Cerebral cavernous malformations (CCMs) are common vascular lesions in the brain affecting 0.1%–0.5% of the population.17,33 CCMs consist of multilobulated abnormal blood vessels lined with endothelial cells without mature vessel walls and may occur in either sporadic or familial forms. Approximately 20% of all cases present with multiple CCMs, which are typically associated with autosomal dominant inheritance.3,4,13 Patients with CCMs are at risk for epileptic seizures, intracerebral hemorrhage, and focal neurological deficits.2,21 Seizures present as the initial symptom in 40%–70% of patients with supratentorial CCMs, and 30%–40% of these cases become resistant to treatment with antiepileptic drugs (AEDs).18,26,30 Surgical resection of CCMs along with perilesional hemosiderin treatment achieves seizure freedom in 70%–80% of patients with a solitary CCM,6,31 although poorer results have been reported in cases with multiple CCM lesions.11,23

MRI-guided laser interstitial thermal therapy (LITT) is a minimally invasive technique that has been used in
the treatment of focal epilepsy, such as mesial temporal lobe epilepsy, hypothalamic hamartoma, CCMs, tuberous sclerosis, insular epilepsy, and focal cortical dysplasia.22,29 Emerging evidence demonstrates that LITT can be a safe and effective alternative to traditional open surgery, with advantages including real-time monitoring of ablation volume with MRI thermometry, protection of off-target sites with temperature thresholds, and minimal patient discomfort. The use of LITT in epilepsy treatment has been most studied in patients with hippocampal sclerosis, in whom the aggregate rate of Engel I outcome is 68%,15.16,19,22,24,29,32 which is similar to the reported rates of 68% for selective amygdalohippocampectomy and 72% for anterior temporal lobectomy.20

Recently, Willie et al. reported a case series of 19 patients with CCM-related epilepsy who underwent stereotactic laser ablation of solitary lesions and achieved seizure outcomes similar to those of open surgery, with minimal adverse effects. No genetic characterization of these patients was reported.30 Here, we provide complementary evidence for the safety and effectiveness of LITT in a series of 6 patients with distinct clinical and genetic features and over 12 months of postoperative follow-up in an independent epilepsy center. This series is to our knowledge the first investigation of LITT to include patients with multifocal CCM lesions.

Methods

Patient Selection

With the approval of the University of Chicago Institutional Review Board, we retrospectively reviewed all patients with CCM-related drug-resistant focal epilepsy who underwent MRI-guided stereotactic LITT at the University of Chicago Medical Center from March 2016 to August 2018. All patients received a standard presurgical evaluation, which included a comprehensive neurological history and examination and inpatient video-EEG monitoring. Brain MRI in each patient showed a CCM with a characteristic “popcorn” appearance, with a rim of hypointensity on T2-weighted sequences and prominent blooming artifact on susceptibility-weighted sequences indicating the presence of hemosiderin. FDG-PET, neuropsychological evaluation, functional MRI (fMRI), and diffusion tensor imaging (DTI) were performed when clinically warranted. In cases of discrepancy between surface EEG and seizure semiology or cavernoma location, stereoelectroencephalography (SEEG) evaluation with depth electrodes was performed.

Patients were offered LITT if the epileptogenic CCM was less than 1.5 cm in diameter (since tissue ablation is limited to within 0.75 cm of the laser fiber), with no evidence of recent hemorrhage. For comparison, during the study period, 5 patients with CCM-related epilepsy underwent open surgical lesionectomy with or without additional perilesional resection. All of these patients were excluded from LITT due to large lesion size and/or recent hemorrhage.

