Emergency Bulletin
Hong Kong College of Emergency Medicine

Acute basilar artery occlusion

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A 48-year-old cleaning worker was found sitting on the floor by colleagues at his workplace. He had vomited a few times and was found unresponsive afterwards.

On arrival, he was drowsy and his initial GCS was E4V1M5. His condition quickly deteriorated and became:

- GCS: E1V1M1, pupils non-reactive
- BP 190/100 mmHg, pulse 120 bpm
- SpO2 95% on 100% oxygen. Afebrile

Physical examination revealed four limbs hypertonia with bilateral up-going plantar response.

What is the initial management?

The patient was assessed in the resuscitation room. We should assess the airway, breathing and circulation status of the patient first.

In view of unconsciousness, he was intubated for airway protection using a video-assisted laryngoscope in a negative pressure room with staff in appropriate personal protective equipment. Mechanical ventilation was also provided.

Cardiac monitoring and end-tidal CO2 monitoring were attached. Two large bore IV accesses were set. Blood tests for complete blood count, liver and renal function, clotting profile, random glucose, POCT blood gas, H'stix and HaemoCue were checked. Further collateral history was taken from his colleagues.

What are the possible differential diagnoses?

Causes of acute onset of coma are shown in table 1.

Metabolic	Hyponatraemia Hypoglycaemia Hypothermia Uraemia Myxedema coma
Vascular	Ischaemic stroke Haemorrhagic stroke Subarachnoid haemorrhage
Intracranial	Brain tumor Ruptured intracranial aneurysm
Infective	Meningitis Encephalitis Brain abscess
Shock	Cardiogenic shock Anaphylactic shock Hypovolaemic shock Neurogenic shock
Toxicological	Salicylate poisoning Tricyclic antidepressants poisoning Severe paracetamol poisoning Alcohol intoxication

Table 1: Causes of acute onset of coma

Progress of patient

Bedside glucose was normal. ECG showed sinus rhythm without ischaemic changes. Post-intubation chest x-ray was clear and the endotracheal tube was in-situ.

Computed tomography(CT) of brain was performed (Figure 1). There was no intracranial haemorrhage, space occupying lesion or midline shift. There was no dense middle cerebral artery(MCA) sign, but there was hyperdense basilar artery sign.

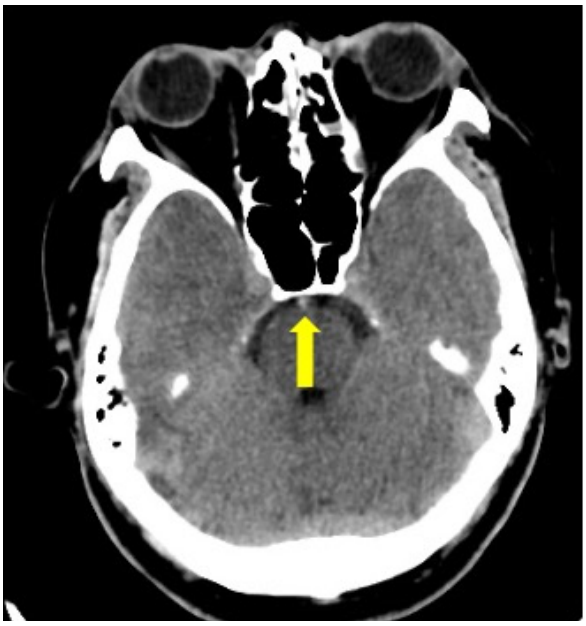

Figure 1: Computed tomography of brain showing hyperdense basilar artery sign (arrow)

What is hyperdense basilar artery sign?

Hyperdense basilar artery sign is the basilar artery equivalent of the dense MCA sign. It indicates the presence of a thrombus inside the artery lumen.

In patients presenting with high probability of posterior circulation stroke based on clinical symptoms, the presence of hyperdense basilar artery sign is a strong predictor of basilar artery thrombosis.[1]

The dense basilar artery sign is present in the non-contrast CT scan in 65% of the cases. It has a sensitivity (60.98-65.85%), specificity (70.73-90.24%) and accuracy (65.85-75.61%).[2,3]

Besides the dense artery signs, are there any other signs of early infarct in non-contrast CT (NCCT) scan of brain?

