



## PRACTICE

### EASILY MISSED?

## Posterior circulation ischaemic stroke

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#### What you need to know

- Posterior circulation stroke causes a wide range of non-specific presenting symptoms
- More than a third of posterior circulation strokes are initially misdiagnosed
- Explore the possibility of posterior circulation stroke in patients with new vertigo or disequilibrium, and those with new headache or changed migraine
- A negative HINTS examination in a patient with isolated vertigo can help rule out posterior circulation ischaemia

A 63 year old man with a history of migraine with visual aura, hypertension, and anxiety presented to the local emergency department with a five day history of headache (see “A patient’s perspective”). This headache started similarly to previous migrainous episodes, but became more severe than usual and was accompanied by intermittent double vision and disturbed balance, speech, and swallowing. The patient was treated in the emergency department with intravenous fluids and analgesia and discharged with a diagnosis of migraine. The following day, his symptoms worsened; clinical examination revealed vertical diplopia, gaze-evoked jerk nystagmus, right sided past-pointing, and an ataxic gait. Computed tomography (CT) of the head and CT angiography demonstrated an acute right superior cerebellar artery territory infarct and thrombus in the V3 and V4 (distal) segments of the right vertebral artery; subsequent brain magnetic resonance imaging (MRI) revealed other posterior circulation infarcts (fig 1).

### What is posterior circulation ischaemia?

The posterior circulation comprises both vertebral arteries, the basilar artery, and the intracranial vessels that they give rise to.<sup>1</sup> Together, these arteries supply the brainstem, cerebellum, medial

and postero-lateral thalamus, occipital lobes, and sometimes parts of the medial temporal and parietal lobes<sup>1</sup> (fig 2).

Posterior circulation ischaemic stroke causes about 20% of ischaemic strokes and affects more than 20 000 people each year in the UK.<sup>2 3</sup> The New England Medical Center Posterior Circulation Registry (NEMC-PCR)<sup>4</sup> provides useful demographic data on those experiencing posterior circulation stroke (defined using brain imaging): patients had a mean age of 60.5 years; 63% were men, and approximately a quarter (24%, n=98) had a preceding transient ischaemic attack.<sup>4</sup>

### Why is it missed?

More than a third (37%) of posterior circulation strokes are misdiagnosed in the emergency department, more than three times as often as anterior circulation strokes.<sup>5</sup> Table 1 presents reasons why posterior circulation strokes might be more difficult to identify, including the absence of both “typical” symptoms and cardiovascular risk factors.

In this patient’s case, the diagnosis was missed because the symptoms of diplopia, dysphagia and dysarthria were either not elicited, or their significance—as “red flags” for posterior circulation disease—was not recognised. Migraine presents particular diagnostic quandaries: migraineurs are more likely to develop headache secondary to acute ischaemia<sup>7 8</sup>; those with aura have a higher incidence of stroke<sup>12 13</sup>; migraine is more often associated with carotid and vertebral artery dissection<sup>14</sup>; and migraine can cause transient neurological deficits without headache (acephalic migraine or “late-life migraine accompaniments”).<sup>15</sup>

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This is one of a series of occasional articles highlighting conditions that may be more common than many doctors realise or may be missed at first presentation. The series advisers are Anthony Harnden, professor of primary care, Department of Primary Care Health Sciences, University of Oxford, and Dr Kevin Barracough, School of Social and Community Medicine, University of Bristol. To suggest a topic for this series, please email us at practice@bmj.com.

