

## Brief Communication

# An Angiographically Occult Arteriovenous Malformation in the Medial Parietal Lobe Presenting as Seizures of Medial Temporal Lobe Origin

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**Summary:** We present an unusual case of a patient who was diagnosed with temporal lobe epilepsy and whose seizures were reduced markedly after excision of an angiographically occult arteriovenous malformation (AVM) located in the left medial parietal lobe. A 38-year-old man had complex partial seizures characterized by motionless staring with oroflimentary and behavioral automatism since the age of 15 years. Magnetic resonance imaging (MRI) demonstrated a small lesion extending from the left posterior cingulate gyrus to the precuneus. There was no MRI evidence of mesial temporal sclerosis. Intracranial EEG recordings showed ictal onset from the left medial parietal lobe propagating to the medial temporal lobes.

Clinical signs appeared when these discharges reached the temporal lobes. After excision of the lesion (which was histologically confirmed as an AVM), together with the marginal cortex, seizures were reduced significantly. Careful diagnostic evaluation of lesions such as this one may reveal an epileptogenic lesion (zone) far from the region where scalp ictal discharges seem to arise. In our case, we hypothesize that false localization was due to propagation of ictal discharges from the parietal focus through the limbic system. **Key Words:** Occult arteriovenous malformation—Complex partial seizure—Parietal lobe—Epilepsy surgery.

The term “occult” or “cryptic” cerebrovascular malformation has been used to describe any vascular malformation that cannot be detected angiographically (1–3). Angiographically occult vascular malformations (AOVMs) have been subclassified into four groups: arteriovenous malformations (AVMs), venous angiomas, cavernous angiomas, and telangiectasias. Lobato et al. (4), reported that 43.8% of AOVMs were AVMs, which were probably occult because of thrombosis (3). The most common clinical presentation of occult AVMs is a seizure disorder. Wharen et al. (5) reported that 72% of patients with occult AVMs have seizures, and the most common presentation is temporal lobe lesions producing complex partial seizures (CPSs; 6). Yeh and Privitera (7) described a patient with a thrombosed AVM in the posterior temporal region, who had CPSs caused by a remote epileptic focus in the medial temporal region. In contrast, in another study, 18% of patients with occult AVMs who had seizures had lesions located in the parietal lobe (8). These patients had simple partial seizures rather than

CPSs. We present the rare case of a patient with CPSs attributed to a medial temporal lobe origin, but whose primary epileptogenic lesion was identified by magnetic resonance imaging (MRI) and intracranial EEG as an angiographically occult AVM in the left medial parietal lobe.

