ROLE OF MRI IN DETECTING GROSS CHANGES OF BRAIN IN PAEDIATRIC PATIENTS WITH GLOBAL DEVELOPMENTAL DELAY

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ABSTRACT:

Background: Developmental delay is common, affecting 1-3% of the population. Developmental delay is defined as significant delay (more than two standard deviations below the mean) in one or more of the following developmental domains.

Objectives: To study gross changes on MRI brain in patients with global developmental delay and to emphasize the importance of MRI in detecting pathological changes in brain.

Methodology: 50 cases of pediatric patients with developmental delay were evaluated over a period of eight months. Detailed MRI Brain scan was done. MR imaging was performed in Plain T1W, post contrast T1W, T2W, FLAIR and DWI sequences were taken as and when necessary.

Results: 33 percentage of the cases were of cerebral dysgenesis, followed by trauma, genetic, metabolic conditions in descending order.

Conclusion: MR imaging remains the ideal scanning modality to evaluate gross changes in brain in pediatric patients with global developmental delay.

Key words: Global developmental delay, incomplete lissencephaly, polymicrogyria, focal cortical heterotopia, semilobar holoprosencephaly, microcephaly, perisylvian syndrome, hemimegalencephaly, corpus callosal agenesis, acute disseminated encephalopathy, post infectious, unilateral open lip schizencephaly, nasoethmoid encephalocele, fibrous cortical dysplasia, post anoxic ischaemic changes.

I. INTRODUCTION AND DEFINITIONS:

Developmental delay is common, affecting 1-3% of the population. Developmental delay is defined as significant delay (more than two standard deviations below the mean) in one or more of the following developmental domains[1] Gross motor, Vision & Fine motor, Hearing, Speech & Language, Social, Emotional & Behavioral

AIMS AND OBJECTIVES:

To study gross changes on MRI brain in patients with global developmental delay and to emphasize the importance of MRI in detecting pathological changes in brain.
CAUSES OF GLOBAL DEVELOPMENTAL DELAY

<table>
<thead>
<tr>
<th>CATEGORY</th>
<th>CONDITIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>GENETIC OR SYNDROMIC</td>
<td>Down’s syndrome, Fragile X syndrome, Klinefelter</td>
</tr>
<tr>
<td>METABOLIC</td>
<td>Phenetonuria, medium chain acyl-Co A Dehydrogenase deficiency, Galactosemia, degenerative disorders of the CNS, Mucopolysaccharidosis, Aminoaciduria-phenylketonuria, Homocystinemia, Histidinemia, Organic aciduria</td>
</tr>
<tr>
<td>ENDOCRINE</td>
<td>Congenital hypothyroidism</td>
</tr>
<tr>
<td>TRAUMATIC</td>
<td>Acquired brain injury, Birth injuries, Hypoxic Ischemic encephalopathy</td>
</tr>
<tr>
<td>ENVIRONMENTAL CAUSES[3]</td>
<td>Children require their basic needs for food, clothes, warmth, love and stimulation to bemet to develop normally. Children in neglectful, abusive, fearful, under stimulated environments may not shownormal development. Iodine deficiency.</td>
</tr>
</tbody>
</table>

| CEREBRAL MALFORMATIONS          | Neuronal migration disorders, Neural tube closure defects, disorder of cleavage and sacculations |
| NEUROECTODERMAL DYSPLASIA       | Tuberous sclerosis                                                                                                                                |
| CEREBRAL PALSY AND DEVELOPMENTAL COORDINATION DISORDER [DYSPRAXIA] [4] | Motor difficulties can prejudice developmental in general                                                                                       |
| INFECTIONS                      | Perinatal e.g. Rubella, CMV, HIV Neonatal meningitis                                                                                            |
| TOXINS[5]                       | Fetal or maternal alcohol or drugs in pregnancy Childhood: lead toxicity                                                                         |

II. METHODS AND MATERIALS

50 cases of pediatric patients with developmental delay were evaluated over a period of eight months. Detail clinical history with clinical examination followed by MRI Brain scan was done. MR imaging was performed using 1.5 Tesla Phillips Achiva. Plain T1W, post contrast T1W, T2W, FLAIR and DWI sequences were taken as and when necessary.

III. RESULTS

50 cases of patients with developmental delay were evaluated. MRI brain is the modality of choice in investigating gross changes occurring in brain, in patients with developmental delay[6][7]. 33 percentage of the cases were of cerebral dysgenesis, followed by trauma, genetic, metabolic conditions in descending order. Percentage wise occurrence of each conditions have been mentioned in the pie chart.

<table>
<thead>
<tr>
<th>CONDITIONS</th>
<th>NORMAL SCAN</th>
<th>POSITIVE SCAN</th>
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</thead>
<tbody>
<tr>
<td>INFECTIONS</td>
<td>YES</td>
<td>YES</td>
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<tr>
<td>TRAUMA</td>
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<td></td>
</tr>
<tr>
<td>GENETIC SYNDROMES</td>
<td>YES</td>
<td>YES</td>
</tr>
<tr>
<td>METABOLIC</td>
<td>YES</td>
<td></td>
</tr>
<tr>
<td>NEURAL TUBE CLOSURE DEFECTS</td>
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<td></td>
</tr>
<tr>
<td>CEREBRAL DYGENESIS</td>
<td></td>
<td>YES</td>
</tr>
<tr>
<td>NON-SPECIFIC LEUKODYSTROPHY LIKE CHANGES</td>
<td></td>
<td>YES</td>
</tr>
<tr>
<td>ENDOCRINE</td>
<td>YES</td>
<td></td>
</tr>
<tr>
<td>ENVIRONMENTAL</td>
<td>YES</td>
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</tr>
</tbody>
</table>
IV. DISCUSSION:

INCOMPLETE LISSENCEPHALY

Axial, cor and sag images show areas of pachy and agyria mainly in the parieto-occipital lobes bilaterally. Shallow sulci are noted in the frontal lobes bilaterally [8].
CLOSED LIP SCHIZENCEPHALY

Using CT, the diagnosis of schizencephaly is sometimes difficult, particularly in closed lip schizencephaly. CT scans in closed-lip may show only a slight outpouching at the ependymal surface of the cleft, and a full-thickness cleft may be difficult to identify on CT scan. The cleft is partially or totally lined by gray matter and extends from the lateral ventricle to the pial surface of the cerebral hemisphere. MRI is the modality of choice. MRI delineates the gray matter lining the cleft, (pathognomonic finding)[9][10]. Associated findings like heterotopias, pachygyria or polymicrogyria are better defined by MRI.

POLYMICROGYRIA

Fig. Polymicrogyria: is seen in the much of the right cerebral hemisphere with undulating of the cortex. Left cerebral hemisphere shows normal cortex and cortico-medullary differentiation
FOCAL CORTICAL HETEROTOPIA

FOCAL heterotopic nodules in the right frontal white matter lobe. CLUSTERS of heterotopic nodules in the right temperoparietal region extending from the cortex to the periventricular white matter. C/F: Pt presented with refractory seizures disorders, mild developmental delay, motor dysfunction.

SEMILOBAR HOLOPROSENCEPHALY

Semilobar holoprosencephaly: Axial T2 & Sag T1WI shows partial temporal and occipital horns with moderate dorsal cyst.

MICROCEPHALY / MICROLISSENCEPHALY

MR: large subarachnoid spaces, impaired myelination, reduced volume of white matter, very simplified gyral pattern with no more than about 5 gyri in each hemisphere and sulci less than 1/3rd depth of normal. Cerebral cortex is thin[11].
Fig. Bilateral perisylvian polymicrogyria: Axial, cor & sag images show abnormality of the insula and fronto-parietal cortex. These findings are best appreciated on SPGR seq( 3D T1 wt GRE seq)

PERISYLVIAN SYNDROME

PERISYLVIAN SYNDROME may be sporadic/ familial. Mutation of several different genes can cause this malformation. C/f: pseudobulbar palsy, epilepsy, mental retardation. Familial cases are less symptomatic[12]. MR shows abnormal cortex and irregularity of the cortical white matter junction with polymicrogyria in the perisylvian region.

HEMIMEGALENCEPHALY

Fig: Hemicagecephaly with bizarre, hamartomatous enlargement of the left hemisphere. Cortex is thickened and convoluted.

Imaging: Cortex is dysplastic, with broad gyri, shallow sulci, and cortical thickening.
Gyral pattern may be normal or agyric. White matter usually shows heterogenous signal due to heterotopias and dysplastic neurons and glia. MR shows involvement of part or all of the hemisphere. A characteristic finding is straightening of the ipsilateral frontal horn of the enlarged ventricle.

**COMPLETE CORPUS CALLOSAL AGENESIS WITH COLPOCEPHALY**

Both ventricles are separated, with dilated trigone and occipital horns of lateral ventricles with complete absence of corpus callosum.

**ACUTE DISSEMINATED ENCEPHALOPATHY**

Multiple asymmetrical lesions which are hyperintense on T2W and FLAIR images in white matter, periventricular region, basal ganglia, parietal occipital lobes, subcortical region and cerebellum on either side of midline. Lesions are hypointense on T2W images, postcontrast shows ring enhancing lesions along the ependymal walls of lateral ventricles.
POST INFECTIVE VIRAL ACUTE DISSEMINATED
Multiple well defined lobulated lesions involving bilateral basal ganglia, periventricular regions, subcortical region and brainstem. They are iso-hypointense on T1W image, hyperintense on T2W, FLAIR and DWI

UNILATERAL OPEN LIP SCHIZENCEPHALY
Well defined triangular cystic lesion extending from margin of body of left lateral ventricle to cortex, having density of CSF which is hypointense on T1W images and hyperintense on T2W images.

NASOETHMOID ENCEPHALOCELE
Well defined expensile cystic lesion seen in nasoethmoid region, more on right side. Lesion is hypointense on T1W images and hyperintense on T2W images, displacing both orbits laterally. Associated findings are trigone colpocephaly with absence of corpus callosum agenesis.

BILATERAL FIBROUS CORTICAL DYSPLASIA
Hyperintense lesions on T2W images and hypointense on T1W images, is seen at cortical region at junction of grey white matter close to post central sulci on both sides, more marked on left side. Post contrast TW image does not show any abnormal enhancement.
POST ANOXIC HYOXIC ISCHEMIC CHANGES

Multiple small hyperintense signals on T2W images in left temporal and parietal lobe, which appear hypointense on T1W and FLAIR image. There is also an edema surrounding the lesion.

V. CONCLUSION

MR imaging remains the ideal scanning modality to evaluate gross changes in brain in pediatric patients with global developmental delay.

REFERENCES: