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Seattle, WA
What will we cover today?

I. Introduction & Overview: Complex Stroke

II. The First 36 Hours of Care

III. Basic Brain Imaging

IV. Critical Transitions of Care:
   - admission to discharge and beyond

V. Complex Stroke Cases & Imaging
   - malignant MCA ischemic stroke
   - complications

v. Questions

1. Hemicraniectomy
2. Hemorrhagic Conversion (spontaneous & post- tPA)
3. Increased Intracranial Pressure
4. Hemorrhage & Underlying Ischemia
   a. Hypertensive IPH
   b. Hypocoaglable States
   c. Cerebral Amyloid Angiopathy
   d. Vasculitis / RCVS
   e. Aneurysm/ AVM

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Introduction and Overview

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CARE OF THE PATIENT WITH ACUTE STROKE

• Early assessment
• Rapid recognition of symptoms
• Activation of emergency services / appropriate intervention(s)
• Ongoing assessment
• Knowledgeable, multidisciplinary care team approach
What is the average door-to-needle time at your institution?
Pre-hospital assessment scales

- Cincinnati Prehospital Stroke Scale
- Los Angeles Prehospital Stroke Screen
- Miami Emergency Neurologic Deficit Checklist

Pre-hospital actions

- Document “Time last seen normal”
- Consider transport to nearest stroke center
- Perform finger stick to obtain blood glucose
- Obtain vital signs including blood pressure
- Facility pre-notification (code stroke/stroke team alert)

Recommended Evaluation Elements

• Patient history

• Physical exam and formal stroke scale (e.g., NIHSS)

• Stat non-contrast CT scan of the brain

• Consider ordering the following diagnostic tests
  – Blood glucose
  – Serum electrolytes / renal function tests
  – Markers of cardiac ischemia
  – Complete blood count, including platelet count
  – Activated partial thromboplastin time
  – Prothrombin time/international normalized ratio
  – Electrocardiogram
  – Oxygen saturation

Time Is Brain: Damage to the Brain During AIS Is a Rapid and Progressive Process

- Cells of the ischemic penumbra are metabolically active and potentially salvageable with timely assessment and management\(^1,2\)
- The infarction expands in the penumbra over time, increasing the area of irreversible brain damage\(^2\)
- Restoration of blood flow to the affected area may interrupt this process\(^3\)

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# TIME = BRAIN

<table>
<thead>
<tr>
<th>Time frame</th>
<th>Neurons lost</th>
<th>Ages the brain by</th>
</tr>
</thead>
<tbody>
<tr>
<td>Every second</td>
<td>32,000</td>
<td>8.7 hours</td>
</tr>
<tr>
<td>Every minute</td>
<td>1.9 million</td>
<td>3.1 weeks</td>
</tr>
<tr>
<td>Every hour</td>
<td>120 million</td>
<td>3.6 years</td>
</tr>
<tr>
<td>10 hours†</td>
<td>1.2 billion</td>
<td>36 years</td>
</tr>
</tbody>
</table>

†The duration of a typical, unmanaged acute ischemic stroke.

Calculations were based on a linear growth function, which does not reflect the actual rate of growth at particular points of time, but yields the average rate of infarct growth over the entire duration of infarct maturation for all possible growth function shapes.

Saver (Stroke, 2006) does not suggest that Activase® (alteplase) will help reverse the process of acute ischemic stroke.

# Clinical Presentation of Acute Ischemic Stroke

<table>
<thead>
<tr>
<th>Clinical Presentation of Acute Ischemic Stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aphasia</td>
</tr>
<tr>
<td>Ataxia</td>
</tr>
<tr>
<td>Dysarthria</td>
</tr>
<tr>
<td>Diplopia</td>
</tr>
<tr>
<td>Cranial nerve palsies</td>
</tr>
<tr>
<td>Hemianopia</td>
</tr>
<tr>
<td>Hemiparesis</td>
</tr>
<tr>
<td>Loss of sensation</td>
</tr>
<tr>
<td>Quadriaparesis</td>
</tr>
<tr>
<td>Visual field disturbances</td>
</tr>
</tbody>
</table>

# Conditions That May Mimic Stroke

<table>
<thead>
<tr>
<th>Conditions That May Mimic Stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcoholic intoxication</td>
</tr>
<tr>
<td>Metabolic disorders</td>
</tr>
<tr>
<td>Cerebral infections</td>
</tr>
<tr>
<td>Migraines</td>
</tr>
<tr>
<td>Drug Overdose</td>
</tr>
<tr>
<td>Conversion disorder</td>
</tr>
<tr>
<td>Epidural hematoma</td>
</tr>
<tr>
<td>Seizure and post-seizure</td>
</tr>
<tr>
<td>Hypoglycemia</td>
</tr>
<tr>
<td>Tumors</td>
</tr>
<tr>
<td>Neuropathies (e.g., Bell’s Palsy)</td>
</tr>
<tr>
<td>Hypertensive encephalopathy</td>
</tr>
</tbody>
</table>

COMPLICATIONS DURING THE FIRST 36 HOURS

- Cerebral edema
- Hemorrhagic conversion
- Pneumonia
- Infection
- Seizures
- Deep vein thrombosis
HEMORRHAGIC STROKE RISK

- **Risk** for IntraCranialHemorrhage 0.5% to 2% per year
- **Anticoagulation increases risk** 10 X
- Risk of cerebral emboli in patients with major cardioembolic sources (prosthetic valve, cardiac thrombus, AF) is high (without anticoagulation)
  - 20-30%
HEMORRHAGIC TRANSFORMATION WITHIN 36 HOURS OF CEREBRAL INFARCT

Relationships With Early Clinical Deterioration and 3-Month Outcome in the European Cooperative Acute Stroke Study I (ECASS I) Cohort

