Hyperacute stroke and TIA management in the L&ACGH Emergency Department

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Outline

- Ischemic Stroke Syndromes
- Bedside examination (linked video)
- Imaging
 - How to read a plain CT head
 - Ischemic vs hemorrhagic stroke
- Who should be managed in LACGH vs KGH?
 - TIA/minor non-disabling stroke
 - Acute, subacute disabling stroke
 - Hemorrhage (parenchymal, subdural, subarachnoid)
- Stroke Mimics

Ischemic Stroke Syndromes

Stroke Syndrome List

- Anterior circulation stroke:
 - Middle cerebral artery
 - Anterior cerebral artery
- Posterior circulation stroke:
 - **Posterior cerebral artery** (occipital lobe, thalamus, medial temporal lobe)
 - Brainstem (midbrain, pons, medulla)
 - Cerebellum
- Five common **lacunar** stroke syndromes
 - Pure motor stroke
 - Pure sensory stroke
 - Sensorimotor stroke
 - Ataxic hemiparesis
 - Clumsy hand-dysarthria





Anterior Circulation Stroke

- MCA and/or ACA
- Occlusion of the ICA can result in ischemia in both MCA and ACA territory simultaneously



Middle cerebral artery



- About two-thirds of all ischemic stroke occurs in the middle cerebral artery territory
- MCA stroke can involve the frontal, temporal, and parietal lobes
- MCA stroke can also involve the basal ganglia through the *lenticulostriate arteries*

 The MCA covers a large territory shown in blue on this CT scan image taken at the basal ganglionic level



MCA covers a large portion of the hemisphere



MCA stroke syndromes

- Left hemisphere (ie, dominant)
- Right hemiparesis
- Right-sided sensory loss
- Right homonymous hemianopia
- Dysarthria
- Aphasia

- Right hemisphere (ie, nondominant)
- Left hemiparesis
- Left-sided sensory loss
- Left homonymous hemianopia
- Dysarthria
- Neglect of the left side of environment

Anterior cerebral artery



ACA covers the medial portion of the brain



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ACA stroke syndrome

- How might this person present?
- What do you think you would find on the exam?



ACA stroke syndrome

- Contralateral leg paresis > arm paresis
- Or, bilateral leg weakness if both ACAs are involved
- Abulia, disinhibition, executive dysfunction
- In some cases, akinetic mutism if bilateral caudate head infarction

Posterior Circulation

- This includes:
 - Vertebral arteries
 - Posterior and anterior inferior cerebellar artery
 - Basilar artery
 - Pontine arteries
 - Superior
 cerebellar artery
 - Posterior cerebral artery



Left PCA infarction on CT



This is a thrombus in the left PCA

PCA stroke syndromes

- The most common syndromes involve the occipital lobe, the medial temporal lobe or the thalamus
- Occipital lobe:
 - Contralateral homonymous hemianopia
 - Cortical blindness (bilateral lesions)
- Medial temporal lobe:
 - Deficits in long-term and short-term memory
 - Behaviour alteration (agitation, anger, paranoia)

PCA perfuses three main areas of the brain



PCA stroke syndromes, cont'd

- Thalamic infarct
 - Contralateral sensory loss
 - Aphasia (if dominant side involvement)
 - Executive dysfunction
 - Decreased level of consciousness
 - Memory impairment





www.medicosite.com

Brainstem stroke syndromes

- Some of the clinical features seen are:
 - <u>Crossed sensory findings (e.g. ipsilateral face</u> and contralateral body numbress)
 - <u>Crossed motor findings (ipsilateral face,</u> <u>contralateral body)</u>

- Gaze-evoked nystagmus

Other findings in brainstem stroke

- Ataxia and vertigo, limb dysmetria
- Diplopia and eye movement abnormalities
- Dysarthria, dysphagia
- Tongue deviation
- Deafness (very rare)
- Locked-in syndrome (can't move any limb, can't speak, can sometimes blink

Midbrain stroke

- Eyelid or eye movement problem on one side, limb weakness or clumsiness on the other side.
- Ipsilateral 3rd nerve palsy
- Contralateral hemiparesis of the arm and leg, sometimes with hemiplegia of the face
- Contralateral hemiataxia







Midbrain

Pontine stroke

- Face weakness or eye movement impairment on one side, limb weakness on the other side
- Ipsilateral signs:
 - Horner's syndrome
 - 6th or 7th nerve palsy (diplopia, whole side of face is weak)
 - Hearing loss (rare)
 - Loss of pain and temperature sense
- Contralateral signs:
 - Weakness in leg and arm
 - Loss of sensation in arm and leg
- Nystagmus, nausea





Horner's syndrome

- Ptosis
- Miosis
- Anhydrosis



Medullary stroke

- Persistent nystagmus with face numbness on one side, limb numbness on the other side
- Ipsilateral signs:
 - Tongue weakness
 - Sensory loss in face
 - Horner's syndrome
 - Ataxia
 - Palate weakness (dysphagia)
- Contralateral signs:
 - Weakness, sensory loss in arm and leg
- Nausea, nystagmus, dysphagia, dysarthria





