Review Article

Open Access

Cranial Ultrasound: A Novel Approach of Neuroimaging in Preterm Infants Suffering from Perinatal Birth Injury

Saba Fatima, Amber Goraya, Abid Ali Qureshi, Hina Azhar

ABSTRACT:

Introduction: Preterm birth is a common cause of neonatal mortality with an additional burden of adverse neurodevelopmental outcomes. It is caused by different factors that can be either perinatal, natal or postnatal leading to white matter injury/intracranial hemorrhages. These lesions can be readily assessed by cranial ultrasound which provides cost-effective, radiation-free, bedside imaging.

Conclusion: Cranial ultrasound is an innovative method to assess brain injury in preterm infants. Ultrasonographic evaluation of preterm brain is recommended as early as possible after birth with interval follow up. Three distinct patterns of brain injury can be seen in preterm infants: Periventricular leukomalacia (PVL), Germinal Matrix-Intraventricular hemorrhage (GMH-IVH) and cerebellar hemorrhages. Germinal matrix hemorrhage is found to be most common pattern with cystic PVL being next among three patterns of brain injury. Ultrasound is an operator-dependent technique with poor visualization of few abnormalities on two-dimensional images. The limitation of conventional ultrasonography opens up new aspects of 3 D scanning with better imaging outcomes.

Keywords: Germinal Matrix-Intraventricular hemorrhage, Preterm birth, Periventricular leukomalacia.

How to cite this Article:
Fatima S, Goraya A, Qureshi AA, Azhar H. Cranial Ultrasound: A Novel Approach of Neuroimaging in Preterm Infants Suffering from Perinatal Birth Injury. J Bahria Uni Med Dental Coll. 2023;13(1):61-66 DOI: https://doi.org/10.51985/JBUMDC202277

INTRODUCTION:

Preterm birth is defined as neonatal birth less than 37 weeks of gestation.[1] It is divided into extremely preterm (less than 28 weeks GA), very preterm (28-32 weeks GA) and late preterm birth (more than 32 weeks GA).[2] Epidemiologically it causes an annual birth rate of 11% globally with an estimated 15 million preterm births.[3] It is one of the major causes of childhood mortality and causes almost one million deaths each year and overall accounts for a risk factor in about 50% of neonatal deaths.[1] Economic burden caused by preterm birth was studied to be more than 2.9 billion pounds in terms of neonatal admissions, health care and educational assistance.[2]

Saba Fatima
Senior Registrar, Department of Pediatric Radiology
The Children's Hospital Lahore
Email: sabaazhar.23@gmail.com

Amber Goraya
Associate Professor, Department of Pediatric Radiology
The Children's Hospital Lahore
Email: amber.goraya@yahoo.com

Abid Ali Qureshi
Professor and Head, Department of Pediatric Radiology
The Children's Hospital Lahore
Email: abidaliqureshi67@gmail.com

Hina Azhar
Assistant Professor, Department of Developmental Pediatrics
The Children's Hospital Lahore
Email: hinaazhar2009@gmail.com

Received: 29-May-2022
Accepted: 14-Nov-2022

These premature neonates are prone to develop multiple problems because of immature organ systems like respiratory, gastrointestinal, cardiac and neurological complications. Premature brain is more prone to injury from multiple pre, peri and postnatal causes due to ischemia, infection or inflammation. The common maternal causes include maternal hypertension, maternal diabetes and anemia while the common fetal causes include birth asphyxia, chorioamnionitis, respiratory distress and sepsis.[4] Perinatal injury causes neurological damage by cumulative effects of hypoxic-ischemic, inflammatory and metabolic processes, which lead to immature auto regulation of cerebral vasculature to hypoxia and thus results in cellular damage.[5]

Damage to premature brain causes intellectual and psychiatric disabilities, like development of cerebral palsy in 15% and behavioral disturbances with impaired academic performance in 30 to 50% of patients.[6] Another study depicts that 10% of patients develop CP with hearing and visual problems while 40% develop cognitive defects, language and educational difficulties.[7] Because of this high incidence of neurodevelopmental problems, prompt and accurate neuroimaging is indicated in preterm neonates. There are multiple techniques for brain imaging with cranial ultrasound being the first-line modality for imaging of neonatal brain as it provides fast, radiation free, cost effective, bedside screening method of imaging. Cranial ultrasound also helps in serial imaging to look for progression of disease process.[8] Three dimensional ultrasound is a new technique of performing cranial imaging which helps in visualization of the brain parenchyma in multiple planes along with reduced scanning time.[9]

