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Predecompression and postdecompression cognitive and affective changes in Chiari malformation type I

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OBJECTIVE The role of the cerebellum in cognitive function and psychiatric symptoms is poorly understood and particularly understudied in patients with cerebellar pathologies such as Chiari malformation type I (CM-I). Additionally, it is unclear if interventions targeted toward the cerebellum might impact these life-altering symptoms. The authors sought to characterize pre- and postoperative cognitive and psychiatric function in CM-I patients as evidence for targeted cerebellar treatment for some cognitive and psychiatric conditions.

METHODS This prospective study included surgical patients with CM-I who reported cognitive or psychiatric dysfunction. Patients completed a preoperative assessment and a parallel assessment 6 months following surgery. Neuropsychological evaluations included a 90-minute standardized assessment of cognitive function across multiple domains and a self-reported assessment of psychiatric symptoms. This clinical sample consisted of 54 patients (mean age 34.17 years, median 14.15 years). Any patient demonstrating preoperative performance below 3.5 SDs within any cognitive domain was excluded (n = 1). All patients underwent preoperative neuropsychological assessment comprising standard clinical tests of processing speed, attention, memory, executive function, and psychiatric symptoms.

RESULTS Preoperatively, CM-I patients performed significantly worse than a representative normative sample on measures of executive function and visuospatial memory and reported more psychiatric symptoms across all domains. On postoperative assessment, 89% of patients showed clinically significant improvements (> 1 SD) in cognitive and/or psychiatric domains.

CONCLUSIONS The authors demonstrate significant, often unrecognized, impairments in cognitive function and psychiatric symptoms in a cohort of CM-I patients. Following targeted surgical posterior fossa decompression, these symptoms improved, suggesting that at least in a subgroup of symptomatic CM-I patients, these symptoms may be treatable. This study highlights the potential role of the cerebellum in cognitive and psychiatric dysfunction.

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KEYWORDS Chiari; decompression surgery; cerebellum; neuropsychology; cognition; psychiatric

The role of the cerebellum in cognitive and psychiatric function is not well understood.[1,2] Given the reciprocal connections between the posterior cerebellum and the prefrontal cortex,[3,4] displaced and compressed caudal cerebellar structures might impact function.[5] Behavior modifying cerebellar lesions in animal models further supports a wholistic view of the cerebellum's neurological role.[6,7] Changes in patients with focal cerebellar pathologies (e.g., lesions) result in myriad cognitive and psychiatric changes, termed the "cerebellar cognitive affective syndrome" (CCAS).[8] This syndrome encompasses cognitive impairments (e.g., executive function, visuospatial function, and language), and psychiatric symptoms (e.g., depression, anxiety, and somatization disorders) from cerebellar pathology.[9,10]

Chiari malformation type I (CM-I) is a congenital or acquired structural condition where the cerebellar tonsils herniate through the foramen magnum (Fig. 1). Tonsillar

ABBREVIATIONS CM-I = Chiari malformation type I; FDR = false discovery rate; NPT = neuropsychological testing; PFD = posterior fossa decompression.

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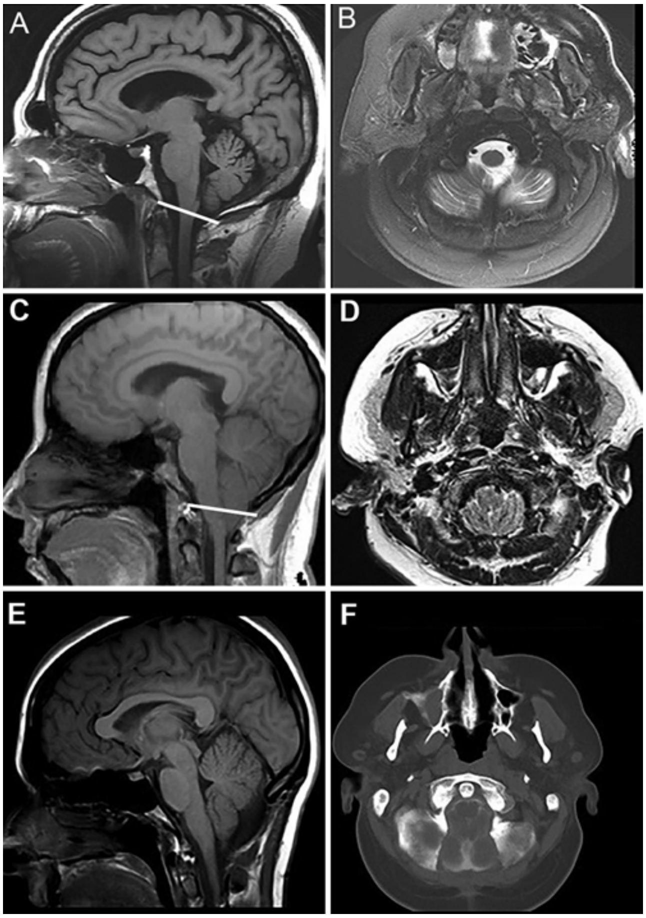

