Hemorrhagic Strokes in Adults

Canadian Best Practice Hyper-Acute Hemorrhagic Stroke Recommendations

Charmaine Martin RN(EC)

Disclosures

- No conflict of interests
- "Passionate" about neuroscience nursing.
  - Spirited
  - Intense
  - Heartfelt
  - Eloquent

Objectives

- Types of Hemorrhagic Stroke
- Incidence / Mortality of Hemorrhagic Stroke
- Outcome Predictors
- Emergency Care
- Neurological Monitoring
- Diagnostic Imaging
- Treatment
- Recovery

Levels of Evidence

- Level A – data from multiple RCT
- Level B – single RCT or nonrandomized studies
- Level C – consensus opinion or experts

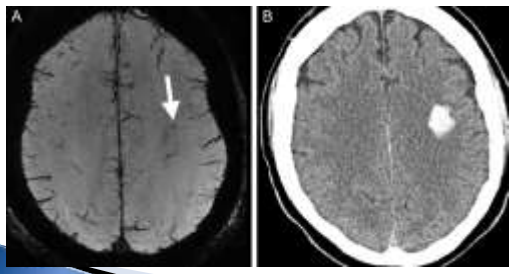
Classes of Evidence

- Class I – Evidence / general agreement that procedure or tx is useful and effective
- Class II – Evidence is conflicting about the useful/ efficacy of a procedure or tx
  - Class II a – Weight of evidence in favor
  - Class II b – Usefulness /efficacy is less well established by evidence / opinion
- Class III – Conditions / evidence that it is not effective / useful – could be harmful

Types of Hemorrhagic Stroke

- ICH – Intracerebral Hemorrhage – tissue
  - Lobar
  - Midbrain
  - Cerebellar
  - IVH – Intraventricular Hemorrhage – ventricles
- SAH – Subarachnoid Hemorrhage – subarachnoid spaces +/– ventricular space (2nd IVH)
  - Emergency treatment only

Hemorrhagic Transformation of a ischemic stroke
Not covered in this presentation



Incidence of Hemorrhagic Stroke

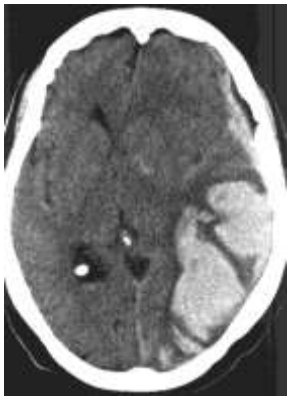
20–30% of all strokes

Mortality of ICH

- ICH strokes caused by hypertension have a 30 day mortality of 10% – 50% depending on size / location of bleed
- 50% of patients are expected to deteriorate within the first 24–48 hours related to cerebral edema and complications associated with the initial stroke.

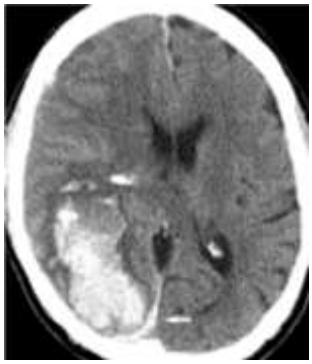
Lobar Hemorrhage (35%)

Located in the cortex &/or subcortical white matter



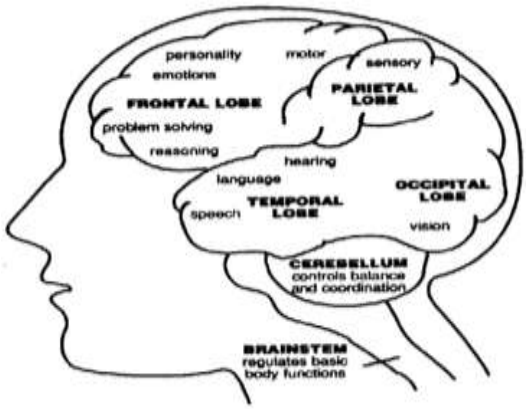
Lobar Hemorrhage

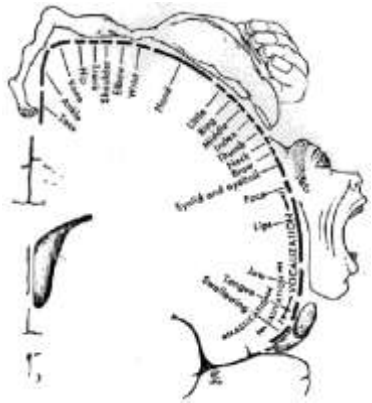
Primary Causes: HTN or Cerebral Amyloid Angiopathy



