TEXAS NEUROLOGICAL SOCIETY JUNE 2026

Broca's Area

The Voice of Texas Neurology

President's Message

President, Yessar Hussain, MD
Austin

It is both an honor and a privilege to serve as President of the Texas Neurological Society. Over the past eight years, TNS has become far more than a professional organization to me, it has grown into a vibrant community of colleagues, mentors, educators, advocates, and friends united by a shared commitment to advancing neurological care across Texas.

Through my journey with TNS, I have had the opportunity to witness the extraordinary dedication of neurologists throughout our state. Whether practicing in academic institutions or private settings, in large metropolitan hospitals or rural communities, our members consistently demonstrate an unwavering commitment to excellence in patient care, education, research, and advocacy.

As I step into this role, one of my primary goals is to strengthen neurology's voice in Texas. Our field is facing an increasingly complex environment, marked by evolving healthcare policies, reimbursement challenges, access disparities, and rapidly changing patient needs. These realities underscore the importance of a unified and proactive presence. TNS is well positioned to expand its advocacy efforts and deepen its engagement at the legislative level, helping to shape policies that directly impact the care we provide and the patients we serve.

Equally important to me is fostering the continued engagement of residents, fellows, and medical students within our society. The establishment of the Residents Committee several years ago marked an important step toward building meaningful connections between trainees and TNS. It has been inspiring to see this initiative grow, with increasing opportunities for mentorship, collaboration, leadership development, and professional networking. Investing in the next generation is essential to ensuring a strong and dynamic future for neurology in Texas.

Looking ahead, I am also committed to broadening our membership and extending our reach across the state. Many talented neurologists have yet to engage with TNS, and I believe our organization offers significant value through education, advocacy, collaboration, and community. By continuing to grow and strengthen our collective voice, we not only support one another as professionals but also enhance the care delivered to patients and communities throughout Texas.

I am deeply grateful for the opportunity to serve this remarkable organization. I look forward to working alongside all of you in the year ahead as we continue to build a stronger, more connected future for neurology in Texas.

Sincerely,

Yessar Hussain, MD
President, Texas Neurological Society

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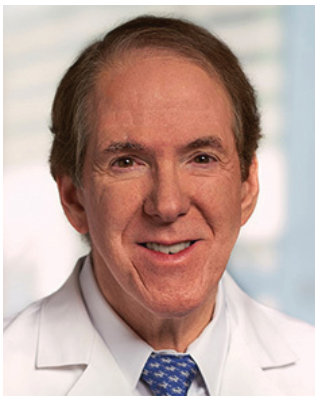


RANDOLPH W. EVANS, MD

This Issue

I thank our officers and other contributors for their excellent submissions to this issue. We look forward to seeing you at the TNS Summer Conference at La Cantera in San Antonio 7/31/26-8/1/26. Program director Leorah Freeman; Erin Furr-Stimming, chair; and the education committee have planned an excellent program.

Artificial intelligence

Many of us are integrating AI into our practice, writing, and research. I prefer to type but ambient AI is excellent for many. For point of care research, I now go directly to AI rather than UpToDate, pubmed, or google. Open Evidence is excellent. To research topics for writing, I now start with Open Evidence rather than PubMed.

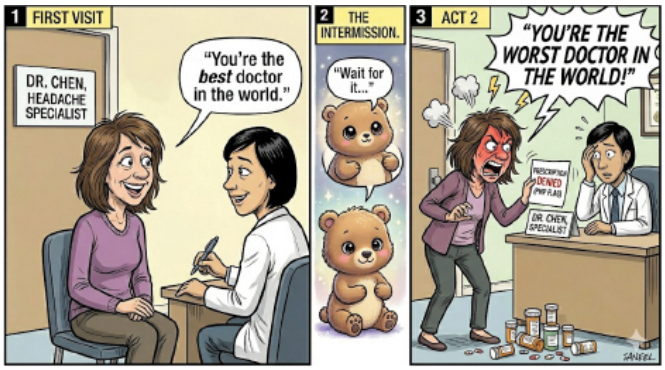
For many journals, you must disclose your use of AI but it has been a challenge for editors to recognize AI submissions. Hallucinations have become less but are still present. To paraphase, trust somewhat but verify. Trust but verify is an over 100-year-old Russian proverb which President Reagan first used during the signing of the INF Treaty with General Secretary Gorbachev in 1987.

I have generated the following case report with the assistance of Open Evidence which I trust somewhat but verified and modified a lot. This editor has no problem with the use of AI for writing as long as you verify and acknowledge the AI contribution. A major purpose of Broca's is education. However, another purpose is to give all of us including students, residents, fellows, and clinicians an opportunity to develop and practice their research, writing, and cognitive thinking skills. Passive use of AI (cognitive off-loading) robs you of developing critical thinking and writing skills (How do you get into the New England Journal? Practice, practice, practice. Or cry, rewrite, cry again). Once developed, using AI as a tool can enhance critical thinking when you evaluate and refine AI outputs.

I don't miss the bad old days when I would spend hours at the TMC library researching topics, pulling and lugging around bound journals, and copying articles for hours (I think I still have $100 on my old library card). I don't miss using my Underwood typewriter with carbon paper or later IBM Selectric and proofing as I went along. Starting in the digital dark ages, I am still amazed at near instant access to full text articles, spell and grammar check, using a pc for word processing, reference manager, and now AI. I recently did spring cleaning in my office and threw away filing cabinets of correspondence with contributing authors and manuscripts and indexed folders of copied articles on numerous topics.

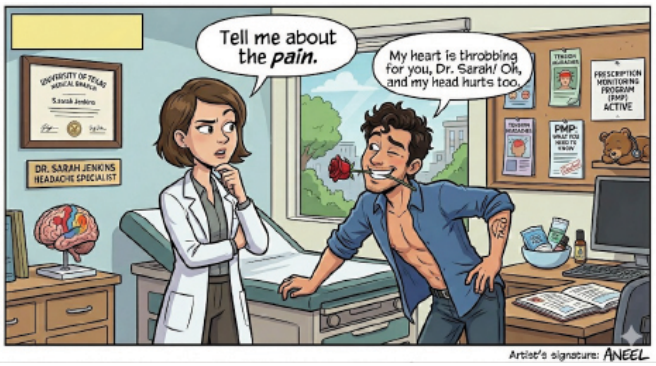
I use AI to help prepare slides for lectures. Gemini can make cartoons for you. I give specific instructions and then refine it. Here are a few I've done for an upcoming lecture. Not sure if they are funny but they're fun to create. Try it for your next lecture. Or maybe, cartoons aren't a good idea for lectures (or Broca's).

Patients without borders



Superdocs: the physician supermarket

Patients without boundaries. Love in all the wrong places



The great escape

HEADACHE WITH A THUNDERCLAP ONSET

This is a 59 year old woman with a history of occasional severe migraines without aura for 25 years lasting up to 24 hours with rizatriptan. Past medical history of hyperlipidemia on rosuvastin. She reported a 3 week history of a similar headache but with a thunderclap onset of 10/10 which ended 6 days before presentation. Neurologic exam was normal. She went to a TMC ED and was given a "migraine cocktail" without relief. A scan of the brain was not done.

What is a thunderclap headache? What are the possible causes? How often do migraines have a thunderclap onset?

Day and Raskin coined the term, thunderclap headache, in a 1986 case report of a 42 year old woman with a sudden, severe headache "like a thunderclap" found to be associated with an unruptured intracranial aneurysm. ICHD-3 defines a thunderclap headache as a severe headache of sudden onset that reaches maximum intensity in less than 1 minute. [3]

This patient should have had imaging in the ED because she met the Ottawa SAH rule which requires investigation if one or more of the following findings are present: symptoms of neck pain or stiffness; age 40 years or older; witnessed loss of consciousness; onset during exertion, thunderclap headache; and limited neck flexion on exam. [4]

Thunderclap headache has a long list of possible causes including the following: vascular-hemorrhagic (aneurysmal SAH, sentinel leak, intracerebral hemorrhage, retroclival hematoma); vascular-non-hemorrhagic (RCVS, cerebral venous sinus thrombosis, cervical artery dissection, ischemic stroke, acute hypertensive crisis, PRES); endocrine (pituitary apoplexy); CSF-related (spontaneous intracranial hypotension, third ventricle colloid cyst); infectious (meningitis, encephalitis); and primary/idiopathic (primary thunderclap headache, primary cough headache, primary sexual headache, primary exertional headache, and new daily persistent headache). [5,6,7]

In a multi-center ED study of 4536 patients with headaches, 644 presented with a thunderclap headache. [8] Serious pathology was found in 10.9% vs 6.6% without a thunderclap onset. 87.7% had a non-serious or benign cause.

Perhaps 75% of patients with SAH have a thunderclap headache. In a study of 106 neurologically intact patients with spontaneous SAH who all received analgesics, the shortest time to complete headache resolution was 10 hours with headache resolution in 5% within 24 hours and 10% at 48 hours after the ictus. [9] In a retrospective study of 66 people who had an aneurysmal SAH, up to 47% had persistent headaches at a mean time of 3.1 years with the tension-type more common than migraine-like phenotype. [10]

Was testing indicated in this case? Yes. ESR was 2. CRP was <.3. MRI of the brain with and without contrast showed a tiny chronic microhemorrhage (or cerebral microbleed, CMB) in the right frontal lobe. MRA of the head and neck and MRV of the head were negative. The MRI was done with and without contrast to help exclude spontaneous intracranial hypotension since diffuse pachymeningeal thickening is present in about 75% of cases. [11]

What is the significance of this CMB? What is the management? Can triptans be used in the presence of this CMB?