RESULTS: Risk of early neurological deterioration and of 3-month death was severely increased after PH2, indicating that large hematoma is the only type of hemorrhagic transformation that may alter the clinical course of ischemic stroke.
HEMORRHAGIC CONVERSION
• The presence of petechiae or confluent petechial hemorrhage confined to the ischemic zone

CEREBRAL EDEMA
• Accumulation of fluid in the intracellular or extracellular spaces of the brain;
• Occurs during first 24-48 hours
• Large hemispheric strokes – greater risk
• Often associated with hemorrhagic strokes or a hemorrhagic conversion

VASOSPASM
• More common with subarachnoid hemorrhage related to aneurysm

PNEUMONIA
• Often related to dysphagia and aspiration
HEMORRHAGIC CONVERSION IN ISCHEMIC STROKE

- Present in 15% of all ischemic strokes
- Present in up to 30% of cardioembolic strokes
- May be related to distal migration or lysis of an embolus resulting in reperfusion of the ischemic tissue, which can become hemorrhage depending on extent of ischemic vascular injury
- Detection related to imaging (MRI with T2 weighted sequences, and diffusion and perfusion-weighted imaging more sensitive than CT for early detection) - up to 60% conversion when re-imaged at 3 weeks.
- Autopsy demonstrates hemorrhagic transformation in 50-70% of patients undergoing anticoagulation [note – majority of hemorrhagic conversions in patients not receiving anticoagulation are asymptomatic]
PEARLS RELATED TO HEMORRHAGIC CONVERSION

• Visualization in patient with ischemic stroke may provide insight into underlying mechanism of stroke
  – May influence ongoing therapy

• Early anticoagulation in ischemic stroke adds risk to hemorrhagic conversion

• **Risk:Benefit ratio** of early anticoagulation influenced by the cardioembolic source of stroke as well as size of infarct [current data indicates the risk for recurrent stroke within first 5-7 days after ischemic event 5-8% without anticoagulation {patient with AF}]
  – Although heparin reduces the risk of recurrence of cardioembolic stroke – risk of symptomatic ICH offsets benefits
ANTICOAGULATION AND HEMORRHAGIC CONVERSION

• Occurrence and timing of hemorrhagic conversion not impacted by anticoagulation
  – Magnitude and clinical impact of hemorrhagic conversion has higher association
  – Large infarct
  – Excessive anticoagulation
  – Higher risk for symptomatic hemorrhagic transformation

• Current recommendation – delaying anticoagulation for 1-2 weeks (7-10 days)
  – Oral warfarin (without heparin) may be used in patients with nonvalvular AF or recent MI

• Typical patient with ischemia and AF is unlikely to benefit from anticoagulation within the first 1-2 days, especially with large stroke
• Patients who may benefit from early anticoagulation
  – Documented thrombi in left atrium
  – Mechanical prosthetic valve
  – Intracardiac thrombus
  – Congestive heart failure

• Consider other risk factors for brain hemorrhage
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Stroke Types

- Normal
- Ischemic Stroke
- Intracerebral Hemorrhage
- Subarachnoid Hemorrhage
**Imaging for Stroke Assessment**

**CT-Angiography**
Rapidly images large vessels in the neck and many first- and second-order arteries in the brain.

**Non-contrast CT**
Is the most practical and least time consuming initial brain imaging test for evaluation of potential stroke and can rule out hemorrhage.

**CT-Perfusion**
Provides cerebral blood flow, cerebral blood volume, and mean transit time maps.

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Basic Brain Imaging: CT

• X-ray beams (photons) pass through a patient’s body and are collected by computed tomography “CT” detector.

• A gray scale is created showing different shades of gray depending on the degree of absorption of the X-ray beams.

• TERMINOLOGY: a structure or lesion is “hyperdense” (LIGHTER shade of gray/white) or “hypodense” (DARKER shade of gray)
Basic Brain Imaging: Houndsfield Units ("HU’s")

CT absorption scale is measured in Houndfield units ("HU’s"). The scale is +1000 to -1000. The higher the HU’s, the more hyperdense. So bone is white, air is black.

<table>
<thead>
<tr>
<th>Substance</th>
<th>HU</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone</td>
<td>1000 (white)</td>
</tr>
<tr>
<td>Calcification</td>
<td>140-200 (white)</td>
</tr>
<tr>
<td>Acute blood</td>
<td>56-76 (white)</td>
</tr>
<tr>
<td>Gray matter</td>
<td>32-41 (actually whiter than “white” matter)</td>
</tr>
<tr>
<td>White matter</td>
<td>23-34</td>
</tr>
<tr>
<td>CSF</td>
<td>0</td>
</tr>
<tr>
<td>Fat</td>
<td>-30 → -100 (darker)</td>
</tr>
<tr>
<td>Air</td>
<td>-1000 (black)</td>
</tr>
</tbody>
</table>

Neuroradiology: The Requisites, Grossman and Yousem;
There are 3 views in neuroimaging: axial, coronal and sagittal. Most head CT's will show axial cuts only, like the one to the left.

MRI will usually show all 3 views: axial, coronal and sagittal view.

The convention is that the patient’s right side of the brain will be shown on the left side of the image. In an axial view, picture the patient lying in the scanner with the feet sticking out at you.
Basic Brain Imaging: Tips for Reading a CT

- Pay attention to what a normal head CT’s look like.

- There is a WIDE range of normal and a wide range in quality of exams.

- As with reading chest x-rays,
  - symmetry is your friend;
  - it is helpful that there are 2 halves to every brain;
  - side by side comparison can reveal a lesion or give assurance that the study is normal.

