SPECTRUM OF NEONATAL AND INFANTILE INTRACRANIAL ABNORMALITIES USING TRANSCRANIAL NEUROSONOGRAPHY (TCNSG)

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ABSTRACT

BACKGROUND

The aim of the study is to evaluate the spectrum of intracranial abnormalities among neonates and infants undergoing transcranial neurosonography.

MATERIALS AND METHODS

Serial sonographic scans from neonates and infants admitted to the paediatric intensive care unit between February 2015 and February 2016 by radiologists.
Design/Setting- Observational study at teaching hospital.

RESULTS

In our study, out of 124 newborns and infants who were clinically suspected to have some intracranial pathology, there were 181 abnormal sonographic findings in 93/124 (75%) of patients. Hydrocephalus of various aetiologies was the most common abnormality in 45/181 (24.86%) of patients. Among them, 23 had congenital hydrocephalus and rest of the patients had either infection or haemorrhage as causative factors followed by 8/181 (4.42%) cerebral oedema, ventriculitis in 9/181 (4.97%) and 16/181 (8.84%) cerebral infarcts. Others included 25/181 (13.81%) intraventricular haemorrhage, 33/181 (18.23%) periventricular leukomalacia, 8/181 (4.42%) porencephalic cysts, 8/181 (4.42%) encephalomalacia and 2/181 (1.10%) hypoxic ischaemia. Developmental anomalies such as (1/181, 0.55%) Dandy-Walker malformation and (1/181, 0.55%) hydranencephaly were also observed.

CONCLUSION

Neurosonography offers the most economical and highly accurate techniques for imaging intracranial pathologies in the neonates and infants. It should, therefore, be employed as a routine screening modality for intracranial pathologies in all the newborn babies and infants.

KEYWORDS

Neonate, Infant, Intracranial Lesions, Sonography.

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BACKGROUND

Transcranial Neurosonography (TCNSG) detects a spectrum of neonatal and infantile intracranial abnormalities. NSG can demonstrate various sequelae of ischaemic and infective insults to the brain. These findings can influence parent counselling, treatment planning and rehabilitation of the patients. Additionally, NSG equipped with high-frequency transducers and colour Doppler techniques help in the detection of early lesions of hypoxic-ischaemic encephalopathy. High-frequency transducer becomes essential in the detection of small subdural collections, cortical and subcortical lesions and small cystic lesions, which were previously undetected by conventional scanners. Colour duplex Doppler helps in detecting subtle ischaemic changes and infarcts, altered blood flow patterns and vascular abnormalities. We report intracranial pathologies including congenital anomalies in neonates and infants with clinically suspected intracranial pathologies underwent Transcranial Neurosonography (TCNSG).

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MATERIALS AND METHODS

The study was conducted in the Department of Radiodiagnosis, Viswabharathi Medical College, Kurnool, between February 2015 and February 2016. One hundred and twenty four infants with clinically suspected intracranial pathologies were evaluated by transcranial neurosonography using MyLab 50 Esaote series ultrasound and colour Doppler system using convex 3-4 MHz, 8.2-11 MHz linear array and 5-7 MHz transvaginal probe. The babies were screened in the coronal, sagittal and parasagittal plane using anterior fontanelle as an acoustic window. Additional views were considered where necessary. A minimum of four

images was obtained in two perpendicular planes in patients with abnormal findings. The intracranial lesions detected on ultrasound were described according to Ment et al, 2002.[1] The Ventriculo-Hemispheric Ratio (V/H) was calculated as a percentage according to literature.[2] The third ventricular measurement was taken in La coronal section, which passes through the third ventricle. A value of more than 5 mm was considered abnormal.

Range of V/H	Grade of Hydrocephalus
30-39%	Mild
40-46%	Moderate
47% and above	Severe

STATISTICAL ANALYSIS

Data was reported as averages and standard deviation for continuous variables and as actual numbers and percentages for category variables. Statistical analysis was done using Microsoft spreadsheet 2003.

RESULTS

In our study, out of 124 newborns and infants who were clinically suspected to have some intracranial pathology, there were 181 abnormal sonographic findings in 93/124 (75%) of patients. Hydrocephalus of various aetiologies was the most common abnormality in 45/181 (24.86%) of patients. Among them, 23 had congenital hydrocephalus and rest of the patients had either infection or haemorrhage as causative factors followed by 8/181 (4.42%) cerebral oedema, ventriculitis in 9/181 (4.97%) and 16/181 (8.84%) cerebral infarcts. Others included 25/181 (13.81%) intraventricular haemorrhage, 33/181 (18.23%) periventricular leukomalacia, 8/181 (4.42%) porencephalic cysts, 8/181 (4.42%) encephalomalacia and 2/181 (1.10%) hypoxic ischaemia. Developmental anomalies such as (1/181, 0.55%) Dandy-Walker malformation and (1/181, 0.55%) hydranencephaly were also observed.

75% of infants in our study group had abnormal sonographic patterns. Hydrocephalus of various aetiologies was the most common abnormality detected in these patients. CT scan done in 5 patients correlated well with the sonographic diagnosis. Aqueductal stenosis was found to be the most common cause of congenital non-communicating hydrocephalus. In our study group, the size of the IVH correlated well with the severity of post haemorrhagic hydrocephalus. In infants with HIE, we could detect the global and focal ischaemic lesions. Further evaluation of HIE patients with colour Doppler revealed subtle ischaemic changes by demonstrating decreased flow patterns in the ill-defined echogenic areas. Infarcts were identified as focal echogenic areas with absent arterial pulsations in the periphery. We could detect small 2 to 3 mm lesions of microcytic encephalomalacia and early evolving lesions of PVL. Neurosonographic features were different in preterm and term babies with perinatal asphyxia. While the lesions were predominantly central (IVH and PVL) in the preterm, they were mainly peripheral (subcortical leukomalacia, infarcts and cystic encephalomalacia) in term babies (Table 1).

