Cerebellar Infarction Presenting with Acute Vestibular Syndrome in Two U.S. Air Force Pilots

Roger R. Hesselbrock

BACKGROUND: Cerebellar infarction is an uncommon but serious cause of isolated acute vestibular symptoms, particularly in young, healthy individuals, and can easily be overlooked. We present two cases of cerebellar infarction in U.S. Air Force pilots, one of which occurred during flight.

CASE REPORTS: A 41-yr-old man developed acute vertigo, disequilibrium, nausea, and headache, with progressive slow symptomatic improvement, and presented to medical attention 4 d after symptom onset. Brain magnetic resonance imaging showed right inferomedial cerebellar infarction. Echocardiography discovered patent foramen ovale and atrial septal aneurysm. A 40-yr-old man developed severe vertigo, nausea, and vomiting during initial aircraft descent. Head computed tomography scan was performed acutely and was normal. Initial assessment was benign paroxysmal positional vertigo. Brain magnetic resonance imaging 1 mo after symptom onset showed a small right inferior cerebellar infarction. Patent foramen ovale and bilateral atrial enlargement were seen on echocardiography. Both pilots made full neurological recoveries and were eventually returned to flight status.

DISCUSSION: Central causes of isolated acute vestibular symptoms are uncommon and are often not considered in otherwise healthy individuals. Cerebellar infarction is one of these uncommon but increasingly recognized causes of acute vestibular symptoms. As evaluation and management of central causes are much different from peripheral conditions, prompt localization confirmation is paramount. Accurate evidence-based bedside screening methods are available for rapid localization. Awareness of the possibility of central etiologies and careful clinical evaluation with application of bedside screening methods in patients with acute vestibular symptoms will reduce the number of inaccurate diagnoses.

KEYWORDS: dizziness, vertigo, stroke.

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Acute vestibular symptoms such as dizziness and vertigo are not an unusual occurrence, and are often accompanied by nausea, vomiting, unsteadiness, and other constitutional symptoms. These symptoms account for 4–5% of urgent care or emergency department encounters and up to 500,000 visits annually in the United States.[8,12] There are many causes for such symptoms, ranging from benign peripheral etiologies such as labyrinthitis associated with upper respiratory infections to more severe central etiologies, including cerebrovascular disease and neoplasms. In the young, healthy population that is typical of most aviators, benign self-limited peripheral causes are most often expected in the setting of acute vestibular symptoms. However, unexpected central etiologies such as cerebellar or brainstem infarction do occur uncommonly.[1,4] Awareness of this possibility and prompt recognition of signs suggestive of central causes are important, as their evaluation and management are quite different than for peripheral vestibular etiologies. We present two cases of military pilots with acute vestibular symptoms, one with occurrence during flight, who were later discovered to have small medial cerebellar infarctions.

CASE REPORTS

The first patient was a 42-yr-old male transport pilot without any reported intercurrent illness who developed sudden onset

From the Aerospace Medicine Consultation Division, U.S. Air Force School of Aerospace Medicine, Wright-Patterson Air Force Base, OH.

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Address correspondence to: Col. Roger R. Hesselbrock, USAFSAM/FECN, Bldg. 840, 2510 Fifth Street, Wright-Patterson AFB, OH 45433-7913; roger.hesselbrock@us.af.mil.

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of tumbling/spinning sensation, nausea, and centrally located headache. These symptoms improved over time, but he continued to have headache and mild imbalance, which prompted him to seek medical attention 2 d after symptom onset. A noncontrast brain magnetic resonance imaging (MRI) study was obtained and showed a subacute right medial cerebellar infarct (**Fig. 1** and **Fig. 2**). Although by this time his clinical symptoms had resolved, he was admitted to a local hospital for further evaluation. Computed tomography (CT) angiography showed no vascular blockage or other lesions, and detailed laboratory evaluation did not reveal any pertinent abnormalities. He was placed on aspirin and atorvastatin empirically. Echocardiography noted a large patent foramen ovale and he was briefly anticoagulated until final results of hypercoagulability testing were obtained and reviewed. No significant cardiac arrhythmias were seen on 30-d monitoring. He did not receive any clear guidance on closure of the patent foramen ovale. When seen at the U.S. Air Force School of Aerospace Medicine about 15 mo postevent, he was taking aspirin and Crestor. He was clinically asymptomatic and had a normal physical examination and unchanged radiographic findings. Neuropsychological testing showed no clinically significant abnormalities. Transthoracic echocardiography with agitated saline bubble study showed an atrial septal aneurysm with right-to-left shunting at rest, which increased with Valsalva maneuver. He was advised on the potential risks and benefits of continued observation vs. percutaneous closure. He elected to remain on antiplatelet therapy and not pursue closure. He was recommended for return to flying status in nonhigh-performance, multiplace aircraft, with another qualified pilot present during aircraft operations.

