Neonatal neurosonography: A pictorial essay

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Abstract

Neurosonography is a simple, established non-invasive technique for the intracranial assessment of preterm neonate. Apart from established indication in the evaluation of periventricular haemorrhage, it provides clue to wide range of pathology. This presentation provides a quick roadmap to the technique, imaging anatomy and spectrum of pathological imaging appearances encountered in neonates.

Key words: Cranial USG; germinal matrix haemorrhage; neurosonography

Introduction

Sonographic examination of the neonatal brain remains an invaluable assessment tool in experienced hands. Many radiologists are not quite familiar with the spectrum of imaging appearances of the neonatal intracranium due to inadequate exposure. Many specific clinical questions can be resolved by optimally utilizing this simple informative tool. Portability of the study, lack of need to transport baby to radiology services, and the wealth of diagnostic information available from a large open anterior fontanelle makes this study a highly challenging one.[1] This pictorial essay attempts to highlight the common indications for neurosonography, its basic techniques, sonographic anatomy, and the spectrum of pathological imaging appearances seen in neonates. With an ever-growing innovation in technique and an increasing affordability of state-of-the-art equipment, there is a greater need for the radiological community to be well acquainted with various facets of this diagnostic tool.

Indications

The main indication for this examination is the demonstration or exclusion of an intracranial hemorrhage in a preterm neonate. The technique is additionally used for the follow-up of intraventricular hemorrhage (IVH)-related complications to look for evolving findings pertaining to ischemia. Other broader indications include demonstration of congenital structural anomalies, intracranial vascular lesions, and also its use as a simple screening tool in the exclusion of gross intracranial pathology.

Cranial USG Technique

There are essential steps to be followed before the procedure. Most of the examination is performed bedside with the neonate within the incubator. Examination is preferably done through the cranial incubator opening. Care should be taken not to move the infant or to cause any disturbance to incubator temperature. Pressure over the anterior fontanelle is to be avoided, especially in a critically ill premature neonate. All aseptic precautions are to be followed in accordance with the protocols of the neonatal ICU.

Best results are obtained with a high-frequency phased array transducer (5-8 MHz) with a small footprint probe. High-resolution images are obtained in preterm neonates by using probe frequency of 7.5 MHz. Technical difficulty may be encountered in obtaining the best image in the case...
Screening via other supplemental fontanelle and obtaining high-resolution images from them may have added value. This technique has been shown to improve detection of posterior fossa hemorrhages and in the evaluation of the transverse sinuses. Mastoid and posterior fontanelle approaches are useful in the demonstration of a subtle intraventricular bleed in the occipital horn, in patients with suspected holoprosencephaly and demonstration of a minor bleed in the brain stem and adjacent cerebellum. To complete the examination, high-resolution linear-array transducer images are obtained for detailed interrogation of the convexity subarachnoid space and superficial cortex as well as deeper brain structures. Linear images can be adjunctively obtained via any fontanelle for the evaluation of underlying anatomical structure or vessels.

Translation of brain anatomy to sonography needs understanding of sonographic physical principles. The general principles of sonography apply to intracranial study. Cerebrospinal fluid (CSF) spaces are anechoic, whereas the choroid plexus, small hemorrhages, and areas of infarction appear hyperechoic. On ultrasonography (USG), gray matter tends to be hypoechoic and white matter tends to be hyperechoic. Secondly, the normal brain is always nearly symmetric. This fact allows for detection of a small anterior fontanelle [Figures 1 and 2]. Phased array transducers of small footprint and wide insonation angle (up to 140°) help to obtain examination of diagnostic quality.

Neurosonography starts with gray-scale imaging performed via the anterior fontanelle in the coronal and sagittal planes. Generally, six to eight coronal images are obtained, beginning at the anterior frontal lobes and extending to the occipital lobes posterior to the lateral ventricle trigones [Figure 3]. The transducer is then rotated 90° and five sagittal images are obtained, including a midline and two parasagittal views of right and left hemispheres encompassing the peripheral cortex [4][5] [Figure 4]. Color Doppler images for arterial and venous structures may be obtained for the screening of vascular structures [6]. Documentation of Doppler imaging of the circle of Willis and the region of vein of Galen is an essential part of the assessment [Figure 5]. Spectral tracing with peak systolic velocity (PSV), end-diastolic velocity (EDV), and resistive index (RI) need to be recorded for evaluation of ischemia. Power Doppler imaging has also been recommended by some authors to search for areas of hyper- or hypo-vascularity in suspected vascular occlusion, ischemia, or infarction [3].

