Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Association Between Polysomnographic Findings and Postoperative Decompressive Volume in Adults with Chiari Malformation Type I

Harun Emül
Inonu University
İsmail İçlek
Inonu University
Sarp Şahin
Inonu University
Durmuş Emre Karatoprak
Inonu University
Mustafa Namık Öztanır
Inonu University
Hilal Ermiş
Inonu University
Güleç Mert Doğan
Inonu University
Selami Çağatay Önal
conal_tr@yahoo.com

Inonu University

Research Article

Keywords: Chiari malformation type I, polysomnography, posterior fossa decompression, sleep-disordered breathing, 3D volumetry

Posted Date: November 12th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10869258/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Objective

To evaluate the relationship between postoperative posterior fossa decompressive volume and polysomnographic outcomes in adults with Chiari malformation type I.

Methods

This retrospective single-center study included 47 adult patients with Chiari malformation type I who underwent suboccipital craniectomy, C1 laminectomy, and duraplasty. All patients had preoperative and postoperative polysomnography and radiological evaluation. Posterior fossa volume was assessed using both manual morphometric measurements and 3D Slicer-based segmentation. Preoperative and postoperative polysomnographic parameters were compared, and correlations between volumetric parameters and postoperative sleep outcomes were analyzed. Multivariable linear regression was performed to identify predictors of postoperative sleep-disordered breathing outcomes.

Results

The mean age was 37.8 ± 12.8 years, and 10 patients (21.3%) were male. After decompressive surgery, N3 sleep duration increased significantly from 106.4 ± 61.2 to 138.0 ± 60.9 min ($p = 0.008$), and total sleep time increased from 380.3 ± 82.5 to 410.2 ± 60.1 min ($p = 0.044$). Central apnea index decreased from 3.66 ± 12.32 to 1.58 ± 2.77 events/h, but this change was not statistically significant ($p = 0.210$). 3D Slicer posterior fossa volume increase percentage was positively correlated with postoperative total sleep time ($r = 0.293$, $p = 0.048$). Larger 3D Slicer postoperative posterior fossa volume was negatively correlated with apnea–hypopnea index ($r=-0.331$, $p = 0.028$) and mean central apnea duration ($r=-0.309$, $p = 0.041$). In multivariable analysis, age independently predicted postoperative apnea–hypopnea index ($\beta = 0.295$, $p = 0.011$), and preoperative central apnea index independently predicted postoperative central apnea index ($\beta = 0.093$, $p = 0.006$).

Conclusion

Posterior fossa decompression was associated with improved sleep architecture, and volumetric decompression showed significant associations with selected postoperative polysomnographic outcomes.

Introduction

Chiari malformation type I (CM-I) is a structural abnormality of the craniocervical junction characterized by caudal displacement of the cerebellar tonsils through the foramen magnum [1, 2]. Although CM-I may

remain asymptomatic in some individuals, it can be associated with a wide spectrum of clinical manifestations, including headache, neck pain, dizziness, sensory disturbances, lower cranial nerve dysfunction, and sleep-related respiratory abnormalities [3, 4]. In adults, the clinical presentation may be heterogeneous, and symptom severity is not always directly proportional to the degree of tonsillar herniation observed on magnetic resonance imaging.

Sleep-disordered breathing has increasingly been recognized as an important clinical component of CM-I [5]. The involvement of brainstem respiratory centers, altered cerebrospinal fluid dynamics, and compression at the cervicomedullary junction may contribute to central apnea, obstructive events, hypoventilation, and sleep architecture disturbances (6]. Polysomnography provides objective assessment of these abnormalities by evaluating sleep stages, total sleep time, sleep efficiency, apnea–hypopnea index, central apnea index, and central apnea burden [7, 8]. However, the relationship between radiological decompression and postoperative polysomnographic outcomes remains incompletely understood.

Posterior fossa decompression is the main surgical treatment for symptomatic CM-I and aims to restore cerebrospinal fluid flow, reduce neural compression, and increase the functional volume of the posterior fossa and craniocervical junction [9]. While many studies have focused on clinical improvement, syringomyelia regression, and radiological changes after surgery, fewer have examined whether the magnitude of decompressive volume gain is associated with objective improvements in sleep parameters [10]. Volumetric assessment using manual measurements and three-dimensional segmentation methods, such as 3D Slicer, may provide a more quantitative evaluation of postoperative anatomical change than linear measurements alone [11].

The aim of the present study was to evaluate the relationship between postoperative decompressive posterior fossa volume and polysomnographic findings in adults with CM-I. Specifically, we investigated preoperative and postoperative changes in sleep parameters, examined correlations between posterior fossa volumetric measurements and postoperative polysomnographic outcomes, and assessed whether decompressive volume parameters were independently associated with postoperative sleep-disordered breathing indices.

Material and Methods

Study Design and Patient Selection

This retrospective, single-center study included adult patients who underwent surgery for Chiari malformation type I (CM-I) at the Department of Neurosurgery, Turgut Özal Medical Center, İnönü University, between January 2010 and June 2025. CM-I was defined radiologically as cerebellar tonsillar descent of at least 5 mm below the foramen magnum. Patients were considered surgical candidates if they had clinical symptoms attributable to CM-I, such as headache, paresthesia, dysarthria, motor weakness, or balance and coordination disturbances, and/or associated syringomyelia and/or abnormal

polysomnographic findings suggestive of sleep-related respiratory dysfunction. During the study period, 121 patients underwent surgery for CM-I. Of these, 60 patients were excluded because preoperative polysomnography was not available. Among the remaining 61 patients, 7 were excluded because duraplasty was not performed as part of the standard surgical procedure, 4 because postoperative polysomnography was not completed, 2 because preoperative polysomnography had been performed at an external center and did not include all required parameters, and 1 because preoperative radiological images were unavailable. The final study cohort consisted of 47 adult patients. Patients were included if they were aged 18 years or older, had a diagnosis of CM-I, underwent suboccipital craniectomy, C1 laminectomy, and duraplasty, had both preoperative and postoperative polysomnography, had at least 1 year of clinical follow-up, and had available preoperative and postoperative magnetic resonance imaging and non-contrast cranial computed tomography including the craniocervical junction. Patients younger than 18 years, those who underwent non-standard surgical procedures, those without postoperative polysomnography, those with incomplete polysomnographic parameters, those with missing radiological imaging, and those with inadequate follow-up were excluded.

