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

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INTRODUCTION:

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

CAUSES OF GLOBAL DEVELOPMENTAL DELAY

CATEGORY	CONDITIONS
GENETIC OR SYNDROMIC	Down's syndrome, Fragile X syndrome, Klinefelter
METABOLIC[2]	Phenletonuria, medium chain acyl-Co A Dehydrogenase deficiency, Galactosemia,

	degenerative disorders of the CNS, Muccopolysaccharidosis, Aminoaciduria-phenylketonuria, Homocystinemia, Histidinemia, Organic aciduria
ENDOCRINE	Congenital hypothyroidism
TRAUMATIC	Acquired brain injury. Birth injuries, Hypoxic Ischemic encephalopathy
ENVIRONMENTAL CAUSES[3]	Children require their basic needs for food, clothes, warmth, love and stimulation to be met to develop normally. Children in neglectful, abusive, fearful, under stimulated environments may not show normal development. Iodine deficiency.
CEREBRAL MALFORMATIONS	Neuronal migration disorders, Neural tube closure defects, disorder of cleavage and sacculaitons
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

AIMS AND OBJECTIVES

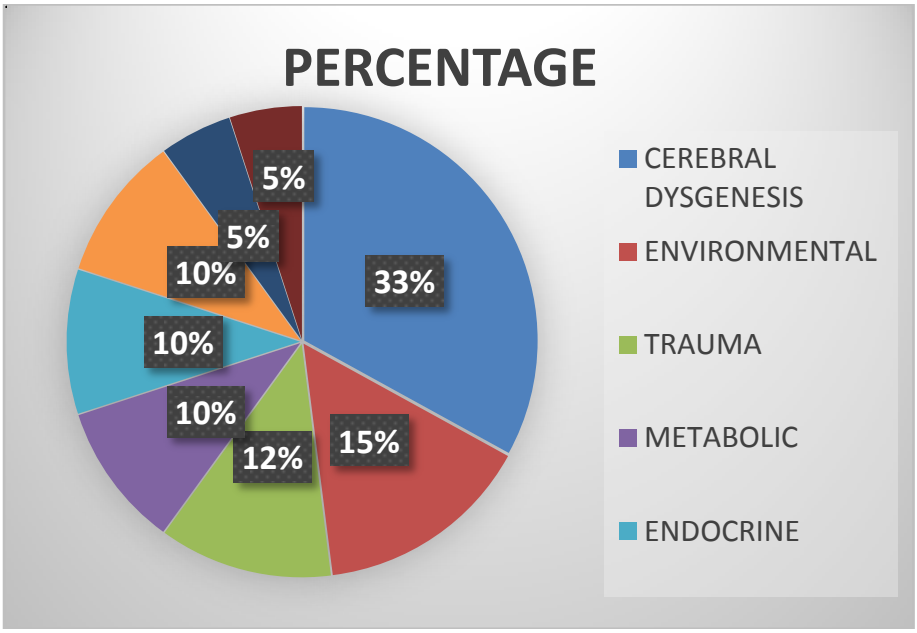
To study gross changes on MRI brain in patients with global developmental delay and to emphasize theimportance of MRI in detecting pathological changesin brain

METHODS AND MATERIALS

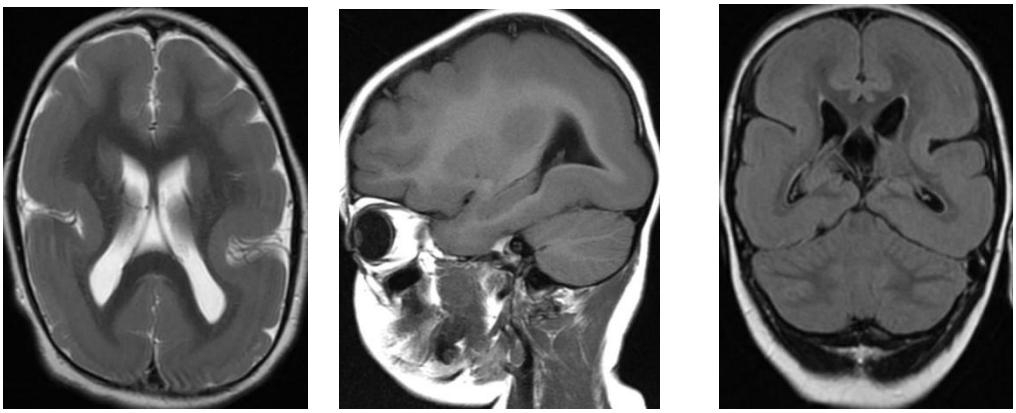
50 cases of pediatric patients with developmental delay were evaluated over a period of eight months. Detail clinical history with clinical examination followed by MRI Brain scan was done.MR imaging was performed using 1.5 Tesla Phillips Achiva. Plain T1W, post contrast T1W, T2W, FLAIR and DWI sequences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

