What did I learn from my last case of Cerebral Hyperperfusion Syndrome?

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Disclosure

Speaker name: Gary Ansel, MD

I have the following potential conflicts of interest to report:

☐ Employment in industry
☐ Stockholder of a healthcare company
☐ Owner of a healthcare company
☒ Other(s)

Royalties: Cook Medical,

☐ I do not have any potential conflict of interest
Great Case Bad Outcome: CAS

• 72 year old patient
• Hx of CAD, HTN, DM
  – BP meds = Beta blocker, Ca\(^+\) blocker and diuretic
• Known chronic occlusion of Right Carotid artery
• Now presents with intermittent near syncope and right arm weakness
• BP on admission 185/98
Baseline Angigram of Lt ICA
Decision to Treat With Carotid Stent
Case Details

• Distal protection utilized
• Came to the procedural unit with some continued hypertension 180/90 (BP meds held)
• Post stent did not drop BP or become bradycardic
• BP controlled with IV NTG with goal < 160mmHg
• Placed back on home meds and by next afternoon discharged with BP of 152/85
Post Discharge Course

• Called into the clinic with complaint of sore neck and mild to moderate headache
• Instructed by staff to check for neck swelling and to acetaminophen ok to use for headache
• Third day became unresponsive and squad called
• Patient found to have intracranial hemorrhage neurosurgery consulted but family decided to not pursue heroics. Pt died next day.
CT Scan 72 hours post carotid stent
So What Did I Learn
Hyperperfusion syndrome

- Failure of normal cerebral autoregulation, secondary to long-standing changes in perfusion pressure

- Maximal dilation of cerebral arterioles for long periods of time causes loss of cerebral blood flow autoregulation in areas of chronically underperfused brain tissue and can result in hemorrhage and/or edema
Hyperperfusion Syndrome

May be acute
May be delayed
Hyperperfusion Syndrome and CAS

• Hyperperfusion: sudden increase CBF after CEA/CAS usually >100% (incidence approx 1.9%)*

• Syndrome occurs also in the presence of relative hyperperfusion of the ipsilateral hemisphere, ranging from 20 to 44% increased CBF (incidence 0.37%)*

• CT Perfusion and TCD have been used to diagnose

Hyperperfusion syndrome

• Risk factors for hyperperfusion syndrome:
  - Sudden increased perfusion in a chronically ischemic hemisphere
  - Severe stenosis or bilateral carotid artery stenosis
  - Previous stroke/ischemia
  - Periprocedural/long standing hypertension
  - Poor collateral blood supply
  - Reduced preoperative cerebrovascular reactivity
Hyperperfusion syndrome

- Risks factors for intracranial hemorrhage once hyperperfusion syndrome exists:
  - The use of anticoagulant & antiplatelet agents
  - Preprocedural cerebral infarctions
  - Preexisting hypertensive microangiopathy
  - Aggressive antiplatelet therapy (periprocedural use of glycoprotein IIb/IIIa inhibitors)
Clinical presentation of Hyperperfusion Syndrome

- seizures in 36%
- hemiparesis in 31%
- both in 33% of patients
- Sever HTN at onset 81%
- Hours to days

Bouri et al. Eur J Vasc Endovasc Surg 2011;41;229-37
Meta analysis of Cerebral Hyperperfusion CEA

• Results: 36 studies were identified
  – CEA:
    • incidence of severe hypertension 19%
    • cerebral hyperperfusion 1%
    • ICH 0.5%
    • mean systolic blood pressure of patients, who went on to develop cerebral hyperperfusion syndrome, was 164 mmHg
    • cumulative incidence of cases rose appreciably above a postoperative systolic blood pressure of 150 mmHg.
Meta analysis of Cerebral Hyperperfusion CEA

• Results: 36 studies were identified
  – CEA:
    • The incidence of cerebral hyperperfusion in the first week was 92% with a median time to presentation of 5 days.
Control Of BP is Essential

