

Corpus Callosum Morphometric Analysis in Paediatric Epilepsy: A Retrospective MRI Study

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ABSTRACT

INTRODUCTION: The corpus callosum (CC) plays a central role in interhemispheric communication and seizure propagation. Structural alteration of the CC in paediatric epilepsy has been inconsistently reported, highlighting the need for further comparative morphometric analysis during this critical neurodevelopmental period. This study aimed to evaluate differences in CC morphometry between epileptic and non-epileptic children aged 6-17 years.

MATERIALS AND METHODS: This retrospective case-control study analysed brain magnetic resonance imaging (MRI) scans acquired between July 2023 and October 2024 at a single tertiary hospital, including 50 epileptic and 42 non-epileptic children. CC morphometry, including anterior-posterior diameter, total length, and thickness of genu, body, isthmus, and splenium, was measured on mid-sagittal T2-weighted images. Statistical analyses were performed using the independent t-tests and Mann-Whitney tests as appropriate. **RESULT:** No significant overall differences were found in CC morphometry between epileptic and non-epileptic groups. However, subgroup analysis revealed significantly reduced body thickness in epileptic children aged 12-14 years and shorter total CC length among epileptic females. In contrast, epileptic children without extra-callosal abnormalities showed greater genu thickness compared to non-epileptic controls. These findings were associated with a moderate-to-large effect size (Hedges' $g > 0.6$), suggesting subtle deviations in callosal maturation patterns. **CONCLUSION:** Epilepsy may be associated with region-specific and sex-related variation in CC morphometry during critical stages of brain maturation. Although these differences are subtle and not evident on conventional MRI, quantitative CC morphometry may serve as a complementary, hypothesis-generating approach to investigating neurodevelopmental alterations in paediatric epilepsy.

Keywords

epilepsy, corpus callosum, morphometry, magnetic resonance imaging, paediatric

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INTRODUCTION

Epilepsy is the most common chronic neurologic condition that, if left untreated, causes lifelong disability, premature death, carries social stigma, and a significant economic burden on a country. In a developing country, paediatric epilepsy incidence ranges between 33 and 82 cases per 10,000 children. However, this number is often higher due to irregular reporting, diagnostic difficulties,

lack of management standardisation, and limited availability of paediatric neurologists, which worsens the accuracy of diagnosis and reporting of these cases¹. Paediatric epilepsy also often associated with structural abnormalities of the brain, including morphometric changes in the corpus callosum.^{2,3}

The corpus callosum (CC) connects two cerebral hemispheres and facilitates interhemispheric communication. It contributes to seizure generalisation by transmitting unilateral seizure activity to the contralateral hemisphere.^{4,5} CC abnormalities are frequently observed alongside neuronal migration disorders, such as lissencephaly, pachygyria, and hemimegalencephaly during brain development, which disrupt brain structure and increase seizure risk.^{6,7} Vice versa, recurrent seizures in epilepsy can have a profound effect on brain development and function. Understanding the morphometry differences as structural projections between epileptic and non-epileptic children is essential for explaining the neurodevelopmental impact of epilepsy.

Studies examining the relationship between CC structure and epilepsy have a long history. However, studies in pediatric epilepsy were limited, and findings have been inconsistent. In one study, thickened CC was observed in 9 of 37 children aged 7-14 years, with genu, isthmus, and splenium thicknesses exceeding those of the adult CC segment.⁷ Another study reported that an epileptic was found in 14 of 39 subjects with CC dysgenesis, ranging from hypoplasia to agenesis of the CC spectrum.⁸ This inconsistency highlights the need for further investigation into specific methods. By focusing on comparative analysis of CC morphometry between epileptic and non-epileptic paediatric groups, this study aims to understand whether epilepsy affects brain structure during critical developmental periods. Our primary hypothesis is that there is a significant difference in CC morphometry between the two groups, especially with specific age and sex-related factors.

MATERIALS AND METHODS

Study Population

We retrospectively and consecutively included 256 head MRI examinations of children aged 6-17 years who underwent head MRI scans at our center from July 2023 to October 2024. We then stratified them into epileptic and non-epileptic groups based on their clinical

referral information. The study initially excluded one epileptic patient with complete CC agenesis and 163 non-epileptic patients due to their brain parenchymal abnormalities, resulting in 50 epileptic subjects and 42 non-epileptic subjects. The clinical profiles were reviewed from the medical record, including seizure onset and clinical companion. This study was approved by the local Ethics Committee in Human Research (1981/LOE/301.4.2/V/2025) before starting the study.