Sl. No.	Sonographic Findings	n=93	%/181
1.	Ventriculitis	9	4.97%
2.	Cerebral oedema	8	4.42%
3.	Cerebral infarcts	16	8.84%
4.	Intraventricular haemorrhage	25	13.81%
5.	Porencephalic cyst	8	4.42%
6.	Subdural collection	25	13.81%
7.	Encephalomalacia	8	4.42%
8.	Hypoxic ischaemia	2	1.10%
9.	Hydrocephalus	45	24.86%
10.	Hydranencephaly	1	0.55%
11.	DWS	1	0.55%
12.	Periventricular leukomalacia	33	18.23%
	Total	**181**	**100.00%**

Table 1. Spectrum of Sonographic Findings of Neonatal and Infantile Intracranial Abnormalities

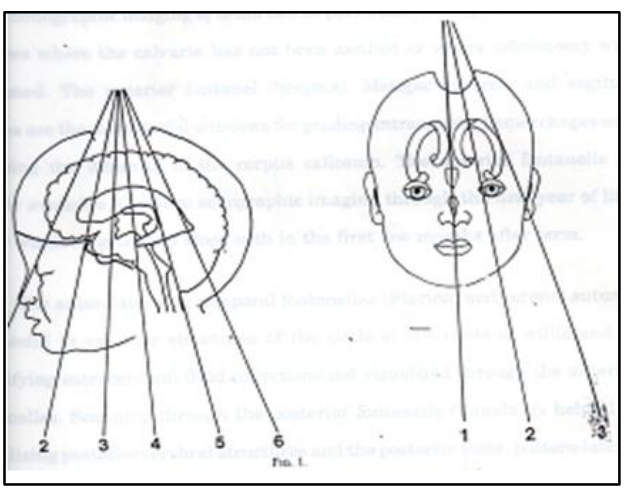

Figure 1. Diagram Representing the Configuration of the Six Coronal and Three Sagittal Planes

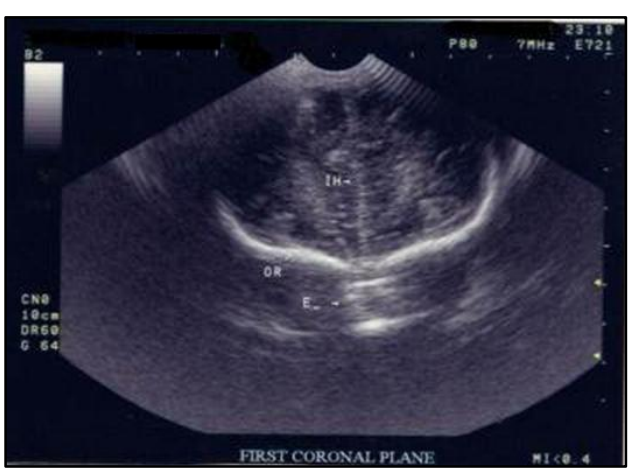

Figure 2. First Coronal Plane

IHF- Interhemispheric Fissure.
OR- Orbital Roof.
EC- Ethmoid Complex.

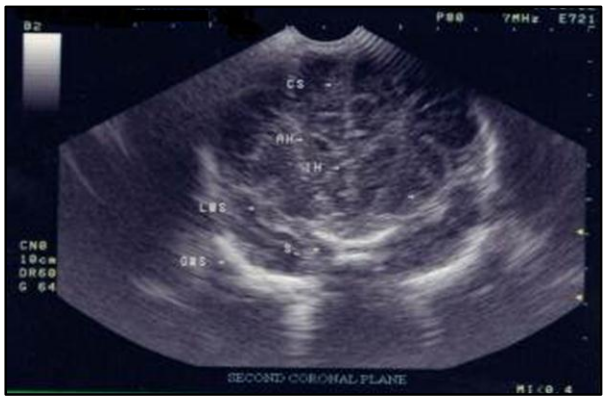

Figure 3. Second Coronal Plane

CS- Cingulate Sulcus.
AH- Anterior Horn.
IHF- Interhemispheric Fissure.
LWS- Lesser Wing of Sphenoid.
S- Sphenoid.
OWS- Oreater Wing of Sphenoid.
Sylvian Fissure with MCA.

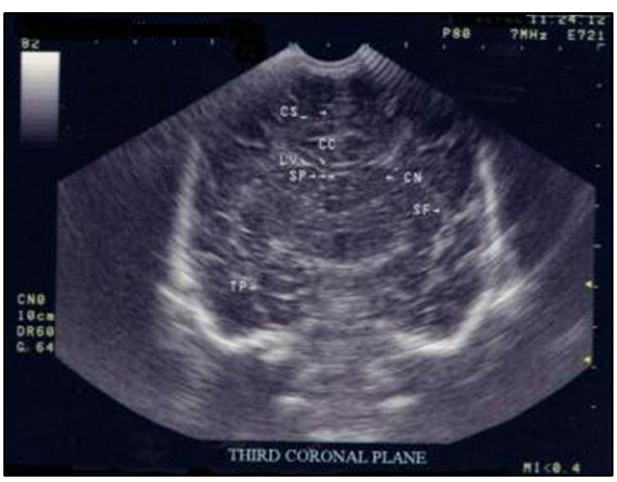

Figure 4. Third Coronal Plane

CS- Cingulate Sulcus.
CC- Corpus Callosum.
LV- Lateral Ventricle.
SP- Septum Pellucidum.
CN- Caudate Nucleus.
SF- Sylvian Fissure.
TP- Temporal Pole.

