**Background:** Premature newborn infants are exposed to a wide spectrum of brain lesions which are clinically silent supporting a possible role of cerebral ultrasound screening. Aim of the study is to describe the pattern of cranial ultrasound abnormalities in preterm infants defining the short term and long term neurologic outcomes. **Material and Methods:** A hospital-based bedside cranial ultrasound was carried out at day 1, 3, 7 and follow-up scan at 3-6 months in the Department of Radio-diagnosis. **Results:** One hundred infants were included. The different abnormalities detected in cranial ultrasound of premature newborn infants include hydrocephalus in 12%, intracranial hemorrhage in 6%, brain edema in 6%, periventricular leukomalacia in 2%, choroid plexus cyst in 1%, intraventricular sepsa in 1% and colocephaly in 1%. **Conclusion:** Gestational age, newborn birth weight and neurologic symptoms were the most important risk factors for detecting brain lesions. The main purpose of cranial ultrasound was the demonstration or exclusion of an intracranial hemorrhage in a preterm neonate.

**Keywords:** Cranial USG, neonatal cranial US, neuroimaging in preterm infant, trans-fontenellar cranial ultrasound, ultrasound imaging of newborn brain

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**INTRODUCTION**

Since the late 1970s, cranial Ultrasonography (USG) is the most widely used neuroimaging procedure in premature infants as it provides information about perinatal brain injury for the prediction of long-term outcomes.[1] Cranial sonography is less expensive, free from radiation, does not require sedation, and its portability allows for bedside evaluation of gravely ill patients.

US helps in assessing the neurological status of the child as clinical examination and symptoms are often nonspecific. It is often not possible to subject every patient to magnetic resonance imaging (MRI) because of the nonavailability of this modality and the cost of investigation. Also, the critical clinical condition of the newborn does not allow shifting the patient to an MRI center. Because the ultrasound facility is available in the majority of care-providing centers, it can be used by the radiologists for screening the newborn to evaluate the immediate risk and possible long-term neurological outcomes. Ultrasound is an ideal and fast bedside examination. It is accomplished by using one or more transducers placed on the large anterior or the small posterior fontanel, which is used as an acoustic window.

**Aims and objectives**

The objective of this study was to screen high-risk preterm infants admitted to the neonatal intensive care unit using transfontanellar sonography on days 1, 3, and 7 of life to identify the various cranial pathologies. Follow-up cranial USG was performed at 2 weeks, and at the age of 1, 2, and 3 months to study the sequel of cranial abnormality.

**MATERIALS AND METHODS**

Premature infants born before 32 weeks of gestational age (GA) with birth weight of 1500–1800 g and admitted into neonatal intensive care unit (NICU) of the Department of Paediatrics, CCM Medical College and Hospital, Durg, Chhattisgarh, India, were examined by ultrasound through anterior fontanel during the...
period from May 2015 to March 2017. The anterior and posterior fontanels were commonly used. The mastoid and temporal fontanels were used to obtain axial images for evaluating posterior fossa. The transverse sinus can be examined through posterior fontanel or foramen magnum. SonoSite Sonomax ultrasound equipment with linear 5–7 MHz probe was used. All antiseptic precautions were taken and sterilized ultrasound gel was used. Transvaginal (TV) probe of 7–12 MHz (kept exclusively for cranial ultrasound) was used to evaluate quickly the areas of the brain near convexity. The examination time on an average was 5–7 min and another 1 min for TV probe to avoid heating effect (if at all it was there). Precaution was taken to avoid undue pressure of the probe on the fontanel. Follow-up ultrasound scan after 7–14 days, 1 month, 3 months, and 6 months was performed in the Department of Radio Diagnosis. Infants with abnormality detected by cranial ultrasound were referred to the neurosurgeon and/or for MRI.

Infants born before 32 weeks GA with birth weight of 1500 g or less were selected for this study.

RESULTS

Average NICU stay for premature infant was 10–14 days. The longest stay was 60 days in one case. Neonatal mortality was 16%. Neurological symptoms were found in 20% premature infants. MRI was performed in 10% and abnormalities were detected in all of them. Congenital anomaly was present in 1%. The follow-up was 3–6 months in 20%. However, 80% were lost in follow-up.

