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Case Report

Diagnostic determination by diffusion tensor imaging of neural axon injury between the 2 hemispheres following traumatic brain injury ☆

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ABSTRACT

Traumatic brain injury (TBI) is caused by an external mechanical force to the head resulting in alteration of brain function. However, the injury to neural tracts and the connections between them is difficult to diagnose using traditional imaging techniques. A 54-year-old woman visited our clinic because of insufficient coordination of her body. Her personal history included severe TBI with a 10-day coma medically treated 10 years previously. She presented with memory impairment and insufficient coordination of her body, suggesting post-concussion syndrome. Her Glasgow Coma Scale score was 15 and the strength testing result was 5/5 for both sides; however, she could not walk. She had been examined at many medical centers, but without a diagnosis of her condition. She was scanned using morphometric magnetic resonance imaging (MRI), which detected a significant reduction in the corpus callosum. MRI-diffusion tensor imaging (DTI) revealed decreased fractional anisotropy (FA) in the white matter of the right temporal lobe and the corpus callosum. FA reflects the degree of anisotropy of water molecules. The decrease in FA in the corpus callosum indicated loss of connection between the 2 hemispheres. MRI tractography was used to describe the number of neural tracts in the corpus callosum. MRI-DTI and MRI tractography served as powerful diagnostic tools, providing imaging results that offered an explanation for our patient's clinical picture.

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Introduction

Traumatic brain injury (TBI) is an acquired injury to the brain from a sudden impact to the head or a penetrating injury that alters normal brain function. According to the world system of general statistics, traffic accidents are the most common cause of TBI [1]. A patient with mild TBI may or may not lose consciousness, but they can show neurological symptoms, such as headache and memory loss, which may last for weeks or even months. A patient with severe TBI usually falls into a coma and can show neurological sequelae after treatment. White matter integrity is critical to brain function. These events may result in long-term structural and functional changes in the brain, causing cerebral atrophy, the steady loss of neurons and the connections between them, and neurodegeneration, the steady functional decline of neurons [2].

Computed tomography (CT) and magnetic resonance imaging (MRI) are routinely employed to detect injuries to the brain parenchyma. MRI is especially advantageous for finding brain microinjuries. However, both normal CT and MRI techniques show low sensitivity in evaluating axonal injury or measuring the patterns of brain volume, leading to poor correlation between diagnosis and final outcome. Diffusion tensor imaging (DTI) is a recently developed imaging modality that shows high sensitivity in detecting axonal disruptions evident as local changes in the diffusion pattern of water molecules within and around axon bundles. The degree of diffusion anisotropy, known as fractional anisotropy (FA), is a sensitive parameter for characterizing local diffusion. Changes in FA reflect changes in the principal direction of the diffusion tensor and can be used to evaluate the condition of white matter connectivity in the brain [3]. TBI leads to a decrease in FA, resulting mainly from a decline in diffusivity along the principal direction. Thus, FA changes measured by DTI are a marker of injury. These signals from DTI are compared to a small group and then normalized tractography is used to quantify disruption along brain pathways. In addition, to evaluate the level of post-TBI cerebral atrophy, image post-processing is applied to measure the volume of brain areas via morphometry. Therefore, the correlation between the level of cerebral atrophy and the loss of neurons can be clearly determined.

Case presentation

A 54-year-old woman visited our clinic at Can Tho Stroke International Services General Hospital, Vietnam because of insufficient coordination of her body. Her personal history included severe TBI with coma for 10 days, which was medically treated about 10 years previously. Some negative outcomes of TBI, including memory impairment and insufficient coordination of the body, suggested post-concussion syndrome. Clinical examination involved application of the Glasgow Coma Scale (GCS), which obtained a score of 15, and strength testing resulting in a score of 5/5 for both sides, although the pa-