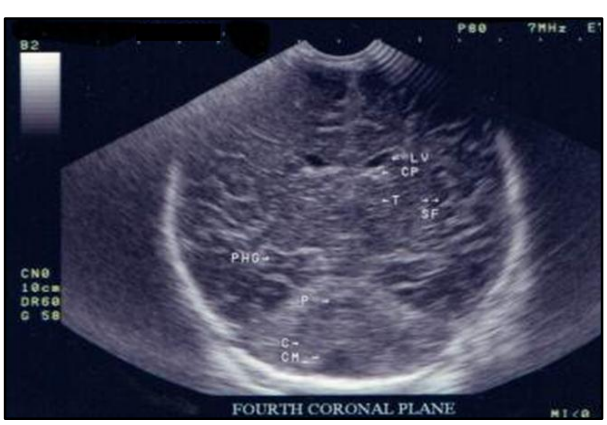

Figure 5. Fourth Coronal Plane

LV- Lateral Ventricle.
CP- Choroid Plexus.
TH- Thalamus.
SF- Sylavian Fissure.
PHG- Parahippocampal Gyrus.
P- Pons.
CB- Cerebellum.
CM- Cisterna Magna.

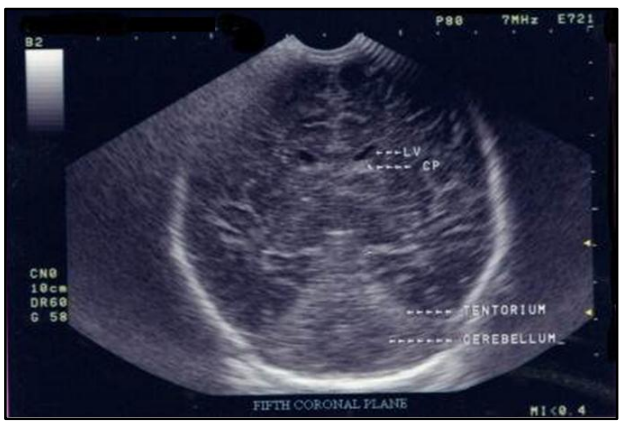

Figure 6. Fifth Coronal Plane

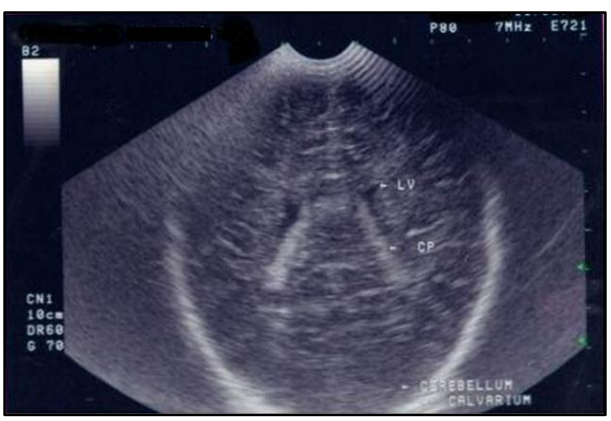

Figure 7. Sixth Coronal Plane

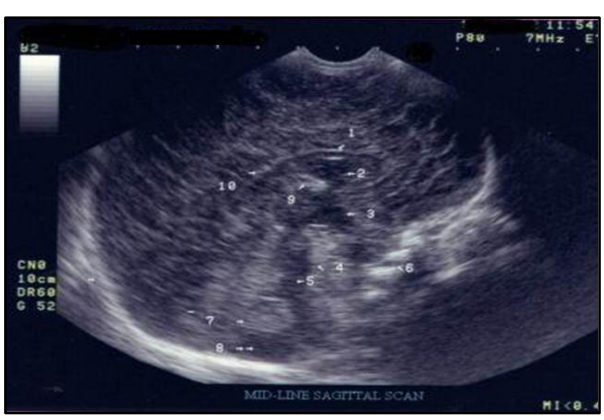

Figure 8. Midline Sagittal Scan

1. Corpus Callosum.
2. Lateral Ventricle.
3. Third Ventricle.
4. Pons.
5. Fourth Ventricle.
6. Clivus.
7. Cerebellar Vermis.
8. Cisterna Magna.
9. Choroid Plexus.
10. Callosal Sulcus.

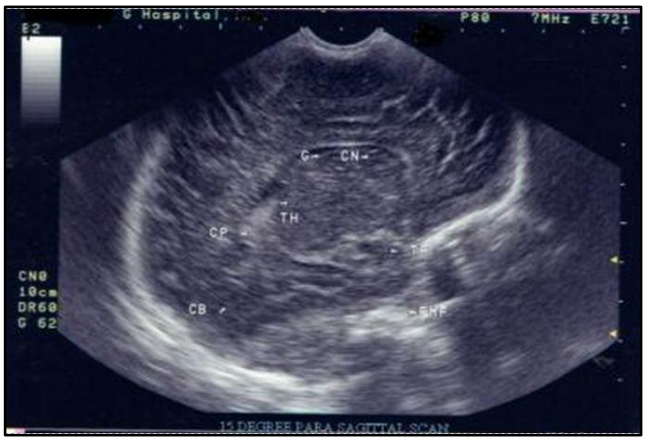

Figure 9. 15-Degree Parasagittal Scan

CG- Caudothalamic Groove.
CN- Caudate Nucleus.
TH- Thalamus.
TP- Temporal Pole.
CP- Choroid Plexus.
CB- Cerebellum.
FMF- Floor of Middle Fossa.