- BP control
  - Reduced odds when BP 140-160
  - No published cases of with BP < 135
  - Theoretically Labetolol and clonidine optimal
  - Vasodilators such as NTG and Nitropresside may worsen already profound autoregulation problem

Bouri et al. Eur J Vasc Endovasc Surg 2011;41;229-37
Anti-hypertensives to Avoid

• calcium channel blockers
• sodium nitroprusside
• Nitrates
• angiotensin II inhibitors

Faroq et al Exp and Trans Stroke Med 2016:8:7
Anti-hypertensives to Consider

- Beta Blockers
- Mixed alpha-adrenergics/beta blocker (labetolol)
- Central actingssuch as Clonidine

Faroq et al Exp and Trans Stroke Med 2016:8:7
## Key Factor prevention and Treatment Summary

<table>
<thead>
<tr>
<th>Treatment modality</th>
<th>Comment</th>
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<tbody>
<tr>
<td>Blood pressure control</td>
<td>Strict control of blood pressure is recommended. Lower blood pressure even in normotensive patients. There are no definite guidelines about blood pressure parameters and management should be individualized. Avoid medications which have vasodilatory effect such as calcium channel blockers. Labetalol and clonidine are better options to treat elevated blood pressure in these patients.</td>
</tr>
<tr>
<td>Timing of carotid surgery</td>
<td>Carotid endarterectomy or stenting should be done within 2 weeks of transient ischemic attack or stroke. Patient is at risk of cerebral hyperperfusion syndrome if they underwent contralateral carotid endarterectomy in past 3 months.</td>
</tr>
<tr>
<td>Type of anesthetic</td>
<td>High doses of volatile halogenated hydrocarbon anesthetics may lead to cerebral hyperperfusion syndrome. Isoflurane is safer to use in these patients but can cause complications at higher doses. Nitrous oxide is also safe but should not be used with isoflurane. Propofol normalizes cerebral blood flow and is a safe option.</td>
</tr>
<tr>
<td>Use of anti-epileptic medications</td>
<td>Prophylactic use of an anti-epileptic drug is not recommended. If patient has lateralized epileptiform discharges or a clinically manifest seizure spell, an anti-epileptic drug may be administered.</td>
</tr>
<tr>
<td>Use of hypertonic saline and mannitol</td>
<td>The evidence about the use of hypertonic saline and mannitol is not strong but may be administered if the patient has cerebral edema. Corticosteroids and barbiturates are not indicated in most cases. Hyperventilation and sedation may be administered if the patient has cerebral edema.</td>
</tr>
</tbody>
</table>
Strategic Flow Chart

Inpatient strategy
- Hourly blood pressure monitoring
- Record headache, vomiting, GCS
- Aim for sBP 140mmHg-160mmHg OR pre-operative sBP if lower

Asymptomatic hypertension
- 160-179mmHg – oral antihypertensive(s)
  - Switch to intravenous if failure to reduce BP after 2 hours or symptoms develop
- ≥180mmHg – intravenous antihypertensive(s)
  - Reassess BP and symptoms hourly until BP is controlled

Symptomatic hypertension
- Headache, GCS<15, seizure
- Intravenous anti-hypertensive(s) aiming for sBP ≤140mmHg (or higher if symptoms resolve)
  - Urgent cerebral and carotid imaging
  - Consider CT Perfusion or TCD monitoring
  - Neurology opinion if oedema or ICH
  - Admit to HDU / CCU for intra-arterial BP monitoring
  - Cardiology opinion for investigation and management of BP

Discharge strategy
- Discharge when BP stable for 24 hours and sBP<160mmHg
- Home BP monitor for 1 week if intravenous antihypertensives used or labile BP
- Return to vascular unit if sBP ≥ 160mmHg or headache, vomiting or drowsy

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Summary
Better to Prevent Than Try to Treat
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