Insults to the Term Brain

Monica Epelman, MD
mepelman@nemours.org
No disclosures
Imaging of the brain in full-term neonates: does sonography still play a role?

Received: 20 December 2005 / Accepted: 24 January 2006 / Published online: 16 May 2006
© Springer-Verlag 2006

Pediatr Radiol
DOI 10.1007/s00247-011-2238-5

Abnormal corpus callosum in neonates after hypoxic-ischemic injury

Monica Epelman • Alan Daneman • William Halliday • Hilary Whyte • Susan I. Blaser

Head Ultrasound and MR Imaging in the Evaluation of Neonatal Encephalopathy: Competitive or Complementary Imaging Studies?

Monica Epelman, MDb,* Alan Daneman, MD, FRCPc,b Nancy Chauvin, MDc, Wolfgang Hirsch, MDc

Neonatal encephalopathy: a prospective comparison of head US and MRI

Monica Epelman • Alan Daneman • Christian J. Kellenberger • Abdul Aziz • Osnat Konen • Rahim Moineddin • Hilary Whyte • Susan Blaser

Received: 19 October 2009 / Revised: 24 January 2010 / Accepted: 1 March 2010
© Springer-Verlag 2010

DOI 10.1007/s00247-015-3357-1

 ADVANCES IN PEDIATRIC NEURORADIOLOGY

Neurosonography: in pursuit of an optimized examination

Alan Daneman1 • Monica Epelman2,3,4

Easily Overlooked Sonographic Findings in the Evaluation of Neonatal Encephalopathy: Lessons Learned From Magnetic Resonance Imaging

David Dinan, MD,* Alan Daneman, MD,* Carolina V. Guimaraes, MD,* Nancy A. Chauvin, MD,* Teresa Victoria, MD, PhD,* and Monica Epelman, MD*
Literature comparing US and MR has deficiencies…

- Retrospective studies
- Long time interval between US and MR exams in the same patient
- US exams not “state of the art”
  - No use of linear transducers
  - No Doppler information
- US images frequently not shown in articles!

BUT...it is generally believed that US is less good at imaging of the periphery of the brain and that in these areas, CT or MRI may provide better assessment.

**HII: Head US**

*Neonatal encephalopathy (NE) is a major cause of mortality and morbidity in newborns.*

*NE occurs in 1 to 6 per 1000 live full-term births and is the most important cause of brain damage in the newborn.*
CP: What is it?

Group of disorders that
• present after birth
• characterized by abnormal control of movement or posture
• absence of recognized underlying progressive disease

Not a single disease, but group of conditions
• different parts of body involved
• other associated disabilities
CP: USA Statistics

✓ 4,000,000 live births / year
✓ 2-3/1,000 live births = cerebral palsy
✓ 8,000-12,000 cerebral palsy/year
✓ 10-15% of CP is acquired through known brain injury, infection or trauma after first month of life
CP: What is it?

Non progressive disorder of posture and movement caused by a defect or insult to the central nervous system

Static encephalopathy with a delayed developmental presentation

→ it may appear to worsen by 2 y.o. of age

However, changes are the result of the deficits becoming more obvious as the child grows and matures.
PRETERM NEONATES

TERM NEONATES

Mild-Moderate Hypotension
PV white matter injury

Severe Hypotension
Deep gray matter (thalami) injury (most metabolically active)

Mild-Moderate Hypotension
Subcortical white matter parasagittal cortex

Severe Hypotension
Thalami, BG, C-S tracts, peri-rolandic cortex injury (most metabolically active)

long penetrators in this 23 weeks premature (red arrows)
HII: Classical US findings

- Focal or diffuse increase in parenchymal echogenicity
- Slit-like ventricles
- Obliteration of the extra-axial spaces
HII: Less recognized US findings

1. Peripheral brain findings

2. Central brain findings

3. Doppler findings
HII: Less recognized US findings

1. Peripheral brain findings
   a. Gray-white matter differentiation
   b. Cortical abnormalities
   c. Subcortical white matter
   d. Extra-axial spaces abnormalities

2. Central brain findings

3. Doppler findings
HIII: Less recognized US findings

1. Peripheral brain findings

2. Central brain findings
   a. Basal ganglia evaluation
   b. Periventricular white matter incl. medullary veins
   c. Active hemorrhage evaluation
   d. Ventricular size evaluation
   e. Brainstem evaluation
   f. Corpus Callosum evaluation

3. Doppler findings
HIII: Less recognized US findings

1. Peripheral brain findings

2. Central brain findings

3. Doppler findings
   a. Resistive indices (RI) fluctuation
   b. Hyperemia
   c. Sinus vein patency evaluation
1. HII: Peripheral US findings

1a. Gray white matter differentiation
   a. Accentuation
   b. Loss
   c. Mixed pattern

1b. Cortical abnormalities
   a. Abnormal cortical thickness and echogenicity

1c. Subcortical white matter
   a. Focal peripheral echogenicities

1d. Extra-axial spaces abnormalities
   a. Subdural collections
   b. Blurring interhemispheric fissure
1a. Gray white matter differentiation

“In infants with extensive white matter disease corticomedullary differentiation is considerably enhanced”


ORIGINAL ARTICLE

White—gray matter echogenicity ratio and resistive index: sonographic bedside markers of cerebral hypoxic–ischemic injury/edema?