## Why does this matter?

Delayed or incorrect diagnosis results in inadequate acute care and poorer outcomes. Posterior circulation strokes have longer “door to needle” times for intravenous thrombolysis than anterior circulation strokes,<sup>16 17</sup> and are more likely to arrive in hospital after the 4.5 hour “time window” for thrombolysis.<sup>18</sup>

Prompt diagnosis is also important to detect and treat two severe life-threatening presentations. Cerebellar infarction can become “malignant”<sup>1</sup> (10–20% of cases), when oedema associated with the infarct results in obstructive hydrocephalus and brainstem compression, necessitating urgent neurosurgical intervention.<sup>19</sup> Basilar artery occlusion, while rare (1% of all strokes), can cause brainstem or thalamic infarction, which can result in severe syndromes such as complete limb and facial paresis with preserved consciousness (the “locked-in” syndrome), reduced consciousness, and oculomotor abnormalities (from midbrain and bilateral thalamic damage, as part of the “top of the basilar” syndrome), coma, and cardiorespiratory disturbances, depending on the site of occlusion.<sup>20</sup> It has an extremely poor prognosis, but can benefit from intra-arterial thrombolysis or mechanical thrombectomy (clot retrieval) even 24 hours after symptom onset.<sup>1</sup>

## How is this diagnosed?

### A careful history

A careful clinical history is key. Focus on the exact onset and nature of symptoms. As in all strokes, the symptoms and signs of posterior circulation stroke typically start suddenly, but onset can be staggered with severe atheromatous disease or small vessel occlusions.<sup>1</sup> Ask about traditional cardiovascular risk factors and atrial fibrillation. Consider features suggesting arterial dissection, such as: new unilateral, posterior neck pain; a history of repetitive or sustained neck movements; or head or neck trauma.

The most common signs and symptoms are shown in figure 3.<sup>1 21</sup> “Dizziness” is the most common symptom of posterior stroke, present in just under half of patients. Less common symptoms, including double vision (15% of cases),<sup>21</sup> visual field loss, disorientation, confusion, and memory loss, are still useful to keep in mind.<sup>2</sup>

Presentation with isolated “dizziness” (vertigo or disequilibrium) or headache is the most common reason for misdiagnosis<sup>5</sup>; take a detailed “posterior circulation” history (focussing on identifying any other posterior circulation symptoms that may not immediately be reported, or risk factors for stroke as described above) from every patient presenting with these symptoms. In particular, it is important to characterise “dizziness” in the history. Distinguish between vertigo (a feeling of rotation or motion), disequilibrium (an unsteady feeling on walking), presyncope (a feeling of an impending loss of consciousness), and light headedness<sup>22</sup>; vertigo and disequilibrium are most suggestive of posterior circulation ischaemia. However, in practice, dizziness often does not fall clearly into any of these categories.

The presence of two or more posterior circulation symptoms should raise a “red flag” (box 1) and should prompt further investigation.

### Box 1: “Red flags” requiring referral or further assessment (based on our specialist opinion)

- Sudden onset vertigo or disequilibrium with one or more additional posterior circulation symptoms (headache, gait or limb ataxia, visual change (that is, diplopia or partial visual field loss), dysarthria, dysphagia, limb weakness)
- Sudden onset vertigo or disequilibrium with a HINTS examination suggesting a central cause (see fig 4)
- In those with known migraine, presentation with any new posterior circulation symptoms, even if these occur with headache, should be referred. Complete a posterior circulation stroke history and examination in those with a change in usual headache pattern (such as changes in duration or severity), with a low threshold for discussion with acute stroke team
- Presence of any two new acute onset posterior circulation symptoms (especially if there are risk factors for stroke) or any new focal posterior circulation neurological signs

## Focused examination

Offer patients a full neurological examination. Focus on eye movements, limb power, and coordination (fig 3). In cases of isolated acute vestibular dysfunction (rapid onset vertigo, nausea, vomiting, and unsteady gait, with or without nystagmus) presenting to hospital, the three-step “HINTS” (Head-Impulse-Nystagmus-Test-of-Skew) examination (fig 4) is reported to have high sensitivity (100%) and specificity (96%) for identifying central lesions.<sup>23</sup> A clear-cut result in a patient with isolated vertigo can help rule out posterior circulation ischaemia.

