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Structural alteration of motor and sensory cortices in Parkinson's disease using magnetic resonance imaging: Automatic brain segmentation

Sahar Shareef [1], Tahir Ali [2], Bunyamin Sahin [3], Amani Elfaki [4], Raeesa Abdel Tawab Mohammad [1], Aly Mohamed Ahmed [1], Amal AlRabiah [1], Tahani Ahmad Al-Matrafi [1], Samar Al-Saggaf [5], Abdul-Aziz Haji Ahmed [1], Muhammad Atteya [1, 6, *]

[1]*Department of Anatomy, College of Medicine, King Saud University, Riyadh, Saudi Arabia*
[2]*Department of Anatomy, Faculty of Medicine, National Ribat University, Khartoum, Sudan*
[3]*Department of Anatomy, Faculty of Medicine, Ondokuz Mayis University, Samsun, Turkey*
[4]*Department of Anatomy, Faculty of Medicine, National University, Khartoum, Sudan*
[5]*Department of Anatomy, Faculty of Medicine, King Abdul Aziz University, Jeddah, Saudi Arabia*
[6]*Department of Histology, Faculty of Medicine, Cairo University, Cairo, Egypt*

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ABSTRACT

Parkinson's disease (PD) is a neurodegenerative disorder commonly diagnosed as motor triad of symptoms including tremor, rigidity and bradykinesia. The degeneration of the Substantia nigra in PD, leads to alterations in the function of cortical areas including primary motor cortex and other non-primary motor areas. The combination of motor and sensory symptoms marked in PD, is the cause of investigating the structural changes of motor and sensory cortices in PD. The aim of this study is to detect the structural changes of cortical thickness and the volume of grey matter and white matter in motor and sensory cortices in PD. This will help in early detection of the disease, monitor response to medication or help in prognosis and prediction of outcomes. Nineteen control volunteers and 18 Parkinson's patients were participated in the study. The ages of participants were ranging between 45-65 years (mean 56.14 years). Structural magnetic resonance imaging was performed and the Digital Imaging and Communications in Medicine (DICOM) images were evaluated using automatic brain segmentation software (BrainSuite). The structural changes in the present study were found in the thickness of the motor cortex and in the volume of grey matter (GM) of the sensory cortex. The thickness of the right motor cortex for males was smaller in Parkinson's patients than control. The volume of the GM of left sensory cortex for males was smaller in Parkinson's patients than control. While no differences were found between Patients and controls in the volume of GM or white matter (WM) in the motor cortex, or in the volume of WM and the cortical thickness in the sensory cortex. Structural changes in specific areas of the brain may influence specific symptoms.

1. Introduction

The primary motor cortex (M1), which is located in the precentral gyrus of the frontal lobe, participates in the initiation of skilled, delicate, and agile voluntary movements (Strominger et al., 2012). M1 is critical for fine motor control, such as the independent use of digits among primate species (Geyer et al., 2000). It is interconnected with the parietal lobe, pre-motor cortex, supplementary motor area, basal ganglia, and cerebellum (Kishore et al., 2014). The primary motor cortex has a critical role in the control of the distal limb muscles on the contralateral side of the body, as well as the axial and proximal limb musculature. It receives its main afferents from somatosensory areas and the premotor and supplementary motor cortex (Strominger et al., 2012).

The primary somatic sensory (somatosensory) cortex (SI) includes the postcentral gyrus of the parietal lobe and its medial extension in the paracentral gyrus (Strominger et al., 2012). It receives inputs of touch and pressure and input from muscle spindle and position of joints. The pain and

* Corresponding Author.
Email Address: atteya.m@gmail.com (M. Atteya)
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temperature are slightly represented in the postcentral gyrus (Strominger et al., 2012).

Parkinson's disease is a neurodegenerative disorder commonly diagnosed as motor triad of symptoms including tremor, rigidity and bradykinesia. The loss of dopaminergic projections caused by the degeneration of the Substantia nigra pars compacta in PD, leads to alterations in the function of cortical areas including primary motor cortex (M1) and other non-primary motor areas (Sabatini et al., 2000). In addition to motor features, patients also suffer from non-motor symptoms such as psychiatric problems, autonomic disturbances, pain, fatigue, and most importantly impaired cognition starting in executive functioning, memory, and spatial behavior during the early stage of the disease (Vingerhoets et al., 2003). For this reason, PD research has expanded its investigation beyond the nigrostriatal region to the whole brain in order to characterize the different symptoms (Koshimori et al., 2015).

