



Case Report

Unusual presentation of basilar artery thrombosis

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J Cerebrovasc Endovasc Neurosurg.
2020 December;22(4):282-286

Received: 28 March 2020

Revised: 1 June 2020

Accepted: 4 June 2020

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Strokes in the territory of the posterior cerebral artery (PCA) may rarely cause acute confusion or delirium, especially when bilateral or the dominant PCA are involved. Delirium as the only initial presentation of basilar artery thrombosis (with no brainstem or long tract findings) is an extremely rare occurrence. In this article, the clinical presentation of our case was an acute confusion with septic shock-like features (tachycardia, hypotension, and leukocytosis) for a few days without any focal deficit. These symptoms pointed more toward a non-focal neurological cause, especially meningoencephalitis. This case highlights the importance of detailed history and thorough evaluation of high-risk patients who present with an acute devastating neurological syndrome. In addition, knowledge of the atypical presentation of stroke should be acquired, and the limitation of an unenhanced computed tomography scan of the brain without vascular imaging should be known. Investigating patients with a sudden acute confusion should be directed toward the evaluation of the etiology in a stepwise manner. However, the pace of investigations should be fast to establish the diagnosis and optimize the outcome.

Keywords Delirium, Basilar artery, Thrombosis, Stroke

INTRODUCTION

Delirium is a serious, non-focal, non-localizing symptom that needs to be evaluated with a broad differential, and its pathophysiological mechanism remains poorly understood.¹⁾ Delirium is an acute illness affecting cognition and consciousness that is transient in nature and has a fluctuating course. It can be a clinical manifestation of a wide variety of systemic and brain disorders.¹⁵⁾

Basilar artery thrombosis is a potentially fatal condition, and one of the most challenging conditions for neurologists to diagnose and manage. It approximately ac-

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counts for 1-4% of all ischemic strokes.¹⁴⁾ Basilar artery thrombosis is caused by local thrombosis of an atherosclerotic stenotic basilar artery. Atherosclerosis is typically extensive and usually not confined to the posterior circulation.⁵⁾ When compared to anterior circulation syndrome, basilar artery thrombosis has a longer prodrome with latency between first prodromal symptom and stroke onset ranging from days to months. The most common prodromal symptoms are nausea, vomiting, vertigo, neck pain, and headache.⁸⁾

Delirium as the only initial presentation of basilar artery thrombosis (with no brainstem or long tract findings) is an extremely rare occurrence. Strokes in the territory of the posterior cerebral artery (PCA) may rarely cause delirium, especially when bilateral or the dominant PCA are involved. The involvement of the medial occipitotemporal gyri seems responsible for the development of delirium. In addition, it may be caused by the disconnection of anterior limbic structures from neocortical inputs by the severance of the pathway linking dominant temporal neocortex and limbic system or by the destruction of posterior limbic structures that were hypothesized to explain what has been known as 'agitated delirium'.⁸⁾ In this article, we describe a patient who presented with delirium and septic shock-like features (tachycardia, hypotension, and leukocytosis) who was misdiagnosed initially as viral meningoencephalitis.

CASE REPORT

A 60-year-old man was admitted with acute onset of confusion, personality change, and intermittent headache. His symptoms worsened over three days prior to admission. There was no history of nausea, vomiting, fever, or seizures. His past medical history was significant for hypertension, but he was not using any medications. He was a non-smoker and never consumed alcohol. In the emergency department, vital signs including blood pressure and temperature were normal. He was confused, disoriented to time and place with no neck rigidity or stiffness. Neurological examination showed

a normal tone and power (muscle strength by medical research council scale) with normal symmetrical deep tendon reflexes. His gait was normal, but the examination of coordination and fundi was difficult to perform. Unenhanced computerized tomography (CT) scan of the brain was normal. The patient was taken to the magnetic resonance imaging (MRI) suite, but he was moving and irritable, which made this imaging modality difficult to perform. Routine investigations revealed normal biochemical tests and inflammatory markers. His white blood cells (WBC) count revealed a leukocytosis (20,000) with normal platelets and hemoglobin. Blood and urine cultures and chest radiographs were unremarkable. Examination of cerebrospinal fluid revealed no WBCs or red blood cells, normal glucose and protein with negative gram stain and culture. Polymerase chain reaction tests were negative for common viral pathogens. Electroencephalogram recordings showed a slow background in the theta range with no epileptiform activity or evidence of encephalitis. Two days after admission, his condition deteriorated, and he became more agitated and combative with a drop in his blood pressure to 80/40 mmHg and tachycardia (120 bpm). He was intubated and mechanically ventilated, but unfortunately, his Glasgow coma scale dropped. His pupils became fixed and dilated. Repeat work-up of septic shock was again unremarkable, and he required inotropic support. Another CT of the brain with imaging of the vascular system was performed, which demonstrated basilar artery thrombosis and ischemic changes of both cerebelli, brainstem, and both thalami and occipital lobes (Fig. 1). He was rebooked for another urgent MRI and magnetic resonance angiography of the brain, but unfortunately, his condition was dismal, and all his brainstem reflexes were absent, and the study was not obtained. He was declared dead two days later.