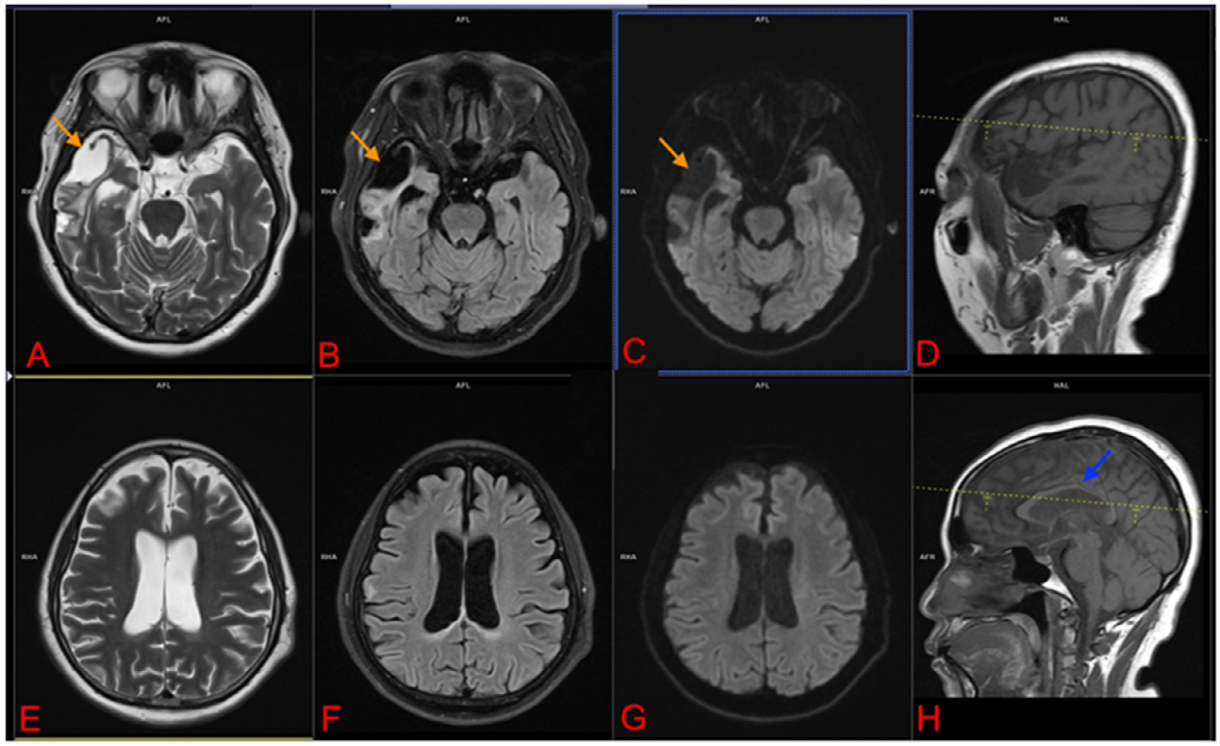

Fig. 1 – (A-D) show the old brain lesion in the right temporal lobe, hyperintensity on T2W, and hypointensity on T1W and FLAIR–DWI (orange arrow). (E-G) show normal cerebral signal intensity on MRI. (H) is the sagittal T1W image of the corpus callosum showing a reduction in its size (blue arrow).

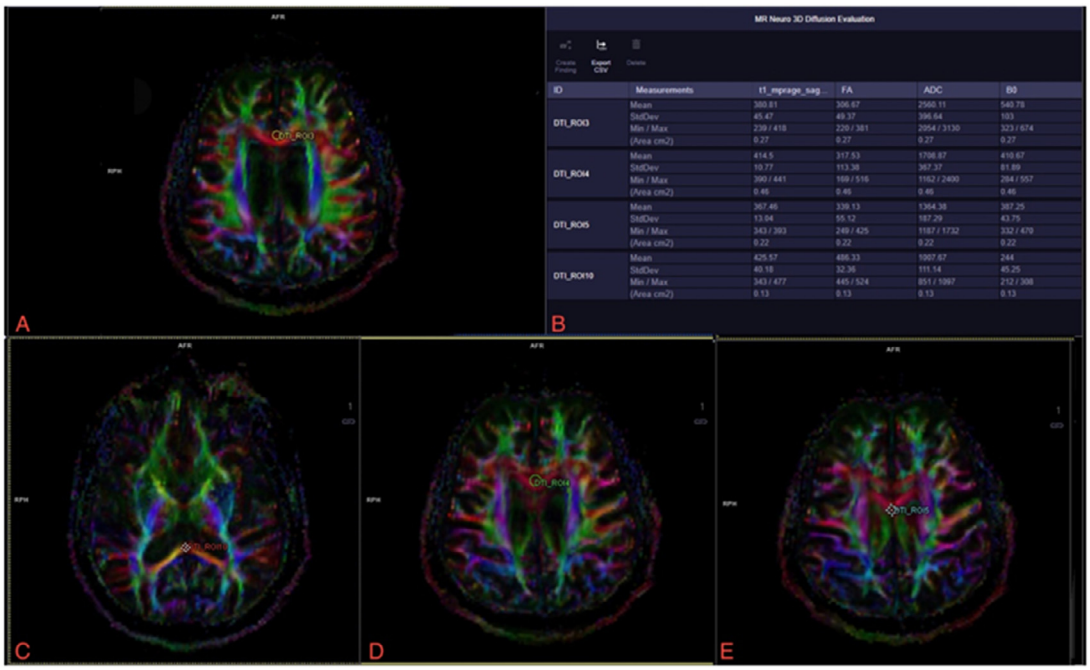

Fig. 2 – Fractional anisotropy (FA) map of the corpus callosum. (A, C, D, and E) show FA measurements of parts of the corpus callosum. (B) shows the results of correlation between the positions of the regions of interest (ROIs).

