Acute stroke treatments

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Contents

- Management of acute ischaemic strokes
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  - Carotid Endarterectomy
- Management of Primary intracerebral haemorrhages)
  - Hypertension
  - Secondary to Oral anticoagulants
- Intracranial cavity and ICP
- Decompressive hemicraniectomy
- Suboccipital decompression craniectomy
Learning objectives:

- Identify acute treatments for ischaemic and haemorrhagic strokes
- List stroke thrombolysis inclusion and exclusion criteria
- Have the knowledge to rapid access patients for potential thrombolysis
- Describe how rtPA therapy works, its side-effects and complications
- Devise appropriate nursing care for patients after thrombolysis
- Understand the need for vascular imaging and carotid endarterectomy
- List the aetiology for primary intracerebral haemorrhages
- Devise appropriate nursing care for patients with haemorrhagic strokes
- Understand Intracranial pressure, most common hearniations and recognise its signs and symptoms
- Devise appropriate nursing care for patients post-decompressive craniectomy
- Be aware of current research and ongoing trials

Ischaemic stroke
The objective is to avoid lesion enlargement
Thrombolysis in acute ischaemic stroke

- Thrombo = Clot (fibrin)
- Lysis = Dissolve
Alteplase

- Recombinant tissue plasminogen activator (r-tPA) – a protein
- Thrombolytic agent
Thrombolysis in acute ischaemic stroke

Conversion of plasminogen to plasmin

Plasmin breaks down fibrin in clot

Breaks up thrombus

Blood flow through blocked vessel
Thrombolysis in acute ischaemic stroke

- ECASS III (2008): benefit in giving up to 4.5 hours of onset
- IST-3 (2012): confirmed benefits of prior trials for up to 4.5 hours of onset; advised thrombolysis aged over 80 years old

(WAKE-UP and EXTEND (2019): 4.5 to 9hrs after symptom onset or wake-up positive results)
Thrombolysis in acute ischaemic stroke

Modified Rankin Score

For 1 patient to have an excellent outcome (mRS 0-1):

- 5 patients NTT within 1.5hrs
- 9 patients NTT between 1.5 – 3hrs
- 15 patients NTT between 3 – 4.5rs
Thrombolysis in acute ischaemic stroke

**Inclusion criteria**

- Clinical presentation of a stroke
- Neurological deficit not improving
- Onset of symptoms less than 4.5 hrs (BAO ≤ 12 hours)
- Age ≥ 18
- No haemorrhage or early ischaemic changes on CT head
- No contraindications

- Up to 20% of stroke patients are eligible for thrombolysis
- Only 12% receive treatment
Thrombolysis in acute ischaemic stroke

**Exclusion criteria**

- **BP persistently > 185/110**
- Any evidence of active bleeding
- Severe sudden onset headache at onset of symptoms (suggestive of SAH)
- Previous stroke, serious head injury, GI or urinary tract haemorrhage, surgery or significant trauma in the last 3 months
- Neoplasm with increased bleeding risk
- Previous history of spontaneous ICH, pancreatitis, oesophageal varices, active hepatitis, portal hypertension or liver cirrhosis
- AVM or aortic aneurysm
- LP within the last 10 days
- External cardiac massage or obstetric delivery within the last 10 days
- Vitamin K antagonist (eg Warfarin) and INR > 1.7; DOACs
- Unfractioned heparin within the last 24 hours and APTT abnormal
- **Treatment dose LMWH within the last 48 hours**
- Known (or strongly suspected) iron deficient anaemia, thrombocytopenia or platelet defect
Thrombolysis in acute ischaemic stroke

**Rapid Assessment**

- History of symptoms and onset time – “*is this a stroke?*”
- Past medical history
- ABCD assessment – treat if necessary
- NIHSS score
- Baseline neurological observations: Vital signs, Glasgow Coma Scale, Glucose
- Bloods (FBC, U&E, Lipids, LFTs, Coagulation, Group & Save)
- Bedside INR (if on Warfarin)
- 2 x large bore Peripheral cannulas
- CT head
Thrombolysis in acute ischaemic stroke

