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Volumetric examination of the cerebellum and cortical visual centres in individuals with nonarteritic anterior ischemic optic neuropathy using automatic segmentation

Nonarteritik anterior iskemik optik nöropatili bireylerde serebellum ve kortikal görme merkezlerinin otomatik segmentasyon kullanılarak volümetrik incelenmesi

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ABSTRACT

Objective: Non-arteritic anterior ischemic optic neuropathy (NAION) is a clinical condition caused by acute ischemic damage to the anterior part of the optic nerve. In this study, brain magnetic resonance imaging (MRI) images of NAION patients and healthy control subjects (HC) were analyzed and compared using an automatic segmentation method.
Materials and Methods: The study included 14 patients diagnosed with NAION and 14 HC. VolBrain method, which provides brain parcellation with Voxel-Based Morphometry (VBM), was used for volumetric analysis of brain MR images. In the statistical analysis of the findings, the Mann-Whitney U test was employed.
Results: We found that cerebrum, gray and white matter, frontal lobe and middle frontal gyri, occipital lobe, calcarine cortex, lingual gyrus, superior and middle occipital gyri volumes were significantly lower in NAION patients compared to the HC ($p < 0.05$). There was no statistically significant difference between the two groups in the volume analysis of the cerebellum and its parts ($p > 0.05$).
Conclusion: Our study reveals that NAION induces volumetric changes in visual and non-visual regions of the cerebral cortex, but not in the volume of the cerebellum.
Keywords: Cerebral cortex, cerebellum, optic neuropathy, MRI, volume

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ÖZET

Amaç: Non-arteritik anterior iskemik optik nöropati (NAİON), optik sinirin ön kısmındaki akut iskemik hasarın neden olduğu klinik bir durumdur. Bu çalışmada, NAİON hastalarının ve sağlıklı kontrollerin (HC) beyin manyetik rezonans görüntüleme (MRG) görüntüleri otomatik segmentasyon yöntemi kullanılarak analiz edildi ve karşılaştırıldı.
Gereç ve Yöntem: Çalışmaya 14 NAİON ve 14 HC tanılı hasta dahil edildi. Beyin MR görüntülerinin volumetrik analizi için Voksel Tabanlı Morfometri (VBM) ile beyin parselasyonu sağlayan VolBrain yöntemi kullanıldı. Bulguların istatistiksel analizinde Mann-Whitney U testi kullanıldı.
Bulgular: NAİON hastalarında cerebrum, substantia grisea ve substantia alba, lobus frontalis ve gyrus frontalis medius, lobus occipitalis, cortex calcarinus, gyrus lingualis, gyrus occipitalis superior ve gyrus occipitalis medius hacimlerinin HC'ye göre anlamlı derecede düşük olduğunu bulduk ($p < 0,05$). Cerebellum ve bölümlerinin hacim analizinde iki grup arasında istatistiksel olarak anlamlı bir fark yoktu ($p > 0,05$).
Sonuç: Çalışmamız NAİON'un cortex cerebri'nin görsel ve görsel olmayan bölgelerinde hacimsel değişikliklere yol açtığını, ancak serebellum hacminde anlamlı bir değişiklik oluşturmadığını ortaya koymaktadır.
Anahtar Kelimeler: Serebral korteks, serebellum, optik nöropati, MRG, hacim

