The role of diffusion tensor imaging (DTI) in developmental brain anomalies in pediatric age group of Egyptian patients

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Aims and objectives

The developmental brain anomalies are commonly encountered in day to day practice, it is very important for every radiologist to be familiar with the basic imaging findings of common anomalies to make correct diagnosis necessary for optimum management of these conditions.

Congenital anomalies of the central nervous system (CNS) often demonstrate aberrant white matter connections, which may be better characterized with diffusion tensor imaging (DTI) and fiber tractography (FT) than with conventional magnetic resonance (MR) imaging, in an earlier stage than conventional T2- or T1-weighted imaging.[1]

DTI is an advanced neuroimaging technique that can be used to characterize the orientational properties and allows quantification of the diffusion process of water molecules in vivo, to be used as surrogate markers of the integrity of white matter (WM) microstructure. [2-4]

The changes in DTI parameters were investigated to gain insight into intraregional and interregional developmental changes in the major WM tracts and infer details about myelination and axonal development. [2]

DTI has already shown promising results in the study of various developmental brain diseases. [5]

And thus came the aim of our study to evaluate the role of Diffusion-tensor imaging (DTI) and fiber tractography (FT) in:

- Assessment of brain white matter changes and developmental CNS anomalies in pediatric age group
- Demonstration of the orientation and integrity of white matter fibers in vivo.
Methods and materials

Patient selection

Case control prospective study included 21 subjects.

Inclusion criteria:

- From neonates to 15 year old having neurological dysfunction referred from (Neurology outpatient clinic, Cairo university children hospital (Cuch)) to Radiology Department; Cairo University Hospitals.
- Ten control subjects of the same age group to get rough measurement for white matter FA values and anatomical tracts configuration.

Exclusion criteria:

- General: Claustrophobic patients, patients with pacemaker or metallic implants
- Specific: patients suffering from ischemic or traumatic brain injuries.

Each patient was subjected to:

- Full history taking.
- Reviewing medical sheet whenever possible.
- Our institutional review board waived the requirement of informed consent; however normal controls subjected to informed consent according to the ethical committee regulations.

MR examination included:

- Non contrast Conventional MR examination including T1WI, T2WI, and FLAIR(fluid attenuation inversion recovery) in axial, sagittal and coronal planes
- Diffusion Tensor imaging medium.

Technique:

- Technique was performed using a standard 1.5 Tesla unit.
- A standard head coil was used.
- Patient position: Supine, 20 were under sedation.
- Scan time was around 12 minutes.

Imaging parameters:
• **T1WI:** TR 450 ms, TE 15 ms, Flip 69°, Matrix 180 x 169, FOV 210 x 236, Number of excitation: 2, Slice thickness: 6.0/1.5
• **T2WI:** TR 3619 ms, TE 100 ms, Flip 90°, Matrix 192 x 165, FOV 210 x 236, Number of excitation: 2, Slice thickness: 6.0/1.5
• **FLAIR:** TR/TI 6000/2000 ms, TE 120 ms, Matrix 208 x 192, FOV 210 x 236 mm, Number of excitation: 2, Slice thickness: 6.0/1.5
• **Diffusion weighted imaging (DWI):** A diffusion weighting factor of b zero and 1000 s/mm², TR 4100 ms, TE 115 ms, Flip 90°, Matrix 132 x 105, FOV 210 x 236 mm, Number of excitation: 1, Slice thickness: 6.0/1.5
• **Diffusion Tensor consisted of:** A single shot, spin-echo echo planar sequence in 16 encoding directions, A diffusion weighting factor of 800 s/mm², TR 8000 ms, TE 67 ms, Flip 90°, matrix 112 x 110, FOV 210 x 236 mm, number of excitations: 2, slice thickness: 2.0/0.00

**Post-processing:**
• All the diffusion-weighted images were transferred to the workstation.
• Images were post-processed using multiple region of interest (ROI) technique in the software assigned for tractography and supplied by the manufacturer.

**The maps obtained were:**
• 3D DTTI (diffusion tensor tractography images)
• 2D axial DTI superimposed upon FLAIR or T2 images

**Tracts drawn:**
• Corpus callosum (CC)
• Cortico-spinal tracts (CST)
• Superior longitudinal fasciculus (SLF)
• Inferior longitudinal fasciculus (ILF)

And guided by the pathology in conventional MRI pathology additional tracts were drawn accordingly (e.g. Superior cerebellar peduncle (SCP), Probst bundle)

**Regional white matter fractional anisotropy to be measured at:**
• Bilateral Frontal white matter regions.
• Bilateral parietal white matter regions.
• Bilateral occipital white matter regions.

