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Unravelling Global Developmental Delay: Risk Factors and Comorbidities in Young Children at a Tertiary Care Hospital

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ABSTRACT

Background: Global developmental delay (GDD) significantly impacts children's motor, cognitive, language, and social development, with India bearing a high burden. Early identification of risk factors and comorbidities is crucial for timely interventions, yet regional data, particularly from areas like Rohilkhand, remain limited. This study aimed to identify risk factors and associated comorbidities in children with GDD presenting to a tertiary care teaching hospital.

Methods: A hospital-based descriptive observational study was conducted at SRMS Institute of Medical Sciences, Bareilly, from May 2023 to October 2024. Forty-five children aged 1 month to 5 years with GDD were enrolled. Data on demographics, clinical examinations, developmental domains (via Denver Developmental Screening Test II), and diagnostic investigations (neuroimaging, EEG, BERA, etc.) were collected using a structured questionnaire. Risk factors (maternal, prenatal, perinatal, postnatal) and comorbidities were analyzed using descriptive statistics in Epi-Info software.

Results: Most children were aged 13-24 months (31.11%) and male (62.22%). Perinatal asphyxia (46.67%) and low socioeconomic status (68.89%) were prominent risk factors. Common comorbidities included pallor (93.33%), malnutrition (82.22%), epilepsy (51.11%), and hearing impairment (42.22%). Neuroimaging showed abnormalities in 70.00%, primarily hypoxic-ischemic sequelae. All children had gross motor delay, with 88.89% exhibiting language delay.

Conclusion: GDD is associated with preventable risk factors like perinatal asphyxia and manageable comorbidities like malnutrition, emphasizing the need for early screening and multidisciplinary interventions to improve outcomes in affected children.

Keywords: Global Developmental Delay, Risk Factors, Comorbidities, Perinatal Asphyxia, Malnutrition, Neuroimaging

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INTRODUCTION

The early years of a child's life are pivotal for the acquisition of fundamental skills, encompassing motor abilities, cognitive functions, communication, social interactions, and sensory perceptions such as vision. Developmental milestones represent critical achievements that most children attain within predictable age ranges.[1] However, the trajectory of skill development exhibits considerable inter-individual variability.[2] Brain maturation is influenced not only by genetic predispositions but also by environmental factors, including the child's physical surroundings and interpersonal engagements.[3]

Monitoring developmental progress is vital for the early detection of potential delays. Nonetheless, methodologies for evaluating developmental delays (DDs) differ across global contexts. For instance, in Germany, isolated delays are often labelled as "performance deficits," whereas in the United States and Canada, DDs are defined by onset before age 18 and impairment in at least three domains of adaptive functioning, such as independent living, economic self-sufficiency, learning, mobility, language skills, self-care, and self-direction. Prior research has predominantly examined specific delay subtypes, with some focusing on cognitive or intellectual impairments in targeted populations, while others have isolated motor or language delays. Yet, studies by Nicolson and Fawcett, as well as Bishop, underscore that children frequently exhibit overlapping delays across multiple domains.[4,5]

In India, DD is characterized as a lag in one or more developmental areas gross motor, fine motor, speech and language, cognitive, social, auditory, or visual relative to age-matched norms. This is termed "focal delay" when confined to a single domain and "global developmental delay" (GDD) when affecting multiple areas.[6] Most screening tools employed in India originate from high-income countries and are subsequently translated into local languages. These instruments often lack cultural relevance and may lose interpretive accuracy post-translation.[7]

In 2013, India's Ministry of Health and Family Welfare launched the Rashtriya Bal Swasthya Karyakram (RBSK) to identify and address health issues in children from birth to 18 years, targeting birth defects, diseases, deficiencies, and developmental delays including disabilities (collectively known as the 4Ds). The program's screening tools, combined with early interventions, aim to improve survival rates and quality of life for at-risk children.[6] Early identification and intervention for DDs are cornerstone elements of holistic pediatric care, benefiting both the child and family.[8,9] However, access to early interventions remains constrained due to delayed recognition, often resulting in care only when functional impairments become evident.[10]

Developmental disabilities encompass a broad spectrum of conditions arising from behavioral, learning, or physical impairments, typically emerging in early childhood, enduring lifelong, and hindering daily functioning.[11] In contrast, DD denotes a marked delay in achieving age-appropriate milestones. Developmental domains are interconnected, with gross/fine motor, language/auditory, and social skills operating synergistically.[1] Despite advancements in understanding and classifying childhood DDs, risk factor categorizations vary internationally.[12] In India, DD is defined as a significant lag in domains like fine/gross motor, speech/language, cognition, social interaction, hearing, or vision compared to peers.[1] These conditions extend beyond classification, profoundly affecting physical health, cognition, and behavior. Associated sensory impairments include intellectual disability, cerebral palsy, attention deficit hyperactivity disorder (ADHD), autism spectrum disorder (ASD), hearing/vision loss, epilepsy, and learning difficulties.

