

# Pearls & Oysters: Symptomatic cerebral vasospasm on conventional angiography following temporal lobe epilepsy surgery

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## PEARLS

- Cerebral vasospasm is a complication of temporal lobe epilepsy (TLE) surgery.
- Cerebral vasospasm is associated with increased incidence of reversible and permanent neurologic deficit after TLE surgery.

## OY-STERS

- Cerebral vasospasm after TLE can be demonstrated as focal stenosis on conventional angiography. Triple-H therapy (hypertension, hypervolemia, and hemodilution), intra-arterial vasodilator administration, and angioplasty should be considered in clinically significant cases.

**CASE REPORT** We present a case of a 39-year-old right-handed woman with longstanding pharmacoresistant left TLE who developed new-onset neurologic deficits 2 weeks after a left anteromedial temporal lobe resection. The patient started having seizures at 26 years of age. Seizures manifested as déjà vu sensation followed by staring and unresponsiveness and right upper extremity jerking lasting 2–3 minutes. These occurred daily with occasional evolution to bilateral convulsions. She was on multiple antiepileptic medications including levetiracetam, carbamazepine, and gabapentin and had previously failed valproic acid and phenytoin. MRI brain was consistent with left mesial temporal sclerosis, and FDG-PET showed hypometabolism of the left temporal lobe. Neuropsychology evaluation revealed congruent findings. After presentation at the interdisciplinary epilepsy surgery conference, she underwent a left anteromedial temporal lobe resection. Initial postoperative course was uneventful, and examination revealed expected deficits consisting of a new right superior quadrantanopsia and mild dysnomia. MRI obtained on postoperative day 1 showed expected postsurgical changes following a partial left temporal lobectomy with minimal amount of blood products along the posteromedial resection edge.

On postoperative day 14, the patient developed acute right upper motor neuron facial weakness and global aphasia. No other new weakness, numbness, or visual disturbances were identified. MRI revealed a subacute left middle cerebral artery (MCA) infarct, and conventional angiography showed multifocal, critical vasospasm in proximal and distal left M1 and the M2-3 segments (figure). The proximal inferior division of the left MCA was nearly occluded. In addition, moderate to severe focal stenosis was present in the distal supraclinoid left internal carotid artery (LICA) and left P2-3 segments. Milrinone 10 mg was infused intra-arterially in the left internal carotid artery, followed by balloon angioplasty throughout the left M1 and LICA. An IV milrinone infusion was continued for 72 hours. She was also given oral nimodipine. Systolic blood pressures were maintained between 140 and 160 mm Hg with IV fluids and vasopressors. Daily transcranial Doppler (TCD) monitoring initially showed elevated left MCA mean flow velocities.

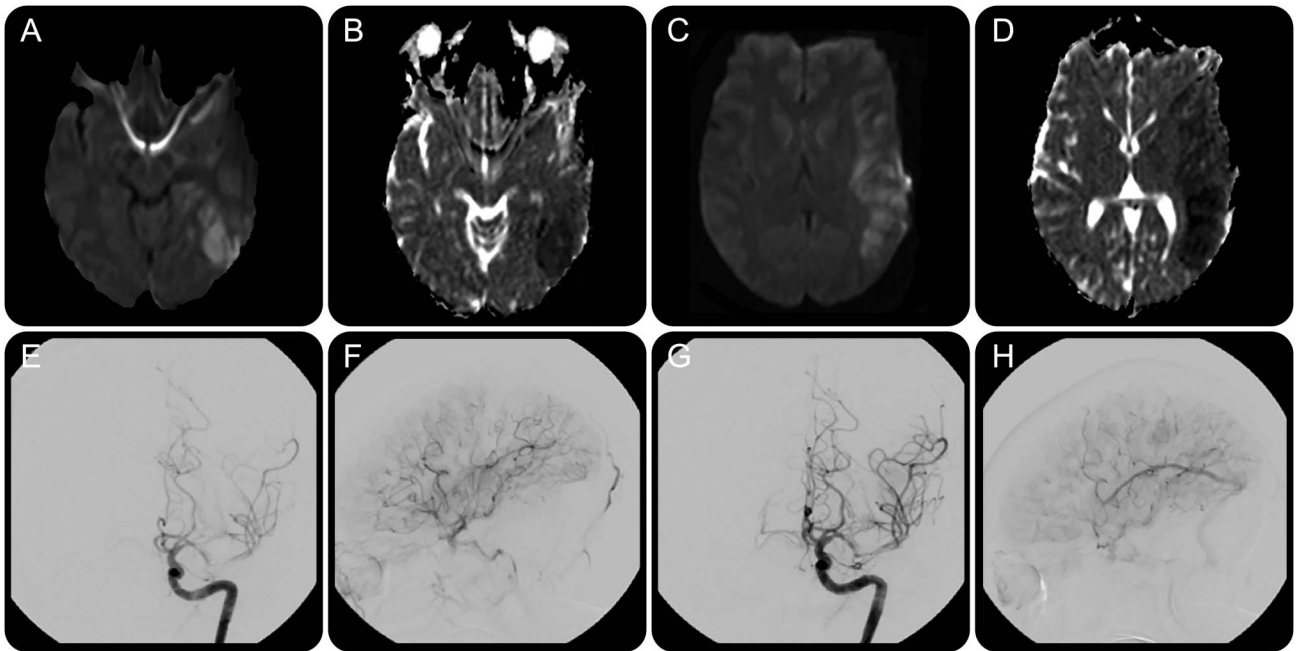
CT angiogram repeated after 3 days showed substantial improvement in vasospasm, and TCD velocities normalized. On clinical examination, verbal comprehension had improved; however, the patient continued to have paucity of spontaneous speech on outpatient evaluation at 14 months.

