Etiology of Developmental Delay in Egyptian Children

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ABSTRACT

Objective: *The aim of this study is to determine the etiology of developmental delay in Egyptian children which may have a great impact on management, prognosis and recurrence risk.* ***Study design:*** *A retrospective chart review was carried out on all children referred for developmental assessment in the child development center in the National Neuro-Motor System Institute from July 2001 to April 2002. A diagnostic study including full history thorough clinical examination and developmental assessment in the 6 main areas of development were done to all cases. A battery of selected investigations including, EEG, EMG, visual and auditory evoked response, screens for metabolic diseases, thyroid function, karyotyping and neuroimaging (CT & MRI) were done if needed for diagnosis.* ***Results:*** *A total of 1161 patients were identified (54%) were males and (46%) females. Neurodevelopmental assessment did not confirm developmental delay in 165 cases (14%). 356 (31%) cases showed mild developmental delay, 398 (34%) were moderately delayed and 342 (29%) were severely delayed. proper history yielded an etiologic diagnosis in 742 cases (65%) of the 1161 cases examined. Etiologic categories included hypoxic ischemic encephalopathy (16%), kernicterus (9%), Down syndrome 6%, epileptic syndromes 6%, cerebral dysgenesis 5%, meningoencephalitis 5%, maternal diseases 5%, congenital infections 4%, ICH 4% and autism 2%. Positive consanguinity was reported in 43% of cases.* ***Conclusion:*** *The high incidence of perinatal etiology in our cases raises the importance of good maternal and neonatal care. Prophylactic measures should be done against neonatal hyperbilirubinaemia. Genetic counseling is essential in consanguine marriage. High risk infant should be followed regularly for early detection of developmental delay and early intervention.* **(Int. J. Ch. Neuropsychiatry, 2004, 1(1): 29-40)**

INTRODUCTION

Developmental disability (DD) is a significant physical, mental, and/or sensory impairment found in various combination of a visual- perceptual-motor, language or behavior nature that can affect major life activities. These conditions are usually caused by central nervous system dysfunction resulting from unusual or stressful biologic factors occurring during pregnancy, labor or shortly after birth. Socioeconomic and environmental situations may bring out latent biologic features[1]. Despite the 10 percent prevalence of developmental disabilities[2], the early identification of such problems remains difficult. Although severe disorders can be recognized in infancy, it is unusual to diagnose speech impairments, hyperactivity, or emotional disorders before the age of three or four years, and learning disabilities are rarely identified before children start school[3]. Despite the difficulty of diagnosing developmental delay, governmental efforts in many countries have recently been made to

promote early identification and intervention and thus to reduce long-term disability[4]. Since in most young children developmental delays are not associated with a specific diagnosis, definitive therapy, or cure, critics might question the necessity for early identification. However, there is increasing evidence that even in the absence of an etiologic explanation; early identification helps both children and their parents[5]. The best chance for effecting developmental change is while the nervous system of the very young child is still malleable and responsive. Development must be monitored within various areas (fine and gross motor, language, cognitive, and psychosocial development), it is not unusual for one of these areas to be overlooked[6]. If the pediatrician considers the developmental assessment as a matter of ongoing surveillance rather than a screening procedure performed at a particular visit, the yield of developmental assessment will be far greater[7].

There is a growing body of research into early interventions for developmentally disabled infants, children, and their families. Perhaps the most important findings in many researches are the roles of the parents in developmental programs for disabled children younger than three years of age[8]. Modern primary health care includes educational and developmental concerns within its domain and therefore information and recommendations specific to the needs of individuals with developmental disabilities is mandatory [9]. Developmental delay is said to exist when a child does not reach developmental milestones at the expected age. The clinical conditions that comprise the developmental disabilities are cerebral palsy, mental retardation, the epilepsies, vision and hearing impairment and multi-handicapping neurosensory conditions, chromosomal abnormalities as Down syndrome and infantile autism. The myelomeningoceles, myopathies, muscular dystrophies, and the host of hereditodegenerative disorders should be included. All these conditions are considered to be major disabilities[10].There are a number of minor dysfunctions that are likely of similar origins as minimal brain dysfunctions, attention deficit hyperactive behavior, developmental language and visual-perceptual-motor disorders, and learning disorders. These conditions are classified as minor developmental disabilities[11].

In this article, we shall review the different etiologic factors of developmental delay observed in children referred for developmental assessment to the child development center in the Egyptian National Institute of Neuromotor Rehabilitation.

