

A Sinister Cause of Recurrent Syncope

Brian L. Risavi^{1*}, Erin M. Reese², Maryrose Kuo¹

¹Lake Erie College of Osteopathic Medicine, Erie, PA, USA

²UPMC Hamot, Erie, PA, USA

Email: *brisavi@lecom.edu

How to cite this paper: Risavi, B.L., Reese, E.M. and Kuo, M. (2025) A Sinister Cause of Recurrent Syncope. *Case Reports in Clinical Medicine*, **14**, 565-572.
<https://doi.org/10.4236/crcm.2025.1410072>

Received: September 8, 2025

Accepted: October 8, 2025

Published: October 11, 2025

Copyright © 2025 by author(s) and Scientific Research Publishing Inc.
This work is licensed under the Creative Commons Attribution International License (CC BY 4.0).
<http://creativecommons.org/licenses/by/4.0/>



Open Access

Abstract

Background: Posterior circulation strokes represent a minority of strokes but are the most difficult to diagnose. Posterior circulation stroke presentations are widely variable, often suggesting benign disorders. History and physical examination are key to timely diagnosis. **Case Report:** A 58 year-old white male presented to the emergency department via Emergency Medical Services (EMS) after experiencing a syncopal event while standing at work. The patient had no symptoms prior to the event. He experienced several additional syncope episodes while lying on the stretcher in the ambulance. Approximately 25 minutes post-arrival, the patient experienced a 15-second episode of unresponsiveness with extensor posturing during which time his skin became flushed and diaphoretic, although his eyes remained open without deviation. He subsequently had four or five more episodes. Of note, he became bradycardic and apneic with these episodes, often as low as 34 beats per minute. Magnetic resonance imaging of the brain showed multiple supra/infratentorial posterior circulation infarcts bilaterally. A follow-up magnetic resonance imaging study 24 hours later demonstrated worsening diffusion abnormalities. Computed tomography (CT) angiogram of the neck demonstrated occlusion of the proximal basilar artery. The patient's mental status declined and intubation was required. The family decided on a do-not-resuscitate order for the patient, and he passed away nine days after admission. **Conclusion:** When a patient has a benign presentation, clinicians should look for high-risk signs and symptoms and exclude them. Posterior circulation strokes should remain high on the differential of patients presenting with recurrent syncope, dizziness, vertigo, nausea, sudden hearing loss, sudden hoarseness, gait disturbances, and focal deficits.

Keywords

Neurology, Acute Medicine, Stroke

1. Introduction

Posterior circulation strokes represent a minority of strokes but are the most difficult to diagnose [1] [2]. Their clinical presentation is widely variable and often non-specific, considering the broad anatomic areas involved [3]. Patients may, in fact, have National Institutes of Health (NIH) stroke scores of zero [4]. Younger patients are frequently misdiagnosed [5] [6]. Posterior circulation strokes are generally ischemic in nature and often present with dizziness or other symptoms suggestive of benign disorders [7]. The patient had high-risk features: recurrent syncope over a short period of time, extensor posturing, and intermittent bradycardia. The history is critically important in evaluating for posterior circulation stroke as symptoms are often very subtle [4]. Physical examination has assumed a more important role in the evaluation and diagnosis of posterior circulation strokes in the acute phase as traditional imaging modalities lack sensitivity. We present an unusual presentation of a posterior circulation stroke. The recurrent syncope, associated with multiple episodes of altered mental status, bradycardia, and extensor posturing, raised concerns for a neurologic cause. When a patient has a benign presentation, clinicians should look for high-risk signs and symptoms and exclude them. This case highlights the broad range of clinical symptoms associated with posterior circulation strokes.

2. Case Presentation

A 58 year-old white male presented to the emergency department at 1625 hours via Emergency Medical Services (EMS) after experiencing a syncopal event while standing at work. The patient had no symptoms prior to the event. He experienced several additional syncopal episodes while lying on the stretcher in the ambulance. His medical history included hypertension, coronary artery disease, hyperlipidemia, and prior coronary artery bypass grafting surgery. He stopped smoking ten years prior and consumed alcohol daily. On arrival in the emergency department, the patient was alert and did not recall the event. Pulse was 62, respiratory rate 16, blood pressure 127/79, and pulse oximetry 97% on room air. He was afebrile. He felt "a little confused" and nauseated. Review of systems was otherwise negative. Physical examination initially revealed no evidence of any focal neurologic deficits. Cardiac examination demonstrated no murmurs, gallops, or rubs. Pulses were +2 in all extremities and no peripheral edema was noted. Physical examination was otherwise unremarkable. The electrocardiogram showed a normal sinus rhythm at a rate of 62 with no acute changes or other abnormalities. Approximately 25 minutes post-arrival the patient experienced a 15-second episode of unresponsiveness with extensor posturing during which time his skin became flushed and diaphoretic, although his eyes remained open without deviation. He did not respond to questions and no seizure activity was observed. He subsequently had four or five more episodes. Of note, he became bradycardic and apneic with these episodes, often as low as 34 beats per minute. Neurologic examination was otherwise normal except as noted above.