The study protocol was approved by the İnönü University Health Sciences Scientific Research Ethics Committee, Scientific Research and Publication Ethics Board (session no: 15; decision no: 2025/8371; date: November 18, 2025). The study was conducted in accordance with the principles of the Declaration of Helsinki. Because of the retrospective design of the study, the requirement for written informed consent was waived by the ethics committee.

Data Collection

Demographic data, clinical records, radiological images, operative details, and polysomnographic findings were obtained from the hospital information system, radiology archive, and sleep laboratory records. Age, sex, body mass index, tonsillar herniation distance, syringomyelia status, posterior fossa morphometric measurements, posterior fossa volumetric parameters, sleep stage durations, total sleep time, sleep efficiency, apnea–hypopnea index, central apnea index, mean central apnea duration, and number of central apneas were recorded.

Radiological Assessment and Posterior Fossa Measurements

All patients underwent preoperative and postoperative magnetic resonance imaging including the craniocervical junction. Non-contrast cranial computed tomography scans were used for posterior fossa morphometric and volumetric assessment. Postoperative radiological measurements were obtained from follow-up imaging performed at least 2 months after surgery.

Tonsillar herniation distance was measured on mid-sagittal magnetic resonance images as the perpendicular distance between the tip of the cerebellar tonsils and the foramen magnum reference line. Syringomyelia was evaluated on cervical or spinal T2-weighted magnetic resonance images and

recorded as present or absent. Representative preoperative and postoperative magnetic resonance images demonstrating syringomyelia regression after decompressive surgery are shown in Fig. 1. Posterior fossa volume was first estimated manually using a morphometric approach based on an ellipsoid geometry assumption. On cranial computed tomography images, craniocaudal height, anteroposterior diameter, and transverse diameter of the posterior fossa were measured on sagittal, coronal, and axial planes. Posterior fossa volume was calculated using the ellipsoid volume formula: where AP represents anteroposterior diameter, TR represents transverse diameter, and CC represents craniocaudal height. Posterior fossa volume increase was calculated as the difference between postoperative and preoperative volumes. The percentage of volume increase was calculated as follows: Volume increase (%) = ((postoperative volume − preoperative volume) / preoperative volume) × 100

Left: Preoperative image showing prominent syringomyelia extending along the cervical spinal cord. Right: Postoperative cervical magnetic resonance image obtained in the second postoperative week after suboccipital craniectomy, C1 laminectomy, and duraplasty, demonstrating marked regression of syringomyelia.

3D Slicer-Based Volumetric Analysis

Posterior fossa volume was also measured using three-dimensional segmentation with 3D Slicer software. Preoperative and postoperative non-contrast cranial computed tomography images were exported in DICOM format and imported into the software. The anatomical borders of the posterior fossa were manually identified, and segmentation was performed on multiplanar views. The software-based volume calculation module was then used to obtain posterior fossa volume in cubic centimeters. Preoperative and postoperative segmented volumes were compared for each patient, and absolute volume increase and percentage volume increase were calculated. A representative example of preoperative and postoperative segmentation-based posterior fossa volumetry is shown in Fig. 2.

Representative preoperative and postoperative non-contrast cranial computed tomography DICOM images of an adult patient with Chiari malformation type I demonstrating segmentation-based posterior fossa volume calculation. The upper panel shows preoperative multiplanar segmentation and three-dimensional modeling of the posterior fossa, with a measured posterior fossa volume of 158.69 cm^3. The lower panel shows postoperative posterior fossa segmentation after decompressive surgery, with a measured posterior fossa volume of 188.32 cm^3.

Polysomnographic Evaluation

All patients underwent full-night polysomnography at the Sleep Disorders Center of Turgut Özal Medical Center using a 55-channel Alice 6 computerized polysomnography system. Postoperative polysomnography was performed as part of the postoperative follow-up protocol at least 2 months after surgery. The polysomnographic recordings included electroencephalography, electrooculography, submental and tibialis electromyography, electrocardiography, oronasal airflow, thoracoabdominal

respiratory effort, oxygen saturation, body position, and snoring parameters. All polysomnographic recordings were manually scored by an experienced physician certified in sleep medicine. Sleep stages, respiratory events, sleep efficiency, total sleep time, obstructive and central apnea events, apnea–hypopnea index, central apnea index, mean central apnea duration, and number of central apneas were recorded. Sleep stages and respiratory events were scored according to the American Academy of Sleep Medicine Scoring Manual, version 2.6.

Surgical Technique

All patients underwent surgery under general anesthesia in the prone position with the head slightly flexed. A midline vertical incision was made from approximately 2 cm above the inion to the C3 level. The occipital bone and upper cervical posterior elements were exposed through subperiosteal dissection. A suboccipital craniectomy of approximately 3×4 cm was performed and extended laterally toward the occipital condyles when necessary. Total C1 laminectomy was then performed. Fibrotic bands between the foramen magnum and C1 were released. The posterior fossa dura was opened in a Y-shaped fashion from the occipital region toward C1, and cerebrospinal fluid flow and dural pulsation were confirmed. Duraplasty was performed using an autologous fascial graft harvested from the paravertebral fascia. The wound was closed anatomically after placement of a free-drainage surgical drain.

Outcome Measures

The primary outcome measures were postoperative polysomnographic parameters and their relationship with posterior fossa decompressive volume parameters. The main volumetric variables were manual posterior fossa volume increase, manual posterior fossa volume increase percentage, 3D Slicer posterior fossa volume increase, and 3D Slicer posterior fossa volume increase percentage. Secondary outcome measures included preoperative-to-postoperative changes in polysomnographic parameters and subgroup comparisons according to syringomyelia status and residual postoperative tonsillar herniation.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows, version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean±standard deviation, and categorical variables were expressed as number and percentage. The normality of continuous variables was assessed using the Shapiro–Wilk test, histograms, and Q–Q plots. Preoperative and postoperative polysomnographic parameters were compared using the paired-samples t-test for normally distributed variables and the Wilcoxon signed-rank test for non-normally distributed variables. Comparisons between independent groups, including syringomyelia status and residual postoperative tonsillar herniation categories, were performed using the independent-samples t-test for normally distributed variables and the Mann–Whitney U test for non-normally distributed variables. Categorical variables

were compared using the chi-square test or Fisher's exact test, as appropriate. Correlations between posterior fossa volumetric parameters and postoperative polysomnographic outcomes were evaluated using Pearson or Spearman correlation analysis according to the distributional characteristics of the variables. Correlation coefficients were reported as r values together with corresponding p values. Multivariable linear regression analysis was performed to identify independent predictors of postoperative sleep-disordered breathing outcomes. Separate regression models were constructed for postoperative apnea–hypopnea index, postoperative central apnea index, and mean postoperative central apnea duration. Clinically relevant covariates were included in the models, including age, body mass index, syringomyelia status, the corresponding preoperative polysomnographic parameter, and 3D Slicer postoperative posterior fossa volume. Complete-case analysis was used for regression models. Regression results were reported as unstandardized β coefficients with 95% confidence intervals. A two-tailed p value of < 0.05 was considered statistically significant.

