Posterior Circulation Stroke

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Overview

- Cases
- Background
- Anatomy
- Signs and Symptoms
- Treatment
- Review of Cases

Case 1

- 50 yo right handed M with a PMHx of HTN, HLD, T2DM, CAD, previous pontine stroke who presents with vertigo with any movement of his head. Additionally he says he has had persistent, severe headache since this morning as well as nausea and vomiting.

Diagnosis?

Case 2

- 46 yo right handed M with no PMHx who had finished running 4 miles on the treadmill when he felt right neck pain, headache, and right facial numbness. He tried to eat a snack but “wasn’t able to hold anything down” and when walking toward his car he felt that he kept walking towards the right and “had to hold the walls” while getting out of his house. He went to an OSH ED and was found to have dysphonia, dysphagia, nystagmus, and singultus. He was transferred to UH for higher level of care.

Diagnosis?

Case 3

- 70 yo right-handed M with no known PMHx who was at home with his wife when he was noted to suddenly have left sided weakness and was acutely unresponsive. His wife thinks he had some twitching of his right arm and leg and noted that he was incontinent of urine. Upon arrival to the ED he was found to have a flaccid left side with leftward gaze deviation. He was still unresponsive.

Diagnosis?

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Background

- Posterior circulation strokes occur in the vertebrobasilar arterial system
  Arteries: vertebrals, basilar, PICA, AICA, SCA, PCAs

- **20% of all ischemic strokes**

- **Difficult to identify and localize**
  - ~ 37% of posterior circulation strokes are misdiagnosed which is > 3x more often than anterior circulation strokes

- Consequences of delayed or missed diagnosis
  - Severe disability
  - Death
  - Inadequate secondary prevention

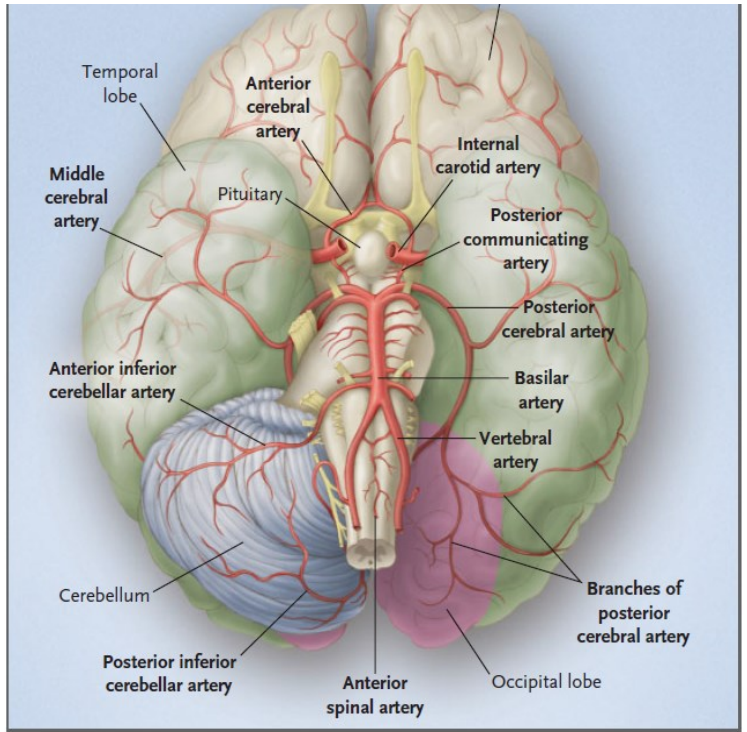

Figure 1. Arterial Supply of the Brain Stem, Cerebellum, Occipital Lobes, Posterior Temporal Lobes, and Thalamus.

The vertebrobasilar arterial supply feeds the brain stem (medulla, pons, and midbrain), cerebellum, occipital lobes, posterior temporal lobes, and thalamus (not visible in this view). The arterial supply consists of the extracranial and intracranial vertebral arteries, which unite to form the basilar artery, which runs midline along the ventral surface of the brain stem, feeding it with small, deep perforators until it merges with the circle of Willis to give off the posterior cerebral arteries.

N Engl J Med. 2005 Jun 23;352(25)2618-26.

BMJ. 2018 Apr 19;361:k1185.
BMJ. 2014 May 19;348:g3175.

Pitfalls in the Diagnosis of Cerebellar Infarction

Sean I. Savitz, MD, Louis R. Caplan, MD, Jonathan A. Edlow, MD

Abstract

Background: Cerebellar infarctions are an important cause of neurologic disease. Failure to recognize and rapidly diagnose cerebellar infarction may lead to serious morbidity and mortality due to hydrocephalus and brain stem infarction.

Objectives: To identify sources of preventable medical errors, the authors obtained pilot data on cerebellar ischemic strokes that were initially misdiagnosed in the emergency department.

