

Clinical commentary

Endovascular ablation of a temporal lobe epileptogenic focus – a complication of Wada testing

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The authors report on a patient with a long history of medically refractory temporal lobe epilepsy. During pre-operative evaluation for anterior temporal lobectomy he underwent a Wada test for speech and memory lateralization. During the procedure, he suffered a focal cerebral infarct, within the right medial temporal lobe, at the site of the epileptic focus. This resulted in the resolution of his seizure disorder. The possibility of endovascular management of seizure disorders of the temporal lobe should be investigated further as a potentially minimal invasive therapeutic option for medically intractable epilepsy.

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The modern era of epilepsy surgery began in the late 19th century with the work of Macewen and Horsley (1, 2). Since that time, the efficacy and safety of the surgical management of epilepsy has been dramatically refined with the development of modern neuro-anesthesia, electrophysiologic monitoring, microsurgical techniques and ever improving neuro-imaging modalities. More recently, surgical resection has been shown to be particularly effective for drug-resistant temporal lobe epilepsy (3). Non-invasive ablative techniques, such as stereotactic radiosurgery, for control of seizure disorders have also been investigated and applied (4, 5). However, control of epileptogenic foci via the endovascular route has not previously been investigated. To the authors' knowledge this is the first report of endovascular control of temporal lobe epilepsy.

Case report

History

A 17-year old left-handed male with medically intractable epilepsy was evaluated at the George Washington University epilepsy center, as a potential candidate for epilepsy surgery. He has suffered from seizures since age 5. At that time he began to

report intermittent visual disturbances, consisting of a passing image of a 'purple animal with ferris wheels for feet.' At age 13, he had an episode of confusion and global amnesia lasting 3–5 min firmly establishing his diagnosis of epilepsy. In January 2000 he suffered his first generalized tonic-clonic seizure. He has continued to have seizures, approximately once per month. His episodes consist of staring spells associated with hand wringing, which progressed to a generalized convulsion lasting 4–5 minutes, followed by prolonged post-ictal confusion. The seizures have been refractory to medical management. Previous medications included Depakote, Lamictal and Topomax. His current regimen consists of Dilantin and Tegretol. There is no past history of traumatic brain injury or febrile seizures. His mother also suffers from an unspecified seizure disorder.

Examination

General medical and neurologic examinations were within normal limits and without focal findings. Neuropsychologic testing showed him to be of average intelligence, currently performing at the sixth grade level. Verbal IQ was 82 with a performance IQ of 81; both in the low average range. Verbal memory was found to be in the normal range with

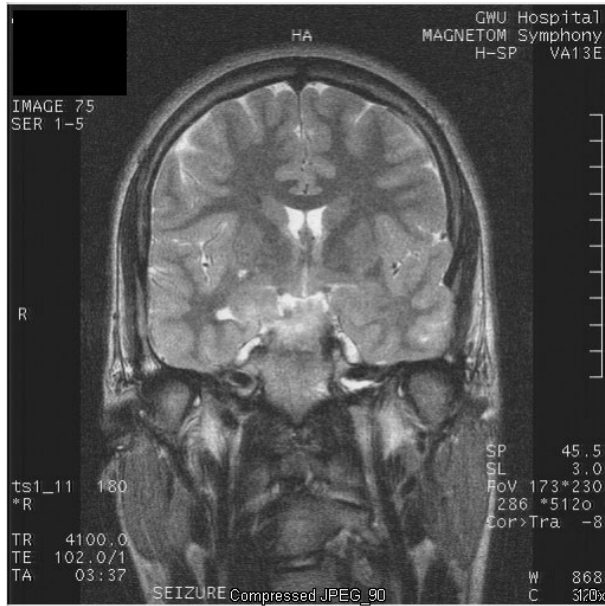


Figure 1. T2-weighted coronal MRI of the brain demonstrating increased signal within the right amygdala consistent with sclerosis.

moderately impaired visual spatial memory as demonstrated by his poor performance on the Rey-Osterrieth Complex Figure reproduction. He also demonstrated mild dysfluency with difficulties primarily in word retrieval.

Neuroimaging and functional studies

Inpatient telemetry with, video electroencephalography (EEG) monitoring off antiepileptic medications confirmed his stereotypic seizure and defined the right temporal lobe as the origin of the discharges. A magnetic resonance imaging (MRI) of the brain, with and without gadolinium enhancement, was obtained and demonstrated increased T2-weighted signal within the right amygdala, consistent with sclerosis (Fig. 1).