Results

The study included 47 adults with a mean age of 37.8 ± 12.8 years; 57.4% had syringomyelia. Mean tonsillar herniation decreased from 10.6 ± 4.1 mm preoperatively to 4.2 ± 4.5 mm postoperatively. The mean 3D Slicer-derived posterior fossa volume increase was 23.8 ± 10.0 cm^3 (16.0 ± 7.1%) (Table 1).

Table 1
Clinical, Radiological, Volumetric, and Baseline Polysomnographic Characteristics of the Study Cohort

Variable	Overall cohort (n = 47)
Clinical characteristics	
Age, years	37.8 ± 12.8
Male sex, n (%)	10 (21.3)
Body mass index, kg/m²	29.1 ± 6.8
Radiological characteristics	
Preoperative tonsillar herniation distance, mm	10.6 ± 4.1
Postoperative tonsillar herniation distance, mm	4.2 ± 4.5
Syringomyelia, n (%)	27 (57.4)
Posterior fossa height, cm	3.3 ± 0.3
Posterior fossa transverse diameter, cm	10.9 ± 0.6
Posterior fossa volumetry	
Manual posterior fossa volume increase, cm³	21.1 ± 9.6
Manual posterior fossa volume increase, %	15.7 ± 7.7
3D Slicer posterior fossa volume increase, cm³	23.8 ± 10.0
3D Slicer posterior fossa volume increase, %	16.0 ± 7.1
Baseline polysomnographic characteristics	
Total sleep time, min	380.3 ± 82.5
Sleep efficiency, %	82.2 ± 12.2
N3 sleep duration, min	106.4 ± 61.2
REM sleep duration, min	54.0 ± 25.3
Apnea–hypopnea index, events/h	8.4 ± 14.9
Central apnea index, events/h	3.7 ± 12.3
Number of central apneas	21.9 ± 69.4

Data are presented as mean ± standard deviation or number (%). Abbreviations: 3D, three-dimensional; REM, rapid eye movement.

Following decompression, N3 sleep duration increased significantly from 106.4 ± 61.2 to 138.0 ± 60.9 min (p = 0.008), and total sleep time increased from 380.3 ± 82.5 to 410.2 ± 60.1 min (p = 0.044). No significant postoperative changes were observed in apnea–hypopnea index, central apnea index, or sleep efficiency (Table 2).

Table 2
Preoperative and Postoperative Changes in Polysomnographic Parameters After Decompressive Surgery

Parameter	Preoperative	Postoperative	p value
Body mass index, kg/m²	29.1 ± 6.8	29.8 ± 6.5	0.713
N1 sleep duration, min	23.2 ± 17.0	24.4 ± 16.7	0.713
N2 sleep duration, min	196.3 ± 81.2	185.5 ± 67.3	0.496
N3 sleep duration, min	106.4 ± 61.2	138.0 ± 60.9	**0.008**
REM sleep duration, min	54.0 ± 25.3	61.0 ± 27.4	0.164
Total sleep time, min	380.3 ± 82.5	410.2 ± 60.1	**0.044**
Sleep efficiency, %	82.2 ± 12.2	83.2 ± 11.3	0.656
Central apnea index, events/h	3.66 ± 12.32	1.58 ± 2.77	0.210
Mean central apnea duration, sec	11.7 ± 7.2	9.9 ± 6.4	0.116
Apnea–hypopnea index, events/h	8.38 ± 14.91	8.67 ± 16.50	0.891
Number of central apneas	21.9 ± 69.4	10.4 ± 18.6	0.195

Data are presented as mean ± standard deviation. Statistically significant p values are shown in bold. Abbreviations: REM, rapid eye movement.

Greater 3D Slicer-derived posterior fossa volume increase was associated with longer postoperative total sleep time (r = 0.293, p = 0.048). Larger postoperative 3D Slicer volume was associated with lower apnea–hypopnea index (r=-0.331, p = 0.028) and shorter mean central apnea duration (r=-0.309, p = 0.041) (Table 3).

Table 3
Correlations Between Posterior Fossa Decompressive Volume Parameters and Postoperative Polysomnographic Outcomes

Postoperative polysomnographic parameter	Manual postoperative posterior fossa volume	Manual posterior fossa volume increase	3D Slicer postoperative posterior fossa volume	3D Slicer posterior fossa volume increase	3D Slicer posterior fossa volume increase (%)
N3 sleep duration, min	r=-0.100	r=-0.014	r=-0.117	r=-0.027	r = 0.023
	p = 0.503	p = 0.926	p = 0.449	p = 0.856	p = 0.882
Total sleep time, min	r = 0.077	r = 0.245	r = 0.048	r = 0.268	r = 0.293
	p = 0.606	p = 0.097	p = 0.756	p = 0.071	p = **0.048**
Sleep efficiency, %	r=-0.008	r = 0.176	r=-0.008	r = 0.193	r = 0.231
	p = 0.956	p = 0.237	p = 0.958	p = 0.199	p = 0.122
Central apnea index, events/h	r=-0.235	r=-0.220	r=-0.221	r=-0.243	r=-0.189
	p = 0.112	p = 0.138	p = 0.149	p = 0.104	p = 0.208
Mean central apnea duration, sec	r=-0.371	r=-0.248	r=-0.309	r=-0.257	r=-0.145
	p = **0.010**	p = 0.092	p = **0.041**	p = 0.085	p = 0.337
Apnea–hypopnea index, events/h	r=-0.112	r=-0.061	r=-0.331	r=-0.060	r=-0.065
	p = 0.452	p = 0.683	p = **0.028**	p = 0.690	p = 0.667
Number of central apneas	r=-0.229	r=-0.188	r=-0.213	r=-0.211	r=-0.153
	p = 0.122	p = 0.205	p = 0.165	p = 0.160	p = 0.310

Data are presented as correlation coefficient and p value. Statistically significant p values are shown in bold. Abbreviations: 3D, three-dimensional.

Patients without syringomyelia had a higher postoperative central apnea index and a greater number of central apneas than those with syringomyelia, whereas decompressive volume gain did not differ between groups (Table 4).