In this review article, we will discuss the basic physical aspects of ultrasound, technique of performing cranial ultrasound with normal anatomical description of neonatal brain and anatomic variants. Various patterns of brain injury in preterm neonates with their ultrasound manifestations will be discussed along with indications for further imaging evaluation

DISCUSSION:

Cranial ultrasound is an important tool that is used for the evaluation of normal and abnormal brain in preterm infants. As it is a bedside screening tool and does not require long sedation as compared to MRI, it is being increasingly used for neuroimaging in neonatal intensive care units.[8]

Technique of cranial ultrasound:

High-frequency sound waves are used to produce images in cranial ultrasound.[10] Sector scanning is the most common mode of imaging in cranial ultrasonography[11] however an article published in AJR recommends the use of linear array transducer.[11] An acoustic window is needed to transmit sound waves from the scan head and the most commonly used window is anterior fontanelle.[12,13] Scanning is performed with a scan head/transducer of 7.5 Hz frequency in sagittal and coronal planes with scanning from frontal to occipital regions and from right to left side of the head.[12] Studies recommend taking 6-8 coronal views with 5 sagittal views.[13,14] Supplemental acoustic windows have also been described to aid in the visualization of the preterm brain like posterior and mastoid fontanelles.[15] Posterior and mastoid fontanelles allow accurate visualization of occipital lobes, atria of ventricles and infra-tentorial structures.[16]

There are many recommendations on timings of cranial ultrasound in preterm infants for early recognition of any brain injury but controversies exist in these recommendations. Perlman et al. gave a scanning protocol based on the weights of preterm infants described in Table 1.

In 2001, the American Academy of Neurology reviewed this protocol and recommended routine cranial ultrasound in preterm infants of <30 weeks gestational age at between 7 and 14 days after birth with repetition of scan between 36 and 40 weeks postmenstrual age.[18] Another study devised scanning protocol based on the gestational age of infants as described in Table 2.

Table 1. Timing protocol for cranial ultrasound based on birth weight (17)

<1000 grams	1000-1250 grams	1250 and 1500 grams
Days 3-5	Days 3-5	Days 3-5
Days 10-14	Around day 28	Pre discharge
Around day 28	Pre discharge	Pre discharge

Table 2. Timing protocol for cranial Ultrasound based on gestational age[19]

Gestational age < 32 weeks	Gestational age >32 weeks
Shortly after birth	Third day of life
Third day of life	Weekly thereafter until discharge
Seventh-day of life	
Weekly thereafter until discharge	

Normal anatomy and anatomical variants on cranial ultrasound:

In the coronal plane, most anterior section passes through frontal horns of lateral ventricles and it reveals frontal lobes separated by falx cerebri and fluid-filled cavum septum pellucidum which is a normal structure in preterm infants. Corpus callosum is also seen at this level above the frontal horns. Posterior to the frontal horns, there are septum pellucidum, frontal horns indented by the head of caudate nuclei which are separated from the lentiform nucleus by internal capsules with Sylvian fissures and temporal lobes more laterally. Vascular anatomy at this plane includes the bifurcation of internal carotid arteries into anterior and middle cerebral arteries. Moving posteriorly, there are bodies of lateral ventricles, thalami, tentorium and brainstem with the lateral part being occupied by basal ganglia, internal capsule and deep white matter of parietal lobes. Further posterior plane of imaging includes trigone and atria of lateral ventricles filled by choroid plexus, cerebellar vermis, quadrigeminal cistern and splenium of the corpus callosum. The most posterior image reveals occipital lobes and optic pathway. In the sagittal plane, midline imaging reveals pulsatile branches of anterior cerebral artery coursing over the cavum septum pellucidum, inferior to which lies the third ventricle, the aqueduct of Sylvius and 4th ventricle. All components of the brainstem with cerebellar vermis can also be visualized at this level. Parasagittal images reveal lateral ventricles with thalami lying at their floor and brain parenchyma of frontal and occipital lobes. The most parasagittal images reveal occipital and temporal horns of lateral ventricles with temporal lobes seen bilaterally. Echogenic choroid plexus is seen filling the ventricles in all sections.[20]

