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TRANS-FONTANELLAR ULTRASOUND EVALUATION OF NEONATES AND INFANTS IN A TERTIARY CARE HOSPITAL IN GARHWAL REFERRED FOR SONOGRAPHY

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ABSTRACT Ultrasound is widely used to diagnose various conditions in neonates and infants like intracranial hemorrhage, hypoxic-ischemic insult, changes of birth asphyxia and congenital disorders of the brain like hydrocephalus, etc as it is easily available, simple and noninvasive and more important is that it lacks ionizing radiation.

The present hospital-based study of cranial sonography (Trans-fontanellar ultrasound) done in the Department of Radiodiagnosis, HNB Base government teaching hospital situated in Srinagar Garhwal, was done with the aim of assessing the burden of intracranial disorders in neonates and infants, especially the premature newborns who get admitted in neonatal intensive care units.

A retrospective descriptive analysis was done on the data of 196 CUSG done.70.4 % CUSG were performed on neonates, 24.5% were performed on age group 29 days to 6 month age group and rest on age more than 6 months. The male-female gender ratio was 1: 0.96. The most common neonatal pathological conditions were changes of Hypoxic- ischemic encephalopathy (HIE) (28.4 %).

Cranial sonography is a useful diagnostic tool in the evaluation of neonatal and infant brain and is a well-established mode of making primary diagnosis.It is preferred because it can be rapidly performed and is a portable, cost-effective test.

KEYWORDS : cranial sonography, hypoxic insult, birth asphyxia, germinal matrix hemorrhage, meningitis

Introduction

Neonatal and infant cranial sonography is an easily performed, noninvasive diagnostic procedure which can be performed along bedside and is being used widely all over the world in hospital and NICU setup as it is a well-established test to detect spectrum of intracranial disorders in neonates and infants like germinal matrix hemorrhage, hypoxic-ischemic insult, changes of birth asphyxia, hydrocephalus, various congenital neurological defects, and all intracranial issues related to preterm babies. Advances in neonatal intensive care unit techniques and medical care have led to increased survival of preterm babies who are low birth weight babies and develop multiple issues related to premature vulnerable brain parenchyma especially the germinal matrix and the periventricular white matter. Ultrasound can be performed along bedside and even in the incubator as it is portable and easy to use and does not need shifting to departmental ultrasound section. Preterm babies are extremely vulnerable to hypothermia, thus ultrasound is preferred over other modalities especially in vulnerable sick preterm as well as term newborns for obvious reasons as also it requires a short time to scan the sick usually low birth weight babies. Incidence of nuchal cord and meconium aspiration in intranatal period with the increasing use of Caesarean section also contributes such a tendency for hypoxic-ischemic insult and birth asphyxia, as such babies are delivered earlier than expected date in some emergent situations leading to prematurity. Other disorders like congenital hydrocephalus and malformations are easily assessed by ultrasound as a primary investigation before proceeding for either CT or MRI which give better anatomical and pathological details in select indicated and triaged cases especially to confirm or refute any primary diagnosis through ultrasound. It is a feasible diagnostic test in the neonatal cranium as upto 12 or even 18 months of age the anterior fontanelle is open and provides a suitable acoustic window to visualize the intracranial structures at ease and the findings are easily reproducible. Incoming sections, we will deal into the details of techniques of cranial sonography, anatomical considerations of the brain, ultrasound results-oriented discussion and the relevant case findings with elaborate discussion over the disorders found in our hospital-based study.

Materials and methodology

A retrospective cross-sectional descriptive analysis was done on data of all the neurosonograms of neonates and infants at the Radiology department of HNB teaching government hospital over a period of 3 years from April 2016 to March 2019.

The Hemwati Nandan Bahuguna (HNB) Teaching Hospital is the associated hospital to Veer Chandra Singh Garhwali Government Institute of Medical Sciences and Research. It serves people of four districts of Uttarakhand namely TehriGarhwal, Rudraprayag, Chamoli, and parts of PauriGarhwal. This tertiary care center is in a remote hilly region at Srikot, Uttarakhand, India on Badrinath highway.

Real-time cranial ultrasound was performed on L & T Sonalisa USG machine and Toshiba Nemio SSA 510A color Doppler machine using a convex array transducer of 3.5 MHz.

A total of 7995 non-antenatal ultrasounds were performed in the study period, out of which for study purposes, 196 cranial ultrasounds of neonates and infants were included as per inclusion criteria.

Ultrasound coupling gel was applied over the anterior fontanelle of child and scanning of brain and surrounding structures was done in coronal as well as sagittal, parasagittal planes across the lateral and third ventricles as shown in **figure 1** illustrating the planes of scanning done with short focal length to visualize entire cerebral cortex and deeper gray matter and posterior fossa structures. Care was taken to avoid hypothermia to the child.

The interpretation of cranial ultrasound was done under the following:

1) Symmetry of cerebral hemispheres, cerebellar structure and size, shape, and echogenicity of ventricles
2) Periventricular area and echogenecity with details and symmetry
3) Falx cerebri and shift of midline structures.
4) Status of the choroid plexus especially Trigonal regions and their symmetry.
5) Echogenicity of cerebral parenchyma, thalami and basal ganglia
6) Sylvian fissure, corpus callosum, cingulate and hippocampal gyri.
7) Any abnormal hyper or hypoechoic area
8) Any mass lesions
9) Cavum septum pellucidum and vergae.
10) Cranial vault and status of scalp, occiput and nape of neck.

Data on all in- and outpatients in whom cranial ultrasound was

performed during diagnostic evaluation over a 3-year period were retrospectively collected.

The data thus obtained from the ultrasound examinations regarding name, age, sex, locality, birth history, birth weight, sibling history, maternal history, clinical symptoms and ultrasound findings were retrieved on the pre-designed format.

