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Research Article

Investigation of Cortical and Subcortical Brain Regional Volumes in Children with Autism Spectrum Disorder Using Automated Segmentation

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Abstract

Aim: This study aimed to examine regional cortical and subcortical brain volume differences in children with autism spectrum disorder (ASD). By comparing the ASD group with healthy individuals, the effects of the disorder on brain morphology will be evaluated. We hypothesized that children with ASD would exhibit increased volumes in selected cortical and subcortical brain regions.

Material and Methods: The brain magnetic resonance images (MRI) of 33 children with ASD and 33 healthy controls were analyzed and compared. The VolBrain automated segmentation method was used to process the MRI and detect volumetric differences. The resulting measurements were statistically analyzed to identify significant volumetric differences between the ASD and control groups.

Results: The findings of this study revealed that children with ASD exhibited significantly larger volumes in the cerebrum, white matter, grey matter, frontal lobe, postcentral gyrus, amygdala, thalamus, putamen, and hippocampus compared to healthy controls ($p < 0.05$). However, no significant differences were observed between the groups in the volumes of the precentral gyrus, parietal lobe, and caudate nucleus ($p > 0.05$).

Conclusion: The present findings indicate that autism spectrum disorder (ASD) is associated with widespread volumetric enlargement across both cortical and subcortical brain regions. These results contribute to a clearer characterization of the neuroanatomical profile of ASD and underscore the value of automated volumetric neuroimaging approaches for detecting disorder-related structural brain patterns.

Keywords: Autism Spectrum Disorder, VolBrain, Cerebrum, White matter, Grey matter

INTRODUCTION

Autism Spectrum Disorder (ASD) is a lifelong neurodevelopmental disorder that originates in early childhood. It is primarily marked by persistent impairments in social communication and interaction, accompanied by restricted, repetitive patterns of behavior, activities, or interests (1). The heterogeneity of symptoms in ASD reflects considerable variation among individuals in terms of clinical features, developmental trajectories, and treatment responses (2). Although the exact etiology of ASD remains unclear, it is widely believed to result from a complex interplay of genetic predisposition, prenatal and perinatal factors, and environmental influences (3). In recent years, the prevalence of ASD has increased markedly, with current estimates indicating that approximately one in every 54 children is diagnosed with the disorder (4). With the rapid and progressive nature of ASD, neuroimaging studies aimed at exploring potential structural neuroanatomical alterations have gained significant momentum (5). Magnetic Resonance Imaging (MRI), as a non-invasive and high-resolution imaging modality, has demonstrated strong potential in detecting clinically meaningful variations and supporting the diagnostic process (6). MRI studies suggest that individuals with ASD may exhibit differences in total brain volume, cortical thickness, and the size of subcortical structures compared to neurotypical individuals (7, 8). In their neuroimaging study on ASD, Riddle et al. reported that volumetric analyses often

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focused either on total brain volume or on a limited number of cortical and subcortical regions (9). Given the complex neurobiological profile of ASD, there remains a need for more comprehensive and systematic volumetric analyses encompassing multiple cortical and subcortical regions within the same cohort. Such an approach may help clarify whether volumetric differences in ASD are localized or distributed across several interconnected brain structures. Furthermore, the use of automated segmentation techniques offers improved reproducibility, objectivity, and methodological consistency, thereby strengthening the reliability of volumetric neuroimaging research. In line with this, the present study aims to contribute to the existing body of knowledge by conducting an extensive volumetric analysis focused on the cerebrum, white matter, grey matter, frontal lobe, precentral gyrus, postcentral gyrus, amygdala, caudate nucleus, thalamus, putamen, and hippocampus in individuals with ASD.

MATERIAL AND METHODS

This research was conducted as a retrospective, cross-sectional, and observational study. All methodological procedures adhered to the ethical standards outlined in the Declaration of Helsinki. Ethical clearance was obtained from the Non-Interventional Clinical Research Ethics Committee of Tokat Gaziosmanpaşa University (E-15235480-050.04-549373).