Methods: Fifteen cases of misdiagnosed cerebellar infarctions were collected, all seen, or reviewed by the authors during a five-year period. For each patient, they report the presenting symptoms, the findings on neurologic examination performed in the emergency department, specific areas of the examination not performed or documented, diagnostic testing, the follow-up course after misdiagnosis, and outcome. The different types of errors leading to misdiagnosis are categorized.

Results: Half of the patients were younger than 50 years and presented with headache and dizziness. All patients had either incomplete or poorly documented neurologic examinations. Almost all patients had a computed tomographic scan of the head interpreted as normal, and most of these patients underwent subsequent magnetic resonance imaging showing cerebellar infarction. The initial incorrect diagnoses included migraine, toxic encephalopathy, gastritis, meningitis, myocardial infarction, and polyneuropathy. The overall mortality in this patient cohort was 40%. Among the survivors, about 50% had disabling deficits. Pitfalls leading to misdiagnosis involved the clinical evaluation, diagnostic testing, and establishing a diagnosis and disposition.

Conclusions: This study demonstrates how the diagnosis of cerebellar infarction can be missed or delayed in patients presenting to the emergency department.

ACADEMIC EMERGENCY MEDICINE 2007; 14:63–68 © 2007 by the Society for Academic Emergency Medicine

Keywords: medical errors, cerebellar infarction, misdiagnosis, stroke

Background cont'd

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[1]
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 positive	More likely to be FAST negative.[3] 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,[5 6] making them more likely to be misdiagnosed in the emergency department[5]
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,[7-9] possibly secondary to the denser perivascular innervation in these arterial territories[9]
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[10] (compared with 2% of all ischaemic strokes[11]), and so strokes in this territory often occur in younger patients without obvious cardiovascular risk factors[2]

BMJ. 2018 Apr 19;361:k1185.

Background cont’d

- Screening tools geared toward anterior circulation stroke
  - NIHSS
  - Cincinnati Pre-Hospital Stroke Scale (FAST)
- CT Head limited in posterior fossa
  - Bone artifact

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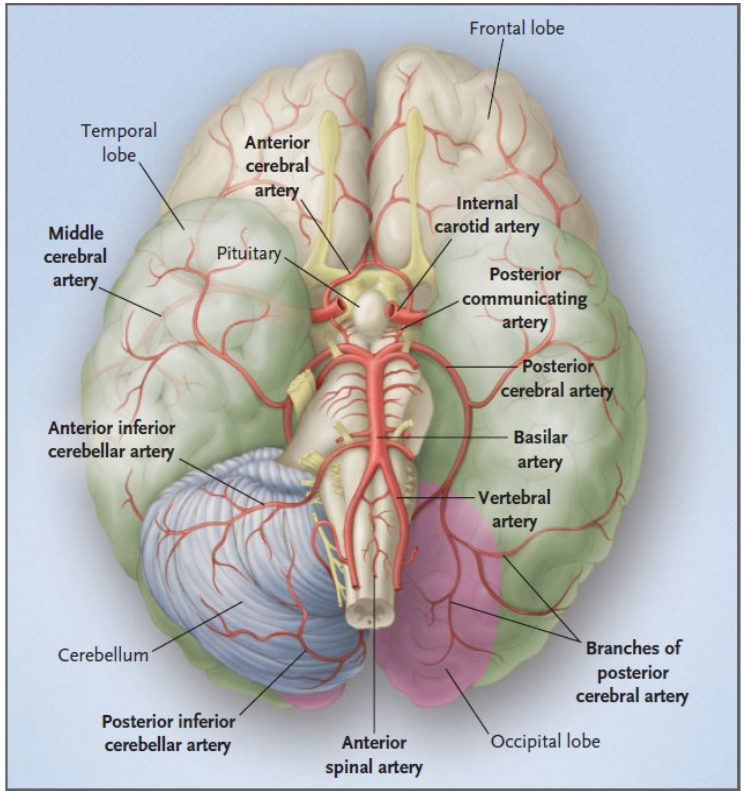
Anatomy



