Stereotactic laser ablation for nonlesional cingulate epilepsy: case report

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Stereotactic laser ablation (SLA) is being increasingly used to treat refractory focal epilepsy, especially mesial temporal lobe epilepsy. However, emerging evidence suggests it can be used for extratemporal lobe epilepsy as well.

The authors report the case of a 17-year-old male who presented with refractory nocturnal seizures characterized by bilateral arms stiffening or rhythmic jerking lasting several seconds. Semiology suggested an epileptogenic zone close to one of the supplementary sensory motor areas. Electroencephalography showed seizures arising from the central region without consistent lateralization. Brain imaging showed no abnormality. An invasive evaluation using bilateral stereo-electroencephalography (SEEG) was utilized in 2 steps, first to establish the laterality of seizures, and second to further cover the mesial cingulate region of the right hemisphere. Seizures arose from the middle portion of the right cingulate gyrus. Extraoperative electrical mapping revealed that the seizure onset zone was adjacent to eloquent motor areas. SLA targeting the right midcingulate gyrus was performed. The patient has remained seizure free since immediately after the procedure with no postoperative deficits (follow-up of 17 months).

This case highlights the utility of SEEG in evaluating difficult-to-localize, focal epilepsy. It also demonstrates that the use of SLA can be extended to nonlesional, extratemporal epilepsies.

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STEREOTACTIC laser ablation (SLA) is one of the newest minimally invasive techniques used in the treatment of refractory focal epilepsy. It was first reported to treat epilepsy in 2012.⁵ Since then this technique has been utilized mainly for mesial temporal lobe epilepsy (MTLE)³² but also for epilepsy caused by hypothalamic hamartomas (HHs),³¹ tuberous sclerosis,²³ periventricular heterotopias,^{12,29} malformations of cortical development,⁶ and cavernous malformations.²⁴ The literature includes descriptions of only a few cases of extratemporal, nonlesional epilepsy treated with SLA, and they are mostly insular cases.^{26,28} Here we describe what is to the best of our knowledge the first known case of nonlesional, cingulate refractory epilepsy, treated successfully with SLA.

We detail the medical and surgical evaluations, treatment performed, and outcome.

Case Report

History and Presentation

This 17-year-old, right-handed male presented with a history of epilepsy since the age of 9. His seizure semiology had been stable and consisted of seizures occurring out of nighttime sleep or daytime naps. He reported an occasional aura of feeling dizzy, fearful, or unwell preceding a few of the seizures. During some of the seizures, he was able to understand people talking around him and even tried to talk back. His parents described the seizures as the

ABBREVIATIONS AED = antiepileptic drug; HH = hypothalamic hamartoma; MTLE = mesial temporal lobe epilepsy; SEEG = stereoelectroencephalography; SLA = stereotactic laser ablation; SOZ = seizure onset zone; SSMA = supplementary sensory motor area.

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FIG. 1. Sagittal T1-weighted MR images showing the location of the most mesial contacts of the depth electrodes. All electrodes have orthogonal trajectories. **A:** Left hemispheric electrodes. **B:** Right hemispheric electrodes. LAC = left anterior cingulate; LMC = left middle cingulate; LMF = left mesial frontal; LOF = left orbito-frontal; LPC = left posterior cingulate; RAC = right anterior cingulate; RMC = right middle cingulate; RMF = right mesial frontal; ROF = right orbito-frontal; RPC = right posterior cingulate.

left arm extending and moving up, occasionally accompanied by the right arm. This was followed by the whole body becoming rigid with a slight, superimposed jitteriness. This semiology is typically described in seizures arising from the supplementary sensory motor area (SSMA) or nearby.⁴ These seizures were short and lasted up to 15 seconds. An additional semiology involved the patient sitting upright, turning to his right, and grabbing at the air with his right hand. Both seizure types were frequent, occurring multiple times every night. The patient had initially been treated with valproic acid, but due to side effects and lack of efficacy, the treatment was changed to a combination of lamotrigine and clonazepam, which produced seizure freedom for about 1 year when the patient was 9–10 years of age. He had then been weaned off antiepileptic drugs (AEDs), and the seizures had subsequently recurred. A combination of levetiracetam, lamotrigine, and lacosamide produced seizure freedom, and he was treated with this combination for 2 years. Neuropsychological evaluation during that time indicated low-average cognitive functioning, with a full-scale IQ of 82 (12th percentile). He was again weaned off AEDs, and again experienced seizure recurrence. Despite resumption of treatment with the same combination of AEDs, the seizures persisted. Different combinations of AEDs, including carbamazepine, oxcarbazepine, and topiramate, were tried without good seizure control. The seizure semiology and frequency did not change over the course of treatment, and the patient developed depression and anxiety. There were no epilepsy risk factors. The patient underwent multiple electroencephalography (EEG) examinations. When seizures were captured on EEG they showed a nonlateralized, either bifrontal or generalized seizure onset; the EEG findings were otherwise normal.