The study included children diagnosed with ASD and a control group composed of healthy children with similar age and sex characteristics. According to a G*Power analysis, a total of 40 participants 20 in each group was determined to be sufficient based on a 95% confidence level (1-α), 95% statistical power (1-β), an effect size of d = 1.2, and a two-tailed hypothesis (10). Archival medical records of 55 children diagnosed with ASD between 2019 and 2024 were reviewed. Based on image quality and data completeness, high-resolution brain MRI scans from 33 children were included in the ASD group. The control group consisted of brain MRI scans from 33 children who underwent imaging during the same period due to non-specific complaints such as headache or dizziness, but who had no diagnosed neurological or systemic disorders. Control participants were matched to the ASD group in terms of age and sex. Because the study was retrospective and based on archival MRI data, height and weight measurements were not consistently available and therefore could not be included in the analysis.

ASD diagnoses were established by a child and adolescent psychiatrist according to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) criteria. Inclusion criteria for the ASD group were: a confirmed clinical diagnosis of ASD and availability of high-quality T1-weighted MRI data. Exclusion criteria included a history of neurological disorders other than ASD, genetic syndromes, major systemic diseases, or structural brain abnormalities. For the control group, inclusion criteria were: absence of any neurodevelopmental, neurological, or psychiatric diagnosis, MRI performed for non-neurological complaints such as headache or dizziness, and normal MRI findings confirmed by a radiologist. Children with incidental structural abnormalities or chronic systemic diseases were excluded.

Brain imaging data used in this study were obtained from the SECTRA archive system of Tokat Gaziosmanpasa University Faculty of Medicine Hospital. The brain images of the participants were acquired using a 3 Tesla MRI scanner (Philips Medical Systems 3.0 Tesla Gyroscan NT) and were based on T1-weighted, three-dimensional volumetric Turbo Field Echo (TFE) sequences. These high-resolution structural images were selected to provide the anatomical detail necessary for accurate volumetric analysis. All MRI data were anonymized prior to analysis, and participants' personal information was protected in accordance with confidentiality protocols. High-resolution anatomical images were obtained in the sagittal plane using a 1 mm^3 isotropic voxel dimension. The acquisition protocol included the following parameters: repetition time (TR) of 8.1 ms, echo time (TE) of 3.7 ms, turbo field echo (TFE) duration of 230 ms, a flip angle of 8°, a matrix resolution of 224 × 224 pixels, and a field of view (FOV) measuring 224 × 224 mm.

To enable objective, rapid, and reproducible calculation of brain volumes, this study employed the VolBrain system (version 1.0; https://volbrain.net/auth). The standard volBrain "HIPS" and "CERES" automated segmentation pipelines were used to obtain global, cortical, and subcortical volumetric measurements. The segmentation process was based on a multi-atlas, patch-based label fusion algorithm, which integrates multiple reference atlases and adapts them to the target image for accurate anatomical labeling (11). The following regions of interest (ROIs) were extracted: total cerebrum, right and left cerebrum, total/right/left cerebral white matter, total/right/left cerebral grey matter, frontal lobe, precentral gyrus, postcentral gyrus, parietal lobe, amygdala, caudate nucleus, thalamus, putamen, and hippocampus. ROI definitions were based on the standardized anatomical parcellation provided by the volBrain atlas framework, ensuring comparability across subjects.

Statistical Analysis

All statistical procedures were conducted using IBM SPSS Statistics software, version 25.0. Prior to data analysis, skewness and kurtosis values were examined to assess the assumption of normality. As the data were found to be normally distributed, it was deemed appropriate to use parametric tests. To determine whether the variables differed based on health status, independent samples t-tests were conducted. All statistical outcomes were interpreted using a 95% confidence interval, with a significance threshold set at $p < 0.05$.

RESULTS

The mean age of children with ASD and healthy controls participating in the study was calculated as 6.93 years. Both groups consisted of 20 boys and 13 girls. No statistically significant differences were found between the groups in terms of age and sex variables ($p > 0.05$).

