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Assessment of changing trends of risk factors contributing to Intellectual Disability – A retrospective study

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ARTICLE INFO ABSTRACT Article history: Background: The etiology of Intellectual Disability is varied and difficult to establish. Reports from India Received 03-02-2021 on the spectrum of underlying causative factors are lacking. So this retrospective study was conducted to Accepted 20-02-2021 identify the various etiologies of Intellectual Disability (ID). Available online 12-03-2021 Materials and Methods: Study was conducted at a tertiary care center. The etiology of ID was ascertained after clinical assessment. Two groups were ascertained Group A those who were born in 2000 and before and group B those who were born in 2001 and after. The spectrum of causative conditions was identified. Keywords: Results: In both groups the prevalence of ID was higher among males than in females. No major difference Etiology in domicile was noted. More than 75% children were off springs of non-consanguineous parents. Most of Intellectual disability (ID) them belong to mild ID (45%) in both groups. The etiological yield was 18.33% in group A and 11.66% in group B with Prenatal cause, followed by perinatal 48.33% in group A and 55% in group B, being the most common and postnatal cause 8.33% and 5% in group A and B respectively .Idiopathic causes constitute 20% in group A and 25% in group B, Few cases fall under multiple etiologies. Conclusion: It is possible to ascertain the diagnosis in most of the cases of intellectual disability.

Conclusion: It is possible to ascertain the diagnosis in most of the cases of intellectual disability. The prenatal and postnatal causes are decreasing due to awareness of Antenatal checkup, and active treatment respectively. But the Perinatal cause, which is preventable one, is increasing, which needs active intervention; also idiopathic cause which needs thorough screening.

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1. Introduction

Intellectual disability, formerly known as mental retardation, is a condition characterized by significant low average intellectual functioning and impairment in an adaptive behavior manifested before age of 18 years. In the last century, the persons with intellectual disability have experienced a radical change in all aspects of life: healthcare, employment, education, recreation, and living situation (World Health Organization, 2000). It has been defined and renamed many times throughout history. Mental retardation, which was in use worldwide till late 20th century, has been now replaced with the term Intellectual disability in most of the countries. Diagnostic and Statistical Manual 5th Revision (DSM-5) has replaced it with Intellectual Disability.^{1,2}

Abbasi moheb L et al studied that the prevalence of ID is 1-3% worldwide and this prevalence is expected to be high in under developing countries because of the nongenetic factors such as malnutrition, poor healthcare and environmental factors.³ Noor et al showed on the basis of IQ level, ID is ranging 2-3%, but the prevalence of ID is roughly reported to 1%.⁴ Actual prevalence varies considerably, with the range as high as 9.7%. This difference in ID arises because of the variations in population studies, study design and case definition. Generally ID is more common in boys than girls and on the average; the ratio is 1.4:1.⁴

* Corresponding author. E-mail address: shankardrpsy@ymail.com (S. Shankar). Kaufman et al classified ID, as genetic or non-genetic, depending on its etiology. The causes of genetic ID, which accounts for only 30% to 50% of all ID cases, include chromosomal abnormalities (e.g. trisomy 21 syndrome), inherited genetic traits (e.g. fragile X syndrome) and single gene disorders (e.g. Prader-Willi syndrome). However, the causes of non-genetic ID are not fully known. It is now suggested that the risk factors for non-genetic ID are extensive, and can be classified as prenatal, perinatal and neonatal factors according to the timing of suffering.⁵ Many prenatal factors (advanced maternal age, low maternal education, third or more parity, maternal alcohol use, maternal tobacco use, maternal diabetes, maternal hypertension, maternal epilepsy and maternal asthma), one perinatal factor (preterm birth) and two neonatal factors (male sex and low birth weight) were significantly associated with increased risk of ID. In at least 30 to 50 percent of cases, physicians are unable to determine etiology despite thorough evaluation (Rauch et al and Baird et al 1985). It is evident that many of the factors and causes of intellectual disability are preventable. In spite of that, the preventive strategy is not addressed well.^{6,7}

Mentally retarded children require more socio psychological support in comparison to their peers. Intellectual disabilities requires certain special needs, such as special education. Intellectual disability is a public concern because it requires involves all domains medical, social, educational level and also it can causes severe impairment. The prevention strategies are not met or not involved in, to reduce the occurrence of intellectual disability. According to recent reviews and meta-analysis, there are many good improvements in disability certification and reservations in education section and more. But in relation to prevention strategy, it is still rudimentary.