INTRODUCTION

The eye is a complex sensory organ that detects light and transmits the rays falling on the retina to the brain via the optic nerve, which facilitates the reception of visual stimuli, plays an important role in the perception of the environment we live in and our integration with the environment. Photoreceptor cells in the retina detect the visual impulse. The visual stimulus is transmitted to the cortical visual centers via the optic nerve and subsequent visual pathways (1, 2). Optic neuropathy (ON) occurs as a result of acute, subacute or chronic damage to the optic nerve. It manifests itself with symptoms such as decreased visual acuity, blurred vision, loss of visual field, and deterioration of color vision (3). The most common types are optic neuritis and ischemic ON. Optic neuritis is observed at a younger age while ischemic ON develops at an advanced age. There are also traumatic, compressive, infiltrative, and metabolic ON types (4). Non-arteritic anterior ischemic optic neuropathy (NAION) refers to the development of an idiopathic ischemic process in the anterior portion of the optic nerve. Patients are generally over the age of 50 years with vasculopathic risk factors (eg, diabetes mellitus, hypertension and obstructive sleep apnea) (5, 6). In individuals with vision loss, stimuli to the eye cannot be adequately transmitted to the cortical visual center. As a result, atrophy occurs in the cortical visual center (7). The pathogenesis of NAION is not yet fully known. A transient ischemia of the short posterior ciliary vessels not triggered by thromboembolic events is suspected. A history of sudden onset scotoma not associated with pain, (sectoral) optic disc swelling, afferent pupillary defect and visual field defect are of diagnostic significance (8). Studies that perform volume analyses are becoming increasingly common in the literature. Automatic programs are used to examine the morphometry of brain parts in detail (9, 10). Using Voxel-Based Morphometry (VBM), the volumes of the whole brain, white matter (WM) and gray matter (GM), brainstem, cerebrospinal fluid (CSF), hippocampus, and cerebellum can be determined (11). In the literature, there are studies investigating the effects of conditions such as optic neuritis, glaucoma, congenital vision loss and late vision loss on brain volume by automatic segmentation method (12-15). However, we did not come across studies investigating the effects of NAION. In the present study, it was aimed to investigate the effect of NAION on both cortical visual centers and areas of the brain responsible for balance and coordination.

MATERIALS AND METHODS

Before starting the study, approval was obtained from Tokat Gaziosmanpasa University Non-Interventional Clinical Research Ethics Committee. (Project No: 23-KAEK-255). Based on the volume of lobus occipitalis, substaintia grisea and substantia alba, According to the 95% confidence (1-α), 95% test power (1-β), d=1.493 effect size,and the two-way hypothesis, the total number of cases was determined as 26, 13 in each group (16). The radiological images were obtained from the Sectra archive system, which contains magnetic resonance (MR) images of the Department of Radiology of Tokat Gaziosmanpaşa University. Brain and cerebellum images of the individuals included in the study were obtained from T1-weighted three-dimensional volumetric TFE (turbo field echo) sequences taken by MR imaging (MRI) devices in the hospital. Of the 22 patients diagnosed with NAION in the archive system between 2019 and 2024, we included 14 patients who had MR images. The images of 14 HC (healthy control subjects) were used as a control group. Since the study was retrospective, informed consent was not applied. For volumetric analyses, the VolBrain (v.1.0, http://volbrain.upv.es), a free online web-based system for brain MRI data processing system, was employed. VolBrain is a fully automated segmentation technique based on multi-atlas patch-based label fusion segmentation technology (17). VolBrain gives fast results and there is no need for additional processes such as installation and assembly (9). We saved the obtained images to an external memory. Registration was completed by entering the https://volbrain.upv.es/instructions.php website. After the membership and registration processes, the sagittal 3D-T1 DICOM brain MR images on our computer were converted to NIfTI (Neuroimaging Informatics Technology Initiative) format. In the process of converting MR DICOM (Digital Imaging and Communications in Medicine) images to NIfTI format, the dcm2niigui application was used with RadiAnt and MRIcron. AssemblyNet was used for brain measurement and CERES was used for cerebellum measurement by entering the system over the internet. Brain and cerebellum parceling were performed and the volumes of the relevant areas were reported in pdf and cvs formats. Depending on the density of the site, it took an average of 10 minutes to receive all this data for each person's image.

Statistical analysis

The normal distribution of data and the homogeneity of variance were evaluated using the Kolmogorov-Smirnov test and the Levene test, respectively. Because of the anormal distribution in the study groups, Mann-Whitney U-tests were used to compare differences between the groups. Analyses were completed by using the Statistical Package for Social Sciences (SPSS Inc., Chicago, IL) version 26.0 program. The statistical significance for all analyses was set at $p < 0.05$.

RESULTS

The mean age of the NAION and HC groups evaluated in the present study was 62.5 (years). Both groups had nine men and five women. There was no significant difference between the groups in terms of age and gender (p>0.05).

