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Original Article

Comparing thalamic volumes between focal cortical dysplasia patients and healthy individuals

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Abstract: Background: Focal cortical dysplasia (FCD) is a congenital deformity caused by FCD maturation, differentiation, and neuronal migration. Magnetic resonance imaging (MRI) is one of the most popular and consistent procedures for diagnosing FCD. Limited research has evaluated the relationship between the FCD and thalamic volume. Therefore, we conducted the current study to compare thalamic volumes between patients with FCD and healthy individuals. Methods: The current study was a cross-sectional study of patients with FCD referred to Kashani and Milad Hospitals in Isfahan City in 2019-2021. All patients who met the inclusion criteria were enrolled in the study using the census method. The study population was divided into two groups: patients with FCD and healthy controls. MRI was performed on patients with FCD using a Siemens 1.5 or 3 Tesla MRI device. The data were analyzed using SPSS Statistics for Windows (IBM SPSS Statistics for Windows, Version 18.0). Results: Among the 60 patients, 30 had FCD with a mean age of 22.6 ± 9.3 years, and 30 were healthy with a mean age of 26.3 ± 3.6 years. The only significant difference observed was between the right and left thalamic volumes in the FCD group (P = 0.042). The thalamus on the involved side was significantly smaller than that on the non-involved side in patients (*P*-value < 0.001). However, no significant differences were observed in the absolute value of the difference between the left and right thalamic volumes when comparing all patients with FCD to those in the non-FCD group (P = 0.054). Conclusion: Our study showed that in patients with FCD, the thalamus on the involved side was significantly smaller than that on the noninvolved side.

Keywords: Focal cortical dysplasia, magnetic resonance imaging, thalamic volume

Introduction

Focal cortical dysplasia (FCD) is a congenital deformity hypothesized to be caused by abnormal maturation, differentiation, and neuronal migration. However, the specific cause remains unknown [1]. The disorder is characterized by atypical neuronal aggregations mislocalizing in the cortical or subcortical regions [2]. FCD is distinguished by organizational anomalies, such as misaligned neurons compared to the typical radial orientation, and aberrant structural properties of neurons, including a significant decrease in myelin content [3]. It is generally acknowledged that FCD is the leading cause of drug-resistant epilepsy among pediatric populations [4].

The diagnostic standards for FCD primarily rely on a combination of clinical, imaging, and histopathological findings. Magnetic resonance imaging (MRI) is the gold standard imaging modality for diagnosis, often revealing characteristic features, including cortical thickening, blurring of the gray-white matter junction, increased T2/FLAIR signal abnormalities, transmantle sign, and abnormal gyral or sulcal patterns [5]. However, not all FCD cases are visible on MRI, particularly type I lesions, making diagnosis challenging in some patients [6]. The histopathological classification by the International League Against Epilepsy (ILAE) remains the definitive diagnostic standard, encompassing subtypes based on cortical dyslamination and neuronal abnormalities [7].

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Clinically, FCD typically presents with drug-resistant focal epilepsy, often with onset in childhood or adolescence. Seizures may manifest as focal with or without impaired awareness, and sometimes progress to generalized tonic-clonic seizures. Additional clinical features can include cognitive impairment, intellectual disabilities, focal neurological deficits, and less commonly, headaches or movement disorders. Unlike other neurological conditions, there are no specific laboratory findings diagnostic for FCD since it represents a structural brain malformation. However, neurophysiological studies such as electroencephalography (EEG) and advanced imaging techniques like positron emission tomography (PET) are critical in identifying the epileptogenic zone.

Current treatment strategies for FCD center around surgical resection of the dysplastic cortex, which offers the best opportunity for seizure control or freedom, particularly in patients with drug-resistant epilepsy. Surgical outcomes are generally favorable, with approximately 50-75% of patients achieving seizure freedom after complete lesion resection. Alternative treatment approaches include antiepileptic drugs (AEDs), although these often demonstrate limited efficacy in FCD-related epilepsy. Emerging therapeutic options include mammalian target of rapamycin (mTOR) inhibitors, ketogenic diets, and neurostimulation techniques such as vagus nerve stimulation (VNS).

The prognosis of patients with FCD varies significantly based on several factors. Early surgical intervention tends to improve seizure outcomes and cognitive prognosis. Favorable prognostic indicators include older age at seizure onset and clear lesion visibility on MRI. Conversely, MRI-negative lesions and younger age at seizure onset predicted poorer outcomes. Long-term seizure freedom is achievable in over half of patients following surgical intervention, and cognitive function may improve with early post-operative seizure control.