Stereotactic Laser Ablation

Patients were placed in an MRI-compatible stereotactic head frame (CRW Integra Neurosciences). A 1.0-mm-section stereotactic CT was obtained intraoperatively and subsequently fused with 1.0-mm-section volumetric gadolinium-enhanced T1- and T2-weighted MR images using StealthStation S7 (Medtronic). Intravenous antibiotics were administered at the beginning of each procedure. With the patient under generalized anesthesia, an applicator assembly consisting of a 1.6-mm-diameter outer polycarbonate cooling catheter containing an inner 400-μm-diameter optical fiber (Visualase Medtronic) was implanted to target CCMs via a 3.2-mm twist drill perforation. At the end of the implantation, another intraoperative CT was obtained to confirm the laser fiber position, and coregistration to the MRI was again performed to ensure exact fiber position in the planned trajectory to the targeted CCMs. The patient was then transferred from the operating room to a dedicated 3-Tesla MRI scanner (Philips) for MRI-guided thermal ablation.29

A low-power (5 W) test ablation was performed to visualize the expected heat distribution from the fiber and test the phase-shift imaging for temperature monitoring at temperatures between 37°C and 80°C. Real-time MR images of the expected damage around the probe, calculated with the Arrhenius equation, were monitored to estimate the volume of permanent damage based on the identification of voxels exposed to 70°C, i.e., all voxels within the 70°C isothermic line. LITT resulted in an ellipsoid ablation volume as assessed by intraoperative real-time MRI. The goal of ablation was to include the targeted CCM and adjacent hemosiderin-stained brain in the volume heated to a minimum of 70°C. For lesions adjacent to eloquent cortex or major white matter tracts (e.g., the optic radiation), preoperative fMRI or DTI was used in the placement of virtual safety markers, which triggered automatic laser shutoff if any marker reached a temperature of 43°C.

At the completion of the ablation procedure, a 1.0-mm-section gadolinium-enhanced T1-weighted sequence with contrast was acquired to assess the final ablation volume (Fig. 1). The laser probe and cranial fixation device were then removed. The stab incision was closed with a single suture. Patients were subsequently transferred to the neurointensive care unit for postoperative care and treated prophylactically with a 3-day taper of dexamethasone to reduce postablative edema. All patients were treated by the same neurosurgeon (P.C.W.).

Assessment of Ablation Volume

Pre-, intra-, and postoperative MR images were analyzed in Visage 7 (Visage Imaging). Preablation CCM volumes were measured by tracing the T2-hypointense hemosiderin rim on each slice. Immediate postablation volumes were measured by tracing the enhancing borders on gadolinium-contrast T1-weighted images, which signify the area of blood-brain barrier breakdown occurring above 50°C and below 65°C when necrosis sets in with complete vascular obliteration. The postoperative hemosiderin volume was measured as the volume of T2 hypointensity on the last available follow-up MRI, at least 3 months after surgery. A representative example of volume measurement is depicted in Fig. 2. Preoperative and postoperative T2 volumes were compared via the Wilcoxon sign-rank test using SAS Studio 3.8 (SAS Institute Inc.).
Assessment of Seizure Outcomes

Seizure outcomes were assessed during clinical follow-up visits or phone interviews. The seizure occurrence status, adverse effects, and medication status were determined at the time of the most recent follow-up or phone call. Preoperative AEDs were maintained for at least 6 months after LITT and in some cases were reduced thereafter if patients remained seizure free. Seizure outcomes were categorized according to Engel’s classifications, including class I: seizure free without auras (IA), seizure free with auras only (IB), disabling seizures after surgery but remained seizure free for more than 2 years (IC), and seizures upon medication withdrawal only (ID); class II: fewer than 3 seizure days per year; class III: greater than 80% reduction in seizure frequency; and class IV: less than 80% reduction in seizure frequency.10,34

Results

Patient Characteristics

The median age was 47 (range 24–64) years; the median duration of epilepsy was 9 (range 2–26) years, and the median duration of postoperative follow-up was 25 (range 12–39) months. Two patients (patients 2 and 6) had familial CCM disease with a KRIT1 gene mutation and multiple CCMs; one of these (patient 2) underwent simultaneous ablation of 2 CCMs.

The 7 presumed epileptogenic CCMs in the 6 patients were distributed in the temporal (n = 3), frontal (n = 1), parietal (n = 1), and occipital (n = 2) regions (Fig. 3). Patient 2 had 2 subcentimeter CCMs in the left temporal and right frontal lobes, and 2 additional punctate lesions (possible CCMs) in the right frontal gyrus and right pons; EEG demonstrated epileptiform discharges over both the left temporal and right frontal regions, and so both lesions were ablated. Patient 6 had multiple CCMs with EEG showing ictal discharges from the right anterior temporal region correlating with the right anterior temporal CCM, which was subsequently ablated. Patient 3 had a prior lesionectomy without significant seizure improvement; SEEG showed a discrete region of ictal onset in the hemosiderin margin adjacent to the left optic tract, which was subsequently ablated with LITT (Table 1).