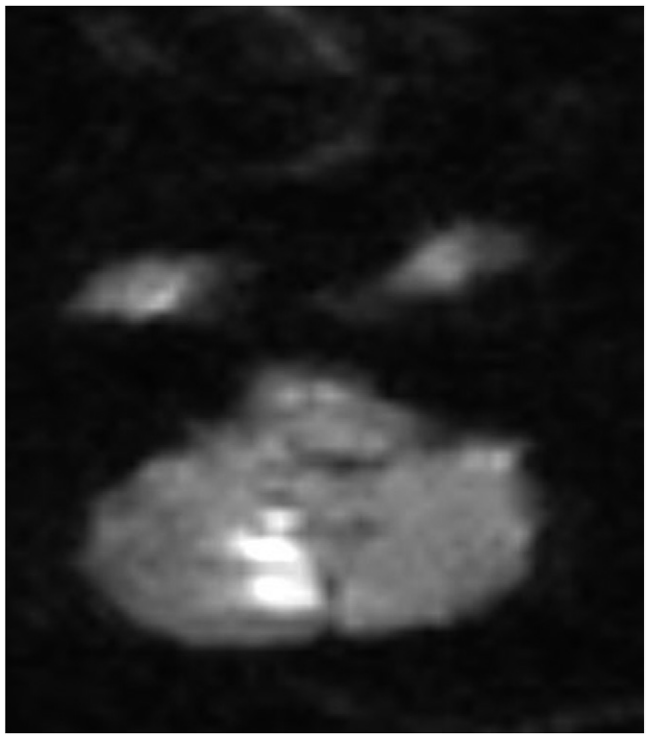

Fig. 1. Patient 1. Diffusion-weighted imaging sequence.

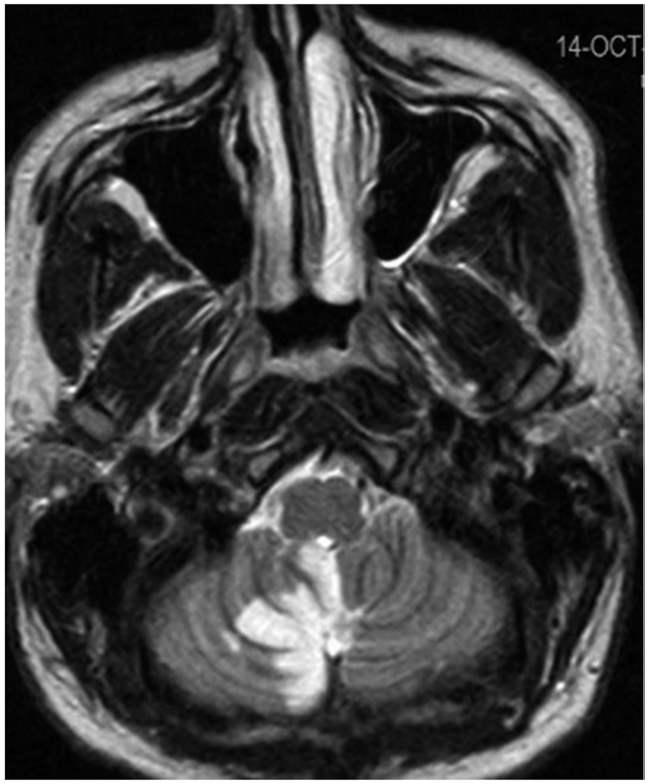

Fig. 2. Patient 1. Fluid-attenuated inversion recovery sequence.

The second patient was a 40-yr-old male tanker pilot who had been experiencing mild upper respiratory symptoms but felt safe to fly. He was completing a routine air refueling mission and had started initial descent when he experienced sudden onset of a sensation of clockwise barrel roll accompanied by a sense of eye movement, which worsened when he tried closing his eyes. After about 15 min he developed worsening nausea and then had multiple emesis episodes. No speech abnormalities were observed, but he felt off balance (no direction predominance). He denied headache, otalgia, aural fullness, tinnitus, hearing loss, numbness, or weakness. He could stand up but could not maintain balance, again without directional predominance. He had a negative noncontrast head CT scan and was treated with antiemetics, without success. He was assessed initially as having an upper respiratory infection with associated vestibular dysfunction. He was able to fall asleep and, on awakening, his imbalance and vertigo had disappeared, but he had "one of the worst headaches" he had ever experienced. Pain was dull and diffuse in location, 8–9/10 in intensity, unaccompanied by nausea, vomiting, photophobia, or phonophobia. He was given an Epley maneuver that seemed to reproduce his symptoms. This led to the working diagnosis that his symptoms were related to benign paroxysmal positional vertigo. He was then referred to otolaryngology and was seen about 1 mo later; examination findings were normal. Brain and internal auditory canal MRI study was read as showing no internal auditory canal abnormalities, but did show abnormal areas in the right cerebellum that enhanced with contrast and were suspicious for infarction (**Fig. 3** and **Fig. 4**). Neurology

