Stroke

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Emergency Medicine Training Hub
Ballarat & Grampians Region
5th December 2013
Learning objectives

Diagnostic reasoning for stroke
- Lacunar vs cortical
- Primary vs Secondary Haemorrhagic
- Traumatic vs atraumatic SAH

Stroke management in an ED – including thrombolysis

Pre reading
- Seizure Management Protocol
What is a stroke?

Extra point: What is the cat called?
Stroke

- Term is a literal translation of the Greek word for ‘Apoplexia’ and indicates a violent striking down by God.
Stroke

- Irreversible damage to the brain by vascular insult i.e. Bleed or infarct
- Symptoms last longer than 24 hours (vs TIA)
- PET / MRI scanning shows permanent neuronal damage in 30-50% despite apparent symptom resolution in ‘TIAs’* – suggests a scale of injury rather than discrete entities

*Stroke 2009;40:2276-2293
Mandatory statistical slide

- 1 in 10-20 deaths (dependent on country)
- 30% 1 year and 50% 5 year mortality (>45y.o.)
- Cost >$5bn/year
Types of stroke

Arterial vascular events:
- Ischaemic (see next slide)
- Haemorrhagic
  - Primary
    - Hypertensive (note the acute response)
    - Subarachnoid (likely hypertensive factors also)
  - Secondary
    - Ischaemic transformation
  - Vasculitis e.g. secondary to amphetamine use
Figure 1. Schematic drawing of hyperacute stroke in the left middle cerebral artery territory.

Infarct core

Penumbra

Benign oligemia

Bandera E et al. Stroke 2006;37:1334-1339

Copyright © American Heart Association
Stroke process ED (NICE UK)
Stroke process ED (NICE UK)

1. Suspected Stroke
   - Exclude hypoglycaemia.
   - Use the Face Arm Speech Test (FAST) to screen for diagnosis of Stroke.

2. Positive screen
   - Establish diagnosis rapidly using a validated tool e.g. ROSIER

3. Indications for immediate scanning
   - Thrombolysis if indicated (<3hr from symptom onset)
   - Scan immediately (ideally within the next slot or definitely within 1 hr)

4. Assessment for brain scanning
   - No immediate indications for scanning

5. Scan as soon as possible (within 24 hrs)

6. Admit to an acute stroke unit for specialist monitoring and treatment
ROSIER (Recognition of Stroke in the Emergency Room)

Assessment  Date: ____________________  Time: ____________________

Symptom onset  Date: ____________________  Time: ____________________

GCS  E=___  M=___  V=___  BP= ___ / ___  *BG= ________

*If BG < 3.5 mmol/L, treat urgently and reassess once blood glucose normal

Has there been loss of consciousness or syncope?  Y (-1)  □  N (0)  □

Has there been seizure activity?  Y (-1)  □  N (0)  □

Is there a NEW ACUTE onset (or on awakening from sleep)

I.  Asymmetric facial weakness  Y (+1)  □  N (0)  □

II.  Asymmetric arm weakness  Y (+1)  □  N (0)  □

III.  Asymmetric leg weakness  Y (+1)  □  N (0)  □

IV.  Speech disturbance  Y (+1)  □  N (0)  □

V.  Visual field defect  Y (+1)  □  N (0)  □

Total Score  ______ (-2 to +5)

Provisional diagnosis

□  Stroke  □  Non-stroke (specify) ____________________________

Note: Stroke is unlikely, but not completely excluded if total scores are ≤0.
Stroke process ED (NICE UK)

Suspected Stroke

Exclude hypoglycaemia.

Positive screen

Use the Face Arm Speech Test (FAST) to screen for diagnosis of Stroke.

Establish diagnosis rapidly using a validated tool e.g. ROSIER

Indications for immediate scanning

Thrombolysis if indicated (<3hr from symptom onset)

Assessment for brain scanning

Scan immediately (ideally within the next slot or definitely within 1 hr)

No immediate indications for scanning

Scan as soon as possible (within 24 hrs)

Admit to an acute stroke unit for specialist monitoring and treatment

Consider alternative diagnosis (stroke remains a possible diagnosis)
Stroke process ED (NICE UK)
Stroke process ED (NICE UK)

- long duration of symptoms
- depressed level of consciousness
- unexplained progressive or fluctuating symptoms
- Papilloedema or neck stiffness
- severe headache at onset
- history of trauma or falls
- on anticoagulant treatment or has a known bleeding tendency.
Ischaemic Stroke
Ischaemic stroke

- Anticipate treatments and/or complications
  - Large vessel
    - Swelling from oedema, drop GCS, require RSI
    - Possible treatment with interventional radiology
    - Large areas for bleeding with tPA – avoid?
    - May require or benefit from neurosurgical intervention
  - Small vessel – lacunar
    - Less severe event
    - Possibly thrombolysis?
Localising and differentiating - Cortical and lacunar stroke

