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Evaluation Relationship between Multiple Sclerosis and Corpus Callosum Atrophy by MRI in Females

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Abstract

Background: Axon destruction in the Corpus callosum is known as one of the most important elements in multiple sclerosis. It is noteworthy that corpus callosum atrophy usually occurs in a patient with multiple sclerosis. MRI can show macroscopic pathological areas caused by multiple sclerosis with high sensitivity.

Objective: Quantitative callosal measurement in patient with MS and compare with healthy group.

Methods: Midsagittal corpus callosum areas were determined in 30 controls with normal MRI and 30 patients (Female) with definite multiple sclerosis. Groups included individuals of different age ranging from 20 to 40 years. Corpus callosum area included callosal length, body, genu and splenium width measured by straight line. The length of the brain was measured by measuring the anterior-posterior dimension from the frontal pole to the occipital pole.

Result: The mean midsagittal corpus callosum length, body width, genu width and splenium width were 67±3.14, 5.56±2.26, 7.7±1.57 and 13.66±2.32 in patient and 68.83±4.2, 5.63±0.8, 10±1.14 and 15.6±214 in healthy group. The present study showed a significant difference in thickness of genu and splenium parts of corpus callosum in patient compare with healthy group.

Conclusion: Corpus callosum morphology abnormalities can be assessed by using an available MRI device. In this study, it was also found that MS caused a corpus callosum atrophy that could be measured by a quantitative MRI.

Keywords: Atrophy; Corpus callosum; MRI imaging; MS disease; Measurement

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Introduction

The Corpus callosum, a regular and dense white matter, plays the role of connection and communication between the two hemispheres, which can be viewed and evaluated by Magnetic Resonance Imaging (MRI) [1]. It is involved in various cerebral activities that cause to organize the role of the cortex areas [1,2]. Histological studies have shown that white matter of corpus callosum formed in various regions from the anterior (genu and rostrum) medial (body) and posterior (splenium). Regions of the corpus callosum principally associate different anterior and posterior cortical areas in two hemisphere [2]. The anterior fibers will connect between the two frontal lobes, the anterior fibers of the trunk will transmit the motor information, the posterior fibers of the trunk comprise the somatosensory information, the isthmus plays a role in the transmission of auditory information, and ultimately the splenium section transmits visual data [3-5]. The corpus callosum contains a collection of heterogeneous and complex fibers. The nerve fibers in the sensory area, visual area and primary motor, in addition to the thickness, have more myelin than the associative areas [6]. The corpus callosum has been shown to be altered in conditions for example dyslexia, schizophrenia, even when visual assessment of the MR images reveals normal findings. A quantitative assessment of corpus callosum can be useful as a prognosis for the progression of diseases of the nervous system such as Alzheimer and multiple sclerosis disease. Multiple Sclerosis (MS), known as an inflammatory disease of the central nervous system, is one of the chronic autoimmune diseases [7] (Figure2). Disease symptoms develop in MS with the damage of myelinated axons and the destruction of myelin in the central nervous system, which are indicated in the form of MS plaques in the MR images [8]. When disease affects a previously normal CC, interhemispheric disconnection

symptoms occur that include visual, scmesthetic, kinesthetic, auditory, and complex function impairment. Typical features of the callosal syndrome syndrome include neurological disorders such as unilateral motion disability in the dominant hemisphere, difficulty in the movement of the opposite hand, homolateral sensory defect, and homolateral vision and hearing impairment by on dichotic tests [9]. It should be noted that the principal cause of MS for researchers is not properly known. MS involves the white matter of central nervous system. There is a concern that MS is the most common neurologic disease among young adults. Most patients are between the ages of 20 and 40, but the disease may even start in childhood or after age 60. It is also important to know that the proportion of female to male patients is about 7 to 3 [10]. Recent data provide evidence for primary involvement and neurodegeneration of central and cortical gray matter. MRI is now the dominant laboratory method for diagnosis of MS. MS lesions is usually easily detected and often is characteristic. Conventional MRI techniques are now widely accessible to community and academic neurologists. The lesions appear bright and high signal on T2-weighted and FLAIR sequences, indicating a higher than normal water content of inflammatory lesions. These MRI sequences can accurately determine the size of the MS plaques and their position. Lesions are usually iso signal on T1-weighted images, indicating that the tissue itself is intact. Lesions may be present in many areas of the brain, but most typically they are found adjacent to the lateral ventricles, oriented perpendicular to them, and in the corpus callosum (best seen on midline sagittal FLAIR images and in the cerebellar peduncles) [11]. MS plaques directly touch the ventricular wall following the location of the small venules. In contrast, small vascular lesions are usually seen several millimeters away from the ventricular wall. Usually, global and focal cerebral atrophy are determined by measuring the brain and the spinal cord [12]. Atrophy correlates with axonal and neuronal loss, and physical and cognitive impairment. Atrophy of corpus callosum and presence of demyelinating lesions in callosal or sub-callosal regions are basic findings in patients with multiple sclerosis [13]. Using MR imaging system can investigate of cerebral anatomy and function. Several neuroimaging studies have utilized the mid sagittal regions of the corpus callosum to show differences in morphology related to sex, aging, handedness, and pathologic states.

