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Case Report

Delays in Diagnosis and Treatment of a Basilar Artery Occlusion in a Young Female

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Abstract

We report a case of a 30-year-old female with a history of intravenous drug abuse, who arrived to the ED after 5 hours of delay from the initial EMS call from her mother. She has a history notable for an incompletely treated methicillin-resistant Staphylococcus aureus (MRSA) bactermia. Upon admission, she was unresponsive and hypotensive so she received antibiotics and hydration for sepsis. After initially being missed in the ED, a CT angiogram of the head was eventually done that found a basilar artery occlusion. After a mechanical thrombectomy, she was intubated and admitted to the NICU. A MRI showed innumerable areas of infarcts as well as subarachnoid hemorrhage responsible for an evolving rostral hydrocephalus. Recovery was unlikely and after discussion with the family and the palliative care team, the patient was discharged 2 days later to hospice to receive comfort care. We report this case to show how initial delay as well as misdiagnosis of basilar artery occlusion could lead to irreversible neurological devastation and death.

Introduction

The posterior circulation of the brain derives from cervical arteries. The vertebral arteries that arise from the subclavian arteries fuse to become the basilar artery (BA).1 The basilar artery is responsible for the irrigation of vital areas of the brain including the cerebellum, brainstem, thalamus, occipital and medial temporal lobes.2 Posterior circulation strokes (PCS) affect around 100 000 people in the USA annually. They account for 20 % of ischemic strokes.3 In this case report, we discuss how the misdiagnosis and delay of treatment of a basilar artery occlusion (BAO) can lead to devastating outcomes and death.

Case Description

We hereby report the case of a 30-year-old female with a medical history of intravenous drug abuse, hepatitis C, a MRSA infection of the mitral valve and a groin infection that was not completely treated. The history goes back to five hours before admission when the patient's mother was not able to reach her over the phone. The mother then contacted the Emergency Medical Services (EMS). However, when the EMS arrived to site, they were uncomfortable with breaking down the door. They had to wait 4 hours for the mother to arrive with the key. Upon entering the house, they found an unconscious patient covered with vomit. The last contact the mother had with the patient was the afternoon before. There was a five hour delay from the time EMS arrived to her place to the time she was admitted.

At the Emergency Department (ED), the initial assessment found a hypotensive (97/60 mmHg) and tachypneic (36/min) patient. The initial neurological assessment shows an unresponsive patient, with asymmetrical pupils (left: 5mm; right: 3mm). Her members extended to noxious stimuli. Her initial blood workout shows anemia (Hb= 7.9 g/dL) and a low platelet count (129 000/µL). On the suspicion of sepsis, she was started on piperacillin-tazobactam and vancomycin as well as intensive hydration.

Neurology was then consulted. On the neurological examination, they found a comatose patient with a Glasgow Coma Scale (GCS) of 4 and her initial National Institute of Health Stroke Scale (NIHSS) was of 30. A head CT with both CT angiography (CTA) and CT perfusion (CTP) show basilar artery occlusion. (Figure 1) The occlusion was associated to a limited opacification versus occlusion involving the distal right V2 and proximal V3 segments and on the CTP, a 3mm penumbra in left cerebellum, no core infarct respectively.

Since her last known well could not be accurately determined, she could not receive intravenous thrombolysis and was instead planned for mechanical thrombectomy. This was 3 hours after her arrival to the emergency department. The mechanical thrombectomy confirmed the occlusion involving the proximal basilar artery with extension into bilateral posterior cerebral arteries. She was treated with combined distal aspiration stent retriever maneuver five times leading to a TICI 2b reperfusion. Afterwards, she was transferred to the NICU. Overnight, she developed septic shock. She was put on vasopressors with a goal MAP > 65.

After stabilization, she underwent an MRI the second day that showed innumerable areas of evolving infarction within the brain stem, cerebellum, and both cerebral hemispheres, and an increasing pressure in the posterior fossa resulting in the evolving rostral hydrocephalus and a small amount of hemorrhage layering dependently within the occipital horns of both lateral ventricles. (Figures 2 and 3) Increased signal intensity in the perimesencephalic and interpeduncular cisterns noted on the FLAIR images is suggestive of interval development of subarachnoid hemorrhage that could also account for the evolving hydrocephalus.

The neurological examination at this point showed a comatose young woman with dysconjugate gaze. She had spontaneous downward saccades of her right eye. Her left eye did not move but had exotropia and slight down-and-out deviation. Decerebrate posturing of all extremities was noted with noxious stimulation. Swelling in her lower limbs necessitated an ultrasound study that did not find a significant abnormality related to infarcts. However, multiple infarcts were found on the spleen, kidneys and brain.

