TIA and Minor Stroke

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Disclosures

• No financial disclosures
Objectives

• Introduction
• Epidemiology
• Assessment of possible stroke/TIA
  • History/Exam
  • Testing
• Secondary stroke prevention treatments
• A few words on acute stroke treatments
Stroke facts

• Major cause of disability and death worldwide
• 5\textsuperscript{th} leading cause of death in US (moved from 4\textsuperscript{th} in 2013)
  • ~ 130,000 Americans annually
  • 1 of every 20 deaths $\rightarrow$ 1 death every 4 minutes
• >795,000 strokes in the US each year
  • ~25% are recurrent stroke
• Costs $34 billion+ annually in the US
Stroke Death Rates, 2008-2010
Adults, Ages 35+, by County

Rates are spatially smoothed to enhance the stability of rates in counties with small populations.

Data Source:
National Vital Statistics System
National Center for Health Statistics

Age-Adjusted Average Annual Rates per 100,000
- 13.5 - 72.7
- 72.8 - 81.2
- 81.3 - 88.9
- 89.0 - 100.1
- 100.2 - 300.1
- Insufficient Data
Definition of Stroke

- Neurologic symptoms due to vascular lesion of the CNS (Brain, spinal cord, or retina)
  - Infarct = due to ischemia ("clot")
  - Hemorrhage ("bleeding") into brain (IPH), ventricles (IVH), or subarachnoid space (SAH)
    - Non-traumatic
    - Not SDH
    - Not EDH
- Other etiologies excluded
Definition of Stroke

• Infarct / ischemic stroke - 87%
  • Permanent injury OR ≥24 hours of symptoms \(\rightarrow\) infarct
  • <24 hours \(\rightarrow\) TIA (typically <1 hour, w/o lesion on CT or ?MRI)
• Intracerebral hemorrhage (ICH), intraparenchymal hemorrhage (IPH) - 5-10%
• Subarachnoid hemorrhage - 5%

• Symptoms caused by reversible edema without infarct or hemorrhage do not qualify as stroke.
• Neither does traumatic IPH
When to suspect stroke?

- Timing (sudden onset)
- Focal deficits
  - Unilateral weakness (including facial droop) and numbness
  - Speech impairment (aphasia, dysarthria)
  - Neglect
  - Vision impairment (field cut, diplopia)
  - Ataxia/incoordination, especially if unilateral
  - Vertigo...
  - Loss of, or decreased level of consciousness?
- Risk factors
Stroke risk factors

- Non-modifiable
  - Age
  - Gender
  - Race/ethnicity
  - Genetic factors
  - Migraine history

- Modifiable
  - Hypertension
  - Tobacco use
  - Diabetes
  - Dyslipidemia
  - Heart disease, Atrial fibrillation
  - Lack of exercise
  - Heavy alcohol use
  - Drug use
Assessment of possible stroke/TIA

- **History:**
  - Current (and prior) symptoms with course
  - Time of onset/LKN
  - PMHx, medications

- **Exam:**
  - Mental status including language assessment (fluency, naming, repetition, comprehension) and dysarthria
  - Cranial nerves (attention to visual fields, gaze preference, facial droop or subtle flattening, sensation)
  - Motor (pronator drift, subtle weakness) and coordination
  - Sensory including dual stimuli (inattention/neglect)
  - Gait testing
Localization – where is the stroke?

Remember the neuro-anatomy –

What brain regions could cause the sx?

Is there a single region to explain everything?

What blood vessels supply that region?

<table>
<thead>
<tr>
<th>Clues to cortical localization:</th>
<th>Clues to brainstem:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Aphasia (dominant hemisphere, Broca’s or Wernicke’s areas)</td>
<td>• Cranial nerve involvement, especially if multiple in similar region</td>
</tr>
<tr>
<td>• Neglect (non-dominant parietal)</td>
<td>• Diplopia, gaze palsy (CN 3, 4, and/or 6)</td>
</tr>
<tr>
<td>• Gaze deviation - towards side of stroke (frontal eye fields)</td>
<td>• Crossed face and body findings</td>
</tr>
<tr>
<td>• Homonymous hemianopsia (typically occipital cortex)</td>
<td>• Really severe bulbar involvement</td>
</tr>
</tbody>
</table>
Deficits by vascular territory

- **MCA**
  - Contralateral hemiparesis and hemi-sensory loss (face/arm > leg)
  - Dominant hemisphere = aphasia, non-dominant
  - +/- gaze preference toward side of brain lesion
  - +/- homonymous hemi/quadrantopsia (optic radiations)