The results were entered and tabulated in Microsoft Excel sheet, and descriptive analysis was done.

This study has been approved by the Institutional Ethical Committee of Veer Chandra Singh Garhwali Government Institute of Medical Sciences and Research.

Results and result-based discussion: - [Kindly Refer to Tables 1 to 5]

A total 7995 non-antenatal ultrasound cases were performed in the study period of 3 years from April 2016 to March 2019, out of which 196 cranial ultrasounds were performed over infants and neonates forming about 2.45% of all ultrasound studies done. 29 cases of all cranial ultrasound cases were normal in term neonates and infants, forming 15.31 % of all cases. 20 cranial ultrasounds were rejected on account of the poor acoustic window in children having closed anterior fontanelles.

For analysis, age was categorized in three age groups; less than 29 days (neonates), 29 days to 6 months and more than 6 months.

Of 196 cranial ultrasounds; 70.4 % were performed on neonates, 24.5% were performed on age group 29 days to 6 month age group and rest on age more than 6 months.The male-female gender ratio was 1: 0.96.

The most common neonatal pathological conditions which we came across in our study of neurosonography were changes of Hypoxic- ischemic encephalopathy (HIE) (28.4 %) and germinal matrix bleeds (11.22%).

Changes of birth asphyxia were seen in 36 cases (18.4%) forming a major subset of pathological conditions in neonates and infants. Entire brain parenchyma appears echogenic in such cases with usually slit-like ventricles due to cerebral edema, with thalamus appearing bright on both sides in the majority of cases**[Figure 9].** Twelve cases showed changes of hypoxic-ischemic encephalopathy with moderate to severe peritrigonal bilateral hyperechogenecities with lateral ventricular prominence, with a subependymal cyst in almost 6 cases.

Apart from 15.31% (29 cases) normal cranial ultrasound studies of term babies and infants; out of total 196 cases of cranial ultrasounds, we found developing the premature brain in almost 12.24% cases (24 cases). Premature developing brain on ultrasound has poor cerebral sulcal delineation and persistent cavum septum pellucidum in almost all cases and the majority had cavum vergae. They are seen as anechoic cystic midline spaces between lateral ventricles. They also had mild hypomyelinated periventricular hyperechogenecities bilaterally, especially in peritrigonal areas. Lateral ventricles were usually bit prominent.

Germinal matrix hemorrhage or subependymal bleed on ultrasound were seen as a focal echogenic area in caudothalamic recess along the floor of lateral ventricles in about 22 cases (11.22%) [**Figure 2**]. They had mild ventricular dilatation in 6 cases, Grade 2 neonatal ICH was seen in three cases seen as an asymmetrical enlargement of choroid plexus in lateral ventricles in preterm newborns [**Figures 3 & 4**].

21 cases of the subependymal cyst were seen out of which there were 13 isolated cases of the subependymal cyst and 8 cases were associated with either of birth asphyxia or hypoxic insult or mild ventriculomegaly [**Figure 6**].

Mild ventriculomegaly was seen in eight neonates and infants and represented periventricular white matter loss in the neonatal period due to hypoxic insult or in some cases represented sequelae of meningitis. Some may presumably be due to sequelae of neonatal ICH.

Intracranial infections (meningitis /meningoencephalitis) were seen in 19 cases (9.7%) with findings of echogenic cerebral sulcal spaces with bright tentorium cerebri and/ or interhemispheric fissure. Most of them had ventricular dilatation. Few cases of meningitis had diffuse cerebral edema. Diffuse cerebral edema without obvious cause was seen in three cases, presenting with seizures and showed slightly reflective brain parenchyma with slitlike ventricles and effaced basal cisterns.

Incidental unilateral lateral ventriculomegaly(usually on the left side) was seenin 5 newborns and early infants. Bilateral basal ganglia calcification was seen in association with hypoxic insult in a one month infant.

Focal occipital focal parenchymal calcification on the left side was seen in a three-day child.

A neonate of fifteen days showed an occipital midline skin covered swelling obviously missed on antenatal ultrasound, ultrasound showed a meningoencephalocele with brain parenchyma herniating through midline occipital bony defect of 9 - 14 mm and external swelling containing herniated brain with its meningeal coverings [**Figure 7**].

A rare unusual case of cystic encephalomalacia was seen on ultrasound in a 13-day old male child with large irregular cystic periventricular areas and cerebral parenchymal distortion [**Figure 10**]. It might represent either sequelae of TORCH group of infection (usually HSV-2 infection) or sequelae of high-grade ICH.

An 8 days old male child with large head size and wide open anterior fontanelle had Dandy-Walker malformation with moderate obstructive hydrocephalus**[Figure 12**].

Two cases of congenital aqueductal stenosis were seen with typical sunset eyes, large head, bulging fontanelles with obstructive hydrocephalus in a 3-day child and a 7-month infant respectively.[**Figure 8]**

Scalp collections called as cephalhematoma or subgaleal hematomas were seen in 10 neonates who developed boggy scalp swellings immediately after birth. Usually, they were seen in parieto-occipital scalp region [**Figure 11**].

One infant of 7 months had fallen from the bed and sustained an undisplaced fracture of left parietal bone with overlying scalp hematomas and was detected by ultrasound.