Differential diagnosis includes acute aortic syndrome, acute coronary syndrome, cardiac arrhythmia, channelopathies, cerebrovascular accident, pulmonary embolism, and seizure. CT of the head (non-contrast) was done in the ED and revealed no acute abnormalities. The magnetic resonance imaging (MRI) was done (with and without contrast) the following morning at 1130 hours when the patient's condition deteriorated, followed by the electroencephalogram (EEG) and echocardiogram. The electrocardiogram revealed a normal sinus rhythm without acute changes. The EEG revealed no evidence of seizure activity. An echocardiogram revealed an ejection fraction of 60% and was otherwise normal. Magnetic resonance imaging of the brain (**Figure 1**, T2 weighted) showed multiple supra/infratentorial posterior circulation infarcts bilaterally including the following areas: basis pontis, midbrain, corpus callosum, left occipital lobe, and both mesiotemporal lobes. Through shared decision making, due to the extensive nature of the basilar artery clot and brainstem anatomy infarction involved, it was decided that no interventions would be initiated. A follow-up magnetic resonance imaging study 24 hours later (**Figure 2**, T2 weighted) at 1128 hours demonstrated worsening diffusion abnormalities. CT angiogram of the neck followed at 1400 hours and demonstrated occlusion of the proximal half of the basilar artery. The patient's mental

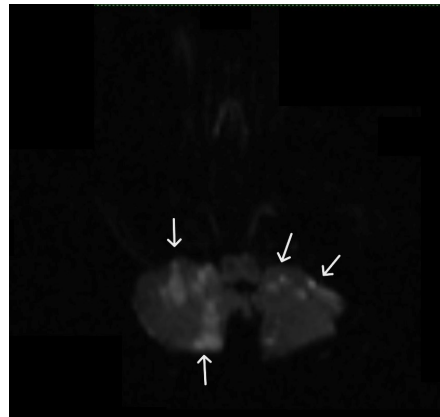


Figure 1. Initial MRI.

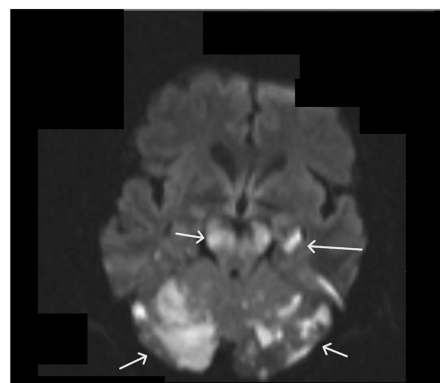


Figure 2. 24-hour follow-up MRI.

status declined and intubation was required. The patient received supportive care and ventilatory support. The family made the patient do not resuscitate and he passed nine days after admission. Patient consent for publication was obtained from family.

3. Discussion

This case demonstrates the difficulty in diagnosing posterior circulation strokes. Clinical presentation is often non-specific and frequently lacking in focal findings. The most common etiologies include ischemia, cardioembolic, large artery atherosclerosis, and small artery disease [3] [8]. Vertebral artery dissection and patent foramen ovale are important causes in young adults [9]. Dizziness is a common complaint and often benign. However, posterior stroke can often manifest with dizziness, particularly as the disease process progresses over time [10]. In fact, dizziness/vertigo are the symptoms most correlated with missed strokes [11]. Approximately 10% of posterior strokes present with isolated spontaneous vertigo [12]. Other data indicate that missed posterior strokes frequently present with altered mental status and headache [13] [14]. Posterior strokes may mimic vestibular neuronitis or labyrinthitis [10]. Concerning symptoms include neurologic deficits, unilateral hearing loss, gait ataxia, and direction-changing nystagmus [15].

