Sonography of the Infant Brain
Hemorrhage and Ischemia

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I have no disclosures

Historical Perspective:
pneumoencephalography

Learning Objectives

Learn the ultrasound appearance of hemorrhagic and hypoxic-ischemic insults to the brain of premature and fullterm infants

Learn the imaging and clinical sequelae of these insults

Transducer Frequency

8-5 MHz
9-4 MHz
5-2 MHz
12-5 MHz
17-5 MHz

Technique

coronal planes

Technique

sagittal planes
When routine coronal, sagittal, and mastoid images do not completely image abnormalities, use whatever approach works!

- Oblique views via anterior fontanelle (demonstrate shunt tube)
- Posterior fontanelle views (posterior fossa lesions)
- Axial views (course of shunt tube, extraaxial fluid collection, brainstem, cerebral peduncle, various intracranial vessels)

### Anatomy

**coronal anterior**

- Orbits
- Interhemispheric fissure
- Periventricular blush frontal horns

**anterior/mid coronal**

**mid/posterior coronal**
Anatomy
posterior coronal

Extraaxial Fluid Spaces

Sinocortical width : 0.4 - 3.3 mm
Craniocortical width : 0.3 – 6.3 mm
Interhemispheric width : 0.5 – 8.2 mm

Corpus Callosum

- Largest medial interhemispheric commissure
- Fibers interconnect cerebral hemispheres
  - sharing memory and learning
- Forms during 3rd-4th fetal month
- Bud from lamina terminalis


Anatomy
mid sagittal

Anatomy
mid sagittal
Anatomy

mid sagittal

Genu
Body
Splenium
Cerebellum
Cisterna magna
Brainstem

Presence, number, configuration of gyri proportional to gestational age of infant

Anatomy

parasagittal

Sylvian fissure
Insula
Frontal horn
Body
GM
Caudate nucleus
Thalamus
Caudothalamic groove
Glomus choroid plexus
Periventricular blush
Occipital horn
Temporal horn

Ultrasound Diagnosis

hemorrhage

Highly sensitive and accurate
Rapid evaluation
Portable capability
No radiation
No contrast
No sedation
Useful for serial followup
Useful predictor of outcome

Intracranial Hemorrhage (ICH)
Intraventricular Hemorrhage (IVH)

premature infant

✓ < 32 weeks gestation
✓ < 1500 gms birth weight
✓ Incidence 35-50% prior to 1970s
✓ Reduction to 15-20% very low BW infants (antenatal corticosteroids, postnatal surfactant)
✓ 90% within 1st 7 days of life (1/3 within 1st 24 hours)

Symptomatology

✓ None
✓ Sudden decrease hematocrit
✓ Rapid increase head circumference
When to perform US

- Optimal cost-effective timing to screen premature infants 1-2 weeks
- Screening most often 3-7 days of age
- If none found, repeat US at approximately term (36-40 weeks) to look for sequelae (hydrocephalus, periventricular leukomalacia)
- If ICH, clinical follow-up determines timing for follow-up scan

Risk Factors ICH

**premature infant**

- Extreme prematurity and low birth weight
- Hypoxia
- Hypertension
- Hypercapnia
- Hyperosmolarity
- Hypocoagulation

Risk Factors ICH

**premature infant**

- Hypernatremia
- Rapid volume increase
- Pneumothorax
- PDA
- Intracerebral vasodilatation
  - vasoactive substances (e.g., prostaglandins)
- Factors associated with ↑ or ↓ cerebral blood flow

Germinal Matrix

- Fine network blood vessels & primitive neural tissue
- Highly cellular, richly vascular, metabolically active in developing brain
- Zone neuronal/gial proliferation
- Early gestation forms entire wall ventricular system
- Regresses after 3rd month gestation
- 24 weeks persists mainly over head caudate nucleus anterior to caudothalamic groove
- Involutes by 32 weeks

Intracranial Hemorrhage

**US classification premature infants**

**Classification ICH**

*premature infant*

- **Gr 1** = GMH
- **Gr 2** = IVH, no ventricular dilatation, ± GMH
- **Gr 3** = IVH, ventricular dilatation, ± GMH
  - **Gr 3a** = mild IVH, mild ventricular dilatation, ± GMH
  - **Gr 3b** = moderate IVH, moderate ventricular dilatation, ± GMH
  - **Gr 3c** = severe IVH, severe ventricular dilatation, ± GMH
- **Gr 4** = IVH, ventricular dilatation, ± GMH, IPH