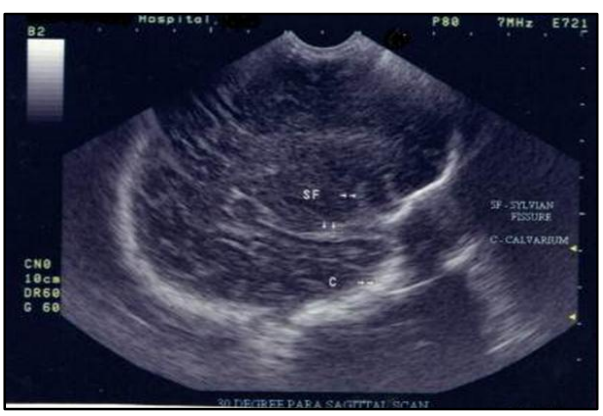

Figure 10. 30-Degree Parasagittal Scan

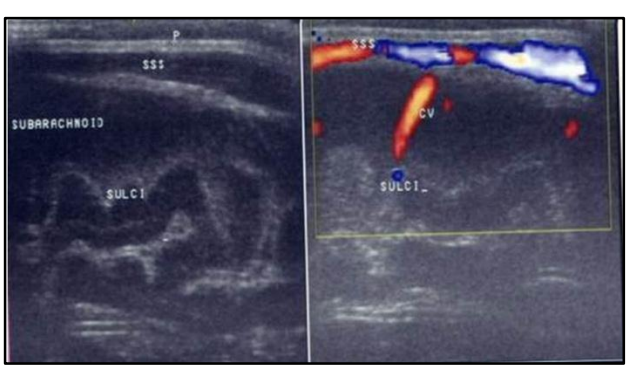

Figure 11. Colour Flow Doppler with 11 MHz Showing Superficial Cortical Vessels in Subarachnoid Space

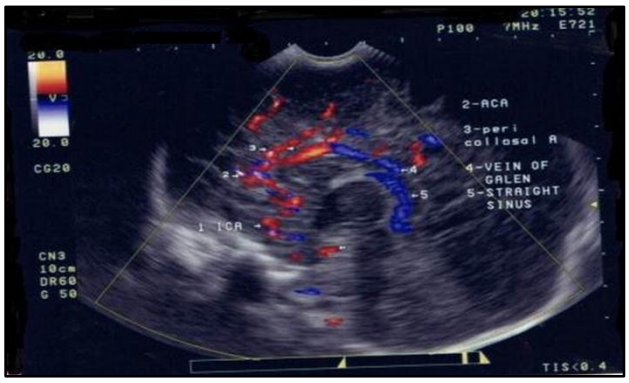

Figure 12. Sagittal Plane Colour Flow Doppler Depicting ACA and Pericollosal Artery