The results of this study are depicted in Tables 1 and 2 and in Figures 1-17.

DISCUSSION

Neuroimaging or transfontanellar neonatal brain scanning is an important test in the diagnosis of hemorrhage and other acquired and congenital brain pathology of the newborn. It is less expensive, free from ionizing radiation, does not require sedation, and its portability makes it the preferred diagnostic modality to evaluate the gravely sick premature newborn.

A brief discussion of normal ultrasound anatomy and variations is essential for proper neurosonographic interpretation to avoid pitfalls in diagnosis.

Two-dimensional or grayscale imaging

The cranial ultrasound is performed with a linear-array transducer. Six to eight coronal plane images are obtained beginning in the frontal lobe and progressing posteriorly to the occipital lobe past the trigone of the lateral ventricle [Figures 1 and 2].

Next, the transducer is turned 90° on the anterior fontanel to obtain five more images in the sagittal and parasagittal planes. The first image is a midline sagittal view, which includes corpus callosum and cerebellar vermis. Additional parasagittal views are obtained laterally on either sides including lateral horns and choroid plexus [Figure 3]. The final view can be made on either side through the lateral thalamus.

The echogenic structures in the midline that form the choroid plexus and cerebellar vermis have an appearance like a woman in Victorian-era dress, the “lady in the dress” sign. The corpus callosum is seen superior to the cavum septum pellucidum (CSP) and inferior to the cavum is the third ventricle, thalamus, and brain stem [Figure 4]. This is true sagittal midline view.

Additional fontanels are used for better visualization of structures. The mastoid fontanels enable inspection of the cerebellar hemispheres and in detection of posterior fossa hemorrhage and congenital anomalies. Mastoid fontanels are also used to evaluate the size and appearance of the fourth ventricle and cistern magna. The transverse sinus can be examined through posterior fontanels or foramen magnum.

Pitfalls and normal variants

An understanding of normal variation is essential for neurosonographic interpretation. CSP is present in up to 50%–61% of normal neonates [Figure 1]. 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 periventricular leukomalacia (PVL). Massa intermedia can vary in size in normal and pathological conditions. When posterior fontanel approaches are used, prominent calcar avis and lobulated glomus of the choroid should be observed as normal variations. Other variations in anatomy include immature sulcation in premature newborn before 24 weeks GA containing only Sylvian fissures and persistent fetal fluid-filled spaces such as CSP, cavum vergae, and cavum veli interpositi. It should not be mistaken for other malformations in posterior fossa such as arachnoid cysts or Dandy–Walker malformation of cerebellum; a mega cisterna magna measures greater than 8 mm in either sagittal or axial plane present in 1% of infants is a benign finding of no clinical significance. Asymmetric ventricular size in 20%–40% of infants; choroid plexus variants; periventricular halos and cystic lesions [Figure 5].
The fetal or preterm infant’s brain is vulnerable to both hemorrhagic and ischemic injury during the late second trimester and early third trimester. By 32 weeks of postconceptual age, the germinal matrix hemorrhage (GMH) is found only along the ventricular surface of the caudate nucleus and at its border with the thalamus. It normally involutes by 34–36 weeks of postconceptual age. The incidence of GMH and/or intraventricular hemorrhage (IVH) is infrequent after that time. Hemorrhagic and ischemic injuries often occur, even though different pathophysiological processes lead to the lesions.

The vascular structure of the cerebral white matter (WM) in mid- to late gestation includes long penetrating arteries that originate from anterior, middle, or posterior cerebral artery. The end zones of these arteries are especially prone to hypoperfusion and ischemia, and thus, there is an increased likelihood of ischemic necrotic damage along the course or end zones of the arteries or in the periventricular area. Nonhemorrhagic cerebral infarction, ventriculomegaly, or cystic lesions such as PVL or porencephaly may evolve WM injuries.

Ventriculomegaly that occurs in the absence of IVH is most often secondary to the loss of cerebral WM that has been damaged or failed to develop normally.

