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Original Research Article

Evaluation of Prevalence and Causes of Developmental Delays among Children

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Abstract:

Background: Developmental delays (DD) affect approximately 1–3 % of children worldwide and encompass impairments in motor, cognitive, language, social, and adaptive domains. Early identification of causative factors—genetic, environmental, perinatal, nutritional, and socioeconomic—is critical to optimize interventions and outcomes.

Objective: To evaluate the prevalence and causes of developmental delays among children aged 6 months to 5 years at a tertiary paediatric centre, and to analyse associated risk factors.

Methods: In this prospective observational study, 300 children presenting with suspected DD were enrolled over 18 months. Detailed histories, standardized developmental assessments (Bayley Scales of Infant Development III), laboratory investigations, neuroimaging, and genetic testing (where indicated) were performed. Data were analysed using SPSS 25.0: continuous variables as means ± SD (Student's t-test), categorical as proportions (χ^2 test); multivariate logistic regression identified independent risk factors ($p < 0.05$ significant). Observation tables summarize demographic, clinical, and etiologic data.

Results: Of 300 children (mean age 24.7 ± 13.2 months; 59% male), 42% had global DD, 34% language delay only, and 24% motor delay only. Aetiologies: 28% preterm/perinatal hypoxia, 22% genetic syndromes (e.g. Fragile X, Down), 18% environmental (lead exposure, low socioeconomic status), 14% metabolic/nutritional (iron-deficiency anaemia, hypothyroidism), and 18% idiopathic. Multivariate analysis identified low birth weight (OR 2.8; 95% CI 1.7–4.6), perinatal hypoxia (OR 3.4; 95% CI 2.0–5.8), and low maternal education (OR 1.9; 95% CI 1.1–3.3) as independent predictors of DD.

Conclusions: Perinatal complications and socioeconomic factors are leading causes of DD in our cohort. Strengthening perinatal care, early developmental screening, genetic counselling, and addressing environmental/nutritional risks are essential to reduce the burden of DD.

Introduction

Developmental delay is a public health problem in low- and middle-income countries. However, there is no summarized evidence in low- and middle-income countries on developmental delay, and primary studies on this issue show varied and inconclusive results. Developmental delays (DD) refer to significant lag in one or more domains—gross/fine motor, cognition, speech/language, social, and adaptive skills—relative to age expectations. The global prevalence of DD is estimated at 1–3 %, with higher rates in low- and middle-income countries due to suboptimal perinatal care and environmental exposures [1, 2]. DD predisposes children to lifelong disability, educational underachievement, and social challenges [3].

A multitude of factors contribute to DD. Perinatal complications—prematurity, birth asphyxia, intracranial hemorrhage—account for 20–30 % of cases. Genetic abnormalities (chromosomal, single-gene syndromes) underlie 15–25 %. Nutritional deficiencies (iron, iodine) impair neurodevelopment, while environmental toxins (lead, mercury) and low socioeconomic status exacerbate risk. Additionally, idiopathic DD persists in 20–30 % despite extensive workup.

Early detection and intervention within the "first 1,000 days" are critical for neural plasticity. Current guidelines recommend routine developmental surveillance at each well-child visit and standardized

screening at 9, 18, and 30 months. Yet, in many settings, children present late, missing optimal intervention windows.

This study aims to delineate the spectrum of DD and its etiologies in a tertiary paediatric population, identifying modifiable risk factors to inform prevention and early intervention strategies.

Materials and Methods

Study Design and Setting A prospective observational study conducted from January 2023 to June 2024 at the LN Medical College & Research Centre & JK Hospital, Bhopal, India

Participants: Children aged 6 months–5 years referred for developmental concerns.

Inclusion: Those with ≥ 2 weeks delay in any developmental domain per parental report and screening.

Exclusion: Known CNS infections, severe congenital anomalies incompatible with life, or parental refusal.

Ethical Approval

Institutional Ethics Committee clearance obtained. Written informed consent from caregivers.

Assessment Protocol

1. **History & examination:** Perinatal history, family history, socioeconomic status (Kuppuswamy scale), environmental exposures.
2. **Developmental evaluation:** Bayley Scales of Infant and Toddler Development, Denver II for supplemental screening.
3. **Laboratory investigations:** CBC, serum ferritin, thyroid profile, blood lead levels if indicated.
4. **Neuroimaging:** MRI brain for children with global DD or focal deficits.
5. **Genetic testing:** Chromosomal microarray or targeted gene panels for dysmorphic features or positive family history.

Data collection and definitions

- **Global DD:** ≥ 2 standard deviations below mean in ≥ 2 domains on BSID-III.
- **Isolated domain delay:** ≥ 2 SD below mean in one domain only.
- **Etiologic classification:** Perinatal (prematurity < 32 weeks, Apgar < 5 at 5 min), genetic, nutritional/metabolic, environmental/toxic, idiopathic.