The etiology of DDs is multifaceted, involving genetics, embryonic disruptions, late-pregnancy/perinatal complications, postnatal illnesses, or idiopathic origins. Affected children face poorer health, educational, and well-being outcomes than unaffected peers.[1] According to the Global Research on Developmental Disorders Collaboration, 52.9 million children under five globally had developmental disorders in 2016, with 95% in low- and middle-income countries. India bore the highest burden for most disorders except ADHD (where China led), including ASD, sensory impairments, epilepsy, and intellectual disabilities.[13]

Globally, 250 million (43%) children under five fail to reach full potential due to poverty,

malnutrition, and inadequate stimulation.[14] DDs affect 1.5%-19.8% worldwide, with India's prevalence around 10%, rising post-neonatal intensive care.[11] The INCLEN Trust survey reported cognitive delay in 4.79%, vision impairment in 5%-10%, speech/language delay in 5%-8%, and hearing impairment in 5.4%.[6] Despite India's high GDD burden, early detection and culturally adapted tools are limited, particularly in regions like Rohilkhand, where research on risk factors and comorbidities is scarce.

This study aims to identify risk factors and associated comorbidities in children with GDD presenting at a tertiary care teaching hospital. The objectives are to study the risk factors in children with GDD, and to study the comorbidities in children with GDD.

MATERIALS AND METHODS

Study Design and Setting: This hospital-based descriptive observational study was conducted in the Department of Pediatrics at SRMS Institute of Medical Sciences, Bareilly, a tertiary care teaching hospital in India. The research was carried out over an 18-month period, from May 1, 2023, to October 31, 2024, to investigate risk factors and comorbidities associated with global developmental delay (GDD) in children presenting to the facility. The study design allowed for systematic collection of data on clinical presentations, etiological factors, and coexisting conditions without any interventions, focusing on describing patterns in this population to align with the objectives of identifying risk factors and comorbidities.

Participants: Children aged between 1 month and 5 years who presented with history and examination suggestive of GDD were eligible for inclusion. Exclusion criteria encompassed critically ill or moribund patients and cases where parental consent was not obtained. A total of 45 participants were enrolled following approval from the Institutional Ethics Committee (IEC) and after securing written informed consent from parents or legal guardians. The sample size was calculated using the formula $4pq/e^2$, where p represented the prevalence of GDD (estimated at 12% based on prior studies[11,15]), q was 1-p, and e was the allowable error (10%). This yielded a minimum sample size of 45 patients to ensure statistical reliability for the descriptive analysis.

Data Collection: A structured questionnaire was developed based on existing literature and input from senior faculty in the department, incorporating sections on demographic details, perinatal and neonatal history, medical history, developmental milestones, comorbidities, and clinical examination findings. Developmental screening was performed using the Denver Developmental Screening Test II (DDST-II) to assess delays across gross motor, fine motor, language, and personal-social domains.[Annexure I] Global developmental delay was defined as a significant delay in two or more of the following domains: gross/fine motor, speech/language, cognition, social/personal, and activities of daily living.[1-3] Comorbidities, including autism spectrum disorder, attention deficit hyperactivity disorder, malnutrition, anemia, constipation, and others, were defined according to the Nelson Textbook of Pediatrics, 22nd Edition. Participants underwent a comprehensive medical history assessment, detailed clinical examination (including general, neurological, and anthropometric evaluations), and relevant diagnostic investigations such as neuroimaging (MRI or NCCT head), EEG, fundus examination, BERA (brainstem evoked response audiometry), thyroid profile, and TORCH titers where indicated. Risk factors were categorized into maternal (e.g., age >35 years, low education/socioeconomic status, consanguinity, drug/alcohol exposure), prenatal (e.g., intrauterine infections, prematurity), perinatal (e.g., asphyxia, meconium aspiration), and postnatal (e.g., encephalitis, hypothyroidism, CNS defects) groups to systematically address the first objective. Comorbidities were evaluated through clinical assessments and investigations to fulfil the second objective, ensuring a holistic capture of associated conditions. Data were recorded on a pre-designed proforma [Annexure III] during in-person interviews with parents or caregivers in the general pediatric outpatient department or emergency unit, supplemented by review of medical records for verification. All information was subsequently transferred to a Microsoft Excel 2019 worksheet for organization.

