ISSN- O: 2458 - 868X, ISSN–P: 2458 – 8687
National Library of Medicine - ID: 101731606

International Journal of Medical Science and Innovative Research (IJMSIR)
IJMSIR : A Medical Publication Hub
Available Online at: www.ijmsir.com
Volume – 8, Issue – 3, June – 2023 , Page No. : 166 – 179

Role of magnetic resonance imaging inevaluation of developmental delay in pediatric patients

[1]Dr. Mrs. Lovely Kaushal, Professor & Head, Department of Radiodiagnosis, Gandhi Medical College, Bhopal, Madhya Pradesh, India.

[2]Dr. Swati Goyal, Associate Professor, Department of Radiodiagnosis, Gandhi Medical College, Bhopal, Madhya Pradesh, India.

[3]Dr. Anita Uikey, Junior Resident, Department of Radiodiagnosis, Gandhi Medical College, Bhopal, Madhya Pradesh, India.

[4]Dr. Sonal Gupta, Junior Resident, Department of Radiodiagnosis, Gandhi Medical College, Bhopal, Madhya Pradesh, India.

[5]Dr. Mustafa Javed, Junior Resident, Department of Radiodiagnosis, Gandhi Medical College, Bhopal, Madhya Pradesh, India.

Corresponding Author: Dr. Anita Uikey, Junior Resident, Department of Radiodiagnosis, Gandhi Medical College, Bhopal, Madhya Pradesh, India.

Citation this Article: Dr. Mrs. Lovely Kaushal, Dr. Swati Goyal, Dr. Anita Uikey, Dr. Sonal Gupta, Dr. Mustafa Javed, "Role of magnetic resonance imaging inevaluation of developmental delay in pediatric patients", IJMSIR- June - 2023, Vol – 8, Issue - 3, P. No. 166 – 179.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Background: In this cross-sectional study, we study the role of magnetic resonance imaging (MRI) as a non-invasive imaging modality for the evaluation of developmental delay in pediatric patients & to correlate MRI with clinical findings.

Methodology: This study was conducted as an observ ational study in the Department of Radiodiagnosis, GMC Bhopal, during the study period of 1 year on patients with developmental delay history. The study comprised 100 patients who underwent MRI Brain scan study. Magnetic Resonance Imaging (MRI) was done and clinical findings are being correlated with MRI findings

Results: Out of 100 participants, 91% were found to have abnormal MRI findings. Gross motor delay, fine motor delay, social/ adaptive delay, and language delay were observed in 85%, 74%, 78%, and 73% of study parti cipants respectively. On MRI, the majority (90%) of the participants reported white matter involvement while 61% of participants reported HIE in MRI. Association between gross motor, fine motor, social/adaptive & language delay with MRI results of the study participants was found to be statistically significant.

Conclusion: The developmental delay has multiple a etiologies, most of them which cannot be diagnosed without the use of neuroimaging for example the degree of perinatal hypoxic insult and structural brain ab normalities, the in-depth evaluation of the brain is being provided by MRI. MRI serves as an accurate and non-

invasive, non-ionizing imaging method for the evaluation of brain anatomy and pathology.

Keywords: MRI, Developmental delay, hypoxic-is chemic insult.

Introduction

Development is a complex and continuous process that begins in utero and progresses until maturity and begins from conception[1,2]. This whole process includes structural as well as functional stages of progress or growth. A person goes through a lot of changes from the beginning right from the foetal stage until a full-grown individual. When a child is not capable to perform certain activities at a certain stage of life, may be a concern and often is termedas Developmental Delay '(DD)[1]. DD is neither a disease nor a diagnosis but a symptom or clinical presentation[3-7]. It used to diagnosed when a child shows a significant lag (2 SD less than the mean in one domain or 1.5 standard deviations inany two domains) in the acquisition of age-appropriate developmental mile stones [8,9].

The relevant domains of development evaluated include gross motor function, fine motor function, speech/ language, personal/ social milestones, and cognition [8].

Global DD (GDD) is defined as under-achievement in two/more domains [5,8-11]. GDD might occur due to static/ progressive disorders in the CNS (central nervous system). Patients who are having these disorders, regression, stability, or disease progression can develop [12]. Whereas, specific DD (SDD) is defined as insufficient progress in a single domain [8,9].

Although comprehensive data for the Indian population is not available, studies from other countries have shown some developmental disability in about 15% of children, and according to WHO, the overall global burden of disease is estimated at approx. 5 percent of all children who are less than the age of 14 years display some developmental disability [8]. Many of them believed that GDD patients constitute nearly 5 to 10 percent of those presenting as outpatients at numerous medical centers [2,13]. Additionally, GDD alone has been reported in 1-3 percent of the pediatric population aged 5 years or younger than that. [8,9,14,15].

The rate of development differs from one child to another and depends on various factors [2,16]. During this whole process, genetic as well asenvironmental, and nutritional factors with chronic diseases can have adverse effects on developmental milestones in four domains that is fine &gross motor, social, and language skills [14,17]. Known various causes of DD are a perinatal hypoxic insult, metabolic defects, structural brain abnormalities & anomalies, toxins, infections, genetic syndromes, and endocrine, traumatic, and environmental factors [8,18]. These causes cannot be identified only based on physical examination or patient history [19].

The diagnosis meaningfully impedes the value of the life of the patient & its full participation in the life of the family, school, and community. In such cases it depends on the clinician 's ability to detect and diagnose the cause with a multimodality approach which always includes neuroimaging [2,3,20] To evaluate of a child with DD, early diagnosis and treatment helps in counselling parents regarding the outcome of their child & identifying any possible risk of recurrence [1].

The diagnosis of DD is not done immediately after birth but during infancy or early childhood. Many times the diagnosis is done only when the child enters the school [2,3,21].

The degree of DD is further categorized as mild (defined as functional age which is < 33 percent below the chronological age), moderate (defined as functional age 34- 66 per percent chronological age), and severe (fun ctional age >66 percent of chronological age) [7].

Patients with DD are required to give a detailed history and physical examination, chromosomal & metabolic studies, MRI is characteristically used only as a 2nd-line modality [8]. Diagnosis is important as it enables clinicians todefine various treatment plans and conduct surveillance for known complications as well as it is used to provide prognosis and condition-specific family support (in cluding family-planning choices). This ensures the best overall outcomes for child and their family. [7]

All cases of delayed milestones must undergo neuro imaging as suggested by the AAN (American Academy of Neurology) [1,22]. Neuroimaging provides significant information as proof of previous injuries and specific abnormalities that would indicate a group/specific disease[12]. Magnetic resonance imaging (MRI) is more favored, compared to CT [1].