Discussion:-

Why cranial ultrasound be performed if at all? Data says the most common cause of neonatal mortality is preterm birth and low birth weight[7]. Why so?- neonatal intracranial hemorrhage and periventricular leukomalacia (part of Hypoxic ischemic encephalopathy)[1, 2, 3, 4, 5, 6, 7, 8]. The value of cranial ultrasound cannot be debated especially in developing countries as World Health Organization (WHO) recommends as primary modality of diagnosis in preterm and term neonates[6]. The brain is one of the first organs in body investigated since 1974 through the 1970s by ultrasound[1]. The cranial vault has natural acoustic window provided by anterior and posterior fontanelles in neonates and infants, till the time it closes by 12 months when it becomes difficult to get clear images[1, 2, 3, 4, 5]. The main indication initially was hydrocephalus but now other indications have preceded this one like issues of preterm babies like hypoxic insult, birth asphyxia, intracerebral hemorrhage, congenital malformations and other, with ever-increasing survival of preterm babies as early as 25 to 28 weeks with low birth weight

(LBW) as low as 900 gm to 1500 gm wt[1, 2]. This is now possible because of high-end ultrasound machines with sector probes with a frequency of 5 MHz and having short focal length instead of previously used convex array transducer with lower frequency[1]. Sector probes can probe deeper into the cranial cavity and parieties which are not seen clearly with convex probes. Scanning is done with sector or convex array probe with frequency of 5 - 7 MHz through acoustic window provided by anterior fontanelle in coronal and sagittal, parasagittal planes across the midline so as to visualize entire brain parenchyma with ventricular system[1, 2, 3]. [**Figure 13**] In addition additional views are obtained by scanning in axial plane through the thin squamous temporal bone across squamosal sutures and anterolateral fontanelles[1, 2, 3]. There is usually no need of sedation as preterm babies are relatively immobile and usually sick, in moving babies cineloop facility helps to grab optimal images[1]. Cumbersome risky travel to CT suite and MRI suite is avoided by judicious use of cranial ultrasound[5]. Pitfalls do occur in neurosonography in form of overriding of cranial bones due to moulding during delivery and dense scalp hair when copious amounts of gel need to be applied[1]. At the ouset, it should be note that in complex situations like congenital anomalies and white matter disorders in term infants, and cranial trauma, CT or MRI is preferred, so also in cases of cerebral calcifications, CT is preferred[5]. In the large head, in term infants, we need to use 3.5 MHz frequency probe. Color Doppler study can be used to visualize the intracranial vessels and sinuses in relevant cases[1, 2, 3]. So also ultrasound is good alternative to CT when cost is an issue as it gives comparable results[8]. A spinal sonogram can be done in the infant's spine [**Figure 14**] to detect spina bifida. **Figure 5** depicts the utility of spinal sonogram in detecting lipoma myelomeningocele (skin covered) in a student of a local college.

Normal sonographic anatomy of neonatal brain:-

The various clinical indications of neurosonography include preterm babies, low birth weight, seizures, boggy anterior fontanelle, split sutures, abnormal neurological findings, back midline swelling, swelling in nape of neck, large head size, listless baby, child does not accept feed, birth asphyxia, tachycardia, tachypnoea, cranial trauma, cephalhematomas, critically ill preterm and term neonates[5, 6, 8]. Germinal matrix bleeds present with hypotonia, another indication for neurosonography[8]. Cranial ultrasound is also indicated in pretems who need ECMO (extrtacorporeal membrane oxygenation) as any intracranial bleed above grade 1 is contraindication for ECMO[5].

We will first delve into expected normal sonographic features of preterm and term neonate.

The fluid-filled (CSF) echo-free ventricular system provides a readily identifiable framework for demonstration of anatomical structures in the brain, the two cerebral hemispheres, cerebellum and deeply seated gray matter nuclei like basal ganglia and thalami which line third ventricle, periventricular white matter and choroid plexuses situated in the lateral ventricles[1, 2, 3]. On ultrasound, cerebellum appears like an echogenic leaf-like structure in posterior fossa behind brainstem[1].

We can identify the following other details like:-

1) the ventricular system which compromises of two lateral ventricles, third ventricle which communicates with lateral ventricles via foramina of Monro on either side and the fourth ventricle which connects with the third ventricle via cerebral aqueduct of Sylvius[1, 2, 3]. The width of lateral ventricles is about 10.2 to 13 mm (Lou et al J Pediatr 1979, 94: 118) at the level of the foramen of Monro and behind the foramen of Monro, it is 9 mm) (Levine M, Sturte D, Arch Dis Child 1981, 56)[1].
2) Solid structures like caudate nucleus lie beneath the anterior horn of lateral ventricles forming its inferior border, whereas the thalamus forms the lateral wall of the third ventricle[1, 2, 3]. Between caudate nucleus and thalamus on either side lies the germinal matrix in the caudothalamic recess or groove[1, 2, 3]. The germinal matrix is a highly vascular friable structure which is prominent till 35 weeks of gestation[1, 2, 3]. It is important because it has a critical role in the proliferation of cells which give rise to the neurons in the cerebral cortex and basal ganglia and consists of the immature friable tiny capillary network which are prone to bleed in the precarious preterm newborns in any event of hypoxia, ischemia or asphyxia[1, 2].
3) Mild degrees of asymmetry of lateral ventricles is common in neonates usually left being slightly larger than right[1].
4) The thalami are slightly echogenic than adjoining brain parenchyma, connected in the midline by a rounded small Massa intermedia[1]. The third ventricle is in the midline and is slitlike[1,2,3].
5) Cavum septum pellucidum and cavum vergae are CSF filled cystic spaces in the midline, seen as anechoic areas in between two lateral ventricles, seen in 95 % of preterm babies and only 15 % of term babies[1].
6) Corpus callosum is a midline largest commissure seen as hypoechoic lining above the roof of lateral ventricles[1].
7) Cerebral sulci and fissures are seen as prominent linear markings in the cerebral hemispheres[1,2,3,4,5].