Motor disabilities could potentially restrict patients' physical activity and together with non-motor symptoms worsen their quality of living during the lifespan (Andreadou et al., 2011). PD is considered as the second most prevalent geriatric neurodegenerative disorder after Alzheimer's disease (Nussbaum and Ellis, 2003). In some patients with PD, some non-motor symptoms even appear earlier than motor symptoms or in the early stage of PD (Weintraub et al., 2004). Thus, it should not be surprising to find that sensory disturbances occur in PD, may well contribute to the motor disturbances that are the hallmark of this condition (Conte et al., 2013).

Impairments in gait and balance, some of the most disabling symptoms in Parkinson's disease (Coelho et al., 2010) are multifactorial in origin and partly arise from impaired integration of sensory feedback from vestibular, visual, and proprioceptive sensory systems (Wright et al., 2010). This observation has motivated the study of sensorimotor cortex in PD.

Many neurological and psychiatric diseases have been found to harbor structural changes in patients' brains. Early and accurate diagnosis of these diseases is crucial in improving the treatment of these diseases. Magnetic resonance (MR) brain images are frequently used to assist diagnosis of these diseases. Detecting brain structural changes from magnetic resonance images can facilitate early diagnosis and treatment of neurological and psychiatric diseases (Chen et al., 2014).

A study done by Dalaker et al. (2009) has detected that no significant differences in whole brain volume between PD patients and age-matched normal control, and no GM-WM differences between the groups (Dalaker et al., 2009). However, another study found decreased GM volume in the frontal, parietal and temporal cortices of PD patients compared with matched healthy controls, and that the increased cognitive dysfunction was associated with more pronounced GM atrophy (Song et al., 2011). Other studies have shown that cortical thinning occurs in PD (Lyoo et al., 2010) and that it is associated with disease duration (Jubault et al., 2011).

This study was designed to assess the volumes of GM and WM and the thickness of motor and sensory cortices in the patients with PD on magnetic resonance images (MRI) using automatic brain segmentation method.

2. Materials and methods

The study comprised 37 subjects. The control volunteers were 19 (10 male, 9 female) and the Parkinson's patients were 18 (10 male, 8 female). The ages of participants were ranging between 45-65 years (mean 56.14 years). The study was approved by the Ethical Committee of the Ondokuz Mayis University, Samsun, Turkey. Patients and controls consented to all procedures; they all signed informed consent to participate in the study.

2.1. Criteria of selection

The patients were adult Turkish subjects; clinically diagnosed as Parkinson's patients. The controls were Turkish volunteers with no history of psychiatric disorders and drug medication. They were matched with patients on the basis of gender and age. Patients and controls were excluded in case of drug abused, tumors, psychiatric problems, or any central neurological problems.

2.2. MRI acquisition

Structural MRI was done in the radiology section, Ondokuz Mayis University, Samsun, Turkey. MRI was performed on SIEMENS 1.5 Tesla scanner. T1-weighted images obtained using three-dimensional acquisition by Magnetization Prepared Rapid Acquisition (MP-RAGE) produces good grey/white matter contrast in coronal section.

2.3. Analysis of the MR images

In order to obtain the quantitative evaluation of the regions of interest (ROIs), morphometric measurements were conducted blinded to clinical data by using two types of software. They are ImageJ software, which was downloaded from http://rsb.info.nih.gov/ij and BrainSuite software, which was downloaded from http://brainsuite.org. ImageJ was used for manipulating the images and BrainSuite for images analysis.

2.3.1. Manipulation of the images using imageJ

ImageJ is produced and distributed by the National Institute of Health (NIH) in the United States of America. The software is in the public domain and it runs on any computer systems.

Measurements from images can be stored separately.

In this study, ImageJ was used to reorient the images. The DICOM images were transferred to ImageJ software and automatically converted into a stack (Fig. 1). The images were resliced, from bottom to top, and opened as axial images, then later flipped horizontally to return to the correct orientation (Fig. 2). Finally, the images were saved as Analyze file format in order to be transferred to the BrainSuite software for segmentation and volume estimation.