The current study was designed to determine the relationship between corpus callosum atrophy, as an axonal injury, between patients with definitive multiple sclerosis and normal patients.

Patients and Methods

This descriptive study was performed in the Kianpars Imaging center, Ahvaz, Iran. MRIs of 30 (female) healthy volunteers and 30 (female) participants with MS were used. In this study, individuals with different groups were present in the range of 20 to 40 years. The brain MRI was done with a Signa unit (General Electric, USA) operating at field strength of 3 Tesla. T1 weighted sagittal, T2 weighted sagittal and axial T2 Flair were obtained using a spin echo pulse Sequence. The slice thickness was 4 mm for sagittal images and 5 mm for axial images.

Images of selected subjects were recalled on the monitor and the image showing the mid-sagittal section of the corpus callosum was magnified. The anteroposterior length of the Corpus Callosum (CCL) was measured by a straight line joining the anterior-most point of the genu to posterior-most point of the splenium. The Width of the Genu (WG) was the distance from the anterior-most point of the genu to the anterior-most point of the inner concavity of the anterior part of the corpus callosum. The Width of the Trunk (WT) is measured as the dorso-ventral width of the corpus callosum in the midpoint. The Width of the Splenium (WS) was the vertical distance between two points at the dorsal and ventral margins of the splenium at its widest part (Figure 2) and the brain length was longitudinal dimension of the brain from the frontal to the occipital pole. Figure 1 shows MS plaques in T2 flair imaging weighted with magnified. Figure 3 shows abnormal corpus callosum in patient with MS disease. Data were statistically analyzed using SPSS program.

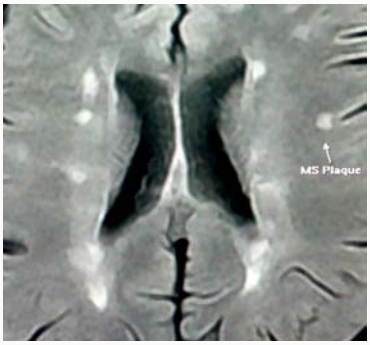

Figure 1: MR T2 Flair imaging show MS plaque.

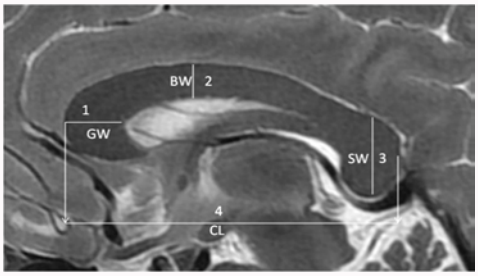

Figure 2: Normal MR T2 Imaging.1) Genu width, 2) Body width, 3) Splenium width and 4) Callosal length.

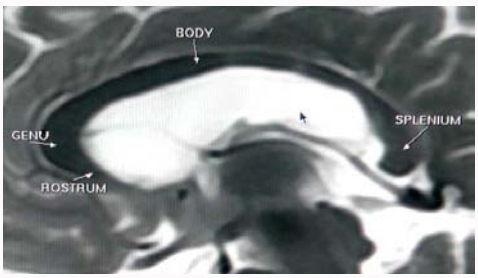

Figure 3: MR T2 shows corpus callosum in patient with MS.

The independent t-test for unpaired samples was then calculated to determine if significant differences were present between healthy and diseased groups.

Result

The mean value for the corpus callosum length was 67±3.14 mm in patient group and 68.83±4.2 mm in healthy group (*P*-Value 0.246).

The mean value for the body width was 5.56±2.26 mm in patient group and 5.63±0.8 mm in healthy group (*P*-Value 0.88).

The mean value for the genu width was 7.7±1.57 mm in patient

Table 1: Means of corpus callosum parts for control and patient groups. Scale in millimeters.