Figure 1: Diagrammatic illustration showing the size of fontanelle limiting the sonographic window

Figure 2: Anatomical picture of the anterior fontanelle; size variation in normal neonates as shown by CT volume-rendered reconstruction

Figure 3: Illustration of coronal USG examination, showing frontal and posterior parietal planes

Figure 4: Illustration of sagittal and parasagittal USG examination, showing mid-sagittal and ventricular planes
of early changes of infarction or focal ischemia. Bilateral symmetric changes due to a systemic process may lead to errors in interpretation. A third fact involves interpretation of visible layers of the normal cortex. The superficial pia mater should be seen as a thin, well-defined hyperechoic layer immediately overlying the hyperechoic cortical gray matter, which in turn overlies the hyperechoic white matter.\(^9\)

Failure to distinctly visualize these normal layers is indicative of abnormalities such as focal hemorrhage or infarct.\(^9\) Lastly, the periventricular white matter is normally homogeneous in echogenicity and is equally or less echogenic than the adjacent choroid plexus.\(^9,10\) Asymmetry or heterogeneity of the periventricular white matter is suggestive of an abnormality, such as periventricular leukomalacia (PVL). Normal anatomical illustrations are shown in Figures 6-9.

An understanding of normal variation is essential to neurosonographic interpretation. Cavum septum pellucidum is present in up to 50–61% of normal neonates\(^10\). Minor asymmetry in the frontal horns or bodies of ventricles is often observed. Also, the echogenicity of periventricular parenchyma is variable. Being relatively echogenic in premature neonates, it might be wrongly interpreted as PVL. Massa intermedia can be quite variable in size in normal and pathological conditions [Figure 11]. When posterior fontanelle approaches are utilized, prominent calcar avis and lobulated glomus of the choroid should be observed as normal variations.\(^7\)

### Intracranial Hemorrhage

**Germinal matrix hemorrhage**

One of the most important indications of neurosonography is the demonstration of intracranial hemorrhage in a premature infant. Routine screening cranial USG should be performed in all infants of under 30 weeks gestation, once between 7 and 14 days of age and should be optimally repeated between 36 and 40 weeks postmenstrual age.\(^11-13\) In term infants, non-contrast computed tomography (CT)

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<th>Classification of findings</th>
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<td>Grade 1</td>
<td>Germinal matrix hemorrhage</td>
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<td>Grade 2</td>
<td>Blood within the ventricular system, but not distending it</td>
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<td>Grade 3</td>
<td>Intraventricular hemorrhage with ventricular dilatation</td>
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<td>Grade 4</td>
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*Reference 14, 15, †References 16, 17, ‡References 12, 13, §Measurements at the midbody of the lateral ventricle on sagittal image (Modified from MENT et al.\(^11\)). USG: Ultrasonography
Figure 8: Coronal and parasagittal images of preterm (28 weeks) infant showing intracranial anatomy. Note the smooth surface of the immature brain. Distribution of the lobes colored.

Figure 9 (A-D): Doppler images (A and B) demonstrating circle of Willis. Coronal USG demonstrating the vein of Galen. (C) Parasagittal study showing the small periventricular veins in the region of caudothalamic groove (D).

Figure 10 (A, B): Coronal USG demonstrating cavum septum (star) (A) and relation of vascular structures (B). Note that there are low-level echoes of cerebral gray and white matter, with subtle differences in the echoes of cortex and white matter. CSF spaces in Sylvian and inter-hemispherical region (arrows) are hyperechoic. Cerebellum is hyperechoic in relation to cerebral hemispheres.

Figure 11 (A, B): (A) Sagittal USG demonstrating a relatively large massa intermedia (arrow) (B) Illustrates the relatively hyperechoic peritrigonal white matter(open arrow) in a normal neonate.

Figure 12A: The figure demonstrates the location and the extent of the germinal matrix (colored pink).
is preferred. Areas of potential bleeding arise in the zones of primitive germinal matrices, which are more extensive around the periventricular regions of the lateral ventricles and around the temporal horns [Figure 12A]. The extent of germinal matrix diminishes with progressive maturity, limiting it to the region of caudothalamic groove. Germinal matrix hemorrhages are seen as areas of increased echogenicity in the region of the caudothalamic groove. Germinal matrix hemorrhages were classified into four categories by Papile based on the extent of hemorrhage [Figure 12B][14] [Table 1].

