

**Original Article**

# A CLINICO-EPIDEMIOLOGIC STUDY OF NEUROLOGIC ASSOCIATIONS AND FACTORS RELATED TO SPEECH AND LANGUAGE DELAY

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## ABSTRACT

**Objectives:** SLD is a significant neurodevelopmental complication and association of several neurologic disorders. The current study evaluates the neurologic associations and factors related to SLD.

**Methods:** A one year retrospective study was done at Tertiary Care Center in India. Medical records of patients examined at Pediatric Neurology Clinic for SLD, between December 2009 and December 2010 were studied and the collected data was analyzed.

**Results:** The prevalence of SLD was 16.27% and the Male : Female ratio was 2.76:1 in this study. GDD, Seizure disorder and ADHD were major comorbid conditions. Significant associations seen were; PIH, IUGR, Oligohyraminos, Perinatal- Neonatal resuscitation, LBW, LSCS, neonatal Icterus and seizures. There was a positive family history of SLD in 23.85% patients. EEG was epileptiform in 45.87% patients and features suggestive of Perinatal ABI due to varied etiology was the major neuroradiologic finding followed by congenital structural abnormalities. No specific diagnosis was possible in 33.94% patients, but 17.43% had Primary Epilepsy syndrome and 17.43% had a suspected Neurogenetic etiology.

**Conclusion:** Exact etiological diagnosis of SLD is challenging & not possible on many occasions. Most important associations include seizure disorder, ADHD, autism, neonatal resuscitation and Epileptiform EEG with or without clinical seizure activity. Most of these patients have either an epilepsy syndrome or suspected Neurometabolic or Neurogenetic etiology.

**Key words:** Speech and Language Delay, Neurologic Associations, ADHD, Autism, GDD, Epilepsy

## GLOSSARY

SLD = Speech and Language Delay;  
GDD = Global Developmental Delay;  
GMD = Gross Motor Delay;  
ADHD = Attention Deficit Hyperactivity Disorder;  
EEG = Electro Encephalogram;  
ABR = Auditory Brainstem Response;  
CT Scan = Computerized Tomography Scan;

MRI = Magnetic Resonance Imaging;  
LBW = Low Birth Weight;  
PIH = Pregnancy Induced Hypertension;  
IUGR = Intra Uterine Growth Retardation;  
LSCS = Lower Segment Caesarean Section;  
ABI=Acute brain injury;  
CALM = Café u lait macule

## INTRODUCTION

Speech and language development in children is a dynamic process starting at birth and continuing throughout infancy and early childhood. It requires a favorable environment including overall normal brain structure and pathologically and functionally normal speech and language areas. Any insult to the developing brain may result in either GDD or delay/impairment in specific areas of neurodevelopment, depending upon the area of the brain bearing the brunt of the pathology.

Despite significant technological advances in early detection and diagnosis of neurologic disorders the neuro-physio-pathologic basis of SLD still remains unclear, complicating management of SLD. To complicate further there is paucity of literature studying the exact determinants of neurological aspects of SLD.

In the aforementioned context the present study was conducted to examine the epidemiology, evaluate the prevalence, associations and factors related to SLD in patients examined in a Pediatric Neurology Clinic of a Tertiary Care Center at Jodhpur, Rajasthan, India.

## MATERIALS AND METHODS

Medical records of patients evaluated in the Pediatric Neurology Clinic of Umaid Hospital, Dr. S. N. Medical College Jodhpur, Rajasthan, India, between December 2009 and December 2010 for SLD were retrieved and analyzed. Data extracted from the case records included personal and epidemiological data, diagnosis, details of developmental, antenatal, perinatal, postnatal and family history, clinical examination, EEG, ABR and neuroimaging findings. Data were expressed as mean, standard deviation (SD), and frequency was expressed as a percentage. Computation of P values was done by t-test and chi-squared analysis.  $P < 0.05$  was considered statistically significant.

## RESULTS

Over the 1-year period, a total of 670 patients attended Pediatric Neurology Clinic. The prevalence of SLI amongst patients attending the clinic was 16.27% (109/670), 80 (73.40%) were males and 29 (26.60%) were females, with a Male: Female ratio of 2.76:1, ( $P < 0.0001$ ). Mean

age at presentation was 6.1 +/- 4.72 years (range, 4.5 month to 28 years). Most of the patients were in the age group of 1-6 year age. 13 (11.93%) patients had a neuroregression in Speech and language due to varied reasons after a normal age appropriate neurodevelopment. The specific Speech and Language problems seen in the patients are depicted in table 1.