tient could not walk or coordinate her movements. The patient's musculoskeletal condition was normal and the results of her blood test and biochemical tests were also normal. A brain MRI was indicated for the patient. She was scanned using non-contrast MRI (Siemens 3.0 Spectra system), and the brain parenchyma, the vascular component, was visualized with Syngovia software version 4.1. The results of basic MRI pulse sequences showed an abnormality in the right temporal lobe, with hypointensity on T1-weighted and fluid-attenuated inversion recovery-diffusion-weighted imaging (FLAIR-DWI) and hyperintensity on T2-weighted, suggesting a chronic cerebral lesion. Sagittal T1-weighted imaging of the corpus callosum showed a remarkable decrease in its size (Fig. 1). The volume rendering technique image reconstructed by ToF-3D pulse showed no abnormality in the internal and external brain artery.

Because of the image obtained from sagittal T1W, the patient was then scanned using morphometric MRI. The results showed a significantly reduced midsagittal area of the corpus callosum, which was only 2.7 cm^2 in size. The patient was then scanned again using the DTI technique, and neuron tracts were reconstructed with a database of DTI tracts. Images of axon bundles were obtained in axial, sagittal, and coronal views of the corpus callosum. FA was calculated at several regions of interest in the brain using Syngovia 4.1, and other parameters were also measured at the same time. The results indicated a decrease in brain parenchyma, especially in the temporal lobe and corpus callosum. The FA value of the white matter of the right temporal lobe, determined by MRI-DTI, was 0.142 and those of the genu, body, and splenium of the corpus callosum were 0.306, 0.339, and 0.486, respectively (Fig. 2). The decline in FA in the corpus callosum suggested a diagnostic determination of loss of the connection between the 2 hemispheres. In addition, images of neuron bundles reconstructed by the tractography technique in Syngovia showed a lack of neurons in the corpus callosum, especially in the body (Fig. 3). There were clear imaging features of the correlation between neurons and parts of the corpus callosum on the sagittal view.

Discussion

Traumatic brain injury is a significant health problem and a financial burden on the healthcare system as well as the economy because of diminished worker productivity. The general public's awareness of TBI is limited. TBI can range from mild to severe and may result in structural and functional changes in the brain associated with cerebral atrophy, the gradual loss of neurons and the connections between them, and neurodegeneration, the gradual functional decline of neurons [2].

Cerebral atrophy as a response to TBI causes a decline in brain function, neurons, and the connections between them. The understanding of region-specific brain connections can help to make an accurate diagnosis and determine the treatment. Interestingly, the duration of loss of consciousness is correlated with specific atrophy in the corpus callosum; 3.863 cm^3 of the corpus callosum is lost per day of coma in patients with severe TBI [3].

Traditional imaging techniques such as CT and standard MRI can easily detect injuries to the brain parenchyma, including hemorrhage, edema, and contusions, but their sensitivity