Table 4
Comparison of Central Sleep Apnea-Related Parameters According to Syringomyelia Status

Parameter	Without syringomyelia	With syringomyelia	p value
Age, years	36.8 ± 12.9	38.5 ± 12.8	0.644
Preoperative tonsillar herniation distance, mm	11.7 ± 4.0	9.8 ± 4.0	0.115
Postoperative tonsillar herniation distance, mm	4.5 ± 5.0	4.0 ± 4.1	0.701
Posterior fossa height, cm	3.2 ± 0.3	3.4 ± 0.3	**0.028**
Posterior fossa transverse diameter, cm	11.0 ± 0.7	10.9 ± 0.6	0.348
Preoperative central apnea index, events/h	7.6 ± 18.4	0.7 ± 0.7	0.057
Preoperative number of central apneas	45.9 ± 102.9	4.2 ± 4.2	**0.040**
Postoperative central apnea index, events/h	2.7 ± 3.9	0.7 ± 0.9	**0.012**
Postoperative number of central apneas	17.8 ± 26.2	4.9 ± 6.0	**0.018**
3D Slicer posterior fossa volume increase, cm^3	23.2 ± 9.1	24.3 ± 10.9	0.722
3D Slicer posterior fossa volume increase, %	15.9 ± 6.7	16.0 ± 7.5	0.953

Data are presented as mean±standard deviation. Statistically significant p values are shown in bold. Abbreviations: 3D, three-dimensional.

Residual tonsillar herniation ≥ 6 mm was associated with differences in selected N2 and N3 sleep-stage parameters; however, postoperative total sleep time, sleep efficiency, apnea–hypopnea index, and central apnea index were comparable between groups (Table 5).

Table 5
Postoperative Polysomnographic Findings According to Residual Tonsillar Herniation

Parameter	Residual herniation < 6 mm	Residual herniation ≥ 6 mm	p value
Age, years	39.4 ± 11.2	34.9 ± 15.0	0.258
Preoperative tonsillar herniation distance, mm	9.6 ± 3.9	12.4 ± 4.0	**0.026**
Postoperative tonsillar herniation distance, mm	1.2 ± 1.7	9.0 ± 3.3	**< 0.001**
Preoperative N3 sleep duration, min	89.6 ± 50.6	136.1 ± 68.2	**0.011**
Preoperative total sleep time, min	358.4 ± 82.4	418.9 ± 69.1	**0.014**
Postoperative N2 sleep duration, min	203.84 ± 64.51	153.06 ± 61.15	**0.011**
Postoperative N3 sleep duration, min	123.8 ± 62.7	162.9 ± 50.0	**0.033**
Postoperative total sleep time, min	410.4 ± 47.3	409.8 ± 79.7	0.978
Postoperative sleep efficiency, %	82.9 ± 9.1	83.7 ± 14.7	0.814
Postoperative central apnea index, events/h	1.6 ± 2.8	1.5 ± 2.8	0.885
Postoperative apnea–hypopnea index, events/h	9.8 ± 18.4	6.7 ± 12.6	0.542
Postoperative number of central apneas	11.1 ± 17.4	9.2 ± 20.9	0.741

Data are presented as mean±standard deviation. Statistically significant p values are shown in bold. Residual tonsillar herniation was categorized using a 6-mm cutoff. Abbreviations: N2, non-rapid eye movement stage 2 sleep; N3, non-rapid eye movement stage 3 sleep.

In multivariable analysis, age independently predicted postoperative apnea–hypopnea index (β = 0.295, p = 0.011), whereas preoperative central apnea index independently predicted postoperative central apnea index (β = 0.093, p = 0.006). Postoperative 3D Slicer-derived posterior fossa volume was not an independent predictor of the evaluated sleep-disordered breathing outcomes (Table 6).

Table 6
Multivariable Linear Regression Analysis for Postoperative Sleep-Disordered Breathing Outcomes

Dependent variable	Predictor	β coefficient	95% CI	p value
Postoperative apnea–hypopnea index	Preoperative apnea–hypopnea index	0.143	-0.056 to 0.342	0.155
	Age, years	0.295	0.073 to 0.517	**0.011**
	Body mass index, kg/m²	0.086	-0.328 to 0.500	0.677
	Syringomyelia	-0.670	-5.825 to 4.486	0.794
	3D Slicer postoperative posterior fossa volume, cm³	-0.078	-0.182 to 0.026	0.137
Postoperative central apnea index	Preoperative central apnea index	0.093	0.029 to 0.156	**0.006**
	Age, years	0.047	-0.021 to 0.115	0.170
	Body mass index, kg/m²	-0.002	-0.129 to 0.125	0.970
	Syringomyelia	-1.462	-3.057 to 0.134	0.071
	3D Slicer postoperative posterior fossa volume, cm³	-0.005	-0.037 to 0.027	0.757
Mean postoperative central apnea duration	Preoperative mean central apnea duration	0.242	-0.019 to 0.502	0.068
	Age, years	0.140	-0.016 to 0.296	0.078
	Body mass index, kg/m²	-0.071	-0.370 to 0.228	0.632
	Syringomyelia	0.088	-3.519 to 3.695	0.961
	3D Slicer postoperative posterior fossa volume, cm³	-0.049	-0.121 to 0.023	0.179

Multivariable linear regression analysis was performed using complete-case analysis. β coefficients are unstandardized regression coefficients. Syringomyelia was coded as absent = 0 and present = 1. Statistically significant p values are shown in bold. Abbreviations: 3D, three-dimensional; CI, confidence interval.

Discussion

In this retrospective single-center study of adults with Chiari malformation type I, we evaluated the association between postoperative decompressive posterior fossa volume and polysomnographic outcomes. The most clinically relevant findings were that decompressive surgery was associated with significant increases in N3 sleep duration and total sleep time, while apnea–hypopnea index and central apnea index showed numerical but statistically non-significant postoperative changes. Importantly, volumetric parameters were associated with selected postoperative sleep outcomes: 3D Slicer-derived posterior fossa volume increase percentage was positively correlated with total sleep time, whereas larger postoperative posterior fossa volume was negatively correlated with apnea–hypopnea index and mean central apnea duration. In multivariable analysis, age independently predicted postoperative apnea–hypopnea index, and preoperative central apnea index independently predicted postoperative central apnea index. These findings suggest that decompressive surgery may improve sleep architecture in adults with CM-I and that volumetric assessment, particularly three-dimensional posterior fossa volumetry, may provide clinically meaningful information beyond conventional linear measurements.