Brain ultrasonography (USG) has a main role in the finding & management of neonatal disease in preterm as well as term infants. USG is widely known as a non-invasive, radiation-free, & reproducible procedure that can be also performed at the bedside, in the intensive care unit, and on the intubated ventilated baby. But it has some limitations of being operator dependent and is not used once the fontanelles close.[2,22]

CT is performed in the infant when USG does not satisfy the clinical question or when an acoustic window is not available. CT also aids in detecting acute or subacute processes such as intracranial hemorrhage, cerebral edema, hypoxic - ischemic injury, infarction, hydro cephalus / shunt dysfunction, neo plasm, or abnormal collections. But it has the major drawback of using harm ful radiation.[2,23]

The diagnostic modality of choice for the evaluation of the brain at all ages is Magnetic resonance imaging (MRI). MRI provides great anatomical detail and has high sensitivity and specificity in detecting brain patho logic findings. [2]Therefore, it is the preferred imaging modality of choice for the infant or child with DD. [2] MRI aids in visualizing any structural abnormalities and a etio logical causes of DD [1]. It can differentiate myelinated from unmyelinated white matter in infant [2]. The ab normal neuro radiological results were seen in 60–65% of cases and the most common aetiology was traumatic/ neuro vascular aetiology. The most shared cause is hypoxic-ischemic injury whereas the most common structural abnormality is at the ventricular level. The second most crucial structure involved in the DD is the corpus callosum (hypoplasia and agenesis). [1]

Careful evaluation and investigation can reveal a cause in 55-85% of cases with DD.

Materials & methods

The present study was carried out for a period from August 2021 to September 2022. The study was per formed at Gandhi Medical College, & associated Hospital (Hamidia Hospital) Bhopal, which acts as a referral center catering to the needs of people from different strata of society, predominantly the lower and lower middle class. A total of 100 participants were enrolled in the study. It is a Facility based cross-sectional study. Children aged more than 6 months and less than 10 years clinically presented with developmental referred from the department of pediatrics are included in this study. Children with a history of acute encephalopathy and children with neuro-regression are excluded. All the patients first underwent MRI after taking written MRI Consent from legal guardians. After obtaining informed consent, and explaining the purpose of the study to the participants/legal guardians, data collection was done and information was recorded on a predesigned, pretested, and semi-structured proforma. The proforma included socio-demographic variables such as age, gender, contact number, address, developmental delay

history, etc. All patients underwent an MRI scan with a 1.5 Tesla machine by using a Brain coil and various required sequences is taken & images are obtained.

MRI Protocols used are

Various MRI sequences used in the study are T2 axial, T2 coronal, T1 sagittal, FLAIR axial, T2*GRE, DWI & ADC Map, T1 axial, T2 sagittal.

Figure 1: T2 axial planning.

Data was entered and analysis done using Epi info version. The MRI observations were compared with various parameters of DD.

Statistical analysis

Data was entered into MS Excel 2007, and analysis was done with the help of Epi info Version 7.2.2.2. Frequency and percentages were calculated. Quantit ative variables were expressed as the mean and standard deviation. Categorical data was expressed as a per centage. Microsoft Office was used to prepare the graphs. Chi-square/ Fischer 's exact test was applied for comparison. P <0.05 was taken as statically significant.

Results

The present study entitled–Role of magnetic resonance imaging in Evaluation of Developmental Delay in pediatric patients was carried out for a period of one year. A total of 100 participants were enrolled in the study. Out of 100 participants, 91 had anomalous MRI findings. The majority (78%) of the study participants were aged less than two years. 12% of participants were between 2 to 5 years of age while 10% were aged more than 5 years. The mean of study participants was found to be 24.44 $\pm$ 28.2 months. 53% of participants were males while 47% were females. 85% of the participants reported delayed development of gross motor milestones, 74% of the participants reported delayed develop ment of fine motor milestones, 78% of the study parti cipants reported delayed development of social and adaptive milestones and 73% of the study participants reported delayed development of language milestones.

Table 1: Age distribution of children.

Sn.	Variables	Frequency	Percent
1	> 6 months to 2 years	78	78.0
2	> 2 to 5 years	12	12.0
3	> 5 years	10	10.0
	Total	100	100.0

Table 2: Distribution of participants based on Gender

Sn.	Variables	Frequency	Percent
1	Male	53	53.0
2	Female	47	47.0
	Total	100	100.0

Figure 2: Distribution of participants based on various developmental milestones.

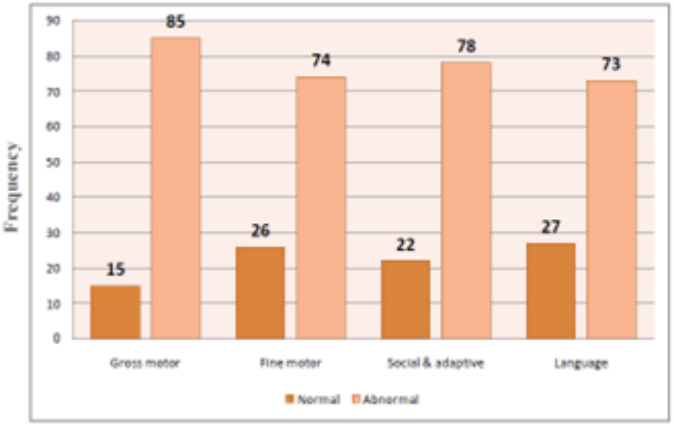


Table 3: Distribution of participants based on Brain structures (Structural morphology) involved in MRI

Sn.	Structure involvement	YES		NO	
		No.	Percent	No.	Percent
1	Ventricle	52	52.0	48	48.0
2	White matter	90	90.0	10	10.0
3	Grey matter	6	6.0	94	94.0
4	Corpus Callosum	3	3.0	97	97.0
5	Basal Ganglia	8	8.0	92	92.0
6	Brain Stem	4	4.0	96	96.0
7	Cranial Vault	2	2.0	98	98.0

Figure 3: Distribution of white matter region involvement

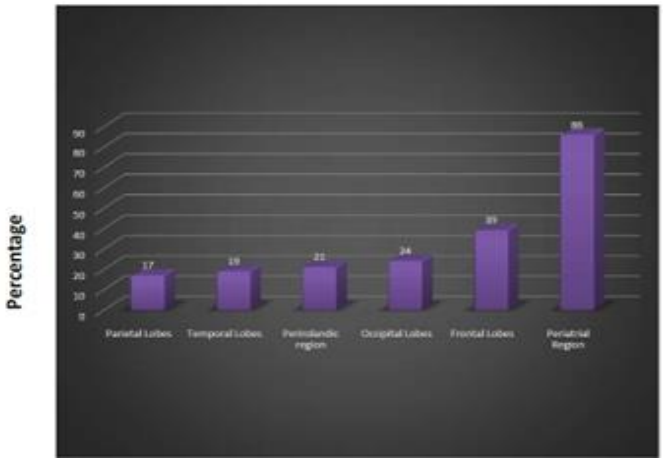


Figure 4: Distribution of study participants based on MRI findings.

