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Blended Motor-Sensory Nerve Bundles on Diffused Tensor Imaging: Evidence of Brain Plasticity in a Patient with 36-year Sequelae from Encephalitis

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Abstract

BACKGROUND: Brain plasticity refers to the extraordinary ability of the brain to modify its structure and function after changes within the body or in the external environment. However, brain plasticity is not easily detectable using non-invasive imaging modalities.

CASE REPORT: In this article, we report the case of a 36-year-old male patient with sequelae of encephalitis. The patient had general epilepsy with multiple hospital admissions. Imaging performed using a 3.0 Tesla magnetic resonance imaging scanner showed that his cerebral hemispheres were asymmetrical, both morphologically and tractographically; we observed a scar at the right temporo-occipital region and atrophy of the right temporal lobe, hippocampus, and the pons. Diffusion tensor imaging (DTI) reconstruction showed asymmetrical corticospinal and thalamocortical tracts, with partial damage of the posterior thalamocortical tract by the scar. Blended motor-sensory nerve bundles were observed on the left side of the patient's brain exclusively and not on the right side or healthy subjects. DTI quantification showed a lower line number, lower fractional anisotropy, and higher apparent diffusion coefficient in the patient compared with healthy subjects and in the patient himself, with decreased functionality on the side of the scar.

CONCLUSION: Non-invasive DTI with 3D image reconstruction in this patient showed evidence of brain plasticity in the corticospinal and thalamocortical tracts, which can be used to inform diagnosis and plan treatment strategies.

Introduction

Diffused tensor imaging (DTI) is a magnetic resonance imaging (MRI)-based technique that was developed recently to visualize the nerve pathways between regions of the brain and between the brain and the spinal cord [1], [2], [3]. Conventionally, MRI is the best imaging method to monitor and evaluate brain lesions. Its high spatial resolution and good tissue contrast, especially for brain tissue, render it the first choice for assessing brain damage. DTI is used to reconstruct nerve pathways by recording the diffusion of water molecules in myelinated nerve tissues. When water is in a free environment (e.g., cerebrospinal fluid), the water molecules diffuse in random directions, the so-called isotropic diffusion [4], [5]. However, in tissues, especially in myelinated nerve tissues, water molecules diffuse only in certain directions, namely, the direction of nerve fibers or bundles, which is called anisotropic diffusion and allows the visualization of the conduction pathways in the central nervous system [6]. When the pathway is damaged because of encephalitis, cerebral infarction, cerebral hemorrhage, or brain tumor, the anisotropic diffusion is disrupted, which creates an interrupted image of the nerve bundle [7], [8], [9].

To quantify abnormalities, DTI includes three indicators that are used to evaluate the integrity of the conduction pathway, namely, the number of lines, the partial anisotropic index fractional anisotropy (FA), and the apparent diffusion coefficient (ADC) [10].

The partial FA, which varies from 0 to 1, is the most common index used to evaluate the integrity of the nerve conduction pathway on DTI: FA = 0 when the diffusion process is isotropic, that is, the water molecule diffuses randomly in different directions, such as the diffusion of water molecules in the cerebrospinal fluid; and FA = 1 when the diffusion is anisotropic, that is, the diffusion occurs only in one direction. FA increases when the density of the conduction pathway is thick, the axon is highly differentiated, the diameter of the axon is large, and the level of myelination in white matter is high. In turn, FA decreases when myelin or the axon is injured or interrupted [11].

ADC is an index that is used to measure the diffusion amplitude of water molecules in the nerve tissues. ADC is low when the pathway in the white matter is tightly organized (intact) and high when the pathway loses its normal structure (damaged) [11]. In the assessment of the integrity of the neural structure, the values of FA and ADC tend to be contradictory [12].