FIG. 1. A: Sagittal T1-weighted MRI sequence demonstrating a normal midline cerebellar position relative to the foramen magnum (*line*). **B:** Axial T2-weighted MRI sequence demonstrating a CSF (*white area*) signal around the cervicomedullary junction. **C:** Sagittal T1-weighted MRI sequence demonstrating a Chiari malformation with cerebellar descent below the foramen magnum (*line*). **D:** Axial T2-weighted MRI sequence demonstrating profound loss of CSF volume at the level of the foramen magnum due to cerebellar tissue crowding. **E:** Sagittal T1-weighted MRI sequence demonstrating postoperative expansile duraplasty and craniectomy providing space for unobstructed CSF flow and dramatic improvement in cerebellomedullary compression. **F:** CT myelogram demonstrating wide bony decompression of the foramen magnum promoting ventral and dorsal CSF continuity.

displacement results in cerebellar and, in some cases, cervicomedullary compression. Common CM-I symptoms include Valsalva-induced occipital headaches and upper-extremity paresthesia in addition to a broad spectrum of additional symptoms, including complaints about cognitive function.[5,11] CM-I results in a stereotypical mechanical compression localized to the foramen magnum. Studies evaluating cognitive function in CM-I demonstrate disparate findings with some commonalities emerging. Preoperatively, patients with CM-I have been shown to have deficits in memory[12–14] and attention.[14–17] Others investigating postoperative function showed dysfunction in executive function, verbal learning, and language,[13,18,19] and persistent visuospatial memory deficits before and after posterior fossa decompression (PFD).[20] However, while some studies employed standard measures,[14,21] conclusions are limited by the use of cognitive screening tools with low sensitivity, single domain analysis (e.g., memory), or the utilization of nonclinically validated measuring tools. Additionally, recent work has demonstrated the presence of psychiatric symptoms (i.e., depression and anxiety) in CM-I patients, highlighting them as potential symptomatological features.[14,16,21,22]

The present study characterizes pre- and postoperative cognitive and psychiatric function in a subset of CM-I patients who reported typical physical symptoms of CM-I and/or cognitive and psychiatric symptoms. Specifically, we assessed cognitive function and psychiatric symptoms across multiple domains and explored the impact of PFD on within-individual changes pre- to postoperatively and provide evidence for a potential role of cerebellar-mediated pathology in cognitive and psychological dysfunction in at least a subpopulation of CM-I patients.

Methods

Patients in the current study presented for evaluation at the University of Pittsburgh from 2015 to 2023. All patients reporting cognitive or psychiatric symptoms were offered neuropsychological testing (NPT) pre- and postoperatively and completed a comprehensive neurological examination, including MRI. Postoperative imaging was completed 3 or 6 months postoperatively. Of the 135 patients who completed preoperative NPT, 55 returned for postoperative NPT. There were no demographic or neuropsychological differences between patients who returned versus those who did not (Supplemental Table 1). A diagnosis of symptomatic CM-I was made based on reported symptoms and imaging. All patients underwent PFD by a single surgeon (R.M.F.). The neuropsychological battery was administered preoperatively, and patients were asked to return 6 months postoperatively to complete an assessment. Patients demonstrating preoperative performance below 3.5 SDs on any measure were excluded to eliminate the impact of extreme outliers and preserve a normal distribution verified using a modified z-score method that identified the same outlier.[23] Thus, 54 patients were included (mean age 34.2 years [range 16–57 years], median 14.2 years) (Supplemental Table 1). This prospective study was approved by the University of Pittsburgh Institutional Review Board and all patients provided written informed consent.

Study Design and Procedures

The goal of surgery in patients with symptomatic CM-I is to provide space in the posterior fossa to eliminate direct pressure on the inferior cerebellum, medulla oblongata, and upper spinal cord[24] and by reestablishing unobstructed CSF flow. Surgical goals were achieved by bone decompression of the foramen magnum, intradural exploration using an operating microscope with lysis of arachnoid of adhesions, tailored cautery tonsillar volumetric reduction, and expansile duraplasty. Progressively more during the study period, in most patients the C1 arch was preserved with intentional shaving of the top of the arch. Complete removal of the dorsal aspect of the C1 arch was reserved for patients with tonsillar herniation rostral to the C1 arch. Prior to PFD surgery, patients completed a standardized neuropsychological assessment of cognitive function and psychiatric symptoms (see Supplemental Methods and Tables). Six months postsurgery, 55 patients returned to complete a subsequent neuropsychological assessment. This included 1 patient who was excluded due to preoperative performance that fell below 3.5 SDs on two measures, resulting in a sample of 54 (mean age 34.2 years [SD 10.6 years], range 16–57 years).