Webster et al discussed that in spite of so many interventions like nutritional supplementation, periodic checkups, anomaly scan and genetic screening, the prevalence of intellectual disability remains same. Are these interventions meant to prevent intellectual disability? Or what is the cause? For such high prevalence of intellectual disability for that we need to know the current etiology or risk factors for mental retardation. This helps us to focus on that issue in prevention strategies.⁸ Moreover, ID has a great influence on quality of life and productivity, which involves not only affected children but also their families and the society.

The aim of this study is to describe etiological characteristics of people with intellectual disability in the last 17 years. There is huge scope for prevention of avoidable and treatable causes for ID.

2. Objectives of the Study

The present study on mental retardation was carried out with the following objectives:

- 1. To assess the etiological pattern of mental retardation found among the children born on 2000 or before (Group A).
- 2. To assess the etiological pattern of mental retardation found among the children born on 2001 or after (Group B).
- 3. To compare the groups in terms of sociodemographic characteristics ,degree of intellectual impairment and etiological factors responsible for the Intellectual disability.

3. Materials and Methods

3.1. Ethical consideration

The ethical approval and permission to conduct the study was granted by institutional ethical committee and written informed consent was obtained from the parents of children with intellectual disability.

3.2. Study design

It was a retrospective record based observation study. The study period was from Oct 2018 to Oct.2019. This retrospective study was conducted at the Department of psychiatry. The patients attending the outpatient child guidance clinic/ disability camps with record of ID/ID from Oct 2018 to Oct 2019 with the following inclusion and exclusion criteria were included in the study. Children with ID born on 2000 or before are categorized as Group A and Children with ID born on 2001 or after are categorized as Group B

3.3. Sample selection

Using purposive sampling method, a total of 120 cases (60 cases for group A and 60 Cases for group B) were selected as study participants over 12 months period.

3.4. Inclusion criteria

- 1. Persons with Intellectual disability of age 3 and above attending Child Guidance Clinic were taken up for the study
- 2. Parents willing to give Informed consent.

3.5. Exclusion criteria

- 1. Children with age less than 3
- 2. Those who are critically ill, or with severe behavior disturbances were excluded from the study
- 3. Parents not willing to participate.

3.6. Methodology

By following strict inclusion and exclusion criteria, cases were selected and categorized into two groups depending on their year of birth - those born on 2000 or before taken as group A and those born on 2001 or after taken as group B. All children underwent detailed clinical evaluation as per a structured proforma that included perinatal events, regression of milestones, seizures, behavioural problems, symptoms suggestive of inborn errors of metabolism, hypothyroidism, three generation pedigree and social history. Examination included anthropometric, dysmorphic, neurological and ophthalmologic assessment. Totally 120 patients included into the study were subjected to IQ measurements done by the clinical psychologist.IQ assessment was done using Binet-Kamat test of intelligence or Malin's Intelligence Scale for Indian Children. The term IQ, stands for Intelligence Quotient, which is a ratio of Mental Age and Chronological Age multiplied by 100 (i.e. MA / CA x 100 = IQ), the resultant IQ was categorized into Mild, moderate, severe and profound based on ICD-10 classification .Socio demographic profile assessed using socioeconomic scale

3.6.1. Binet-Kamat test of intelligence⁹

Bombay-Karnatak version of Binet-Simon Intelligence Scale, otherwise popularly known as Binet Kamat Test or just BKT, is one of the old tests of intelligence that has been in use since several decades in India which includes both verbal and performance tests. This can be used in Age group between 3 to 22 years and adults with mental handicap.

3.6.2. Malin's Intelligence Scale for Indian Children

Malin's Intelligence Scale for Indian children (MISIC) (the Indian adaptation of Wechsler's Intelligence scale for children) was used to assess the IQ of the children, which gives scores on Verbal IQ, Performance IQ and full scale IQ. This scale has 11 sub-tests which generate a verbal IQ, performance IQ and a total IQ score.¹⁰

3.6.3. ICD-10 classification

The severity of ID was graded into mild (IQ 50-69), moderate (IQ 35-49), severe (IQ 20-34) and profound (IQ under 20) using ICD-10 classification. Mental retardation/ID was defined as significant deficits in cognitive function and adaptive behavior. The severity of ID/ID was determined by Intelligence Quotient (IQ) level measured by the psychologist using a standard method.¹¹

