“Low Flow” TIA/Stroke Events

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Case

A 65 year old man, with a past history of arterial hypertension and smoking, presents after suffering a 15 minute episode of left-sided weakness.
☐ Transient thrombosis?
☐ Embolism with rapid lysis?
☐ “Low flow” event?
Cerebral Ischemia

- Focal symptomatology
  - TIA or Ischemic Stroke (IS)
    - Is there cerebrovascular disease?
- Diffuse symptomatology
  - Syncope
    - Abrupt decrease in cerebral perfusion
Case

69 yo man presented to the ED with a 30-m episode of R-sided weakness
- Transient thrombosis?
- Embolism with rapid lysis?
- "Low flow" event?

- Transient event in the setting of marginal LMCA’LICA perfusion
Case

- 44 yo woman experienced brief episodes of RUE “limb shaking” weakness, typically when she would quickly get up from a sitting or lying position
- PMHx included increased BMI, dyslipidemia, and severe, long-standing DM
Exam

- Supine: BP 144/86, RRR at 72
- Standing: BP 110/65, RRR at 66
- Moderate distal neuropathic findings
- o/w normal
Case: Summary

- Young diabetic patient with autonomic neuropathy and symptomatic orthostasis
- "Low flow" TIAs referable to her LMCA in the setting of a severe intracranial LICA lesion
- TIAs have resolved post-PTA/stent
- Cerebral vasocapacitance studied w/ PET during room air and then after induced hypercapnia
- 32 pts w/ unilateral TIAs
  - N=08 w/ “low flow” TIAs
  - N=24 w/ little/no evidence of “low flow” events
“Low flow” subset defined by
- Exertional, positional, orthostatic, or dysrhythmic induction of SSx
- Multiple events (4.5 v 20, on ave)
- Shorter events (2.5 v 20-m, on ave)
- Arm-predominant “limb shaking” SSx
- Continued events in spite of anti-thrombotic therapies
TIA/IS Pathophysiology

- Embolic theory
  - What is the source of embolic material?
- Thrombotic theory
  - Why did the vessel close?
- "Low flow" theory
  - What caused the diminished flow?
  - Is one artery narrower than the rest?
Case

- 76 yo was found down and was noted to have severe L-sided weakness and L-sided neglect
- LKW \(~48\text{-h before presentation to the ED}\)
Artery-to-artery embolization from RICA ulcerated athero-stenosis to RMCA
IS causative mechanisms
Borderzone (Watershed) Infarcts

- **Definition**
  - Ischemic lesions situated along the “borderzones” between the territories of two major arteries

- **Typical locations**
  - ACA-MCA
  - MCA-PCA
  - Deep irrigation zone of MCA
  - Between the major cerebellar arteries
10% of all brain infarctions
May be bland or hemorrhagic or mixed
Mechanisms include
- Systemic hypotension
- w/ occlusion of ICA
- Due to microemboli
- w/ cerebral thromboangiitis obliterans
- Sharp drop in systemic BP
  - Most frequent cause of borderzone infarction (BZI)
  - Reduction of CBF most severe in terminal areas of vascular beds
- ACA-MCA most common location
Case

- 55 yo presented in transfer 2-d after the onset of L-sided weakness
- Prior Hx of brief episodes of L-sided limb-shaking weakness *when getting up quickly*
- PMHx included diabetic autonomic insufficiency with severe orthostatic hypotension
“Low flow” TIAs followed by “low flow” stroke in the deep irrigation zone of and in the setting of an acute RICA occlusion
BZI and ICA Occlusion

- Historically
  - BZI in the setting of ICAO considered due to reduced blood flow

- Alternatively or in addition
  - Vascular occlusions could be caused by microemboli from the occluding thrombus before the lumen becomes completely blocked
Borderzone (Watershed) Stroke

- The newer dogma
  - Stroke 2005;36:567-577
Embolism and hypoperfusion play synergistic role in BZI.