### Table 1: Abnormal USG findings in 30 cases

| USG Finding                  | No. of cases | No. of males | No. of females |
|-----------------------------|--------------|--------------|----------------|
| Congenital hydrocephalus    | 10           | 5            | 5              |
| Posthemorrhagic hydrocephalus| 2            | 1            | 1              |
| IVH                         | 6            | 3            | 3              |
| PVL                         | 2            | 1            | 1              |
| Cystic lesion               | 2            | 1            | 1              |
| Congenital anomaly          | 1            |              |                |
| Ventricular septa           | 1            |              |                |
| Brain edema                 | 6            | 3            | 3              |

### Table 2: Distribution of cranial USG findings at different days of examination

| Day of USG scanning | USG findings                  | No. of cases |
|---------------------|-------------------------------|--------------|
| 1                   | Normal                        | 70           |
| 3–5                 | I/V Hemorrhage                | 6            |
| 7–10                | PVL                           | 2            |
| 30                  | Cystic lesion                 | 1            |
| 1–3                 | Brain edema                   | 6            |
| 2                   | Ventricular septa             | 1            |
| 1                   | Congenital hydrocephalus      | 10           |
| 1                   | Post-hemorrhagic hydrocephalus| 2            |
| 1                   | Congenital anomaly            | 1            |
| 3                   | Venous infarction             | 1            |

### Pathophysiology of common abnormalities

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The vascular structure of the cerebral white matter (WM) in mid- to late gestation includes long penetrating arteries that originate from anterior, middle, or posterior cerebral artery. The end zones of these arteries are especially prone to hypoperfusion and ischemia, and thus, there is an increased likelihood of ischemic necrotic damage along the course or end zones of the arteries or in the periventricular area. Nonhemorrhagic cerebral infarction, ventriculomegaly, or cystic lesions such as PVL or porencephaly may evolve WM injuries. Ventriculomegaly that occurs in the absence of IVH is most often secondary to the loss of cerebral WM that has been damaged or failed to develop normally. 
Twin pregnancy carries a higher risk of neonatal death, cerebral palsy, and intrauterine death.\(^{[15]}\)

The outcome abnormalities found in GMH and/or IVH with WM damage, however, can range from subtle cognitive abnormalities to borderline or severe mental retardation.\(^{[21,22]}\)

**Ultrasound findings**

GMH is also known as periventricular hemorrhage or preterm caudothalamic hemorrhage. The highly vascular germinal matrix is located in the subependymal region between the caudate nucleus and thalamus. The germinal matrix is only transiently present as a region of thin-walled vessels, migrating neuronal components, and vessel precursors.\(^{[23]}\) The germinal matrix is mature by 34 weeks GA so that the hemorrhage becomes very unlikely after this age. Most GMHs occur in the first week of life. These hemorrhages start in the caudothalamic groove (Figure 6) and may extend into the lateral ventricle and periventricular brain parenchyma. Most infants are asymptomatic or show subtle signs that are easily overlooked.\(^{[23]}\)

Among infants who developed GMH and/or IVH, at least one-third had echodensities as early as 1 h after birth, indicating an antenatal or immediate postnatal onset.\(^{[24]}\) By 32 weeks, the risk of GMH is greatly reduced and disappears completely by 40 weeks.\(^{[25]}\)

Papile *et al.*\(^{[26]}\) graded hemorrhages as follows: grade 1, IVH is confined to the GM; grade 2, IVH involves bleeding into the ventricle (Figure 7); grade 3, IVH has distended and enlarged the ventricle (Figure 8); and grade 4, bleeding within the brain parenchyma.

Grade 1 and 2 bleeds generally have a good prognosis. Grade 3 and 4 bleeds have variable long-term deficit, however, outcome in grade 3 bleed is usually good if no parenchymal injury has occurred.
PVL is also known as hypoxic-ischemic encephalopathy (HIE) of the preterm. It is a WM disease that affects the periventricular zones. The other causes such as infection and vasculitis play an additional role. Normally, the echogenicity of the periventricular WM should be less than that of the choroid plexus. While the GMHs are a result of acute hemodynamic changes, PVL represents a relatively insidious cerebral parenchymal insult. Chronic hypoxemia or hypoperfusion leads to PVL.

PVL occurs most commonly in premature infants born at less than 33 weeks GA (38% PVL) and with less than 1500g of birth weight (45%). Sonographic grading of PVL was described by de Vries et al. PVL grade 1–3 is a disease of preterm neonates, whereas PVL grade 4 is seen in full-term neonates. PVL can be differentiated from hemorrhages because PVL lacks mass effect. About 2% preterm neonates born before 32 weeks develop cystic PVL, which has been identified via cranial ultrasound on the first day of life, indicating that the adverse event was at least 2 weeks prenatal rather than perinatal or postnatal. US is highly reliable in the detection of cystic WM injury (PVL grade 2 or more) but has significant limitations in the demonstration of non-cystic PVL grade 1, which is considerably more common than cystic WM injury.

Porencephalic cysts: Large foci of intraventricular/intraparenchymal bleed could lead to a cavitating destructive lesion in brain parenchyma, which
communicates with the ventricular system leading to the formation of a porencephalic cyst [Figure 10]. Porencephalic cysts are often a sequel of grade 4 hemorrhages. The diagnosis of a severe ventriculomegaly does not pose difficulties [Figure 11], but the identification of mild ventricular dilatation depends on the establishment of normality threshold parameters.\textsuperscript{[28-30]}

2. Ventricular enlargement, Levene index, and ventricular index: In daily practice, the 95th percentile of the ventricular measurements can be used as upper normality threshold.

For up to 40 weeks GA, the Levene-index should be used and after 40 weeks, the ventricular index.\textsuperscript{[30]}

The Levene index is the absolute distance between the falx and the lateral wall of the anterior horn in the coronal plane at the level of the third ventricle.
The ventricular index is the ratio of the distance between the lateral sides of the ventricles and the biparietal diameter [Figure 12]. It should be remembered that when the ventricular system widens, the frontal horns tend to enlarge in the craniocaudal direction more than the left to right dimension. These measurements can be taken in an axial plane through the temporoparietal bone.\[30\]

Prenatally diagnosed isolated ventriculomegaly, in the absence of associated anomalies, is not a strong predictor of cognitive or motor abnormalities.\[31\]

3. Choroid plexus cysts are often incidental findings without clinical consequences [Figure 13]. In prenatal US, these cysts can be predictive of trisomy 18. However, cysts arising close to foramen of Monro need follow-up as they may obstruct the flow of cerebrospinal fluid and result in raised intracranial pressure.\[23\]

4. Ventricular septa [Figure 14]: Schellinger et al.\[32\] in a series of 24 patients described the presence of septa at birth (congenital septa) in 7 neonates. IVH and infection are the major causes of true intraventricular septa. Among 17 cases of acquired septa, there were true intraventricular septa and the pseudosepta that originated outside the ventricles but later became part of the ventricular system due to cavitating PVL. Sonography is the diagnostic method of choice. Septa are associated with a high incidence (62%) of shunt failure.

Other abnormalities in cranial USG

Intracranial sonography can demonstrate many unsuspected cranial abnormalities including wide subarachnoid spaces, Dandy–Walker syndrome, agenesis of corpus callosum, Arnold–Chiari malformation, vascular malformation, dysplastic atrophic brain, large cephaloceles, and dermoid cyst.

Wide subarachnoid spaces with low-level internal echoes are observed in intracranial subarachnoid hemorrhages and meningitis. Normal subarachnoid spaces measured as sinocortical width are usually less than 3.5 mm wide.\[33\]

MRI is superior in demonstrating congenital anomalies of the brain and HIE. Figure 15 reveals colpocephaly in a premature newborn suspected of agenesis of corpus callosum.

5. Germinolytic cysts are located at Caudo-thalamic notch (CTN) and are tear-shaped and these children have no neurological sequel. Etiology is not known.

6. Mineralizing vasculopathy can be seen in the thalamostriatal and lenticulostriatal arteries and is caused by calcification of the arterial wall. A wide range of perinatal, acquired, and nonspecific clinical conditions may result in this sonographic finding.

7. Benign macrocrania, also known as extraventricular obstructive hydrocephalus, is seen in children between 6 months and 2 years. The head circumference is above the 97th percentile. After the age of 2 years, the head size normalizes. Some state that such children may have slight developmental delay.