Neuropsychological Assessment

Cognitive function was evaluated across several domains: executive function (set-shifting, inhibition, and feedback integration), attention (visual scanning, simple attention, and complex attention), processing speed (psychomotor speed and information processing), visuospatial memory (immediate recall and delayed recall), verbal memory (verbal learning and delayed recall), and language (phonemic fluency and semantic fluency) (see Supplemental Methods and Tables). This battery was administered by a neuropsychologist (L.C.H.) preoperatively and approximately 6 months postoperatively. All raw scores were converted to standardized z-scores, which represent performance relative to age-, education-, and sex-based normative samples. To assess potential within-individual change, scores were examined pre- and postoperatively.

Psychiatric symptoms were assessed using a self-report questionnaire that included 53 items assessing psychiatric symptoms across multiple domains including depression, anxiety, somatization, obsessive-compulsive behavior, and hostility (see Supplemental Methods and Tables).

Statistical Analysis

All data were analyzed using IBM SPSS version 28 (IBM Corp.). First, to characterize cognitive function and psychiatric symptoms preoperatively, z-score means and SDs are reported for all measures and represent performance relative to a representative normative sample, with 0 representing the normative sample mean. Next, one-sample t-tests were used to assess whether any measures of cognitive function or psychiatric symptoms significantly differed from the norm-based means. Finally, to assess within-individual change from pre- to postoperative assessment (i.e., postoperative score − preoperative score), paired-samples t-tests were utilized. A false discovery rate (FDR) correction was applied for all statistical comparisons. For domains with significant preoperative deficits and significant pre- to postoperative change, analyses were conducted to characterize patterns of clinically significant improvement, defined as change > 1 SD, a commonly used metric.[25] Chi-square tests were used to examine whether preoperative scores (below vs above the median) were associated with clinically significant improvements (> 1 SD). The current study was not preregistered. Demographic characteristics are described in Supplemental Table 1.

Results

Preoperative Assessment

Table 1 presents means and SDs for preoperative assessments across all cognitive and psychiatric domains, as well as results from one-sample t-tests. An FDR correction was implemented to control multiple comparisons.

Cognitive Function

Patients exhibited lower performance relative to the normative sample on an executive function measure of feedback integration and visuospatial memory (immediate recall and delayed recall). Patients in the current sample performed better than the normative sample on a processing speed measure of simple psychomotor speed and on a language measure of semantic verbal fluency.

Psychiatric Symptoms

In the sample, patients were significantly more impaired than the normative sample across all domains including depression, anxiety, somatization, obsessive-compulsive behavior, and hostility.

Postoperative Assessment

Analyses were conducted to characterize patterns of clinically significant change following surgery, defined as change > 1 SD. Most patients in the sample (48/54, 89%) showed clinically significant improvements in either cognitive or psychiatric domains. Approximately one-third of patients (17/54) demonstrated improvements in both domains by the 6-month follow-up assessment. Within the sample, 56% (30/54) of patients demonstrated clinically significant improvement in visuospatial memory (immediate recall and delayed recall). Similarly, 65% (35/54) of patients demonstrated clinically significant improvement in at least one psychiatric domain.

Table 1 presents means and SDs for postoperative assessments across all cognitive and psychiatric domains. Paired-samples t-tests were conducted to determine the impact of surgery on cognitive function and psychiatric symptoms. An FDR correction was implemented to control for multiple comparisons.

Cognitive Function

Patients demonstrated improvements on visuospatial memory measures of immediate recall (Fig. 2A) and delayed recall (Fig. 2B).

Psychiatric Symptoms

Patients evidenced significant improvements across psychiatric domains of depression, anxiety (Fig. 3B), somatization (Fig. 3C), obsessive-compulsive (Fig. 3D), and hostility (Fig. 3E) symptoms. Preoperatively, patients reported significant distress in each of these domains when compared with the normative sample (Table 1); however, postoperatively, depression, anxiety, and hostility symptoms no longer differed from the normative sample. Most patients with multiple psychiatric symptoms improved in multiple domains (Supplemental Table 3). Postoperative somatization and obsessive-compulsive symptoms also improved, although they remained clinically elevated relative to the normative sample.