Video EEG was performed in 5 patients, and interictal

![FIG. 1. Real-time imaging of CCM laser ablation. A: Preoperative T2 MRI showing right uncal CCM. B: MR thermometry immediately at cessation of ablation. C: Damage map at the end of ablation; orange areas were heated to 70°C or higher. Note that gaps in thermal and damage maps correspond to hemosiderin artifact. D: Immediate postablation postcontrast T1 MRI. E: T2 MRI at 6 months after LITT, showing focal encephalomalacia and redistributed hemosiderin.](image)

![FIG. 2. Volumetric analysis. Volumes were measured by tracing areas on consecutive MRI cuts. Shown are representative cuts with the borders of preoperative CCM, including a T2-hypointense hemosiderin ring (A, yellow outline), ablation volume as measured by contrast enhancement immediately postablation (B, orange outline), and postoperative hemosiderin adjacent to encephalomalacia on follow-up T2-weighted MRI (C, blue outlines).](image)
and ictal discharges were recorded in 3 patients. SEEG recording with depth electrodes was performed in 2 patients. Neuropsychological tests were obtained in 2 patients. Functional MRI was performed in 4 patients with lesions adjacent to the optic pathway. Retinotopic tractography was performed in patient 4 and incorporated in stereotactic targeting and planning of the ablation to minimize damage to the primary visual pathway (Fig. 4).

LITT Procedural Metrics

Ablation metrics are listed in Table 2. For 4 of 7 (57%) treated CCMs, the laser fiber was pulled back 1 or 2 times during LITT to ensure complete ablation of the lesion. The median power was 9.0 (range 5.1–9.2) W; lower power was used for the paracalcarine CCM (patient 4). LITT was performed over a period of 138–321 (median 186) seconds, resulting in total energy delivery of 1.1–2.9 (median 1.3) kJ. Hemosiderin within CCMs created an artifact on MR thermography (Fig. 1). The temperature buffering effect from heat convection seen with arteries and CSF spaces was not seen in the vicinity of CCMs; in fact, CCMs underwent rapid heating, likely due to high iron content.

Lesion Volumetrics

The median preoperative CCM volume (T2-hypointense hemosiderin ring) was 0.7 (range 0.2–3.2) mL. The

### TABLE 1. Patient demographics and clinical characteristics

| Patient No. | Age at Op (yrs) | Sex | Age at Onset (yrs) | Epilepsy Duration (yrs) | Baseline Seizure Type | Baseline AEDs | Prior Op |
|-------------|----------------|-----|-------------------|------------------------|-----------------------|--------------|---------|
| 1           | 53             | F   | 34                | 19                     | Focal aware and focal impaired aware | LTG, GBP    | No      |
| 2           | 24             | M   | 18                | 6                      | Focal impaired to bilateral tonic-clonic | LEV, OXC    | No      |
| 3           | 40             | M   | 38                | 2                      | Focal aware and focal impaired aware | LEV, LCM, PHT | Lesionectomy |
| 4           | 64             | M   | 38                | 26                     | Focal impaired to bilateral tonic-clonic | LCM         | No      |
| 5           | 59             | M   | 47                | 12                     | Focal aware and focal impaired aware | LEV, OXC    | No      |
| 6           | 25             | F   | 22                | 3                      | Focal impaired to bilateral tonic-clonic | CLZ, OXC    | No      |

CLZ = clobazam; GBP = gabapentin; LCM = lacosamide; LEV = levetiracetam; LTG = lamotrigine; OXC = oxcarbazepine; PHT = phenytoin.
median ablation volume (contrast-enhancing region on immediate postablation MRI) was 2.3 (range 0.5–4.6) mL. On follow-up imaging performed a median of 6 (range 4–11) months after ablation, the ablated CCMs appeared as T2 hyperintensity with a thin margin of T2-hypointense signal, consistent with liquefied necrosis and redistributed hemosiderin (Fig. 3). The median volume of the T2-hypointensity on follow-up MRI was 0.5 (range 0.1–2.9) mL, which was a median of 24% (range 9%–44%) of the preoperative T2-hypointense lesion volume and was significantly lower than the preablation volume (\(p = 0.02\)) (Table 2). Multiple postoperative MR images were available for 4 of 7 CCMs; no change in target lesion size or morphology was noted beyond 3 months postoperatively.