Purulent meningitis is an infection caused by *Neisseria meningitidis* that often damages the brain and meninges. Without timely diagnosis and treatment, patients may die or experience severe sequelae, including mild paralysis, paralysis, epilepsy, and many irreversible brain damages. These sequelae cause disabilities, thus affecting the living and working capacity of many patients [13], [14], [15]. In the 1970s and 1980s, purulent meningitis was an under detected but substantial burden among infants and young children in Vietnam [16], [17]. Brain damage after encephalitis causes functional defects in the areas in which it dominates. However, over time, the human brain makes structural and functional adjustments to adapt to and overcome some of these defects. This phenomenon is often referred to as "brain plasticity" [18], [19]. Brain plasticity refers to the extraordinary ability of the brain to modify its structure and function after changes occurring within the body or in the external environment. Brain plasticity takes place most strongly during childhood, with changes commonly observed during learning; however, that process continues throughout the life of the individual [20]. It can be observed at multiple scales, from microscopic changes in individual neurons to large-scale changes that can be observed using diagnostic imaging methods, such as functional MRI or DTI [20], [21]. Regarding patient care, research on brain plasticity provides a better definition of the patients' functional state, better individual prognosis, improvement of treatment strategies, and progress in the understanding of how the nervous system acts in response to disease [22]. At present, studies related to brain plasticity using DTI are scarce.

This case report aimed to provide evidence of brain plasticity manifested on the corticospinal and thalamocortical tracts using DTI.

Case Report

The patient was a 36-year-old man. His mother had a complicated labor and forceps were used to deliver him. Seven days post-discharge from the maternity ward of a general hospital, the patient suffered from diarrhea and dehydration, which were accompanied by persistent high fever; he was hospitalized again and treated at the pediatric department for purulent meningitis. After nearly 3 months of hospitalization, the patient was discharged with sequelae of epilepsy. The lesion was located on the right temporo-occipital lobe so that he had difficulty moving his left hand, and his fingers were always folded. The first epileptic seizure occurred when he was 5 years old, with the expressions of slight twitching, foaming from the mouth, and occurring over a short period of about 40 s. His family sent him for re-examination and, based on his previous medical record, he was treated with an antiepileptic drug, Gardenal. Because the seizures appeared more regularly, he left school at the age of 10, before finishing the 5th grade. He sometimes felt the seizures in advance, and he usually sat down or shouted for help. When he grew up, the seizures became more regular, with longer duration and more severe expressions, including distorted mouth, twitching limbs, tight grip, rolling eyes, screaming, foaming from the mouth, and, finally, exhaustion with shortness of breath. He experienced hand and leg fractures, chin chipping, and tearing of the skin of the scalp several times.

He received treatment at the neurology department twice, in September 2003 and in April 2015. Because the seizures became frequent, the medical team at the hospital kept prescribing antiepileptic drugs. Recently, he visited our hospital and was assessed using hematology, biochemistry, electroencephalography (EEG), X-ray, MRI, and DTI analyses.

During the whole treatment, the physician changed his prescription 14 times, but the disease did not subside. The most recent episode before this study occurred in early April 2015, during which the patient had seizures every 1–2 min. He could hardly control himself and exhibited delirium speaking, blurred vision, and inability to determine the direction. His treating physician started treatment on April 13, 2015, with a request for re-examination after 10 days, but the patient was admitted to the emergency room at the Internal Medicine Department 3 days later. After the treatment period, at the time of the study (2016), he was temporarily stable and still taking medication as an outpatient.

DTI

A Philips Achieva 3.0 Tesla scanner (Netherlands) with a SENSE NV 16-channel coil was used in this study to acquire MRI and DTI data. The scan range covered an area from the skull base to the top of the skull, and sequences of T1W, T2W, FLAIR, and diffusion-weighted imaging (DWI) were used. The imaging parameters were as follows: Acquisition matrix, 128 × 128; FOV, 230 × 230 mm^2; TR, 10172 ms, 93 ms; EPI factor, b 0 and b 1000 s/mm^2; and section thickness, 2 mm (acquired isotropic voxel sized, 1.8 × 1.8 × 2 mm^3).

Fiber tracking

DWI and DTI data were analyzed using the software installed in the Philips Extended MR

Workspace 2.6.3.1. A 2D color map of FA was constructed to seed region of interests (ROIs) according to known anatomy. Three sensory pathway's ROIs were placed in the commissura cerebelli, the thalamus, and the posterior part of the capsula interna. Three motor pathway's ROIs were placed in the precentral gyrus, the posterior limb of the internal capsule, and the pons [10].

The High Directional Resolution DTI software was employed to reconstruct 3D images of sensory thalamocortical and motor corticospinal tracts, which were used to analyze the morphology, number of lines, FA, and ADC.