Given her poor neurologic status, an assessment of the palliative team as well as a discussion with the family led to the decision of limiting the care to comfort treatments and a discharge towards a hospice.

A few days later, she passed away peacefully.

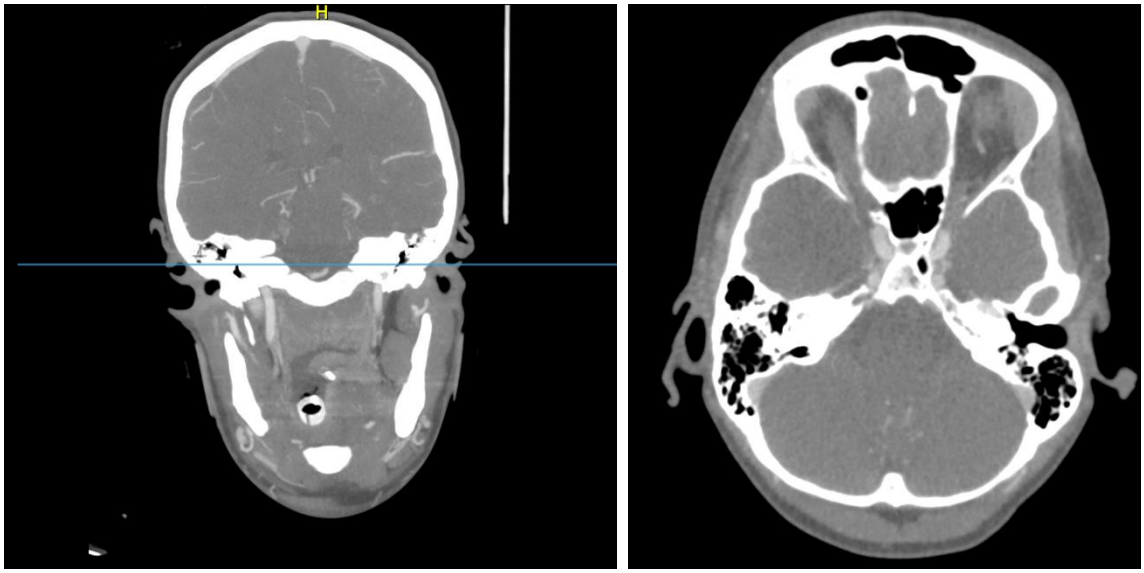

Figure 1. Head CT showing the basilar occlusion on both coronal and axial cuts

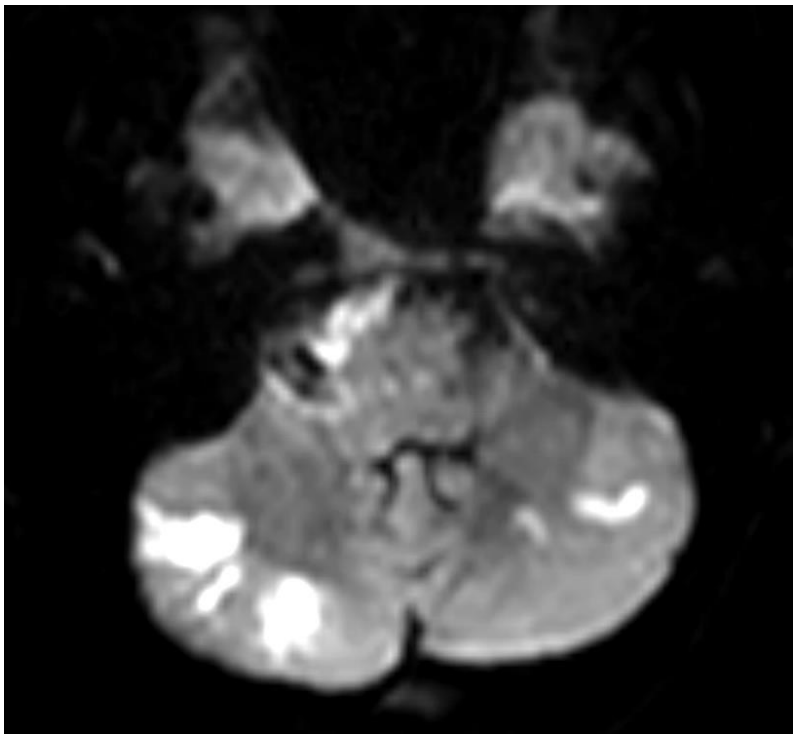

Figure 2. MRI showing diffusion restricting lesions in the cerebellum

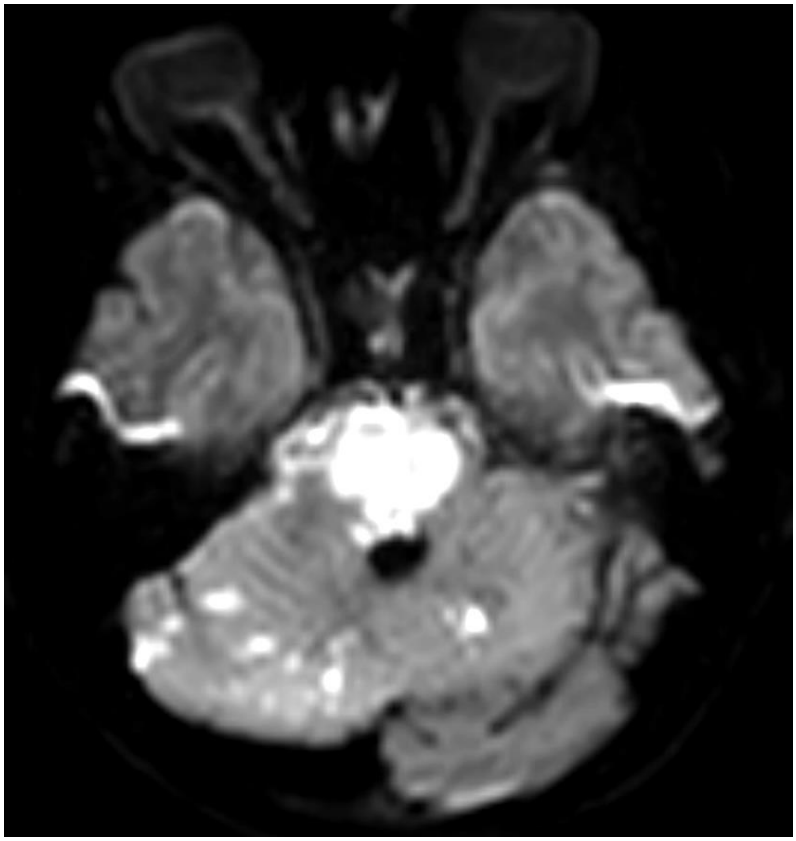

Figure 3. MRI showing diffusion restricting lesions on the brainstem

Item	Description	Score Options	Baseline	24 hours later	Discharge (2 days later)
1a. LOC	Level of consciousness	0 = Alert; 1 = Drowsy; 2 = Obtunded; 3 = Coma	3	3	3
1b. LOC Questions	Month, age	0 = Both correct; 1 = One correct; 2 = None	2	2	2
1c. LOC Commands	Open/close eyes, grip	0 = Both correct; 1 = One correct; 2 = None	2	2	2
2. Best Gaze	Horizontal eye movement	0 = Normal; 1 = Partial palsy; 2 = Forced deviation	0	2	2
3. Visual	Visual fields	0 = No loss; 1 = Partial	3	3	3

		hemianopia; 2 = Complete; 3 = Bilateral hemianopia			
4. Facial Palsy	Facial movement	0 = Normal; 1 = Minor; 2 = Partial; 3 = Complete	3	3	3
5a. Motor Arm (L)	Arm drift (10s)	0 = No drift; 1 = Drift; 2 = Some effort; 3 = No effort; 4 = No movement	3	3	4