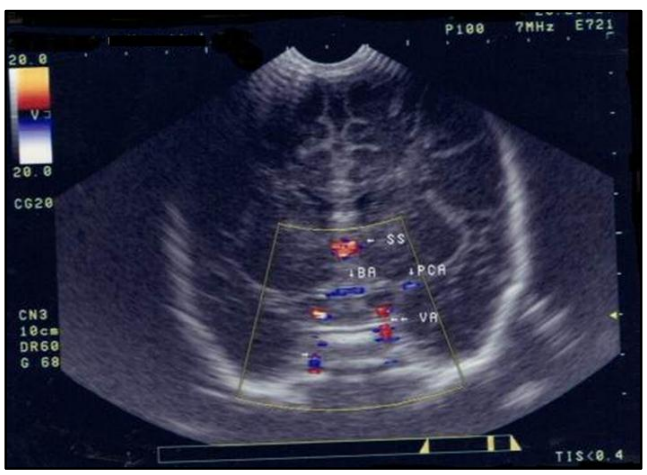

Figure 13. Coronal Plane- Colour Flow Doppler

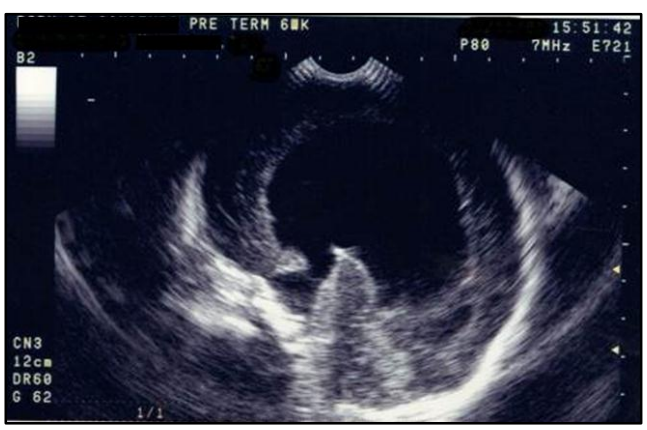

Figure 14. Congenital Non-Communicating Hydrocephalus with Aqueductal Stenosis

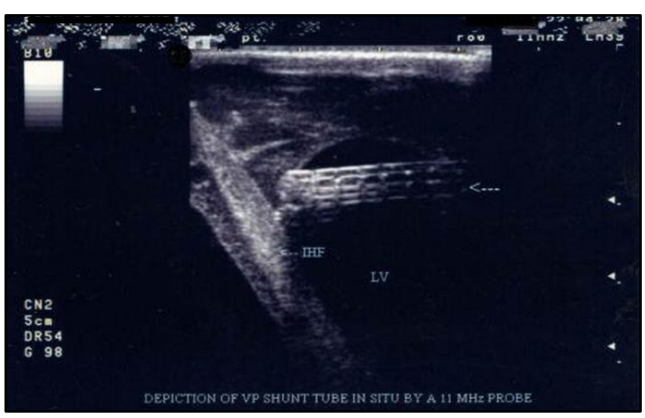

Figure 15. Depiction of VP Shunt Tube in Situ by an 11-MHz Probe

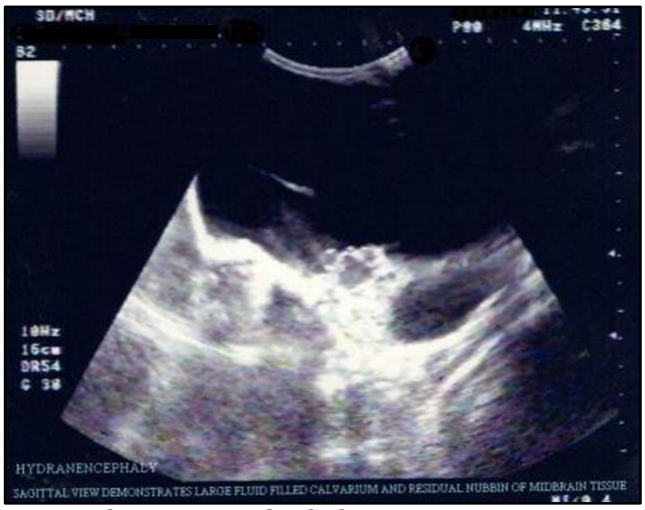

Figure 16. Sagittal View Demonstrates Hydranencephaly, Large Fluid Filled Calvarium and Residual Nubbin of Midbrain Tissue

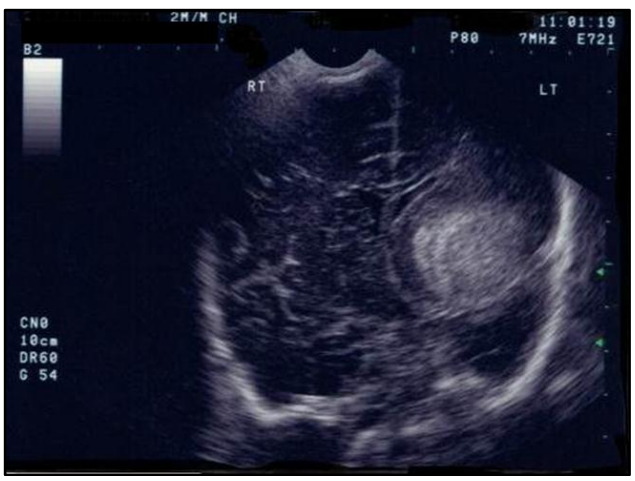

Figure 17. Large Echogenic Parenchymal Haematoma with Compression of the Ipsilateral Ventricle and Some Midline Shift

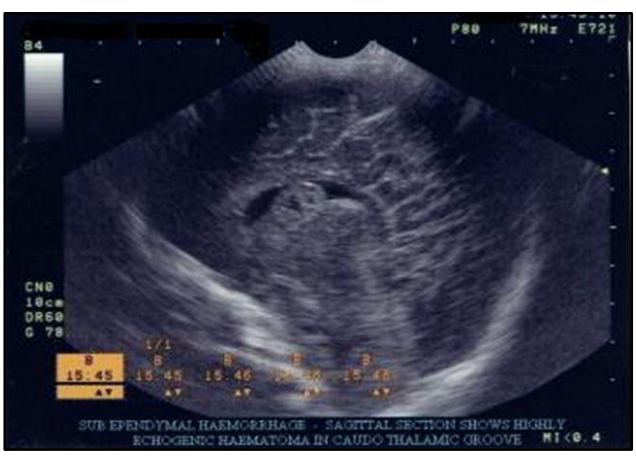

Figure 18. Subependymal Haemorrhage- Sagittal Section Shows Highly Echogenic Haematoma in Caudothalamic Groove

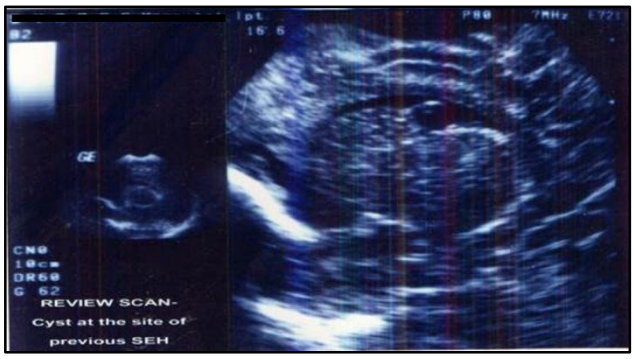

Figure 19. Review Scan- Cyst at the Site of Previous SEH

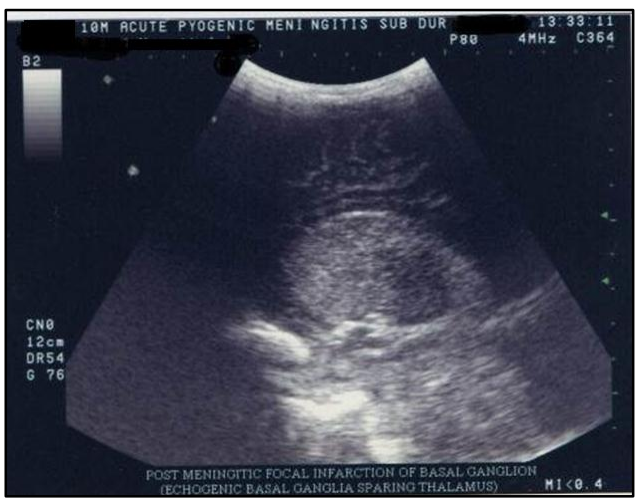

Figure 20. Postmeningitic Focal Infarction of Basal Ganglion (Echogenic Basal Ganglia Sparing Thalamus)