2.3.2. Analyzing of the MR images using BrainSuite

The MR images of the subjects were analyzed using automatic segmentation software namely the BrainSuite Version 13a. The BrainSuite is a collection of software tools that enable automated processing of MRIs of the human brain. It includes many features of processing, analyzing, and visualizing MRI data. The major functionality of these tools is to extract and parameterize the inner and outer surfaces of the cerebral cortex and to segment and label grey and white matter structures (Jacobson and Marcus, 2011).

BrainSuite software was performed on a Toshiba personal computer Core i5, 2.30 GHz, RAM 4GB. The software analyzed the MR images in two stages: Cortical Surface Extraction sequence (CSE) and Surface and Volume Registration. Stage one takes about 20 minutes to run while stage two lasts 2 hours and 15 minute, for neach image.

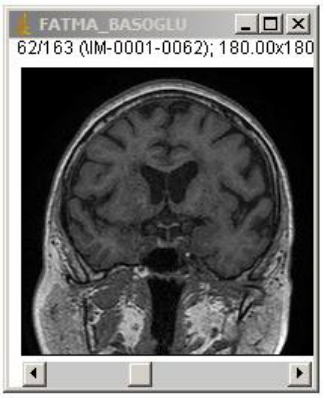
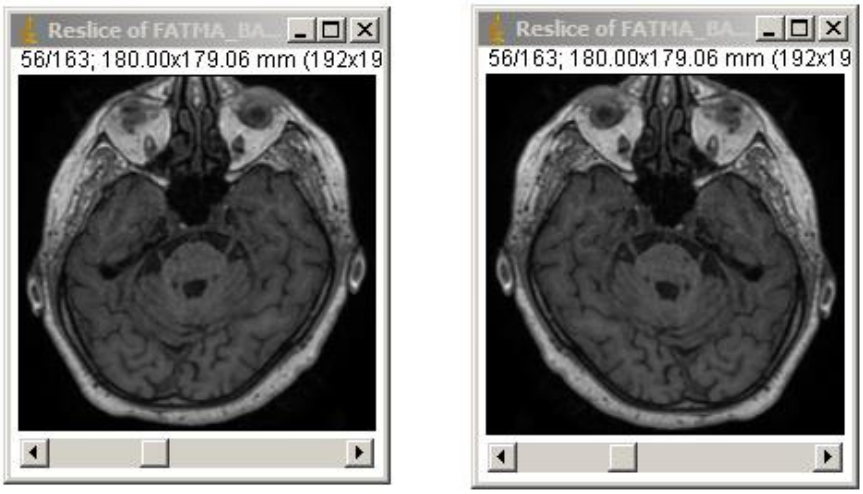


Fig. 1: DICOM images opened in the imageJ as stack



(a) **(b)**

Fig. 2: Dicom images resliced from bottom to top (a) and flipped horizontally (b)

Cortical surface extraction features (CSE)

CSE is at the core of the BrainSuite and is an important first step in conducting many of the types of analysis. Given a T1-weighted MRI image, the CSE automatically skull-strips the image; classifies white matter, grey matter, and cerebrospinal fluid (CSF), and generates 3D models of the inner cortical and pial surfaces. To perform CSE; the Analyze file format, which was prepared through ImageJ, was dragged to the BrainSuite window, and the sagittal, coronal and axial images were shown there in three windows (Fig. 3).

The *CSE* stage was chosen by clicking on *cortex* icon in the BrainSuite *tab bar*. The CSE stage includes ten steps. The 1st step is the *skull stripping*, which is controlled and adjusted manulally to garantee the delineation of the brain tissue, while the rest steps were done automatically. It produces 3-D model of the brain in which the left and right hemispheres are displayed in different colors (Fig. 4).