Data Image and Protocol

We used MRI image data obtained from both 3T (Magnetom SKYRA, Siemens AG Healthcare Sector, Erlangen, Germany) and 1.5T (Optima MR360, GE Healthcare, Wisconsin, USA) scanners. Morphometric measurements were performed on T2-weighted mid-sagittal images with a slice thickness of 4-5mm and an interslice gap of 0.4-0.5mm.

Although MRI examinations were acquired using both 1.5T and 3T scanners, slice thickness and interslice gaps were comparable across systems. Morphometric measurements were restricted to clearly defined mid-sagittal images using standardised selection criteria to minimise acquisition-related variability.

The mid-sagittal slice was manually selected based on clear visualisation of the cerebral aqueduct, third ventricle, and full rostro-caudal extent of the corpus callosum. All measurements were performed using Horos DICOM Viewer (version 3.3.6, Horos Project) with standardised zoom and window settings applied consistently across all cases to ensure consistency.

Morphometric parameters included antero-posterior diameter (APD), length of the CC (LCC), genu thickness (GT), body thickness (BT), isthmus thickness (IT), and splenium thickness (ST) as illustrated in Figure 1.

All measurements were performed once by a single observer who was blinded to the clinical diagnosis and group allocation at the time of measurement, following

a predefined standardised measurement protocol under the supervision of the three board-certified radiologists. Repeat measurements for inter- or intra-observer reliability analysis were not performed due to the retrospective nature of the study and the absence of duplicated standardised imaging datasets. In the epileptic group, findings of brain abnormalities outside the CC were also recorded.

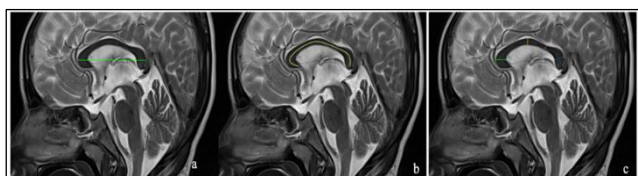


Figure 1: Morphometry measurement method in T2 FSE sequence in midline sagittal sections (a) antero-posterior diameter; (b) length of the CC (LCC); (c) segmental thickness: genu thickness (GT) in green, body thickness (BT) in yellow, isthmus thickness (IT) in orange, and splenium thickness (ST) in blue

Statistical Analysis

Demographic data were tested for homogeneity using the Chi-Square and Mann-Whitney tests. Analytical data were first tested for normality using the Shapiro-Wilk test. If the data were normal, the analysis proceeded with an independent T-test. For non-normal data, the Mann-Whitney test was applied. The analysis compared the epileptic and non-epileptic groups. We also performed a comparative analysis between the two groups based on age and sex. Furthermore, we compared the CC morphometry between the epileptic group without abnormalities outside the CC and the non-epileptic group. All the tests were performed using the 26th version of IBM SPSS statistical analysis with a significance value of $p < 0.05$.

RESULT

Demographic Result

Out of 92 patients, 49 (53.3%) were boys, and 43 (46.7%) were girls, with 38 subjects (41.3%) aged 6-8 years, 26 subjects (28.3%) aged 9-11 years, 18 subjects (19.6%) aged 12-14 years, and 10 subjects (10.8%) aged 15-17 years. Table I shows the summary of clinical and imaging findings characteristic of the epileptic group, while Figure 2 shows some recorded extra-callosal abnormalities.

Table I: Clinical and imaging findings characteristic of the epileptic group

Characteristic	n (%)
Seizure onset	Infant (0-2 years)
	16 (32)
	Early Childhood (3-5 years)
	10 (20)
	Middle Childhood (6-12 years)
Clinical companion	19 (38)
	Adolescent (> 12 years)
	1 (2)
	No data
	4 (8)
Imaging findings of abnormalities outside the CC	Brain infection history
	2 (4)
	Developmental disability
	8 (16)
	Intellectual disability
No extra-callosal abnormality	5 (10)
	No clinical companion
	35 (70)
	Cortical disorders
	10 (20)
Hamartoma	1 (2)
	Hippocampal disorders
	9 (18)
	Ventricular disorders
	1 (2)
Brain infarction	1 (2)
	Brain infection
	2 (4)
	Mixed disorders
	4 (8)
No extra-callosal abnormality	22 (44)

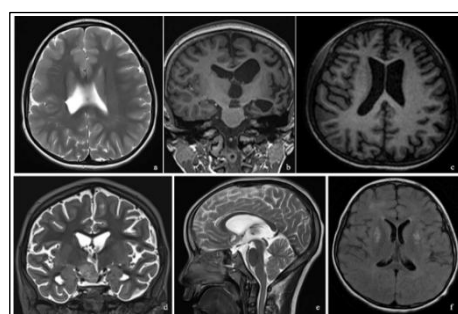


Figure 2: Some recorded extra-callosal abnormalities (a) *pachygyria* of the right frontoparietal lobe; (b) mixed of the left hemi atrophy, *hydrocephalus*, and hippocampal atrophy; (c) *polymicrogyria* of the right frontotemporoparietal lobe; (d) hamartoma; (e) *hydrocephalus*; (f) infarction of the bilateral basal ganglia and the right frontal subcortical

Statistical Result

No significant differences in corpus callosum morphometry were observed between epileptic and non-epileptic groups across all measurement parameters, as all P values exceeded 0.05 (data not shown). However, in the comparative analysis between the two groups based on age (Table II), there is a significant difference in BT parameter in the 12-14 years' age category ($p = 0.03$, Cohen's $d = 1.12$, Hedges' $g = 1.07$), in which the non-epileptic group has a thicker CC body when compared with the epileptic group with a large effect size. Meanwhile, in the comparative analysis based on sex (Table III), there is no significant difference in all parameters in the male category but there is a significant difference in LCC parameter in the female category ($p = 0.013$, Cohen's $d = 0.73$, Hedges' $g = 0.72$), in which the female epileptic group has lower

LCC compared with the non-epileptic group with a moderate-to-large effect. We also analysed the CC morphometry between the epileptic group without extra-callosal abnormalities and the non-epileptic group (Table IV). There is a significant difference in the GT parameter ($p=0.012$, Cohen's $d=0.63$, Hedges' $g=0.62$), with the epileptic group having greater GT compared with the non-epileptic group. It has a moderate effect size.

Table II: CC morphometry comparison between the epileptic and non-epileptic groups based on age

Age	Parameters	Mean (SD) (mm)		P value	Cohen's d	Hedges' g
6-8 years	APD	62.43 (3.59)	62.16 (6.08)	0.872		
	LCC	81.33 (4.54)	78.90 (8.50)	0.275		
	GT	9.17 (1.34)	9.39 (2.07)	0.704		
	BT	5.05 (0.75)	5.03 (1.19)	0.957		
	IT	2.88 (0.61)	2.77 (0.70)	0.368		
	ST	8.16 (1.00)	8.06 (2.01)	0.839		
9-11 years	APD	63.27 (2.79)	63.32 (5.51)	0.53		
	LCC	82.42 (5.51)	80.57 (7.52)	0.50		
	GT	9.41 (1.46)	9.37 (2.39)	0.97		
	BT	5.39 (1.25)	5.09 (1.82)	0.76		
	IT	3.30 (1.13)	3.26 (0.98)	0.92		
	ST	9.11 (1.48)	8.92 (2.01)	0.79		
12-14 years	APD	68.89 (2.93)	67.13 (2.66)	0.20		
	LCC	87.31 (5.33)	83.27 (10.53)	0.34		
	GT	9.57 (0.91)	10.15 (2.68)	0.54		
	BT	5.29 (1.00)	3.90 (1.39)	0.03	1.12	1.07
	IT	2.81 (0.98)	2.76 (0.62)	0.89		
	ST	9.94 (1.20)	8.90 (2.19)	0.25		
15-17 years	APD	67.26 (4.48)	67.44 (4.65)	0.95		
	LCC	85.69 (5.18)	82.16 (7.79)	0.42		
	GT	10.79 (2.15)	10.84 (1.64)	0.97		
	BT	5.02 (1.24)	5.32 (1.42)	0.73		
	IT	3.87 (1.35)	3.98 (1.30)	0.90		
	ST	10.30 (1.88)	9.90 (2.06)	0.76		

