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CONTENTS

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Aims and Scope of Indian Journal of Developmental and Behavioural Pediatrics (IJDBP)

IJDBP is a specialty journal in Developmental and Behavioural pediatrics published by Indian Academy of Pediatrics Chapter of Neurodevelopmental Paediatrics

The Journal welcomes Original papers, Review articles, Case reports and other articles relevant to child development & Behaviour including :

- Neuro developmental disorders,
- Developmental delays,
- Behavioural issues,
- Autism,
- Attention deficit hyperactivity disorder,
- Learning difficulties,
- Intellectual disabilities,
- Evidence based role of early intervention,
- Family centred multidisciplinary intervention,
- Neurogenetic disorders affecting child development,
- Neuroimaging & Neurological issues affecting child development,
- Corrective and assistive surgeries
- Home environmental and environmental issues affecting child development,
- Medical conditions
- Low birth weight and High-risk neonate requiring neonatal intensive care & its outcome,
- Preventive aspects in adolescents and pregnancy.
- Management of conditions covered in Rights of Persons with Disability Act,2016 of GOI.

It aim to promote advances in research in the field of child development and Behavioural issues so that latest evidenced based information is shared to enhance the quality of care and improve lives of children with special needs and their families.

The journal will be National Double Blind Peer review Open access journal published Quarterly. We will accept for publication manuscripts that were not published earlier in any form. The journal is devoted to publishing quality papers based on original innovative and most advance research in the field of developmental behavioural pediatrics.

The Journal aims to have the highest possible ethical and publication standards by scrutinizing the papers, through peer review assisted by eminent experts from prestigious teaching institutes from the country. For all Manuscripts submitted the journal will employ a plagiarism detection system for detecting plagiarism against previously published work.

INVITED GUEST EDITOR

PRIORITIES FOR NEURODEVELOPMENTAL SERVICES IN THE GLOBAL SOUTH

Life expectancy, care and quality of life for children and adults with neurodisabling conditions have improved remarkably in the 21st century. The awareness of the support needs of children with disabilities and their families are increasingly recognised by society and prioritised by service providers and governments. For any country the context of environment, society, economy, health informatics (among others) determine the priorities for service development. These priorities are necessarily different for low and middle income economies when compared with high income countries where there are well established service structures. In this article I provide a personal view on the priorities for neurodevelopmental service development in the global south.

There are certain key principles and processes which form the basis for what I consider as the building blocks for national/regional service development. Among the main principles are the following:

- implementation of the International Classification of Functioning, Disability and Health (ICF) which moves away from the medical model of 'fixing' (cure) to one of Functioning (doing) and Participation (1). Most neurodevelopmental conditions are not curable, so the focus needs to move to living meaningfully through participation and independence. Implementing it in clinical practice requires tools like the F-Words in Child Disability' developed by CanChild and available in many world languages(F-words for Child Development - CanChild). It also encourages the use of Family Centred Services (FCS) , in which the child and family's needs are the priority in planning services (https://iaacd.net/research/fcs).
- Adopting an Evidence-informed Care framework, ensuring there is proven effectiveness of interventions. The interventions also have to be relevant to the context (environment/culture) of the child and family.
- Encouraging a 'Non-categorical way of thinking' for professionals and family members. There are many commonalities across different neurodisabling conditions. A neurodevelopmental approach to management of functional difficulties e.g. sleep, behaviour, continence etc, using shared principles is valid for many different diagnoses.
- Adopt a Life Course Health Development approach, i.e. a 'whole life' approach in which plans for adult living for persons with neurodevelopmental conditions start in childhood (2). It keeps in mind the health trajectory of the medical condition, expected changes and challenges to anticipate. It also allows clinicians to be trained to manage these childhood-onset conditions as they grow into adulthood.
- Shared Partnership with People with Lived Experience (PWLE) is needed clinically, strategically at policy level and across organisations (3). This ensures the focus is on what is actually important to the affected children and families, not just what doctors or therapists think are important.

In addition to these key concepts and principles of practice, there are some 'process' issues to prioritize. These are as follows:

- There is a need to develop Care Multi-professionals Networks of Care in Neurodisability, allowing development of standards, promoting teaching, training and research(4).
- Data processing: There is a need to systematically collect and analyse local/national/regional data to know what the service priorities are and to assess effectiveness of interventions (5).

- Last but not the least, it is important to ensure that national 'universal' child development programmes are linked to any disability specific programmes like screening, assessment and intervention. Having connected programmes across child development and disability allows not just better resource utilisation, but also a more coherent programme of support to encourage and nurture development of ALL children.

These ideas are not exclusive to other ideas to build strong foundations, but they are essential. Implementing them as a priority on a national basis will enable development of a strong national child-onset disability provision.

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With Regards & Best Wishes,

Arnab Seal
President, International Alliance of Academies of Childhood Disability
Honorary Senior Lecturer, University of Leeds, UK

EDITORIAL

Looking back on 2025, I feel proud of how far we've come. One moment that stands out is the publishing of the IAP-NDP Consensus guidelines on High Risk Infant Developmental Supportive Care in the June issue of Indian Pediatrics. For me, that wasn't just a professional achievement—it was proof of our shared commitment to the most vulnerable newborns. This year, I've seen children with special needs move from the edges of our concern to the center of innovation in education, health care, and inclusion. We're finally shifting from offering "support at the margins" to building a world where belonging is the starting point. In our classrooms, clinics, and neighborhoods, I see technology and policy finally converging on a simple, beautiful ethical premise: every child in our care deserves more than just access—they deserve an experience intentionally shaped around their unique strengths.

I've been especially moved by the changes in assistive technology. What used to be clunky devices are now adaptive tools that respond to each child in real time. AI-driven learning and communication apps adjust difficulty and pacing like a conversation, not a clinical intervention. Speech-generating devices, accessible e-books, and personalized reading software are narrowing long-standing gaps in literacy and language. For children who have waited too long to be heard, this has been life-changing.

I am particularly moved by the evolution of smarter assistive technology . Immersive learning has also opened new doors. Virtual and augmented reality are no longer novelties—they're part of everyday intervention plans. I've watched children rehearse crossing a busy street, navigating a noisy classroom, or practicing a social conversation in safe, repeatable environments. By tailoring these spaces to their sensory needs, and using gamified platforms to build teamwork and emotional regulation, we're reframing "screen time" as a place of connection and growth.

Perhaps the accomplishment of 2025 that touches me most deeply is the way inclusive education tools have begun to reach children far beyond our well-funded specialist centers. Teletherapy and online platforms are bringing speech, occupational, and behavioral services into living rooms, easing the burden of distance and cost for families in rural or underserved areas. Global partnerships are funding accessible e-books in multiple languages and low-bandwidth platforms, reminding us that inclusion must extend beyond the urban school gate.

I am also encouraged by how we are using data to uphold dignity and amplify the family voice. Our new platforms for individualized education planning are using analytics and AI for more than just paperwork; they are exposing hidden inequities and prompting us to intervene earlier when it matters most. Data dashboards that track functional progress, participation, and emotional well-being are helping us move away from cold, narrow test scores and toward a much richer definition of success for our neurodivergent learners. It was heartening to see the members of IAP advocating for the changes facilitating family engagement for special needs children. Rightfully so, families and self-advocates are claiming their place at the table, challenging old narratives, and pushing us to adapt systems to children rather than the other way around.

Still, I know the work isn't finished. These accomplishments, as impressive as they are, remain heartbreakingly unequally distributed. Too many children with disabilities are still out of school or sitting in classrooms without meaningful support. Innovation without equity is just another form of exclusion. Our challenge for 2026 and beyond is to make sure these breakthroughs become everyday rights for every child, not isolated projects for a lucky few.

Best Regards

Dr. Zafar Mahmood Meenai

FRCPCH(UK), MSc paediatric Neurodisability (UK)

Editor-in-Chief, IJDBP

Effect of early intervention strategies on children with developmental delays using a multidisciplinary approach—a single-group design study

Monika Kundu Srivastava [1], Insha Mohammad[2], Swati Seth [3]
1. Consultant Occupational Therapist (Former HOD, OT), District Early Intervention Centre, Noida, RBSK NHM, Uttar Pradesh (Operationalised by HLFPPT), India
2. HOD Psychology, District Early Intervention Centre, Noida, RBSK NHM, Uttar Pradesh (Operationalised by HLFPPT), India
3. HOD Paediatrics, District Early Intervention Centre, Noida, RBSK NHM, Uttar Pradesh (Operationalised by HLFPPT), India

Address for correspondence
Monika Kundu Srivastava
Consultant Occupational Therapist (Former HOD, OT), District Early Intervention Centre, Noida, RBSK NHM, Uttar Pradesh (Operationalised by HLFPPT), India

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Abstract

Objectives: To determine the effectiveness of early intervention strategies on children with developmental delays or at risk for delays using a multidisciplinary approach.

Methods : A single-group design study was conducted at an early intervention centre at the district level over a period of 1 year. Fifty-two children who had neurodevelopmental delays (NDD) and 1 child who was 'at risk' for delay were assessed on the Developmental Screening Test (DST) for changes in their development quotients (DQ) before and after medical, occupational therapy, psychology and speech therapy intervention. The 53 children selected for the study had cerebral palsy (n=26), autism (n=21), cerebral palsy and autism (n=2), and a miscellaneous group (n=4).

Results: Forty children showed improvements in DQ after intervention and 12 children showed deterioration. There was no change in the DQ of 1 child. Post intervention, the mean DQ improved significantly from 55.15±21.67 (DST 1) to 60.83±23.42 (DST 2), $p<0.05$(CI 95%; -1.24 to -10.11). Though the study revealed no significant changes in individual groups, positive trends (improvements) were seen in cerebral palsy only, autism only, cerebral palsy and autism, and the miscellaneous group. Additionally, there were no significant changes in DQ scores reported gender-wise though females did show a greater improvement than males in the study.

Conclusion : Early intervention strategies using a multidisciplinary approach are effective in improving the fine motor, gross motor, language and personal-social skills of children with neurodevelopmental delays.

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Keywords:

- Early intervention
- Multidisciplinary approach
- neurodevelopmental delay (NDD)
- development quotient (DQ)
- Development Screening Test (DST)

Introduction

The rapid advances in medical technology have successfully increased the survival of high-risk babies, thus adding to the number of babies who might end up with developmental delays and disabilities. [1] These conditions may affect day-to-day functioning due to impairment in physical, learning, language, or behaviour areas, and are usually lifelong. [2]

The global prevalence of developmental delay in children is reported as 1% to 3%, while the World Health Organization (WHO) estimates that 15% of the world's population lives with some form of disability [3]. Over 43 % of children under the age of 5 years are at risk of not fulfilling their full developmental potential[4]. According to the 2016, Global Burden of Diseases, Injuries, and Risk Factors (GBD) study, India had the highest number of children affected with developmental disabilities (around 1.15 crore) and the highest cases of years living with disability (YLD) in the world (739 per 1 lakh). [5]

During early childhood, i.e., the first 5 years of life, the developing brain grows rapidly and is most sensitive to stimulation. The first 1,000 days, i.e., from 0 through 2 years are a unique period of opportunity when the foundations of optimum health, growth, and neurodevelopment across the lifespan are established. [6] Early intervention strategies enhance brain development[7]. Timely and periodic assessments of young children's development make it possible to identify and treat developmental disabilities at the earliest possible point of manifestation and to prevent loss of developmental potential. [8] It can also help identify developmental risk factors and target effective anticipatory guidance to provide parents with strategies for promoting optimal developmental outcomes. [8]

Despite the Integrated Child Development Services (ICDS), one of the world's largest and most unique outreach programs for early childhood care and development since 1975,[9] provisions for early identification and intervention for infants and young children in the Persons with Disabilities (Equal opportunities, Protection of Rights and Full Participation) Act of 1995[1] [now known as Rights of Persons with Disabilities (RPWD)] Act, 2016, the National Policy for Persons with Disabilities (2006)[10], and the *Rashtriya Bal Swasthya Karyakram* (RBSK) initiated under the National Health Mission (NHM) and launched in 2013[3], India still lacks routine developmental screening and surveillance[3] to handle the 67,385 babies born daily about one-sixth of the world's child births. [10]

Almost 54 % of deliveries take place in government hospitals [11] and having an early intervention centre in all such hospitals that cater to newborn and high-risk babies within the government sector should be prioritised. A common approach to early identification that ensures optimal intervention and limitation of secondary disabilities is developmental surveillance. [8] Each child often requires a multi-professional approach to the diagnosis and management and it is essential to ensure that children have access to the most appropriate range of support and interventions.[8] The availability of early detection and intervention services such as pediatrics, occupational therapy, speech therapy and audiology, psychology, optometry, dentistry, special education, social work, and basic laboratory facilities under one roof has been documented to be effective in the management of many childhood situations spanning from healthy child surveillance to inpatient mental health care. [1, 8]

A multidisciplinary approach brings together scientific knowledge from professionals who are trained to assess and deliver remediation and rehabilitation services. The strategies used by the professionals in early intervention include motor, sensory, cognitive-perceptual, psychosocial and emotional dimensions that are critical for the overall growth and development of children.

However, as there are very few studies that have been documented in Indian government hospital settings using a multidisciplinary approach for early detection and intervention, the present study was

undertaken to determine the effectiveness of early intervention strategies on infants and children at one such centre.

Materials and Methods

The study was conducted at the District Early Intervention-Centre of Excellence (DEIC-COE), located in NOIDA (GB Nagar District), Uttar Pradesh, India. The DEIC, caters to children from 0 through 6 years of age referred from the neonatal intensive care unit (NICU) of the Super Speciality Pediatric Hospital located within the same campus, other medical facilities and also walk-in patients without referrals. A single-group study was considered for ethical reasons. Fifty–two children from 0 through 6 years with neurodevelopmental delays (NDD) and 1 child 'at risk' for delay were included in the study from among all the children registered as out-patients with the DEIC from July 2018 through July 2019. Verbal consent was taken from the parents of the children included in the study.

The 53 children included had received some or the other form of intervention or guidance from all the different speciality departments of pediatrics, psychology, occupational therapy, speech therapy and audiology, dentistry and optometry. Special education intervention was provided to children above the age of 3 years. A minimum of two readings of development quotient (DQ) on the Developmental Screening Test (DST)—one pre-intervention (DST 1) and at least one post-intervention (DST 2)—were mandatory for inclusion in the study.

Those children who did not require active intervention from psychology, occupational therapy and speech therapy were excluded from the study. Those children who did not comply with the follow-up schedules were also excluded.