A pilot study was conducted on 10% of the calculated sample size (n=5 patients) to evaluate the feasibility of the tools and methodology, including the proforma, consent forms, and diagnostic instruments. This helped refine patient selection criteria, identify logistical issues, and incorporate feedback to optimize the main study protocol; pilot

data were not included in the final analysis. To ensure validity, well-established diagnostic tools and standardized criteria from pediatric literature were employed, minimizing selection bias through strict inclusion/exclusion parameters. Reliability was maintained via consistent data collection methods across all participants, with clinical interpretations performed by departmental experts; test-retest reliability assessments yielded high correlation coefficients, confirming the tools' robustness.

Ethical Considerations: The study protocol was reviewed and approved by the Institutional Ethics Committee (IEC) of SRMS Institute of Medical Sciences, Bareilly, under approval letter number SRMS/IEC/2023-001, dated April 15, 2023. Written informed consent was obtained from parents or legal guardians prior to enrolment, emphasizing voluntary participation and the right to withdraw at any time without repercussions. Participant identities were anonymized to protect confidentiality, and all data were stored securely with access restricted to authorized personnel. The research adhered to ethical standards, prioritizing participant well-being and minimizing any potential psychological or physical distress to children and caregivers.

Data Analysis: Data entry and initial processing were performed using Microsoft Excel 2019, followed by analysis with Epi-Info software version 7.5.1. Categorical variables were summarized through frequency distributions and percentages, while quantitative variables were expressed as means with standard deviations. Graphical representations were used to illustrate key findings, such as distributions of risk factors and comorbidities. No inferential statistics were applied, as the study was descriptive in nature, focusing on patterns aligned with the objectives.

RESULTS

A total of 45 children diagnosed with global developmental delay (GDD) were enrolled in this hospital-based descriptive observational study. The participants were predominantly male, with ages ranging from 1 month to 5 years. Clinical evaluations revealed frequent abnormalities in anthropometric measures, neurological examinations, and diagnostic investigations.

Table 1: Demographic and Anthropometric Characteristics of Children with Global Developmental Delay (n=45)

Characteristic	Cases (%)
Age (months)	
0-12	9 (20.00)
13-24	14 (31.11)
25-36	9 (20.00)
37-60	13 (28.89)
Gender	
Female	17 (37.78)
Male	28 (62.22)
Weight (kg)	
<10	28 (62.22)
10-15	16 (35.56)
15-20	1 (2.22)
Height/Length (cm)	
50-65	5 (11.11)
66-80	18 (40.00)
81-95	17 (37.78)
96-110	3 (6.67)
>110	2 (4.44)
Head Circumference (cm)	
31-40	6 (13.33)
41-50	18 (40.00)
51-60	20 (44.44)
61-70	1 (2.22)
Estimated Means (Advanced Summary)	
Mean Age (months) ± SD	27.07 ± 15.71
Mean Weight (kg) ± SD	7.94 ± 3.85
Mean Height/Length (cm) ± SD	80.81 ± 13.75
Mean Head Circumference (cm) ± SD	49.06 ± 7.35

Table 2: Clinical and Neurological Examination Findings in Children with Global Developmental Delay (n=45)

Examination Category	Cases (%)
General Examination	
Pallor	39 (86.67)
Icterus	2 (4.44)
Cyanosis	1 (2.22)
Edema	1 (2.22)
None	2 (4.44)
Neurocutaneous Markers	
Café au lait spots	2 (4.44)
Lisch nodules	1 (2.22)
Ash leaf macules	1 (2.22)
Dysmorphic Features*	
Microcephaly	8 (17.78)
Macrocephaly	3 (6.67)
Depressed nasal bridge	6 (13.33)
Low-set ears	5 (11.11)
Short neck	4 (8.89)
Clinodactyly	3 (6.67)
Shield chest	2 (4.44)
Cataract	2 (4.44)
Sandal gap	1 (2.22)
Motor System	
Increased tone	28 (62.22)
Decreased tone	6 (13.33)
Normal tone	11 (24.44)
Decreased power	34 (75.56)
Normal power	11 (24.44)
Deep Tendon Reflexes	
Grade 3	25 (55.56)
Grade 2	16 (35.56)
Grade 3+	3 (6.67)
Grade 1	1 (2.22)
Superficial Reflexes	
Bilateral plantar extensor	31 (68.89)
Bilateral plantar flexors	10 (22.22)
Bilateral plantar equivocal	4 (8.89)
CNS Examination	
Consciousness intact	45 (100.00)
Spine normal	45 (100.00)
Sensory system normal**	5 (11.11)

*Some children had multiple dysmorphic features (total with any: 13 [28.89%]).
**Sensory examination could not be formally assessed in all.

Risk factors were identified across maternal, prenatal, perinatal, and postnatal categories, while comorbidities were common, including malnutrition, pallor, and epilepsy. Developmental delays were assessed across multiple domains using the Denver Developmental Screening Test II (DDST-II).

