98), 0.046</td>
</tr>
<tr>
<td>Dyslipidaemia</td>
<td>64.3%</td>
<td>86.9%</td>
<td>0.03</td>
<td>0.34 (0.11 to 1.05), 0.06</td>
</tr>
<tr>
<td>Baseline diastolic BP</td>
<td>78</td>
<td>70</td>
<td>0.04</td>
<td>1.04 (0.995 to 1.08), 0.08</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>14.3%</td>
<td>38.1%</td>
<td>0.09</td>
<td>NS</td>
</tr>
<tr>
<td>Shunt used</td>
<td>28.6%</td>
<td>16.8%</td>
<td>0.14</td>
<td>NS</td>
</tr>
<tr>
<td>Degree of stenosis</td>
<td>93.6%</td>
<td>87.8%</td>
<td>0.17</td>
<td>NS</td>
</tr>
<tr>
<td>Baseline systolic BP</td>
<td>148</td>
<td>144</td>
<td>0.44</td>
<td>NS</td>
</tr>
<tr>
<td>Contralateral stenosis</td>
<td>53.4%</td>
<td>50.3%</td>
<td>0.77</td>
<td>NS</td>
</tr>
</tbody>
</table>

Maas et al, J of Neurol, Neurosurg & Psych. 84(5):569-72, 2013 May.
Other studies suggest additional patient factors in the development of CHS

- Diabetes
- Long standing uncontrolled HTN
- Age > 75
- Recent carotid procedure within the past 3 months
- High-grade ipsilateral and contralateral stenosis
- Female sex
- Vascular malformations
- Cerebrovascular reactivity
- Symptomatic status
  - Higher the degree of neuro deficit the higher the risk
# Postoperative complications after CEA with selective shunting

<table>
<thead>
<tr>
<th>Postoperative complications</th>
<th>Group A (n = 59), No. (%)</th>
<th>Group B (n = 1036), No. (%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major stroke</td>
<td>2 (3.4)</td>
<td>10 (1.0)</td>
<td>.130 (NS)</td>
</tr>
<tr>
<td>Minor stroke</td>
<td>0 (0.0)</td>
<td>6 (0.6)</td>
<td>1.0 (NS)</td>
</tr>
<tr>
<td>Mortality</td>
<td>2 (3.4)</td>
<td>6 (0.6)</td>
<td>.63 (NS)</td>
</tr>
<tr>
<td>Seizures</td>
<td>3 (5.1)</td>
<td>0 (0.0)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>MI</td>
<td>0 (0.0)</td>
<td>2 (0.2)</td>
<td>1.0 (NS)</td>
</tr>
<tr>
<td>Permanent cranial nerve palsy</td>
<td>0 (0.0)</td>
<td>3 (0.3)</td>
<td>1.0 (NS)</td>
</tr>
<tr>
<td>Hematoma requiring re-exploration</td>
<td>0 (0.0)</td>
<td>7 (0.7)</td>
<td>1.0 (NS)</td>
</tr>
</tbody>
</table>

Group A treated < 8 Weeks after symptoms of TIA or Stroke
Group B treated > 8 Weeks after symptoms or for asymptomatic disease

Diagnosis of CHS

- **Clinical symptoms**
  - Headache
    - Migraine like
  - Poorly defined neurologic symptoms
    - Non lateralizing
    - Waxing and waning
  - Hypertension
    - Non specific but always present

- **Imaging**
  - CT
    - Edema, bleeding
  - MRI
    - Diffusion and perfusion weighted images
    - Can show relative perfusion
  - CT SPECT
    - Flow imaging
  - TCD
    - Can be quantitative but largely shows trends in cerebral flow

- **EEG**
  - If seizures suspected
62 year-old man 10 days post right CEA
Mild left hemiparesis, headache, blurred vision in the right eye

Cranial MRI
A) white matter edema
B) local hemorrhage
Fundus exam of right eye showing new retinal hemorrhages
Perfusion Imaging

69 year old male with confusion one day post CEA
Hypertension to the 160s systolic
CT perfusion performed

A) left middle cerebral artery distribution increased relative cerebral blood flow
B) increased relative cerebral blood volume
C) decreased mean transit time
D) Non-contrast CT shows no hemorrhage or edema
C-ARM flat detector measurement of cerebral blood flow

- On the table assessment of cerebral perfusion
- Neuro parenchymal blood volume (PBV) system
  
  Syngo Neuro PBV IR, Siemens Medical Solutions, Erlangen, Germany

- Sensitivity and specificity to predict CHS not well defined
- May prove to be useful in some high risk cases that one would expect to have hyperperfusion

Treatment

• Mainstay of therapy ---- Control BP
  • Systolic BP less than 120 to 140 mm Hg

• Beta-Blockers and clonidine
  • effectively decrease cerebral perfusion

• Ca+ channel blockers and nitrates should be avoided
  • can cause cerebral vasodilatation

• Anticonvulsant if seizures present
  • Prophylactic treatment not indicated
Outcomes

• Early treatment prevents most morbidity/mortality

• Neurologic findings resolve
  • Unless there is intracranial hemorrhage

• Some evidence of cognitive decline persisting
  • Despite lack of ICH or other focal findings
Suspect CHS with ill defined neurologic events post carotid revascularization

- Need high index of suspicion
- Especially in patients with higher risk
  - Symptomatic vs asymptomatic
  - Stroke vs TIA
  - Emergent vs elective treatment
    - Early treatment of symptomatic patients
- No shunt vs shunt
- CEA vs stent
- **HYPERTENSION VS NORMOTENSION**
  - Key to risk and key to treatment
Save the Date

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