Anthropometric data was collected at the outset after registration of the child which included height in centimetres, weight in kilograms, head and chest circumference in centimetres, and mid-upper arm circumference (MUAC) in centimetres. A

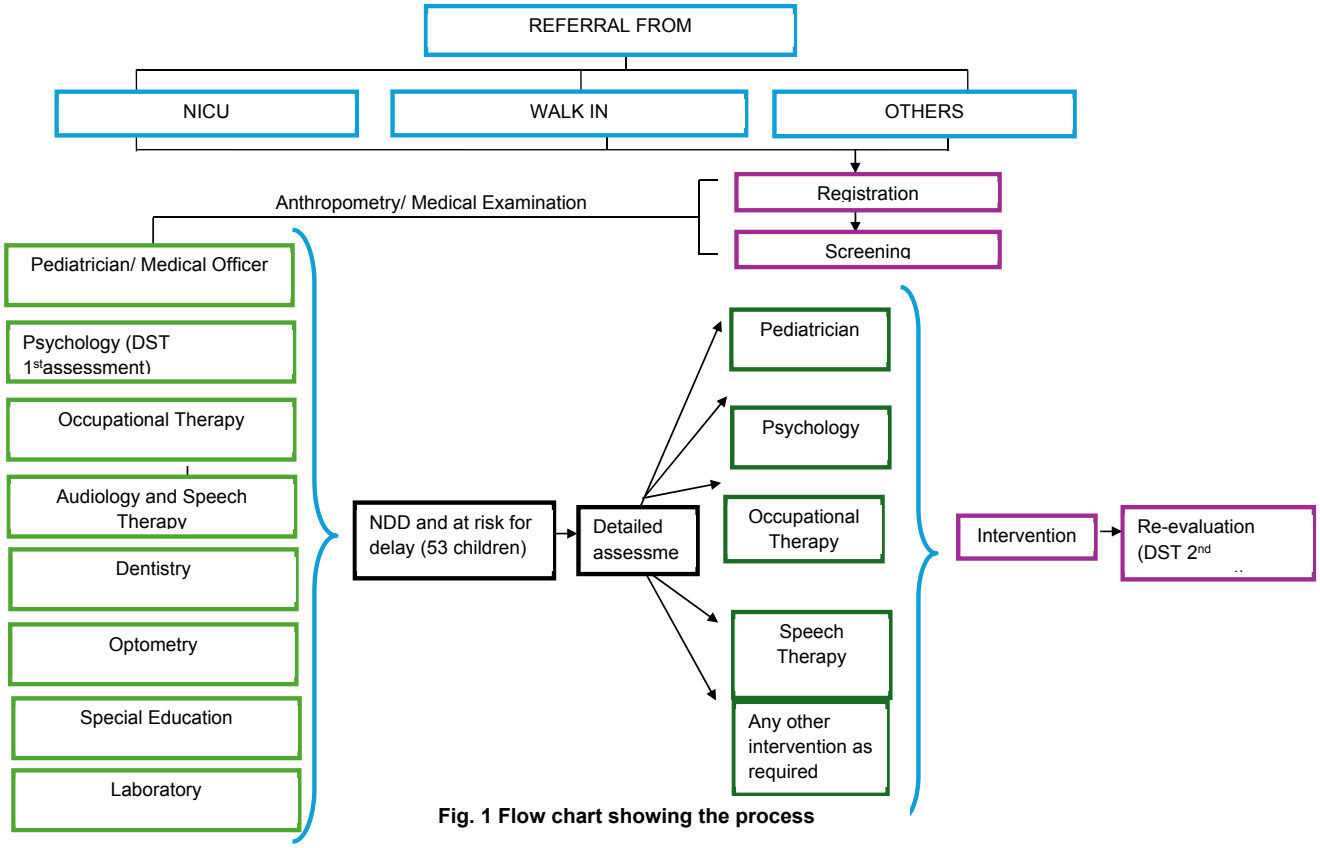

Fig. 1 Flow chart showing the process

detailed medical history was taken and a thorough examination was done by the paediatrician / medical officer. All the children were screened by the heads of department of psychology, occupational therapy, speech therapy and audiology, dentistry, optometry and special education for possible delays. Routine blood testing was done wherever indicated. The counsellor advised every parent on the nutritional requirements of the child and the social worker coordinated the appointment schedules on an 'as needed' basis vis-à-vis the parent and the respective departments.

If the screening process revealed a NDD, a detailed assessment was done by all concerned department heads. Intervention commenced thereafter by the respective specialists for remediation and rehabilitation. Each specialist followed their own protocols of assessment and intervention as detailed by their respective specialities. Parents or caretakers were counselled and explained the importance of their active participation in the intervention process. Each child was given a home programme. Regular assessments were done by all concerned speciality departments to monitor progress of the child. The first post-intervention scoring was done after at least 3 months of intervention, except for 03 children who were re-evaluated before 3 months due to reasons beyond our control **(Fig. 1)**.

The 53 children comprised of 26 who had cerebral palsy (CP), 21 with autism (ASD), 02 with CP and ASD, 03 children had delays including 01 female child who had a syndromic manifestation and was under evaluation, and 01 child was 'at risk' for delay **(Table 1)**.

Table 1 Baseline demographics of participants

	CP	ASD	CP & ASD	Delay/ At risk
Male	13	16	01	02
Female	13	05	01	02
Total	**26**	**21**	**02**	**04**

For the purpose of the study, the Developmental Screening Test (DST) was used as the outcome measure to determine pre- and post- intervention results. The DST is a screening test to ascertain the development of the child according to their respective age. It was developed by Bharath Raj (1977, 1983) and was designed to measure the developmental sequences of children from birth through 15 years of age. It consists of 88 items which assesses the overall development of the child (fine motor, gross motor, language and personal social skills). The administration was done by a semi-structured interview with a parent or a person well acquainted with the child. The attained score i.e., the DQ was then converted to IQ to get the results of the level of development of the child.

Results

Fifty-three children with delays/ at risk for delays were investigated. All children received medical, occupational therapy, psychology and speech therapy intervention. Paired sample statistics, using t-test revealed a significant difference between DST 1 (mean=55.15; SD=21.67) and DST 2 (mean=60.83; SD=23.43) scores on the complete sample (n=53), $p<.05$ (CI 95%, -1.24 to -10.11) as shown in **Table 2 and Fig. 2**.

Table 2 Comparison of DST 1 and DST 2 scores on the participants

Paired Samples Test										
Mean		Paired Differences					t	df	Sig. (2-tailed)
		Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference					
				Lower	Upper				
Pair 1	dst1 - dst2	-5.68113		16.07785	2.20846	-10.11274	-1.24953	2.572	52	.013

Fig. 2 A significant difference was found between DST 1 and DST 2 scores on the participants

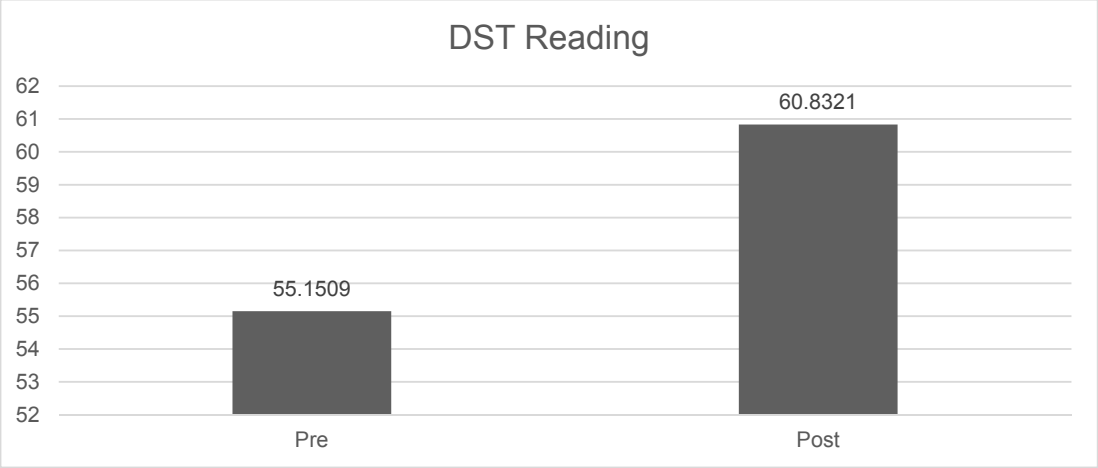

Table 3 indicates that most of the children (40 out of the 53 children) who participated in the study showed improvement in their overall development that included fine motor, gross motor, language and personal-social skills**.** The DQ of 1 child did not change but development continued to be age appropriate and hence has been considered as an improvement in the study. Only 12 children showed a deterioration in scores **(Table 4)**.

Table 3 Category-wise improvement (DQ) seen in 40 children

No change in category but increase in scores	Profound to Severe	Severe to Moderate	Severe to Mild	Moderate to Mild	Moderate to Borderline	Mild to Borderline	Mild to Dull Normal/ Age appropriate	Borderline to Age appropriate
20	01	02	01	03	03	06	01/01	02

Table 4 Category-wise deterioration (DQ) seen in 12 children

No change in category but decrease in scores	Moderate to Severe	Mild to Moderate	Borderline to Mild	Age appropriate to Borderline
05	01	03	01	01

Table 5 shows the trends for individual groups. The improvement trend suggests that the intervention program benefitted the individual groups (only CP: 58%; only ASD: 86%; CP & ASD/ Delay: 100%). However, results of Mann Whitney U Test for only CP and only ASD did not suggest a significant change in DST1 and DST 2 (p=0.108) scores due to the small sample size.

The least improvement was seen in dyskinetic type of CP and in moderate ASD. Score trends in **Table 5** also indicate that females appear to have benefitted from treatment more than males in 2 of the 4 major groups (only CP: 60%; only ASD: 100 %). The other 2 groups showed an equal improvement rate. However, the study did not reveal any significant gender differences with respect to the DST 1 and DST 2 on total sample size (n=53; p=0.304), only CP (n=26, p=0. 545), or only ASD (n=21; p=0.240).

Table 5 Changes for DST 2 for all diagnosis

1.	CP	TOTAL (M/F)	IMPROVEMENT (M/F)	IMPROVEMENT (M/F)	DETERIO-RATION	DETERIORA-TION
	Spasticity	4 (3/1)	3 (2/1)	75	1 (1/0)	25
	Hypotonia	6 (3/3)	4 (2/2)	66.7	2 (1/1)	33.3
	Ataxia	1 (1/0)	1 (1/0)	100	0 (0/0)	0
	Mixed	5 (3/2)	3 (1/2)	60	2 (2/0)	40
	Dyskinetic	10 (3/7)	4 (0/4)	40	6 (3/3)	60
	TOTAL	**26** (13/13)	**15** (6/9)	**58** (40/60)	**11** (7/4)	**42** (63.6/36,4)
2.	**ASD#**					
	Minimal	10 (7/3)	9 (6/3)	90	1 (1/0)	10
	Mild	1(0/1)	1 (0/1)	100	0 (0/0)	0
	Mild-Moderate	5 (5/0)	4 (4/0)	80	1 (1/0)	20
	Moderate	1 (1/0)	0 (0/0)	0	1 (1/0)	100
	Severe	1 (1/0)	1 (1/0)	100	0 (0/0)	0
	OTHERS (INCLEN)	3 (2/1)	3 (2/1)	100	0 (0/0)	0
	TOTAL	**21** (16/5)	**18** (13/5)	**86** (81/100)	**3** (3/0)	**14** (19/0)
3.	**CP and ASD**	2 (1/1)	2 (1/1)	100	0 (0/0)	0
4.	**Delay/ Atrisk**	4 (2/2)	4 (2/2)	100	0(0/0)	0

ASD#: Eighteen children were diagnosed using CARS. Three other children were assessed on INCLEN

An additional finding from the study was that that there was no correlation between DST 2 scores and the time interval for each child (p=.962) i.e., irrespective of whether DST 2 was done after 1,3,6, or even 12 months, changes were seen in scores.

Discussion

The primary purpose of the study was to determine the effect of early intervention strategies on children with developmental delays from 0 through 6 years of age using a multidisciplinary approach. Results indicated that 41 out of the 53 children (including the child who progressed age appropriately) improved in their overall development that included fine motor, gross motor, language and personal social skills. Families of the all 41 children were keenly involved with their children and strictly adhered to the instructions provided at the hospital and complied with the prescribed home program.

Twelve children showed deterioration on DST 2 scores. Compliance and follow-up vis-à-vis the treatment program was a concern for 11 children except 1 child who had CP and was in the severely acute malnourished (SAM) category. A total of 9 children had CP of who 8 had marked involuntary movements that affected their performance on fine motor skills, gross motor skills and language component of the DST; 3 children had ASD with major language delays. It can be said that lack of

compliance could be a major factor for deterioration because there were 4 other children in the CP group with marked involuntary movements who showed improvement and had good compliance and follow-up. Out of the 3 children with ASD who showed fall in DST 2, there was one child each in the mild, mild-moderate and moderate category. Other than these 3 children, all the other children in their respective groups showed improvements. Poor compliance and follow-up were major issues with the 3 children as well.

The findings were supported by a study conducted as a weekly supervised home-based developmental activity program for children with global developmental delay (GDD). Demographic data and baseline DASII (Development assessment scale for Indian infants) scores were obtained before intervention. Parents were trained for home-based intervention program with weekly follow-ups at the early intervention centre. After 6 months, DASII was repeated and difference in means of motor and mental quotients noted. There was a significant difference between pre- and post- therapy DASII scores in motor and mental scales with large effect size (Cohen's d >0.7) indicating that children across all severities of GDD showed improvement from home-based therapy supervised by weekly institute visits. [12]

A systematic review of studies on whether early developmental intervention programs provided post hospital discharge prevent motor and cognitive impairment in preterm infants suggested that early developmental interventions improve cognitive outcomes up to preschool age and also motor outcomes during infancy. However, these effects were small. [13]

Another study examined the age of referral and the effect of early intervention for physically challenged children. Fifty children were referred before 9 months of age, and they were compared with 55 children referred after 9 months of age. At 18 months of age, the children in the earlier referred group showed greater developmental progress in acquisition of skills in all of the six areas tested: perceptual-fine motor ($p<0.0003$), cognition ($p< 0.0001$), language ($p< 0.00(4)$, social-emotional ($p<0.001$), self-care ($p<0.0001$), and gross motor ($p<0.002$). The results show that, at least in the short term, there is a critical age for onset of intervention to achieve the most benefit for the developmentally disabled child [14]. A related study done on children with autism that involved different treatment modalities concluded that very early intensive therapy decreases the effect of autism. [15]

The above positive results could be due to the fact that early detection of abnormal white matter maturation is important in the design of preventive, protective, and rehabilitative strategies for the management of infants as white matter injury and abnormal maturation are thought to be major contributors to the neurodevelopmental disabilities observed in children and adolescents who were born preterm. [16]

Early intervention strategies include early educational and neuroprotection strategies that take advantage of cerebral plasticity and encompass all interventions that promote normal development and prevent disabilities, including organisational, therapeutic and environment□modifying measures, such as early stimulation programs. [7]

A study on premature infants admitted in the neonatal intensive care unit concluded that multisensory stimulation is an effective non-pharmacological method used in the development of premature infants to reduce stress and improve neuromuscular development. [17] Occupational therapy, psychology and speech therapy/audiology use a multisensory approach for intervention.

Early intervention programs have shown some small long-term benefits in heavier low-birth-weight babies. Cognitive and academic skills for heavier LBW (HLBW) premature children were better than for lighter LBW (LLBW) ones at the age of 8 years. However, attenuation of the large favourable effects was observed in both the heavier and lighter LBW groups at 3 years of age [18]. Favourable results were also seen only in HLBW youth at 18 years of age who had received early intervention. [19]

There are studies that indicate that early intervention approaches also enhance peer-related social competencies [20], benefit adult competencies and reduce violent behaviour[21] of young children with developmental delays.

Involvement of the families was also an important reason that the children who participated in the study showed improvements. Families understand their child's strengths, abilities, and special needs; know their rights and advocate effectively for their child; help their child develop and learn; have support systems; and are able to gain access to desired services and activities in their community. [22] Another study found that interventions that focus on parent–infant relationships have a greater impact on cognitive outcomes at infancy and preschool age than intervention programmes that focus on either infant development or parent support. [23].

The results of the study were also similar to programs such as NIDCAP (Newborn Individualized Developmental Care and Assessment Program) in Sweden and the IHDP (Infant Health and Development Program) created in the United States. The similarities were that efficacy was greatest with programs involving both the parents and the child; long-term stimulation improved cognitive outcomes and child-parent interactions; cognition showed greater improvements than motor skills and larger benefits were obtained in families that combined several risk factors including low education attainment by the mothers. [23]

Results for only CP and only ASD did not suggest a significant pre- and post-DST due to the small sample size. However, the positive trends towards improvement for both groups separately (only CP: 58 % ; only ASD: 86 %), indicated that the intervention programme was benefitting the individual groups as well.

The positive trends in CP can be supported by a systematic review of studies the effect of early intervention in infants at very high risk of cerebral palsy on child and on family outcome. [24].

A study done on children with ASD from birth to 3 years of age group to review the early intervention methods and studies available on each method revealed that early intervention gives better outcomes. [25] The positive trends in ASD can be supported by other studies as well. [26, 27] The present study did not reveal any gender differences with respect to the pre- and post-test DST scores. This could be due the small sample size for male only and females only. However, 80 % of the females included in the study showed an improvement vis-a-vis 68 % of males on DST 2. This can be corroborated by studies which demonstrate that girls have an advantage over boys in their cognitive function as measured by intelligence tests (Doyle and PFL Evaluation Team, 2016). [28] Another study of children aged two to four years found that girls had significantly stronger verbal and non-verbal abilities than boys. [28] Both the studies however, suggested that the possible advantage for girls in cognitive development during early childhood appeared to reduce over time. [28]

Limitations of the Study

The sample size of 53 children comprised only of children with NDD or at risk for delay. Through the study we were unable to determine the effects of early intervention with confidence on a specific group of children such as CP or ASD due to a small sample size. The study did not take into consideration the birth history of the child or whether the child had taken therapy prior to intervention as part of the present study as most children did not have the required documentation. Age of referral to the Centre, presence of co-morbidities such as epilepsy, heart conditions, etc., (past or current), and socioeconomic status was also not considered. As most of the children had feeding issues due to sensory or physiological limitations, the current nutritional status too was not considered

Conclusion

The study has proven that following a multidisciplinary team approach in early intervention settings improves the effectiveness of care. The present study in a government set-up strengthens the assumption that quality care need not come at a high price. Future studies could analyze various factors such as affect of birth history, nutritional status, co-morbidities, socioeconomic status, prior intervention received and parental motivating factors on DST scores. Whether treatment in a hospital set-up only, a home program only, or a combination of both is necessary for optimal results could also be studied.