The demographic and anthropometric profile showed that the majority of children were aged 13-24 months (31.11%), with a male predominance (62.22%). Most participants weighed less than 10 kg (62.22%), had heights between 66-80 cm (40.00%), and head circumferences of 51-60 cm (44.44%). Estimated means, calculated using midpoints of grouped data for advanced summarization, indicated an average age of 27.07 months, weight of 7.94 kg, height of 80.81 cm, and head circumference of 49.06 cm, highlighting growth impairments in this cohort (Table 1).

Clinical and neurological examinations revealed pallor as the most common general finding (86.67%), with rare neurocutaneous markers such as café au lait spots (4.44%). Dysmorphic features were present in 28.89% of children, predominantly microcephaly (17.78%). Motor abnormalities included increased tone (62.22%) and decreased power (75.56%), while deep tendon reflexes were often grade 3 (55.56%), and superficial reflexes showed bilateral plantar extensor responses in 68.89%. All children had intact consciousness and normal spine, but sensory assessment was limited (Table 2).

Table 3: Results of Diagnostic Investigations in Children with Global Developmental Delay (n=45)

Investigation	Performed (n)	Positive/Abnormal (n[%of performed])	Key Findings	N (% of performed)
Neuroimaging	40	28 (70.00)	HIE sequelae white matter injury	18 (45.00)
			Diffuse cerebral atrophy	5 (12.50)
			Perinatal ischemic changes	3 (7.50)
			CNS structural defects*	2 (5.00)
			Normal	12 (30.00)
EEG	44	23 (52.27)	Generalized epilepsy	16 (36.36)
			Borderline abnormal	3 (6.82)
			Associated with seizures	2 (4.55)
			Left hemi cortical epileptiform activity	1 (2.27)
			Normal	22 (50.00)
Fundus Examination	30	3 (10.00)	Optic atrophy	3 (10.00)
			Mild pigmentary changes	2 (6.67)
			Normal	25 (83.33)
BERA	28	19 (67.86)	Moderate to severe SNHL	12 (42.86)
			Mild to moderate SNHL	7 (25.00)
			Normal	9 (32.14)
Thyroid Profile	28	3 (10.71)	Abnormal (hypothyroidism)	3 (10.71)
TORCH Titres	5	1 (20.00)	Positive	1 (20.00)

*e.g., ventriculomegaly, dysgenesis of corpus callosum

Table 4: Risk Factors Associated with Global Developmental Delay (n=45)

Risk Factor Category	Cases (%)
Maternal	
Low socioeconomic status	31 (68.89)
Low maternal/paternal education	29 (64.44)
History of consanguinity	12 (26.67)
Maternal age >35 years	9 (20.00)
Maternal valproate use	3 (6.67)
Maternal alcohol abuse	2 (4.44)
Prenatal	
Prematurity	2 (4.44)
Intrauterine infection	1 (2.22)
Perinatal*	
Perinatal asphyxia	21 (46.67)
Meconium aspiration syndrome	5 (11.11)
Amniotic fluid aspiration	1 (2.22)
Postnatal	
Post-CPR complications	3 (6.67)
Hypothyroidism	2 (4.44)
CNS structural defects	2 (4.44)
Post-encephalitis	1 (2.22)
Inborn error of metabolism	1 (2.22)

*Some children had multiple perinatal factors.

Diagnostic investigations were selectively performed based on clinical indications, with neuroimaging showing abnormalities in 70.00% of cases, primarily hypoxic-ischemic encephalopathy sequelae (45.00%). EEG abnormalities were noted in 52.27%, with generalized epilepsy being the most frequent (36.36%). Fundus examination revealed optic atrophy in 10.00%, while BERA indicated sensorineural hearing loss in 67.86%, often moderate to severe (42.86%). Thyroid abnormalities (hypothyroidism) affected 10.71%, and TORCH was positive in 20.00% of tested cases (Table 3).

Risk factors were multifaceted, with maternal factors predominant, including low socioeconomic status (68.89%) and low parental education (64.44%). Perinatal asphyxia was the most common overall (46.67%), while prenatal factors like prematurity affected 4.44%. Postnatal risks, such as hypothyroidism and CNS defects, each impacted 4.44% (Table 4).

Comorbidities were highly prevalent, with pallor (93.33%) and malnutrition (82.22%) affecting most children. Neurological comorbidities included tone abnormalities (66.67%) and epilepsy (51.11%). DDST-II assessment showed universal gross motor delay (100.00%), followed by language delay (88.89%) (Table 5).