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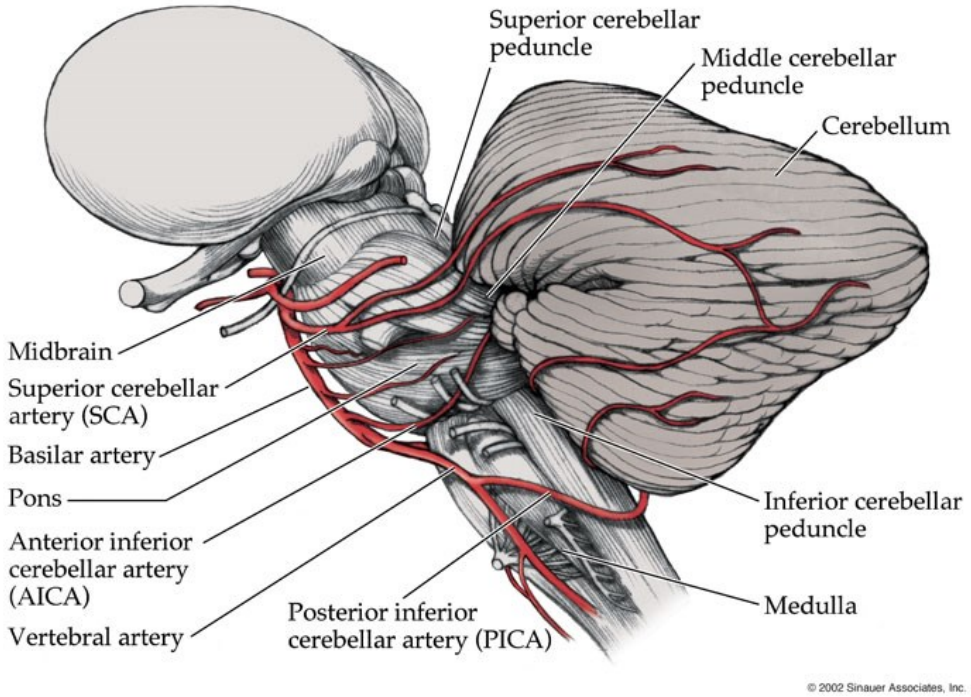
Anatomy cont’d



Blumenfeld (2010, p. 649)

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Posterior Circulation Stroke

- **Most common symptoms:**
  - Hemiplegia
  - Facial/lingual palsy
  - Sensory loss

- ***Highest predictive value for posterior circulation:**
  - Horner's syndrome
  - Crossed sensory/motor deficits
  - Quadrantanopia
  - CN III palsy

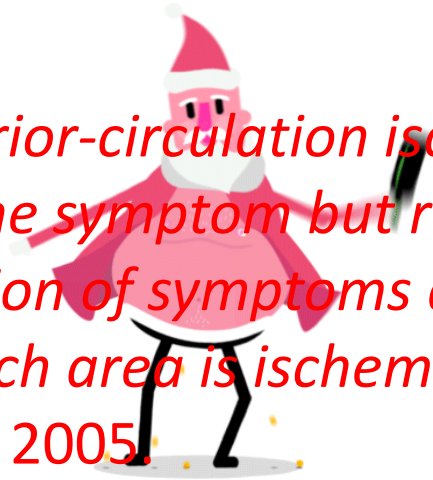
*BUT SENSITIVITY OF ONLY 1.3-4%

Posterior circulation strokes are harder to recognize and assoc. w/ prolonged door-to-needle time.

Stroke.2012;43:2060-2065

Signs and Symptoms

- <u>5 D's:</u>
- 1. Dizziness/vertigo
- 2. Diplopia
- 3. Dysarthria
- 4. Dysphagia
- 5. Dystaxia (Ataxia)



"Posterior-circulation ischemia rarely causes only one symptom but rather produces a collection of symptoms and signs depending on which area is ischemic." – Savitz and Caplan 2005.

[redacted]

N Engl J Med. 2005 Jun 23;352(25)2618-26.