- **ACA**
  - Contralateral leg weakness and sensory loss
  - Disinhibition, AMS/personality changes
  - Urinary incontinence

- **PCA**
  - Contralateral homonymous hemianopsia

- **Vertebro-basilar artery**
  - Contralateral hemiparesis (face=arm=leg)
  - Severe dysarthria, dysphagia
  - Ataxia/dysmetria, vertigo
  - Diplopia, nystagmus
  - Cranial nerve deficits
Pathophysiology of Ischemic Stroke

Ischemic (87% of total strokes (US))

- Large Artery Atherosclerosis: 30%
- Small Vessel: 20%
- Cryptogenic: 25%
- Cardioembolic: 20%
- 5% Unusual
Pathophysiology of Ischemic Stroke

Embolic
- Cardio-embolic: 20%
- Athero-embolic: 30%
- Embolic, unknown source*: ~25%
- Other (paroxysmal emboli, hypercoagulable state, dissection with embolism, etc): 5%

Non-embolic
- (small vessel, lacunar, thrombotic, atherothrombotic): 20%
Localization

Clues to lacunes

- Lacunar infarct = small infarct (2–20 mm) in the subcortical structures from occlusion of a single small perforating artery (deep cerebral white matter, basal ganglia, thalamus, or pons)

- No cortical features (*unless thalamic mimics)
## Classic Lacunar syndromes:

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Clinical findings</th>
<th>Typical location (vessels supplying)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pure motor (33-50%)</td>
<td>Motor deficit of face, arm, and mildly the leg; mild dysarthria</td>
<td>PL internal capsule (Lenticulostriate branches, MCA) or pons (pontine perforators, BA)</td>
</tr>
<tr>
<td>Ataxic hemiparesis (2nd most common)</td>
<td>Weakness and clumsiness of the arm and leg, often gait changes</td>
<td>Pons or PLIC</td>
</tr>
<tr>
<td>Clumsy hand-dysarthria</td>
<td>Ataxia, dysmetria, or dysdiadochokinesia of the distal upper limb with dysarthria</td>
<td>Basilar pons (pontine perforators, BA)</td>
</tr>
<tr>
<td>Pure sensory</td>
<td>Sensory deficit (numbness, tingling, pain, or burning) of face/arm/leg</td>
<td>Thalamus (PCA branches)</td>
</tr>
<tr>
<td>Mixed sensorimotor</td>
<td>Hemiparesis and hemibody sensory deficit</td>
<td>Lateral pons or thalamocapsular region</td>
</tr>
</tbody>
</table>

Bamford, J et al. *Stroke* 1987
ABCD² score

- Risk assessment tool to predict short term stroke risk after TIA
- Developed out of UCSF
- Optimized to assess the 48 hr (2 day) stroke risk, but also gives the 7 day and 90 day stroke risk
- Higher ABCD² scores are associated with greater risk of stroke after TIA
  - Low risk: hospital observation may be unnecessary without another indication
  - Moderate risk: consider hospital observation or urgent assessment and risk factor control
  - High risk: hospital observation often worthwhile

**ABCD² score**

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Value</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>≥ 60 years</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>&lt;60 years</td>
<td>0</td>
</tr>
<tr>
<td>BP</td>
<td>≥ 140/90</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>&lt;140/90</td>
<td>0</td>
</tr>
<tr>
<td>Clinical features</td>
<td>Unilateral weakness</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Speech disturbance without weakness</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>0</td>
</tr>
<tr>
<td>Duration of symptoms</td>
<td>≥ 60 minutes</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>10-59 minutes</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>&lt; 10 minutes</td>
<td>0</td>
</tr>
<tr>
<td>Diabetes hx</td>
<td></td>
<td>1</td>
</tr>
</tbody>
</table>

Score ranges 0 to 7

ABCD² score and risk calculation

<table>
<thead>
<tr>
<th>Risk group</th>
<th>Score</th>
<th>2 days</th>
<th>7 days</th>
<th>90 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>0-3</td>
<td>1.0%</td>
<td>1.2%</td>
<td>3.1%</td>
</tr>
<tr>
<td>Moderate</td>
<td>4-5</td>
<td>4.1%</td>
<td>5.9%</td>
<td>9.8%</td>
</tr>
<tr>
<td>High</td>
<td>6-7</td>
<td>8.1%</td>
<td>12%</td>
<td>18%</td>
</tr>
</tbody>
</table>