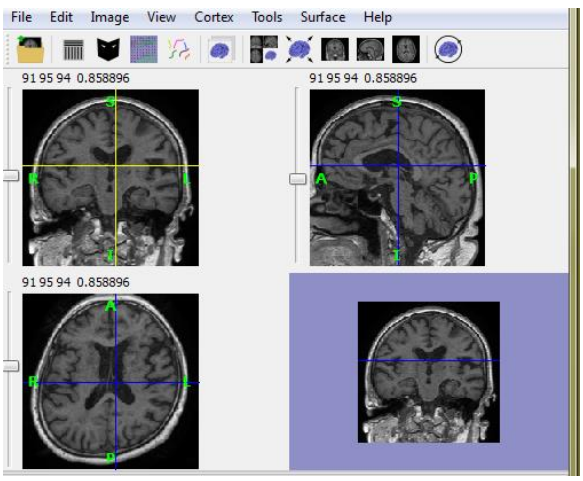


Fig. 3: An analyze file opened in BrainSuite

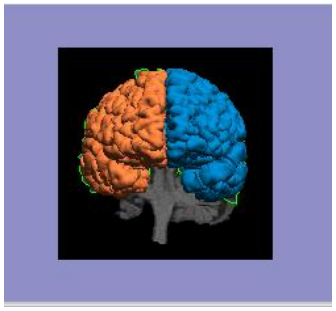


Fig. 4: Split hemispheres (A 3-D Model of the brain after completion of skull strip)

2.4. Statistical analysis

Statistical package for social science (SPSS version 20) was employed for all statistical analyses. Different tables and graphs were used to produce the findings. Independent sample T-test was used to compare the mean volume of grey and white matter of motor and sensory cortices, between controls and Parkinson's patients and to compare the mean thickness of motor and sensory cortices between controls and Parkinson's patients.