PS Pinto¹, A Tekes¹⁻³, S Singhi¹, FJ Northington²⁻³, C Parkinson²⁻³ and TAGM Huisman¹⁻³

¹Division of Pediatric Radiology, Russell H. Morgan Department of Radiology and Radiological Science, Johns Hopkins Hospital, Baltimore, MD, USA; ²Division of Neonatology, Department of Pediatrics, Johns Hopkins Hospital, Baltimore, MD, USA and ³Neuro Intensive Care Nursery Program, Department of Pediatrics, Johns Hopkins Hospital, Baltimore, MD, USA
1a. Gray white matter differentiation

Normal G-W differentiation

**Coronal**

**Sagittal**

Peripheral-angled views

*These images show the normal G-W differentiation, The sulci are of medium echogenecity *(green line)* The cortex is slightly hypoechogenic *(yellow)* when compared to adjacent subcortical white matter *(★)*
These images show abnormal accentuation of the G-W differentiation. The sulci are thick and hyperechogenic, the cortex is hypoechogenic and the subcortical white matter is hyperechogenic in the regions of restricted diffusion.
1a. Gray white matter differentiation

Loss G-W differentiation

These images show loss of the G-W differentiation on both, US and DWI.
1b. Cortical abnormalities

Abnormal cortical thickness and echogenicity in cortical laminar necrosis

Follow up US, 24 hs later show diffuse blurring of the sylvian fissures (green arrows) and abnormal thickness and echogenicity of the parietooccipital cortex (red arrows).

Corresponding axial noncontrast T1W image shows laminar hyperintensities in the bilateral parietooccipital cortex consistent with cortical laminar necrosis.
US images show patchy abnormal subcortical areas of increased echogenicity in the left perisylvian region (red arrows) Corresponding to the left perisylvian infarct with hemorrhagic conversion /hematoma seen on MRI
1d. Extra-axial spaces abnormalities: Subdural collections

Meconium aspiration. Evaluation prior to ECMO. US images show abnormal fluid collection encircling the right cerebral hemisphere (red arrows).

Corresponding CT images show bilateral subdural hemorrhages.
1d. Extra-axial spaces abnormalities:
Blurring of the inter-hemispheric fissure
Group B streptococcus (GBS)

- common, partially preventable
- if maternal cervical colonization,
  - 10% \(\Rightarrow\) systemic GBS (1-5 per 1000 births)
  - \(\Rightarrow\) meningitis in 5-10%
- ↑ risk in: PROM, prematurity, maternal chorioamnionitis
- VLBW 70% mortality
- full term: insidious onset, meningitis/osteomyelitis
  - 5% mortality
  - significant hydrocephalus, developmental delay, seizures
Thrombophlebitis

- organizing exudate with fibroblast proliferation fills sulci, extends along (and bathes) vessels
- ... leading to vascular occlusion

Courtesy Dr Susan Blaser
2. HII: Central US findings

2a. Basal Ganglia evaluation
   - Echogenicity pattern → important

2b. Periventricular white matter incl. medullary veins

2c. Active IVH

2d. Ventricular size evaluation
   - Easiest to assess, but least useful

2e. Brainstem evaluation
   - Less common → still important

2e. Corpus Callosum evaluation
2a. Basal ganglia abnormalities

Fullterm neonate with a history of profound HII. Coronal US image shows diffuse increased echogenicity of the basal ganglia (red arrows) and slitlike ventricles. Axial DWI shows restricted diffusion in the bilateral posterior putamen (green arrows) and ventrolateral thalami (blue arrows).
2a. Basal ganglia abnormalities
2a. Basal ganglia abnormalities

Coronal US image shows bilateral areas of abnormally increased echogenicity involving the thalami (red arrows). Note blurring of the interhemispheric fissure (blue arrows).

Corresponding axial T2-weighted image reveals extensive edema in the bilateral thalami (green arrows), periventricular white matter and within both frontal regions. These findings are characteristic of deep cerebral venous thrombosis involving the bilateral thalamoperforating veins and bilateral internal cerebral veins.
2a. Basal ganglia abnormalities

There is subtle increased echogenicity on the right basal ganglia (yellow arrow) when compared to the left (green arrow).