**Table 1: Specific Speech and Language Problems**

| Speech and Language Problems    | Patients |
|---------------------------------|----------|
| Echololia                       | 2        |
| Poor social reciprocal behavior | 2        |
| Stereotyped speech/language     | 5        |
| Auditory agnosia                | 3        |
| Poor morphology of speech       | 30       |
| Poor comprehension              | 17       |
| Poor fluency (unclassified)     | 13       |
| No name repeating               | 9        |
| Stuttering                      | 11       |
| Stammering                      | 9        |

GDD was seen in 52.29% of these patients, ADHD in 33.94%, Autism was present in 13.76% and GMD (with age appropriate social neurodevelopment) in 12.84% of the patients. Seizure Disorder was present in 38.53% patients and was a major co-morbid condition. Table 2 depicts the types of seizures present.

**Table 2: Types of Seizures seen in patients (n=42)**

| Type of Seizure                 | No. (%) <sup>*</sup> |
|---------------------------------|----------------------|
| <b>Primary Generalized</b>      |                      |
| Absence                         | 1(2.38)              |
| Atypical Absence                | 0(0.0)               |
| Myoclonic                       | 5(11.91)             |
| Atonic                          | 0(0.0)               |
| Tonic                           | 1(2.38)              |
| Clonic                          | 1(2.38)              |
| Tonic-Clonic                    | 12(28.57)            |
| <b>Partial Seizures</b>         |                      |
| Simple                          | 2(4.76)              |
| Complex                         | 3(7.14)              |
| Secondary Generalized           | 1(2.38)              |
| <b>Mixed and other seizures</b> | 16(38.1)             |

\* 42 patients had history of seizures

Mothers of only 50.46% patients regularly visited Antenatal Clinics during their pregnancy. 12.84% mothers had miscarriages

during earlier pregnancies. 7.34% patients were offspring of a consanguineous marriage with significant inbreeding. 13 (11.93%) required NICU admission after birth. Mean birth weight was 2.62 +/- 0.72 kg. (Table 3).

**Table 3: Antenatal, Perinatal and Postnatal History**

| History                               | No. (%)*  |
|---------------------------------------|-----------|
| <b>Antenatal History</b>              |           |
| PIH                                   | 17(15.60) |
| IUGR                                  | 7(6.42)   |
| Oligohyraminos                        | 7(6.42)   |
| Threatened Abortion                   | 5(4.59)   |
| Anemia                                | 5(4.59)   |
| APH                                   | 4(3.67)   |
| PROM                                  | 3(2.75)   |
| Decreased Fetal Movements             | 3(2.75)   |
| Thyroid                               | 2(1.83)   |
| Leaking PV                            | 1(0.92)   |
| Late Quickening                       | 1(0.92)   |
| Fever                                 | 1(0.92)   |
| GDM                                   | 1(0.92)   |
| Secondary Infertility                 | 1(0.92)   |
| Persistent Vomiting                   | 1(0.92)   |
| Respiratory Distress During Pregnancy | 1(0.92)   |
| <b>Perinatal History</b>              |           |
| Neonatal Resuscitation                | 28(25.69) |
| LBW                                   | 22(20.18) |
| LSCS                                  | 20(18.35) |
| Home                                  | 14(12.84) |
| Delayed Cry                           | 11(10.09) |
| Pre-term                              | 10(9.17)  |
| Post-term                             | 2(1.83)   |
| Forceps                               | 2(1.83)   |
| Vacuum                                | 1(0.92)   |
| <b>Postnatal History</b>              |           |
| Icterus                               | 10(9.17)  |
| Seizures                              | 8(7.34)   |
| Excessive Cry                         | 7(6.42)   |
| Poor feeding                          | 6(5.50)   |
| Constipation                          | 3(2.75)   |
| Neonatal Encephalopathy               | 3(2.75)   |