## Choosing investigations

Computed tomography (CT) is often the first line investigation for suspected acute posterior circulation stroke, but lacks sensitivity, particularly in the brainstem.<sup>2</sup> Magnetic resonance (MR) imaging sequences are more sensitive, and high signal on diffusion weighted imaging is nearly always seen in acute infarction. However, a recent meta-analysis<sup>24</sup> found that, although only 6.8% (95% confidence interval 4.9% to 9.3%) of patients with acute ischaemic stroke have negative diffusion weighted imaging, this is five times more common in posterior circulation stroke (odds ratio 5.1, 95% CI 2.3 to 11.6, P<0.001). Clinical assessment and concurrent vascular imaging (CT or MR angiography to identify relevant occlusions or stenosis) can also be important to help clinch the diagnosis.

## How is it managed?

Immediately refer all patients with suspected posterior circulation ischaemia to specialist stroke services where they exist; in the UK, this should be to a hyperacute stroke service. Intravenous thrombolysis is effective treatment for acute ischaemic posterior circulation stroke<sup>25–27</sup> if given within 4.5 hours of symptom onset.<sup>28</sup> Little data about mechanical thrombectomy for the posterior circulation is available (in contrast with anterior circulation stroke), with the exception of proven basilar artery occlusion.<sup>12</sup> Patients with large cerebellar infarcts require monitoring on a neurocritical care unit (or equivalent) to facilitate prompt referral for neurosurgical intervention, if needed.

As with all strokes, the underlying causes and risk factors, including atrial fibrillation, require investigation and treatment.

## Education into practice

- Think about the last time you referred a patient to an acute stroke team with suspected posterior circulation ischaemia—how did it go, and what would you do differently now?
- Diagnosing posterior circulation ischaemia can be challenging, even for stroke specialists—why do you think this is the case, and what aspects of the history and examination could you use to help?
- How confident do you feel performing the HINTS assessment? What about your colleagues? How might you improve this?

## A patient's perspective

I am 63 years old, not obese, and relatively fit apart from borderline hypertension. I am generally healthy, although I did start getting the kind of migraine headaches presaged by visual disturbance (a "colourless kaleidoscope") about seven years ago; these were generally cleared up by over-the-counter medication straight away.

More recently, I experienced a particularly intense, painful migraine that, unusually, lasted for the best part of a week, with occasional loss of balance and intermittent double vision. I thought these symptoms were all related to migraine, but after five days my symptoms continued to worsen, and I felt very unwell, so I visited my local emergency department. While in the waiting room, I experienced severe difficulty swallowing, and speech was difficult; the doctor on duty referred me to a nurse, who in turn referred me to a second doctor. My daughter had to push me between the nurse and second doctor in a wheelchair, as I had lost my balance. I was monitored for two to three hours, given aspirin, oxygen, and a drip before being discharged, despite having high blood pressure and having lost my balance completely.

I went home and went to bed, but in the small hours of the morning I awoke choking and unable to breathe, and my wife called an ambulance. The ambulance staff were exceptional and took me to another hospital, as it has a dedicated, acute stroke unit, which they felt that I needed immediately. I had had a stroke in my right-hand rear brain and lost all balance and had hugely compromised eyesight. I was released within 48 hours and continue my rehabilitation at home.

## How were patients involved in the creation of this article?

The case description is based on a real patient, who also wrote the "patient's perspective" describing his experiences.

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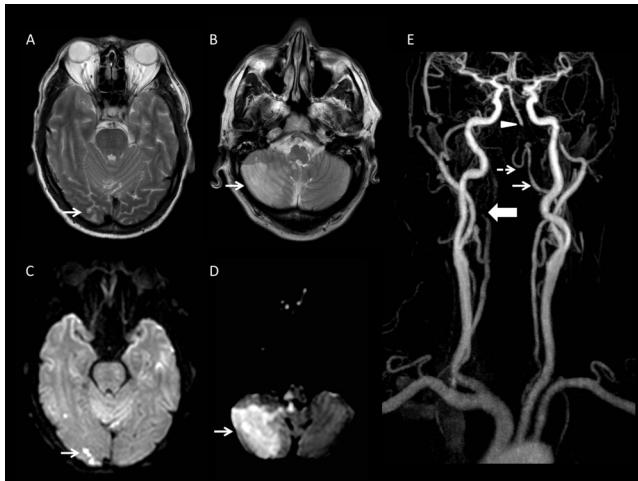
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## Table