Pathological conditions in neonatal and infant brain:-

The major intracranial disorders which we routinely come across the clinical practice on ultrasound in neonates and infants are as follows[1,2,3,4,5]:

1) Hypoxic-ischemic encephalopathy (hypoxic insult) 2) Birth asphyxia 3) Neonatal intracerebral hemorrhage (ICH) 4) Hydrocephalus 5) Periventricular leukomalacia (PVL) 6) Intracranial infections 7) Congenital malformations 8) Space-occupying lesions 9) cranial trauma.

We will now go into details of each relevant disorder more so which we came across in our study;

1) Neonatal intracerebral hemorrhage[1,2,3,4,5]:

With the advent of greatly improved obstetric care and advance in neonatal intensive care medicine, there is steadily increasing survival rate of premature babies even as early as 22 - 23 week of gestation with birth weight as low as 900 gm to 1500 gm wt. Thus in Indian scenario, we find the increasing incidence of germinal matrix hemorrhage which is being detected reliably on cranial ultrasound[3]. It forms one of the major indications of cranial sonography[3].

The germinal matrix bleeds especially during 25 to 28 week along caudothalamic recess with spillage of blood across ependyma into lateral ventricles due to hypoxia and hypercapnia in an event of hypoxic insult or asphyxia with resulting vasodilatation. This occurs in preterm babies who have respiratory issues like hyaline membrane disease.

Neonatal ICH can be classified by **Papile**[3] into four ultrasonic grades

1) Grade 1- subependymal bleeds
2) Grade 2- mild intraventricular hemorrhage with minimal ventricular dilatation
3) Grade 3- Intraventricular hemorrhage with marked ventricular dilatation
4) Grade 4- Intraparenchymal hemorrhage (venous hemorrhagic infarct) [Volpe et al][5]

Grade 1 bleeds usually have self-limiting course, as only 2% suffer from a neurological handicap. It usually resolves completely or leaves behind a thin echogenic gliotic scar or infrequently results into a small echo-poor subependymal cyst, which persists for a year or two until getting obliterated by growing brain[1, 2, 3]. Grade 2 to 4 bleeds undergo lysis of clots with ventricular dilatation due to fibrin deposition blocking narrower points of ventricular system[1, 2]. Ventricular dilatation might get resolve in grade 2 bleeds but remains persistent in grade 3 & 4 bleeds leading to hydrocephalus, raised intracranial tension and its neurological manifestations[1, 2]. It might require CSF drainage by lumbar puncture or even ventriculoperitoneal shunting[1, 2]. Such cases with ventricular dilatation need serial ultrasound follow-ups to look for persistent

hydrocephalus and plan for VP shunt accordingly[1,2]. Grade 4 bleeds usually present near germinal matrix in frontoparietal lobes eventually leading to cystic brain necrosis and porencephaly[1,2]. Thus grade 3 & 4 bleeds have severe neurological deficits in the form of spastic diplegia and quadriplegia in almost 50 to 96% cases[1,2]. Rarely bleeds can develop in cerebellum[1].

In short, a screening ultrasound needs to be performed to detect ICH in all preterm infants born under 30 weeks of gestation once in initial 2 weeks and another in the 4th week of postnatal period[1,2].

Ventriculomegaly is graded as mild (0- 5 mm), moderate (10- 15 mm) and severe (> 15 mm) as assessed by ultrasound measuring lateral ventricular width in the sagittal plane at midbody level. (Ment et al)[3]. On ultrasound, the intraventricular bleeds appear as contour abnormalities of echogenic choroid plexus with the extension of such echogenicity anterior to the foramen of Monro[1, 2, 3]. Trigonal choroid plexus appears bulky with asymmetry[1,2]. It is reported that upto 5% of cases of IVH need VP shunting[2].

2) Hypoxic-ischemic encephalopathy (HIE)[1,2,3,4,5]:

It is one other major indications of neurosonography as it has major clinical and socioeconomic implications, being a major cause of cerebral palsy and neurodevelopmental impairment[1,2]. It results in periventricular leukomalacia in 5- 10% of preterm newborns and in such cases have poor prognosis[1,2]. Hypoxia indicates reduced blood oxygenation whereas ischemia indicates reduced blood perfusion and both terms indicate the same process[1]. It results from preterm white matter injury and the severity can be graded on the basis of ultrasound findings as(Di Vries grading)[1,3]:

1) Grade 1- increased periventricular echogenicity lasting upto 7 days
2) Grade 2- increased periventricular echogenicity lasting beyond 7 days
3) Grade 3- increased echogenicity with a tiny periventricular cyst formation
4) Grade 4- coalescing periventricular larger cystic areas
5) Grade 5- coalescing larger cystic areas reaching upto subcortical white matter.

These changes by definition are bilateral and the severity grading is possible only through serial postnatal ultrasound follow ups[1,2]. The cysts represent cerebral infarction with necrosis, in the water shed areas between the penetrating ventriculopetal arteries and the deep ventriculofugal arteries, located 3- 10 mm from the lateral ventricular borders[1,2]. The highly reflective periventricular flare is due to vascular congestion, early white matter necrosis and hemorrhage with development of echo-poor foci within 2- 6 weeks eventually converting into periventricular leukomalacia pattern[1]. Apart from hypoxic insult in the perinatal period, hypotensive crises, premature rupture of membranes and intrauterine infections all lead to PVL[1]. MRI has an important role to play in cases who thrive better despite PVL[1]. On ultrasound, cases of PVL eventually show changes of cerebral atrophy and widened extracerebral CSF spaces[2]. The higher incidence of HIE of almost 28.42 % in our study might be due to the higher prevalence of preterm deliveries in hills on account of an obviously difficult life and cumbersome travel in hilly terrains

3)Acute Ischemic insult (Status Marmoratus)[1,2]:

It is due to an acute episode of profound hypoxia leading to thalamic and/or basal ganglia hyperechogenecities with diffuse cerebral edema, which on USG is seen as effaced cerebral sulcal spaces with obliteration of basal cisterns[1,2]. These thalamic changes are due to neuronal necrosis and gliosis. Though ultrasound features are nonspecific in case of cerebral edema, still it can be used as primary modality however CT is supposed to be a diagnostic test of choice in diffuse cerebral edema. MRI also has a role in acute brain ischemia seen as hyperintense areas on DWI[1].