Preliminary Evaluation

The patient was admitted to the epilepsy monitoring unit. Interictal EEG showed midline spikes at Cz dur-

ing wakefulness and sleep. Seizures were captured, and the semiology was either bilateral asymmetrical tonic, bilateral asymmetrical clonic, or automotor seizure with manual automatisms. All seizures had ictal patterns that were nonlocalizable with further evolution of the ictal discharge in the midline at Cz.

Brain MRI and PET examinations revealed no abnormality. A neuropsychological re-evaluation indicated slowed developmental progress, with cognitive functioning in the borderline impaired to the low-average range. The patient's full-scale IQ was 73 (4th percentile), but the profile was nonlateralizing. His behavioral history and formal testing performances were indicative of impaired attention, and he continued to have symptoms of depression.

The case was presented to a multidisciplinary surgical epilepsy conference and the conclusion was that the patient was likely suffering from epilepsy arising from the SSMA or a nearby region based on the semiology and EEG.

Stereoelectroencephalography

A bilateral stereoelectroencephalography (SEEG) study was suggested to lateralize the seizure onset zone (SOZ) as a first step, as utilized in previous cases.^{7,8,13,14} SEEG electrodes were placed using a Leksell stereotactic frame (Elekta) with Framelink planning software (Medtronic). Antibiotics were given within 1 hour of incision with each surgical procedure but were not given during the postoperative monitoring period. Electrodes were placed bilaterally in an orthogonal fashion to cover the posterior, middle, and anterior cingulate gyri, premotor regions, and orbito-frontal regions (Fig. 1). The cingulate was chosen for sampling due to its proximity to the SSMA. Based on the laterality of the ictal pattern, further electrodes could be placed to determine the extent of the SOZ. This 2-step approach has been used before with subdural electrodes.^{2,27}

SEEG showed abundant epileptiform discharges arising from the deepest contacts in the right middle cingulate

FIG. 2. SEEG findings (A–E) and MRI (F–H). *Blue tracings* represent the left hemispheric contacts while *black tracings* represent right hemispheric contacts. **A:** Interictal discharges. Run of beta paroxysmal fast lasting 1 second followed by repetitive (*red arrow*), periodic discharges of spikes with a frequency of 25–35 Hz seen in contacts RMC 1–2, located in the most mesial aspect of the middle portion of the cingulate gyrus (*red box*). **B and C:** Close-up of the periodic interictal discharges. **D:** SEEG seizure pattern with onset of the paroxysmal fast (25 Hz) activity in contacts RMC 1–2, the same contacts showing the interictal discharges (*red arrow*). The EEG onset precedes the clinical onset by 6 seconds. **E:** Close-up of the SEEG onset. **F–H:** Coronal (F) and axial (H) T1-weighted and sagittal FLAIR (G) sequences showing the location of contacts RMC 1–2 in the middle of the right cingulate gyrus (*red circles*).

electrode (Fig. 2A–C). Thirteen typical seizures showed an ictal pattern of fast activity localized to the same contacts that showed the interictal discharges (Fig. 2D–H). The left hemispheric contacts did not show any epileptiform discharges.

The next step was to further delineate the extent of the SOZ in the right mesial hemispheric surface by placing more SEEG electrodes in the right mesial superior frontal gyrus (SSMA location) and around the right midcingulate electrode that showed the interictal and ictal discharges in a “cage-like” orthogonal approach. We attempted to make the electrodes roughly equidistant to neighboring existing electrodes and the right middle cingulate electrode

within the allowable limits of the surrounding vasculature (Fig. 3A). These additional electrodes were placed 7 days after the initial bilateral implantation, during the same hospital admission, without removing any old electrodes. Eleven typical seizures were captured with this new set of electrodes over a 3-day period. The SOZ did not change compared to the previous evaluation, and the most mesial right midcingulate contacts still showed the seizure onset discharge of fast activity with early spread to posterior contacts located in what was presumably the primary foot motor area (Fig. 3B and C).