- **Cortical - Large vessel**
  - Motor **and** sensory **and** cortical signs
    - Hemiparesis (Upper and lower suggests more than one large vessel)
    - Sensory unlikely to be complete loss, different to other side
    - Philosophising, attention (R), language (L) – ‘thinking’
  - Falling level of consciousness (airway)
Cortical or lacunar

- Lacunar – small vessel
  - Able to mentate
  - ‘The wiring’ for sensory and motor functions
  - Sensory or motor symptoms (can be large area)
  - ‘Stuttering chorus’ (multiple small vessels)
## Oxford / Bamford Stroke Classification

<table>
<thead>
<tr>
<th>Signs</th>
<th>Lacunar</th>
<th>Partial anterior circulation</th>
<th>Total anterior circulation</th>
<th>Posterior circulation</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Motor or sensory deficit</td>
<td>2 of following: motor or sensory deficit; higher cortical dysfunction; hemianopia</td>
<td>All of: motor or sensory; cortical; hemianopia</td>
<td>Isolated hemianopia; brain stem signs; cerebellar ataxia</td>
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</table>

The type of stroke is then coded by adding a final letter to the above:

- **I** – for infarct (e.g. TACI)
- **H** – for haemorrhage (e.g. TACH)
- **S** – for syndrome; intermediate pathogenesis, prior to imaging (e.g. TACS)
Cerebral Cortex and Associated Body Regions

- Sensory
- Motor

- Eye
- Nose
- Face
- Lips
- Jaw
- Teeth
- Gums
- Tongue
- Fingers
- Forefinger
- Hand
- Forearm
- Elbow
- Arm
- Trunk
- Hip
- Knee
- Toes
- Leg/general
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Cortical or lacunar?

- L sided weakness – face, arm and leg
- Patients thinking ability
  - Poor language skills / innattention
- Rapidly becoming drowsy
Cortical or lacunar?

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- Patients thinking ability
  - Poor language skills / innattention
- Rapidly becoming drowsy

LARGE VESSEL
Therefore . . .

- **ACA** – Contralateral leg weakness, possible emotional, long term memory issues

- **MCA** – Contralateral face and arm weakness, speech problems

- **PCA** – Ipsilateral homonymous hemianopia, cerebellar signs, brain stem signs
Ischaemia

- Some irreversible damage likely (PET/MRI)
- Time dependent events – penumbra
- Subsection of strokes likely to benefit from thrombolysis
  - Early to thrombolysis
  - No complicating factors (Diabetes)
  - Smaller area of infarct
# Stroke Thrombolysis – evidence for efficacy?

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<th>STEMI thrombolysis</th>
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<td>small number of patients studied (&lt;10,000 patients in RCTs)</td>
<td>large number of patients studied (&gt;60,000 patients in RCTs)</td>
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<td>most studies negative, positive studies (see next slide) not replicated with the same outcomes and methodology</td>
<td>every study across all populations positive benefit found</td>
</tr>
<tr>
<td>3-4.5h time frame for benefit (better if earlier)</td>
<td>clear mortality benefit (2%) and small functional benefit</td>
</tr>
<tr>
<td>few patients suitable for treatment</td>
<td>simple workup with well defined patient selection criteria (i.e. meets STEMI criteria)</td>
</tr>
<tr>
<td>complex workup with uncertain patient selection criteria</td>
<td>consistent pathological process (acute plaque rupture causing vascular occlusion)</td>
</tr>
<tr>
<td>inconsistent pathological process (a large number are embolic or unknown)</td>
<td>6 hour time frame for benefit</td>
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<tr>
<td>rapid response/ benefit not present (based on NINDS I in first 24 hours)</td>
<td>many patients suitable for treatment (unless at a center where PCI is preferred)</td>
</tr>
<tr>
<td>very high risk (at least 3% risk of death) — clear early harm</td>
<td>greater benefit for large infarcts</td>
</tr>
<tr>
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<td>rapid response/ benefit</td>
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Stroke thrombolysis - evidence

- NINDS I and II (1995)
- ECASS-III (2008)
- IST-3
NINDS I and II

● NINDS I
  ● Resolution of stroke or 4 point improvement (4 to 0 not the same as 22 to 18) at 24 hours – NO DIFFERENCE

● NINDS II
  ● favorable outcome at 3 months using a global endpoint derived from 4 assessment scales
  ● placebo group had more severe strokes at baseline
  ● Thrombolysis benefit for up to 3 hours — <90 min OR 1.71 (1.06-2.7) — 91-180 min OR 1.12 (0.71-1.76) (not significant)
ECASS-III (2008)

