ACUTE STROKE: MANAGEMENT

Killian O’Rourke MD
Department of Neurology
Mater Misericordiae University Hospital, Dublin
The Definition of Stroke/ TIA

A clinical syndrome characterized by the sudden onset of a focal neurological deficit presumed to be on a vascular basis.
Differential Diagnosis: What else could it be?

- Diagnosis is made on history
- Don’t take what you are told for granted
“When is a door not a door????????……..”
Conditions Misdiagnosed as Stroke
N.B. NIHSS 1 - 4

- Migraine
- Syncope, Postural Hypotension
- Seizure
- Vertigo
- Transient Global Amnesia
- Anxiety/Hyperventilation
- Confusion
- Unexplained Fall
- Peripheral Nerve Palsy
- Structural Intracranial Lesions
- Metabolic Disorders
- CNS Infections
- MS
- Local Eye Disorders
What things do you need to know?

- **Onset**: sudden V gradual
- **Modalities Involved**: motor, speech etc.
- **Localisation**: anatomical area involved
- **Accompanying symptoms**: headache etc.
- **History**: seizures, migraines, etc.
THREE STROKE TYPES

Focal Brain Dysfunction

Ischemic Stroke
- Clot occluding artery
- 85%

Intracerebral Hemorrhage
- Bleeding into brain
- 10%

Subarachnoid Hemorrhage
- Bleeding around brain
- 5%

Diffuse Brain Dysfunction
CAUSES (ETIOLOGIES) OF ISCHEMIC STROKE: SIX MAIN CATEGORIES

OLDER PATIENTS (> 55)
- Large-artery atherosclerosis
- Small-artery disease

OLDER PATIENTS (> 55)
- Cardioembolism
- Hypotension

YOUNGER PATIENTS (≤ 55)
- Hypercoagulable states
- Nonatherosclerotic vasculopathies

Correct therapy depends on cause of stroke!
“cause” & “risk factor” are not synonymous—must treat both!
ACUTE ISCHEMIC STROKE (AIS) & TIA
LOW BLOOD FLOW TO FOCAL AREA OF BRAIN

- Pathophysiology:
  - Usually embolism
  - Sometimes thrombosis in situ

- Acute therapy:
  - Reperfusion (Thrombolysis or Thrombectomy)
  - Do NOT lower BP
  - Avoid aspiration / IV glucose

**Ischemic stroke** = Infarction with sequelae

**Transient ischemic attack** = No infarction and no sequelae
"TIME IS BRAIN: SAVE THE PENUMBRA"

Penumbra is zone of reversible ischemia around core of irreversible infarction—salvageable in first few hours after ischemic stroke onset

Penumbra damaged by:
- Hypoperfusion
- Hyperglycemia
- Fever
- Seizure
Identification of penumbra through MRI perfusion-diffusion mismatch or perfusion CT may replace time as the major indication for emergency acute ischemic stroke therapies.
Pooled analysis of IV rTPA in 2,776 subjects from NINDS, ECASS I, ECASS II, ATLANTIS (Lancet 2004;363:768-74)

Figure 3: Model estimating odds ratio for favourable outcome at 3 months in rt-PA-treated patients compared with controls by OTT

Adjusted for age, baseline glucose concentration, baseline NIHSS measurement, baseline diastolic blood pressure, previous hypertension, and interaction between age and baseline NIHSS measurement.
STROKE EMERGENCY BRAIN IMAGING: NONCONTRAST CT SCAN

Intracerebral Hemorrhage
- CT detects all ICHs immediately

Subarachnoid Hemorrhage
- CT detects 90% of SAHs; if SAH suspected & CT negative, must LP
60 y.o., left hemiparesis and neglect: onset 70 mins
57 y.o., aphasia + right face/arm weakness: onset 90 mins
73 y.o., aphasia + right hemiparesis: onset 2 hours
78 y.o., dense neglect, left hemiplegia: onset 3 hours
48y.o, Male

