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Research Article

Features of changes in fractional anisotropy of different brain parts during the progression of Parkinson's disease

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ABSTRACT

BACKGROUND: Parkinson's disease is a neurodegenerative disease, in second place in terms of incidence in the world after Alzheimer's disease. It is currently believed that the presymptomatic stages of Parkinson's disease are mainly associated with degeneration of the subcortical and vegetetive nervous systems, and lesions of the cerebral cortex appear on later stages of the disease, however, it is of interest to study in more detail the involvement of the pathways of the brain in the pathological process in depending the disease progression.

OBJECTIVE: to study features of damage to the brain pathways during the progression of Parkinson's disease by magnetic resonance tractography.

MATERIAL AND METHODS: 88 patients with Parkinson's disease were examined (stage II disease — 42 people, stage III — 46 people according to the Hoehn and Yahr scale). The control group consisted of 35 people who did not differ in gender. All patients included in the study underwent a neurological examination, as well as magnetic resonance imaging of the brain with diffusion tensor imaging.

RESULTS: We found that with increasing stage of Parkinson's disease, there was a significant increase in fractional anisotropy in the hippocampus, insular cortex, and inferior and superior temporal sulcus cortex in patients with Parkinson's disease; we also noted a significant decrease in putamen fractional anisotropy.

CONCLUSION: the tractography study of the brain pathways during disease progression is a promising method that allows us to clarify in the pathogenesis of Parkinson's disease, including the role of extra-nigral pathology in the development of some non-motor disorders.

Keywords: cerebral cortex; cerebral white matter; fractional anisotropy; magnetic resonance imaging; Parkinson's disease; progression; tractography.

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Научная статья

Особенности изменения фракционной анизотропии различных отделов головного мозга при прогрессировании болезни Паркинсона

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АННОТАЦИЯ

Актуальность. Болезнь Паркинсона — нейродегенеративное заболевание, по частоте встречаемости находящееся на втором месте в мире после болезни Альцгеймера. В настоящее время считается, что предсимптомные стадии болезни Паркинсона в основном связаны с дегенерацией подкорковой и вегетативной нервной систем, а поражения коры головного мозга появляются на более поздних стадиях заболевания. Однако представляет интерес более детальное изучение вовлечения проводящих путей головного мозга в патологический процесс при прогрессировании болезни.

Цель исследования. Изучить особенности изменения фракционной анизотропии различных отделов головного мозга при прогрессировании болезни Паркинсона по данным магнитно-резонансной трактографии.

Материалы и методы. Было обследовано 88 пациентов с болезнью Паркинсона (II стадия заболевания — 42 человека, III стадия — 46 человек по шкале Хен и Яра). Группу сравнения составили 35 человек, не отличавшихся по полу и возрасту. Всем включенным в исследование пациентам проводились неврологический осмотр и магнитно-резонансная томография головного мозга с выполнением диффузно-тензорной визуализации. Данные трактографии проецировались на стандартную маску головного мозга.

Результаты. С увеличением стадии болезни Паркинсона нами было обнаружено у пациентов достоверное увеличение фракционной анизотропии гиппокампа, коры островковых извилин, а также было отмечено достоверное уменьшение фракционной анизотропии скорлупы.

Заключение. Трактография является перспективным исследованием, позволяющим уточнить патогенез болезни Паркинсона, в том числе роль экстранигральной патологии для развития ряда немоторых нарушений.

Ключевые слова: белое вещество головного мозга; болезнь Паркинсона; кора головного мозга; магнитно-резонансная томография; прогрессирование; трактография; фракционная анизотропия.

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ЭКО•ВЕКТОР

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BACKGROUND

Parkinson's disease (PD) is a neurodegenerative disorder with the second-highest incidence worldwide, after Alzheimer's disease. The clinical manifestations of PD mostly result from the demise of dopaminergic neurons within the substantia nigra (SN). However, as this disease advances, neurodegenerative transformations occur in nearly all brain structures [1].

Several studies on PD, alongside morphometric indices, have evaluated the brain's structural connections using diffusion-tensor magnetic resonance imaging (DT MRI). This technique enables the assessment of microstructural brain disorders *in vivo* and relies on measuring the magnitude and directionality of water molecule diffusion within each voxel of the image. Water diffusion occurs along a preserved axon fiber because it has an insulating myelin sheath, indicating its direction and integrity. This is the basis for acquiring DT images. DT MRI visualizes and quantitatively evaluates the condition of brain conductive pathways [2].