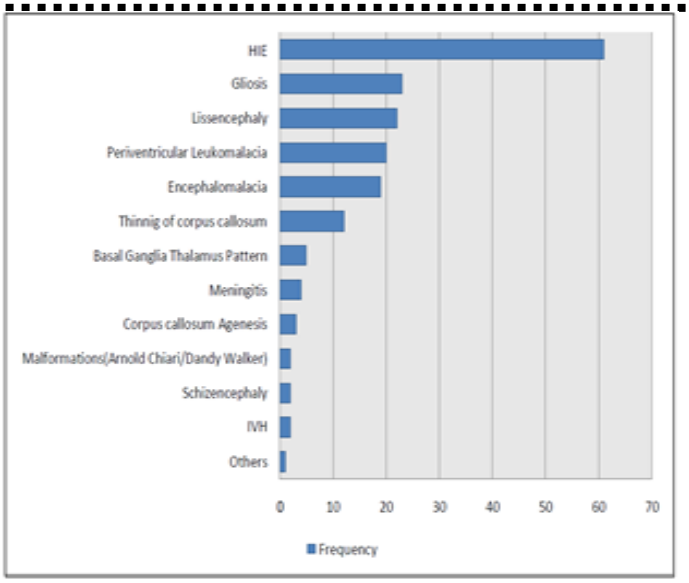
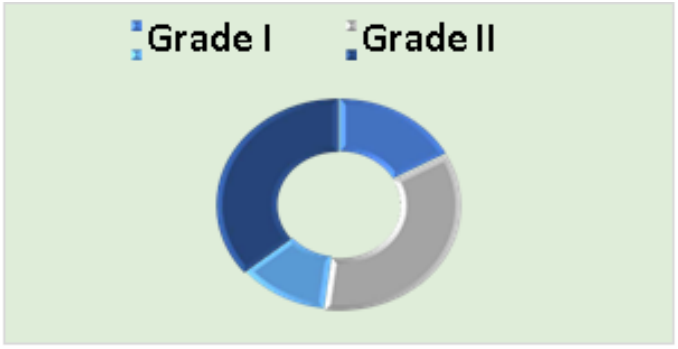


Figure 4 displays the distribution of study participants based on MRI findings. 61% of participants reported HIE in MRI. Gliosis, lissencephaly, periventricular leuko malacia, encephalomalacia, and thinning of the corpus callosum were observed in 23%, 22%, 20%, 19%, and 12% as MRI findings, respectively. Basal ganglia, meningitis, and corpus callosum agenesis were involved in 5%, 4% and 3% of findings respectively. IVH, schizencephaly, and malformations were involved in 2% of findings each. 1% of each of the study participants reported hydranencephaly, heterotopias, early subacute bleeding bleed, Colpocephaly, Thrombus sagittal sinus, and Trigonocephaly.

Figure 5: Distribution of study participants based on HIE grading.



Among 64 participants reporting hypoxia as etiology,

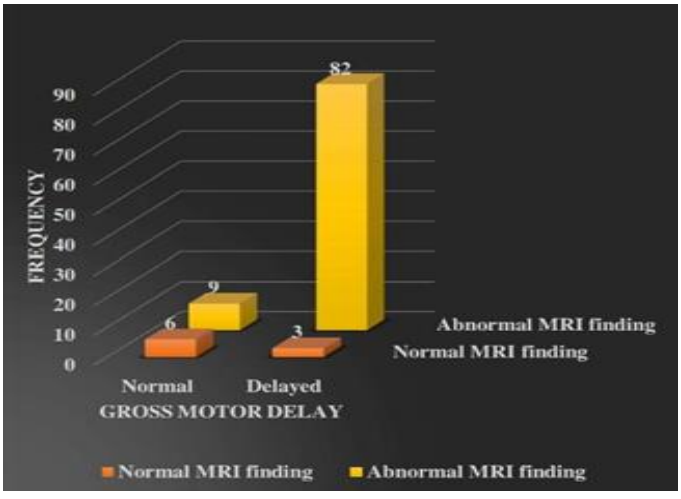
35% reported moderate hypoxia, 17% reported mild hypoxia and the rest 12% reported severe hypoxia.

Table 4: Association between Gross motor delay with MRI findings among study participants

			MRI Findings		Total
			Normal	Abnormal	
Gross Motor	Normal	Count	6	9	15
		% within MRI Findings	66.7%	9.9%	15.0%
	Delayed	Count	3	82	85
		% within MRI Findings	33.3%	90.1%	85.0%
Total		Count	9	91	100
		% within MRI Findings	100.0%	100.0%	100.0%

Above table displays that association between gross motor delay with MRI findings of the study participants was found to be highly significant with p<0.001. 90.1% of study participants with abnormal findings on MRI presented with gross motor delay.

Figure 6: Comparison of MRI findings with Gross motor DD



Above table displays that association between gross motor delay with MRI findings.

Table 5: Association between Fine motor delay with MRI findings among study participants

			MRI Findings		Total
			Normal	Abnormal	
Fine Motor	Normal	Count	7	19	26
		% within MRI Findings	77.8%	20.9%	26.0%
	Delayed	Count	2	72	74
		% within MRI Findings	22.2%	79.1%	74.0%
Total		Count	9	91	100
		% within MRI Findings	100.0%	100.0%	100.0%

Above table displays that association between fine motor delay with MRI results of the study participants was found to be statistically significant (p=0.001). 79.1% study participants with atypical results on MRI presented fine motor delay.

Figure 7: Comparison of MRI findings with Fine motor DD

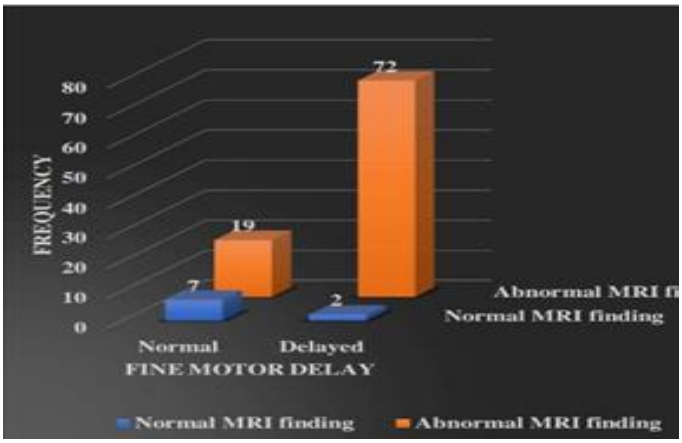
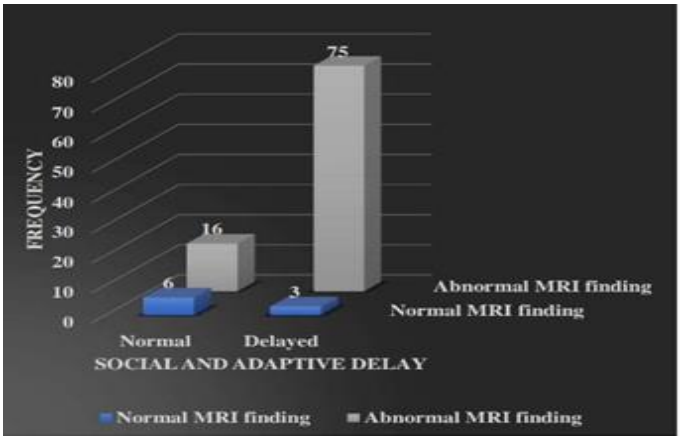