**Statistical analysis:**

**Concerning the demographic data of the patients:**
Cases with positive conventional MRI findings were seven males and four females.

Cases in the age group from (0-5) years were eight patients.

Cases in the age group from (6-10) years was only one patient.

Cases in the age group from (11-15) years were two patients.

Concerning the study outcome:

Nine cases out of eleven (81.8%) cases with positive conventional MRI findings showed additional DTI findings.

Combined structural and anatomical disorders were noted in five out of eleven cases.

Demonstrative control subject:

An eleven years old normal female. Fig. 1 on page 6 Fig. 2 on page 6 Fig. 3 on page 7 Fig. 4 on page 7 Fig. 5 on page 8

Table one : Regionl white matter FA values.

<table>
<thead>
<tr>
<th>white matter region</th>
<th>right</th>
<th>left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal</td>
<td>0.68</td>
<td>0.68</td>
</tr>
<tr>
<td>parietal</td>
<td>0.71</td>
<td>0.71</td>
</tr>
<tr>
<td>occipital</td>
<td>0.66</td>
<td>0.66</td>
</tr>
</tbody>
</table>
**Fig. 1:** An eleven years old normal female. 3DDTI showing the normal orientation and the average thickness of the corticospinal tract.

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**Fig. 2:** 3DDTI showing the average thickness and orientation of corpus callosum.

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**Fig. 3:** 3DDTI showing the average thickness and orientation of the superior longitudinal fasciculi.

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**Fig. 4:** 3DDTI showing the average thickness and orientation of the inferior longitudinal fasciculi.
**Fig. 5:** 2D color map superimposed upon axial FLAIR measuring average white matter FA at different regions of interest (ROI). Frontal, parietal and occipital white matter.

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Results

A case was reported by conventional MRI as complete corpus callosum agenesis, by DTI-FT it was proved that it was partial callosal agenesis! To our knowledge no such finding was previously reported. There was sparse fibers at the region of the genu taking H shaped pattern as described by Adolf [6] in cases of partial agenesis. There was also relative decrease in the Periventricular white matter FA more advanced on the frontal region (table 2) Fig. 6 on page 13 Fig. 7 on page 13 Fig. 8 on page 14 Fig. 9 on page 14

Table 2: Regional white matter FA values

<table>
<thead>
<tr>
<th>white matter region FA</th>
<th>right</th>
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<tbody>
<tr>
<td>Frontal</td>
<td>0.19</td>
<td>0.19</td>
</tr>
<tr>
<td>parietal</td>
<td>0.3</td>
<td>0.33</td>
</tr>
<tr>
<td>occipital</td>
<td>0.25</td>
<td>0.26</td>
</tr>
</tbody>
</table>

A case was reported by conventional MRI as Joubert-dandy complex with mid brain typical molar tooth appearance, by the DTI-FT the SCP was thin attenuated which was against that was described by Lee[1] that the SCP was thickened and elongated SCP with a horizontal configuration can be seen. Fig. 10 on page 15 Fig. 11 on page 16 Fig. 12 on page 16

A case was reported by conventional MRI with Diffuse non uniform sulcal arrangement "polymicrogyra" with multiple bilateral periventricular white matter signal alteration hyper-intense in T2 and FLAIR with vacuolated appearance proven Zellweger syndrome. by DTI-FT there was thick callosal fibers and crossing cortico-spinal tract cranially, associated with decreased mild decrease in the FA of the frontal and parietal regions(table3). Fig. 13 on page 17 Fig. 14 on page 18

Table 3: Regional white matter FA values

<table>
<thead>
<tr>
<th>white matter region FA</th>
<th>right</th>
<th>left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal</td>
<td>0.23</td>
<td>0.24</td>
</tr>
<tr>
<td>parietal</td>
<td>0.14</td>
<td>0.2</td>
</tr>
<tr>
<td>occipital</td>
<td>0.4</td>
<td>0.35</td>
</tr>
</tbody>
</table>
A case was reported in this study conventional MRI with pachygryrii and cortical dysplasia by DTI-FT there was disruption of the callosal fibers posteriorly at the lesion of the defective sulcation around the dysplasia ,as described by Lee[1]