Extraoperative electrical stimulation successfully mapped the SSMA, primary hand and fingers motor area,

FIG. 3. Full right hemispheric coverage shown on a T1-weighted MR image (A, C, and D) obtained after insertion of the second set of electrodes and SEEG tracings (B). **A:** Second set of depth electrodes added to the right hemisphere in a “cage-like” approach around the contacts that showed the SOZ in the previous phase. The original electrodes are indicated in white, while the additional electrodes are indicated in yellow. All electrodes have orthogonal trajectories. **B:** SEEG page of the right hemispheric contacts only showing a seizure pattern after the addition of further electrodes. The ictal onset remains in RMC 1–2 (midportion of the cingulate gyrus). An early spread within 4 seconds is seen in contacts RSP 1–5, located superior and posterior to RMC 1–2. **C:** Red circle shows the EEG onset and yellow circle the early spread. Again, the EEG onset precedes the clinical onset by 4 seconds. **D:** Results of extraoperative motor mapping. Contacts RSP produce twitching of the left shoulder, left hand, and left foot concordant with the SSMA. Contacts RMPC produce twitching of the left foot, concordant with the cingulate motor region. Stimulation was performed per the following parameters: pulse rate 50 Hz, pulse duration 0.2 msec, and train duration 2 seconds. Stimulation was conducted at 2.0 mA, 3.0 mA, up to a maximum of 4.0 mA or until the occurrence of clinical symptoms or an after-discharge. RAMC = right anterior middle cingulate; RMPC = right middle posterior cingulate; RSA = right superior anterior; RSM = right superior middle; RSP = right superior posterior.

and the cingulate motor area (Fig. 3D). Those functional regions lay posterior to the presumed SOZ in the right midcingulate. Following mapping, all of the electrodes were removed, and the patient was discharged 10 days after the initial SEEG implantation.

Stereotactic Laser Ablation

Based on these results, the options of an open resection versus stereotactic laser ablation (SLA) were reviewed and offered to the family. Open resection was to be performed via an awake craniotomy with intraoperative motor mapping to maximize the resection. SLA was offered as a

procedure with less invasiveness and a lower chance of seizure freedom. After much deliberation over a period of months, the decision was made to proceed with SLA of the middle portion of the cingulate gyrus, corresponding to the contacts that showed the earliest ictal changes. In the absence of a lesion, the posterior border of the ablation was based on the stimulation results to spare eloquent cortex with positive motor responses, including left foot clonic jerking from stimulation of the posterior portion of the cingulate, felt to represent a cingulate motor area.^{1,18} The anterior border of the ablation was defined by the next anterior cingulate electrode that did not show epileptogenic

FIG. 4. A: Sagittal postcontrast T1-weighted and axial T2-weighted MR images showing the laser cannula prior to ablation. This trajectory is chosen to avoid passing through the primary motor cortex or corticospinal tracts. **B:** Sagittal and coronal postcontrast T1-weighted MR images obtained immediately after the treatment showing the created ring-enhancing ablation lesion in the right middle portion of the cingulate gyrus.

activity. The full thickness of the cingulate (medial-lateral and superior-inferior) was targeted to achieve maximal efficacy.

Roughly 6 months after the SEEG evaluation, the patient underwent SLA utilizing the Visualase (Medtronic) system. Under general anesthesia, a Leksell frame was applied for stereotaxy. A reference CT scan was obtained and co-registered to existing MRI studies. A parasagittal frontal approach was used to minimize the risk of injury to motor pathways (Fig. 4A). A 2-mm forehead incision was utilized. The cooling cannula and optical fiber with diffuser tip were placed using framed stereotactic guidance. The patient was placed in the MRI scanner for the ablation under MRI thermometry control. Two ablative lesions were made along the long axis of the middle portion of the cingulate. The posterior lesion was created with 8.9 W $\times$ 2.5 minutes. The diffuser tip was then backed out 9 mm along the tract and a second contiguous lesion was made with 8.9 W $\times$ 2 minutes. An immediate contrast-enhanced T1 sequence confirmed that the planned lesion was created (Fig. 4B). The patient was admitted for overnight observation and discharged the following day on a dexamethasone taper.