- The most basic head CT will have approximately 32 axial cuts measuring 0.5 cm each. Get used to going through a study the same way every time (bottom to top or top to bottom).
Basic Brain Imaging: Tips for Reading a CT

- Experiment with viewing different numbers of axial cuts at a time – you can scroll through a study looking at just a single image at a time or you can look at 2, 4 or 6 images at a time.

- Many people find it valuable to look at more than one cut at a time -- to get the whole picture and to see if a lesion extends beyond a single cut. However you often may want to focus on a single image to get the best detail of a potential abnormality.

- Pay attention to structure density. Look for gray-white differentiation.

- Pay attention to ventricle size. Again this will help to give you a sense of the broad range of normal. If the ventricles seem large, do the sulci also seem large?
Normal Head CT of 59 y/o man at upper level of midbrain to thalami, basal ganglia. On CT gray matter is lighter or “more hyperdense” than white matter. Note the good grey-white differentiation especially on the 2 lower images.
Normal head CT of 38 year-old starting at the pons/4th ventricle, going up to midbrain, then to the thalami and internal capsule, finally to the centrum semi-ovale. Note the normal size of the ventricles – though there is a wide range of normal.
Basic Brain Imaging

HEAD CT OPTIONS:

- Plain CT
- CT with contrast
- CT Angiogram ("CTA")
  - Head
  - Neck
- CT Perfusion

NORMAL
Basic Brain Imaging

HEAD CT OPTIONS:

- Plain CT
- CT with contrast
- CT Angiogram (“CTA”)
- CT Perfusion

Plan Head CT Best for:
- Trauma: skull/face/orbit fracture
- Detecting acute blood subarachnoid hemorrhage
- Intraparenchymal hemorrhage
- Subdural hematoma
- Detecting calcification

Lesions commonly missed on CT:
- Acute ischemic stroke
- Tumor
- Abscess
- Demyelinating lesion
Basic Brain Imaging

HEAD CT OPTIONS:

- Plain CT
- **CT with contrast**
- CT Angiogram (“CTA”) Head &/or Neck
- CT Perfusion

**CONTRAST = IODINE**
- administered via 18 ga or larger ante-cubital IV
- flows through cerebral vessels and leaks out in areas of blood-brain barrier breakdown
- order a CT with contrast when you’re worried about a focal lesion such as: tumor, abscess or other focal infection; you still miss lesions that can be seen only w/MRI but will increase yield over plain CT
- Terminology: area of contrast outline is “enhancing”

**CONTRAINDICATIONS**
- Creatinine >1.5 (there are exceptions)
- Iodine allergy (there are exceptions)
On the plain CT, bilateral areas of hypodensity are seen. On the post-contrast, multiple ring-enhancing lesions are seen. The imaging is consistent either with infection, such as toxoplasmosis or tumor, especially metastases.
Basic Brain Imaging

HEAD CT OPTIONS:

- Plain CT
- CT with contrast
- **CT Angiogram** ("CTA")
  - Head &/or Neck
- CT Perfusion

**CTA**

- Images cranial blood vessels.
- Iodine contrast is injected through 18-gauge ante-cubital IV.
- Images are taken that show the contrast material in the vessels (principally the arteries).
  - static picture (snapshot) of vessels
  - can be reconstructed to show 3-D
- Contraindications are the same as for CT with contrast.
Basic Brain Imaging

HEAD CT OPTIONS:

- Plain CT
- CT with contrast
- CT Angiogram ("CTA")
  Head &/or Neck
- CT Perfusion

CTA Neck vs Head (Brain)

- Typically a CTA of the NECK will image from the top of the aortic arch to the Circle of Willis.

- Consider CTA of neck to look for...
  - carotid/vertebral dissection
  - stenosis or occlusion of extracranial or proximal intra-cranial artery (e.g.: internal carotid stenosis, basilar tip thrombosis)

- This study is frequently requested on acute ischemic stroke patients to assess for stenosis, clot or dissection.
**LEFT:** is an image from a normal neck CTA. It is at the level just above the carotid bifurcation. The left ICA and ECA are somewhat larger than the right. Also seen are the vertebral arteries as they ascend in the vertebral foramina.

**RIGHT** is the “reformatting” of the CTA. Multiple images are formatted together so that a long portion of vessels may be seen in multiple views. Here is a coronal view of the internal carotids. The distal basilar artery and its branches can be seen intra-cranially.
Basic Brain Imaging

HEAD CT OPTIONS:

- Plain CT
- CT with contrast
- **CT Angiogram**
  - (“CTA”)
  - Head &/or Neck
- CT Perfusion

---

**CTA Neck vs Head (Brain) cont.**

- CTA of the BRAIN enhances vessels from the skull base to the vertex – it will not give a good view of extra-cranial vessels.

- Consider CTA of brain to evaluate for suspected:
  - aneurysm
  - AVM or other vascular anomaly

- It is **important to specify** “CTA neck” or “CTA brain” – if you ask for both, the radiologist may protest and/or the study may be technically sub-optimal.
From left to right: plain CT, CTA, CTA reconstruction. The cause of this sub-arachnoid hemorrhage is evident even on the plain CT. There is an aneurysm at the basilar tip. It is more sharply outlined in the CTA. The CTA reconstruction shows the Circle of Willis with the large aneurysm very nicely. When you suspect a subarachnoid hemorrhage on plain CT, you should order a CT-angiogram of the brain to assess for aneurysm.
Basic Brain Imaging

HEAD CT OPTIONS:

- Plain CT
- CT with contrast
- CT Angiogram (“CTA”)
  Head &/or Neck
- CT Perfusion