4) Ischemic insult in term infants[1,2]:

Prolonged ischemia leads to a) selective neuronal necrosis - on ultrasound seen as diffusely reflective brain parenchyma including similar changes in basal ganglia, thalami, cerebellum due to edema as well as ischemia, associated with loss of grey-white matter differentiation[1]. Eventually, there is gliotic scarring with cerebral atrophy[1].

5) Changes of birth asphyxia[1,2,3]:

It leads to a significant number of deaths and serious neurological handicaps. On ultrasound, there is usually increased reflectivity of brain parenchyma with slit-like ventricular system and effaced basal cisterns. It eventually leads to widespread gliosis and cystic encephalomalacia.

6) Intracranial infections[1,2,3]:

They are meningitis, meningoencephalitis,ventriculitis, and brain abscess, occurring due to TORCH group of infections in utero like CMV, toxoplasmosis, group B streptococcus, E coli, HSV-2 and other bacteria[1, 2]. On ultrasound, meningitis is seen as the presence of echogenic cerebral sulcal spaces resulting from inflammatory exudates around pial and subarachnoid vessels in sulcal and fissural spaces[1, 2]. Eventually, there develops ventriculomegaly and widening of extracerebral CSF spaces seen as subdural effusions[1,2]. The hydrocephalus is of communicating type as exudates block strategic points like arachnoid villa, cerebral convexity in addition to the foramen of Monro, aqueductal region, foramina of Magendie and Luschka[1].

Cytomegalovirus infection is the commonest and most serious of all intracerebral infections, leading to necrotizing inflammation of germinal matrix and periventricular white matter with resulting periventricular calcifications, microcephaly and polymicrogyria[1, 2]. Toxoplasmosis of the brain leads to ventriculomegaly and cerebral calcifications even in utero, especially in basal ganglia, thalami and periventricular as well as cortical areas[1,2].

Mineralizing reactive vasculopathy resulting from congenital infection leads to highly reflective thalamostriate and lenticulostriate vessels with striking appearances due to striated bright branching pattern bilaterally on ultrasound[1]. We found at least two to three such cases associated with acute ischemia and infections.

Ventriculitis is seen in 65- 90% of cases of bacterial meningitis and is one of the major causes of neonatal mortality. On ultrasound, it is seen as a dilated ventricular system with internal low-level echoes, irregular choroid plexuses, and thick irregular echogenic lining of ventricles[1,2]. Trapped ventricles are common due to band and septae formation as a result of glial proliferation, fibrinous basal arachnoiditis with especially dilated trapped fourth ventricle[1,2].

7)Congenital anomalies[1,2,3]:

We will consider only a few conditions which we encountered in our study, as the entire list of anomalies of the brain of congenital origin with details is beyond the scope of this article. Most of these anomalies can be diagnosed in utero on antenatal ultrasound as early as 18- 19 weeks gestation. Congenital malformations which are the result of primary errors of development or from destructive injuries in utero can be divided as:-

a) **Disorders of cytogenesis and histogenesis[1]**: which includes Sturge Weber syndrome, Tuberous sclerosis, Neurofibromatosis type 1& 2 and aneurysmal malformation of vein of Galen.

b) **Disorders of organogenesis[1]**: which include disorders due to failure of neural tube closure like - anencephaly, encephalocele, meningocele, myelomeningocele, and other like Arnold Chiari formation, dysgenesis of the corpus callosum and Dandy-Walker Malformation.

c) Disorders of diverticulation[1]: includes Holoprosencephaly and its three subtypes.

d) Disorders of sulcation and migration[1]: like lissencephaly,

agyria/pachygyria complex, and schizencephaly.

e) Destructive brain lesions[1]: Hydranencephaly and porencephaly.

Now we will consider a few relevant congenital brain malformations;

1) Encephalocele / meningoencephalocele[1,2]:
Encephalocele is commonly seen in the occipital region which on ultrasound shows posterior herniating sac covered with meninges and contains varying amounts of dysplastic brain, ventricles, vessels, and CSF with a cranial defect seen along the neck of swelling. Cervico-occipital encephaloceleis associated with type 3 Chiari malformation, in which there is a severe form of cerebellar herniation through foramen magnum downwards, with hindbrain abnormalities, the fourth ventricle appears elongated and kinked. The cerebellum is hypoplastic. Lateral ventricles are dilated. We found two cases of encephalocele with one having dysgenesis of the corpus callosum.

2) Dysgenesis of corpus callosum[1,2]:
Corpus callosum is the major commissural structure connecting two cerebral hemispheres. It is formed in 8- 12 weeks of gestation and is derived from lamina terminalis. Any insult in utero before 12 weeks of gestation leads to either agenesis or partial dysgenesis of the corpus callosum. Then the midline fibers instead of crossing across midline become oriented posterio-anteriorly as the bundle of Probst lying along widely placed parallel lateral ventricles along superomedial aspects indenting them with characteristic elongation and superior displacement of the third ventricle. There is dilatation of occipital horns of lateral ventricles (colpocephaly). The anterior horns of lateral ventricles are narrowed and peaked (Viking horn appearance). The cerebral sulci are seen diverging radially away from the third ventricle. Dysgenesis of the corpus callosum is associated with other cerebral anomalies like Chiari 2 malformation, encephalocele, Dandy-Walker (DW) malformation, interhemispheric cyst, and intracranial lipomas. Sometimes there is an absence of posterior portion.