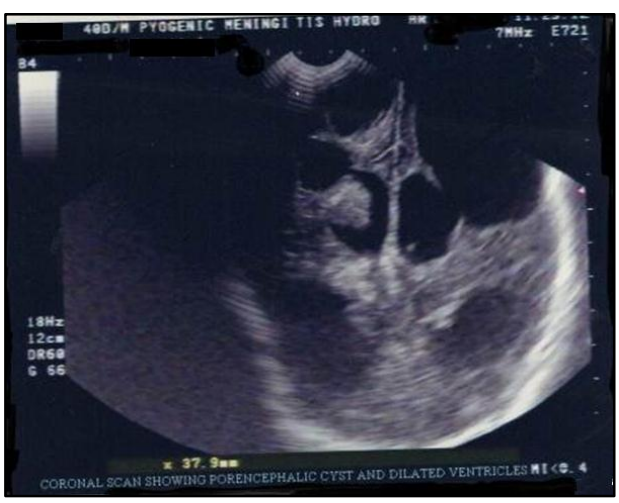

Figure 21. Coronal Scan Showing Porencephalic Cyst and Dilated Ventricles

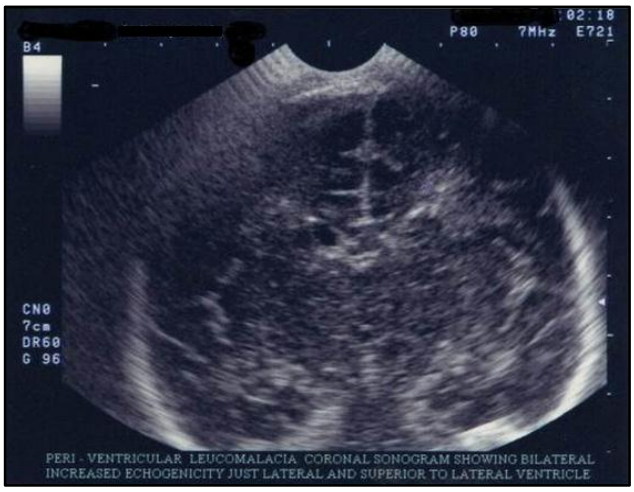

Figure 22. Periventicular Leukomalacia Coronal Sonogram Showing Bilateral Increased Echogenicity Just Lateral and Superior to Lateral Ventricle

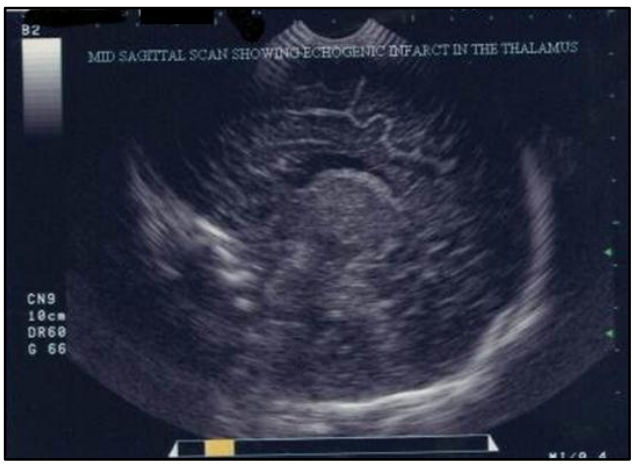

Figure 23. Mid Sagittal Scan Showing Echogenic Infarct in the Thalamus

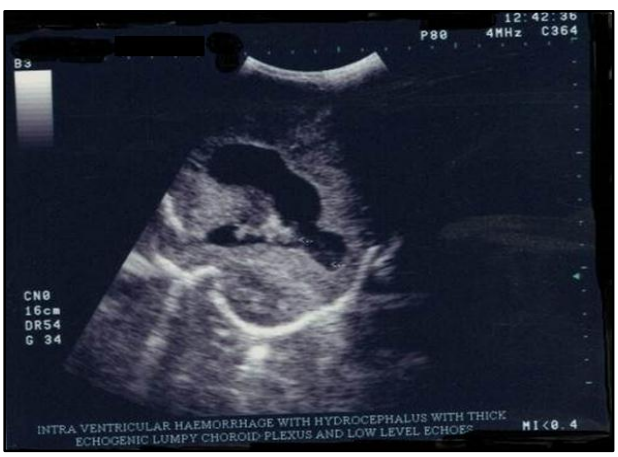

Figure 24. Intraventricular Haemorrage with Hydrocephalus With Thick Echogenic Lumpy Choroid Plexus and Low Level Echoes

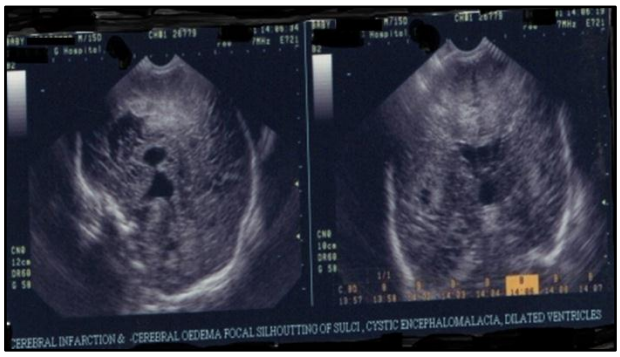

Figure 25. Cerebral Infarction and Cerebral Oedema Focal Silhouetting of Sulci, Cystic Encephalomalacia, Dilated Ventricles