The MR images confirm the areas of abnormality on the left and show this more floridly than the US.
2b. Periventricular white matter abnormalities incl. medullary veins
Evaluation of medullary veins
Fetal MRI. 20 weeks gestation. Normal medullary veins (arrows) can be seen in HASTE and EPI sequences.

Fetal MRI. 28 weeks gestation. Normal medullary veins (arrows) can be seen in EPI sequences.

Courtesy Dr. Tamara Feygin
HII: Hypoperfusion

Medullary venous congestion in twin-twin transfusion

Courtesy Susan Blaser MD
Ex-premature at 31 wks 11 day old

Periventricular hemorrhagic venous infarction

It is important to differentiate GM-IVH from bilateral hemorrhagic PVL. These two entities are distinct in their neurodevelopmental outcome, which is more favorable for the GM-IVH.
2b. Periventricular white matter abnormalities incl. medullary veins

Sagittal US images show increased periventricular echogenicity consistent with PVL (red arrows). These findings are most conspicuous on the image on the left obtained with a linear transducer and reflect small areas of ischemia and/or hemorrhage in medullary vein distribution (green arrows).
2b. Periventricular white matter abnormalities incl. medullary veins

6-day-old with large SCT and acute drop in hemoglobine. Head US shows a left grade 1 IVH and abnormal accentuation of the periventricular medullary veins, noted as periventricular linear echogenicities (yellow arrows). These are believed to play an important role in the physiopathology of PVL
Us movies depict active left grade 1 IVH
Normal brainstem echo pattern 
(green arrows)

Asymmetry with heterogenous echotexture of the abnormal left cerebral (yellow arrow) peduncle

Corresponding abnormal brainstem restricted diffusion in the left cerebral peduncle (yellow arrow)
2d. Brainstem evaluation

Neonate with HII. Asymmetry of the cerebral peduncles with abnormal increase in echo-texture on the left (yellow arrows) noted on axial US scans through the mendosal suture.

Corresponding DWI confirms restricted diffusion within the left cerebral peduncle (blue arrows).
2e. Ventricular size evaluation

*Least useful, may be normal in severe cases*

Full-term baby with home delivery and abruptio placenta. PPHN, hypoxemia, respiratory distress and abnormal EEG. US shows **normal ventricular size**. Note moderate accentuation of the GW differentiation, abnormal basal ganglia echogenicity and blurring of the interhemispheric fissure.

Corresponding MRI shows extensive bilateral restricted diffusion involving the cerebral hemispheres bilaterally with sparing of the unmyelinated white matter. The ventricles are normal in size, shape, and configuration.
Newborn with severe HII. The corpus callosum (red arrows) is thickened and shows abnormal increase in echogenicity.
3. HII: Doppler US findings

3a. Resistive indices fluctuation
3b. Assessment of hyperemia → Dr Faingold
2c. Sinus vein patency evaluation
3a. Resistive indices (Ris) fluctuation

In this patient the Doppler study of the ACA shows fluctuation of the resistive index in images taken 2 minutes apart.

FLUCTUATION of the RI’s during the same exam, probably due to loss of autoregulation.
In this patient hyperemia is noted in the basal ganglia bilaterally on Color and Power Doppler and this was confirmed on the MRA. In addition prominent lenticulostriate vessels are appreciated on T2 weighted images as flow voids.
Dynamic Cerebral CDS - CPI

Courtesy Dr R. Faingold, Montreal, QC, Canada
Metabolic disorder
Neonatal Cerebral Sinovenous Thrombosis
- Transient abnormal protein C with infection and birth
- Poor outcome

US and MRI images show extensive superior sagittal vein thrombosis. No flow is elicited in the superior sagittal sinus (arrows) on color Doppler imaging despite adequate low settings (as low as 2.2 cm/s)
Optimal US results...

- Real time imaging
- Multiple transducers
  - High frequency (~8 MHz)
  - Vector, curved array, linear
- Multiple windows and focused views
  - Angled views to include the periphery of the brain; Central focused views; Posterior fossa
- Doppler - spectral, color, power
- Implementation of rounds including:
  - Sonographers
  - Neuroradiologists
  - Neonatologists

Implementation of these practices and techniques will improve the reliability of HUS to depict HII related changes
Thank You
for your attention!!! –
Any questions?

♦ Special Thanks:
  • Drs. Alan Daneman and Susan Blaser, Toronto, Sick Kids, Canada
  • Drs. Ricardo Faingold and Natalia Gorelik, Montreal Children’s Hospital, Canada
  • Drs. Tamara Feygin, Zimmerman, Nancy Chauvin & Tedi Victoria, CHOP, Philadelphia, USA