The quantity of DT MRI studies in patients with PD is quite less. Nonetheless, available data demonstrate that this approach can enhance the quantity and value of the diagnostic information obtained.

A study [3] discovered that cognitive disorders in patients with PD are linked to specific characteristics of brain tractography. A reduction in working memory capacity was consistently associated with reductions in fractional anisotropy (FA) of the brain's conductive pathways in the left temporal region, head and tail of the right hippocampus, head of the left hippocampus, knee of the corpus callosum, and left orbitofrontal region. The "disruption of ascending fibers within the corpus callosum" may serve as a neuroimaging biomarker for dementia progression in PD.

Langley et al. used DT MRI data from 20 patients with stage III PD assessed according to the Hoehn and Yahr scale and from 17 healthy volunteers [4]. The authors identified changes in regional FA within the SN. A substantial decline in the FA of the SN was observed in patients with PD compared with that in control participants, specifically in rostral areas more than in caudal sections. Abnormalities were observed in the SN of the hemisphere opposite to the limbs, exhibiting the most significant neurological symptoms.

The role of extranigral pathology in causing nonmotor disorders and the link between cognitive disorders and psychotic symptoms were observed by the staff of the Department of Nervous Diseases of the Academy [5, 6]. However, the precise progression of the conductive pathway changes from PD stages II to III remains inadequately understood.

The discrepancy in the obtained results can be attributed to the limited sample of patients studied and variances in the methodologies employed. In most studies, structural interrelationship disorders were found in patients with PD, mainly in the SN and striatopallidal complex and in some cases in the cerebellum. In addition, patients with cognitive impairments exhibited a decrease in FA within the left temporal region, hippocampus, and corpus callosum. These observations hint at the possible nature of disease progression and potentially introduce new therapeutic approaches.

If the confirmed data from subsequent studies demonstrate that the measurement of FA of the listed tracts can be useful for the differential diagnosis of various parkinsonism syndromes, the data can help in prescribing appropriate therapy at an earlier stage.

Currently, cortical lesions manifest in the later stages of the disease. However, there remains interest in studying the precise involvement of brain structures in the pathological process during disease progression [7].

This study aimed to examine the distinctness of lesions in cerebral conductive pathways during the progression of PD using MR tractography data.

MATERIALS AND METHODS

Eighty-eight patients with PD were assessed based on the diagnostic criteria set by the British Brain Bank [8]. These patients were divided into 2 groups according to the disease stage and criteria stipulated by the Hoehn and Yahr scale: 42 patients with stage II and 46 with stage III. All study participants were receiving therapy with dopaminergic drugs at the time of inclusion.

The control group comprised 35 individuals of comparable age who exhibited initial symptoms of cerebral circulation insufficiency.

The patients were subjected to neurological examinations that included evaluations of their motor functions. The degree of motor disorders, such as hypomimia, gait disorders, posture, trunk bradykinesia, rigidity, tremor, and postural instability, was measured according to Section III (motor functions) of the Unified PD Rating Scale (Fahn S, Elton S, et al.), a dedicated tool for diagnosing such disorders. The clinical stage of the disease was evaluated using the Hoehn and Yahr scale. All patients and controls underwent DN MRI.

A 3.0-T brain MRI was conducted using an 8-channel head coil on a Philips Achieva machine. The obtained results were projected onto a standard brain mask.

FA was measured on a scale from 0 to 1, where 0 indicates unrestricted diffusion and 1 represents the complete directional movement of all water molecules along the conductor's axis. The functionality of a white matter conductor is contingent on its FA level, which is directly related to its integrity.

Statistical analysis was performed on 390 gray and white matter structures. Data were processed using Statistica 10.0 from StatSoft.

Data with normal distribution were described using the mean (*M*) and standard deviation (*SD*). Reliable differences were determined by a significance level of $p < 0.05$.