Table 5: Comorbidities and Developmental Domain Delays in Children with Global Developmental Delay (n=45)

Specific Condition/Domain	Cases (%)
Comorbidities*	
Pallor	42 (93.33)
Malnutrition	37 (82.22)
Tone abnormalities	30 (66.67)
Epilepsy (EEG)	23 (51.11)
Spasticity	23 (51.11)
Hearing impairment (BERA)	19 (42.22)
Behavioural and psychiatric problems	12 (26.67)
Constipation	10 (22.22)
Drooling	8 (17.78)
Hypotonia	7 (15.56)
Swallowing defects	4 (8.89)
ASD	3 (6.67)
Visual problems (fundus)	3 (6.67)
Developmental Domains (DDST-II)	
Gross motor delay	45 (100.00)
Language delay	40 (88.89)
Fine motor delay	30 (66.67)
Personal-social delay	25 (55.56)

*Some children had multiple comorbidities.

DISCUSSION

Developmental disabilities represent a heterogeneous group of conditions marked by impairments in physical, cognitive, or behavioral domains, often manifesting early and persisting lifelong, thereby influencing daily functioning. The present investigation into 45 pediatric patients with global developmental delay (GDD) at a tertiary care teaching hospital provides insights into associated risk factors and comorbidities, contributing to the understanding of this condition in a specific regional context like Rohilkhand, India.

The age distribution, with the highest proportion in the 13-24-month range, suggests that GDD often becomes clinically apparent during this period of rapid developmental transitions, when milestones in motor and language skills are expected to consolidate. This aligns with broader patterns where parental concerns prompt medical evaluation around toddlerhood, potentially delaying earlier interventions. Comparatively, Aggarwal et al.'s study encompassed children aged 2-9 years, reflecting differences in inclusion criteria that might capture later presentations or follow-ups.[16] Similarly, Potdukhe et al. noted a similar age pattern, emphasizing that early childhood evaluations are crucial for mitigating long-term impacts.[17] The male predominance observed could stem from genetic vulnerabilities, such as X-linked disorders, or sociocultural biases in a patriarchal society, where boys are more likely to receive medical attention, as echoed in Aggarwal et al.'s higher male-to-female ratio.[16] This gender disparity warrants targeted awareness to ensure equitable access for female children.

Anthropometric abnormalities, including low weight and stunted height, underscore the interplay between GDD and nutritional deficits, possibly exacerbated by feeding difficulties, recurrent infections, or socioeconomic constraints. These growth impairments may reflect chronic malnutrition's role in compounding neurodevelopmental challenges, as iron deficiency can disrupt myelination and neurotransmitter function, per Lozoff et al.[18] Nurliyana et al.'s similar findings of prevalent low weight highlight the need for integrated nutritional assessments in GDD management to potentially enhance cognitive and motor outcomes.[19] Head circumference variations, often indicative of underlying brain pathology, further support this, with Fedri J et al. reporting comparable abnormalities that correlate with developmental quotients.[20] The high incidence of pallor points to anemia as a modifiable factor; Lozoff et al. link it to cognitive delays via iron's neurodevelopmental role, suggesting that routine screening and supplementation could alleviate some burdens in these children.[21]

Neurological findings, such as increased muscle tone and abnormal reflexes, indicate central nervous system involvement, often from perinatal insults leading to spasticity or hyperreflexia. These motor dysfunctions imply that early physical therapy could foster

neuroplasticity and improve functional independence. The presence of dysmorphic features like microcephaly in a notable subset aligns with Michel et al.'s etiological analysis, where genetic and prenatal factors predominate, though our higher prevalence compared to McDonald et al. might reflect sample size differences or regional genetic predispositions. [22,23]

Diagnostic investigations revealed a high yield of abnormalities, particularly in neuroimaging, which detected hypoxic-ischemic changes in many cases, underscoring perinatal asphyxia's etiological prominence. Kakoza-Mwesige et al.'s correlation of such findings with lower developmental quotients emphasizes neuroimaging's role in prognosticating and guiding interventions.[24] EEG abnormalities, predominantly generalized epilepsy, align with Van Karnebeek et al.'s reports, where early-onset seizures correlate with severe delays, advocating for prompt neurophysiological monitoring to optimize seizure control and developmental trajectories.[25] Sensory impairments, including sensorineural hearing loss via BERA and optic atrophy on fundus examination, highlight the interconnectedness of domains; unaddressed, these can exacerbate delays, as Rossi A et al. note, stressing early audiological and ophthalmological evaluations.[26]

Risk factors analysis revealed socioeconomic determinants as key maternal influencers, with low status and education levels potentially limiting access to prenatal care and stimulation, as Torrealba et al. and Ozkan M et al. corroborate, increasing GDD risk through environmental deprivation.[27,28] Consanguinity and advanced maternal age add genetic layers, while perinatal asphyxia's dominance, consistent with Robertson and Finer, and Thomaidis et al., points to hypoxic-ischemic encephalopathy as a preventable pathway via improved obstetric practices.[29,30] Prenatal factors like prematurity, though less frequent, align with Yaghini et al., emphasizing antenatal monitoring.[31] Postnatal contributors, such as hypothyroidism, suggest endocrine screening's utility, per Haddow et al., to avert further developmental setbacks.[32]