Early infarct signs in NCCT scan of brain can be subtle, which include:[4-8]

1. Loss of gray-white matter differentiation in the basal ganglia (e.g. obscuration of the lentiform nucleus
2. Loss of insular ribbon or obscuration of sylvian fissure
3. Cortical hypoattenuation and sulcal effacement

However, visualization of the posterior fossa structures in non-contrast CT scan is suboptimal due to obscuration by artifacts produced by skull base bony structures. Indeed, early ischaemic signs for posterior circulation stroke on CT are not well-established.[9]

Contrast injection greatly enhance the sensitivity and specificity of CT. On cross-sectional images and angiographic reconstruction, basilar artery occlusion appears as a filling defect within the vessel and localize the site of occlusion.

What is the diagnosis for the patient?

The most likely diagnosis is acute ischaemic stroke due to basilar artery occlusion (BAO).

Basilar artery and the vertebral artery are part of the posterior circulation blood supply. 20% of ischaemic events in the brain involve posterior circulation and basilar artery occlusion accounts for about 1 % of all stroke.[10,11] It is a neurological emergency that can cause brainstem or thalamic ischemia.

The most frequent causes of posterior circulation large artery ischemia are atherosclerosis, embolism and dissection.[1] (Table 2)

Causes	Percentage
Atherosclerosis	26-36%
Emboli	30-35%
Other causes including dissection of vertebral artery	6-8%
undetermined	22-35%

Table 2: Causes of basilar artery occlusion

The commonest location of atherosclerotic occlusive disease within the posterior circulation is the proximal portion of the vertebral artery in the neck.[12-14] (Figure 2)

Dissection usually involves the extracranial vertebral artery just before it enters the foramen transversarium at C5 or C6, or in the very distal part of the artery in the neck before it penetrates the dura mater to enter the cranial cavity.[15]

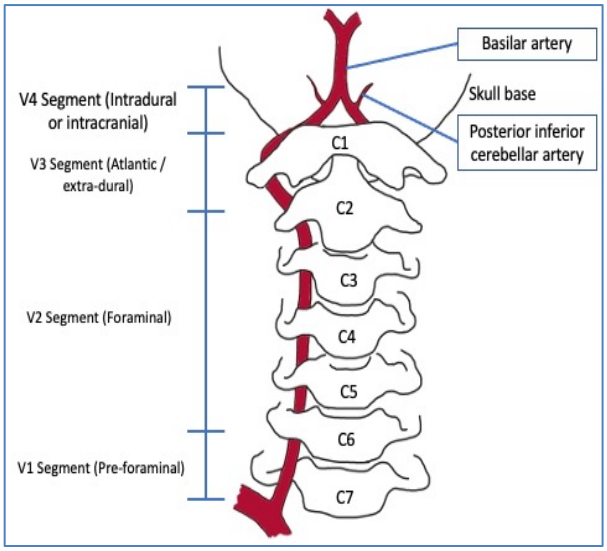

Figure 2: Different segments of vertebral artery

Which territory does the basilar artery supply?

The basilar artery is formed by the union of the vertebral arteries and terminates by dividing into two posterior cerebral arteries. In general, the basilar artery is divided into three segments. (Figure 3)

- Proximal segment: from the vertebral artery junction to the anterior inferior cerebellar artery
- Middle segment: from anterior inferior cerebellar artery to the superior cerebellar artery
- Distal segment: basilar artery distal to the orifices of the superior cerebellar artery

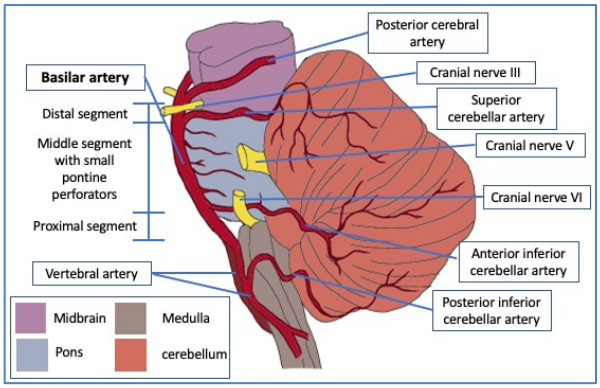

Figure 3: Anatomy of the basilar artery

Numerous small pontine branches arise from the front and sides of the middle basilar artery along its course and supply the pons.