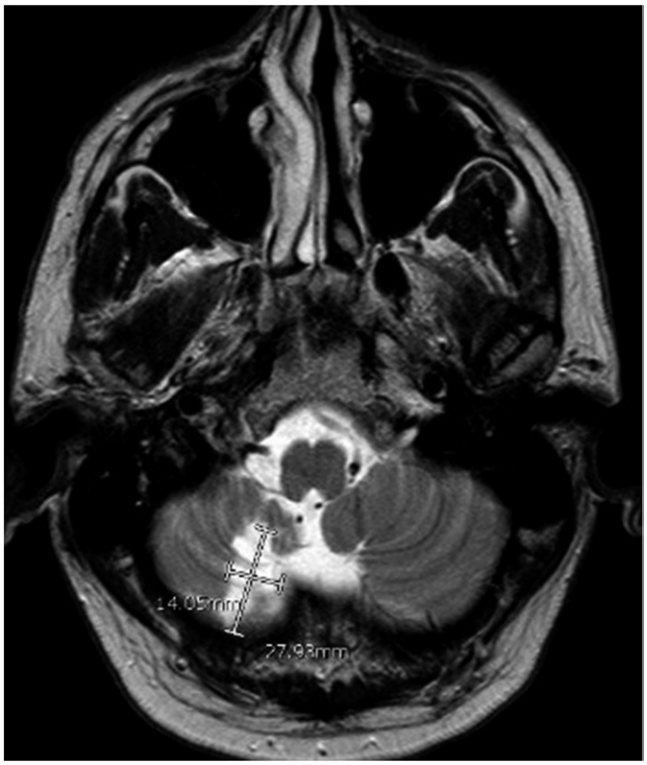

Fig. 3. Patient 2. T2-weighted sequence.

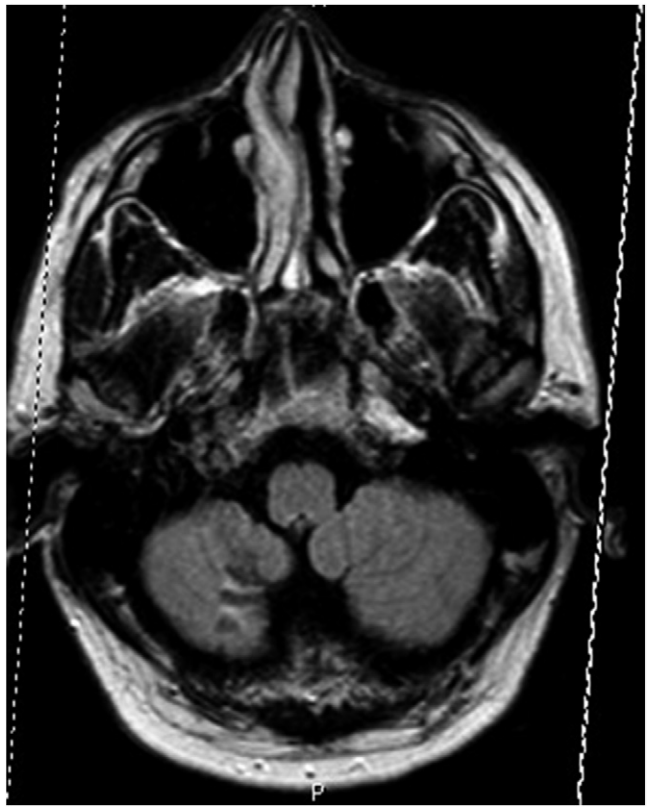

Fig. 4. Patient 2. Fluid-attenuated inversion recovery sequence.

evaluation noted elevated blood pressure and increased lipoprotein(a) level. Repeat imaging studies showed a right medial cerebellar hypodensity consistent with the history of infarct. CT angiography of the head and neck showed normal blood vessels. Transesophageal echocardiogram with bubble study noted a patent foramen ovale. Closure was not recommended, but continued antiplatelet therapy and observation were advised. When seen at the U.S. Air Force School of Aerospace Medicine approximately 4 yr postevent, the patient was taking aspirin, atorvastatin, and lisinopril. He was clinically asymptomatic and had a normal physical examination and unchanged radiographic findings. Neuropsychological testing showed no clinically significant abnormalities. Transthoracic echocardiography with agitated saline bubble study showed a small patent foramen ovale with minimal right-to-left shunting at baseline which did not increase with Valsalva maneuver. He was recommended for return to flying status in nonhigh-performance, multiplace aircraft, with another qualified pilot present during aircraft operations.