Statistical Analysis: Data entered in SPSS 25.0. Continuous variables as mean ± SD; categorical as frequencies and percentages. Group comparisons by Student's t-test or χ^2 test. Multivariate logistic regression to identify independent predictors of DD; variables with $p < 0.1$ on univariate entered model. $p < 0.05$ considered significant.

Observation Tables

Table 1: Demographic and Clinical Characteristics (n = 300)

Variable	Value
Age, months (mean ± SD)	24.7 ± 13.2
Male, n (%)	177 (59)
Preterm birth (< 37 wks), n (%)	96 (32)
Low birth weight (< 2.5 kg), n (%)	84 (28)
Socioeconomic status (lower class, n %)	142 (47)
Family history of DD, n (%)	36 (12)

Table 2: Distribution of Developmental Domains Affected

Delay Type	n (%)
Global DD	126 (42)
Language only	102 (34)
Motor only	72 (24)

Table 3: Etiological Classification

Aetiology	n (%)
Perinatal complications	84 (28)
Genetic syndromes	66 (22)
Environmental/toxic	54 (18)
Nutritional/metabolic	42 (14)
Idiopathic	54 (18)

Results

Demographics and delay profiles: Among 300 children (mean age 24.7 ± 13.2 months; 59% male), 42% had global DD, 34% language-only, and 24% motor-only delays. Preterm birth occurred in 32%, low birth weight in 28%.

Etiology: Perinatal complications (prematurity, hypoxia) predominated (28%), followed by genetic

syndromes (Down, Fragile X, Rett; 22%), environmental/toxic exposures (lead, low SES; 18%), nutritional/metabolic (iron deficiency anaemia, congenital hypothyroidism; 14%), and idiopathic (18%).

Risk factor analysis: Univariate analysis (Table 4) showed low birth weight, perinatal hypoxia, low maternal education, and low SES significantly associated with global DD ($p < 0.05$).

Table 4: Univariate Associations with Global DD

Factor	DD (n = 126)	No DD (n = 174)	p-value
Low birth weight	48 (38)	36 (21)	0.001
Perinatal hypoxia	54 (43)	30 (17)	< 0.001
Maternal education ≤ 5 yrs	68 (54)	60 (34)	0.002
Low SES	74 (59)	68 (39)	0.001

Multivariate logistic regression (Table 5) identified low birth weight (OR 2.8; 95% CI 1.7–4.6), perinatal hypoxia (OR 3.4; 95% CI 2.0–5.8), and low maternal education (OR 1.9; 95% CI 1.1–3.3) as independent predictors.

Table 5: Multivariate Logistic Regression for Global DD

Predictor	OR	95 % CI	p-value
Low birth weight	2.8	1.7–4.6	< 0.001
Perinatal hypoxia	3.4	2.0–5.8	< 0.001
Maternal education ≤ 5 yrs	1.9	1.1–3.3	0.02
Low SES	1.3	0.8–2.2	0.24

Statistical Analysis: Continuous variables were normally distributed (Kolmogorov–Smirnov test); between-group comparisons used Student's t-test. Categorical variables analysed via χ^2 test. Logistic regression for multivariate analysis. All tests two-tailed; $p < 0.05$ significant.

Discussion

In this cohort, perinatal factors and socioeconomic determinants predominated among DD aetiologies. Our perinatal complication rate (28 %) aligns with reports ranging 20–30 %. Genetic syndromes comprised 22 %, like 15–25 % in prior series. Environmental and nutritional causes (32 % combined) underscore the impact of lead exposure and micronutrient deficiencies in low-resource settings.

Low birth weight and perinatal hypoxia emerged as strong independent predictors, reflecting the need for improved antenatal, intrapartum, and neonatal care. Low maternal education also conferred risk, likely via reduced health literacy and delayed recognition of DD. Although idiopathic DD (18 %) persisted despite extensive workup, emerging genomic tools may elucidate underlying etiologies. Early standardized screening (e.g., BSID-III) enabled identification of isolated delays: isolated language delay (34 %) and motor delay (24 %), consistent with other studies. Language delays often predate learning disabilities; targeted speech therapy can improve prognosis.

Albaradie R et al studied the prevalence of seizures in children with developmental delay and identify the characteristics of such patients; to examine the association of GDD with epilepsy and to determine the effect of certain risk factors on this association. The prevalence of epilepsy in GDD patients was 56%; the epilepsy and non-epilepsy groups differed significantly in age. The most common type of seizure was generalized onset motor, which were observed in 37.5% of the sample. Problems during labor occurred in 15% of the sample; consanguineous marriage occurred in 61.6% of the participants. Neither of these factors differed significantly in the epilepsy and non-epilepsy groups. Advanced paternal age did differ significantly in the two groups (p=0.003). The prevalence of epilepsy is high in children with GDD, and of the factors studied here, the most significant variables affecting this correlation are the type of seizure and advanced paternal age.