Head Impulse

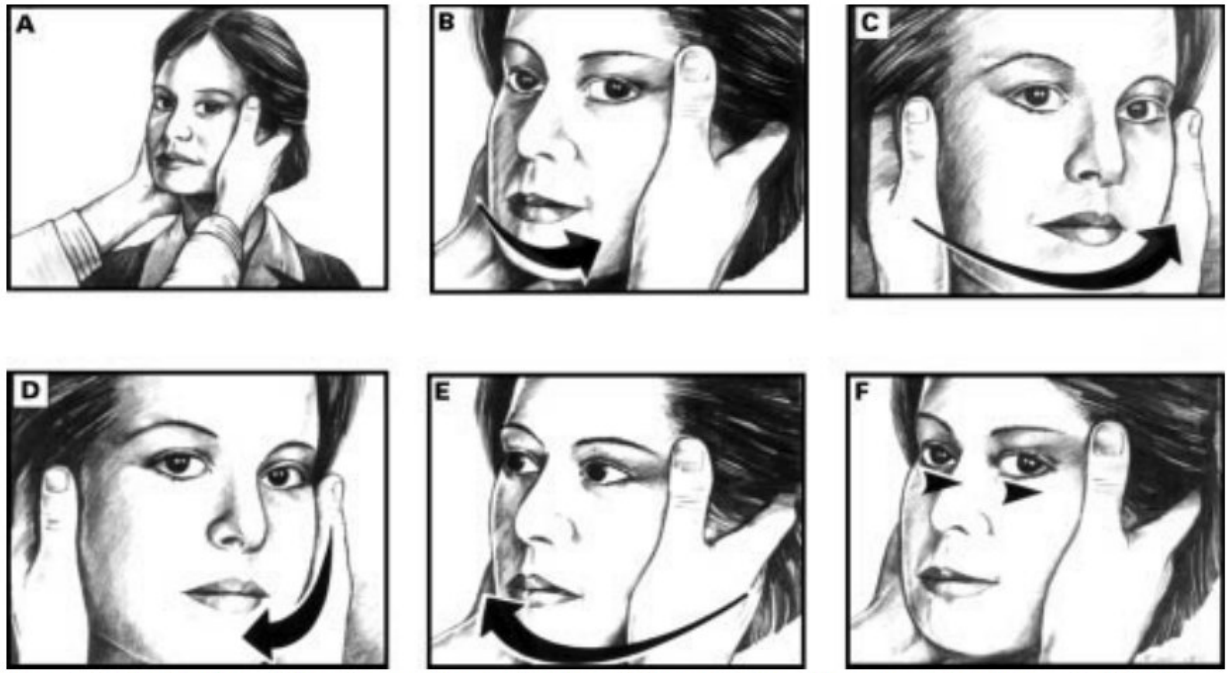
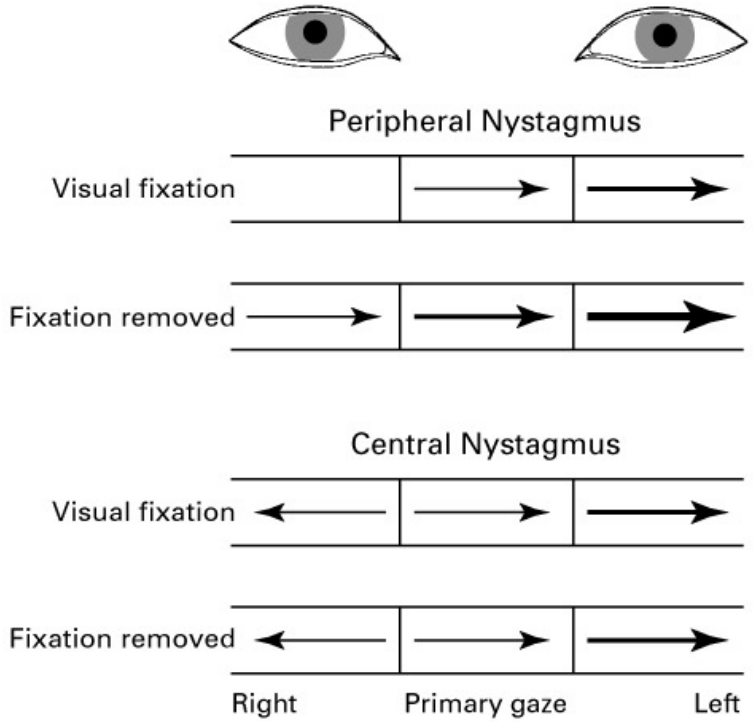


Figure 2 The head impulse test. The examiner turns the patient's head as rapidly as possible about 15 degrees to one side and observes the ability of the patient to keep fixating on a distant target. The patient illustrated has a right peripheral vestibular lesion with a severe loss of right lateral semicircular canal function. While the examiner turns the patient's head to toward the normal left side (top row) the patient is able to keep fixating on target. By contrast, when the examiner turns the patient's head to the right the vestibulo-ocular reflex fails and the patient cannot keep fixating on target (E) so that she needs to make a voluntary rapid eye movement—that is, a saccade, back to target (F) after the head impulse has finished; this can be easily observed by the examiner. It is essential that the head is turned as rapidly as possible otherwise smooth pursuit eye movements will compensate for the head turn. (Based on a figure from Halmagyi and Curthoys.[26])

J Neurol Neurosurg Psychiatry. 2000 Feb;68(2):129-34.

Direction Changing Nystagmus



N Engl J Med. 1998 Sep 3;339(10):680-685.