**Treatment**

- Re-check neurological observations
- Severe Hypertension
  - Labetalol 10-20mg IV bolus every 10 min (max 300mg)
  - Labetalol infusion 2-8 mg/min
  - GTN infusion 50mg in 50mls, 1 to 10 ml/hr
Thrombolysis in acute ischaemic stroke

Treatment

- Consent
- Establish patient weight and calculate dose: 0.9mg/Kg (max 90mg)
- Prepare and Administer medication (do not shake via swirl only):
  - 10% bolus (over 1-2 min)
  - 90% infusion (over 1 hr) via an infusion pump

Eg. 70 kg
0.9 mg ---- 1 Kg
X ---- 70Kg x = 0.9 x 70 = 63 mg (ml)

10% of 63 = 6.3 or 6 mg (ml)
90% of 63 = 57mg (ml)
Thrombolysis in acute ischaemic stroke

Treatment
Thrombolysis is an acute treatment option for which type of stroke?

A – Ischaemic stroke
B – Haemorrhagic stroke
46 year old female, right handed

**HPC:** Last seen well at 11:00 by her partner. At 12:00 while having lunch, sudden onset of left sided weakness.

**PMH:** smoker

**Medications:** Nil

**Allergies:** nil

**FM:** mother MI at 60

**SH:** lives with husband and 2 children. Works as a cleaner

Paramedics arrived on scene and took patient to the nearest HASU, alerting ED

**ED arrival:** 13:15
Case study 1

- LUL, severe weakness (power 1/5)
- LLL, no response (power 0/5)
- Left homonymous hemianopia
- Left facial droop
- Right gaze preference
- NIHSS 16

**Vital signs:**
- E4 V4 M6
- Blood Pressure 200/110
- HR 75 Sinus rhythm
- SaO2 98% in Air
- RR 15
- T 36.5ºC
- BM 6.5 mmol/L
Can we thrombolyse this patient right now?

A – Yes, absolutely. Let’s do it!

B – No way

C – I have no idea
Case study 1

NIHSS 16

Vital signs:
E4 V4 M6
Blood Pressure 200/110 HR 75 SR
SaO2 98% in Air RR 15 T 36,5ºC BM 6.5 mmol/L

Can we thrombolyse this patient right now?
A – Yes, let’s do it!
B – No way
C – I have no idea
Thrombolysis in acute ischaemic stroke

Treatment

- Monitor side-effects/complications and treat
  - Allergic rash
  - Bronchospasm
  - Severe Hypotension
  - Anaphylactic reaction
  - Bleeding/ICH (including bruises)
- Stop rtPA if severe hypotension, bronchospasm, anaphylactic shock
- Management of suspected ICH as per local policy

- ABCD
- Chlorpheniramine 10mg IV
- Hydrocortisone 200mg IV
- Salbutamol 2.5 – 5 mg
- Adrenaline 0.5mg im (0.5 ml of 1:1000)
- Fluid challenge
Transfer to HASU

- Flush the entire IV line at the end of the infusion
- Avoid all treatments/procedures with associated risk of bleeding (eg. NG tube, NSAIDs, anticoagulants)
- Repeat Brain imaging at 24 hours → antiplatelet therapy
Repeat brain imaging if ICH is suspected

NIHSS at 2, 24, 48 and 72 hours

Neurological observations as per local policy (Glasgow Coma Scale, pupil assessment, limb power, NEWS)
  - 15 min for 2 hours
  - 30 min for 6 hours
  - 1 hrly for 16 hours

Know the clinical syndrome and monitor for lesion extension

Continuous cardiac monitoring

Maintain BP < 185/110

Monitor for signs/symptoms of raised ICP
Tenecteplase

- Genetically variant of alteplase
- Longer half-life
- Greater binding affinity with fibrin \( \rightarrow \) better thrombolytic effect
- Administered as bolus only
- Tenecteplase dose 0.25mg/Kg before thrombectomy associated with higher recanalization and better functional outcomes in comparison with alteplase (Campbell et. al 2018 – phase II trial)
- Tenecteplase (dose 0.4mg/Kg) not superior than alteplase in patients with mild stroke (Logallo et al. 2017 – phase III trial)
- Current trials: EXTEND-IA TNK Part 2: dose 0.4 mg/Kg; TASTE; ATTEST-2; TEMPO-2
Case 2

