Transient Ischemic Attack
(and the mimics)

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Disclosures

• None.
What is a **TIA**?

Why limit it to a time point?

TIA is pathophysiological = brain angina.
Transient Ischemic Attack

- Definition now a tissue based definition (new); not time
- AHA/ASA 2009 definition
  - “Transient ischemic attack (TIA): a transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction.”
  - MRI
  - ‘Acute neurovascular syndrome’ if no imaging available
- TIA should be thought of as equivalent to crushing retrosternal chest pain of unstable angina
  - Brain parenchyma devoid of nociceptors
  - Other major clue is loss of cerebral function (vision, speech, motor, sensory); so need to know common “stroke syndromes,” “mimics” and some neuroanatomy basics

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Transient Ischemic Attack

• Diagnostic certainty will depend on the extent of evaluation the individual patient receives.
  • This concept is not unique to brain ischemia

• Brain DWI MR imaging currently and serum diagnostic studies likely in the future (“brain troponins”) increase diagnostic certainty

• No longer using terms like RIND
  • reversible ischemic neurological deficit
TIA and Stroke Risk: Frontloaded

Epidemiology

• Dr. Charles Miller Fisher
  • House officers and students learn neurology “stroke by stroke”

• >50% of neurological admissions

• 50,000 new /year in Canada

• 3\textsuperscript{rd} most important cause of death
  • after heart disease and cancer

• The \textbf{single most} important cause of adult disability
  • 30% of survivors require daily assistance
What are stroke / TIA Risk Factors?
Risk Factors

**Non-modifiable**
- Age
- Ethnicity: Black, Asians
- Male gender
- Migraine *(with aura)*
- **Family history**
  - Stroke in first degree relative
- **Genetics**
  - Sickle cell
  - Dyslipoproteinemia
  - Vasculopathies
  - Cardiomyopathies
  - Mitochondrial diseases

**Modifiable**
- **HTN** – 3-4 x
- **DM** – 2-4 x with HTN
- **SMOKING** – 2-3 x
- **Hyperlipidemia**
- **CAD**
- **Afib** – 5-6 x
- **Stroke, TIA, Carotid stenosis**
- OSA
- EtOH
- Vasoactive drugs (cocaine)

Medical conditions $\rightarrow$ Hypercoagulable states (hyperviscosity syndromes) i.e. Malignancy
What is a “rational” approach to TIA / Stroke Care

1. Recognition
2. Syndromes
3. Neuroanatomy
Recognition

• Which of the following is likely to represent and acute CVA?
  A. Sudden fever and confusion with inability to count months year backwards, count to 10 or repeat more than 4 digits. Non-focal exam.
  B. Right sided headache with phono and photophobia, nausea and vomiting with + Romberg. Preceded by 15 minutes of shimmering lights.
  C. 23 yo M out partying, ataxic gait, dysarthria and vomiting
  D. Acute R facial droop (not sparing forehead), preceded by retro-auricular pain and vesicles in inner ear.
  E. Acute R gaze deviation, L hemiplegia and hemi-sensory neglect
Recognition

• Which of the following is likely to represent an acute CVA?

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  Right MCA territorial infarct  
  (clinical can hint at ischemic vs hemorrhagic)
“Classic” Clinical Presentation

• **Sudden**
• **Focal** weakness, language impairment, gaze deviation, hemianopia
• **Neuroanatomically based**
• **New** (and not old deficit from prior CNS insult)
  • Search for “warning symptoms” in week prior
    • Amaurosis fugax
      • A dark veil (or window shade) dropping down
      • Painless
      • Fully reversible over minute
    • Sentinel TIA’s in same/differing territories
TIA/Stroke Mimics: Paroxysmal neurological symptoms

- **Migraines with aura**
  - Visual, somatosensory, dysphasic speech, motor, brainstem – craniobulbar complaints

- **Seizures (focal)**
  - Motor (limb flinging TIA)
  - Sensory (numbness in anatomical region with a lesion, ie. Sensory seizures from stroke to sensory cortex)
  - Post ictal Todd’s paralysis (motor, eye-field)

- **Somatization**
  - Sensory

- **Demyelinating disease**
  - Should not be on the DDx as brief symptoms and usually wrong demographic