CT Perfusion Study

- Iodine or xenon is infused/inhaled (respectively).
- Cerebral blood flow is measured.
- Hypo-perfused areas are differentiated from normal brain and from infarcted brain.
- Sometimes performed in setting of acute CVA to discern if there is salvageable tissue.
- Really need experience neuro-radiologist to read 2/2 variability in studies from center to center.
Basic Brain Imaging

HEAD CT OPTIONS:

- Plain CT
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CT Perfusion may help ID penumbra

Graphic of penumbra
CT- Perfusion

Helps determine cerebral blood flow and which areas are at risk.
Hyper-dense MCA sign

The sign is typically seen within 90 minutes of the ischemic event, and thus, it is very important for radiologists to recognize this sign. It can save the patient in 'golden hour' of thrombolysis (3 hours for intravenous tPA, and 6 hours for intra-arterial thrombolysis). This sign has approx. 100% sensitivity, however only 30% specificity.

http://radiopaedia.org/articles/hyperdense-mca-sign
MRI- Introduction

An MRI scanner consists of a large and very strong magnet in which the patient lies. A radio wave antenna is used to send signals to the body and then receive signals back. These returning signals are converted into images by a computer attached to the scanner. Imaging of almost any part of your body can be obtained in any plane.

http://radiopaedia.org
Basic Brain Imaging: **MRI- Introduction**

**Advantages**
- Visualize early ischemic changes
- Ability to image without the use of ionizing radiation (x-ray) unlike CT scanning
- Multiple plane imaging (Axial, Sagittal, Coronal, or Oblique) without moving the patient.
- Ability to be reconstructed in multiple planes
- Superior soft tissue contrast films making it ideal for the brain, spine, joints and other soft tissue body parts
- Some images can be obtained without contrast, unlike CT or conventional angiography
- Advanced techniques for visualization of both brain activity and the underlying networks

**Disadvantages**
- There are a number of disadvantages and challenges to implementing MRI scanning.
- MRI scans are more expensive than CT scans and take longer to acquire so patient comfort is sometimes an issue. Additionally images are subject to unique artifacts that must be recognized and abated.
- MRI scanning is not safe for patients with metal implants and foreign bodies.
- Careful attention to safety measures to avoid serious injury and requires special MRI compatible equipment and stringent adherence to safety protocols

http://radiopaedia.org/articles/
DWI major role in the following clinical situations

- early identification of ischemic stroke
- differentiation of acute vs chronic stroke
- differentiation of acute stroke vs mimics
- differentiation of epidermoid cyst from arachnoid cyst
- differentiation of abscess from necrotic tumors

- assessment of cortical lesions in CJD
- differentiation of herpes encephalitis from diffuse temporal gliomas
- grading of gliomas and meningiomas (need further study)
- assessment of active MS plaque (old plaques will not be bright)

http://radiopaedia.org/articles/diffusion-weighted-imaging-1
Basic Brain Imaging:

MRI - Diffusion Weighted Imaging

DWI EARLY (acute) ischemic stroke

DWI OLD (subacute) ischemic stroke.

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Basic Brain Imaging
Resources for neuroanatomy & imaging reviews:

UCLA Neuroradiology has compiled “A collection of the best educational sites”; the website is http://www.neuropat.dote.hu/nrad2.htm


UBM Medica Network http://radiopaedia.org/
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V. Questions
What are transitions of care?

“Transitions of care”

the movement of patients between health care staff and practitioners, settings, and home as their condition and care needs change.

PCP  •  PCP or specialist in an outpatient setting

HOSP  •  to a hospital physician and nursing team during an inpatient admission

SNF  •  yet another care team at a skilled nursing facility

PCP  •  Back home to make appointment with PCP
Interactions Within Stroke Systems of Care


Stroke
Volume 44(10):2961-2984
September 23, 2013
Guiding principles for field triage of patients with suspected acute stroke.

<table>
<thead>
<tr>
<th>Patient with abnormal vital functions in need of acute resuscitation</th>
<th>Transport to nearest hospital for stabilization of vital signs</th>
<th>Once vital functions stabilized, transfer to nearest CSC (or PSC if long distances)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient with acute onset of stroke symptoms within 6-8 hours</td>
<td>Transport patient to closest PSC or CSC if &lt;15-20 minutes transport time</td>
<td>If PSC and/or CSC &gt;15-20 minutes away, go to closest ASRH</td>
</tr>
<tr>
<td>Patient with acute stroke and seen initially at an ASRH</td>
<td>ASRH might use telemedicine to help evaluate the patient and to make transfer recommendations</td>
<td>Transfer to nearest PSC or CSC based on stroke type, patient's medical condition, treatment options</td>
</tr>
</tbody>
</table>

Higashida R et al. Stroke 2013;44:2961-2984
Examples of care transitions among staff, specialists, and care areas:

HYPERACUTE ISCHEMIC STROKE PATIENT

Higashida R et al. Stroke 2013;44:2961-2984
Examples of care transitions among staff, specialists, and care areas:

**HYPERACUTE HEMORRHAGIC PATIENT: IPH/SAH**

Higashida R et al. Stroke 2013;44:2961-2984
Examples of care transitions among staff, specialists, and care areas:

**HYPERACUTE ISCHEMIC STROKE ..... POSSIBLE INTERVENTION (ENDOVASCULAR OR SURGICAL)**

Higashida R et al. Stroke 2013;44:2961-2984
What are transitions of care?

ANY CRITICAL HAND-OFF

EMS
- Presentation handoff w/ ED team

ED
- ED RNs initial team work up & stroke code activations; continuous team communication, collaboration, coordination

ANGIO
- Stat RNs handoff to Radiology RNs

NICU
- Recovery & handoff to NICU RNs; stroke pathway care

3WH
- Stroke pathway care, continuous status monitoring, discharge planning

4WH
- Handoff to inpt rehab, cont .status assessment. Rehab discharge planning.