- primary outcome disability at 90 days
  - dichotomized as a favorable outcome (mRS score of 0 or 1) or an unfavorable outcome (mRS of 2 to 6)
  - favoured alteplase (52.4% vs. 45.2%; OR 1.34; 95% CI 1.02 to 1.76; P=0.04)
  - If mRS 0-2 compared with mRS 3-6 then no significant difference observed

- NB: mRS of 2 = mild disability
  6 = dead
IST-3 – use of rtPA after 3 hours all ages

- Multicentre international RCT
- A pilot phase (<10% total) was blinded and then in the main trial it was open label
- Originally planned for 6000 but recruitment insufficient
- Big differences in the sub-groups at baseline
- Only +ve results in the trial were in the sub-groups
IST-3 results

- Recruitment slow - 2 pts/yr/centre
- half over 80 years old, mainly treated at 4.2hrs (i.e. virtually all outside the current european licence (which is the point of the trial and contradicts previous trials)
- pts who got tPA more likely to go to HDU (24%v17%) than those who didn’t.
- High early deaths (11% v 7%) but then improved by 6 months (16% v 20%). Overall mortality was identical at 6 months
- found a **2% benefit in primary outcome** (alive and independent, 35% v 37%) at 6 months - No statistical significance. You could call this an NNT of 50 if it was real.
Using rtPA

- **DOSE**
- Give Heparin 5000 IU as bolus (why?)
- 0.9mg/kg alteplase (r-TPA) (maximum 90mg) over 60 minutes (10% given as a bolus)
Haemorrhagic conversion post rTPA

Arrow shows fluid level consistent with coagulopathy and increases risk of ongoing bleed.
Treatment of anticoagulation mediated ICH

- FFP 4 units
- Cryoprecipitate 1 unit
- Platelets 1 unit
- Vit K 5-20mg if warfarinised
- Bloods should be taken prior to reversal and for ongoing management of coagulopathy
  - X-match
What else can we do?

Solutions
Intravenous thrombolysis is the second most powerful The ultimate aim is to keep the time to treatment as short
acute ischaemic stroke (AIS) intervention available (after as possible
stroke unit care). The earlier treatment of AIS with
thrombolysis is initiated, the better the outcome!
Take every effort to shorten time to treatment
Most effective are:

Early recognition of stroke symptoms, including public
Rapid EMS dispatch
Implementation of pre-hospital protocols:
This resulted in a 4-fold increase in the absolute number of
significant increase in the proportion of patients with
intra-thrombolysis rates were seen in acute ischaemic stroke
patients transported by helicopter
The shortest hospital arrival times and highest

Establishment of stroke networks
Priority and direct transfer to specialised stroke
Management by multidisciplinary teams
Act fast to initiate treatment with thrombolysis as early as
possible
Stroke experts are needed
Joint teaching activities (national training program for stroke)
Annual meeting with all physicians in the area, involved in
stroke care for continuous training
Registries to evaluate the network
Improving AIS management pre-hospital
Use public awareness campaigns:
Target the general public as stroke witnesses
Symptom and risk factor awareness
Awareness to take action

Keep the message easy
Public awareness campaigns can increase ambulance
dispatches for stroke
Rapid patient recognition and reaction to stroke warning
signs
Immediate transfer to appropriate stroke centres
If available, use helicopter emergency service (HEMS):

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Ensure that many patients achieve a door-to-needle time (DTN) ≤60 min
Rapid implementation of existing in-house stroke protocols

Shorten time to treatment
Monitoring of processes to detect weaknesses and
possibilities for improvement
Pre-notification that patient is on the way and direct access
Rapid triage by emergency physician or paramedic before
arrival
Vital parameters stabilised (O2, temperature)
POC test for blood glucose (100-180 mg/dl) and INR
BP optimum (150-160 mmHg systolic)
NIHSS assessment
Priority CT access
Use of scales such as ASPECTS
Rapid read, images should always be analysed by the
neurologist and the radiologist
Set-up that allows weighing the patient, e.g. lying in CT
TeleConsultation: provide stroke expertise from a distance,
if no stroke expert is available close-by
TeleStroke can help to achieve treatment goals (best
within TeleStroke Units)

Other therapy

The initiation of an Acute Brain Care Unit (ABC-Unit) and aRapid laboratory testing and point of care (POC) test multidisciplinary treatment protocol significantly reduced the length of time between the arrival of the patient at the emergency room and initiation of treatment (from 86 to 26 minutes) 

The common goal of pre- and in-hospital management strategies is for all teams to work together as an integrated network

Target: Stroke9

Target: Stroke is a multidimensional initiative from the AHA/ASA to improve stroke care

Aim: to ensure that as many patients as possible with AIS achieve a DTN ≤60 min

10 key best practice strategies, associated with faster DTN:

Advance hospital notification by EMS
Rapid triage protocol and stroke team notification
Single call to active stroke team
Stroke tools
Rapid acquisition and interpretation of brain imaging

Follow-up will be after 1 year, in line with Get With The Guidelines-Stroke (GWTG-Stroke) data and rate of improvement in the DTN

References


Lambert Y. Presentation at the ESC in Hamburg, 2011


Audebert H. Presentation at the ESC in Hamburg, 2011.


