

Spontaneous limb movements and posturing secondary to acute basilar artery occlusion: a potentially devastating seizure mimic

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ABSTRACT

Basilar artery occlusion is a devastating but treatable form of ischaemic stroke with high morbidity and mortality rates. The diagnosis is often challenging due to considerable heterogeneity of clinical signs and symptoms. We report a case of an acute basilar artery occlusion presenting with decreased level of consciousness associated with rhythmic tonic movements of the four extremities, mimicking seizure activity. The patient was treated with intravenous thrombolysis and subsequently gained good recovery. Awareness of this entity is required to recognise this potentially treatable, but otherwise devastating seizure mimic.

CASE REPORT

A 70-year-old woman presented to the emergency department with sudden decreased level of consciousness 1 h after symptoms onset. She had a history of atrial fibrillation and hypertension but no prior history of syncope or seizures. Upon arrival at hospital, her vital signs were stable. She was intubated due to decreased level of consciousness (Glasgow coma scale score=7/15). Neurological examination was notable for sporadic rhythmic, symmetrical, extensor posturing of both upper and lower extremities at a frequency of less than 1 Hz. This posturing appeared as intermittent tonic movements of the four extremities lasting up to 5 s followed by periods of normal tone. These occurred spontaneously but also upon light stimulation without any clonic components. Cranial nerve examination showed a right gaze deviation, preserved bilateral pupillary light response but absent corneal and oculocephalic reflexes. There was no facial asymmetry or weakness in four limbs. Deep tendon reflexes were

normal with bilateral extensor plantar responses. Laboratory investigations showed normal haemoglobin, platelets and white blood cell counts. Coagulation studies showed a mild elevation of international normalized ratio (INR) at 1.6 (normal <1.3) with a normal activated partial thromboplastin time (aPTT). Liver enzymes and renal function tests were normal. The initial diagnosis was that of a generalised tonic seizure; however, we entertained the possibility of an acute stroke, given the acute onset of symptoms and the brainstem findings on neurological examination.

An unenhanced CT brain scan was normal (figure 1A). CT angiogram showed a 9-mm distal basilar artery occlusion extending up from the mid-pontine segment, sparing the tip of the basilar artery. (figure 1B).

We made a diagnosis of pontine, or decerebrate spasms, secondary to acute basilar occlusion likely from a cardioembolic source due to the history of atrial fibrillation with subtherapeutic anticoagulation. We gave an intravenous tissue plasminogen activator 2 h from symptoms onset. The patient remained stable after thrombolytic therapy. By 24 h, the spasms had completely subsided and she was awake, alert and responsive. A repeat CT brain scan showed bilateral cerebellar infarctions in the superior cerebellar artery territories (figure 1C). She was extubated at 72 h and was able to walk with minimal deficit. She was discharged home with good recovery 3 weeks after the initial event.

DISCUSSION

Cerebral infarctions involving the posterior circulation encompass 20% of all

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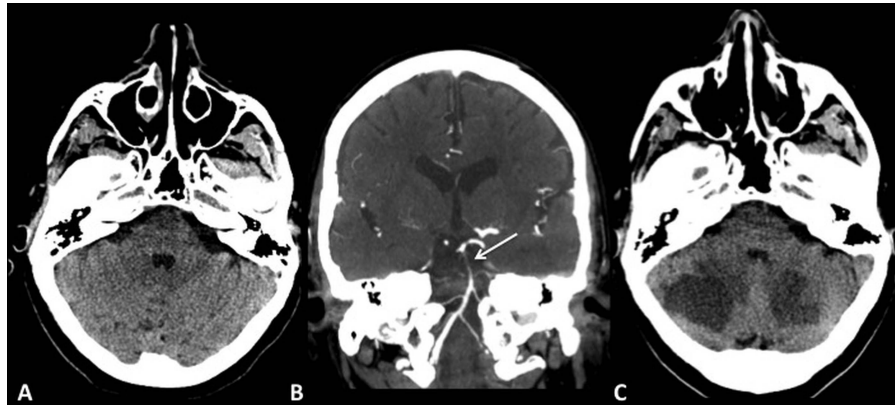


Figure 1 (A) Axial image of the baseline non-contrast CT scan of the head through pons and cerebellum. (B) Coronal view of the CT angiogram showing a 9-mm distal basilar artery occlusion (arrow). (C) Follow-up CT scan showing bilateral cerebellar strokes.

ischaemic strokes; basilar artery occlusion is responsible for approximately 5% of these.¹ The most common mechanism of basilar occlusion is *in situ* atherosclerotic disease, either isolated to the basilar artery or as part of a diffuse process of intracranial and extracranial large artery atherosclerosis. The remaining cases are due to embolic disease either from a proximal arterial source or from cardiac causes, such as valvular disease and atrial fibrillation.²

Patients with basilar artery occlusion have high rates of mortality and morbidity especially when they present with decreased level of consciousness. If recognised early, the outcomes may improve with reperfusion therapies such as intravenous thrombolysis. The considerable heterogeneity of presenting symptoms and signs, ranging from decreased level of consciousness, weakness, cranial nerve abnormalities, tetraplegia/quadruplegia, to locked-in syndrome can make the diagnosis difficult.³ Our case presented with involuntary movements resembling those related to seizure.

Spontaneous limb movements and posturing have been recognised and reported due to acute brainstem ischaemia for many years.² These convulsive-like movements can range from rhythmic tremor and dystonic movements to generalised tonic-clonic activities.⁴ The diagnosis in these cases remains extremely challenging, even by neurologists. The presence of brainstem signs and symptoms plus the acuity of presentation should prompt clinicians to consider brainstem ischaemia as an aetiology.

The pathophysiology of these spontaneous movements in brainstem ischaemia is not well understood. The role of pontine reticular formation in seizure generation and propagation has been extensively studied in experimental models.⁵ In animal models, stimulation of the brainstem reticular formation (in medulla, pons and midbrain) produces rhythmic tonic activities followed by short-lasting clonic jerks, without the concurrent cortical discharges classically recorded with cortical seizures on scalp EEGs. It is well recognised that the disruption of the descending suprasegmental motor pathways between the red nucleus and the vestibular nuclei can

result in a decerebrate posturing.⁶ Others propose this relates to a type of release phenomenon of descending pyramidal input on descending tracts originating in the midbrain.⁷ It is therefore possible that pontine hypoperfusion related to an acute basilar occlusion can produce decerebrate spasms. In our case, we hypothesise that the spontaneous movements were caused by ischaemia to the descending pathways including corticospinal, vestibulospinal and reticulospinal tracts.

The goal of acute treatment in basilar artery occlusion is to achieve recanalisation by early initiation of intravenous, intra-arterial thrombolysis, endovascular reperfusion therapies or a combination of the above. The outcome is directly related to the clinical symptoms, length and location of the occlusion, degree of achieved recanalisation and time to treatment; without recanalisation there is practically no chance of a favourable outcome.

In summary, acute occlusion of the basilar artery represents a neurological emergency, one where the diagnosis is challenging due to the heterogeneous clinical presentation, but when suspected can be confirmed with prompt vascular imaging. Neurologists and emergency physicians should have a very high index of suspicion and consider spontaneous limb movements and posturing as a manifestation of acute brainstem stroke to obtain urgent vascular imaging and involve their acute stroke service in the care of these patients.

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