Postoperative Course

The patient became seizure free immediately after the procedure. As of the most recent follow-up visit, 17 months postoperatively, he remained seizure free.

Repeat neuropsychological testing 10 months after surgery reflected stable functioning without evidence of cognitive decline. His performance indicated borderline impaired general intellectual abilities (full-scale IQ 70, 2nd percentile). He continued to present with symptoms of depression, which likely contributed to variability in attention, but was reported to have made some improvements in his emotional functioning over time.

Discussion

SLA, also known as laser interstitial thermotherapy (LiTT), was first introduced in 1976 by Fritz Heppner.²⁰

It involves the stereotactic placement of a small catheter to deliver destructive energy via laser to a discrete target. In the modern iteration of this technology, these ablations are guided by real-time MRI thermometry to better predict the area of destruction while preserving adjacent eloquent tissue. A cooling cannula containing an optical fiber with a diffuser tip is precisely placed within the target using stereotactic guidance. The laser energy is delivered while the patient is continually scanned in the MRI machine to provide real-time feedback regarding the lesion created.

SLA has gained popularity in managing refractory focal epilepsy for several reasons. The ability to access areas of the brain that are hard to reach with open surgery, such as basal or medial regions, is one of the reasons why SLA is being used more for certain types of epilepsies, such as mesial temporal lobe epilepsy (MTLE)^{16,17} and hypothalamic hamartomas (HHs).^{10,31} Surgical manipulation and exposure of functionally important regions required with open surgery can be avoided with SLA.^{8,9} Other advantages include shorter hospital stay (often without ICU admission), a minimal incision, less postoperative pain, and no risk of bone flap infection (as a craniotomy is not required). Although the time under anesthesia is not very different from traditional resections, the actual procedure time is shorter and most of the anesthesia time is used for planning the procedure, transporting the patient, applying stereotaxy, obtaining imaging, etc.^{25,30,32}

Most of the available experience with SLA comes from small case series or individual case reports. The vast majority of the series studied either MTLE or HH. Table 1 summarizes the available data from the largest series of SLA in MTLE.

There are only a few reports on using SLA for lesional, extratemporal epilepsy. Pathologies include periventricular nodular heterotopia,^{11,29} focal cortical dysplasia,^{3,6,11,23} tuberous sclerosis,⁵ cavernous malformations,²⁴ and encephalomalacia.¹⁹

Reports on treating nonlesional extratemporal epilepsy with SLA are mostly restricted to insular epilepsy. The