Magnitude of Effect

Chi-square tests were conducted to assess associa-

TABLE 1. Preoperative and postoperative cognitive function and psychiatric symptoms in 54 surgical patients with CM-I

	Norm-Based Comparisons								Pre- to Postop Change			
	Preop				Postop							
	Mean	SD	t-Statistic	p Value	Mean	SD	t-Statistic	p Value	Mean Δ	SD	t-Statistic	p Value
Estimated IQ	0.21	0.75	2.08	0.28	0.33	0.74	1.2	0.09	0.12	0.29	−2.39	0.26
Cognitive domains												
Executive function												
Set shifting	0.19	1.14	1.20	0.28	0.47	0.99	**3.40***	**0.004**	−0.27	0.89	−2.22	0.063
Inhibition	0.23	0.69	**2.44***	**0.03**	0.44	0.86	**3.78***	**0.001**	−0.21	0.70	−2.21	0.063
Feedback integration	−0.42	0.85	**−3.65†**	**0.002**	−0.29	0.79	**−2.7†**	**0.02**	−0.13	0.71	−1.37	0.235
Attention												
Visual scanning	0.24	1.06	1.68	0.14	0.28	1.03	1.99	0.086	0.04	0.86	−0.31	0.796
Simple attention	−0.22	0.85	−1.87	0.11	−0.21	0.85	−1.79	0.122	0.01	0.79	−0.08	0.936
Complex attention	0.04	0.92	0.34	0.74	0.10	0.94	0.77	0.525	0.06	0.97	−4.2	0.752
Processing speed												
Psychomotor speed	0.46	0.98	**3.43***	**0.003**	0.67	0.81	**6.03***	**<0.0001**	0.21	−1.67	−0.39	0.184
Information processing	−0.17	0.94	−1.35	0.23	−0.07	0.97	−0.52	0.67	0.10	−0.75	0.42	0.551
Visuospatial memory												
Immediate recall	−0.53	1.28	**−3.04†**	**0.007**	0.49	0.92	**3.87***	**0.001**	1.02	1.37	**−5.44‡**	**<0.001**
Delayed recall	−0.51	1.17	**−3.18†**	**0.005**	0.43	0.96	**3.27***	**0.005**	0.94	1.32	**−5.22‡**	**<0.001**
Verbal memory												
Verbal learning	0.15	1.17	0.93	0.39	0.51	1.29	**2.88***	**0.013**	0.36	1.10	−2.38	0.052
Short delay	−0.26	1.16	−1.64	0.14	0.01	1.33	0.05	0.96	0.27	1.28	−1.54	0.208
Long delay	−0.29	1.23	−1.71	0.65	−0.06	1.23	−0.33	0.78	0.23	1.22	−1.39	0.235
Language												
Phonemic fluency	0.07	1.04	0.50	0.65	0.15	1.09	1.01	0.40	0.08	0.80	−7.3	0.551
Semantic fluency	0.43	0.99	**3.19***	**0.05**	0.63	1.18	**3.95***	**0.001**	0.20	0.99	−1.52	0.208
Psychiatric domains												
Depression	0.66	1.02	**4.76§**	**<0.0001**	0.16	1.04	1.15	0.34	−0.50	0.89	**4.14‡**	**0.0004**
Anxiety	0.87	1.08	**5.94§**	**<0.0001**	0.31	1.15	1.99	0.09	−0.56	0.95	**4.34‡**	**0.0003**
Somatization	1.62	0.96	**12.43§**	**<0.0001**	0.81	1.19	**5.01§**	**<0.0001**	−0.81	1.11	**5.37‡**	**<0.0001**
Obsessive-compulsive	1.81	1.06	**12.55§**	**<0.0001**	1.03	1.19	**6.39§**	**<0.0001**	−0.77	1.16	**4.89‡**	**<0.0001**
Hostility	0.69	0.90	**5.66**	**<0.0001**	0.21	0.99	1.5	0.19	−0.49	0.86	**4.17‡**	**0.0004**

Boldface type indicates statistical significance.
* Higher performance than the normative sample performance.
† Lower performance than the normative sample.
§ Greater psychiatric symptoms than the normative sample.
‡ Significant change from pre- to postoperative performance.

tions between preoperative function and the magnitude of clinically significant improvements. Patients who performed the worst preoperatively evidenced the most clinically meaningful improvement (> 1 SD). Among patients whose visuospatial memory performance was below the mean preoperatively, 81% (21/26 patients; $\chi^2 = 18.9$, $p < 0.001$) demonstrated clinically meaningful improvement on immediate recall and 77% (20/26 patients; $\chi^2 = 14.5$, $p < 0.001$) on delayed recall. Within neuropsychiatric symptoms, two domains emerged showing a significant association between preoperative distress and degree of change. First, those with higher reported symptoms of depression evidenced the greatest reduction of associated distress postoperatively with 82% (9/11; $\chi^2 = 7.0$, $p = 0.008$) evidencing clinically meaningful change. Likewise, 79% (15/19) of patients with the greatest degree of distress over cognitive complaints showed the greatest reduction of symptom burden ($\chi^2 = 6.5$, $p = 0.021$).