- 36 y.o male, right handed
- PMH: Nil
- SH: mRS 0, lives with wife
- 6:00-6:45am: collapse, vomiting and headache. Taken to ED @ Northwick Park Hospital
Case 2

- 7:00am: CT head – nil acute infarct. No bleed

CTA – L M1/M2 thrombus with ICA dissection

- NIHSS 21

- 9:00am: Thrombolysed. CXH contacted

- 9:45am: Patient still at NWPH, waiting for ambulance and drop GCS 9/15 E3 V1 M5. Repeat CT – early L MCA changes
Transferred to CXH. On arrival NIHSS 19

Thrombectomy: recanalization at 12:22.

Post-thrombectomy: improved, more alert, obeying commands with visual cues. NIHSS 17

16:00: repeat CT - evolving MCA with early mass effect

20:00: decision made for surgical intervention

21:16: Knife to skin
Case 2
Within 72-92 hours

Surgical procedure under GA

Aim to relieve increased ICP as result of oedema of cerebral tissue caused by large cerebral hemisphere lesion, or space-occupying lesion

Removes part of the skull and opens the dura → Brain herniates outwards
When oedema/mass occurs within a compartment, the brain shifts from a compartment of high pressure to one of a lesser pressure.

Intracranial Pressure (ICP)

- 3 compartments
  - 2 Supratentorial – falx cerebri
  - 1 Infratentorial
Subfalcine herniation

- Subfalcine herniation (3)
  - Most common herniation
  - Midline shift
  - Headache
  - Contralateral leg weakness
  - Possible hydrocephalus

Eg. Malignant MCA infarct/space-occupying lesion (8%) - 80% mortality rate
Signs and symptoms of raised ICP

- Restlessness
- Headache
- Nausea and/or vomiting
- Photophobia
- Reduce GCS (or effort)
- Increase focal neurology
- Seizure
- Increased BP with associated Bradicardia (*Cushing 1900*)
- Meningism (*neck stiffness, headache and photophobia*)
- Changes in the pupil size and reaction of the pupils to light
- Think about the clinical syndrome!
Decompressive hemicraniectomy

- **Traditional conservative management**: sedation, hyperventilation, barbiturates and osmotic therapy (no evidence from RCTs)

- **DHC** (evidence from RCTs) – death and disability (mRS 4 and 5) at 12 months
  - Major disability
  - Death
  - Less 60 years versus older 60 years

*Age should not be an exclusion criteria!*
Decompressive hemicraniectomy

Refer to neurosurgery within 24 hours of symptom onset and treat within 48 hours if:

- Pre-morbid mRS < 2
- Clinical diagnosis of MCA syndrome
- NIHSS > 15
- Decrease in the level of consciousness to a score of 1 or more on NIHSS 1a
- At least 50% of the MCA territory in brain imaging
Posterior fossa herniation

- Cerebellum, midbrain, pons, medulla
- 3rd to 12th CN impairment
- Cardiac centres in medulla
- Medullary respiratory centre and pontine respiratory centre
- 4th ventricle - CSF obstruction - hydrocephalus
Suboccipital decompressive craniectomy

- Is effective for cerebellar infarction
- EVD and Craniectomy
- EVD increased mortality because of upward herniation
Suboccipital decompressive craniectomy

- 57 y.o male

**HPC:** 4pm acute onset dizziness + vomiting. Lasted 2 hours then settled.
8pm symptoms returned. Went to bed.
2am woke up, vomiting + room spinning

- LAS arrival: noted RUL + RLL weakness
- Examination in ED: nystagmus, R facial droop, RUL, RLL weakness. GCS 15/15
- CT – old R basal ganglia infarct. Nil acute          DWI MRI – bilateral cerebellar infarcts
Suboccipital decompressive craniectomy

- CTA – Thrombus in R VA
8:30am: drop GCS (E3 V5 M6), ataxia, worsened dysarthria, R CN VI palsy, upper airway noises, tachypnea
Impression: Posterior fossa malignant swelling 2nd bilateral cerebellar infarcts and hydrocephalus

- 11am (just before intubation): Noted L sided weakness

- **Plan:** 1) R frontal EVD for posterior fossa decompression
  2) Posterior fossa decompression
Do all stroke patients have carotid dopplers?