Grade 1 hemorrhage is limited to the region of the caudothalamic groove, usually less than a centimeter in size [Figure 13]. Grade 2 hemorrhages extend into the adjacent ventricles, but do not cause ventricular dilatation. Grade 3 hemorrhages show ventricular extension and show minimal increase in the ventricular dimensions [Figures 14 and 15]. Grade 4 hemorrhages, presently considered as venous infarct, present with a predominant intraparenchymal component of hemorrhage, often with a sizable mass effect [Figures 16-19]. Posterior fossa hemorrhage is uncommon and can be demonstrated when sizable. Hemorrhages of grade 3 and 4 are associated with neurological deficits or learning disability.[11] Evolution of hemorrhage is demonstrated with follow-up sonograms [Figure 20].

**Periventricular Leukomalacia**

While the germinal matrix hemorrhages are a result of relatively acute hemodynamic changes, PVL represents a relatively insidious cerebral parenchymal insult. Chronic hypoxemia or hypoperfusion leads to PVL. On
pathological studies on infants more than 6 days of age, there is high incidence of PVL in low-birth-weight infants between 900 and 2200 g. \(^1\) Sonographic grading of PVL was described by Di Vries.\(^{15-16}\) Grade 1 PVL generally presents as an increase in the parenchymal echoes in the periventricular region, mainly around the trigone, lasting less than 7 days [Figure 21]. These early sonographic findings are described as a periventricular flare. Some element of uncertainty of interpretation is noted in the extremely premature infant, in whom periventricular parenchyma is often echogenic [Figure 13]. Grade 2 PVL presents as persistent periventricular hyperechogenicity lasting more than 7 days. Grade 3 PVL presents with relatively advanced parenchymal changes leading to microcyst formation [Figure 22A and B]. These changes are more evident in the parietal region and also extend to the frontal areas. Grade 4 PVL represents with multiple coalescing cystic areas in the cerebral parenchyma. Grade 4 changes represent advanced cystic changes reaching up to the level of cortex. Grade 3 and 4 changes are often associated with neurological sequelae such as diplegia and paraplegia in later life in upto 50% of patients [Figure 23]. Owing to the low sensitivity of sonography in the detection of non-hemorrhagic, non-cavitary parenchymal injury, additional imaging studies are usually necessary.\(^1\)

**Acute Ischemia**

Evaluation of diffuse brain edema is technically challenging on neurosonography. As the size of the ventricles varies considerably, ventricular size is unreliable as a parameter...
in assessing the mass effect. The usual observation in the cases of ischemia is a combination of diffuse increase in the echogenicity of ganglionic areas with associated obliteration of cisterns and small capacity of the ventricles [Figure 24].

CT and/or magnetic resonance imaging (MRI) still remain as superior techniques in assessing diffuse intracranial ischemia [Figure 25]. Serial Doppler examination of the intracranial vessels and circle of Willis is helpful in evaluating the severity of intracranial ischemia. Diastolic flow, reflected in Resistive Index (RI) is a measure that will indicate the hemodynamic status of intracranial flow.\(^{[2,12]}\)
Porencephalic Cyst

Large foci of intraventricular/intraparenchymal bleed could lead to a cavitating destructive lesion in the brain parenchyma. After resolution and evacuation of the hematoma, the cavity of the lesion communicates with the ventricular system, leading to the formation of a porencephalic cyst [Figure 26]. Porencephalic cysts, which are, often, a sequel of grade 4 haemorrhages are usually associated with higher neurodevelopmental defects\(^1\)

Congenital CNS Anomalies

Structural information is easily available in premature and mature infants on sonography. Initial evaluation of anomalies can be concluded with reasonable certainty. Hydrocephalus contributes to a large number of cases that can be diagnosed and followed up by neurosonography. Extent of hydrocephalus, level of obstruction, and thickness of the cerebral mantle can be obtained for subsequent follow-up. Biventricular, bifrontal ratio is measured at the level of foramen of Monro for quantitative follow-up of hydrocephalus [Figures 27 and 28]. Grossly dilated ventricular cavities are noted in aqueductal stenosis [Figure 29], agenesis of corpus callosum with midline cyst [Figure 30A and B] and hydrancephaly [Figure 31]. Evaluation through the mastoid fontanelle may be helpful for demonstrating the aqueduct obstruction [Figure 32]. Distinguishing between obstructive versus non-obstructive hydrocephalus is vital, with the former needing neurosurgical consultation for prompt management. Although periventricular transparenchymal passage fluid is not reliably detected in acute obstructions, serial follow-up of ventricular size by sonography helps in distinguishing obstructive, progressive hydrocephalus versus balanced, stable hydrocephalus.