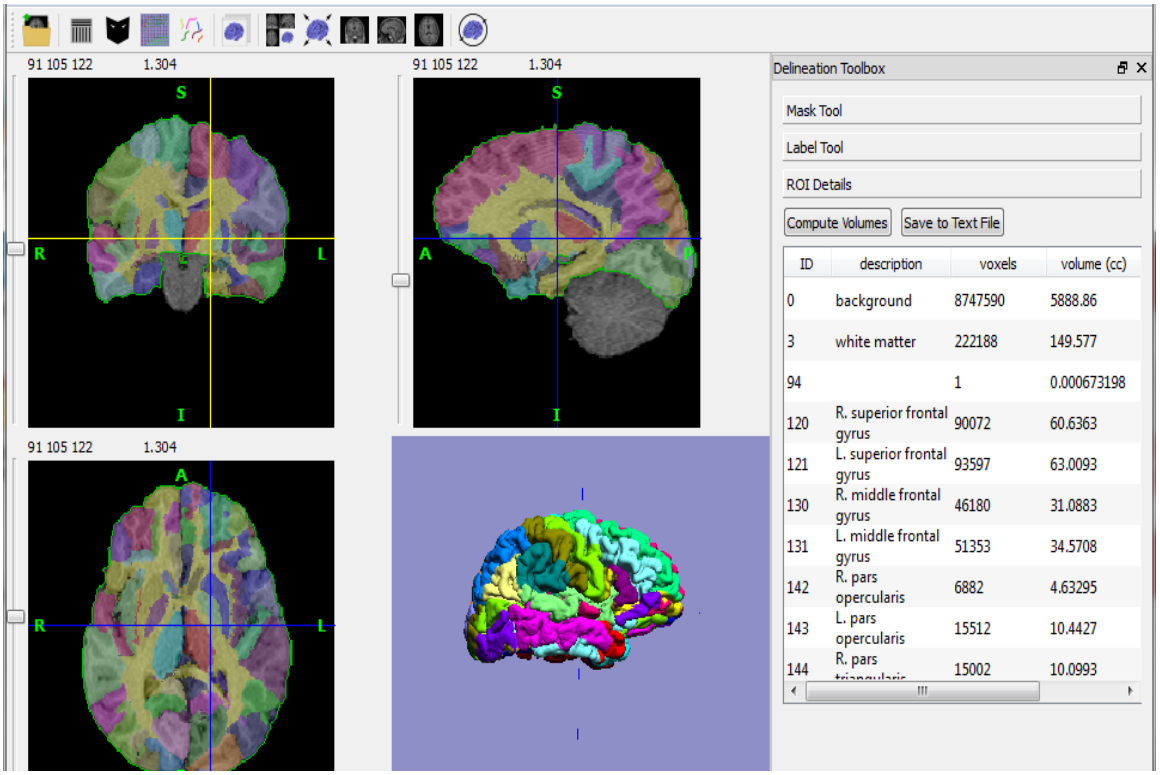


Fig. 5: Automatic labeling of brain ROIs in BrainSuite

	U	V	W	X	Y	Z	AA	AB	AC	AD	AE	AF
1	Region of Interest	Mean_Thickness (mm)	GM_Volume (cm^3)	CSF_Volume (cm^3)	WM_Volume (cm^3)	Total_Volume (GM+WM) (cm^3)	Cortical_Area_mid (cm^2)	Cortical_Area_inner (cm^2)	Cortical_Area_pial (cm^2)		Right Frontal Lobe	Left Frontal L
2	182=R. pre-central lobule	2.94	10.94	4.17	16.10	27.04	50.87	48.49	58.12		152.73	
3	183=L. pre-central lobule	2.59	9.14	6.11	14.19	23.33	43.23	41.74	48.59			
4	222=R. post-central gyrus	3.00	11.73	5.62	13.38	25.11	57.69	52.50	68.58			
5	223=L. post-central gyrus	3.13	13.21	7.99	13.78	26.99	62.36	56.12	75.70			
6	182=R. paracentral lobule	3.13	3.29	1.40	2.79	6.08	14.61	13.21	17.06			
7	183=L. paracentral lobule	3.37	3.20	1.04	3.95	7.14	14.23	12.10	17.96			
8												
9												
10	Right Motor Cortex	4.50	12.58	4.87	17.50	30.08	58.17	55.09	66.65			
11	Left Motor Cortex	4.27	10.74	6.63	16.16	26.90	50.35	47.79	57.57			
12	Total Motor Cortex	4.39	23.33	11.49	33.66	56.98	108.52	102.88	124.22			
13	Right Sensory Cortex	4.56	13.38	6.32	14.77	28.15	64.99	59.11	77.11			
14	Left Sensory Cortex	4.81	14.81	8.51	15.76	30.56	69.47	62.17	84.68			
15	Total Sensory Cortex	4.69	28.19	14.83	30.53	58.72	134.47	121.28	161.79			

Fig. 6: Outputs of BrainSuite on master sheet

3. Results

The structural MR imaging was done for both control and Parkinson's groups using the same scanning parameters. The MR images of the subjects were automatically segmented using brain segmentation software, the BrainSuite. The quantitative data obtained by the automatic brain segmentation and parcellation including the volume of GM, volume of WM and the cortical thickness of motor and sensory cortices information are given here.

3.1. Evaluation of cortical thickness of motor cortex

Regarding the sex difference across the groups, the thickness of the right motor cortex for males was

smaller in Parkinson's group than control group (P=0.03), while no females significant difference across the control and Parkinson's groups was seen (P>0.05) (Table 1).

Table 1: Cortical thickness (cm^2 ±SD) of motor cortex in controls and patients

Gender	Groups	Right	Left	Total
Male	Control	4.84 Ḿ ±0.28	4.81 ±0.38	4.82 ±0.26
	Parkinson's	4.55 Ḿ ±0.27	4.55 ±0.32	4.55 ±0.29
Female	Control	4.60 ±0.25	4.48 ±0.30	4.54 ±0.23
	Parkinson's	4.75 ±0.29	4.71 ±0.34	4.69 ±0.24
Male and Female	Control	4.72 ±0.29	4.65 ±0.38	4.69 ±0.28
	Parkinson's	4.63 ±0.29	4.62 ±0.33	4.61±0.27

Ḿ = sig. (p<0.05)

3.2. Evaluation of volume of grey matter of motor cortex

Regarding the volume of grey matter of right, left, and total motor cortex; there were no statistical significant differences between the males and females within the groups, between the groups, or across the groups of control and Parkinson's (P>0.05) (Table 2).

3.3. Evaluation of volume of white matter of motor cortex

Regarding the volume of white matter of right, left, and total motor cortex; there were no statistical significant differences between the males and females within the groups, between the groups, or across the groups of control and Parkinson's (P>0.05) (Table 3).

3.4. Evaluation of cortical thickness of sensory cortex

The mean thickness of right, left, and total sensory cortices, showed no statistical significant difference between males and females within the groups, between the groups, and across the groups of controls and Parkinson's (P>0.05) (Table 4).

3.5. Evaluation of volume of grey matter of sensory cortex

The volume of grey matter of the right, left, and total sensory cortices, revealed no statistical significant differences between males and females within the groups and between the groups of controls and Parkinson's (P>0.05) (Table 5). Regarding the sex difference across the groups, the volume of grey matter of the left sensory cortex for males was smaller in Parkinson's patients than controls (P=0.03), while no difference in right and in total sensory cortices. No females significant difference across the controls and Parkinson's groups was detected, regarding the volume of grey matter of sensory cortex (P>0.05) (Table 5).

