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)

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

OTHER HISTORY

- Causes: atraumatic (spontaneous) and traumatic hemorrhage
- Single/multiple hemorrhages
- Predisposing conditions:
  a. Older age
  b. Hypertension
  c. Dementia (cerebral amyloid angiopathy)
  d. Vascular anomaly (arteriovenous 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 increase 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 exclude 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. **Anti-hypertensives** 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. **Anticoagulation reversal** (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: Idracizumab 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 prothrombin complex concentrate if idracizumab 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 O[>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

REFERENCES & ACKNOWLEDGEMENT


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