MATERIALS AND METHODS

One thousand one hundred and sixty one children with developmental disabilities were collected from the child development center in the National Institute of Neuromotor Rehabilitation from July 2001 to April 2002. Each child was subjected to:

History taking

A systematic history taking probing for risk factors was done for each child. Areas of concern include biologic risk due to a prenatal or perinatal insult, environmental risk due to a poor caretaking environment, and established risk due to a clearly diagnosed disorder in infancy.

Physical Examination

Risk factors for developmental delay can also be suggested from abnormalities detected in the physical examination. Measurement of head circumference may detect microcephaly or macrocephaly. Dysmorphic features may suggest a chromosomal abnormality. A structural examination of the eyes and functional assessment of the child's vision was performed with use of simple eye-tracking exercises (such as having the infant follow a bright object across the midline and observing for congruence of gaze while tracking). Fundus examination and Visual evoked response was also used to evaluate central vision. If deafness is a concern, hearing was tested with brain-stem evoked potentials. Dermatological examination was done to identify any ectodermal diseases, such as tuberous sclerosis or neurofibromatosis that can be associated with developmental delay. The physical examination also included a developmentally oriented neurological examination with attention to the persistence of primitive reflexes such as a prominent Moro reflex, either hypertonia or hypotonia, or evidence of asymmetry of tone or muscle strength.

Laboratory Assessment

Laboratory assessment done to our patients depended on results of clinical examination. If dysmorphic features were noted, chromosomal studies were done. When the child shows signs of progressive motor or cognitive delay metabolic screening tests for amino acids, mucopolysaccarides and organic acids were done. Children with abnormal muscle tone were screened with a creatine phosphokinase measurement and electromyography because of the possibility of muscular dystrophy. Thyroid-function testing was performed in children who show evidence of developmental delay together with any physical findings associated with thyroid disease, such as thickened tongue, umbilical hernia, and coarse skin. Electroencephalography, computed tomographic scanning, or magnetic resonance imaging was done for children with asymmetric findings on the neurologic examination, weakness in the upper or the lower extremities, spasticity abnormal head growth, seizures, blindness, deafness, or other substantial impairments.

Developmental assessment:

The developmental assessment for each child encompasses a profile of 6 scales in: gross and fine motor development, Cognition, Language, self care and psychosocial development. The profile contains 274 items and is performed in less than an hour by a developmental pediatrician[12].

Gross Motor Development

Motor milestones were used in organizing a developmental review. In addition to overt milestones as rolling over, sitting, standing, and hopping, more subtle indicators were also observed. For instance, the early presence of unilateral dominance or handedness in a child less than 15 months of age may suggest a hemiplegia of the opposite side even without evidence of hypertonicity[13].

Fine Motor Development

Assessment of fine motor abilities began in early infancy. For example, the failure of a baby to unclench his or her fist voluntarily by the age of three months may be an early

indicator of cerebral palsy[6]. Fine motor function at early ages was assessed with a set of blocks. The child was watched at six months of age for hand-mouth activity, and at eight months he or she should be able to bang two blocks together. By 1 1/2 years, the baby may begin to play with the blocks and by 2 years to build a short tower. A three-year-old should be able to make a tower of 6 to 8 blocks.

Language Development

Assessment of language needs to incorporate both the extent of the child's language performance (expressive as well as receptive) and the characteristics of the environment in which the child is learning. language milestones was assessed with a formal testing instrument[14] a surveillance tool designed for routine office use that helps identify receptive, expressive, or visual abnormalities that may contribute to speech delay. This scale has reasonable sensitivity and specificity when it is administered to children less than three years old. It may be helpful to have the child imitate chewing movements, tongue thrusting, and repetition of syllables. This procedure can uncover oral motor problems in phonation and expression.

Cognitive Development

In infants and toddlers, motor and language milestones are often the best proxy for true cognitive assessments. An early hint of difficulties in cognition may come at eight to nine months if a child does not appreciate object permanence. The child who cannot recognize that a hidden object is still present may not be making the appropriate mental connections. The child of 1 to 1 1/2 years old should begin to demonstrate an understanding of cause and effect. Parents were asked whether the child loves to throw a toy down just so the parent can pick it up. The child's laughter indicates an understanding of cause and effect. As the child grows older, cognitive abilities could be tested more formally, with attention to the understanding of size and shape relations, symbolic thoughts, and play, as well as the development of more formal language.[15]

Psychosocial Development

Psychosocial delay manifests itself as behavioral abnormalities that may be early indicators of difficulties in emotional development. The quantity, severity, nature, and duration of these abnormalities can characterize the child as having behavior problem, or psychosocial delay[16]. Severe sleep disturbances, over excitability, or apathy, and toddlers and preschoolers with signs of extreme aggressiveness, fearfulness, or substantial defecation problems were referred for psychological or behavioral testing[17].