Brain morphology and nerve bundles on MRI and DTI

In healthy subjects, the corticospinal and thalamocortical tracts of each hemisphere were adjacent to each other, but always separated; moreover, they were symmetrical on the right and left sides (Figure 1). The brain parenchyma was rather homogeneous.

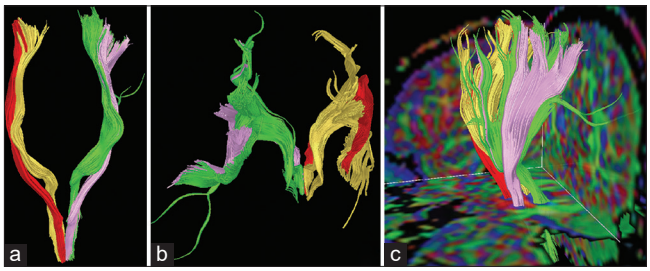

Figure 1: Diffusion tensor imaging images of the thalamocortical (yellow and green) and corticospinal (red and pink) tracts of both brain hemispheres of a healthy adult. (a) Coronal view; (b) axial view from above; and (c) sagittal view, with the tracts superimposed with the brain parenchyma. (a-c) The motor and sensory bundles are arranged symmetrically between the two hemispheres and are separate on each side. This phenomenon can be observed in all healthy subjects analyzed (Vu Duy Lam, personal communication)

Conversely, at the right temporo-occipital region, we detected a star-shaped neurological scar containing cerebrospinal fluid (hyperintensive on T2W images and hypointensive on T1W and FLAIR images) very close to the occipital horn of the right ventricle; the scar had a wrinkled border, a size of 1.8 × 2.4 × 2.0 cm, and was seemingly pulled by the surrounding brain parenchyma (Figure 2).

The brain symmetry in the patient was lost, with atrophy observed in the right temporal lobe, hippocampus, and pons (Figure 3). The corticospinal and thalamocortical tracts were asymmetrical on the two sides (left and right), especially the left motor corticospinal tract, which originated posteriorly in the parietal cortex. The posterior half of the right sensory bundle was ruptured by the scar in the brain parenchyma at the right temporo-occipital area, whereas the right

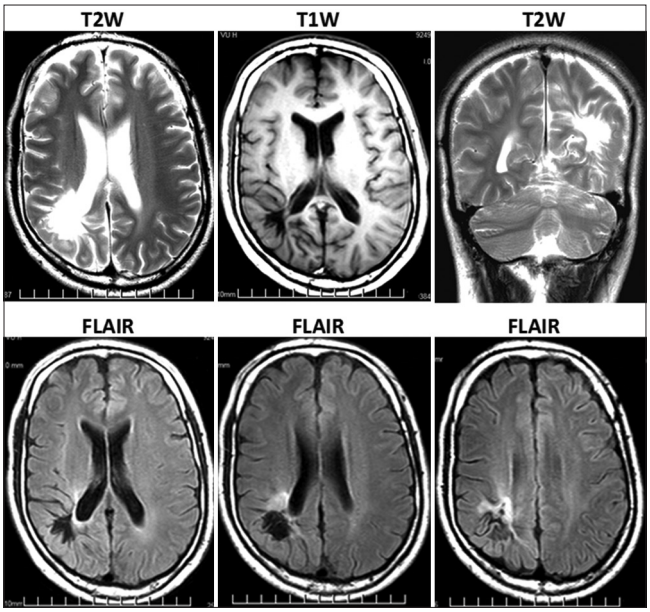

Figure 2: Conventional magnetic resonance images of a 36-year-old male patient. T2W, T1W, fluid-attenuated inversion recovery (FLAIR) axial, and T2W coronal images showing a star-shaped neurological scar in the right temporo-occipital region, which contained fluid (hyperintensive on T2W images, hypointensive on T1W and FLAIR images), had wrinkled edges, and exhibited a size of 1.8 × 2.4 × 2.0 cm; this scar was located very close to the occipital horn of the right ventricle

sensory bundle was much slimmer than that observed on the left side (Figures 4 and 5).

More specifically, in the left hemisphere of the patient, several strands of the left sensory tract became separated from the bundle and interspersed with lines of the left motor tracts, behind which it originated posteriorly in the parietal cortex (Figure 6). This was not observed on the right side.

