

Basilar Artery Occlusive Disease

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Chief Complaint: Left-sided Weakness

History of Present Illness:

71-year-old male with past medical history of hypertension (HTN), chronic obstructive pulmonary disorder (COPD) and Opioid use disorder presents with left-sided weakness for the past 12 hours. The patient reports no symptoms prior to falling asleep but woke up with immediate weakness in his left leg, dizziness, and blurred vision. This was followed with an inability to walk as he could not support his full body weight on his left side. The patient initially received medical attention at Norwegian American Hospital Emergency Department and was later transferred to UIC with suspected stroke. The patient described the weakness in his left hand and left leg as they were “not doing what he wanted it to do.” He denies any previous similar episodes, pain or nausea and states he did not realize if he was talking funny. Patient is worried because he has never felt this way.

Past Medical History:

HTN (uncontrolled), Opioid Use Disorder, COPD

Past Surgical History:

Abdominal surgery after GSW in remote past

Medications:

HTN, doesn't recall name and ran out
Albuterol, but ran out

Allergies: NKDA

Social History:

Tobacco: Denies, but significant secondhand exposure
Alcohol: 1-2 beers on occasion
Illicit drug use: uses 2-3 bags of heroin per day, snorts and previous IVDU in the 80's

Review of Systems: positive findings

Eyes: Blurred vision, *Respiratory:* Wheezing, *Neuro:* L sided weakness, dizziness as above in HPI

OBJECTIVE: 71-year-old male with past medical history of HTN, COPD, Opioid Use Disorder, transferred from Norwegian American Hospital with L sided weakness. Imaging suggestive for basilar artery occlusion. MRI demonstrates multiple lesions consistent with R PCA stroke.

Vital Signs:

BP: 195/103
Pulse: 54
RR: 18

Labs/Imaging:

MRI: multifocal area of non-hemorrhagic early acute ischemic change involving the posterior limb of the right internal capsule extending into the right thalamus, occipital lobe, splenium of the corpus callosum and in the white matter adjacent to the atrium of the right lateral ventricle, which extends along the temporal structures
ECG by 12-lead: sinus brady

Physical Exam:

SPO2: 100
Height (cm): 78.01
Weight (kg): 34.76

National Institutes of Health Stroke Scale: total= 8

Facial Palsy Score= 2, partial paralysis in lower face
Motor LUE Score= 1, drift but doesn't hit bid; limb holds 90 degrees, but drifts down before full 10 seconds
Motor LLE Score= 1, drift but doesn't hit bid; limb holds 90 degrees, but drifts down before full 10 seconds
Dysmetria Score= 2, present in two limbs
Dysarthria Score=2, unintelligible in absence of aphasia

Diagnosis

❖ Basilar Artery Occlusive Disease with lesions consistent in the right posterior cerebral artery.

❖ Progression of disease:

- ❖ Patient initially presented with symptoms consistent with neurological deficits including: sudden ipsilateral weakness, dizziness and vision changes
- ❖ Following this presentation patient was evaluated using National Institute of Health Stroke Score, scoring positive for 8 points
- ❖ Imaging included MRI confirmed suspected stroke, which showed multiple lesions following the posterior cerebral artery consistent with occlusion from atherosclerotic plaque.

❖ Assessment and plan.

- ❖ 1. Plan: continuous neurology checks, dysphagia screening prior to any oral intake, speech therapy
- ❖ 2. Plan: medication management for previous conditions & newly diagnosed

