International Journal of Endorsing
HEALTH SCIENCE RESEARCH

IJEHSR

Clinical Article

Incidental pathologies on magnetic resonance imaging in delayed milestones pediatric patients.

Ghazala Shahzad[1], Aashfa Hasan Shaikh[1], Vashdev Khimani[2], Peer Asad Aziz[3], Zeeshan Nasir[3] & Pir Abdul Ahad[4]

[1]Department of Radiology, Liaquat University of Medical & Health Sciences (LUMHS), Jamshoro-Pakistan.
[2]Department of Neurosurgery, Liaquat University of Medical & Health Sciences (LUMHS), Jamshoro-Pakistan.
[3]Liaquat University of Medical & Health Sciences (LUMHS), Jamshoro-Pakistan.
[4]Suleman Roshan Medical College, Hyderabad-Pakistan.

Doi: 10.29052/IJEHSR.v9.i1.2021.118-123
Corresponding Author Email: pirasadaziz@hotmail.com
Received 14/06/2020
Accepted 10/09/2020
First Published 01/03/2021

OPEN ACCESS

Abstract

Background: Delayed in achieving milestones is a rare but still devastating effect with a vast amount of etiologies resides in it. At least 89% of patients with development delay showed additional clinical features. One of the best techniques used to investigate such patients is Magnetic Resonance Imaging (MRI). MRI helps in the early diagnosis of children's delayed milestones that helps for parental counselling and identifies any recurrence risk in patients' siblings. The study aims to identify the incidental pathologies on MRI in patients with developmental delay.

Methodology: It is a cross-sectional study of MRI that includes 22 patients diagnosed with additional clinical features associated with developmental delay. All the patients enrolled in the study were recruited from Liaquat University Hospital Jamshoro and Hyderabad, developing decay for six months from January 2019 to June 2019. History and clinical examination with MRI of the included study participants were made on GE 1.5 Tesla with appropriate sequence after sedation. Various anatomical structures like ventricles, corpus Collosum etc., were further examined systematically.

Results: A total of 22 patients were enrolled in the study, including 11 male and 11 female. MRI findings suggest that most of the presentations, i.e. 45.5% in the study, were around 2-5 years. Furthermore, mostly founded incidental pathologies were atrophic brain (13.6%), enlarged arachnoid Space (13.6%) and Sinusitis (13.6%). Besides this, Canavan Disease and sigmoid sinus thrombosis were found together. MRI scans suggest that sinusitis usually involved mastoid antrum most often and, in some cases, it is found together with a subdural hematoma and mucosal enlargement.

Conclusion: In conclusion, MRI findings in delayed milestones patients show atrophic brain (13.6%), enlarged arachnoid spaces (13.6%) and sinusitis (13.6%). All these are not uncommon pathologies that can be the inductive factor for the pathogenesis of delayed milestone brains. This suggests that further cross-sectional studies are required to develop the findings associated with clinical features regarding incidental pathologies in developmental delay in pediatric patients.

Keywords

Magnetic Resonance Imaging (MRI), Incidental Pathologies, Developmental Delay.

Check for updates

Introduction

From conception to maturity, the human brain itself grows through a continuous development process, and it can get amended by genetic, environmental, nutritional and chronic diseases. This can result in the significant delayed achievement of Milestones, which can be evaluated by a motor that includes gross & fine, along with social and language skills. These skills can be labeled as development delay if one or more than one skill is delayed[1-4]. The estimation of 5-10% children, including 1-3% younger than 5 years and 40,000-120,000 children born each year out of four million annual births in the United States and Canada, reported to have development delay but still the exact prevalence is not known[5].

The evident revelation of development delay can occur during infancy, early childhood and early school years[1]. However, it does not represent the diagnosis, but it is the manifestation of various etiologies that includes genetic, metabolic, vascular, malformation syndrome, traumatic, infections, toxins and environmental. Besides this, the careful assessment can reveal the cause in around 55-85% of individuals with developmental delay[1,5-9]. High proportion with a wide attribute in patient's selection criteria are reported including, abnormalities in children with obvious clinical diagnosis[9].

Investigations on pediatric patients with developmental delay suggest that brain MRI is one of the major and important investigations in these individuals, and about 60-84% had an abnormal scan. These scans show that most findings are found in ventricles and corpus callosum[1,4,8,10]. However, there is no paper on incidental findings associated with development delay patients associated with MRI scans. Therefore, by pointing out these findings, further investigation can be done that can highlight certain factors associated with developmental delay. Our study aims to identify the incidental MRI finding in patients with a developmental delay that could further help assess certain factors associated with developmental delay.

