Post-Subdural Hematoma Transient Ischemic Attacks
Hypoperfusion Mechanism Supported by Quantitative Electroencephalography and Transcranial Doppler Sonography

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Case Presentation
An 81-year-old right-handed woman with hypertension developed mild left hemiparesis over 1 week. Computed tomography of the head showed a right acute on chronic subdural hematoma (SDH). She underwent burr hole evacuation, her weakness resolved, and she was discharged to home after 4 days.

One week later, she experienced transient left hemiplegia and confusion lasting a few hours. On arrival to the emergency department, her symptoms had resolved. Blood pressure was 140/90 mm Hg. Repeat head computed tomography showed a small residual SDH and mild right hemispheric edema; both hematoma volume and edema were stable compared with those of previous imaging. Magnetic resonance imaging showed no diffusion restriction underlying the SDH, and magnetic resonance angiography showed normal intra- and extracranial vasculature. Electroencephalography (EEG) showed no epileptiform activity; however, there was mild slowing in the right hemisphere. Quantitative EEG (qEEG) analysis showed reduced alpha/delta ratio within the right hemisphere (Figure 1). Levetiracetam was empirically started, and she was admitted for close observation.

By day 2 of admission, she developed left face, arm, and leg weakness, right gaze deviation, and left-sided neglect. Blood pressure at the time was 110/78 mm Hg. EEG showed no epileptiform activity, but significant worsening of the right hemispheric slowing. qEEG analysis showed a further decrease in the alpha/delta ratio. Transcranial Doppler (TCD) sonography showed low mean flow velocities in the right middle cerebral artery (MCA) and a high pulsatility index on the right hemisphere compared with the left. Repeat brain magnetic resonance imaging/magnetic resonance angiography again showed normal intra- and extracranial vasculature. Careful review of the patient’s video EEG record revealed a 10-second suppression of all frequencies correlating with a syncopal event that immediately preceded the onset of her neurological symptoms (Figure 1). Her blood pressure after the event was 92/53 mm Hg. During this event, there was worsened right hemisphere slowing on EEG. Antihypertensive medications were discontinued, and she was given an intravenous fluid bolus for blood pressure augmentation. Her symptoms resolved over the next 2 hours.

By day 3 of admission, she was back to her neurological baseline. Her systolic blood pressure was 140 to 160 mm Hg, and TCD sonography showed increased flow in the right MCA. Her EEG was less slow in the right hemisphere, and qEEG showed recovered alpha activity with a normal alpha/delta ratio (Figure 2). Repeat TCD showed increased right MCA mean flow velocities. She was discharged to a rehabilitation facility with no weakness.

Discussion
An SDH is a collection of blood that accumulates below the dural layer, outside the brain and the arachnoid space. It is usually the result of a traumatic head injury, causing rupture of a bridging vein coursing from the cortical surface to the overlying dura. Because of age-related brain atrophy, bridging veins are under greater tension in the elderly, and minor trauma can lead to bleeding. Acute SDHs are associated with a high mortality and morbidity rate because of increased intracranial pressure (ICP), mass effect, and brain tissue shifts, as well as underlying brain contusion, cerebral edema, and secondary ischemic injury. The synergistic effects of these factors are complex. Emergent surgical evacuation is effective at improving outcomes; however, cerebral edema can persist for days to weeks after evacuation.1 It is believed that the blood constituents, namely hemoglobin and iron, play a major role in causing delayed neuronal injury and perpetuating cerebral edema.2

Pathophysiology of Brain Injury After SDH
The exact pathophysiology of brain injury after SDH is unknown. Many factors may play a role, including hematoma volume, increased ICP, trauma-related factors, blood constituents, brain metabolism, spreading depolarization, and ischemia. These factors may lead to secondary events and exacerbate neuronal damage.