Clinical Presentation

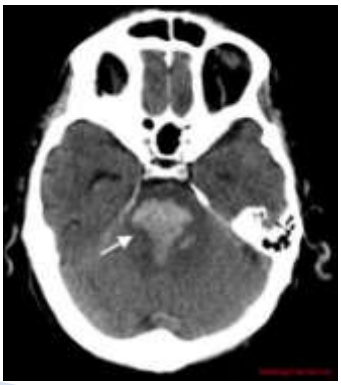
- Lobar Bleeds
- Neurological deficits based on the location of the bleed
- Continuous progression of neurological symptoms based on size of bleed and degree of intracranial pressure.



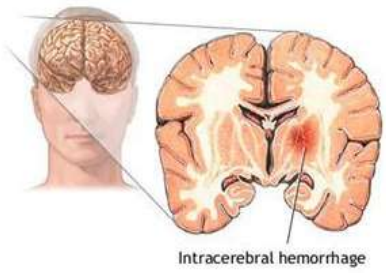


Midbrain Hemorrhage (50%)

Primary Cause: HTN



Deep ICH

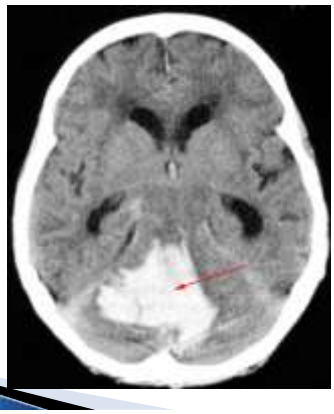


Clinical Presentation Midbrain & Brainstem Bleeds

- Vomiting
- Rapid LOC – if bleed is large / in the pons or brainstem
- Pupillary changes: eye bobbing; gaze palsies; pinpoint pupils; diplopia
- Cranial Nerve changes (eg. Dysarthria, dysphagia)
- Hemiparesis without sensory (corticospinal tracts)
- Hemisensory changes

Cerebellar Hemorrhage (10%)

Primary Cause:
HTN
Tumour
Vascular Abnormality

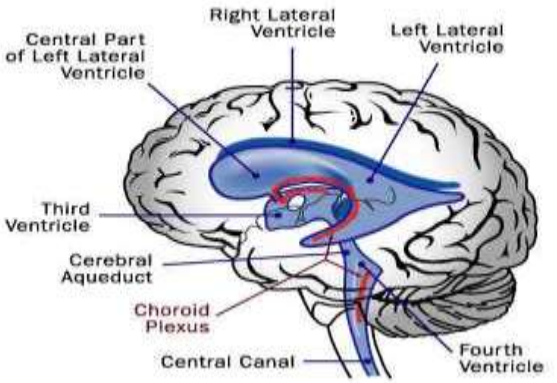


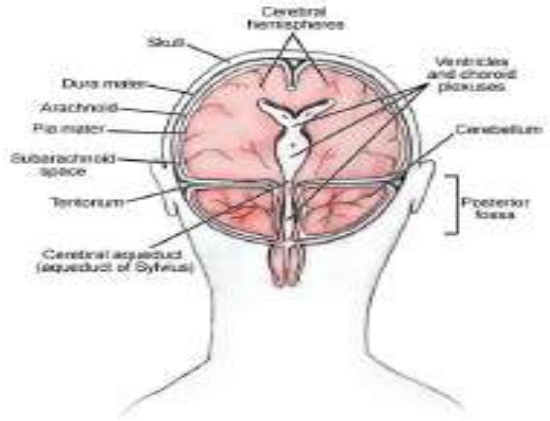
Clinical Presentation Cerebellar Bleeds

- Ataxia
- Nausea & Vomiting
- Dysarthria
- Dysphagia
- Diplopia / Nystagmus

- High Risk for Rapid Deterioration in 1st 72 hrs
- Best Candidates for Surgical Intervention