After completing the developmental assessment a graph was used to chart the child's performance on each of the profile scales. The graph was completed by marking the highest item number on which the child earned to pass on each scale and connecting the points. A developmental age was established for each child for the 6 profile scales, and a developmental quotient (DQ) was calculated (DQ = developmental age/Actual age × 100) [12]

RESULTS

One thousand one hundred sixty one children were examined, age ranged between 6 and 54 months, mean age 14±8 months. Six hundred twenty six were males (54%). The

etiological factors encountered from history taking in our cases were illustrated in table (1). In 36% of cases no cause could be detected from the history while hypoxic ischemic encephalopathy caused developmental disability in another 16% of cases, the next most common cause were kernicterus (9%) and complications of prematurity in another 8% of cases. Twenty four percent of cases showed other causes with more or less similar percentage as, meningo-encephalitis, intracranial hemorrhage, maternal diseases, TORCH infection, and neonatal convulsions (fig 1).
Regarding developmental assessment, 14% percent of our patients showed normal development. Mild (DQ 75-55), moderate (DQ 50-35) and severe developmental delay (DQ <35) were encountered in 31%, 34% and 29% respectively (table 2).

The final clinical diagnoses reported after physical examination and investigations were illustrated in table (3). Cerebral palsy was the most common clinical diagnosis encountered in 523 cases (45%). Non specific mental retardation comes next to CP and was diagnosed in 376 cases (32%). Other clinical diagnoses encountered less commonly and illustrated in fig (2).

Correlation between etiology and DQ were illustrated in table (4). High percentage of normal DQ 56% and mild developmental delay in 60% were illustrated in cases with irrelevant history.

Regarding the correlation between clinical diagnosis and DQ (table 5), 34% and 46 % if normal DQ occurred in cases with normal clinical examination and cases with CP respectively. Neither of cases with normal clinical examination nor cases with Down syndrome showed severe developmental delay. Cases with neurodegenerative disorders (NDD) and cases with non specific mental retardation always had abnormal DQ. The majority of cases with NDD had severe developmental delay (fig 4).

Table 1. Etiological factors from history taking.

Etiology	number	percent
Unknown	419	36
HIE	184	16
Kernicturus	105	9
Prematurity	92	8
Meningoencephalitis	75	6.5
Maternal Disease	87	7.5
Torch Infection	65	5.5
ICH	65	5.5
Convulsion	69	6
Total	1161	100

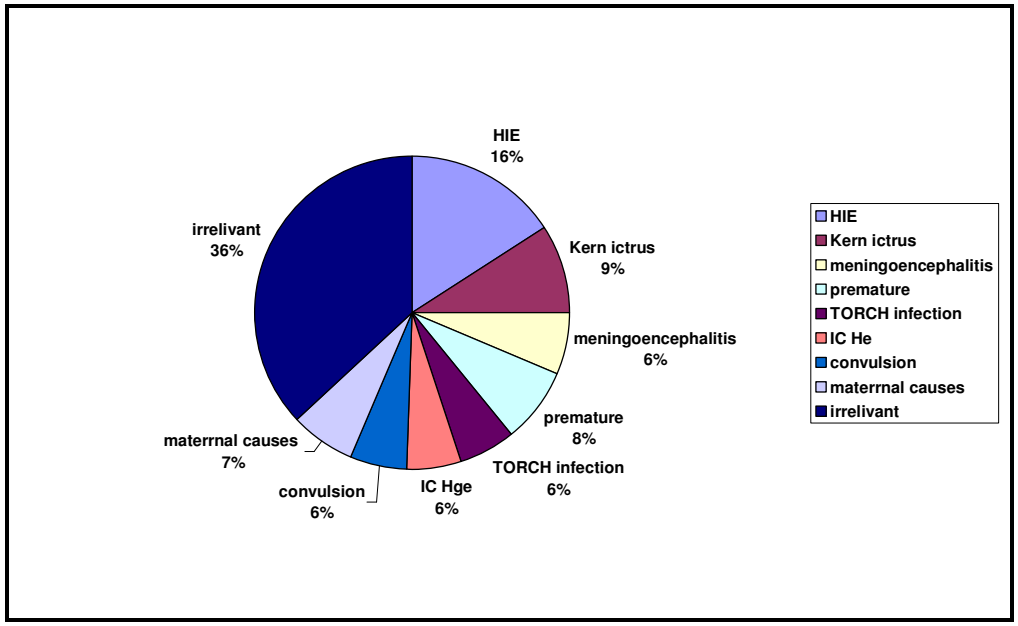

Fig. (1): Etiological factors from history taking.