Table III: CC morphometry comparison between the epileptic and non-epileptic groups based on sex

Parameters	Mean (SD) (mm)		<i>P</i> value	Cohen's d	Hedges' g
	Non epileptic	Epileptic			
MALE					
APD	63.81 (4.91)	65.53 (4.46)	0.207		
LCC	82.88 (5.93)	82.98 (7.26)	0.958		
GT	9.57 (1.64)	9.97 (2.28)	0.483		
BT	5.48 (0.90)	4.98 (1.46)	0.162		
IT	3.26 (1.12)	3.29 (1.01)	0.912		
ST	8.92 (1.84)	8.91 (2.10)	0.980		
FEMALE					
APD	65.31 (2.94)	63.13 (6.28)	0.424		
LCC	83.8 (4.71)	78.22 (9.12)	0.013	0.73	0.72
GT	9.42 (1.20)	9.39 (2.23)	0.967		
BT	4.73 (0.94)	4.73 (1.56)	0.873		
IT	2.88 (0.66)	2.70 (0.77)	0.419		
ST	9.11 (0.89)	8.43 (2.05)	0.147		

Table IV: CC morphometry comparison between the epileptic with no abnormalities outside the CC and non-epileptic groups

Parameters	Mean (SD) (mm)		P value	Cohen's d	Hedges' g
	Non Epileptic	Epileptic			
APD	64.46 (4.20)	64.60 (4.81)	0.904		
LCC	83.27 (5.40)	81.46 (7.08)	0.257		
GT	9.50 (1.45)	10.45 (1.54)	0.012	0.63	0.62
BT	5.16 (0.98)	5.56 (1.12)	0.144		
IT	3.10 (0.96)	3.30 (0.80)	0.289		
ST	9.00 (1.49)	9.59 (1.61)	0.148		

DISCUSSIONS

CC plays a pivotal role in the pathophysiology of epilepsy, serving as the main commissural pathway that connects the two cerebral hemispheres and facilitates interhemispheric communication. Structural abnormalities such as CC agenesis or dysgenesis can disrupt these interhemispheric connections and contribute to seizure generation and propagation. Although the CC consists primarily of white matter and lacks active neuronal bodies, it can facilitate the bilateral spread of seizure activity by transmitting excitatory impulses from one hemisphere to the other.^{4,5}

In the present study, no significant differences were observed in overall CC morphometric parameters between epileptic and non-epileptic groups (data not shown). This finding aligns with previous reports that found no major volumetric or linear differences in CC structure among children with epilepsy.^{9,10} However, morphometric measurements reflect only macroscopic geometry and may not directly correspond to microstructural integrity. Studies using diffusion tensor imaging (DTI) have demonstrated that even in the absence of morphometric differences, epileptic children may show altered white matter organisation, including reduced fractional anisotropy (FA) and increased mean diffusivity (MD) in callosal fibres.¹⁰⁻¹² These results suggest that CC morphology alone represents only one aspect of callosal anatomy. Future analyses integrating morphometric and diffusion metrics could provide a more comprehensive understanding of white matter abnormalities in epilepsy.

Recent work has also proposed alternative morphometric approaches, such as surface-based and voxel-based morphometry, which offer greater sensitivity in detecting subtle shape variations.¹³ Incorporating these advanced methods in future pediatric epilepsy research may refine morphometric characterisation and improve reproducibility across imaging centers.

The epileptic cohort in this study was biologically heterogeneous, encompassing a wide range of etiologies including structural abnormalities, developmental disorders and acquired brain lesions. Such heterogeneity may have diluted global morphometric differences when epilepsy is analysed as a single entity and underscores the importance of stratified analyses in pediatric epilepsy research.

When analysed by age, a significant difference was observed in body thickness (BT) among subjects aged 12-14 years, with thinner BT in the epileptic group. This developmental period is critical for callosal maturation, as substantial myelination and remodeling occur throughout adolescence. Previous studies have shown significant increases in callosal area and callosal-to-supratentorial ratios between early childhood and mid-adolescence.^{12,14} Dysregulated maturation during this sensitive window may therefore explain the observed BT thinning in epileptic adolescents. Interictal discharges and neuroinflammation have been implicated as potential mechanisms interfering with normal callosal development.¹⁵⁻¹⁷ The effect size of this difference was large, suggesting a substantial deviation in callosal body thickness associated with epilepsy in this age group. Nevertheless, given the modest sample sizes and retrospective design, these results should be interpreted cautiously and require confirmation in larger, prospective cohorts.