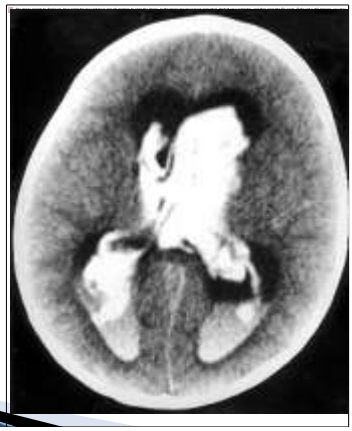
The Ventricular System of the Human Brain





Intraventricular Hemorrhage

Primary Cause:
HTN
Vascular Abnormality



Clinical Presentation Intraventricular Bleed

- Initially: severe abrupt headache, nausea, vomiting, confusion / disorientation
- Neck Rigidity
- Rapid Loss of Consciousness
- Sluggish or Fixed Pupils
- Arrhythmias / Respiratory Changes
- Treatment: ABCs & EVD + osmotic therapy

Mortality of IVH

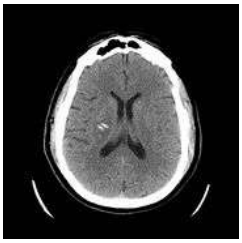
- 80% mortality
  - Sudden increased intracranial pressure
  - 3rd ventricular hematoma resulting in diencephalic or mesencephalic signs
    - Tachycardia
    - Hypertension--→ hypotension
    - Whole body tremors – looks like seizure
    - Downward gaze
  - 4th ventricular compression – cushing's response

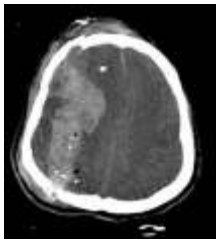
Predictors of Stroke Outcome

- Size does matter & Clinical Presentation

ICH blood volume & GCS on admission most powerful predictor of death by 30 days
Evidence B

- Increase in hematoma size results in a 5 fold increase in death / poor outcomes





Size of ICH

Predictors of Acute Outcome

- Increased temperature > 37.5 C– marker of death (Odds ratio 1.2)
- Increased age > 85 yrs 10x morbidity
- Increased Intracranial pressure
- Time from onset of bleed until hospitalization

Predictors of ICH Outcome

- Blood thinners – warfarin at therapeutic levels (2.5–3.5) increases risk of hematoma expansion (54% vs 16 % no coumadin)
  Odds ratio 6.2
- INR> 4.5 doubles risk

Predictors of ICH Outcome

- Hydrocephalus independent indicator of 30 day death
- Location of bleed can help predict clinical deficits and functional outcomes
  - Example: Left temporal ICH; Cerebellar ICH
- Etiology of bleed: HTN vs Cerebral Amyloid Angiopathy
  - HTN – slower to recover
  - CAA – faster to recover → Higher risk rebleed

Cerebral Amyloid Angiopathy

- Effects elderly population:
  - 65-75yrs 3%
  - 75-85yrs 8%
  - >85yrs 12%
- Higher risk for re-bleeds
  - 21% after 1st bleed;
  - 35-50% after 2nd bleed.

Cerebral Amyloid Angiopathy

Pathology

Deposition of congophilic material in small to medium size vessels in the brain – similar to the plaques of Alzheimer.