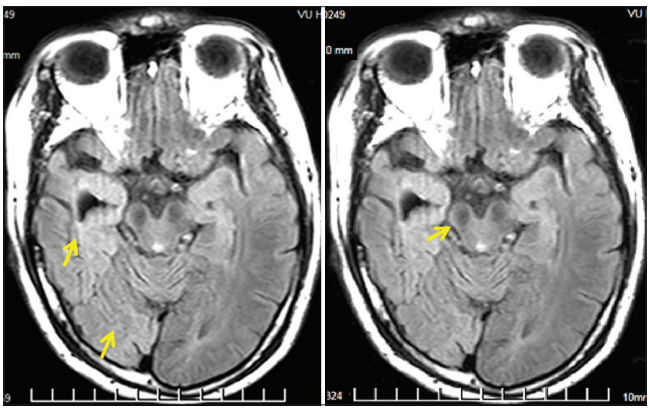

Figure 3: Two fluid-attenuated inversion recovery axial images of the patient's brain showing atrophy of the right temporal lobe, hippocampus, and pons (arrows)

Quantification of nerve lines, FA, and ADC

The number of lines, FA, and ADC on each side of the corticospinal and thalamocortical tracts of the patient are shown in Table 1.

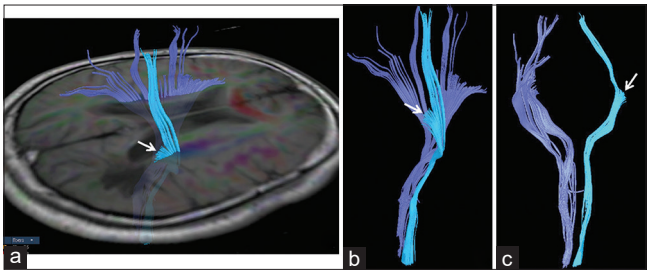

Figure 4: Diffusion tensor imaging of the thalamocortical tract of the patient. (a) Sagittal view, with the tracts superimposed with the brain parenchyma; (b) sagittal view without the brain parenchyma; and (c) coronal view. (a-c) The posterior half of the right sensory bundle was ruptured (arrows) by the scar in the brain tissue at the right temporo-occipital area, and the right sensory bundle was much slimmer than that on the left side

There was a significant decrease in the number of nerve lines (both corticospinal and thalamocortical) on the right (injured) compared with the left (intact) side. For the corticospinal tracts, the FA and ADC values of the right corticospinal tract were comparable to those of the left side. For the thalamocortical tracts, FA was significantly smaller and ADC was significantly higher on the right side.

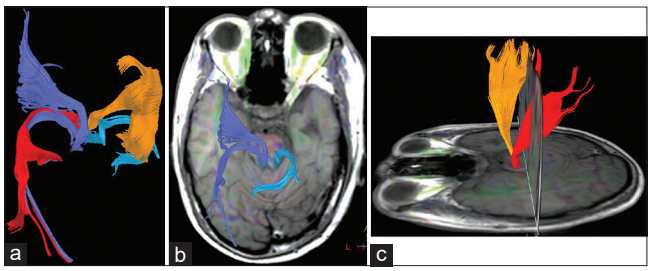

Figure 5: Diffusion tensor imaging images of the thalamocortical (blue and navy) and corticospinal (orange and red) tracts of both sides of the brain of the current patient. (a) Axial view from above; (b) thalamocortical tract on an axial view, superimposed with the brain parenchyma; and (c) corticospinal tracts on a sagittal view. (a-c) The corticospinal and thalamocortical tracts are asymmetrical on the two sides (left and right), especially the left motor corticospinal tract, which originated posteriorly in the parietal cortex

Discussion

Scarring

The location and function of the area of scarring explained the brain morphology and some of the clinical features of the patient. The lesion was located mainly in the subcortical white matter, near the posterior limb of the right internal capsule, where there were no neuronal bodies; they were mainly centripetal and centrifugal conduction tracts. The primary visual cortex (V1) in the nearby right occipital region was not damaged. Therefore, the clinical symptoms of the patient were reflected in sensory and corresponding motor functions, such as twitching of the left hand; however, there were no visual abnormalities. White matter damage occurring over a long period (36 years) had reduced the centripetal and centrifugal impulses, causing atrophy of the right temporal lobe, hippocampus, and pons.