All parameters related to the cerebrum were lower in the NAION group than in the HC group. Cerebrum total, right and left volumes (p values are 0.013, 0.022 and 0.012 respectively), frontal lobe (FL) total, right and left volumes (p values are 0.022, 0.027 and 0.019 respectively), WM total, right and left volumes (p values are 0.006, 0.009 and 0.004 respectively) and GM total, right and left volumes (p values are 0.035, 0.039 and 0.027 respectively) were significantly lower in the NAION group. There was no significant difference between the two groups in the volume of the middle frontal gyrus (MFG) on the right side (p = 0.098). The volumes of certain regions of the occipital lobe were lower in the NAION group compared to the HC group. Occipital lobe (OL) total, right and left volumes (p = 0.002), calcarine cortex (CC) total, right and left volumes (p values are 0.002, 0.008 and < 0.001 respectively), lingual gyrus (LG) total, right and left volumes (p values are 0.001, < 0.001 and 0.007 respectively), middle occipital gyrus (MOG) total and left volumes (p values are 0.018 and 0.031 respectively) and superior occipital gyrus (SOG) total, right and left volumes (p values are 0.029, 0.027 and 0.031 respectively), were significantly lower in the NAION group than in the HC group

There was no significant difference between the two groups in total, right and left cerebellum volumes (p values are 0.215, 0.232 and 0.291 respectively).

There was a significant decrease in right lobule X cortical thickness and left lobule IX volume in the NAION group compared to the HC group (p values are 0.033 and 0.048 respectively). There was no significant difference between the two groups in terms of cortical thickness and volume of other lobules (p > 0.05)

Table 1. Distribution of the cerebrum and its parts volumes control (HC) and nonarteritic anterior ischemic optic neuropathy (NAION) groups.

Volume (cm3)	NAION (n=14) median	(min-max)	HC (n=14) median	(min-max)	p
CerebrumT	957.04	(785.26-1127.1)	1068.02	(935.21-1228.2)	**0.013**
CerebrumR	481.3	(392.45-569.50)	536.46	(466.60-615.48)	**0.022**
CerebrumL	475.75	(392.80-557.50)	532.12	(468.60-612.80)	**0.012**
CWMT	366.70	(283.22-435.62)	426.45	(360.41-483.71)	**0.006**
CWMR	184.38	(141.40-220.20)	213.32	(180.55-242.76)	**0.009**
CWML	182.39	(141.80-215.42)	212.19	(179.86-240.96)	**0.004**
CGMT	586.83	(502.04-704.70)	652.91	(563.20-744.50)	**0.035**
CGMR	294.90	(251.01-355.90)	328.02	(280.20-372.71)	**0.039**
CGML	291.95	(251.03-348.80)	324.89	(282.95-371.84)	**0.027**
FLT	180.76	(152.98-230.50)	202.88	(172.97-239.64)	**0.022**
FLR	90.11	(77.57-115.90)	102.70	(85.73-119.52)	**0.027**
FLL	90.27	(75.41-114.60)	100.70	(87.24-120.12)	**0.019**
MFGT	38.14	(31.83-45.41)	43.19	(36.26-51.87)	**0.039**
MFGR	19.11	(15.00-23.40)	20.19	(18.14-25.27)	0.098
MFGL	19.73	(15.60-22.09)	20.85	(17.90-27.11)	**0.013**

CerebrumT: Cerebrum total, CerebrumR: Cerebrum right, CerebrumL: Cerebrum left, CWMT: Cerebrum WM total, CWMR: Cerebrum WM right, CWML: Cerebrum WM left, CGMT: Cerebrum GM total, CGMR: Cerebrum GM right, CGML: Cerebrum GM left, FLT: Frontal lobe total, FLR: Frontal lobe right, FLL: Frontal lobe left, MFGT: Middle frontal gyrus total, MFGR: Middle frontal gyrus right, MFGL: Middle frontal gyrus left, All comparisons were conducted using the Mann Whitney U test. *p < 0.05

Table 2. Distribution of the occipital lobe and its parts volumes control (HC) and nonarteritic anterior ischemic optic neuropathy (NAION) groups.