Cerebral Amyloid Angiopathy

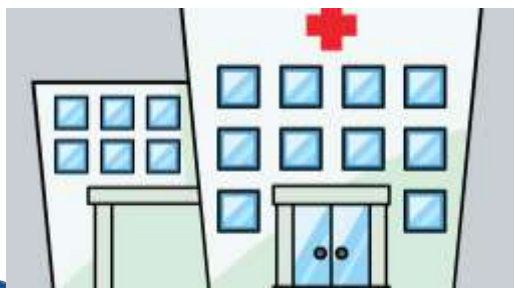
- Diagnosis : gradient echo MRI and/or tissue sample. Definitive diagnosis- post-morteum autopsy
- Suspect :Occurs sporadically through the lobar regions of the brain
- Prognosis: clinically better outcomes with 1st bleed compared to HTN bleeds

Emergency Diagnosis & Assessment of Hemorrhagic Stroke

- Prompt recognition and treatment as medical emergency (Evidence Level A)
- Human brain 22 billion neurons
- Every minute stroke is not treated–1.9 million neurons die

Time is Brain

Identify, Stabilize, Consult → Stroke Center



Treatment of ICH / IVH

Goals/ Options:

1. Stopping / slowing initial bleeding during 1st hours of onset
2. Removing blood from parenchyma / ventricles to eliminate cause of injury
3. Management of ICP and decreased cerebral perfusion
4. Supportive management: ABCs, glucose, fever, nutrition and DVT prophylaxis

"I think they are having a stroke?"

Can you tell the difference between Ischemic vs Hemorrhagic Stroke upon initial presentation?

NO – need radiological imaging
CT scan or MRI immediately

Level A

Ischemic vs Hemorrhagic Stroke

Helpful Hints:

- Sudden focal neurological deficits usually while patient is active (location of bleed)
- Symptoms progression worsens over time
- Vomiting (increased ICP)
- Headache ICH > ischemic but <SAH)
- Instability in neurological / cardiopulmonary (size of bleed / ICP)
- Hypertension

Diagnostic Imaging

- CT and MRI are each first choice imaging options Level A
- CT head plain – superior at demonstrating ventricular extension.
- CT (with contrast)/ CTA can identify tumor, AVM, aneurysm.
- MRI / MRA superior for posterior fossa, recent strokes, vasculature

Clinical Assessment Tools

NIHSS – for alert or drowsy patients.
Level B

GCS – for obtunded, semi or fully unconscious patients
Level B

Canadian Neurological Scale (CNS) – baseline and every 30-60 minutes for 48-72 hrs.
Level C

Common Errors :Neuro assessment

GCS – central vs. peripheral stimuli
– Limbs positioned at mid-abdomen, flexed
– volitional vs. posturing movements
Sedation reduction for regular examination

Pupil – location, size, reactivity

Confusion or Language deficit
– comprehension ; expression

Objectively measure level of arousal / sedation with a standardized tool (RASS)

HHS Neuroassessment

- GCS
  - Eye opening response
  - Best verbal response
  - Best motor response
- Speech assessment
  - Receptive
  - Expressive
  - Global
  - Normal
- Pupils
  - Ambient, constriction, consensual
- Movement/strength
  - Normal
  - Mild weakness
  - Severe weakness (3)
  - Spastic flexion
  - Extension
  - No response

- Agitation and sedation scale (ie. RASS)
- VS – BP, temp, pulse, RR, O2 sat

Assessing Level of Consciousness

- GCS (Glasgow Coma Scale) assesses patient's level of consciousness by assessing two components: AROUSAL & AWARENESS
- Arousal – state of awakefulness. Measured by assessing ability to open eyes to voice / pain
- Awareness – interaction with and reaction to environmental stimuli. Measured by best verbal response and best motor response.

Orientation

- Ask more detail –
  - place – city, building; floor;
  - Date – day, month, year, season – record information.
- The details that fall away will be the early clues to deterioration
- Perform speech assessment if confusion/inappropriate responses

Speech Assessment

- Receptive deficit
  - Assess ability to comprehend language and recognize words
- Expressive deficit
  - Assess ability to articulate speech, assign meaning to words
- Global Aphasia
  - Receptive and expressive deficits identified, no clear speech
- Normal speech
  - Completes a normal receptive and expressive exam

Making Motor Assessment Better

- Maximizing stimulation –
  - Voice – gentle to louder
- Inflict central pain – trapezius squeeze, supraorbital pressure (limit sternal rub)
- Ensure hands are free to respond to central pain, placed midline
- Peripheral pain – only done if a limb is nonresponsive

Best Motor in GCS

1. **Obeys instructions**:
   - holding up fingers, thumbs up, stick out tongue (CN XII)

2. **Central stimulus**: place arms flexed at lower abdomen and unrestrained

Localize: Attempt to remove pain source purposefully

Withdraw: Removes limb away from pain source and away from body.