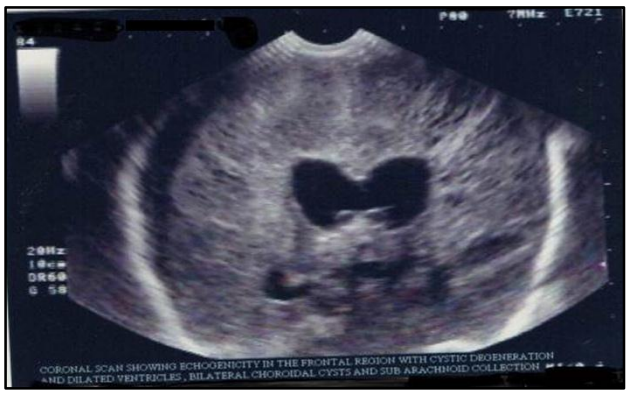

Figure 26. Coronal Scan Showing Echogenicity in the Frontal Region with Cystic Degeneration and Dilated Ventricles, Bilateral Choroidal Cysts and Subdural/Arachnoid Collection

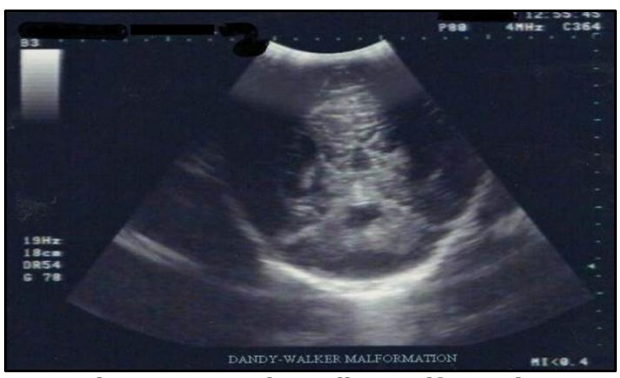

Figure 27. Dandy-Walker Malformation

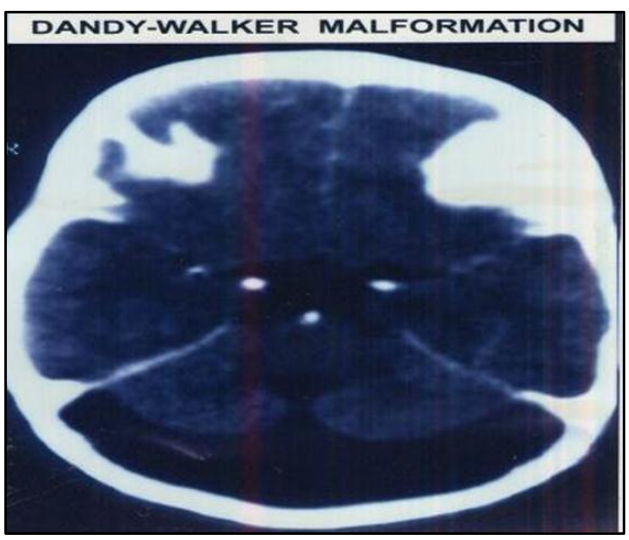

Figure 28. Dandy-Walker Malformation

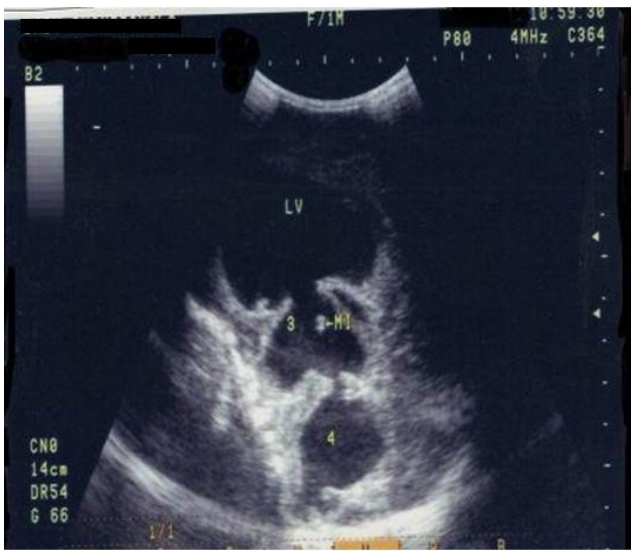

Figure 29. Communicating Hydrocephalus

DISCUSSION

A broad range of intracranial pathologies can be identified in the infant and newborn using transcranial ultrasonography. It is most useful for demonstrating haemorrhage, ventricular size, pulsatility of the major cerebral vasculature and screening for any congenital malformations. Neurosonography represents the primary modality for the evaluation of the preterm brain in gestational immature neonates and is required in every infant as screening sonography.[3]

In our study, (75%) of newborns and infants who were clinically suspected to have some intracranial pathology had abnormal sonographic findings. We could also demonstrate the anatomical structures including basal ganglia, internal capsule and external capsule, claustrum and sylvian fissure along with pulsation of middle cerebral artery within it.

Using the high resolution, real-time ultrasonography of the head in infancy Babcock and DS, Han BK[4] found that 69.11% of patients had hydrocephalus of various aetiologies, the most common abnormality. Hydrocephalus in a neonate should be assumed to be of congenital origin only when other reasons have been ruled out.[5] Twenty three of our patients had congenital hydrocephalus. Out of them, seven had associated meningocoele, two patients had occipital encephalocele, others were having associated anomalies such as giant cisterna magna and Dandy-Walker malformation, 12/23 had non-communicating hydrocephalus, 7/12 of them showed obstruction at the level of the aqueduct, 4/7 had slit-like narrowing at the level of the aqueduct, 3/7 only a presumptive diagnosis was made by dilated lateral ventricles and third ventricle with normal or no visualised fourth ventricle, 3/12 at the level of the foramen of Monro and 2/12 at the level of the 4th ventricle.