In children diagnosed with ASD, the total, right, and left hemisphere volumes of the cerebrum (p = 0.01, 0.01, and 0.01, respectively), the total, right, and left volumes of the cerebral white matter (white matter) (p = 0.02, 0.01, and 0.03, respectively), and the total, right, and left volumes of the cerebral grey matter (grey matter) (p = 0.01, 0.01, and 0.01, respectively) were found to be significantly higher compared to healthy children.

Table I. In children diagnosed with ASD, the total, right, and left volumes of the frontal lobe (p = 0.01, 0.01, and 0.02, respectively), as well as the total, right, and left volumes of the postcentral gyrus (p = 0.02, 0.02, and 0.04, respectively), were found to be significantly greater compared to healthy controls. However, no statistically significant differences were observed between the groups in the total, right, and left volumes of the parietal lobe (p = 0.06, 0.06, and 0.08, respectively) and the precentral gyrus (p = 0.15, 0.17, and 0.22, respectively)

Table II. When the volumes of subcortical structures were examined, children with ASD exhibited significantly larger total, right, and left volumes of the amygdala (p = 0.02, 0.02, and 0.02, respectively), thalamus (p = 0.01, 0.01, and 0.02, respectively), putamen (p = 0.01, < 0.001, and 0.02, respectively), and hippocampus (p = 0.01, < 0.001, and 0.01, respectively) compared to healthy controls. However, no statistically significant differences were found between the two groups in the total, right, and left volumes of the caudate nucleus (p = 0.07, 0.08, and 0.07, respectively) Table III.

Table I. Comparison of Cerebral Volumes Between ASD and Control Groups

Volume (cm³)	ASD		CONTROL		t	p
	Mean	SD	Mean	SD		
Cerebrum total	1102.49	143.0	1010.49	135.13	2.686	0.01
Cerebrum right	553.68	72.82	504.41	66.67	2.867	0.01
Cerebrum left	548.76	70.24	505.5	69.43	2.516	0.01
Cerebrum white matter total	396.1	63.13	361.47	51.23	2.447	0.02
Cerebrum white matter right	198.73	32.17	179.98	26.24	2.595	0.01
Cerebrum white matter left	197.35	31.02	181.45	25.9	2.26	0.03
Cerebrum grey matter total	706.34	81.12	648.98	89.21	2.733	0.01
Cerebrum grey matter right	354.93	41.33	324.93	43.58	2.869	0.01
Cerebrum grey matter left	351.36	39.87	324.0	46.25	2.573	0.01

ASD = Autism Spectrum Disorder group; Control = Neurotypical control group.
All comparisons were conducted using independent samples t-tests. A p-value < 0.05 was considered statistically significant.

Table II. Comparison of Frontal and Parietal Lobe Volumes Between ASD and Control Groups

Volume (cm³)	ASD		CONTROL		t	p
	Mean	SD	Mean	SD		
Frontal lobe total	218.29	26.07	198.51	35.68	2.572	0.01
Frontal lobe right	109.75	13.13	98.77	18.76	2.755	0.01
Frontal lobe left	108.5	13.07	99.68	17.39	2.33	0.02
Precentral gyrus total	29.03	3.69	27.44	5.15	1.444	0.15
Precentral gyrus right	14.56	2.12	13.67	2.98	1.393	0.17
Precentral gyrus left	14.38	1.79	13.74	2,36	1.246	0.22
Parietal lobe total	134.22	14.76	126.44	17.7	1.939	0.06
Parietal lobe right	68.0	8.24	63.85	9.25	1.925	0.06
Parietal lobe left	66.16	6.78	62.55	9.27	1.805	0.08
Postcentral gyrus total	26.24	2.77	24.22	4.08	2.35	0.02
Postcentral gyrus right	12.66	1.54	11.56	2.14	2.388	0.02
Postcentral gyrus left	13.53	1.42	12.63	2.06	2.067	0.04

ASD = Autism Spectrum Disorder group; Control = Neurotypical control group.
All comparisons were conducted using independent samples t-tests. A p-value < 0.05 was considered statistically significant.