SDH causes dramatic elevation of ICP, reduction of cerebral perfusion pressure, and cerebral blood flow (CBF). Increased ICP can lead to increased microcirculatory resistance, which may lead to decreased CBF2 (Law of Poiseuille). The size of hematoma is often less marked than the size of
secondary ischemic lesions. There is a mismatch between increased energy metabolism and decreased CBF underneath the blood clot, leading to enhanced release of excitotoxic amino acids culminating in secondary brain injury. In the severely ischemic cortex underlying the hematoma (mean CBF <25 mL/100 g per minute), glutamate and aspartate content increase by >750% over basal levels. Hlatky et al studied 33 patients after a surgical evacuation of acute SDH using an oxygen probe and microdialysis. The authors found significantly decreased oxygen tension and increased lactate and pyruvate levels in patients who developed delayed injury.

SDHs and Transient Ischemia

Transient neurological symptoms in the setting of SDHs have been reported, though they are uncommon and the underlying mechanisms uncertain. The most frequently proposed theory is reduced CBF and subsequent ischemia from vessel compression caused by cerebral edema. In patients with traumatic brain injury, microvascular collapse, endothelial swelling, and perivascular edema may also restrict CBF and impair oxygen diffusion. In this setting, CBF becomes tenuous and vulnerable to reductions in blood pressure. Focal seizure activity is another proposed mechanism of transient symptoms because postevacuation seizures are common, occurring in 25% of patients. Other theories include fluctuating ICP because of changes in head position or Valsalva maneuver, spreading depression, and repeated small hemorrhages. Given their brief nature, determining the underlying mechanism of transient symptoms is often challenging. Our patient experienced transient hemiplegia 1 week after her initial presentation that seemed to be precipitated by mild hypotension. Allowing the patient’s systolic blood pressure to increase from 100 to 140 mm Hg by stopping her antihypertensive medication and hydrating her with intravenous fluids resolved her symptoms.

Continuous Electroencephalogram and qEEG Monitoring

Continuous EEG monitoring is traditionally used for the diagnosis and management of complex seizure disorders. Because EEG signal correlates with CBF, continuous EEG monitoring is used in the operating room for the detection of ischemia during neurovascular interventions. With mildly reduced CBF, the EEG initially demonstrates loss of fast (beta) frequencies. When CBF drops to 18 to 25 mL/100 g per minute, the EEG background slows to 5 to 7 Hz. At 12 to 18 mL/100 g per minute, the EEG shows delta slowing (1–4 Hz). At very low CBF levels, there is progressive suppression of all frequencies.

qEEG is a digital analysis method that transforms the EEG into power spectra by fast Fourier transformation. This compresses the EEG data, making review more efficient. Decreased percentage of faster frequency bands (alpha and beta) coupled
with increased percentage of slower frequency bands (delta and theta), that is a reduced alpha/delta ratio, has been shown to be a sensitive marker of focal brain ischemia. In our patient, at baseline on day 1, there was decreased fast activity and mild to moderate slowing within the right hemisphere. Using qEEG, the alpha/delta ratio was found to be reduced. This ratio significantly worsened after the syncopal event on day 2. During that event, EEG showed diffuse suppression of all frequencies. After the patient regained consciousness, her EEG showed a significant worsening of the alpha/delta ratio in the right hemisphere, while her left hemisphere recovered to baseline. Clinically, this worsening correlated with her left hemiparesis and right gaze deviation. On day 3, after blood pressure augmentation, there was significant improvement in the right hemisphere alpha/delta ratio, associated with neurological improvement. The EEG changes correlated with the clinical events and were most likely related to decreased CBF, which recovered after blood pressure augmentation.

**TAKE-HOME POINTS**

- Secondary ischemic injury after subdural hematoma may occur as a result of brain edema and local microvascular collapse.
- Electroencephalography changes (in raw data, as well as on quantitative electroencephalography) can reflect brain ischemia. Continuous electroencephalography monitoring can be useful in patients with transient neurological symptoms.
- Transcranial Doppler sonography can be useful in patients with subdural hematoma and transient neurological symptoms in determining the mechanism of transient symptoms.
Disclosures

None.

References


Key Words: EEG • subdural hematoma • transcranial Doppler • transient ischemia
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