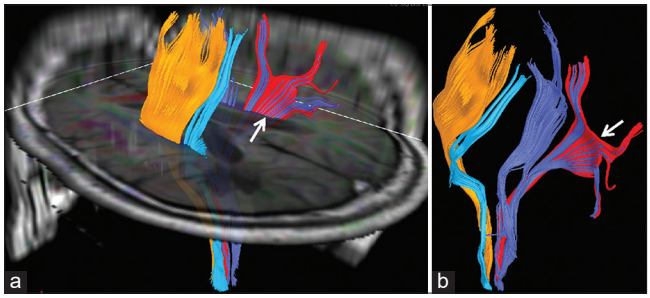

Figure 6: Diffusion tensor imaging images of the thalamocortical (blue and navy) and corticospinal (orange and red) tracts of both sides of the brain of the current patient on an oblique sagittal view. (a and b) The right motor (orange) and the sensory (blue) bundles are quite separate from each other, whereas the motor lines (red) on the left alternate between the sensory lines (navy) (arrows)

Table 1: Number of lines, fractional anisotropy, and apparent diffusion coefficient of the corticospinal and thalamocortical tracts

Parameters	Side	Corticospinal tract	Thalamocortical tract
Number of lines	Left	399	155
	Right	178	61
FA	Left	0.479 ± 0.181	0.458 ± 0.171
	Right	0.493 ± 0.202	0.428 ± 0.187
ADC	Left	0.934 ± 0.440	0.885 ± 0.401
	Right	0.896 ± 0.513	1.086 ± 0.615

ADC: Apparent diffusion coefficient, FA: Fractional anisotropy.

This atrophy of the right temporal lobe and hippocampus, associated with a parenchymal defect in the right temporo-occipital area, also explained the progression of the patient's general seizures. He had typical symptoms of temporal seizures, as follows: Before the seizures, the patient had warning signs, then suddenly lost consciousness and experienced convulsions of the whole body, eye rolling, distorted mouth with foaming, and uncontrollable urination, which lasted for about 2–3 min. At the end of the episode, the patient breathed fast, then lays still and gradually regained consciousness. After the seizure, the patient did not remember what had happened during the episode. Sometimes, he exhibited psychomotor disorders. On the patient's EEG, paroxysmal epilepsy waves were observed predominantly in the right temporal region [23].

On MRI, the scar was located at the right temporo-occipital parenchyma and had a size of 1.8 × 2.4 × 2.0 cm; it had persisted for 36 years, from the age of 5 months until the patient was 36 years old, when this study was conducted. We hypothesized that, during his childhood, as the patient's brain continued to grow and the brain and skull volume increased, the brain parenchyma was always pulled toward the nerve scar (fixed area), causing spontaneous and excessive discharge in the brain, thus increasing the frequency of the patient's seizures as he aged.

Blended motor-sensory nerve bundles

On DTI images of the patient's brain, the posterior half of the right sensory thalamocortical

bundle was ruptured by a neurotic scar. Specifically, some fibers of the left sensory thalamocortical bundle (the opposite side of the lesion) were separated from the large bundle and intermingled with the fibers of the motor bundle of the same side, at the back. This phenomenon was not observed on the injured side of the patient's brain (i.e., the right side) and was not detected in either of the two hemispheres in healthy subjects [24] (Lam, personal communication). In addition, the motor bundle on the left side originated posteriorly in the parietal cortex, causing a morphological imbalance of the two bundles between the two hemispheres [25].

On microscopic anatomy, the internal capsule consists of ascending sensory fibers and descending motor fibers. In the internal capsule of the two hemispheres, the motor and sensory bundles are located next to each other, but are separated from each other [26]. Originating from pyramid-shaped cells in the premotor cortex, primary motor cortex, and supplementary motor area in the precentral sulcus of the frontal lobe, the motor fibers travel down to the internal capsule in the corticospinal bundle and then continue downward and stop at the anterior horn of the opposite spinal cord. Although these motor and sensory bundles are adjacent to each other, their fibers should never intermix with each other [27].

To the best of our knowledge, this was the first observation of such blended nerve bundles on DTI; moreover, this change was thought to result from brain plasticity.