A CMB is a small less than 10 mm (usually 2-5 mm) deposit of hemosiderin typically due to leakage from capillaries or arterioles detected on MRI SWI or GRE as small, round, dark voids. Basal ganglia, thalamus, and brainstem CMBs are typically caused by hypertensive vasculopathy while lobar, cortical, or cortical-subcortical CMBs are a hallmark of cerebral amyloid angiopathy. [12]

CMBs are present in up to 5-21% of the general population, 30-40% of patients with ischemic stroke, and 60-68% of patients with primary intracerebral hemorrhage. [13] CMBs increase with older age and are present in 6.5% in ages 45-50, 18% of people ages 60-69 years and 38% of those over 80 years.

In this case, the CMB is an incidental finding associated with age-related small vessel disease in a non-hypertensive person. Migraine is associated with infratentorial CMBs at older ages. [14] In most cases, CMBs should not prevent the use of antithrombotics or anticoagulants for stroke prevention when otherwise indicated. [15] A single incidental chronic lobar CMB is not a stated contraindication to triptan use based upon my literature review. A gepant could certainly be tried if there is any concern.

What was the cause of the headache in this case?

Primary thunderclap headache is a consideration but her headache exceeded the 10 day ICHD-3 duration criterion

Reversible cerebral vasoconstriction is possible even with a normal MRA because in RCVS, vasoconstriction peaks at 2-3 weeks and may be resolving by 3 weeks. [16] In a prospective study, only 6% had a single thunderclap headache. Patients have a mean of 4 attacks during weeks 1-4. [17] Our case had no triggering factors which include orgasm, physical exertion, an acute stressful or emotional situation, pregnancy, environmental exposure (including high altitude and cold water exposure), and a variety of vasoconstrictive agents and other medications (such as triptans, phenypropranolamine, diet pills, SSRIs and SNRIs, and cocaine.

Pioneering neurologist, C. Miller Fisher, used the term "crash migraine" to describe acute, high-intensity headaches similar to those caused by saccular aneurysm rupture but with normal LP and angiography. [18] Is "crash migraine" real? Most authorities attribute "crash migraine" to primary thunderclap headache or RCVS. However, if the headache is similar to their prior migraine and testing is negative, why couldn't migraine present with a thunderclap onset. Evans and Turner have reported primary NDPH with a thunderclap onset in 3.6%.[7] It is also possible that these patients had undetected RCVS which was the trigger for NPDH. Bottom line: don't assume a thunderclap headache is migraine. Do the imaging and LP as indicated!

How common is status migrainosus (SM)? What is the duration? What treatments are effective for migraine status?

If our case's headache did not have a thunderclap onset, we would classify it as status migrainosus.

A retrospective population based incidence study in Olmsted County, Minnesota found the following: median age 35; 88.6% female; 36.3% with chronic migraine; 35.7% with a history of aura; incidence rate 26.6 per 100,000 with peak incidence between ages 40-49 years; median attack duration 5 days; most frequent triggers were stress and too much or too little sleep; and recurrence in 14.8% at a median of 58 days following the initial attack. [19]

In a retrospective cohort study at the Mayo headache center, the lifetime prevalence of SM among migraine patients was 43.9% with a 1 year incident of 24.2%. [20] The median attack duration was 10 days with a range of 3-330 days) with a median severity of 8/10. The patients tended to have chronic migraine with high rates of aura.

In a narrative review of SM, Robblee et al proposed that "A revised definition of SM should move beyond a rigid 72-h threshold and give greater weight to functional impairment, treatment response, and more explicit definitions of attack duration that clarify how non-headache phases are handled." [21]

Numerous treatments are tried for SM including NSAIS, neuroleptics, steroids, DHE, triptans, gepants, perhaps IV eptinezumab, IV valproic acid, and pericranial nerve blocks [22] with modest efficacy and an absence of high-quality RCTs. In an observational study of 54 patients, the success rate for rendering patients pain free within 24 hours and maintaining pain-free status for 48 hours was as follows: dexamethasone, 7/29 (24%) for nerve blocks, 1/9 (11%) for ketorolac and 1/9 (11%) for naratriptan. [23] We all know this from the many patients we see with persistent SM after getting "migraine cocktails" in the ER and our trials of outpatient medications and nerve blocks.

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By Dr. Kim Monday, TNS Legislative Chair, and Tom Holloway, TNS Lobbyist

Since the end of the 89th Texas Legislature, our legislative team has shifted focus from passing legislation to making it real — securing seats at tables where rules will be written, helping hospitals prepare for new grant programs, pressing state agencies to act on overdue reforms, and laying the groundwork for our agenda in the 90th Texas Legislature.

DPRIT: Litigation & Appointments

In November, Texas voters overwhelmingly approved a $3 billion appropriation to set up the Dementia Prevention and Research Fund of Texas (DPRIT). While this funding was initially expected to be transferred from state general revenue to the new DPRIT account on January 1, a constitutional challenge filed in Travis County District Court continues to delay implementation.

Three pro-se plaintiffs sued the Secretary of State, alleging that voting machines in 251 of Texas's 254 counties were not properly certified before the Constitutional Amendment Election on November 4, 2025. While a Travis County judge ruled against the plaintiffs in February, the case remains active pending appeal and funding remains paused until the case is finally adjudicated.

While the delay is unfortunate, it has not changed TNS's preparation. Under the enabling statute of SB 5, the Governor, Lieutenant Governor, and Speaker are each responsible for appointing members to the DPRIT Oversight Committee, which will set research priorities and establish grant criteria. TNS nominated Dr. Louise D. McCullough, Huffington Chair of Neurology at UTHealth Houston, as the strongest candidate to represent neurology on the DPRIT Board.

Dr. McCullough's board application has been submitted to both Speaker Dustin Burrows and Lt. Governor Dan Patrick for review, and TNS remains engaged with both offices regarding the importance of strong, day-one representation from Texas neurologists as DPRIT begins its mission.

Mobile Stroke Unit Grant Program: Applications & Awards

Perhaps TNS' most significant victory from the 89th Texas Legislature was the establishment of the Mobile Stroke Unit Grant Program; a $5 million appropriation to support the acquisition, deployment, and operation of mobile stroke units at hospitals across the state.

In January, HHSC formally released its application for the Mobile Stroke Unit grant program, with several health systems submitting responses. As of this writing, HHSC has not yet announced its award decisions, though agency staff have indicated that initial grant selections are imminent and confirmed that a second round of grant applications is planned for fiscal year 2027.

TNS continues to support health systems as they prepare applications for the next cycle, including UTMB-Galveston, which is preparing an application through its existing stroke program, and Covenant Medical Center in Lubbock, which is positioning to expand mobile stroke care into West Texas. As deployment data comes in from first-cycle awardees, TNS will continue to build the case for expanded mobile stroke services funding in the 90th Legislature.

DSHS Sunset Review & the Medical Advisory Board

This interim, the Texas Sunset Commission is undertaking its first comprehensive review of the Texas Department of State Health Services in more than a decade, providing TNS with a unique opportunity to address several long-standing issues with the agency's Medical Advisory Board.

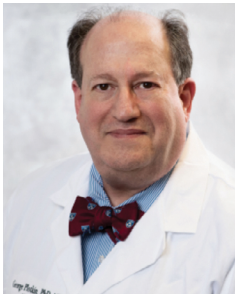
The Medical Advisory Board (MAB) is a panel of physicians that advise the Department of State Health Services (which subsequently advises the Texas Department of Public Safety) on driver licensing for Texans whose medical conditions could affect their ability to drive safely. The bulk of the cases that reach the MAB sit squarely within neurology: seizure disorders, post-stroke deficits, cognitive decline, Parkinson's disease, and traumatic brain injury. The MAB's Guide for Determining Driver Limitations effectively serves as the clinical rulebook for whether and under what conditions these patients can keep safely retain their licenses.

Unfortunately, DSHS has not approved an update to the MAB's clinical guidelines since 2014, citing cost and compliance concerns that would arise from an elevated review process. In practical terms, this means that over a decade of advances in seizure management, stroke recovery, and cognitive screening that have not made their way into the framework governing Texans' driving privileges. Needless to say, the failure to update or appropriately adhere to these guidelines represents a serious public safety concern for all drivers on Texas roads.

On March 10, TNS members Dr. Gerlyn Friesenhahn and Dr. Azreena Thomas met with senior staff at the Sunset Advisory Commission to outline the structural and organizational issues within the MAB. TNS continues to work with Sunset Commission staff to mandate the adoption of updated clinical driving guidelines and remove the administrative barriers that have stalled the board from acting on physician recommendations in the past.

The Sunset Commission is expected to publish its official report on DSHS this October, followed by a public hearing where physician representatives from TNS will provide testimony related to MAB composition, the public safety risks posed by refusing to update the board's clinical guidelines, and the administrative barriers preventing effective collaboration between the MAB, DSHS, and DPS. A decision meeting follows a month later, and lawmakers will take up Sunset's recommendations as a bill when the Legislature convenes in January 2027.

When Faces Warp but Vision Is Normal: A Cortical Cause of Visual Distortion



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Clinical Vignette

A 54 year old woman presented with a three day history of distorted faces. When speaking to her husband, his left eye appeared higher than the right and his mouth seemed pulled sideways. She recognized him immediately and denied diplopia, blurry vision, or loss of vision. Objects in the room appeared normal.