\*Out of 109 patients examined at Paediatric Neurology Clinic for SLD

Seizures during infancy were seen in 19 (17.43%) patients. 6 (5.50%) patients had history of inconsolable crying during infancy. Family history for SLD was seen in 23.85% patients along with seizure disorder in 22.94%, Mental Retardation in 11.93% and Stroke before 40 yrs

of age in 5.50% patients. Microcephaly in 3 (2.75%) and Macrocephaly was present in 2 (1.83%) patients. Coarse facies, prominent ears, Hypertelorism, Dolicocephalic Head and Cherubic facies constituting Facial Dimorphism were seen in 7 (6.42%) patients. CALM (Hypopigmented and Hyperpigmented spots) were seen in 3 (2.75%) patients. EEG was done in 86 (78.90%) patients and was Epileptiform in 50 (45.87%) patients; Focal epileptiform discharges were seen originating from Parietal region (42.86%), Frontal region (25.71%), Central region (20%), Occipital region (8.57%) and Temporal region (2.86%). ABR and Neuroimaging were not performed in all patients; ABR findings were available in 41(37.61%), CT scan findings in 28 (25.69%) patients and MRI in 18 (16.51%) patients. (Tb- 4).

**Table 4: Investigations of study subjects**

| Investigation (No. of patients)                 | No. (%)*  |
|---|-----------|
| <b>EEG (n=86)</b>                               |           |
| Epileptic form                                  |           |
| Focal   | 16 (18.6) |
| Multifocal                                      | 12 (13.9) |
| Generalized                                     | 9 (10.5)  |
| Interictal Epileptiform                         | 8 (9.3)   |
| Multifocal & Generalized                        | 5 (5.8)   |
| Slow for Age                                    | 5 (5.8)   |
| Fast Beta Activity                              | 3 (3.5)   |
| Non specific                                    | 3 (3.5)   |
| Normal  | 25 (29.1) |
| <b>ABR (n=41)</b>                               |           |
| Abnormal audiometric brain stem evoked response | 10(24.4)  |
| Bilaterally increased hearing threshold         | 3(7.3)    |
| Normal  | 28(68.3)  |
| <b>CT Scan (n=28)</b>                           |           |
| Perinatal ABI                                   | 14(50.0)  |
| Congenital structural defects                   | 4(14.3)   |
| Postnatal ABI                                   | 2(7.1)    |
| Normal  | 8(28.6)   |
| <b>MRI (n=18)</b>                               |           |
| Perinatal ABI                                   | 10(55.6)  |
| Congenital structural defects                   | 2(11.1)   |
| Postnatal ABI                                   | 0(0.0)    |
| Normal  | 6(33.3)   |

\*Out of the no. of patients in which this test was done

Due to limitations related to clinical and diagnostic inputs, a specific diagnosis could not be made in 37 (33.94%) patients, however 19

(17.43%) had a Primary Epilepsy syndrome, 19 (17.43%) a suspected Neurogenetic etiology, 11 (10.09%) had cerebral Palsy and 7 (6.42%) had a suspected Neurometabolic etiology. Other etiologies seen were Perinatal Asphyxia in 4 (3.67%) patients, Stroke in 3 (2.75%), Hearing Deficits in 3 (2.75%), Primary Neuro-developmental condition (Primary Autism) in 2 (1.83%), Acute Brain Injury in 2 (1.83%), Rheumatic Chorea in 1 (0.92%), and possible sequelae to only Prematurity (no other attributable factor) in 1 (0.92%) patients.

## DISCUSSION

It was observed in the current study that 16.27% of visits to the Pediatric neurology Clinic at Umaid hospital, Jodhpur were for Speech and Language delay. Luthra S and Dharamvir<sup>1</sup> reported a higher prevalence of 48% in Advanced Pediatrics Neurodevelopment Indoor Unit No other study was available to compare the prevalence of SLD in a pediatric Neurology clinic. However the prevalence of SLD in preschool children has been reported to range from 2.02-19% in various studies<sup>2-4</sup>.

It has been clinically recognized now for many years that SLD is more common in males than females and the male: female ratio in the present study was 2.76:1 which is similar to several other studies<sup>5,6</sup>. Beitchman and co-workers<sup>3</sup> however reported a male:female ratio of 0.98:1 for speech only, 0.98:1 for language only and 0.82:1 for speech or language and 0.46:1 for both speech and language delay. Tomblin<sup>7</sup> however observed that the ratio was equal with no sex predilection. In the current study parental bias due to a social preference for male children may also contribute to more male children attending a specialized health care facility.