Home
- 2-day, 7-day, 30- & 90-day calls/ MRS assessments. Self-management and/or combination of family or caregiver support & outpt care coordination, continuous status monitoring. F/U w/ PCP & Stroke clinic. Rehab

Clinics
- Stroke Clinic, Rehab/ therapies, f/u diagnostics, care coordination w/ PCP.
What is the mandate?

REGULATORY MANDATE:

To reduce both readmission rates and adverse events, hospitals have the mandate to improve the effectiveness of transitions of care in which they play a role. (CMS & Joint Commission Stroke)

CONSEQUENCES: Failure to meet standards

1. Poor patient outcomes.

2. Penalties: Hospitals with unacceptably high readmission rates for Medicare and Medicaid patients will soon face financial penalties under the Patient Protection and Affordable Care Act.

3. Denial of Comp. Stroke Certification (TJC)
The problem: Ineffective transitions of care

Ineffective care transitions lead to **preventable adverse events**, higher hospital readmission rates and avoidable costs. 4

A study of physician-to-physician communication estimated that 80% of serious medical errors involve miscommunication during the hand-off between medical providers.5

“**falling through the cracks**”, occurs in every type of health care setting, but it is especially problematic when patients leave the hospital to receive care in another setting or in discharge to home.


Lessons Learned:
Check the safety brakes before you let go!
Critical Transitions of Care:

**Nursing Challenges to Consider**

- Wide variation in symptoms to identify & monitor
- Neurologic assessment continuity (variation of assessment skills)
- Documentation: EMR systems & content complex
- Acuity: Very High
- Balancing multiple high risk health problems
- Complex Pathophysiology
- Coordinating complicated care with resource limitations
- Multiple disciplines, services, and hand-offs involved
Best-evidence transitional care practices

• Multidisciplinary communication, collaboration and coordination – including patient/caregiver education – from admission through transition. A care team –

• Clinician involvement and shared accountability during all points of transition

• Comprehensive planning and risk assessment throughout hospital stay.
Best-evidence transitional care practices

- **Standardized transition plans, procedures and forms.**

- **Standardized training.**

- **Timely follow-up, support and coordination after the patient leaves a care setting.**

- **If a patient is readmitted within 30 days, gain an understanding of why.**
HAND-OFFs & DOCUMENTATION

**BEST PRACTICE MUST HAVE...**

Documentation showing tracking of patient status between ALL transfers with comparable assessments!
Post-TPA and Post-procedure Monitoring Challenges

Post-tPA:
Q 15 min X 2 hours, then q 30 minutes X 6 hours, then q 1 hour X 16 hours, then per unit protocol.

Document on post-procedure tracking tools:
tPA started, completed and each of the timed VS/Neuro/Post-Angio checks (where applicable)

Post-Intervention & Post-Angiography Monitoring:
limited evidence to guide the “BEST PRACTICE” for exact assessment & monitoring protocol.
### Sample Post-Angio Tracking Sheet:

#### Front & Back

**Post-Neuro Angiogram Assessment Tracking**

Start documenting assessments immediately after procedure.

**HEMOSTASIS**

<table>
<thead>
<tr>
<th>Time:</th>
<th>Date</th>
</tr>
</thead>
</table>

**Q 15 MIN x 4 (1st hour)**

<table>
<thead>
<tr>
<th>Vital Signs, Invasive Site, Pulses, Neuro Status (Q Hour)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Q 15 MIN</td>
</tr>
<tr>
<td>VITAL SIGNS</td>
</tr>
<tr>
<td>INVASIVE SITE ASMT</td>
</tr>
<tr>
<td>PULSES</td>
</tr>
<tr>
<td>NEURO</td>
</tr>
</tbody>
</table>

**Q 30 MIN x 2 (2nd hour)**

<table>
<thead>
<tr>
<th>Vital Signs, Invasive Site, Pulses, Neuro Status (Q Hour)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Q 30 MIN</td>
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**Document in ORCA**

This sheet is only a handoff tool, not part of the permanent record.

**ICU Frequent Assessment**

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**DO NOT SCAN IN PERMANENT RECORD**

For QI tracking purposes only.

9/18/2014 C. Artuso - V. Johnson

Comprehensive Stroke Center at Harborview

Version 5.2014.7.8
CASE STUDIES
COMPLEX STROKE PATIENTS
Introduction and Overview

I. Introduction & Overview: Complex Stroke

II. The First 36 Hours of Care

III. Basic Brain Imaging

IV. Critical Transitions of Care:
   - admission to discharge and beyond

V. Complex Stroke Cases
   - malignant MCA ischemic stroke
   - complications

V. Questions

1. Hemicranietomy
2. Hemorrhagic Conversion (spontaneous & post- tPA)
3. Increased Intracranial Pressure
4. Hemorrhage & Underlying Ischemia
   a. Hypertensive IPH
   b. Hypocoaglable States
   c. Cerebral Amyloid Angiopathy
   d. Vasculitis / RCVS
   e. Aneurysm/ AVM
Malignant Edema

- Peak edema usually at about 3 days, but can range from 1-5
- Peak edema from hemorrhagic stroke can occur later
- Large strokes require careful monitoring for neurologic deterioration
- Decompressive hemicraniectomy may be needed
Hemicraniectomy

Decreases the risk of death in cases of malignant edema with large, complex ischemic strokes.

