MAYO CLINIC

CASE REPORT

Refractory Left Focal Motor Status Epilepticus as Initial Clinical Presentation of Acute Basilar Artery Thrombosis

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Abstract

Seizures are uncommon with posterior circulation strokes. They are more often associated with anterior circulation strokes, with only a limited number of cases of status epilepticus reported to be related to brain stem ischemia. The literature includes case reports of generalized tonic-clonic seizures and associated status epilepticus as an initial presentation of acute basilar artery thrombosis. However, there are only rare cases reporting focal motor seizure as status epilepticus in the setting of acute basilar artery thrombosis, an important clinical presentation that should prompt evaluation for acute brain stem ischemia.

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Convulsion-like movements without electroencephalographic (EEG) evidence of cortical discharges have been described in brain stem strokes and as an initial presentation of an impending basilar artery occlusion (BAO).[1] A retrospective case series at one institution identified 35 patients with poststroke seizure, but only 7 patients had a brain stem lesion.[2] In this series, 14 patients had status epilepticus as the initial stroke presentation. Although seizure as an initial presentation of stroke is extremely rare, when it occurs it is typically in the setting of anterior circulation strokes; only a limited number of cases of status epilepticus have been reported in the context of brain stem ischemia due to basilar artery involvement in adults.[2-5]

We report a unique case of refractory focal motor status epilepticus as the initial presentation of an acute basilar artery thrombosis with resulting posterior circulation ischemia. Awareness of this presentation is important and should prompt urgent investigation for basilar artery thrombosis and acute brain stem ischemia.

REPORT OF CASE

A previously healthy 69-year-old right-handed man presented to the emergency department after his coworkers noted that he experienced left hand shaking and subsequently became unresponsive.

On admission, his initial Glasgow Coma Scale score was 5, the Mayo Full Outline of UnResponsiveness (FOUR) score was 8, and the National Institutes of Health Stroke Scale score was 26. Initial blood pressure was 203/168 mm Hg. Clinically, the patient was stuporous and exhibited persistent, rhythmic tonic-clonic activity of the left upper and lower extremity. Given concerns for focal motor status epilepticus, he was treated immediately with lorazepam and levetiracetam and ultimately required continuous propofol infusion for seizure control. He was resuscitated, intubated for airway control, and underwent neuroimaging.

Initial head computed tomography (CT) revealed no evidence of cortical lesions, hemorrhage, or acute ischemic stroke changes. His complete blood cell count, comprehensive metabolic profile, liver function tests, urinalysis, and toxicology screen results were unremarkable with negative findings on blood culture, and his serum glucose level was 210 mg/dL (to convert value to mmol/L, multiply by 0.0555). Computed tomographic angiography of the head and neck revealed an occlusive thrombus in the proximal-mid basilar

