

Convulsive-like Movements in Brainstem Stroke

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Background: Involuntary convulsive-like movements sometimes occur in patients with brainstem strokes. These movements vary in nature, frequency, and trigger, including fasciculation-like, shivering, jerky, tonic-clonic, and intermittent shaking movements. Some are interpreted as decerebrate postures or seizures. It is important to recognize this type of motor phenomenon since it may be a diagnostic clue for early diagnosis and treatment of brainstem strokes.

Design: Case report and review of the literature

Observation: A 72-year-old man presented with impaired consciousness and jerks of the upper limbs mimicking seizures. These episodes consisted of brief clonic contractions of the proximal and distal upper extremities. They were observed in paroxysms lasting for 3 to 5 seconds. Magnetic resonance imaging showed

large midpontine infarction. Magnetic resonance angiography revealed the absence of basilar artery blood flow. No seizure discharges were observed in the electroencephalogram. Anticoagulation with intravenous heparin was started. Two days after admission, the patient had a cardiac arrest and died. We review the frequency and nature of convulsive-like movements in brainstem stroke in the literature.

Conclusions: Movements associated with brainstem lesions are not easily differentiated from convulsions. Unexpected onset and inexperience of the observers limit the characterization of this phenomenon. Convulsive-like movements in brainstem stroke may occur more frequently than reported. Early detection of this motor phenomenon may have practical implications.

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SINCE 1868, spontaneous movements have been described in patients with brainstem lesions.¹⁻⁴ Tonic postures and jerks or twitching of the limbs in stroke patients are variously reported as seizures or abnormal movements.^{2,3} They were originally described in association with decerebration. The movements often are of sudden onset and may persist for a variable time.

We describe a patient with brainstem ischemia and convulsive-like movements and review the literature.

REPORT OF A CASE

A 72-year-old man presented with impaired consciousness and "seizures." He was eating when he suddenly developed weakness on his left side and fell down. His wife observed tonic postures in the 4 limbs while the patient was on the floor. Two days before admission, he reported dizziness and the persistence of visual images even when his eyes were closed. He

had a history of arterial hypertension and hypercholesterolemia. He had no history of migraine, seizures, or stroke.

In the emergency department, he was immediately connected to a respirator. On neurological examination, he was quadriparetic and unresponsive. Pupils were 3 mm and reactive to light. He had rightward gaze conjugate palsy. Lower brainstem reflexes were present. He had spontaneous jerking of the upper limbs lasting for 3 to 5 seconds. These episodes consisted of sudden, stereotypical, brief clonic contractions of the proximal and distal upper extremities. These movements repeated in paroxysms at irregular intervals regardless of the position of his arms. They were different from the classic decerebrate posture or myoclonus. During the movements, there was no change in the level of consciousness. No movements were detected in the face or lower limbs.

Results of laboratory studies, including complete blood cell count, electrolyte levels, glucose level, partial thromboplastin time, and prothrombin time, were

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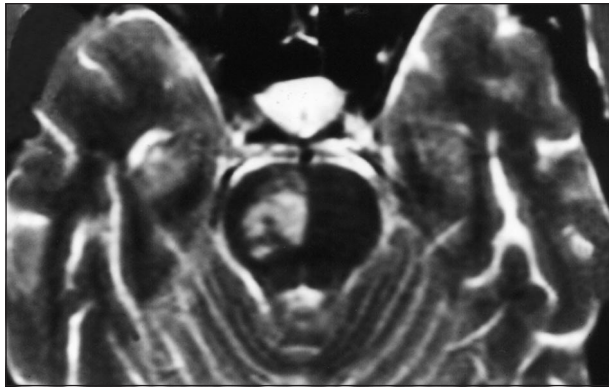


Figure 1. Magnetic resonance image at presentation showing T2-weighted hyperintense signal affecting the right pons in the base and tegmentum.

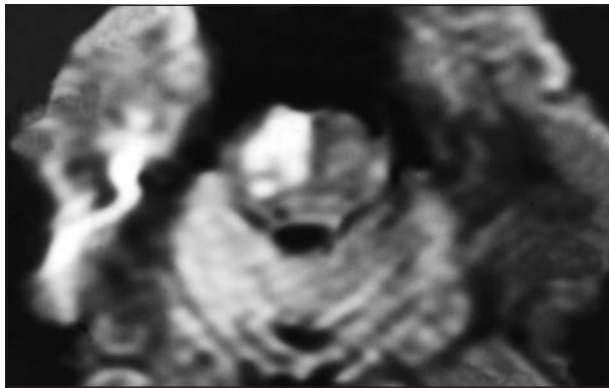


Figure 2. Diffusion-weighted image sequence demonstrating marked signal changes in the basis pontis and tegmentum.

normal. An electroencephalogram (EEG) showed no cortical discharges. A diffusion-weighted magnetic resonance image (MRI) on admission showed a pontine infarction involving the right basis and the tegmentum (**Figure 1** and **Figure 2**). A magnetic resonance angiogram showed absence of basilar artery blood flow.