Other anomalies that can be diagnosed using neurosonography include Dandy–Walker syndrome [Figure 33], agenesis of the corpus callosum [Figures 34 and 35], Arnold–Chiari malformation, and vascular malformations. Unusual course of the anterior cerebral artery in cases of agenesis of the corpus callosum, described as sunburst appearance, can be conclusively shown by gray-scale and Doppler examination. Also,

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**Figure 26:** Coronal USG and the high-resolution parasagittal view demonstrating a large porencephalic cyst communicating with the left lateral ventricular cavity. The patient earlier had a large grade 4 hemorrhage

**Figure 27 (A, B):** Coronal illustration at the level of interventricular foramen, showing the measurements for bifrontal/ventricular ratio (A), USG in a patient with hydrocephalus showing measurement of ventricular/bifrontal ratio (B)

**Figure 28:** Coronal images at two levels showing hydrocephalus secondary to germinal matrix hemorrhage on the left side. Note the dilated third ventricle due to obstruction at the level of the aqueduct
Figure 29: Severe hydrocephalus due to congenital aqueductal stenosis. Sagittal image (C) showing a dilated third ventricle and completely collapsed fourth ventricle. Obstruction is at the level of aqueduct (arrow).

Figure 30 (A, B): (A, B) Coronal and sagittal USG demonstrating severe hydrocephalus in a patient with agenesis of the corpus callosum and midline interhemispheric cyst (open arrows).

Figure 31: Severe hydrocephalus mimicking hydrancephaly. There is a minimal residual cerebral mantle. Note the small posterior fossa associated findings like colpocephaly and the additional anomalies of the posterior fossa can be shown [Figure 35]. Hydrancephaly and severe hydrocephalus, although easily demonstrated, cannot be distinguished with certainty on sonography alone. Dysplastic atrophic brain is occasionally seen on sonography. These lesions are seen without the context of ischemia in the early perinatal period. Typically, the brain is small in size, hyperechoic without differentiation of the gray and white matter, and shows multiple cysts of varying size. Lesions are bilateral and often asymmetric [Figure 36A and B]. Direct examination over the cranial swelling can be performed by sonography. Large cephaloceles [Figure 37], dermoid cyst, and cephalhematoma can be diagnosed.

Examination through the mastoid fontanelle can be very useful in differentiating holoprosencephaly from gross hydrocephalus. Additional detailed information regarding the posterior fossa can also be obtained through this route.

Mineralizing Vasculopathy

An appearance of linear bright branching streaks or patches, either unilaterally or bilaterally along the basal ganglia region is suggestive of mineralizing vasculopathy [Figures 38 and 39]. These hyperechoic streaks resembling a branched candlestick are due to the calcification of the walls of the thalamostriatal and lenticulostrital medium-sized perforating arteries, associated with wall hypercellularity, intramural and perivascular deposition of amorphous basophilic material. Mineralizing vasculopathy is a
Vascular Lesions

The dilated vessels of the arteriovenous malformations tend to appear as cystic lesions on neurosonography [Figure 40]. Doppler images might also demonstrate dilated feeding and draining vessels. The typical spectral pattern for
intracranial arteriovenous malformations is irregular and biphasic. Small vascular malformations and peripherally located cortical lesions cannot be detected by sonography. However, large asymmetric arteriovenous malformation can be detected by Doppler flow and on demonstration of large feeding veins. Aneurysmal malformation of the vein of Galen is one of the conditions occasionally diagnosed by screening sonography. Typical location of the lesion in posterior-superior third ventricular region between the cerebral hemispheres, with low-level internal echoes and swirling effect on Doppler examination makes it possible for a conclusive sonographic diagnosis. Information regarding the feeding arteries and the subtle changes in the parenchyma certainly calls for alternative imaging methods like MRI. Another vascular lesion that can be demonstrated is the thrombosis of the superior sagittal sinus. Although not recommended for routine screening, previously demonstrated thrombus can be followed up by sonographic methods.

Miscellaneous

Intracranial sonography can demonstrate many unsuspected cranial abnormalities. Demonstration of wide subarachnoid spaces with low-level internal echoes is observed in intracranial subarachnoid hemorrhages and meningitis. The demonstration is facilitated by the use of a high-resolution linear probes. Normal subarachnoid spaces, measured as sinocortical width, are usually less than 3.5 mm wide. Other lesions which can be shown are large mass lesions in the hemispheres,
notably teratoma, lipoma of corpus callosum, and unusual infantile neuroglial neoplasia arising from cerebral hemispheres. Occasionally, large arachnoid cysts and subdural hematomas are demonstrated by screening examination. Choroid plexus cysts are yet another commonly observed lesion in perinatal sonography. They are incidentally detected in neonates (8.8%), resolving spontaneously without complications when detected in isolation.[21] They may have syndromic associations or may occasionally present as isolated lesions [Figure 42].

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References