Seizure Outcomes

Overall, 5 of 6 (83%) patients achieved seizure freedom (Engel I), of whom 4 (67%) were seizure free without auras (Engel IA), and 1 patient (patient 1) had a single seizure 4 days after ablation but at the time of this report had remained seizure free for more than 24 months (Engel IC). The remaining patient (patient 4) had rare seizures (Engel II).

Cognitive outcomes were not assessed because pre- and postablation neuropsychological tests were not consistently obtained in the studied patients. All patients were discharged within 24 hours of postablation. Reduced dosage without reducing the number of AEDs was achieved in patients 1 and 6. Reduction of the number of AEDs was achieved in patients 2, 3, and 5. AED was not immediately changed in patient 4, and a second AED (pregabalin) was subsequently added (Table 3).

Adverse Effects

There were no hemorrhagic complications related to either laser ablation or laser fiber trajectories. Patient 1 reported blurry vision and difficulty reading shortly after ablation of her left parietal, paratrigonal CCM. Humphrey visual field (HVF) testing completed 6 weeks after LITT revealed a small right homonymous visual field deficit that

| Patient No. | CCM Location       | Power (W) | Time (secs) | Energy (kJ) | Pullbacks | Postop MRI (mos) | T2 Hypointensity Vol (mL) | Seizure Outcome (Engel class) |
|-------------|--------------------|-----------|-------------|-------------|-----------|-----------------|--------------------------|-------------------------------|
| 1           | Lt paratrigonal    | 9.0       | 260         | 2.3         | 1         | 11              | 2.27                     | 3.36 24% IC                   |
| 2a†         | Lt temporal pole   | 9.2       | 146         | 1.3         | 0         | 5               | 0.73                     | 1.89 32% IA                   |
| 2b          | Rt frontal         | 9.0       | 138         | 1.2         | 0         | 5               | 0.18                     | 2.28 44% IA                   |
| 3           | Lt parieto-occipital | 9.1      | 321         | 2.9         | 2         | 6               | 3.19                     | 4.63 9% IA                    |
| 4           | Lt paracalcarine   | 5.1       | 225         | 1.1         | 1         | 4               | 0.31                     | 0.47 15% II                   |
| 5           | Rt uncus           | 9.0       | 139         | 1.3         | 1         | 6               | 0.87                     | 3.82 11% IA                   |
| 6           | Rt temporal pole   | 9.0       | 110         | 1.0         | 0         | 3               | 0.73                     | 0.83 33% IA                   |

* Number of times the fiber position was adjusted during ablation to ensure complete ablation of lesion.
† 2a and 2b are results for the left and right hemispheres, respectively, of patient 2.
was not appreciated on pre- or postoperative clinical examination (Fig. 5). The patient’s blurry vision improved over time, and HVF at 6 months after LITT demonstrated decreased size of her visual field deficit. Of note, HVF testing was not performed prior to LITT. Patient 3 had a preexisting right superior quadrantanopia, which was not worsened after LITT. Patient 1 was readmitted for a single breakthrough convulsive seizure. There was no readmission related to laser ablation. No delayed complications were identified.