Small embolic material prone to lodge in distal field arterioles would be more likely to result in cortical micro-infarcts when chronic hypoperfusion prevails.
Infarcts and ICA disease

- Cortical watershed infarcts
  - CWIs
- Internal watershed infarcts
  - IWIs

Autopsy series
- 10% of all brain infarcts are BZI

Imaging series
- 19 to 64% of lesions may be BZI
Susceptibility of the watershed areas thought to result from distal field anatomy, where perfusion is lowest.

However, cortical distal fields may not always correspond to the ‘classic’ watershed areas in situations where:

- A ‘shift’ has occurred because of additional hypoplasia or stenosis of proximal ACA, PCA, or MCA.
Markers of hemodynamic failure

- Occurrence of syncope at stroke onset
- Fluctuating hand-only weakness
- Limb-shaking events

BZI distal to ICA disease more likely w/ non-competent circle of Willis

Microemboli and BZI

- Arising from unstable carotid plaques
- Arising from “stump” of occluded ICA
CWI or CBZI

Most common type

Anterior CWI or CBZI

- ACA-MCA
- Superiorly as a linear strip on the superior convexity close to the interhemispheric fissure
- Or a thin fronto-parasagittal wedge extending from the anterior horn of the lateral ventricle to the frontal cortex
Posterior CWI or CBZI
- MCA-PCA
- Parieto-temporo-occipital wedge extending from the occipital horn of the lateral ventricle to the parieto-occipital cortex
IWI or IBZI
- Can affect the corona radiata
- Can affect the territories between the deep and superficial perforators of the MCA
- Can affect the territories between the perforators of the ACA and MCA, in the centrum semiovale area
IWI or IBZI
- Confluent IWI are large cigar-shaped infarcts along the lateral ventricle
- Partial IWI as a single lesion or in a chain-like ("rosary-like") pattern

There, of course, is considerable inter- and intra-individual variability when dealing w/ BZI
Discussion

- Literature on cerebral perfusion in those w/ disease of the ICA
  - Evidence in favor of both hemodynamic and embolic mechanisms for "low flow" stroke
  - The mere presence of hemodynamic impairment on testing DOES NOT necessarily mean that it is the cause of a BZI
IWI or IBZI

- “Rosary-like” pattern of infarction
- Hemodynamic
  - Given length and diameter of perforators and low density of arterioles at this level, CPP is likely to be lower in this terminal area than in overlying cortex
- Reduced CPP may not have the same influence on all superficial and deep perforators
IWI or IBZI

- “Confluent” pattern of infarction
  - Hemodynamic
  - May be caused by longer lasting impairments of cerebral perfusion

- May predict impending cortical stroke
CWI or CBZI

- Anterior location
  - Hemodynamic
- Anterior cortical and internal
  - Hemodynamic
- The MCA and ACA both are only supplied by the ICA, so a critical ICA stenosis or occlusion will exert maximal effect on anterior watershed zone especially if collaterals poor or process of occlusion rapid
Some newer studies

- Isolated CWI or CBZI
  - Embolic causes evident...
Demonstration of hemodynamic failure at baseline DOES NOT prove that any subsequent stroke is purely flow-mediated.

Patients w/ impaired perfusion reserve may be more likely to have stagnant flow, which in turn increases the risk of thrombus formation and then artery-to-artery embolism.
Marginally perfused areas, like in the watershed zones, may be more susceptible to the effect of emboli because of the already stressed or exhausted vascular reserve or even partly exhausted OEF reserve.
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<thead>
<tr>
<th></th>
<th>ICA Territory</th>
<th>VB Territory</th>
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<tbody>
<tr>
<td>Embolic</td>
<td>~80%</td>
<td>~50%</td>
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<tr>
<td>“Low flow”</td>
<td>~20%</td>
<td>~50%</td>
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Stroke Mechanisms, In General

- >70% are ischemic
  - 40% are related to cerebral thrombosis
  - 30% are related to cerebral embolism
- ~20% are related to cerebral hemorrhage
- 05 to 10% may be stroke “mimics”