Various anatomic variants are commonly found due to improvement in technique and technology of ultrasound and these variants are important to know to avoid misinterpretation as pathologies.[21] Persistent fetal fluid spaces are commonly found and to name as added cavum septum pellucidum, cavum verge and cavum interpositum.[21] Most of these spaces are close by birth in full-term infants while 85% of cavum septum pellucidum closes by 3-6 months of age.[22] These spaces are differentiated from their anatomical location, space anterior to the foramen of Monro and between frontal horns is cavum septum pellucidum while one posterior to foramen of Monro and fornices is cavum verge.[23] Smaller choroid plexus cysts less than 1 cm are commonly found in a neonatal autopsy but are insignificant[20] while multiple, bilateral and larger cysts have significance due to association with chromosomal abnormalities.[24] Lenticulostriate vasculopathy is a nonspecific finding which can both be a normal variant and pathological with later due to thickening of walls of arteries secondary to TORCH infections, chromosomal abnormalities and metabolic disorders.[25]

Patterns of brain injury in preterm neonates and their ultrasound features:

Various patterns of acquired perinatal brain injury have been described in preterm neonates with three types of structural abnormalities including white matter injuries, intracranial hemorrhages and cerebellar injuries.[26] White matter injury is the most common of brain injury and according to a study, it encompasses periventricular leukomalacia, punctuate white matter injury, intraparenchymal hemorrhage, ischemia and ventriculomegaly with volume loss.[27] While a particular terminology has been devised by Joseph Volpe in 2009 to cover the neuropathological spectrum of preterm infants called "Encephalopathy of Prematurity". This entity consists of periventricular leukomalacia (PVL) and severe germinal matrix-intraventricular hemorrhage (GMH-IVH) with its complications.[28] Out of these defined patterns, PVL with its associated neuronal/axonal diseases is the most common pattern found in 50 %or more of preterm infants while GMH-IVH is only found in 5 % of infants born prematurely.[29] Another important component of preterm brain insult is cerebellar injury which accounts for 19% of the cases as proved in a study.[30]

1. Periventricular leukomalacia: Periventricular leukomalacia (PVL) relates to the injury to cerebral white matter in preterm infants. It consists of two components: 1) a focal necrotic component which is further of two types and 2) a diffuse gliotic component which is due to astrogliosis and microgliosis. Macroscopic necrotic components in combination with gliotic changes evolve into cystic lesions hence referred to as cystic PVL while microscopic necrotic foci with surrounding diffuse gliosis change into glial scars and known as non-cystic PVL.[28,29] The overall incidence was proved to be 26.4%[31] and 16.3 %[32] in two different studies. The former study was based on the preterm African population with the role of genetic and environmental factors leading to higher incidence and hence making it a less reliable study than later.

Cystic PVL is easily seen by cranial ultrasound however it is present in only 3% of preterm infants[33] while the EPIPAGE study demonstrates it in 5% of infants.[34] The later EPIPAGE study is a better one as it includes a large sample size with more accurate results. Non-cystic PVL is difficult to assess on ultrasound however its incidence is reportedly high as compared to cystic PVL. Cystic PVL progresses from areas of in homogeneously increased echogenicities on ultrasound to evolve into complete cystic lesions.[35] These commonly occur along the distribution of long penetrating arteries particularly in watershed areas alongside trigone of lateral ventricles. They appear as multiple small variable-sized cysts having anechoic fluid.[36] Generally, cystic PVL is categorized by four grades described in table 3.