Best Motor Response

Peripheral stimulus to the limb that does not move to assess motor response and strength.

- For peripheral stimulus use interphalangeal joint (not nailbed)

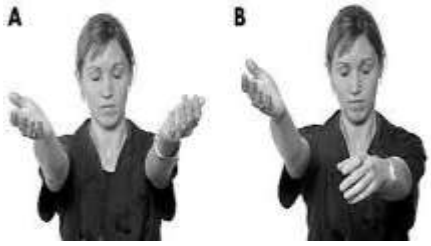
- Spastic flexion and extension are brain stem responses in an **acute setting**.
- Hand grasp – can be a reflex

Muscle Movement & Strength

- Normal Power → 5 /5
- Mild Weakness → 4 /5
- Severe Weakness → 3/ 5 – elevate above gravity – not sustained
  - 2/5 – movement without gravity eliminated
  - 1/5 flicker

- Spastic Flexion
- Extension
- No Response → 0/ 5

Arm Drift



Pupil Assessment

Compare:
- Size
- Shape
- Reaction to light
- Accommodation

SCORE	BEHAVIOUR	DESCRIPTIVE	
+4	Combative	Violent, immediate danger to staff.	
+3	Very agitated	Aggressive. Pulls or removes tube(s) or catheter(s).	
+2	Agitated	Frequent non purposeful movements. May fight ventilator/ ventilation.	
+1	Restless	Anxious, apprehensive but movements are not aggressive or vigorous.	
0	Alert & Calm		
-1	Drowsy	Not fully alert, but has sustained awakening to voice (eye opening & eye contact (>10 sec).	VOICE
-2	Light Sedation	Briefly awakens to voice (eye opening & eye contact (<10 sec).	VOICE
-3	Moderate Sedation	Movement or eye opening to voice (no eye contact).	VOICE
-4	Deep Sedation	No response to voice, but movement or eye opening to physical stimulation.	PHYSICAL STIMULATION
-5	Unrousable	No response to voice or physical stimulation.	PHYSICAL STIMULATION

When to Worry about the Patient Changing

- Greater risk of instability & deterioration in 1st 24 – 48 hours – EDEMA

INTRACRANIAL PRESSURE

- Brain tissue 80%
  - CSF 10%
  - Blood 10%

Monitor for Signs of Increased ICP

1. Increasing Headache
2. Vomiting
3. Cranial Nerve VI palsy and/or upward gaze
4. LOC ↓
5. Cushing Reflex (" ship has sailed")
   a. Bradycardia
   b. Respiratory Depression
   c. Hypertension

Level B

ICP management

Head of bed elevation 30 degrees – improves jugular outflow / lowers ICP. Head midline– avoids compression of jugular veins.

Evidence A

Osmotic Therapy – to be used in cerebral edema

Evidence B

1. Mannitol
2. 3% Normal Saline

ICP Management with Mannitol

Mannitol draws out fluid from both edematous and non-edematous brain tissue

Mannitol increases preload and CPP – thus decreasing ICP through cerebral autoregulation.

Major problem – hypovolemia and induced hyperosmotic state

ICP Management
3% Normal Saline / ICP Drains

- Hypertonic 3% normal saline solutions
  - Bolus 150-200 mls
- Target serum osmolality to 300-320mOsm/kg

- ICP drain required ->to Neurosurgery
  - high risk of morbidity / mortality with hemorrhage / infection (bacterial colonization 6-22%)

Risk for Bleeding?