Parameter	N	Mean ± SD	Min	Max
CCL: patient	30	67 ± 3.14	60	74
control	30	68.83 ± 4.2	59	75
GW: patient	30	7.7 ± 1.57	5	12
control	30	10 ± 1.14	8	12
BW: patient	30	5.56 ± 2.26	3	12
control	30	5.63 ± 0.8	4	7
SW: patient	30	12.66 ± 2.32	6	16
control	30	15.06 ± 2.14	9	19
FOL: patient	30	157.67 ± 7.9	151	164
control	30	163.65 ± 6.6	161	170

Table 2: Showing comparison of thickness of different parts of Corpus callosum in control and patient groups with its significance.

Parameter	Mean ± SD	P-value	T	df
CCL: patient	67 ± 3.14	0.246	-1.173	58
control	68.83 ± 4.2			53.35
GW: patient	7.7 ± 1.57	0.000	-7.292	58
control	10 ± 1.14			52.992
BW: patient	5.56 ± 2.26	0.88	-.152	58
control	5.63 ± 0.8			36.248
SW: patient	12.66 ± 2.32	0.019	-2.423	58
control	15.06 ± 2.14			57.645
FOL: patient	157.67 ± 7.9	0.000	-4.075	58
control	163.65 ± 6.6			97.67

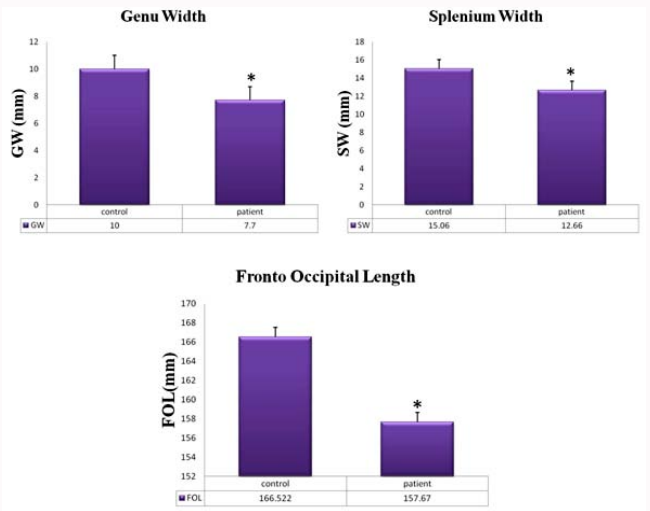

Figure 4: Genu and Splenium width and Fronto-Occipital length of control and patient groups. Values are expressed as mean±SD for 2 groups. *p<0.05, * indicate comparison to the control group, which indicates a significant difference between the control group and the patient group in all three charts.

group and 10±1.14 mm in healthy group (*P*-Value 0).

The mean value for the splenium width 12.66±2.32 mm in patient group and 15.06±2.14 mm in healthy group (*P*-Value 0.019).

The mean value for the fronto occipital length 157.76±7.9 mm in patient groups and 163.65±6.6 mm in healthy group (*P*-Value 0).

The present study showed a significant difference in thickness of genu and splenium parts of corpus callosum in patient compare with healthy group, but corpus callosum length and body thickness was not significantly difference between patient and healthy groups.

Also, the fronto occipital length showed strong significant between two group (Table 1and 2).

In this study observed decrease size in patient group include genu and splenium width, also the fronto occipital length decrease compare healthy group (Figure 4 and 5).

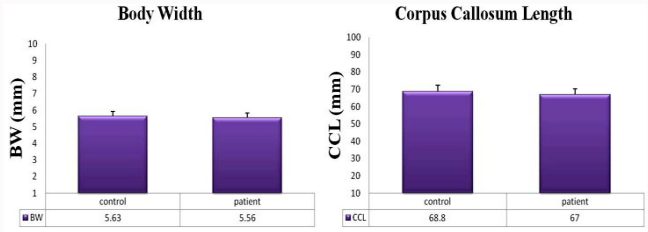

Figure 5: Body width and Corpus Callosum length of control and patient groups. Values are expressed as mean±SD for 2 groups, which does not indicate a significant relationship.

Discussion

Multiple Sclerosis (MS) is a complicated autoimmune and inflammatory disease that results in neurological damage to the brain and the spinal cord [14], causing irreversible atrophy in the areas mentioned [15]. At present, the basic and accepted method of imaging a nervous system that is used to evaluate the progress of MS is an MRI technique that can show both lesion and atrophy.

