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Vertebrobasilar occlusion and vertebral artery syndrome

Synonyms: vertebrobasilar stroke, vertebrobasilar insufficiency

Occlusion or impairment of the vertebrobasilar blood supply affects the medulla, cerebellum, pons, midbrain, thalamus and occipital cortex. This results in a number of clinical syndromes and is caused primarily by atherosclerosis.

Anatomy – brief review

The vertebral arteries branch off the subclavian arteries, passing cephalad through the costotransverse foramina of the sixth to second cervical vertebrae. They enter the skull through the foramen magnum merging at the pontomedullary junction to form the basilar artery which divides into two posterior cerebral arteries at the upper pons. At the base of the brain the carotid and basilar systems join to form the circle of Willis. This arrangement of collateral circulation may allow adequate brain perfusion even with occlusion of a main vessel.

Important points to consider when assessing clinically are:

- Superior cerebellar arteries from the basilar artery supply the lateral aspect of pons and midbrain together with the superior surface of cerebellum.
- The cerebellum is supplied by branches from the basilar artery (long circumferential, posterior cerebral, anterior inferior cerebellar and superior cerebellar arteries).
- The medulla is supplied by the posterior inferior cerebellar artery and by direct smaller branches from the vertebral arteries.

- The pons is supplied by small and large branches from the basilar artery.
- The midbrain and thalamus are supplied by penetrating arteries from the posterior cerebral arteries.
- The occipital cortex is perfused by the posterior cerebral artery.

Pathophysiology

- **Atherosclerosis:** the most common vascular disease affecting the vertebrobasilar system^[1]:
 - This affects large vessels, causing narrowing and occlusion.
 - It needs to produce stenosis at the origins of both vertebral arteries to cause vertebrobasilar ischaemia.
 - Even with vertebral artery occlusion, collaterals (circle of Willis) may prevent ischaemia.
 - Ischaemia of the hind brain is likely to develop with the association of carotid artery disease (often at the bifurcation of the carotid artery), vertebral artery stenosis and intracerebral disease.
 - Note that the circle of Willis is only intact in 50% of patients.
- **Lipohyalinosis:** affects small vessels, eventually causing occlusion. This frequently occurs in association with hypertension. Lipohyalinosis also causes weakening of the vessel walls and rupture of the vessels, leading to focal haemorrhage. Almost all intracerebral haemorrhages come from such rupture of small penetrating vessels.
- **Embolic occlusion of the vertebrobasilar system:** is uncommon and emboli are typically from the aortic arch, subclavian artery and vertebral arteries. Emboli usually occlude the basilar artery.
- **Vertebrobasilar artery dissection.**

Epidemiology

A relatively uncommon form of stroke. However:

- [Stroke](#) is the fourth^[2] biggest cause of death in the UK.
- 25% of lesions causing stroke occur in the vertebrobasilar circulation^[1].

Risk factors associated with stroke

See also the separate [Cerebrovascular Events](#) article.

- Age.
- Hypertension.
- Alcoholism and problem drinking.
- Coronary and other heart disease.
- Diabetes mellitus.
- Drug misuse^[3] ^[4].
- Family history of stroke.
- Giant cell arteritis^[5].
- Obesity and physical inactivity^[6].
- Previous stroke.
- Race.
- Smoking^[1] ^[7].

Presentation

Death or major disability is the result of occlusion of large vessels in the vertebrobasilar system. However, many lesions arise in smaller vessels with a wide variety of focal neurological deficits. Stroke scoring systems to evaluate patients have been developed but are of limited use for vertebrobasilar stroke^[8].

History

Onset and duration of symptoms may vary with aetiology.

- Basilar artery thrombosis may be preceded by transient ischaemic attacks for days or weeks prior to occlusion (seen in half of patients who experience a vertebrobasilar stroke).
- Embolic events cause sudden and dramatic symptoms without prodrome.
- It may (rarely) be brought on by turning the head (temporarily occluding one vertebral artery, with insufficient collaterals due to advancing atherosclerosis).

Symptoms reported with vertebrobasilar strokes include:

- Vertigo - common - and this may be the only symptom.
- Nausea and vomiting.
- Disturbance of consciousness.
- Headache.
- Visual disturbance (oculomotor signs such as nystagmus, diplopia and pupillary changes).
- Visual field defects.
- Speech disturbance (for example, dysarthria and dysphonia).
- Sensory changes in the face and scalp.
- Ataxia.
- Contralateral motor weakness (may cause a 'drop attack').
- Sensory disturbance affecting pain and temperature.
- Incontinence.