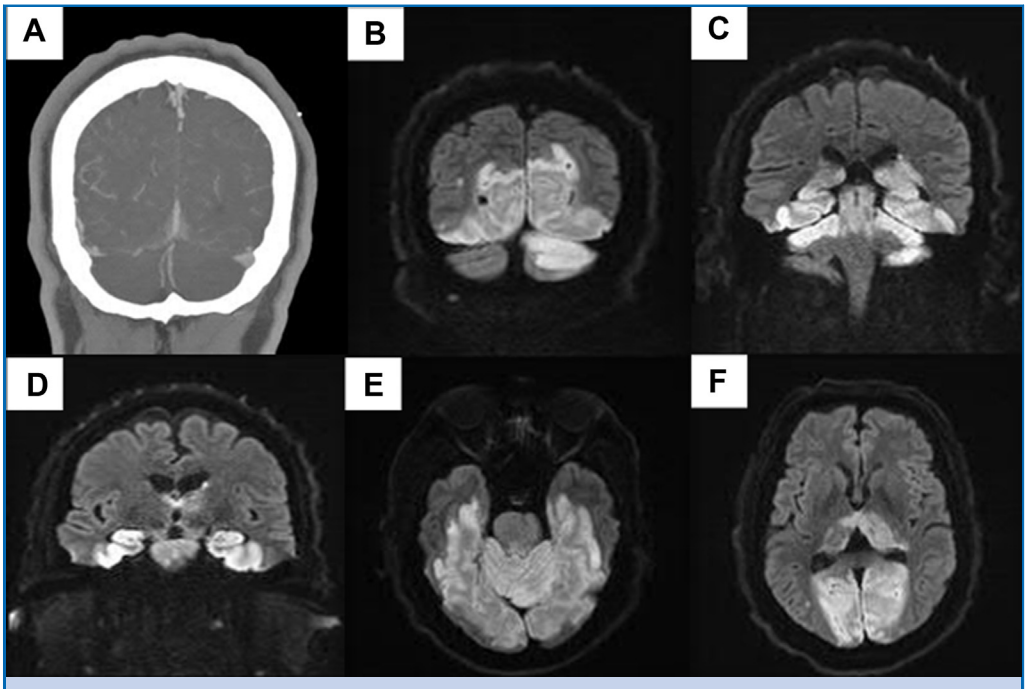

FIGURE 1. A, Representative coronal image of the initial computed tomographic angiogram of the head and neck, which revealed proximal-mid basilar artery thrombosis with no evidence of a vertebral dissection. B-F, Magnetic resonance imaging diffusion-weighted sequences show extensive acute infarcts bilaterally involving the thalami, midbrain, pons, inferior and medial temporal lobes, and occipital lobes consistent with widespread infarcts involving the posterior circulation. There was minimal inferior parietal involvement, which was thought to be within the posterior cerebral artery territory. The medulla and inferior cerebellum were spared. This distribution is concordant with the basilar thrombosis seen on angiography. Although sulcal effacement was present, there was no development of significant mass effect, obstructive hydrocephalus, or evidence of hemorrhagic transformation.

artery without evidence of predisposing vertebral artery dissection and no evidence of intracranial hemorrhage or early ischemic changes (Figure 1A). Cerebral CT perfusion imaging revealed a diffusely prolonged mean transit time in the posterior circulation involving bilateral cerebellar hemispheres, the brain stem, and bilateral temporal lobes, suggestive of a large ischemic penumbra. The patient underwent emergency thrombectomy within 2 hours after presentation.

Continuous video EEG obtained after the endovascular intervention revealed electrographic seizures arising over the left frontotemporal head region, as well as moderate to severe generalized slowing of the background activity (Figure 2). Brain magnetic resonance imaging the next day revealed widespread acute posterior circulation infarcts involving the thalami, mid-brain, pons, inferior and medial temporal lobes, and occipital lobes with relative sparing of the medulla and inferior cerebellum concordant with the suspected geographic distribution affected by the basilar thrombosis previously demonstrated on angiography (Figure 1B-F).

Clinically, the patient's condition continued to decline, with the loss of all brain stem reflexes except for spontaneous breathing with ventilator assistance and a right corneal reflex. Findings on repeated clinical examinations were consistent with severe brain stem injury and coma. Ultimately, the patient's family elected tracheostomy and parenteral feeding.

DISCUSSION

Basilar artery occlusion is a life-threatening condition and a diagnostic challenge. The largest study evaluating stroke-associated seizure (the Seizures After Stroke Study [SASS]), reported seizure in 8.9% of patients and the subsequent

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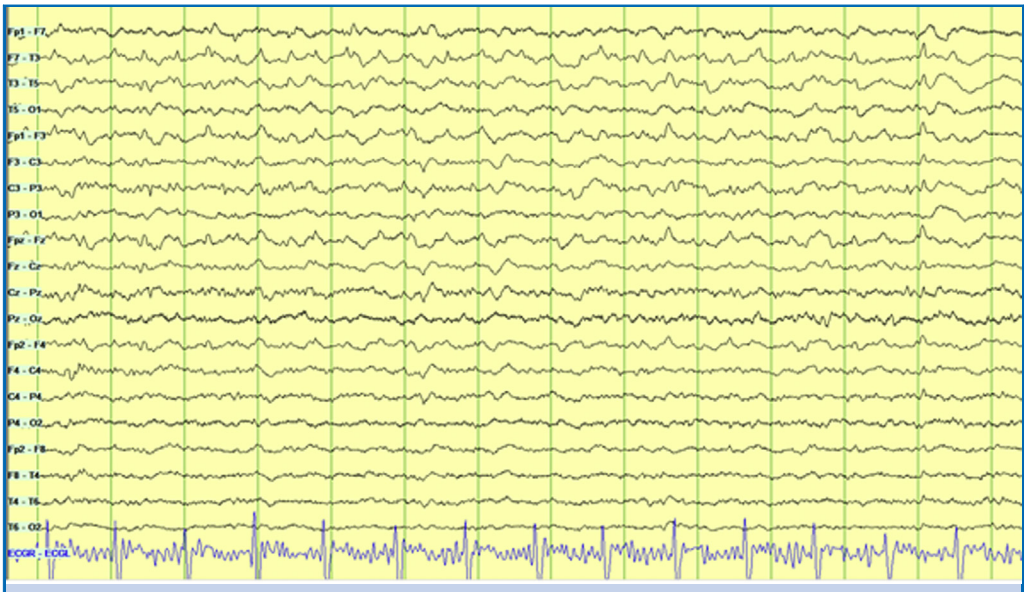

FIGURE 2. Representative electroencephalographic epoch demonstrating an electrographic seizure arising over the left frontotemporal head region, as well as moderate to severe generalized slowing of the background activity. The patient had recurrent brief electrographic seizures lasting 20 to 30 seconds consisting of rhythmic theta activity evolving into delta or recurrent sharp waves over the left frontotemporal region (Sensitivity 7 mcV/mm; Filter 1 to 70 Hz; interval between 2 vertical lines: 1 sec).

development of epilepsy in 2.5% of patients who had either ischemic or hemorrhagic stroke.[6] The study reported greater probability of seizure occurrence in hemorrhagic stroke survivors compared with ischemic stroke survivors.[6] Another study by De Reuck et al[7] revealed partial anterior circulation syndrome to be the only independent predictor of seizure in patients with cerebrovascular disease. Therefore, our description of a rare case of ischemic stroke involving posterior circulation infarction with focal seizure presentation is important.