DISCUSSION

Acute basilar artery occlusion is a rare devastating type of stroke that represents only 1-4% of all strokes. It

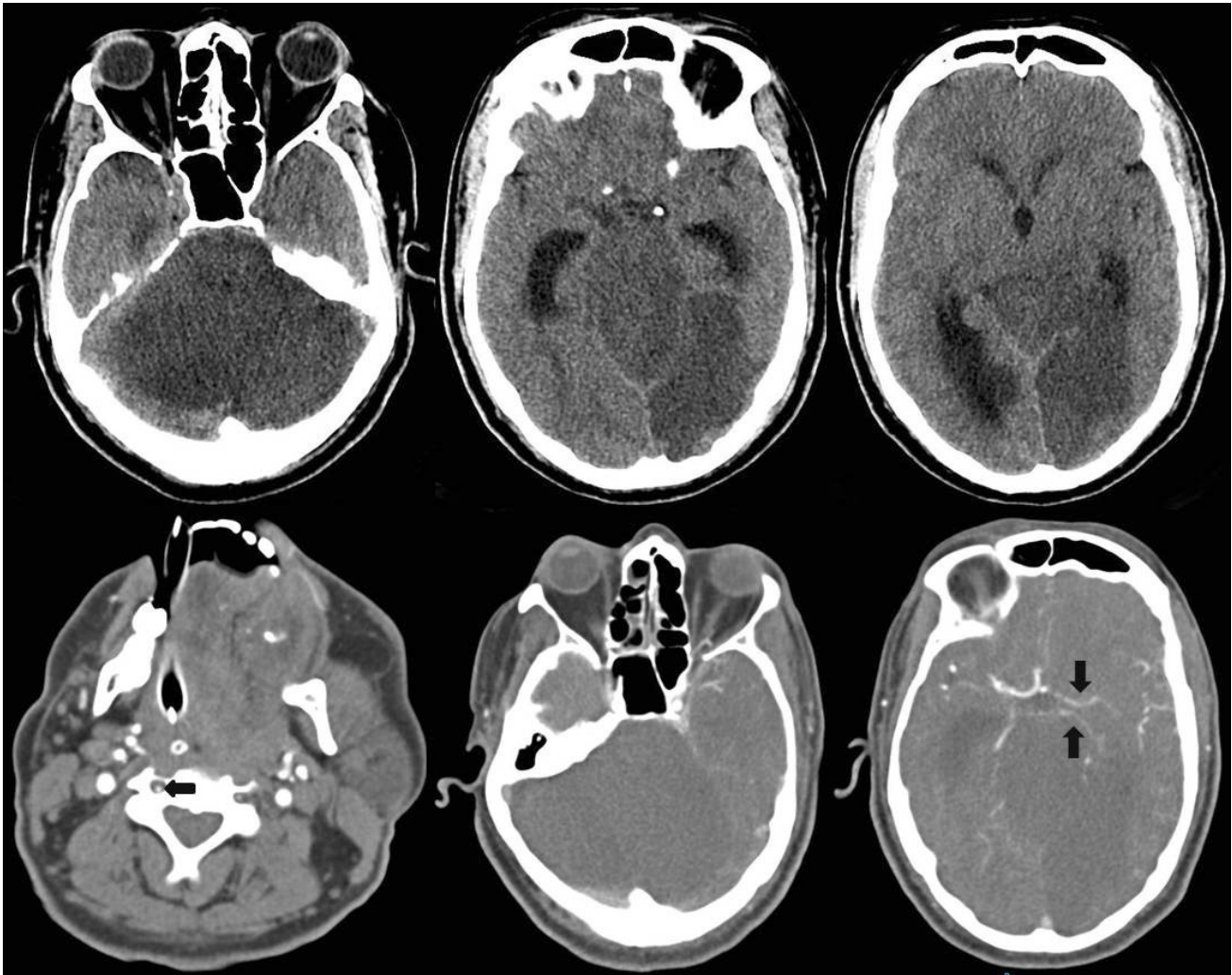


Fig. 1. CT scan of the brain showing bilateral cerebellar, occipital, cortical, and subcortical hypo-attenuation more evident at the left side associated with unilateral left-sided thalamic area of hypoattenuation suggesting acute ischemic insult/infarction secondary to vertebrobasilar system occlusion. Non-visualized basilar artery as well as its branches with patent anterior circulation denoting vertebrobasilar total occlusion at the time of the scan. CT, computerized tomography.