Ophthalmologic examination was unremarkable. Visual acuity and confrontation visual fields were intact. Neurologic examination revealed no motor or sensory deficits. MRI of the brain demonstrated a small lesion in the right occipito temporal region.

Discussion

Distortion of facial features with preserved identity recognition is most consistent with prosopometamorphopsia, a rare cortical visual perceptual disorder.

Patients often describe faces that appear stretched, tilted, shifted, or asymmetric. The key detail is that they still recognize the person in front of them. They know who they are seeing, but something about the arrangement of features feels wrong. Objects in the environment are frequently unaffected.

This helps distinguish prosopometamorphopsia from prosopagnosia, where identity recognition is impaired, and from visual hallucinations, where faces are seen that are not actually present. In this condition, perception is real but the structure of the face is distorted.

The syndrome typically localizes to the ventral visual stream, particularly the occipito temporal cortex and fusiform gyrus. These regions integrate facial features into a coherent whole. When that integration is disrupted, the result is distortion rather than loss of recognition.

Right hemisphere lesions are reported more often, reflecting the dominant role of the right hemisphere in holistic facial processing. The right sided lesion in this patient fits with that functional lateralization. Reported causes include focal ischemia, seizures, tumors, demyelinating disease, and inflammatory processes.

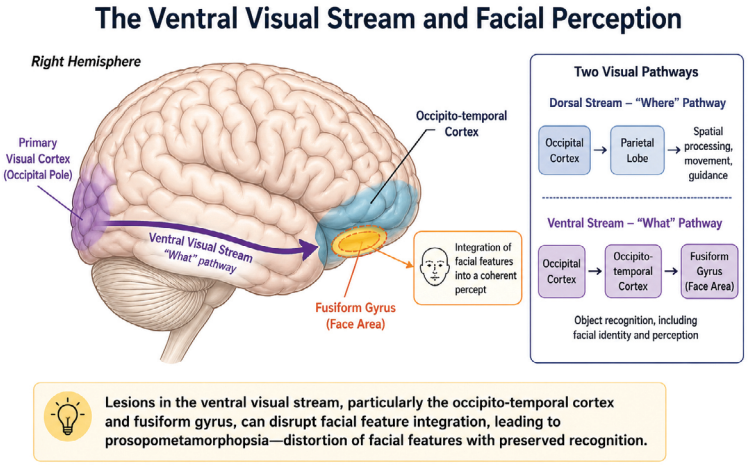
Because visual acuity and ophthalmologic examinations are usually normal, patients are often first referred for eye evaluation or attributed to migraine aura or psychiatric causes. Taking the time to carefully characterize the complaint can prevent unnecessary workup and guide localization early.

When evaluating abnormal facial perception, it helps to clarify whether identity is intact, whether objects are also distorted, whether there is any visual field loss, and whether acuity is preserved. Asking whether specific features are displaced or whether the entire face appears altered can further refine localization. These details often point to the ventral visual stream before imaging confirms it.

Teaching Pearl

Distortion with preserved identity points to dysfunction of facial feature integration rather than loss of recognition.

In a patient with normal acuity and intact visual fields, distorted faces should prompt consideration of a ventral stream cortical lesion rather than primary ophthalmologic disease. Recognizing this pattern allows timely neuroimaging and avoids unnecessary diagnostic detours.



Neuropathic Itching

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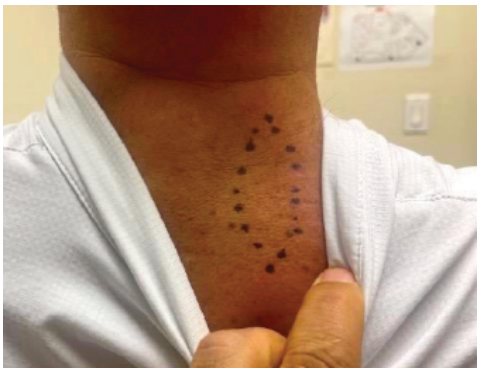
A 55 year old diabetic man presented with severe itching of a small oval area of about 3cm diameter located in the posterior cervical and upper thoracic region, that started 9 months earlier with no triggers. Video 1 No skin rash, neck pain, or weakness. The itching is intermittent and is associated with tingling and numbness in that area. Cervical spines MRI showed age appropriate DJD.



The most likely diagnosis is:

A. Herpes zoster infection.

B. Migratory neuritis of Wartenberg (MNW)

C. Notalgia paraesthetica

D. C8 radiculopathy

E. A skin disorder

The answer is C.

Notalgia paresthetica (NP) is a chronic sensory neuropathy characterized by localized pruritus and dysesthesia, most commonly affecting the upper back (1). Recent studies support a multifactorial pathogenesis for NP, with compression or irritation of the dorsal rami of thoracic spinal nerves playing a central role. diabetes is a risk factor. Imaging and electromyography have shown variable utility in diagnosis, while histological findings often lack specificity. The distribution is atypical for herpes zoster or C8 radiculopathy. One fixed lesion is not typical for MNW.

Unlike conventional itch, neuropathic itch develops in normal skin from excess peripheral firing or dampened central inhibition of itch pathway neurons (2). Neuropathic itch is a symptom of the same central and peripheral nervous system disorders that cause neuropathic pain. It may respond to Benadryl lotion or tablets or Lidoderm patches 5%.

Citations

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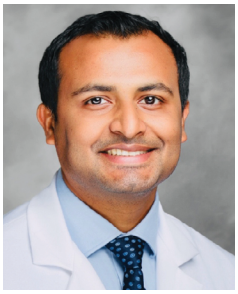
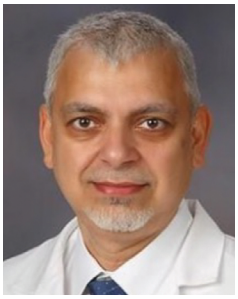
From Scope to Stroke: Cerebral Air Embolism Presenting as Acute Ischemic Stroke After Endoscopy





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Introduction

Cerebral air embolism (CAE) is a rare but potentially catastrophic cause of acute neurological deterioration. It occurs when gas bubbles or air enter the vascular system, either the arterial or venous, and obstruct blood flow within the cerebral vasculature, leading to ischemia and subsequent secondary inflammatory injury. (1) Although CAE is commonly associated with invasive vascular procedures, including head and neck surgeries and central line placement, CAE has increasingly been reported in gastrointestinal interventions such as endoscopy, particularly in the setting of mucosal injury and air insufflation. (2) Recognition of symptoms of CAE is often delayed due to its rarity and the unique and transient nature of initial imaging findings. Early diagnosis and management are critical, and prompt intervention, including hyperbaric oxygen therapy (HBOT), can significantly improve outcomes when available.

This case report describes an acute ischemic stroke secondary to cerebral air embolism following esophagogastroduodenoscopy (EGD), followed by a focused review of the literature.

Case description:

A 65-year-old right-handed African-American female with a history of hypertension, hyperlipidemia, sinoatrial exit block, bipolar disorder, hypothyroidism, Chronic Kidney disease (CKD) stage III, Gastroesophageal reflux disease (GERD) underwent esophagogastroduodenoscopy under anesthesia for concerns of dysphagia. Immediately upon waking up from anesthesia, she was found to have acute neurological deficits including left-sided weakness, left hemibody numbness, left hemispheric neglect and right gaze deviation with an NIHSS score of 18. On examination, she was awake and oriented, had fluent speech with intact comprehension. Cranial nerve examination revealed right gaze preference with inability to cross the midline, left facial droop and left-sided severe weakness with Medical Research Council motor scale of 1 and 5 on the right side. Sensory examination showed left-sided deficit with neglect to double simultaneous stimulation.

A noncontrast CT scan of the head in the ED revealed no acute infarction but demonstrated scattered foci of intravascular gas, including air in the straight sinus and along the right frontal and parietal cortical surfaces. CT angiography of head and neck showed no large vessel occlusion but did confirm multiple areas of intravascular gas cortically. MRI brain demonstrated right frontoparietal cortical restriction diffusion without any corresponding FLAIR changes consistent with diffusion-FLAIR mismatch, suggestive of potential reversible ischemia as well as pneumocephalus. As the patient's presentation was within the thrombolytic window timeframe, Tenecteplase(TNK) was administered approximately within 3 hours of the symptom onset following risks versus benefit discussion with the patient and the family. The patient was subsequently transferred to a tertiary hospital, where she was admitted to stroke unit and subsequently underwent two sessions of hyperbaric oxygen treatment(HBOT). Her hyperbaric treatment course was complicated by bilateral optic barotrauma. Her hospital course was complicated by dysphagia requiring percutaneous endoscopic gastrostomy tube/PEG, supraventricular tachycardia, hypotension and metabolic derangement. Stroke workup revealed normal ejection fraction without any evidence of patent foramen ovale or intracardiac shunt in TTE. LDL 113 mg/dl(reference range:<100 mg/dl), HgbA1C 5.4%, TSH 0.97 µIU/mL (reference range 0.34-5.66)

CT chest abdomen and pelvis revealed no evidence of perforated viscus, but increased gas within the stomach, small bowel and transverse colon. Endoscopic findings included severe gastroesophageal junction stenosis with diffuse esophageal candidiasis on biopsy results, suggesting mucosal disruption of the esophageal gastric lumen as a potential portal of air injury in the setting of significant air insufflation during the procedure. Despite complications and significant stroke related morbidities, the patient demonstrated partial neurological recovery and was discharged to a skilled nursing facility for subsequent rehabilitation.