The anterior inferior cerebellar artery (AICA) originated from the proximal middle segment. It supplies the inferior cerebellum including the inferior and middle cerebellar peduncles

The superior cerebellar artery (SCA) arises near the distal segment of the basilar artery. It supplies the superior cerebellum, pons, pineal body, superior medullary velum and tela choroidea of the third ventricle.

What are the clinical presentations of acute stroke due to basilar artery occlusion?

In the BASICS registry, 19% of patients had prodromal transient ischemic attacks and 19% had prodromal minor stroke.[16] The most common prodromal symptoms were vertigo, nausea, and headache.[17]

The clinical presentations correlated with the site and extent of the basilar artery occlusion and the type of collateral blood flow.[18] Around 30-60% of patients with BAO present with coma.[17]

There are a number of posterior circulation stroke syndromes (Table 3), but these classical symptoms or syndrome are often incomplete or absent.[19]

The posterior circulation is rich in collaterals and therefore, clinical manifestations of disturbed basilar artery flow are highly variable. It is an easily missed diagnosis and a high index of suspicious is required.[20,21]

How is acute basilar artery occlusion managed?

Regarding acute management, options of reperfusion therapies include intravenous thrombolysis, intra-arterial thrombolysis, mechanical thrombectomy and a combination of these approaches.

Intravenous thrombolysis (IVT)

Intravenous alteplase (recombinant tissue plasminogen activator – rtPA) is the mainstay of treatment for acute ischaemic stroke.

The Third International Stroke Trial showed that treatment with IV-rtPA within 6 hours of symptoms onset was associated with improvements in functional outcome and quality of life without effect on overall survival rate.[22]

Therefore, it is suggested that all eligible patients with acute ischemic stroke should be treated with intravenous thrombolysis, including those with stroke in the posterior circulation.

Intra-arterial thrombolysis (IAT)

The use of intra-arterial thrombolytics in posterior circulation stroke has been previously studied.[23-26] The Basilar Artery International Cooperation Study (BASICS) prospectively observed and compared outcomes of patients with basilar artery occlusion and found no conclusive evidence of superiority for IA-thrombolysis over IV-thrombolysis and prompted the need for a randomized control trial.[16]

The American Heart Association / American Stroke Association recommends IA-rtPA in selected patients with middle cerebral artery occlusion within 6 hours of symptom onset but no recommendation is made for IA thrombolysis in posterior circulation stroke including basilar artery occlusion.[27]

Mechanical thrombectomy (MT)

Mechanical thrombectomy is proven effective only for selected patients with acute ischemic stroke caused by a proximal intracranial arterial occlusion in the anterior circulation. Trials that establish the benefit of it largely excluded patients with posterior circulation infarcts.[28-32]

Although the benefit is uncertain, mechanical thrombectomy may be a reasonable treatment option for patients with acute ischaemic stroke caused by occlusion of the basilar artery, vertebral arteries or posterior cerebral arteries when performed at centers with appropriate expertise. [33,34]

For stroke rehabilitation, a multidisciplinary approach is adopted. Speech therapists will assess the quality of speech and swallowing process for any risk of choking. Physiotherapists will help improve muscle strength and train patients to walk. Occupational therapists will assess and train patient to cope with activities of daily living. Clinical psychologists and medical social workers will address psychosocial aspects including adjustment reaction and loss of income during hospitalization and rehabilitation.[35]

Progress of patient

Hyperacute stroke team was activated for thrombolysis. After a conjoint discussion with on-call neurologist and interventional radiologist, computed tomography of cerebral angiogram was performed and confirmed the

location of occlusion at distal basilar artery. Initially, the National Institute of Health Stroke Scale (NIHSS) score of the patient was 34/42.