**Table 1 | Reasons why posterior circulation ischaemia strokes are more likely to be missed than anterior circulation strokes**

Reason	Anterior circulation (carotid artery territory) ischaemia	Posterior circulation ischaemia
Wide range of symptoms	Brain regions supplied by anterior circulation relatively well defined; tend to present with classical and well known stroke symptoms (motor, sensory, and/or speech or visuospatial disturbance)	Posterior circulation supplies several brain regions with differing functions (fig 2), and has greater anatomical variability; this means that ischaemia can present with a wide range of symptoms and signs, some of which (such as vertigo, reduced conscious level, diplopia) are not as well known as stroke symptoms <sup>1</sup>
Absence of "typical" symptoms	Presentation with "typical" stroke symptoms such as speech disturbance and limb weakness mean that "FAST" (Face Arm Speech Test) assessment used by paramedics and promoted as part of the "Act FAST" public health campaign is likely to be positive	More likely to be FAST negative. <sup>3</sup> Posterior circulation strokes often present with lower scores on the National Institutes of Health Stroke Scale (NIHSS; the most commonly used measure of stroke severity) than anterior circulation strokes, <sup>5,6</sup> making them more likely to be misdiagnosed in the emergency department <sup>5</sup>
Presentation with non-specific symptoms	Non-specific symptoms such as headache, nausea, vomiting, reduced consciousness are not common (usually only in the context of large ischaemic strokes)	Presentation with non-specific symptoms more likely. In particular, headache is more common in posterior circulation strokes, <sup>7,9</sup> possibly secondary to the denser perivascular innervation in these arterial territories <sup>9</sup>
Absence of cardiovascular risk factors	Usually strongly associated with cardiovascular risk factors (such as hypertension, diabetes, hypercholesterolaemia, smoking, family history of cardiovascular disease)	Arterial dissection (both spontaneous and traumatic) is responsible for a quarter of posterior circulation strokes <sup>10</sup> (compared with 2% of all ischaemic strokes <sup>11</sup> ), and so strokes in this territory often occur in younger patients without obvious cardiovascular risk factors <sup>2</sup>

## Figures



**Fig 1** Magnetic resonance imaging (MRI) of brain of the patient described in the case history. Axial T2 sequences (A, B) and axial diffusion weighted sequences (C, D) show acute infarcts (arrows) in the right occipital lobe (A, C) and right cerebellum (B, D). Contrast enhanced magnetic resonance angiography (E) shows an abrupt occlusion of the right vertebral artery (thick arrow). The left vertebral artery (thin arrow) continues via a tortuous route, before terminating in the posterior inferior cerebellar artery (interrupted arrow). The basilar artery (arrowhead) receives no flow from either vertebral artery, and instead shows retrograde filling from the circle of Willis

### Other cortical regions (including medial temporal and parietal lobes)

Blood supply—Supplied by posterior cerebral artery in some but not all people

Ischaemia symptoms—Neuropsychological such as memory deficits, alexia, acalculia, agraphia, prosopagnosia