There may be a history of associated risk factors.

Examination

Diagnosis can be made from careful clinical examination but the complexity of signs requires careful and detailed neurological examination. Common findings:

- Abnormal level of consciousness.

- Hemiparesis or quadriparesis (usually asymmetrical).
- Bulbar manifestations (facial weakness, dysphagia, dysarthria, dysphonia).
- Also common are pupillary and oculomotor abnormalities.

The physical findings can be very varied and complex according to the precise location within the brain that is affected:

- Abducens nucleus, horizontal gaze centre (located in the pontine paramedian reticular formation) and medial longitudinal fasciculus lesions cause oculomotor signs. These can result in:
 - Ipsilateral lateral gaze palsy.
 - Conjugate gaze palsy.
 - Ocular bobbing.
- Midbrain syndromes:
 - Vertical gaze palsy.
 - Third cranial nerve palsy.
- Pontine syndromes:
 - Tremor, ataxia and mild hemiparesis.
 - Horizontal gaze palsy.
 - Cranial nerve VI and VII palsies.

- Medullary syndromes:
 - Loss of facial pain and temperature sensation (ipsilateral).
 - Horner's syndrome.
 - Ataxia (ipsilateral).
 - Tongue, soft palate, vocal cord, sternocleidomastoid paralysis (ipsilateral).
 - Contralateral loss of pain and temperature sensation elsewhere.
- Posterior cerebral artery syndromes:
 - Contralateral hemianopia with macular sparing.

Clinical syndromes and scenarios

Various different combinations of signs and symptoms have been described.

Clinical syndromes

A detailed account is too complex here but examples of syndromes described include the following:

- Lateral medullary or Wallenberg's syndrome:
 - Usually from occlusion of the vertebral artery.
 - Occasionally from occlusion of the posterior inferior cerebellar artery.
 - Involvement of the vestibular system causes nausea, vomiting and vertigo.
 - Ipsilateral features:
 - Ataxia from cerebellar involvement.
 - Horner's syndrome from damage to descending sympathetic fibres.
 - Reduced corneal reflex from descending spinal tract damage.
 - Nystagmus.
 - Hypacusis.
 - Dysarthria.
 - Dysphagia.
 - Paralysis of palate, pharynx and vocal cord.
 - Loss of taste in the posterior third of the tongue.
 - Contralateral findings:
 - Loss of pain and temperature sensation in the trunk and limbs (anterior spinothalamic tract).
 - Tachycardia and dyspnoea (cranial nerve X).
 - Palatal myoclonus (involuntary jerking of the soft palate, pharyngeal muscles and diaphragm).
- Medial medullary or Dejerine's syndrome:
 - An uncommon lesion caused by vertebral artery or anterior spinal artery occlusion.
 - Causes ipsilateral tongue paresis with deviation to the side of the lesion.
 - Contralateral hemiplegia with facial sparing.
 - Ipsilateral loss of vibration and proprioception.
- Cerebellar infarction:
 - Causes inco-ordination, clumsiness, intention tremor, ataxia, dysarthria, scanning speech.
 - Early diagnosis is important, as swelling may cause brain stem compression.

- Locked-in syndrome:
 - Caused by infarction of the upper ventral pons.
 - Usually dramatic and sudden, causing quadriplegia with preserved consciousness.
- Internuclear ophthalmoplegia:
 - Horizontal gaze palsy.
 - In younger patients it may be caused by multiple sclerosis.
- One-and-a-half syndrome:
 - Ipsilateral conjugate gaze palsy and internuclear ophthalmoplegia.
 - Causes inability to move the ipsilateral eye and only abduct the contralateral eye with nystagmus.
- Millard-Gubler syndrome:
 - Ventral pontine syndrome.
 - Diplopia with facial paresis and contralateral hemiparesis.
- Top-of-the-basilar syndrome:
 - Sudden confusion and amnesia.
 - Visual symptoms.
 - Usually caused by embolism.
- Raymond-Céstan syndrome:
 - Upper dorsal pons affected.
 - Ataxia and tremor ipsilaterally.
 - Weakness of mastication.
 - Contralateral loss of sensory modalities.
 - May involve facial weakness and hemiparesis.
- Foville's syndrome:
 - Lower dorsal pontine area affected.
 - Ipsilateral paresis of the whole face.
 - Loss of ipsilateral horizontal gaze.
 - Contralateral hemiplegia with facial sparing.
- Weber's syndrome:
 - Ventral midbrain affected.
 - Ipsilateral mydriasis, cranial nerve III palsy and ptosis.
 - Contralateral hemiplegia.
- Benedikt's syndrome:
 - Dorsal midbrain affected.
 - Ipsilateral oculomotor effects as in Weber's syndrome.
 - Contralateral tremor, ataxia or chorea.