Methodology

A Cross-sectional study designed for MRI of brain scans with 22 patients presenting with additional clinical features is associated with development delay in Liaquat University Hospital Jamshoro and Hyderabad; for six months (January to June 2019). The patients recruited were referred from the Pediatric and Neurosurgery ward to the radiology department for MRI. The history and clinical examination were made, and the patient's files were studied with a multi-disciplinary approach. A designed questionnaire was filled that included already set variables, which were adopted from the previous study[10].

Patients with an already known genetic disorder, metabolic disorder, protein-energy malnutrition, current acute or chronic infection and developmental delay with no additional features were excluded from the study. The patients, who clarify the exclusion criteria, went under MRI scans of the brain on the machine, GE 1.5 Tesla with sequenced used T1W, T2W, FLAIR, and diffusion-weighted DWI and ADC sequences. Strict sedation protocol was followed. For infants, the "feed and scan" technique was used. In contrast, for older children, oral or IV drugs including Syrup Chloral Hydrate 5 to 10 mg/kg were used with consent and under anesthetics and pediatrician supervision. All children were monitored with pulse oximetry, and continuous respiration was advocated during the entire scan, followed by strict vigilance for two hours after scan.

The patients were placed in the supine position, and immobilization of the head was achieved by surrounding the head with an air-evacuated bag of polystyrene balls. Systematically, the scans were taken by starting with ventricles, corpus callosum, grey and white matter, along with basal ganglia, brain stem and cerebellum. The discreet data was then spread on SPSS software version 23, and results were drafted out.

Results

A total of 22 patients were enrolled, with 11 males and 11 females in the present study. Most of the

presentations, i.e. 45.5%, were around the age of 2-5 years, while 95.5% delivered in term gestational age, 86.4% normal obstetric history and 54.5% have no history consanguinity. Complete demographic data are shown in table 1.

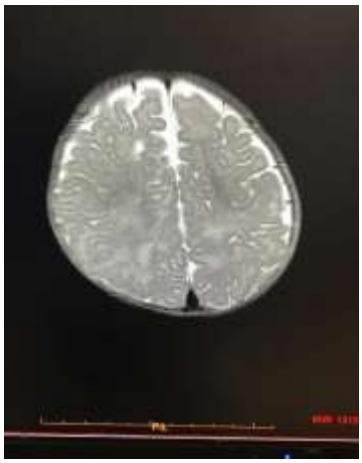
Table 1: Demographic data of patients included in the study.

Variable		n(%)
Age Groups	3 months-1 year	7(31.8)
	2-5 years	10(45.5)
	6-8 years	4(18.2)
	9-12 years	1(4.5)
Gender	Male	11(50)
	Female	11(50)
Gestational Age	Preterm	1(4.5)
	Term	21(95.5)
Obstetric history	Normal	19(86.4)
	Bad	3(13.6)
Consanguinity	Present	10(45.5)
	Absent	12(54.5)

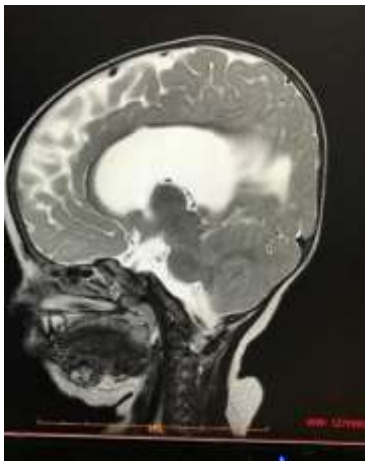
Additional Pathological findings that were found along with a predesigned questionnaire (Table 2) suggest that Canavan Disease and sigmoid sinus thrombosis were found together. At the same time, the dysplastic changes were either diffuse or focal. Furthermore, our results suggest that sinusitis usually involved mastoid antrum most often, and in some cases, it is found along with subdural hematoma and mucosal enlargement. Besides the Atrophic brain, the MRI scans show that enlarged subarachnoid space and sinusitis are common findings regarding others (Table 2 / Figure 1 & 2).

Table 2: Additional Radiological Findings.