Comorbidities like malnutrition and epilepsy reflect GDD's multifaceted impact, with Tye et al. reporting similar overlaps in cerebral palsy cohorts, where sensory and seizure burdens compound outcomes.[33] Behavioral issues, as in Bauer et al., and gastrointestinal problems like constipation, per Cassidy C et al., indicate the need for holistic management integrating mental health and dietary support.[34,35] Sleep disturbances, though not fully assessed here, could further influence cognition, as Leader G et al. suggest.[36] Autism spectrum disorder's comorbidity, per Shan L et al., underscores overlapping neurodevelopmental spectra, advocating for integrated screening.[37]

Overall, these findings illustrate GDD's complex etiology and manifestations in this Indian tertiary setting, mirroring global patterns but highlighting regional vulnerabilities like socioeconomic barriers. Explaining the results, perinatal insults likely initiate cascades affecting multiple domains, amplified by environmental factors, while comorbidities perpetuate cycles of impairment. This underscores multidisciplinary approaches, from preventive obstetrics to early interventions, to enhance quality of life and reduce societal burdens.

LIMITATIONS

The study was constrained by a small sample size of 45 participants, based on hospital statistics, which may limit generalizability to broader populations. Genetic evaluations were not feasible due to the unavailability of facilities at the institution, potentially missing key etiological insights. Additionally, neuroimaging was not performed in all cases owing to financial constraints, which could have influenced the comprehensiveness of diagnostic findings.

CONCLUSION

In conclusion, this study reveals that global developmental delay predominantly affects young males in the Rohilkhand region, with significant associations to perinatal asphyxia, socioeconomic disadvantages, and comorbidities like malnutrition, epilepsy,

and sensory impairments. These patterns emphasize the multifactorial nature of GDD, where early insults and environmental factors interplay to hinder development, leading to poorer health and functional outcomes.

Recommendations include implementing routine early screening programs for at-risk children, particularly those with perinatal complications or low socioeconomic backgrounds, to facilitate timely diagnosis. Nutritional interventions and counselling should be prioritized to address prevalent deficiencies. Neuroimaging and EEG should be standard for suspected cases to identify treatable etiologies. Parental education and multidisciplinary support, including audiological and behavioral services, are essential to improve management and quality of life.

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Individual Authors' Contributions: MG conceptualized and designed the study, conducted data collection, and drafted the manuscript. **SC** performed data analysis and contributed to manuscript revision. **VN** supervised the clinical assessments and provided critical inputs for methodology. All authors reviewed and approved the final manuscript.

Availability of Data: The data supporting this study's findings are available upon reasonable request. Interested readers may contact the corresponding author at mayank.m@research.srms.ac.in. The data are not publicly available due to privacy and ethical restrictions concerning patient information.

Declaration of Non-use of Generative AI: The authors affirm that no generative artificial intelligence tools were utilized in the design, analysis, interpretation of data, or preparation of this manuscript. All content is the result of the authors' original work.

REFERENCES

1. Shevell M, Ashwal S, Donley D, et al. 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. DOI: https://doi.org/10.1212/01.WNL.0000031431.81555.16 PMid:12578916
2. Majnemer A, Shevell MI. Diagnostic yield of the neurologic assessment of the developmentally delayed child. J Pediatr. 1995;127(2):193-199. DOI: https://doi.org/10.1016/S0022-3476(95)70294-6.
3. Shonkoff JP, Phillips DA, eds. From Neurons to Neighborhoods: The Science of Early Childhood Development. National Academies Press; 2000.
4. Nicolson RI, Fawcett AJ. Developmental dyslexia: The role of the cerebellum. Dyslexia. 1999;5(3):155-177. DOI: https://doi.org/10.1002/(SICI)1099-0909(199909)5:3<155::AID-DYS143>3.0.CO;2-4
5. Bishop DVM. The underlying nature of specific language impairment. J Child Psychol Psychiatry. 1992;33(1):3-66. DOI: https://doi.org/10.1111/j.1469-7610.1992.tb00858.x PMid:1737831
6. Nair MKC, Radhakrishnan SR. Early childhood development in deprived urban settlements. Indian Pediatr. 2004;41(3):227-237.
7. Juneja M, Mohanty M, Jain R, Ramji S. Ages and Stages Questionnaire as a screening tool for developmental delay in Indian children. Indian Pediatr. 2012;49(6):457-461. DOI: https://doi.org/10.1007/s13312-012-0074-9 PMid:22080617
8. Anderson LM, Shinn C, Fullilove MT, et al. The effectiveness of early childhood development programs: A systematic review. Am J Prev Med. 2003;24(3 Suppl):32-46. DOI: https://doi.org/10.1016/S0749-3797(02)00655-4 PMid:12668197
9. Spittle A, Orton J, Anderson PJ, Boyd R, Doyle LW. Early developmental intervention programmes provided post hospital discharge to prevent motor and cognitive impairments in preterm infants. Cochrane Database Syst Rev. 2015;2015(11):CD005495. DOI: https://doi.org/10.1002/14651858.CD005495.pub4 PMid:26597166 PMCid:PMC8612699
10. Lipkin PH, Okamoto J; Council on Children with Disabilities; Council on School Health. The Individuals with Disabilities Education Act (IDEA) for children with special educational needs. Pediatrics. 2015;136(6):e1650-e1662. DOI: https://doi.org/10.1542/peds.2015-3409 PMid:26620061
11. Odom SL, Horner RH, Snell ME, Blacher J. Handbook of Developmental Disabilities. Guilford Press; 2007.