Table 3. Ultrasound grading for Cystic PVL[35]

GRADE	ULTRASOUND APPEARANCE
I	Increased echogenicity areas in the periventricular region
II	Increased echogenicities evolving into small frontoparietal cysts
III	Periventricular frontoparietal and occipital lobe cysts
IV	Extensive subcortical cysts

Non-cystic PVL also called as diffuse PVL is difficult to image on ultrasound however it is found in the majority of cases as proved to be 69%[33] and 80%[37] in two different studies. It is difficult to assess on ultrasound however its incidence is reportedly high as compared to cystic PVL. Miller et al concluded that non-cystic PVL is present in 69% of patients[33] while Leijser et al in their study proved it to be present in 80% of infants.[37] However both these studies had various limitations, like small and limited sample size with timings of performing ultrasound not following any established protocols.

It can be seen as periventricular echogenicities with no progression into cystic lesions.[38] However several studies demonstrate that diffuse white matter injury can progress into brain atrophy which manifests in the form of enlarged ventricles and subarachnoid spaces, widening of Sylvian fissure with reduced gyral folding. Brain atrophic changes had been described in particular in 18% of patients by Horsch et al[39] while Skiold et al proposed these changes to be found in 4.8% patients.[40] The work done by later is more reliable as it provides a quantitative scoring system for different features of brain atrophy.

Generally, the diagnostic accuracy of ultrasound for PVL is variable as proposed to be 68 % by Debellion et al[41] and 90% by Trounce et al[42]. The later study appears to be more appropriate as it compared the ultrasound findings with histopathological sampling at autopsy while in the former study MRI brain was used as a reference standard tool.

2. Germinal matrix – intraventricular hemorrhage and its complications:

Germinal matrix hemorrhage is one of the common abnormalities in the preterm brain which are diagnosed on ultrasound. Germinal matrix itself consists of proliferated neurons and glial cells having vessels of single-cell origin. It starts involution by the end of second trimester and only a small amount remains till 32 weeks in the region of caudothalamic groove. By term age almost complete involution occurs causing decreased risk of hemorrhage in term infants. Germinal matrix hemorrhage has been divided into 4 different grades by Papile et al described in table 4.

Table 4. Grading for Germinal Matrix Hemorrhage[43]

Grades	Description
Grade I	Subependymal hemorrhage
Grade II	Intraventricular hemorrhage without dilatation
Grade III	Intraventricular hemorrhage with dilatation
Grade IV	Intraventricular hemorrhage with parenchymal extension

The overall incidence of hemorrhage was proved to be 44.6 % and 64.4 % in two different studies. These studies also documented individual incidences of different grades which were 52.4 and 40% for grade I, 30.9 and 11 % for grade II, 11.9 and 25.7 % for grade III, 4.7 and 2.8% for grade IV.[44,45] The ultrasound picture of grade I hemorrhage is echogenicities noted in caudothalamic groove on sagittal scans and inferolateral to frontal horns on coronal scans. Grade II hemorrhage is seen as echogenicities filling the part or whole of ventricles while in grade III there is associated ventricular dilatation. Grade IV is ventricular hemorrhage associated with adjacent infarction.[46]

Cumulative incidence of germinal matrix-intraventricular hemorrhage was proved to be 44.6 %[44] and 64.4 %[45] in two different cohort studies which also mentioned individual incidences of different grades with grade I being most common and grade IV being least common in both studies. However the former study appears to be better due to its large sample size and description of the number of hemorrhages with relation to the gestational age of born infants. Ultrasound appearance is of echogenic material in caudothalamic groove and within the ventricular system according to different grades.

3. Cerebellar Haemorrhages:

Cerebellar injury is one of the frequent findings in neonates born prematurely. Germinal matrix present along the fourth ventricle adjacent to the cerebellum can undergo injury with a similar pathogenic mechanism as that of the lateral ventricle germinal matrix. Conventional sonograms with anterior fontanelle are less accurate in the determination of cerebellar hemorrhage however mastoid fontanelle view allows optimal visualization and assessment of this pathology. Sonographically it is seen as echogenicity involving cerebellar hemispheres or vermis with abnormal size or shape of the fourth ventricle. The incidence of cerebellar hemorrhages on cranial sonograms is low as proved to be 9 %[47] and2.8 %[48] in two different studies. Limperopoulos et al in the former study used a big sample size for a case-control study making it a better incidence predictor than latter.