Cerebellar stroke

- Ataxia, vertigo, nystagmus, nausea, vomiting, dysarthria
- Often headache and nystagmus
- Can also have rapid deterioration in level of consciousness



Cerebellar infarction

- Infarction causes edema resulting in mass effect, herniation and compression of the fourth ventricle
- This can lead to rapid deterioration in level of consciousness
- Surgical decompression is often necessary in these circumstances



- Pure motor stroke usually arises from infarction in the posterior limb of the internal capsule; course is often stuttering over hours to days:

• **Pure sensory stroke** usually arises from thalamic infarction



- Sensorimotor stroke can arise from infarcts at the junction between the thalamus and the internal capsule
- As the name implies, the symptoms consist of weakness and sensory loss with no visual field deficit, aphasia, neglect or other symptoms



- Ataxic hemiparesis often arises from infarction in the corona radiata
- Ataxia is unilateral and is in excess of the mild weakness found on exam



- Clumsy hand-dysarthria is caused by infarction in the pons, but can also occur in corona radiata and the internal capsule
- Contralateral facial weakness with dysarthria and dysphagia occurs with contralateral hand weakness/ataxia, and sometimes weakness in the arm or leg



Summary of Stroke Syndromes

- MCA stroke: hemiparesis, sensory loss, hemianopia, and either aphasia or neglect
- ACA stroke: leg weakness and executive dysfunction
- **PCA stroke**: hemianopia, pure sensory infarct (thalamus), memory impairment, decreased level of consciousness
- **Brainstem strokes**: crossed sensory or motor findings, nystagmus, ataxia, dysarthria, diplopia, vertigo, Horner's syndrome
- **Cerebellar strokes**: ataxia, nystagmus, vertigo, nausea, headache and rapid deterioration in consciousness
- Lacunar strokes: pure motor, pure sensory, sensorimotor, ataxic hemiparesis, clumsy hand-dysarthria

Neurological examination for stroke

- <u>https://www.youtube.com/watch?v=4ASM1mzKVGc</u>
- Can also go to the Stroke Network of Southeastern Ontario website: <u>https://www.strokenetworkseo.ca/best-practice-education/acute</u> and then click on the link for Assessments
Imaging (focused on CT)

Objectives:

- Recognize basic anatomical structures on a plain CT head
- Recognize acute thrombus in the MCA
- Recognize acute ischemic stroke
- Recognize acute intracranial hemorrhage

Reading a plain CT head: 6 slices

- One approach is to focus on the following levels on an axial CT:
 - 1. Medulla, Cerebellum, and Vertebral Arteries
 - 2. Pons, and Basilar Artery
 - 3. Midbrain, and Proximal Middle Cerebral Arteries
 - 4. Basal ganglia and Insula
 - 5. Corona radiata
 - 6. Centrum semiovale

















Recognizing acute thrombus



- Thrombus can have a variable appearance on CT depending on whether:
 - It is in an artery running parallel to the plane of the image or perpendicular
 - It is in a vein or artery

Thrombus in RMCA M1 segment





Thrombus in the LMCA M2 segment

10 W 90 : L 40



Venous thrombus in the superior sagittal sinus













Detecting early cerebral ischemia on CT scan

- Loss of grey-white differentiation
 - You may have to adjust the brightness and contrast (the "window width" and "window level")
- Loss of sulci
- Use the same system every time you look at a CT for possible acute stroke
 - For example, the Alberta Stroke Program Early CT Score (ASPECTS)

Alberta Stroke Program Early CT Score



C = caudate, L = lentiform, I = insula, IC = internal capsule

M1, M2, M3 = anterior, lateral, posterior MCA territory; M4 to M6 are above the lentiform nuclei

Right hemiparesis and aphasia: Where is the infarct?



Can you see the infarct using ASPECTS?



Case

 77 year old female with left hemiparesis, left homonymous hemianopia, left side sensory loss




































































Intracranial Hemorrhage

 http://radiopaedia.org/articles/intracranialhaemorrhage

Subarachnoid hemorrhage, acute



Lobar hemorrhage, acute



Intraventricular hemorrhage, acute



Epidural hemorrhage, acute



Subdural hematoma, acute



Subdural hematoma, chronic



Subdural hemorrhage, acute on chronic



Where do I send my patient?