- **Structural**
  - Mass-lesion, SDH, ICH

- **Metabolic**
  - Hypo/hyperglycemia
  - Hypo/hypercalemia
  - Hypo/hypernatremia

- **Toxic**
  - Toxidromes
    - History and Rx list clarify

CMAJ 2004;170(7):1134-7
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### Visual symptoms of migraine aura vs transient ischemic attack (TIA)

<table>
<thead>
<tr>
<th>MIGRAINE AURA</th>
<th>TIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>15–30 minutes</td>
<td>3–10 minutes</td>
</tr>
<tr>
<td>Dynamic, bright, multicolored</td>
<td>Static, dark</td>
</tr>
<tr>
<td>Forms geometric patterns</td>
<td>Dimming of vision</td>
</tr>
</tbody>
</table>
Spend the time here...

• Really push the patient on what they mean
• Have them draw out any visual phenomena
• Also slow down and clarify the sensory disturbance
  • Migratory and fleeting?
  • Migratory and “march-like”?
  • Hemi-body exactly to midline?!  
  • Complete anesthesia to all modalities?
Top 7 symptoms unlikely to be TIA

1. Postural **dizziness** alone
2. Tingling of **all 4 extremities**
3. **Syncopal** events
4. Momentary word finding trouble that is not new
5. Brief positional and **recurrent numbness** of one limb
6. **Scintillating or flashing** visual disturbances
7. Almost anything with **hyperventilation** or **chest pain**
Top 6 symptoms likely to be TIA

1. Vertigo only if present with brainstem symptoms
2. Hemibody numbness
3. Double vision, crossed numbness or weakness, slurred speech, ataxia of gait
4. Monocular or hemifield visual loss (not blurring of entire visual field)
5. Speech disturbance for a defined period of time (definite dysarthria, muteness or marked word finding difficulty, paraphasic speech)
6. Hemibody weakness
Basic Neuroanatomical Concepts

• Anterior vs. posterior circulation
• Cortical Vs Subcortical
• Side independent [localizable]
  • Motor, sensory, visual, auditory, frontal eye fields (gaze deviation / preference)
• Side Specific [localizable]
  • Left (dominant) hemisphere
    • Language
  • Right hemisphere
    • Neglect / visuospatial
  • Mathematics, L vs. R, finger agnosia
  • Apraxia
  • Memory
What is the Recommended TIA Work-Up?

Should be the default when you are not certain...

i.e, risk factors (mod or non mod.), language barrier etc.
Canadian Best Practice Recommendations for Stroke Care: Summary
(updated 2015)

Superb reference and website for all health care professionals.
Evidence based and thoroughly referenced.
Regularly updated.

CMAJ 2008;179(12 SUPPL):S1-S25

http://www.strokebestpractices.ca/
Focused Evaluation

• What is the critical information to garner from a TIA evaluation

1. Age, sex
2. PmHx: see risk factors slide
3. Symptoms: only motor, sensory, speech or both
4. BP and ECG
5. Total duration of symptoms (5 min?, 25 min? 24 hrs?)
6. When? (today, yesterday, 6 months ago?)
What is an expedited work-up? (AHA, CSC)

• **Neuroimaging**
  - CT (C-)
  - MRI with DWI

• **Vascular imaging (extracranial & intracranial)**
  - CUS
  - CTA (results comparable to CUS and MRA).
  - MRA (2D TOF) or MRA with contrast
  - TCD (microembolic signals)
  - Conventional angiography

• **Cardiac and ‘other’ testing**
  - ECG (r/o afib, SSS, arrhythmia, LVH)
  - TTE, TEE
  - Holter +/- Loop Recorder

• **Routine bloodwork**
  - CBC, SMA7, Coags, E+, FLP, CK, LFT’s
  - Hypercoagulable work-up depending on age / cryptogenicity of stroke-TIA

*local strengths in that expertise in vascular imaging dictate what to select as first line (also other medical conditions i.e. PM or renal failure)*
Canadian Stroke Strategy

• Carotid imaging should be performed within 24 hours of a carotid territory transient ischemic attack or non disabling ischemic stroke (if not done as part of the original assessment) unless the patient is clearly not a candidate for carotid endarterectomy [Evidence Level B].
Echocardiography (AHA, CSC)

• Should you hold the patient overnight for an echo?
  • “TIAs require urgent evaluation, but there is little evidence that early echocardiographic evaluation has a higher yield.”

