SPONTANEOUS INTRACEREBRAL AND INTRACEREBELLAR HEMORRAHGE

Spontaneous intracerebral hemorrhage

Hemorrhage in the brain occurring in the absence of immediate trauma.

- Primary : occurring in the absence of a structural disease process
- Secondary : is associated with a congenital or structural lesion in the brain

Historical review

- Hippocrates(400 BC) : Sanguineous apoplexy
- Hoffman(17 century): first introduced the concept of ICH
- Maceven(1888): first success operation for spontaneous ICH
- Bagley : first described surgical indication based on the location of the hematoma
- Mckissock et al (1961) : No difference in outcome after either surgical or medical management

Epidemiology

- Accounts for 10-13 % of all strokes
- Disproportionately high mortality and disability rate
- Overall incidence 10-15 cases per lakh
- Uncommon in age < 45 years, incidence increases thereafter with age(350 per lakh in >80yrs
- With the advent of CT- 30 days survival rate has increased from 8% to 44%

Risk factors

- Age
- Race
- Hypertension
- Diabetes
- Prior cerebral infarction
- Coronary artery disease

Etiologies

- Hypertension (50%)
- Vascular Malformation- aneurysm and AVM
- Coagulopathy
- Tumors
- Vasculopathy: CAA, Moyamoya, vasculitis
- Drug related
- Stroke

Pathology and pathogenesis

- Occurs predominantly in deep location
- Most common: putamen
- Other common location : subcortical white matter, cerebellum, thalamus
- Most common cause is hypertension: hence typically caused by involvement of perforating arteries: 100- 400 um.
- Target arteries: lenticulostriate and thalamoperforating arteries, paramedian branches of basilar and SCA and AICA

Contd..

- Also, autoregulation of blood flow is altered
- Vessels are less able to compensate for increased BP
- If BP is more than the limit of autoregulation- Rupture of vessels
- Small satellite hemorrhages: the marginal hemorrhages of Stemmer
- May split the tissue/ compress

Contd…

Clot Resolution: Six phase process

- Invasion by macrophages
- Development of surrounding edema
- Development of microvessels at the margin of clot
- Resorption of edema
- Lesion contraction
- Gliosis

Evolution of SICH

Hyperacute :0-24 hours- clot evolution

Acute : 1-7 days- intracellular deoxy Hb

Early Subacute : 1-2 weeks- intracellular Meth Hb

Late subacute : 2-4 weeks – extracellular Meth Hb

Early Chronic : 1-6 months- Extracellular Meth Hb, Hemosiderin

Late Chronic >6 months: Hemosidrin

CBR changes in ICH

- Hibernation phase
- Reperfusion phase
- Normalization phase

Hibernation phase

- Duration 48 hrs
- Areas of decreased perfusion in periphery of hematoma
- Concomitant decrease in cerebral metabolism
- Decrease in cerebral metabolism exceeds that in CBF
- Decreased O2 requirement, cerebral tissue is not ischemic

Reperfusion phase

- 2 days to 2 weeks
- Pattern of CBF restoration is heterogenous
- 3 different pattern of subacute blood flow: continued hypoperfusion, improved perfusion, hyperperfusion
- Metabolic activity is increased
- Ischemic injury

Normalization phase

- > 2 weeks
- Heterogenous pattern of resolution of mass effect and hematoma
- Revascularization in peripheral region of neurogliotic tissue

Distribution of Hypertensive Hemorrhage

Site	Percentage
1.Putamen	35-50
2Subcortical white matter	30
3.Cerebellum	16
4. Thalamus	10-15
5. Pons	5-12

Supratentorial hemorrhage: Symptoms and Signs

- Abrupt or acute/ Subtle
- Depends on location and size of hematoma
- General symptoms: Headache, vomiting, Dizziness, confusion, Imbalance, somnolence , stupor and coma
- General Signs; truncal and appendicular ataxia, Dysarthria, neck rigidity, EOM palsy, hemiparesis, pupillary abnormalities, decerebrate posturing