Table III: Comparison of Subcortical Structure Volumes Between Groups

Volume (cm³)	ASD		CONTROL		t	p
	Mean	SD	Mean	SD		
Amygdala total	1.88	0.31	1.63	0.52	2.366	0.02
Amygdala right	0.96	0.17	0.83	0.27	2.362	0.02
Amygdala left	0.92	0.15	0.8	0.25	2.338	0.02
Caudate nucleus Total	6.09	0.87	5.49	1.67	1.853	0.07
Caudate nucleus Right	3.06	0.46	2.75	0.89	1.797	0.08
Caudate nucleus Left	3.03	0.42	2.74	0.8	1.874	0.07
Thalamus Total	14.87	1.87	13.58	2.05	2.669	0.01
Thalamus Right	7.44	0.93	6.72	1.05	2.94	0.01
Thalamus Left	7.43	0.94	6.86	1.06	2.311	0.02
Putamen Total	8.92	1.29	7.83	1.76	2.867	0.01
Putamen Right	4.46	0.66	3.85	0.89	3.189	0.002
Putamen Left	4.45	0.64	3.98	0.92	2.447	0.02
Hippocampus Total	6.67	1.19	5.73	1.4	2.939	0.01
Hippocampus Right	3.38	0.61	2.91	0.62	3.085	0.002
Hippocampus Left	3.28	0.59	2.81	0.83	2.648	0.01

ASD = Autism Spectrum Disorder group; Control = Neurotypical control group.
All comparisons were conducted using independent samples t-tests. A p-value < 0.05 was considered statistically significant.

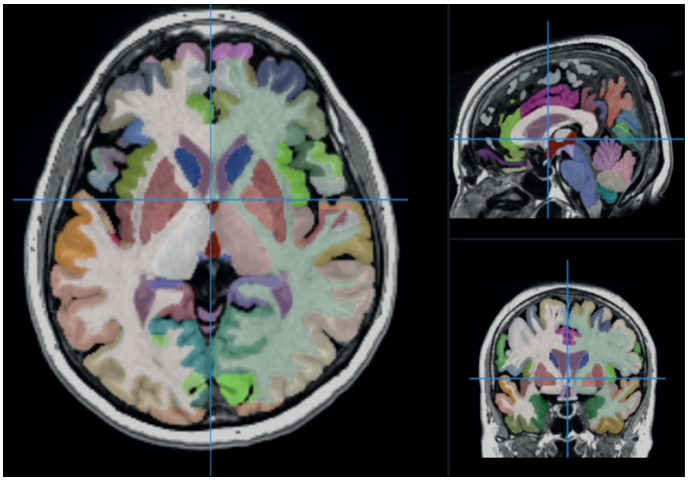

Figure I. Cerebrum image of a 9-year-old child with ASD analyzed by VolBrain.

DISCUSSION

This study distinguishes itself from previous research in the literature by providing a volumetric analysis of a wide range of cortical and subcortical brain regions in children diagnosed with Autism Spectrum Disorder (ASD). In this context, the comprehensive evaluation of the cerebrum, cerebral white matter (white matter), cerebral grey matter (grey matter), frontal lobe, precentral gyrus, postcentral gyrus, amygdala, caudate nucleus, thalamus, putamen, and hippocampus offers a valuable contribution to the current body of knowledge.