Panel: Eligibility criteria for the pooled analysis

Inclusion criteria
Age 18–60 years
Clinical deficits suggestive of infarction in the territory of the MCA with a score on the National Institutes of Health stroke scale (NIHSS) >15
Decrease in the level of consciousness to a score of 1 or greater on item 1a of the NIHSS
Signs on CT of an infarct of at least 50% of the MCA territory, with or without additional infarction in the territory of the anterior or posterior cerebral artery on the same side, or infarct volume >145 cm³ as shown on diffusion-weighted MRI
Inclusion within 45 h after onset of symptoms
Written informed consent by the patient or a legal representative
Hemicraniectomy: outcome comparison

Figure 1: Distributions of the scores on the mRS and death after 12 months for patients treated with or without decompressive surgery
CASE STUDY: MCA with Unstable Edema, Hematoma, Bone Flap [7/1]

56 year old male patient, found down at home, left side paresis

- Returned from work at 2 AM; loud thump heard in AM – several hours to gain access to home
- History of hypertension; has not seen physician in many years
- Smokes 1 ppd, social alcohol use; family hx heart disease
- Initial CT – large right MCA infarct

Large right MCA infarct on CT’

VS: Temp: 36 °C (96.8 °F) HR: 69 RRR: 14 SpO2: 96 % BP: 188/120 mmHg
  [BP treated with labetolol in ED]
Well developed, well-nourished male patient; 81.65 kg

Pupils equal, reactive to light; dense left-sided hemiparesis; able to converse with some hesitation; c/o right side headache;

7/01 CT – Subacute right middle cerebral arterial distribution infarction with moderate cerebral edema and mass effect on the right lateral ventricle.

Carotid dopplers – occluded right ICA

Admitted to general neurology floor – admission medications included Aspirin, Lipitor, Lovenox, Pepcid, Insulin, Ativan; IV NSS @ 100 ml/hr
OVERVIEW and ADMISSION

WBC – 17.1 (4.5-11.0uL)
Glucose 125 mg/dL (65-99)
PT 12.6 sec (12.9-15 sec)
Admission VS: Temp: 36 °C (96.8 °F) HR: 62 RR: 35 SpO2: 95 % BP: 191/117 mmHg
ECG - NSR

Admitting Diagnosis: Stroke: multiple risk factors including HTN, smoking, family history.
Currently sinus rhythm. Right MCA distribution with vasogenic edema.
Day 2 – hospital admission

Patient c/o persistent headache – focused on right side; increasingly lethargic progressing to obtunded

Stat imaging reveals increased right sided edema; midline shift, slight hemorrhage, symmetry reperfusion

**NEUROSURGERY CONSULTATION** – prepared for craniectomy

**PREOPERATIVE DIAGNOSIS:** Malignant middle cerebral artery syndrome secondary to massive right middle cerebral artery infarct with mass effect, midline shift, and neurologic deterioration.

**POSTOPERATIVE DIAGNOSIS:** same as pre-operative dx

**OPERATION:** Right frontotemporal parietal hemicraniectomy with dural enhancement with graft.
HOSPITAL COURSE

Hospital day 2 [7/3] - patient underwent craniectomy for increased cerebral edema

Hospital day 5 [7/6] – increasing WBC ct; bronchoscopy, intubated for respiratory failure; dx. pneumonia – likely r/t aspiration (potentially pre-hospital admit); pan sensitive e-coli; pleural effusion; chest tube

Hospital day 16 [7/17] – CT right sided subdural hematoma not significantly changed in size; associated mass effect is decreased with only trace midline shift remaining; Basilar cisterns patent; scalp hematoma decreased in size. No evidence of hemorrhagic transformation is seen compared with 07/10/2014.
HOSPITAL COURSE [continued]

**Respiratory Failure** – intubated; tracheostomy performed

**Cardiac** – slightly elevated troponin; high risk for cardiac cath; recommended medical management; aortic atherosclerosis

**Dysphagia** – PEG placed

**Elevated LFTs** – statin changed to pravastatin
Discharge DX:
Acute right MCA stroke; unstable cerebral edema; s/p hemicranectomy; left-sided hemiparesis; PEG for nutrition; pneumonia (resolving); s/p pleural effusions; s/p chest tubes for evacuation; subdural hematoma (resolving); s/p midline shift (resolving); compromised airway (trach)

DC Medications: ASA 81mg po od; Lisinopril 5 mg bid; metoprolol 50 mg bid; pravastatin 80 mg od;

Discharged to LTAC on hospital day 28
CASE STUDY: MCA STROKE WITH HEMORRHAGIC TRANSFORMATION POST tPA

53 year old Asian female patient transferred from distant community hospital after telestroke evaluation and treatment

Patient’s husband reports that he was awakened when she tried to get out of bed at 0330 AM and fell to the floor; patient has slurred speech with facial droop, left-side hemiparesis

Past medical history – significant for atrial fibrillation

Evaluated via telemedicine; initial CT WNL; cervical spine films WNL

Treated with rt-PA at 0630; elective intubation for airway protection
CBC and CMP were normal except glucose 237. Digoxin level 0.8. INR was 1.0, troponin normal at 0.11.

EKG showed non-specific ST changes in lead III.

The patient was intubated, sedated on propofol drip, and mechanically ventilated upon arrival.

Sedation D/C to assess neuro status.

Patient is awake and pointing to her ET tube with her R hand; left side flaccid

Repeat CT without contrast – no change at 6 hours, some subjective fullness of the R MCA Patient is bradycardic in the 40s-50s, BP in the 120s-140s systolic, oxygen saturation 100%. ABG 7.42/35/204/100. CXR is clear.
INITIAL EVALUATION cont.

