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Original Research Article

CLINICAL PROFILE OF PERSONS WITH INTELLECTUAL DISABILITY: ASSOCIATION OF PARENTAL CONSANGUINITY WITH SEVERITY, COMORBIDITIES AND BEHAVIOURAL PROBLEMS

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ABSTRACT

Background: Intellectual disability (ID) is a neurodevelopmental disorder characterised by limitations in intellectual functioning and adaptive behaviour. It is often accompanied by comorbidities that exacerbate impairment and increase the caregiver burden. Parental consanguinity may increase the risk of autosomal recessive disorders that cause ID; however, its association with ID severity, comorbidities, and behavioural issues remains unclear. **Objectives:** To characterise parental consanguinity, intellectual disability severity, medical and psychiatric comorbidities, and behavioural problems; compare these outcomes by consanguinity history; and assess their associations with consanguinity.

Materials and Methods: This cross-sectional study, conducted at Government General Hospital, Vijayawada, included 100 individuals aged 5 years and older with ID and their caregivers, selected by convenience sampling. We used the VSMS and the Aberrant Behaviour Checklist for assessment. We analysed the data using chi-square, Fisher–Freeman–Halton, and Mann–Whitney U tests. We reported effect sizes as Cramer's V and rank-biserial correlation. We set statistical significance at $p < 0.05$.

Results: Of the 100 participants, 48% had parental consanguinity, 60% were male, 40% were aged 10 years or younger, and 58% belonged to a lower-middle socioeconomic group. Third-degree consanguinity was the most common (58.3%). Mild and moderate ID affected 51% and 43% of participants, respectively. ID severity was higher among participants with consanguinity ($U=1508.5$, $p=0.043$), although the categorical analysis was not statistically significant ($\chi^2=5.046$, $p=0.080$). We observed no significant associations between consanguinity and comorbidities.

Conclusion: Participants with parental consanguinity tended to have greater ID severity, although the categorical analysis did not reach statistical significance. Consanguinity was not significantly associated with medical, psychiatric, or behavioural issues, suggesting that it may inform clinical assessment but does not determine severity or related features.

Keywords: Intellectual Disability, Parental Consanguinity, Comorbidities, Behavioural Problems.

INTRODUCTION

Intellectual disability (ID) is a neurodevelopmental disorder characterised by significant difficulties in intellectual functioning and adaptive behaviour that arise during development. Difficulties in adaptive functioning affect the conceptual, social, and practical aspects of daily life, impacting individuals, families, and healthcare systems.[1] The WHO, via ICD-11, provides a standardised framework for

diagnosing and classifying ID [1]. It is a global public health concern, with prevalence varying by population, criteria, age, and case-ascertainment methods. A meta-analysis estimated a prevalence of about 10.4 per 1,000 people, though figures varied.[2] In India, Russell et al. reported a lifetime prevalence of around 2% among children and adolescents, with an adjusted estimate of 1.4%.[3] These figures indicate a significant burden and highlight the need for proper identification, assessment, and support services.

Individuals with intellectual disability frequently have associated medical, psychiatric, developmental, and behavioural conditions. Common comorbidities include epilepsy, cerebral palsy, autism spectrum disorder, attention-deficit/hyperactivity disorder, sensory impairments, and other medical or psychiatric disorders.[4,11] These comorbidities may further affect adaptive functioning, quality of life, service requirements, and caregiver burden.[4,11] Behavioural problems are also common among individuals with intellectual disability. These may include irritability, aggression, hyperactivity, self-injurious behaviour, stereotyped behaviour, and other challenging behaviours. Their frequency and severity may contribute substantially to caregiver burden and difficulties in daily functioning.[5,6]

Parental consanguinity is an important genetic and demographic factor in intellectual disability. Consanguineous unions increase the likelihood that offspring inherit identical pathogenic variants, particularly those causing autosomal recessive disorders, and are associated with certain congenital and genetic conditions.[7,8] Such unions remain common in South Asia, the Middle East, and North Africa.[8] In India, prevalence varies by region; the National Family Health Survey-4 reports 9.9% among women aged 15–49, with higher rates in the south.[9] Research across populations has examined links between consanguinity and intellectual disability. Consanguinity may increase the risk of recessive conditions that cause intellectual disability, but it does not alone establish its genetic cause.[7,8] Indian studies also report familial intellectual disability and other neurodevelopmental or neurological conditions in consanguineous families.[10,12] However, evidence remains limited on whether consanguinity relates to intellectual disability severity, medical and psychiatric comorbidities, or behavioural problems.