Discussion

We demonstrate that a patient population with an isolated cerebellar pathology differs from a representative normative sample across domains of cognitive and psychiatric function. In this cohort, 89% (48/54) of CM-I patients with cognitive or psychiatric symptoms who underwent PFD evidenced clinically significant improvement (> 1 SD) in cognitive function and/or reductions in psychiatric symptoms. Specifically, 56% (30/54) of patients evidenced improved cognitive function, 65% (35/54) evi-

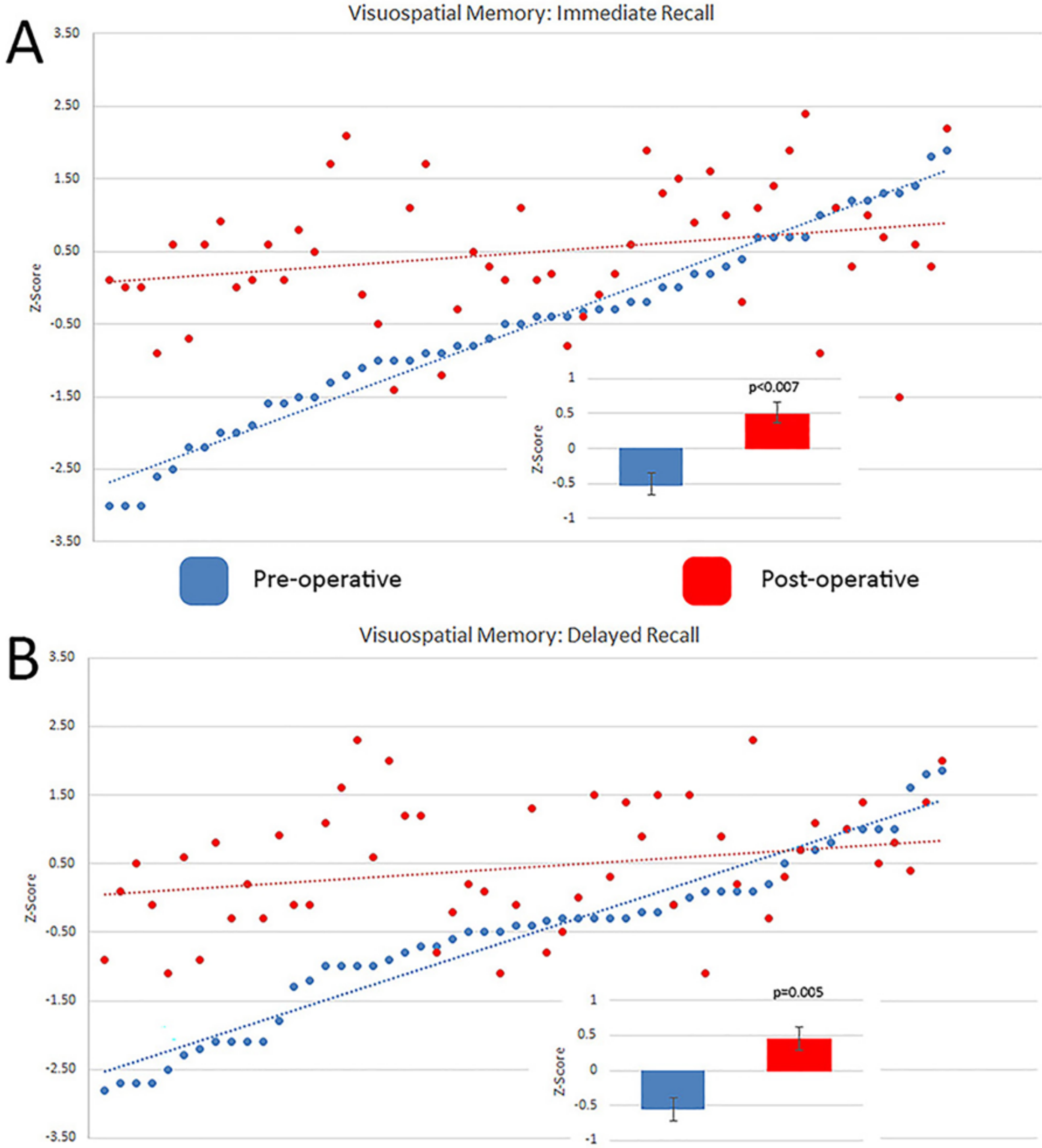

FIG. 2. Pre- and postoperative patient performance on a visuospatial memory task. Each pair of *blue* (preoperative) and *red* (postoperative) *dots* represent scores from the same patient, with lower z-scores indicating poorer performance and higher z-scores indicating better performance. Panel **insets** show the paired-samples t-test demonstrating significant within-individual improvements in visual memory immediate delay (**A,** *upper*), and delayed recall (**B,** *lower*) by 6-month postoperative follow-up. Figure is available in color online only.

denced significant reduction in psychiatric symptoms, and 31% (17/54) evidenced improved cognitive function and reduced psychiatric symptoms.