Disadvantages and future perspectives of cranial ultrasound:

Despite being highly sensitive for diagnosing most of the brain injuries in preterm infants, cranial ultrasound still has uncertainty concerning its use. The uncertainty is based upon two factors, one is inter-observer reliability and accuracy because it is an operator-dependent technique. Second, there are forms of brain injury not readily determined on ultrasound using conventional or even supplemental views, like non-cystic diffuse PVL, neuronal-axonal injuries and cerebellar hemorrhages as already described.[38]

Considering these disadvantages of conventional ultrasound, it is the need of hour to work on new aspects of three dimensional ultrasonography which will help in opening new horizons for prompt imaging of neural elements in preterm neonates.[49] It will allow the imaging of entire brain in single volumetric sweep with reconstruction of images in multiple planes thus helping in visualization of parenchymal changes not easily visible on 2 D ultrasound. It also helps in rapid bed side scanning along with storage of data for later manipulation in form of multi planar displays, volume calculations and tomographic images.[49] It was proved in a study performed in California that the scanning time with 3D ultrasound was less than conventional 2D imaging along with better imaging results with three dimensional ultrasound.[50]

Limitations:

The literature used for this article consisted of mainly review articles with few being randomized control trials and cohort studies. Further, the sample size for most of the studies was small and limited to a population of some specific area making them region biased studies and less accurate for epidemiological analysis. Another major limitation was the lack of any standardized quantification in most studies with the use of subjective ultrasound findings for describing various abnormalities which led to vivid interpretation of results.

CONCLUSION:

Cranial ultrasonography is a radiation free screening method for imaging of neonatal brain which helps in assessment of anatomical variants as well as major pathological imaging patterns. Considering inter observer accuracy difference and limitations of two dimensional imaging, 3-D ultrasound is a new aspect of prompt bed side neonatal brain imaging.

Authors Contribution:
Saba Fatima: Concerned with concept, data collection, analysis and article writing
Amber Goraya: Concerned with concept, data collection, analysis and article writing
Abid Ali Qureshi: Concerned with review of article
Hina Azhar: Concerned with review of article

REFERENCES:

1. Blencowe H, Cousens S, Chou D, Oestergaard M, Say L, Moller AB, et al. Born too soon: the global epidemiology of 15 million preterm births. Reproductive health 2013;10(1):1-4.
2. Boyle AK, Rinaldi SF, Norman JE, Stock SJ. Preterm birth: Inflammation, fetal injury and treatment strategies. Journal of reproductive immunology 2017; 119:62-6.

