

Basilar Artery Occlusion Presenting as a Tonic-Clonic Seizure

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Stroke-associated seizures are usually due to ischemic involvement of the cerebral cortex or intracerebral hemorrhage [1]. Cases of subcortical lesions inducing stroke are less common but have been described [2]. Non-epileptic involuntary movements of the arms and legs due to ischemic lesions in the brainstem have also been reported [3]. On the other hand there are a few reports of seizures associated with ischemic lesions of the brainstem. The pathophysiology and mechanism of

such seizures are not known. We report a case of a seizure as a presenting symptom of basilar artery occlusion resulting in an ischemic stroke of the pons.

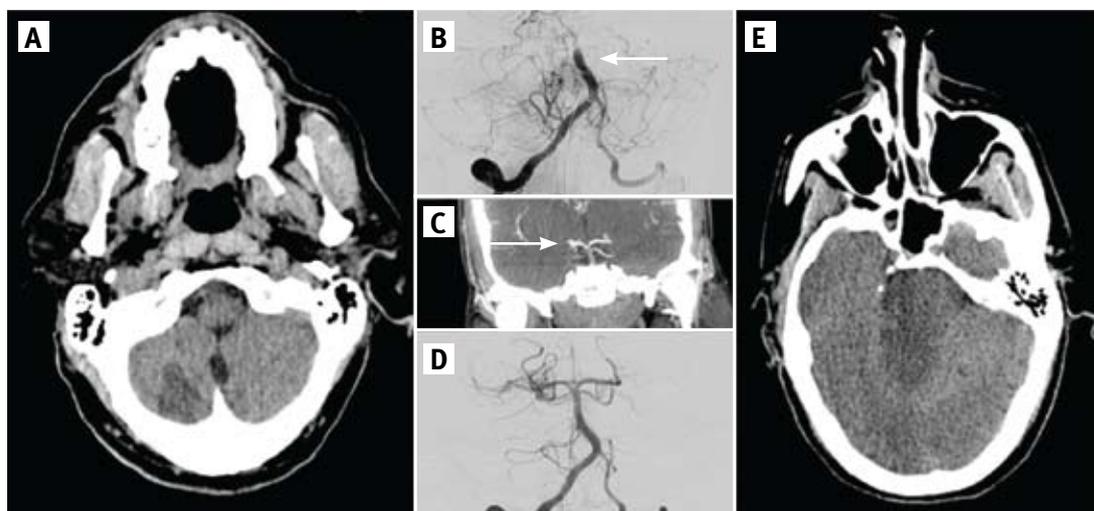
PATIENT DESCRIPTION

A 53 year old man with no history of epilepsy was admitted to the emergency department with complaints of vertigo, nausea and a single episode of vomiting. On neurologic examination eye movements were intact, there was no nystagmus or pyramidal signs, and instability of gait was noted. On computed tomography without contrast media a hypodense lesion was observed on the right cerebellum [Figure A].

Two days after admission the patient became confused. Several minutes later

the staff noticed vocalization followed by loss of consciousness and tonic-clonic movements with small amplitude in all four limbs. The presentation was seen by a neurologist who interpreted it as generalized tonic-clonic seizure, and since the condition continued for several minutes, status epilepticus was suspected. Intravenous phenytoin was administered with good seizure control at the end of the loading dose. Bedside monitor electroencephalogram performed half an hour after the movements stopped showed no epileptic activity. Urgent CT showed no hemorrhage or signs of new stroke.

Two hours from seizure onset, while the patient was regaining consciousness, right hemiplegia, right gaze limitation and Babinsky sign on the right were observed.



[A] CT of the brain after admission to the hospital showing left cerebellar stroke **[B]** A cutoff sign of the basilar artery is demonstrated on angiography **[C]** CT-angiogram of the brain after seizure showing a filling defect in the mid-basilar artery

[D] Angiogram at the end of the mechanical thrombectomy of the basilar artery occlusion showing open basilar artery **[E]** Brain CT after the procedure demonstrating large hypodense lesion in the left pons

Urgent neck and brain CT-angiography demonstrated right vertebral artery dissection and a filling defect in the mid-basilar artery [Figure C]. IV heparin was started and the patient was transferred to the angiography suite where stenting of the vertebral artery dissection and mechanical thrombectomy of the basilar artery occlusion were performed. Recanalization of the basilar artery was achieved 8 hours after seizure onset [Figure D]. Despite treatment the patient developed a large infarct in his left pons [Figure E] and remained mute, left hemiplegic and with lateral and vertical gaze palsy.

COMMENT

Seizure mechanism in the acute phase of stroke is unknown but may be related to the acute focal metabolic derangement including local acidosis, brain edema, and altered electrolyte balance and neurotransmitter activity. Although the majority of stroke-related seizures involve the cerebral cortex, there are reports of seizures as a presenting symptom of lacunar stroke [2]. Our case demonstrates generalized tonic-clonic seizure as a presenting symptom of basilar artery occlusion without evidence of cortical involvement on imaging studies.

Since recanalization was achieved within a few hours we cannot exclude the possibility of a transient occipital cortex ischemia that caused sufficient metabolic derangement for the seizures to occur. The other possibility is that contrary to the common notion, seizure is possible

without cortical involvement. In their report in 1978, Nathanson et al. [4] present four cases of patients with brainstem damage who developed seizures of axial structures; the authors hypothesized that these were epileptic seizures originating from the brainstem and not the brain cortex. The only report we found of basilar artery occlusion presenting as seizure describes two Japanese patients who were admitted with convulsions, loss of consciousness and hemiplegia [5]. Like our patient, they had brainstem, thalamic and cerebellar signs of ischemia on brain diffusion-weighted magnetic resonance imaging and no cortical involvement.

Involuntary movements at the onset of basilar artery occlusion or brainstem ischemic stroke have been described in a few papers [3]. These describe series of patients with vertebrobasilar stroke and wide clinical presentations that include fasciculation-like, shivering, jerky, tonic-clonic, and intermittent shaking movements. In all the cases EEG was normal, consciousness was preserved, and there was no effect of anti-epileptic drugs. In a review summarizing these cases [3], the conclusion was that these were non-epileptic seizures. One of the mechanisms suggested was disruption of inhibitory projection of the cortex to the spine or brainstem. In our case, although resembling these cases in some aspects such as normal EEG and no evidence of cortical involvement, the patient was unconscious, there was vocalization at the onset of movements and the convulsions stopped at the end of the loading dose of phenytoin.

In conclusion, our case demonstrates that acute basilar occlusion can present in a manner undistinguishable from generalized tonic-clonic seizure. If indeed this represents a true seizure, the mechanism may be related to occipital cortex ischemia or activation of subcortical seizure generators. Neurologists and emergency room physicians need to be aware of this and must perform a CT angiogram. Cases of atypical features of epilepsy such as basilar occlusion represent a deadly condition that may be treated if diagnosed in time. In our case the recent cerebellar stroke and the lack of seizure history in addition to the focal signs on examination provided the clues to a timely diagnosis and treatment.

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