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Original Article

Developmental Quotient Profile and Clinical Correlates in Children with Neurodevelopmental Delay: A Cross-Sectional Study

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ABSTRACT

Background: Quantifying developmental level helps characterize the severity of delay and may identify potentially preventable perinatal correlates. Evidence from resource-constrained referral settings remains limited.
Methods: In this hospital-based cross-sectional study, 78 children aged 6 months to 5 years with neurodevelopmental delay beginning in the first year of life underwent structured history, neurological examination and Gesell-based developmental assessment. Developmental age and developmental quotient (DQ) were derived. DQ distribution and its reported associations with gestational category, neonatal complications, birth asphyxia, seizures, intranatal complications, motor findings and microcephaly were examined. MRI outcome tables were excluded and are reported in a separate manuscript.
Results: Mean chronological age was 26.32 months, mean developmental age 10.02 months and mean DQ 40.04. Fifty-nine children (75.6%) had DQ 20–49, 13 (16.7%) had DQ 50–70, four (5.1%) had DQ <20 and two (2.6%) had DQ >70. Birth asphyxia was recorded in 55 (70.5%), prematurity in 14 (17.9%), fetal distress in 11 (14.1%) and neonatal sepsis in four (5.1%). Median DQ was lower with abnormal motor findings than without them [36.5 (IQR 30.0–46.0) vs 44.0 (35.5–51.0); p=0.037]. DQ did not differ significantly by birth asphyxia, any neonatal complication, prematurity, seizure disorder, intranatal complication or microcephaly.
Conclusion: Marked developmental impairment predominated in this referred cohort. Abnormal motor findings were associated with lower DQ. Perinatal histories remain clinically important but should not be interpreted as independent determinants of developmental severity in this dataset.

Keywords: *developmental quotient; neurodevelopmental delay; birth asphyxia; neonatal complications; developmental assessment; perinatal risk factors.*

INTRODUCTION

Developmental delay in early childhood may affect motor, language, cognitive, adaptive and personal-social domains. Developmental assessment describes the child's current functional level, guides early intervention and supports etiologic evaluation; it does not by itself identify the cause.[1–3]

Perinatal and neonatal insults—including hypoxic–ischemic injury, prematurity, infection and severe jaundice—may adversely influence neurodevelopment. Their contribution varies by population and quality of obstetric and neonatal care, and associations in clinic-based studies may be distorted by referral patterns and retrospective recall.[4–7]

This manuscript focuses on the developmental phenotype and perinatal correlates in children already identified with developmental delay. Its objective was to describe DQ severity and evaluate associations with recorded antenatal, intranatal and neonatal factors. MRI findings and MRI–clinical correlations are intentionally excluded to prevent overlap with the companion imaging article.

METHODS

Study design and setting: This descriptive cross-sectional study was performed at Chacha Nehru Bal Chikitsalaya, affiliated with Maulana Azad Medical College, Delhi over one year.

Participants: Children aged 6 months to 5 years attending the outpatient, pediatric neurology or child development clinics were eligible when developmental delay was the primary complaint and had begun in the first year of life. Clinically apparent or strongly suspected neurodegenerative disorders and delay secondary to non-cerebral chronic or acquired conditions were excluded. Seventy-eight children were enrolled.

Developmental assessment: Development was assessed using Gesell-based clinical methods across motor, adaptive/fine-motor, language and personal-social domains. Developmental age was estimated and DQ calculated as developmental age divided by chronological age ×100. The DQ was grouped as <20, 20–49, 50–70 and >70.

Perinatal variables: Structured history covered gestation, antenatal problems, delivery, intranatal events, neonatal complications and selected family and past history. Because several events could coexist, antecedent frequencies were not mutually exclusive.

Statistical analysis: Results are summarized as mean, median with interquartile range (IQR), number and percentage. DQ distributions between children with and without each clinical feature were compared using two-sided Mann–Whitney U tests because DQ was non-normally distributed in several subgroups. A p value <0.05 was considered statistically significant. Analyses were reconstructed from all 78 master-chart records.

Ethical considerations: Ethics approval was obtained from the IEC of Maulana Azad Medical College, Delhi.

RESULTS

Table 1. Age and sex distribution of participants (n=78)

Age group (months)	Total, n (%)	Male, n (%) within group	Female, n (%) within group
6–12	13 (16.7)	10 (76.9)	3 (23.1)
13–36	48 (61.5)	34 (70.8)	14 (29.2)
37–60	17 (21.8)	14 (82.4)	3 (17.6)
Total	78 (100)	58 (74.4)	20 (25.6)

Table 2. Chronological age, developmental age and developmental quotient by age group

Age group (months)	Mean chronological age (months)	Mean developmental age (months)	Mean DQ
6–12	9.85	5.05	52.20
13–36	21.65	8.38	38.64
37–60	52.12	18.44	34.68
Overall	26.32	10.02	40.04

Table 3. Distribution of developmental quotient (n=78)

DQ category	No.	%
<20	4	5.1
20–49	59	75.6
50–70	13	16.7
>70	2	2.6

Table 4. Recorded possible antecedents of developmental delay

Antecedent	No.	%
Birth asphyxia	55	70.5
Prematurity/low birth weight	14	18.0
Fetal distress	11	14.1
Neonatal sepsis/meningitis	4	5.1
Intrauterine growth restriction	3	3.8
Twin pregnancy	2	2.6
Kernicterus	2	2.6
Congenital brain malformation (any recorded)	8	10.3

Table 5. Developmental quotient according to clinical and perinatal features

Feature	Present, n	DQ median (IQR), present	Absent, n	DQ median (IQR), absent	p value
Preterm birth	14	32.3 (25.4–44.8)	64	38.5 (32.5–47.6)	0.108
Any associated symptom	54	36.8 (29.2–46.8)	24	45.0 (34.5–48.5)	0.087
Seizure disorder	40	35.5 (28.8–46.2)	38	43.7 (33.5–47.9)	0.143
Any neonatal complication	64	36.8 (29.6–47.0)	14	42.5 (36.0–48.0)	0.138
Birth asphyxia	55	37.0 (29.0–46.0)	23	42.0 (33.9–50.0)	0.094
Any intranatal complication	40	37.5 (31.2–47.6)	38	37.5 (32.8–47.0)	0.936
Abnormal motor finding	57	36.5 (30.0–46.0)	21	44.0 (35.5–51.0)	0.037
Microcephaly	24	35.5 (31.2–45.1)	54	40.0 (31.3–48.0)	0.369

Two-sided Mann–Whitney U tests applied. IQR, interquartile range.

DISCUSSION

The cohort showed substantial developmental impairment: three quarters of children had DQ 20–49 and the overall mean DQ was approximately 40. The lower mean DQ in older age groups may reflect delayed referral, increasing divergence from age expectations, survival or referral selection, or measurement limitations; the cross-sectional design cannot distinguish these explanations.

Birth asphyxia was the most frequently recorded antecedent, followed by prematurity/low birth weight and fetal distress. Prospective work in low- and middle-income settings has demonstrated adverse developmental trajectories after birth asphyxia, while outcomes after prematurity vary with gestational age, neonatal morbidity and access to follow-up and early intervention.[5–7]

Although children with birth asphyxia had a numerically lower median DQ, the difference was not statistically significant (p=0.094). The only significant subgroup difference was a lower DQ among children with abnormal motor findings (p=0.037), a clinically coherent association because motor function contributes directly to the developmental phenotype and to Gesell-based performance.

Aetiologic evaluation of developmental delay increasingly prioritizes careful phenotyping, hearing and vision assessment, chromosomal microarray and genomic testing when appropriate, with targeted metabolic testing and neuroimaging based on clinical clues. Regardless of etiology, timely referral for early intervention should proceed in parallel with diagnostic investigation.[1–3]

LIMITATIONS

This was a small, single-centre, referral-based cross-sectional study without a typically developing comparison group. Perinatal exposures were largely history based and potentially overlapping. Domain-specific developmental results, assessor reliability and instrument standardization were not reported. Several subgroup comparisons were exploratory and no correction was made for multiple testing. The cross-sectional design precludes causal inference, and multivariable modelling was not justified by the modest sample and event distribution.

CONCLUSION

Most children in this specialist-clinic cohort had marked developmental impairment, with DQ 20–49 forming the largest group. Abnormal motor findings were associated with lower DQ, whereas birth asphyxia, neonatal complications, prematurity and seizures were not significantly associated with DQ in the patient-level reanalysis. The results support structured developmental surveillance and early intervention while cautioning against assigning developmental severity to individual perinatal antecedents from this cross-sectional dataset.

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