Ultrasound imaging of the neonatal brain

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Learning objectives

1. To evaluate the ultrasound brain anatomy and anatomical landmarks in neonates

2. To analyze the different patterns of brain injury, differentiating two groups, premature and term infants

Background

Brain lesions are a major cause of morbi-mortality in neonates. Hypoxic-ischemic injuries have an incidence of 1-8 cases/1000 births and are responsible for up to 25% of all neonatal deaths.

In this age group, usually they present with nonspecific manifestations that also appear in different pathologies like enterocolitis, various infections and metabolic disorders [1, 2].

Hence, it was necessary to develop a rapid and cost-effective method with good sensibility in order to depict the hypoxic-ischemic lesions.

The impact of the correct diagnostic and treatment is crucial: perinatally it frequently has a vital importance and later on influences the neurological and functional outcome.

Imaging findings OR Procedure details

We reviewed the ultrasound (US) imaging studies obtained in a group of premature and term neonates.

The studies were performed with high frequency linear array transducer (10 mHz), by direct contact with the anterior fontanelle, using a standardized protocol with sagittal and coronal images of the whole brain.

We evaluated the anatomy in premature and term infants (Fig. 1 on page 6 - Fig. 5 on page 10).
Among the group of patients with pathology we classified brain injuries according to the two different groups:

I-Hypoxic-ischemic brain injury in the premature infants (Fig. 6 on page 11 - Fig. 9 on page 15):

1. White matter injury
2. Germinal matrix hemorrhage
3. Intraventricular hemorrhage
4. Periventricular (PV) hemorrhage
5. Infarction
6. Posterior fossa injury

II-Hypoxic-ischemic brain injury in the term infants (Fig. 10 on page 17 - Fig. 15 on page 24):

1. Cerebral cortex injury
2. Boundary zone injury

The hypoxic-ischemic brain injury is due to decreased cerebral blood flow or systemic hypoxemia [2].

It can have various stages. *Mild hypoxia* affects the boundary regions of vascular territories (watershed lesions). *Severe hypoxia* leads to a deeper involvement: the basal ganglia and the perirolandic cortex [2].

We analyzed the findings and correlated them with follow up MRI images.

**Indications for routine US screening:**

- *birth weight* less than 1500 g
- *gestational age* less than 35 weeks
- *suspicion* of intracranial hemorrhage or hypoxic-ischemic brain injury [2, 3].

**US of the neonatal brain:**

- efficient tool for identifying *hypoxic-ischemic injuries* (Table 1 on page 34) and *hemorrhagic lesions* (Table 2 on page 34)

- the normal PV white matter is less bright than the choroid plexus
- in the ischemic lesions and also in the hemorrhagic ones it shows increase of echogenicity

- depicts between 28% and 80% of histologically demonstrated periventricular leukomalacia (PVL)

- difficulty in depiction of mild homogeneous densities (that are associated with transient signal anomalies or even normal findings on follow-up MRI and with no effect on the patient outcome)

- irregular and inhomogeneous US densities are associated with more severe MRI findings [1-3].

**MRI utility** (Table 3 on page 35):

- in 2/3 of the infants with hypoxic-ischemic injuries provides additional information over US

- PV or cortico-subcortical areas of T2 hyperintensities in *ischemic lesions*

- better characterization of the *cysts* (showing more numerous or more extensive cysts than the US)

- better depiction of *hemorrhagic lesions*: increased signal intensity on T1WI and decreased on T2WI (in areas not normally myelinated at this age, as myelination gives similar intensities)

- important role in the group with heterogeneous echodensities (superior evaluation of the hypoxic-ischemic damage)

- extensive hemorrhages and intensities on MRI can *predict the development of cystic leukomalacia*

- when no dilated ventricles, MRI could relieve *small intraventricular hemorrhages* not detected by US

- for *subarachnoid bleeding*, MRI or CT is preferable to US [2].

**Periventricular leukomalacia** - stages:

- acute phase: increased echogenicity of the PV white matter, appearing within 24 to 48 hours after a hypoxic-ischemic event (Fig. 6 on page 11, Fig. 7 on page 12)

- late subacute phase: 2-4 weeks, cysts can arise in the hyperechogenic areas

- chronic phase: the cysts resolve, with development of ventricular enlargement [1, 2].
Hemorrhagic lesions:

- in 1/3-1/2 of all infants of less than 35 weeks gestational age or weighing less than 1,500 g
- in 2/3 of the infants which require assisted ventilation
- the spectrum of the hemorrhagic lesions: petechial, focal and extensive (at least three lobes involved in the last one); the higher the grade, the greater the tendency to cystic degeneration
- the frontoparietooccipital PV regions are the more frequently involved, but also frequently found in the subcortical white matter [3-5]
  - **Subependimal hemorrhage** (Fig. 16 on page 26, Fig. 17 on page 26)
- 90% of hemorrhages occur in the germinal matrix overlying the caudate head and 10% in the vicinity of the body of the caudate nucleus
- highly echogenic region of convex borders
- **best seen on coronal sections:** inferolateral to the lateral ventricle, just posterior to the foramen of Monro
- on sagittal scans: anteriorly and laterally to the choroid plexus (also echogenic); an useful anatomic landmark - the thalmocaudate notch
- tendency to develop cystic spaces [4, 5]
  - **Intraventricular hemorrhage** (Fig. 18 on page 27 - Fig. 24 on page 33)
- complicates about 80% of subependymal hemorrhages
- echogenic intraventricular densities
- **best diagnostic clue:** search the frontal and occipital horns on sagittal scans
- anterior to the caudate nucleus in the frontal horns (be aware that the choroid plexus shouldn't be present anterior to the foramen of Monro)
- in preterm neonates: extension pattern from the thalmodacuate notch (germinal matrix hemorrhage) within the lateral ventricle to its occipital horns
- in term infants: intraventricular bleeding is rare; the choroid plexus is the major source of the intraventricular hemorrhage [4, 5]
  - **Other hemorrhage locations**
- cerebellar, subarachnoid and small extraaxial hematomas

-the incidence of intracerebellar hemorrhage is up to 25% of preterm neonates less than 34 weeks of gestational age

-the cerebellar vermis, inherently echogenic, shouldn't be confused with blood

-they are unreliably visualized by US; consequently, the study should be completed by MRI or CT [4, 5].

**Complications:**

- **Of hypoxic-ischemic lesions**
  - cystic transformation, multicystic leukomalacia
  - basal ganglia necrosis
  - PVL: white matter loss, ventricular dilatation, abnormal T2 hyperintensity in the periventricular region [2]

- **Of hemorrhages**
  - hydrocephalus, early or late (up to 1/3 of the hemorrhages, resulting from obstruction at any level - foramen of Monro, aqueduct of Sylvius or foramen of IV ventricle - or by inflammatory mechanisms) [4, 5]

- Both
  - significant neurologic sequelae, some of long-term morbidity or even incapacitating
  - mortality (up to 1/3 of the hemorrhages)
  - generally proportional to the extension [2, 5].

**Images for this section:**
Fig. 1: Sagittal brain US at the midline. Anatomical landmarks
Fig. 2: Coronal brain US at the level of the central sulcus
Fig. 3: Coronal brain US showing the superior frontal and precentral sulci
Fig. 4: Sagittal brain US at the level of the inferior frontal gyrus and the sylvian fissure
Fig. 5: Sagittal brain US centered in the insular cortex (circle)
Fig. 6: Coronal US. Premature infant. Increased echogenicity of the PV white matter (arrows) due to hypoxic-ischemic injury in acute phase. See also fig. 7 - 9.
**Fig. 7:** Sagittal scan, the same patient as in fig. 6, 8 and 9. Hyperechogenicity of the PV white matter (arrows) due to hypoxic-ischemic injury
**Fig. 8:** Axial FLAIR sequence, 6 months after the US examination (fig. 6 and 7), shows severe encephalomalacic changes
**Fig. 9:** Coronal T2*WI, the same patient as in fig. 6-8. Extensive hyperintensity of the white matter representing severe encephalomalacic changes. Chronic phase of hypoxic-ischemic encephalopathy

![Image of T2*WI](image)

**Fig. 10:** Coronal US. Term infant. Hyperechogenicity of the white matter (arrows) corresponding to the superficial territory of the left middle cerebral artery (MCA). Continuation in fig. 11-15
Fig. 11: Continuation of fig. 10. Sagittal US showing hyperechogenicity of the white matter in the left MCA territory (arrows). MRI was indicated (see next images)
**Fig. 12:** Axial T2WI, the same patient as in fig. 10 and 11. Cortico-subcortical hyperintensity of the superficial territory of left MCA corresponding to the area depicted by US.
**Fig. 13:** Continuation of fig. 12. DWI showing diffusion restriction in the left MCA superficial territory, confirming the stroke
**Fig. 14**: Continuation of fig. 13. ADC map confirming the diffusion restriction in the area visualized on DWI
Fig. 15: Axial-FLAIR image, the same patient as in fig. 10 - 14, 1 month after the acute presentation. Cystic encephalomalacy in the posterior part of the superficial territory of left MCA.