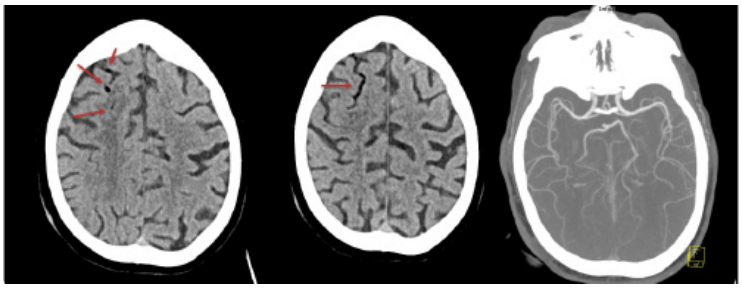

Figure 1: CT head and CT angiogram brain. Arrow demonstrating scattered foci of intravascular air along the periphery of the right frontal lobe.

Continued: From Scope to Stroke: Cerebral Air Embolism Presenting as Acute Ischemic Stroke After Endoscopy

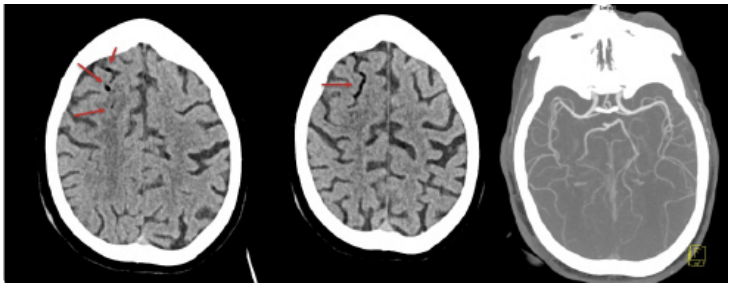

Figure 3: MRI brain demonstrating right frontoparietal cortical diffusion restriction and pneumocephalus along right frontoparietal convexity.

Discussion:

Cerebral air embolism occurs when gas/air enters either the arterial or the venous circulation and travels along the anterior or the posterior cerebral vasculature into the brain. (1) Arterial emboli directly occlude the cerebral vessels especially at the capillary junction, and venous air can travel to the brain via retrograde flow or paradoxical embolism through intracardiac shunt or pulmonary shunts.(2) Even small volumes of the air can cause significant neurological deterioration by mechanical obstruction, subsequently leading to endothelial damage and activation of the inflammatory anticoagulation cascade triggering formation of microthrombi and subsequent infarction.(3)

CAE commonly results from iatrogenic causes including central venous catheter placement/manipulation, surgical procedures specially involving the neck or head in a sitting position, cardiopulmonary bypass, hemodialysis, or endoscopic, lung overinflation, decompression sickness or barotrauma with mechanical ventilation. (3) Predisposing factors could include low venous pressure, upright positioning, right-to-left shunt (e.g., patent foramina ovale) which facilitates the air entry or paradoxical embolism into the arterial system.

The unique feature of the air emboli in the venous circulation is the ability to travel retrograde, especially if they are in jugular veins, due to the lower specific gravity/density compared to the blood, causing cerebral involvement even without arterial access. (4)

Although rare, CAE is a recognized complication of endoscopic procedure especially upper GI endoscopy. (2)(3)(5) The risk increases if there is coexisting mucosal injury (example after biopsy, dilatation, or esophageal infection such as candidiasis in this case) and associated high-pressure insufflation. Air under pressure may enter the circulation through the disrupted mucosa subsequently into the venous or the portal system, or directly into arterial circulation in the presence of vascular injury or shunting. (5) This case in particular highlights the multiple risk factors leading to cerebral air embolism including esophageal stenosis, procedure or insufflation and mucosal injury secondary to candidiasis.

The neurological manifestation of cerebral air embolism depends on the location and extent or severity of the embolization. Symptoms include but are not limited to hemiparesis, neglect, aphasia, seizures, altered mentation, etc. Sometimes the imaging finding may be transient, and absence of the intravascular air does not completely exclude the diagnosis, however, as these imaging presentations are rare, they can be easily missed. (6)

CT imaging is often the first-line modality in the diagnosis and may demonstrate hypodense intravascular air within the cerebral vasculature. Precordial Doppler is very sensitive for intraoperative monitoring which helps to detect any tiny amounts of air and is gold standard surveillance in very high-risk procedures. (7) MRI with DWI imaging is more sensitive for detection of ischemic injury, however clinical suspicion remains paramount especially in the setting of periprocedural setting and where MRI brain is not readily available.

Initial management focuses on preventing further entry of the air into the cerebral vasculature and reducing burden of the cerebral air emboli. Administration of 100% oxygen, laying the patient in left lateral decubitus along with Trendelenburg positioning (Durant maneuver) along with maintenance of hemodynamic stability is cornerstone for the initial management. (8) Hyperbaric oxygen therapy, wherever available, is a definitive treatment. Hyperbaric oxygen treatment helps in cerebral air embolism with multiple mechanism including reduction in the size of the air bubbles (Boyle's law), increasing delivery of the oxygen to the ischemic tissue, increasing the nitrogen reabsorption from the vascular tissue and also reduction in the cerebral inflammation and subsequent cerebral edema. Intervention with hyperbaric oxygen treatment ideally within 6 hours is associated with improved clinical and neurological outcomes. (9)

Thrombolytics such as Tenecteplase or tPA are not the standard of the treatment for cerebral air embolism, as the stroke etiology is likely due to mechanical obstruction of the blood vessels rather than a thrombotic mechanism. However mechanical obstruction with air emboli, will subsequently lead to endothelial injury leading to activation of the coagulation cascade and microthrombi formation. Microhemorrhages due to capillary damage and reperfusion injury has been documented too. (3) In cases where cerebral air embolism presents as acute ischemic stroke, and the diagnosis is uncertain, thrombolysis may be administered (as occurred in this case, which did not lead to complication including cerebral bleeding). Various emerging therapies such as mechanical aspiration thrombectomy, adjunctive lidocaine infusion have been reported in various case reports/series, however lack of robust randomized control trials. (10) There is no established role of antiplatelet treatment for cerebral air embolism. However, they can be used in the setting of previous stroke for secondary stroke prevention or other cardiac indication. Heparin has been used in some animal studies but is not recommended due to hemorrhagic transformation of the infarcted tissue. (11) The use of lidocaine as a potential neuro-

Continued: From Scope to Stroke: Cerebral Air Embolism Presenting as Acute Ischemic Stroke After Endoscopy

protective effect in cerebral air embolism, as evidence from animal study and limited human data, is not a standard of care. Possible mechanisms of neuroprotection could be due to suppression of ischemic depolarization, decrease in glutamate cytotoxicity or decrease intracellular air accumulation, however human evidence of neuroprotective effect is limited and can only be used as an adjunct in treatment, but clinical evidence is insufficient. (12)

Historically, cerebral air embolism carries very high mortality rates as high as 80 to 100%, however with the use of hyperbaric oxygen treatment, in available centers, mortality has been significantly reduced with many survivors experiencing favorable neurological outcomes. (13)

Conclusion

Cerebral air embolism (CAE) is a severe condition that can cause acute ischemic stroke and should be considered in patients presenting with acute neurological deficits following invasive procedures involving blood vessels or air insufflation, including endoscopy. Recognition of symptoms is crucial, as early imaging, including CT, may be inconclusive, and delays in treatment can worsen long-term outcomes. Immediate supportive measures and prompt initiation of hyperbaric oxygen therapy remain the cornerstone of management. This case underscores the importance of maintaining a high index of suspicion for cerebral air embolism in the periprocedural setting and highlights the need for preventive strategies during endoscopic interventions to avoid this catastrophic complication. Limited evidence exists regarding the role of thrombolysis, antiplatelet therapy, or lidocaine in the treatment of stroke following cerebral air embolism.

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Broca's Area

CASE STUDIES

Recurrent Syncopal Episodes in a Patient with Idiopathic Intracranial Hypertension

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Disclosures:

The authors have no relevant financial or non-financial conflicts of interest to disclose

Introduction

Idiopathic intracranial hypertension (IIH) is characterized by elevated intracranial pressure (ICP) without an identifiable structural cause. It most commonly affects obese women of childbearing age and typically presents with headache, transient visual obscurations, diplopia, and pulse-synchronous tinnitus. Medication exposures, particularly retinoids and tetracycline-class antibiotics, are recognized risk factors. The most notable physical examination finding is papilledema. Diagnosis is established through brain magnetic resonance imaging (MRI) to exclude mass lesions or hydrocephalus, followed by lumbar puncture confirming an elevated opening pressure with normal CSF composition. First-line treatment includes weight loss and carbonic anhydrase inhibition, primarily acetazolamide. In cases with progressive or vision-threatening disease, surgical interventions such as dural venous sinus stenting, CSF shunting, or optic nerve sheath fenestration may be required.[1]

Although IIH classically presents with visual and headache-related symptoms, less common manifestations have been reported. In a retrospective case–control study of 54 IIH patients by Morden et al. (2021), headache and visual disturbance were present in 96.2% of cases, while tinnitus (25.9%) and dizziness or syncope (20.0%) were less frequently observed.2 Syncope is not traditionally considered a key feature of IIH, and evidence supporting an association is limited but growing.