LD is most common developmental concern voiced by parents of children between the ages of 1 and 3 years of age; in the current study the mean age of patients at presentation was 6.1 +/- 4.72 years. This may be due to general ignorance among parents and care givers regarding the presence of serious or trivial underlying neurologic conditions and also the fact that many parents would first visit a traditional faith healer and may be very late to seek consultation from a specialist.

In the present study SLD in 52.29% patients was a part of GDD, reflecting a global insult to the brain. Associated GMD is also commonly found

in children with SLD<sup>8</sup> and it was supported by a prevalence of 12.84% in our study.

Attention deficit hyperactivity disorder was seen in almost one third patients (33.94%) in the current study. An extensive review of the literature did not reveal studies evaluating this observation; however, the prevalence of language impairment has been reported up to 45% among children meeting the criteria for attention deficit hyperactivity disorder (ADHD)<sup>9,10</sup>. ADHD is usually not a cause of the language disorder, but can make speech and language therapy difficult because the child has difficulty paying attention, staying still, and following through. Autism was present in 13.76% of our patients. Unfortunately, despite the availability of good screening methods it is very difficult to tease apart autism and SLD due to other causes.

A Family history of SLD was found in 23.85% patients, which is similar to what has been reported by Pruitt S L et al (24%)<sup>11</sup>. Family history of Seizures 22.94%, Mental Retardation 11.93% and Stroke before 40 yrs of age in 5.50% of patients was also seen in this study; there association with SLD has not been reported earlier. A strong family history of SLD and other neuro-developmental disorders may indicate a genetic etiology.

Damage to the developing brain before or around the time of birth can lead to poor neurological and cognitive outcome in children. The exact etiology of more specific learning disabilities, especially those affecting speech and language development are however unclear. In the present study many maternal, prenatal, perinatal and postnatal events were seen to be present in patients with speech and Language Delay (Table 3); many of these are preventable, mandating early detection and management.

Seizure disorder was a major co morbid condition present in 38.53% patients. Interictal Epileptiform discharges (IED) were seen in 45.87% patients (Table 4). Focal IED originated from Parietal region in 42.86% and Frontal region in 25.71% which may anatomically correspond to Broca's area (posterior inferior frontal gyrus of the brain). Magnetic encephalography and Functional MRI studies are recommended to further evaluate this observation.

Many neuro-radiologic studies have suggested that atypical patterns of asymmetry of auditory

and perisylvian cortex and reduced cerebral volume are a risk factor for specific language impairment<sup>12,13</sup>. In the present study due to technical limitations exact radio-anatomic elaboration was not feasible, however, in the current study neuroimaging revealed congenital structural brain defects including lissencephaly, Polymicrogyria, Agenesis of Corpus Callosum, Cerebral hypoplasia and Cerebellar hypoplasia in 11-15 % patients and perinatal and postnatal ABI in approximately 60% patients in whom Neuroimaging was performed.

## CONCLUSION

SLD may result due to varied etiology and is commonly associated with delayed or abnormal neurodevelopment in other spheres, seizures, epileptiform EEG with or without seizures, ADHD and autism. Children born to mothers with PIH have a higher chance of having SLD although the mechanism for the same is not very clear. Mothers with Oligohydramnios and IUGR may also have a higher chance of having SLD in their children. Important Perinatal and natal associations with higher chance of SLD were resuscitation at birth, LBW, LSCS delivery, neonatal icterus and neonatal seizures. A family history of SLD, seizure disorder, mental retardation and stroke before the age of 40 years were an important association in children having speech and language abnormalities.

Epilepsies and epileptiform EEG constituted a major association; theoretically there is a higher chance that many of these patients may be eventually amenable to treatment if the discharge focus is one of the speech and language areas.

A specific diagnosis was not possible in approximately one third (33.94%) of our patients potentially increasing the possibility that many of the treatable conditions may have been missed. We therefore recommend a meticulous neurometabolic workup in any patient with the relevant clinical setting because majority of the neurometabolic conditions are treatable.

Despite several technical limitations and being a retrospective study with epidemiological design, we hope that this study will go a long way to eventually solve queries and controversies associated with neurologic aspect of SLD. More studies focusing on individual factors and associations are recommended to be designed in

order to further explore this difficult and challenging field.

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