The current results join burgeoning work demonstrating the underestimated role of the cerebellum in cognition[12–14,18,20,26] and affect.[22,27–29] Cognitive function across multiple domains (executive function and visuospatial memory) differed relative to normative samples. This is broadly consistent with other studies[13–16,18,21,30] showing differences in executive function[13,16,18,21] and visuospatial memory.[20,31] Additionally, elevated psychiatric symptoms were observed preoperatively, including depression, anxiety, somatization, obsessive-compulsive behavior, and hostility, broadly consistent with other work.[18,22,27] Our observation of diminished visuospatial memory is consistent with the role of the cerebellum in visuospatial organization/memory[20,32,33] and prior work showing the role of the cerebellum in implicit learning and visuospatial process-

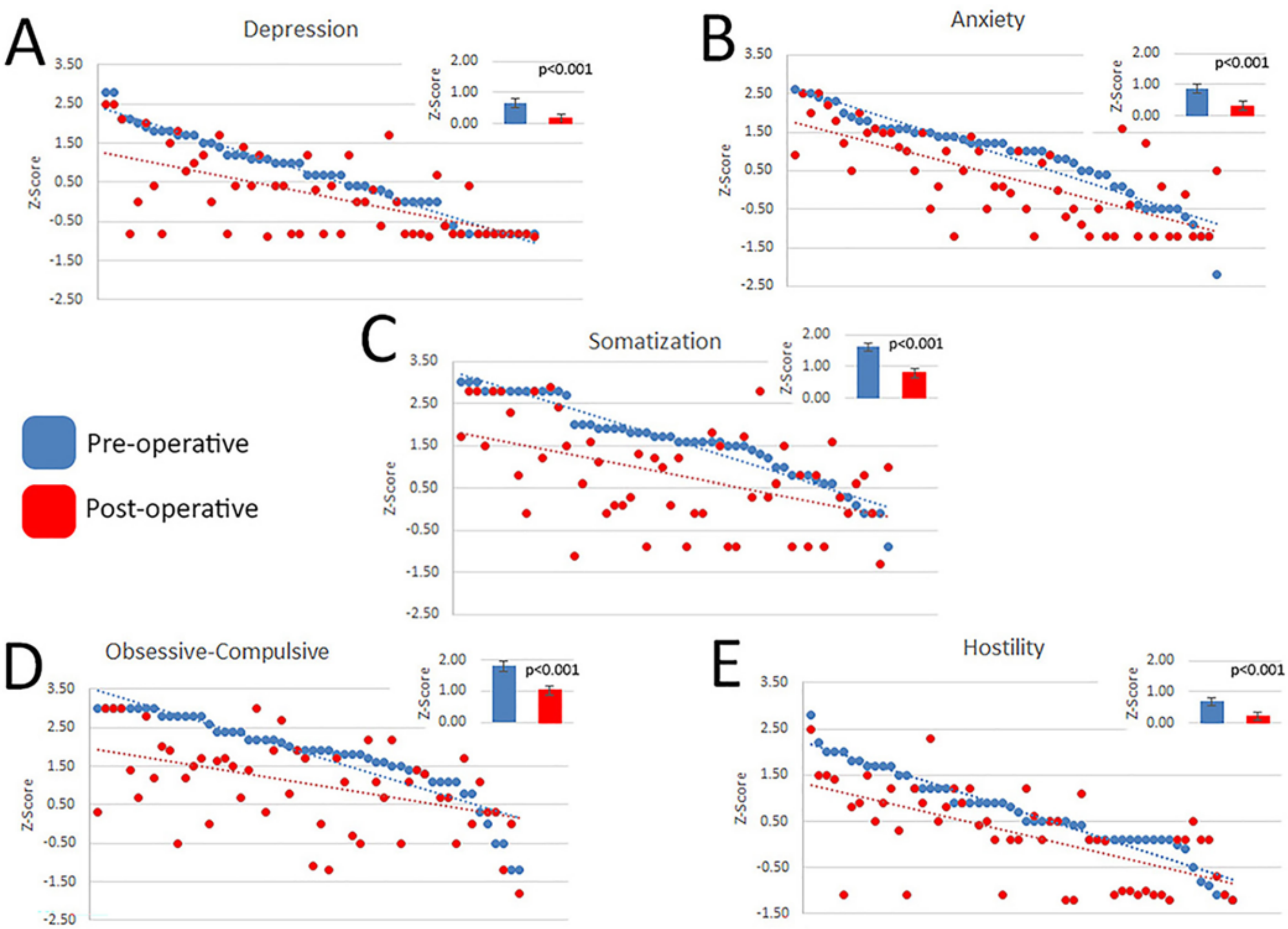

FIG. 3. Pre- and postoperative symptom levels across all psychiatric domains: depression (**A**), anxiety (**B**), somatization (**C**), obsessive-compulsive behavior (**D**), and hostility (**E**). Each pair of *blue* (preoperative) and *red* (postoperative) *dots* represent scores from the same patient, with higher z-scores indicating more symptoms and lower z-scores indicating fewer symptoms. Panel **insets** show results from the paired-samples t-test and demonstrate significant within-individual improvements across all psychiatric domains by the 6-month postoperative follow-up. Figure is available in color online only.