A 2023 case report by De Simone et al. described a 57-year-old woman with daily orthostatic syncope and IIH-related headaches; lumbar puncture reduced both symptoms, suggesting a potential mechanistic link between elevated ICP and transient cerebral hypoperfusion.[3] The authors proposed that reduced intracranial compliance and dynamic dural venous sinus collapsibility might predispose patients to ICP fluctuations sufficient to impair cerebral perfusion and trigger syncope. Similarly, a 2024 report by Flores et al. detailed a 26-year-old woman with IIH symptoms and posture-related syncope; while her headaches and visual disturbances improved with acetazolamide and lumbar puncture, the effect on syncope was unclear.[4]

Although cognitive dysfunction, cranial neuropathies, olfactory disturbances, and syncope have been documented in IIH, these symptoms rarely serve as the presenting or predominant complaint. Here, we describe an unusual case in which recurrent syncope was the patient's primary symptomatic burden and improved significantly following targeted IIH treatment.

Case

The patient is a 29 year-old female with a past medical history of truncus arteriosus s/p surgical repair, OSA, and obesity who presented with recurrent syncopal episodes that occuredr three times per week for the last seven years. She experiences a prodromal headache followed by loss of consciousness for 1-2 minutes with a rapid return to baseline afterwards. She experiences daily headaches with photophobia, phonophobia, and blurry vision, with at least two severe headaches per month. Trials of propranolol and topiramate provided minimal relief. She had been seen by a cardiologist prior who ruled out cardiovascular etiology.

Her history was notable for a diagnosis of IIH at age 12 based on elevated opening pressure during a lumbar puncture; her symptoms had resolved with acetazolamide at that time and it was later discontinued. An MRI brain and cervical spine performed one month before presentation revealed at least three punctate nonspecific T2 hyperintense lesions in the juxtacortical and corpus callosal white matter, along with mild cervical degenerative changes. Idiopathic intracranial hypertension was considered at the time and differentials included migraine headaches, vasovagal syncope, and psychogenic syncope.

Notable physical exam findings include weight of 276 lbs with BMI of 50.48 kg/m^2. Visual acuity was grossly normal to confrontation without findings of papilledema on fundoscopic exam. Otherwise features of her neurological exam were unremarkable.

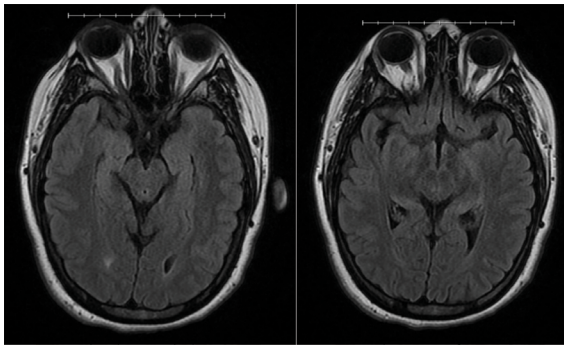

Figure 1 - FLAIR sequences showing tortuosity of bilateral optic nerves.

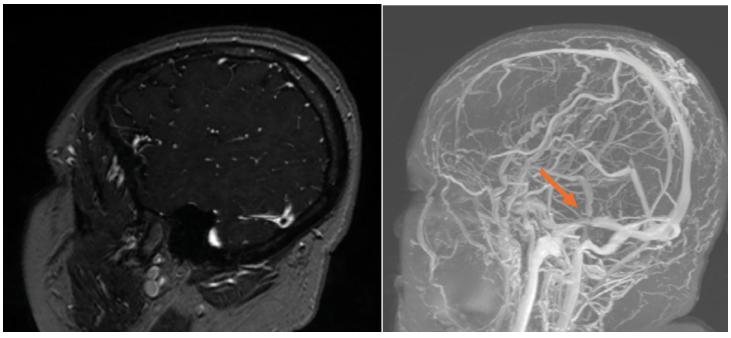

Figure 2 - MRV postcontrast sequences demonstrating moderate stenosis of the distal right transverse sinus.

Continued: Recurrent Syncopal Episodes in a Patient with Idiopathic Intracranial Hypertension

A repeat MRI brain with contrast demonstrated scattered supratentorial FLAIR hyperintensities in a nonspecific pattern compatible with microvascular ischemic change versus demyelinating disease, without enhancement. On close inspection, mild tortuosity of the bilateral optic nerves was notable (Figure 1). MRV showed focal stenosis of the distal right dominant transverse sinus and distal nondominant left transverse sinus (Figure 2).

Lumbar puncture was performed and resulted in an opening pressure of 32 cm H2O. There were 0 nucleated cells, protein was 17, and oligoclonal bands were absent. A follow up lumbar puncture 1.5 mo later resulted in an opening pressure of 25 cm H2O. The results of these studies support the diagnosis of idiopathic intracranial hypertension.

Management

The patient was counseled on weight reduction as a key disease-modifying strategy and was later restarted on acetazolamide 250mg once daily. For migraine prophylaxis, she initiated monthly fremanezumab (Ajovy). By 6 months after her initial evaluation, she had achieved a 40-lb weight loss through dietary modification and exercise alone. Her headaches significantly improved, and syncopal episodes decreased from three per week to approximately once episode over the span of 6 months.

Discussion

This case emphasizes syncope as an uncommon but clinically relevant manifestation of idiopathic intracranial hypertension. Although headache and visual disturbances remain the hallmark features of IIH, the patient's recurrent syncopal episodes, occurring multiple times per week and reliably preceded by acute, severe headaches, suggest a physiologic connection between transient rises in intracranial pressure and loss of consciousness. Her substantial improvement following multiple lumbar punctures, weight reduction and low dose acetazolamide therapy further supports the role of ICP elevation in triggering these events.

Only a limited number of published cases have described syncope in association with IIH, and the underlying pathophysiology remains incompletely understood. However, several plausible mechanisms have been proposed (3).

- **Reduced intracranial compliance:** Patients with IIH may have diminished capacity to buffer small increases in intracranial volume. Even modest fluctuations in CSF pressure could therefore transiently reduce cerebral perfusion pressure below the threshold for maintaining consciousness.
- **Dynamic dural venous sinus collapsibility:** Venous sinus stenosis or pressure-sensitive collapse, commonly observed in IIH, may cause episodic impairment of cerebral venous outflow. This can acutely elevate ICP, reduce cerebral perfusion, and potentially provoke syncope -- particularly in situations involving postural change or Valsalva-like maneuvers.
- Autonomic dysregulation secondary to chronic ICP elevation: Chronic pressure-related distortion of brainstem autonomic centers may impair cardiovascular reflexes, lowering the threshold for vasovagal or orthostatic syncope. This mechanism may coexist with impaired cerebral perfusion and amplify susceptibility to transient loss of consciousness. Pain from the preceding headache with underlying impairment in baroreflex response may be a trigger for vasovagal syncope.[5,6]

The patient's clinical course aligns with the limited literature noting that intervention aimed at reducing ICP, such as lumbar puncture or acetazolamide, can ameliorate both headache and syncopal episodes. Her marked reduction in event frequency following weight loss also reinforces the well-established relationship between obesity, ICP elevation, and IIH disease activity. Overall, this case expands existing evidence that syncope, while uncommon, may represent an under-recognized manifestation of IIH, particularly in patients who report brief, headache-associated episodes with rapid return to baseline. Increased clinical awareness of this presentation may prevent unnecessary cardiac or psychiatric workups and prompt earlier evaluation of ICP-related etiologies.

Further investigation into the hemodynamic and autonomic changes associated with IIH, including real-time ICP monitoring, venous sinus flow studies, and autonomic testing, may help clarify the mechanisms linking elevated ICP to transient global cerebral hypoperfusion and syncope. Improved understanding of these processes could refine diagnostic criteria, guide targeted treatment, and identify patients at increased risk for this atypical presentation.

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Atypical Presentation of Optic Neuritis in a Patient with Active Hepatitis C Infection: A Case Report

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Abstract

Introduction:

Optic neuritis is associated with demyelinating diseases such as multiple sclerosis (MS), but atypical presentations, particularly bilateral optic nerve involvement or coexisting systemic symptoms, necessitate broader diagnostic consideration.

Case presentation:

This case report describes a 50-year-old male with untreated, active hepatitis C virus (HCV) infection who presented with bilateral optic neuritis. Cerebrospinal fluid (CSF) analysis revealed elevated myelin basic protein, positive oligoclonal bands, and an elevated immunoglobulin G index and an HCV viral load of 9.3 million International Unit/milliliter (IU/ml). Magnetic resonance imaging (MRI) of the orbit showed bilateral optic neuritis with perineuritis. The patient had no history of interferon therapy and lacked markers of cryoglobulinemia, making this a rare case of optic neuritis possibly associated or seen with chronic Hepatitis C viral infection. The patient was treated with intravenous immunoglobulin (IVIG) without corticosteroids with some improvement in the vision.

Conclusion:

This case adds to the expanding spectrum of multiple sclerosis (MS) mimics and underscores diagnostic challenges associated with atypical optic neuritis presentations, particularly in the setting of coexisting systemic viral infections.

1. Introduction

Optic neuritis (ON) is defined as an inflammatory disease of the optic nerve and represents the most common cause of acute optic neuropathy in young adults. It is classically associated with demyelinating disorders such as multiple sclerosis (MS), presenting with acute, unilateral, painful vision loss, red color desaturation, and a relative afferent pupillary defect. However, bilateral involvement of optic nerves, atypical age of onset, or systemic features should prompt evaluation for alternative etiologies, including autoimmune conditions (e.g., Neuromyelitis Optica Spectrum Disorder (NMOSD), Myelin Oligodendrocyte Glycoprotein-IgG Associated Disorder (MOGAD), sarcoidosis, Sjögren's syndrome, systemic lupus erythematosus (SLE)) and infectious or para-infectious processes [1].