Test of Skew Deviation

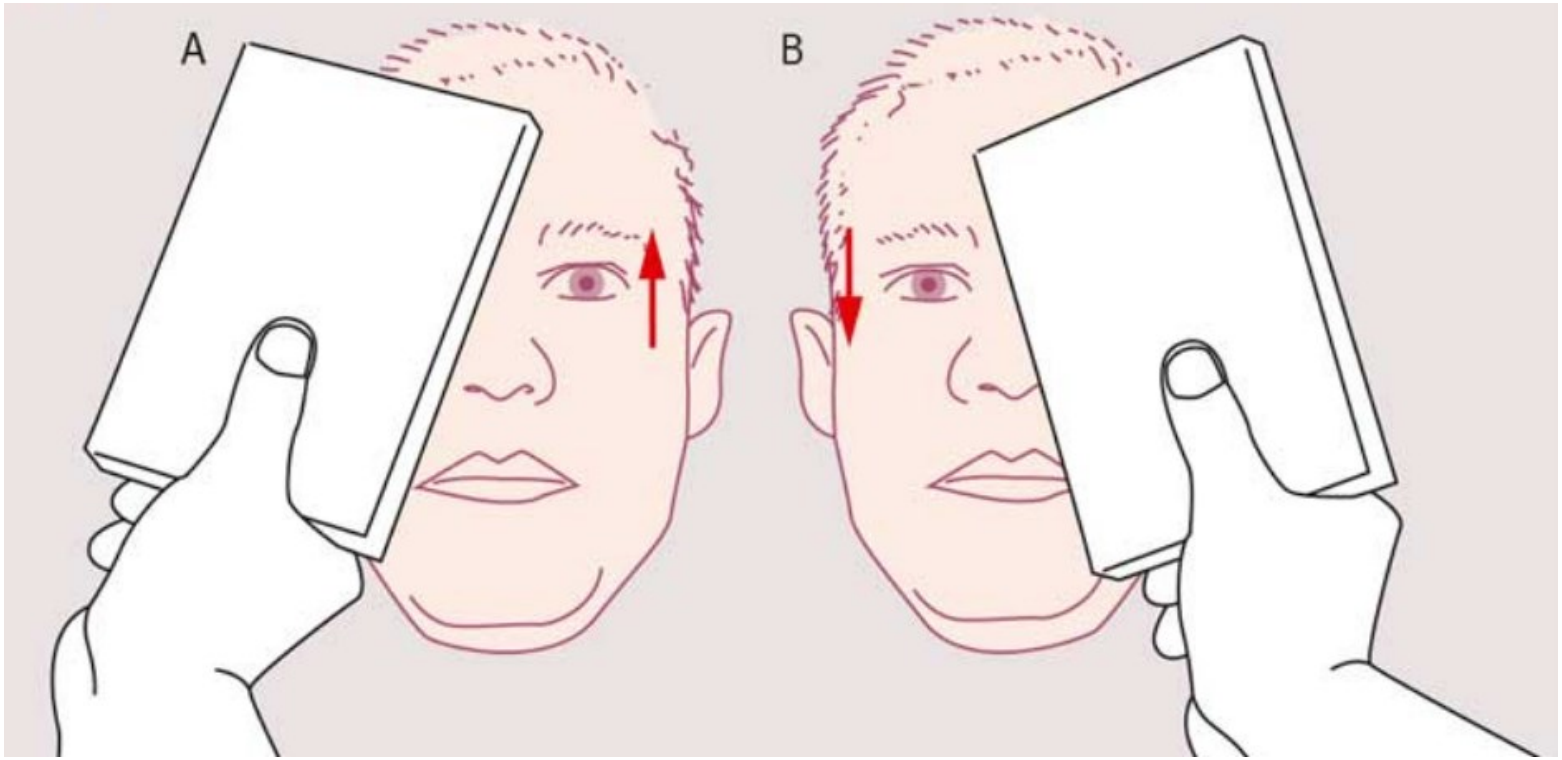


TABLE 1. HINTS to INFARCT

Findings suggesting a benign (peripheral) etiology
1) Unidirectional horizontal–torsional nystagmus with a contralesional fast phase
2) Normal vertical ocular alignment with cross-cover testing
3) Abnormal head impulse test (visible refixation saccade with ipsilesional head impulses)
Findings suggesting a dangerous etiology
1) Direction-changing (gaze-evoked) nystagmus; spontaneous vertical, vertical–torsional, or pure torsional nystagmus
2) Abnormal vertical ocular alignment with cross-cover testing suggestive of skew deviation
3) Normal head impulse test in the planes of right and left horizontal canals
4) Presence of unilateral sensorineural hearing loss with prolonged vertigo (HINTS-plus)

HINTS, Head Impulse, Nystagmus, Test of Skew; INFARCT, Impulse Negative, Fast-phase Alternating, Refixation on Cover Test.

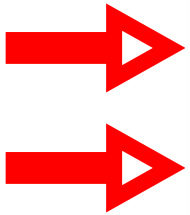
Journal of Neuro-Ophthalmology. 38(2):244-250, June 2018.