Putaminal Hemorrhage

- Most common ICH
- Specific S/S- hemiparesis, hemisensory syndrome, homonymous hemianopsia, Horizontal gaze palsies(dominant hemisphere) and hemineglect (non-dominant hemisphere)

Caudate Hemorrhage

- 5% to 7%
- Abrupt headache and vomiting followed by decreased level of consciousness
- Disoriented
- Neck rigidity
- Occ seizures and horizontal gaze palsies

Thalamic hemorrhages

- 10-15% cases
- Acute onset sensorimotor deficits
- Vomiting with or without headache,
- Altered consciousness
- Ocular findings- upward gaze palsies, convergence, miotic, unreactive pupils due to tectal compression, retraction nystagmus on upward gaze and skew deviation

Lobar Hemorrhage

- CAA – most common cause
- Headache
- Vomiting
- Seizures
- Deep coma and hemiparesis are rare

Intraventricular hemorrhage

- Generally seen as extension of intraparenchymal hemorrhage(80%) or may be isolated
- Higher mortality rate
- Initial treatment with EVD – shunt may be required if permanent hydrocephalus develops
- No role of direct surgery

Diagnostic studies

- Routine investigations
- Evaluation of heart, peripheral vessels, and kidney
- Screening for blood dyscrasias, infections, and vasculitides
- CT And MRI: both for initial diagnosis as well as for surgical planning

CT classification of basal ganglionic hemorrhage (Kanaya and Kuroda)

Class Type	Criteria
1	External capsule
2	Capsular
3a	Cp without V
3b	Cp with V
4a	Ca + p without V
4b	Ca + p with V
5	Thalamus or subthalamus

CT classification of thalamic hemorrhage (Kanaya and Kuroda)

Class	Criteria
1a	Localized to thalamus
1b	localized to thalamus with V
2a	internal cap without V
2b	internal Cap with V
3a	hypothalamus/midbrain without V
3b	with V

Prognostication Criteria

- Basal ganglionic clot > 85 ml or 6% of brain volume
- Cerebellar clot >3 cm in diameter
- Involvement of posterior limb of internal capsule
- Medial limb of hematoma< 28 mm from pineal
- Lateral edge >32 mm from pineal

Infratentorial Hemorrhage Signs and Symptoms

Cerebellar Hemorrhage

- 5-10%
- Hypertension and anticoagulation
- H/A, vomiting, neck stiffness, gait ataxia, Dysarthria, LOC in delayed and neglected cases
- Appendicular Ataxia, ipsilateral gaze palsies, peripheral facial palsy
- Usually deteriorate fast and in unpredictable manner- urgent surgical intervention

Brainstem Hemorrhage

- Pons- most common site
- Basilar long and short perforating arteries
- H/A, vomiting, Focal pontine signs- facial or limb numbness, deafness, diplopia, quadriparesis, hemiparesis, autonomic disturbances
- Signs- abnormal breathing pattern, apnea, cranial nerve and long tract deficits, decerebrate posturing and multiple oculomotor findings
- Reactive, miotic, pinpoint pupils, absent horizontal gaze movements, ocular bobbing

Nonhypertensive hemorrhage

Aneurysm and AVM

- Second leading cause of SICH
- 40% of aneurysm patients have SICH
- High suspicion if clot is frontal or temporal in location, patient is young and nonhypertensive
- Diagnosis requires CT, MRI, IADSA
- Treatment- surgery, GK, embolization

Hematologic disorders

- Thrombocytopenia-ITP, decreased marrow production, Inherited disorders of function (vWD)
- Coagulation factors deficiency- Hemophilia, DIC, liver disease

Spontaneous bleeding may occur if

- Platelets<10000
- Clotting factor activity is < 1%

Tumors

- ~ 4% cases of SICH
- Pituitary tumors, metastatic tumors especially Bronchogenic ca, melanoma, choriocarcinoma, RCC, malignant gliomas
- Diagnosis is usually suspected on unusual appearance of hematoma on CT
- Surgery may be indicated depending on the clinical significance of clot and underlying disease.