Fig. 16: Coronal US at the level of the caudate head in a term neonate. Left germinal matrix hyperechogenicity (arrow) without ventricular involvement, corresponding to grade I hemorrhage.
Fig. 17: Continuation of fig. 16. Sagittal US showing hemorrhage restricted to the subependymal region (arrow), corresponding to grade I hemorrhage
Fig. 18: Coronal US in premature infant. Bilateral hyperechogenicity of the germinal matrix associating ventricular contamination, without hydrocephalus, corresponding with grade II hemorrhage
Fig. 19: Continuation of fig. 18. Sagittal US demonstrating a better visualization of the ventricular hemorrhage in this case
**Fig. 20:** Coronal US in extremely premature infant. Germinal matrix hemorrhage with extension into the ventricular system and subsequent hydrocephalus: grade III hemorrhage
Fig. 21: Continuation of fig 20. Coronal US demonstrating a very high sensibility for the depiction of the ventricular involvement in grade III hemorrhages.
Fig. 22: Continuation of fig. 20 - 21. Sagittal scan confirming the lesions visualized in the coronal plane
Fig. 23: Coronal US. Premature neonate with very low birth-weight. Germinal matrix hemorrhage with extension into the adjacent frontoparietal brain parenquima and also into the ventricular system with slight enlargement: grade IV hemorrhage
**Fig. 24:** Continuation of fig 23. Another coronal scan with better characterization of the right lateral intraventricular hemorrhage and the hydrocephalus

<table>
<thead>
<tr>
<th>SONOGRAPHIC GRADE</th>
<th>APPEARANCE</th>
<th>PVL GRADE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>PV densities &lt; 7 days</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>PV densities &gt; 7 days</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>PV densities evolving into localized cysts</td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>PV densities leading to extensive cysts</td>
<td>3</td>
</tr>
<tr>
<td>5</td>
<td>PV and subcortical white matter densities, evolving into extensive PV and subcortical cysts</td>
<td>4</td>
</tr>
</tbody>
</table>

*Table 1* Ultrasound findings and grading scale in hypoxic-ischemic brain injury. Adapted from De Vries et al [1]

**Table 1:** US grading scale in hypoxic-ischemic brain injury
### Table 2: Classification of periventricular hemorrhages in neonatal period

<table>
<thead>
<tr>
<th>GRADE OF THE HEMORRHAGE</th>
<th>CHARACTERISTICS</th>
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<tbody>
<tr>
<td>I (Fig 16-17)</td>
<td>restricted to subependymal regions</td>
</tr>
<tr>
<td>II (Fig 18-19)</td>
<td>grade I + extension of hemorrhage into normal sized ventricles</td>
</tr>
<tr>
<td>III (Fig 20-22)</td>
<td>grade II + dilated ventricles</td>
</tr>
<tr>
<td>IV (Fig 23-24)</td>
<td>grade III + extension into adjacent brain parenchyma (more frequently frontoparietal)</td>
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Adapted from Papile et al [3]

### Table 3: US and MRI comparison in the evaluation of the spectrum of hypoxic-ischemic brain injury in neonates

<table>
<thead>
<tr>
<th>ULTRASOUND</th>
<th>MRI</th>
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<tbody>
<tr>
<td><strong>ADVANTAGES</strong></td>
<td><strong>DISADVANTAGES</strong></td>
</tr>
<tr>
<td>- screening procedure</td>
<td>- operator dependent</td>
</tr>
<tr>
<td>- availability</td>
<td>- false negative cases: mild changes, small amount of blood in no dilated ventricles or subependymal hemorrhage in the body of the caudate</td>
</tr>
<tr>
<td>- lower cost</td>
<td>- not suitable for depiction of hemorrhage in some locations like subarachnoid, small extraaxial hematomas, posterior fossa</td>
</tr>
<tr>
<td>- good sensitivity for important lesions (which influence the patient outcome)</td>
<td>- not appropriate for screening</td>
</tr>
<tr>
<td>- acceptable (small) false positive rate</td>
<td>- less available</td>
</tr>
<tr>
<td>- real time exploration</td>
<td>- higher cost</td>
</tr>
<tr>
<td>- innocuous</td>
<td>- movement artifacts</td>
</tr>
<tr>
<td></td>
<td>- sedation</td>
</tr>
<tr>
<td></td>
<td>- technique of choice for diagnosis and follow-up of hypoxic-ischemic injuries of moderate or severe grade</td>
</tr>
<tr>
<td></td>
<td>- evaluates late sequelae of neonatal hypoxic-ischemic brain injury</td>
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<tr>
<td></td>
<td>- identifies the hemorrhage that isn’t possible through US</td>
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<tr>
<td></td>
<td>- DWI for the infarct diagnosis</td>
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<td></td>
<td>- spectroscopy: quantification of anoxic metabolites</td>
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</table>

- no operator dependent (reproducibility)
- higher sensitivity and specificity
- good predictor of the final diagnosis and outcome
- can provide additional information (better differentiation and quantification of abnormalities, especially at an earlier stage)

**Table 3** US and MRI comparison in the evaluation of the spectrum of hypoxic-ischemic brain injury in neonates

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Conclusion

US may provide important information about **brain anatomy and development** in neonates.

US should be the **initial examination in all neonates at high risk** for hypoxic-ischemic lesions and intracranial hemorrhage and it is important **to evaluate** these injuries **early** in their course.

The **pattern of involvement** differs between **premature** and **term infants**.

US provides useful data and can be used in the **follow up** to document the evolution of lesions.

References


Personal Information