ON has been linked to various viral infections, including hepatitis C virus (HCV). Neurological complications in chronic HCV infection are most often peripheral while central nervous system (CNS) involvement, including optic neuropathy, is rare [2]. HCV-associated ON has been reported sporadically in active or treated infection, often linked to interferon-alpha therapy, which may exacerbate autoimmune processes [3]. Diagnosing HCV-associated ON is challenging due to its rarity and nonspecific clinical/radiological features. Thorough evaluation for cryoglobulins, complement, rheumatoid factor, and vasculitis markers is essential, alongside exclusion of other neurologic autoimmune or inflammatory conditions [4].

This case report describes an atypical bilateral ON in a patient with active, untreated HCV infection, illustrating the potential overlap between infectious and demyelinating processes and emphasizing the need to consider broader etiologies in atypical ON presentations.

2. Case Report

A 50-year-old right-handed dominant Caucasian male with past medical history of hypertension, untreated Hepatitis C without systemic symptoms and polycythemia vera presented with a three-week history of progressive bilateral vision loss—more severe in the right eye (with altitudinal field defect) and inferior filed involvement in the left eye. He also had intermittent right-sided retro-orbital pressure-like headaches, nausea, gait imbalance, two brief episodes of tinnitus and unintentional 20-pound weight loss. He reported 10–12 sexual partners over the past decade.

Neuro-ophthalmologic examination revealed grade 4 optic disc edema in the right eye and grade 2 in the left, with associated macular edema and findings consistent with intermediate/posterior uveitis. There was no relative afferent pupillary defect (RAPD) present on exam. Visual acuity was "counting fingers" in the right and 20/50 in the left eye. Neurological exam demonstrated hyperreflexia (3+) in the upper and lower extremities with normal Achilles reflexes, impaired light touch and pinprick sensation in a glove-and-stocking distribution (likely chronic from tuberculosis prophylaxis with isoniazid or his untreated hepatitis C infection), preserved vibration and proprioception were preserved, and negative Romberg sign.

Initial laboratory investigations, including Complete Blood Count (CBC), Magnesium and Comprehensive Metabolic Panel were largely unremarkable, apart from mildly elevated liver enzymes. An infectious disease panel confirmed HCV antibody positivity, with viral load of 9.3 million IU/mL and genotype 3A. Serology for EBV and CMV Immunoglobulin G (IgG) positivity with negative Immunoglobulin M (IgM) (consistent with prior exposure rather

Continued: Atypical Presentation of Optic Neuritis in a Patient with Active Hepatitis C Infection: A Case Report

than acute infection). Hepatitis B surface antigen (HBsAg), HSV 1/2, Toxoplasma, Bartonella, rapid plasma reagin (RPR) were negative. Tuberculosis (TB) evaluation, including QuantiFERON- TB Gold, tuberculosis polymerase chain reaction, and chest radiography yielded negative results. Cryoglobulin testing was negative. Autoimmune workup revealed an Anti-nuclear antibody (ANA) (1:640, nucleolar pattern). Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were within normal limits. Additional autoantibodies, including anti-double stranded deoxyribonucleic acid (DNA), anti-Smith, and rheumatoid factor, were negative. Angiotensin-converting enzyme (ACE) level was <10 (normal range), and serum lysozyme was normal.

Cerebrospinal fluid (CSF) analysis revealed lymphocytic pleocytosis with hypoglycorrhachia and elevated protein levels, consistent with an inflammatory or infectious process (see table below). CSF cultures and meningitis/encephalitis panel were negative. NMO/MOG antibodies were negative. Myelin Basic Protein was elevated at 24.5 ng/ml (normal: 0.00–5.50 ng/mL). Oligoclonal bands were also found to be positive with 9 bands detected in the CSF only with IgG Index elevated at 1.81 (Ref Interval: 0.28-0.66). MRI of the orbits with contrast demonstrated bilateral optic nerve enhancement with perineural involvement, more prominent on the right consistent with bilateral optic neuritis/perineuritis, suggestive of an inflammatory and/or infectious process (Figure 1). MRI brain with contrast was unremarkable for intracranial lesions.

CSF chemistry

CSF Cell Count

Test	Result	Reference Range & Units
Glucose (CSF)	45	50–80 mg/dL
Total Protein (CSF)	89.0	15.0–45.0 mg/dL
Unspun Color	Colorless	—
Unspun Clarity	Clear	—
Spun Color	Colorless	—
Spun Clarity	Clear	—
Sediment	No sediment observed	—

Meningitis/Encephalitis Panel

Test	Result	Reference Range & Units
Appearance	Clear	—
WBC Count	24	0–5 /µL
RBC Count	2	— /µL
BF Segs%	1%	0–7%
BF Lymphs%	98%	28–96%
BF Macrophage%	1%	16–56%

Table 1: CSF analysis showing chemistry, cell count and viral panel

Component	Result
Escherichia coli K1	Negative
Haemophilus influenzae	Negative
Listeria monocytogenes	Negative
Neisseria meningitidis (encapsulated)	Negative
Streptococcus agalactiae	Negative
Streptococcus pneumoniae	Negative
Cytomegalovirus	Negative
Enterovirus	Negative
Herpes simplex virus 1	Negative
Herpes simplex virus 2	Negative
Human herpesvirus 6	Negative
Human parechovirus	Negative
Varicella zoster virus	Negative
Cryptococcus neoformans/gattii	Negative

Plasma exchange (PLEX) was considered; however, given the absence of cryoglobulinemia, diagnostic uncertainty regarding the underlying mechanism, and multidisciplinary concern from infectious diseases and ophthalmology in the setting of untreated HCV infection with high–viral load, IVIG was selected as the initial therapeutic approach. After the confirmation of normal serum immunoglobulin A (IgA) levels, the patient received a 5-day course of IVIG 400 mg/kg for immune-mediated optic neuritis.

Following multidisciplinary consultation with infectious diseases and ophthalmology, corticosteroids were withheld due to concerns regarding potential exacerbation of active hepatitis C virus replication. Unfortunately, only minimal improvement in vision, and his headache persisted following treatment with IVIG. He was discharged with a diagnosis of bilateral optic neuritis, considered most consistent with possible Hepatitis C-associated neuroinflammation. Initiation of direct-acting antiviral therapy for HCV was deferred, as the patient's impending incarceration preceded the scheduled treatment approval and initiation timeline. He was discharged with close follow-up arranged with infectious disease (for HCV treatment), neurology, and ophthalmology.

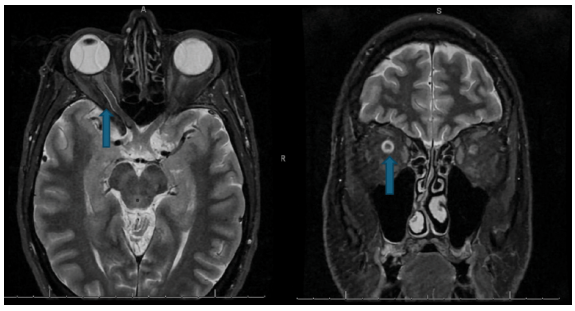

Figure 1: Coronal T2 STIR sequence (left) showing hyperintense signal prominently within the right optic nerve than the left with an axial T2 sequence (right) showing hyperintense signal along a segment of the right optic nerve.

Continued: Atypical Presentation of Optic Neuritis in a Patient with Active Hepatitis C Infection: A Case Report

3. Discussion

This case presents an atypical constellation of findings that challenge conventional diagnostic pathways for optic neuritis and demyelinating disease. The patient's bilateral optic neuritis, uveitis, and inflammatory CSF profile with elevated myelin basic protein and oligoclonal bands raised initial consideration of a central inflammatory process, including demyelinating etiologies. However, the presentation deviates from the classic monophasic optic neuritis seen in conditions like clinically isolated syndrome (CIS) or early multiple sclerosis (MS), which typically presents as unilateral vision loss, a positive relative afferent pupillary defect, and is more common in younger adults [4]. The absence of MRI lesions fulfilling the McDonald criteria for MS combined with bilateral macular edema and posterior uveitis further complicates the diagnostic picture. Although optic neuritis can occur in MS, their simultaneous bilateral optic disc edema with macular edema and intermediate/posterior uveitis is highly atypical for MS and should prompt consideration for alternative infectious or systemic inflammatory etiologies [5] as in this patient. Visual evoked potentials were not obtained and may have provided additional insight into the degree and pattern of optic nerve involvement.

A distinctive feature of this case is the presence of untreated chronic Hepatitis C virus (HCV) infection, with a high viral load and genotype 3A, without evidence of cryoglobulinemia or interferon therapy. Although ocular manifestations have been reported in HCV infection, optic neuritis remains a rare and incompletely characterized complication [6]. Most previously described cases of HCV-associated demyelination or optic neuritis occurred in the context of interferon-alpha therapy, which was not a factor here. There is limited but emerging evidence that HCV may contribute to immune-mediated neuroinflammation through mechanisms such as molecular mimicry or direct triggering of autoimmune responses in the central nervous system, supported by isolated reports of HCV Ribonucleic acid (RNA) detections in brain tissue or demyelinated lesions [7]. The patient's positive ANA with a nucleolar pattern, lymphocytic pleocytosis with hypoglycorrhachia, and elevated IgG index provide additional support for a possible HCV-associated neuroinflammatory process rather than classic MS.