8. Evaluation of diffuse brain edema is technically challenging on neurosonography. The usual observation in the cases of ischemia is a diffuse increase in the echogenicity of the ganglionic areas with associated obliteration of cisterns and small capacity of the ventricles; however, evaluation of ventricular size is an unreliable parameter in assessing the mass effect. Computed tomography and/or MRI still remain as superior techniques in assessing diffuse intracranial ischemia.\[34\]

9. HIE: In preterm infant, GMH–IVH is the most common manifestation of hypoxic brain injury. Significant ischemic component may result in infarction. PVL and hemorrhage are the primary manifestations of HIE in premature infant.

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**Table 3: Grading of PVL**

| Grades | PVL (increased periventricular echogenicity) |
|--------|---------------------------------------------|
| 1      | Persisting more than 7 days                 |
| 2      | Developing into small periventricular cysts (Swiss-cheese appearance) |
| 3      | Developing into extensive periventricular cysts, occipital and frontoparietal |
| 4      | In deep WM developing into extensive subcortical cysts |

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Figure 17: MCA Doppler through temporal fontanel with normal waveform in a preterm newborn of gestational age 31 weeks, RI 0.56
The sensitivity of cranial ultrasound examinations as predictors of later neurodevelopment abnormalities has been reported as 16% at 1 and 2 weeks after birth, increasing to 53% at 6 weeks and 58% if performed when a child is at term-corrected age.\(^\text{[35]}\) The specificity of cranial ultrasound examinations has been 99%–100% in all age groups.\(^\text{[36]}\)

**Doppler imaging**

The Doppler evaluation of vasculature is done for patency and resistance to flow [Figures 16 and 17]. Arteries are usually evaluated in the coronal plane, whereas venous structures are best imaged in sagittal plane. First the circle of Willis is investigated with color Doppler imaging through the anterior or temporal fontanels.\(^\text{[36]}\) Color Doppler images are obtained in the sagittal plane to evaluate the sagittal sinus and vein of Galen. Power Doppler imaging can be used to detect signs of ischemia that may present as areas of hyper- or hypovascularity.\(^\text{[6]}\)

Additional fontanels are used for better visualization of structures. The mastoid fontanels enable inspection of the cerebellar hemispheres and in detection of posterior fossa hemorrhage and congenital anomalies. Mastoid fontanels are also used to evaluate the size and appearance of the fourth ventricle and cistern magna. The transverse sinus can be examined through posterior fontanels or foramen magnum. A value of 0.6–0.9 is used to estimate a normal Resistance Index (RI) in premature and full-term infants.\(^\text{[37]}\) Lower values may indicate acute hypoxia or ischemia, whereas higher values may suggest cerebral swelling.\(^\text{[9]}\)

**Limitations of cranial USG:**

1. Limited overview in posterior fossa and convexity of the brain.
2. Absence of US signs in ischemia in full terms in first 24 h.
3. Difficulty in detecting migration disorders and cortical dysplasia.
4. Mostly performed with freehand 2D imaging, hence operator-dependent.

**Recommendations:**

1. Routine screening cranial ultrasound for all infants born at 32 weeks GA or earlier
2. Follow-up examination at about the second week allows diagnosis of hemorrhagic lesions and at the sixth week of life to detect cystic lesions or ventriculomegaly to predict long-term outcomes.

The potential benefits and harmful consequences of misinterpretation of cranial USG examination should be communicated to parents. Infants who have hemorrhagic lesions or any WM or cystic lesions require follow-up after discharge from NICU to facilitate the timely initiation of interventions.

**Conclusion**

The variety of information obtained from screening of preterm infants by transfontanellar sonography reveals the importance of this safe, cost–effective, and bedside modality to assess neurological complications in seriously ill newborns and to disclose to the parents the long-term neurological outcome apart from early referral to neurosurgeon for further management. The use of this valuable diagnostic tool by radiologists will enable them to improve skill by acquainting themselves with normal anatomical structures, anatomic variants, imaging pitfalls, and pathological processes with the help of grayscale imaging.

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**Conflicts of interest**

There are no conflicts of interest.

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