3.6. Evaluation of volume of white matter of sensory cortex

The volume of white matter of right, left, and total sensory cortices, showed no statistical significant differences between the males and females within the groups, between the groups, or across the groups of controls and Parkinson's patients (P>0.05) (Table 6).

Table 2: Mean volume (cm^3 ±SD) of grey matter of motor cortex in controls and patients

Gender	Groups	Right	Left	Total
Male	Control	12.88 ±1.74	12.20 ±1.22	25.08 ±2.28
	Parkinson's	11.45 ±1.62	11.16 ±1.17	23.46 ±3.25
Female	Control	11.35 ±1.50	10.67±1.29	22.02±2.48
	Parkinson's	11.71 ±1.86	11.06 ±0.98	22.85 ±2.84
Male and Female	Control	12.15 ±1.77	11.48 ±1.45	23.63 ±2.79
	Parkinson's	11.56 ±1.68	11.12 ±1.06	23.21±3.01

Table 3: Mean volume (cm^3 ±SD) of white matter of motor cortex in controls and patients

Gender	Groups	Right	Left	Total
Male	Control	15.83 ±2.47	14.47 ±2.33	30.30 ±4.65
	Parkinson's	16.23 ±3.00	15.41 ±2.89	30.38 ±4.25
Female	Control	14.25 ±2.40	13.59 ±1.91	27.84 ±4.13
	Parkinson's	13.96 ±3.41	14.18 ±2.52	28.14 ±5.46
Male and Female	Control	15.08 ±2.50	14.05 ±2.13	29.13 ±4.47
	Parkinson's	15.22 ±3.30	14.87 ±2.72	29.33 ±4.84

Table 4: Cortical thickness (cm^2 ±SD) of sensory cortex in controls and patients

Gender	Groups	Right	Left	Total
Male	Control	4.68±0.27	5.17±0.43	4.92 ±0.30
	Parkinson's	4.50±0.18	4.89±0.33	4.69 ±0.24
Female	Control	4.59±0.27	4.91±0.18	4.75 ±0.19
	Parkinson's	4.69 ±0.33	4.93 ±0.25	4.76 ±0.30
Male and Female	Control	4.64 ±0.27	5.05 ±0.36	4.84 ±0.26
	Parkinson's	4.58 ±0.26	4.90 ±0.29	4.72 ±0.26

Table 5: Mean volume (cm^3 ±SD) of grey matter of sensory cortex in controls and patients

Gender	Groups	Right	Left	Total
Male	Control	12.20 ±1.64	16.50Ḿ ±1.93	28.45 ±2.87
	Parkinson's	11.88 ±1.87	14.61Ḿ ±1.31	26.48 ±2.72
Female	Control	11.54 ±1.41	14.65 ±2.64	26.19 ±3.26
	Parkinson's	11.77 ±1.41	14.04 ±2.38	25.74 ±3.31
Male and Female	Control	11.87 ±1.52	15.62 ±2.42	27.32 ±3.20
	Parkinson's	11.83 ±1.63	14.40 ±1.73	26.20 ±2.87

Ḿ = sig ($p<0.05$)

Table 6: Mean volume (cm^3 ±SD) of white matter of sensory cortex in controls and patients

Gender	Groups	Right	Left	Total
Male	Control	11.98 ±2.02	13.53 ±1.38	26.01 ±3.81
	Parkinson's	13.19 ±3.57	14.23 ±1.49	27.42 ±4.90
Female	Control	11.39 ±2.39	13.09 ±1.70	24.48 ±3.96
	Parkinson's	11.36 ±3.74	12.26 ±2.34	25.62 ±7.77
Male and Female	Control	11.69 ±2.17	13.30 ±1.52	25.24 ±3.85
	Parkinson's	12.38 ±3.66	13.42 ±2.07	26.62 ±6.20

4. Discussion

4.1. Thickness of motor cortex

The result of the present study revealed that the thickness of the right motor cortex for males was smaller in Parkinson's patients than control. The present finding agrees with studies that found thinning in right precentral area (Pereira et al., 2012; Kim et al., 2014). The present finding disagreed with Koshimori et al. (2015), who found significant reduction in cortical thickness in the left precentral gyrus for PD patients instead of the right cortex (Koshimori et al., 2015).