FCD has been demonstrated to impact thalamic development, specifically in volume and structural connections. According to research, the thalamic volume in the hemisphere affected by FCD is significantly reduced compared to the contralateral hemisphere [8, 9]. Furthermore, these patients' thalamus and thalamocortical pathways had higher odds of having high apparent diffusion coefficient (ADC) values, which may indicate changes in the microstructural integrity of these regions [10].

The purpose of surgical treatment in patients with FCD is primarily directed towards managing epilepsy rather than addressing thalamic volume changes. However, understanding thalamic involvement may provide insights into the broader network effects of FCD [11]. Structural MRI studies have been employed to quantify anomalies in thalamic volume and thalamocortical circuits in patients with FCD [12]. Additional investigations have explored the coordination of cortical and thalamic activity during sleep-related epilepsy induced by FCD type II, revealing positive correlations between FCD volume and both thalamic and lesion regions [13].

These findings suggest a potential relationship between FCD, thalamic volume alterations, and the manifestations of epilepsy. However, few studies have comprehensively evaluated the relationship between FCD and thalamic volume changes in patients and healthy individuals. Therefore, we conducted the current study to compare thalamic volume between patients with FCD and healthy individuals, aiming to better understand the extent of structural changes beyond the primary cortical lesion.

Material and methods

Study design

The current study was a cross-sectional study conducted on patients with FCD who were referred to Kashani and Milad Hospitals in Isfahan city between 2019-2021. The study protocol was approved by the Isfahan University of Medical Sciences Research Committee and certified by the Ethics Committee (IR.MUI.MED.REC.1401.366).

Inclusion and exclusion criteria

The inclusion criteria for patients included in the study were patients with FCD who provided informed and written consent. The diagnosis of FCD was made using MRI and the ILAE classification [14] and was confirmed by a pathologist after pathological examination. The control group included other participants who underwent brain MRI for various reasons but did not have FCD. The exclusion criteria were patients

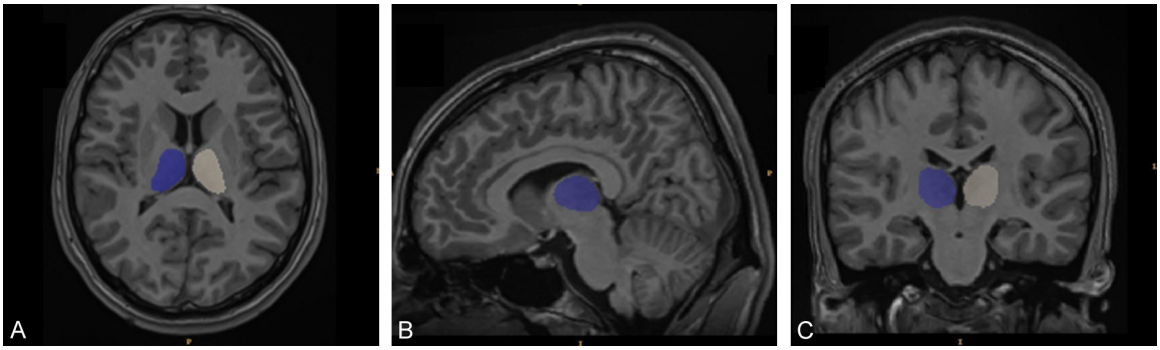

Figure 1. One example of manual lesion labeling using the ITK-SNAP software. Lesion volume was calculated based on the 3D T1 volumetric sequence. A. Axial view; B. Coronal view; C. Sagittal view.

who underwent functional hemispherectomy or corpus callosotomy, and unwillingness to participate in the study.

Study population

All patients that met the inclusion criteria entered the study using census method. MRI were performed for all patients. Then, all participants were divided into case and control groups based on the inclusion criteria. Demographic data of patients including age, gender, and FCD side were obtained. In this study, the evaluation of thalamic volume was investigated in a comparative way. Therefore, as the number of patients included in the study increased, we included MRIs of healthy people with no history of brain disease as a control group.