3) Dandy-Walker malformation[1,2]:
It is a cystic balloon-like dilatation of fourth ventricle with large posterior fossa and inferior vermian/cerebellar hypoplasia, it occurs due to atresia of foramina of Magendie and Luschka. Dandy-Walker variant includes lesser degrees of vermian hypoplasia and fourth ventricle. Dandy-Walker variant, mega cisterna magna and retrocerebellar cyst all form DW spectrum. In true DW malformation, there is dilatation of lateral ventricles as in our case detected on ultrasound. The occipital horns of lateral ventricles show typical divergence due to posterior fossa cyst and elevated tentorium. Torcularherophile is displaced upwards.

3) Porencephaly[1,2]:
It results from the destruction of brain parenchyma in the first or early second trimester. The resulting cystic areas may or may not communicate with the ventricular system and are not lined by grey matter. It results from in utero infections; cerebral infarction or germinal matrix hemorrhages and represents encysted necrotic damaged brain.

4) Hydrocephalus[1,2,3]:
Hydrocephalus results from excessive accumulation of CSF in the ventricular system and/ or subarachnoid spaces due to excessive CSF secretion, or obstruction of CSF flow either by extraventricular or intraventricular cause. In communicating hydrocephalus, there is an extraventricular obstruction to CSF flow either at the level of arachnoid villi or cerebral convexities. In obstructive hydrocephalus, there is intraventricular obstruction of CSF flow proximal to outlet foramina of the fourth ventricle. In neonates and infants, the various causes of hydrocephalus are:

i) Post-hemorrhagic hydrocephalus
ii) Infections like meningitis and ventriculitis
iii) Tumors and cysts obstructing the ventricular system at various sites
iv) Congenital causes aqueductal stenosis (X-linked), Chiari 2 malformation, DW malformation
v) Post-traumatic communicating hydrocephalus
vi) Benign communicating hydrocephalus of infancy or chronic subdural hygromas of infancy due to transient impairment of arachnoid villi.
vii) Choroid plexus papilloma which secretes excess CSF.

In congenital aqueductal stenosis, there is dilatation of the lateral ventricles with a normal fourth ventricle due to stenosis of cerebral aqueduct of Sylvius. Clinically they present with a large head with full or bulging anterior fontanelle with an increasing head circumference on serial measurements.

5) Space-occupying lesions in neonates and infants[1,2]: they are as follows;

a) **Neoplastic lesions**, very rare (2/100000), including astrocytomas, ependymomas, primitive or developmental tumors, choroid plexus papilloma, mostly are supratentorial in location.All cases of intracerebral tumors need to undergo either CT or MRI for a complete evaluation, accurate characterization of tumor. Most of the intracranial tumors appear echogenic on ultrasound but some have mixed echogenicity. Ependymoma and teratomas occur in infancy.

b) **Cysts** including porencephalic cyst, arachnoid cyst.A cyst is a fluid-filled cavity within or adjacent to the brain and has mass effect[1]. Congenital cysts are seen in association with dysgenesis of the corpus callosum and alobar Holoprosencephaly[2].

c) **Vascular malformations** like congenital arteriovenous malformations and vein of Galen aneurysmal malformation[1, 2].

5) Traumatic brain lesions[1]:
They include accidental trauma, birth-related trauma and non-accidental injury (NAI)[1].

Birth-related trauma results from an abnormal presentation like breech, or surgical instrumentation like forceps-assisted delivery or ventouse application for extracting fetal head. It can lead to scalp injury with cephalhematoma or extracerebral bleeds like extradural or subdural bleeds and also can cause parenchymal bleeds, usually in cerebellum[1]. They present with bruising and boggy scalp swellings. There may be over-riding of cranial bones at sutures. Ultrasound can help in identifying cephalhematoma, intracranial bleeds and sometimes even fractures of the cranial vault. But it can miss EDH in parieties along with blind spots just near the anterior fontanelle[1]. CT helps in such dicey cases[1].

Conclusion:-
Cranial sonography is a useful diagnostic tool in the evaluation of neonatal and infant brain and is a well-established mode of making primary diagnosis in wards and bedside, including NICU, setup in hospitals and private clinic. Our study shows the various spectrum of findings in newborns especially the premature newborns and also the term infants along with older infants including hypoxic insult, birth asphyxia, hydrocephalus, germinal matrix hemorrhages, encephaloceles, periventricular leukomalacia, cephalhematomas, and others. It is preferred because it can be rapidly performed and is a portable, cost-effective test. It helps in follow up of patients with intracerebral hemorrhage and hypoxic insult. It lacks ionizing radiation. It is very much feasible in hospitals which do not have high-end equipments like CT and MRI. Our study shows this effectiveness of ultrasound of neonatal brain through anterior fontanelle in detecting such good number of positive cases which help in management decisions by clinicians and help prognosticate the case and can help clinicians to decide whether a neurosurgical procedure be performed like VP shunting in aqueductal stenosis or perform lumbar puncture to detect meningitis by CSF sampling or advice medical management in relevant cases.