The major pathological change in PD is a degeneration of nigrostriatal dopaminergic neurons. However, PD is a network disorder and is known to involve almost the whole brain (Braak et al., 2003). Despite less vulnerable cortical structures gradually become affected as the disease progresses, several recent MR-based imaging analyses of PD revealed that cortical atrophy exists even in early stages of disease development (Jubault et al., 2011). Among several other methods, cortical thickness analysis has been introduced as an effort to overcome the difficulties in analysis of highly convoluted cerebral cortex and have demonstrated focal cortical thinning pattern in neurologic diseases. However, the results using cortical thickness analysis in PD patients were not consistent between the studies (Kim et al., 2014). Cortical areas associated with motor functions are known to be damaged in PD even in early stage by recent imaging analyses (Zarei et al., 2013).

Kim et al. (2014) study on right-handed patients revealed a significant cortical thinning in motor-related areas, only in patients with left-sided disease onset (LPD), but not in patients with right-sided disease onset (RPD). More strenuous exercise performed by the right side of the body may explain the absence of significant cortical thinning in areas of the motor related cortex in the RPD group. These results suggest the possibility that enhanced physical activity by handedness may have a neuroprotective effect on contralateral cerebral structures (Kim et al., 2014).

Studies showed that the thickness of the cortical area did not correlate with the severity of motor symptoms including tremor, rigidity or bradykinesia (Zarei et al., 2013; Kim et al., 2014). Koshimori et al. (2015) stated that the biological underpinnings of reduced cortical thickness are not fully understood, although the MRI techniques used in their study were validated methods to assess structural changes. In the healthy elderly, cortical thinning may occur in the brain areas where no neuronal loss is found (Freeman et al., 2008). Cortical thinning might be associated with the reduced size of neuronal cell bodies, reduced dendritic arborization, and/or the loss of presynaptic terminals (Pellicano et al., 2012). PD has been associated with cytoskeletal damage of various neuronal cells including dopaminergic, glutamatergic, cholinergic, tryptaminergic, GABAergic, noradrenergic and adrenergic neurons (Foley and Riederer, 1999).

Dysfunction of basal ganglia circuitry and/or cerebello-thalamo-cortical circuitry is suggested as a possible mechanism for generation of tremor in PD (Rodriguez-Oroz et al., 2009; Lewis et al., 2011). In addition, increased excitability of primary motor cortex, increased responsiveness of basal ganglia circuitry to peripheral afferent stimuli have been suggested as a possible mechanism explaining rigidity (Rodriguez-Oroz et al., 2009). Therefore, the result of reduced thickness of primary motor cortex in the current study might be due to the effect of disease on cortical circuitry in PD.

Pereira et al. (2012) and Zarei et al. (2013) mentioned that the neurodegenerative process in PD is reflected by cortical folding and especially cortical thickness measurements (Pereira et al., 2012; Zarei et al., 2013). Although such measurements are presumed to detect changes in the cortical GM layer of PD patients, pathological postmortem confirmation of these changes is needed. Therefore, future studies assessing a wide sample of PD patients in different stages of the disease and involving postmortem pathological confirmation, are essential to further assess the relationship between the clinical course and the neurodegeneration in PD (Zarei et al., 2013). The pattern of cortical thinning that Zarei et al., (2013) found on Parkinson's patients, was different from that previously reported for normal aging in Salat et al. (2004). Therefore, the result of reduced cortical thickness found in the

present study, might be due to the effect of disease on patients and not due to aging effect.

4.2. Grey matter volume of motor cortex

The present study showed no significant difference in grey matter of motor cortex for Parkinson's patients in comparison with controls. The present finding agreed with Brenneis et al. (2003), who found no significant volume difference in GM between PD patients and controls (Brenneis et al., 2003). Brenneis suggested in his finding that impairment of GM of motor cortex in PD consists of functional deficits rather than structural changes (Brenneis et al., 2003). This suggestion could explain the finding of the present study which was in accordance with Brenneis finding.

The present findings disagree with Goldman et al. (2014), who found reduced GM volume in bilateral precentral gyrus (Goldman et al., 2014), and with other studies that found reduced GM in left precentral gyrus (Kostic et al., 2012; Gonzalez-Redondo et al., 2014). The precentral gyrus is the origin of the pyramidal tract; therefore, Shao et al. (2014) suggested that the impairment of the precentral gyrus may partially contribute to the motor deficits of PD. Because of the complex and varied clinical manifestations of PD and different clinical data in studies, there are often varying results.