3.6.4. Social status by Socio economic scale

Social status by Socio economic scale (S.E.Gupat and B.P.Sethi et al 1978, Kuppusamy et al 1961). The Social status by Socio economic scale (S.E.Gupat and B.P.Sethi 1978, Kuppusamy 1961). Socioeconomic scale consists of scores based on three variables namely education, occupation, and income. On the basis of ten point scale it consists of ten categories which were grouped with 5 social class namely very high, high, upper middle, lower middle and very low. The 10 point scale consists of 200 scores with equal class interval. The inter rater reliability was found to be very high (R=0.9).¹²

3.6.5. Data analysis

All data including socio-demographic information was gathered from the family interviews. Accuracy checks and necessary corrections were made before and after the data were entered into the database. Data analysis was carried out using the Statistical Package for Social Sciences (SPSS 23.0 version) software for Windows and descriptive statistics were applied, including frequency of each variable and its distribution.

4. Results and Discussion

4.1. Sociodemographic Details

Socio-economic background of the Mentally-Retarded children: See Table 1

The sex-wise distribution of mentally-retarded children shows that, 66.6% (40) in group A and 65% (39) in group B were males, whereas females' proportion is 20 in number in group A and 21 in group B. This shows that mental retardation was more among males than females. Similar to this study, Eyman et al studied that the prevalence of ID was higher among males than in females (p<0.001) which are in support with other studies.¹² Also Croen and Luckassan et al showed that ID in males was two times more common, a finding similar to but slightly higher than previous literature reports. There is high proportion of male case of ID in many studies including this study. This may be due to fact that only male child are brought for evaluation and female child with ID are not brought for evaluation, for various social and cultural factors.^{13,14}

Maternal age at the time of child birth majority are between 20 to 30 years in both groups with slight higher number of persons 11(18.33%) with above 30 age in group B when compared 6 (10%) in group A but that is statistically not significant. Similarly paternal age at the time of child birth majority were between 20 to 30 years in both groups. There is slight change in education level for father and mother in the two groups, but that is not significant statistically. Krishnasubha et al studied Age of the mother at the time of children delivery i.e. maternal ages in study and control groups, it is showing that more number of individuals (13 cases) are observed in the age group above 30 years of maternal age, as it may be assumed that the advanced maternal age is one of the influencing factor of mental retardation.¹⁵ Admit nagarkar et al also discussed the paternal age in study and control groups i.e. age at the time of children birth, it is indicating that, more number of individuals (16 cases) are observed above 35 years of paternal age when compared with controls.¹⁶

Variable		Group An-60Born in 2000 and before	%	Group Bn-60 Born 2001 after	%	CHI Square P value
C	М	40	66.66	39	65	0.037
Sex	F	20	33.33	21	35	0.847
	<20yrs	6	10	4	6.66	
Maternal age	20 -30yrs	48	80	45	75	1.9674 0.373932
	>30yrs	6	10	11	18.33	
	<20yrs	1	1.66	1	1.66	
Paternal age	20 -30yrs	46	76.66	44	73.33	0.1873 0.91060
	>30yrs	13	21.66	15	25	
Matamal	Primary	29	48.33	26	43.33	
aducation	Secondary	21	35	22	36.66	0.3687 0.83164
cultation	College	10	16.66	12	20	
Paternal education	Primary	24	40	22	36.66	
	Secondary	22	36.66	21	35	0.4005 0.818512
	College	14	23.33	17	28.33	
Maternal	Yes	13	21.66	18	30	1 0874 0 207050
employment	No	47	78.33	42	70	1.0674 0.297039
Paternal	Yes	56	93.33	58	6.66	0 7018 0 402105
Employment	No	04	6.66	02	3.33	0.7010 0.402195
Domicilo	Rural	34	56.66	33	55	0.0338
Domicile	Urban	26	43.33	27	45	0.854147
	Lower	25	41.66	24	40	
Socioconomic	Lowermiddle	19	31.66	20	33.33	
status	Uppermiddle	13	21.66	14	23.33	0.1465 0.929385
	High	2	3.33	1	1.66	
	Very high	1	1.66	1	1.66	

Table 1: Socio demographic characters

Table 2: Consanguineous marriage, birth order and family history of intellectual disability

Variable		Group A n-60	%	Group B n-60	%	CHI Square P value	
C	No	45	75	49	81.66		
Marriage	2 nd degree	7	11.66	6	10	0.9394 0.625176	
Mainage	3 rd degree	8	13.33	5	8.33		
	1^{st}	40	66.66	41	68.33		
Birth Order	2^{nd}	17	28.33	18	30	1.0409 0.594248	
	3^{rd}	3	5	1	1.66		
Family history of	Yes	7	11.66	4	6.66	0 0008 0 242581	
intellectual disability	No	53	88.33	56	93.33	0.9006 0.342381	