Nerve bundle conduction pathways

Table 2 compares the results of the quantification of nerve bundles, FA, and ADC of the patient with those of healthy subjects in studies conducted on the same platform at our hospital, including the corticospinal tract [24] and thalamocortical tract (Lam, personal communication).

Table 2: Number of lines, fractional anisotropy, and apparent diffusion coefficient of the patient and healthy subjects

Parameters	Side	Corticospinal tract		Thalamocortical tract	
		Patient	Healthy subjects	Patient	Healthy subjects
Number of lines	Left	399	496.5	155	315
	Right	178	498.9	61	400
FA	Left	0.479 ± 0.181	0.512 ± 0.071	0.458 ± 0.171	0.479 ± 0.025
	Right	0.493 ± 0.202	0.530 ± 0.090	0.428 ± 0.187	0.480 ± 0.024
ADC	Left	0.934 ± 0.440	0.832 ± 0.101	0.885 ± 0.401	0.829 ± 0.055
	Right	0.896 ± 0.513	0.840 ± 0.101	1.086 ± 0.615	0.817 ± 0.007

ADC: Apparent diffusion coefficient, FA: Fractional anisotropy.

There was an overall decrease in the number of lines and FA and an increase in ADC in the patient's brain compared with the respective tract and side of healthy subjects. The change was more significant on the right side for each tract, and more significant on the thalamocortical (sensory) tract for each side. This overall change demonstrated the reverse effect of the lesion on general brain functionality and conduction pathways in the patient. Changes in each parameter either in the patient himself, between brain sides and types of tracts, or in comparison with healthy subjects were linked to the characteristics of the nerve scar and can be explained by the adaptation of the brain over time, that is, brain plasticity.

A significant decrease in the number of lines was observed in the right corticospinal (motor) and thalamocortical (sensory) tracts corresponding to the injured side compared with that detected on the intact (left) side. The decrease was more significant for the sensory lines because the posterior half of the right sensory bundle was damaged by the scar (Figure 4).

The location of the scar explained the changes in FA and ADC observed between sides and tracts. As FA reflects the degree of myelination and ADC reflects the level of intact tissue, scarring or changes in tissues result in lower FA or higher ADC values. In the present case, the scar was located on the right occipital lobe; thus, its impact was more significant on the corresponding right thalamocortical tract. The FA value of the right and left corticospinal tracts was similar, whereas the FA value of the right thalamocortical tract was smaller than that of the left. Similarly, the ADC value of the right thalamocortical tract was the highest because of the loss of the normal structure of the nerve conduction pathway and the maximum amplitude of fluctuation of the water molecules in the fibers of the sensory bundle.

In normal humans, the FA index of the motor and sensory bundles between the two hemispheres is different because of the different degrees of myelination between the right and left sides. Moreover, this index also varies according to age, gender, and characteristics of the individual [28]. Therefore, we recommend using the comparison as a guide, but avoid statistical comparisons.

Clinical presentation

All of the morphological and tractographical changes mentioned above were responsible for the severe clinical symptoms observed in the patient. Injury in the brain, which occurred on the pathway of the conduction bundle and persisted for a long time, caused profound changes in many areas of the brain, making it difficult to explain all the associations between morphological changes and clinical symptoms.

To adapt to the aforementioned structural and functional defects, over time, the patient's brain made adjustment between the right and left sides, as manifested in the observed changes in the motor and sensory pathways: On the opposite side of the injury, the motor fibers interfered with the sensory fibers,

and the motor bundle strongly bent posteriorly. These manifestations are called brain plasticity and are not detected in healthy individuals.

Conventional brain MRI and DTI in this patient not only contributed to the diagnosis and quantification of the lesions but also helped neurosurgeons to plan treatment, primarily based on the images of the important nerve conduction pathways in the brain, such as the motor and sensory pathways. Surgeons use this type of information to determine the surgical entrance, remove the nerve scar and the pulling parenchyma, and leave a neat structure, without compromising further the important conduction pathways [8], [9]. This provided the potential for effective treatment of the temporal seizures in the patient.

This study was preliminary, as it was based on the actual observations made in one patient with brain parenchymal injury secondary to encephalitis. To identify other manifestations of brain plasticity on MRI, additional patients with other types of illness and long-term monitoring are required, to observe the changes in brain structure and function over time under the influence of changes occurring inside and outside the body.