Authors conclusion

'Zero-cost' or 'minimum-cost' quality care in a clinical setting given with a home program and regular follow-ups increases the likelihood of parent motivation and compliance thereby improving favourable outcomes such as: (a) preventing occurrence or development of a condition, (b) reducing the impact and magnitude of disability or delay in development, (c) providing supportive programmes for the complicated disability, and (d) to maximize the residual ability. More such early intervention centres offering multidisciplinary team services for detection and intervention should be commissioned through either government schemes or public-private partnerships at the district as well as the village level.

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Parental Play and Screen Time in Children with Autism Spectrum Disorder (ASD): A Comparative Study in the Indian Context

Aakriti Khajuria[1], Sunny Babber[2], Sandeepa Bailum[2], Ravinder K. Gupta[3] Urvi Gupta[4]

1. Aakriti Khajuria Post Graduate Student, Dept. Of Psychiatry, Acharya Shri Chander College of Medical Sciences (ASCOMS), Jammu ,J&K
2. Sunny Babber (Associate Prof., Dept. Of Community Medicine, ASCOMS) Sandeepa Bailum (Associate Prof., Dept. Of Community Medicine, ASCOMS)
3. Urvi Gupta (Clinical Psychology Scholar, Sharda University, Greater Noida)
4. Ravinder K. Gupta (Prof & HOD, Dept. Of Pediatrics, ASCOMS) (Corresponding author)

Address for correspondence
Dr Ravinder K. Gupta (Prof & HOD, Dept. Of Pediatrics, Acharya Shri Chander College of Medical Sciences (ASCOMS), Jammu,J&K
Urvigupta00@gmail.com
Mob 9419251787

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Abstract

Background : In recent years, a noticeable decline in parental play has coincided with a significant rise in screen exposure among young children. These evolving trends may have profound implications for social and communication development, particularly in children with Autism Spectrum Disorder (ASD).

Objective: To assess the impact of screen time and parental play on children diagnosed with autism spectrum disorder, and to compare these factors with typically developing children.

Methodology: This comparative cross-sectional study involved a sample of 70 children aged 2 to 8 years. Thirty- five children diagnosed with ASD, according to the DSM-5 (Diagnostic and Statistical Manual) criteria and attending the Postgraduate Department of Pediatrics, ASCOMS & Hospital were included and 35 children in the same age group without autism were taken as controls. Data was collected from the primary caregivers of both the groups and was analyzed using appropriate statistical tests and $p < 0.05$ was considered statistically significant.

Results: Children with ASD had significantly higher screen time and lower frequency of parental play compared to typically developing peers ($p = 0.001$). Only 14.29% of ASD parents reported daily play, versus 40% in the control group. Screen exposure was notably higher in the ASD group, with none reporting zero screen time. A significant negative correlation was found between screen time and parental play in the ASD group ($r = -0.561$, $p = 0.001$). Socio-demographic factors showed no significant influence ($p > 0.05$).

Conclusion: The study highlights a significant association between increased screen time and reduced parental play in children with ASD. These findings underscore the need for greater awareness and interventions to promote parent-child interaction, increase human engagement and regulate screen exposure in this population.

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Keywords:

- Autism Spectrum Disorder
- Human engagement
- Screen Time
- Parental Play

Introduction

Autism spectrum disorder (ASD) is a neurodevelopmental condition characterized by persistent challenges in social communication and interaction, coupled with restricted, repetitive behaviors. According to the International Classification of Diseases (ICD-11), ASD involves early-onset impairments in reciprocal social interaction and communication, along with a range of atypical, inflexible behaviors, interests, or activities, relative to expected developmental and cultural norms [1]. The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) similarly defines ASD by two core domains: (a) deficits in social-emotional reciprocity, nonverbal communicative behaviors, and relationships, and (b) restricted or repetitive patterns of behavior, interests, or activities [2]. In recent years, there has been a noticeable rise in the reported cases of autism spectrum disorder across the globe, with India being no exception. According to various epidemiological studies and clinical observations, the prevalence of autism in India has seen a marked increase, likely due to improved awareness, better diagnostic tools, and changing environmental and societal factors. Despite this upward trend, autism in India remains an under-researched and often misunderstood condition. Social stigma, limited access to specialized healthcare, and a lack of comprehensive national data continue to hinder early diagnosis and effective intervention. Epidemiological data in India suggest that ASD prevalence ranges between approximately 0.1% and 1.4% [3]. Research suggests that autism may be caused by a combination of genetic and environmental factors and various other factors such as viral infections, medicines, complications during pregnancy or air pollutants. Researchers are exploring about the impact of screen time, parental play and family type on ASD.

Screen time refers to the amount of time an individual spends using devices with screens such as smartphones, tablets, computers, televisions, and gaming. The global growth of electronic media usage among children has caused concerns regarding screen time impact on child development. In India also, rising screen exposure among very young children has emerged as a pressing public health concern. A study conducted in Tamil Nadu, India reported a mean screen time of 2.39 hours per day, with 73% of children under five exceeding recommended limits, and excessive usage strongly linked to developmental delays, particularly in language and communication domains [4]. Clinicians have raised red flags as they have noted an increasing number of preschoolers showing symptoms resembling “virtual autism spectrum disorder” (VASD) attributing this trend to screen use of 4-6 hours daily, especially when introduced before six months of age. Many studies suggest that screens have a major impact on children’s neurodevelopment and may increase their risk of developing ASD [5].

Parental play is defined as interactive, reciprocal play sessions between caregivers and their children and is a cornerstone of healthy development, especially in children with ASD. This dynamic interplay fosters joint attention, language acquisition, socio-emotional bonding, and cognitive growth [6]. A comparative cross-sectional study at a government mental-health clinic in Visakhapatnam revealed that children with ASD averaged over two hours of daily screen exposure and engaged in active parent-child play fewer than twice per week while typically developing peers received daily play and minimal screen time [7]. In recent decades, India has witnessed significant socio-cultural shifts, including a transition from joint to nuclear family structures, increased workforce participation among both parents, rapid urbanization, and a declining presence of extended family members such as grandparents. These changes, coupled with the widespread digitalization of everyday life, have profoundly altered traditional caregiving dynamics. As parental availability decreases and screen-based entertainment becomes more common, opportunities for meaningful play interactions between parents and children are often diminished, especially among children with developmental conditions like ASD.

Aim and Objectives :

1. To assess the impact of screen time and parental play on children diagnosed with Autism Spectrum Disorder (ASD) and to compare these factors with typically developing children.
2. To find the association if any, between screen time and parental play in children with ASD.

Materials And Methods :

This research was designed as a comparative cross-sectional study conducted among Indian children

after getting approval from the Institutional Independent Ethical Committee with reference no. ASCOMS/IEC/2024/Meeting-II/FM/24 dated 18/05/24, in the Post-graduate Department of Pediatrics from 01/10/2024 to 31/05/2025, at Acharya Shri Chander College of Medical Sciences and Hospital (ASCOMS & H) in Jammu & Kashmir, India. This comparative cross-sectional study included 70 children aged 2–8 years. The case group comprised 35 children with a prior diagnosis of ASD or newly diagnosed in the outpatient department (OPD) based on DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition) criteria. The control group included 35 typically developing children visiting the OPD for reasons unrelated to ASD. Inclusion and exclusion criteria were applied to both groups to ensure appropriate participant selection. Data were collected from the primary caregivers of both groups using a structured questionnaire. Written informed consent was obtained from the parents or legal guardians of all participating children prior to inclusion in the study.

Inclusion Criteria :

- Children aged 2 to 8 years.
- For the cases group: Children diagnosed with autism spectrum disorder as per the DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition) criteria, with no co-morbid neurodevelopmental disorders.
- For the control group: Children without any clinical signs or diagnosis of neurodevelopmental disorders, as verified by the treating pediatrician or psychiatrist, attending the Pediatrics or Psychiatry Outpatient Departments of ASCOMS & H.

Exclusion Criteria

- Both groups: Children with co-morbid neurological or sensory impairments (e.g., epilepsy, visual or hearing deficits).
- Both groups: Children whose parents or guardians did not provide consent.

Statistical analysis was performed by using IBM SPSS 21, descriptive data was expressed in terms of percentages and proportions, while continuous data was expressed in terms of mean and standard deviation and was compared with the help of appropriate statistical tests (chi-square & t-test). Pearson correlation coefficient was calculated to find the correlation between screen time and parental play time in children with ASD. P-value less than 0.05 was considered as statistically significant otherwise non-significant.

RESULTS

Table 1. Comparison of socio demographic profile of children with ASD and without ASD

Socio demographic	**ASD (n=35)**		**Control (n=35)**		**p-value**
Mean age of children ±SD	3.45 ± 1.23		3.81 ± 1.38		0.253 (N.S)
Gender					
Boy	24	68.57	25	71.43	0.794 (N.S)
Girl	11	31.43	10	28.57	
Parental status (main caregiver)					
Father	12	34.29	08	22.86	0.289 (N.S)
Mother	23	65.71	27	77.14	
Education status of parents					
Post-graduation	8	22.86	11	31.43	0.722 (N.S)
Graduation	17	48.57	15	42.86	
High school	10	28.57	09	25.71	

Economic status of parents					
Lower class	09	25.71	8	22.86	0.932 (N.S)
Lower-middle class	13	37.14	11	31.43	
Middle class	06	17.14	06	17.14	
Upper-middle class	06	17.14	08	22.86	
Upper class	01	2.86	02	5.71	
Residence					
Rural	17	48.57	15	42.86	0.631(N.S)
Urban	18	51.43	20	57.14	

N.S indicates statistically non-significant

Table 1 shows the comparison of socio demographic profile of children with ASD and without ASD. Among the thirty-five samples of ASD children (cases) aged 2-8 years, 24 were male, and 11 were female. Among the normal (control) children aged 2-8 years, 25 were male, and 10 were female. In both groups, the mother was the primary caregiver. In our study 22.86% parents of ASD children were post graduates whereas 31.43% parents of neurotypical children were post graduates.in both groups, majority of parents had completed their graduation while 28.57 % and 25.71% parents of children with and without ASD had studied till high school. In both the cases and controls, majority of the families belonged to lower middle-income socioeconomic status with 37.14% in the ASD group and 31.43% in non- ASD group respectively. In cases, only 2.86% families belonged to the upper class whereas in the control group 5.71% families were from upper class. About 51.43% families of ASD children belonged to urban area while 57.14% families of typically developing (neurotypical) children belonged to urban areas. The influence of socioeconomic variables on the study population was insignificant in the present study $p>0.05$.

Table 2. Comparison of Parental play of children with ASD and without ASD

Parental play of children	**ASD (n=35)**		**Control (n=35)**	
	Frequency	**Percentage**	**Frequency**	**Percentage**
Never	0	0	0	0
Less than once a week	14	40	02	5.71
One or two times a week	09	25.71	03	8.57
Several times a week	07	20	16	45.71
Once or twice a day	04	11.43	10	28.57
Several time a day	01	2.86	04	11.43
p-value	0.001*			

* indicates statistically highly significant

Table 2 shows comparison of the frequency of parental play between children with autism spectrum disorder (ASD) and those without ASD. Among the parents of children with ASD (n=35), 40% reported playing with their child less than once a week, whereas only 5.71% of parents in the control group (n=35) reported the same. In contrast, more frequent play interactions (i.e., once or more per day) were reported by 40% of the parents in control group compared to only 14.29% of parents in the ASD group. The difference in play frequency between the two groups was statistically significant (**p = 0.001**), indicating that **parents of children with ASD engage in play less frequently than parents of typically developing children**.

Table 3. Comparison of screen time of children with ASD and without ASD

Screen time of children	ASD (n=35)		Control (n=35)	
	Frequency	Percentage	Frequency	Percentage
Never	0	0	05	14.29
Rarely	09	25.71	16	45.71
Seldom	08	22.86	08	22.86
Sometimes	10	28.57	04	11.43
Often	18	51.43	02	5.71
p-value	0.001*			

* indicates statistically highly significant

Table 3 shows comparison of screen time of children with ASD and without ASD. A significant difference was observed in the screen time exposure between children with autism spectrum disorder (ASD) and those without ASD. Among children with ASD (n = 35), a larger proportion were reported to use screens frequently, with 28.57% using screens "sometimes" and 51.43% "often." In contrast, only 11.43% and 5.71% of children in the control group (n = 35) fell into these respective categories. Conversely, 14.29% of typically developing children reportedly never used screens, while none of the children with ASD were reported to have zero screen exposure. The observed difference in screen time distribution between the two groups was statistically significant (**p = 0.001**), indicating that **children with ASD tend to have higher screen time compared to their typically developing peers.**

Table 4. Correlation coefficient between screen time and parental play in children with ASD (n=35)

Correlation	Correlation coefficient 'r'	p-value
Screen time of ASD children	r= -0.561	0.001*
Parental play		

* indicates statistically highly significant

In **Table 4** Pearson correlation analysis was conducted to examine the relationship between screen time and parental play among children with autism spectrum disorder (ASD). The results revealed a statistically significant **negative correlation** between the two variables (**r = -0.561, p = 0.001**), indicating that **increased screen time in children with ASD was associated with decreased frequency of parental play**.

DISCUSSION :

This study aimed to compare the socio-demographic profiles, parental play frequency, and screen time exposure between children with autism spectrum disorder (ASD) and typically developing children, and to examine the correlation between screen time and parental play in children with ASD. The socio-demographic characteristics in this study revealed no statistically significant differences between children with ASD and those without ASD, indicating well-matched groups. The present study observed a higher prevalence of ASD among boys (68.57%) compared to girls (31.43%), which is consistent with findings from multiple global and Indian meta-analyses. Internationally, ASD is more commonly diagnosed in males, with global male-to-female ratios estimated to range from 3:1 to 4:1 [8]. This trend has also been consistently observed in the Indian context. A meta-analysis by Raina et al. [9], which examined the prevalence of autism in Indian children, reported a similar gender bias, with males being significantly more affected. Biologically,

genetic susceptibility and neurodevelopmental differences have been proposed as contributors. Socio-culturally, underdiagnosis in females, due to subtler symptom presentations and gender-based expectations may also skew the observed ratio. The finding that mothers were the primary caregivers across both groups aligns with Indian cultural norms where caregiving responsibilities are typically undertaken by mothers[10].In terms of parental education and socioeconomic status, most families in both groups belonged to the lower-middle or middle-income brackets, and the majority of parents were educated up to graduation level. This reflects patterns observed in other Indian studies, where children with ASD are not necessarily concentrated in low socioeconomic groups, but diagnosis and access to services are often better among more educated families [11].

Parental play is widely recognized as a cornerstone for social, emotional, and cognitive development in early childhood. It facilitates language acquisition, emotional bonding, and the development of critical social skills, especially in children with developmental delays such as ASD. A significant finding of this study was the reduced frequency of parental play in the ASD group compared to controls ($p = 0.001$). While 40% of ASD parents played with their child less than once a week, only 5.71% of control parents did so. On the other hand, daily play was far more common in the control group. This mirrors findings from Indian studies such as Desai et al.[12], which reported that parents of children with ASD often face challenges engaging in play due to behavioral difficulties and communication barriers in the child. Additionally, time constraints due to occupational and household responsibilities, particularly for mothers who often serve as the primary caregivers can further restrict the frequency and quality of such interactions[13]. Traditionally, Indian joint families have provided a socially rich and interactive environment for children. In such settings, children typically engage with multiple caregivers that is parents, grandparents, siblings, and extended relatives which may foster better social stimulation, early language development, and emotional bonding. This type of human engagement is especially valuable in early childhood, a critical period for the development of social and communication skills. Presence of multiple caregivers can help in early identification of developmental delays and may reduce the severity of social deprivation often experienced by children with ASD. However, the rising trend of nuclear families in urban India, driven by migration, urbanization, and economic demands, has led to a reduction in such naturalistic social support systems[14]. This transition may contribute to delayed detection of ASD symptoms and reduced day-to-day human interaction, potentially exacerbating developmental challenges in affected children. Furthermore, in nuclear families, the caregiving burden typically falls on a single parent, most often the mother leading to stress and reduced time for engaging in developmental activities such as parental play[15].