Other causes

- Vasculopathy, vasculitis
- Drugs
- Hemorrhagic transformation of ischemic stroke
- Delayed post-traumatic intracerebral hematoma (DTICH)\
- Mycotic aneurysm

Medical Management

- Control BP
- ICP monitoring and Control of ICP
- Prevent seizures
- Hemodilution
- Neuroprotectants e.g. muscimol (GABA antagonist), MK 801 (NMDA antagonist)
- Moderate hypothermia (32-33 C)
- Barbiturate coma- requires mandatory ventilation and pressure support

Surgical Techniques

Stereotactic Aspiration with Fibrinolytic and Mechanical Assistance

- Mechanical devices- 4 mm cannula encasing an archimides screw
- Ultrasonic aspirator , modified nucleotome under USG guidance
- Endoscopically with ultrasonic guidance
- Open craniotomy and hematoma evacuation

Fibrinolysis

- Fibrinolysis using catheter inside the hematoma by t-PA
- Urokinase(5000- 6000IU bd)

Open Evacuation

- Craniotomy and evacuation of clot

Prognostic factors

- Hematoma location
- Hematoma volume
- Timing of surgery
- Level of consciousness
- Age

Trials evaluating the role of surgery in patients of SICH

Junela et al J Neurosurgery 1989

- RCT
- 52 patients (surgical-26, medical-26)
- Patients with decreased level of consciousness and severe neurologic deficits were included
- 6 months mortality 46 in surgery group c.f. 38%, functional independence at 6 months is 7% in surgery group c.f.31% medical group

Auer et al J Neurosurgery 1989

- 100 patients (50/50)
- Supratentorial h'age >10cc included
- Patients with surgical apparent cause were excluded
- Stereotactically guided endoscopic drainage
- Early mortality rate< 1 week – 14% in surgery group c.f. 28.1%
- 6 months mortality rate – 42% c.f.70%

- Hematoma size<50cc- good functional outcome
- Hematoma size>50cc – lower mortality rate but no difference in outcome
- Most benefit was seen in patients with lobar hematoma and subcortical hematoma
- No benefit in putaminal and thalamic bleed

Chen et al

- n=127 (64/63)
- Craniotomy and evacuation
- No significant difference in mortality in two groups
- Increased risk of dependence and death after surgery

Morgenstern et al (STICH) trial

- N=34 (17/17)
- Cases with intraventricular hemorrhages were excluded from the study
- mortality at 1 month – 6% in surgically treated group c.f. 24% in medically treated group
- Both the groups were not comparable

Zuccarello et al Stroke 1999

- N = 20 (9/11)
- No significant difference was seen in both the groups

Variables affecting outcome

1. Hematoma location
   - Basal ganglia bleed mortality rate ~50%
   - Mckissok et al – 75% mortality c.f.62%
   - Brambilla et al – 71.5% mortality in surgical treated basal ganglia bleed c.f. 26.1%
   - Auer et al demonstrated higher rate of good outcome in surgically treated lobar hematoma c.f. basal ganglia hematoma

2. Hematoma volume

- Endoscopic evacuation of hematoma > 50cc (n=22) decreased mortality c.f. medical Rx but didn't improve survival quality
- Endoscopic evacuation of hematoma <50cc (n=28) didn't decrease mortality but improve survival quality

Auer et al JNS 1989;70:530-35

3. Timing of surgery

- Morgenstern et al – demonstrated improved outcome in patients treated with in 12 hrs of symptoms
- Higher rehemorrhage rate in patients treated within 4 hrs of onset c.f. the patients treated with 12 hours of onset of symptoms
- Higher mortality rate in patient with rehemorrhage

4. Level of consciousness

- Brambella et al demonstrated worse outcome in patients with GCS<7
- Broderick et al showed that 30 days mortality rate in patients with GCS<8 and hematoma volume >60cc is 91% c.f. 19% mortality rate in patients with GCS>8 and hematoma volume <30cc

Conclusion

- Spontaneous intracerebral hemorrhage is a significant cause of mortality and morbidity.
- Mortality and morbidity approaches 50%
- Surgery may not be beneficial in all cases.
- In selected patients, Early surgery may be beneficial.