RESULT

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. CONDITIONS	NORMAL SCAN	POSITIVE SCAN
INFECTIONS		YES
TRAUMA		YES
GENETIC SYNDROMES	YES	YES
METABOLIC	YES	
NEURAL TUBE CLOSURE DEFECTS		YES
CEREBRAL DYGENESIS		YES
NON-SPECIFIC LEUKODYSTROPHY LIKE CHANGES		YES
ENDOCRINE	YES	
ENVIRONMENTAL	YES	



INCOMPLETE LISSENCEPHALY



Axial, coronal and sagittal 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

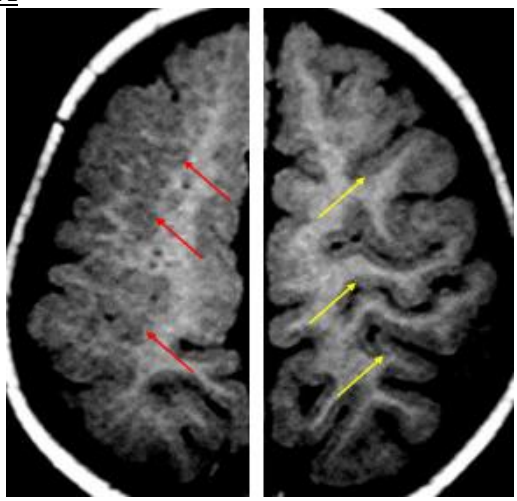


Fig. Polymicrogyria: is seen in the much of the right cerebral hemisphere with undulating of the cortex. Left cerebral hemisphere shows normal cortex and cortico-medullary differentiation.

FOCAL CORTICAL HETEROTOPIA

Cluster heterotopia

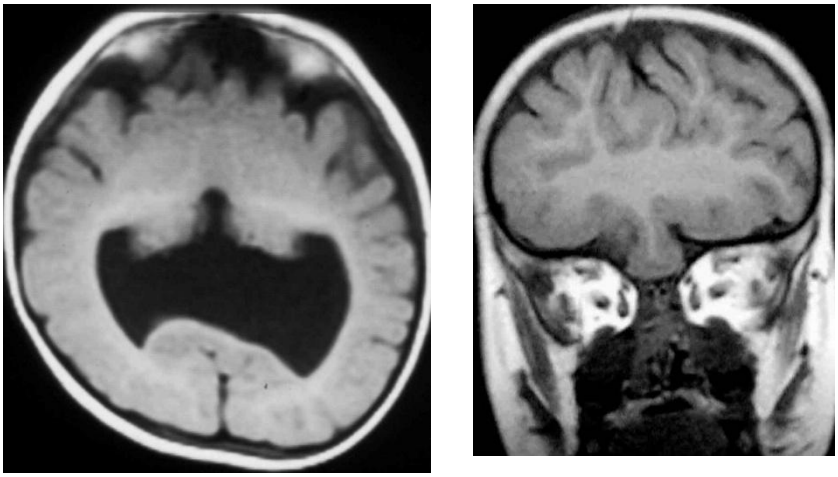
FOCAL heterotopic nodules in the right frontal white matter lobe.
CLUSTERS of heterotopic nodules in the right temperoparietal region extending from the cortex to the periventricular white matter.
C/F: Pt presented with refractory seizures disorders, mild developmental delay, motor dysfunction.

SEMILOBAR HOLOPROSENCEPHALY

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

SEMILOBAR HOLOPROSENCEPHALY

Axial & Coronal T1WI shows partial temporal and occipital horns with fusion of the frontal lobes and thalami.