Discussion of Disease Process/Clinical Correlations

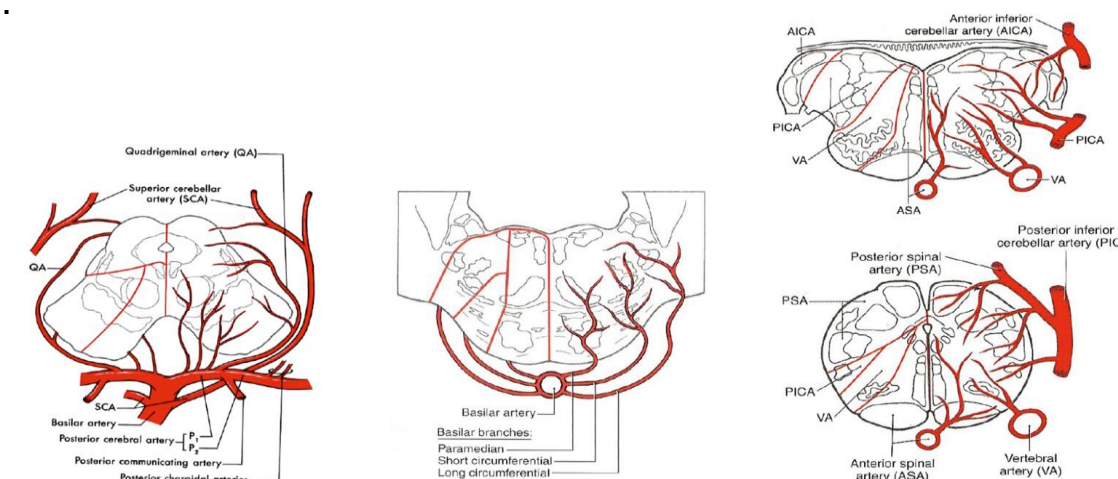
❖ Basilar Artery Occlusion Disease is often the result of artery to artery (intraarterial) thrombi from underlying atherosclerosis. Most often presentation is ischemia of the pons, which presents as case discussed above. Severity of the occlusion depends on the location and progression of the ischemia. The basilar artery includes many anastomoses, which often spares the function of many structures.

❖ Etiology of Atherosclerosis:

- Atherosclerosis is a disease in which plaques build up within the arteries and may compromise blood flow to essential structures following the blood flow.
- Plaques are composed of fats, calcium deposits and cholesterol. As they progress over time they may calcify and decrease normal blood flow. When a piece of the plaque is dislodged from the initial deposition site in the artery it may travel to other areas of the body, often the brain.
- The causes of plaque development vary, but often include poor diet, lack of exercise, lifestyle choices, hypertension, diabetes and other chronic conditions.
- This patient is housing and food insecure. He is diagnosed with uncontrolled hypertension and has a history of drug use and smoke exposure. A combination of these factors support the environment for plaque development.

❖ Paramedian pons:

- The primary blood supply to the paramedian pons is supply directly by the basilar artery.
- The paramedian pons contains a descending long motor tract and oculomotor fibers. Ischemia to this area presents as both ocular and motor deficits.
- This patient had evident atherosclerosis, and development of an embolism that compromised a portion of the right basilar artery supplying blood to the paramedian pons. The symptoms included both ocular and motor deficits to the left side of the body. Consistent with pons occlusion.



Current Research and New Treatments

- ❖ Prognosis of basilar artery occlusive disease (BAOD) was evaluated by comparing the cognitive and functional profiles of 28 patients with education and level-matched healthy controls (Table 1). Patients with BAOD were found to have significant impairments in verbal episodic memory (immediate and delayed recall) and visuospatial episodic memory (immediate and delayed recall and recognition)¹
- ❖ New treatments for basilar artery occlusion and ischemia when basilar trunk accessibility is not feasible involve retrograde revascularization with thyrocervical collaterals for anterograde mechanical aspiration and stenting of the occluded artery² (Figure 1)
- ❖ A case report of a 79-year old Caucasian Italian woman depicted the first use of transcranial doppler to monitor and diagnose basilar occlusive disease. Researchers conclude that this methodology should be applied in all situations of unexplained acute loss of consciousness or signs of brain dysfunctions.³
- ❖ A 2015 study of the Basilar Artery International Cooperation Study depicted that poor prognosis of patients with severe basilar artery occlusion was related to treatment beginning 9 hours after symptom onset⁴

Table 1

Comparison of cognitive outcomes (raw score) between BAOD patients and healthy controls.