Our patient presented to the emergency department with left upper extremity convulsive movements. Because of concerns about focal motor status epilepticus, the patient was stabilized with antiseizure drugs (ASMs). Subsequent neuroimaging revealed a proximal-mid BAO. Continuous EEG monitoring after the endovascular intervention revealed left frontotemporal electrographic seizures lasting 20 to 30 seconds, as well as moderate to severe generalized slowing of the background activity. Taken together, this clinical presentation suggestive of focal motor status epilepticus resolving with ASM administration and repeated EEG confirmation of electrographic seizures represent a unique presentation of refractory focal motor status epilepticus.

Studies have found that most patients with ischemic strokes have a total or partial infarction of the anterior circulation, compared with only 15% to 20% of patients with a posterior circulation infarction.[8] Among posterior circulation—related strokes, BAO is a rare event, implicated in only 1% to 4% of cases.[8] Some of the common etiologies of posterior circulation infarction include cardioembolism, atherosclerosis, arterial dissection, vertebrobasilar dolichoectasia, and vascular anatomic variation.[9]

A BAO has a longer prodrome compared with other cerebral artery occlusions. Some of the common presenting symptoms for a posterior circulation stroke include dizziness, unilateral limb weakness, dysarthria, headache, nausea, and vomiting.[9] Early symptoms of a BAO are nonspecific; they include nausea, vertigo, headache, and neck pain.[8] In addition, there can be a delay of days to weeks between the initial prodrome and BAO stroke onset. Basilar artery occlusion is, therefore, difficult to diagnose as many of these common early and nonspecific symptoms mimic nonstroke etiologies. The severity of a BAO depends on

the magnitude and location of the occlusion. For example, a mid-basilar occlusion with bilateral pontine ischemia can cause locked-in syndrome, which has a mortality of 75%.[8] Occlusions at other regions can cause quadriplegia and cranial nerve III palsy. Thus, an early diagnosis of BAO is critical to enhancing the chances of patient survival. Ongoing studies such as the Basilar Artery International Cooperation Study (BASIC) are key to the development of proper treatment plans for BAO.[8]

In general, posterior circulation infarction is associated with focal impaired awareness seizure.[10] Basilar artery occlusion—associated seizures have been identified in only few cases with predominant generalized tonic-clonic seizure presentation or brief convulsion-like movement in one extremity without status epilepticus.[3,11-15] Our patient presented with early left-sided motor movements that progressed to refractory focal motor status epilepticus. Therefore, a patient presenting with refractory focal motor seizure in the context of no history of seizures and associated neurologic changes should prompt a differential diagnosis that includes BAO even when findings on screening head CT are normal.

Determining whether the presenting movements represent a seizure from cortical lesions or convulsion-like movements from corticospinal tract (CST) infarction is critical. Understanding of this underlying etiology is important as it can alter the patient's care management and chances of survival considerably. One distinguishing feature between the two is that seizures have a more waxing and waning presentation whereas CST infarctions present with stable paresis or paralysis. Saposnik and Caplan[1] hypothesized the pathophysiology of convulsion-like movements in brainstem infarction as a consequence of CST ischemia. Patients can have development of hypertonia, clonus, and hyperreflexia, which can be confused as seizure symptoms. These symptoms can be explained by either spontaneous discharge from the CST or as a result of loss of inhibitory signals to the stretch reflexes from the reticulospinal tract that run adjacent to the CST.[16,17] In turn, the loss of these inhibitory signals leads to activation of the descending reticular formation pathway, causing convulsion-like movements.[14] These movements are treated in the context of ischemia and would not warrant the use of ASMs.

Conversely, seizure-associated strokes are a consequence of cortical lesions developed after hypoxic conditions and build-up of toxic metabolites. The hypoxia is caused by poststroke hypoperfusion, followed by cellular changes such as blood-brain barrier disruption, glutamate accumulation, and neuronal damage resulting in early epileptiform discharges.[18] Even though one could postulate the focal motor status epilepticus presentation of our patient as convulsion-like movements, our evidence with the response to antiseizure medications and the cortical irritability on EEG suggest cortical lesions as the most likely underlying mechanism. This case is of particular interest because focal seizures have not previously been described in the context of BAO, and clinically, one could easily think of uncontrolled epilepsy alone as an etiology. However, this patient did not have a history of epilepsy, and his clinical course is most consistent with an underlying stroke-related refractory focal motor status epilepticus.

CONCLUSION

This rare case provides compelling evidence of an important association for focal motor status epilepticus, acute basilar thrombosis, and posterior circulation cerebral ischemia. Recognition of this association is important because it may serve as a diagnostic clue for early diagnosis and interventional treatment of posterior circulation strokes.

Abbreviations and Acronyms: **ASM** = antiseizure medication; **BAO** = basilar artery occlusion; **CST** = corticospinal tract; **CT** = computed tomography; **EEG** = electroencephalogram

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