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].

PERISYLVIAN SYNDROME

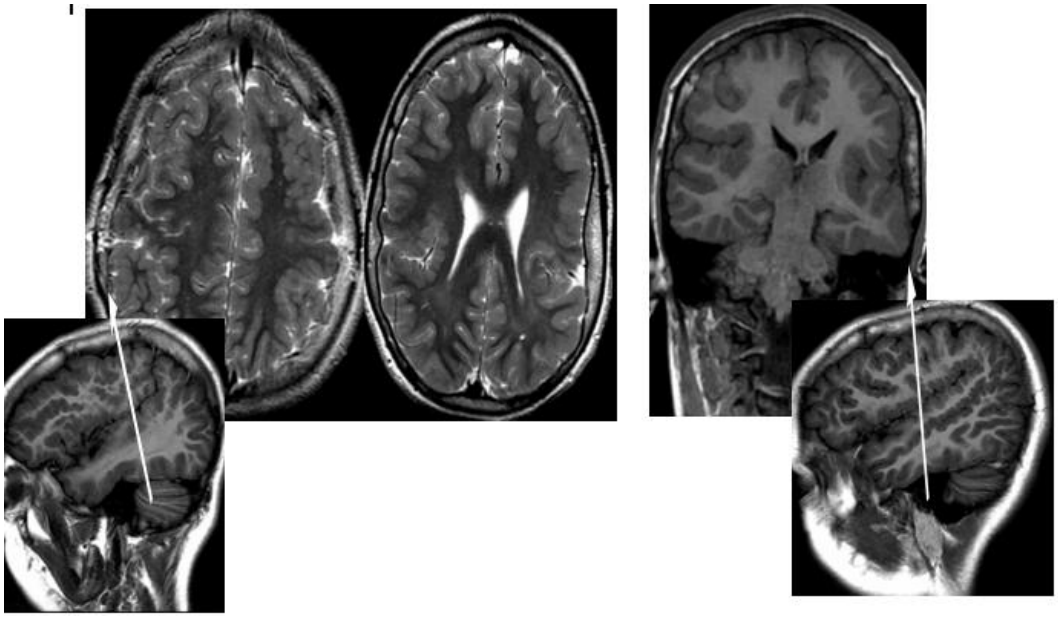


Fig. Bilateral perisylvian polymicrogyria: Axial, cor & sag images show abnormality of the insula and fronto-parietal cortex. These findings are best appreciated on SPGR sequences (3D T1 wt GRE seq)

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
Imaging: Cortex is dysplastic, with broad gyri, shallow sulci, and cortical thickening.
Gyral pattern may be normal or agyric. White matter usually shows heterogenopus 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.

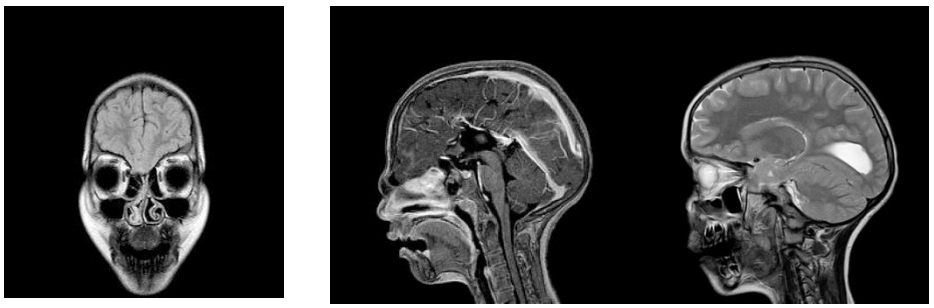
COMPLETE CORPUS CALLOSAL AGENESIS WITH COLPOCEPHALY
Both ventricles are seperated, with dilated trigone and occipital horns of lateral ventricles with complete absence of corpus callosum

ACUTE DISSEMINATED ENCEPHALOPATHY
Multiple asymmetrical lesions which are hyperintense on T2W and FLAIR images in white matter, periventricular region, basal ganglia, parietal occipiatal 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 TW, FLAIR and DWI

UNLATERAL OPEN LIP
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

NASOETHMOIDEN CEPHALOCELE



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 dosent 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 odema surrounding the lesion.

CONCLUSION

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

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