Other therapy

Intravenous thrombolysis is the second most powerful acute ischaemic stroke (AIS) intervention available (after stroke unit care). The earlier treatment of AIS with thrombolysis is initiated, the better the outcome!

Keep the message easy

The ultimate aim is to keep the time to treatment as short as possible

Public awareness campaigns can increase ambulance dispatches for stroke

Rapid patient recognition and reaction to stroke warning signs

Rapid triage by emergency physician or paramedic before arrival

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Take every effort to shorten time to treatment

Most effective are:

Early recognition of education

Establishment of stroke

Priority centres or stroke units

Management by multidisciplinary team

Act fast to initiate treatment as early as possible

Stroke Symptom Identification

Interaction between blood, brain, and body

Regional committee with emergency medical services (EMS), emergency department (ED), neurology, radiology, rehabilitation teams, patient organizations. Ideally, emergency ambulance equipped with CT scanner, access to lab, and health authorities to organise stroke and point-of-care lab, care at the regional level (3 per year)

Joint teaching activities (national training program for stroke)

Annual meeting with all physicians in the area, involved in acute stroke care for continuous training

Registries to evaluate the network

Improving AIS management pre-hospital

Use public awareness campaigns:

Target the general public as stroke witnesses

Symptom and risk factor awareness

Awareness to take action

Rapid read, images should always be analysed by the neurologist and the radiologist

Set-up that allows weighing the patient, e.g. lying in CT

TeleConsultation: provide stroke expertise from a distance, if no stroke expert is available close-by

TeleStroke can help to achieve treatment goals (best within TeleStroke Units)

Decision to treat and initiation of the therapeutic regimen, e.g. with the bolus intravenous injection of iv rt-PA in the imaging suite

Decision whether to perform additional imaging or rescue treatment in the imaging department

Ensure that as many patients as possible with AIS achieve a door-to-needle time (DTN) ≤ 60 min

Rapid implementation of existing in-house stroke protocols

Other therapy

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**Solutions**
- Intravenous thrombolysis is the second most powerful acute ischaemic stroke (AIS) intervention available (after stroke unit care). The earlier treatment of AIS with thrombolysis is initiated, the better the outcome!
- Take every effort to shorten time to treatment
- Most effective are:
  - Early recognition of stroke symptoms, including public
  - Rapid EMS dispatch
  - Establishment of stroke centres or stroke units
  - Act fast to initiate treatment

**Keep the message easy**
- The ultimate aim is to keep the time to treatment as short as possible
- Public awareness campaigns can increase ambulance dispatches for stroke
- Rapid patient recognition and reaction to stroke warning signs

**Shorten time to treatment**
- Monitoring of processes to detect weaknesses and possibilities for improvement
- Pre-notification that patient is on the way and direct access to imaging
- Rapid triage by emergency physician or paramedic before arrival

**Intravenous thrombolysis**
- PA, rt-PA, or tissue plasminogen activator
- Best in stroke within 1 hour
- Highly effective for AIS
- Decision whether to perform additional imaging or rescue treatment in the imaging department
- Ensure that as many patients as possible with AIS achieve a door-to-needle time (DTN) ≤60 min

**Rapid implementation of existing in-house stroke protocols**

**Keeping the public informed**
- Use public awareness campaigns:
  - Improving AIS management pre-hospital1-5
  - Target the general public as stroke witnesses
  - Use public awareness campaigns:
  - Symptom and risk factor awareness
  - Awareness to take action

**Rapid in-hospital diagnosis and treatment**
- Optimisation of infrastructure will:
- Increase thrombolysis rates
- Improve safety and reduce risk of bleeding complications

**Other therapy**

How many words dedicated to Stroke unit care?
How many words dedicated to Stroke unit care?

Answer: 4
Who edits the stroke forum website?
Who edits the stroke forum website?
Causes of morbidity and mortality in Stroke

- Mass effect and local noxious effect of insult
  - Theoretically treated in thrombolysis
- Pneumonia
  - Aspiration
  - Non-aspiration
- Venous thrombosis
- Falls
Stroke Units

- Reduced probability of death or being disabled (odds ratio 0.81, 95% CI 0.72-0.91; p=0.0001)

- The potential benefit was significant across all age ranges and clinical characteristics, except for unconsciousness

- The difference in survival between the two groups was most pronounced during the first month after admission.

- More intense the stroke care is, the better the outcome.
Of note on stroke forum site:

Benefits of stroke units

- Improved quality of care
- Facilitated patient access to thrombolysis and specialised stroke care
- Timely evaluation
- Improved early survival across age groups
- More cost effective than care on other hospital wards/teams
- Reduced incidence of post-stroke complications, such as urinary tract infections, pneumonia, and death.
Of note on stroke forum site:

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Other therapy via ED

- Telemedicine (largely linked to stroke thrombolysis)
- Head up
- Rails up
- NBM, speech therapy

Neurosurgery - Hemicraniectomy

- Prevents coning
- Allows perfusion in penumbra
- Selection of patients
  - Right brain - Hemiparesis and language deficits (less appropriate)
  - Low falls risk
Treatment of hypertension in ischaemic stroke – rarely req

- Treatment if syst BP >220 mmHg or dias. >120 mm Hg.
- For thrombolysis syst BP <185 mm Hg for 24 hours.
- Agents
  - Labetalol 10-20mg titrate over 2mins rpt 20mins
  - Nicardipine 5-15 mg/h titrate in 2.5mg adjustments every 5 mins
  - Sodium nitroprusside 0.5 mcg/kg/min IV infusion
TIA
TIA

- Stroke versus TIA
- Stroke WHO 1970s - "neurological deficit of cerebrovascular cause that persists beyond 24 hours or is interrupted by death within 24 hours".
- TIAs <24 hours of neurological deficit
  - They are associated with a significant increase in the risk of subsequent stroke, recurrent TIA, cardiovascular events or death.
Risk of stroke is increased after a TIA
Assessing risk

- ABCD-2
  - Admit – Investigation next 24 hours
  - Discharge – Investigations in next week
- CHADs / CHADs vasc
- ROSIER
Stroke mimics

- Hypoglycaemia
- Syncope
- Seizures
- Sepsis
- Infective endocarditis = suspect if fever, IVDU, or heart murmur/valvular heart disease
- Space occupying lesion
- Subdural haematoma
- Anticoagulation complication e.g. intra cerebral haemorrhage
- Temporal arteritis.
TIA process ED (NICE UK)

Suspected TIA

- Neurological symptoms fully resolved
  - Yes
  - No
    - See stroke algorithm

- Is history compatible with TIA?
  - Yes
  - No
    - Consider alternative diagnosis

Start Aspirin 300 mg and consider the following:
- treatment with statins
- blood pressure management
- lifestyle management.

Assess risk of stroke using a validated scoring system such as ABCD²

- ABCD² <4
  - Specialist assessment and investigation within 1 week
  - Best medical treatment (e.g. control of blood pressure, antiplatelet drugs and cholesterol lowering through diet and drugs and smoking cessation)

- ABCD² ≥4
  - Specialist assessment and investigation within 24 hours
  - Best medical treatment (e.g. control of blood pressure, antiplatelet drugs and cholesterol lowering through diet and drugs and smoking cessation)

Where
# ABCD² Score for TIA

Estimates the risk of stroke after a TIA.

<table>
<thead>
<tr>
<th>Feature</th>
<th>Value</th>
<th>Points</th>
</tr>
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<tbody>
<tr>
<td>Age ≥ 60</td>
<td>YES</td>
<td>+1</td>
</tr>
<tr>
<td>BP ≥ 140/90 mmHg</td>
<td>YES</td>
<td>+1</td>
</tr>
<tr>
<td><strong>Clinical Features of the TIA</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unilateral Weakness</td>
<td></td>
<td>-2</td>
</tr>
<tr>
<td>Speech Disturbance without Weakness</td>
<td></td>
<td>+1</td>
</tr>
<tr>
<td>Other Symptoms</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td><strong>Duration of Symptoms</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;10 Minutes</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>10-59 Minutes</td>
<td></td>
<td>+1</td>
</tr>
<tr>
<td>≥ 60 Minutes</td>
<td></td>
<td>+2</td>
</tr>
<tr>
<td><strong>History of Diabetes</strong></td>
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According to the validation study, 4-5 points: Moderate Risk.

- 2-Day Stroke Risk: 4.1%.
- 7-Day Stroke Risk: 5.9%.
- 90-Day Stroke Risk: 9.8%.
### ABCD^2 Score for TIA

**Estimates the risk of stroke after a TIA.**

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<td>+1 NO</td>
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#### Note 2 day risk:

- 3 or less = 1%
- 4 or 5 = 4.1%
- 6 or 7 = 8.1%

5 points

According to the validation study, 4-5 points:
- Moderate Risk
  - 2-Day Stroke Risk: 4.1%
  - 7-Day Stroke Risk: 5.9%
  - 90-Day Stroke Risk: 9.8%
Beginning Warfarin (not for ED)

### CHA₂DS₂-VASc Score for Atrial Fibrillation Stroke Risk

Calculates stroke risk for patients with atrial fibrillation, possibly better than the CHADS₂ score.