5b. Motor Arm (R)	Arm drift (10s)	0–4 (as above)	3	3	4
6a. Motor Leg (L)	Leg drift (5s)	0–4 (as above)	3	3	4
6b. Motor Leg (R)	Leg drift (5s)	0–4 (as above)	3	3	4
7. Limb Ataxia	Finger-nose, heel-shin	0 = Absent; 1 = One limb; 2 = Two limbs	0	0	0
8. Sensory	Pinprick	0 = Normal; 1 = Mild; 2 = Severe	0	1	0
9. Best Language	Aphasia	0 = No aphasia; 1 = Mild; 2 = Severe; 3 = Mute	3	3	3
10. Dysarthria	Speech clarity	0 = Normal; 1 = Mild; 2 = Severe; or Intubated/unassessable	2	Intubated	2
11. Extinction/Inattention	Neglect	0 = None; 1 = Mild; 2 = Severe	0	2	0
Total NIHSS	(0–42)	Sum of above	30	33	36

Table 1. The evolution of the NIHSS for the patient from admission to discharge

Discussion

Since the presentation of posterior strokes is usually more insidious, frequently with nonfocal symptoms like confusion and altered mental status, patients with PCS are twice more likely to be misdiagnosed than patients with anterior strokes.[4] This is especially true for young patients, even in academic hospitals.[5] In the current case, the patient presented with altered consciousness, hypotension, tachypnea and anemia. All of these symptoms are consistent with septic shock, especially in a patient with a history of IV drug use and recent incompletely treated MRSA.[6] However, the initial diagnosis and focus on sepsis delayed the diagnosis and treatment of BAO in this case.