3. Torchin H, Ancel PY, Jarreau PH, Goffinet F. Epidemiology of preterm birth: Prevalence, recent trends, short-and long-term outcomes. Journal de gynecologie, obstetriqueetbiologie de la reproduction 2015; 44(8):723-31.
4. Anjari M, Counsell SJ, Srinivasan L, Allsop JM, Hajnal JV, Rutherford MA, et al. The association of lung disease with cerebral white matter abnormalities in preterm infants. Pediatrics 2009;124(1):268-76.
5. Novak CM, Ozen M, Burd I. Perinatal brain injury: mechanisms, prevention, and outcomes. Clinics in perinatology 2018; 45(2):357-75.
6. Neil JJ, Inder TE. Imaging perinatal brain injury in premature infants. Seminars in perinatology 2004; 28(6): 433-443
7. Cerisola A, Baltar F, Ferrán C, Turcatti E. Mechanisms of brain injury of the premature baby. Medicina 2019; 79:10-4.
8. Diwakar RK, Khurana O. Cranial sonography in preterm infants with short review of literature. Journal of pediatric neurosciences 2018; 13(2):141.
9. Stanojevic M, Hafner T, Kurjak A. Three-dimensional ultrasound-a useful imaging technique in the assessment of neonatal brain. Journal of perinatal medicine 2002; 30(1):74-83.
10. Accardo J, Kammann H, Hoon Jr AH. Neuroimaging in cerebral palsy. The Journal of pediatrics 2004; 145(2):19-27.
11. Blickman JG, Jaramillo D, Cleveland RH. Neonatal cranial ultrasonography. Current problems in diagnostic radiology 1991; 20(3):93-119.
12. Lowe LH, Bailey Z. State-of-the-art cranial sonography: Part 1, modern techniques and image interpretation. AJR 2011; 196(5):1028-33.
13. Meijler G, Steggerda SJ. Neonatal cranial ultrasonography. Springer; 2012 Jan 25.
14. Rumack CM, Wilson SR, Charboneau JW, Levine D. Diagnostic Ultrasound. St. Louis, MO. Elsevier Mosby, 2005, https://doi.org/ 10.7863/jum.2005.24.5.737
15. Anderson N, Allan R, Darlow B, Malpas T. Diagnosis of intraventricular hemorrhage in the newborn: value of sonography via the posterior fontanelle. AJR. 1994; 163(4):893-6.
16. Buckley KM, Taylor GA, Estroff JA, Barnewolt CE, Share JC, Paltiel HJ. Use of the mastoid fontanelle for improved sonographic visualization of the neonatal midbrain and posterior fossa. AJR. American journal of roentgenology 1997; 168(4):1021-5.
17. Perlman JM, Rollins N. Surveillance protocol for the detection of intracranial abnormalities in premature neonates. Archives of pediatrics & adolescent medicine 2000; 154(8):822-6.
18. Ment LR, Bada HS, Barnes P, Grant PE, Hirtz D, Papile LA, et al. Practice parameter: Neuroimaging of the neonate: Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. Neurology 2002; 58(12):1726-38.
19. Leijser LM, de Vries LS, Cowan FM. Using cerebral ultrasound effectively in the newborn infant. Early human development 2006; 82(12):827-35.
20. Gupta P, Sodhi KS, Saxena AK, Khandelwal N, Singhi P. Neonatal cranial sonography: a concise review for clinicians. Journal of pediatric neurosciences 2016; 11(1):7.
21. Lowe LH, Bailey Z. State-of-the-art cranial sonography: part 2, pitfalls and variants. American Journal of Roentgenology 2011; 196(5):1034-9.
22. Epelman M, Daneman A, Blaser SI, et al. Differential diagnosis of intracranial cystic lesions at head US: correlation: with CT and MR imaging. RadioGraphics 2006; 26:173–196.
23. Bronshtein M, Weiner Z. Prenatal diagnosis of dilated cava septipellucidietvergae: associated anomalies, differential diagnosis, and pregnancy outcome. Obstet Gynecol 1992; 80:838–842
24. Siegal M. Pediatric sonography, 53rd ed.Philadelphia, PA: Lippincott, Williams, and Wilkins, 2002.
25. Teele RL, Hernanz-Schulman M, Sotrel A. Echogenic vasculature in the basal ganglia of neonates: A sonographic sign of vasculopathy.Radiology 1988; 169:423-7.
26. Cerisola A, Baltar F, Ferrán C, Turcatti E. Mechanisms of brain injury of the premature baby. Medicina 2019; 79:10-4.
27. Mirmiran M, Barnes PD, Keller K, Constantinou JC, Fleisher BE, Hintz SR, et al. Neonatal brain magnetic resonance imaging before discharge is better than serial cranial ultrasound in predicting cerebral palsy in very low birth weight preterm infants. Pediatrics 2004; 114(4):992-8.
28. Volpe JJ. Brain injury in premature infants: a complex amalgam of destructive and developmental disturbances. The Lancet Neurology 2009; 8(1):110-24.
29. Volpe JJ. Neurology of the newborn. Vol. Philadephia: Saunders Elsevier. 2008.
30. Parodi A, Rossi A, Severino M, Morana G, Sannia A, Calevo MG, et al. Accuracy of ultrasound in assessing cerebellar haemorrhages in very low birthweight babies. Archives of Disease in Childhood-Fetal and Neonatal Edition 2015; 100(4):289-92.
31. Nzeh DA, Ajayi OA. Sonographic diagnosis of intracranial hemorrhage and periventricular leukomalacia in premature African neonates. European journal of radiology 1997; 26(1):77-82.
32. Fawer CL, Calame A, Perentes E, Anderegg A. Periventricular leukomalacia: a correlation study between real-time ultrasound and autopsy findings. Neuroradiology 1985; 27(4):292-300.
33. Miller SP, Cozzio CC, Goldstein RB, et al. Comparing the diagnosis of white matter injury in premature newborns with serial MR imaging and transfontanel ultrasonography findings. AJNR 2003; 24:1661–1669.
34. Larroque B, Marret S, Ancel PY, Arnaud C, Marpeau L, Supernant K, et al. White matter damage and intraventricular hemorrhage in very preterm infants: the EPIPAGE study. The Journal of pediatrics 2003; 143(4):477-83.
35. deVries LS, Eken P, Dubowitz LM. The spectrum of leukomalacia using cranial ultrasound. Behavioural brain research 1992; 49(1):1-6.
36. Inder TE, Volpe JJ. Mechanisms of perinatal brain injury. InSeminars in fetal and Neonatal medicine 2000; 5(1):3-16.
37. Leijser LM, de Bruïne FT, Steggerda SJ, van der Grond J, Walther FJ, van Wezel-Meijler G. Brain imaging findings in very preterm infants throughout the neonatal period: part I. Incidences and evolution of lesions, comparison between ultrasound and MRI. Early human development 2009 Feb 1; 85(2):101-9.