A case was reported in this study conventional MRI as "subependymal periventricular diffuse nodular heterotopia" by DTI-FT there was no significant extra information apart from the increased FA in the heterotopic grey matter than the normal grey matter which coincides by the radial migration of the grey matter described by Lee[1]

A case was reported in this study with conventional MRI as a large CSF filled inter-hemispheric cyst ,by DTI- FT the lower part of the CST and superior cerebellar peduncle was splayed around the cyst especially on the left side by virtue of it's mass effect

A case was reported in this study by the conventional MRI as corpus callosum agenesis , the diagnosis was confirmed by the DTI-FT and the misdirected antro- posterior probst bundle was shown this coincides with Lee [1]

A case was reported in this study by conventional MRI with complementary MRS with Diffuse white matter T2 hyper intensity in the deep and periventricular and subcortical fibers (MRS revealed large lactate peak small MI peak ;no reduction of NAA, no increase of choline ;radiologically suggesting Leukodystrophy proven adrenoleukodystrophy , by DTI- FT there was interruption of callosal fibers in its central portion and non visualized superior longitudinal fasiculi ; the fractional anisotropy were reduced on both sides in frontal parietal and occipital regions(table 4) coinciding with the axonal loss described by Ishak[7] Fig. 15 on page 18 Fig. 16 on page 19

Table 4:Regional white matter FA values

<table>
<thead>
<tr>
<th></th>
<th>right</th>
<th>left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal</td>
<td>0.1</td>
<td>0.2</td>
</tr>
<tr>
<td>parietal</td>
<td>0.2</td>
<td>0.1</td>
</tr>
<tr>
<td>occipital</td>
<td>0.11</td>
<td>0.1</td>
</tr>
</tbody>
</table>

A case was reported by conventional MRI with Bilateral diffuse altered signal intensity of both deep white matter ;bright in T2WI and dark in T1WI and FLAIR, sparing deep grey matter i.e. both basal ganglia and thalami and lateral capsule suggesting leukodystrophy ,by DTI-FT showed complete anatomical disruption of all white matter tracts as described by axonal loss theory by Ishak[7] together with global decrease of FA in frontal ,parietal and occipital region.
A case was reported by conventional MRI as unilateral hemispheric parenchymal atrophy with ex-vacuo ventricular dilatation. Diffuse white matter T2 - hyper intense signal areas were seen (Rasmussen encephalitis), by DTI-FT there was defective callosal fibers posteriorly and there was significant decrease of FA on the left side coinciding with axonal loss theory of Ishak [7].

A case was reported in this study by the conventional MRI and complementary MRS showing Diffuse bilateral symmetrical cerebral and cerebellar areas of white matter abnormal signal eliciting bright T2WI, low in T1WI and FLAIR and involving both deep and subcortical U fibers, bilateral abnormal signal of deep grey matter relatively sparing the putamina with total involvement of the brain stem were also seen. MRS shows increased NAA peak, MI peak, small GLs and small lactate. "Canavan disease," by DTI-FT there was diffuse decrease of the FA of the frontal, parietal and occipital regions. As suggested by Ishak [7].