Sudden onset spinning had to grab the counter, vomiting, unable to walk, R neck pain at 16.00

Presented to A&E at 16.25 Dx ?Vestibular Neuronitis and referred medically
AIS EMERGENCY THERAPY:
IV TISSUE PLASMINOGEN ACTIVATOR (T-PA)

- Must give < 4.5 h—earlier you give it, better the outcome
- Stroke onset = last time known to be normal
- Do NOT give if INR > 1.7
- Do NOT give if BP > 185/110
- Correct glucose if > 8mMol
- Disability risk ↓ 30% despite ~5% symptomatic ICH risk
- Lawsuits for not giving >>> lawsuits for giving
ED: Acute Ischaemic Stroke (AIS) 24/7

I. Triage—10 min
- Review t-PA criteria
- Page acute stroke team
- Draw pre t-PA lab tests*

II. Medical Care—25 min
- Place O2, 2 NS IVs
- Obtain BP, weight, NIHSS
- Obtain 12-lead ECG
- Send patient to CT

III. CT & Labs—45 min
- Obtain lab results
- Read CT
- Return pt to ED

IV. Treatment—60 min
- Start IV t-PA
- Monitor for ICH sx
  - HTN, headache
  - N/V, ↓ neuro status

*FBC, PT/INR, glucose, cardiac enzymes, renal
OTHER AIS THERAPIES: MAYBE IA, YES ASA, NO HIGH-DOSE HEPARIN

- Intra-arterial t-PA
  - Only preliminary evidence to date, not FDA approved
  - Studies ongoing, esp. combined w/ IV t-PA

- MERCI or SOLITAIRE device
  - Mechanical embolectomy devices
  - Both FDA approved, but controlled trial results pending

Window for MCA occlusion 6 hours but do NOT preclude IV t-PA w/in 4.5 h
Window for Basilar Occlusion: “< 12 hours”

- Aspirin
  - Aspirin 325 mg per day begun within 48 h of stroke onset decreases morbidity & mortality (may begin 24 h after t-PA)

- Heparin(s)
  - Insufficient evidence to recommend routine use of high-dose IV heparin, LMW heparin, or heparinoid as Rx for AIS per se
The AIS-BP Relationship

In AIS, high BP is a response, not a cause—don’t lower it!

- BP increase is due to arterial occlusion (i.e., an effort to perfuse penumbra)
- Failure to recanalize (w/ or w/o thrombolytic therapy) results in high BP and poor neuro outcomes
- Lowering BP starves penumbra, worsens outcomes
AIS IS NOT A HYPERTENSIVE EMERGENCY!

AHA guidelines state “Do not Rx unless BP > 220/120,” but also state:
- No data to suggest 220/120 is dangerous & requires Rx
- Evidence that BP lowering worsens outcomes is concerning
- Goal is to avoid overtreating pts until definitive data available

- Only definite indications to ↓ BP emergently in AIS:
  - AMI, CHF, Aortic dissection, ARF, or HTN encephalopathy
  - Candidate for thrombolysis and BP > 185/110
MAY LOWER BP SLIGHTLY PRE T-PA

If all t-PA criteria met except sustained BP > 185/110:

- Ensure 2 IVs (NS @ 75 cc/h)
- Calm patient, empty bladder
- Recheck BP, lower slightly if necessary

- SBP > 220 or DBP > 120
  - No BP med, No t-PA
- SBP > 185 and ≤ 220 or DBP > 110 and ≤ 120
  - Lower BP pre-t-PA

Avoid excessive lowering of BP just to give t-PA—
“Don’t kill the penumbra to save the penumbra”
LOWERING BP IN T-PA PATIENTS

Labetalol 10-20 mg IV
- May repeat q 10-15 min
- Pre-t-PA: only use a 2nd dose only if necessary