A – Yes

B – No

C – Don’t know
Why?

- Is it an ischaemic stroke?
- Is it an anterior circulation ischaemic stroke?
- Will the person be a candidate for carotid endarterectomy?
  - Pre-morbid mRS
  - Co-morbidities
Vascular imaging for anterior circulation
Carotid Endarterectomy

- Surgical procedure under local or general anaesthesia
- Aims to remove atherosclerotic plaque in the arteries
- Considered for patients with symptomatic severe carotid artery stenosis (50-99%)
- Reduces the risk of stroke or death
- Optimise BP, cholesterol levels, diabetes, lifestyle advice, antiplatelet treatment pre and post CEA
78 years old male

**HPC:** At 2pm sudden onset of RUL weakness and word finding difficulty

**PMH:** DM2, HTN, Smoker

At 4pm, presented as a thrombolysis call.

On Examination, NIHSS 0

CT head: Nil acute infarct. No bleed

Clinical impression: L MCA TIA

C. Dopplers: L ICA 65-75%
Primary Intracerebral haemorrhages
Acute Primary intracerebral haemorrhage

Primary Intracerebral haemorrhage

- Hypertension
- AVM or aneurysms
- Intracerebral tumours
- Haematological disorders
Acute Primary Intracerebral haemorrhage

- Airway protection
- Management of HTN
- Reversal of coagulopathy
- Surgical intervention
Management of hypertension

**INTERACT 2 Trial**

- SBP reduction to < 140 mmHg within 1 hour and during 7 days

### Systolic BP control

**Median (iqr) time to treatment**, hr. - intensive 4 (3-5), standard 5 (3-7)

<table>
<thead>
<tr>
<th>Intensive group to target (&lt;140mmHg)</th>
<th>Systolic BP time trends</th>
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<tbody>
<tr>
<td>462 (33%) at 1 hour</td>
<td>1 hour - Δ14 mmHg (P&lt;0.0001)</td>
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<tr>
<td>731 (53%) at 6 hours</td>
<td>6 hour - Δ14 mmHg (P&lt;0.0001)</td>
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**Mean Systolic Blood Pressure (mm Hg)**

- **Intensive**
- **Standard**

**Key secondary outcome**

**Ordinal shift in mRS scores (0-6)**

- Odds ratio 0.87 (95%CI 0.77 to 1.00); P=0.04

**Intensive**

- 8.1%
- 21.1%
- 18.7%
- 15.9%
- 18.1%
- 6.0%
- 12.0%

**Standard**

- 7.6%
- 18.0%
- 18.8%
- 16.6%
- 19.0%
- 8.0%
- 12.0%

**Disability but independent**

- **Major disability**

- **Death**
Management of hypertension

**INTERACT 2 Trial**

- Early BP control
- Target SBP < 140 mmHg
- Sustained BP control
If on Warfarin:
1) Reverse anticoagulation with Vitamin K and prothrombin complex concentrate (PCC) - Octaplex
2) Initiate Octaplex 1ml/min for 5 minutes  →  2-3 mls/min after
3) Check INR 30 min post-treatment

If on Dabigatran: Reverse anticoagulation with Idarucizumab 5g

If Apixaban, Edoxaban, Rivaroxaban: reverse with PCC
Patients with PICH who develop hydrocephalus should be considered for surgical intervention or insertion of an EVD.
Case study 3

74 years old lady

**HPC:** 6am seen well. At 8.40am tonic clonic seizure and left sided weakness

**PMH:** Right lumpectomy for breast cancer, depression, anxiety, cognitive impairment

**SH:** ex-smoker, heavy alcohol consumption (4 bottles wine/day). Lives with husband but currently in residential home for respite care, mRS 3

**On Examination in ED at 9.30am:**

NIHSS 19

BP 220/110  HR 140 sinus tachycardia  SaO2 95% in air  BM 8.3

GCS 14/15 (E4 V4 M6)  LUL 0/5  LLL 0/5
Case study 2

Are you going to thrombolyse this patient?
A – Yes
B – No
C – Maybe
D – I don’t know
Day 2 of admission, 6:40am:

GCS E2 V1 M4. NIHSS 36

EVD @ 10am.
References