DISCUSSION

Accurate assessment of patients with acute vestibular symptoms can be challenging, particularly if symptoms are isolated and if no concerning examination findings are present.[11] This is even more so in the younger age group, where peripheral causes of vestibulopathy are by far the most common. Most peripheral etiologies for acute vestibular symptoms are benign and self-limiting, generally short lived with low potential for long-term residual effects. However, even in a young, otherwise healthy patient, central causes of acute vestibular symptoms do occur and can easily be overlooked. Prompt recognition of potential central causes for acute vestibulopathy is important, as the diagnostic and management approach is not only quite different, but is also more time-critical than for peripheral conditions. For example, posterior fossa ischemic swelling may not be clinically or radiographically apparent initially, but is progressive over 48–72 h and can lead to life-threatening compressive changes that could require urgent neurosurgical intervention.[12] Identification of stroke also facilitates appropriate risk stratification and implementation of measures to prevent further ischemic events.

An optimal diagnostic assessment should be timely, targeted, accurate, and cost effective. Bedside assessment, which includes both history and physical examination, must be the first step in almost all patients with acute vestibular symptoms. In combination with the ABCD2 score (Age, Blood pressure, Clinical features, Duration, and Diabetes) and general neurological examination, specialized oculomotor-based testing can risk-stratify stroke in patients with acute dizziness and more accurately guide the need for cerebral imaging studies.[5] This is important, as the preferred imaging modality, MRI, is not always readily available, may not show hyperacute cerebral infarctions, and, if performed indiscriminately, is estimated to increase annual healthcare expenditures by more than $1 billion.[7] Historical features that increase concern for a central

process include presence of vascular disease risk factors, the "four Ds" (symptoms of diplopia, dysarthria, dysphagia, and dysphonia), sudden deafness or tinnitus, and acute neck or occipital pain.[11] A simple three-step combination oculomotor examination method, HINTS (Head-Impulse, Nystagmus, Test of Skew), can be performed at the bedside in less than 1 min and was shown in a recent cross-sectional study to be 100% sensitive and 96% specific for stroke.[3] Another recent cross-sectional study of high-risk patients with acute vestibular symptoms compared HINTS with ABCD2 for assessing stroke, and found that HINTS had greater sensitivity and specificity (96% and 84%, respectively) than ABCD2 alone (61% and 62%, respectively).[9] Other signs concerning for central nervous system causes of acute vestibular symptoms include focal examination abnormalities such as ataxia, dysmetria, and dyssynergia.[11]

Regarding causality, cardioembolic events account for about 20–30% of all ischemic strokes.[10] While the distribution of cerebral blood flow favors anterior circulation territory (carotid artery system) in cardioembolic stroke, posterior circulation territory (the vertebrobasilar artery system) cardioembolic strokes do occur, and actually accounted for an estimated 24% of posterior circulation strokes in one 407-patient series.[2] It is interesting that both of our patients were found to have cardiac septal abnormalities on echocardiography, which may have contributed to their strokes. One patient had a small patent foramen ovale with little right-to-left shunting even during Valsalva maneuver, and the second patient had a larger patent foramen ovale with associated atrial septal aneurysm and significant right-to-left shunting at rest. Neither patient elected to have patent foramen ovale closure and both patients were advised to continue indefinite antiplatelet therapy.

In addition to focal neurological deficits that could compromise aircraft operation, cerebellar injury can also produce cognitive impairments in executive function, reasoning, and judgment, which, if unrecognized, could have adverse aeromedical effects.[6] This concept is not universally accepted or recognized, but should be considered, especially in the assessment of aviators with cerebellar damage for suitability to return to aviation duties. Screening of cognitive function would be a reasonable starting point in the aviator with cerebellar damage who wishes to return to flying. Functional/operational assessments may also be indicated and can provide valuable complementary information to facilitate aeromedical disposition.

Cerebellar infarction is an uncommon cause of acute vestibular symptoms and can easily be overlooked during initial assessment. Clinicians should always be cognizant of this possibility when assessing any patient who presents with acute vestibular symptoms. A careful evaluation with application of targeted evidence-based bedside examination methods such as HINTS is paramount to most accurately predict the underlying site and processes responsible for clinical symptoms. Using this approach, the number of inaccurate diagnoses can be reduced, resulting in a more timely, efficient, cost-effective diagnostic and management strategy. In addition to vascular imaging and serologic testing, echocardiographic screening for structural cardiac abnormalities is indicated if the diagnosis of cerebellar infarction is confirmed.

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Author and affiliation: Roger R. Hesselbrock, M.D., B.S., Aerospace Medicine Consultation Division, U.S. Air Force School of Aerospace Medicine, Wright-Patterson AFB, OH.

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