Screen time among children with ASD is a complex issue. On one hand, caregivers often use digital media as a coping mechanism to manage challenging behaviors or to provide temporary engagement, especially in settings where support services are limited. This practice is particularly common in India, where awareness about ASD-specific interventions remains limited and where socio-economic constraints often make screen-based devices the most accessible and affordable option for keeping a child occupied. The present study highlights a significant association between increased screen time and autism spectrum disorder (ASD), with children diagnosed with ASD exhibiting considerably higher screen exposure compared to their neurotypical peers. Our findings showed that more than half (51%) of the ASD group used screens “often,” and no child in this group had zero exposure. This aligns with the growing body of literature, including Indian studies such as Sidiq M et al.[16-18], which emphasize the increasing reliance on digital screens among children with developmental disorders. Research conducted in Andhra Pradesh; India concluded that toddlers with more than 4 hours of screen time per day were at high risk for developing Autism and a statistically significant correlation was established between the duration of screen use and the score of the Modified Checklist for Autism in Toddlers-Revised scale, with increased screen time showing greater autistic-traits. Yaakov Ophir et al.[19] suggest that high screen time is associated with delayed language development, reduced eye contact, impaired attention span, and diminished social reciprocity, all of which are core concerns in ASD.

The present study revealed a significant moderate

negative correlation (r = -0.561, p = 0.001) between screen time and parental play among children with ASD. Although less studies have been conducted to study this correlation yet the present study aligns with recent Indian evidence which suggests a correlation between increased screen time and a higher likelihood of autistic-like symptoms in young children, particularly when combined with reduced parental play. Another study suggests that increase in parental play and reducing screen time is effective in improving social functioning in children with ASD[20]. Reduced parental engagement not only limits play-based learning opportunities but may also reinforce the child's preference for passive digital content over reciprocal social activities.

Conclusion :

This study underscores the critical interplay between screen time and parental play in children with autism spectrum disorder (ASD) within the Indian context. While socio-demographic variables were similar across groups, children with ASD exhibited significantly higher screen exposure and reduced frequency of parental play compared to their typically developing peers. The observed moderate negative correlation between screen time and parental play highlights a concerning trend that is greater screen use may be displacing meaningful parent and child interactions that are vital for social and cognitive development. These findings reinforce the need for increased parental awareness, increased human engagement culturally sensitive intervention strategies, and systemic support to promote screen-free, play-based engagement in families of children with ASD. As nuclear families become the norm and digital devices more accessible, it is imperative to guide caregivers in balancing technological use with active, developmental play.

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Signs of Autism in Infancy: Review Article

A. Shafi[1], S. Raghavan[2], B. Koshy[3], S. Philip Oomen[4]

1. Postdoctoral Fellow, Department of Developmental Pediatrics, Christian Medical College, Vellore, Tamil Nadu
2. Associate Professor, Department of Developmental Pediatrics, Christian Medical College, Vellore, Tamil Nadu
3. Professor, Department of Developmental Pediatrics, Christian Medical College, Vellore, Tamil Nadu
4. Professor and Head, Department of Developmental Pediatrics, Christian Medical College, Vellore, Tamil Nadu

Address for correspondence
Dr. Samuel Philip Oomen
Professor and Head, Department of Developmental Pediatrics, Christian Medical College, Vellore, Tamil Nadu
E-mail: docspo@gmail.com

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Abstract

Background: Autism Spectrum Disorder (ASD) is a neurodevelopmental condition with an early onset, often evident before the age of three. While diagnosis typically occurs later, a growing body of evidence confirms that subtle but significant signs of ASD are frequently present in infancy [.1] Early identification can substantially improve developmental outcomes by facilitating timely intervention during a critical period of brain plasticity.[2]

Objective: This review synthesizes current literature on the early behavioral and neurodevelopmental signs of autism evident in the first year of life, highlighting findings from prospective, retrospective, and high-risk infant sibling studies.

Methods: A narrative review approach was adopted. Peer-reviewed publications between 1990 and 2024 were analysed, including clinical studies, parental report research, video analyses, and prospective cohort studies of high-risk infants.

Results: Key early markers include a decline in eye fixation from 2-6 months[3], limited social engagement and smiling[1], delays in joint attention and gesture use[1], motor delays[1], and atypical affect. Neurobiological studies reveal early brain changes, such as cortical surface area hyper expansion, that precede the full expression of behavioural symptoms.[4]

Conclusion: Infants later diagnosed with ASD often show observable signs within the first year. Recognizing these signs across social, communicative, motor, and sensory domains requires systematic observation and validated tools. Clinicians and early intervention teams must be trained to identify and act upon these subtleties to facilitate timely diagnosis and support.

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Keywords:

- Autism Spectrum Disorder
- Infancy
- Early Signs
- Developmental Surveillance
- Neurodevelopment

1. Neurodevelopmental Basis of Early Autism Signs

The first year of life marks a critical period for brain development, including synaptogenesis, pruning, and network specialization. Disruptions in these processes are increasingly linked to the earliest signs of ASD. [1, 10] Structural MRI studies of high-risk infants have identified a specific developmental cascade: a hyper expansion of the cortical surface area between 6 and 12 months of age, which precedes an overgrowth in total brain volume observed between 12 and 24 months.[4] This rate of brain volume overgrowth in the second year of life has been directly correlated with the severity of social deficits at 24 months.[4]

Functional MRI and EEG studies demonstrate altered connectivity in brain networks, particularly those sub serving face processing and social attention, as early as 6 months of age.[5] Atypical activity in the fusiform face area, amygdala, and superior temporal sulcus has been implicated in the failure of typical eye contact and social gaze development.[5] Furthermore, the genetic underpinnings of ASD are profound, with heritability estimated to be around 80-90%.[6] This risk is polygenic, involving common variants and rare mutations in genes related to synaptic function, such as *SHANK2* and *SHANK3*.[7] Epigenetic mechanisms, like DNA methylation, may link these genetic predispositions to environmental risk factors.[8,9[

2. Review of Methodologies for Early Detection

2.1 Retrospective Video Analysis

Retrospective studies analyzing home videos taken before diagnosis remain valuable for detecting subtle, naturalistic markers. These studies consistently show that infants later diagnosed with ASD exhibit reduced eye gaze, fewer reciprocal smiles, and less imitation compared to typically developing peers. [10]

2.2 Prospective High-Risk Infant Sibling Studies

Infants with an older sibling with ASD have a significantly higher recurrence risk. Prospective studies of these high-risk cohorts provide invaluable insight into the earliest divergence from typical development.[1] By 12 months, siblings later diagnosed with ASD can be distinguished from other infants based on atypical eye contact, visual tracking, and difficulties with disengaging visual attention[1] .Other common early signs in this group include decreased orienting to name, poor visual tracking, reduced motor coordination, and blunted affect by 6–12 months.[1]

2.3 Parental Report and Recall Studies

Parent-reported concerns often precede formal clinical recognition. Parents frequently describe a lack of eye contact, unresponsiveness to their name being called, and limited babbling or use of gestures. Studies have validated that parental concerns, especially when coupled with observational screening, have significant predictive value for a later ASD diagnosis.[11]

3. Core Domains of Early Autism Manifestations

3.1 Social Attention and Eye Contact

While typically developing infants show preferential looking to faces and eyes from birth, a key early sign in ASD is not an initial absence of eye contact, but rather a **decline in eye fixation between 2 and 6 months of age**. [3] This derailment of a fundamental social adaptive process is one of the earliest known indicators of social disability.[3] This pattern is often accompanied by reduced social smiling and poor synchrony with caregivers[1,12]

3.2 Response to Name and Auditory Responsiveness

Failure to consistently respond to one's name by 9–12 months is one of the most robust early markers of ASD.[1,12] This reflects underlying impairments in social orienting and auditory attention, rather than a hearing deficit.

3.3 Joint Attention and Gestural Communication

Joint attention involves both initiating and responding to a shared focus with another person. Deficits in these behaviors, particularly a lack of declarative pointing (pointing to share interest) and showing objects to others, emerge by 12 months in infants with ASD.[1,12]

3.4 Atypical Temperament and Affect Regulation

Infants who later develop ASD are often described as having a blunted affect, low adaptability, heightened negative affect, and poor self-soothing behaviors. [13] These temperamental traits can impact early caregiver-infant bonding.

3.5 Early Language and Vocal Patterns

While some infants with ASD show delayed or absent babbling, others may exhibit more subtle differences, such as producing fewer canonical (consonant-vowel) vocalizations and fewer socially directed sounds by 9-12 months.[14] Unusual crying patterns have also been noted as early as one month of age[5]

3.6 Motor Delays and Unusual Movements

Subtle motor delays are frequently observed alongside social-communicative challenges. These can include poor head control, delays in sitting or pull-to-sit, and atypical reaching and grasping, which may be evident by 6–9 months.[15] Early stereotyped motor movements, such as hand-flapping or body rocking, may also emerge before age one.[16]

3.7 Sensory Processing Atypicalities

Hyper reactivity (e.g., aversion to certain textures or loud noises) and hypo reactivity (e.g., apparent insensitivity to pain, visual fixation on spinning objects) are core features of ASD and are frequently observed in infancy.[17]

3.8 Repetitive and Unusual Play Patterns

By 9-12 months, infants with ASD may show unusual interest in toys, such as atypical visual inspection or repetitive manipulation (e.g., spinning or lining up objects) rather than engaging in functional or symbolic play.[18]

4. Onset Patterns and Developmental Trajectories

The onset of ASD symptoms can follow several patterns:

Early-Onset Pattern: Characterized by clearly observable delays or atypical behaviors before 12 months of age.[19]

Regressive Pattern: Affects up to 30% of children, who lose previously acquired skills—most often language or social interaction—typically between 15 and 24 months.[10]

Plateau Pattern: Children initially meet early milestones but demonstrate a slowing or plateauing of developmental progress after the first year.[20]

5. Predictive Validity of Early Signs

5.1 High-Specificity Markers

Certain signs have very high predictive value for a later ASD diagnosis. These include:

A decline in eye fixation from 2–6 months.[3]

Lack of response to name by 12 months.[12]

Absence of joint attention behaviors (e.g., pointing to share interest) by 12-14 months.[21]

Atypical brain development markers on MRI, such as cortical surface area hyper expansion.[4]

5.2 Multi-Domain Clustering

While individual signs are important, the combination of impairments across multiple domains—social, motor, language, and sensory—significantly improves predictive accuracy.[11,22] A persistent pattern of early atypicalities without developmental catch-up is strongly predictive of ASD.

6. Implications for Clinical Practice

The evidence strongly supports the integration of ASD-specific surveillance into routine pediatric care.

Developmental Surveillance: Paediatricians should actively monitor for the dynamic signs of ASD, such as the trajectory of eye contact and the emergence of joint attention, in all well-baby checks.

Screening Tools: While many screening tools are designed for toddlers (16-30 months), their use highlights the feasibility of early detection. In the Indian context, translated and validated versions

of tools like the **Modified Checklist for Autism in Toddlers, Revised with Follow-up (M-CHAT-R/F)** have shown good psychometric properties.[23] Indigenous tools like the **Indian Autism Screening Questionnaire (IASQ)**[24] and the **Chandigarh Autism Screening Instrument (CASI)**[25] are also valuable resources for community screening.

Parental Concerns: Paediatricians must elicit and validate parental concerns, as they are often the first to notice subtle developmental deviations and are reliable predictors of a later diagnosis.[11,26]

7. Gaps in Literature and Future Directions

Despite significant progress, several gaps remain. There is a critical need for more longitudinal studies that link early signs to long-term outcomes, particularly within diverse populations like India.[27] Further research is required to develop and validate culturally and linguistically appropriate screening tools to improve early detection in low-resource settings.[28] Finally, integrating promising biological markers, such as EEG and neuroimaging, into clinical screening protocols could revolutionize pre-symptomatic identification and intervention.[29]

8. Conclusion

Autism Spectrum Disorder frequently emerges in the first year of life through a constellation of subtle but observable signs affecting social gaze, reciprocity, motor function, and sensory responsiveness. Converging evidence from behavioral and neurobiological research underscores that these are not isolated symptoms but manifestations of an underlying divergence in neurodevelopment. For clinicians, particularly in settings like India where awareness and resources may be limited, a high index of suspicion and a focus on these early markers are paramount. An integrated approach that combines caregiver insight, systematic clinical observation, and the use of validated tools is essential for turning the promise of early detection into a reality for more children.

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Evaluation of neurodevelopmental outcome in Leigh's Syndrome/Leigh's like disorder– A hospital-based study.

Pravallika Y. L[1], Sanjay K.S[2], Vykuntaraju K Gowda[3]

1. Department of Pediatrics, Indira Gandhi Institute of Child Health, Bengaluru, Phone No. 9949500840,
Email ID: ypravallika412@gmail.com
2. Department of Pediatrics, Indira Gandhi Institute of Child Health, Bengaluru.
Email ID: dr.sanjay.ks@gmail.com
3. Department of Pediatric Neurology, Indira Gandhi Institute of Child Health, Bengaluru.
Email ID. drknvraju08@gmail.com

Address for correspondence
Dr Vykuntarajju K Gowda ,
Professor and HOD
C 101, Sobha Magnolia Apartments,
Bannerghatta Main Road, BTM layout,
Opposite Jal Bhavan, Bengaluru 560029.

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Abstract

Background: Leigh's syndrome is also called Leigh's disease or Subacute necrotising encephalomyelopathy. It is characterized by bilaterally symmetrical and subacute necrotic lesions in the basal ganglia, thalamus, the brainstem, and the posterior columns of the spinal cord. Genetically, it can be due to a defect in either mitochondrial or nuclear genes. The disease involves multiple organ systems, causing a heterogeneous presentation. There is a lack of literature enlightening on the pattern and severity of developmental retardation in Leigh syndrome; hence, this study was carried out.

Objective: To describe the neurodevelopmental profile in cases of Leigh syndrome / Leigh-like disorder.

Materials and methods: This was a hospital-based cross-sectional observational study of cases fulfilling Leigh's syndrome based on the Mitochondrial Disease Criteria (MDC) between 4 months and 42 months, who were followed up at the tertiary care center from March 2024 to August 2024. Children who were not hemodynamically stable were excluded. Their developmental assessment was done using the Bayley Scale of Infant and Toddler Development IV, and the level of impairment in all domains was noted.

Results: A total of 70 children were included. In our study, the developmental retardation in Leigh syndrome/Leigh-like disorder showed a global involvement, with major involvement being the fine motor domain, followed by gross motor domain, followed by expressive communication, followed by receptive communication, and the cognitive domain was the least affected.

Conclusion: There was significant involvement of global developmental delay in patients with Leigh syndrome/Leigh-like disorder, with predominant motor involvement and relative sparing of cognition. Early intervention focusing on improving motor coordination with improve the lifestyle of the child and family.

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- lissencephaly,
- neuronal migration
- gene

Introduction

Leigh Syndrome (LS) was first described by Denis Archibald Leigh in 1951 as a Subacute Necrotising Encephalopathy (SNE) and is a complex and incurable early-onset neurodegenerative disease. Online Mendelian Inheritance in Man Database (OMIM 256000) has defined LS by these cardinal characteristics: "neurodegenerative disease with variable symptoms due to mitochondrial dysfunction caused by hereditary genetic defect accompanied by bilateral Central Nervous System (CNS) lesions that can be associated with further abnormalities in diagnostic imaging" [1]. It is the most common presentation of mitochondrial diseases in the pediatric population [2,3,4,5]. The incidence is known to be 1 in 40,000 births [2,6,7,8,9] but can also be as high as 1 in 2,000 in certain populations [10]. The incidence in our country is still unknown.

The clinical spectrum is known to be widely heterogeneous, though the characteristic neuropathology features are typically consistent [2]. The most common clinical features are ataxia, hypotonia, developmental delay, seizures, poor feeding or feeding difficulties associated with dysphagia, persistent vomiting, failure to thrive, abnormal ocular disturbances [1]. There can be respiratory dysfunction, which often leads to death [11]. In older infants or young children, LS may present with ataxia, dystonia, or intellectual decline. Later, the children present with episodic regression within periods of clinical stability [11].

Literature shows similarity of Leigh syndrome with Thiamine deficiency, causing Wernicke's encephalopathy, leading to Leigh-like disease [12]. Large community-based studies are needed to differentiate thiamine deficiency leading to Leigh-like disease or genetic defects causing Leigh syndrome [13]. Though developmental delay is a known condition of Leigh syndrome/Leigh-like disorder, the exact severity and the pattern of developmental delay remain unknown. Hence, the study was chosen to shed light on the pattern of involvement of various developmental domains in children with Leigh syndrome/Leigh-like disorder.