## CASE REPORT

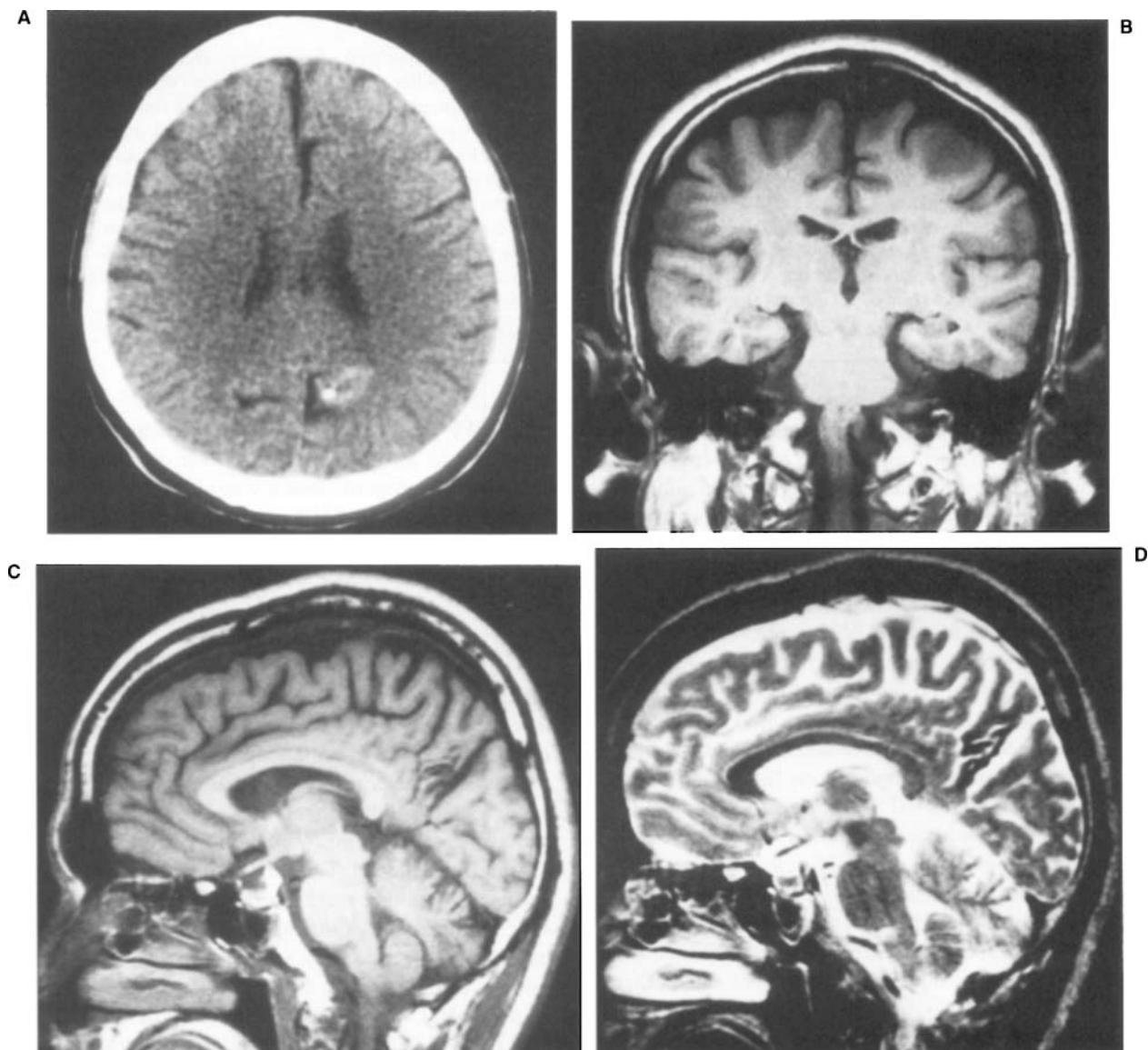
The patient was a 38-year-old man with seizures since the age of 15 years. He was diagnosed with “temporal lobe epilepsy” but was hospitalized for surgical treatment after failure of antiepileptic drugs (AEDs) to control seizures. As a result of a seizure, he had burned his hands and lost all his fingers except both thumbs. He also had a history of chronic subdural hematoma because of falls during seizures. The seizures were mainly complex partial in type and were characterized by a motionless stare, oroflimentary movements, and behavioral automatism. They occasionally secondarily generalized. Neurologic examination was normal, but his intelligence quotient (IQ) was slightly lower than the normal range (full IQ; 84). A carotid amobarbital test revealed that the left hemisphere was dominant for speech function. Computed tomography (CT) revealed only a small calcified

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area and no enhancement of a lesion in the left medial parietal lobe (Fig. 1A). The MRI showed no hippocampal atrophy (Fig. 1B) and a small lesion with hypointensity signal on both  $T_1$ - and  $T_2$ -weighted images. This extended from the left posterior cingulate gyrus to the precuneus of the left parietal lobe and included hemosiderin deposits and calcification (Fig. 1C and D). The lesion did not enhance after administration of gadolinium. Cerebral angiography was normal. The patient underwent video/scalp-EEG monitoring, and five seizures were recorded. These were stereotyped in their electro-clinical features and did not include lateralizing signs such as unilateral automatisms, head turns, or any hand dystonic postures. An interictal scalp EEG showed bilateral spikes over the frontal and temporal regions, right

greater than left. However, ictal scalp EEG was unable to define the location of the epileptogenic region. Because temporal lobe epilepsy was suspected, depth and subdural electrodes were implanted in both temporal lobes, and subdural electrodes were also placed over the site of the MRI-detected lesion in the left medial parietal lobe. Video/intracranial-EEG monitoring showed that ictal discharges originated from the left medial parietal lobe and then propagated to the right medial temporal lobe and finally to the left medial temporal lobe. Clinical symptoms appeared when the ictal discharge reached the right temporal lobe (Fig. 2). At surgery, the lesion was excised, along with the surrounding marginal cortex. This was done after cortical mapping had failed to demonstrate any identifiable function in this region. Histo-



**FIG. 1.** Computed tomography (A) demonstrating only calcification in the left medial parietal lobe. A coronal  $T_1$ -weighted magnetic resonance (MR) image (B) demonstrating no hippocampal atrophy in either temporal lobe. Sagittal  $T_1$ -weighted (C) and  $T_2$ -weighted (D) MR images demonstrating a small hypointense lesion in the left medial parietal lobe.