Table 6: Association between Social Adaptive delay with MRI findings among study participants

			MRI Findings		Total
			Normal	Abnormal	
Social and Adaptive	Normal	Count	6	16	22
		% within MRI Findings	66.7%	17.6%	22.0%
	Delayed	Count	3	75	78
		% within MRI Findings	33.3%	82.4%	78.0%
Total		Count	9	91	100
		% within MRI Findings	100.0%	100.0%	100.0%

Figure 8: Comparison of MRI findings with Social and Adaptive DD

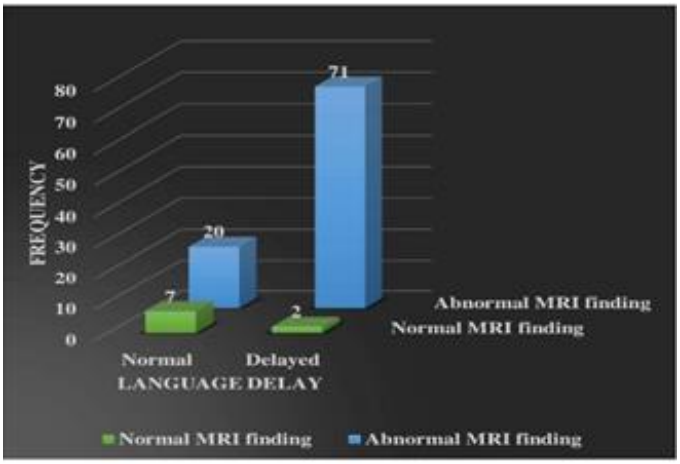


Above table displays that association between social & adaptive delay with MRI results of study participants was found to be statistically significant with p=0.003.82.4% study participants with atypical results on MRI presented social and adaptive delay.

Table 7: Association between Language delay with MRI findings among study participants.

			MRI Findings		Total
			Normal	Abnormal	
Language	Normal	Count	7	20	27
		% within MRI Findings	77.8%	22.0%	27.0%
	Delayed	Count	2	71	73
		% within MRI Findings	22.2%	78.0%	73.0%
Total		Count	9	91	100
		% within MRI Findings	100.0%	100.0%	100.0%

Figure 9: Comparison of MRI findings with Language DD



Above table displays that association between language delay with MRI results of the study participants was found to be statistically significant (p=0.001). 78.0 % study participants with atypical results on MRI presented language delay.

Discussion

The present study named ―Role of magnetic resonance imaging in the Evaluation of Developmental Delay in pediatric patients was carried out over a period of one year. Comprehensive information on MRI findings brain in DD is not available for the pediatric Indian population [24,25,26], and therefore, the research was conducted to study its role to evaluate developmental delay and correlate MRI with clinical findings. And also to determine the efficacy of MRI in planning the management of children with development delay. A total of 100 participants were enrolled in the study. This was similar to study by Randhawa et al (2022)[8], which was a cross-sectional study and joined 60 pediatric patients (3 months to 12 years).

In our study mean age of study participants was found to be 24.44 ± 28.2 months. Majority (78%) of study participants were < than 2 years. 12% of participants were between to 2 to 5 years of age while 10% were aged more than 5 years. Similar result was shown by Habibullah H et al (2020)[17] where many of the children with abnormalities in MRI brain imaging were in the age group between 2–5 years (29%) and with the next peak at 8–12 years (25.8%). Alamri A et al (2020)[27], the mean age of the study group at the time of the first clinical evaluation was 37.9 months (±32.5 standard deviation). Hafiz H et al (2019)[28], the majority presented between 3 months–12 years of age. KAUR T et al (2018)[2], 27.14% of patients were < one year whereas the majority (60%) were aged 1 to 5 years. Ali AS et al (2015)[29], the majority (56.7%) of participants were aged 2 years and below.

In our study, 53% of participants were males while 47% were females. Similar results of male preponderance have been detected in various studies: Randhawa et al (2022) [8]- 55%, Chaudhary K et al in 2021- 54.8 %, Habibullah H et al (2020)[17]- 58.0%), Alamri A et al (2020)[27]- 53%, Hafiz H et al (2019)[28]- 58%, KAUR T et al (2018)[2]- 68.5%, Palve R et al(2016)[3]- 60%, Ali AS et. al (2015)[29]- 56.8% were male and female AA etal (2011) [14] - 57.4%. Although, Elanchezhian D et al (2020) [12], in their study, found 52.4% female and 47.6% male participants.

In our study gross motor delay, fine motor delay, social/ adaptive delay, and language delay were observed in 85 %, 74%, 78%, and 73% of study participants res pectively. Similar results were observed in a study by Chaudhary K et al in 2021[1] where there were 40.38 %, 30.77 %, 20.19 %, and 9.62 % patients with motor (fine and gross) delay, language/ speech delay, social/ emotional delay, and cognitive impairment respectively. Similarly, Alamri A et al (2020) [27] also reported motor delay in 56%, language delay in 6%, and social/ cognitive delay in 1% of the participants.

As per a recent study, on MRI, the majority (90%) of the participants reported white matter involvement which was followed by ventricular involvement (52%). 6%, 3%, 8%, 4%, and 2% of participants reported involvement of grey matter, corpus callosum, basal ganglia, brain stem, and cranial vault respectively. Similar outcomes were found in various studies.

Chaudhary K et al in 2021[1] reported 49 (47.12 %), 43 (41.35 %), 30 (28.85%), 11 (10.58 %), 8 (7.69 %), and 3 (2.88%) participants with atypical MRI findings in ventricles, white matter, corpus callosum, grey matter, brainstem, cerebellum respectively. Alamri A et al (2020) [27], reported corpus callosum dysgenesis (77/ 153, 50%) and incomplete inversion/ malrotation of the hippocampus (50/153, 33%). Thin/hypoplastic corpus callosum was a more common finding (60/77) than complete or partial agenesis (17/77). Hafiz H et al (2019)[28], Palve R et al[3] in 2016, and Ali AS et al

(2015)[29], also observed that white matter and ventricles and mainly the corpus callosum were the most commonly affected anatomical structures.