MRI assessment

The MRI scan was performed with a Siemens 1.5 or 3 Tesla MRI device. The positioning was done parallel to the inferior border of the corpus callosum on the axial plane and perpendicular to the hippocampus on the craniocaudal plane. 3D T1-weighet gradient-echo imaging was performed with a section thickness of 1.5 mm and fluid-attenuated inversion recovery (FLAIR) spin-echo was performed with a section thickness of 3 mm.

Then the volume of the thalamus in FCD patients alone and with healthy patients was compared based on demographic information.

Image analysis

First, we examined the images obtained visually to determine the presence of artifacts such as gross motion artifacts. If the image quality was appropriate, we processed them using the Volbrain site. Volbrain is an online MRI brain volumetry system that works fully automatically.

We used Volbrain to delineate the thalamus and calculate bilateral thalamic volumes in both groups with and without FCD. The parameters were Bilateral Thalamic volume (BTV), Right Thalamic volume (RTV), Left Thalamic volume (LTV), Difference between Left and Right (DBLRV), Absolute Value of Difference between Left Thalamic volume and Right Thalamic volume (AVDBLRV), Right Side Mass (RSM), Left Side Mass (LSM). We also calculated the abovementioned parameters for a group without FCD.

Two experienced individuals in medical image analysis manually corrected all measurement masks obtained from the Volbrain site. The edited masks were then reviewed by a neurologist, an expert in anatomical labeling, and blinded to the participant's group. The correction was performed using a slice-by-slice method in the axial direction and verified in the coronal and sagittal directions using ITK-SNAP (**Figure 1**).

Afterward, the template library and the subject to be segmented must be in the same stereotactic space. A spatial normalization was performed based on a linear affine registration to the Montreal Neurological Institute (MNI152) space.

Statistical methods

Categorical data were reported as frequency (percent), and continuous data as mean (SD) or

median (IQR). The Kolmogorov-Smirnov (or Shapiro-wilk) test along with q-q plot, and kurtosis, and skewness statistics were used to test the normality in the data. Demographics characteristics were compared between patients with FCD and healthy subjects using independent samples t-test (for age), and χ^2 test (for gender). Thalamic volume values within FCD and healthy groups were compared by two-paired samples t-test. To minimize the effect of confounders (such as age, and gender) we used analysis of covariance (ANCOVA) for comparison between groups. For all analyses, statistically significant level was set at *p*-value < 0.05. Data were analyzed using SPSS Statistics for Windows (IBM SPSS Statistics for Windows, Version 18.0.).

Results

Populations

Our study examined 60 people with a mean (SD) age of 24.6 (6.9) years, of whom 33 (55.0%) were men. Among the 60 cases, 30 had FCD with a mean age of 22.6 ± 9.3, and 30 were healthy with a mean age of 26.3 ± 3.6. The demographic information of the surveyed people is in **Table 1**.

Thalamic volume measurement

The investigated subjects were compared based on thalamic volume, the results of which are presented in **Table 2**.

According to **Table 2**, the only significant difference observed was between right and left thalamic volumes in the FCD group (P = 0.042). There were no significant differences observed in the absolute value of the difference between the left and right thalamic volumes when comparing all patients with FCD to the control group (*P*-value = 0.054) and when comparing FCD patients on the right side to the control group (*P*-value = 0.021). No significant differences were observed in any other cases.

As shown in **Table 3**, the thalamus on the involved side was significantly smaller than that on the non-involved side in patients with FCD (*P*-value < 0.001). However, there was no noticeable variation in the sizes of the left and right thalami in the control group.

Discussion

In the current study, we evaluated the thalamic volume of patients with FCD compared to non-FCD individuals. The only significant difference observed was between right and left thalamic volumes in the FCD group. The size of the thalamus on the involved side is significantly smaller than on the non-involved side in FCD patients. Also, no significant differences were observed in the absolute value of the difference between the left thalamic volume and right thalamic volume when comparing all patients with FCD to the non-FCD group. According to the best of our knowledge, our study is the first to compare the change in thalamic volume in FCD patients with that of healthy individuals. In the rest of the studies that have been done, only the FCD side has been compared with the opposite side.