This case adds to the growing spectrum of MS mimics and highlights the diagnostic challenges posed by atypical optic neuritis, particularly in the context of systemic viral infections. The lack of disseminating brain MRI lesions, the presence of multiple CSF markers of CNS inflammation, and the decision to prioritize intravenous immunoglobulin over corticosteroids due to infection risk highlight the need for careful diagnostic and therapeutic stewardship in such complex presentations.

4. Conclusion

This case urges clinicians to maintain a broad differential, particularly in middle-aged patients with systemic comorbidities and atypical neuro-ophthalmic features. It also raises important questions about the interface between chronic viral infections like HCV and immune-mediated demyelinating syndromes, warranting further investigation.

DECLARATIONS

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Ethics approval (include appropriate approvals or waivers): IRB approval was not needed as patient information was de-identified

Consent to participate: Written informed consent was obtained from the patient. Written Consent for publication (include appropriate statements). Written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review the upon request.

Availability of data and material: None

Code availability: Not applicable

Authors' contributions

Oreoluwa Morakinyo, Divya Sharma and Vijaya Valaparla wrote the main manuscript text. Oreoluwa Morakinyo, Divya Sharma prepared figure 1 and table 1. Laura Wu and Chilvana Patel supervised and made corrections. All authors read, reviewed and approved the manuscript.

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Algal Blooms and Amyotrophic Lateral Sclerosis: A Systematic Review

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Abstract

Smoking, herbicides, pesticides, heavy metals, silica, and numerous other environmental factors are thought to raise the risk of developing Amyotrophic Lateral Sclerosis (ALS). Researchers believe that toxic blue-green algae blooms may be a contributing factor as they uncover clues that seem to link a few ALS cases to people's proximity to coastal areas and lakes. We intend to summarize the updated information on the controversial relationship between cyanotoxins and ALS in the context of ongoing algal blooms caused by climate change. In 1954, a high incidence of Amyotrophic Lateral Sclerosis / Parkinsonism dementia complex was noticed among the Chamorro people, in Guam. This was attributed to their diet being contaminated with cyanobacteria-derived beta-N-methylamino-L-alanine (BMAA). Scientific literature established the fact that transfer RNA (tRNA) synthetases can aminoacylate amino acid analogs to their cognate counterparts. BMAA is incorporated at phenylalanine, proline, alanine, and glutamate sites during protein synthesis, thereby amplifying their potential for neurotoxicity. This misincorporation is one mechanism that may contribute to the onset of sporadic neurodegenerative diseases. The National Center for Environmental Health of the Centers for Disease Control and Prevention is currently conducting a study in Florida called the Cyanotoxins in Air Study to examine the health effects of exposure to cyanotoxins in the air. We emphasize the importance of using a variety of scales of analysis, including satellite remote sensing of cyanobacterial algal blooms, to establish the link between ALS and algal blooms.

Introduction

Lou Gehrig's disease is a fatal neuromuscular condition that worsens over time and is characterized by muscle weakness, wasting, fasciculations, and increased reflexes. Three novel ALS risk genes were discovered by a study, which also found a specific genetic correlation between ALS and autoimmune diseases (Li et al, 2021). Despite advancing knowledge of the genetic causes of ALS, most cases are sporadic and lack a distinct family history. It is believed that several environmental factors, including silica, heavy metals, pesticides, herbicides, and smoking, increase the risk of developing ALS (Weisskopf & Ascherio 2009; Bozzoni et al., 2016; Anderson, 2022). While patients may experience different initial triggers, once the disease has progressed to its full extent, most patients experience similar final mechanisms of motor neuron degeneration.

Since motor neurons are the longest cells in the body, even the slightest disruption of cellular hemostasis or transport by an environmental toxin could have devastating consequences (Banack SA et al.,2010). Therefore, identifying an environmental trigger for ALS has significant implications. It was discovered that samples of cyanobacterial blooms collected over a couple of years contained the neurotoxic amino acid, β-N-methylamino-L-alanine (BMAA) (Metcalf et al., 2008). As they find evidence that appears to link a few ALS cases to people's proximity to coastal areas and lakes, researchers believe that toxic blue-green algae blooms may be a contributing factor. In light of ongoing algal blooms brought on by climate change, we want to provide an update on the contentious connection between cyanotoxins and ALS.

Discussion

The Chamorro people of Guam were discovered to have a high incidence of Amyotrophic Lateral Sclerosis/Parkinsonism Dementia Complex (ALS/PD) in the 1950s (Chris C. Plato et al., 2003). It was attributed to their cycad seed-based diet. The specialized roots of cycads (Cycas micronesica) contain cyanobacteria and the concentration of protein-bound BMAA is up to a hundred times higher than that of free BMAA in the seeds and flour (Banack SA et al., 2010). The consumption of flying foxes or cycad seeds by the Chamorro people of Guam may have generated sufficiently high cumulative doses of plant neurotoxins that led to neuropathologies (Cox PA, Sacks OW, 2002). From 1945 to 1972, epidemiologic studies documented 350 ALS cases unique to the Chamorro population (Reed DM & Brody JA, 1975). Filipino migrants to Guam have been diagnosed with ALS with a high mortality rate of six times that of the continental US (Garruto RM et al., 1981). A study examined brain tissues of Chamorro people of Guam who died from ALS/PDC, revealing the presence of beta-methylamino-l-alanine (BMAA) as a free amino acid in 83% of patients and as a protein-associated amino acid in 100% of Chamorro individuals (Murch SJ et al., 2004).

Researchers are studying BMAA, produced by cyanobacteria and diatoms, as a potential risk factor for neurodegenerative illnesses (Ra D et al., 2021). The ability to transfer RNA (tRNA) synthetases to aminoacylate amino acid analogs to their cognate counterparts has been established by a growing body of scientific literature (Rubio Gomez MA, Ibba M, 2020). BMAA is incorporated at the phenylalanine, proline, alanine, and glutamate sites during protein synthesis, increasing the potential for neurotoxicity (Dunlop RA et al., 2013). One mechanism that could play a role in the sporadic onset of neurodegenerative diseases is this misincorporation. A study using biochemical assays discovered that human alanyl-tRNA synthetase uses BMAA as a substrate and that BMAA inhibits the enzyme's associated amino acid activation and editing functions (Han NC et al., 2020).

Historically, BMAA was primarily produced by cycad-associated cyanobacteria, which accumulate in cycad seeds consumed by fruit bats or flying foxes which are eventually consumed by humans or directly used to make flour (Nunes-Costa D et al., 2020). Data on BMAA concentrations in South Florida waters suggests that

Continued: Algal Blooms and Amyotrophic Lateral Sclerosis: A Systematic Review

Guam's situation is not unique, and BMAA could be found in high concentrations in aquatic animals worldwide where cyanobacteria blooms occur (Brand LE et al., 2010). BMAA bioaccumulation in aquatic animals, particularly fish, raises concerns about long-term human health risks due to its presence in limnic ecosystems (Jiao Y et al., 2014). An ALS patient in Florida was linked to chronic exposure to BMAA through shellfish consumption, suggesting direct human exposure to neurotoxic amino acids may contribute to the etiology of ALS (Banack SA et al., 2014). In China, BMAA was discovered in freshwater aquaculture ecosystems, affecting organism growth and food quality (Wu X et al., 2019). Urbanization, agriculture, and industrial development impact harmful planktonic and benthic cyanobacterial bloom dynamics in aquatic ecosystems, with nitrogen and phosphorous discharge affecting algal bloom potentials (Paerl H, 2008; Dong Y et al., 2014). In 2009, the finding of BMAA in cyanobacterial scum material and its predominance in Dutch urban waters raised serious concerns about the potential for chronic effects from low-level doses (Faassen EJ et al., 2009). The N-hydroxysuccinimide ester of N-butyl nicotinic acid (C4-NA-NHS) improved BMAA detection and localized BMAA in blue mussel tissues, suggesting that effective purification is necessary before consumption (Andrýs R et al., 2015). Research on Lake Finjasjön (Sweden) water samples and BMAA bioaccumulation patterns in plankti-benthivorous and piscivorous fish indicated that older fish and fish with different feeding habits may have higher BMAA concentrations (Lage S et al., 2015). Baltic Sea (Northern Europe) BMAA occurrence contradicts pelagic sources, with phytoplankton communities showing positive levels, while benthic invertebrates and fish species have no measurable levels (Zguna N et al., 2019). Figure. 1 summarizes the biomagnification of BMAA in Guam and the aquatic ecosystem.

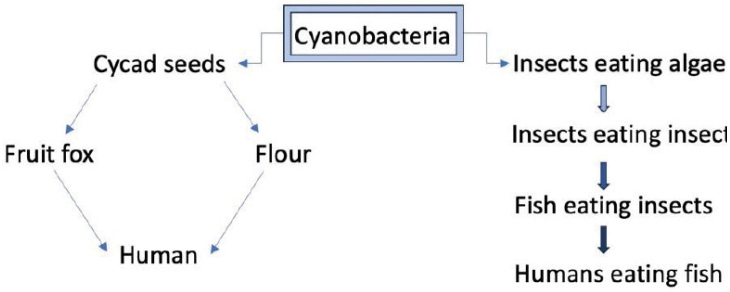

Figure. 1: Biomagnification of BMAA in Guam and the aquatic ecosystem.

