INTRACEREBRAL HEMORRHAGE AND STROKE

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Neuroscience Outreach
INTRACEREBRAL HEMORRHAGE

- Is the result of a rupture of a blood vessels into the parenchyma
- Is classified into primary (80%) and secondary hemorrhage (20%)
  - Primary: more common in older aged population
  - Secondary: mostly pathological and should always be considered for those under 45 with NO history of HTN
- Multiple causes exist including HTN, trauma, AVM or tumour
- Slightly more common in men, younger population, and certain races
- Baseline hematoma size is a strong predictor of outcome
Hemorrhagic Stroke

- Accounts for 12-15% of all stroke
- Is the most fatal form of stroke (50%)
- Has the highest morbidity of all stroke types
  - Severe persistent deficits occur in 30-40%;
  - only 20% regain functional independence
  - Surgical evacuation of hemorrhage not effective in LT outcomes
INTRACEREBRAL HEMORRHAGE CAUSE

Primary
- HTN
- Amyloid angiopathy

Secondary
- Vascular lesion
- Coagulopathy
- Tumour
- Ischemic conversion
- Trauma
- Drugs
- Idiopathic
HTN
- Often young
- Small volume
- Usually deep, cerebellar
- Rare cortical

AA
- Older (but may have HTN)
- Large volume
- Usually lobar
- Higher risk of re-hemorrhage

Imaging can reveal multiple old hemorrhage (MRI Flair)
Can direct care
May be difficulty to tell HTN/AA or other cause in large volume hemorrhage
HTN AND ICH

Often are found are areas of bifurcation
HTN AND ICH
Hemorrhage location and HTN

Cerebellar Hemorrhage
BLOOD VESSELS, HTN AND HEMORRHAGE

HTN Crisis....no overt MCA, ACA
Symptoms

- Patients get very sick very fast
- Sudden onset of focal neurological deficit
  - Cerebellum = ataxia
- Is rare to wake with symptoms
- Early symptoms of increasing ICP
  - Headache
  - Nausea
  - Vomiting
  - Decreased LOC
  - 50% symptom progression (from expanding ICH + edema)

3x more likely than ischemia
**EARLY CRITICAL MANAGEMENT**

- Recognize that no effective targeted therapy exists for ICH

But things to think about:

- Hematoma expansion occurs in 35-50%
  - Usually occurs within the 1st 3 hours after acute hemorrhage
    - 26% >33% hematoma growth
    - 12% >33% hematoma growth at 24%
  - Is worsened with anti-coags
  - More often in AA hemorrhage, vascular pathology (AVM/aneurysm)
  - Can often be identified by the presence of the ‘spot sign’ on CT
The presence of contrast enhancement within ICH, visible on CTA. Suggests active, dynamic hemorrhage. Is a predictor of ICH growth and poorer outcomes.
On admission  
Presence of spot sign  
CT 4 hours later

Length x width x height/2 = volume
Dx: Hemorrhagic stroke

Consider medications, past history

Routine labs, Coags

BP management

Assess for ICP
ICH PROGRESSION

ICP
BP
Coags
Clot

Care provided as per stroke

Danger zone
C R I T I C A L  M A N A G E M E N T : 1 S T  3 6  H O U R S

Care needs to include

- Life support: ABC
- ICP assessment and documentation
  - Know what the symptoms reflect
  - Pay attention to initial hemorrhage location***
    - Cortical versus cerebellar
- BP-intensive reduction
- Control of hematoma size
- Seizure management
- Assess for other injury-great % of patients fall
Hemorrhage location

- Cortex
- Thalamus
- RAS, brainstem
HEMORRHAGE LOCATION AND ICP SYMPTOMS

Headache, nausea, focal neurology (motor weakness), mild change in LOC, vomiting

Motor and/or sensory loss, obvious changes in LOC, change in pain perception

Comatose, decerebrate/decorticate, intubated, brainstem findings

Can provide guidance to rate of onset of ICP symptoms
ICP

Symptoms present when ICP >20

Early ICP symptoms results from:
- Cerebral irritation, meningeal pressure
- Hemorrhage volume
- Edema surrounding the hemorrhage