GTN infusion
WORRYING ABOUT THE LUNGS: ASPIRATION, DYSPHAGIA, & OXYGEN

- Weak oropharyngeal muscles common
- Neurogenic dysphagia: liquids worse than solids (purees best)
- Stroke pts on ventilator: 2/3 mortality, most survivors disabled

Recommendations (science):
- Keep patient 100% NPO until evaluation
- Use NG feeding tube if necessary (& IV NS 75-125 cc/h)
- Evaluate with video fluoroscopy whenever possible

Recommendations (art):
- Maintain > 30°
- Maintain O2 sat. 95% w/ 2-4L O2
STROKE UNIT CARE: PREVENT COMPLICATIONS

- Aspiration (NPO until swallowing evaluation)

- Deep-vein thrombosis
  - Sequential compression devices (if stroke < 48 h)
  - Heparin 5000 q8h or enoxaparin 40 mg/d

- Urinary tract infection (avoid Foley catheters)
- Constipation (docusate sodium for all)

- Decubitus ulcers (move q2h, out of bed TID by day 2)
- Upper GI bleed (Ranitidine)

- Fever (paracetamol + antibiotics as indicated)
Beware: Recurrent stroke after TIA and minor stroke

Coull et al BMJ 2004
SECONDARY STROKE PREVENTION: ANTIPLATELET AGENTS FOR ARTERIAL DISEASE

- **Aspirin**
  - Prevents MI & stroke
  - Stroke rec 50-365 mg/d, but MI rec 75-162 mg/d
  - Low dose with less side effects, > 1200 mg/d ineffective
  - Enteric coating, NSAIDs may lessen efficacy

- **Clopidogrel 75 mg per day**
  - Prevents MI and stroke
  - Routine combination with aspirin not indicated in stroke pts – possibly justified in polyvascular cases
  - PPIs lessen efficacy

- **Aspirin / dipyridamole XR 25/200 twice daily**
  - Headache common side effect of dipyridamole
SECONDARY STROKE PREVENTION: WARFARIN FOR CARDIOEMBOLISM

- Underused for atrial fibrillation/flutter
- Starting dose 5 mg OD in elderly
- INR monitoring
  - Target 2.5, range 2.0-3.0 (mechanical HVR 2.5-3.5)
  - Reflects dose 2-3 days ago, stabilizes in 10-14 days
  - Vitamin K (greens, NG feedings, Ensure)
  - Other meds, EtOH, cranberry juice
  - Dose and formulation changes
- Limit holding for procedures (e.g., dental, GI, surgery)
SECONDARY STROKE PREVENTION: CAROTID STENOSIS PROCEDURES

- Carotid Endarterectomy (CEA)
  - Clear benefit if 70-99% stenosis
  - Some benefit if 50-69% stenosis
  - Accept complication rate < 6%

- Carotid Angioplasty/Stenting (CAS)
  - Now, option only in high-risk pts
    - Restenosis after CEA
    - Radiation-induced stenosis
    - Increased medical risk for CEA
    - Contralateral carotid occlusion
  - Cerebral protection devices improving, trials continue

% stenosis = (D-N)/D by contrast angiography
Proposed thrombolytic therapeutic paradigm in the 0 – 8 hour time-window

Patients with acute ischemic stroke 0 – 8 hour window from onset

No

Consider Endovascular therapy

Eligible for alteplase 0 – 4.5 hour window

Yes

Intravenous tPA 0.9 mg/kg

Intravenous tPA 0.9 mg/kg

Yes (30 min)

TCD/MRA/CTA monitoring

Large vessel occlusion

No

Standard therapy

Salvageable brain* PWI/DWI/ASPECTS

Yes

Standard therapy

Mechanical thrombectomy

* PWI/DWI or Angio/DWI or Angio/ASPECTS mismatch

Decision making strategy
1. Evidence based medicine – standard of care
2. Randomized clinical trial (DIAS 3&4, IMS III, etc)
3. Good clinical practice following predefined protocols of inclusion criteria & treatment modality