results in severe disability and significant morbidity.¹¹⁾ In a study done by Wijdicks et al.¹⁶⁾ in 1996 with a total of 25 patients, 22 of them died, and the three survivors had “locked-in” syndrome. In another study done by Schonewille et al.¹³⁾ (The Basilar Artery International Cooperation Study [BASICS]), 26 of 27 patients with basilar artery occlusion who did not receive any kind of therapy died within four weeks, and one patient was bedridden and incontinent in a long-stay facility. Early diagnosis within the window of intervention allows the use of intravenous thrombolysis, intra-arterial thrombolysis, and mechanical endovascular procedures

(thrombectomy). Such procedures allow for recanalization and subsequent improvement in the functional outcome. In basilar artery occlusion, the time window for intervention has often been longer than the typical window used in the anterior circulation.⁷⁾ The degree of baseline ischemia evaluated by CT using posterior circulation acute stroke prognosis early CT score is more important than the time of intervention.³⁾

Up to two-thirds of patients with basilar artery strokes have prodromal events including transient ischemic attack, minor strokes, and other symptoms such as headache, dizziness, vertigo, altered level of consciousness,

and motor and sensory symptoms. In a study done by Organek et al.¹²⁾ in 2015, they reported five patients with mild transient symptoms as long as two weeks before the catastrophic worsening. These symptoms include headache, fluctuating motor weakness, coma, dizziness, nausea, vomiting, and sensory symptoms. These results were similar to another study done by Ferbert et al.⁶⁾ who documented a case series of 85 patients with basilar artery occlusion. Around 60% of their patients had prodromal symptoms, mainly vertigo, nausea, headache, and neck pain. Again, these prodromal symptoms had onset about two weeks before a formal diagnosis.

Until recently and based on anecdotal reports and poorly conducted studies, anticoagulation with intravenous heparin was the therapy of choice for acute basilar artery thrombosis. This therapy is of little benefit with a mortality of 88% and “locked-in” syndrome in the remaining 12%.⁹⁾ Recently, intravenous or intra-arterial use of thrombolytic agents has been introduced with successful recanalization rates ranging from 44-88%. Although better, the mortality rate has remained high because of recurrent thrombosis, hemorrhagic complications, and irreversibility of the stroke itself. The overall good outcome with no or mild deficit is 20-25%.²⁾

Unenhanced CT is relatively insensitive in detecting early signs of posterior circulation ischemia, as seen in our patient with normal initial CT of the brain. This is due to imaging artifacts that limit its usefulness.¹⁵⁾ In patients with clinical neurological findings suggestive of posterior circulation ischemia, further evaluation with CT angiography, magnetic resonance angiography, or conventional four vessels cerebral angiography is required. These imaging techniques will precisely visualize the basilar artery and rule out filling defects due to occlusion.⁴⁾ Although the second CT brain with angiography demonstrated basilar artery thrombosis with accompanied ischemic changes in our case, the diagnosis was late. An MRI of the brain is more sensitive than an unenhanced head CT for the detection of acute stroke, especially posterior circulation strokes. The diffusion-weighted imaging sequence paired with an apparent diffusion coefficient map are the most sensitive

sequences for acute stroke (90-94% sensitivity).⁴⁾¹⁰⁾ We recommend obtaining intracranial vessel imaging on all patients who are at high risk for intracranial strokes and presenting with unexplained acute delirium to rule out basilar artery occlusion.

CONCLUSIONS

The clinical presentation of our case was an acute confusion with septic shock-like features (tachycardia, hypotension, and leukocytosis) for a few days without any focal deficit. These symptoms pointed more toward a non-focal neurological cause, especially meningoen- cephalitis. A high index of suspicion for basilar artery occlusion should be practiced in patients with acute confusion of sudden onset and lack of features sugges- tive of an alternative diagnosis. This case highlights the importance of detailed history and thorough evaluation of high-risk patients who present with an acute devas- tating neurological syndrome. In addition, knowledge of the atypical presentation of stroke should be acquired, and the limitation of an unenhanced CT of the brain without vascular imaging should be known. Investigat- ing patients with a sudden acute confusion should be di- rected toward the evaluation of the etiology in a stepwise manner. However, the pace of investigations should be fast to establish the diagnosis and optimize the outcome.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the find- ings specified in this paper.

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