Procedure

The patient was readmitted to the hospital 3 months later for Wada testing, in preparation for right anterior temporal lobectomy. The epilepsy team felt it was critical to define hemispheric dominance for both speech and memory given his left-handedness and poor memory performance on neuropsychological testing. He was taken to the interventional neuroradiology suite and the Wada test, with selective middle cerebral artery injection, was performed under video EEG monitoring. A dose of 2.6–3.0 cc (1 mg/cc concentration diluted in normal saline) per injection of methohexital (Eli

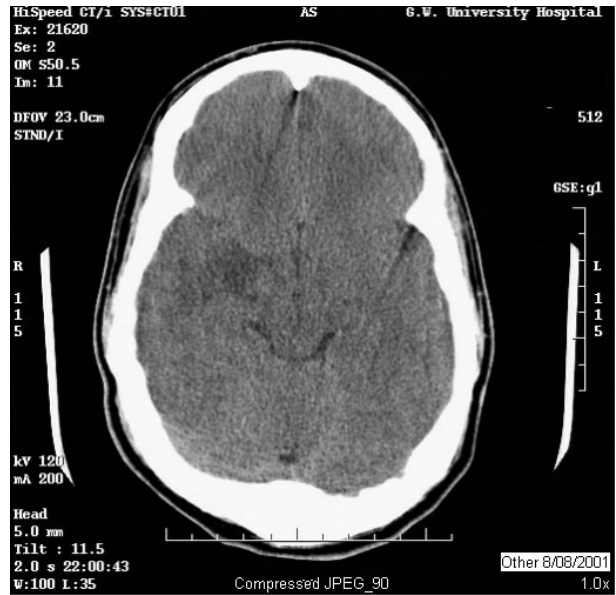


Figure 2. Non-contrast CT scan of the head showing hypodensity in the area of the right mesial temporal lobe.

Lilly Co., Indianapolis, IL, USA) was used. Two injections were necessary on each side due to the short half-life of the agent. Near the end of the procedure, the EEG monitoring began to show right-sided periodic lateralizing epileptiform discharges, which progressed into a generalized convulsive episode. An immediate non-contrast computed tomography scan of the head was obtained demonstrating decreased signal in the right medial temporal lobe (Fig. 2). The patient was readmitted to the telemetry-monitoring unit where his neurologic examination remained normal without evidence of visual, sensory, motor or cerebellar dysfunction. No further clinical evidence of seizure activity was noted. Mild slowing on the EEG was noted over the right temporal lobe that returned to baseline prior to discharge. He was discharged to home the next day and continued on his pre-procedural medication regimen.

Post-procedure course

The patient returned in follow-up 3 weeks post-procedure. At that time an MRI of the brain with and without gadolinium enhancement was obtained, demonstrating a 1-cm area of enhancement in the right amygdala and uncus, consistent with a completed infarct (Fig. 3). He has subsequently undergone repeat neuropsychologic testing which shows no decrement in speech or memory function and is unchanged from his pre-procedural evaluation. At his last follow-up evaluation, 11 months after his endovascular procedure, he is seizure free on a regimen of Dilantin and Tegretol.



Figure 3. Gadolinium-enhanced T1-weighted axial MRI of the brain demonstrating a 1-cm area of enhancement within the right mesial temporal lobe consistent with a completed infarct.

Discussion

Since 1948 when Dr Juhn Wada first observed the temporary effects of intracarotid Amytal (amobarbital) injection, its utility in the evaluation of cerebral dominance and memory lateralization have been well documented (6, 7). Many epilepsy centers routinely employ selective middle cerebral artery barbiturate injection in their preoperative evaluation of patients with temporal lobe epilepsy.

The neurologic complications of Wada testing have been well documented in the literature. These include stroke, seizure, retinal artery occlusion, and emotional lability (8, 9). The patient presented here suffered a radiographically documented stroke during his Wada testing. The most likely etiology for this infarct is catheter emboli, a well-known complication of cerebral angiography, occluding a small perforating branch of the anterior choroidal artery (10). It is unlikely that the anterior choroidal artery itself was occluded given that the remainder of its vascular territory (i.e., globus pallidus, internal capsule, and optic tract) was uninvolved either clinically or radiographically. Following this event the patient suffered no neurologic decline and is now fortuitously seizure free.

Peri-procedural complications in the past have led to the development of new therapeutic options such as Cooper's inadvertent ligation of the anterior choroidal artery and subsequent control of Parkinson's disease (11). It is possible that in the future selective, controlled endovascular techniques may be used to embolize the supply to precise areas of the temporal lobe and thereby ablate an epileptogenic focus.

Conclusion

There continues to be significant evolution of the surgical management of temporal lobe epilepsy. Endovascular techniques aimed at a controlled ablation of epileptogenic foci should be investigated further as a minimally invasive option for the treatment of seizure disorders.

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