Table 1:showing list of pathological conditions found on neurosonography

Sr No	Pathological Conditions found	No of cases(n=196)	% cases
1	Developing premature brain	24	12.24%
2	Germinal matrix bleeds	22	11.22%
3	Birth asphyxia	36	18.7%
4	Hypoxic insult	12	6.12%
5	Meningitis	19	9.7%
6	Bland ventriculomegaly	08	3.06%
7	Congenital hydrocephalus	06	4.1%
8	Diffuse cerebral edema	03	1.53%
9	Normal study	29	15.3%
10	Unilateral ventriculomegaly	05	2.5%
11	Subependymal cyst	21	6.63 %
12	Meningoencephalocele	02	1.2%
13	B/L basal ganglia calcification	01	0.51%
14	Cephalhematoma	10	5.1%
15	Skull bone fractures	01	0.51%
16	Porencephaly	02	1.2%
17	Parenchymal calcifications	01	0.51%
18	K/C/O Down's syndrome	01	0.51%
19	Dermoid cyst scalp	01	0.51%

Table 2:- Age group of neonates and infants referred for neurosonography

Sr no.	Age group	No of cases (n=196)	% cases
1	< 28 month	138	70.40%
2	>29 days to 6 months	48	24.5%
3	6 months and above	10	5.1%

Table 3:- Sidedness of some focal intracranial lesions

Sr No	Condition	Right-sided	Left-sided	IVH// B/L
1	Germinal matrix bleed	05	12	03 IVH
2	Subependymal cyst	03	15	03 B/L

Table 4:- Causes of ventriculomegaly

Sr No	Causes of Ventriculomegaly	No of cases
1	Periventricular leukomalacia	08
2	Intracerebral bleed	04
3	Meningitis	19
4	Congenital hydrocephalus	06
5	Unilateral ventriculomegaly	05

Table 5:- Various causes of scalp lesions.

Sr No	Scalp lesions	No of cases
1	Cephalhematoma	10
2	Fracture related	01
3	Benign lesion	01
4	Meningoencephalocele	02

Figure 1- Illustration showing scanning planes of Trans-fontanellar ultrasound of newborn in various coronal and sagittal parasagittal planes by an ultrasound probe placed over the anterior fontanelle.

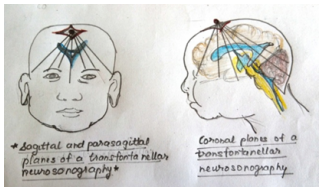


Figure 2- Coronal neurosonogram showing acute left germinal matrix hemorrhage seen as an echogenic focal area in the left caudothalamic recess along the floor of the anterior horn of lateral ventricle.

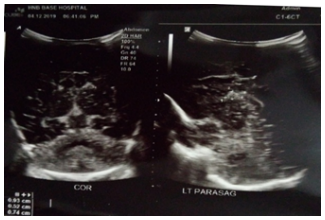


Figure 3-Neurosonogram of a preterm newborn showing left acute GMH with left lateral ventricular hemorrhage with minimal ventricular dilatation (grade 2 ICH)

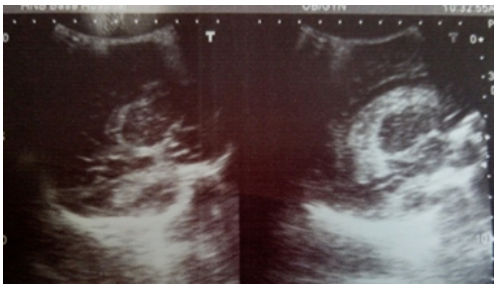


Figure 4- Grade 2 neonatal ICH in left lateral ventricle with echogenic brain parenchyma (changes of birth asphyxia).

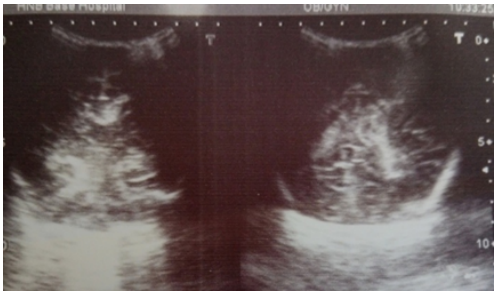


Figure 5- An example of a spinal sonogram of 23-year-old student showing Spinal Bifida manifesta at L3, L4, L5 vertebral levels with lipomyelomeningocele.

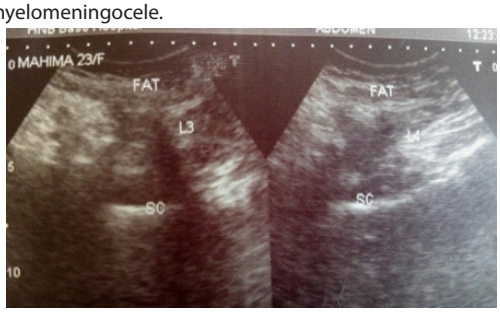


Figure 6- Left subependymal cyst in a parasagittal neurosonogram in LBW preterm baby with poor cerebral sulcal delineation.

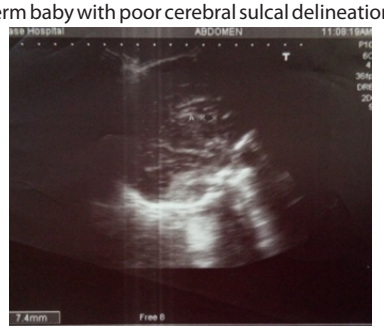


Figure 7- Linear probe scan through external occipital swelling in the midline in an infant showing herniated brain parenchyma through 18 x 13 mm defect and external skin covered swelling of 54 x 39 mm. (Cervico-occipital encephalocele) .

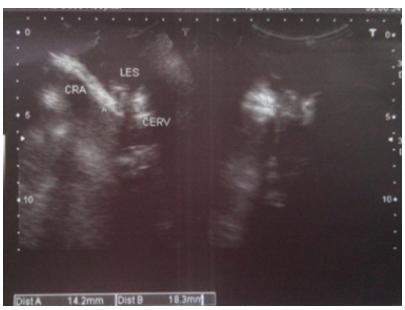


Figure 8- Moderate hydrocephalus due to congenital aqueductal stenosis on coronal neurosonogram.