MRI findings	n(%)
Atrophic brain	3(13.6)
Canavan Disease findings	1(4.5)
Sigmoid sinus thrombosis	1(4.5)
Chiari II malformation	1(4.5)
Congenital malformation	1(4.5)
Dandy-Walker malformation/variant	2(9.1)
Delayed myelination, demyelination	2(9.1)
Dysplastic changes	2(9.1)
Enlarged subarachnoid space	3(13.6)
Hypoxic Ischemic Encephalopathy	1(4.5)
Joubert Syndrome	1(4.5)
Sinusitis	3(13.6)
Periventricular leukomalacia	1(4.5)
Prominent Cisterna Megna	1(4.5)
Subdural hematoma	1(4.5)
Tonsillar herniation	1(4.5)

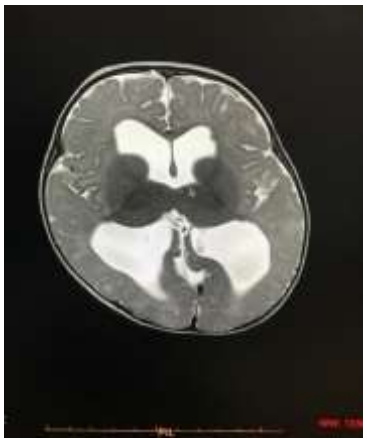


Axial TWI shows the diffuse symmetric hyperintense area in a white matter involving both cerebral hemispheres according to the patient's age (8 months) represents delayed myelination.

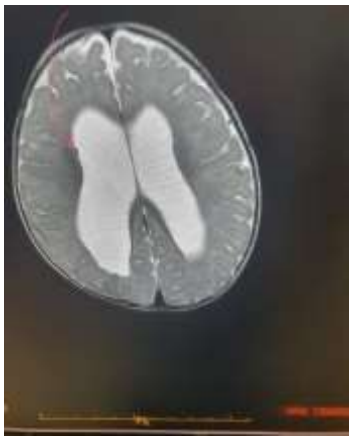


TWI mild sagittal shows diffuse thinning of genu and body of corpus callosum with complete splenium representing corpus callosal dysgenesis.

Figure 1: MRI Scans showing diffused areas of cerebral hemispheres and corpus callosum in patients with delayed myelination and dysgenesis.



Axial TWI shows fenestrated falx posteriorly with absent splenium.



TW axial image shows grey matter nodules in bilateral sup endymal location of lateral ventricles representing grey matter heterotopia.

Figure 2: MRI Scans showing fenestrated falx and sup endymal lateral ventricles location, representing absent splenium and grey matter heterotopia.

Discussion

Evaluation of 22 patients present with development delay or delay milestone achievement was observed in this study. The patients were referred from the pediatrics and neurosurgery ward to the radiology department for a Magnetic resonance scan. As cited by the previous study, all the patients with developmental delay ''development delay plus'' showed some MRI scan abnormality concerning the previous study, which showed only 89% finding on MRI scan[10]. It may be due to the difference in inclusion criteria of this study compared to the last study.

As all of the patients included in our study show some abnormal findings on MRI scan with most of the presentation around the age of 2-5 years (45.5%), (95.5%) delivered in term gestational with no gender prediction. This is the same finding in contrast to the previous finding that shows peak age of 3 to 12 months with male preponderance[1, 10].

Associated condition in this study including, brain atrophy, Canavan disease, Chiari II malformation, Congenital malformation, Dandy-Walker malformation, demyelination, Joubert Syndrome, Hypoxic Ischemic encephalopathy, Enlarge subarachnoid space with subdural hematoma or without and Sigmoid sinus thrombosis with sinusitis, all are historically proven association with developmental delay in literature without its etiological perspective[11-18]. Studies suggest that congenital and developmental anomalies have distinctive clinical and radiological findings. It is suggested that these identifications play a major role in preventing recurrence of development delay and counseling parents[19].

The study's major drawback is that it does not discuss clinical features and radiological findings associated with Delayed milestone patients. Besides it, the results of the study only highlight the associated pathology. It also does not indicate the co-presentation of two or more incidental pathologies in a single patient. This leads to further research and needs to be discussed in further Cross-Sectional studies in the future.

Conclusion

It is concluded that MRI findings in delayed milestones patients show atrophic brain (13.6%), enlarged arachnoid spaces (13.6%) and sinusitis (13.6%), which are not uncommon pathologies than others. This can be the inductive factor for the pathogenesis of delayed milestone brains. The results of the study do not discuss clinical features and radiological findings associated with Delayed milestone patients. Besides it, the results of the study only highlight the associated pathology. Therefore, further cross-sectional studies are required to be conducted that develop the findings associated with clinical features.