Cerebral volume enlargement in individuals with ASD is considered an early indicator of neurodevelopmental deviations. Courchesne et al., in a study comparing a total of 586 individuals aged 1 to 50 years with and without ASD, reported that during childhood, cerebrum volume particularly in the frontal and temporal lobes was significantly greater in children with ASD than in typically developing peers (12). Similarly, Hazlett et al., in a longitudinal study following 106 high-risk infants and 42 low-risk infants between 6 and 24 months of age, found that those with early cerebral overgrowth were more likely to receive an ASD diagnosis later in development (13). In line with these findings, the increased cerebrum volume observed in our ASD group may reflect early neurodevelopmental changes that impact brain structure. In the literature, such volumetric increases have been suggested to indicate cerebral hyperplasia and may represent a potential biomarker specific to ASD (12, 13). This enlargement is thought to be associated with atypical developmental trajectories in processes such as neuronal proliferation, synaptogenesis, and myelination (14). On the other hand, some studies have reported inconsistent findings, suggesting that the absence of significant volumetric differences may be due to variability in sample age, ASD subtypes, imaging protocols, or analysis methods (8, 15).

In individuals diagnosed with ASD, increased volume of cerebral white matter , particularly during childhood, has been frequently reported (12). This increase is suggested to be concentrated along the projection pathways between the frontal and occipital lobes, potentially indicating atypical neuronal transmission and excessive connectivity (16). Moreover, it has been reported that this white matter enlargement may decrease over time and normalize with age (17). With regard to cerebral grey matter, studies have identified volume increases especially in the frontal, temporal, and parietal cortical regions in individuals with ASD. These differences have been associated with cognitive functions commonly affected in ASD, such as social cognition, attention, language, and executive functioning (18, 19). In line with this literature, the significantly greater white and grey matter volumes observed in the ASD group in our study may be interpreted as findings consistent with the neurodevelopmental basis of ASD.

In the literature, several studies have highlighted increased frontal lobe volume in individuals with ASD. Carper et al. and Hazlett et al. reported significantly greater frontal lobe volumes in children with ASD compared to control groups. This volumetric enlargement has been suggested to be associated with core ASD symptoms such as stereotyped movements, lack of inner speech, deficits in social communication, and impairments in cognitive functioning (13, 20). However, it has also been emphasized that findings regarding frontal lobe volume may vary with age. These differences are particularly pronounced in early childhood but tend to diminish during adolescence and adulthood (8, 12). Mahajan et al., in a study involving 30 children with ASD, 33 with ASD and comorbid Attention-Deficit/Hyperactivity Disorder (ADHD), and 63 healthy controls, reported increased precentral gyrus volume in the ASD group in both structural and functional terms. The researchers suggested that this increase may be related to hyperactivity specific to the motor cortex, stereotyped behaviors, and deficits in bodily awareness (21). However, findings regarding the precentral gyrus remain inconsistent in the literature, and it has been noted that factors such as sample age, imaging techniques, segmentation methods, and ASD subtypes may significantly influence outcomes (8, 19). In our study, although the precentral gyrus volume was greater in the ASD group, this difference did not reach statistical significance.

Findings related to parietal lobe volume in ASD show considerable variability in the literature. Brieber et al., in their study involving 30 children and adolescents with ASD, 33 with comorbid ASD and ADHD, and 63 typically developing individuals, reported significantly increased grey matter volume in the left inferior parietal cortex and right supramarginal gyrus. They suggested that these volumetric increases may be associated with impairments in attention and social cognition (22). Although the volumetric measurements revealed a greater parietal lobe volume in individuals with ASD compared to healthy controls, this difference did not attain statistical significance. This may suggest that the parietal lobe is less structurally affected in ASD, or that volumetric analyses may be limited in detecting subtle regional alterations. These findings may also reflect the morphological heterogeneity and inter-individual variability commonly observed in ASD. Studies on the postcentral gyrus suggest that this region displays atypical features not only functionally but also structurally (21, 23). Wang et al., in their study comparing data from 31 boys with ASD and 33 typically developing boys aged 3–7 years, reported a significant increase in grey matter volume in the left postcentral gyrus. The authors suggested that this volumetric increase was linked to the severity of impairments in domains such as social reciprocity, verbal and non-verbal communication, and self-care functionality. (23) Moreover, volumetric enlargement in this region was found to be linked to altered functional connectivity patterns with other brain areas, and these patterns were significantly correlated with the severity of autism symptoms. These findings suggest that ASD is not limited to deficits in social and communicative functions, but rather represents a pervasive neurodevelopmental disorder that also affects sensory perception and processing mechanisms.