Therefore, the present study was undertaken to assess the clinical profile of individuals with intellectual disability and to examine the association between parental consanguinity and the severity of intellectual disability, as well as with medical and psychiatric comorbidities and behavioural problems. Understanding these associations may improve clinical assessment, help identify associated conditions, and enable appropriate counselling and support for affected individuals and their families.

MATERIALS AND METHODS

Place of study: Department of Psychiatry, Government General Hospital, Siddhartha Medical College, Vijayawada

Type of study: Cross-sectional observational study

Study Sample: Persons with Intellectual disability and their caregivers, where applicable.

Type of sampling: Convenience sampling

Sample Size: 100

Inclusion Criteria

1. Those who give consent to participate in the study
2. Persons who fulfil the ICD-11 diagnostic criteria for Intellectual Disability
3. Caregivers/informants available to provide relevant clinical and behavioural information, where required.
4. Patients aged over 5 years, of either gender

Exclusion Criteria

1. Caregiver/informant unable or unwilling to provide reliable information or uncooperative during assessment.

Study Tools

1. Informed written consent form
2. Semi-structured proforma to collect the data
3. **ICD-11:** The ICD-11 diagnostic criteria were used to establish the diagnosis and classification of intellectual disability.[1]
4. **Modified B. G. Prasad Socioeconomic Status Scale:** Socioeconomic status was assessed using the 2026 revised Modified B. G. Prasad scale, which classifies families into five socioeconomic classes based on monthly per-capita family income.[14]
5. **Vineland Social Maturity Scale (VSMS):** The VSMS was used to assess adaptive behaviour and social maturity. It provides estimates of Social Age and Social Quotient based on functioning across different domains of adaptive behaviour.[13]
6. **Aberrant Behaviour Checklist (ABC):** The ABC is a 58-item caregiver-rated scale used to assess behavioural problems in individuals with intellectual and developmental disabilities across five domains: irritability, lethargy/social withdrawal, stereotypic behaviour, hyperactivity/noncompliance, and inappropriate speech.[5]

Study Procedure: After obtaining approval from the Institutional Ethics Committee, we recruited participants who met the inclusion criteria. We used a semi-structured pro forma to collect participants' sociodemographic details, then administered the VSMS and ABC.

Statistical Analysis: Data were analysed using SPSS Statistics 28.0. Categorical variables were reported as frequencies and percentages; continuous variables as mean ± standard deviation (SD) or median and interquartile range (IQR), as appropriate. We assessed normality before selecting statistical tests.

We examined associations between parental consanguinity and intellectual-disability severity using Pearson's chi-square test or, for small expected cell counts, the Fisher–Freeman–Halton exact test. We also compared severity distributions using the Mann–Whitney U test, as severity was ordinal. We compared medical and psychiatric comorbidities between groups using the Fisher–Freeman–Halton exact test. Cramer's V was used to describe association strength where appropriate. We summarised Aberrant Behaviour Checklist (ABC) scores as median (IQR) and compared them using the Mann–Whitney U test, with rank-biserial correlation as the effect-size measure. Tests were two-tailed; $p < 0.05$ was considered statistically significant.

RESULTS

Table 1: Socio-Demographic Characteristics (N = 100)

	Variable	n (%)	Consanguinity	
			Present (n=48), n (%)	Absent (n=52), n (%)
AGE (in years)	10 and younger	40 (40%)	18 (37.5)	22 (42.3)
	11 – 20	31 (31%)	15 (31.3)	16 (30.8)
	21 - 30	17 (17%)	8 (16.7)	9 (17.3)
	31 and above	12 (12%)	7 (14.6)	5 (9.6)
GENDER	Male	60 (60%)	29 (60.4)	31 (59.6)
	Female	40 (40%)	19 (39.6)	21 (40.4)
SOCIOECONOMIC STATUS	Upper	2 (2%)	0 (0.0)	2 (3.8)
	Upper Middle	4 (4%)	1 (2.1)	3 (5.8)
	Middle	33 (33%)	16 (33.3)	17 (32.7)
	Lower Middle	58 (58%)	29 (60.4)	29 (55.8)
	Lower	3 (3%)	2 (4.2)	1 (1.9)