### Thalamus

Blood supply—Posterior cerebral artery

Ischaemia symptoms—Sensory loss or disturbance

### Occipital lobes

Blood supply—Posterior cerebral artery

Ischaemia symptoms—Visual field defects

### Brainstem (midbrain, pons, medulla)

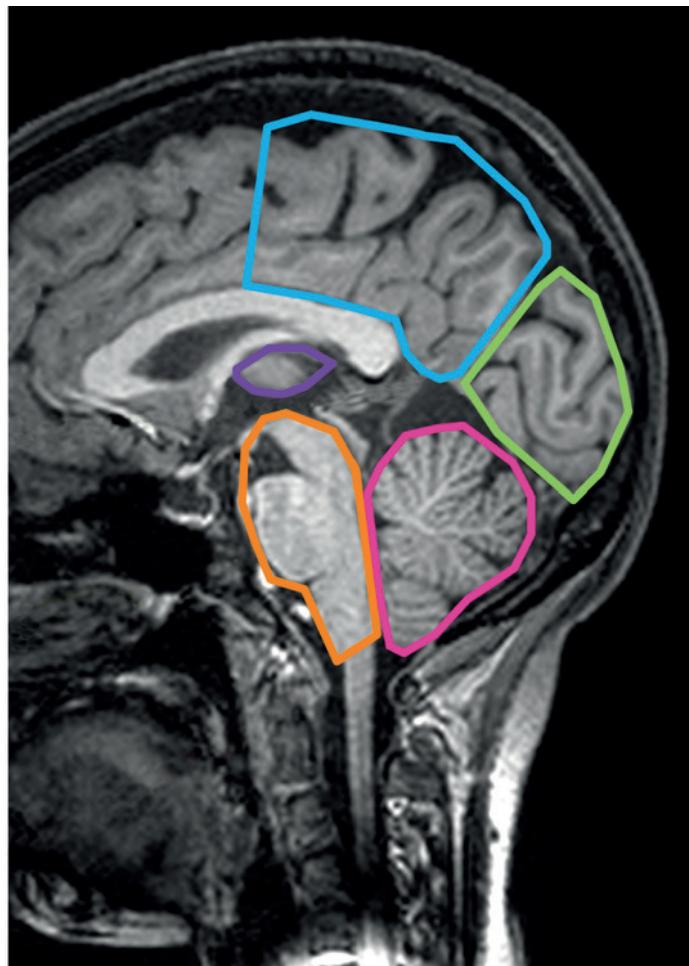
Blood supply—Basilar, superior cerebellar, and anterior inferior cerebellar arteries

Ischaemia symptoms—Limb weakness, sensory loss, cranial nerve palsies; classical brainstem syndromes with crossed signs; “locked-in” syndrome; “top of the basilar” syndrome

