IOSR Journal of Nursing and Health Science (IOSR-JNHS)
e-ISSN: 2320–1959.p- ISSN: 2320–1940 Volume 11, Issue 3 Ser. IV (May. – June. 2022), PP 40-44
www.iosrjournals.org

Basilar Artery Occlusion Syndrome

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Abstract: *Basilar artery occlusions (BAO) are a subset of posterior circulatory strokes. The special issues relevant to BAO include variable and uncertain symptoms at onset, resulting in delays in diagnosis, high morbidity and mortality, and uncertain management. Despite imaging techniques, diagnosis and therefore, treatment is often delayed.(1) The presentation of basilar artery occlusion varies depending on the cause of the occlusion and the location of the ischemia. The areas of the brain provided by the posterior circulation are difficult to visualize and usually require angiography or magnetic resonance imaging.(2)*

Keywords: *occlusion of the basilar artery, thrombosis of the basilar artery, intra-arterial treatment, intravenous thrombolysis, thrombosis of the basilar artery, stroke.*

Date of Submission: 25-05-2022 Date of Acceptance: 07-06-2022

I. Introduction:

Basilar artery occlusion (BAO) is a potentially fatal diagnosis, but it is one of the most difficult diseases to diagnose and treat. Posterior circulatory strokes account for approximately 15% to 20% of all ischemic strokes. BOA is a subset of this category, accounting for 1% to 4% of all ischemic strokes. Unlike hemispheric ischemia, where there is usually a sudden onset of focal symptoms, BOA syndromes can mimic other conditions without a stroke, leading to a delay in neurological evaluation.(1) Intervention may lead to complete neurological recovery despite severity of initial deficits.(2)

Basilar artery occlusion. The basilar artery is a major component of the posterior circulation (contributing to the circle of Willis) for the structures of the posterior cranial fossa, including the brainstem and cerebellum.(3) The basilar artery passes anteriorly to the brainstem and is formed by joining the vertebral arteries.

- **Basilar artery occlusion** can have a variety of clinical manifestations ranging from transient weakness or paresthesia to almost complete paralysis.(3)
- **Complete occlusion of the proximal or middle basilar artery** is accompanied by retained oculomotor function and consciousness, but all other voluntary muscle movements are lost.(3)
- **Complete occlusion of the distal basilar artery** can cause ischemia in the midbrain and thalamus, most often leading to oculomotor abnormalities and changes in alertness and behaviour.(3)
- **Partial occlusions of the basilar artery** can lead to a variety of deficits depending on the location and severity of the occlusion and the anatomical regions affected.(3)

Basilar artery occlusion is associated with a poor prognosis, but the emergence of high-quality, reliable and non-invasive technology (e.g. MRI) allows early diagnosis of BAO in patients with partial occlusion, limited ischemic lesions. Although results continue to be poor, progress in pharmacological and mechanical thrombolysis and endovascular therapy may increase the survival rate and limit the rate of disability.(4)

The location of the arterial occlusion is closely related to the symptoms.

Given the anatomy of the posterior circulation and the circle of Willis, the clinical manifestations depend on the location of the occlusion, the extent of the thrombus, and the collateral flow. Normally, blood flows in an anterograde manner from the vertebral arteries to the basilar artery to its terminal branches. This flow pattern may vary. If the proximal segment of the basilar artery is occluded and the occlusion results from a slow progressive stenosis, collateralization occurs in the cerebellum in the circumferential branches of the basilar artery. Additionally, the flow can be reversed from the posterior cerebral artery (PCA) to the distal basilar artery.(4)

The most common causes of posterior circulatory ischemia are similar to those of anterior circulatory strokes and include embolism, large artery atherosclerosis, penetrating disease of the small arteries, and arterial dissection.(1)

The mechanism of occlusion of the basilar artery is different depending on the segment of the vessel that is occluded.

- Atherosclerotic occlusive disorder predominantly affects the middle segment of the basilar artery followed by the vertebrobasilar junction.

- Embolism, either from a cardiac or arterial cause, is much more common in the distal third of the basilar artery and vertebrobasilar junction.
- Arterial dissection is much more common in the extracranial vertebral artery. It is often associated with a previous neck injury or chiropractic adjustment. Intracranial dissections are unusual.(4)
- Rare causes that are more specific to the posterior circulation include the cervical spine or skull base fracture, cervical instability, arteritis, meningitis, aneurysms, hereditary arteriopathy and neurosyphilis.(1)

BAO syndrome is presented under the form of a posterior circulation stroke.