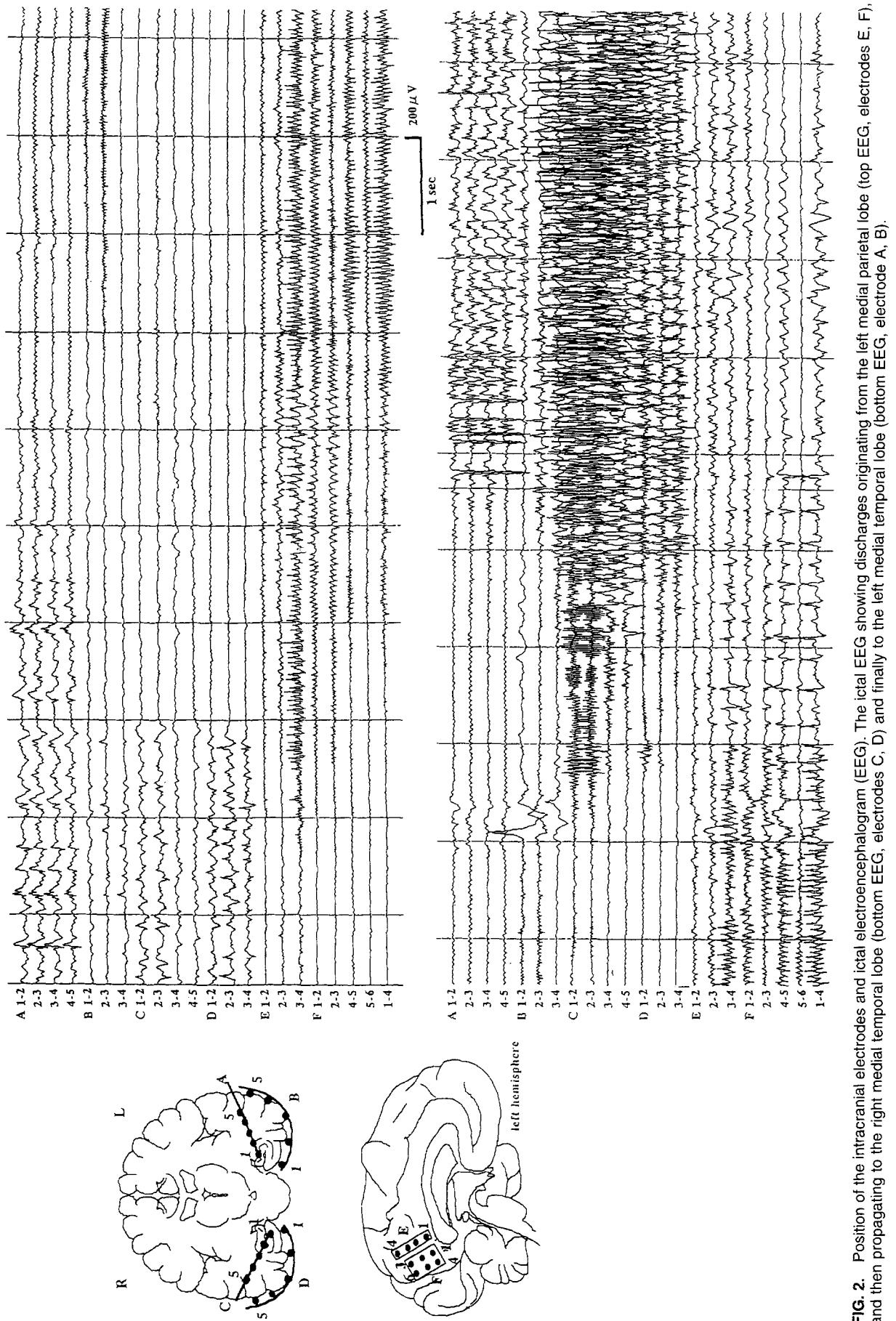
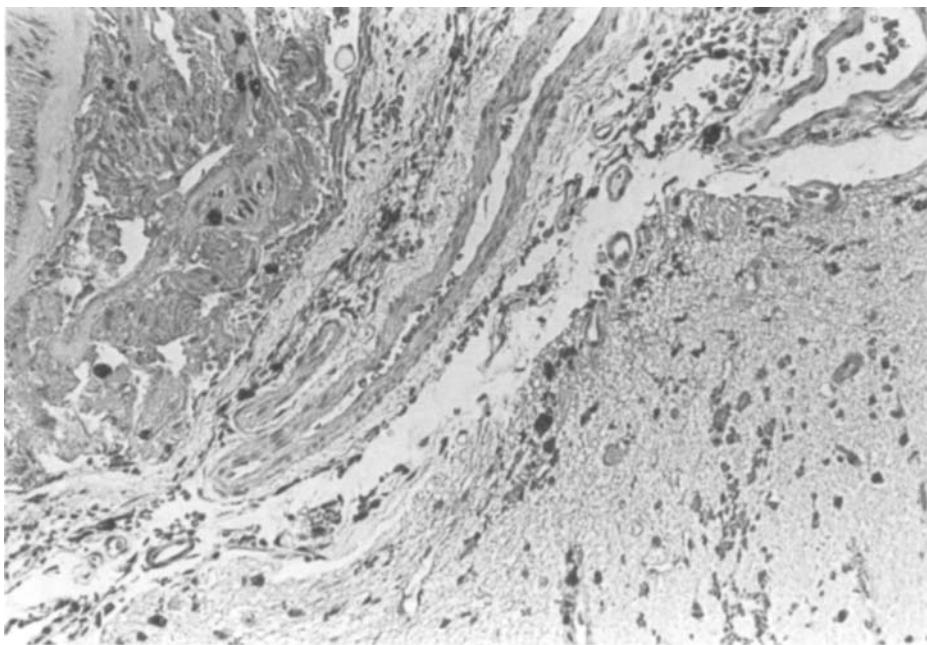