**US Classification ICH**

*premature infant*

- Identification infants in whom structural changes may be expected to resolve or progress
- Identification infants who may require intervention
- Most gr 1 and 2: nl followup US
- Most gr 3a: nl followup US or no change
- Most gr 3b, 3c, 4: persistent structural changes

**Germinal Matrix Hemorrhage**

- Typically begins in ganglionic eminence of GM
- Uniformly echogenic mass ant to caudothalamic groove
- May be unilateral or bilateral
- Larger hemorrhages may cause focal compression of inferolateral margin of ventricle
- If hemorrhage significant, may rupture through ependyma into ventricular system

**31 week asymptomatic premie**

**GMH may result in subependymal cysts**
Intraventricular Hemorrhage
- May result from intraventricular extension of GMH
- May be unilateral or bilateral
- Blood initially appears brightly echoic
- May be difficult to identify in non-dilated ventricle
- Large hemorrhage forms cast of ventricle
Grade 3b
moderate IVH
moderate ventricular dilatation
± GMH

Grade 3b IVH
posterior fossa

Grade 3c
severe IVH
severe ventricular dilatation
± GMH

Grade 3c

IVH

✓ Clot undergoes internal liquefaction
✓ Clot retracts from ventricular walls
✓ Clot fragmentation, absorption
✓ Septation may persist
✓ Ependymal lining thickened, hyperechoic as response to chemical ventriculitis from IVH (7 days – 6 weeks)

IVH resolution process
Ventricular Enlargement

- Initially, due to distention by hemorrhage
  - Resolves, persists, progresses
- Post-hemorrhagic hydrocephalus
  - Obstructive (usually at aqueduct)
  - Communicating
    - CSF not resorbed by arachnoid granulations
    - Inflammatory ependymitis may cause persistent ventriculomegaly
- RI > 0.8 sign of increased ICP
- LPs, ventricular punctures, reservoirs, shunts

Posthemorrhagic Hydrocephalus

Intraparenchymal Hemorrhage

**Grade 4**

**IVH**

**Ventricular Dilatation**

± **GMH**

**Parenchymal hemorrhage**

**Grade 4**

**Intraparenchymal Hemorrhage**

**IPH**

- Most often due to hemorrhagic periventricular infarction
- Associated with IVH in 80% of cases
- Occurs on side with more severe IVH
- Frontoparietal distribution
- Porencephalic cyst results (communicating)
Grade 4
1 month later

Grade 4
another month later

IPH
caused by periventricular hemorrhagic infarction

- Due to increased pressure terminal v SE region
  (drains medullary vv in periventricular white matter)
- GMH - IVH complex obstructs terminal v
- Increased periventricular pressure impairs blood flow

26 week gestation
GMH

Terminal Veins
displaced by GMH bilaterally

Periventricular Hemorrhagic Infarction


Morbidity & Mortality with ICH

Premature infants
relates to hydrocephalus and parenchymal damage to descending white matter tracks

- Grades 1 & 2: risk same as gr 0 (12 - 18%)
- Grades 3 & 4: risk of major neurological handicap
  - Developmental delay
  - Mental retardation
  - Motor disabilities
  - Contralateral hemiparesis gr 4

Intracranial Hemorrhage
term infant

- Less common than in premature infants
- Risk factors
  - 25% no definite pathogenic factor
  - Birth trauma
  - ECMO
  - Hemorrhagic infarction
  - Coagulation defects
  - Apnea

Intracranial Hemorrhage
term infant

- Many are asymptomatic
- May present with seizures
- Location - cerebrum, cerebellum subarachnoid, subdural, epidural
- Small for gestational age
- Higher incidence in vaginal deliveries

FT NB TOF S/P Cardiac Cath
seizure

SAH/SDH Term Infants

- US offers less complete evaluation of extra-axial fluid spaces
- Associated with asphyxia, trauma, DIC
- Stand-off pad, high frequency linear transducer, or axial trans temporal view may aid in diagnosis
- Unilateral/bilateral hypoechoic fluid collections surrounding the brain parenchyma
**Enlarged Extraaxial Fluid Spaces**