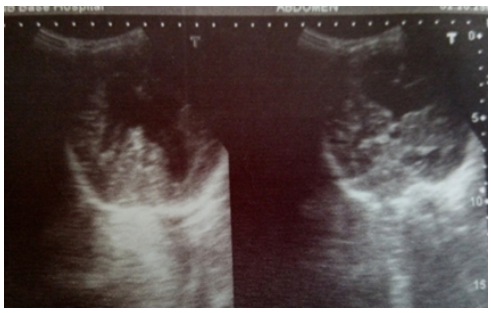


Figure 9- Bilateral thalamic hyperechogenecities in a newborn having an acute episode of asphyxia due to aspiration of milk.

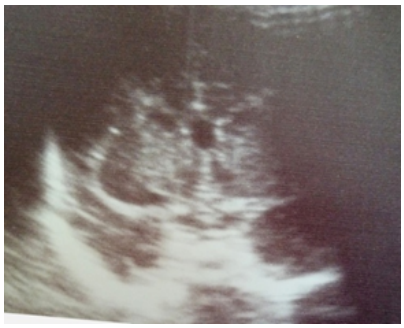


Figure 10- Cystic encephalomalacia (porencephaly) in an infant as seen on neurosonography.

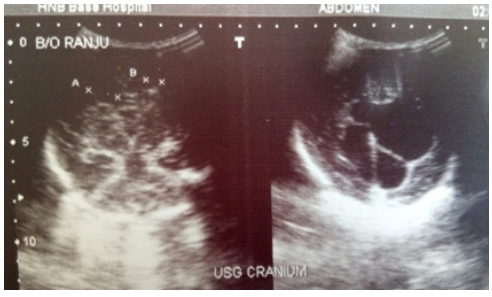


Figure 11- Linear probe scan of the scalp in a case of cephalhematoma in the left parieto-occipital scalp region, seen as an anechoic area in subgaleal location.

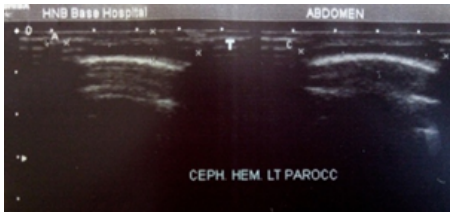


Figure 12- Moderate to severe congenital obstructive hydrocephalus due to Dandy-Walker malformation of the posterior fossa in an infant on neurosonogram.

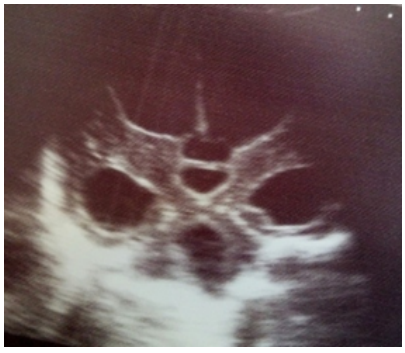


Figure 13:- Normal Neurosonogram.

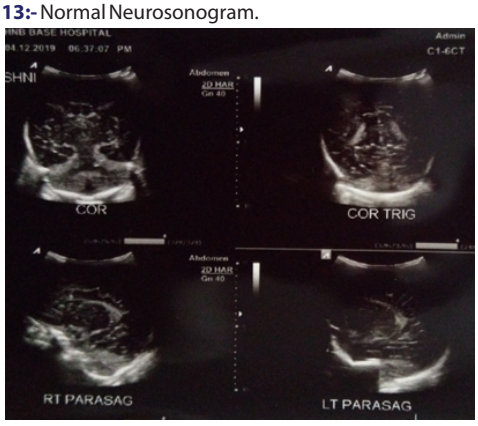
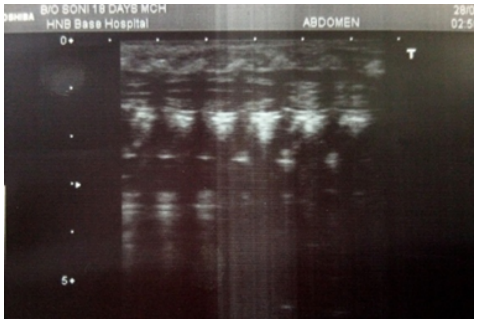


Figure 14:- Normal neonatal spine sonography demonstrating the lower end of the cord with a central canal and cauda equina in the spinal canal.



REFERENCES

1) The neonatal brain, Tim Jaspen: Chapter from Textbook of Clinical Ultrasound (Volume 2), Editor David Cosgrove, October 2000 publication, Churchill Livingstone publication, pg. 1055- 1103.
2) Cranial sonography, Rajeev Jain, SeemaSawhney: Chapter from textbook of Diagnostic Radiology series (Paediatric Radiology), Editor Manorama Berry et al, 1997, First edition, Jaypee brothers medical publishers limited, pg. 225- 235.
3) Venkatraman Bhat, Varun Bhat, Neonatal neurosonography: A pictorial essay, IJRI, Nov 2014, 24(4): 389-400,DOI: 10.4103/ 0971- 3026.143901.
4) N Vainak et al, Neonatal brain ultrasound - a practical guide for the young Radiologists, Electronic presentation online system, European society of Radiology, DOI: 10.1594/ecr2014/c-0527.
5) Arie Franco, Kristopher Neal Lent; Dove Press journal,26/9/2013, Reports in Medical imaging, 2013: 6, 93-103, http://dx.doi.org/10.2147/RMI.S46473.
6) Carsten Kruger, Naftali Naman; Cranial ultrasound in neonates and infants in Rural Africa; SAJCH September 2010 Volume 4 (3): 83-87.
7) Vittal Prasad Chinta, Evaluation of cranial sonography indices in infantsand neonates; IJMSPH 2016; 5(4): 1492- 1495.
8) Joao Guilherme Bezerra Alves et al; Transfontanellar Ultrasonography in infants: Comparative study with cranial computed tomography; Journal of Tropical Paediatrics, June 1997; Volume 43: 182-183.