Zablotsky B et al studied prevalence and trends of developmental disabilities among children Parents reported physician or other health care professional diagnoses of attention-deficit/hyperactivity disorder; autism spectrum disorder; blindness; cerebral palsy; moderate to profound hearing loss; learning disability; intellectual disability; seizures; stuttering or stammering; and other developmental delays. There were overall significant increases in the prevalence of any developmental disability (16.2%–17.8%, P < .001), attention-deficit/hyperactivity disorder (8.5%–9.5%, P < .01), autism spectrum disorder (1.1%–2.5%, P < .001), and intellectual disability (0.9%–1.2%, P < .05), but a significant decrease for any other developmental delay (4.7%–4.1%, P < .05). The prevalence of any developmental disability increased among boys, older children, non-Hispanic white and Hispanic children, children with private insurance only, children with birth weight ≥2500 g, and children living in urban areas and with less-educated mothers. The prevalence of developmental disability among US children aged 3

to 17 years increased between 2009 and 2017. Changes by demographic and socioeconomic subgroups may be related to improvements in awareness and access to health care.

Wondmagegn T et al did a systematic review and meta-analysis on prevalence and determinants of developmental delay among children in low-and middle-income countries. It aimed to assess the pooled magnitude of confirmed developmental delay and its determinants among children in low- and middle-income countries. The pooled prevalence of developmental delay in low- and middle-income countries was high as compared to that in high-income countries. Maternal education level and weight at birth were significantly associated with developmental delays. Therefore, strategies should be designed to decrease the rate of low birth weight and the number of illiterate mothers living in low- and middle-income countries.

Shatla MM et al studied prevalence and factors associated with developmental delays among preschool children in Saudi Arabia. Overall prevalence of children with DDs was 16.4%. The most prevailing DDs were the communication, problem solving, and personal/social skills (5.6%, 5.5% & 4.6% respectively). Lower rates of DDs were identified for fine motor, and gross motor milestones (1.9%, and 1.5% respectively. The study recognized a high prevalence of DDs especially for communication, problem solving, and personal/social skills. It spotted several modifiable risk factors, and recommended early screening of preschool children for prompt recognition and timely intervention.

Poverty and its associated factors put children at risk for developmental delay. The aim of the study by Wei QW was to describe the neurodevelopment of children under three years of age in poverty-stricken areas of China and explore possible associated factors. Developmental delay was explored with the five-domain, structured, parent-completed Ages and Stages Questionnaire. The Zung Self-Rating Depression Scale was used to assess depressive symptoms of the caregiver. The high prevalence of developmental delay in children younger than three years in poverty-stricken areas of China and the presence of risk factors for developmental delay such as inadequate learning resources and activities in the home, caregiver depression, and low family income highlight the need for early identification and interventions

An Egyptian community-based study was done by Metwally AM et al in national prevalence and profile of single and multiple developmental delays among children aged from 1 year up to 12 years. All enrolled children were assessed according to Vineland Adaptive Behavior Scales, (VABS) as a reliable screening questionnaire for identifying categories of DDs that were verified by paediatrics' specialists. The overall prevalence of children with DDs was 6.7%. The prevalence of a single DD was 3.9% versus 2.8% multiple DDs. Communication deficit was the most prevalent type (5.3%). Lower prevalence was identified for fine motor delay (1.0%), gross motor delay, and socialization deficit (1.5% each). The detected prevalence of DDs is within the estimated range of prevalence of DDs for the paediatric population. The majority of the detected risk factors are preventable. Developmental screening is recommended to be implemented in all primary care settings as a routine practice.

Limitations: Single-centre design may limit generalizability. Genetic testing was not uniform due to cost constraints, possibly underestimating genetic etiologies. Long-term outcome data were not captured.

Implications: Strengthening maternal–child health services to prevent preterm birth and perinatal hypoxia is paramount. Routine developmental surveillance and addressing modifiable risks (nutrition, environment) can mitigate DD burden. Enhanced access to genetic counselling and early intervention programs is recommended.

Conclusion

Developmental delays in this population are multifactorial, with perinatal complications and socioeconomic factors being predominant and modifiable causes. Integrating improved perinatal care, universal developmental screening, nutritional support, environmental risk reduction, and early therapeutic interventions are vital to minimize the impact of DD. Future multicentre longitudinal studies with comprehensive genomic evaluation are warranted.

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