Sex-based comparison (Table III) revealed a significantly shorter total CC length (LCC) in females with epilepsy compared to non-epileptic females with moderate effect size. This observation is consistent with previous

findings showing a steeper developmental slope and earlier callosal maturation in females.^{14,18} Because females experience accelerated CC development, any neurodevelopmental disturbance may become more pronounced earlier in adolescence. While our sample size limits generalisation, this pattern warrants further longitudinal evaluation.

Linear CC measurements without normalisation to intracranial volume or total brain size may partially reflect individual differences in head size or developmental stage. Therefore, the observed age- and sex-related differences should be interpreted as relative morphometric trends rather than absolute anatomical differences.

Analysis restricted to epileptic children without extra-callosal abnormalities (Table IV) showed thicker genu (GT) compared to the non-epileptic peers. This result is consistent with the findings of Schupper et al, who reported increased CC thickness in a subset of children with epilepsy.⁷ Given that callosal development follows a rostro-caudal pattern, where posterior regions mature earlier,^{14,19} a thicker genu may reflect asynchronous anterior callosal development rather than a definitive compensatory growth pattern. The effect size of this difference was moderate, suggesting that the observed alteration reflects a meaningful deviation in callosal growth trajectory among epileptic children. This moderate magnitude reinforces the notion that epilepsy may subtly influence regional neurodevelopment rather than producing gross structural abnormalities, potentially representing adaptive remodeling in response to disrupted interhemispheric signaling.

Overall, the observed differences suggest that epilepsy may be associated with deviations in normal callosal maturation patterns. However, these findings should be interpreted with caution, given the retrospective and correlative nature of the study. The results do not imply causality but highlight subtle morphometric trends that

may reflect altered neurodevelopmental timing in paediatric epilepsy.

Several limitations should be acknowledged. First, its retrospective design inherently limits control over data completeness and uniformity. MRI and clinical data were derived from pre-existing hospital records, restricting the ability to account for important confounders such as epilepsy subtype (generalised versus focal), seizure frequency, illness duration, and anti-epileptic drug exposure. These factors are known to influence both global brain morphology and corpus callosum development, and their absence may have introduced bias.

Second, inter- and intra-observer reliability analyses were not performed, which may limit measurement reproducibility. However, to minimise variability, all measurements were performed using predefined anatomical landmarks, fixed zoom and window settings, and supervised by experienced radiologists. Future prospective studies should incorporate repeated measurement and reliability testing. Nevertheless, the use of different scanner field strengths and slice parameters may still have influenced linear measurement precision and represent a limitation of this study.

Third, corpus callosum dimensions were not normalised to intracranial or total brain volume. Although the age range was restricted to 6-17 years to minimise developmental variation, linear measurements may still be influenced by head size, sex-related brain differences, or maturational stage. This limitation should be considered when interpreting the observed sex- and age-related morphometric differences.

Finally, the relatively small sample size limits the statistical power to detect subtle morphometric differences. A larger, prospectively collected and multicenter data set would allow for more robust subgroup analysis, particularly regarding epilepsy types, duration, and sex-specific maturation patterns.

Future research should incorporate longitudinal follow-up to track callosal development over time and combine conventional morphometry with diffusion tensor imaging (DTI) or advanced surface-based approaches. Integration of these methods may yield a more comprehensive understanding of structural and microstructural alterations associated with pediatric epilepsy and improve the interpretability of neurodevelopmental trajectories

CONCLUSION

While epilepsy does not produce a uniform reduction in CC morphology across all paediatric age groups, specific morphometry differences may emerge during critical periods of brain maturation and appear to be influenced by sex, even in the absence of overt parenchymal abnormalities on conventional MRI. Recognising these subtle findings contributes to a better understanding of the neurodevelopmental impact of pediatric epilepsy and supports the role of quantitative neuroimaging as a hypothesis-generating, complementary approach in research settings.

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