White matter regions involvement on MRI, in our study, were as follows: 86% of peri atrial region > frontal lobes (39%) > occipital lobes (24%) > peri Rolandic region (21%) > temporal lobes (19%) > parietal lobes (17%). Other MRI findings were as: 61% of partakers reported HIE in MRI. Gliosis, lissencephaly, periventricular leukomalacia, encephalomalacia, and thinning of the corpus callosum were observed in 23%, 22%, 20%, 19%, and 12% as MRI findings, respectively. Basal ganglia, meningitis, and corpus callosum agenesis were involved in 5%, 4%, and 3% of findings respectively. IVH, schizencephaly, and malformations were involved in 2% of findings each. 1% of each of the study partakers reported hydranencephaly, heterotopias, early subacute bleeding, Colpocephaly, Thrombus sagittal sinus, and Trigonocephaly. A study by Chaudhary K et al (2021)[1] also found similar results as 4 (6.35 %) patients each with aqueductal stenosis, tuberous sclerosis, Dandy-Walker malformation, and hydrocephalus with thinning of corpus callosum respectively. Isolated hydrocephalus and Chiari malformation were seen in 2 (3.17 %) patients each. Alexander disease, metachromatic leukodystrophy, wide open schizencephaly, porencephalic cyst, lissen cephaly pachygyria spectrum, polymicrogyria, hemime galoencephaly and agenesis of the cerebellum was observed in 1 (1.59 %) patient respectively. Elanchezhian D et al in 2020 [12] found terminal zone myelitis, heterotopia, pachygyria, and corpus callosal agenesis with Dandy-Walker malformation. White matter abnormalities including nonspecific changes, hypo myelination, demyelination/ leuko encephalopathy, peri ventricular leukomalacia, and volume loss, with non-specific WM changes being the most frequent (31/104; 30%), Alamri A et al (2020)[27]. Palve R et al (2016) [3] found that 13 cases had the following syndrome comp lexes: dandy walker malformation, hemisphere hypo plasia, dandy walker variant, holoprosencephaly, and open & closed-lip schizencephaly. Cerebral atrophy with encephalomalacia changes were also seen in 6 cases. Hypoplasia of the CC and ventriculomegaly were also seen in 10 cases. These were some studies with similar results. In our study, most (64%) of the participants reported hypoxia. This was followed by traumatic or neurovascular disease in 16%, congenital/ developmental etiology in 13%, and metabolic/ degenerative etiology in 6% of the participants. Among 64 participants reporting hypoxia as etiology, 35% reported moderate hypoxia, 17% reported mild hypoxia and the rest 12% reported severe hypoxia.

Similarly, Randhawa et al (2022) [8], hypoxic insult was observed in 36.7% of patients, and structural abnormality in the brain and metabolic/ white matter abnormality were observed in 20% and 5% of patients respectively. As per Chaudhary K et al (2021) [1,] there were 41(39.42%), 31 (29.81%), 24(23.08%), 6 (5.77%), and 2 (1.92%) patients in the etiological category of normal, traumatic/ neurovascular, congenital/ developmental, metabolic/ degenerative and nonspecific respectively. The distribution of patients based on diagnosis. There were 31 (49.20 %) patients with hypoxic-ischemic injury/ encephalopathy and 5 (7.94 %) with an isolated abnormality of the corpus callosum.

As per our study, the association between gross motor delay ($p<0.001$), fine motor delay ($p=0.001$), social/ adaptive delay (0.003), and language delay ($p=0.001$) with MRI results of the study participants was found to be statisticallysignificant. 90.1%, 79.1%, 82.4%, and 78% of study participants with atypical results on MRI presented with fine motor, gross motor, social & adaptive, and

language delay. Similarly, Bouhadiba et al (2000) [30]showed a study on 224 children with DD & he observed that in 109 cases (48.6%) with positive findings in brain MRI. Alamri A et al (2020)[27], correlation of the various clinical variables with the presence of MRI abnormalities revealed that children with mainly motor DD were more likely to have abnormal MRI findings 84/122 (69%,P= 0.03). KAUR T et al (2018)[2] also found that a statistically significant association was detected between DD (fine motor, gross motor, social and adaptive delay, and language delay with MRI findings with p<0.001. As per a current study, the association of clinical presentation with MRI results of the study participants was found to be statistically significant. 97.8% of study participants with atypical results on MRI presented clinically with signs and symptoms of DD. (p<0.001). this was dissimilar to the results by KAUR T et al (2018)[2] who reported that no association was seen between the occurrence of seizure as a clinical symptom and MRI findings. However, MRI imaging is an essential part of the comprehensive evaluation of children with DD. Many specific aetiological and pathophysiologic conditions that lead to DD can be detected easily. Evidence supports that early diagnosis & treatment of developmental disorders leads to improvement.

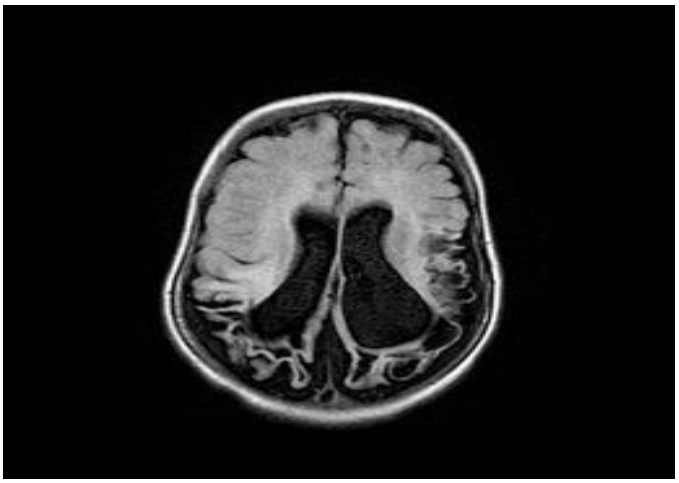


Figure 10: FLAIR axial Sequence in MRI showing multiple cystic lesions of CSF signal intensity with adjacent focal dilatation of ventricles likely Cystic Encephalomalacia

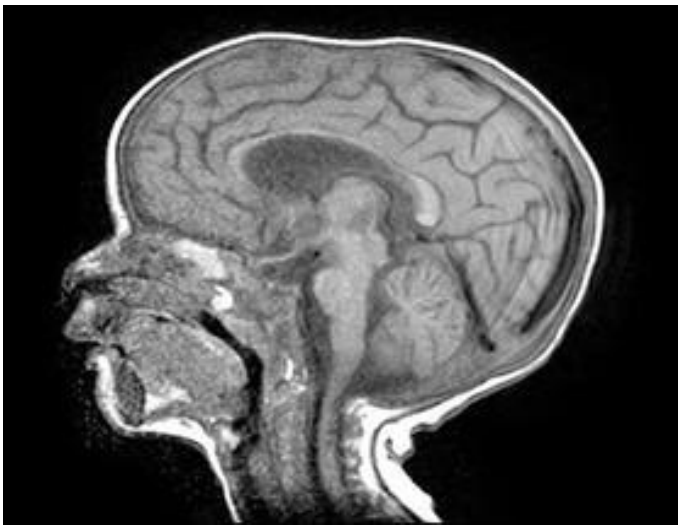


Figure 11: T1 SAG Sequence showing Thinning of corpus callosum predominantly noted in the body of corpus callosum