Sleep-disordered breathing in CM-I is thought to result from complex interactions between hindbrain compression, impaired cerebrospinal fluid flow, and dysfunction of respiratory control pathways at the cervicomedullary junction. Gagnadoux et al. reported a high frequency of sleep apnea in patients with Chiari malformation and syringomyelia, with a substantial central apnea component, and demonstrated postoperative reduction in central apnea index after decompression [12]. In contrast, in our cohort, central apnea index decreased from 3.66 ± 12.32 to 1.58 ± 2.77 events/h but did not reach statistical significance, possibly because the baseline central apnea burden was lower and interindividual variability was high. Marino et al. emphasized that polysomnography is essential for identifying sleep-disordered breathing in Chiari malformation, particularly because symptoms and imaging severity may not reliably predict respiratory abnormalities [13]. Our findings support this concept by showing that objective PSG parameters, especially sleep architecture measures such as N3 sleep duration and total sleep time, may capture clinically relevant postoperative changes that are not fully reflected by apnea indices alone.

The postoperative improvement in N3 sleep duration and total sleep time observed in our study suggests that decompressive surgery may influence not only respiratory events but also global sleep organization. Sader et al. reported marked polysomnographic improvement after decompression in patients with Chiari 1.5 malformation and sleep-related breathing disorders, indicating that restoration of craniocervical junction anatomy may improve sleep physiology [14]. Similarly, Jarrell et al. evaluated sleep-disordered breathing in Chiari type I malformation and highlighted the potential role of foramen magnum decompression in improving sleep-related respiratory abnormalities, although outcomes may vary according to clinical and radiological characteristics [15]. Compared with these studies, our adult cohort showed a more selective pattern of improvement, with significant gains in N3 sleep duration and total sleep time rather than statistically significant reductions in apnea–hypopnea or central apnea indices. This difference may reflect age-related differences, baseline disease phenotype, timing of postoperative PSG, or the relatively mild mean apnea burden in our cohort.

A central strength of the present study is the use of both manual and 3D Slicer-based posterior fossa volumetry to quantify decompressive anatomical change. Kobets et al., in a systematic review of volumetric segmentation in posterior fossa-related pathologies, emphasized that segmentation-based approaches provide more detailed anatomical quantification than conventional two-dimensional measurements, although they may be influenced by operator-dependent variability and segmentation workload [16]. Mantha et al. also showed that CSF-space volumetric change after posterior fossa decompression may correlate with clinical outcome in Chiari type I malformation, supporting the concept that volumetric changes can be clinically meaningful [17]. In our study, 3D Slicer postoperative posterior fossa volume was negatively correlated with postoperative apnea–hypopnea index and mean central apnea duration, whereas 3D Slicer posterior fossa volume increase percentage was positively correlated with total sleep time. These findings suggest that segmentation-based volumetry may better capture functionally relevant decompressive effects than linear measurements alone.

Syringomyelia is an important radiological feature of CM-I and is often interpreted as a marker of impaired cerebrospinal fluid dynamics at the foramen magnum. Chotai et al. demonstrated that syrinx regression after posterior fossa decompression may occur early but can continue over time, indicating that postoperative structural recovery is dynamic rather than immediate [18]. Ciaramitaro et al. reviewed adult Chiari-related syringomyelia and emphasized the heterogeneity of clinical and radiological presentation in adults [19]. In our cohort, syringomyelia was present in more than half of the patients, but patients without syringomyelia had higher preoperative and postoperative central apnea burden. This finding is noteworthy because it suggests that sleep-related respiratory dysfunction in CM-I may not simply parallel the presence of syringomyelia. Instead, central apnea burden may be more closely related to brainstem compression, respiratory network vulnerability, or local craniocervical CSF-flow disturbance than to syrinx formation alone.

Residual postoperative tonsillar herniation was associated with differences in selected sleep-stage parameters but not with postoperative apnea–hypopnea index or central apnea index. Xiao et al. reported that combining cerebrospinal fluid hydrodynamics with fourth ventricle outlet morphology may improve prognostic prediction after CM-I decompression, underscoring that postoperative outcome cannot be explained by a single linear anatomical parameter [20]. El-Hajj et al. also reported that a subset of patients may continue to experience deficits or recurrent symptoms after posterior fossa decompression, supporting the need for multidimensional postoperative assessment [21]. Consistent with these observations, our findings indicate that residual tonsillar position alone may be insufficient to explain postoperative sleep-disordered breathing. The lack of independent association between 3D Slicer postoperative posterior fossa volume and PSG outcomes in multivariable models further suggests that postoperative sleep physiology is influenced by multiple factors, including age, baseline central apnea burden, body habitus, syringomyelia status, and the degree of functional rather than purely anatomical decompression.

The broader literature also supports a high but heterogeneous burden of sleep-related breathing disorders in Chiari malformation. Dauvilliers et al. found sleep apnea syndrome in 67.4% of 46 patients

with Chiari malformation, including 73% of adults, with both obstructive and central phenotypes and recommended systematic screening, particularly before surgery [22]. In a prospective controlled adult study, Botelho et al. reported higher apnea–hypopnea index values and a greater proportion of central apneas in patients with craniovertebral junction malformations than in healthy controls [23]. Henriques-Filho and Pratesi observed an abnormal apnea–hypopnea index in 65 of 103 patients with Chiari type I or II malformations and proposed that central apneas and REM sleep behavior disorder may be markers of brainstem dysfunction [24]. In a prospective cohort of 90 adults with CM-I/CM-1.5, Ferré et al. found sleep-related breathing disorders in 50%, predominantly with hypopneic/obstructive events; greater severity was associated with male sex, older age, excess weight, and hydrocephalus [25]. Leu similarly emphasized that CM-I-associated sleep-disordered breathing may be obstructive, central, or mixed and can occasionally be the presenting manifestation of the malformation [26]. These observations reinforce that respiratory involvement in CM-I is not restricted to central apnea and that polysomnographic findings should be interpreted within the broader clinical phenotype.