**DISCUSSION** Surgery for medically refractory TLE results in better seizure control, improved quality of life outcomes, and reduction in mortality rates, as compared to continued medical therapy.<sup>1,2</sup> Temporal lobe resection is mostly implemented using an anterior temporal resection approach or via a selective amygdalohippocampectomy using trans-sylvian or transcortical techniques.<sup>3</sup>

Though the overall risk of morbidity has been low (8%–10.8%), TLE surgery has been associated with medical and neurologic complications. Medical complications include CSF leak (5.8%), aseptic meningitis (2.7%), pneumonia (0.7%), intracranial hematomas (1.5%), deep vein thrombosis/pulmonary embolism (0.7%), and hydrocephalus (1.4%). Reported neurologic complications include visual field defects (20.2%), cranial nerve deficits (2.4%),

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Diffusion-weighted and apparent diffusion coefficient images demonstrate acute left middle cerebral artery (MCA) territory infarction (A-D). Cerebral angiogram shows vasospasm in the supraclinoid left internal carotid artery and left M1-3 segments with decreased perfusion in the left MCA territory (E) and luxury perfusion in the left frontal lobe (F). Balloon angioplasty of the left M1 segment and intra-arterial milrinone improved perfusion in the left MCA (G). Left temporal lobe ischemia is associated with luxury perfusion and early venous drainage into the left vein of Labbé (H).

dysphasia (14.9%), hemiparesis (3.6%), status epilepticus (0.2%), and psychiatric symptoms (7.7%).<sup>1,3,4</sup> Cerebral vasospasm is not commonly identified as a major complication of TLE surgery, yet when assessed using serial TCD ultrasound, postoperative vasospasm can be detected 32.7%–70% of the time, regardless of the resection method used.<sup>5–7</sup> Female sex and presence of higher amount of blood in the resection cavity on postoperative CT has been identified as predisposing factors for the development of vasospasm, and correlate with an increased incidence of transient and persistent neurologic deficits.<sup>7</sup> Evidence of firm causal relationships between cerebral vasospasm and persistent neurologic deficits is lacking in large studies and has been demonstrated in one prior case report showing severe symptomatic narrowing on MRI and TCD after TLE successfully treated with hydration and nimodipine.<sup>8</sup>

The pathophysiology of cerebral vasospasm after temporal lobe resection is unclear; however, it is potentially similar to large-vessel vasospasm seen following aneurysmal subarachnoid hemorrhage (SAH). Vasospasm in patients with SAH is attributed to multiple mechanisms including free hemoglobin-induced oxidative stress, activation of myosin light chain kinase facilitating smooth muscle contraction, upregulation of vasoconstricting peptides such as endothelin-1 by inflammatory mediators, and impairment of feedback loops that restore normal arterial

tone, all contributing to high incidence of vasospasm in the 3- to 21-day period following SAH.<sup>9,10</sup> Our patient developed symptoms on postoperative day 14, similar to the expected time period for vasospasm following SAH. The association between cerebral vasospasm and higher bleeding volumes on postoperative CT scans of patients undergoing TLE surgery supports the pathophysiologic similarity between these 2 entities,<sup>7</sup> although clear causation has not been established.

Our case directly demonstrates intracranial vasospasm occurring after temporal lobectomy using conventional angiography. Resultant focal cerebral ischemia was further associated with neurologic deficit. Furthermore, we demonstrate a therapeutic strategy in select patients with symptomatic vasospasm after TLE. A combination of intra-arterial and IV vasodilator administration, balloon angioplasty, and intensive medical therapy was used to reverse angiographic vasospasm in the patient and partially alleviate the neurologic deficits. Further studies will be important to identify at-risk groups and to delineate the role of TCD and other imaging modalities in detecting clinically significant vasospasm after temporal lobe surgery.

#### AUTHOR CONTRIBUTIONS

Dr. Rao contributed to acquisition of data, analysis, and draft creation and revisions. Dr. Narayanan contributed to study concept, design, analysis, and interpretation. Dr. Nanjireddy contributed to acquisition of data

and analysis. Dr. Mittal contributed to study concept, design, analysis interpretation, and critical revision for intellectual content. Dr. Basha contributed to manuscript revisions and editing and the critical review of the manuscript for important intellectual content and study supervision.

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### DISCLOSURE

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