- Compared with anterior circulation stroke syndromes, posterior circulation ischemia may have a longer prodrome and an evolution that is important to recognize.(1)

Symptoms associated with basilar artery occlusion may be sudden, especially in embolic and distal occlusions, but many patients with proximal atherosclerotic occlusions have prodromal symptoms.

Patients may experience almost complete recovery if treatment is provided promptly.

The time from onset of symptoms to diagnosis in the emergency department is often significantly delayed in occlusion of the basilar artery.(3)

- A progressive course of symptoms or transient ischemic attacks in the vertebro-basilar territory is found in patients with atherosclerotic occlusion.

50% of patients suffer from transient ischemic attacks or a progressive and lingering course of the disease from a few days to a few weeks before occlusion.

The most common symptoms include:

- Motor deficits such as hemiparesis or tetraparesis and facial paresis: 40 - 67% of cases (5,6)
- Dysarthria and poor speech: 30 - 63% of cases
- Dizziness, nausea and vomiting: 54 - 73% of cases
- Headache: 40 - 42% of cases
- Visual impairment: 21 - 33% of cases
- Altered affect: 17 - 33% of cases
- In some cases, convulsive movements with herald hemiparesis - may be the only diagnostic indications
- Occasionally, patients may present with isolated vertigo or dizziness without other neurological symptoms, but this is very rare.
- The presence of vascular risk factors, headache and inability to walk may suggest a diagnosis of vertebrobasilar insufficiency.
- Any other associated neurological signs of brain cell dysfunction support the diagnosis of vertebrobasilar insufficiency.(4)
- Dizziness and vertigo, common early symptoms of BAO, are nonspecific.(2)

➢ The distinction between central and peripheral vertigo is clinically important because delayed recognition of vertigo leads to missed opportunities for thrombolysis and / or thrombectomy and may increase the risk of death and disability.(7)

Based on the temporary profile of symptoms, basilar artery thrombosis can occur in at least three different ways:

- **Sudden onset** of severe motor and bulbar symptoms with reduced affect
- **Gradual or disordered course** of posterior circulation symptoms which eventually become progressively disabling motor and bulbar symptoms with reduced affect.(4)
- **Prodromal symptoms,** including double vision, dysarthria, vertigo and paresthesia. These symptoms precede the symptoms of single-phase basilar artery thrombosis by several days or even months.(4) The use of a stroke risk stratification tool, such as the ABCD2 score, which assigns points (0-7) based on 5 clinical factors: age 60 or older = 1; blood pressure ≥ 140/90 = 1; clinical features (unilateral weakness = 2, speech disorders without weakness = 1 and any other symptoms = 0); duration of symptoms (<10 minutes = 0, 10-59 minutes = 1 and ≥60 minutes = 2); and diabetes = 1, may also help stratify patients at higher risk.(1)

Clinical signs:

- Abnormal levels of affect and motor signs, such as hemiparesis or quadriparesis (usually asymmetrical), in more than 70% of patients.
- Bulbar and pseudobulbar signs present in 74% of patients.
- Pupil abnormalities, oculomotor signs and pseudobulbar manifestations (e.g. facial weakness, dysphonia, dysarthria, dysphagia) in more than 40% of patients.
- Neurological examination should be used to look for additional signs and symptoms of localization.(7)
- Oculomotor paralysis, oropharyngeal dysfunction, ataxia and limb weakness are the most common signs.
- Abnormal eye movements, asymmetrical pupils, dysmetria, respiratory problems and impaired consciousness.

- Alternative vertical or horizontal nystagmus in the primary gaze is of central etiology until proven otherwise.(1)