Conclusion

We described the significant changes in morphology and tractography that occurred in the brain of a patient with 36-year sequelae from encephalitis. His brain damage secondary to encephalitis resulted in the development of a scar on the right temporo-occipital region, which altered the structure of the right and left conduction pathways. These changes are thought to be attributable to brain plasticity, which compensated for the sensory and motor deficiencies in the right hemisphere. DTI with 3D image reconstruction is a non-invasive imaging technique that allows the detection of brain plasticity in the corticospinal and thalamocortical tracts in the human brain and can be used to inform diagnosis and plan treatment strategies.

Ethical Approval

The research was approved by the Ethics Committee of Hospital 108, Hanoi, Vietnam (Number. 531/HDDD).

Informed Consent

Informed consent was obtained from the patient included in the study. The patient and his family agreed both to participate in the diagnosis and treatment with understanding and allow our research group to use their studying image, data, and information in writing, publishing scientific article.

References

1. Barbas H, Pandya DN. Architecture and frontal cortical connections of the premotor cortex (area 6) in the rhesus monkey. J Comp Neurol. 1987;256(2):211-28. https://doi.org/10.1002/cne.902560203
   PMid:3558879
2. Hasan KM, Walimuni IS, Abid H, Hahn KR. A review of diffusion tensor magnetic resonance imaging computational methods and software tools. Comput Biol Med. 2011;41(12):1062-72. https://doi.org/10.1016/j.compbiomed.2010.10.008
   PMid:21087766
3. Scannell JW, Burns GA, Hilgetag CC, O'Neil MA, Young MP. The connectional organization of the cortico-thalamic system of the cat. Cerebral Cortex. 1999;9(3):277-99. https://doi.org/10.1093/cercor/9.3.277
   PMid:10355908
4. Basser PJ, Mattiello J, LeBihan D. MR diffusion tensor spectroscopy and imaging. Biophys J. 1994;66(1):259-67. https://doi.org/10.1016/S0006-3495(94)80775-1
   PMid:8130344
5. Beaulieu C, Allen PS. Water diffusion in the giant axon of the squid: Implications for diffusion-weighted MRI of the nervous system. Magn Reson Med. 1994;32(5):579-83. https://doi.org/10.1002/mrm.1910320506
   PMid:7808259
6. O'Donnell LJ, Westin CF. An introduction to diffusion tensor image analysis. Neurosurg Clin North Am. 2011;22(2):185-96. https://doi.org/10.1016/j.nec.2010.12.004
   PMid:21435570
7. Ciccarelli O, Catani M, Johansen-Berg H, Clark C, Thompson A. Diffusion-based tractography in neurological disorders: Concepts, applications, and future developments. Lancet Neurol. 2008;7(8):715-27. https://doi.org/10.1016/S1474-4422(08)70163-7
   PMid:18635020
8. Jones DK. Studying connections in the living human brain with diffusion MRI. Cortex. 2008;44(8):936-52. https://doi.org/10.1016/j.cortex.2008.05.002
   PMid:18635164
9. Lazar M. Mapping brain anatomical connectivity using white matter tractography. NMR Biomed. 2010;23(7):821-35. https://doi.org/10.1002/nbm.1579
   PMid:20886567
10. Löbel U, Sedlacik J, Güllmar D, Kaiser WA, Reichenbach JR, Mentzel HJ. Diffusion tensor imaging: The normal evolution of ADC, RA, FA, and eigenvalues studied in multiple anatomical regions of the brain. Neuroradiology. 2009;51(4):253-63. https://doi.org/10.1007/s00234-008-0488-1