- Posterior cerebral artery occlusion:
 - Most often causes occipital lobe infarction with hemianopia and macular sparing.
 - Variable effects from thalamic syndrome to varieties of cortical visual disturbance.

Differential diagnosis

Symptoms associated with vertebral artery occlusive disease include dizziness, vertigo, diplopia, perioral numbness, blurred vision, tinnitus, ataxia, bilateral sensory deficits, and syncope, all of which can be caused by other disease entities, including cardiac arrhythmias, orthostatic hypotension, and vestibular disorders^[9]. Therefore, other diagnoses to consider include:

- Secondary brain tumours.
- Primary brain tumours – specifically, cerebellopontine angle tumours.
- Lesions in the supratentorial hemispheric region, causing brain stem compression and herniation.
- [Subarachnoid haemorrhage](#).
- [Meningitis](#).
- Basilar [migraine](#).
- [Multiple sclerosis](#).
- [Guillain-Barré syndrome](#)^[10].

[Giant cell arteritis](#) has been identified as a cause of vertebrobasilar infarcts^[5].

Investigations

- Basic blood tests, including FBC, ESR, blood chemistry, clotting and lipid profile.

- Screen for hypercoagulable states if aged under 45 – for example:
 - Lupus anticoagulant and anticardiolipin antibodies.
 - Antithrombin III deficiency.
- Imaging studies:
 - Immediate brain imaging with non-enhanced CT is recommended for all people with suspected acute stroke if they have indications for thrombectomy (which includes those with strokes involving the basilar as well as the posterior cerebral artery, as long as there is potential to salvage brain tissue) as well as those with a known bleeding tendency, depressed level of consciousness, unexplained progressive or fluctuating symptoms or those on anticoagulant therapy^[11].
 - In those with suspected stroke who do not have indications for immediate brain imaging, non-enhanced CT scanning should occur within 24 hours^[11].
 - MRI scanning is better than CT scanning, particularly for ischaemia, demyelination, tumours and diseases of blood vessels.
 - Diffusion-weighted MRI sequences may be used to show infarct core volume in order to assess suitability for thrombectomy^[11].
 - MR angiography is usually performed in conjunction with MRI scanning.
 - New MRI techniques allow even better definition of pathology^[12].
- ECG:
 - Mandatory for all stroke patients.
 - 20% of stroke patients have an arrhythmia and about 2% have had previous myocardial ischaemia (indicating the presence of cardiovascular disease).

- Echocardiography:
 - Identifies valvular defects, vegetations and other sources of emboli, particularly in young patients with basilar artery occlusion.
- Cerebral angiography:
 - Used less because of non-invasive imaging.
 - Useful when thrombolysis or recanalisation is contemplated.

Management

Where should patients be treated?

- Where possible, patients should be treated in specialist stroke units.
- Specialist neurological intensive care may be required for:
 - Patients who are candidates for thrombolysis or other interventional treatments, including thrombectomy.
 - Fluctuating neurological symptoms.
 - Impaired levels of consciousness.
 - Unstable haemodynamic indices.
 - Cardiorespiratory problems.

What treatments can be given?

- General measures:
 - Prevent aspiration pneumonitis.
 - Early attention to continence programmes.
 - Careful monitoring of catheters to avoid infection.
 - Control of body temperature.
 - Control of blood glucose.