Table 3: Degree of intellectual diability – Group A

Degree	IQ range and mean	No N-60	%
Mild	51 TO 69 59.22	27	45%
Moderate	36 TO 48 41.52	23	38.33%
Severe	21 TO 33 25.88	9	15%
Profound	<20	1	1.66%

Table 4: Degree of intellectual disability – group B

Degree	IQ range and mean	No N-60	%
Mild	53 to 68 60.36	27	45%
Moderate	37 to 48 42.76	25	41.66%
Severe	25 to 34 32.71	7	11.66%
Profound	<20	1	1.66%

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Table 5: Comorbidities			
Comorbidities	Group A n-60	Group B n-60	
Present	31 (51.66%)	28 (46.66%)	
Epilepsy	5	5	
Hyperactivity	4	2	
Autism	3	2	
Psychosis	1	0	
Behavioural	8	7	
Sensory	3	2	
Cerebral palsy	20	7	

Table 6: Etiologies

Category and cause		Group a N=60	Total N=60	%	Group b	Total N=60	%
	Genetic	8			5		
	Infections	0		18.33	0	7	11.66
Prenatal	Gestational	1	11		0		
	DM Eclampsia	2			2		
	Others	0			0		
	Prolonged	2			2		
	Labor	8	9 29 48.33 2		9		
Natal	High forceps	2		2	22	55	
	Prematurity	3			3	33	55
	Preterm	14		17			
	Asphyxia						
	Infections	1			1		
Postnatal	Seizures	1			1		
	Hyperbilirubi	1	5	8.33	0	3	5
	Nemia	1			1		
	Metabolic Nutritional	1			0		
Multiple	Multiple etiology	3	3	5	2	2	3.33
Idiopathic	No causes	12	12	20	15	15	25

Fathers were employed and no change in both groups, whereas number of mothers employed in group B (30%) is more when compared to group A (21.66%). Mother of the children in the study sample belongs to all grade of education with predominantly Primary level in both groups 48.33% (group A) and 43.33% (group B). Also Father of the patients in the study sample had all grade of education with predominantly Primary level in both groups 40% (group A) and 36.66% (group B). Majority of the mother of intellectually disabled child were unemployed irrespective of their education status in both groups. Whereas father of the 90% more were going for job in both groups.

More than 50% of the study population belongs to rural background, 56.66% in group A and 55% in group B, the remaining are from urban area. In both the groups, the rural population outweighs the urban population. Krishnasubha et al showed more number of individuals were observed in urban area in both study and control groups (study group: 81% and control group: 67%.¹⁵). Most of the study population belongs to rural area, because of the fact that, the sample is chosen from government tertiary care centre, where mostly rural population turn up for checkup.

With regards to socioeconomic status most of the families falls under lower category in both group. In group A Out of 60 mentally challenged children 1 child (1.66%) belongs to Very high, 2(3.33) in high, 13(21.66) in upper middle, 19(31.66) in lower middle and 25(41.66) in lower class. Similarly in Group B 1(1.66%) belongs to Very high, 1(1.66) in high, 14(23.33) in upper middle, 20(33.33) in lower middle and 24(40) in lower class, the distribution being almost similar in both groups. Gopala krishnan thakur et al showed similar reports in social economic status. His study revealed that poor income group were more vulnerable to give birth of child with mental retardation. The social background also emerged as a crucial factor in having an impact of well being of the children.¹⁷

Majority of the children's in both the groups were born from non consanguineous marriage. Only few 10 to 11% from second degree and 13.33 % and 8.33% from 3^{rd} degree consanguineous marriage in group A and B respectively.(Table 2). In a study by Darr et al similar marriage pattern was observed among many Pakistanis¹³ and showed significant association of consanguinity and mild ID.¹⁸ Birth order of most of the kids are first born child (66.66%) in group A and 68.33% in group B, only very few percentages of kids were of second and third order birth. ID seemed to be prevalent more in the 1^{st} order of birth rather than the consecutive ones. Only 10 patients were of higher order. Family history of intellectual disability is 11.66% in group A and 6.66% in Group B. Is similar with other studies like roshanak et al found There were 19 (29.7%) male patients and 45 (70.3%) female patients. First degree relatives with ID were found in the families of studied patients, among these relatives, 48% were female and 52% were male.¹⁹