ing and attention.[34–36] While practice effects and regression (or progression) to the mean are ever-present factors in interpreting change, the stability of all other cognitive measures suggests that the improved visuospatial memory is not primarily driven by these factors; furthermore, lower scores may be less prone to drifting toward the norm.[25]

Critically, the patients in this cohort exhibit significant psychiatric symptoms, with higher levels of depression, anxiety, somatization, obsessive-compulsive behavior, and hostility relative to normative samples. Several studies have also shown affective dysfunction in CM-I patients,[13,18,22,27,30,31] with anxiety and depression commonly reported. Changes in affect are well described as part of the manifestation of injury and pathology in the vermis and perivermian regions of the cerebellum,[10,37,38] playing a critical role in affect regulation via direct connections to the ventral tegmental and limbic areas.[10,38,39] Accordingly, the lack of broad recognition of psychiatric distress in such patients may result in misinterpretation or misattribution of their presentation as psychosomatic by medical providers.

While some studies have compared pre- and postoperative cognitive function in CM-I,[12,13,20–22,27,40] only one other study has used a within-patient design.[20] Unique to our study, patients who performed the most poorly preoperatively demonstrated improved visuospatial memory. The other study, using a similar within-group design, did not show improvement of visuospatial deficits, and changes were not seen across other domains.[20] Although the potential impact of a placebo effect cannot be ruled out, a key surgical difference might provide additional insight. Bone-only decompression provides some decompression with purportedly less morbidity but has an inferior efficacy rate due to a less thorough decompression.[41] Seaman et al.[20] provided fortuitus insight, as most patients (13/19) in that study underwent a bone-only decompression and while all experienced resolution of their headaches, no improvement in cognitive or psychiatric function was observed in any patient, suggesting that headache resolution in and of itself did not result in cognitive or psychiatric improvement. In the Seaman et al. study, the visuospatial memory test results are not reported between the different (intra- vs extradural) surgical groups, and the much smaller sample might also account for their reported stability in cognitive functions. By contrast, in the current study 30 of 54 patients evidenced significantly improved visuospatial memory, all of whom underwent an intradural procedure with direct cerebellar decompression and thorough confirmation of CSF pathways at the fourth ventricle. During each surgery, extensive dissection of the foramen magnum to the edge of the paracondylar protuberances was performed, with all medial bone being removed. A wide duraplasty was performed with additional decompression provided by lysing all arachnoid tonsillar adhesions, fourth ventricle obstructive membranes were resected when present, and when appropriate, gentle cauterization of the pia to promote rostral contraction to achieve focal foramen magnum decompression. The difference between bone-only and intradural decompression might be related to a greater degree of decompression,[42] thereby lowering the likelihood that the current results are purely the result of a placebo effect. Interestingly, in the current sample, patients who performed most poorly preoperatively generally demonstrated the greatest improvements postoperatively. The predictive utility of cognitive factors on surgical outcome and recovery remains unclear, although continued research may provide important prognostic information.

Studies have demonstrated behavior changes, especially aggression, with cerebellar lesioning in animal models.[6,7] Others have identified cerebellar dysfunction as a contributor to social inflexibility and dysfunction in a range of conditions.[43] Higher levels of psychiatric symptoms are commonly reported in patients with cerebellar pathology. For example, emotional lability is classically associated with iatrogenic injury to the cerebellar vermis.[44] In CM-I patients,[14,16,21,27] a small number of studies have demonstrated post-PFD changes in elevated psychiatric symptoms.[22,27] In this cohort, all psychiatric domains (depression, anxiety, somatization, obsessive-compulsive behavior, and hostility) showed significant improvement after PFD. Following PFD, depression, anxiety, and hostility normalized, and while somatization and obsessive-compulsive symptoms improved, they did not normalize.

One common challenge to surgical studies is the rarity of a sham control group, and the same is true of the current study. Insight on the magnitude of placebo effects on psychiatric symptoms can be taken from the deep brain stimulation literature.[45,46] For example, Holtzheimer et al.[46] found that at 6 months, 20% of sham control patients reported a 40% or greater improvement in depression symptoms, corresponding to approximately a 1-SD improvement among cohort participants. This is lower than the 65% of patients with greater than 1 SD in at least one psychiatric domain and 58% with the same magnitude of improvement in at least one cognitive domain within our cohort. Furthermore, the fact that all patients in the Seaman et al. study[20] experienced headache resolution and no improvement in cognitive or psychological symptoms provide further evidence that the cognitive and psychological improvement in our study is unlikely to be a sole function of headache reduction/resolution.