In our study, significant volumetric increases were identified in subcortical structures such as the amygdala, thalamus, putamen, and hippocampus in children with autism. In the literature, enlargement of the amygdala has been associated with impairments in social cognition and emotional processing, as well as with symptoms of social withdrawal and anxiety (24, 25). Increased thalamic volume is suggested to reflect developmental alterations in sensory-motor integration and attentional processes (21, 26). The enlargement of the putamen has been linked to motor coordination impairments, stereotyped behaviors, and repetitive movement patterns (7, 27). Additionally, it has been suggested that the increased hippocampal volume observed in individuals with ASD may be associated with impairments in social cognition and adaptive functioning (28). In contrast to certain previous findings, our analysis revealed no statistically significant difference in caudate nucleus volume a key subcortical region between individuals with ASD and healthy controls. Similar findings have also been reported in some previous studies involving individuals with ASD (29, 30).

Although our findings align with evidence of widespread neurodevelopmental alterations in ASD, their functional implications remain indirect, as behavioral measures, symptom severity, and functional connectivity were not assessed; therefore, associations with specific cognitive or clinical domains should be interpreted cautiously.

From a methodological perspective, a key strength of this study is the simultaneous evaluation of multiple cortical and subcortical regions using an automated, atlas-based segmentation approach, which enhances reproducibility and reduces operator-dependent variability. The use of standardized MRI acquisition parameters and a matched control group also supports the internal consistency of the findings. However, these strengths should be considered alongside methodological limitations, including the retrospective design, relatively modest sample size, and the absence of intracranial volume normalization and anthropometric covariates, which may influence volumetric comparisons.

This study has several limitations. First, its cross-sectional design does not allow for the evaluation of developmental trajectories in brain structure volumes over time. Consequently, longitudinal studies are needed to better clarify how volumetric differences relate to neurodevelopmental processes in ASD. Second, the sample was derived from a single center, and no stratification was performed according to ASD

subtypes, symptom severity, or comorbid clinical conditions. This may restrict the generalizability of the findings across the broader autism spectrum. In addition, the study focused exclusively on structural volumetric data and did not incorporate functional or connectivity-based neuroimaging measures. Future research using multimodal imaging approaches, including functional MRI and connectivity analyses, would provide a more comprehensive understanding of ASD-related brain alterations. Another important limitation is the absence of anthropometric measures such as body weight and height. Variations in nutritional status and overall body size both of which may plausibly differ in children with ASD could represent potential confounding factors in regional brain volume comparisons. Without accounting for these variables, it is difficult to fully distinguish disorder-related neuroanatomical differences from global body-size effects.

CONCLUSION

Our findings support our initial hypothesis that children with ASD exhibit widespread volumetric alterations across both cortical and subcortical brain regions. These results indicate that ASD is not limited to impairments in social communication and behavior, but rather reflects a broad neurodevelopmental condition involving multiple neurocognitive systems, including motor control, sensory processing, memory, and emotional regulation. By simultaneously evaluating a wide range of brain structures using an automated and standardized segmentation approach, this study extends previous research that has often focused on a limited number of regions, thereby contributing to a more comprehensive understanding of the neuroanatomical profile of ASD.

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Conflict of Interest

The authors declare that they have no conflict of interest.

Author Contributions

ATU: Protocol/project development, Data collection or management, Manuscript writing/editing; HIS: Data analysis, Manuscript writing/editing; SG: Data analysis, Manuscript writing/editing; FK: Data analysis, Manuscript writing/editing

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Ethical approval

All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Approval for the study was Tokat Gaziosmanpaşa Faculty of Medicine Clinical Research Ethics Committee (E-15235480-050.04-549373).

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