- **Oculomotor signs** are common and may be associated with the above-mentioned syndromes. They usually reflect the involvement of the centre of the vertical gaze in the middle brain and / or the abducens nucleus, the centre of the horizontal gaze located in the paramedian reticulate formation adjacent to the abducens nucleus and / or the longitudinal medial fasciculus. Injuries to these structures result in the following:
- Abducens ipsilateral paralysis
- Paralysis of the ipsilateral conjugate gaze
- Internuclear ophthalmoplegia
- A syndrome and a half caused by an injury simultaneously affecting the paramedian reticular formation and the medial longitudinal fasciculus, resulting in ipsilateral conjugate paralysis and internuclear ophthalmoplegia.(4)
- Eye shortening, which locates the lesion to the brainstem. It is characterized by a rapid downward movement of the eyeball, with a subsequent return to the primary position.
- Strabismus deviation.
- Other signs of pontine ischemia include limb tremor, ataxia (usually associated with mild hemiparesis), facial weakness, dysarthria, dysphagia, and hearing loss.(4)
- **One of the most devastating locations for a BAO is a mid-basilar occlusion with bilateral pontine ischemia.(1)** These patients may appear comatose, but they may be fully conscious and paralyzed only with limited vertical eye movements. This **"blockage syndrome"** has a high mortality rate of about 75% in the acute phase.
- Another BOA syndrome involves occlusion at the distal apex of the BA, where the superior cerebellar artery (SCA) and posterior cerebral artery (PCA) represent the final terminal branches. This **"upper basilary syndrome"** can cause ischemia of the midbrain, thalamus, lower temporal lobes, and occipital lobes.(6) The pupillary light reflex is often affected, so the pupils react to light slowly and incompletely or not at all.(6)
- **The described signs may be present in different combinations.**
- **Recognized syndromes commonly associated with basal artery occlusion are:**
- **Locked syndrome;** It is caused by the infarction of the brainstem base secondary to the occlusive disease of the proximal and middle segments of the basilar artery leading to quadriplegia. Because the tegument of the brainstem is not affected, the patient has an undamaged level of the affect, vertical eye movements and blinking being preserved. Coma associated with oculomotor abnormalities and quadriplegia indicate proximate and middle basilar occlusive disease with pontine ischemia.(4,5)
- **Top of basilar artery syndrome:** has diencephalic ischemia caused by occlusion of the rostral basilar artery, usually an embolus. Patients present with changes of the affect. They may suffer from visual symptoms such as hallucinations and / or blindness. Paralysis of nerve 3 and pupillary abnormalities are also common. Motor abnormalities include abnormal or postural movements.(2,4,5,6)
- Risk factors are those found in patients with stroke, **the most common risk factor is hypertension which occurs in 70% of cases. It is followed by diabetes, coronary artery disease, peripheral vascular disease, smoking and hyperlipidemia.(7)**

Laboratory:

- Complete blood count, electrolyte values, level of creatinine, prothrombin time, partially activated thromboplastin time and lipid profile.
- Young patients (<45 years of age) or patients who do not show atherosclerosis should be investigated for the presence of procoagulant conditions: Protein C, protein S and antithrombin III deficiencies, Lupus anticoagulant and anticardiolipin antibodies.
- • Creatine kinase levels, cardiac isoenzyme levels and troponym levels

Imaging studies:

- Cranial CT is the first imaging method
- CT without contrast is not particularly sensitive for the diagnosis of acute ischemic stroke, especially in the posterior form (50-70%), in patients with AB thrombosis (1)
- Contrast cranial CT
- The gold standard for vascular imaging is cerebral angiography
- MRI and magnetic resonance angiogram MRA are more sensitive than CT to identify ischemia and vascular occlusion
- MRA can identify vertebral, basilar occlusion with sensitivity up to 97% and a specificity of 98%
- MRA has limitations because it overestimates the degree of stenosis. Severe stenoses may resemble vascular occlusion.
- Transcranial Doppler (TCD) is useful for assessing cerebrovascular disease (but it is often inaccurate).

- In patients with basilar artery disease the reported sensitivity is 72% and the specificity is 94%
- TCD is useful to follow up any initial assessment that demonstrates the lesion.

- **A posterior stroke should not be ruled out with early negative MRI, especially with persistent neurological deficits.(1)**

- Echocardiography and rhythm monitoring are used to detect a source of heart embolism.(2)

Prognosis:

- Reclassification of the occluded vessel (2) is the most important prognostic factor in patients with BAO.

- Other factors associated with a good prognosis include younger age (<60 years), involvement of a single posterior circulation area and less severe presentation at installation (2) GCS> 10, low NiH scale score and absence of coma.

Treatment in the acute stage of BAO

- There is no consensus for best management in the acute phase of BAO.