• TEE is more sensitive than TTE for atheroma of the aortic arch and abnormalities of the interatrial septum (eg, atrial septal aneurysm, PFO, atrial septal defect), atrial thrombi, and valvular disease.

• Holter monitoring is abnormal in a minority of unselected patients with TIA.
  • The longer you can obtain, higher yield
  • Consider loop recorder, as technology permits / evolves
How do we triage TIA’s into “HIGH” vs. “LOW” risk?

And does it matter?
Canadian Best Practice: Timing of Initial Assessment

• Patients presenting to GP/community physician \( \leq 1 \) week of event should have:
  
  • *Immediate* clinical evaluation and investigations to establish the diagnosis, r/o stroke mimics, and develop a management plan [Evidence Level B].
  
  • Patients who *cannot* be evaluated as an outpatient within 24h from clinical presentation should be transported to an ED that has access to *neurovascular* imaging facilities and *stroke expertise* [Evidence Level B].
Canadian Best Practice: Timing of Initial Assessment

• Patients presenting to a GP/community physician > 1 week following a suspected TIA or non-disabling ischemic CVA should be seen by a stroke specialist on a less urgent basis, generally within one month of presentation [Evidence Level B].

• Out with the ABCD2 score...
Treatment: *Always err on side* of caution if unsure

- Ensure patient on:
  1. Anti-platelet agent
     - start on *all* potential ischemic phenomena
     - discuss with neurologist about continuing at follow-up
  2. Statin (LDL <2.0mmol/L or 50% baseline value)
  3. +/- BP control, once status of carotid arteries and cerebrovascular tree known

- Smoking cessation
  - *Best* moment to intervene when you inform patient they may have had a minor stroke

- Education (**FAST**):
  - Never let patient leave the office without reviewing this
  - Emphasis on T
When Stroke Strikes, Act F.A.S.T.

**FACE**
Smile.
Does one side of the face droop?

**ARMS**
Raise both arms.
Does one arm drift downward?

**SPEECH**
Repeat a sentence.
Are they able to speak clearly? Can they repeat the sentence?

**TIME**
Time is critical.
Call 911. Get to the hospital immediately. Brain cells are dying. Every Minute Counts!
SUMMARY

Emergency Investigations

CT head (non-contrast)
• MRI (ideal)
ECG and HOLTER or Loop Recorder
CTA NECK and COW/VB circulation + CAROTID DUPLEX
ECHOCARDIOGRAM (TTE or TEE depending on situation)
CXR
CHECK fasting lipids, glucose, HbA1C, CBC, SMA7, LFT, CK

Well defined indications/contra-indications
• IV-rTA
• Mechanical thrombectomy
SUMMARY

Emergency / Urgent Measures

IV-TPA +/- mechanical thrombectomy

*Anti-platelet*

*Statin*

*Anti-HTN Rx*

If no high grade large vessel stenosis with stuttering hemodynamics

  *Indicative of “at risk” parenchyma*

? Carotid intervention (2 weeks)

? Atrial fibrillation (3-14d)

Secondary Prevention

*Anti-platelet*

*Statin*

*Anti-HTN Rx (s)*

*Smoking cessation*

*Exercise*

*Diet (balanced and salt intake)*

*Weight BMI (18.5-24.9)*

*ETOH*

*Hormone (E) removal / replacement*

*Screen and Tx OSA*

*Malignancy search where appropriate*

When in doubt *call* your friendly neighbourhood neurologist for advice and guidance ☺
Bottom Line

• Recognize the severity **TIA or minor stroke**
  • Maximize medical therapy *wherever* you see the patient
  • *Treat* what is treatable / or correctible in the acute phase, do not “consult” and forget about it.
    • May work at an academic center, will not work in a community practice.
  • Ensure appropriate (and timely) follow-up
  • Be fluent with the major organizational guidelines (Canadian Best Practice Guidelines and Uptodate.com)
    • Try and standardize routine stroke care based on the evidence (EBM)

• **Secondary prevention** is of utmost importance
Thank you for your attention.

Questions?