The study included 100 participants with intellectual disability. Parental consanguinity was present in 48 (48%) participants and absent in 52 (52%). By age, 40 (40%) were 10 years or younger, 31 (31%) were 11–20 years, 17 (17%) were 21–30 years, and 12 (12%) were 31 years or older. Among participants with parental consanguinity, 18 (37.5%) were 10 years or younger, 15 (31.3%) were 11–20 years, 8 (16.7%) were 21–30 years, and 7 (14.6%) were 31 years or older. Among those without parental consanguinity, the corresponding figures were 22 (42.3%), 16 (30.8%), 9 (17.3%), and 5 (9.6%), respectively. The cohort included 60 (60%) males and 40 (40%) females. The consanguinity-present group comprised 29 (60.4%) males and 19 (39.6%) females, whereas the consanguinity-absent group comprised 31 (59.6%) males and 21 (40.4%) females. Regarding socioeconomic status, most participants were in the lower-middle group (58%), followed by the middle group (33%). The upper-middle, lower, and upper groups accounted for 4%, 3%, and 2%, respectively. In the consanguinity-present group, 29 (60.4%) were in the lower-middle group and 16 (33.3%) in the middle group. In the consanguinity-absent group, 29 (55.8%) were in the lower-middle group and 17 (32.7%) in the middle group.

Table 2: Parental Consanguinity (N = 100) & Degree of Consanguinity

Consanguinity	n (%)	Degree	n (%)	% of consanguineous subgroup
Present	48 (48%)	2nd degree	13 (13%)	27.1
		3rd degree	28 (28%)	58.3
		4th degree	5 (5%)	10.4
		5th degree	2 (2%)	4.2
Absent	52 (52%)	NA	NA	NA

Among the 48 participants with parental consanguinity, 28 (58.3%) had third-degree consanguinity, 13 (27.1%) second-degree, 5 (10.4%) fourth-degree, and 2 (4.2%) fifth-degree.

Table 3: Severity of Intellectual Disability According to Parental Consanguinity

Severity category	n (%)	Consanguinity n (%)		Cumulative %
		Present (n=48)	Absent (n=52)	
Mild	51 (51%)	19 (39.6%)	32 (61.5%)	51.0
Moderate	43 (43%)	26 (54.2%)	17 (32.7%)	94.0
Severe	6 (6%)	3 (6.3%)	3 (5.8%)	100.0
Total	100	48	52	

Inferential Analysis of Consanguinity Versus Severity of Intellectual Disability

Test	Statistic	df	p-value
Pearson χ^2	5.046	2	0.080
Fisher–Freeman–Halton exact	—	—	0.080
Mann–Whitney U (ordinal code)	U = 1508.5	—	0.043

Effect Sizes for Consanguinity Versus Severity of Intellectual Disability

Effect size	Value	Interpretation
Cramer's V	0.225	Small–moderate association
Rank-biserial correlation	−0.209	Small effect

Of the 100 participants, 51 (51%) had mild intellectual disability, 43 (43%) had moderate intellectual disability, and 6 (6%) had severe intellectual disability. Among participants with parental consanguinity, 19 (39.6%) had mild, 26 (54.2%) had moderate, and 3 (6.3%) had severe intellectual disability. Among those without parental consanguinity, 32 (61.5%) had mild, 17 (32.7%) had moderate, and 3 (5.8%) had severe intellectual disability.

Severity categories did not differ significantly between groups (Pearson's chi-square test: $\chi^2 = 5.046$, df = 2, p = 0.080). However, when severity was analysed as an ordinal variable, the Mann–Whitney U test showed a statistically significant difference between the two groups (U = 1508.5, p = 0.043), with a shift towards higher severity categories in the consanguinity-present group. Participants with parental consanguinity showed a modest shift towards higher ordinal severity scores, although the categorical severity distribution did not differ significantly on chi-square testing. The effect size was Cramer's V = 0.225, indicating a small association, while the rank-biserial correlation was −0.209, indicating a small effect.

Table 4: Medical Comorbidities and their association with Consanguinity

Medical comorbidity category	Overall, n (%)	Present (n=48), n (%)	Absent (n=52), n (%)	Test statistic/p-value
Cerebral Palsy	3 (3.0)	2 (4.2)	1 (1.9)	Fisher–Freeman–Halton exact p = 0.786
Epilepsy	22 (22.0)	11 (22.9)	11 (21.2)	
Nil	75 (75.0)	35 (72.9)	40 (76.9)	
Total	100 (100.0)	48 (100.0)	52 (100.0)	

Epilepsy was present in 22 (22%) participants, while cerebral palsy was present in 3 (3%). Overall, 75 (75%) participants had no recorded medical comorbidity. Epilepsy was present in 11 (22.9%) participants with parental consanguinity and 11 (21.2%) without. Cerebral palsy was present in 2 (4.2%) and 1 (1.9%), respectively. Medical comorbidities did not differ significantly between the two groups (Fisher–Freeman–Halton exact test, p = 0.786).