4.2. Degree of intellectual disability

In group A, 45% of children has mild Intellectual disability and 38.33% had moderate, 15% had severe and 1.66% had profound subtypes. In group B, 45% of children had mild, 41.66% had moderate and 11.66%, 1.66% severe and profound respectively. The mean IQ was around 53.33. Severe retardation was evident in around 15% and 11.66% of the patients in group A and B respectivel. Profound 1 case in both. (Tables 3 and 4)

On comparison of both groups more number of children with moderate intellectual disability were present in group B but this is statistically not significant. Many studies showed similar results like for example, Shagun aggarwal et al found Mild ID is seven to ten times more common than moderate or severe ID.²⁰SG kumar et al also studied similar reports However, recent census data reported that majority had mild disability (151, 83.4%), followed by moderate (21, 11.6%), severe (8, 4.4%), and profound (1, 0.6%) disability.²¹ Maulik and roshanak et al concluded as mild, severe, moderate and profound types of 9 ID with 45.5%, 27.3%, 18.2% and 9.1% respectively in their study. Severe and Profound ID were diagnosed at a much earlier age than mild and moderate types.^{19,22}

4.3. Comorbidities

Intellectual disability was mostly associated with comorbidities. Co-morbidities were reported in 51.66% (31 out of 60) patients in group A and 46.66% patients (28 out of 60) in group B. Co-morbidities thus seem to be a very common accompaniment with ID, in more than half of the study population. (Table 5). The different co-morbidities recorded in the study population, include epilepsy, hyperactivity, autism, psychosis, behavioural disturbance sensory impairments, and cerebral palsy. Psychosis was not present in group B, whereas the other comorbidities are almost same in both groups. Presence of comorbidities is associated with additional impairment in functioning which is the main reason for burnout for parents of children with intellectual disability.

Many studies also reported that comorbidities are very commonly present with intellectual disability. Singhi et al discussed that the majority of ID children (60/64, 93%) had coexisting motor, seizure, vision, and/or hearing and speech disabilities.²³ Jan et al also showed Majority of these (63%) had spastic cerebral palsy, as has been reported earlier19. Previous studies have also concluded prenatal insult as the commonest cause of cerebral palsy.^{24,25}

4.4. Etiologies

Discussion on etiologies can be categorized into prenatal, perinatal, postnatal, multiple etiologies and idiopathic (Table 5).

4.5. Prenatal cause

In group A, 11 children had prenatal cause. Among that 8 children had genetic causes, 2 had eclampsia and 1 had uncontrolled gestational diabetes. In group B, 7 children had prenatal causes. In that 5 children had genetic causes and 2 had eclampsia. Both the groups have similar etiology, with slight genetic preponderance in group A than group B, which may be due to proper antenatal screening, but the difference has no statistical significance. Genetic causes include down syndrome, tuberous sclerosis, fragile X syndrome, Aicardi syndrome. Other prenatal causes includes eclampsia, and gestational diabetes. Majority of this group has severe intellectual disability. The contribution of different etiologies to ID/ID was similar to previous literature reports. Tuberous sclerosis, Cornelia de Lange syndrome, Bardet Biedl syndrome, Noonan syndrome and congenital muscular dystrophy were found in more than one patient. 25

D.Phalke et al found Genetic causes constituted the most common category accounting for 51/83 (61.4%) of the cases, followed by perinatal acquired 17 (20.4%), CNS malformations 10 (12%), external prenatal 3 (3.6%) and postnatal acquired causes 2(2.4%).²⁶ Similarly Silky jain et al showed Down syndrome was the most common cause accounting for 14.8% of all cases. Chromosomal disorders constituted 36.5% (19/52) of genetic causes single gene 48% and recognizable syndromes 11.5%.²⁷ The etiological spectrum in our study resembles that in most of the Western studies and also the study by Tikaria, et al,²⁸ unlike the study by Jauhari, et al²⁹ where perinatal causes constituted the most common category.

4.6. Natal cause

In group A 29 cases that is 48.33% are due to natal cause. Almost half of the children with intellectual disability fall under this group. The main causes are prolonged labour, forceps (no other cause was present except for this forceps use, so it will be taken as inadequate application of high forceps, also all cases had history of prolonged labour), prematurity, preterm, low birth weight and asphyxia. In this forceps, high forceps was used in 8 deliveries, birth asphyxia was present in 14 cases. In group B 33 cases that is 55% from natal cause. More than half of the cases falls under this category, of which high forceps in used in 9 deliveries and birth asphyxia in 17 cases, which is more compared to group A. This shows that in spite of so many advancements in antenatal or obstetrical care, the perinatal insult is in increasing trend, but the difference is statistically not significant.