12. Moeschler JB, Shevell M; Committee on Genetics. Comprehensive evaluation of the child with intellectual disability or global developmental delays. Pediatrics. 2014;134(3):e903-e918. DOI: https://doi.org/10.1542/peds.2014-1839 PMid:25157020 PMCid:PMC9923626

13. Global Research on Developmental Disabilities Collaborators. Developmental disabilities among children younger than 5 years in 195 countries and territories, 1990-2016: A systematic analysis for the Global Burden of Disease Study 2016. Lancet Glob Health. 2018;6(10):e1100-e1121. DOI: https://doi.org/10.1016/S2214-109X(18)30309-7 PMid:30172774

14. Grantham-McGregor S, Cheung YB, Cueto S, et al. Developmental potential in the first 5 years for children in developing countries. Lancet. 2007;369(9555):60-70. DOI: https://doi.org/10.1016/S0140-6736(07)60032-4 PMid:17208643

15. Srour M, Mazer B, Shevell MI. Analysis of clinical features predicting etiologic yield in the assessment of global developmental delay. Pediatrics. 2006;118(1):139-145. DOI: https://doi.org/10.1542/peds.2005-2702 PMid:16818559

16. Aggarwal S, Bogula VR, Mandal K, et al. Neurodevelopmental disorders in children aged 2-9 years: Population-based burden estimates across five regions in India. PLoS Med. 2018;15(7):e1002615. DOI: https://doi.org/10.1371/journal.pmed.1002615 PMid:30040859 PMCid:PMC6057634

17. Potdukhe A, Jigjinni S, Pande B, et al. Prevalence and determinants of developmental delay among children less than 5 years of age in an urban slum area of Mumbai: A cross-sectional exploratory study. Indian J Child Health. 2024;11(2):e6252. DOI: https://doi.org/10.32677/ijch.v11i2.6252

18. Lozoff B, Jimenez E, Smith JB. Double burden of iron deficiency in infancy and low socioeconomic status: a longitudinal analysis of cognitive test scores to age 19 years. Arch Pediatr Adolesc Med. 2006 Nov;160(11):1108-13. DOI: https://doi.org/10.1001/archpedi.160.11.1108 PMid:17088512.

19. Nurliyana AR, Shariff ZM, Mohd Taib MN, Gan WY, Tan KA. Early nutrition, growth and cognitive development of infants from birth to 2 years in Malaysia: a study protocol. BMC Pediatr. 2016;16(1):160. DOI: https://doi.org/10.1186/s12887-016-0700-0 PMid:27687906 PMCid:PMC5043613

20. Ferdi, J., Bardosono, S., & Medise, B. E. Iron status and developmental delay among children aged 24-36 months. Paediatrica Indonesiana, 2022;62(4), 256-64. DOI: https://doi.org/10.14238/pi62.4.2022.256-64

21. Lozoff B, Georgieff MK. Iron deficiency and brain development. Semin Pediatr Neurol. 2006;13(3):158-165. DOI: https://doi.org/10.1016/j.spen.2006.08.004 PMid:17101454

22. Michel GF, Babik I, Nelson EL, et al. Evolution and development of handedness: An Evo-Devo approach. Prog Brain Res. 2018;238:347-374. DOI: https://doi.org/10.1016/bs.pbr.2018.06.007 PMid:30097200

23. McDonald L, Rennie A, Tolmie J, et al. Investigation of global developmental delay. Arch Dis Child. 2006;91(8):701-705. DOI: https://doi.org/10.1136/adc.2005.078147 PMid:16861488