Table 5: Psychiatric Comorbidities and their association with Consanguinity

Psychiatric comorbidity category	Overall, n (%)	Present (n=48), n (%)	Absent (n=52), n (%)	Test statistic/p-value
ADHD	15 (15.0)	6 (12.5)	9 (17.3)	Fisher–Freeman–Halton exact p = 0.703; Cramer's V = 0.148.
ASD	10 (10.0)	4 (8.3)	6 (11.5)	
ADHD & ASD	9 (9.0)	6 (12.5)	3 (5.8)	
Others	3 (3.0)	1 (2.1)	2 (3.8)	
Nil	63 (63.0)	31 (64.6)	32 (61.5)	
Total	100 (100.0)	48 (100.0)	52 (100.0)	

Among the participants, 15 (15%) had ADHD, 10 (10%) had ASD, 9 (9%) had both ADHD and ASD, 3 (3%) had other psychiatric comorbidities, and 63 (63%) had no psychiatric comorbidity. Among participants with parental consanguinity, ADHD was present in 6 (12.5%), ASD in 4 (8.3%), both ADHD and ASD in 6 (12.5%), and other psychiatric comorbidities in 1 (2.1%). Among those without parental consanguinity, the corresponding figures were 9 (17.3%), 6 (11.5%), 3 (5.8%), and 2 (3.8%), respectively. The distribution of psychiatric comorbidities did not differ significantly between participants with and without parental consanguinity (Fisher–Freeman–Halton exact test, p = 0.703; Cramer's V = 0.148).

Table 6: Behavioural problems and their association with Parental Consanguinity

ABC Domain	Consanguinity Present (n=48), Median (IQR)	Consanguinity Absent (n=52), Median (IQR)	Mann–Whitney U	Rank-biserial r	p-value
ABC Total	66.0 (53.0–91.0)	63.0 (51.8–87.5)	1378.0	−0.104	0.371
Irritability	21.5 (15.0–30.0)	20.5 (13.0–30.0)	1325.0	−0.062	0.597
Lethargy	22.5 (13.8–35.0)	22.0 (14.0–30.3)	1297.5	−0.040	0.735
Stereotypy	10.0 (0.8–14.0)	4.5 (0.0–13.0)	1436.5	−0.151	0.187
Hyperactivity	7.0 (2.0–28.0)	8.0 (2.0–30.0)	1236.0	0.010	0.936
Inappropriate Speech	2.0 (0.0–4.0)	1.0 (0.0–3.3)	1408.0	−0.128	0.255

We assessed behavioural problems using the Aberrant Behaviour Checklist (ABC). The median ABC total score was 66.0 (IQR: 53.0–91.0) in participants with parental consanguinity and 63.0

(IQR: 51.8–87.5) in those without parental consanguinity. The difference was not statistically significant (Mann–Whitney U = 1378.0, p = 0.371). No statistically significant differences were observed across the individual ABC domains: irritability (p = 0.597), lethargy (p = 0.735), stereotypy (p = 0.187), hyperactivity (p = 0.936), and inappropriate speech (p = 0.255). Overall, parental consanguinity was not significantly associated with behavioural problems as measured by the ABC.

DISCUSSION

This study assessed 100 individuals with intellectual disability, focusing on the association between parental consanguinity and severity, comorbidities, and behavioural problems. Consanguinity was present in 48%, mostly third-degree (58.3%) and second-degree (27.1%). This high rate aligns with Indian data showing persistent consanguineous marriages in certain regions. Sharma et al. noted regional differences, with higher rates in southern states.[9] Lakhan et al., from Barwani, observed familial clustering of various conditions among consanguineous families.[12] These findings highlight the importance of considering parental consanguinity in clinical evaluations, though it alone doesn't confirm a genetic cause.

Overall, 51% of participants had mild, 43% moderate, and 6% severe intellectual disability. Moderate disability was more common than mild in the consanguinity-present group (54.2% vs 39.6%); the reverse was true in the consanguinity-absent group (61.5% vs 32.7%). The groups did not differ significantly in the distribution of categorical severity (Pearson $\chi^2 = 5.046$, df = 2, p = 0.080). However, ordinal analysis found a significant shift towards higher severity with parental consanguinity (Mann–Whitney U = 1508.5, p = 0.043). Effect sizes were small (Cramer's V = 0.225; rank-biserial correlation = -0.209). This suggests a modest distributional difference, not that consanguinity causes more severe disability. Earlier work by Madhavan and Narayan found that, among persons with intellectual disability, the combination of family history of intellectual disability and parental consanguinity was associated with a higher risk of intellectual disability in offspring.[10] Their findings provide a possible genetic context for the present observation, particularly because consanguinity increases the probability of homozygosity for pathogenic variants associated with autosomal recessive conditions.[7,8] However, direct comparison with the present study is limited because Madhavan and Narayan examined familial risk rather than severity categories among already diagnosed individuals. Similarly, Lakhan et al. demonstrated familial patterns of intellectual disability and neurological or psychiatric disorders in consanguineous families. Still, they did not specifically establish an association between consanguinity and severity of intellectual disability.[12]