Additional studies also help contextualize the postoperative and radiological findings of the present cohort. Botelho et al. prospectively studied 25 adults with craniovertebral junction malformations and found that decompression significantly reduced total, obstructive, and central respiratory events; the central apnea index decreased from 13.81 to 1.68 events/h, whereas the reduction in mean apnea–hypopnea index from 26.68 to 12.98 events/h did not reach conventional statistical significance ($p = 0.06$) [27]. Spence et al. described two children in whom isolated central sleep apnea led to the diagnosis of CM-I and improved dramatically after posterior fossa decompression [28]. In pediatric cohorts, Dhamija et al. linked perimedullary subarachnoid-space effacement and greater tonsillar descent with higher central apnea and arousal indices [29], whereas Khatwa et al. found that sleep-disordered breathing was associated with greater foramen magnum crowding and tonsillar herniation, with postoperative improvement of central apnea in surgically treated children [30]. Finally, Ferré et al. developed and externally validated prediction models in cohorts of 90 and 74 patients, showing that sleep-related breathing disorder risk reflects a combination of age, sex, Chiari subtype, and clivus length rather than a single morphometric variable [31]. Taken together, these data are consistent with our finding that postoperative sleep physiology is multifactorial and may be better characterized by integrating polysomnography with quantitative and conventional imaging rather than relying on tonsillar position or decompressive volume alone.

This study has several limitations. Its retrospective, single-center design and relatively small sample size may limit the generalizability of the findings and reduce the statistical power to detect modest associations, particularly for apnea-related parameters with high variability. Although postoperative polysomnography was performed at least 2 months after surgery, the exact timing was not identical for all patients. Because sleep parameters and syringomyelia regression may continue to change over time, variability in follow-up timing may have influenced postoperative outcomes. Posterior fossa volume was assessed using both manual morphometric estimation and 3D Slicer-based segmentation; however, these measurements may be affected by image quality, slice thickness, anatomical boundary definition, and operator-dependent variability. Interobserver and intraobserver reliability analyses were not

performed. Potential confounding factors such as craniofacial anatomy, medication use, smoking status, cardiopulmonary comorbidities, and detailed symptom severity scores could not be fully controlled. In addition, standardized patient-reported outcome measures and validated sleep quality scales were not available. Prospective multicenter studies with larger cohorts, standardized follow-up intervals, reliability testing for volumetric measurements, and long-term clinical assessment are needed to confirm these findings.

Conclusion

In conclusion, decompressive surgery in adults with Chiari malformation type I was associated with significant improvement in selected polysomnographic parameters, particularly N3 sleep duration and total sleep time. Although postoperative apnea–hypopnea index and central apnea index did not show significant overall improvement, larger postoperative posterior fossa volume and greater decompressive volume gain were correlated with more favorable sleep-related outcomes, including longer total sleep time, lower apnea–hypopnea index, and shorter central apnea duration. These findings suggest that quantitative posterior fossa volumetry, especially 3D Slicer-based assessment, may provide additional value in evaluating the functional impact of decompressive surgery. Further prospective studies with larger cohorts and standardized long-term polysomnographic follow-up are needed to confirm the relationship between decompressive volume and sleep physiology in Chiari malformation type I.

Declarations

Conflict of Interest

The authors declare that they have no conflict of interest.

Funding

This study received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request, subject to institutional and ethical restrictions.

Ethical Approval

The study protocol was approved by the İnönü University Health Sciences Scientific Research Ethics Committee, Scientific Research and Publication Ethics Board (session no: 15; decision no: 2025/8371; date: November 18, 2025). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Author Contributions

H.E. contributed to the conception and design of the study, data collection, data analysis, visualization, and drafting of the manuscript. I.I.,S.S.,D.E.K.,M.N.O.,H.E.,G.M.D. contributed to data collection and data analysis. S.Ç.Ö. contributed to the study conception and design, supervision, interpretation of the data, and critical revision of the manuscript. All of the authors read and approved the final manuscript.

Informed Consent

Because of the retrospective design of the study, the requirement for written informed consent was waived by the ethics committee.

References

1. Bauer DF, Niazi T, Qaiser R, Infinger LK, Vachhrajani S, Ackerman LL et al (2023) Congress of Neurological Surgeons Systematic Review and Evidence-Based Guidelines for Patients With Chiari Malformation: Diagnosis. Neurosurgery 93(4):723–726
2. Zisakis A, Sun R, Pepper J, Tsermoulas G (2023) Chiari Malformation Type 1 in Adults. Adv Tech Stand Neurosurg 46:149–173
3. Langridge B, Phillips E, Choi D (2017) Chiari Malformation Type 1: A Systematic Review of Natural History and Conservative Management. World Neurosurg 104:213–219
4. Ciaramitaro P, Ferraris M, Massaro F, Garbossa D (2019) Clinical diagnosis-part I: what is really caused by Chiari I. Childs Nerv Syst 35(10):1673–1679
5. Abel F, Tahir MZ (2019) Role of sleep study in children with Chiari malformation and sleep disordered breathing. Childs Nerv Syst 35(10):1763–1768
6. Pomeraniec IJ, Ksendzovsky A, Yu PL, Jane JA (2015) Jr. Surgical History of Sleep Apnea in Pediatric Patients with Chiari Type 1 Malformation. Neurosurg Clin N Am 26(4):543–553
7. Seol J, Chiba S, Kawana F, Tsumoto S, Masaki M, Tominaga M et al (2024) Validation of sleep-staging accuracy for an in-home sleep electroencephalography device compared with simultaneous polysomnography in patients with obstructive sleep apnea. Sci Rep 14(1):3533
8. Patel SR, Genuardi MV, DeSensi R, Fudim M, Lund LH, Mehra R et al (2025) Real world characteristics of central sleep apnea: a review of 29,000 consecutive patients undergoing polysomnography. Sleep Breath 29(5):296
9. Lo JJ, Shiino S, Shapiro SZ, Hines BL, Komune N, Donofrio CA et al (2026) Chiari I Malformation: Review and Update of Current Treatment Options. Clin Anat
10. Ibrahimy A, Wu T, Mack J, Scott GC, Cortes MX, Cantor FK et al (2023) Prospective, Longitudinal Study of Clinical Outcome and Morphometric Posterior Fossa Changes after Craniocervical Decompression for Symptomatic Chiari I Malformation. AJNR Am J Neuroradiol 44(10):1150–1156
11. Huang Y, Huang J, Guan C, Liu T, Que S (2025) Volumetric measurement of cranial cavity and cerebral ventricular system with 3D Slicer software based on CT data. BMC Med Imaging 25(1):64