RESULTS

Statistically significant differences were discovered in the FA of several brain regions in patients with PD and controls. Specifically, the left hippocampus, short gyrus islet cortex, inferior temporal sulcus cortex, putamen, right superior temporal tract, left middle temporal tract, left and right red nucleus (RN), and subthalamic nucleus (STN) on the left showed significant variation (Table 1).

As the PD stage increased, a significant increase in FA was found in the left hippocampus, insular gyrus cortex, superior and inferior temporal sulci, right and left RN, and left STN. Moreover, the FA of the putamen on the left side consistently decreased.

Patients with PD showed a significant decrease in the FA of the rubrospinal tract, medial longitudinal fasciculus, and inferior cerebellar peduncle on the right side (Figures 1–3).

DISCUSSION

The use of DT MRI in PD studies is limited; however, evidence indicates that the progressive decline in the function of various tracts can disrupt the integrity of the conductive pathways between cortical and subcortical regions associated with specific cognitive functions [9].

The study identified significant increases in the FA in certain gray matter structures of the brain in patients with PD. Specifically, the left hippocampus, insular gyrus cortex, and inferior and superior temporal sulcus cortices showed notable changes.

According to Chen et al., FA values were reported to be higher in the hippocampus and temporal gyrus regions of the cerebral cortex in patients with PD than in healthy controls [10]. The hippocampus and temporal cortex are responsible for cognitive functions, which suggests that the increase in FA may be attributed to compensatory functions.

Herz et al. conducted a meta-analysis of functional neuroimaging studies on motor tasks and reported consistently reduced FA in the posterior putamen of patients with PD while performing motor tasks [11]. In this study, we replicated these findings by observing a nearly twofold decrease in FA in the left hemisphere of patients with stage III PD according to the Hoehn and Yahr scale.

Table 1. Fractional anisotropy indices of the brain regions investigated in patients with Parkinson's disease that showed significant differences

Structure	Comparison group, *M* ± *SD*	Stage II on the Hoehn and Yahr scale, *M* ± *SD*	Stage II on the Hoehn and Yahr scale, *M* ± *SD*	*p*-value
Left hippocampus	–	0.194 ± 0.004	0.216 ± 0.007	0.024
Insular cortex gyri	–	0.179 ± 0.004	0.197 ± 0.004	0.014
Inferior temporal cortex	0.226 ± 0.127	0.236 ± 0.007	0.268 ± 0.005	<0.05
Superior temporal cortex	0.248 ± 0.006	0.249 ± 0.006	0.276 ± 0.005	<0.05
Left putamen	–	0.340 ± 0.004	0.196 ± 0.007	0.046
Left RN	–	0.415 ± 0.017	0.489 ± 0.022	0.023
Right RN	–	0.474 ± 0.01	0.532 ± 0.02	0.026
Left STN	0.358 ± 0.046	0.531 ± 0.03	0.512 ± 0.036	<0.05
Right rubrospinal tract	0.431 ± 0.422	0.402 ± 0.38	–	0.009
Right medial longitudinal fascicle	0.46 ± 0.44	0.421 ± 0.4	–	0.037
Right inferior cerebellar peduncle	0.4 ± 0.39	0.37 ± 0.35	–	0.047

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In addition, patients with PD exhibited an increase in FA within both the left and right RN and STN regions.

The RN is an objective subcortical center that plays a pivotal role in movement coordination. Its bilateral connection with the cerebellum and cortex allows it to proficiently participate in motor control. Presumably, increased FA amplifies RN activity to compensate for the dysfunctional thalamostriatocortical circuit.

STN plays a crucial role within the basal ganglia–thalamocortical circuit. Studies have suggested that STN overactivity may be responsible for the symptoms of PD [12]. Moreover, Wang et al. reported that problems concerning basal ganglia and thalamocortical connections may result in bradykinesia and rigidity [12].

Patients with newly diagnosed and moderately severe PD exhibit heightened functional connectivity between the STN and sensorimotor cortex, which implies that these changes occur in early disease stages [13]. This study revealed that the FA of the left hemisphere of the STN increases with disease progression.

In addition to the gray matter, modifications affect the neuronal pathways of the brain. Patients diagnosed with stage II PD showed FA reduction in the rubrospinal tract, medial longitudinal bundle, and inferior cerebellar peduncle on the right.