**Discussion**

**Treatment Modalities for CCM-Associated Epilepsy**

Surgical resection is considered the gold standard for the treatment of drug-resistant CCM-related epilepsy. Pure lesionectomy achieves postoperative seizure freedom in 70%–80% of patients with sporadic seizures or those in whom their first seizure occurred less than 1 year before surgery. Worse outcomes (44%–55% seizure freedom) have been reported in cases with multifocal CCMs, in part due to uncertainty about which lesion or lesions are epileptogenic. There is also a reduced chance of seizure freedom in cases with longer preoperative duration of epilepsy. In cases where the epileptogenic lesion is clearly defined, early resection in CCM-related epilepsy is generally recommended. Several studies have demonstrated that resection of CCMs along with perilesional hemosiderin provides a better chance for seizure freedom, particularly when guided by intraoperative electrocorticography. However, traditional approaches carry the additional morbidity of craniotomy and microsurgical tissue manipulation, which often deters patients from considering surgery.

Stereotactic radiosurgery (SRS) is a noninvasive technique that can be considered for patients with symptomatic CCMs near eloquent cortex or in deep brain structures that are considered high risk for surgical resection. However, the effectiveness of SRS for CCM-related epilepsy appears to be lower than that of surgical resection, with reported seizure freedom rates ranging from 44% to 62%.

### TABLE 3. Postoperative seizure outcomes

| Patient No. | Clinical Follow-Up (mos) | Seizure Outcome (Engel class) | Postop Seizures | Postop AED Status | Adverse Events |
|-------------|--------------------------|-----------------------------|----------------|-------------------|---------------|
| 1           | 35                       | IC                          | Focal impaired to bilateral tonic-clonic LTG↓, GBP | Visual field deficit |
| 2           | 24                       | IA                          | No             | OXC (2→1)         | No            |
| 3           | 31                       | IA                          | No             | LEV, OXC (3→2)    | No            |
| 4           | 26                       | II                          | Focal aware and focal impaired aware LCM, PGL | No                |
| 5           | 18                       | IA                          | No             | OXC (2→1)         | No            |
| 6           | 12                       | IA                          | No             | ONF, OXC↓         | No            |

PGL = pregabalin; ↓ = reduced dosage; → = decreased number.

![FIG. 5. Postoperative visual field deficit in patient 1. On the left, coronal T2-weighted MRI before (A) and 11 months after (B) LITT. On the right, a small right homonymous visual field deficit (dark areas in right visual field, contiguous with physiologic blind spot) noted on HVF testing at 6 weeks after LITT (C) that improved by 6 months after LITT (D).](image-url)
the seizure freedom rate after SRS is generally lower than that for surgical resection. In a series of 30 patients with CCM-related epilepsy, surgical resection was significantly more effective than SRS (79% vs 25%). In another study of 49 patients with CCM-related epilepsy, only 53% of patients were seizure free after treatment with SRS. Additionally, seizure improvement has a long latency of 10 to 12 months after radiation, and significant postradiation cerebral edema, headache, and radionecrosis with mass effect may occur, which often requires hospital admission and treatment with dexamethasone. Radiosurgery is also not recommended for patients with familial CCM due to the risk of new CCM formation.

**Clinical Outcomes With LITT**

Early data have demonstrated that LITT might be a safe and effective alternative to lesionectomy for CCM-related epilepsy. McCracken et al. first reported the technical feasibility of LITT for the treatment of CCM-related epilepsy, with seizure freedom achieved in 4 of 5 (80%) patients after at least 12 months of follow-up.25 In a recent follow-up study of 19 patients with solitary, nonfamilial CCMs from the same center by Willie et al., 14 of 17 (82%) patients with > 12 months of follow-up achieved Engel class I outcomes, of whom 10 of 17 (59%) were Engel IA.35 The findings in this study are in line with those of McCracken et al. and Willie et al., Overall, 5 of the 6 (83%) patients achieved seizure freedom (Engel I), of whom 4 (67%) were seizure free without auras (Engel IA). One patient was seizure free for > 24 months after a single seizure on postoperative day 4 (Engel IC), and the remaining patient continued to have rare seizures (Engel II). To date, no patient in this series has become seizure free and discontinued all AEDs. This result is in part due to the long duration of preoperative seizures in this series (range 2–19 years, median 9 years).

Nondisabling focal neurological deficits were seen in 1 of 7 (14%) patients who underwent ablation in the present study, compared with 2 of 17 (12%) patients with ablations in the series by Willie et al. No hemorrhagic complications were seen in our patient sample or the aforementioned studies.