<table>
<thead>
<tr>
<th>Age in Years</th>
<th>&lt;65</th>
<th>65-74</th>
<th>≥75</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>Male</td>
<td>Female</td>
<td></td>
</tr>
<tr>
<td>Congestive Heart Failure History</td>
<td></td>
<td>+1</td>
<td>NO</td>
</tr>
<tr>
<td>Hypertension History</td>
<td>+1</td>
<td>YES</td>
<td></td>
</tr>
<tr>
<td>Stroke/TIA/Thromboembolism History</td>
<td>+2</td>
<td>NO</td>
<td></td>
</tr>
<tr>
<td>Vascular Disease History</td>
<td>+1</td>
<td>YES</td>
<td></td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>+1</td>
<td>NO</td>
<td></td>
</tr>
</tbody>
</table>

**4 points**

Stroke risk was 4.0% per year according to Yip et al's 2010 stroke study and the European Society of Cardiology's guidelines.

One recommendation suggests a 0 score is "low" risk and may not require anticoagulation; a 1 score is "low-moderate" risk and should consider antiplatelet or anticoagulation, and score 2 or greater is "moderate-high" risk and should otherwise be an anticoagulation candidate.

Consider falls risk . . .
Admit the following patients:

- Any patient with ABCD2 scores ≥ 4
- Crescendo TIA - 2 or more TIA in a week
- Continuing symptoms or residual deficit
- CT finding of stroke or other significant abnormality
- Known severe stenosis in a vascular territory corresponding to the TIA symptoms
- Those already taking anti-platelet therapy
- Suspected cardiac source of emboli (e.g. valvular disease or replacement, AF, MI)
- AMI < four weeks ago - needs inpatient echocardiogram
- Carotid bruit - needs inpatient Carotid Doppler
- Persistent (three or more readings) hypertension > 200 systolic +/or 120 diastolic
- Those with TIAs who had severe symptoms/ signs, even if fully recovery
- Diagnostic uncertainty
- Otherwise unwell, unstable diabetes or multiple active co-morbidities or unsafe for discharge.
Discharge

- Antiplatelet therapy
- TIA referral form

- Imaging
  - Echocardiogram
  - Carotid Doppler

- Bloods
Antiplatelet therapy:

- Start aspirin 150mg od and dipirydamole 200mg SR bd unless allergic to aspirin - give clopidogrel 75mg od (no dipirydamole)
- **or** patient has AF - persistent or paroxysmal - refer CAPAC for enoxaparin and to start warfarinisation unless contra-indicated (see AF guideline).
- If ABCD <4 and patient is already taking aspirin - either adding dipirydamole SR 200mg to aspirin or changing aspirin to clopidogrel 75mg is appropriate.
Haemorrhagic Stroke
Types of stroke – arterial event

- **Ischaemic**
- **Haemorrhagic**
  - Primary
    - Hypertensive (not the acute response)
    - Subarachnoid
  - Secondary
    - Isch. transformation, AVM, tumours, coagulopathy
    - Cerebral Amyloid (accumulation, increases with age)
    - Vasculitis e.g. secondary to amphetamine use
Hypertensive Stroke

- Basal Ganglia / Putamen (>50%)
- Thalamus
- Pons
- Cerebellum
Hypertensive Stroke

A – Cerebral lobes

B – Basal Ganglia

C – Thalamus

D - Pons

E - Cerebellar

Imaging

- CT
  - Non-contrast for initial diagnosis (ischaemic vs bleed, other)
- CT with contrast
  - For those patients at risk of further bleeding
- MRA
  - Good screening test for vascular lesions esp young, non-hypertensive pts
Early complications in ED

- 38% hematoma expansion is noted within three hours of ICH onset and that hematoma volume is an important predictor of 30-day mortality (Brott et al 1997; Qureshi et al 2005).
- Headache and Nausea and vomiting (3x isch stroke)
- Neurological/GCS deficit
Treatment of BP in hypertensive bleed

Rarely required

- If systolic BP >200 mm Hg or MAP >150 mm Hg: IV infusion protocol

- If systolic BP >180 mm Hg or MAP is over 130 mm Hg and:
  - raised ICP then as above and maintain a CPP of 60 mm Hg
  - no evidence raised ICP then consider modest reduction of BP (target MAP of 110 mm Hg or target BP of 160/90 mm Hg) and closely monitor

- In patients presenting with a systolic BP of 150 to 220 mm Hg, acute lowering of systolic BP to 140 mm Hg is probably safe (?required)
Case 1
PF - 64 y.o. male