12. Gagnadoux F, Meslier N, Svab I, Menei P, Racineux JL (2006) Sleep-disordered breathing in patients with Chiari malformation: improvement after surgery. Neurology 66(1):136–138
13. Marino AC, Farzad F, Jane JA (2023) Jr. Chiari I Malformation and Sleep-Disordered Breathing. Neurosurg Clin N Am 34(1):35–41
14. Sader N, Hader W, Hockley A, Kirk V, Adeleye A, Riva-Cambrin J (2021) The relationship between Chiari 1.5 malformation and sleep-related breathing disorders on polysomnography. J Neurosurg Pediatr 27(4):452–458
15. Jarrell M, Caudill C, Haji F, Leon T, Rozzelle CJ, Maddox MH et al (2024) Sleep-disordered breathing in children with Chiari type I malformation. J Neurosurg Pediatr 34(4):393–401
16. Kobets AJ, Alavi SAN, Ahmad SJ, Castillo A, Young D, Minuti A et al (2024) Volumetric segmentation in the context of posterior fossa-related pathologies: a systematic review. Neurosurg Rev 47(1):170
17. Mantha S, Coulthard LG, Campbell R (2021) CSF-space volumetric change following posterior fossa decompression in paediatric Chiari type-I malformation: a correlation with outcome. Childs Nerv Syst 37(12):3861–3869
18. Chotai S, Chan EW, Ladner TR, Hale AT, Gannon SR, Shannon CN et al (2020) Timing of syrinx reduction and stabilization after posterior fossa decompression for pediatric Chiari malformation type I. J Neurosurg Pediatr 26(2):193–199
19. Ciaramitaro P, Migliaretti G, Ferraris M, Garnero A, Morana G, Carucci P et al (2023) Syringomyelia Associated with Chiari 1 Malformation in Adults: Positive Outcome Predictors after Posterior Fossa Decompression with Duraplasty. J Clin Med. ;12(8)
20. Xiao Y, Liu Y, Wang Z, He K, Zhang Z, Chen S et al (2023) Combined Cerebrospinal Fluid Hydrodynamics and Fourth Ventricle Outlet Morphology to Improve Predictive Efficiency of Prognosis for Chiari Malformation Type I Decompression. World Neurosurg 176:e208–e18
21. El-Hajj VG, Öhlén E, Sandvik U, Pettersson-Segerlind J, Atallah E, Jabbour P et al (2024) Long-term outcomes following posterior fossa decompression in pediatric patients with Chiari malformation type 1, a population-based cohort study. Acta Neurochir (Wien) 166(1):460
22. Dauvilliers Y, Stal V, Abril B, Coubes P, Bobin S, Touchon J et al (2007) Chiari malformation and sleep related breathing disorders. J Neurol Neurosurg Psychiatry 78(12):1344–1348
23. Botelho RV, Bittencourt LR, Rotta JM, Tufik S (2003) A prospective controlled study of sleep respiratory events in patients with craniovertebral junction malformation. J Neurosurg 99(6):1004–1009
24. Henriques-Filho PS, Pratesi R (2008) Sleep apnea and REM sleep behavior disorder in patients with Chiari malformations. Arq Neuropsiquiatr 66(2b):344–349
25. Ferré Á, Poca MA, de la Calzada MD, Moncho D, Romero O, Sampol G et al (2017) Sleep-Related Breathing Disorders in Chiari Malformation Type 1: A Prospective Study of 90 Patients. Sleep. ;40(6)
26. Leu RM (2015) Sleep-Related Breathing Disorders and the Chiari 1 Malformation. Chest 148(5):1346–1352

27. Botelho RV, Bittencourt LR, Rotta JM, Tufik S (2010) The effects of posterior fossa decompressive surgery in adult patients with Chiari malformation and sleep apnea. J Neurosurg 112(4):800–807
28. Spence J, Pasterkamp H, McDonald PJ (2010) Isolated central sleep apnea in type I Chiari malformation: improvement after surgery. Pediatr Pulmonol 45(11):1141–1144
29. Dhamija R, Wetjen NM, Slocumb NL, Mandrekar J, Kotagal S (2013) The role of nocturnal polysomnography in assessing children with Chiari type I malformation. Clin Neurol Neurosurg 115(9):1837–1841
30. Khatwa U, Ramgopal S, Mylavarapu A, Prabhu SP, Smith E, Proctor M et al (2013) MRI findings and sleep apnea in children with Chiari I malformation. Pediatr Neurol 48(4):299–307
31. Ferré Á, Poca MA, de la Calzada MD, Moncho D, Urbizu A, Romero O et al (2019) A Conditional Inference Tree Model for Predicting Sleep-Related Breathing Disorders in Patients With Chiari Malformation Type 1: Description and External Validation. J Clin Sleep Med 15(1):89–99