Higher risk features of TIA

• Stuttering symptoms
  • Concerning for recurrent occlusion and recanalization of vessel (high grade carotid stenosis or impending occlusion of small vessel)
    • ABCD³ gives 2 points if dual TIA (within last 7 days)

• Positive neuroimaging correlate
  • MRI with diffusion restriction (imaging stroke)
  • High risk carotid lesion: identification of >50% stenosis of ICA
  • ABCD³-I score gives 2 points for DWI positive lesion and 2 points for ICA stenosis >50%
### ABCD² vs ABCD³ score

<table>
<thead>
<tr>
<th>Criteria</th>
<th>ABCD²</th>
<th>ABCD³</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age ≥ 60 years</td>
<td>1 point</td>
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</tr>
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</tr>
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<td></td>
<td></td>
</tr>
<tr>
<td>- Unilateral weakness</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>- Speech disturbance w/o weak.</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Duration of symptoms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- ≥ 60 minutes</td>
<td>2</td>
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<tr>
<td>- 10-59 minutes</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Diabetes hx</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Dual TIA (hx preceding TIA 7d)</td>
<td>NA</td>
<td>2</td>
</tr>
<tr>
<td>Imaging: ipsilateral ICA ≥ 50% sten.</td>
<td>NA</td>
<td>2</td>
</tr>
<tr>
<td>Imaging: DWI correlate</td>
<td>NA</td>
<td>2</td>
</tr>
<tr>
<td>Total possible</td>
<td>0-7</td>
<td>0-13</td>
</tr>
</tbody>
</table>

**Kaplan Myer Survival Curves:**

Workup of possible stroke/TIA

- Urgent assessment:
  - Evaluation within 12hrs and all testing within 48hrs if possible
- Labs: CBC, electrolytes and Cr, fasting glucose, lipids, A1c*
- CT head, ±MRI
- Vessel imaging (Carotid ultrasound*, CTA, MRA, or cerebral angiogram)
  - *carotid U/S does not evaluate the posterior circulation
- ECG/telemetry, 30 day cardiac monitor
- TTE
Common Stroke Mimics

- 38.6% of acute stroke patients in a 6 hour time window were found to have a stroke mimic in a recent study (193/498 patients)

Rother et al. *Stroke* 2014
Treatment

• Acute treatments:
  • TPA – not given for TIA or minor stroke (NIH ≤ ~4)
  • Mechanical thrombectomy
• Long-term secondary prevention:
  • Antithrombotic agent
  • Hypertension control
  • Lipid control
  • Diabetes control
• Lifestyle modification:
  • Physical activity
  • Diet
  • Smoking cessation
  • Weight management
Antithrombotic agents
Depends on the etiology

- Thrombotic (small vessel):
  - Single antiplatelet agent*
    - If previously on nothing, start ASA 81mg
    - If already on ASA, advance to clopidogrel (consider 5 day overlap or load)
    - Dipyridamole ER with aspirin (Aggrenox) approved for prevention of recurrent stroke, however often poorly tolerated (headaches)
Antithrombotic agents

- CHANCE trial
  - Clopidogrel vs placebo + ASA x 21d
  - TIA and minor stroke w/in 24h
  - Stroke risk: PLX group 8.2%, PBO group 11.7%, p<0.001
  - Hemorrhage risk (systemic and ICH) 0.3% both groups
  - Applicable?

- POINT trial: active/ongoing trial sponsored by NIH
  - High risk TIA or minor stroke w/in 12h
  - Clopidigrel vs placebo + ASA x90 days
Antithrombotic agents

Depends on the etiology

- Cardio-embolic (AF, cardiac thrombus, mechanical valve):
  - Anticoagulation (warfarin, apixaban, rivaroxaban, dabigatran, edoxaban, heparin, fondaparinux, etc)
    - All patients with non-valvular atrial fibrillation should be anticoagulated to reduce stroke (CHADS2 or CHADSVASc score ≥2)
    - ASA is not really effective in atrial fibrillation
Oral AC vs Aspirin