Table 2. Degrees of Developmental Delay (DD).

Degree Of DD	Number	Percent
Normal (DQ>75)	165	14%
Mild (DQ75-55)	356	31%
Moderate (DQ55-35)	398	34%
Severe (DQ<35)	242	21%
Total	1161	100%

Table 3. Number and percentage of each clinical diagnosis.

clinical diagnosis	number	percent
Normal	101	8
Cerebral Palsy	543	48
Mental Retardation	322	28
Congenital Anomalies	87	7
Down Syndrome	72	6
Autism	24	2
Neurodegenerative	12	1
Total	1161	100

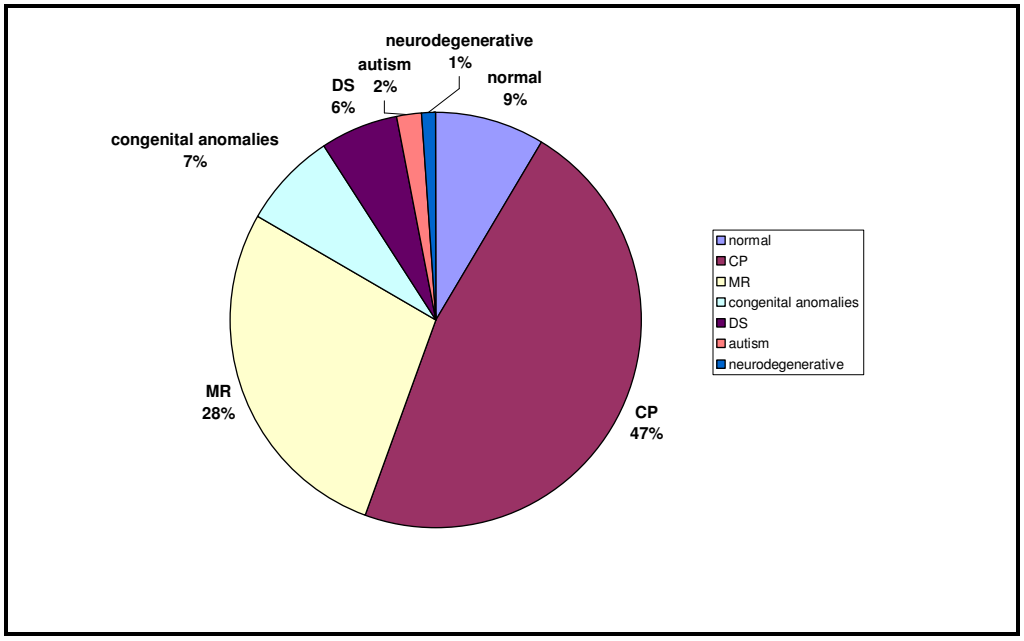

Fig. (2): Clinical diagnosis encountered.

Table 4. Correlation between etiology and DQ**.**

Etiology	normal		mild		moderate		severe		total	
	No	%	No	%	No	%	No	%	No	%
Irrelevant	93	56	215	60	71	18	40	16	419	35
HIE	9	5	25	7	103	26	47	19	184	16
Kernictrus	7	4	17	5	54	14	27	11	105	9
Prematurity	8	5	19	5	41	10	24	10	92	8
Meningo-encephalitis	6	4	17	5	20	5	32	13	75	7
Maternal Disease	10	6	12	3	26	8	39	16	87	7
Torch Infection	8	5	9	3	31	9	17	7	65	6
ICH	9	5	7	1	39	10	10	4	65	6
Convulsion	15	9	35	10	13	4	6	3	69	6
Total	165	99	356	99	398	99.	242	99	1161	100