Volume (cm3)	NAION (n=14) median	(min-max)	HC (n=14) median	(min-max)	p
OLT	81.83	(67.25-94.01)	94.51	(81.95-108.11)	**0.002**
OLR	41.53	(34.35-47.71)	48.12	(41.80-52.72)	**0.002**
OLL	40.78	(32.91-46.30)	45.99	(40.15-56.10)	**0.002**
CCT	7.04	(5.10-8.30)	8.50	(7.40-11.06)	**0.002**
CCR	3.60	(2.54-4.13)	4.33	(3.71-5.58)	**<0.001**
CCL	3.42	(2.54-4.28)	4.12	(3.07-5.80)	**0.008**
CuneusT	9.15	(7.22-11.97)	10.64	(8.87-12.03)	0.051
CuneusR	4.59	(3.69-6.22)	5.26	(4.64-7.01)	0.054
CuneusL	4.59	(3.53-6.20)	5.22	(3.96-6.26)	0.073
LGT	16.18	(14.16-19.98)	19.34	(17.30-23.04)	**0.001**
LGR	8.33	(6.70-10.89)	10.25	(9.14-11.84)	**<0.001**
LGL	8.32	(5.9-10.14)	9.17	(7.96-11.21)	**0.007**
OFGT	8.92	(6.59-11.96)	9.09	(5.86-13.09)	0.462
OFGR	4.25	(2.25-5.34)	4.19	(3.01-6.36)	0.963
OFGL	4.82	(3.34-6.62)	5.46	(2.85-7.93)	0.26
IOGT	14.30	(11.80-19.57)	16.49	(12.17-19.82)	0.135
IOGR	7.76	(5.17-10.27)	7.79	(5.96-9.97)	0.566
IOGL	6.68	(6.03-9.45)	8.44	(6.01-10.36)	0.098
MOGT	11.07	(7.86-14.10)	12.39	(11.22-15.75)	**0.018**
MOGR	5.89	(4.09-7.75)	6.42	(5.53-8.19)	0.06
MOGL	5.39	(3.69-7.74)	6.28	(4.82-8.18)	**0.031**
SOGT	7.96	(6.10-11.51)	10.28	(6.88-13.22)	**0.029**
SOGR	4.41	(3.38-7.38)	5.54	(4.20-7.93)	**0.027**
SOGL	3.78	(2.32-5.46)	4.53	(2.58-6.19)	**0.031**
OPT	5.76	(3.76-7.22)	5.84	(4.23-8.08)	0.421
OPR	2.65	(1.61-3.63)	3.04	(1.61-4.13)	0.223
OPL	3.07	(2.01-3.88)	2.92	(2.17-3.95)	0.945

OLT: Occipital lobe total, OLR: Occipital lobe right, OLL: Occipital lobe left, CCT: Calcarine cortex total, CCR: Calcarine cortex right, CCL: Calcarine cortex left, CuneusT: Cuneus total, CuneusR: Cuneus right, CuneusL: Cuneus left, LGT: Lingual gyrus total, LGR: Lingual gyrus right, LGL: Lingual gyrus left, OFGT: Occipital fusiform gyrus total, OFGR: Occipital fusiform gyrus right, OFGL: Occipital fusiform gyrus left, IOGT: Inferior occipital gyrus total, IOGR: Inferior occipital gyrus right, IOGL: Inferior occipital gyrus left, MOGT: Middle occipital gyrus total, MOGR: Middle occipital gyrus right, MOGL: Middle occipital gyrus left, SOGT: Superior occipital gyrus total, SOGR: Superior occipital gyrus right, SOGL: Superior occipital gyrus left, OPT: Occipital pole total, OPR: Occipital pole right, OPL: Occipital pole left, All comparisons were conducted using the Mann Whitney U test. *$p < 0.05$

Table 3. Distribution of the cerebellum and its parts volumes control (HC) and nonarteritic anterior ischemic optic neuropathy (NAION) groups.