Our findings partially align with previous research, revealing novel insights. Rezayev et al. [12] reported a significant decrease in thalamic volume in the FCD hemisphere compared to the contralateral hemisphere in pediatric patients. Similarly, we observed that the thalamus on the involved side was significantly smaller than that on the non-involved side (P < 0.001). However, our study extends beyond this by comparing patients with FCD with healthy controls, revealing that while ipsilateral-contralateral differences exist within patients with FCD, the absolute volumetric differences between patients with FCD and healthy individuals did not reach statistical significance (P = 0.054). This finding contrasts with what might be expected based on other epilepsy syndromes. Studies on temporal lobe epilepsy have consistently shown bilateral thalamic volume reductions compared to healthy controls [15, 16]. The absence of significant bilateral thalamic atrophy in our FCD cohort suggests that the pathophysiological mechanisms in FCD may differ from those in other epileptic conditions, possibly due to the focal and developmental nature of the cortical malformation.

The reduced thalamic volume in FCD patients has significant clinical implications beyond seizure generation. Since the thalamus is crucial for sensory integration, attention, and cognition, its volume reduction likely contributes to both epileptogenesis and the neurocognitive

Table 1. Demographic characteristics of patients and healthy subjects

Variables	Focal Cortical Dysplasia			Control (N=30)	*P*-value			
	Total (N=30)	RSM (N=13)	LSM (N=17)		Between case and control	Between RSM and control	Between LSM and control	Between RSM and LSM
Age (year), mean (SD)[1]	22.6 (9.3)	21.2 (11.3)	23.6 (7.9)	26.3 (3.6)	0.07	0.19	0.23	0.54
Sex (male), No (%)[2]	16 (53.3)	7 (53.8)	9 (52.9)	17 (56.6)	1.00	1.00	1.00	1.00

SD = standard deviation, RSM = Right Side Mass, LSM = Left Side Mass. [1]: Independent t-test; [2]: Chi-square.

Table 2. Comparison of thalamic volume between groups

Variables	Focal Cortical Dysplasia			Control (N=30)	*P*-value*			
	Total (N=30)	RSM (N=13)	LSM (N=17)		Between case and control	Between RSM and control	Between LSM and control	Between RSM and LSM
BTV (mm^3), mean (SD)	15441.0 (1466.1)	15547.6 (1396.9)	15252.9 (1607.6)	15441.8 (1526.9)	0.99	0.82	0.75	0.51
RTV (mm^3), mean (SD)	7710.4 (739.4)	7696.0 (715.1)	7735.7 (802.6)	7797.4 (762.2)	0.71	0.68	0.83	0.86
LTV (mm^3), mean (SD)	7730.6 (744.1)	7851.6 (385.1)	7517.2 (815.6)	7644.4 (803.9)	0.72	0.39	0.67	0.14
AVDBLRV (mm^3), mean (SD)	179.5 (137.1)	157.4 (97.8)	218.5 (184.9)	312.9 (205.9)	0.054	0.021	0.19	0.042

SD = standard deviation, BTV = Bilateral Thalamic volume, RTV = Right Thalamic volume, LTV = Left Thalamic volume, DBLRV = Difference between Left and Right, AVDBLRV = Absolute Value of Difference between Left Thalamic volume and Right Thalamic volume, RSM = Right Side Mass, LSM = Left Side Mass. *Independent t-test.

Table 3. Comparison of thalamic volume between Groups

Variables		RTV (mm^3), mean (SD)	LTV (mm^3), mean (SD)	*P*-value*
Focal Cortical Dysplasia group (N=30)	LSM (N=17)	7735.7 (802.6)	7517.2 (815.6)	<0.001
	RSM (N=13)	7696.0 (715.1)	7851.6 (385.1)	<0.001
	Total (N=30)	7710.4 (739.4)	7730.6 (744.1)	0.54
Control group (N=30)		7797.4 (762.2)	7644.4 (803.9)	0.14

SD = standard deviation, BTV = Bilateral Thalamic volume, RTV = Right Thalamic volume, LTV = Left Thalamic volume, DBLRV = Difference between Left and Right, AVDBLRV = Absolute Value of Difference between Left Thalamic volume and Right Thalamic volume, RSM = Right Side Mass, LSM = Left Side Mass. *Paired t-test.

impairments commonly seen in FCD patients (attention deficits, memory problems, executive dysfunction). The asymmetric involvement may explain lateralized symptoms. Clinically, recognizing thalamic changes could improve prognostic assessments, guide treatment strategies, and help predict surgical outcomes by understanding structural alterations beyond the primary cortical lesion [11, 17-21].