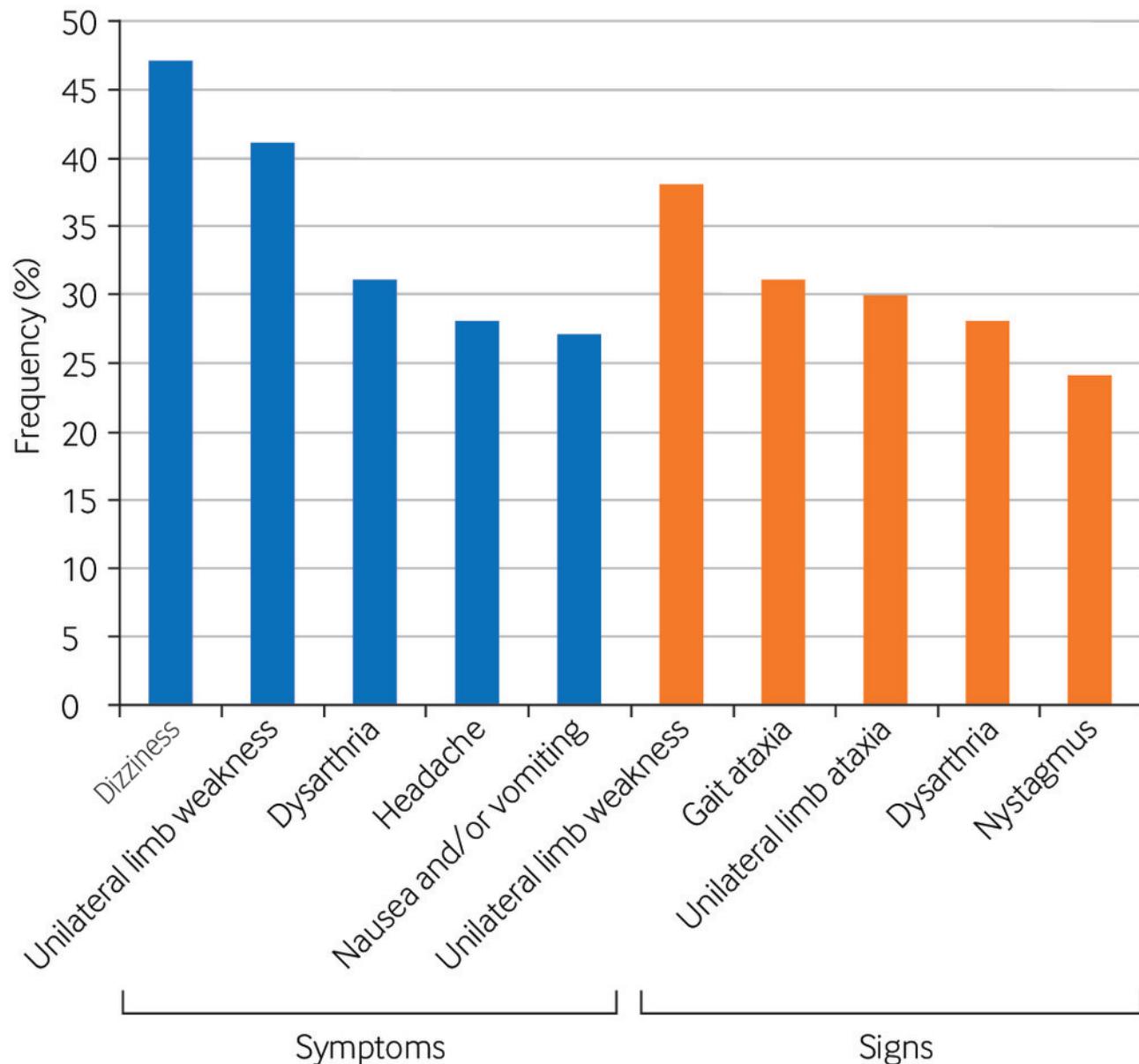
### Cerebellum

Blood supply—Superior, anterior inferior, and posterior inferior cerebellar arteries

Ischaemia symptoms—Vertigo, ataxia, nystagmus, and other cerebellar signs



**Fig 2** Brain regions supplied by the posterior circulation and regional symptoms of posterior circulation ischaemia



**Fig 3** Presenting signs and symptoms in 407 patients with posterior circulation strokes (data from the New England Medical Centre Posterior Circulation Registry<sup>121</sup>)

### Horizontal head impulse test (h-HIT)



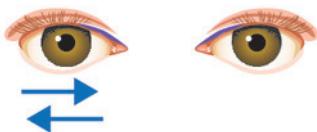
This involves a rapid rotation of the patients' head by the examiner, either from a central to a lateral position (10-20 degrees), or vice versa.

Ask the patient to fixate on a central target. The examiner then rotates the patient's head as described above.

The normal response is for the eyes to remain fixed upon their target.

The presence of a corrective eye movement is **abnormal** and indicates a peripheral lesion.

### Nystagmus



**Nystagmus due to peripheral lesions** is usually present at rest, horizontal, unidirectional and the direction does not change with eye position (although the nystagmus may disappear when looking in the direction of the short phase).

**Nystagmus due to central causes** is often gaze-evoked and may change direction in different eye positions.

### Skew deviation



Access using the alternate cover test.

Ask the patient to fixate on a central target; the examiner then covers each eye in turn.

**The normal response** is for the eyes to remain in their resting position.

The presence of a refixation saccade is an **abnormal response**; this manifests as a small vertical corrective movement that occurs once the cover is removed. This indicates a central lesion.

The presence of either a normal h-HIT or multidirectional nystagmus or abnormal skew is highly suggestive of a central lesion

**Fig 4** The Head-Impulse-Nystagmus-Test-of-Skew (HINTS) examination (adapted from Schulz et al<sup>1</sup> and Kattah et al<sup>23</sup>). A video of the examination can be found at <https://collections.lib.utah.edu/details?id=177180>