Parameter	NAION (n=14) median	(min-max)	HC (n=14) median	(min-max)	p
CerebellumT cm3	112.04	(95.44-130.83)	117.04	(101.42-144.05)	0.215
CerebellumR cm3	55.82	(47.51-66.29)	58.97	(51.33-74.43)	0.232
CerebellumL cm3	55.34	(47.93-64.54)	58.07	(50.09-69.62)	0.291
X RCT mm	3.43	(2.85-3.85)	3.83	(3.00-4.26)	**0.033**
IX LGM cm3	2.44	(1.42-3.93)	2.92	(1.87-3.52)	**0.048**

CerebellumT: Cerebellum total, CerebellumR: Cerebellum right, CerebellumL: Cerebellum left, RCT: right cortical thickness, LGM: left grey matter, All comparisons were conducted using the Mann Whitney U test. *$p < 0.05$

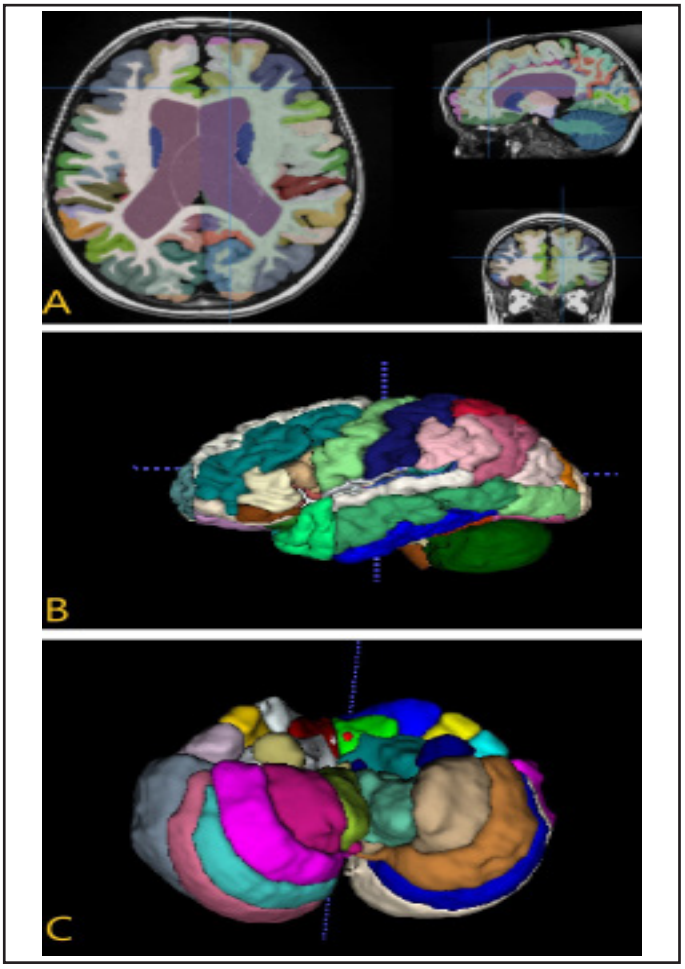

Figure 1. A. Segmentation images created by Volbrain. Brain image of the 63-year-old patient diagnosed with nonarteritic anterior ischemic optic neuropathy
B. 3D segmentation images generated by ITK-SNAP. Brain segmentation of a 63-year-old patient diagnosed with nonarteritic anterior ischemic optic neuropathy
C. 3D segmentation images generated by ITK-SNAP. Cerebellum segmentation of a 63-year-old patient diagnosed with nonarteritic anterior ischemic optic neuropathy

DISCUSSION

Optic nerve disorders can occur as a component of isolated vision loss or a neurological disease. The etiology and clinic of ON types differ from each other. Understanding the cause of the disease provides information about the prognosis of the patient's vision loss. This allows for the prediction of future health risks and additional investigations and treatments (18). Given the impact of optic nerve disorders on neural structures beyond the visual system, recent research has explored whether neuromodulation techniques could influence not only cognitive and perceptual functions but also visual processing and recovery. Neuromodulation is the stimulation and reorganization of nerve cells. Neuromodulation techniques can have modulatory effects on visual perception and memory. This allows the development of visual perception and memory by increasing neural plasticity. Studies on the relationship between neural stimulation techniques and cognitive and visual processes have shown that significant changes can occur in functions such as memory and perception (19, 20). These findings suggest that optic nerve disorders may not be limited to vision alone but could also be associated with broader neurological effects.