Cognitive functions	Cognitive tests	Patients Mean (SD)	Controls Mean (SD)	p
Short-term memory	Digit Span	10.0 (2.52)	10.85 (2.73)	0.20
	HVLT-R immediate recall	18.04 (5.10)	21.85 (3.11)	0.002
	HVLT-R delayed recall	5.04 (2.85)	6.81 (1.78)	0.008
	HVLT-R recognition	8.86 (2.22)	10.00 (1.11)	0.04
Long-term memory (visuospatial episodic memory)	BVMT-R immediate recall	14.78 (10.11)	19.59 (6.52)	0.04
	BVMT-R delayed recall	5.59 (4.65)	7.67 (2.90)	0.03
	BVMT-R recognition	4.85 (1.35)	5.70 (0.54)	0.01
	Language			
Naming	BNT	43.89 (8.77)	48.48 (5.21)	0.04
	Executive function			
Monitoring rules	Phonemic Verbal Fluency	23.73 (10.67)	26.62 (8.09)	0.27
	Category Fluency	12.11 (4.43)	13.59 (2.62)	0.09
Mental flexibility	MWCST	3.29 (1.8)	4.48 (1.50)	0.01
Perceptive ability	Perceptive and visuospatial ability			
	Fragmented Letters	18.52 (2.08)	19.15 (0.93)	0.64
Visuospatial ability	Position Discrimination	18.14 (1.94)	19.59 (0.75)	0.002
	Attention and processing speed			
Processing speed	SDMT	18.14 (12.10)	28.26 (10.57)	0.001
	TMT Part A	70.61 (55.26)	50.85 (20.28)	0.15
Sustained attention	TMT Part B	202.13 (86.95)	135.27 (48.08)	0.01
Set-shifting attention	Stoop C	49.00 (22.34)	33.58 (7.29)	0.001
Selective attention	MMSE	25.09 (3.68)	27.44 (1.45)	0.01

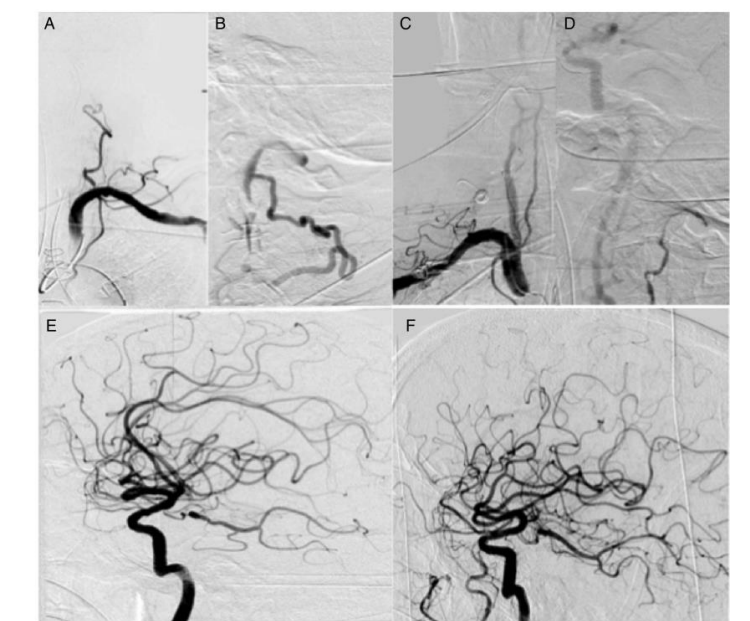


Figure 1 Left subclavian anteroposterior (A) and lateral (B) injection demonstrating a completely occluded left vertebral artery (VA) with distal recanalization of the V3 segment via tortuous muscular collaterals from the thyrocervical trunk. Right subclavian anteroposterior (C) and lateral (D) injection showing a hypoplastic VA with no intracranial opacification of the posterior circulation. Right (E) and left (F) internal carotid artery lateral injection demonstrating diminutive posterior communicating arteries bilaterally with no retrograde flow down the basilar.

Conclusion. Basilar artery occlusion can have devastating results involving cognitive and functional deficits or even death. Prompt diagnosis and monitoring via transcranial Doppler and emergent treatment with revascularization can help in reducing mortality and poor prognosis.

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Thank your mentor for their contributions and their time.