Moreover, BAOs are associated to a high mortality rate even months after treatment.[7] The mainstay of acute treatment is intravenous thrombolysis (if last-known well is within 4.5 hours) and endovascular thrombectomy.[8] In this case, the last-known well of the patient was unknown and consequently, she could not benefit from chemical thrombolysis. Furthermore, the success rate of mechanical thrombectomy depends on the timely recognition of stroke.[9] A matched study shows that basilar artery strokes are subject to significant delays when compared to left middle artery strokes. Indeed, the median delay from symptom onset to ED arrival was shown to be of 7 hours and 32 minutes. The median delay from ED arrival to diagnosis was shown to be of 7 hours. Only 19 % of BA strokes were recognized within the IV thrombolysis window compared to 62 % of LMCA strokes.[10]

In the present case, delays before both arrival to the ED and diagnosis occurred. The total delay from contacting the EMS until thrombectomy was of 8 hours. This significantly decreases that chances of successful outcomes with mechanical thrombectomy, despite successful reperfusion. The patient did not regain consciousness after thrombectomy, thus showing the importance of early recognition and treatment of BAO.

In the medical literature, multiple cases report misdiagnosis of BAO. Notably, a case of a young woman in post-partum presenting with convulsive activity and encephalopathy was presumed to have toxin-related seizures, encephalitis, or serotonin syndrome, and only later was found to have BAO on a CT angiogram and multiple strokes on the MRI.[11] Another report describes the case of a 60 years old man with confusion, personality change, intermittent headaches, and leukocytosis that had an initial normal CT scan. He was first diagnosed with sepsis and only a second CT after he deteriorated and was intubated found BAO and ischemic changes. He passed two days after admission.[12]

Moreover, drug abuse is related to an increase in arterial events with multiple possible etiologies.[13] A paper from 2002, reports the case of a 25-years old female, with a history of cocaine and ecstasy abuse, that presented initially with confusion, dysarthria and right hemiplegia, 72 hours following substance use.

She underwent a CT scan 9 hours later, that showed no abnormality and an MRI and MR angiography later done showed images compatible with BAO. She was referred to the stroke center 30 hours after the onset of symptoms, where she had thromboaspiration that reduced her NIHSS from 17 to 7.[14] Another paper reports the case of a 75 years old man with a history of cocaine use, presenting with asymmetrical and dilated pupils, right-arm jerks and extensor posturing with noxious stimuli that was diagnosed to have BAO on imaging. He could not benefit from endovascular treatment and kept deteriorating after admission until deceasing shortly afterwards from pneumonitis.[15]

Another case series from Sudan described the course of BAO in three patients, two women of 36 and 30 years old and a man of 85 years old. While the context differed between each patient, a snakebite for the first, chronic bifrontal headache during pregnancy for the second and end-stage dialysis in the third, they all showed different initial symptoms patterns, ranging from left-sided weakness to fever, decrease level of consciousness and even loss of consciousness. All three patients were treated with anticoagulation. The third patient died during dialysis.[16]

Infective endocarditis is a severe complication in patients with IV drug abuse, and Staphylococcus aureus is the most common causative agent. A full course of antibiotics reduces drastically the hazard of further events related to the infection.[17] In the case of our patient, the discontinuation of antibiotics against medical advice before full course was permissive of further complications. Brain strokes occur in 10 to 35 % of patients with left-sided IE.[18]

When including silent CVC, the toll raises to 65 % of patients with IE.[19] The ETT study in this case showed a leaflet on the mitral valve, which probably led to the thrombus that lodged in the BA of our patient. Furthermore, factors like thrombocytopenia were associated with a higher risk of hemorrhage in patients, which increases the in-hospital mortality from 22 % to 63.6 %.[20] Our patient had a low platelet count and FLAIR images were suggestive of subarachnoid hemorrhage.

A high initial NHSS has been correlated to a higher risk of mortality after acute ischemic stroke.[21] Since the scale weighs items that are more frequently related to anterior strokes such as aphasia, motor deficits and gaze preference, the severity of posterior strokes can be underestimated.[21] In the current case, the patient had an initial NHSS of 30, showing the initial severity of the stroke. The NHSS kept worsening after admission despite thrombectomy, and the patient eventually transitioned to comfort care and passed away.

Conclusion

In conclusion, this case shows the tricky presentation of basilar artery strokes, which leads in most cases to diagnostic and treatment delays. Accurate diagnosis and timely intervention increase the chances of successful reperfusion and neurologic recovery. Delays in diagnosis and treatment can lead to catastrophic outcomes. This case intends to raise awareness on BAO in young patients, in particular those who present risk factors like IV drug use, in order to improve outcomes.

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