Table 1. Key Clinical Features in Patients With Peripheral Versus Central AVS

Symptoms, Signs, and Imaging at Presentation	PAVS (n=25)	CAVS (n=76)	NLR Central (95% CI)
Associated symptoms	12%	41%	0.67 (0.53–0.85)*
Acute auditory symptoms	0%†	3%	0.97 (0.94–1.01)
Headache or neck pain	12%	38%	0.70 (0.56–0.88)*
General neurological signs (including truncal ataxia)	0%	51%	0.49 (0.39–0.61)*
Facial palsy	0%	1%	0.99 (0.96–1.01)
Hemisensory loss	0%	3%	0.97 (0.94–1.0[illegible])
Crossed sensory loss	0%	0%	0.97 (0.94–1.0[illegible])
Dysphagia/dysarthria	0%	3%	0.97 (0.94–1.01)
Limb ataxia	0%	5%	0.95 (0.90–1.00)
Mental status abnormality (lethargy)	0%	7%	0.93 (0.88–0.99)
Hemiparesis (including UMN facial weakness)	0%	11%	0.89 (0.83–0.97)
Severe truncal instability (cannot sit unassisted)	0%	34%	0.66 (0.56–0.77)*
Obvious oculomotor signs	0%	32%	0.68 (0.55–0.80)*
Dominantly vertical or torsional nystagmus	0%	12%	0.88 (0.81–0.96)
Oculomotor paralysis (3-4-6, INO, gaze palsy)	0%	21%	0.79 (0.70–0.89)*
Subtle oculomotor signs	4%	100%	0.00 (0.00–0.11)*
Direction-changing horizontal nystagmus	0%	20%	0.80 (0.72–0.90)*
Skew deviation present or untestable	4%‡	25%§	0.78 (0.67–0.91)*
h-HIT normal or untestable	0%	93%‖	0.07 (0.03–0.15)*
Initial imaging abnormal¶	92%	97%	0.33 (0.05–2.22)
Acute infarct or hemorrhage±chronic lesions	0%	86%	0.14 (0.08–0.25)*
Other acute pathology±chronic lesions	0%	1%	0.99 (0.96–1.01)
Only chronic lesions (leukoaraiosis)¶	92%	11%	11.18 (2.95–42.35)*

*$P<0.05$ for difference of proportion present in PAVS versus CAVS.

A Positive HINTS Exam: 100% Sensitive and 96% Specific for the presence of a central lesion.



Stroke. 2009 Nov;40(11):3504-10.

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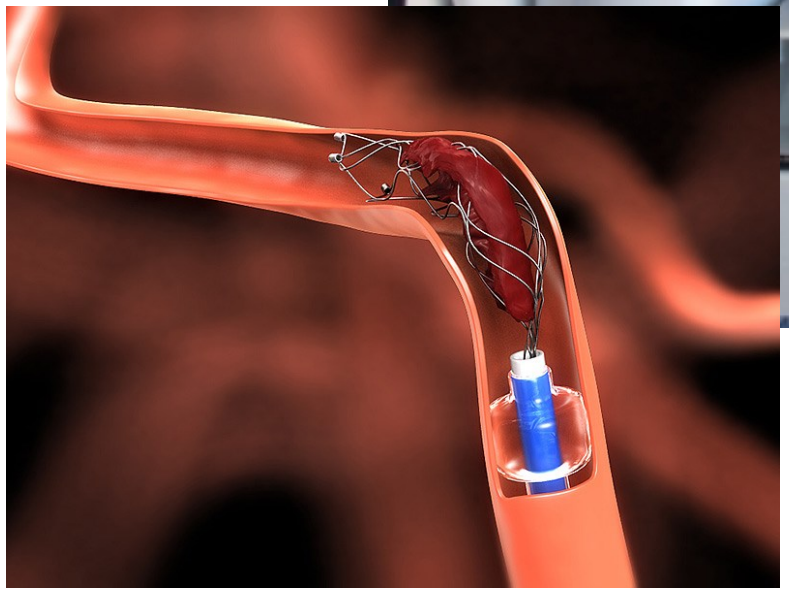
Treatment