Worsening ICP results from:
- Pressure on vital centers (thalamus, brainstem)
- Cytotoxic edema
- Hemorrhagic expansion
ICP MANAGEMENT

When symptomatic
- HOB at 30 degrees, neutral alignment-watch collars
- Limit CNS metabolic demands: fever, glucose, seizures, analgesia, sedation
- Mannitol or 3% hypertonic saline
  - Draws fluids out of the cells
- Hyperventilate:
  - Short term hypocapnia causes vasoconstriction
  - Goal: PCO2 25-35mm/Hg
  - 1 mm/Hg ↓ in PCO2 ↓ CBF by 2%
  - Immediate results-lasts 6-24 hours
**Blood Pressure Management**

**BP**
- No studies show optimal BP
- Should be frequently monitored for 48 hours
- Goal: systolic target of 140mm/Hg
  - Intensive reduction more urgent in those with anti-coag on board
  - Beta-blockers: Labetolol is drug of choice. No nitroprusside (increases ICP)
  - Lower targets (below 140 not associated with greater outcomes
- Maintain CPP > 80 for perfusion
  - ICH is thought to disrupt auto-regulation of CPP
  - CPP = MAP-ICP
    - MAP = systolic + 2x diastolic /3 (usually 70-100 mm/Hg)
- Long term BP targets require individualization
# BP Management

<table>
<thead>
<tr>
<th>Management of BP</th>
<th>Suggestions</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP &gt;200 or MAP &gt;150</td>
<td>Continuous IV infusion</td>
<td>Aggressive reduction BP q 5 mins</td>
</tr>
<tr>
<td></td>
<td>(Labetolol)</td>
<td></td>
</tr>
<tr>
<td>Systolic BP &gt;180 or MAP &gt;130</td>
<td></td>
<td>Maintain CPP &gt;80</td>
</tr>
<tr>
<td>ICP symptoms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP &gt;180 or MAP &gt;130</td>
<td></td>
<td>Moderate reduction to 140</td>
</tr>
<tr>
<td>No ICP symptoms</td>
<td></td>
<td>BP q 15 mins</td>
</tr>
<tr>
<td>Systolic BP 150-200</td>
<td></td>
<td>Aggressive or moderate reduction</td>
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</tbody>
</table>

Suggest aggressive management within 4.5 hours of symptom onset
COAGULATION MANAGEMENT

Emergency Room
CT-scan to confirm ICH

Team interaction
Neuroradiologist
Anesthesiologist
Neurosurgeon
Neurologist
Hematologist

* Consult hematology

PCC: prothrombin complex concentrate
FFP: large volume

Aspirin
Ticlopidine
Clopidogrel
Prasugrel
Ticagrelor

VKAs
INR > 1.5

Platelet transfusion
(Class II recommendation)

Vitamin K (5-10mg IV)
+PPC or FFP
+Check INR after the injection
(Class I recommendation)
Discuss administration of additional FFP/PPC if INR > 1.5

Heparins
aPTT > 2 times normal ranges

Protamine sulfate (slow IV)
(Class I recommendation)
Dosing based on time from cessation of heparin

Dabigatran
Hemodialysis

No recognized antidotes
(from Kaatz et al.)
Oral activated charcoal

Rivaroxaban
Apixaban

rt-PA

Platelet transfusion +PCC or FFP
(Class II recommendation)

Discuss surgical indications

* Consult hematology

Journal of Anaesthesia (2013), 119, 218-227
ER/ICU CONSIDERATIONS SUMMARY

Things you need to worry about:

- VS dysregulation
- ?Large volume hemorrhage
- Presence of spot sign
- Early presentation to ER
- Anti-coagulant use
- Obvious symptoms of ICP
CRITICAL AND NEUROSURGICAL CONSULTS

Non surgical
- Small
- Deep hemorrhage
- No/mild deficit
- GCS <4
- Loss of brainstem function
- Severe coagulopathy