The Mormina et al. study analyzed the FA of the conductive pathways of patients with PD at the base of the peduncles and cerebellar hemispheres, comparing results from those with the disease for more than 5 years to healthy volunteer controls [14]. A decrease in FA was found in the cerebellar hemispheres of patients with PD, likely due to the inclusion of patients with longer disease duration.

Another study discovered an association between higher PD stages and longer disease duration with cerebellar white matter changes and decreased FA [15].

The medial longitudinal fasciculus belongs to the global coordinator and cognitive system [16]. The malfunction of this pathway causes coordinator and postural disorders in PD and cognitive decline.

Furthermore, patients with PD exhibited a decrease in FA within the rubrospinal tract. The rubrospinal tract primarily transmits information related to the nature of purposeful movements and the occurrence of tremors that are symptomatic of PD.

CONCLUSIONS

The data indicate a reduction in the FA in the brain's conductive pathways and subcortical formations, implying their dysfunction. By contrast, FA increases in different cortical regions. The latest established finding is a result of the cortical compensatory response to lesions of the conductors and basal ganglia.

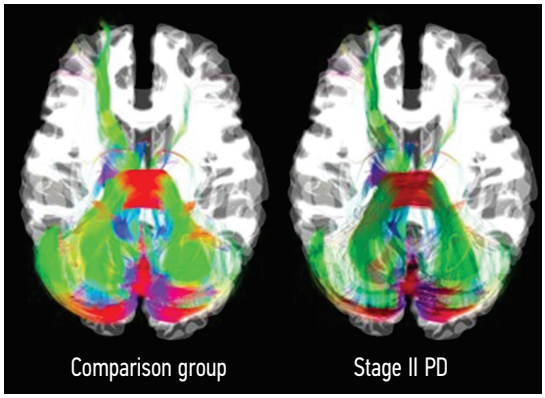

Fig. 1. Visual comparison of the right inferior cerebellar peduncle between the controls and patients with stage II Parkinson's disease (PD) using the Hoehn and Yahr scale

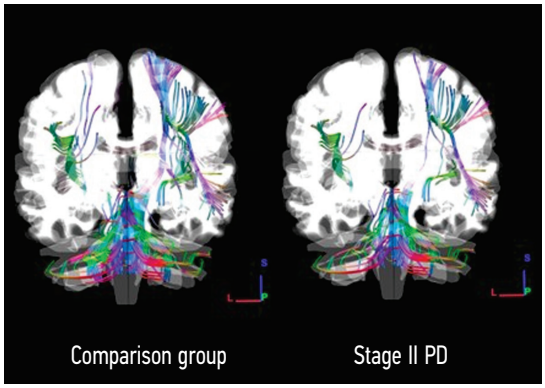

Fig. 2. Visual comparison of the right rubrospinal tract between the controls and patients with stage II Parkinson's disease (PD) using the Hoehn and Yahr scale

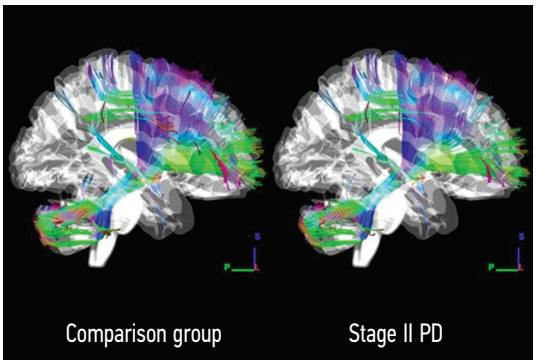

Fig. 3. Visual comparison of the right medial longitudinal fascicle between the controls and patients with stage II Parkinson's disease (PD) using the Hoehn and Yahr scale

Thus, MR tractography augments the amount and caliber of diagnostic information acquired from patients with PD and serves as a method of evaluating disease progression. Moreover, this approach enhances and broadens the understanding of PD causes.

ADDITIONAL INFORMATION

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Conflict of interest. The authors declare no obvious and potential conflicts of interest related to the publication of this article.

Ethical review. The study was approved by the local ethical committee of the Kirov Military Medical Academy of the Ministry of Defense of the Russian Federation (Minutes No. 243 of 10/20/2018).

Author contributions. All authors made a significant contribution to the study and preparation of the article and read and approved the final version before publication.

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