TABLE 1. Summary of the largest published case series of SLA for MTLE

Authors & Year	No. of Pts (hem)	Age at Op	Pathology	FU	Seizure Outcome	Add'l Resection	Complications	Neuropsych FU
Curry et al., 2012	1	NR	MTS	12 mos	Engel I	No	None	NR
Willie et al., 2014	13 (8 lt, 5 rt)	Range 16–64 yrs, mean 24 yrs	MTS: 9; mesial temporal signal change on T2WI: 1; mesial temporal atrophy: 1; nml: 2	Range 5–26 mos, mean 14 mos	Engel I: 6; Engel III: 3; Engel IV: 4	No	Hemianopia: 1; resolved acute SDH: 1	NR
Waseem et al., 2015	7 (3 lt, 4 rt)	Range 54–67 yrs, mean 60 yrs	MTS: 5; nml: 2	Range 1–1.3 yrs, mean 1 yr*	Engel I: 4; Engel II: 1	No	Partial visual field deficit: 2	4 pts followed up; no change in FSIQ: 3; drop in FSIQ: 1
Lewis et al., 2015	1	12.5 yrs	MTS w/ FCD	NR	Engel IV	No	Inaccurate fiber placement	NA
Wu et al., 2015	10 (8 lt, 2 rt)	Range 11–66 yrs	MTS: 10	≥6 mos	Engel IA: 4; Engel IB/C: 4; Engel II: 1; Engel IV: 1	NR	Trans diplopia: 1; contralateral sup quadrantanopia: 1	NR
Kang et al., 2016	20 (14 lt, 16 rt)	Range 11–66 yrs	MTS: 18; LGG: 1; nml: 1	6 mos, 1 yr, 2 yrs, & 3 yrs	6 mos—Engel I: 8; Engel III: 1; Engel IV: 1 1 yr—Engel I: 4; Engel II: 1; Engel III: 1; Engel IV: 2 2 yrs—Engel I: 3 3 yrs—Engel I: 1	4 ATL not incl in FU after SLA	Brain edema & int hemorrhage followed by rt sup quadrantanopia: 1; trans CN IV palsy: 1; HA: 4; trans worsening in mood: 2; insomniac: 1; scalp numbness: 1; suicide: 1	6 pts followed up at 10.2 mos on avg (5 lt hem, 1 rt); sig decrease in total learning CVLT raw scores in 3 pts operated on on lt; sig change in CVLT delayed recall, WMS, & LM II post-ablation raw scores
Dredla et al., 2016	2 (2 lt, 0 rt)	39 & 59 yrs	Nml: 2	Pt 1: 2 yrs; pt 2: NR	Engel I: 2	No	None	2 pts followed at >1 yr postop; preserved visual naming ability; semantic verbal fluency decline (not stat sig); sig verbal memory decline
Jermakowicz et al., 2017	23 (12 lt, 11 rt)	Mean 40.9 yrs	MTS: 15; nml: 8	12–29.5 mos	Engel IA: 8; Engel IB: 7; Engel II: 4; Engel III: 2; Engel IV: 2	No	Partially resolved lt hemianopia: 1	20 pts followed up at 8.4 mos; sig decline in delayed verbal memory for dom-hem pts only; sig cog decline in 8 pts, dom > non-dom
Greenway et al., 2017	15 (9 lt, 6 rt)	Range 22–66 yrs	MTS: 11; nml: 2; frontal gray matter heterotopia: 1	Range 6–45 mos, mean 26 mos	Engel I: 5; Engel II: 1; Engel III: 4; Engel IV: 5	NR	NR	6–36 mos: sig decline in verbal or visual memory in 15; naming showed better results: w/ only 1 pt showing a decline

Add'l = additional; ATL = anterior temporal lobectomy; avg = average; CN = cranial nerve; cog = cognitive; CVLT = California Verbal Learning Test; dom = dominant; FCD = focal cortical dysplasia; FSIQ = full-scale IQ; FU = follow-up; HA = headache; hem = hemisphere; incl = included; int = internal; LGG = low-grade glioma; LM = logical memory; MTS = mesial temporal sclerosis; NA = not applicable; neuropsych = neuropsychological; nml = normal; NR = not reported; pt = patient; SDH = subdural hematoma; sig = significant; stat = statistically; sup = superior; trans = transient; T2WI = T2-weighted imaging; WMS = Wechsler Memory Scale.
 * Reported for 5 patients.

largest study of SLA in insular epilepsy reported on 20 children with lesional and nonlesional MRI findings, and in that study Engel I was achieved in 50% of cases at a mean of 20 months follow-up.²⁶ To our knowledge only 1 other case of nonlesional frontal lobe epilepsy treated with SLA has been described.²⁹

Conclusions

Our case highlights the possibility of extending the substrates of epilepsy that can be potentially treated with SLA. In cases similar to ours, we recommend that a careful invasive evaluation be performed to determine the extent of the epileptogenic zone as accurately as possible. In our case it was useful to perform a second stage of denser coverage after initial localization to define a smaller target. Finding a clearly focal ictal pattern on EEG is essential, as targeting regions with unclear ictal patterns could mean disrupting the early spread region without actually removing the “pacemaker” of the epilepsy. This may lead to poor seizure outcomes. The deep location and proximity of the epileptogenic zone to highly functional regions are also points of consideration in choosing SLA over open resection, as the theoretical risk of injuring nearby structures may be less than with open surgery. Finally, performing SLA as a first step means that in the case of failure to control seizures, an open evaluation or resection can still be performed later with a surgical field that is free of scar tissue affecting the surgical approach.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Marashly, Lew. Acquisition of data: all authors. Analysis and interpretation of data: all authors. Drafting the article: Marashly. 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: Marashly.

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