Visible focal lesions in MRI can be considered as one of the important diagnostic features of MS, but these lesions are poorly correlated with clinical progression. It is noteworthy that neuroxoneal atrophy is an irreversible process, while focal lesions fluctuate over time.

Therefore, changing the volume of the brain and the spinal cord can be more and more associated with the disability of long-term MS.

Corpus Callosum (CC) contains the main fibers of connection and communication between the different lobes of the two cerebral hemispheres. As the largest brain interface, the exact performance of this particular neuroanatomical structure has not yet been determined [16].

Midsagittal callosal area may have the highest number of small diameter transmission fibers within the CC [17]. Decreased midsagittal callosal thickness, which provides more spatially detailed information than parcellated area measures, presumably also reflect decreases in the amount of fiber between the two hemispheres, or a decrease in the percentage of myelination in the nerve fibers.

Autopsy series show that MS plaques are classically associated with local volume loss, due principally to decreased myelin in the axon sheath. In a series of 20 MS brains reported by Barnard [13], corpus callosum atrophy was accompanied by both demyelination and axon loss, but the relative contributions of these factors were not determined. Post-death studies as well as recent researches using MRI have shown that atrophy of callosum atrophy is one of the common findings in patients with MS [13] and when compared with patients who are in the early stages of MS, there is a significant reduction in the area of corpus callosum as well as in corpus callosum controlled for brain size [18].

This is the first report of the mean and range of the length, width, and area of the corpus callosum in patient with definitive multiple sclerosis in south of Iran, and can be considered as a basis for comparison between different diseases that alter the corpus callosum structure.

Klawiter et al., in their study, used quantitative MRI and showed that there is a meaningful relationship with the reduction of gray matter in the brain, not the white matter, with Corpus Callosum

Atrophy (CCA) in MS patients [19].

In this study, as indicated in the results, five components of genu width, width of splenium, body width, corpus colosum length and fronto occipital length were evaluated.

In this research, we used a quantitative MRI and it was proved that significant abnormalities were made to the corpus callosum structure in MS.

We showed a significant difference in thickness of genu and splenium parts of corpus callosum in patient compared with healthy group. There is a large variation in callosal size in different groups.

Since the genu and splenium region are specifically linked to the frontal lobes through minor forceps and occipital lobes through major forceps, thus, the atrophy of these areas due to MS may be the cause of motor disorders as well as diplopia and blurred vision, which is evident in MS patients.

Simon et al., found significant differences between controls and MS patients, in both the mean thickness of CC [20], and its area [21]. The association of MS [22,23] and callosal atrophy has been described with a prevalence ranging from 2% to more than 50%. This atrophy has essentially been determined visually. However, Jean Pierre et al., demonstrated quantitatively, following the works of Dietemann et al., that CC atrophy occurs mainly in long-standing MS cases, after 10 years of evolution. In more recently affected patients, no significant callosal difference with controls was identified, except in those patients with severe progressive MS [24].

Dietemann et al., [25] subjectively evaluated the size and morphology of CC in MS, and correlated the involvement of CC with the duration and severity of the disease.

But Martola et al., in a study for four decades, have shown that atrophy of corpus callosum occurs in patients with MS independent of MS course and physical disability. They showed that CCA was not related to age, sex, onset of disease, and duration of MS [26].

Our observations support prior reports of predominantly positive associations between multiple sclerosis and corpus callosum measures. Research has shown that MS plaques are more likely to be in periventricular white matter [27]. However, little information is available about the effects of various diseases on the corpus callosum, which is a structure that forms a large part of the periventricular tissue [28]. This research, using quantitative measurements, confirms previous studies (*in vivo*) [29] and as well as postmortem studies [13] which represent the role of MS in corpus callosum atrophy. The results of the research showed that in the MS group, the mean of the corpus callosum area significantly decreased, while the cause of the atrophy was still not clearly known.

Conclusion

In conclusion, by using MRI images and analyzing their quantitative data, significant atrophy was observed in some parts of the corpus callosum. Which itself is due to the MS's destructive effects and can also justify the MS symptoms. Although the mechanism of the MS effect on corpus callosum atrophy is not well defined, but a more accurate assessment of MRI images can provide a reasonable link between the rate of corpus callosum atrophy and the severity of motor and neurological disorders. Finally, one question arises that the atrophy of these parts of the corpus callosum occurs only on the MS? This could be of interest to researchers.

Suggestion

Assessment multiple sclerosis and corpus callosum atrophy with brain size in CT scan and MRI in female and male.

Acknowledgments

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