Neurological assessment and diagnosis of posterior circulation ischaemic stroke as a result of vertebral artery dissection in trauma patients guideline

Document Version Control

<table>
<thead>
<tr>
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Scope

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<th>Site</th>
<th>Department, Division or Operational Area</th>
<th>Applicable to:</th>
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<tr>
<td>Royal Perth Hospital</td>
<td>Trauma Service</td>
<td>Medical, Nursing, Allied Health</td>
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Definitions

**Posterior circulation ischaemic stroke (PCA Stroke)**

PCA stroke is a clinical syndrome associated with ischaemia related to stenosis, in situ thrombosis, or embolic occlusion of the posterior circulation arteries—the vertebral arteries in the neck, the intracranial vertebral, basilar, and posterior cerebral arteries, and their branches. In trauma patients, PCA stroke is more commonly caused by vertebral artery dissection, which may be traumatic or iatrogenic (e.g. spinal surgery, Internal Jugular CVC insertion).

General Information

Posterior cerebral artery (PCA) stroke is less common than stroke involving the anterior circulation. An understanding of PCA stroke phenomenology and mechanisms requires knowledge of neurovascular anatomy and of the structure-function relationships of this region of the brain. Identifying mechanisms of stroke is essential so that appropriate preventive therapies may be instituted.
The most common causes of posterior circulation stroke are occlusion or embolism due to large artery vertebrobasilar, proximal aorta and arch) atherosclerosis or dissection, and embolism from the heart. Dissection of the extracranial vertebral artery is also an important cause of stroke, especially in young patients.²
Diagnosis and management

The diagnosis of posterior circulation ischaemic stroke is based on rapidly developing clinical signs of focal (or occasionally global) disturbance of cerebral function, with no apparent cause other than that of vascular origin.2

An index of suspicion for posterior circulation stroke should be maintained in patients presenting with acute neurological symptoms. In the initial assessment phase it is important to establish the onset and tempo of symptoms and establish whether the patient has experienced typical or characteristic posterior circulation stroke symptoms such as acute diplopia, visual field disturbance, or swallowing difficulties.2

Neurological assessment

- If a patient develops any decrease in level of consciousness, the priority is to promptly identify and treat alterations in ABCGS (Airway, Breathing, Circulation, Glucose or Seizures) that may be causing the deterioration.
- If the neurological change persists despite normalization of the ABCGS, a detailed neurological assessment should be performed. The examination should attempt to determine if focal findings are present (suggesting a structural abnormality, such as stroke) or absent (suggesting generalized neurological depression, as seen with sedation or septic encephalopathy).
- Change is the most important finding in any neurological assessment and should be reported promptly to ensure timely medical intervention (if warranted). To ensure that neurological findings are communicated effectively at change of shift, clinicians should perform a neurological examination together with the oncoming shift.3

Detailed neurological assessments should be clearly documented in the patient's medical record.

Common symptoms seen in posterior circulation ischaemia2

<table>
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<tr>
<th>Motor deficits</th>
<th>weakness, clumsiness, or paralysis of any combination of the arms and legs, up to quadriplegia, sometimes changing from one side to another in different attacks</th>
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<td>“Crossed” syndromes</td>
<td>ipsilateral cranial nerve dysfunction and contralateral long motor or sensory tract dysfunction</td>
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<tr>
<td>Sensory deficits</td>
<td>numbness, including loss of sensation or paraesthesia in any combination of extremities, sometimes including all four limbs or both sides of the face or mouth</td>
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<tr>
<td>Homonymous hemianopia</td>
<td>a visual defect affecting the two right or the two left halves of the visual fields of both eyes</td>
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<tr>
<td>Ataxia</td>
<td>imbalance, unsteadiness or disequilibrium</td>
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Vertigo | with or without nausea and vomiting
---|---
Diplopia | as a result of ophthalmoplegia
Dysphagia or dysarthria | 
Isolated reduced LOC | as a result of bilateral thalamic or brainstem ischaemia (especially from rostral basilar artery occlusion)

**Clinical features according to anatomical location and vascular territory affected**

| Lateral medulla | intracranial vertebral artery infarct, also known as Wallenberg syndrome
| | nystagmus, vertigo, ipsilateral Horner’s syndrome, ipsilateral facial sensory loss, dysarthria, hoarseness and dysphagia
| | contralateral hemisensory loss in the trunk and limb – pain and temperature
| Medial medulla | ipsilateral tongue weakness and later hemiatrophy of the tongue
| | contralateral hemiparesis of the arm and leg
| | hemisensory loss – touch and proprioception
| Pons | hemiparesis or hemisensory loss, ataxic hemiparesis, dysarthria, horizontal gaze palsy
| | complete infarction causes “locked in syndrome” with quadriplegia, loss of speech, but preserved awareness and cognition and sometimes preserved eye movements
| Top of the basilar (distal basilar occlusion) | somnolence, confusion (from thalamic infarction)
| | bilateral loss of vision, unawareness or denial of blindness (from bilateral occipital infarction)
| Posterior inferior cerebellar artery | truncal ataxia, vertigo (limb ataxia may occur, especially if the inferior cerebellar peduncle is affected)
| Posterior cerebral artery | contralateral homonymous hemianopia (from occipital infarction)
| | hemisensory loss – all modalities (from thalamic infarction)
| | hemi-body pain (usually with burning quality) down one side of the body (face, arm and leg) as a result of thalamic infarction
| | if bilateral, may have poor visual-motor coordination, inability to understand visual objects

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How is posterior circulation ischaemic stroke diagnosed?²

- Posterior circulation stroke is diagnosed on the basis of history and clinical examination, assisted by imaging.
- History suggestive of potential or known injury to the vertebral arteries (as in Denver Criteria in blunt trauma, or penetrating injury to the neck and upper thorax. Consider iatrogenic causes such as post cervical spine surgery, or Internal Jugular Vein CVC insertion.
- NB. Insertion site for CVC insertion should be determined according to clinical grounds, with a preference for subclavian vein in most patients. IJV CVC insertion poses a higher chance of arterial cannulation due to its anatomical proximity to the carotid artery; left internal jugular catheters have poor flow rates and high risk of thrombosis, and should therefore be avoided. To reduce the risk of mechanical complications, use of ultrasound is recommended in all IJV CVC insertions.⁴
- Assessment for homonymous visual field deficits; eye movement; and looking for Horner’s syndrome (ptosis, small pupil (miosis), and anhydrosis on the same side), bilateral small or fixed pupils, and ataxia may aid early diagnosis.

Imaging

- Urgent CT or magnetic resonance imaging (MRI) to exclude haemorrhage.
- Brain and vessel CT Angiography to identify basilar artery occlusion, in order to initiate thrombolysis therapy MRI with diffusion weighted imaging for suspected posterior circulation stroke.
- MRI is far more sensitive than CT in the diagnosis of acute ischaemic stroke for all vascular territories (80-95% sensitivity in the first 24 hours versus 16% sensitivity with CT).
- Sensitivity may be lower in the posterior circulation with a false negative rate of 19% reported in the literature for early MRI.²

Consider referring patients at risk of deterioration in the acute phase to Neurointerventional Radiology (NIIS WA), Neurology, Neurosurgery and Intensive Care

Related Standards

- NSQHS 9.4.1  Mechanisms are in place to escalate care and call for emergency assistance
- RPH NPS Assessment of Full Neurological Observations
- RPH CPS Central Line Insertion and Management
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References