Posterior strokes may demonstrate a wide range of findings. Dysphagia, dysarthria, diplopia, weakness/numbness (particularly bilateral), and vertigo have been reported [15]. Visual field deficits, nystagmus and other abnormal eye movements, nausea/vomiting, and crossed deficits also occur [10]. Hoarseness, dysarthria, and dysphagia have been associated with lateral medullary stroke. In addition, Horner's syndrome and decreased pain/temperature sensation of the face often occur (unilateral) [16].

Dizziness is a non-specific complaint and frequently benign. There are, however, multiple serious etiologies. Fluid and electrolyte abnormalities, acute coronary syndrome, cardiac arrhythmias, stroke, anemia, and hypoglycemia may all contribute to the complaint of dizziness [10]. Consequently, it is imperative to discern a detailed history of the patient's onset of dizziness. The acronym ATTEST is a useful aid: associated symptoms, timing and trigger, bedside exam signs, and additional testing as needed [10]. Associated symptoms refers to other symptoms such as vestibular complaints or focal deficits. Timing and triggers refers to the onset and if it is triggered by a specific circumstance, such as turning the head and if it is constant or intermittent and its duration. Examination signs includes such findings as nystagmus, skew deviation, or focal deficits. Testing might include the HINTS exam and appropriate imaging, such as MRI or CT angiography [10].

Recognizing that posterior strokes can present with symptoms suggestive of vestibular neuritis or labyrinthitis, it is imperative to assess the history closely [10]. The clinician must determine if the symptoms are triggered or exacerbated by movement [10]. Gaze testing, alternate cover test, HIT (head impulse test), targeted neurologic exam with emphasis on cranial nerves, cerebellar testing, and gait testing are important components of the evaluation [10]. The TiTrATE acro-

nym is helpful in obtaining useful information from the history [17]. This stands for timing, triggers, and targeted exam of patient symptoms. Four acute syndromes are of concern. Triggered episodic, spontaneous episodic, trauma/toxic acute, and spontaneous acute [17]. Eye movement evaluation is important in triggered episodic vestibular syndromes and spontaneous acute vestibular syndromes. The goal is to identify serious pathology, e.g., posterior stroke. Triggers for episodic vestibular syndromes may be present at baseline or exacerbated from those at baseline. It is important to note that worsening dizziness with head movement may be suggestive of a central cause as well as a peripheral cause, particularly when occurring in conjunction with abnormal nystagmus [17]. Episodic features may include both benign and serious causes. Vertebrobasilar insufficiency and cardiac arrhythmias are diagnostic considerations. Acute traumatic/toxic vestibular syndromes are typically a single episode after onset of head trauma, intoxication, carbon monoxide exposure, or medication effect [17]. Spontaneous acute vestibular syndromes worsen with head movement. Vestibular neuritis is an example, however, a posterior stroke may present with this symptomatology, particularly when associated hearing loss occurs. The spontaneous occurrence versus triggered occurrence is more suggestive of a central etiology.

Abnormal eye movements play an important role in diagnosing posterior stroke as well. The HINTS (head impulse, nystagmus, test of skew) exam has been shown to be 100% sensitive and 96% specific for posterior stroke [18] [19]. The horizontal head impulse test, direction-changing nystagmus, and test of skew are more useful in the acute setting than CT/MRI imaging [17]. Nystagmus may be spontaneous or gaze-evoked. It is important to note the direction of the fast phase of the nystagmus, and if it is horizontal, vertical, or torsional. Asymmetry is a concerning finding. Gaze-evoked nystagmus is used to assess the presence of direction-changing nystagmus, which is strongly suggestive of a central etiology [16]. Test of skew is another component of the HINTS exam and evaluates the presence of a brainstem lesion. The clinician covers one eye in an alternating fashion to assess for skew deviation. A normal response is the absence of vertical correction [8] [16].

Given the broad anatomic areas affected by this patient's posterior strokes, it is clear why such a diverse clinical presentation occurred. Symptoms frequently mimic benign pathology, with misdiagnosis nearly three times that of anterior circulation strokes, even when patients are evaluated by neurology [6] [20]. Current evidence-based decision-making dictates that the physical examination is far more reliable compared to imaging for the initial acute evaluation when a posterior circulation stroke is suspected. The focus is on the timing and triggers of the dizziness, as well as bedside ocular exam findings utilizing HINTS. Emergency physicians, however, should receive formal training in the HINTS exam [21]. Most posterior circulation strokes are ischemic in nature. Treatment may include intravenous thrombolysis, endovascular intervention for basilar artery strokes, antiplatelet drugs, lipid lowering drugs, and blood pressure control [22] [23]. Specific cardiac abnormalities, such as atrial/ventricular septal defects, may be poten-

tial sources for an embolic stroke. Cardiology consultation was obtained and no additional testing was performed to rule out cardiac abnormalities.