Anticoagulation with intravenous heparin was started. The next day, the patient was in a coma with no motor response. His pupils were fixed and nonreactive to light. At that time, a new diffusion-weighted MRI showed a large midpontine hyperdense lesion involving the bilateral base and tegmentum. No cerebral lesions were detected. The vertebral arteries in the neck were normal, suggesting a proximal basilar artery occlusion. He had a cardiac arrest and died 48 hours later.

COMMENT

We report brief clonic jerking movements that resemble those occurring in convulsions in a patient with pontine infarction due to basilar artery occlusion. The MRI and the EEG showed no cerebral abnormalities or discharges. Curiously, our patient exhibited bilateral arm movements at the time of the first MRI (when the stroke was asymmetric). We hypothesize that corticospinal tracts were ischemic bilaterally, although only the right side of the pons was infarcted. However, it is difficult to understand why the movements were initially observed in the arms and not in the lower limbs.

Table 1. Convulsive-like Movements in Patients With Pontine Hemorrhage

Source, y	No. of Patients	No. With Convulsive-like Movements	Method	Lesions in Patients With Convulsive-like Movements
Dana, ² 1903	46	3	Autopsy	Tegmentum (pons)
Silverstein, ¹⁴ 1967	50	11	Autopsy	3 Basis pontis and tegmentum, 7 massive, 1 basis pontis

Involuntary movements of the limbs are occasionally seen in patients with acute strokes.⁵ These movements vary in nature, amplitude, frequency, and triggers. They include fasciculation-like, shivering, jerky, tonic-clonic, and intermittent shaking movements. Some movements are not easily differentiated from convulsions. Witnesses present during the episodes often report them as “seizures.” The spared consciousness and variability may be evidence against an epileptogenic mechanism. The upper and lower extremities have been equally involved, and the movements can be unilateral or multifocal. The rapid and unexpected onset, low incidence, and inexperience of the observers limit the characterization of this phenomenon.

In 1868, Nothnagel⁴ described the presence of a “convulsive pontine center.” Later, Jasper and Droogleever⁶ and Penfield⁷ postulated a “centrencephalic system” to characterize a group of neurons located in the midbrain reticular formation that functioned as a pacemaker for seizures. The role of the brainstem in the generation of tonic convulsions was studied more than 4 decades ago; however, the subject is still controversial.⁸ Experimental evidence in rat and cat models showed that stimulation of the reticular formation of the midbrain, pons, or medulla results in tonic seizures with absence of discharges in the cortex or at the site of the stimulus.^{9,10} Kreindler et al,⁹ Bergman et al,¹¹ and others^{12,13} showed that brainstem stimulation induced tonic or clonic movements depending on the magnitude of the stimulating current.

Recently, Kohsaka et al¹² studied sequential changes of brainstem function before and during 3-Hz spike and wave complex discharges in patients with typical absence seizures using simultaneous auditory evoked potentials and EEG. They found changes in the wave III component of the auditory evoked potentials preceding the onset of cortical paroxysmal discharges. This evidence reintroduced the classic concept of a centrencephalic system as a seizure-generating mechanism. Similar findings have been obtained in animals and humans with decreased basilar artery blood flow.¹³

Table 1 and **Table 2** show previous reports of abnormal involuntary movements in patients with hemorrhagic and ischemic pontine strokes. In 1903, Charles Dana² studied 46 patients with pontobulbar hemorrhages. Three of them had “twitching of the face, or of the limbs or both.” He also noted forced laughter-crying episodes associated with this motor phenomenon when the contralateral thalamus was involved. Dana and other