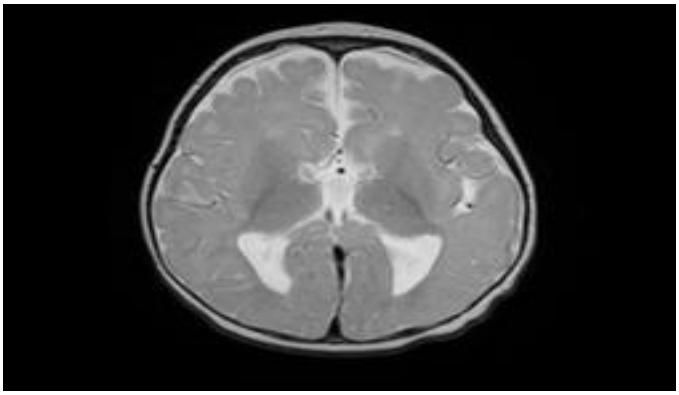


Figure 12: T2 AX Sequence showing Copocephaly

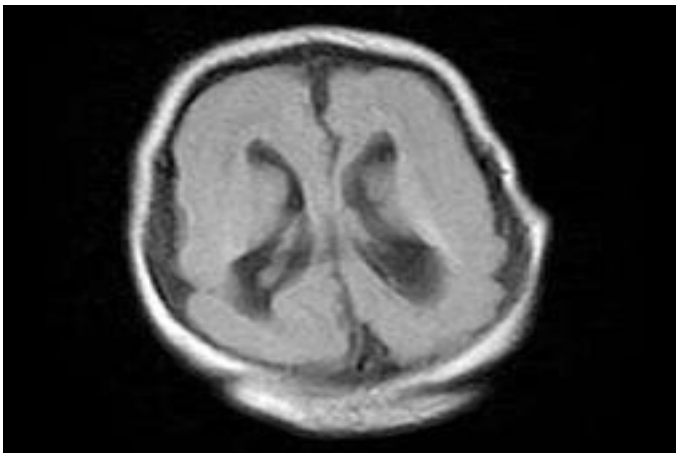


Figure 13: In a patient with history of DD, T2/FLAIR sequences shows abnormally broadened gyri with

reduced sulci giving relatively smooth appearance to the brain – suggestive of Lissencephaly.

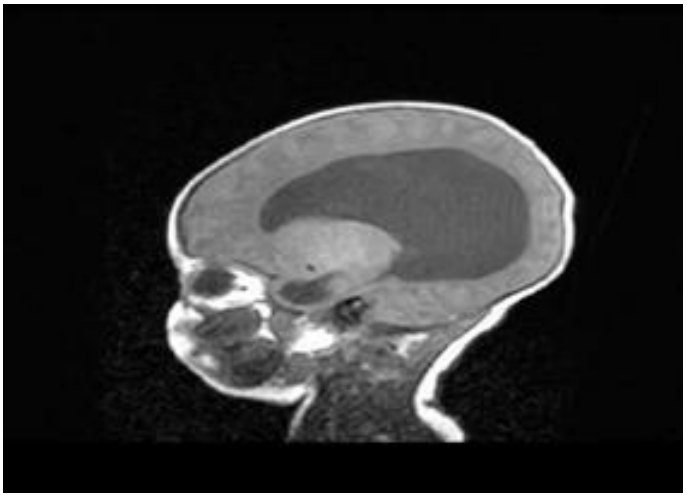
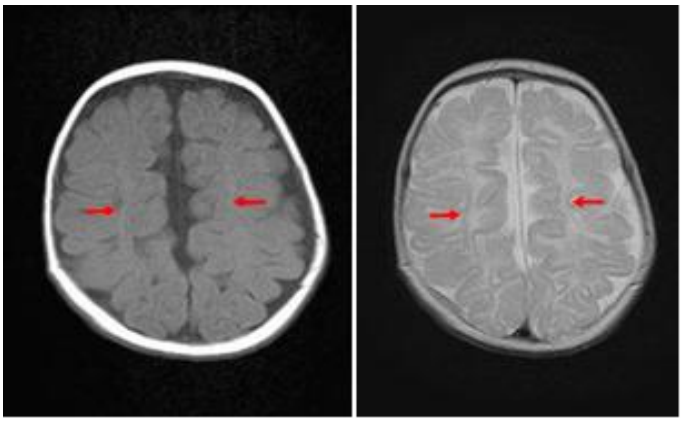


Figure 14: T1 SAG Sequence showing Agenesis of the corpus callosum



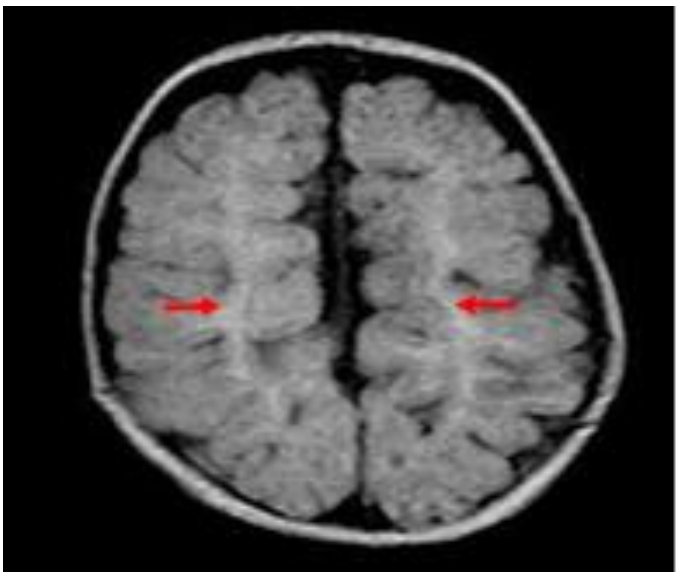
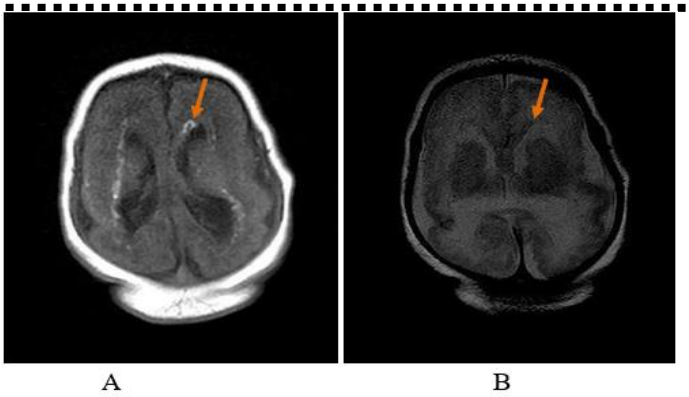
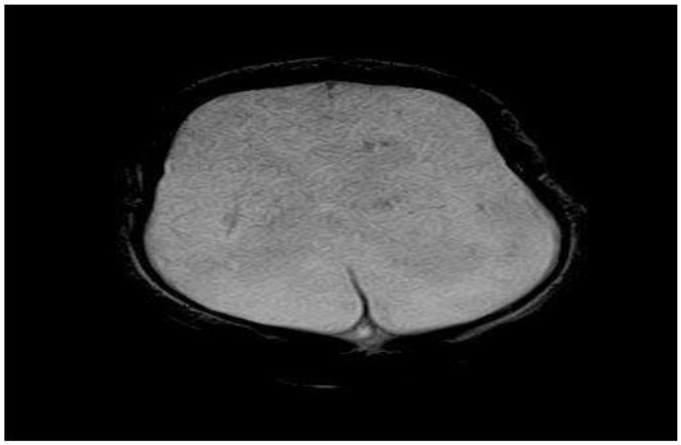