In this study, we compared the brain parts of healthy individuals and NAION patients using the VolBrain automatic segmentation method. We used the VolBrain method because it provides detailed brain morphology and volume analysis. In optic nerve lesion, significant atrophy is observed in both the nerve and the WM volume in the visual pathways which are the continuation of optic the nerve. This atrophy in the visual pathways negatively affects the projection of stimuli to the visual cortex and causes a decrease in the visual cortical volume (7). Co-modeling of neuroanatomical connections between the eye and the cortical visual center in visual impairment indicates changes consistent with transneuronal degeneration. Transneuronal degeneration is the process by which damage to one neuron leads to the degeneration of other neurons connected to that neuron. Retinal problems and lesions in the optic nerve and the pathways in its continuation can spread through transneuronal degeneration and affect different parts of the brain (21). In the present study, we evaluated the changes in cortical visual centers and cerebellum volume due to NAION.

In a study, it was reported that optic neuritis caused significant atrophy in total brain and WM volumes in the right and left hemispheres. No difference was observed in GM volumes (14). The lack of a significant decrease in gray matter volume may be due to the duration of optic neuritis. Various studies have reported that complete vision loss led to a significant level of atrophy in the GM volume of the brain compared to healthy individuals (7, 13). Some of the corticocortical pathways that connect the lobes in the brain hemispheres extend from the FL to the parietal lobe and OL at the back, and some from the temporal lobe to the OL. In individuals with vision loss, besides these pathways, atrophy is also observed in the corticospinal, thalamic and cerebellar pathways. This shows that in vision loss not only the OL but also other parts of the brain that connect with it are affected in terms of volume. It was reported that atrophy in the connective pathways in WM is responsible for volumetric changes in brain lobes (16). Similarly, in the present study, significant atrophy was observed in the total, WM and GM volumes of the brain.

The FL is the center of motor control. It is also responsible for functions such as the control of emotions, reasoning and planning. The anterior visual center and frontal visual area (FVA) in the MFG are connected to the OL. It takes part in controlling eye movements and visual reflexes. A significant decrease was reported in the GM volume of the bilateral FVA and also in the WM volume of the left FVA in individuals with optic neuritis (21). In another study, the effect of optic nerve atrophy on brain volumes was investigated in patients with glaucoma and a

significant decrease in FVA volume was reported. This was thought to be due to a decrease in volume in the WM pathways that provide the connection between the occipital cortex and the MFG (16). Decreased communication between the visual cortex and the FL may cause the FL to be adversely affected in terms of volume (22). It was revealed that increased intraocular pressure in patients with glaucoma causes atrophy of the optic nerve. In addition, it was reported that the volume of the visual cortex and the volume of the associated FVA decreased (15). In the present study, significant decreases in total and left hemisphere volumes were found in the FVA. Due to the high use of right hands and right eye dominance in society, left hemisphere dominance of the brain is more common. We are of the opinion that the effect on the left hemisphere volume is due to this (23). In NAION, patients' motor activities and perception of stimuli are affected. Accordingly, we believe that there could be volumetric changes in the relevant regions of the brain (24).

A literature survey indicates that conditions such as optic neuritis, dysthyroid ON, glaucoma and macular degeneration which cause vision problems lead to atrophy in the visual cortex volume (14, 16, 24, 25). This is due to a decrease in the stimuli projected to the visual cortex. Optic nerve deformation causes anterograde (retina to cortex) and retrograde (cortex to retina) trans-synaptic functional changes. These changes may cause impairment in the transmission the visual stimuli through the optic nerve and visual pathways and in establishment of the connection with the visual cortex (14). In a study investigating the visual cortex in individuals with congenital visual loss, significant atrophy was found both in Brodmann 17 and 18 areas and in the afferent fibers that convey the stimulus (25). Atrophy due to axonal degeneration in the optic nerve lesion negatively affects the WM volume in the optic nerve and subsequent visual pathways as well as the projection of stimuli to the visual cortex. This causes a decrease in the volume of the visual cortex (7).

