INTRACEREBRAL HEMORRHAGE (ICH)

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ICH is the second most common cause of stroke.

PRESENTING COMPLAINT: acute onset headache, one sided weakness, lethargy, slurred speech, seizure

FINDINGS

- A "Look, Listen, Feel", check airway, may require airway protection
- B Normal, decreased or increased RR, check SpO2
- C Check pulse and BP
- D Variable altered * slurred speech, dizziness, lethargy, confusion, coma focal deficits, anisocoria
- E Inspection: trauma, wounds, visible bleeding,
- Upc n/a
- Lpc non-specific, blood glucose, CBC, PT, PTT, INR, creatinine, BUN, ABG

***V** (verbal), **P (**pain), **U** (unconsciousness), **D** (delirious)

$\mathbf{U_{PC}}$ (point of care ultrasound) $\mathbf{L_{PC}}$ (point of care labs)

OTHER HISTORY

● Causes: atraumatic (spotaneous) and traumatic hemorrhage
● Single/multiple hemorrhagies
● Predisposing conditions:
  a. **Older age**
  b. **Hypertension**
  c. Dementia (cerebral amyloid angiopathy)
  d. Vascular anomaly (areriovenous malformation, cavernous malformation, intracranial aneurysm)
  e. Coagulopathies (and liver disease)
  f. Intracranial tumor
  g. **Anticoagulant therapy**
  h. Drugs: cocaine, amphetamines
  i. Infection: septic embolism, mycotic aneurysm
     ● CNS: fungal, rarely bacterial or viral
     ● CVS: endocarditis
  j. Other: genetic variations, high alcohol intake, lower cholesterol and low LDL,vasculitis,
● **Three categories: subdural, epidural and intraparenchymal hematoma**
● Symptoms (according to the size and location of the ICH):
    - Putamen/globus pallidus: hemiplegia, hemisensory loss, homonymous hemianopsia, gaze palsy
    - Internal capsule: contralateral hemiparesis, dysarthria and sensory deficit
    - Cerebellum: walking disturbances, vomiting, headache, neck stiffness, facial weakness,

gaze palsy

- Thalamus: hemiparesis, hemisensory loss, homonymous hemianopsia (transient), upgaze palsy with miotic pupils, aphasia (if hemorrhage in the dominant hemisphere), neglect syndrome (if hemorrhage in the nondominant hemisphere)
- Lobes:
    - Frontal: contralateral plegia or paresis of the leg, , abulia, apathy; inferior frontal lobe ICH more common of traumatic origin
    - Temporal: seizures; often traumatic
    - Parietal: left (Gerstmann's syndrome, motor apraxia), right (contralateral hemineglect syndrome, anosagnosia, constructional apraxia)
    - Occipital: contralateral homonymous hemianopsia
- Pons: coma, paralysis, miosis, absent horizontal eye movements, facial palsy, deafness, dysarthria
- Intraventricular: Potential risk of obstructive hydrocephalus

DIFFERENTIAL DIAGNOSIS

- Ischemic stroke, intracranial mass lesion, cerebral venous and sinus thrombosis, intracranial abscesses
- neurologic symptoms and signs may <u>increase</u> gradually over minutes or a few hours

OTHER INVESTIGATIONS

- Severity Score: ICH Score (30-day mortality prediction score)
- Labs: CBC, Coagulation parameters (PT with INR, aPTT, TCT if patient on direct anticoagulants, Thrombin time for DTI, Xa level for Xa inhibitor), cardiac-specific troponin, toxicology screening, urinalysis with urine culture, pregnancy test in women of childbearing age
- Other labs based on comorbidities (liver function tests etc.)
- Monitoring: BP, ECG, ICP
- ECG -prolonged QT interval, ST-T wave changes (neurogenic cardiac damage), can mimic myocardial infarction
- Imaging: emergent non-contrast CT of the heard, consider non-invasive angiography to exlude underlying vascular anomalies, consider MRI after stabilization to determine cause;
- Repeat CT scan if there is neurologic decline or after 6 hours of initial scan in patients at high risk of expansion
- Baseline chest X-ray

THERAPEUTIC INTERVENTIONS

- Medications:
    A. <u>Anti-hypertensives</u> to maintain BP<140 mmHg
        a. IV Push: labetalol 10mg or hydralazine 10mg
        b. Infusion: nicardipine 5-15 mg/h or clevidipine 1-21 mg/h
    B. <u>Anticoagulation reversal</u> (management depends on anticoagulant)

a. Discontinue the anticoagulant
b. Vitamin K receptor antagonist (warfarin): 10 mg IV vitamin K (no faster than 1mg/min or Fresh Frozen Plasma
c. Heparin/Low Molecular Weight Heparin: protamine sulfate (max. 50 mg)
d. Tissue Plasminogen Activator (TPA): 10 units cryprecipitate (repeat dosing to maintain fibrinogen>150 mg/dL). Alternative agents: 10-15 mg/kg tranexamic acid or aminocaproic acid 4-5 g IV
e. Direct thrombin inhibitors: Activated charcoal if patient presents within 2 hours medication administration. Dabigatran: Idraucizumab 5g (2x 2.5g/50mL consecutive infusions or as a bolus) if patient presents within 3-5 half-lives of medication administration or if renal impairment is felt to impair the clearance of the medication Consider hemodialysis of four-factor protrombin complex concentrate if idraucizumab is not available.
f. Other novel anticoagulants (Factor Xa inhibitors, apixaban, betrixaban, edoxaban, rivaroxaban): andexanet alfa alpha (bolus and 2-hour infusion, dose based on specific drug and time of last drug intake) or four-factor Prothrombin Complex Concentrate (50 IU/kg)if within 3-5 half-lives of medication administration. Activated charcoal for patients presenting within 2 hours of medication ingestion. If a PCC is used, the patient should not receive andexanet

C. Osmotherapy: for patients with significant edema as a temporizing measure prior to surgery, or in patient who are not surgical candidates.
a. Mannitol: load with 1-2g/kg, then continue 01.5 g/kg Q6h (hold for serum osmolality>320 or OG>10). May be given peripherally.
b. Hypertonic saline: 30ml of 23.4% NaCl via CVL.

- Procedures: Consider hematoma evacuation, particularly for large lobar hemorrhages or in patients with progressive neurologic decline, brainstem compression and/or hydrocephalus
- Consult: Neurology, Neurosurgery, Critical Care
- Anxiolysis&Sedation: Avoid opioids (risk of respiratory compromise, intracranial vasodilation and precipitation of herniation). Limit/avoid sedation to allow frequent neurologic assessment.
- **"Assess-Treat-Reassess"**

ONGOING TREATMENT

- Monitor neurologic status Q1-4h
- Continuous BP management, osmotherapy for increasing edema, physical/occupational therapy consultation
- Consider CT angiogram to evaluate for underlying vascular malformation, MRI for evidence of CAA or (tumor) mass
- Management of complications:
  a. Cerebral edema/herniation: osmotherapy or surgical evacuation
  b. Hydrocephalus: external ventricular drainage or surgical evacuation
  c. Respiratory compromise: intubation
- Prophylaxis: Deep vein thrombosis with compression devices and compression stockings
  Stress ulcer prophylaxis in intubated patients

CAUTION

- Complications: expansion of ICH, progressive edema, obstructive hydrocephalus, brainstem compression, brain herniation, neurogenic pulmonary edema, neurogenic cardiac damage (LV wall motion affected, LV dysfunction – mimics MI), recurrent hematoma (r underlying vascular anomaly, cerebral amyloid angiopathy), death

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