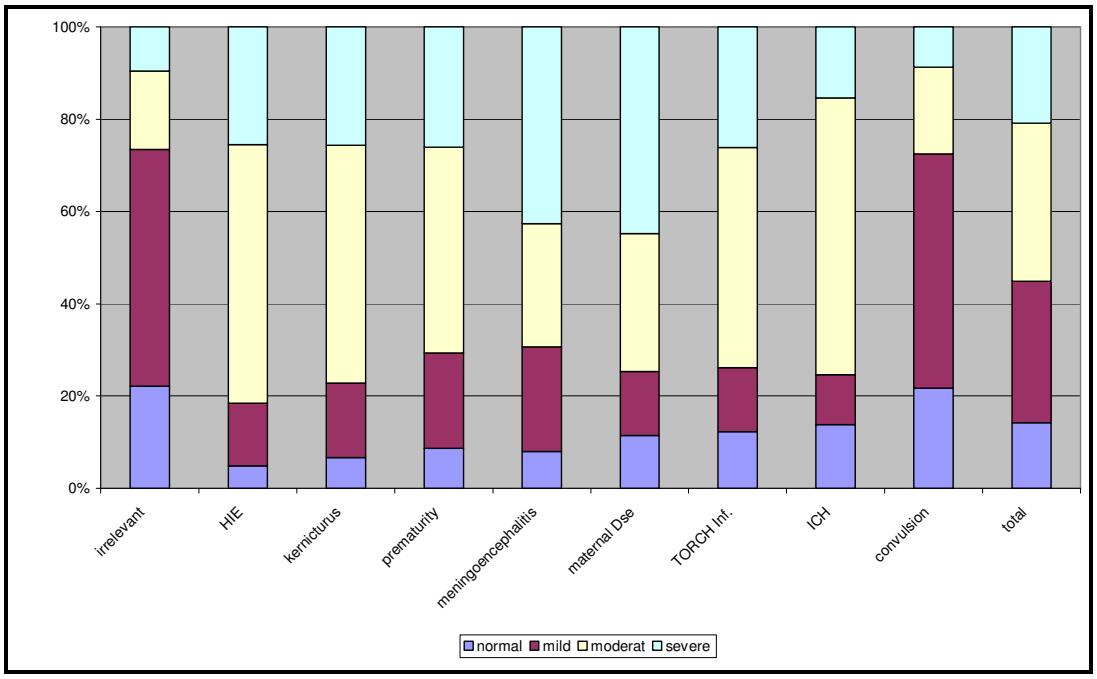

Fig. (3): Correlation between etiology and DQ.

Table 5. Correlation between clinical diagnosis and DQ.

Clinical diagnosis	normal		mild		moderate		severe		total	
	No	%	No	%	No	%	No	%	No	%
Normal	56	34	45	12.5	0	0	0	0	101	9
Cerebral Palsy	76	46	153	43	202	51	112	46	543	47
Mental Retardation	0	0	89	25	135	34	98	40	322	28
Congenital Anomalies	21	13	23	6	23	6	20	8	87	7
Down Syndrome	7	4	36	10	29	7	0	0	72	6
Autism	5	3	8	2	6	1.5	5	2	24	2
Neurodegenerative	0	0	2	0.5	3	0.75	7	3	12	1
Total	165	100	356	99	398	100	242	99	1161	100

DISCUSSION

Pediatricians should be concerned with the achievement of optimum development of all children.[18] Early identification of developmental delay is mandatory as it helps both children and their parents[5]. If parents understand where their children are along a developmental trajectory, they can tailor their expectations and provide equipment, stimulation, and toys to match the child's "readiness" for a particular type of experience at each stage. Early identification allows the family members to feel that they are doing all they can to assist the child[19] and prevent secondary emotional disability. In some instances it is possible to come up with a diagnosis of a genetic, metabolic, or infectious disease, early identification can often prevent further damage. In the present study the mean age attending the child development center was 14 months ±8, when the parents were asked about the reason why not coming earlier the majority did not know that there is something abnormal with their children and their doctors did not inform them about the importance of early identification and early intervention. Glascoe, [20] found that fewer than 30% of children with developmental disabilities are detected by their health care providers. So he concluded that the in-office services designed to detect and address developmental and behavioral problems are insufficient. The success of early identification of children with developmental and behavioral problems is influenced by the manner in which pediatricians elicit, recognize, and select clinical information and derive appropriate impressions. Parents are ready sources of clinical information, and they can be asked to provide many broad types of data[21]. Establishing the presence of developmental delay can be challenging. The wide normal variation among children often makes it easy for a subtle finding to be passed over. Moreover, since delay must be monitored within various areas (fine and gross motor, language, cognitive, and psychosocial development), it is not unusual for one of these areas to be overlooked[6]. If the pediatricians consider the assessment of delay as a matter of ongoing surveillance rather than a screening procedure performed at a particular visit, the yield of developmental assessment will be far greater[7].

Perinatal risk factors were detected from the history of 76% of our cases. This is relatively high percentage and reflects the insufficient antenatal natal and postnatal care. Every effort should be directed in this issue especially for prevention of kernicterus which was elicited in (9%), hypoxic ischemic encephalopathy in (16%) and complication of prematurity in (8%). Garaizar and Parats-Venas [22] studied 111 patients with developmental delay. They found hypoxic-ischemic encephalopathy in 20%, lesions due to prematurity in 26%, intrauterine infection or neonatal meningitis in 11%, unexplained vascular brain lesions in 10%, and late intrauterine brain lesions in 33%.

Thompson et al.[23], studied the contribution of early biological and psychosocial risk factors to developmental outcome of low birth weight infants. Development was assessed at 4 years of age. Biological risk, assessed by the Neurobiologic Risk Score, accounted for significant portions of the variance in the perceptual-performance (17%) and motor (35%) dimensions of the child development. The findings were discussed in terms of early

markers for low birth weight infants who require careful follow-up and early intervention targets to promote developmental outcome.

Kohlhauser et al.[24], studied the developmental and neurological outcome of 72 very-low-birth-weight infants at 1 and 2 years corrected for post conceptional age. They also assessed the variables predicting outcome. They found that at 1 year 24% of the children were neurologically normal and the rate increased to 61% at 2 years. The rate of cognitively normal children remained constant (58% at 1 year and 59% at 2 years) indicating that developmental status at 1 year was predictive for the second year. They concluded that this early period is important, for the identification of developmental deficits and for establishing early adequate interventions. Chaudhari et al.[25], undertook a prospective study to determine the development of preterm babies. They followed one hundred and seventy two preterm babies and 36 control babies for a period of 18-24 months. They assessed psychomotor development using the Bayley Scales of Infant Development. They found that higher the birth weight better was the mean motor developmental quotient at 18 months. Also uncomplicated preterm babies showed higher mean developmental quotients at 18 months than preterm babies with additional complications. The incidence of cerebral palsy in their series was low (4%). Gutbrod et al.[26], related small gestational age to early developmental delay and later language problems; however, they found that neonatal complications may have a larger detrimental effect on long term cognitive development of infants than whether they are born small gestational age or not. Holst et al.[27], investigated the impact pregnancy related risk factors, complicated delivery, and perinatal morbidity on subsequent development in children.

Garaizar and Parats-Venas[22] found the following neurological sequela in their cases, cerebral palsy (68%), epilepsy (47%), mental retardation (45%), learning disorders (34%), microcephaly (19%), visual disturbance (14%) and hyperkinesis in (10%). Cerebral palsy was also the most common clinical diagnosis encountered in our cases (45%). Non specific mental retardation comes next to CP and was diagnosed in (32%). Other congenital anomalies were reported in 7% and Down syndrome in another 6% of cases. Yamada[28] did a medical screening for cerebral palsy, mental retardation and Down syndrome. They reported an incidence of 2, 8 and 1 per 1000 respectively. The rate of perinatal brain damage with cerebral palsy was 81.8%. The same study was done by Suzuki et al.[29] in another city in Japan. They had the same conclusion but the rate of Down syndrome was higher (2/1000). Okumura et al.[30], studied the etiology of cerebral palsy in 76 cases. Congenital CNS anomalies were noted in 8 cases. The main perinatal factors were asphyxia, dyspneic conditions needing mechanical ventilation prolonged apneic spells, hyperbilirubinemia. All their full term birth children were accompanied with asphyxia in which 43% had intracranial hemorrhage.

Conclusion:

The high incidence of perinatal etiology in our cases raises the importance of good maternal and neonatal care. Prophylactic measures should be done against neonatal hyperbilirubinaemia. Genetic counseling is essential in consanguineous marriage.

All high risk infants should receive careful pediatric follow up that includes developmental screening. A system of tracking and monitoring high risk infants during infancy and childhood would allow for early identification of developmental delay.

There must be a cooperation network between each neonatal intensive care unit and an institute for the handicapped children. Early detection of developmental delay permits enrollment in an early intervention programs. Longitudinal studies are needed to evaluate the plasticity of the brain after early damage and the possible role of intervention in brain reorganization.

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