Hanson et al. conducted a study on individuals with unilateral macular degeneration. They reported that the cortical thickness of the occipital polus and calcarine sulcus and the volume of the calcarine gyrus decreased when visual stimuli were not perceived (25). In the present study, similar to the literature, significant atrophy was detected in the OL volumes where the visual centers are located in individuals with NAION. NAION causes damage to the optic nerve and visual pathways. We are of the opinion that the lack of perception of visual stimuli leads to a decrease in WM and GM volumes, resulting in the impairment of visual stimuli projection into the visual cortex.

There are studies in the literature reporting that despite vision loss, atrophy does not occur in the brain and even there is an increase in connections. It was found that in hamsters with vision loss but normal hearing function, the auditory cortex was developed and more connections were established with the visual cortex (26). In their study on individuals with congenital vision loss, Ptito et al. found an increase in the volume of the auditory center and somatosensory center, and a thinning of the cortical thickness of both centers (26). This can be explained by the increase in sensitivity to auditory and tactile stimuli due to the effect of compensatory plasticity in the brain in visual perception deficiency (13). It is thought that a connection between the visual cortex and other sensory centers develops with cross-model plasticity (27). In another study conducted with individuals with complete vision loss, a significant increase was shown in the volume of the left FVA compared to the healthy control group (12). It was hypothesized to be related to compensatory reactions in the absence of visual stimuli. The individuals with vision loss have more physical contact, so tactile stimuli also develop. In addition, fine motor abilities are strengthened when using the Braille alphabet. As a result, it is predicted that there may be an increase in volume in the FVA due to the increase in the number, volume and myelination of axons (28). Non-visual sensory modalities such as touch and hearing are being used more to obtain information about the shape, movement and localization of objects. As a result, it was reported that supramodal brain areas are affected, thereby leading to a volumetric increase in the premotor and primary motor area (29).

The absence of visual input in congenital blindness leads to atrophy of various components of the afferent and efferent visual pathways. Despite its reduced volume, the visual cortex shows functional activity when measured by various neuroimaging techniques, suggesting a possible contribution of thalamocortical (30) and corticocortical (31) connections in cross-modal plasticity in congenital blindness. These pathways contribute to neural plasticity by enabling the formation of new areas of connection to the cortex.

The cerebellum is linked to higher-order functions such as balance, coordination, motor control, complex motor learning, cognitive functions and behavior (32, 33). Since the I-VI and VIII lobules of the cerebellum communicate with the sensorimotor areas in the brain, these parts are the sensorimotor areas of the cerebellum (34). Lobules VI, VII, Crus I–II, VIII and IX of the cerebellum were identified as cognitive supramodal regions. These areas play an important role in the planning coordinating of movement (35). The flocculonodular lobe, formed by lobule X, is the vestibular component of the cerebellum. It is the area where stimuli from the inner ear connect. It is important for the control of the body according to the position of the head and neck (36). In a study evaluating individuals with optic neuritis, a significant volume increase was found in the lobules of the cerebellum related to the plan and coordination of movement, and a significant volume decrease in the sensorimotor areas (14). Another study in individuals with monocular vision loss found a similar increase in volume in the cerebellum (37). Despite the

the inability to receive visual stimuli, the volume increases in Crus II, lobule VIIB and lobule VIIIA of the cerebellum are estimated to be due to compensatory reactions occurring in the cerebellum (14). In the present study, unlike the literature, there was no significant difference in the volume of many regions of the cerebellum. We are of the opinion that the lack of significant change in cerebellum volume in NAION may be related to the duration of the disease, and that compensatory reactions may not have developed yet.

The present study had some limitations. The most important one is that the number of cases was limited. In addition, the onset and prognosis of NAION could not be determined because we benefited from MR images taken in the last five years due to the archiving system. Lifestyle factors such as the presence of chronic diseases such as diabetes mellitus and hypertension, medication use, physical activity and smoking, which may affect brain volume, could not be determined. In addition, the lack of data on patients limits the assessment of the progression of volumetric changes over time.

CONCLUSION

In conclusion, our study investigating the effect of NAION on brain volumes revealed that cortical visual centers as well as the FL, which is the control center of motor function, was affected volumetrically in optic nerve damage, but there was no significant volumetric change in the cerebellum.

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