Figures

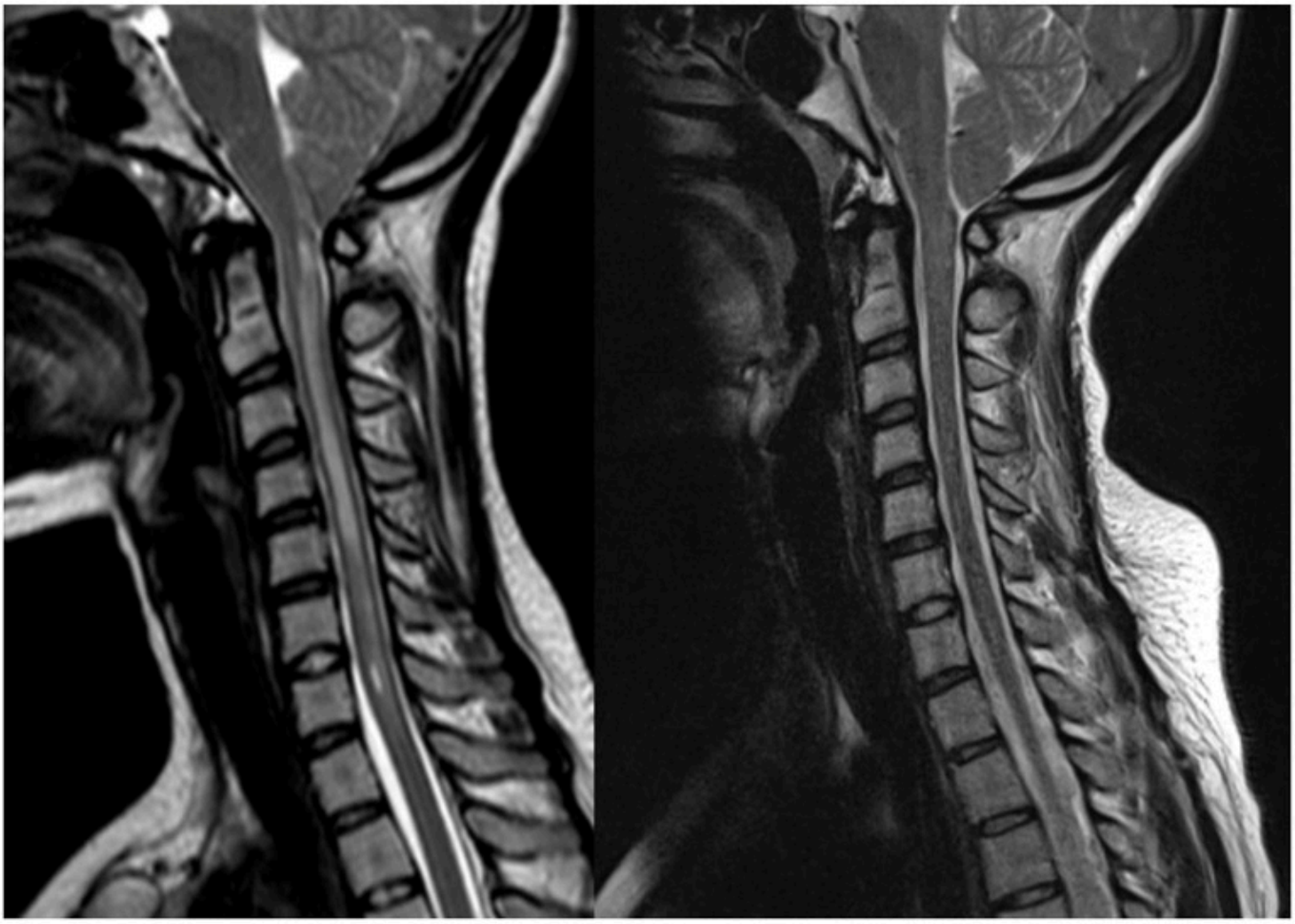


Figure 1

Preoperative and postoperative cervical T2-weighted sagittal magnetic resonance images of an adult patient with Chiari malformation type I and syringomyelia.

Left: Preoperative image showing prominent syringomyelia extending along the cervical spinal cord. Right: Postoperative cervical magnetic resonance image obtained in the second postoperative week after suboccipital craniectomy, C1 laminectomy, and duraplasty, demonstrating marked regression of syringomyelia.

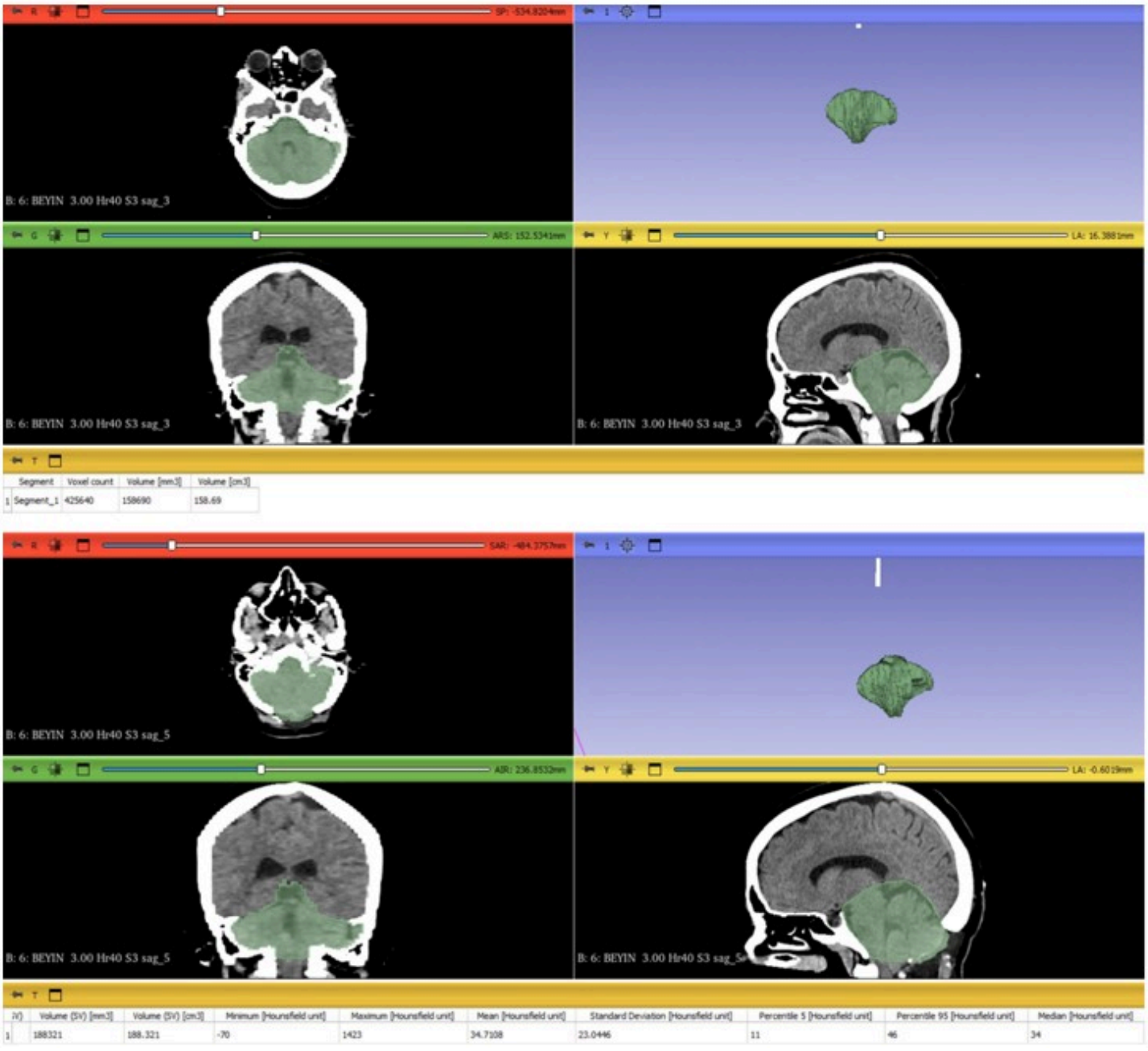


Figure 2

Three-dimensional segmentation-based posterior fossa volumetry using 3D Slicer software

Representative preoperative and postoperative non-contrast cranial computed tomography DICOM images of an adult patient with Chiari malformation type I demonstrating segmentation-based posterior fossa volume calculation. The upper panel shows preoperative multiplanar segmentation and three-dimensional modeling of the posterior fossa, with a measured posterior fossa volume of 158.69 cm^3. The lower panel shows postoperative posterior fossa segmentation after decompressive surgery, with a measured posterior fossa volume of 188.32 cm^3.