Three issues affect the care pathway for the patient at LACGH

- Disabling vs non-disabling stroke presentation
- Time last known well
- Imaging
- Please also refer to the link:

https://www.strokenetworkseo.ca/best-practice-education/acute and click on Physician Guide for Stroke Transfers

Disabling stroke presentation

- If the patient can get to KHSC before 6 hours after time last known well then activate Acute Stroke Protocol and send to KHSC without scanning the patient
- If the patient can't get to KHSC before **6 hours**:
 - If ACT-FAST positive, i.e. possible EVT candidate, then activate ASP and send to KHSC
 - If not ACT-FAST positive then call Stroke service at KHSC to discuss transfer (not as ASP)
 - If in doubt, call Stroke service at KHSC

Non-disabling stroke or TIA presentation

- If within the first 48 hours of presentation then should do CT and CTA from aortic arch to circle of Willis before ED discharge
 - What if CT at LACGH is not available? In that case, please make arrangements for CT/CTA to be done at KGH as has been done in the past before LACGH acquired CT facilities.
- If more than 48 hours after presentation of symptoms, then do CT/CTA ideally within 24 hours
 - If CTA is not available, carotid Doppler is a reasonable second-line vascular imaging choice
 - Referral to Stroke Prevention Clinic

Non-disabling stroke or TIA, CT/CTA is normal

- Refer to KHSC Stroke Prevention Clinic
- If patient is on no antiplatelet agents, then start dual antiplatelet therapy:
 - Clopidogrel 300 mg initial dose Day 1, then 75 mg OD for an additional 20 days
 - ASA 160 mg initial dose Day 1, then ECASA 81 mg OD (indefinitely)
- If patient is already on one antiplatelet agent, then add a second agent for 21 days
Non-disabling stroke or TIA, CTA shows >50% internal carotid artery stenosis that is symptomatic

- CT head may or may not show infarction.
- For >50% symptomatic ICA stenosis, call Vascular Surgery.
 - Patient may be considered for urgent carotid endarterectomy
 - Some surgeons may prefer to hold clopidogrel if the patient is going to be taken urgently for CEA

Non-disabling stroke, intracranial hemorrhage

- For subdural or subarachnoid hemorrhage, call Neurosurgery first
- For parenchymal hemorrhage, call Stroke first
 - Most of these patients will either be followed up quickly in Stroke Prevention Clinic at KHSC or transferred to KGH for further workup by the Stroke Service

Stroke Mimics

Migraine

- The four most common aura symptoms in order of frequency:
 - Scintillating scotoma
 - Paresthesia or dysesthesia
 - Aphasia
 - Weakness
- The key feature on history is slow evolution of symptoms
- If in doubt, call Stroke Service for advice
- Stroke Prevention Clinic follow up is also reasonable if there is doubt about the diagnosis

Posterior Reversible Encephalopathy Syndrome (PRES)



Headache, confusion, seizure, visual impairment. Can sometimes appear to have aphasia, hemianopia.

By Rashmi Chawla, Daniel Smith and Paul E Marik - Near fatal posterior reversible encephalopathy syndrome complicating chronic liver failure and treated by induced hypothermia and dialysis: a case report. J Medical Case Reports 2009, 3:6623. doi: 10.1186/1752-1947-3-6623, CC BY 3.0,

https://commons.wikimedia.org/w/index.php?curid=6834 041

PRES

- Blood pressure is often high (sBP > 200) but patients can have only mild hypertension or normotension in special situations:
 - Immunocompromised
 - Recent administration of IVIG
 - Renal or liver disease
 - Eclampsia
 - SLE

JAMA Neurology | Original Investigation

Recrudescence of Deficits After Stroke Clinical and Imaging Phenotype, Triggers, and Risk Factors

Mehmet A. Topcuoglu, MD; Esen Saka, MD, PhD; Scott B. Silverman, MD; Lee H. Schwamm, MD; Aneesh B. Singhal, MD

JAMA Neurol. 2017;74(9):1048-1055. doi:10.1001/jamaneurol.2017.1668 Published online August 7, 2017.

- Transient worsening of post-stroke deficits or reemergence of strokerelated deficits in the setting of toxic, metabolic, or physiological derangements is common but making this diagnosis can be difficult because it usually depends on MRI to prove there is no new infarction
- Common triggers include: infection, hypotension, hyponatremia, fever, stress, insomnia, benzodiazepine use
- If not sure about the diagnosis, call Stroke Service on call to discuss the case

Other Stroke Mimics

- Hyperglycemia or hypoglycemia
- Seizure and post-ictal aphasia or Todd's paresis
- Drug intoxication
- Brain tumor
- Metabolic abnormalities
 - Hyponatremia (weakness, seizure, confusion), hypercalcemia (confusion, aphasia), hypomagnesemia (weakness, numbness, nystagmus, ataxia)
- CNS infections
 - HSV encephalitis
 - CNS abscess
 - Meningitis
- Multiple sclerosis

Final Word

- CT and CTA is great, but don't replace the value of history, exam and recognition of stroke syndromes and stroke mimics
- KHSC has Stroke Service on call 24/7 to answer any questions any time
 - Care pathways are in place for Acute Stroke and KHSC is available to handle any stroke inpatient need
- Please feel free to email me at: <u>Albert.Jin@kingstonhsc.ca</u>