Question about anticoagulant use

Measure platelet count

Measure INR & PTT

Level A

Stop Bleeding

- Wafarin :
  - Prothrombin Complex Concentrate (PCC) or Fresh Frozen Plasma (dose 15 -20 ml/kg)
    Evidence B
  - Vitamin K – if on warfarin – dose 10 mg IV – takes 6 hours to normalize INR.
    Evidence B
- ASA: stopped immediately Evidence C

Stop Bleeding

- Heparin drip – reverse with protamine sulfate
  Evidence B
- Recent TPA – replace clotting factors/platelets
  Evidence B
- Novel Oral Anticoagulant– 24 hours to clear system. Risk for major hemorrhage <=6%
  - Urgent consult with a hematologist in absence of direct reversal agents

  Evidence C

Continue Anti-Coagulation

Persisting Strong Indication for Anti-coagulation (ie. Mechanical valve or recent cardiac stents) – consult experts (ie. Hematologist, thrombosis, cardiologist)

Medical Treatment to Stop Bleeding

- Trials of Recombinant Activated Factor VIIa
  - *Not recommended for use outside of a clinical trial (Evidence A)*

Acute BP management

- High BP correlated with increased volume of hemorrhage, therefore increased ICP

- **Monitor BP** :
  - q 15 minutes until stabilized→
  - then q30-60 minutes for 24-48 hrs

Acute Blood Pressure Management Factors to Consider

Optimal BP should be based on individual factors

- Chronic hypertension (Normal BP)
- ICP
- Age
- Presumed cause of bleed
  - (HTN, amyloid angiopathy, vascular malformation)
- Interval since onset of bleed

Evidence C

Acute BP – General Guidelines

- Aggressively lowering BP with increased ICP decreases CPP and worsens brain injury / death. *Evidence C*

- Isolated SBP> 210mmhg – not shown to worsen

- BP >=180 mmhg + high ICP –reduce BP with controlled medications, such as Labetolol drip *Evidence B*

Acute BP – General Guidelines

Target SBP : Less than 180mmhg

Evidence C

? Target less than 160mmhg

safety studies completed

clinical outcomes improved – Interact 3

Evidence B

- After 48 hrs, further BP lowering to optimize stroke prevention (as able)

Evidence B

Management of Glucose

- Hypoglycemia and hyperglycemia –
  Monitor early and treat appropriately
  Target 6–10
- Insulin should be started for patients with stroke and hyperglycemia.
- Ongoing studies needed to determine the optimal level of glycemic control

Pain Control

- Sedation / analgesia – minimize pain – but difficult to be able to assess patient–

Level II a B evidence

- Short Acting Narcotics preferred

Prevention / Treatment of Seizures

- Seizures occur commonly
  - may be nonconvulsive
- Studies with continuous EEG – 28% had seizure activity in 1st 72 hrs, esp. lobar bleeds
- Seizures associated with increased midline shift ➡ HIGH ICP

Seizure

Treatment

1. IV Benzodiazepines
2. IV Dilantin

Evidence B

Neurology to assess long-term medication requirements

- Prophylactic treatment not recommended

Evidence C

- Rule out seizure if clinical status declines without explanation

Untreated seizures lead to poor outcomes

Temperature Management

Fever worsens outcomes – independent prognostic factor

- Temperature > **37.5** needs to be treated
- Lowering temperature assists with redistribution of oxygen and lowering glucose

When to Refer to a Neurosurgeon?

Surgical Candidates

- Features supporting surgery include:
  - recent onset of hemorrhage
  - patients with intermediate levels of arousal (obtundation-stupor).
  - involvement of the non-dominant hemisphere ?
  - location of the hematoma near the cortical surface.

Timing of Surgery

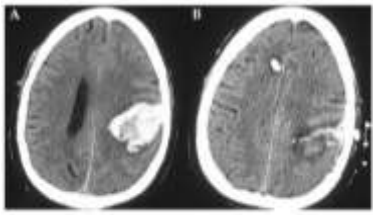
- Operative removal within 12 hours

  Evidence B

- Delayed evacuation by craniotomy offers little benefit. Surgical resection in patients in coma with deep hemorrhages may actually worsen outcome.