Three main mechanisms may explain thalamic volume reduction: (1) Primary neurodevelopmental abnormalities during embryogenesis that simultaneously affect both cortical and thalamic development, (2) Secondary effects from chronic seizures causing trans-synaptic degeneration and excitotoxic damage along thalamocortical pathways, and (3) Compensatory brain reorganization attempting to maintain functional connectivity despite cortical dysplasia, resulting in subcortical structural remodeling [22-24].

A series of studies that have been conducted have examined the changes in the volume of the thalamus in children without a history of any particular disease. In 2018, Tutunjia et al. demonstrated that the first four years of life carry the most growth in the thalamus size, following which only the transverse dimension seemed to keep increasing until puberty. Also, anterior-posterior (AP) diameter measurements showed a comparable trend to the thalamic volume. According to research by Courchesne et al. [25], grey matter volume grows until about 6-9 years old, falling linearly. Other studies have found a substantial reduction in thalamic volume from 4-18 years old after accounting for intracranial volume [26, 27]. Another study by Ge et al., conducted on patients over 20 years old, found a consistent decrease in grey matter volume starting very early in adulthood, while white matter volumes decrease only later in life [25].

However, several acute and chronic clinical circumstances might impact the basal ganglia and thalami bilaterally. Diseases such as systemic metabolic syndrome, vascular issues, degenerative diseases, infections, and tumors are examples [28, 29]. Additionally, thalamic atrophy in pediatrics with the absence of epilepsy can be quantified and observed [30]. However, only a few studies have found a correlation between FCD and thalamic volume [12, 13, 31].

In 2018, Rezayev et al. [12] conducted a study on pediatrics with FCD to evaluate the thalamocortical abnormalities. Among cases, 75 and 68 patients were analyzed for thalamic volume and diffusion tensor imaging (DTI), respectively. A significant decrease in the volume of the FCD hemisphere thalamus was detected as compared to the contralateral hemisphere. In comparison to controls, there was an observed reduction in tract volume, length, count, fractional anisotropy (FA) of thalami, and FA of thalamocortical pathways in FCD patients. FCD patients had higher odds of exhibiting high ADC in both the thalamus and thalamocortical pathways. However, thalamic volume was significantly reduced in the FCD hemisphere as compared to the contralateral hemisphere, there was no significant difference in FA or ADC of the thalamus, nor thalamocortical tracts. This suggests that the observed hemispheric reduction of thalamic volume may not be explained solely by a loss of myelination, as it can be inferred that measures of the properties of water diffusivity (FA and ADC) would be altered. While studies have previously measured FA and ADC in specific brain regions, almost all of the literature on DTI imaging in FCD cases did not use controls in their methodology; instead, statistical measures were used to compare the ipsilateral and contralateral hemispheres, assuming that the contralateral is a healthy equivalent of the control [32-34].

Another study conducted by Jin et al. [13] evaluated the MRI and clinical findings associated with SRE in individuals with FCD type II. Among the 77 cases, 36 and 41 patients had SRE and non-SRE, respectively. They measured small lesion sizes, defined as those with a volume less than 3,217 mm^3. A small FCD lesion was seen in 60.9% of SRE patients, which was significantly more than the non-SRE group. However, there was no statistically significant difference in thalamic volume or seizure semiology between the SRE and non-SRE groups. Also, the volumes of FCD type II showed no correlation with the volume of the ipsilateral and contralateral thalamus, controlling for age, gender, and intracranial volume. Our study is in contrast with this study. In our investigation, the size of the thalamus on the involved side is

significantly smaller than on the non-involved side in FCD patients. However, we did not evaluate the SRE in our population. Also, as mentioned, we compared the thalamic volume of FCD patients with healthy individuals.

Our research has limitations. First, there is the relatively small size of the population that was investigated. Second, the failure to evaluate the rate of seizures in the study population, as well as the effect of thalamic volume on the chance of seizures. And thirdly, different types of FCD were not compared in this study. While the volume of the thalamus may be different in different types of FCD.

Conclusion

According to our knowledge, our study is the first to compare the change in thalamic volume in FCD patients with that of healthy individuals. Our study showed that among patients with FCD, the size of the thalamus on the involved side is significantly smaller than on the non-involved side. However, there were no significant differences in total right and left thalamic volumes in FCD patients compared to the healthy subjects. Overall, we suggest that future research investigating the relationship between specific FCD subtypes, thalamic volume changes, and epilepsy phenotypes/severity. Also, it is better that to use lager sample size and multicentral evaluation.

Disclosure of conflict of interest

None.

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