Intravenous tissue plasminogen activator was given followed by intra-arterial thrombectomy by interventional radiologist. He was admitted to the intensive care unit for monitoring and then transferred to the acute stroke unit later.

What is the prognosis of basilar artery occlusion?

Generally, basilar artery occlusion has a poor outcome. For patients receiving conventional treatment of symptomatic basilar artery occlusion, 80% of patients are associated with poor outcome.[36]

For patients undergoing endovascular stroke treatment, despite high recanalization rates, poor clinical outcomes are still common.[37] An initial low NIHSS score and a high DWI posterior circulation Acute Stroke Prognosis Early CT Score before endovascular treatment were reported to be independent predictors of good outcome.[38]

Progress of patient

There was successful recanalisation with complete antegrade reperfusion. The patient regained consciousness and was extubated on day 2 after admission. NIHSS score becomes 0/42.

After further stroke rehabilitation, he regained full motor function and limb power, functional swallowing and speech. He was ambulatory and able to walk unaided at discharge.

Table 3 Posterior circulation stroke syndrome

Vascular territory	Anatomical location	Stroke syndrome	Clinical findings
Vertebral artery	Medulla and cervical spinal cord	Medial medullary or Dejerine syndrome	Contralateral arm and leg weakness, hemibody loss of tactile, vibration, position sense, ipsilateral tongue paralysis
Posterior inferior cerebellar artery (PICA)	Inferior posterior cerebellar hemisphere, inferior vermis, lateral medulla	Lateral medullary or Wallenberg syndrome	Vertigo, nausea, vomiting, ipsilateral facial numbness and dysmetria, Horner's syndrome, dysphagia and ataxia dysphonia, contralateral hemisensory loss below the face
Basilar artery			
Proximal basilar	Bilateral lower pons	Locked-in syndrome	Quadriplegia, horizontal gaze paralysis, bifacial paralysis, may preserve vertical eye movements
Mid-basilar	Lateral pontine perforators	Lateral mid-pontine syndrome	Ipsilateral loss of facial sensation and motor function of the trigeminal nerve, ipsilateral dysmetria
	Medial pontine perforators	Medial mid-pontine syndrome (Foville syndrome)?	Ipsilateral dysmetria, contralateral weakness, gaze deviation
Top of the basilar	Midbrain, thalamus and mesial temporal lobes and occipital lobes	Top of the basilar syndrome	Somnolence,peduncular hallucinosis, converency nystagmus, skew deviation, vertical gaze paralysis, oscillartory eye movements, Colliers sign (retraction and elevation of eyelids)
Anterior inferior cerebellar artery (AICA)	Ipsilateral labyrinth, lateral pontine	Lateral pontine syndrome	Ipsilateral dysmetria, hearing loss, Horner's syndrome, choreiform

	tegmentum and brachium pontis, ICP		dyskinesia, contralateral decrease temperature and pain sensation
Superior cerebellar artery (SCA)	Dorsolateral upper brainstem and cerebellum and superior cerebellar peduncle	Superior cerebellar artery syndrome	Ipsilateral limb ataxia, vertigo, nystagmus, dysarthria and gait ataxia
Posterior cerebral artery (PCA)			
Unilateral PCA	Occipital lobe	Contralateral homonymous hemianopsia	Homonymous hemianopsia with macular sparing
	Dominant occipital lobe plus splenuym of corpus callosum	Alexia without agraphia	Homonymous hemianopsia and alexia without agraphia
	Ventral occipital cortex; infracalcarine	Achromatopsia	Loss of color differentiation contralateral to the side of the lesion, can be associated with a quadrantanopsia
	Optic radiation or supracalcarine	Inferior quadrantanopsia	Inferior quadrantanopsia
	Myers loop (temporal lobe) or infracalcarine	Superior quadrantanopsia	Superior quadranopsia
Bilateral PCA	Both occipital lobe	Cortical blindness	Bilateral cortical blindness
		Anton's syndrome	Bilateral cortical blindness with denial of blindness and confabulations or visual hallucination

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