- Thrombolytics
  - LKW <4.5h
- MER
  - 24h



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Case 1

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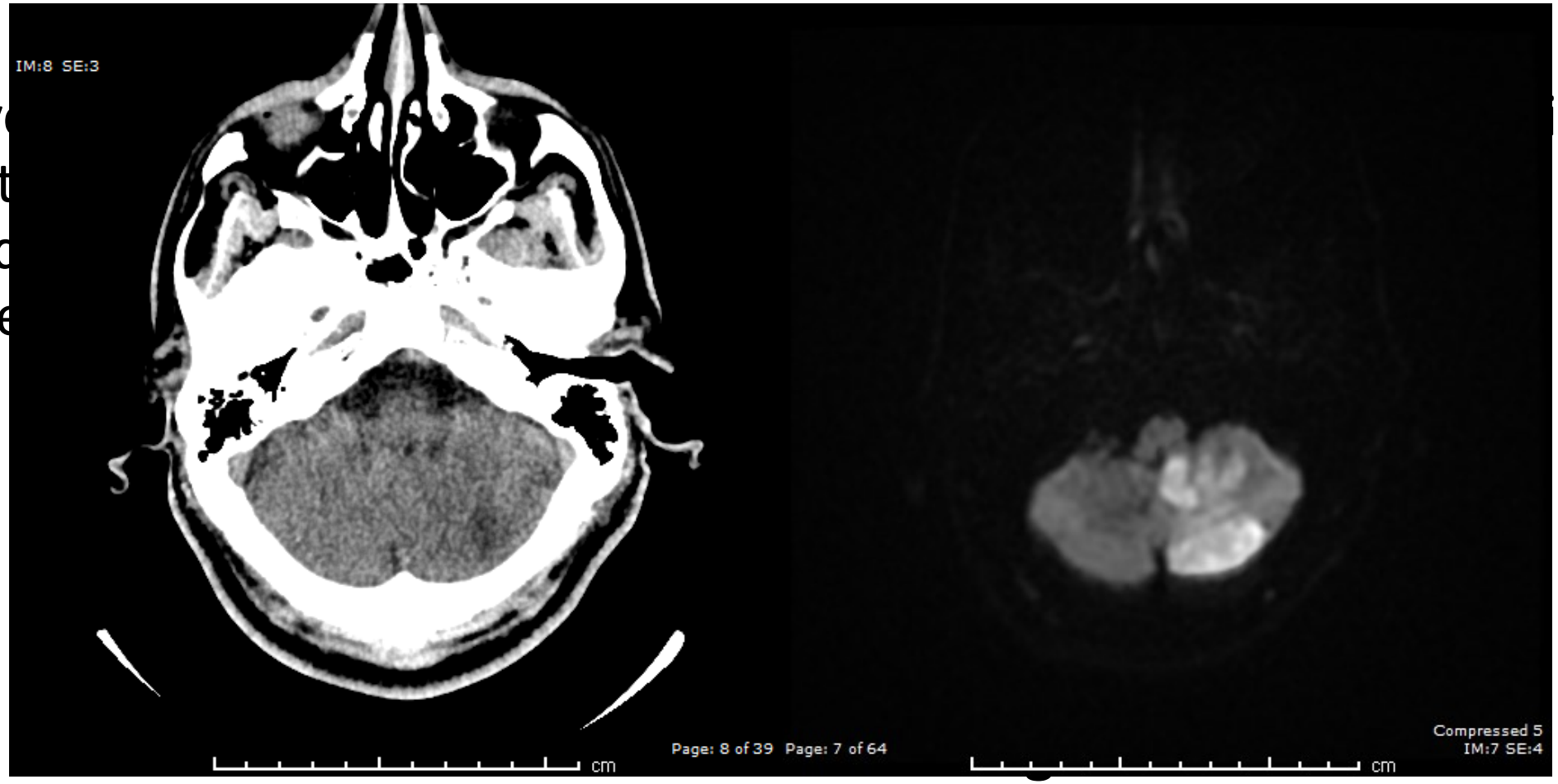
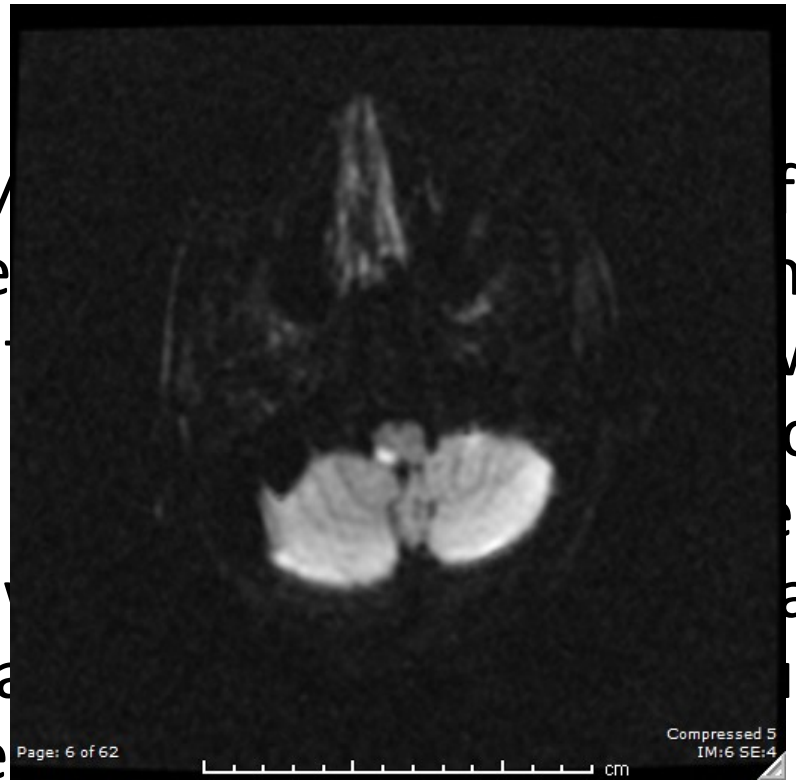