**Imaging and Therapeutic Mechanism**

The mechanisms by which LITT of CCMs results in seizure freedom are unclear. CCMs are thought to cause epilepsy via hemosiderin deposition, gliosis, inflammation, ischemia, and venous hypertension in the surrounding tissue.4 In a previous study, histopathologic analysis after open resection of 2 CCM lesions treated with LITT showed gliosis, hemosiderin-laden macrophages, and collapsed hyalinized vessels, consistent with destruction of the cavernous vascular spaces that characterize CCMs.35 In the present study, LITT targeted the CCM, including the hemosiderin ring in the adjacent parenchyma. Follow-up MRI at 3–11 months after ablation demonstrated focal necrosis with trace redistributed T2-hypointense hemosiderin (Figs. 1 and 2). On T2-weighted MRI, postablation hemosiderin volume was significantly lower than preoperative lesion volume (median 24% of preoperative volume, range 9%–44%, p = 0.04). LITT might result in seizure freedom by destroying epileptogenic bordering parenchyma. Seizure-free outcome was achieved despite residual hemosiderin on imaging, and as with open surgery it remains unclear with this approach how much additional hemosiderin removal is needed to achieve a seizure-free state.

**Contributions of the Present Study**

Although this case series has fewer patients than the study by Willie et al.,35 it has unique clinical features that illustrate the benefit of LITT for CCM-associated epilepsy. First, 2 (patients 2 and 6) of the 6 patients had familial CCM disease with multifocal lesions, whereas such patients were excluded from prior LITT studies.35 These patients may have multiple potentially epileptogenic CCMs, as in the case in patient 2, who underwent simultaneous ablation of a right frontal CCM and left temporal CCM, avoiding the need for bilateral craniotomies. Second, in 3 of the 6 patients, CCMs were located adjacent to the visual pathway, where open resection would carry a high risk for functional deficit. In patient 5, CCM location along the calcarine sulcus was felt to preclude safe surgical resection; retinotopic mapping by fMRI was performed to target the lesion accurately without inflicting damage to the visual system (Fig. 4), and this lesion was precisely ablated without postoperative visual field deficit. Only 1 patient (Fig. 5) had a new postoperative visual field deficit, which was nondisabling and improved over time. Real-time MR thermometry helps prevent off-target injury and postoperative functional deficits with voxel-level lesion demarcation and thermal safety limits at the margin of eloquent cortex or major white matter tracts, as assessed by preoperative fMRI or DTI. Third, the present study is the first to describe a case of LITT used for SEEG-proven epileptogenic hemosiderin after prior lesionectomy, with Engel IA outcome. While there are clear differences between CCM lesions that have undergone prior lesionectomy and those that have not, we have included this case in our series because the presumptive mechanism of epileptogenesis (i.e., hyperexcitable hemosiderin-stained tissue) is similar, and LITT circumvented the need for a second craniotomy and microsurgery in an area abutting the optic radiation.4 Interestingly, this patient had the highest LITT energy delivery and greatest hemosiderin volume reduction, consistent with a significant role for ablation of hemosiderin-laden brain tissue in the therapeutic mechanism. Finally, to our knowledge this is the first report to quantitatively describe the lesion volume reduction resulting from LITT for epilepsy-associated CCM.

**Conclusions**

The findings in this study support the technical feasibility and safety of LITT for the treatment of CCM-related epilepsy, with seizure outcomes similar to those of open surgery. LITT appears promising as a first-line surgical method with high efficacy and precision for the treatment of CCM-related epilepsy. In particular, LITT permits the treatment of CCMs in cases where the risk-to-benefit ratio of craniotomy and lesionectomy is unfavorable, such as in cases with eloquent areas and deep lesions. LITT also provides minimally invasive treatment options for very small
lesions and familial cases with multiple epileptogenic CCMs for which open surgery is not practical.

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Conception and design: Tao, Issa, Chen, Wu, Rose, Collins, Awad, Warnke. Acquisition of data: all authors. Analysis and interpretation of data: all authors. Drafting the article: Satzer, Tao. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Satzer. Statistical analysis: Satzer, Tao. Study supervision: Tao, Warnke.

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