- European Atrial Fibrillation Trial:
  - 1007 pt’s with non-rheumatic Afib and recent TIA/minor stroke, AC (warfarin, goal INR 3 (2.5-4%)) vs ASA 300mg vs placebo
  - Primary endpoint: Annual rate of vascular event (vascular death, stroke, MI, or systemic embolism)
    - 8% in AC group vs 17% in control group (HR 0.53), p=0.001
    - 15% in ASA group vs 19% in placebo (HR 0.83), NS difference, p=0.12
  - Low annual major bleeding risk both groups (AC 2.8% vs ASA 0.9% vs PBO 0.7%, p<0.001)
  - AC more effective than ASA
    - 90 events prevented if 1000 pt’s treated with AC for 1 year, major bleeds 21 per 1000 pt’s
    - Only 40 events prevented if 1000 pt’s treated with ASA for 1 year

Oral AC vs Aspirin

- Annual stroke rates
  - 4% in AC vs 12% in control group
    - HR 0.34, RRR 66%
    - p<0.0001 → highly significant!
  - 10% in ASA group vs 12% in placebo
    - HR 0.86, RRR 14%
    - p=0.31, NS difference

NOAC agents vs Warfarin for nvAF

• Individual NOAC agent studies were underpowered, trend toward benefit over warfarin*

![Graph showing comparison of NOAC agents vs Warfarin]

NOAC vs Warfarin Meta-analysis

- Primary endpoint: 19% reduced risk for stroke or systemic embolism, \( p<0.0001 \)
  - 8% reduced risk for ischemic stroke, \( p=0.10 \)
  - 51% reduced risk for hemorrhagic stroke, \( p<0.0001 \)
  - 10% reduced risk of death (all cause mortality), \( p=0.0003 \)
- 52% lower risk of ICH, \( p<0.0001 \), but 25% increased risk of GI bleed, \( p=0.043 \)

NOAC vs Warfarin Meta-analysis

- Aggregate risk of major bleeding with a non-significant trend toward lower risk (14% RRR, p=0.06)
- Individual trials major bleeding shows significantly lower in ARISTOTLE (Apixaban) and ENGAGE AF-TIMI 48 (Endoxaban), and trend lower for RE-LY (Dabigatran)

OR of GIB: apixaban 1.23, dabigatran 1.58, endoxaban 0.31, rivaroxaban 1.48

Antithrombotic agents
Depends on the etiology

- Athero-embolic (stenosis $\geq 70\%$ in supplying vessel, 50\% in ICA)
  - Dual antiplatelet (aspirin + clopidogrel) for 3 month period* (longer is harmful), followed by single antiplatelet lifelong
  - Symptomatic carotid stenosis is often recommended for surgical treatment (CEA, stent for selected patients)
    - Medical therapy can be used for high risk cases or poor surgical candidates
- Vertebral or intracranial atherosclerosis are treated with medical management initially
  - Stenting in this population is no longer recommended until failure of medical therapy, and is currently being studied for this population
Surgical intervention for carotid disease

• Symptomatic carotid stenosis >50% has been shown to benefit from urgent CEA (or CAS*)
• NASCET trial
  • CEA reduced ipsilateral stroke, major stroke, and death in patients with symptomatic carotid stenosis 70-99%
    • Ipsilateral stroke at two years: 26% medical arm vs 9% surgical arm (ARR 17%, p<0.001)
    • Major/fatal ipsilateral stroke, 13.1% medical arm vs 2.5% surgical arm (ARR 10.6%)
• ECST trial: showed benefit at ≥80% stenosis
• Follow-up analysis of the NASCET trial data showed more modest benefit for 50-69% stenosis
  • Ipsilateral stroke at 5 years 15.7% surgical arm vs 22.2% medical arm (ARR 6.5%, p=0.045), NNT 15
  • This same trial showed no statistical benefit for patients with <50% stenosis.
• Stenting is accepted for patients with high operative risk, due to major stroke and death rates being similar.
  • Classically, it is believed that CEA has higher MI risk and CAS has higher stroke risk

**Surgical intervention for carotid disease**

- Symptomatic carotid stenosis >50% has been shown to benefit from urgent CEA or CAS*, within 2 weeks
- NASCET trial
  - CEA reduced ipsilateral stroke, major stroke, and death in patients with symptomatic carotid stenosis 70-99%
    - Risk of any ipsilateral stroke at two years were **26%** in medical arm and **9%** surgical arm. ARR 17% (P<0.001).
    - For a major or fatal ipsilateral stroke, the corresponding estimates were 13.1% and 2.5% — an absolute risk reduction of 10.6%.
- ECST trial: showed benefit at ≥80% stenosis
- Follow-up analysis of the NASCET trial data showed more modest benefit for 50-69% stenosis
  - 5 year rate of ipsilateral stroke of 15.7% in surgical arm and 22.2% in medical arm. ARR 6.5% (P=0.045), NNT 15 (5 years)
  - This same trial showed no statistical benefit for patients with <50% stenosis.
- Stenting is accepted for patients with high operative risk, due to major stroke and death rates being similar. Classically, it is believed that CEA has higher MI risk and CAS has higher stroke risk