4. Conclusion

Posterior circulation stroke presentations are widely variable, often suggesting benign disorders. History and physical examination are key to timely diagnosis. Imaging studies have insufficient sensitivity in the acute presentation and have limited utility. Clinicians should look for high-risk symptoms and exclude them. Posterior circulation strokes should remain high on the differential of patients presenting with recurrent syncope, dizziness, vertigo, nausea, sudden hearing loss, sudden hoarseness, gait disturbances, and focal deficits.

Author Contributions

Dr. Risavi: Conceptualization, data curation, formal analysis, writing, review

Dr. Reese: Formal analysis, writing, review

Maryrose Kuo: Writing, review, and editing

Consent

Written informed consent was obtained from the patient's son to publish this report in accordance with the journal's patient consent policy.

Data Availability Statement

Data supporting this case report are available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

- [1] Merwick, A. and Werring, D. (2014) Posterior Circulation Ischaemic Stroke. *BMJ*, **348**, g3175. <https://doi.org/10.1136/bmj.g3175>
- [2] Tarnutzer, A.A., Berkowitz, A.L., Robinson, K.A., Hsieh, Y. and Newman-Toker, D.E. (2011) Does My Dizzy Patient Have a Stroke? A Systematic Review of Bedside Diagnosis in Acute Vestibular Syndrome. *Canadian Medical Association Journal*, **183**, E571-E592. <https://doi.org/10.1503/cmaj.100174>
- [3] Markus, H.S., van der Worp, H.B. and Rothwell, P.M. (2013) Posterior Circulation Ischaemic Stroke and Transient Ischaemic Attack: Diagnosis, Investigation, and Secondary Prevention. *The Lancet Neurology*, **12**, 989-998. [https://doi.org/10.1016/s1474-4422\(13\)70211-4](https://doi.org/10.1016/s1474-4422(13)70211-4)
- [4] Kerber, K.A. and Newman-Toker, D.E. (2015) Misdiagnosing Dizzy Patients: Common Pitfalls in Clinical Practice. *Neurologic Clinics*, **33**, 565-575. <https://doi.org/10.1016/j.ncl.2015.04.009>
- [5] Tarnutzer, A.A., Lee, S., Robinson, K.A., Wang, Z., Edlow, J.A. and Newman-Toker, D.E. (2017) ED Misdiagnosis of Cerebrovascular Events in the Era of Modern Neu-