Majority of the cases falls under moderate and mild intellectual disability. Piecuch et al. 1997 showed Complications of prematurity, especially in extremely lowbirthweight infants, or postnatal exposure to Lead can also cause mental retardation.³⁰ Kolevzon et al found Birth asphyxia, birth trauma, complicated delivery, severe prematurity and very low birth weight are perinatal problems, which may result in ID.³¹

4.7. Post natal cause

Total of 5 cases in group A and 3 cases in group B are due to postnatal etiology. The causes include infection, seizure, nutritional, metabolic and hyperbilirubinimia in group A and infection, seizure and metabolic in group B. Majority of the cases falls under moderate and mild intellectual disability. Post natal cause studied by many authors showed similar results. One such study by Leonard et al found infancy and childhood period is postnatal period. Problems such as Japanese encephalitis, Bacterial meningitis, Tuberculosis, head injury, prolonged malnutrition and chronic lead exposure during this period may result in ID.³²

Durkin et al found that another very important cause of ID is malnutrition. This malnutrition affects the mental ability in the people of the areas affected by famine. As a result of these famines, people face the problem of nutritional deficiency, which ultimately results in increase in mental retardation.³³ Postnatal cause is decreasing due to proper nutrition and care, which needs to be addressed continuously.

4.8. Multiple etiology

In majority of studies, single factor was taken up for the study. But in this study, we have included a category of multiple etiology use, in cases with more than one factor responsible for development of intellectual disability. This category children have more than one cause, either prenatal, perinatal or postnatal or multiple factors with in the same domain. For example perinatal cause with birth asphyxia, low birth weight, prematurity and postnatal with infections, seizure or injury. Majority of the cases falls under severe intellectual disability. Group A comprise 3 and group B comprises 2 cases. All of them belong to severe and

profound disability.

4.9. Idiopathic etiology

No visible cause for intellectual disability, there is no signs of physical anomalies, no history of material cause, obstetric cause or natal and post natal cause, such cases are categorized as idiopathic. In this study group A has 12 cases and group B has 15 cases. Majority of the cases has various degree of intellectual impairment. Many studies discussed about the etiology is idiopathic. Rosenfeld, Harripaul and Simone identified individuals with idiopathic ID/ID, autism spectrum disorders, or multiple congenital anomalies, chromosomal microarray analysis (CMA) is recommended as the first-line diagnostic test since it offers a much higher diagnostic yield (15 % to 20 %) compared with G-banded karyotype analysis (3 %). 34-36 Kaufman et al showed very high percentage upto 60% of ID there is no identifiable cause. Considering this, the proportion of idiopathic cases is similar to what was previously reported.⁵ McLaren and Bryson studied that in 30 to 40% of cases, the cause remains unknown. Phalke et al studied mentally challenged children and observed that in 31.3% children causes were unknown or idiopathic.³⁷

A definite diagnosis of ID/DD may not be possible in 30-60 per cent patients, although figures ranging from 10-80 per cent have been reported in different studies. Finding a definite cause is especially difficult in mild ID cases where psychosocial factors play a major role. This indicates the need to establish genetic services as part of paediatric care or integration of such services as part of care in institutions for mentally challenged individuals. The diagnosis of these genetic disorders is important for management of these persons as well as offering prenatal diagnosis services to the family. A high yield of a targeted diagnostic approach was also observed in the workup of these patients.

With this study we came to know that natal cause is still most common cause of intellectual disability of which birth asphyxia is the most common cause and still increasing particularly the high use of high inclination forces which results in mild to moderate degree of intellectual disability. Despite growing advances in medical field, the natal cause is increasing trend, as there is lack of awareness about the cause. At the time of delivery only maternal and infant mortality is given high priority, where future complication is not given importance. So there is need for awareness about birth complication and its future implications, which can be done through various Government training programmes or scheme. Though genetic cause and post natal cause is slightly lower in group B that is new ages but that is statistically not significant, the prevalence of down's syndrome is still same in both groups warrants through prenatal trisomy evaluation and genetic counseling.

Similarly the post natal cause in both the group is very low. Awareness of warning signs and early intervention prevents this cause of intellectual disability

There are few cases with intellectual disability having mixed cause and categorized separately and degree of impairment is severe. This needs to be given special importance as most of the studies studied single etiology. Idiopathic cause is still high in both groups.