Kaplan Myer curves, >70% stenosis:

Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade stenosis
Surgical intervention for carotid disease

- Symptomatic carotid stenosis >50% has been shown to benefit from urgent CEA or CAS*, within 2 weeks
- NASCET trial
  - CEA reduced ipsilateral stroke, major stroke, and death in patients with symptomatic carotid stenosis 70-99%
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- Stenting is accepted for patients with high operative risk, due to major stroke and death rates being similar. Classically, it is believed that CEA has higher MI risk and CAS has higher stroke risk.

Kaplan Myer curves, >70% stenosis:

Stay tuned: There are ongoing trials looking at asymptomatic carotid stenosis (CREST-2), and there may be trials in the future for current medical management vs surgical intervention

Antithrombotics for the ‘other’ categories

- Very dependent on etiology
- Most often single antiplatelet is enough for secondary stroke prevention
- Ongoing ESUS trials comparing AC with ASA
- PFO closure?
  - Not typically recommended unless young patient with multiple recurrent strokes
Treatment of other modifiable risk factors:

- Hypertension control
  - Goal BP <140/90 for all stroke patients as outpatient
  - Permissive HTN while admitted or for initial 24-48 hours (perfusion of pneumbra)

- Lipid control
  - Goal LDL <100 for all stroke patients (high dose*)
  - <70 for athero-embolic strokes, or if significant other vascular risk factors

- Diabetes control
  - A1c as near-normal as possible, <7

- Evaluation for OSA (high comorbidity rate)

AHA guidelines for prevention of stroke stroke.ahajournals.org/content/45/7/2160
SPARCL trial, NEJM 2006
Lifestyle modification after stroke

AHA guidelines stress the importance of lifestyle modifications.

• Physical activity
  • 40 minutes of moderate to vigorous physical activity at least 3-4 days a week

• Diet
  • Mediterranean diet (emphasis on vegetables, fruits, and whole grains and includes low-fat dairy products, poultry, fish, legumes, olive oil, and nuts; limits intake of sweets and red meats) can reduce stroke up to 18%

• Smoking cessation

• Weight management (given effect on CV risk factors)
  • Alcohol use should be limited to light to moderate consumption (2 drinks per day for men and 1 per day for women)

AHA guidelines for prevention of stroke, stroke.ahajournals.org/content/45/7/2160
A few words about what we are doing for acute stroke therapies...
Stroke Therapy Timeline

- **Prayer**
  - Until 1996

- **IV tPA**
  - 1996

- **IA tPA (off-label)**
  - 1999

- **MERCI/ Penumbra**
  - 2004/2008

- **Solitaire / TREVO**
  - 2012
NINDS trial: tPA in stroke

Given within 3 hrs from LKN (select population eligible)

<table>
<thead>
<tr>
<th>tPA (%)</th>
<th>Placebo (%)</th>
</tr>
</thead>
</table>

**Favorable outcome at 3 mos**
- Rankin: 45 (improve by 1+ grades on mRS) 25
  NNT 3.1
- NIHSS: 34 21

**Poor outcomes**
- Sx ICH: 6.4 0.6 NNH 17.2**
- Mortality: 17 21 Lower?! (NS)

** NNH for worsened outcome on modified rankin related to SICH is between 29.7 and 40.1
ECASS-3 trial: tPA 3-4.5 hours

90 Day Outcomes:

<table>
<thead>
<tr>
<th></th>
<th>Placebo (%)</th>
<th>rtPA (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rankin 0,1</td>
<td>45</td>
<td>52</td>
</tr>
<tr>
<td>NIHSS 0,1</td>
<td>43</td>
<td>50</td>
</tr>
<tr>
<td>Mortality</td>
<td>8%</td>
<td>8%</td>
</tr>
<tr>
<td>Symptomatic ICH</td>
<td>4%</td>
<td>8%</td>
</tr>
</tbody>
</table>

NNT 14.2, p = 0.04
p = 0.04

Not that statistically significant!