- Head circumference > 97%

**Seizure**

**SDH/SAH**

- SDH - vessels compressed onto brain surface
- SAH - vessels traverse fluid - "cortical vein sign"

**Cerebellar Hemorrhage**

- Uncommon event in premies/fullterm infants
- In preemies associated with GMH/IVH
- In fullterm neonate associated with traumatic delivery and coagulation defects
- GMH 4th ventricle
- PDA/acidosis
- Poor prognosis
- Frequently fatal
- Mastoid view helpful

**Hypoxic-Ischemic Insults to Infant Brain**

- Determines region of brain damage
- Watershed changes during last trimester
- Premature
  - periventricular leukomalacia
- Full-term
  - cortical/subcortical regions
Periventricular Leukomalacia (PVL)

- Hypoxic-ischemic event premature infant

- 4-15% low birth weight infants (<1000 g)
- Prevalence increases with duration of survival
- Association maternal chorioamnionitis
- Edema from infarction/hemorrhage

Periventricular white matter (coagulation necrosis):
- Frontal cerebral white matter near foramina of Monroe
- Level of optic radiations adjacent to trigones lat ventricle

PVL pathogenesis

- Related to:
  - Immature vasculature in periventricular watershed
  - Junctional zone of end aa lacks collateral circulation
  - Lack of cerebrovascular autoregulation in premature
  - Vulnerable glial cells (actively differentiating into astrocytes & oligodendroglia)

PVL sonographic findings: 1st 10 days

- Generally bilateral symmetric coarse, globular, broad bands of echogenicity in PV white matter (28% patients)
  - White matter gliosis
  - Hemorrhage
  - Edema due to cortical infarction extending to deep white matter

PVL sonographic findings: 2-3 weeks

- Multiple small cysts due to necrosis and cavitation
Periventricular leukomalacia (PVL)

- 1-3 months
  - Periventricular white matter undergoes coagulation necrosis
  - Followed by phagocytosis of the necrotic tissue
  - Decrease myelination/dilatation lateral ventricles
  - More severe cases - cystic cavities
  - Differentiate from grade IV GMH (clearly separate from ventricle)

- Cysts single/multiple (mm - 2 cm diameter)
  - Small cysts may collapse and disappear
  - White matter gliotic scars develop

- 
  - ↓ cerebral myeline and ↑ vents (region of trigone and occipital horns)
  - Larger cysts resolve or become non-communicating porencephalic cysts

Hypoxic-Ischemic Encephalopathy (HIE): fullterm (FT) infants

- 36-40 wks gestation: watershed moves towards cortex
- 44 wks: watershed between endfields of A, M, & PCAs completely peripheral
- Involves cortical and subcortical white matter

Hypoxic-Ischemic Encephalopathy (HIE):

- Initially (cerebral edema)
  - Slit-like ventricles
  - Obliteration extra-axial fluid spaces, sulci, interhemispheric fissure
  - ↑ cerebral echogenicity diffuse of focal (subcortical, PV white matter, thalamus, basal ganglia)
Brain Edema

- Loss of diastolic flow
- Retrograde diastolic flow
- No detectable flow in cerebral arteries

HIE FT Infants

US findings

- Follow-up scans
  - Atrophy (↑ vents, ↑ extra-axial fluid in sulci and interhemispheric fissure)
  - Multicystic encephalomalacia (2° to necrosis)
  - Head circumference useful to differentiate brain atrophy from hydrocephalus

Cerebral Cortical Infarction

- Uncommon in neonate and young infant
- Predisposing factors
  - Prematurity, severe birth asphyxia, congenital heart disease (L→R), polycythemia/hyperviscosity, trauma, meningitis, thromboembolism (from placenta or systemic circulation)
  - Most often MCA distribution
- Acute phase
  - Absent gyral definition, absent vascular pulsations, altered parenchymal echogenicity, territorial distribution, midline shift, ventricular compression

Cerebral Edema

Atrophy

Premature infant - follow-up
Cerebral Cortical Infarction

- Stage of resolution
  - Pulsations gradually return, cystic spaces develop
- Seizures and hemiplegia common following stroke in FT infant

33 week old male born with thrombocytopenia, petechiae
Summary

- Discussed the ultrasound appearance of hemorrhagic and hypoxic-ischemic insults to the brain of premature and fullterm
- Discussed the imaging and clinical sequelae of these insults

Thank You!