  Class III, Evidence A

Surgical Treatment – Craniotomy



Patients with Lobar clot within 1 cm of surface

Evidence B

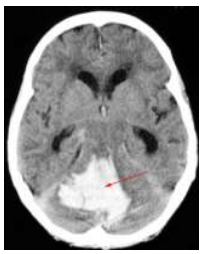
Surgical Treatment – Cerebellar

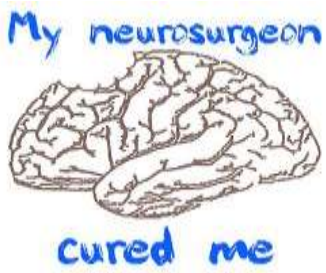
Cerebellar hemorrhage
> 3 cm with:

- Decline in Neurological status
- Hydrocephalus with increasing brainstem herniation

SURGICAL REMOVAL OF CLOT URGENTLY

Class I Evidence B





Surgical Treatment – ? Craniotomy

- Features in favor of less aggressive therapy include
  - serious concomitant medical problems
  - advanced age
  - stable clinical condition
  - onset of hemorrhage > 96 hours

Evidence A

  - inaccessibility of the hemorrhage

Infusion of urokinase into the clot
Endoscopic Mechanical Removal of the Clot

Class II b Evidence B

Prevention of DVT

- Graduated compression stockings ?
- Pneumatic compression *Class I Evidence B*
- Prophylactic heparin – after documentation of cessation of bleeding

Low dose subcutaneous Heparin / Fragmin may be considered after 3-4 days

Class II b Evidence B

Benefit to:
Prevent Stroke
Prevent DVT
Treat DVT / PE
Prevent Stent Occlusion

Risk
Rebleeding
Respiratory Distress or Death

Factors to Consider in using Anticoagulation

Age; Overall status; Mobility
Comorbidities (a.fib; cardiac stent; artificial heart valves; DVT; PE)
Location and Size of Bleed
Cause of Bleed – condition stabilized
Number of days from the bleed; Residual blood

Mobilization / Rehabilitation

- Early mobilization and rehabilitation when the patient is stable is recommended

  Evidence C

- Rehabilitation started within 20 days of bleed has 6x greater response

Prevention of Recurrent ICH

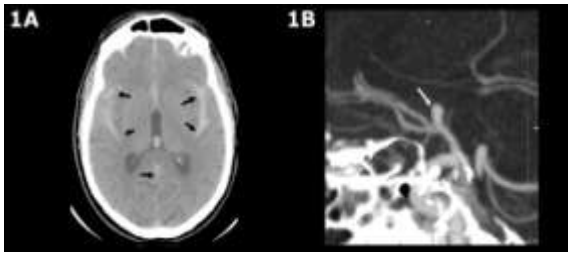
- Treating hypertension in nonacute setting is the most important step to reduce risk – *Class I Evidence A*
- Smoking, heavy alcohol use, cocaine use are risk factors for ICH – discontinuation is recommended *Class I, Evidence B*

Goals of Care

- Goals of Care should be established early after patient 's admission.
- DNR discussions should not occur until 24–48 hrs to assess patients response, or when the patient's condition is worsening despite optimal medical care.

Communicate with the Patient Family and/or POA

Subarachnoid Hemorrhage / Aneurysm



I have the worst headache of my life

- Headache –sudden, severe, thunderclap
- Headache during exertion

NOTE: Headache different from my normal migraines.

- Neck stiffness / pain with limited neck flexion
- Age >= 40
- Nausea & Vomiting

Sensitivity 100%; Specificity 53%

Subarachnoid Hemorrhage

Subarachnoid Hemorrhage

- SAH : 9 people / 100,000 aneurysmal subarachnoid hemorrhage

(with / without intraventricular blood extension)

Risk Factors:

- Female
- Middle Aged – 45–55 yrs
- Smoking
- HTN
- Family History of Aneurysm
- Binge Drinking and/or cocaine use

Mortality of SAH

- 1/3 die (33%) before they get to the hospital
- 5 – 10% will die while in hospital
  - Poor clinical presentation
  - Complications of treatment
- 1/3 will have clinical significant deficit
- 1/3 good recovery