Figure 15: T1/T2/FLAIR Hyperintensity noted in bilateral centrumsemiovale in a preterm baby – HIE changes



A B



C

Figure 16: In a patient with history of DD with micro cephaly, T1WI (A) MR axial section shows hyper intensities, T2WI(A) axial section shows hypo intensities involving along ependymal lining of bilateral lateral ventricles (C) shows GRE blooming in the same region-representing calcifications. In view of history of micro cephaly – case of congenital cytomegalovirus (TORCH)

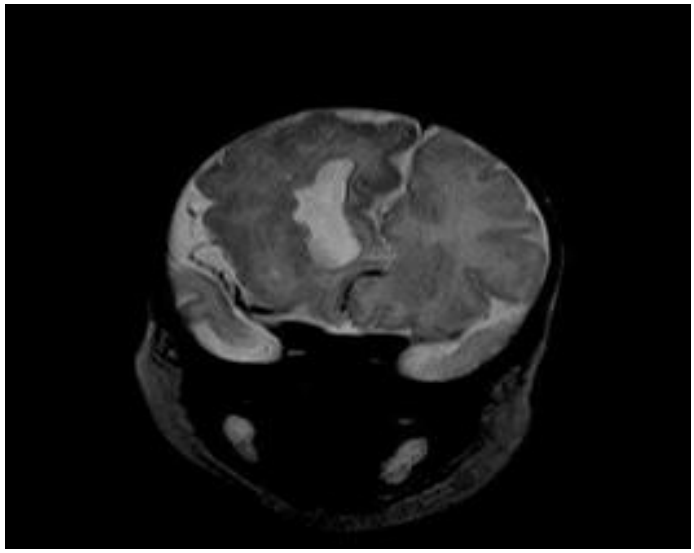


Figure 17: In a patient with a history of DD, T2 WI COR

shows asymmetrical dilatation of the right lateral ventricle with grey matter intensity in the periventricular region – a case of per ventricular heterotopia.

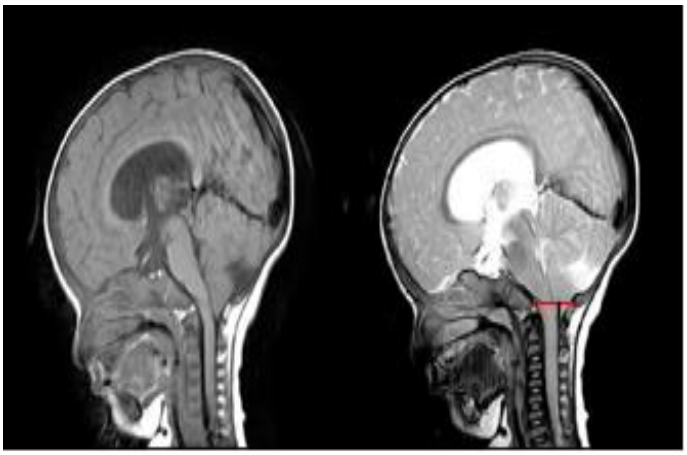
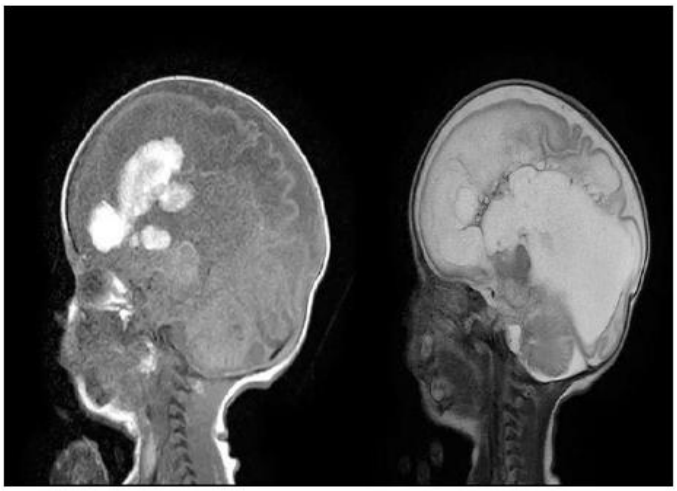
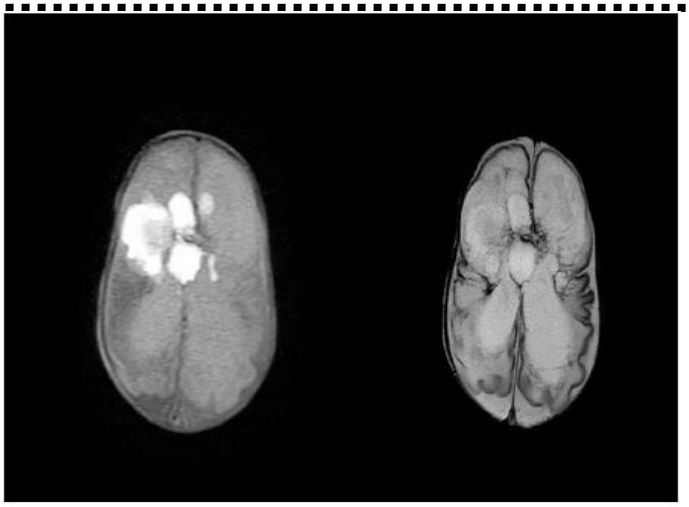


Figure 18: In a patient with history of DD, T1WI (A)&T2WI (A) sagittal section shows caudal descent of peg like tonsils through foramen magnum suggesting tonsillar herniation with hydrocephalus. Tonsil is >5mm below McRae line depicted as colour red – case of Arnold Chiari type I malformation.

(McRae line – A radiographic line drawn on mid sagittal section that connects anterior & posterior margins of foramen magnum (Basion to opisthion)



A B



C D

Figure 19: In a patient with history of DD, sag T1WI (A), sag T2WI (B) and T1WI (C) axial section shows hyper intensities in bilateral frontal lobes, subependymal white matter along bilateral lateral ventricles and in frontal horn of lateral ventricle with GRE blooming (B) – case of intra parenchymal bleed with intraventricular haemor rhage with agenesis of corpus callosum.

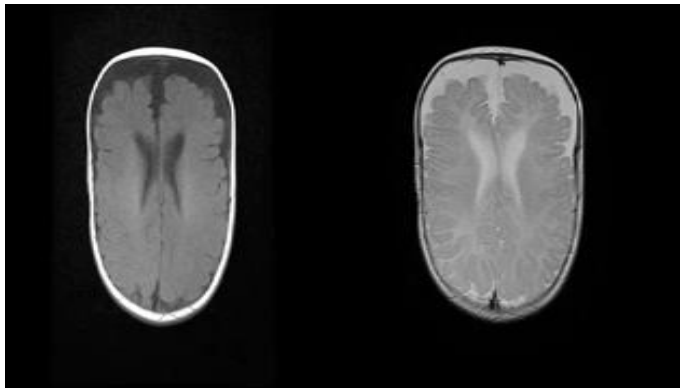


Figure 20: In a patient with a history of DD, T1WI (A) & T2WI (B) axial shows prominent subarachnoid spaces along bilateral frontal convexities in near symmetrical pattern with widened anterior inter-hemispheric CSF space & increased AP diameter– case of Benign enlarge ment of subarachnoid space in infancy with dolichol cephaly.