PMid:19132355

11. Beaulieu C, Allen PS. Determinants of anisotropic water diffusion in nerves. Magn Reson Med. 1994;31(4):394-400. https://doi.org/10.1002/mrm.1910310408
PMid:8208115
12. Provenzale JM, Isaacson J, Chen S, Stinnett S, Liu C. Correlation of apparent diffusion coefficient and fractional anisotropy values in the developing infant brain. Am J Roentgenol. 2010;195(6):W456-62. https://doi.org/10.2214/AJR.10.4886
PMid:21098179
13. Passingham RE, Stephan KE, Kötter R. The anatomical basis of functional localization in the cortex. Nat Rev Neurosci. 2002;3(8):606-16. https://doi.org/10.1038/nrn893
PMid:12154362
14. Mufson EJ, Brady DR, Kordower JH. Tracing neuronal connections in postmortem human hippocampal complex with the carbocyanine dye DiI. Neurobiol Aging. 1990;11(6):649-53. https://doi.org/10.1016/0197-4580(90)90031-t
PMid:1704107
15. Van Buren JM, Borke RC. Variations and Connections of the Human Thalamus. Electronic Resource. Berlin, Germany: Springer-Verlag; 1972.
16. Kim SA, Kim DW, Dong BQ, Kim JS, Anh DD, Kilgore PE. An expanded age range for meningococcal meningitis: Molecular diagnostic evidence from population-based surveillance in Asia. BMC Infect Dis. 2012;12:310. https://doi.org/10.1186/1471-2334-12-310
PMid:23164061
17. Oberti J, Hoi NT, Caravano R, Tan CM, Roux J. An epidemic of meningococcal infection in Vietnam (southern provinces). Bull World Health Organ. 1981;59(4):585-90.
PMid:6797748
18. Berlucchi G. The origin of the term plasticity in the neurosciences: Ernesto Lugaro and chemical synaptic transmission. J Hist Neurosci. 2002;11(3):305-9. https://doi.org/10.1076/jhin.11.3.305.10396
PMid:12481483
19. Mateos-Aparicio P, Rodríguez-Moreno A. The impact of studying brain plasticity. Front Cell Neurosci. 2019;13(66):66.
20. Mansvelder HD, Verhoog MB, Goriounova NA, editors. Synaptic Plasticity in Human Cortical Circuits: Cellular Mechanisms of Learning and Memory in the Human Brain? 2019. Great Britain, Amsterdam: Elsevier Science B.V; 2019.
21. Berlucchi G, Buchtel HA. Neuronal plasticity: Historical roots and evolution of meaning. Exp Brain Res. 2009;192(3):307-19. https://doi.org/10.1007/s00221-008-1611-6
PMid:19002678
22. Beisteiner R, Matt E. Brain plasticity in fMRI and DTI. In: Stippich C, editor. Clinical Functional MRI: Presurgical Functional Neuroimaging. Berlin, Heidelberg: Springer Berlin Heidelberg; 2015. p. 289-311.
23. Singh A, Trevick S. The epidemiology of global epilepsy. Neurol Clin. 2016;34(4):837-47. https://doi.org/10.1016/j.ncl.2016.06.015
PMid:27719996
24. Lam VD. Lesion Assessment of Corticospinal Tracts and Some Indices of Diffusion Tensor Imaging (DTI) in Association with Motor Function on Cerebral Infarction Patients, PhD Thesis. Hanoi: University of Calgary's Digital Repository; 2019.
25. Kreilkamp BA, Weber B, Richardson MP, Keller SS. Automated tractography in patients with temporal lobe epilepsy using TRActs constrained by underlying anatomy (TRACULA). Neuroimage. Clinical. 2017;14:67-76. https://doi.org/10.1016/j.nicl.2017.01.003
PMid:28138428
26. Mårtensson J, Lätt J, Åhs F, Fredrikson M, Söderlund H, Schiöth HB, *et al*. Diffusion tensor imaging and tractography of the white matter in normal aging: The rate-of-change differs between segments within tracts. Magn Reson Imaging. 2018;45:113-9. https://doi.org/10.1016/j.mri.2017.03.007
PMid:28359912
27. Kamali A, Kramer LA, Butler IJ, Hasan KM, Kamali A, Kramer LA. Diffusion tensor tractography of the somatosensory system in the human brainstem: Initial findings using high isotropic spatial resolution at 3.0 T. Eur Radiol. 2009;19(6):1480-8. https://doi.org/10.1007/s00330-009-1305-x
PMid:19189108
28. Inano S, Takao H, Hayashi N, Abe O, Ohtomo K. Effects of age and gender on white matter integrity. AJNR Am J Neuroradiol. 2011;32(11):2103-9. https://doi.org/10.3174/ajnr.A2785
PMid:21998104