Medical comorbidities were present in a subset of participants, with epilepsy in 22% and cerebral palsy in 3%, while 75% had no comorbidity. Epilepsy prevalence was similar regardless of parental consanguinity (22.9% vs 21.2%), and cerebral palsy was rare in both groups. Comorbidity distribution did not differ significantly (p=0.786). The presence of epilepsy and cerebral palsy aligns with the heterogeneity of intellectual disability, which often includes neurological and other disorders.[4,11] Lakhan et al. also found epilepsy and cerebral palsy in families with consanguineous children 12, but their focus was on familial patterns, not comorbidity prevalence by consanguinity. The lack of a significant difference suggests parental consanguinity wasn't associated with comorbidities, though a small sample size and few cases of cerebral palsy may have limited detection. The study population had psychiatric comorbidities: ADHD in 15%, ASD in 10%, both in 9%, other in 3%, and 63% with none. The distribution did not differ significantly by parental consanguinity (p=0.703; Cramer's V=0.148). These findings align with the literature showing substantial psychiatric and developmental comorbidity in people with intellectual disability.[4,11] Matson and Cervantes highlighted comorbidity as a key feature involving multiple conditions.[11] Although genetic links with consanguinity may influence some neurodevelopmental disorders, no clear association was found with overall psychiatric comorbidities, likely due to the multifactorial nature of these conditions.

We assessed behavioural problems using the Aberrant Behaviour Checklist. The median total ABC score was 66.0 (IQR 53.0–91.0) in participants with parental consanguinity and 63.0 (IQR 51.8–87.5) in those without. The difference was not statistically significant (Mann–Whitney U = 1378.0, p = 0.371). None of the five ABC domains—irritability, lethargy, stereotypy, hyperactivity, or inappropriate speech—differed significantly between the two groups.[5] These findings suggest that parental consanguinity was not significantly associated with behavioural problem severity, as measured by the ABC, in the present sample. This is relevant because behavioural problems are common among individuals with intellectual disability and may substantially influence functioning and caregiver burden.[5,6] The absence of an association in the present study is also consistent with the possibility that behavioural difficulties are influenced by multiple factors, including the severity of intellectual disability, psychiatric comorbidity, communication difficulties, environmental circumstances, and caregiver-related factors, rather than parental consanguinity alone. Nevertheless, interpret the lack of significant differences cautiously, given the study's cross-sectional design, convenience sampling, and a sample size of 100 participants.

Overall, the findings indicate that parental consanguinity was associated with a modest shift towards higher ordinal severity of intellectual disability, but was not significantly associated with the categorical severity distribution on chi-square testing, medical comorbidities, psychiatric comorbidities, or behavioural problems measured by the ABC. The findings therefore provide some evidence of an association between parental consanguinity and the distribution of intellectual disability severity, while not demonstrating a corresponding association with the other clinical outcomes assessed. Previous Indian studies have primarily examined the occurrence of intellectual disability and familial patterns in relation to consanguinity.[10,12] In contrast, the present study additionally examines severity, comorbidity, and behavioural outcomes within a single clinical sample. Larger, multicentre studies with detailed family histories, genetic evaluation, and longitudinal follow-up would help clarify whether the observed relationship between parental consanguinity and severity reflects underlying genetic mechanisms or other clinical and familial factors.

CONCLUSION

The present study observed a modest shift towards higher ordinal severity categories among participants with parental consanguinity. However, chi-square testing showed no significant difference in the distribution of intellectual disability severity categories. We found no significant association between parental consanguinity and medical comorbidities, psychiatric comorbidities, or behavioural problems. These findings suggest that parental consanguinity may be relevant to the clinical assessment of people with intellectual disability, while recognising that consanguinity alone does not establish a causal relationship with severity or associated clinical features.

Limitations: The cross-sectional design cannot establish causality between parental consanguinity and clinical characteristics. Convenience sampling at one tertiary-care centre may limit generalisability, and the small sample—especially the severe intellectual disability subgroup—may have reduced power to detect associations. Without genetic investigations, the study cannot identify mechanisms underlying the association between consanguinity and severity. It also did not comprehensively assess family history, prenatal and perinatal factors, or environmental influences.

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