The precise mechanism of CM-I–induced cognitive and psychiatric alterations and PFD-related improvement is unclear. The differential impacts of episodic elevated intracranial pressure and direct mechanical compression of the cerebellum on neural function cannot be delineated based on the current results, and the postoperative improvements cannot be attributed to the alleviation of one over the other. Persistent or transient intracranial pressure elevation in CM-I may impact cognition and psychiatric symptoms. Future work investigating the interplay between neuroanatomical and cognitive aspects of CM-I will offer valuable prognostic indicators for PFD outcome. While open questions remain, we demonstrate that cognitive and psychiatric dysfunction, often underappreciated components of the condition, affect patients with cerebellar-specific pathologies such as CM-I. Critically, this dysfunction is not fixed and may be reversible in some cerebellar conditions. This work provides a foundation for more in-depth studies to delineate the proportion of patients with cognitive and psychiatric symptoms attributable to cerebellar pathologies to help delineate a specific syndrome in this condition and likelihood of improvement with PFD or other targeted cerebellar therapies.

Limitations

This study is limited by the lack of a control group. Adding healthy and nonsurgical control groups will help delineate precise aspects of CM-I, separating nonspecific aspects of headache pain, psychiatric symptoms, and surgical intervention. Also, a specific headache pain scale was not used for all patients, and including this is critical for future work given the prevalence of this symptom and its valence (along with other physical symptoms) as a part of outcome. Pain specifically is important to consider as a factor in the improved outcomes after PFD where chronic pain does exert influence on cognitive performance across multiple cognitive domains such as processing speed, attention, and memory.[23] Pain/symptom reduction is likely a factor in the current results,[47] although the specific nature of the cognitive deficits and changes in the current study and the absence of broader cognitive dysfunction reflect a more CM-I–specific finding. When noted in the chart, pre- and postoperative headaches, National Institutes of Health pain scales, presence of dysphagia, and upper-extremity paresthesias were compared and all statistically improved (Supplemental Table 2). Importantly, not all patients returned for postoperative neuropsychological evaluation, potentially biasing the magnitude of the effect. However, it is clear from the data that at least a proportion of patients with cognitive or psychiatric symptoms improve following PFD and that our available headache data are equivalent between patients who returned for postoperative neuropsychological assessment and those who did not (Supplemental Table 2). In the same vein, this cohort consisted of patients who are self-reporting cognitive dysfunction and therefore might not be reflective of all patients with CM-I and might also present a confirmation bias given their reported symptomatology. That said, perceived cognitive dysfunction does not necessarily equate to objective deficits, as reported cognitive problems do not necessarily correlate with measured function in neurological populations.[48,49] We acknowledge that at present, we do not endorse PFD in CM-I patients with neuropsychiatric complaints but without any of the other well-established CM-I symptoms. This is an important question that requires additional study. Further, while methodological advantages exist from a single-surgeon/-institution study, generalizability is a relative limitation. Finally, in the current study we are unable to comment specifically on the direct physiological changes from surgery (e.g., tractography) on cognition, a promising avenue of future study.

Conclusions

We demonstrate significant, often unrecognized, impairments in cognitive function and psychiatric symptoms in a cohort of patients with focal cerebellar pathology. Following targeted surgical intervention relieving cerebellar compression and thorough reestablishment of CSF flow, these symptoms can be alleviated. This study highlights the role of the cerebellum in cognitive and psychiatric dysfunction and suggests that cerebellar pathology should be considered as a potential neuroanatomical site for similar dysfunction from other etiologies.

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Conception and design: Friedlander, Henry, McDowell, Crittenden, Nowicki. Acquisition of data: Friedlander, Henry, McDowell, Stephenson, Fernández-de Thomas, Nowicki, Mantena. Analysis and interpretation of data: Friedlander, Henry, McDowell, Crittenden, Byrd, Fernández-de Thomas, Nowicki, Strick. Drafting the article: Henry, McDowell, Crittenden, Byrd, Nowicki. Critically revising the article: Henry, McDowell, Fernández-de Thomas, Nowicki. Reviewed submitted version of manuscript: Friedlander, Henry, McDowell, Stephenson, Fernández-de Thomas, Chang, Nowicki, Strick. Approved the final version of the manuscript on behalf of all authors: Friedlander. Statistical analysis: Friedlander, Henry, Crittenden, Byrd. Administrative/technical/material support: Henry, Stephenson. Study supervision: Friedlander, Henry.

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