- History of liver failure likely secondary to alcohol. (Not previously under gastr.).
- Had a fall - weak on right side RUL/RLL (4/5).
- Petechiae noted on thorax, neavae on chest

Investigations of choice?
### U&Es, LFTs

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
<td>135 mmol/L (136-146)</td>
</tr>
<tr>
<td>Potassium</td>
<td>2.9 mmol/L (3.5-5.0)</td>
</tr>
<tr>
<td>Chloride</td>
<td>95 mmol/L (95-110)</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>29 mmol/L (22-31)</td>
</tr>
<tr>
<td>Urea</td>
<td>3.5 mmol/L (3.0-10.0)</td>
</tr>
<tr>
<td>eGFR</td>
<td>&gt;^90 mL/min/1.73m2 (&gt; 60)</td>
</tr>
<tr>
<td>Creatinine</td>
<td>52 umol/L (60-110)</td>
</tr>
<tr>
<td>Total Bilirubin</td>
<td>80 umol/L (&lt; 20)</td>
</tr>
<tr>
<td>Conjugated Bilirubin</td>
<td>54 umol/L (0-7)</td>
</tr>
<tr>
<td>Alanine Aminotransferase</td>
<td>45 U/L (&lt; 45)</td>
</tr>
<tr>
<td>Aspartate Aminotransferase</td>
<td>123 U/L (&lt; 35)</td>
</tr>
<tr>
<td>Alkaline Phosphatase</td>
<td>163 U/L (35-110)</td>
</tr>
<tr>
<td>GGTP</td>
<td>173 U/L (&lt; 50)</td>
</tr>
<tr>
<td>Total Protein</td>
<td>68 g/L (65-85)</td>
</tr>
<tr>
<td>Albumin</td>
<td>26 g/L (36-48)</td>
</tr>
<tr>
<td>Globulin</td>
<td>42 g/L (22-38)</td>
</tr>
</tbody>
</table>
## FBC

<table>
<thead>
<tr>
<th>Haemoglobin</th>
<th>9.3 g/dL (13.0-18.0)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haematocrit</td>
<td>28.1 % (40.0-54.0)</td>
</tr>
<tr>
<td>Red Cell Count</td>
<td>2.81 x10*12/L (4.50-6.50)</td>
</tr>
<tr>
<td>MCV</td>
<td>100 fL (80-96)</td>
</tr>
<tr>
<td>MCH</td>
<td>33.1 pg (27.0-32.0)</td>
</tr>
<tr>
<td>MCHC</td>
<td>33.1 g/dL (32.0-36.0)</td>
</tr>
<tr>
<td>White Cell Count</td>
<td>5.9 x10*9/L (4.0-11.0)</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>2.9 x10*9/L (2.0-8.0)</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>1.8 x10*9/L (1.0-4.0)</td>
</tr>
<tr>
<td>Monocytes</td>
<td>1.1 x10*9/L (&lt; 1.0)</td>
</tr>
<tr>
<td>Basophils</td>
<td>0.1 x10*9/L (&lt; 0.2)</td>
</tr>
<tr>
<td>Eosinophil Count</td>
<td>0.1 x10*9/L (&lt; 0.5)</td>
</tr>
</tbody>
</table>

| Platelet Count | 65 x10*9/L (150-450) |
Clotting

Platelet Count: 65 x10^9/L (150-450)
APTT: 33 secs (26-36)
Prothrombin Ratio (INR): 1.5 (< 1.3) (72.4)
Fibrinogen: 1.6 g/L (2.0-4.0)
TCT: 19 seconds (16-22)
Case 2
92 yo female

- ‘Thump’ heard in grandma’s room (generally independent)
- Grandma on floor
- Laceration to occiput, laying on her back
- GCS 3 for 10 minutes
- GCS 14 on arrival in ED (‘Collared’)
- Vomited x1 in ED
Investigations

Sodium  140 mmol/L(136-146)
Potassium  4.6 mmol/L(3.5-5.0)
Chloride  107 mmol/L(95-110)
Bicarbonate  20 mmol/L(22-31)
Urea  8.2 mmol/L(3.0-10.0)
eGFR  48 mL/min/1.73m2(> 60)
Creatinine  89 umol/L(40-80)

Haemoglobin  14.8 g/dL(11.5-16.5)
Haematocrit  43.4 % (37.0-47.0)
Red Cell Count  4.64 x10*12/L(3.80-5.80)
MCV  94 fl(80-96)
MCH  31.9 pg(27.0-32.0)
MCHC  34.1 g/dL(32.0-36.0)

Neutrophils  11.8 x10*9/L(2.0-8.0)
Lymphocytes  4.2 x10*9/L(1.0-4.0)
Monocytes  1.2 x10*9/L (< 1.0)
Basophils  0.0 x10*9/L (< 0.2)
Eosinophil Count  0.2 x10*9/L (< 0.5)