- Current Medications
  - Lisinopril
  - Digoxin
  - Coreg
  - Spironolactone
  - Insulin- novalog

- No allergies

- Denes smoking, rare alcohol use

- Vital signs on admission: BP 160/64; HR; 63; RR 17

- Physical exam after rt-PA – increased ability to move left leg; no sensation left side; left arm flaccid
HOSPITAL COURSE

• Hospital Day 2 – increasing cerebral edema; evidence of hemorrhage on CT scan; deteriorating neurologic condition
  – Craniectomy performed
  – Improved neurologic condition

• Patient made steady improvement with therapy; residual dysarthria; dysphagia; right dense hemiparesis (U > L); rate controlled atrial fibrillation; on Coumadin - therapeutic

• Fever unknown origin – found associated with CVL – central line discontinued; broad spectrum antibiotics continued x 10 days; WBC normal; fever WNL

• DM – A1C 8.8

• Discharged home, Metformin 1000 mg bid; Lisinopril 20 mg od; aldactone 50 mg od; Novolag with meals; Crestor 5 mg od; Coreg 80 mg od; lanoxin 250 ug od
COMPLEX STROKE

IMAGING REVIEW
80 y.o. female with hx of dementia, depression/anxiety, hypertension, hyperlipidemia, paroxysmal atrial.fib, breast cancer s/p mastectomy

• Onset of left leg weakness, left facial droop and slurred speech today

• At around 2:15pm, patient was walking around the block with a walker. The caregiver noticed that the patient was dragging her left foot with walking, had slurred speech, and left facial droop.

• Daughter was notified, who came home and noticed the above findings. She quickly assessed that the patient had sensation in all her extremities.
IMAGING RESULTS

- Head CT negative for bleed; Frontal lobe predominant cerebral atrophy, small vessel ischemic changes and cerebral white matter
- Symptoms improving; tPA not administered
ED AND HOSPITAL COURSE

Admission VS: Temp: 36.4 °C (97.5 °F) HR: 63 RR: 24 SpO2: 94 % BP: 118/60 mmHg

Weight: 98.88 kg

Laboratory Results: (abnormal only)

Glucose: 109 mg/dL

BUN: 31 (8-26 mg/dL)

Creatinine: 1.27 (0.6 – 1.1 mg/dL)

Protime 12.6 (12.9 – 15 sec)

EKG: Normal Sinus Rhythm

Carotid U/S reveal bilateral PE
Patient was admitted and placed on full anticoagulation. She had complete resolution of her neurologic symptoms and MRI failed to demonstrate acute ischemic event. She remained in sinus rhythm on telemetry; discharged 3 days later with final diagnosis of transient ischemic attack. Remains on Coumadin and Lovenox.

Patient re-evaluated in the ED 9 days post discharge with persistent episodes of vomiting and lack of interest in food. No acute neurologic changes were noted. Patient evaluated by PMD; video swallow study performed [unremarkable]; MRI ordered.

Patient readmitted post MRI with dx. Hemorrhagic conversion post ischemic stroke.
MRI: generalized cerebral atrophy, with prominence of the ventricles and sulci.

New heterogeneous, T2 hypointense, T1 hyperintense lesion within the right cerebellar hemisphere, with loss of signal on gradient echo sequences, consistent with an intraparenchymal hemorrhage. This measures 4.2 x 3.8 x 2.3 cm. Mass effect from the hemorrhage severely narrows the 4th ventricle, although the lateral ventricles have increased only minimally in size, previously 5.1 cm right to left, currently 5.3 cm right to left. A third ventricle measures 13 mm, not significantly changed from 12 mm previously.
D/C Status

Patient discharged to assisted living
Remains on ASA only – accepted risk of devastating embolic event r/t pulmonary embolus
Family reviewed risks/benefits of anticoagulation, however given patient’s advanced dementia and risk of additional neurologic deterioration opted for ASA.
Malignant Edema & Hemicraniectomy, plus...
Case: Malignant Edema & Hemicraniectomy, plus...

56 year old African-American male with a large left middle cerebral artery infarct associated with total occlusion of the Left Internal Carotid Artery w/ left carotid dissection in the setting of HTN & cocaine use. Presents approximately 8 hours after worsening symptoms.

Magnetic Resonance Imaging (MRI) 5/20/2009
Case: Malignant Edema & Hemicraniectomy, plus...

Progressive edema & concern for herniation warranting surgery.

5/21/2009 head CT
s/p hemicraniectomy.
COMPLEX STROKE

Case: Malignant Edema & Hemicraniectomy, plus...
Case: Malignant Edema & Hemicraniectomy, plus…

Let’s Brainstorm…

STROKE NURSING & TRANSITIONAL CARE CONSIDERATIONS.
Hemorrhagic Presentations…
What’s Hiding?
Case: Hemorrhage presentation

61 yo M flying to Seattle en route developed severe HA, AMS, N/V. Head CT at OSH ED revealed a R thalamic/BG bleed with intraventricular extension and hydrocephalus. Typical Hypertensive CT appearance?
Case: Hemorrhage Presentation & Finding Brain Metastases

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Case: Hemorrhage Presentation & Finding Brain Metastases

Let’s Brainstorm…

STROKE NURSING & TRANSITIONAL CARE CONSIDERATIONS.
Hemorrhage Presentation…
True Hypertensive Bleed
50 yo Hispanic F with history of untreated hypertension and limited access to health care.

Presented to local hospital with c/o severe headache, BP 220/120 & quickly declining level of consciousness. Consultation with Stroke Phone attending, imaging review, acute treatment for HTN with labetalol and airlift transport to Harborview.

1 month post-discharge pt doing well with minimal residual affects.
CASE STUDY: Hemorrhage Presentation…
True Hypertensive Bleed

Hypertensive Intracranial (intraparenchymal) Hemorrhage:

- HTN most common cause of IPHs
- Typically round or oval hyperdense appearing mass
- 80% Mortality with large IPHs with interventricular extension.
- Acute Treatment: control hypertension and hydrocephalus.