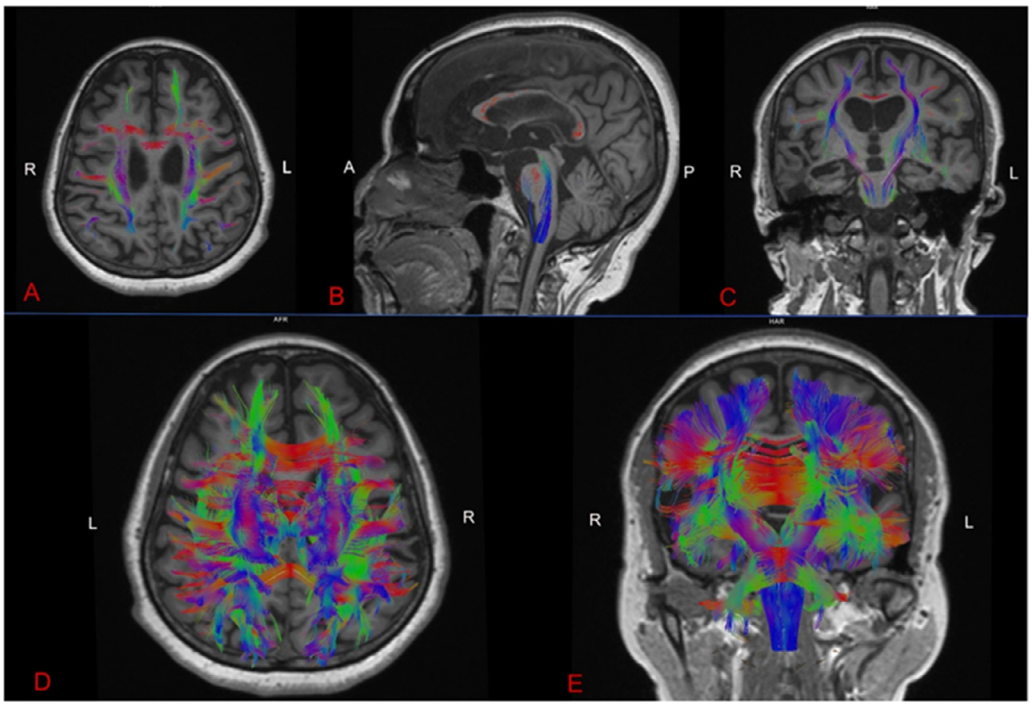

Fig. 3 – Right (R) and left (L) neuron tracts visualized by tractography. (A-C) show the axial, sagittal, and coronal views, respectively, of the neuron tracts in the corpus callosum. (D and E) are 3D images of axon bundles showing the lack of neuron fibers in the corpus callosum.

is low for evaluating axonal injury or measuring brain volume patterns. DTI is a recently developed imaging modality with high sensitivity in detecting axonal disruptions in terms of local changes in the diffusion pattern of water molecules within and around axon bundles. Changes in FA due to injury are indicative of changes in white matter connectivity [4].

Our patient had severe TBI and was in a coma for 10 days. At present, her GCS score is 15, indicating mild concussion, and her muscle strength is 5/5 for both sides; she has minor memory problems. However, our patient cannot walk and she has difficulty in maintaining coordination of her body. These clinical features indicate a lesion involving the brain structure connecting the 2 hemispheres. The result of MRI using basic pulse sequences showed an old cerebral lesion in the right temporal lobe; but it was not sufficient to determine a diagnosis.

The sagittal area of the corpus callosum, obtained by morphometric MRI, measured 2.7 cm^2. According to Laissy et al., the average sagittal area of the corpus callosum of adults is 6.36 cm^2, which indicates a notable decrease in our patient's corpus callosum [5]. Hemanth Kumar et al. found that the average FA of the corpus callosum is 0.63 ± 0.02. MRI-DTI showed a decrease in FA in the white matter of the right temporal lobe (0.142) and a sharp decline in FA in the corpus callosum (0.348, 0.346, and 0.156 in the genu, body, and splenium, respectively) of our patient. This result indicates a notable loss of neurons in the corpus callosum, which affects the connection between the 2 hemispheres [6]. The tractography images showed a remarkable lack of neuron tracts, especially in the body of the corpus callosum. The clinical picture of our patient is explained by this discovery.

Conclusion

In conclusion, TBI is a significant health problem that causes neurological sequelae in the future, especially cerebral atrophy, the steady functional decline due to the loss of neurons and the connections between them, which is extremely difficult to diagnose using traditional imaging techniques. Advanced MRI techniques, such as tractography and DTI, are critical to the diagnosis of types of injury to the brain. Tractography and DTI are modern, highly sensitive techniques for detecting axonal disruptions that are manifested as local changes in the diffusion pattern of water molecules within and around axon bundles. They were effective diagnostic tools, and the imaging data they produced gave an explanation for the clinical picture of our patient.

Authors' contribution

Nguyen-Duong Quoc Anh and Tran Chi Cuong contributed equally to this article as first authorship. Le Minh Thang and Nguyen Minh Duc contributed to write original draft. Nguyen-Duong Quoc Anh and Tran Chi Cuong contributed to undergo diagnostic procedure, collect and interpret the imaging. Le Minh Thang and Nguyen Minh Duc made substantial contributions to collect patient data and clinical data analysis. All authors have read, revised, and approved the final published version of the manuscript. All authors were responsible for submission of our study for publication.

Statement of ethics

Ethical approval was not necessary for the preparation of this article.

Data availability statement

All data generated or analyzed during this study are included in this article and/or its online supplementary material files. Further enquiries can be directed to the corresponding author.

Patient consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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