5. Limitation

- 1. Sample size is small. To address specific risk factors and develop preventive strategy, we have planned to extend the study in future, by including more number of samples which may give more clues about the specific cause for ID.
- 2. Confined to one geographic area so cannot be generalized.
- 3. No similar pattern of studies to compare, no control group, the descriptive nature of study, and no genetic analysis or metabolic screening are the other limitations of this study.
- 4. Problem of recall bias in both groups.

6. Conclusion

The present study findings suggest that currently the perinatal complications are associated with increased risk of developing intellectual disability, implying that this occurrence may be reduced with appropriate antenatal, perinatal, and neonatal healthcare interventions. Specific awareness programmers addressing the natal cause of ID should be given utmost importance in near future, thereby bringing down the prevalence of ID, as it remains the most common cause and is in increasing trend. Knowledge of the causes of intellectual disability can help to reduce cases by at least 25% by practicing primary prevention. Identifying patients with a high risk of non-genetic ID is important for early detection and intervention of ID, which would benefit both clinical practice and public health. Intellectual disability with no specific causes need to addressed specifically and with aids in prevention strategies. We need to address this by give awareness programmers to medical personals, reproductive age groups women's and general public, use of media for this purpose.

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8. Conflict of Interest

The authors declare they have no conflict of interest.

References

1. American Association on Mental Retardation (AAID). In: Mental retardation: Definition, classification, and systems of supports. 10th edn. Washington, DC; 2002.

- American Psychiatric Association. In: Diagnostic and statistical manual of mental disorders-5. 5th edn. Arlington, VA: American Psychiatric Association; 2013.
- Abbasi-Moheb L. Mutations in NSUN2 cause autosomal-recessive intellectual disability. Am J Hum Genet. 2012;90(5):847–55.
- Noor A. CC2D2A, encoding a coiled-coil and C2 domain protein, causes autosomal-recessive mental retardation with retinitis pigmentosa. *Am J Hum Genet*. 2008;82(4):1011–8.
- Kaufman L, Ayub M, Vincent JB. The genetic basis of non-syndromic intellectual disability: a review. J Neurodev Disord. 2010;2(4):182– 209. doi:10.1007/s11689-010-9055-2.
- Rauch A, Hoyer J, Guth S, Zweier C, Kraus C, Becker C, et al. Diagnostic yield of various genetic approaches in patients with unexplained developmental delay or mental retardation. *Am J Med Genet*. 2006;140A(19):2063–74. doi:10.1002/ajmg.a.31416.
- Baird PA, Sadovnick AD. Mental retardation in over half-amillion conservativelivebirths: An epidemiological study. *Am J Ment Deficiency*. 1985;89:323–30.
- Webster R, Oliver MA. Geostatistics for environmental scientists. John Wiley & Sons; 2007.
- 9. Kamat V. A revision of the Binet scale for Indian children:(Kanarese and Marathi speaking). Br J Educ Psychol. 1934;4(3):296–309. doi:10.1111/j.2044-8279.1934.tb02959.x.
- Malin's intelligence scale for Indian children (MISIC). Indian J Mental Retard. 1971;4:15–25.
- World Health Organization. The ICD-10 Classification of Mental and Behavioural Disorders: Clinical descriptions and diagnostic guidelines. Geneva: World Health Organization; 1992.
- Kuppuswamy B. Manual of Socioeconomic Status (Urban). In: 1st Edn. Delhi: Manasayan; p. 66–72.
- Croen LA, Grether JK, Selvin S. The Epidemiology of Mental Retardation of Unknown Cause. *Pediatr*. 2001;107(6):e86. doi:10.1542/peds.107.6.e86.
- Luckasson R, Borthwick-Duffy S, Buntinx WHE, Coulter DL, Criag EM, Reeve A, et al. Mental retardation: definition, classification and systems of supports. Washington DC: AAID; 1992.
- Krishnasubha S, Lakshmikalpana V, Ramesh M, Sudhakar G. A Case - Control Study on Risk Factors of Mental Retardation from an Urban Area of North Coastal Andhra Pradesh. *J Life Sci.* 2010;2(2):93–8. doi:10.1080/09751270.2010.11885158.
- Nagarkar A. Jagdish Prashad Sharma The clinical profile of mentally retarded children in India and prevalence of depression in mothers of the mentally retarded. *Indian J Psychiatry*. 2014;56(2):165–70.
- Thakur GK. A Study Of Mental Retardation Amongst Children In The Context Of Their Socio - Economic Background. *J Business Manag* Soc Sci Res. 2014;3(7).
- Darr A, Modell B. The frequency of consanguineous marriage among British Pakistanis. J Med Genet. 1988;25(3):186–90. doi:10.1136/jmg.25.3.186.
- RJazayeri, Saberi SH, S M, Saberi MD, Soleymanzadeh M, D M. Etiological characteristics of people with intellectual disability in Iran. *Neurosci.* 2010;15(4):258–61.
- Aggarwal S, Bogula VR, Mandal K. Rashmi Kumar* & Shubha R. Phadke Aetiologic spectrum of mental retardation & developmental delay in India. *Indian J Med Res.* 2012;136:436–44.
- Kumar SG, Premarajan KC. SS Kar1 Epidemiology of mental disability using Indian Disability Evaluation Assessment Scale among general population in an urban area of Puducherry. *India J Clin Nurs*. 1998;7:505–11.
- Maulik PK, Harbour CK. Epidemiology of Intellectual Disability. Cambridge University Press; 2013.
- Singhi PD. Clinical Spectrum of Cerebral Palsy in North India– An Analysis of 1000 Cases. J Trop Pediatr. 2002;48(3):162–6. doi:10.1093/tropej/48.3.162.
- Jan MM. Cerebral palsy; Comprehensive review and update. Ann Saudi Med. 2006;26:123–32.
- Multicentric study on genetic causes of mental retardation in India. ICMR Collaborating Centres & Central Co-ordinating Unit. *Indian J Med Res.* 1991;94:161–9.