- Treatment to maintain cerebral blood flow:
 - This in practice means managing blood pressure.
 - Over-enthusiastic treatment of hypertension should be avoided.
 - Treat hypertension, if hypertensive emergency exists, with one or more of these concomitant medical issues:
 - Hypertensive encephalopathy.
 - Hypertensive nephropathy.
 - Hypertensive cardiac failure/myocardial infarction.
 - Aortic dissection.
 - Pre-eclampsia/eclampsia^[11].
 - In patients considered for intravenous thrombolysis, blood pressure reduction to 185/110 mm Hg or lower should be considered^[11].
 - Treat hypotension:
 - Intravenous fluids to maintain intravascular volume with isotonic fluids.
 - Inotropes or vasopressors may be required.
 - Occasionally, there is a need to use a pulmonary artery catheter to monitor central venous pressure and pulmonary capillary wedge pressure with congestive cardiac failure and pulmonary oedema.

- Treatments for respiratory complications:
 - Assess respiratory drive, gag reflex, cough reflex (to expel secretions).
 - Consider endotracheal intubation (Glasgow Coma Scale less than 8).
 - Sedation and muscle relaxation may be needed if agitated or resisting mechanical ventilation but these will have to be reversed before a full neurological assessment can be carried out.
- Thrombolysis (see also the separate [Thrombolytic Treatment of Acute Ischaemic Stroke](#) article):
 - Local intra-arterial thrombolysis results in better recanalisation results than intravenous thrombolysis^[13]. However, there is a lack of data on improvements in long-term outcomes for both of these treatments. Given the non-invasive nature of intravenous thrombolysis, it is preferred by some^[13]. The National Institute for Health and Care Excellence (NICE) recommends intravenous thrombolysis where it is being considered^[11].
- Thrombectomy:
 - Thrombectomy in combination with intravenous thrombolysis where indicated/not contra-indicated should be considered as soon as possible for people who were previously well, who have confirmed occlusion of the basilar artery on CT or magnetic resonance angiography (MRA), as long as there is potential to salvage brain tissue^[11].
 - Imaging to inform salvageable brain tissue is key because if a person with basilar artery occlusion has irreversible bilateral damage to the pons, they may be left with locked-in-syndrome with complete face and body paralysis but clear consciousness, even if the basilar artery is opened^[11].

- Antiplatelet agents:
 - Antiplatelet agents should be used as in other strokes - eg, aspirin.
 - NICE also advises the use of antiplatelet agents in acute stroke resulting from arterial dissection^[11].
- Anticoagulation:
 - Intravenous heparin has been used in acute stroke. This shows no benefit except for some improved outcome in large vessel disease after seven days.
- Angioplasty:
 - The place in management is yet to be decided by research evidence. Stenting of vertebrobasilar arteries needs larger comparative trials to further evaluate the technique^[14].
- Surgery to form an anastomosis between the vertebral artery and external carotid^[15].

What forms of rehabilitation may be required?

- Nursing factors:
 - Maintain skin, nutrition and patient safety.
 - Communication with other therapists.
 - Communication with relatives, and education about stroke and its effects.
- Physiotherapy:
 - Variable needs according to severity.
 - Assessment and development of a programme of care.
 - Mobilisation and strengthening.
 - Chest care.

- Occupational therapy to help with bathing, dressing and grooming. Education of relatives.
- Speech therapy. This may involve speech and language skills but also safety skills, assessment of swallowing and education of carers and family.

What referrals may be required?

- The therapies above.
- Referral to social services.
- Neuropsychology.
- Neuropsychiatry.

Complications

The neurological deficit can be further complicated by:

- Pneumonia (particularly [aspiration pneumonia](#)).
- [Deep vein thrombosis](#) and [pulmonary embolism](#).
- [Myocardial infarction](#).

Prognosis

This will depend on the extent of disease; however:

- Acute basilar artery occlusion has a very high mortality rate.
- As few as 2-5% of people with basilar artery occlusion make a full neurological recovery in the absence of interventions to achieve recanalisation or reperfusion ^[11].
- Annual stroke rates for patients with symptomatic intracranial vertebral and basilar artery stenosis are 8% and 11% respectively ^[9].

Certain syndromes may have a good prognosis in terms of long-term functional outcome but still carry a risk of death in the acute phase from, for example, aspiration pneumonia in the lateral medullary syndrome. One study suggested that basilar artery diameter >4.3 mm could be a marker for high risk of fatal stroke ^[16].

Prevention

This depends on the cause; however, prevention strategies include:

- Warfarin for atrial fibrillation. Better adherence to guidelines would prevent stroke^[17].
- Treatment of hypertension.
- Treatment of hyperlipidaemias.
- Management of risk factors for stroke.

See also the separate [Stroke Prevention](#) article.

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