Materials and methods

The objective is to describe the neurodevelopmental profile in cases of Leigh syndrome / Leigh-like disorder. Cases fulfilling Leigh-like disorder based on Mitochondrial Disease Criteria (MDC)[14], being followed up in a tertiary care center in Bangalore, were included from March 2024 to August 2024. In this hospital-based cross-sectional observational study, all cases between 4 months to 42 months of age diagnosed with Leigh sy like disorder with a disease durati 3 months. Hemodynamically unst within 2 weeks of hospital admiss consent from the parents and ethical obtained from the institutional ethical committee.

Demographic details and history of the study participants were taken. Age was corrected for prematurity. Their physical examination with neurological examination findings was noted. Other investigation reports like CBP (Complete Blood Picture), TMS (Tandem Mass Spectrometry), GC-MS (Gas Chromatography Mass Spectrometry), WES (Whole Exome Sequencing), MGS (Mitochondrial Genome Sequencing), MRI brain, lactate levels, Vitamin B_{12} levels, etc., were noted, if available. The neurodevelopment of the study participants was assessed using the Bayley scale of Infant and Toddler Development IV (BSID IV) [15], and the score was recorded. The same score was used to assess the level of impairment and was recorded as: Developmental age and Percentage delay.

Results :

A total of 70 children, who were previously diagnosed with Leigh syndrome/Leigh-like disorder, between the ages of 4 months and 42 months, were taken for the study. Table 1 shows details of history with Mean with Standard Deviation, Median with IQR, and range. Table 2 shows details of examination findings with Mean with Standard Deviation, Median with IQR, and range. Table 3 shows details of Investigation findings with Mean with Standard Deviation, Median with IQR, and range. Table 4 shows details of developmental assessment with Mean with Standard Deviation, Median with IQR, and range. Figure 1 shows clinical and MRI images of a child during acute presentation. Figure 2 shows clinical and MRI images of the child during the follow-up period.

Table 1: Table showing the details of history

History	Mean ± SD || Median (IQR) || Min-Max
Age (Months)	18.57 ± 8.49 || 17.27 (12.20-22.57) || 3.83 - 41.17
Gender	
Male	38 (54.3%)
Female	32 (45.7%)
Place Of Origin: State	
Karnataka	50 (71.4%)
Andhra Pradesh	8 (11.4%)
West Bengal	4 (5.7%)
Bihar	2 (2.9%)
Maharashtra	2 (2.9%)
Tamil Nadu	2 (2.9%)
Rajasthan	1 (1.4%)
Uttar Pradesh	1 (1.4%)
Place Of Living: District	
Ananthapur	4 (6.7%)
Bellary	2 (3.3%)
Bengaluru	7 (11.7%)
Chikkaballapur	5 (8.3%)
Chikkamagaluru	1 (1.7%)
Chittor	2 (3.3%)
Dabbaguli	1 (1.7%)
Hassan	1 (1.7%)
Kolar	7 (11.7%)
Mandya	6 (10.0%)
Raichur	1 (1.7%)
Ramnagar	6 (10.0%)
Satyasai	2 (3.3%)
Tumkur	15 (25.0%)
Age Of Detection (Months)	3.80 ± 3.72 || 3.00 (2.00-3.38) || 1.00 - 24.00
Consanguinity	
None	58 (82.9%)
2nd Degree	3 (4.3%)
3rd Degree	9 (12.9%)
Family history	
No	60 (85.7%)
Similar Complaints in Siblings	3 (4.3%)
Seizure Disorder In 1st Cousin	2 (2.9%)
Seizure Disorder in Uncle	2 (2.9%)
Seizure Disorder in Siblings	1 (1.4%)
Sibling Death	1 (1.4%)

Similar Complaints In 1st Cousin	1 (1.4%)
Developmental Retardation Timing: Before Acute Onset (Yes)	20 (28.6%)
Weakness (Yes)	70 (100.0%)
Motor regression (Yes)	22 (31.4%)
Seizures (Yes)	55 (78.6%)
Lethargy (Yes)	64 (91.4%)
Poor sucking (Yes)	64 (91.4%)
Tremors (Yes)	10 (14.3%)
GI symptoms (Yes)	11 (15.7%)
Failure to thrive (Yes)	14 (20.0%)
Respiratory symptoms (Yes)	4 (5.7%)

Table 2: Table showing details of examination findings

Examination	**Mean ± SD || Median (IQR) || Min-Max**
weight for age percentile	
<3rd	14 (20.0%)
3rd to 15th	52 (74.3%)
15th to 50th	3 (4.3%)
50th to 85th	1 (1.4%)
height for age percentile	
<3rd	22 (31.4%)
3rd to 15th	47 (67.1%)
15th to 50th	1 (1.4%)
Head Circumference Standard Deviation	
<-3	8 (11.4%)
- 3 To - 2	60 (85.7%)
- 2 To 1	2 (2.9%)
Neurocutaneous Markers	
None	61 (87.1%)
Café-au-Lait Spot	4 (5.7%)
Mongolian Spot	4 (5.7%)
Hypopigmented Macule	1 (1.4%)
CNS examination: HMF and speech (Speech Delay)	70 (100.0%)
CNS examination: Bulk	
Normal	50 (71.4%)
Wasting	20 (28.6%)
CNS examination: Tone	
Normal	27 (38.6%)

Hypertonia	27 (38.6%)
Hypotonia	11 (15.7%)
Variable	5 (7.1%)
CNS examination: Reflexes	
Normal	16 (22.9%)
Exaggerated	54 (77.1%)
CNS examination: Sensory	
Normal	70 (100.0%)
Abnormal	0 (0.0%)
CNS examination: Extrapyramidal	
Normal	25 (35.7%)
Dystonia	41 (58.6%)
Choreoathetosis	4 (5.7%)
Ataxia	13 (18.6%)
CNS Examination: Skull And Spine	
Normal	70 (100.0%)
Abnormal	0 (0.0%)

Table 3: Table showing details of investigation findings

Investigations	**Mean ± SD || Median (IQR) || Min-Max**
Hemoglobin (g/dL)	9.89 ± 1.26 || 10.00 (9.00-11.00) || 7.00 - 12.00
Homocystine (micmol/lit)	19.52 ± 16.71 || 13.00 (9.25-24.75) || 3.00 - 70.00
B12 (pg/mL)	469.53 ± 371.19 || 350.00 (219.00-589.15) || 139.60 - 2000.00
pH	7.26 ± 0.10 || 7.27 (7.20-7.33) || 7.00 - 7.47
pCO2	57.10 ± 41.35 || 44.00 (37.00-49.40) || 27.60 - 158.00
Base excess	-4.49 ± 2.52 || -3.90 (-6.65--2.22) || -8.70 - -1.50
NH3	71.53 ± 52.95 || 53.50 (35.25-101.40) || 10.00 - 169.00
Lactate (mg/dL)	34.33 ± 18.71 || 31.00 (23.00-42.20) || 11.00 - 109.20
Whole-Exome Sequencing	
ARFGEF1 gene, likely pathogenic	1
MT-CYB gene, uncertain significance	1
Thiamine metabolism dysfunction syndrome, likely pathogenic	1
Tandem Mass Spectrometry	
Normal	70 (100.0%)
Abnormal	0 (0.0%)
2D ECHO	

Normal	9
HOCM	1
Mild Left Ventricular Hyperintensities	1
Mild TR	1
Mild TR + Severe PAH	1
Severe PAH	1
Mother's Hb (g/dL)	10.50 ± 0.93 || 10.50 (10.00-11.00) || 9.00 - 12.00
Mother's B12 (pg/mL)	224.50 ± 69.80 || 215.50 (195.00-282.50) || 120.00 - 303.00
Mother's Homocystine (mmol/L)	37.00 ± 31.86 || 30.00 (12.50-48.50) || 11.00 - 96.00

Table 4: Table showing details of developmental assessment

Development	Mean ± SD	Median (IQR)	Min - Max
Cognitive age equivalent (Months)	14.23 ± 8.39	12.50 (8.00-19.00)	0.7 - 36.0
Cognitive Percentage delay	28.10 ± 20.45	22.90 (14.50-38.54)	0.8 - 100.0
Receptive Communication age equivalent (Months)	12.27 ± 8.02	11.00 (6.00-16.75)	0.7 - 33.0
Receptive communication Percentage delay	39.31 ± 23.05	35.60 (21.14-52.57)	4.3 - 100.0
Expressive Communication age equivalent (Months)	11.82 ± 7.79	11.00 (5.50-16.00)	0.7 - 32.0
Expressive communication Percentage delay	41.36 ± 23.35	37.10 (21.18-62.09)	0.8 - 100.0
Fine motor age equivalent (Months)	11.05 ± 7.30	10.00 (5.00-14.75)	0.7 - 32.0
Fine motor Percentage delay	44.00 ± 23.67	37.33 (25.45-61.76)	2.4 - 100.0
Gross motor age equivalent (Months)	11.16 ± 7.39	10.00 (5.25-16.50)	0.7 - 32.0
Gross motor Percentage delay	42.26 ± 30.46	40.28 (22.70-63.03)	-95.5 - 100.0

Discussion

Leigh syndrome is a progressive neurodegenerative disorder with onset usually in infancy or early childhood and a characteristic neuropathology [12]. It is one of the common disorders of mitochondrial etiology [1]. This study aimed to describe the developmental profile in cases of Leigh syndrome/ Leigh-like disorder. Understanding the pattern of involvement of developmental retardation will help us in initiating early intervention in these children, and to counsel on the prognosis.

The Bayley Scale of Infant and Toddler Development IV (BSID IV) can be done from age 16 days to 42 months [14]. The inclusion criteria of this study involved children diagnosed with Leigh syndrome/ Leigh-like disorder, with a disease duration of at least 3 months. Hence, in our study, we involved children aged between 4 months to 42 months of age. Among them, the mean age in months was 18.57 ± 8.49. Since this study was done when the child was brought for follow-up, the age distribution does not give any significant information.

In our study, 54.3% of the participants were male and 45.7% were female children. In a study done by Chan-Mi Hong in South Korea, to see the clinical characteristics of early onset and late onset Leigh syndrome, 46.4% were male and 53.6% were female [2]. In another study done by Xueli Chang in China, which is a meta-analysis on Leigh syndrome, there were a total of 279 patients, out of which 162 (58%)

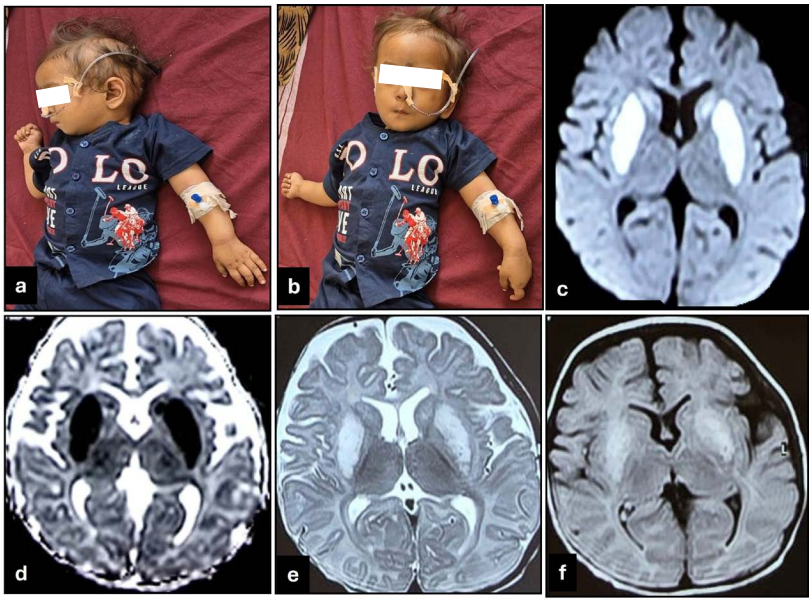

Figure 1. An 8-month-old infant with recurrent vomiting, regurgitation of feeds, and encephalopathy of subacute onset. Irritability reduced on day 3 of treatment; however, persistent oral dyskinesia compelled the need for naso-gastric feeds. Typical cortical fisting, limb dystonia with intermittent neck extension and arching were noted along with hypopigmented sparse hairline representing micronutrient deficiency (a and b). On neuroimaging, axial DWI (c) showed bilateral symmetric areas of diffusion restriction in the Putamen and caudate with signal drop in corresponding areas in ADC (d), suggestive of acute cytotoxic striatal injury. Axial T2WI and T2-FLAIR (e and f) depict diffuse frontotemporal cerebral atrophy with bilateral symmetric homogenous hyperintense signal changes in the putamen in T2-FLAIR(f).

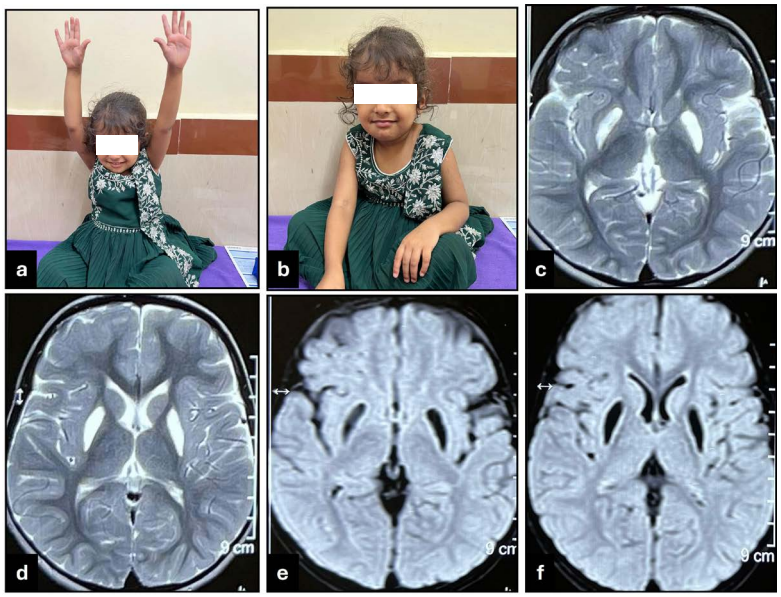

Figure 2. A three-year-old girl, seen during a follow-up visit at the child neurology clinic, with a significant sentinel event of acute encephalopathy, metabolic acidosis, and hyperlactatemia requiring a prolonged stay at the critical care unit at 3 months of age. Subsequently, she had lost all her milestones, only to regain them over the next 3 years with a metabolic cocktail and physiotherapy. Currently, she is ambulatory without support, albeit with an abnormally wide gait. She faces minor difficulty in performing activities of daily living due to dystonia (a and b). MRI Brain obtained 3 years after recovering from the encephalopathic crisis. Axial T2WI (c and d) shows bilateral symmetric, homogenous hyperintense signal changes involving the putamen. T2-FLAIR (e and f) shows symmetric hypointensity in the corresponding areas of T2 hyperintensities (c and d) involving bilateral putamen, suggestive of gliosis.

were male and 117 (41.9%) were female [16]. The difference in gender distribution in different studies could be due to the difference in the overall gender distribution of the population.

In the current study, the majority (71.4%) of the participants were from Karnataka state, followed by 11.4% from Andhra Pradesh. Among those from Karnataka, 25% of the patients are from the Tumkur district of Karnataka. The study was conducted in Karnataka and Andhra Pradesh, which are close to the state, explains the state-wise distribution. Tumkur district could be involved more, which might be secondary to any genetic involvement from the region or a nutritional cause. Further studies are required to know whether the major involvement of the Tumkur region is due to any causative factor prevalent in that area or is incidental.

Data retrieved from parents retrospectively during follow-up showed that, in the present study, the mean age of detection of the condition, in months, was 3.72 months, with a range of 1-24 months. In a study done by Xueli Chang in China, 77.5% of the children were diagnosed before age 2 years [16]. Another study done by Chan-Mi Hong showed that the median age of first clinical presentation was 9 months [2]. Since Leigh is a mitochondrial disorder [1], the initial presentation is usually in infancy, as they fall into metabolic crisis early.

In our study, 82.9% of the study population did not have a history of consanguinity, 4.3% had second-degree consanguinity, and 12.9% had third-degree consanguinity. There is not much available data on the impact of consanguinity on Leigh syndrome. Since consanguinity is more common in South India, further studies may be required to assess this association [17]. In the present study, 85.7% of the participants did not have a significant family history. In a study done by Chan-Mi Hong in South Korea, among the total participants of 110, only 16 had a significant family history [2]. This low percentage of involvement of other family members can be attributed to the fact that the heritability of Leigh syndrome is varied, as there is involvement of both mitochondrial and nuclear genes, with varying heritability. Further, nutritional causes like Thiamine deficiency also contribute to this disorder[1].

The onset of developmental retardation in our study was seen before the child had a metabolic crisis in 28.5% of the participants. This information was extracted by the recall method from parents, and there could be a good chance of recall bias. More information on other causes of developmental disorders could have given a better analysis.