- roimaging. *Neurology*, **88**, 1468-1477.
<https://doi.org/10.1212/wnl.0000000000003814>
- [6] Arch, A.E., Weisman, D.C., Coca, S., Nystrom, K.V., Wira, C.R. and Schindler, J.L. (2016) Missed Ischemic Stroke Diagnosis in the Emergency Department by Emergency Medicine and Neurology Services. *Stroke*, **47**, 668-673.
<https://doi.org/10.1161/strokeaha.115.010613>
- [7] Lee, H. (2014) Isolated Vascular Vertigo. *Journal of Stroke*, **16**, 124-130.
<https://doi.org/10.5853/jos.2014.16.3.124>
- [8] Blum, C.A. and Kasner, S.E. (2015) Transient Ischemic Attacks Presenting with Dizziness or Vertigo. *Neurologic Clinics*, **33**, 629-642.
<https://doi.org/10.1016/j.ncl.2015.04.005>
- [9] von Sarnowski, B., Schminke, U., Grittner, U., Tanislav, C., Böttcher, T., Hennerici, M.G., *et al.* (2017) Posterior versus Anterior Circulation Stroke in Young Adults: A Comparative Study of Stroke Aetiologies and Risk Factors in Stroke among Young Fabry Patients (sifap1). *Cerebrovascular Diseases*, **43**, 152-160.
<https://doi.org/10.1159/000454840>
- [10] Edlow, J.A., Gurley, K.L. and Newman-Toker, D.E. (2018) A New Diagnostic Approach to the Adult Patient with Acute Dizziness. *The Journal of Emergency Medicine*, **54**, 469-483. <https://doi.org/10.1016/j.jemermed.2017.12.024>
- [11] Saber Tehrani, A.S., Kattah, J.C., Kerber, K.A., Gold, D.R., Zee, D.S., Urrutia, V.C., *et al.* (2018) Diagnosing Stroke in Acute Dizziness and Vertigo: Pitfalls and Pearls. *Stroke*, **49**, 788-795. <https://doi.org/10.1161/strokeaha.117.016979>
- [12] Tsang, B.K.T., Chen, A.S.K. and Paine, M. (2017) Acute Evaluation of the Acute Vestibular Syndrome: Differentiating Posterior Circulation Stroke from Acute Peripheral Vestibulopathies. *Internal Medicine Journal*, **47**, 1352-1360.
<https://doi.org/10.1111/imj.13552>
- [13] Liberman, A.L. and Prabhakaran, S. (2017) Stroke Chameleons and Stroke Mimics in the Emergency Department. *Current Neurology and Neuroscience Reports*, **17**, Article No. 15. <https://doi.org/10.1007/s11910-017-0727-0>
- [14] Nouh, A., Remke, J. and Ruland, S. (2014) Ischemic Posterior Circulation Stroke: A Review of Anatomy, Clinical Presentations, Diagnosis, and Current Management. *Frontiers in Neurology*, **5**, Article No. 30. <https://doi.org/10.3389/fneur.2014.00030>
- [15] Nelson, J.A. and Viire, E. (2009) The Clinical Differentiation of Cerebellar Infarction from Common Vertigo Syndromes. *The Western Journal of Emergency Medicine*, **10**, 273-277.
- [16] Edlow, J.A. and Newman-Toker, D. (2016) Using the Physical Examination to Diagnose Patients with Acute Dizziness and Vertigo. *The Journal of Emergency Medicine*, **50**, 617-628. <https://doi.org/10.1016/j.jemermed.2015.10.040>
- [17] Newman-Toker, D.E. and Edlow, J.A. (2015) TiTrATE: A Novel, Evidence-Based Approach to Diagnosing Acute Dizziness and Vertigo. *Neurologic Clinics*, **33**, 577-599.
<https://doi.org/10.1016/j.ncl.2015.04.011>
- [18] Kim, J. and Lee, H. (2013) Vertigo Due to Posterior Circulation Stroke. *Seminars in Neurology*, **33**, 179-184. <https://doi.org/10.1055/s-0033-1354600>
- [19] Kattah, J.C., Talkad, A.V., Wang, D.Z., Hsieh, Y. and Newman-Toker, D.E. (2009) HINTS to Diagnose Stroke in the Acute Vestibular Syndrome. Three-Step Bedside Oculomotor Examination More Sensitive than Early MRI Diffusion-Weighted Imaging. *Stroke*, **40**, 3504-3510. <https://doi.org/10.1161/strokeaha.109.551234>
- [20] Frederick, M., Rajpal, A., Kircher, C. and Faryar, K.A. (2020) The Trouble with Swal-

lowing: Dysphagia as the Presenting Symptom in Lateral Medullary Syndrome. *The Journal of Emergency Medicine*, **59**, 392-395.

<https://doi.org/10.1016/j.jemermed.2020.06.008>

- [21] Edlow, J.A., Carpenter, C., Akhter, M., Khoujah, D., Marcolini, E., Meurer, W.J., *et al.* (2023) Guidelines for Reasonable and Appropriate Care in the Emergency Department 3 (GRACE-3): Acute Dizziness and Vertigo in the Emergency Department. *Academic Emergency Medicine*, **30**, 442-486. <https://doi.org/10.1111/acem.14728>
- [22] Strbian, D., Tsvigoulis, G., Ospel, J., Rätty, S., Cimflova, P., Georgiopoulou, G., *et al.* (2024) European Stroke Organisation and European Society for Minimally Invasive Neurological Therapy Guideline on Acute Management of Basilar Artery Occlusion. *European Stroke Journal*, **9**, 835-884. <https://doi.org/10.1177/23969873241257223>
- [23] Chhabra, N., O'Carroll, C.B., Wang, H., Shahid, A., Mbonde, A.A., Carlin, R.E., *et al.* (2025) Presentation, Treatment and Outcomes of Acute Basilar Artery Occlusion: A Retrospective Analysis. *Journal of Stroke and Cerebrovascular Diseases*, **34**, Article ID: 108153. <https://doi.org/10.1016/j.jstrokecerebrovasdis.2024.108153>