Table 2. Convulsive-like Movements in Patients With Pontine Infarction*

Source, y	Diagnosis	No. of Patients	No. of Patients With Convulsive-like Movements	Method	Lesions in Patients With Convulsive-like Movements
Cannon, ³ 1951	Secondary (acute) vascular brainstem lesions	122	32	Autopsy	NA
Fang and Palmer, ¹⁵ 1956	Basilar artery occlusions	7	5	Autopsy	Tegmentum and inferior olives
Halsey and Downie, ¹⁶ 1966	Vertebrobasilar occlusions	3	1	2 Angiography, 1 autopsy	NA
Fisher, ¹⁷ 1970	Vertebrobasilar occlusions	5	1	Angiography	NA
Kubik and Adams, ¹⁸ 1946	Basilar artery occlusions	18	1	Autopsy	Basis pontis and tegmentum
Silversides, ¹⁹ 1954	Basilar artery occlusions	22	3	Autopsy	NA
Siekert and Millikan, ²⁰ 1955	Basilar artery occlusions	28	4	Autopsy	NA
Silverstein, ²¹ 1972	Pontine infarction (11 basilar occlusions)	83	8	Autopsy	3 Paramedian, 2 tegmentum, 3 basis pontis and tegmentum
Ropper, ²² 1988	Basilar occlusions	8	8	4 Autopsy, 3 CT, 1 MRI	Tegmentum

*NA indicates data not available; CT, computed tomography; and MRI, magnetic resonance imaging.

authors believed that the abnormal movements observed in pontine lesions were not convulsive in nature. In 1967, Silverstein¹⁴ reported on 50 patients with pontine hemorrhages. Eleven patients (22%) had “convulsive seizures,” 3 of them unilateral. These motor movements were the initial symptom in 7 patients (63%). Two of 11 patients with unilateral pontine hemorrhages mainly involving the basis pontis and tegmentum had convulsive seizures, while 7 of 28 patients with bilateral basis pontis lesions and 2 of 11 patients with tegmental hemorrhages had convulsive seizures. These are the 2 largest series reporting convulsive-like movements in pontine hemorrhages.

In 1951, Cannon³ reported convulsion-like episodes in 32 (26%) of 122 patients with acute brainstem lesions, most of them secondary to tumors. Convulsive movements ranged from isolated twitching of the extremities to generalized seizures. However, only 17 patients (14%) in that series had basilar artery occlusions, and the frequency of convulsions was not reported.

Other authors observed similar movements in patients with basilar artery occlusive disease (Table 2). Kubik and Adams,¹⁸ in their pathological study of basilar artery occlusions, found 1 patient (case 3) (6%) with convulsive seizures among 18 individuals studied. Fang and Palmer¹⁵ reported clonic movements in 5 of 7 patients, and Siekert and Millikan²⁰ described “twitching of the extremities” in 3 patients (11%) and generalized convulsions in 1 (4%) of 28 patients with basilar artery occlusions.

Halsey and Downie¹⁶ described 3 patients with “decerebration rigidity” with spared consciousness. One of these patients was a 34-year-old woman who presented with occipital headache and vertigo. Her family observed 3 “severe shaking episodes” with urinary incontinence. Angiography showed proximal basilar artery stenosis. Silverstein²¹ analyzed 83 autopsies of patients with pontine infarcts. Eleven (13%) had basilar artery occlusions. He found 8 individuals who had “convulsive-seizures” (2 focal, 6 generalized), all of whom had “tremors and clonic jerks of the muscles of the extremities.”

The infarct was located in the paramedian area in 3 patients, in the central tegmentum in 2, and in the tegmentum and basis pontis in the remaining 3. Miller Fisher¹⁷ analyzed transient symptoms in patients with vertebral artery occlusion. In 1 patient, jerkings of the left arm with spared consciousness were observed.

In 1988, Ropper²² reported convulsive-like movements in 8 patients with basilar occlusions. Autopsy results were available for 4 patients and showed that the tegmentum was the most commonly affected area. Magnetic resonance imaging or computed tomographic scans of the head were performed for the remaining patients. Thalamic, occipital, and pontine lesions were found in these individuals (Table 2).

Considering all reports that analyzed convulsive-like movements, 66 (23%) of 287 patients with pontine strokes had abnormal movements. The presence of convulsions was more frequent in patients with pontine infarction (23/91 [25%]) than in those with pontine hemorrhage (14/96 [15%]). However, we cannot exclude a selection bias, since we only considered those studies reporting movements in brainstem strokes. Therefore, caution must be taken when interpreting the real frequency of convulsive-like movements in this condition.

In summary, we believe that the movements in our patient may be related to ischemia of the corticospinal tracts rather than to true convulsions with a brainstem nuclear origin. Convulsive-like movements may be seen in patients with either hemorrhagic or ischemic pontine lesions. Recognition of these movements may allow earlier diagnosis of pontine stroke. The pathophysiological mechanisms remain uncertain. Further functional studies are necessary to clarify the nature of this phenomenon.

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