FIG. 2. Position of the intracranial electrodes and ictal EEG showing discharges originating from the left medial temporal lobe (top EEG, electrode A, B) and then propagating to the right medial temporal lobe (bottom EEG, electrode C, D) and finally to the left medial temporal lobe (top EEG, electrode E, F).



**FIG. 3.** Photomicrograph of a surgical specimen showing the features of an arteriovenous malformation (proliferation of dilated arteries with walls of various thickness). Deposition of hemosiderin also can be observed in the perivascular cerebral tissue. H&E, original magnification  $\times 100$ .

logically, the lesion was an AVM (Fig. 3). Seizure activity was reduced significantly postoperatively (twice CPSs), with 1 year of follow-up.

## DISCUSSION

MRI has been very helpful in allowing AOVMs to be diagnosed (9). However, the subtype of AOVM is correctly diagnosed preoperatively in fewer than half of patients. This may be due to the presence of a hematoma, which can obscure the underlying lesion, and to the general insensitivity of CT and MRI in identifying specific pathologic subtypes of AOVMs (10).

Somatosensory auras followed by unilateral clonic and tonic motor seizures are well-recognized features of parietal lobe foci, especially of the perirolandic area (11). A large variety of other clinical findings have been linked with parietal lobe seizures, including epigastric sensations, vestibular and visual hallucinations, automatisms, and arrest reactions (11,12). However, similar seizures might be produced by the spread of discharges to other surrounding areas. Williamson et al. (13) quoted Niedermeyer as stating that, with the exception of the primary sensory area, the parietal lobe does not play a major role in the generation of typical focal seizure patterns. In our patient, ictal discharges originated in the posterior cingulate gyrus and precuneus of the left medial parietal lobe, but this area was silent with respect to the patient's clinical seizures. It was only when the ictal discharges propagated into the temporal lobes that symptoms of CPSs of temporal lobe type occurred. Although several patients whose seizures originated from the lateral parietal areas and spread to the temporal lobes have been

described (12,14), there have been no other reported cases of medial parietal lobe epilepsy confirmed by intracranial EEG propagating to the medial temporal lobes and giving rise to symptoms of typical temporal lobe epilepsy. We believe that it is likely that the cingulum (which shares fibers with the limbic system) and the corpus callosum participated in propagation of ictal discharges to both temporal lobes.

Our patient's seizure frequency and severity were markedly reduced, but not completely controlled, in spite of removing the AVM and the adjacent epileptogenic cortex. Characteristics of postsurgical seizures and interictal EEGs were transient impairment of consciousness and bilateral spikes over the frontotemporal regions. One possible explanation for this is that secondary epileptogenic foci may have been produced in both medial temporal lobes by frequent, long-term activation of the primary focus, as suggested by Yeh and Privitera (7).

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