In our study, the predominant clinical manifestations are, history of motor weakness in 100%, followed by lethargy and poor sucking in 91.4% and seizures in 78.6% of the study participants. In a study done by Chan-Mi Hong, assessing the clinical characteristics of early and late onset Leigh syndrome, seizures were seen in 24% and weakness was seen in 11% of the study population [2]. In another study done by Xueli Chang, weakness was found in 29.3% and seizures were found in 23% of the study population [16]. Leigh syndrome is a heterogeneous disorder with varied clinical presentation [1]. More studies are required among the Indian population in order to understand the difference in the frequency of the major clinical manifestations.

In our study, 20% of the participants weighted age <3rd percentile, and 74.3% fell between the 3rd to 15th percentile. In a meta-analysis conducted by Xueli Chang, out of 204 patients, there were 42 events of failure to thrive, which approximates to 20%, which also correlates with the findings in our study [16]. Our study also showed that 31.4% of the study population had height less than the 3rd percentile for age, and 11.4% had head circumference <-3 Standard deviations for age. Short stature can be expected in this condition, owing to the chronicity, and because of seizure episodes, microcephaly can be a common presentation. There is not much data available to compare these parameters to other studies.

Among our study participants, neurocutaneous markers were seen in 13%, among which the most commonly seen were café au lait spots and Mongolian spots, in 5.7% each. One child (1.4%) had seventh nerve palsy, wasting was seen in 20 children (28.6%), abnormal tone in approximately 62%, which could be hypertonia or hypotonia or variable tone, exaggerated deep tendon reflexes in 77.1%, ataxia in 18.6% and basal ganglia features in about 65%. Since the study was done in any acute crisis, all the children were hemodynamically stable with normal higher mental functions. Leigh syndrome has a heterogeneous clinical presentation, with the commonly involved system being the central nervous system [1].

In this study, GI symptoms were seen in 15.7% and respiratory symptoms were seen in 5.7%. The cardiovascular system was affected in 5 patients, with the available echocardiography. Another study showed that the GI system was affected in 37.3% and the cardiovascular system was involved in 8.2%. These findings confirm a clinically significant involvement of other systems in Leigh disease, with a heterogeneous presentation [1].

In our current study, investigations were done at the time of acute presentation, and they were not available in all the patients, due to multiple reasons. There was elevated lactate, above 30mg/dL, in 30 patients, among the available data. The mean lactate levels were 34.3mg/dL. The mean base excess was -4.49; base excess more than -2 was found in 9 patients, among the available data. This shows metabolic acidosis at acute presentation. A study done by Albert Z. Lim in the United Kingdom showed elevated lactate in 68% of patients [10]. Another study done by Chan-Mi Hong in South Korea also showed lactic acidosis in approximately 60% of the study population [2]. This signifies the anaerobic metabolism that takes place, leading to metabolic acidosis in mitochondrial disorders [1].

Multiple studies have confirmed that developmental delay or regression is seen in Leigh syndrome. Studies investigating the pattern and severity of involvement are lacking. Hence, this study aims to throw light on the developmental profile of children with Leigh syndrome/Leigh-like disorder, by using the Bayley Scale of Infant and Toddler IV. In our study, the mean cognitive age equivalent was 14 months, the mean receptive communication age equivalent was 12 months, the mean expressive communication age equivalent was 11 months, the mean fine motor age equivalent was 11 months, and the mean gross motor age equivalent was 11 months. This is opposed to the mean population age of 18 months. The mean percentage delay in the cognitive domain was 28%, the mean percentage delay in receptive communication was 39%, the mean percentage delay in expressive communication was 41%, the mean percentage delay in the fine motor domain was 44% and the mean percentage delay in the gross motor domain was 42%. This implies that cognition is relatively preserved, and fine motor skills are the most severely affected domain in this study population. There is not much literature on the severity and pattern of developmental delay currently. Further studies are needed to see the validity of our study results.

Conclusions

There was significant involvement of global developmental delay in patients with Leigh syndrome/Leigh-like disorder, with predominant motor involvement and relative sparing of cognition. Early intervention focusing on improving motor coordination with improve the lifestyle of the child and family.

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Clinical and developmental profile of children with West Syndrome in a tertiary care hospital in South India: A prospective observational study

Dr. Ann Mary Catherine[1], Dr. Susan Mary Zachariah[2], Dr. Akbar Mohamed Chettali[3], Dr. Rajappan Pillai[4]
1. Dr. Ann Mary Catherine, MBBS, DNB (Pediatrics) Pediatric Resident, Aster Medcity, Kochi, Kerala, India
2. Dr. Susan Mary Zachariah, MD (Pediatrics), IAP Fellowship in Neurodevelopmental and Behavioural Pediatrics
Senior Specialist – Developmental Pediatrics, Aster Medcity, Kochi, Kerala, India
3. Dr. Akbar Mohamed Chettali, DM (Pediatric Neurology)
Consultant Pediatric Neurologist, Aster Medcity, Kochi, Kerala, India
4. Dr. Rajappan Pillai, MD (Pediatrics), Fellowship [PICU & Retrieval]
Consultant – Pediatric Intensive Care, Aster Medcity, Kochi, Kerala, India

Address for correspondence
Dr. Ann Mary Catherine, MBBS, DNB, Pediatric Resident
Address: Mookenthottam House, Arakuzha, Memadangu P.O., Muvattupuzha, Ernakulam, Kerala, India – 686672
Email: ann.mary.catherine@gmail.com
ORCID iD: 0009-0006-5273-2512

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Abstract

Objectives To describe the etiological, clinical and developmental profile of children with West Syndrome.

Methods A detailed history, clinical examination, DP-3 developmental assessment, EEG and MRI were conducted in the initial encounter. Children were followed up at 6 months to assess seizure control, E-CHESS scores and neurodevelopmental status.

Results Out of 35 children, 62.9 % had Symptomatic West Syndrome. No significant differences were found between Symptomatic and Cryptogenic groups in demographics, seizure type, response to treatment, or E-CHESS scores. Neurodevelopment was adversely affected in all groups influenced by time lag in initiating treatment and E-CHESS scores.

Conclusion The Symptomatic group dominated over the cryptogenic group with potentially preventable perinatal asphyxia as the commonest cause. Neurodevelopment was greatly affected with effects persisting despite seizure control. Delayed treatment and the severity of epilepsy can adversely affect the neurodevelopmental outcomes.

Key Notes Symptomatic predominance in West syndrome is noted. Early intervention is needed to improve developmental outcomes. Seizure control alone does not guarantee good neurodevelopment.

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Keywords:
- West syndrome
- epileptic spasms,
- Neurodevelopment
- Developmental Profile-3
- Hypsarrhythmia

Introduction

West syndrome is a devastating age dependent epileptic encephalopathy characterized by a triad of epileptic spasms (IS), hypsarrhythmia and developmental retardation. It can impair the brain maturation, cognition, and development of the child. It most commonly presents in the second to twelfth months of life. It is often caused by an organic brain dysfunction whose origins may be prenatal, perinatal, or postnatal. This study aimed to explore the etiological and clinical spectrum of children with West syndrome, with a special emphasis on their developmental outcomes. It seeks to enhance paediatricians' understanding of this devastating, age-dependent epileptic encephalopathy.

Methods

This prospective observational study was conducted at our institution from September 2018 to May 2020. Children aged 1 month to 5 years who presented to the emergency, inpatient, or outpatient departments either as known cases of West syndrome or newly diagnosed based on the International League Against Epilepsy (ILAE) definition were included in the study. The study was approved by the institutional ethics committee.

At the initial encounter, a detailed history, clinical findings, developmental assessment (DP-3 scoring), electroencephalogram (EEG), neuroimaging (MRI), and other relevant investigations were performed. Children were followed up six months after the initiation of treatment, with assessments focused on:

- Seizure control
- Adverse outcomes
- Early Childhood Epilepsy Severity Scale (E-CHESS) scoring
- Developmental status based on DP-3 scoring.

The Developmental Profile 3 (DP-3) scoring system evaluates five domains: physical, adaptive, social-emotional, cognitive, and communication. A total standard score and a composite General Development Standard (GDS) score were calculated, with minimum scores of 50 for individual domains and 40 for the composite GDS.

The Early Childhood Epilepsy Severity Scale (E-CHESS) is a tool designed to assess epilepsy severity, aid in evaluating treatment efficacy, and investigate the impact of epilepsy severity on development. It includes five key measures:

1. Duration of seizure occurrence
2. Seizure frequency
3. Number of seizure types
4. Number of anticonvulsant medications used
5. Response to treatment

Each variable was scored from 0 to 3, with higher scores indicating greater severity. Children in the study were categorized into two groups based on their E-CHESS scores: <10 and ≥ 10.

Statistical Analysis

Data analysis was conducted using SPSS 22.0 and R environment version 3.2.2. Descriptive and inferential statistical methods were applied.

- Continuous variables were presented as Mean ± SD (Min–Max).
- Categorical variables were presented as Number (%).
- Statistical significance was set at $P < 0.05$.

Clinical trial number: Not applicable.

Results

A total of 35 children were recruited for the study, of whom 19 (54.3%) were male. The children were categorized into symptomatic (22, 63%) and cryptogenic (13, 37%) groups.

Among the symptomatic group, 13 children (59.1%) were born preterm. Both groups were comparable, except for the significantly higher prevalence of prematurity in the symptomatic group ($P = 0.003$) (Table I). There was no significant association between gender, age distribution, consanguinity, or mode of delivery across the two groups. CNS malformations were observed in 20 children (90.9%) within the symptomatic group.

Seizure type analysis showed that 28 children (80%) presented with flexor spasms. The relationship of spasms with sleep was evaluated, and 21 (60%) had no association with sleep, while 12 (35%) experienced spasms upon awakening.

Most children did not exhibit specific features preceding or following the spasms, and 22 (62.8%) had no additional seizure types apart from infantile spasms. No significant difference was observed in these characteristics between the symptomatic and cryptogenic groups.

A total of 22 children (62.8%) had at least one comorbidity associated with West syndrome, with 17 (77.3%) in the symptomatic group and 5 (38.5%) in the cryptogenic group. Among these, visual impairment was the most common comorbidity in the symptomatic group, while feeding difficulties were more frequently observed in the cryptogenic group (Table 1).

Children with symptomatic West syndrome had significantly higher rates of developmental delay prior to seizure onset than those with cryptogenic West syndrome ($P < 0.01$). In contrast, developmental regression following seizure onset was observed more frequently in the cryptogenic group, which was statistically significant ($P = 0.046$) (Table 1).

The response to treatment was assessed based on complete seizure cessation, partial cessation, or no improvement. Both groups showed comparable responses, with 14 children (63.6%) in the symptomatic group and 8 children (61.5%) in the cryptogenic group achieving complete seizure cessation. Girls demonstrated a better response to treatment compared to boys.

The E-CHESS score at six months post-treatment initiation was comparable between the symptomatic and cryptogenic groups.

At the six-month follow-up, DP-3 scores across different domains and overall developmental scores showed no significant difference between the two groups, except in the physical domain, where the cryptogenic group had better outcomes (Table II). Within each DP-3 domain, developmental scores varied significantly based on treatment response (*ANOVA analysis*). Children with no response to treatment had significantly lower developmental scores compared to those who showed partial or complete cessation.

A significant association was found between higher E-CHESS scores and lower developmental scores at follow-up, with the social-emotional domain being the most affected. Furthermore, children with higher E-CHESS scores demonstrated greater declines in developmental scores, particularly in the social-emotional and communication domains (Table 2).

Early initiation of treatment was associated with better developmental outcomes at follow-up compared to delayed treatment initiation, with the difference in General Developmental Score being statistically significant ($P = 0.017$) (Table 2).

Discussion

A prospective observational study was conducted at Aster Medcity, Kochi, from September 2018 to May 2020 to evaluate the clinical spectrum, aetiology, and outcomes of West Syndrome in children. A total of 35 children meeting the inclusion criteria were enrolled. The mean age at presentation was 10.6 months (range: 3–24 months), comparable to Sehgal R et al.[1] (median: 12 months). The mean age of spasm onset was 4.8 months, aligning with findings by Kalra et al.[2] (5 months). No significant association was observed between seizure onset and gender or preterm gestation. The mean delay in presentation was 5.7 months, likely due to delayed recognition and referral. The male-to-female ratio was 1.18:1, consistent with prior studies [1–3].

A symptomatic aetiology was identified in 62.9% of children, while 37.1% were cryptogenic. Perinatal risk factors were present in 22 children, with antenatal risks in 11. Birth asphyxia was the predominant perinatal factor, followed by neonatal hypoglycaemia and meningitis. Preterm birth was significantly associated with symptomatic West Syndrome. CNS malformations were found in 20 children, including cerebral atrophy, white matter injury, and polymicrogyria. Two children had tuberous sclerosis. No genetic or metabolic causes were identified, possibly due to limited investigations. These findings are consistent with previous studies [1–3].

Flexor spasms were the most common type (80%), followed by extensor and mixed. The mean number of spasms per cluster was 10.5, with a mean of 6.6 clusters per day. Sleep–wake cycle-related spasms were seen in 40%, most commonly on awakening.

Associated seizures developed in 37.1% of children, with two cases evolving into Lennox-Gastaut Syndrome.

Microcephaly was present in 36.4% of symptomatic cases, and 90.9% had pre-existing developmental delay, consistent with findings by Kaushik et al[3]. and Singhi et al[4]. At least one comorbidity was seen in 62.9% of children. Vision impairment was most frequent, followed by hearing impairment, feeding difficulties, and sleep disturbances. Vision impairment was significantly higher in symptomatic cases, as also reported by Sehgal et al[1]. and Kaushik et al[3]. One child had Down syndrome, aligning with literature highlighting increased incidence of infantile spasms and risk of autism spectrum disorder in these children[5–7].

A favourable epilepsy outcome (≥50% reduction in spasms) at six months was achieved in 85.7% of children, while complete cessation occurred in 62.9%. ACTH resulted in complete cessation in 66.6% and partial response in 24.2%. Vigabatrin showed no significant benefit in two children with tuberous sclerosis but offered partial improvement in three ACTH non-responders. These seizure outcomes are consistent with previous studies on epilepsy management and treatment response in West Syndrome [8,9].

Despite seizure control, developmental stagnation or regression persisted in most children, emphasizing the importance of early and aggressive intervention strategies [10]. Symptomatic cases had significant pre-existing developmental delay, whereas cryptogenic cases showed higher post-seizure regression. Delayed treatment initiation significantly impacted communication outcomes. Only 14.3% of children had favourable neurodevelopmental outcomes, and 37.1% had profound delay, similar to Sehgal et al[1]., who reported 8.4% favourable outcomes. In contrast, Partikian et al[11]. reported better cognitive outcomes in US-based populations.

This study highlights the clinical spectrum, aetiology, and outcomes of West Syndrome. The predominance of symptomatic cases aligns with prior studies [1–4], emphasizing the roles of perinatal and structural risk factors. The association between preterm birth and symptomatic cases underscores the need for enhanced neonatal care and early neurodevelopmental assessment.

Although seizure control was achieved in most children, neurodevelopmental outcomes remained poor, with only 14.3% showing improvement. This highlights the need for early intervention programs, including rehabilitation and special education, to reduce long-term disability. Multidisciplinary care involving neurology, developmental paediatrics, and rehabilitation services is essential to improve quality of life.

Study limitations include the small sample size, single-centre design, and short-term follow-up (six months). In addition, genetic and metabolic evaluations were limited, possibly underestimating these aetiologies. Future studies should involve larger multicentre cohorts with longer follow-up to better define prognostic factors and optimize therapeutic strategies[12].