4.3. White matter volume of motor cortex

The results of the current study showed no differences between control and patients regarding the volume of white matter of motor cortex. Supporting the present finding, previous studies found no significant difference in volume of white matter of motor cortex (Brenneis et al., 2003; Pereira et al., 2012).

The clear picture of neurobiological meaning of inconsistency in the volume of motor cortex will emerge only from longitudinal neuroimaging studies on larger groups and histological studies on a suitable animal model. In the present study, the number of subjects was less than 30 in each group, which might be the cause for this inconsistency in the results.

4.4. Thickness of sensory cortex

The present study found that there is no difference in thickness of sensory cortex between patients and controls. The finding was consistent with Jubault et al. (2011), who found no significant difference in thickness of sensory cortex (Jubault et al., 2011). On the other hand, the finding disagreed with Pereira et al. (2012) who found cortical thinning in postcentral area in patients (Pereira et al., 2012), and with Koshimori et al. (2015), who found thickness reduction in left postcentral area (Koshimori et al., 2015). Cortical thinning is reported to be associated with non-motor symptoms such as cognitive dysfunction (Nagano-Saito et al., 2005).

4.5. Grey matter volume of sensory cortex

The present study found that the GM volume for left sensory cortex for males was reduced in Parkinson's patients than control. The present finding agrees with Xia et al. (2013), who found diminished grey matter volume in left postcentral gyrus in patients with Parkinson's disease compared with normal controls. Hence, the volume reduction of grey matter resulted in damage in the function of corresponding brain areas (Xia et al., 2013). An explanation for volume reductions in middle and late adulthood could be due to shrinkage of large neurons (Peters et al., 1998), or rarefaction in the GM microvasculature, resulting in a loss of neurons (Riddle et al., 2003).

A previous study suggested that healthy brain ageing is a process affecting predominantly WM but not GM (Piguet et al., 2009). Hence, the reduction of GM volume found in Parkinson's patients in the present study probably has no link with aging. The result might confirm the presence of neurodegenerative changes in Parkinson's patients.

Sex dimorphism in the GM is subject to the influence of various factors. There is substantial evidence showing that age-related brain decline is sex- and region-specific, although the results are inconsistent. Sex differences of brain volumes loss with ageing were not detected in a longitudinal study (Tang et al., 2001), as well as a cross-sectional study (Raz et al., 2003). It is not yet known if sex affects brain volumes through its interactions with factors other than age, handedness and years of education (Chen et al., 2006). Vascular risk factors may also play a role in sex dimorphism of regional GM in brain, as has previously been reported (Taki et al., 2004). In the present study, the effect of the disease might be more on the males than on the females, since the reduction of GM has been reported only in male patients.

4.6. White matter volume of sensory cortex

The present study did not find any difference in the white matter for sensory cortex, between control and Parkinson's patients. The finding was inconsistent with Raz et al. (1997), who found increased white matter in the right postcentral gyrus (Raz et al., 1997).

There have been remarkable differences in the methods used for extracting brain volume and morphometric data across studies. These differences in methodologies and experimental designs may account for a substantial amount of the heterogeneity reported. Beside the brain segmentation methods, the Voxel-based analyses are also a technique used for volume estimation (Morgen et al., 2011).

5. Conclusion and recommendation

In the present study, the volume changes of GM and WM and the comparisons between controls and Parkinson's subjects were evaluated, by using the brain segmentation techniques. Since the data obtained in this study is independent from the observer's evaluation, the obtained data are unbiased.

Our finding revealed that the thickness of the right motor cortex for males was smaller in Parkinson's patients than control, indicating that the disease resulted in a thickness decrease in male patients. However, no differences in GM and WM were seen in motor cortex between controls and patients. Suggesting that impairment of GM and WM of motor cortex in PD consists of functional deficits rather than structural changes. The finding in sensory cortex includes reduction in the volume of the GM of left sensory cortex for male patients, indicating that the disease resulted in a volume decrease in the males. However, no differences in the cortical thickness or in the volume of WM were seen in sensory cortex between controls and patients.

Disclosure

The authors report no conflicts of interest in this work. The study was approved by the Ethical Committee of the Ondokuz Mayis University, Samsun, Turkey. Patients and controls consented to all procedures; they all signed informed consent to participate in the study.

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