Surgical
- Large hemorrhage (>3 cm)
- Overt deficit
- Diagnosed lesion
- Age: young with large hemorrhage
- Brainstem compression

Is case-based
Surgical evacuation = no improvements in outcomes
ACUTE NEUROSURGICAL GUIDELINES

Eligibility
- Altered LOC w/out intoxicants
- Symptoms of high ICP
- Focal deficit, seizures, lateralizing signs (pupils)
- CT confirmation: non traumatic ICH-cortical/infratentorial

Stabilize
- ABCs
- Reverse Coag (need INR <1.5)
- Treat elevated ICP
- Use only short acting sedatives
- Intubate GCS <8, or <10 for transport

Consult
- CritiCall
- Neurosurgery
- ICU
- Family

Management
- Dilantin load if seizing
- Manage and target BP
- Discuss if transfer appropriate
Midbrain hemorrhage

Medullary hemorrhage
**AFTER 24 HOURS**

- At 72 hours, brain will be ‘settled’ (though swelling can occur up to one week or longer)
- Clot absorption takes about a month
- Injury is the result of the *sudden* insult or repeated insult and the resulting injury to the neuron
- Patient requirements no different from ischemic infarction
AFTER 24 HOURS: WHAT TO EXPECT

Sympathetic storm
- Is the result of sympathetic dysregulation
- Worse in those with greater injury
- Can begin within a few hours of injury
  - Symptoms: fever, cardiac dysrhythmias, Cushing’s ulcer, liver shock

Fever
- Is common (d/t inflammation and hypothalamic hit)
- Increases CNS metabolic demand
- Onset within few hours is considered ‘neurogenic’ (not infectious)
- Requires aggressive management (brain is 1 degree higher)
AFTER 24 HOURS: WHAT TO EXPECT

Seizures

- Occur in 25% of ICH-more often in lobar hemorrhage
- Must be documented through witness or EEG monitoring, or is hx of seizures
- Prophylaxis is NOT recommended
- Should be considered if changes in LOC without obvious cause or out of proportion to ICH

VTE Prophylaxis:

- Can be initiated once clot is stabilized through serial imaging (usually at 72 hours)
- TEDS not effective
- Requires Doppler U/S if >24 post admission and if considering intermittent compression
A WORD ON HTN THALAMIC ICH

- The thalamus has many functions
  - A stroke in one part of the thalamus will not look like a stroke in the other part
- 4 thalamic presentations are reported
  1. Sensory loss +/- motor
  2. Neuropsychiatric, changes in arousal, oculomotor
  3. Frontal lobe symptoms +/- motor or sensory
  4. Visual field cuts +/- motor
SHORT AND LONG TERM OUTCOMES

- Are highly dependent upon location of hemorrhage, time to treatment, surgical intervention and neurological severity, co-morbid illness
- Only 20% return to previous function
- Care similar to that of ischemic stroke
  - Co-morbid management
  - System management: skin, VTE, labs, depression
  - Gradual return of function

Thalamic pain syndrome (central pain/dysesthesia)
  - Can occur weeks-months after stroke on the affected side.
  - Does not respond to regular pain medicines and often doesn’t occur until weeks after the stroke happens

Depression:
  - Very common post stroke
  - Presents as decreased engagement, loss of learned skills post stroke
RECOVERY

The majority of neurological recovery occurs within 6 months

- Upper extremity weakness: function return usually within 1st month
- Lower extremity: no movement within 72 hours, poor prognostic chance of return to full ambulation
- Aphasia: average functional return by 10 weeks
- Dysphasia: rare at one year
- Sensory loss: permanent loss very common
- Visuospatial neglect: 70-80 recovery at 3 months
SUMMARY

Astute management is required for the 1st 24-72 hours

- Be aware of spot signs, large volume, hypertension and use of anti-coags
- Requires very frequent assessment for ICP
- Prepare family

After

- Care similar to that of ischemic stroke
- Co-morbid management
- System management: skin, VTE, labs, depression
- Gradual return of function