38. vanWezel-Meijler G, Steggerda SJ, Leijser LM. Cranial ultrasonography in neonates: role and limitations. InSeminars in perinatology 2010; 34(1): 28-38.

39. Horsch S, Muentjes C, Franz A, Roll C. Ultrasound diagnosis of brain atrophy is related to neurodevelopmental outcome in preterm infants. ActaPaediatrica 2005; 94(12):1815-21.

40. Skiöld B, Hallberg B, Vollmer B, Ådén U, Blennow M, Horsch S. A novel scoring system for term-equivalent-age cranial ultrasound in extremely preterm infants. Ultrasound in medicine & biology 2019; 45(3):786-94.

41. Debillon T, N'Guyen S, Muet A, Quere MP, Moussaly F, Roze JC. Limitations of ultrasonography for diagnosing white matter damage in preterm infants. Archives of Disease in Childhood-Fetal and Neonatal Edition 2003; 88(4):F275-9.

42. Trounce JQ, Fagan D, Levene MI. Intraventricular haemorrhage and periventricular leucomalacia: ultrasound and autopsy correlation. Archives of disease in childhood 1986; 61(12):1203-7.

43. Papile LA, Burstein J, Burstein R, Koffler H. Incidence and evolution of subependymal and intraventricular hemorrhage: a study of infants with birth weights less than 1,500 gm. The Journal of pediatrics 1978; 92(4):529-34.

44. Kadri H, Mawla AA, Kazah J. The incidence, timing, and predisposing factors of germinal matrix and intraventricular hemorrhage (GMH/IVH) in preterm neonates. Child's Nervous System 2006; 22(9):1086-90.

45. Sajadian N, Fakhraei H, Jahadi R. Incidence of intraventricular hemorrhage and post hemorrhagic hydrocephalus in preterm infants. ActaMedica Iranica 2010; 48(4): 260-262.

46. Maller VV, Cohen HL. Neurosonography: Assessing the premature infant. Pediatric radiology 2017; 47(9):1031-45.

47. Steggerda SJ, Leijser LM, Wiggers-de Bruïne FT, van der Grond J, Walther FJ, van Wezel-Meijler G. Cerebellar injury in preterm infants: incidence and findings on US and MR images. Radiology 2009; 252(1):190-9.

48. Limperopoulos C, Benson CB, Bassan H, Disalvo DN, Kinnamon DD, Moore M, et al. Cerebellar hemorrhage in the preterm infant: ultrasonographic findings and risk factors. Pediatrics 2005; 116(3):717-24.

49. Kurian J, Sotardi S, Liszewski MC, Gomes WA, Hoffman T, Taragin BH. Three-dimensional ultrasound of the neonatal brain: technical approach and spectrum of disease. Pediatric radiology 2017;47(5):613-27. DOI 10.1007/s00247-016-3753-1

50. Salerno CC, Pretorius DH, Hilton SV, O'Boyle MK, Hull AD, James GM, et al. Three-dimensional ultrasonographic imaging of the neonatal brain in high-risk neonates: preliminary study. Journal of ultrasound in medicine 2000; 19(8):549-55. https://doi.org/10.7863/jum.2000.19.8.549