Conclusions

The DD has multiple a etiologies, most of them which cannot be diagnosed without the use of neuroimaging for

example the degree of perinatal hypoxic insult and structural brain abnormalities. In this study, MRI brain imaging will provide a high yield of findings that are abnormal and helps to analyse the relative prevalence of several general a etiologies in non-syndromic DD. The present study establishes a variety of morphological appearances of DD on MRI. The most commonly affected age group was less than 2 years. 91% of participants reported abnormal MRI. Gross motor delay, fine motor delay, social/ adaptive delay, and language delay were observed in 85%, 74%, 78%, and 73% of study participants respectively. White matter and ventri cular involvement were most commonly affected among all anatomical structures. The majority of participants reported hypoxia on MRI. As per our findings, there was a significant association between DD and MRI findings.

References

1. Chaudhary K, Kashikar SV, Dhande RP. MRI Assessment of Paediatric DD. Journal of Evolution of Medical and Dental Sciences. 2021 Feb 22;10(8):479-84.

2. KAUR T, SOOD D, Chauhan NS, SONI P. MRI evaluation of brain in Pediatric patients presenting with DD. European Congress of Radiology- ECR 2018.

3. Palve R, Verma P, Chandnani S, KOTHARI S, KUMAR S. MRI evaluation of brain in children with DD. International journal of contemporary medical research. 2016;3(12):3461-.

4. Brain magnetic resonance imaging findings in developmentally delayed children. Momen AA, Jelodar G, Dehdashti H, Int J Pediatr. 2011;2011:386984.

5. Diagnostic evaluation of DD/mental retardation: An overview. Battaglia A, Carey JC, Am J Med Genet C Semin Med Genet. 2003;117C:3-14.

6. Classification of DDs. Petersen MC, Kube DA, Palmer FB Semin Pediatr Neurol. 1998;5:2-14.

7. McDonald LA, Rennie AC. Investigating DD/ impair ment. Paediatrics andChild Health. 2011;21:443–47.

8. Randhawa HS, Bagale S, Umap R, Randhawa J. Brain Magnetic Resonance Imaging-Based Evaluation of Pediatric Patients With DD: A Cross-Sectional Study. Cureus. 2022 Apr 11;14(4).

9. Mithyantha R, Kneen R, McCann E, Gladstone M: Current evidence-based recommendations on investi gating children with global DD. Arch Dis Child. 2017, 102:1071-6. 10.1136/arch dis child-2016-311271

10. Rodriguez DP, Poussaint TY. Neuroimaging of the Child With DD. Top Magn Reson Imaging. 2007; 18(1):75-92.

11. Srour M, Mazer B, Shevell MI. Analysis of Clinical Features Predicting Etiologic Yield in the Assessment of Global DD. Pediatrics. 2006; 118(1):139-145.

12. Elanchezhian D, Kalaivani D. MRI findings in children with global DD. Int J Radiol Diagn Imaging [Internet]. 2020;3(2):07–8. Available from: http:// dx. doi. org/10.33545/26644436.2020.v3.i2a.89

13. Veyrac C, Couture A, Saguintaah M, Baud C. Brain ultra sono graphy in thepremature infant. Pediatr Radiol. 2006; 36(7):626-635.

14. Momen AA, Jelodar G, Dehdashti H: Brain magnetic resonance imaging findings in developmentally delayed children. Int J Pediatr. 2011, 2011:386984. 10.1155/2011/386984

15. Vitrikas K, Savard D, Bucaj M: DD: when and how to screen. Am Fam Physician. 2017, 96:36-43.

16. K. F. Swaiman, ‐General aspects of patient neuro logic history, in Pediatric Neurology, K. F. Swaiman and S. Ashwell, Eds., vol. 1, pp. 1– 14, Mosby, Philadelphia, Pa, USA, 4th edition, 2006.

17. Habibullah H, Albaradie R, Bashir S. MRI evaluation of global DD: a retrospective study. Dubai Medical Journal. 2020;3(1):1-4.

18. Belanger SA, Caron J: Evaluation of the child with

global DD and intellectual disability. Paediatr Child Health. 2018, 23:403-19. 10.1093/pch/pxy093

19. Elanchezhian E, Adaikappan M, Ramesh S, Rajathirajan S. MRI diagnosisin children with global DD. J Med Sci Clin Res. 2016;4:13446-8.

20. A guide to the investigation of children with DD in East Anglia. http:// www. phgfoundation. org/ documents / 85_11430359 70.pdf.AccessedNovember 15, 2017.

21. Shevell MI, Ashwal S, Donley D, et al. Practice parameter: evaluation of the child with global DD: report of the quality standards subcommittee of the American Academy of Neurology and the practice committee of the child neurology society. Neurology 2003;60(3):367-80 Williams HJ.

22. Veyrac C, Couture A, Saguintaah M, Baud C. Brain ultrasonography in the premature infant. Pediatr Radiol. 2006; 36(7):626-635.

23. Rodriguez DP, Poussaint TY. Neuroimaging of the Child With DD. TopMagn Reson Imaging. 2007; 18 (1): 75-92.

24. Kakkar C, Kakkar S, Saggar K, Goraya JS, Ahluwalia A, Arora A. Paediatric brainstem: A compre hensive review of pathologies on MR imaging. Insights into imaging. 2016 Aug;7(4):505-22.

25. Jallo GI, Biser-Rohrbaugh A, Freed D. Brainstem gliomas. Childs Nerv Syst. 2004;20:143–153. doi: 10. 1007/ s00381-003-0870-6.

26. Hegde AN, Mohan S, Lath N, Lim CC. The differential diagnosis for bilateral abnormalities of the basal ganglia and thalamus. Radio graphics. 2011;31:5–30. doi: 10.1148/rg.311105041.

27. Alamri A, Aljadhai YI, Alrashed A, Alfheed B, Abdelmoaty R, Alenazi S, Alhashim A, Benini R. Identifying clinical clues in children with global DD/intellectual disability with abnormal brain magnetic resonance imaging (MRI). Journal of Child Neurology. 2021 May;36(6):432-9.

28. Hafiz H, Raidah A, Bashir S. Magnetic resonance imaging (MRI) evaluation of global DD in Pediatric patients: A retrospective study. Journal of the Neuro logical Sciences. 2019 Oct 15;405:501.

29. Ali AS, Syed NP, Murthy GS, Nori M, Abkari A, Pooja BK,Venkateswarlu J. Magnetic resonance imaging (MRI) evaluation of DD in Pediatric patients. Journal of clinical and diagnostic research: JCDR. 2015 Jan; 9 (1): TC21.

30. S. AA, Syed NP, G.S.N. M, Nori M, Abkari A, B.K. P, J. V. Magnetic Resonance Imaging (MRI) Evaluation of Developmental Delay in Pediatric Patients. J Clin of Diagn Res.2015; 9(1): TC21-TC24.