We observed that 12 patients with hydrocephalus had a history of meningitis. Five patients had tuberculous meningitis, while the rest had nontuberculous bacterial meningitis. The positive sonographic features included hyperechoic ventricular walls with septation and internal echoes in dilated ventricles suggestive of ventriculitis. The other features were cerebral oedema, infarction and subdural collections. Subdural collections were seen as bilateral sonolucent crescents over the frontoparietal areas and were associated with widening of the interhemispheric fissure.

R.M. Fischer et al[6] and S.V.N. Raju et al[7] found that ventriculomegaly was the commonest abnormality in patients with pyogenic meningitis. However, in one study, ventriculomegaly followed by ventriculitis and cerebral oedema were the common findings.[8]

Intraventricular haemorrhage was observed in 25 neonates, 8 had obstructive PHH. Out of them, three patients with small IVH showed only mild ventriculomegaly, while five patients with significant PVH showed moderate ventricular enlargement. Our observations were similar to a study conducted by Fleischer et al.[9]

Majority of the haemorrhages (70%) were detected in the first three days of life. Out of 11 patients with IVH, the youngest was six hours old and the oldest was eight days old. Anand et al suggested that at least one screening scan is essential within the first two days of life to diagnose IVH.[10]

In our study, out of 25 patients with IVH, six had major haemorrhages, eight neonates were premature and two were term babies. Five patients had minor IVH. SEH appeared as a focal area of increased echogenicity in 3 patients overlying both caudate nuclei on the lateral walls of lateral ventricles in the coronal plane. In the sagittal plane, it was seen to lie in the caudothalamic groove. In our series, IPH was bilateral in 1 neonate with grade IV IVH. It was brightly echogenic and relatively well-defined. The degree of brightness was comparable with that of choroid plexus. IPH was differentiated from focal oedema by the extent of brightness.[11] Echogenic ventricular walls were seen in 3 IVH patients suggestive of chemical ventriculitis. In our study, the ultrasound diagnosis of IVH was confirmed by CT scan in only two patients. CT also revealed associated subarachnoid haemorrhage in one of them. Two patients with IVH showed subependymal cysts on follow-up scans. In our study, out of twenty five patients with IVH, eight had ventriculomegaly. Follow-up neuroscans revealed porencephalic cysts in two patients and cystic encephalomalacia in three others. We had 33 infants with PVL, seven were full-term babies and two were preterm. All of them had a history suggestive of birth asphyxia. We could detect cystic lesions as small as 2-3 mm using 7 MHz and 8.2-11 MHz transducers in selected patients. The cystic lesion was located in both the periventricular and subcortical areas in full-term babies while they were mainly periventricular in preterm infants. Also, two infants had widening of IHF and ventriculomegaly suggestive of cerebral atrophy. In our study, the serologic findings suggestive of PVL were increased periventricular echogenicity in three neonates and a cystic lesion in the periventricular and subcortical in six infants. In the three neonates with increased periventricular echogenicity, evaluation with 7 MHz and 11 MHz transducer revealed bilateral coarse globular echodensities. The echodensities were distributed diffusely in the periventricular white matter in 2 neonates and were located adjacent to the trigone of the lateral

ventricular in one neonate. In all of them, the lesion was characteristically symmetrical. We had only two patients presenting with CP. The sonographic findings in these patients were bilateral cystic lesions of greater than 3 mm diameter in the periventricular region. Mild ventriculomegaly and widening of extra cerebral spaces were the associated findings in them suggestive of cerebral atrophy. Similar to the observation of Pidcock, this finding supports the association of cystic PVL with the development of CP. A Case of hydranencephaly in our study, out of 35 patients with HIE, abnormal sonographic findings were detected in 80%. In our series, out of 3 babies with Focal Parenchymal Echodensities (FPE), we could detect the lesions using conventional US scanner in only two neonates. However, colour duplex Doppler could demonstrate subtle FPE with diminished flow pattern in other patients. Also, by utilising 7 and 11 MHz transducers, we could demonstrate the small 2-3 mm cystic lesions of microcystic encephalomalacia in 2 other patients. Follow-up scans in 4 infants with the widening of IHF revealed changes of cerebral atrophy after 4-8 weeks. It indicates that widening of IHF is probably a forerunner of cerebral atrophy in patients with multifocal or diffuse damage of the brain. In our study, a three days old neonate presented with swelling of anterior fontanelle and facial abnormalities was found on neurosonography to have both the cerebral hemispheres replaced by CSF. Falx was identified. The posterior fossa structures were identified. We had one patient with Dandy-Walker malformation in our study. The sonologic findings were those of a large cystic lesion posterior to the cerebellum, enlarged posterior fossa and dilated ventricular system. Cerebellum appeared hypoplastic and vermis was absent. CT scan confirmed the diagnosis. In our study, we had five patients with a ventriculoperitoneal shunt who presented with clinical deterioration and increasing head circumference. Displacement of shunt tube was demonstrated in 2 patients. The other three patients had moderate-to-severe hydrocephalus though the shunt tube was in place.

Limitations of TCNSG- Technical factors like the noise artefact make the visualisation of the side of the brain nearer the transducer and also structures of the posterior fossa difficult. Nonspecific sonographic appearances of some mass lesions need CT scan. Subtle parenchymal abnormalities and small intracranial calcification may go undetected.

CONCLUSION

All preterm babies with or without a history of birth asphyxia should be screened for intracranial haemorrhage in the first week of life. In patients with secondary Intraventricular Haemorrhage (IVH), a pre-discharge scan is sufficient for detection of any ventriculomegaly. In cases with major IVH, repeat scans should be done until stabilisation of post haemorrhagic hydrocephalus. All HIE patients with periventricular increased echogenicity on the initial scan should be rescanned after four weeks. Detection of the cystic lesions of periventricular leukomalacia in these patients predicts cerebral palsy.

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