Conflicts of Interest

None.

Acknowledgement

The Author(s) wish to acknowledge Prof. Dr. Abdul Hameed from Fatima Jinnah Medical College/ Ganga Ram Hospital Lahore.

Funding

None.

Funding

1. Momen AA, Jelodar G, Dehdashti H. Brain magnetic resonance imaging findings in developmentally delayed children. Int. pediatrics. 2011; Article ID 386984.
2. Fenichel GM. Psychomotor retardation and regression. Clinical pediatric neurology: a signs and symptoms approach, 4th ed. Philadelphia: WB Saunders. 2001:117-147.
3. Majnemer A, Shevell MI. Diagnostic yield of the neurologic assessment of the developmentally delayed child. J pediatrics. 1995;127(2):193-199.
4. Battaglia A, Carey JC. Diagnostic evaluation of developmental delay/mental retardation: An overview. In American Journal of Medical Genetics Part C: Seminars in Medical Genetics 2003 Feb 15 (Vol. 117, No. 1, pp. 3-14). New York: Wiley Subscription Services, Inc., A Wiley Company.
5. Shevell MI, Ashwal S, Donley D, Flint J, Gingold M, Hirtz D, Majnemer A, Noetzel M, Sheth RD. Practice

parameter: evaluation of the child with global developmental delay: 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):367-380.

6. McDonald LA, Rennie AC. Investigating developmental delay/impairment. Paed Child Health. 2011;21(10):443-447.
7. Petersen MC, Kube DA, Palmer FB. Classification of developmental delays. In Seminars in pediatric neurology. United States: W.B. Saunders – Elsevier; 1998;5(1):2-14.
8. Koul R, Al-Yahmedy M, Al-Futaisi A. Evaluation children with global developmental delay: A prospective study at Sultan Qaboos University Hospital, Oman. Oman Med. J. 2012;27(4):310.
9. Widjaja E, Nilsson D, Blaser S, Raybaud C. White matter abnormalities in children with idiopathic developmental delay. Acta Radiologica. 2008;49(5):589-595.
10. Ali AS, Syed NP, Murthy GS, Nori M, Abkari A, Pooja BK, Venkateswarlu J. Magnetic resonance imaging (MRI) evaluation of developmental delay in pediatric patients. JCDR. 2015;9(1):TC21.
11. Esmaeeli Nieh S, Madou MR, Sirajuddin M, Fregeau B, McKnight D, Lexa K, Strober J, Spaeth C, Hallinan BE, Smaoui N, Pappas JG. De novo mutations in KIF1A cause progressive encephalopathy and brain atrophy. Ann Clin Translational Neurol. 2015;2(6):623-35.
12. Austin SJ, Connelly A, Gadian DG, Benton JS, Brett EM. Localized 1H NMR spectroscopy in Canavan's disease: a report of two cases. Magn Reson Med. 1991;19(2):439-445.
13. Brill CB, Gutierrez J, Mishkin MM. Chiari I malformation: association with seizures and developmental disabilities. J Child Neuro. 1997;12(2):101-106.
14. Chitayat D, Moore L, Del Bigio MR, MacGregor D, Ben-Zeev B, Hodgkinson K, Deck J, Stothers T, Ritchie S, Toi A. Familial Dandy-Walker malformation associated with macrocephaly, facial anomalies, developmental delay, and brain stem dysgenesis: Prenatal diagnosis and postnatal outcome in brothers. A new syndrome?. Am J Med Genetics. 1994;52(4):406-415.
15. Pindolia K, Chen J, Cardwell C, Cui X, Chopp M, Wolf B. Neurological deficits in mice with profound biotinidase deficiency are associated with demylination and axonal degeneration. Neurobiology Dis. 2012;47(3):428-435.
16. Nickel RE, Galtenstein JS. Developmental prognosis for infants with benign enlargement of the subarachnoid spaces. Developmental Medicine & Child Neurology. 1987;29(2):181-186.
17. Maria BL, Boltshauser E, Palmer SC, Tran TX. Clinical features and revised diagnostic criteria in Joubert syndrome. J Child Neuro. 1999;14(9):583-590.
18. Robertson C, Finer N. Term infants with hypoxic-ischemic encephalopathy: outcome at 3.5 years. DMCN. 1985;27(4):473-484.
19. Rivkin MJ. Developmental neuroimaging of children using magnetic resonance techniques. Ment Retard Dev Disabil Res Rev. 2000;6(1):68-80.