Platelet Count  116 x10*9/L(150-450)
ECG

Bandwidth: 0.50-20 Hz

10 mm/mV  25.0 mm/s
CT Angiogram
CTAngiogram
Subarachnoid haemorrhage

- Non-traumatic vs traumatic
- ?collapse with head injury or subarachnoid with collapse
Subarachnoid haemorrhage - atraumatic

- 20% present with missed ‘herald bleed’
- Re-bleeding in 15% 1-2/52, 40% by 4/52

Symptoms

- sudden onset severe occipital is classic
- suspect in any headache that is unusual for the patient
- sentinel headache (40%)
- transient or persistent loss of consciousness
- vomiting
- seizure or posturing
- meningism
- ophthalmoscopy: subhyaloid retinal haemorrhage, papilloedema
- neurological signs NB: classic CN palsies (e.g. III nerve palsy in large ACOM aneurysm)
- risk factors (next slide)
Predisposition

- Traumatic
  - Older/falls risk
  - Syncope
  - Alcohol/violence

- Atraumatic - Aneurysmal vs non-aneurysmal
  - Younger
  - Hypertension, smokers
  - Genetic predisposition e.g.
    - Berry Aneurysms (ANIB genes 1-11) – ‘familial’
    - Polycystic kidneys
    - Ehlers Danlos Syndrome, fibromuscular dysplasia + others

- CNS disease
Work-up

- Collapse ? Cause

- CT (0-2% false negative first 12 hours, 0-7% upto 24 hrs, 50% at 48 hours)
  - Time to presentation of sentinel bleed may affect accuracy of CT

- LP
  - Overt haemorrhage
  - Xanthachromia (most sensitive at 12 hours)
Circle of Willis

Just before you answer . . .
Circle of Willis – name the bits
Circle of Willis – SAH percentage by anatomy

Most common 60% and vast majority relating to the Anterior cerebral artery

40% in PCOM (note overlap with above)

5-20% posterior circulation
CT for SAH

- Degree of confidence
- Nonenhanced CT for the initial evaluation
- The sensitivity is 93-100% in patients presenting with SAH within 24 hours of symptom onset.
  - 0-7% false-negative rate during this period.
  - 5 days, the sensitivity is approximately 85%
  - 1 week, it is approximately 50%.
What are we looking for on CT?
Blood within the suprasellar cistern
Blood within the anterior inter-hemispheric fissure

Blood within the quadrigeminal cistern
Blood within the anterior interhemispheric fissure

Corpus callosum

Third ventricle

Blood within the Sylvian fissure

Blood within the quadrigeminal cistern
Pathophysiology of symptoms in SAH

- Vascular spasm
- Microcirculation thrombo-embolism
Treatment

ED

- Resuscitation
- BP control
- ICP control – clinically and radiologically detected
  - Head up 30°
  - Mannitol 1g/Kg, usually 1l of 10% (0.25 to 1.5g/kg)

Neurosurgical

- Medical treatment e.g. Nimodipine 60mg 4 hourly
- Physiological management as above - (invasive monitoring)
- MRA, Angiography
- Clips and coils
LP

- May require sedation or RSI for procedure
- Overt haemorrhage or Xanthachromia
  - Any RBCs in 3rd tube should increase suspicion of SAH (RBCs <100 tube 3 – unlikely SAH★)
  - Xanthachromia takes 12 hours to develop (RBCs denature ➔ oxyhemoglobin and bilirubin - yellow hue)
- LP before CT? - normal mentation, no meningism, focal neurology or signs of raised intracranial pressure, no CIs to LP

Prognosis – 30-90% mortality

- Age
- Size of bleed >/<1mm
- Site – Basilar > all others
- Neurological deficit, incl seizures
- Comorbidities
- Complications
What is this . . .
Extra-dural haemorrhage
General Stroke Management

- Optimise physiology – **Resuscitation**
- Intubation
  - Low GCS (<8) or respiratory failure (General)
  - Signs of raised intracranial pressure (for disposition to neurosurgery)
    - Raise foot of bed
    - Mannitol 1-1.5g/kg
    - Maintain CO$_2$ in low normal range
- Aim for MAP below 130mmHg and cerebral perfusion pressure >60mmHg (Broderick et al 1999)
Questions?
Summary

- Resuscitation and optimisation of physiology
- Maintain CPP (MAP-ICP) of 60mmHg
- Differentiating lacunar vs cortical stroke may affect disposition (thrombolysis . . .)
- Primary vs secondary, traumatic vs non-traumatic
Other reading and resources

- http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2291314/
- Spiros talk on education
- CG68 NICE guidelines

Also see lecture notes for each slide for references and notes