- Typical location:
  - Putamen/external capsul (65%)
  - Thalamus (25%)
  - Pons/cerebellum (10%)

www.headneckbrainspine.com

Let’s Brainstorm…

STROKE NURSING & TRANSITIONAL CARE CONSIDERATIONS.
Hemorrhage Presentation…
Now What?
HISTORY 86 y/o F living independently. PMH: h/o hypertension.

- Hospitalized 5 days ago at an OSH with a UTI and mild confusion. Re-admitted at OSH 2 days ago 2/2 increased confusion. Head CT showed Large L frontal IPH. Transfer to HMC.

EXAM: intermittently opens eyes to voice, can say her name and "hospital" but not city. Follows command to lift up arms. couldn't get her to lift legs but she moves them symmetrically in bed.

IMAGING MRI reviewed with Neuro-radiologist

- large L frontal bleed shows both subacute and acute ischemic & hemorrhagic findings.
- the ACA territory, there is a DWI positive area and evidence of laminar necrosis -- most consistent with relatively recent ischemic infarct.
- Intraventricular extension
- IPH in the R temporal-occipital area.
Case: Hemorrhage Presentation… Now What?

Attending’s Comment:

“The imaging is puzzling. Difficult to explain 2 IPH's without h/o significant trauma -- and the L sided lesion is most consistent with hemorrhagic transformation of ischemic stroke. Hemorrhagic metastases are also possible -- though the radiographic picture is not typical for that either.”
Final Impression:
1. Stable appearance of large left IPH and IPH along the trigone of the R lateral ventricle compared to the exam done 6 hours earlier. There is associated mass effect with partial compression of the frontal horns of the lateral ventricles.

2. IVH is now demonstrated, layering in the occipital horns of the lateral ventricles. There is mild dilation of the temporal horns of the lateral ventricles, but unchanged from the exam done 6 hours earlier.

3. No evidence of vascular abnormality underlying hemorrhages.

4. Heterogeneous right thyroid lobe nodule measuring 3.1 cm in diameter. Further evaluation with ultrasound is recommended.

Differentials for her Hemorrhagic Stroke(s) include…

- Hypertensive vs.
- Amyloid Angiopathy causing multiple macro-bleeds vs.
- Malignancy (thyroid nodules found on CT, so some possibility of thyroids metastises) vs
- Hemorrhagic Transformation of ischemic lesions…Less likely as TTE was negative for a thrombus/PFO. CTA performed as part of comprehensive stroke workup revealed normal vessels of the head and neck (unlikely artery to artery embolic/atherosclerosis)
Follow Up:

- repeat MRI brain w&w/o contrast in 8 weeks to further investigate etiology.
- “Her home Aspirin was held and will continue to be held until she follows up in Stroke Clinic given concern for macro-bleed type of cerebral amyloid angiopathy, would likely not restart unless there was a definite indication”.

What is cerebral amyloid angiopathy?
Description.

● CAA usually manifests with a spontaneous lobar hemorrhage. This location helps distinguish CAA-related ICH from hypertensive ICH that more commonly arises in the putamen, thalamus, and pons.

● Transient neurologic symptoms are another manifestation of CAA. These are recurrent, brief (minutes), often stereotyped spells of weakness, numbness, paresthesias, or other cortical symptoms that can spread smoothly over contiguous body parts. The pathogenesis of these spells is not certain; they are not believed to be transient ischemic attacks in most cases.

● CAA-related inflammation is a distinct disease subtype characterized by subacute cognitive decline, seizures and unifocal or multifocal white matter MRI T2 hyperintensities extending to the subcortical white matter or sulci. Clinical and radiographic improvement can occur with immunosuppressive therapy.

● CAA and Alzheimer disease frequently co-exist. CAA may also be associated with a vascular dementia.
Acute superficial lobar hemorrhage in the left frontal lobe seen on CT scan in a patient with cerebral amyloid angiopathy (Panel A). Flair MR image performed one week later shows the high signal intensity of subacute hemorrhage with surrounding edema extending into the subcortical white matter (Panel B). Hemorrhage (now low signal intensity consistent with hemosiderin) and edema are mostly resolved on a three-month follow-up study (Panel C).

_Courtesy of Eric D Schwartz, MD._
Looking back at our case of the 86 yo female with hemorrhage.

Hemorrhage presentation with Cerebral Amyloid Angiopathy suspected or confirmed.

Let’s Brainstorm…
STROKE NURSING & TRANSITIONAL CARE CONSIDERATIONS.
CNS vasculitis in a 37-yo F with systemic lupus erythematosus.
A. infarcts in the right cortical gray matter and subcortical white matter.
B. PCA distribution, L posterior
C. Angio of L ICA w/ stenosis in ACA, MCA, and PCA area.
D. Angio of R ICA w/ lesions in ACA, severe narrowing of distal MCA.
CNS vasculitis in a 37-yr F with systemic lupus erythematosus. 

severe narrowing of distal MCA.
The vasculitis affects any part of the CNS, causing the clinical manifestations to be highly variable and nonspecific [1]. PACNS should be suspected when strokes, frequently recurrent, occur in young patients with no identifiable cardiovascular or hypercoagulable risk factors;

OR in the setting of chronic meningitis, recurrent focal neurologic symptoms, unexplained diffuse neurologic dysfunction, or unexplained spinal cord dysfunction not associated with systemic disease or any other process
Case: Subarachnoid Hemorrhage

19 yo F with sudden onset c/o worst headache of her life, nausea/vomiting and loss of consciousness. Required intervention with aneurysm coiling. Post-op vasospasms experienced.
Subarachnoid hemorrhage results from the bleeding of an artery around the base of the brain. It is the least common type of stroke, accounting for about 5 percent of all strokes.
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