Left cerebellar infarct

Case 2

- 46 yo right handed M finished running 4 miles on the treadmill whe headache, and right facial numbness. He vasn't able to hold anything down" and car he felt that he kept walking towards the walls" while getting out of his house. He as found to have dysphonia, dysphagia s. He was transferred to UH for higher leve



Diagnosis?

Lateral medulla infarct

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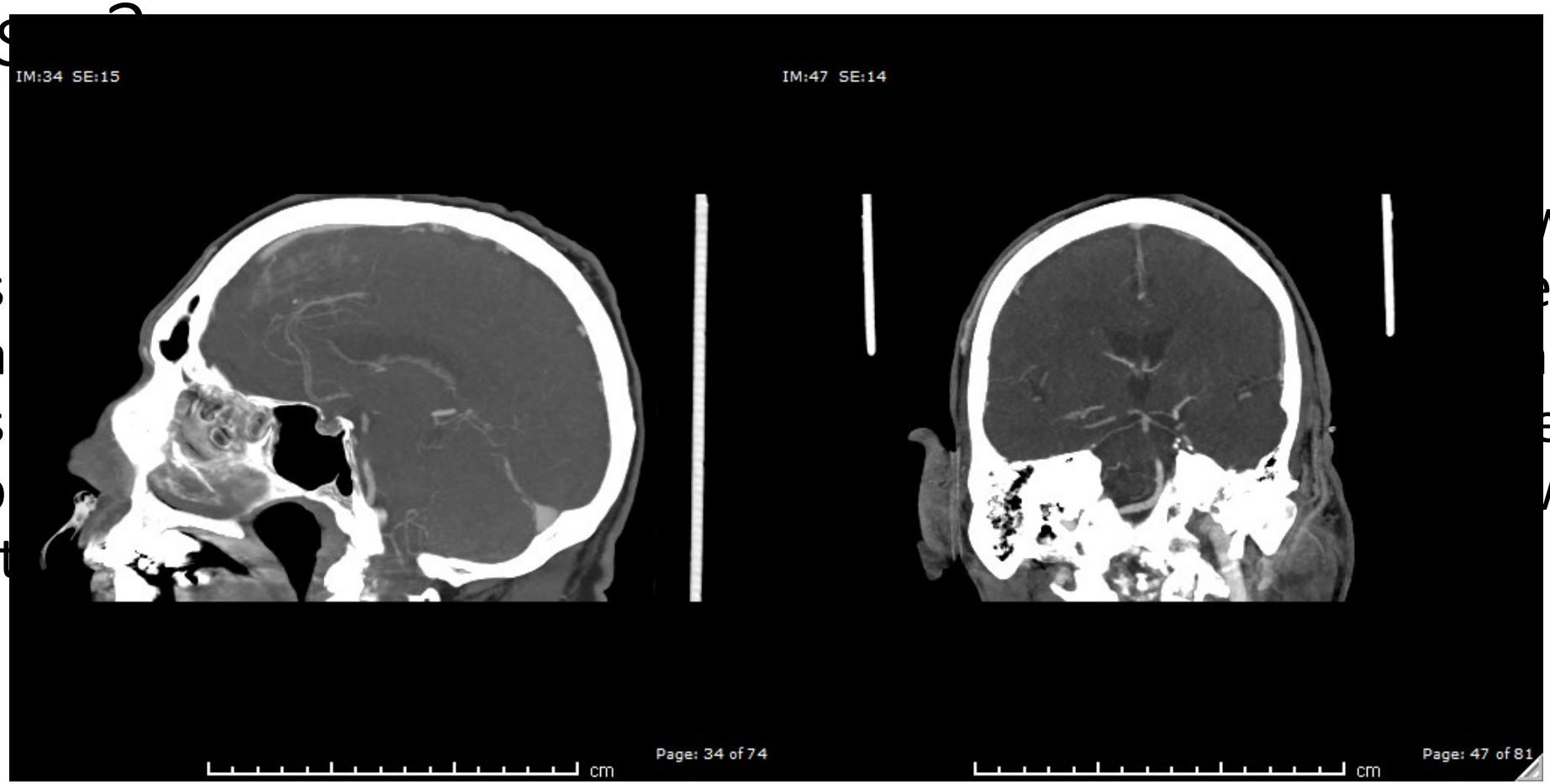

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Diagnosis?

Pre-pontine basilar occlusion