Table 5: cases summary

<table>
<thead>
<tr>
<th>Case N</th>
<th>Diagnosis (Conventional MRI)</th>
<th>Anatomical by DTI</th>
<th>Functional by DTI (FA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 1</td>
<td>Total Callosal agenesis</td>
<td>partial CC agenesis</td>
<td>Periventricular low FA</td>
</tr>
<tr>
<td>Case 2</td>
<td>Joubert dandy variant</td>
<td>attenuated and splayed SCP</td>
<td>No regional white matter or tracts abnormality</td>
</tr>
<tr>
<td>Case 3</td>
<td>polymicrogyra,cortical dysplasia (Zellweger syndrome)</td>
<td>thick cc and distal fiber crossing</td>
<td>Decreased FA in frontal and parietal regions</td>
</tr>
<tr>
<td>Case 4</td>
<td>pachygyria,cortical dysplasia</td>
<td>deviated tracts at the dysplastic grey matter</td>
<td>No regional white matter or tracts abnormality</td>
</tr>
<tr>
<td>Case 5</td>
<td>subependymal heterotopia( diffuse nodular subependymal grey matter like heterotopia)</td>
<td>No tracts abnormality ?</td>
<td>High FA in heterotopic grey matte</td>
</tr>
<tr>
<td>Case 6</td>
<td>Cavum valum interposial cyst (cyst displacing white matter)</td>
<td>No regional white matter or tracts abnormality</td>
<td></td>
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<td>---</td>
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<td></td>
</tr>
<tr>
<td>Case 7</td>
<td>Total Callosal agenesis, with bilateral probst bundles</td>
<td>No regional white matter or tracts abnormality</td>
<td></td>
</tr>
<tr>
<td>Case 8</td>
<td>Adrenoleukodystrophy, defective CC fibers centrally</td>
<td>Diffuse decrease in regional white matter FA</td>
<td></td>
</tr>
<tr>
<td>Case 9</td>
<td>Leukodystrophy, complete anatomical tract disruption, (single ROI)</td>
<td>Diffuse decrease in regional white matter FA</td>
<td></td>
</tr>
<tr>
<td>Case 10</td>
<td>Rasmussen encephalitis, defective CC fibers posteriorly</td>
<td>Unilateral decrease in the regional white matter FA</td>
<td></td>
</tr>
<tr>
<td>Case 11</td>
<td>Canavan, No tracts abnormality ?</td>
<td>Diffuse decrease in regional white matter FA ?</td>
<td></td>
</tr>
</tbody>
</table>

**Study Limitations:**

- Small Sample size.
- Motion artifacts in non-sedated patients.
- Evaluation of the peripheral grey matter with DTI is not of great value because of the low FA of the grey matter and a partial volume effect by cerebrospinal fluid in the sulcus[1]
- The technique is hampered by the lack of a normal standard of reference for age-sex matched white matter fractional anisotropic values.
Fig. 6: 5 months old male presented with metabolic acidosis and respiratory distress grade two. Conventional MRI sagittal, coronal T2WI showing hyper-intense signal in white matter frontal region, non visualized CC[arrow] (A, B),axial FLAIR showing parallel frontal horns of lateral ventricles(C).

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Fig. 7: DWI and 2D color map diffusion images superimposed upon axial FLAIR, showing anterior connection between both hemispheres at the region of the genu (red region). (D)
Fig. 8: 3DDTI reconstructed image, showing the probst bundles on both sides.
Fig. 9: 3DDTI reconstructed image showing the H shaped fibers connected at the region of the genu of corpus callosum

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**Fig. 10:** 40 days female presented with convulsions. Axial T2 FLAIR and sagittal T1W1 showing Hypoplastic cerebellar hemispheres and absence of cerebellar vermis with large retro-cerebellar cystic lesion communicating with the fourth ventricle, deformed midbrain with molar tooth appearance.

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**Fig. 11:** Sagittal, axial and coronal T13D sequence of normal male showing the normal thickness and orientation of the SCP

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**Fig. 12:** 3DDTI and reconstructed images showing attenuated thinned out SCP splayed around retro-cerebellar cyst

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**Fig. 13:** 15 month old male presented with nystagmus and convulsions and history of recurrent UTI and solitary kidney and there was history od died sibling. coronal T2WI,
sagittal T1WI and axial T2WI by conventional MRI showing polymicrogyria, periventricular vacuolated appearance in T2WI.

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**Fig. 14:** 3DDTI showing Heart shaped inter-connected corticospinal tracts (A) and thickened callosal fibers (B) (arrows)

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**Fig. 15:** One and half year old male patient suffering from generalized hypotonia and delayed milestones. Conventional MRI sagittal and coronal T2WI showing the corpus callosum and diffuse hyperintense signal. Axial FLAIR showing diffuse hypointense signal.

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![Fig. 15: MRI images showing hypotonia and delayed milestones](image)

**Fig. 16:** 3DDTTI showing centrally interrupted callosal fibers.

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![Fig. 16: 3DDTTI image showing interrupted fibers](image)
Conclusion

DTI have made it possible to reveal white matter anatomy and to detect neurological abnormalities in pediatric symptomatizing patients, not clearly evident by conventional MRI studies.

The current study clearly obviates the need and potential benefit of having quantitative comparisons between age-sex-matched healthy subjects of different age groups, to get more accurate FA references that could be the base line for more future DTI studies.

DTI is expected to become an important tool for the study of brain anatomy and the diagnosis of various white matter abnormalities, its prognosis and response to treatment.
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References