Table 1. ***Demographic and Baseline Clinical Characteristics of the Study Groups***

Variable	***Symptomatic (n=22)*** ***Number (Percentage)***	***Cryptogenic (n=13)*** ***Number (Percentage)***	***P value***	***Interpretation***
Gender (Male/Female)	14 / 8	5 / 8	0.149	Not significant
Age <6 months at onset	4 (18.2%)	6 (46.2%)	0.083	Earlier onset in cryptogenic group
Preterm birth	13 (59.1%)	1 (7.7%)	0.003	Significant
Mode of delivery (LSCS)[b]	8 (36.4%)	2 (15.4%)	0.259	Not significant
Consanguinity	0 (0%)	2 (15.4%)	0.131	Not significant

Variable	*Symptomatic (n=22)* *Number (Percentage)*	*Cryptogenic (n=13)* *Number (Percentage)*	*P value*	*Interpretation*
Family history of IS[c]	2 (9.1%)	1 (7.7%)	1.000	Not significant
Pre-existing developmental delay	20 (90.9%)	4 (30.8%)	<0.01	Significant
Developmental regression	6 (27.3%)	8 (61.5%)	0.046	Significant
Microcephaly	8 (36.4%)	2 (15.4%)	0.259	Not significant
Facial dysmorphism	6 (27.3%)	0 (0%)	0.064	Borderline significance
Neurocutaneous markers	6 (27.3%)	8 (61.5%)	0.075	Trend toward cryptogenic group
Comorbidities (any)[d]	17 (77.3%)	5 (38.5%)		More common in symptomatic group
Associated seizures	8 (36.4%)	5 (38.5%)	0.901	Not significant

P <0.05 considered significant. Statistical tests used: Chi-square test or Fisher's exact test,

[b] Lower Segment Caesarean Section, [c] Infantile Spasm, [d] comorbidities assessed: visual impairment, hearing impairment, sleep disturbances, feeding difficulties, and recurrent infections

Table 2. ***Developmental and Clinical Outcomes at 6-Month Follow-Up***

Variable	*Symptomatic* *Mean score (SD)*	*Cryptogenic* *Mean score (SD)*	*P value*	*Interpretation*
[a]DP3 – Physical domain	58.27 (9.55)	66.46 (12.90)	0.039[b]	Significant difference
[a]DP3 – Adaptive domain	60.45 (11.23)	66.84 (11.98)	0.122	Not significant
[a]DP3 – Socio-emotional domain	60.60 (11.55)	64.84 (13.77)	0.344	Not significant
[a]DP3 – Cognitive domain	60.68 (14.07)	66.92 (16.15)	0.239	Not significant
[a]DP3 – Communicative domain	65.86 (14.61)	73.23 (16.27)	0.176	Not significant
[c]General Development Score (GDS)	48.50 (12.94)	56.00 (17.00)	0.150	Not significant
[d]E-CHESS score	8.27 (2.45)	7.85 (2.97)	0.649	Similar severity
	Symptomatic *Number (Percentage)*	*Cryptogenic* *Number (Percentage)*		
Complete seizure cessation	14 (63.6%)	8 (61.5%)		Comparable
Partial response	5 (22.7%)	3 (23.1%)		Comparable
No improvement	3 (13.6%)	2 (15.4%)		Comparable

[a]DP3: Developmental Profile-3, [b]P <0.05 considered significant. Statistical analysis used: Student's t-test (continuous), Chi-square or Fisher's exact test (categorical). [c]GDS: General Development Score (maximum = 100). [d]E-CHESS: Early Childhood Epilepsy Severity Scale (range: 0–15; higher = greater severity).

KEY MESSAGE
WHAT THIS STUDY ADDS

This study underscores symptomatic predominance in West Syndrome and highlights the need for early intervention to improve developmental outcomes.

Funding

No funding was received for this study.

Competing Interests

The authors declare no competing interests.

Ethics Approval

The study was approved by the Institutional Research and Ethics Committee, Aster Medcity, Kochi (Ref: AM/EC/67-2018; Date: 03/09/2018).

Consent to Participate

Informed consent was obtained from the parents or legal guardians of all individual participants included in the study.

Human Ethics and Consent to Participate Declaration

The study adhered to the principles of the Declaration of Helsinki. Institutional ethics approval was obtained, and informed consent was secured from parents or legal guardians.

Data Availability

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Exploring Dissociative Symptoms, Self-harm, and Somatisation in Kashmiri Children: Insights from a Hospital-Based Analysis

Dr. Sheikh Mushtaq[1], Mehreena Manzoor Tak[1], Sumiya Hamid[1], Tariq Ahmad Wani[2], Wakeel Ahmad Bhat[1], Zahoor Ahmad Wagay[1], Dr. Syed Karrar Hussain[1],

Aff1- Department of Paediatrics, Government Medical College Srinagar, Srinagar, Jammu & Kashmir, India.
Aff2- King Fahad Medical City, Riyadh, Saudi Arabia.

Address for correspondence
Correspondence :- Mehreena Manzoor Tak
(mehree96_ssh@jnu.ac.in)
9149690019
Department of Paediatrics, GMC Srinagar

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Abstract

Background: Children and Adolescents in conflict-affected regions like Kashmir face heightened mental health risks, including dissociative symptoms, somatisation, and deliberate self-harm (DSH), often driven by trauma, academic stress, and social challenges. There is limited clinical data on adolescent mental health in Kashmir. This study investigates key psychological presentations and their socio-demographic associations in a hospital setting.

Objectives of the Study: 1. To assess the prevalence and predictors of dissociation, self-harm, and somatisation in Kashmiri children. 2. Identify the frequency of dissociation, self-harm, and somatic complaints. 3. Examine associations with socio-demographic factors. 4. Determine predictors of dissociative symptoms

Methodology: A hospital-based cross-sectional study was conducted with 167 children. Data were analysed using SPSS v27. Age comparisons were made using the Mann–Whitney U test, while associations with categorical variables were assessed using Fisher's Exact test. Predictors of dissociation were identified through backward stepwise logistic regression.

Results: The sample consisted of 89.8% females with a mean age of 14.1 years. **Dissociative symptoms** were observed in 29.9% of participants, **somatic complaints** in 49.1%, and **self-harm/suicidal behaviours** in 21.6%. The most common method of self-harm was **organophosphate poisoning** (20.4%). Psychological symptoms, particularly dissociation and panic attacks, were significantly associated with self-harm. Logistic regression showed that adolescents in **9th–12th standard** were 3.48 times more likely to exhibit dissociative symptoms compared to those in lower grades (p=0.003). Children whose parents were in skilled/professional jobs were less likely to report dissociation (OR=0.3, p=0.034). The presence of **somatic complaints** and **self-harm behaviours** significantly predicted dissociative symptoms (both p<0.001).

Conclusion: Dissociation and self-harm are prevalent among Kashmiri children, especially in secondary school girls. School-based screening and context-sensitive interventions are urgently needed.

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- Children
- Kashmir
- Dissociation
- Somatisation
- Self-Harm
- Organophosphate Poisoning

Introduction

The formative years of childhood and adolescence are characterised by fast biological, psychological, and social change as well as increased susceptibility to mental health issues, particularly somatisation, dissociative symptoms, and deliberate self-harm (DSH). Trauma or extreme stress is frequently followed by dissociation, which is characterised as a disturbance in consciousness, memory, identity, or perception[1]. Clinical and longitudinal studies show that teenage dissociation serves as a risk indicator for suicidality and self-harming behaviours[2]. In children and adolescents, somatisation, the presentation of psychological distress as physical symptoms, often co-occurs with affective and dissociative psychopathology, especially in situations where cultural norms prevent the expression of emotions [3].

Cumulative adversities (political instability, exposure to violence, disruptions in education, and economic precarity) presumably increase these risks in conflict-affected areas like Kashmir[4] Research from the area and similar South Asian contexts shows that adolescents have higher rates of anxiety and depression symptoms [5], dissociative presentations, and suicidal behaviours, with stress from school and family strife serving as frequent triggers[6] [7]. However, there is still a dearth of systematic, hospital-based clinical data on the co-occurrence and correlates of DSH, somatisation, and dissociation in Kashmiri children, which limits the development of trauma-informed, context-sensitive interventions and service planning.

Aims and Objectives

Aim. To assess the prevalence and predictors of dissociation, self-harm, and somatisation among Kashmiri children attending a tertiary-care hospital.

Objectives:

1. To estimate the frequency of dissociative symptoms, self-harm behaviours, and somatic complaints.
2. To examine associations between these symptoms and socio-demographic factors (age, sex, schooling level, family structure, residence, parental occupation/education).
3. To identify predictors of dissociative symptoms using multivariable modelling.

Methodology

A descriptive cross-sectional study was conducted at the 500-bedded Children's Hospital, Srinagar, Jammu & Kashmir, India, from January to June 2024. Participants were recruited from adolescents referred by the Department of Paediatrics for psychological evaluation.

Sampling and recruitment

Consecutive sampling was employed: all eligible referrals during the study window were assessed for inclusion. Importantly, paediatricians first evaluated each case and excluded organic causes before referral to the psychiatric/psychological team.

Participants

Inclusion criteria

- Patients aged 7–18 years referred by paediatricians after clinical assessment had excluded organic causes of the presenting symptoms.
- First index presentation to the study team during the study period.
- Provision of written informed consent from the parent/guardian and assent from the adolescent, as applicable.

Exclusion criteria

- Patients with confirmed medical/neurological conditions explaining their symptoms (e.g., epilepsy, traumatic brain injury, metabolic/endocrine disorders).
- Patients with severe intellectual disability or communication impairment precluding reliable assessment.
- Patients with acute intoxication or delirium at the time of evaluation.
- Patients declining consent/assent or withdrawn by guardians.
- Repeat presentations of the same patient during the study period.

Assessment and study tools

Dissociative symptoms were evaluated through structured clinical interviews by psychiatrists using DSM-IV-TR criteria[1]. A structured diagnostic instrument such as the Structured Clinical Interview for DSM Disorders (SCID-I) was not employed; this is acknowledged as a limitation. Somatic and psychological complaints were identified through structured interviews with adolescents and guardians, supplemented by clinical observation. Self-harm behaviours were recorded if intentional self-injury or self-poisoning occurred, irrespective of suicidal intent, and details of the method and intent were documented. Clinicians screened for common comorbid psychiatric conditions (e.g., depression, anxiety, obsessive-compulsive symptoms) during evaluation. These were not systematically assessed with standardised instruments, which may have led to underreporting. Data quality safeguards included standardised case-record forms, consensus meetings within the psychiatric team for atypical cases, and de-duplication checks.

Variables and operational definitions

- Primary outcome: presence of dissociative symptoms as per DSM-IV-TR criteria.
- Secondary outcomes: self-harm behaviour (yes/no; method; stated intent) and somatic complaints (yes/no; type).
- Explanatory variables: age, sex, schooling level, family structure, residence (urban/rural), parental occupation, and parental education.

Statistical analysis

Data were analysed in IBM SPSS v27. Age (non-normally distributed) was summarised as median (IQR) and compared with the Mann–Whitney U test. Categorical variables were analysed using Chi-square or Fisher's Exact tests. Backward stepwise binary logistic regression identified predictors of dissociative symptoms. Diagnostics included multicollinearity checks (variance inflation factors) and goodness-of-fit (Hosmer–Lemeshow test). Adjusted odds ratios with 95% confidence intervals were reported, with significance set at $p < 0.05$.

Ethics

The study was approved by the Institutional Ethics Review Board, Government Medical College Srinagar (Ref. No.: IRBGMC-SGR/Pedia/845). Written informed consent/assent was obtained prior to participation.

Results

The study included 167 adolescents aged between 7 and 18 years, with a mean age of 14.1 ± 1.7 years and a median age of 14 (IQR: 13–15). The sample was predominantly female (89.8%). Most participants were in middle (43.1%) or secondary school (40.1%), and a large majority lived in urban areas (62.9%) and belonged to nuclear families (78.4%). Parental literacy levels were low, particularly among mothers, with 78.9% being illiterate. Nearly half of the participants (49.1%) had parents working in unskilled occupations (Table 1).

Table 1: Socio-demographic Characteristics of the studied Cases (N=167)

Characteristic	Description	N (%)
Age (year)	min - max	7 - 18
	Mean ± SD	14.1 ± 1.7
	Median (P25 - P75)	14 (13 - 15)
Sex	Female	150 (89.8%)
	Male	17 (10.2%)
Child's literacy status	Primary School Level (Ist-5th Standard)	17 (10.2%)
	Middle School Level (6th to 8th)	72 (43.1%)
	Secondary School Level (9th to 10th)	67 (40.1%)
	Higher Secondary School Level (11th to 12th)	11 (6.6%)

Occupation of Parents	Unskilled	82 (49.1%)
	Semi-skilled	41 (24.6%)
	Skilled	40 (24.0%)
	Professional	3 (1.8%)
	Household	1 (0.6%)
Type of family	Nuclear	131 (78.4%)
	Non-nuclear	36 (21.6%)
Family Status	Intact	154 (92.2%)
	Broken	13 (7.8%)
Residence District	Anantnag	11 (6.6%)
	Bandipora	7 (4.2%)
	Baramullah	12 (7.2%)
	Bijbehara	1 (0.6%)
	Budgam	35 (21.0%)
	Ganderbal	5 (3.0%)
	Kulgam	6 (3.6%)
	Kupwara	12 (7.2%)
	Other	7 (4.2%)
	Pulwama	23 (13.8%)
	Ramban	1 (0.6%)
	Shopian	4 (2.4%)
	Srinagar	43 (25.7%)
Dwelling	Rural	62 (37.1%)
	Urban	105 (62.9%)
Father's Education	Illiterate	88 (56.8%)
	Primary School Level (8th-9th)	8 (5.2%)
	Secondary Level (10th to 12th)	42 (27.1%)
	Higher Education (Graduation, M.A, D. Pharma)	17 (11.0%)
Mother's Education	Illiterate	112 (78.9%)
	Primary School Level (8th-9th)	1 (0.7%)
	Secondary Level (10th to 12th)	25 (17.6%)
	Higher Education (Graduation, M.A, D. Pharma)	4 (2.8%)

Clinically, **somatic complaints** were reported by 49.1% of participants, with anxiety and body ache being the most common symptoms. **Psychological symptoms** were present in 35.3% of the adolescents, with **dissociative symptoms** accounting for the majority (29.9%). Self-harm or suicidal behaviours were reported in 21.6% of the sample, with **organophosphate poisoning** being the most common method (20.4%) (Table 2).

Table 2: Symptoms and Chief Complaints in the studied Cases (N=167)		
Characteristic	Description	N (%)
Somatic/Physical Complaints		82 (49.1%)
Number of Somatic/Physical Complaints	One	73 (43.7%)
	Two	9 (5.4%)
Somatic/Physical Complaints type	Anxiety	14 (8.4%)
	Body Ache	14 (8.4%)
	Palpitation	11 (6.6%)
	Headache	10 (6.0%)
	Breathlessness	6 (3.6%)
	Aggression	5 (3.0%)
	Stomach Ache	4 (2.4%)
	Abdomen Pain	3 (1.8%)
	Chest Discomfort	3 (1.8%)
	Seizures	3 (1.8%)
	Chest Pain	2 (1.2%)
	Ticks	2 (1.2%)
	Hematemesis	2 (1.2%)
	Joint Pain	1 (0.6%)
	Leg Pain	1 (0.6%)
	Vomiting	1 (0.6%)
	Heart Palpitation	1 (0.6%)
	Bipolar Disorder	1 (0.6%)
	Coughing	1 (0.6%)
	Pain	1 (0.6%)
	Pain in lower limb	1 (0.6%)
	Anemia	1 (0.6%)
	Blood With Stool	1 (0.6%)
	Esophageal Reflex Grade A	1 (0.6%)
	Enuresis	1 (0.6%)
Psychological Symptoms		59 (35.3%)
Number of Psychological Symptoms	One	54 (32.3%)
	Two	4 (2.4%)
	Three	1 (0.6%)

Psychological Symptom type	Psychological Symptoms-Depression	1 (0.6%)
	Mood Swings	1 (0.6%)
	Forgetfulness	1 (0.6%)
	Dissociation	50 (29.9%)
	Panic Attack	5 (3.0%)
	Lost Consciousness	5 (2.4%)
	OC Symptoms	1 (0.6%)
	Low I.Q	1 (0.6%)
Self-harm/Suicidal Behavior		36 (21.6%)
Number of Self-harm/Suicidal Behavior	One	36 (21.6%)
Self-harm/Suicidal Behavior type	OP poisoning	34 (20.4%)
	Poisoning (Rat Poision)	1 (0.6%)
	Suicide (Hanging)	1 (0.6%)

Early childhood trauma (ECT) was reported by 17.4% of participants. The most frequent causes were family conflict (20.7%), bullying (17.2%), and child sexual abuse (17.2%). Among those reporting self-harm, 67.3% indicated intent to die, while others reported a desire to inflict pain or were unable to articulate their intent. The most frequent methods of deliberate self-harm (DSH) included poisoning (57.1%) and self-inflicted cuts (38.8%) (Table 3).

Table 3: Early Childhood Trauma and DSH in the studied Cases (N=167)

Characteristic	Description	N (%)
Early Childhood Trauma		29 (17.4%)
Cause for Early Childhood Trauma	Accident on Scooty	1 (3.4%)
	Bullying	5 (17.2%)
	chased by dogs which caused her head injury	1 (3.4%)
	Child Sexual Abuse	5 (17.2%)
	Dissociation has given her trauma	1 (3.4%)
	Family Conflict	6 (20.7%)
	Father had passed away	2 (6.9%)
	Had to leave old school	1 (3.4%)
	Mother Died	4 (13.8%)
	Parents divorced	2 (6.9%)
	Separation from parents in Childhood	1 (3.4%)
Only Having thoughts or actions also	DSH	49 (29.3%)
	Only self harm thoughts	45 (26.9%)
	None	73 (43.7%)

Intent of DSH	To kill herself	33 (67.3%)
	To harm & inflict pain	11 (22.4%)
	Didn't Reveal	3 (6.1%)
	Not Known	2 (4.1%)
Mode of DSH	Poisoning	28 (57.1%)
	Cuts	19 (38.8%)
	Burns	1 (2.0%)
	Jumped from Waranda	1 (2.0%)
	Tried hanging	1 (2.0%)

When stratified by self-harm risk, adolescents who had engaged in DSH were significantly older (mean age = 14.8 years) than those with only self-harm thoughts (14.3 years) or no self-harm (13.4 years) ($p < 0.001$). Educational level was also significantly associated with self-harm status ($p < 0.001$), with the majority of DSH cases found among students in secondary school. No significant associations were found between self-harm and gender, family structure, dwelling type, or parental education. However, psychological symptoms, especially dissociation, were significantly more prevalent in the DSH group ($p = 0.003$), while somatic complaints were more common in the self-harm thoughts group ($p = 0.016$) (Table 4).

Table 4: DSH and Self harm in association with the studied factors

Characteristic	Description	None	Self-harm thoughts	DSH	p-value
		N = 73 (43.7%)	N = 45 (26.9%)	N = 49 (29.3%)	
Age (year)	min - max	7 - 17	11 - 18	12 - 17	<0.001
	Mean ± SD	13.4 ± 1.9	14.3 ± 1.4	14.8 ± 1.3	
	Median (P25 - P75)	14 (12 - 15)	14 (13 - 15)	15 (14 - 16)	
Sex	Female	64 (87.7%)	41 (91.1%)	45 (91.8%)	0.716
	Male	9 (12.3%)	4 (8.9%)	4 (8.2%)	
Child's literacy status	Primary School Level (Ist-5th Standard)	14 (19.2%)	3 (6.7%)	0 (0.0%)	<0.001
	Middle School Level (6th to 8th)	34 (46.6%)	23 (51.1%)	15 (30.6%)	
	Secondary School Level (9th to 10th)	21 (28.8%)	16 (35.6%)	30 (61.2%)	
	Higher Secondary School Level (11th to 12th)	4 (5.5%)	3 (6.7%)	4 (8.2%)	
Occupation of Parents	Unskilled	35 (47.9%)	23 (51.1%)	25 (51.0%)	0.316
	Semi-skilled	17 (23.3%)	8 (17.8%)	16 (32.7%)	
	Skilled/Professional	21 (28.8%)	14 (31.1%)	8 (16.3%)	

Type of family	Nuclear	54 (74.0%)	40 (88.9%)	37 (75.5%)	0.134
	Non-nuclear	19 (26.0%)	5 (11.1%)	12 (24.5%)	
Family Status	Intact	66 (90.4%)	43 (95.6%)	45 (91.8%)	0.708
	Broken	7 (9.6%)	2 (4.4%)	4 (8.2%)	
Dwelling	Rural	28 (38.4%)	15 (33.3%)	19 (38.8%)	0.826
	Urban	45 (61.6%)	30 (66.7%)	30 (61.2%)	
Father's Education	Illiterate	38 (55.9%)	26 (61.9%)	24 (53.3%)	0.666
	Primary School Level (8th-9th)	5 (7.4%)	1 (2.4%)	2 (4.4%)	
	Secondary Level (10th to 12th)	15 (22.1%)	12 (28.6%)	15 (33.3%)	
	Higher Education (Graduation, M.A, D. Pharma)	10 (14.7%)	3 (7.1%)	4 (8.9%)	
Mother's Education	Illiterate	52 (81.3%)	31 (75.6%)	29 (78.4%)	0.668
	Primary School Level (8th-9th)	0 (0.0%)	1 (2.4%)	0 (0.0%)	
	Secondary Level (10th to 12th)	10 (15.6%)	7 (17.1%)	8 (21.6%)	
	Higher Education (Graduation, M.A, D. Pharma)	2 (3.1%)	2 (4.9%)	0 (0.0%)	
Number of Somatic/ Physical Complaints	None	37 (50.7%)	16 (35.6%)	32 (65.3%)	0.053
	One	33 (45.2%)	25 (55.6%)	15 (30.6%)	
	Two	3 (4.1%)	4 (8.9%)	2 (4.1%)	
Somatic/Physical Complaints	No	37 (50.7%)	16 (35.6%)	32 (65.3%)	0.016
	Yes	36 (49.3%)	29 (64.4%)	17 (34.7%)	
Number of Psycho-logical Symptoms	None	53 (72.6%)	33 (73.3%)	22 (44.9%)	0.007
	One	17 (23.3%)	12 (26.7%)	25 (51.0%)	
	Two	2 (2.7%)	0 (0.0%)	2 (4.1%)	
	Three	1 (1.4%)	0 (0.0%)	0 (0.0%)	
Psychological Symptoms	No	53 (72.6%)	33 (73.3%)	22 (44.9%)	0.003
	Yes	20 (27.4%)	12 (26.7%)	27 (55.1%)	
Self-harm/Suicidal Behavior	No	51 (69.9%)	36 (80.0%)	44 (89.8%)	0.069
	Yes	22 (30.1%)	9 (20.0%)	5 (10.2%)	
Early Childhood Trauma	No	64 (87.7%)	35 (77.8%)	39 (79.6%)	0.309
	Yes	9 (12.3%)	10 (22.2%)	10 (20.4%)	

In comparing adolescents with and without dissociative symptoms, those with dissociation were significantly older (mean = 14.8 years) and more likely to be in 9th–12th grade (69.4%) ($p < 0.001$). They

also had higher rates of psychological symptoms (55.1% vs. 27.1%, $p < 0.001$) and were more likely to report self-harm ($p = 0.023$). In contrast, somatic complaints were significantly more common among adolescents without dissociation ($p = 0.018$). No significant associations were found between dissociation and gender, family structure, residence, or parental education (Table 5).

Table 5: Dissociative Symptom in association with the studied factors

Characteristic	Description	Non-DSH N = 118 (70.7%)	DSH N = 49 (29.3%)	Total	p-value
Age (year)	min - max	7 - 18	12 - 17	7 - 18	<0.001
	Mean ± SD	13.8 ± 1.8	14.8 ± 1.3	14.1 ± 1.7	
	Median (P25 - P75)	14 (13 - 15)	15 (14 - 16)	14 (13 - 15)	
Sex	Female	105 (89)	45 (91.8)	150 (89.8)	0.78
	Male	13 (11)	4 (8.2)	17 (10.2)	
Child's literacy status	Upto 8th	74 (62.7)	15 (30.6)	89 (53.3)	<0.001
	9th to 12th	44 (37.3)	34 (69.4)	78 (46.7)	
Occupation of Parents	Unskilled	58 (49.2)	25 (51)	83 (49.7)	0.118
	Semi-skilled	25 (21.2)	16 (32.7)	41 (24.6)	
	Skilled/Professional	35 (29.7)	8 (16.3)	43 (25.7)	
Type of family	Nuclear	94 (79.7)	37 (75.5)	131 (78.4)	0.553
	Non-nuclear	24 (20.3)	12 (24.5)	36 (21.6)	
Family Status	Intact	109 (92.4)	45 (91.8)	154 (92.2)	>0.999
	Broken	9 (7.6)	4 (8.2)	13 (7.8)	
Dwelling	Rural	43 (36.4)	19 (38.8)	62 (37.1)	0.776
	Urban	75 (63.6)	30 (61.2)	105 (62.9)	
Father's Education	Illiterate	64 (58.2)	24 (53.3)	88 (56.8)	0.746
	Primary School Level (8th-9th)	6 (5.5)	2 (4.4)	8 (5.2)	
	Secondary Level (10th to 12th)	27 (24.5)	15 (33.3)	42 (27.1)	
	Higher Education	13 (11.8)	4 (8.9)	17 (11)	
Mother's Education	Illiterate	83 (79)	29 (78.4)	112 (78.9)	0.701
	Primary School Level (8th-9th)	1 (1)	0 (0)	1 (0.7)	
	Secondary Level (10th to 12th)	17 (16.2)	8 (21.6)	25 (17.6)	
	Higher Education	4 (3.8)	0 (0)	4 (2.8)	

Somatic/Physical Complaints	No	53 (44.9)	32 (65.3)	85 (50.9)	0.018
	Yes	65 (55.1)	17 (34.7)	82 (49.1)	
	No	86 (72.9)	22 (44.9)	108 (64.7)	<0.001
	Yes	32 (27.1)	27 (55.1)	59 (35.3)	
	Yes	31 (26.3)	5 (10.2)	36 (21.6)	
	Yes	19 (16.1)	10 (20.4)	29 (17.4)	

A backward stepwise binary logistic regression identified four significant predictors of dissociative symptoms. Adolescents in higher grades (9th–12th) were 3.48 times more likely to report dissociation ($p = 0.003$). Those whose parents had skilled or professional occupations were significantly less likely to report dissociation (OR = 0.3, $p = 0.034$). Additionally, the presence of somatic complaints (OR = 0.11, $p < 0.001$) and self-harm/suicidal behavior (OR = 0.11, $p < 0.001$) were both inversely associated with dissociative symptoms (Table 6).

Table 6: Backstep logistic regression analysis predicting association of Dissociative Symptom with the Child's literacy status, Occupation of Parents, Somatic/Physical Complaints, and Self-harm/Suicidal Behavior in the final 10th step of the modal.

Variable(s) entered on step 1: Age (year), Sex, Child's literacy status, Occupation of Parents, Type of family, Family Status, Dwelling, Father's Education, Mother's Education, Somatic/Physical Complaints, Psychological Symptoms, Self-harm/Suicidal Behavior, Early Childhood Trauma.

	Characteristic	Description		OR (LL UL)	p-value
Step-01	Age (year)	min - max		1.05 (0.87 1.26)	0.606
	Sex	Female	Reference		
		Male		0.48 (0.07 3.23)	0.451
	Child's literacy status	Up to 8th	Reference		
		9th to 12th		4.41 (1.4 13.87)	0.011
	Occupation of Parents	Unskilled	Reference		
		Semi-skilled		1.6 (0.5 5.08)	0.427
		Skilled/Professional		0.15 (0.03 0.9)	0.038
	Type of family	Nuclear	Reference		
		Non-nuclear		0.98 (0.28 3.38)	0.974
	Family Status	Intact	Reference		
		Broken		2.46 (0.3 20.15)	0.402
	Dwelling	Rural	Reference		
		Urban		0.74 (0.28 1.98)	0.553
	Father's Education	Illiterate	Reference		
		Primary School Level (8th-9th)		0.86 (0.07 10.85)	0.904
		Secondary Level (10th to 12th)		3.31 (0.68 16.21)	0.139
		Higher Education		6.94 (0.81 59.84)	0.078

	Mother's Education	Illiterate	Reference		
		Primary School Level (8th-9th)		0 (0)	1.000
		Secondary Level (10th to 12th)		1.17 (0.26 5.3)	0.838
		Higher Education		0 (0)	0.999
	Somatic/Physical Complaints	No	Reference		
		Yes		0.04 (0 0.45)	0.009
	Psychological Symptoms	No	Reference		
		Yes		0.27 (0.03 2.91)	0.282
	Self-harm/Suicidal Behavior	No	Reference		
		Yes		0.03 (0 0.46)	0.011
	Early Childhood Trauma	No	Reference		
		Yes		1.56 (0.37 6.6)	0.547
Step-10	Child's literacy status	Up to 8th	Reference		
		9th to 12th		3.48 (1.54 7.85)	0.003
	Occupation of Parents	Unskilled	Reference		
		Semi-skilled		1.25 (0.49 3.24)	0.641
		Skilled/Professional		0.3 (0.1 0.91)	0.034
	Somatic/Physical Complaints	No	Reference		
		Yes		0.11 (0.05 0.28)	<0.001
	Self-harm/Suicidal Behavior	No	Reference		
		Yes		0.11 (0.03 0.34)	<0.001

Discussion

This study investigates the prevalence and correlates of dissociative symptoms, self-harm behaviours, and somatisation in children at a hospital in Kashmir. Of the 167 participants, 29.9% reported dissociative symptoms, while 29.3% revealed a history of deliberate self-harm, and 49.1% reported somatic complaints. Our regression analysis indicated that higher education levels (OR = 3.48, p = .003), somatic complaints (OR = 0.11, p < .001), and self-harm behaviour were significantly associated with dissociative symptoms

These findings are in line with an emerging body of research positioning dissociation as an important marker of psychopathological risk in adolescence. For example, in a large-scale cohort study, Tanaka et al. showed that persistent dissociative symptoms predicted future self-harm (OR = 2.61[8], thus underlining the concept of dissociation being an antecedent rather than a consequence of self-injurious behaviour. Correspondingly, in a study on adolescents with a history of childhood sexual abuse, it was found that dissociation was a strong predictor of non-suicidal self-injury NSSI, with higher parent-reported levels of dissociation scores independently associated with both NSSI and suicide attempts.

Somatisation was a prevailing condition and strongly associated with dissociative symptoms in our sample, which corroborates the findings of Raffagnato et al. (2020), who demonstrated that adolescents characterised by alexithymia

and somatic complaints show more severe psychopathological profiles and a higher risk for self-injurious behaviours[9]. The overlap between bodily symptoms and emotional distress was also reflected in the current sample, with headaches, palpitations, and psychogenic seizures being outstanding in this respect. This suggests that in an emotionally repressive or trauma-exposed environment, the somatic expression becomes a surrogate for unresolved psychological pain.

Moreover, the current study provides further confirmation of the association of early trauma with the development of dissociative and self-injurious behaviour. While only 17.4% of our subjects reported identifiable early childhood trauma, this subgroup disproportionately displayed psychological symptoms and DSH. Luoni et al. (2018) found that children exposed to complex trauma were more likely to present with dissociation, somatic complaints, and mood or psychotic disorders, further supporting a trauma-symptom linkage [10]

Interestingly, higher levels of education, for the 9th–12th-grade category, were significantly associated with dissociative symptoms, with an odds ratio of 3.48, perhaps reflecting greater academic and social pressures faced by older children and adolescents. Previous Indian studies also reported school-related stress as a common precipitant for dissociative episodes. Moyon et al., (2021), in their results, indicate that in the absence of well-structured school-based mental health systems, the academic environment is likely to inadvertently emerge as a source of psychological distress.

The observed gender distribution, with females constituting nearly 90% of the sample, is in keeping with previous studies that have reported a higher prevalence of dissociative and self-harming behaviours among girls. Cultural scripts about emotional expression, internalised distress, and social role expectations might explain this gendered vulnerability.

While the majority of participants indeed came from urban areas and were raised in intact, nuclear families, such protective structural factors failed to significantly buffer them in the development of dissociative symptoms and self-harm. This strongly confirms the need to explore not only family structure but also aspects of emotional climate and relational dynamics within families—areas often neglected by the more common assessment tools

Limitations

This study has several limitations. First, it was conducted in a hospital-based referral sample; therefore, the prevalence estimates may not be generalisable to adolescents in the wider community. Second, while dissociative symptoms were evaluated using DSM-IV-TR based structured clinical interviews, standardized diagnostic instruments such as the SCID-I were not employed, which may affect diagnostic reliability. Third, although comorbid psychiatric conditions (e.g., depression, anxiety, obsessive-compulsive symptoms) were clinically screened, they were not systematically assessed with validated tools, leading to possible underreporting. Fourth, the cross-sectional design prevents establishing causal relationships between dissociation, somatisation, and self-harm. Fifth, an *a priori* sample size calculation was not conducted because the study was exploratory in nature and based on consecutive hospital referrals during the study period. As such, the sample size was determined by the flow of eligible cases rather than statistical power considerations. Finally, the relatively small and female-predominant sample limits subgroup analyses; future research should include larger, community-based, and more gender-balanced cohorts.

Conclusion

Our findings highlight dissociation as a critical transdiagnostic marker linked with both self-harm and somatisation in children. These symptoms likely reflect latent emotional dysregulation shaped by early trauma, academic pressure, and limited avenues for emotional expression. Integrating trauma-informed approaches and school-based mental health interventions may be crucial for early identification and support in high-risk populations like Kashmiri children & adolescents

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