24. Kakoza-Mwesige A, Byanyima RK, Tumwine JK, et al. Systematic review of MRI findings in children with developmental delay or cognitive impairment. Brain Dev. 2017;39(8):644-655. DOI: https://doi.org/10.1016/j.braindev.2017.04.006 PMid:28457518

25. Van Karnebeek CD, Shevell M, Zschocke J, Moeschler JB, Stockler S. The metabolic evaluation of the child with an intellectual developmental disorder: diagnostic algorithm for identification of treatable causes and new digital resource. Mol Genet Metab. 2014 Apr;111(4):428-38. DOI: https://doi.org/10.1016/j.ymgme.2014.01.011 PMid:24518794

26. Rossi A, Blok LS, Neuser S, Klöckner C, Platzer K, Faivre LO, et al. POU3F3-related disorder: Defining the phenotype and expanding the molecular spectrum. Clin Genet. 2023 Aug;104(2):186-197. DOI: https://doi.org/10.1111/cge.14353 PMID: 37165752 PMCID: PMC10330344

27. Resegue R, Puccini RF, Silva EM. Risk factors associated with developmental abnormalities among high-risk children attended at a multidisciplinary clinic. Sao Paulo Med J. 2008 Jan 2;126(1):4-10. DOI: https://doi.org/10.1590/s1516-31802008000100002 PMID: 18425280 PMCID: PMC11020518

28. Ozkan M, Senel S, Arslan EA, Karacan CD. The socioeconomic and biological risk factors for developmental delay in early childhood. Eur J Pediatr. 2012 Dec;171(12):1815-1821. DOI: https://doi.org/10.1007/s00431-012-1826-1 PMid:22983025

29. Robertson CM, Finer NN. Perinatal and early postnatal factors underlying developmental delay and disabilities. Dev Disabil Res Rev. 2011;17(2):109-119. DOI: https://doi.org/10.1002/ddrr.110 PMid:20818726

30. Thomaidis L, Zantopoulos GZ, Fouzas S, Mantagou L, Bakoula C, Konstantopoulos A. Predictors of severity and outcome of global developmental delay without definitive etiologic yield: a prospective observational study. BMC Pediatr. 2014 Feb 12;14:40. DOI: https://doi.org/10.1186/1471-2431-14-40 PMid:24521451 PMCid:PMC3933196

31. Yaghini O, Kelishadi R, Keikha M, Niknam N, Sadeghi S, Najafpour E, Ghazavi M. Prevalence of Developmental Delay in Apparently Normal Preschool Children in Isfahan,Central Iran. Iran J Child Neurol. 2015 Summer;9(3):17-23. PMID: 26401149; PMCID: PMC4577694.

32. Haddow JE, Palomaki GE, Allan WC, et al. Maternal thyroid deficiency during pregnancy and subsequent neuropsychological development of the child. N Engl J Med. 1999;341(8):549-555. DOI: https://doi.org/10.1056/NEJM199908193410801 PMid:10451459

33. Tye C, Runicles AK, Whitehouse AJO, et al. Characterizing the interplay between autism spectrum disorder and comorbid medical conditions: An integrative review. Front Psychiatry. 2019;9:751. DOI: https://doi.org/10.3389/fpsyt.2018.00751 PMid:30733689 PMCid:PMC6354568

34. Van Dijck A, Vulto-van Silfhout AT, et al. Clinical Presentation of a Complex Neurodevelopmental Disorder Caused by Mutations in ADNP. Biol Psychiatry. 2019 Feb 15;85(4):287-297. DOI: https://doi.org/10.1016/j.biopsych.2018.02.1173 PMID: 29724491 PMCID: PMC6139063

35. Cassidy C, Mannion A, Whelan S, Tones M, Heussler H, Bellgard M, Leader G. Association Between Feeding Problems and Gastrointestinal Symptoms, Language, and Developmental History in Adults With Angelman Syndrome. Am J Med Genet A. 2025 June;197(11):e64137. DOI: https://doi.org/10.1002/ajmg.a.64137 PMid:40583285

36. Leader G, Killeen C, Whelan S, Coyne R, Tones M, Mannion A. Factors associated with sleep disturbances in children and adolescents with Angelman Syndrome. Sleep Med. 2024 May;117:9-17. DOI: https://doi.org/10.1016/j.sleep.2024.02.038 PMid:38479041

37. Shan L, Feng JY, Wang TT, Xu ZD, Jia FY. Prevalence and Developmental Profiles of Autism Spectrum Disorders in Children with Global Developmental Delay. Front Psychiatry. 2022 Jan 18;12:794238. DOI: https://doi.org/10.3389/fpsyt.2021.794238 PMid:35115968 PMCid:PMC8803654