Higher ICH risk both groups (longer time)

# patients treated per ICH that occurs:

25 12.5

Hacke, W. *NEJM* 2008
Large vessel occlusions

- Common: 40-50% of all ischemic stroke
- Severe: 5x higher mortality, 3-fold reduction in good outcome
- Respond poorly to intravenous thrombolytic (tPA)
  - Successful opening of occlusion by IV tPA:
    - Middle Cerebral Artery: 35%
    - Carotid Terminus: Less than 10%
- Successful opening of the artery associated with improved outcome
Successful opening of artery and good outcome

- 138 Patients
- Acute MCA occlusion
- All treated with IV-tPA

GOOD OUTCOME AT HOSPITAL DISCHARGE
8% Good Outcome If Artery Did Not Open
66% Good Outcome If Artery Did Open

Riedel et al. Stroke 2011
Time is brain

Probability of good clinical outcome over time to technically successful angiographic reperfusion

The typical LVO patient loses **2 million** neurons/min in the territory at risk

Khatri P et al. *Neurology* 2009
Saver J. *Stroke* 2006
Early Randomized clinical trials
NEW ENGLAND JOURNAL OF MEDICINE, FEB. 2013

3 clinical trials did NOT show benefit of endovascular stroke treatment compared to intravenous tPA treatment.

Why?

1. Poor patient selection (Not selected with CT angiography.)
   20% of patients randomized to IA had no thrombus

2. First generation mechanical devices did not work better than intravenous tPA.
   Devices successful 30-45% of the time.
   IV tPA successful 30-40% of the time.
Modern Randomized clinical trials
NEW ENGLAND JOURNAL OF MEDICINE, 2014-2015

- Improved Outcomes
- Faster times
- Careful selection
- Newer devices
- Improved study design

- MR CLEAN
- ESCAPE
- EXTEND-IA
- REVASCAT
- SWIFT PRIME

A Randomized Trial of Intraarterial Treatment for Acute Ischemic Stroke

Improved Assessment of Rapid Endovascular Treatment of Ischemic Stroke

Randomized Assessment of Rapid Endovascular Therapy for Ischemic Stroke

Thrombectomy within 8 Hours after Symptom Onset in Ischemic Stroke

Stent-Retriever Thrombectomy after Intravenous TPA vs. TPA Alone in Stroke
5 positive Thrombectomy trials in 2015

<table>
<thead>
<tr>
<th>Trial</th>
<th>Outcome</th>
<th>Control group (% with outcome)</th>
<th>Intervention Group (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MR CLEAN</td>
<td>MR 0-2</td>
<td>TPA* 19%</td>
<td>TPA*/INR 33%</td>
</tr>
<tr>
<td>ESCAPE</td>
<td>MR 0-2</td>
<td>TPA 29%</td>
<td>TPA/INR 53%</td>
</tr>
<tr>
<td>EXTEND-IA</td>
<td>MR 0-2</td>
<td>TPA 40%</td>
<td>TPA/INR 71%</td>
</tr>
<tr>
<td>SWIFT PRIME</td>
<td>MR 0-2</td>
<td>TPA 35%</td>
<td>TPA/INR 60%</td>
</tr>
<tr>
<td>REVASCAT (up to 6 hours)</td>
<td>MR 0-2</td>
<td>Control group* 29% (TPA in 80%)</td>
<td>INR group 45% (TPA in 70%)</td>
</tr>
</tbody>
</table>

TPA* If eligible
Number Needed to treat

In order to have one additional stroke patient be independent at 90 days

- MR CLEAN
- ESCAPE
- EXTEND-IA
- SWIFT-PRIME

Primary PCI vs. Thrombolysis for STEMI: Prevention of MI/Stroke/Death
New AHA Guidelines 2015

• Endovascular therapy with a stent retriever is recommended (Class 1 Level A)
  • Proximal MCA or ICA occlusion
  • Within 6 hours of symptom onset

• We have a New Standard of Care for Stroke!
Recent Case Example

• 65yo F, acute onset ALOC with bilateral upper ext weakness with vertigo at 8:30am.
• Outside ER, Telestroke activated
• NIHSS was 15
• IV tPA given ~11:00 am
• Worsened somnolence prior to transfer, intubated
• Transferred to OHSU directly to CT
Transferred to OHSU

- NIHSS on arrival 19X (X=intubated)
Angiography
Angiography
Post-Thrombectomy Run
CTH 24hr-Post Procedure

- TICI3 Revascularization (Complete) at 5 hours from onset
- After Extubation, NIHSS 1 for dysarthria
- 48 hours post, was NIHSS 0
Questions?