Predictors of SAH Outcomes

- Size of bleed – Fischer 1 – 4
- Time to treatment <24 hrs
- Type of surgical tx – coiling vs clipping
  ISAT trials (7.4% decrease in death and disability with coiling)
- Developing Cerebral vasospasm with or without secondary ischemic stroke
  - 5 – 21 days post bleed

Diagnostic Work-up – SAH

- CT head to assess for a SAH
- CT Angiogram – for assessing for aneurysm
- Lumbar puncture – IF CONVINCING HISTORY BUT NO SAH BLOOD ON CT HEAD
  xanthochromia positive in CSF

Referral to a Neurosurgeon ASAP

Summary of Guidelines

1. Prompt identification of symptoms
2. Imaging with CT / MRI
3. Consult Stroke Center
4. Transfer of patient to neuro ICU or stroke units, if patient at risk for neurological deterioration.
5. Monitoring with standardized assessment tool – CNS; NIHSS; GCS

Summary of Guidelines

6. Treatment of seizures (only if they occur)
7. Keep temperature below 37.5 – no extreme cooling
8. Treat ICP with HOB > 30 – Head midline
9. Treat ICP with osmotic diuretics – transfer to neurosurgery as appropriate.
10. Prevent hyperglycemia – exact level under investigation

Summary of Guidelines

11. Treat SBP > 180 mmhg sustained
12. SBP > 180mmhg with high ICP – lower BP slowly to prevent difficulties with CPP
13. New Evidence regarding maintaining SBP < 160mmhg – safe and improved outcomes (Interact 3 study)

Summary of Guidelines

14. History of Anticoagulant use determined ASAP
15. Warfarin induced bleeds – tx with Vitamin K + PCC / FFP; Heparin induced bleeds – tx with protamine sulfate
16. FFP effective at reversing bleeding time but requires high volumes

Summary of Guidelines

17. Pneumatic compression / TEDS prevent DVT
18. After bleeding ceased – DVT tx with heparin / low molecular weight heparin can be considered in 3-4 days.
19. Vena cava filter in patients with proximal DVT with vein occlusion.
20. No determined evidence on if / when to restart warfarin in high risk thromboembolic patients – case by case review

Summary of Guidelines

21. Lobar clots within 1 cm of surface – might be considered for surgery
22. Cerebellar hemorrhage > 3 cm – good evidence for Surgical removal
23. Craniotomy Decision based on individual factors, size and location of bleed, age, probability of good outcomes

Summary of Guidelines

24. Least invasive approaches may be of benefit – endoscopic procedures – need further research.
25. < 7 hours surgery may increase risk of rebleeding.
26. > 96 hrs surgery – no benefit

Summary of Guidelines

27. Early mobilization – starting rehab ASAP
28. Aggressive tx for first 24 hours – unless prior DNR orders.
27. Prevention for future strokes – nonacute setting – outline tx plan for HTN
28. Smoking, heavy alcohol and cocaine use increase risk – stop.

Recovery Curves

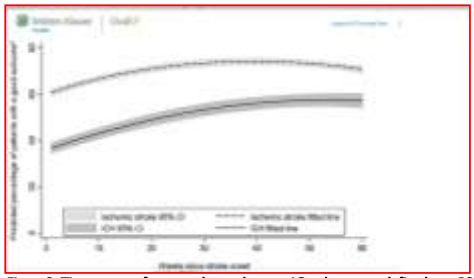


Figure 2. Time course of recovery by stroke type. *Good outcome defined as mRS scores of <3; graphs based on nonmissing values.

http://ovidsp.tx.ovid.com/sp-[illegible]/ovidweb.cgi?&S=NICFFPOFMGDDMCPJNCBLJEFBBPAPAA00&Link+Set=S.sh.47%7c10%7csl_10

Treatment of DVT / PE

- Vena cava filters – reduce risk– long term increases venous thromboembolism

  Class II b Evidence B

- Strong Indication for anticoagulation should be determined on a case-by-case basis

  Evidence C

Contact Information:

Charmaine Martin
martinch@hhsc.ca