- If BAO is suspected, the first step is to check that **the patients can protect their airways** (1), as acute symptoms of BAO may progress to decreased alertness, decreased respiratory impulse and coma.
- The clinician should consider **increasing blood pressure to increase cerebral perfusion** by stretching the head of the flat bed.(7)
- Contrast-free cranial CT and CT angiography should be the next step in diagnostic testing.
- If the CT scan does not show bleeding and the onset of symptoms is in 4,5 hours, the IV tissue plasminogen activator (t-PA) is the standard of care.(6)
- High morbidity and mortality in patients with non-recanalized BAO may lead to consideration of **endovascular treatment for BA revascularization by intra-arterial thrombolysis or thrombectomy.(6)**
- Treatment of basilar artery occlusion involves local intra-arterial thrombolysis (IAT), where possible, or intravenous thrombolysis (IVT).(3)

BAO management:

- **Control of risk factors**

- Diabetic patients are at increased risk for ischemic stroke. The mechanism behind this association is probably due to increased systemic inflammation and arterial stiffness, which increase the likelihood of atherosclerosis. Diabetes also increases the risk of high blood pressure, microvascular disease, lipid abnormalities, and hyperglycaemia, which may increase the likelihood of congestive heart disease and stroke, subsequently.(3)
- Risks associated with oral contraceptives.(3)
- Weight loss supplements, in particular, have been shown to be dangerous.(3) Along with estrogen-like compounds, weight loss supplements often have stimulants. Synephrine is still in use. These substances can cause atrial fibrillation which can increase the risk of stroke.
- Recent research has shown that prolonged QTc may predispose patients to the development of atrial fibrillation. QTc is associated with an increased risk of ischemic stroke. This suggests that QTc may be **an early marker of atherosclerotic disease** (3) to determine the etiology of stroke. The results were dependent on the etiology of the occlusion; patients with an embolic etiology have a 2.4-fold higher risk of reduced progression compared to patients with diffuse or localized atherosclerosis.
- An echocardiogram should be obtained to assess potential cardiac sources of embolism, such as valvular vegetation, intracardiac mass, ventricular thrombus, or permeable oval foramen.
- Patients should be monitored continuously for assessment of atrial fibrillation.(7)

- Predictors of poor outcomes included older age, higher NIHSS score, lack of recanalization, a history of atrial fibrillation, and symptomatic intracranial hemorrhage.
- A pc-ASPECTS> 8 is associated independently with better results.

• Successful recanalization seems to be the most important predictor of a good outcome.

- Heart attack thrombolysis score is a widely used method to describe angiographic findings after endovascular treatment of acute ischemic stroke.

- **There may be a high rate of suspicion for BAO in a patient with dizziness plus any other neurological deficits,** especially fluctuating consciousness, diplopia (or abnormal eye movement on examination), or unstable gait. Altered mood (2) is a common complaint upon presentation.
- **Ischemic course is an important differential diagnosis** that is considered even in younger patients.

- **Arterial dissection should be considered a potential etiology**, especially in patients with a known diagnosis of Turner syndrome. The connective tissue abnormality associated with Turner syndrome may put these patients at a much higher risk for spontaneous arterial dissection (2) compared to the general population.
- **Early diagnosis is crucial.**
- **Proper visualization of the posterior circulation often requires angiography or magnetic resonance imaging.**
- **Intravenous thrombolysis and local arterial thrombolysis are the most commonly used treatment approaches. Recanalization of the occluded vessel significantly improves morbidity and mortality.(2)**
- Particular attention should be paid to the relationship between symptoms and blood pressure.(7)
- Patients should be closely monitored for cerebellar edema that causes compression of the fourth ventricle and / or hernia, which usually peaks 3 to 5 days after a heart attack.(7)
- **There is no clear indication for anticoagulation treatment in acute stroke, with limited exceptions (e.g. heart thrombus) to be considered on a case-by-case basis.(7)**
- Although safe in the case of small infarcts, moderate and large infarcts undergo a hemorrhagic transformation in the first 1 to 2 weeks.(1)

Conclusions: BAO morbidity and mortality remain high despite progress in the prevention and treatment of ischemic stroke. There should be a high rate of suspicion for BAO in a patient with dizziness plus any other neurological deficits, especially fluctuating consciousness, diplopia (or abnormal eye movements on examination) or unstable gait. After airway stabilization, if necessary, the best treatment and optimal treatment time for BAO are not known, but reperfusion approaches involving t-PA IV and / or endovascular therapy are appropriate within 4,5 hours. Data for endovascular treatment over 4,5 hours are based more on a series of cases, but may be considered on an individual basis, especially in those patients with very severe deficits and imaging showing slight or minimal ischemic changes. Further research should focus on faster diagnosis, appropriate imaging and optimal treatment for these patients.

Conflicts of interests: The authors did not report any potential conflicts of interest in research, authorship, and / or publication of this article.

Funding: The authors have not received any financial support for the research, authorship and / or publication of this article.

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Prof. Marieta Dumitrache, et. al. "Basilar Artery Occlusion Syndrome." *IOSR Journal of Nursing and Health Science (IOSR-JNHS)*, 11(03), 2022, pp. 40-44.