- Phalke DB. Study of health status and etiological factors of mentally challenged children in school for mentally challenged. 1992;1(1):15– 20.
- Jain S, Chowdhury V, Juneja M, Juneja M, Kabra M, Pandey S, et al. Indian Pediatr. 2013;50:1125–30.
- Tikaria A, Kabra M, Gupta N, Sapra S, Balakrishnan P, Gulati S, et al. Aetiology of global developmental delay in young children: Experience from a tertiary care centre in India. *Natl Med J India*. 2010;23:263–8.
- Jauhari P, Boggula R, Bhave A, Bhargava R, Singh C, Kohli N, et al. Aetiology of intellectual disability in paediatric outpatients in Northern India. *Dev Med Child Neurol.* 2011;53(2):167–72. doi:10.1111/j.1469-8749.2010.03823.x.
- Piecuch RE, Leonard CH, Cooper BA, Sehring SA. Outcome of Extremely Low Birth Weight Infants (500 to 999 Grams) Over a 12-Year Period. *Pediatr.* 1997;100(4):633–9. doi:10.1542/peds.100.4.633.
- Kolevzon A, Gross R, Reichenberg A. Prenatal and Perinatal Risk Factors for Autism. Arch Pediatr Adolesc Med. 2007;161(4):326–33. doi:10.1001/archpedi.161.4.326.
- Leonard H, Wen X. The epidemiology of mental retardation: Challenges and opportunities in the new millennium. *Ment Retard Dev Disabil Res Rev.* 2002;8(3):117–34. doi:10.1002/mrdd.10031.
- Durkin MS, Khan NZ, Davidson LL, Huq S, Munir S, Rasul E, et al. Prenatal and Postnatal Risk Factors for Mental Retardation among Children in Bangladesh. *Am J Epidemiol*. 2000;152(11):1024–33. doi:10.1093/aje/152.11.1024.

- Rosenfeld J, Patel A, Microarrays. Chromosomal Microarrays: Understanding Genetics of Neurodevelopmental Disorders and Congenital Anomalies. J Pediatr Genet. 2016;6(1):42–50.
- Harripaul R, Noor A, Ayub M, Vincent JB. The Use of Next-Generation Sequencing for Research and Diagnostics for Intellectual Disability. *Cold Spring Harb Perspect Med*. 2017;7(3):a026864. doi:10.1101/cshperspect.a026864.
- Karam S, Aluísio J. Barros c Intellectual Disability in a Birth Cohort: Prevalence, Etiology, and Determinants at the Age of 4 Years . *Public Health Genomics*. 2016;19:290–7. doi:10.1159/000448912.
- Mclaren J, Bryson SE. Review of recent epidemiological studies in mental retardation: Prevalence, associated disorders, and etiology. *Am J Mental Retard*. 1987;92:243–54.

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