By lowering cholesterol, reducing inflammation, and reducing lipid peroxidation, blue-green algae help prevent cardiovascular disease and nonalcoholic fatty liver disease (Ku CS et al., 2013). Blue-green algae dietary supplements offer health benefits, but monitoring these substances for safe consumption is critical. A study used hydrophilic interaction liquid chromatography and tandem mass spectrometry to determine cyanotoxins in nine commercial supplements (Aparicio-Muriana MDM et al., 2023). The Centers for Disease Control and Prevention's (CDC) National Center for Environmental Health is conducting a research study called Cyanotoxins in Air Study (CAST). People who live or work in Florida on Lake Okeechobee, the St. Lucie River, the Caloosahatchee River, or the Cape Coral Canals are eligible to participate in the study. A study reported a high incidence of sporadic ALS in nine patients near Lake Mascoma in New Hampshire, which could be related to chronic exposure to cyanobacterial neurotoxins like BMAA (Caller TA et al., 2009). Torbick N et al., used satellite remote sensing to develop phycocyanin concentration maps in northern New England to assess relationships with ALS cases (Torbick N et al., 2017).

L-serine, an amino acid produced by astrocytes, has been shown to have neuroprotective effects (Jeon H et al., 2022). The possibility that L-serine could mitigate the adverse impacts of BMAA arises from the fact that it inhibits BMAA from increasing the number of oxidized proteins in cells and chaperone-mediated autophagy (Quinn AW et al., 2021). Vervets fed with BMAA showed ALS-type pathological changes (protein aggregates in motor neurons and microglial activation), while vervets fed with BMAA and L-serine exhibited reduced neuropathological changes (Davis DA et al., 2020). L-serine specifically increased the activity of the autophagic lysosomal enzymes cathepsins B and L but not any of the proteasome-hydrolyzing activities (Dunlop RA & Carney JM et al., 2021). A randomized clinical trial examining the effects of oral L-serine (30g/day) on ALS patients revealed that it was generally safe, well tolerated, and did not accelerate functional decline (Levine TD et al., 2017).

Pelagic flagellates and Pseudo-nitzschia chain-forming diatoms are examples of extreme events that aid in understanding the frequency, intensity, and geographic scope of harmful algae blooms in the future (Trainer VL et al., 2020; Kelchner H et al., 2021). Cyanobacterial growth and bloom potentials in freshwater and marine ecosystems are significantly impacted by environmental disturbances like nutrient enrichment and climate change (figure. 2) (Paerl HW & Paul VJ, 2012). Excess nitrogen and phosphorus in the environment can fuel the growth of cyanobacteria (Nwankwegu AS et al., 2019).

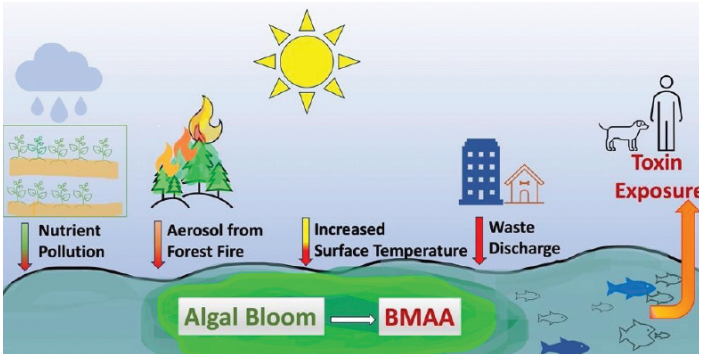

Figure. 2: Factors that contribute to cyanobacterial algal bloom.

Continued: Algal Blooms and Amyotrophic Lateral Sclerosis: A Systematic Review

The emphasis on establishing a link between ALS and algal blooms is a critical endeavor with significant public health implications. Educational and public awareness campaigns can encourage responsible behavior to reduce the potential health risks associated with algal blooms.

Conclusion

Implementing regulations on nutrient pollution, improving wastewater management, and sustainable agricultural practices are a variety of restoration efforts. Satellite remote sensing of cyanobacterial algal blooms allows researchers to track the spatial and temporal distribution of algal blooms across large bodies of water, making it a valuable tool in this research (Schaeffer BA et al., 2022). To establish the connection between ALS and algal blooms, we emphasize the significance of using a variety of scales of analysis, including satellite remote sensing of cyanobacterial algal blooms. A comprehensive approach can guide prudent choices regarding policy and research directions, improving the management of algal blooms, decreasing pollution, and enhancing public health outcomes.

Disclosure:

The authors have no disclosures to report.

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Broca's Area

CASE STUDIES

Delusional Parasitosis (Ekbom Syndrome) After Subarachnoid Hemorrhage

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Abstract

Cognitive impairment are common complaints following neurological events such as a subarachnoid hemorrhage (SAH) and can be linked to structural brain injury. However, psychiatric disorders may similarly cause memory impairments that may mimic neurologic cognitive decline especially in the setting of a recent neurological insult. Here we share the case of a 73-year-old lady who presented with subjective concerns of memory loss since discharge from the hospital for subarachnoid hemorrhage (SAH). Since neurocognitive screening tests and neurologic exam was unremarkable, diagnosis was not made until physical exam revealed several punctate excoriations at various healing stages and the patient revealed an underlying delusional parasitosis. We thus ordered appropriate laboratory tests and imaging studies and strongly encouraged psychiatric consultation. This case showcases the diagnostic complexity of pseudodementia secondary to Ekbom's delusional parasitosis due to its unlikely presentation in neurology. First, SAH as a neurologic trigger for subjective memory concerns can obscure early recognition of pseudodementia as the underlying etiology in patients presenting with subjective cognitive impairment. Second, delusional parasitosis is a rare somatic subtype of delusional disorder whose diagnosis may be obscured by the absence of overtly bizarre behavior beyond the fixed delusion. Third, pseudodementia secondary to psychotic disorders is less frequently described than that associated with other psychiatric conditions, such as depression. Taken together, this case underscores how the interplay between neurologic and psychiatric processes can obscure true diagnosis of cognitive impairment, emphasizing the need for comprehensive evaluation to avoid anchoring bias and delayed recognition of the underlying etiology.

Introduction

The evaluation of cognitive complaints is common following subarachnoid hemorrhage (SAH), with patients frequently reporting deficits in memory, attention, and executive function (1, 2). These along with mental fatigue, language difficulties, and reduced visuospatial ability can significantly impact quality of life among SAH survivors. As such, these concerns often prompt neurological evaluation as they are often attributed to structural brain injury. However, subjective cognitive decline does not always reflect objective impairment (3). Psychiatric disorders including mood disorders, psychotic disorders, and somatic-type delusional disorders may present with complaints of memory loss in the absence of measurable deficits (4, 5). This phenomenon has been described in the context of pseudodementia in which psychiatric illnesses present with symptoms that mimic cognitive decline (5). Thus, it is important to fully investigate organic and psychogenic etiologies in patients suffering from cognitive impairment.

In patients with recent neurologic injury, new cognitive complaints may be readily attributed to structural brain pathology (6). This bias may be potentiated by the subjective perception of "brain injury" by the patient after an organic neurologic event. As a result, this diagnostic framing may obscure alternative etiologies especially when psychiatric symptoms are initially unrecognized. Reports describing psychotic disorders presenting as cognitive decline in the setting of a recent cerebrovascular event remain limited (7). Here we present a case of subjective memory complaints following recent SAH that were ultimately attributable to underlying delusional parasitosis. Initial comprehensive neurologic evaluation revealed no objective cognitive impairment. However, further assessment revealed an underlying delusional parasitosis, suggesting that the patient's perceived cognitive decline was more consistent with psychiatric illness rather than post-hemorrhagic neurologic sequelae. Our case highlights the importance of maintaining a broad differential diagnosis when evaluating cognitive complaints after a neurologic injury and the importance of comprehensive neuropsychiatric assessment.

Case presentation

Patient is a 73-year-old, right-handed lady, with complex cardiac history, obesity, chronic kidney disease stage 3, obstructive sleep apnea, restless leg syndrome, and chronic anemia, accompanied by husband, presenting with concerns of progressive memory loss. She was recently hospitalized after presenting to the emergency for sudden onset severe headache. Initial vitals showed BP 201/82, and she was treated for hypertensive crisis. Computed tomography of the head without contrast showed a 5.6 mm subarachnoid hemorrhage and patient admitted for observation in the intensive care unit. Further work-up revealed that neurosurgical intervention was not indicated. She was treated with nimodipine and remained stable throughout hospital course.

On presentation to clinic, she reported an increased concern for memory decline over the last 18 months that drastically worsened since hospital discharge. She reported an inability to remember events as clearly as she did previously and was having increased difficulty comprehending her crochet and knitting patterns. She is in a wheelchair due to chronic back and bilateral knee pain but is otherwise able to perform basic and instrumental activities of daily living independently. She denies any problems with sleep.

Physical examination of the heart, lungs and abdomen were unremarkable. However, many punctate excoriations in various stages of healing were noted on the ears, extensor surfaces of both forearms, and lateral aspects of the right leg below the knee. Neurologic examination was unremarkable. Cranial nerves I-XII were intact.

Continued: Delusional Parasitosis (Ekbom Syndrome) After Subarachnoid Hemorrhage

Motor strength, muscle tone, and sensation were preserved in all four extremities. Upper and lower deep tendon reflexes 2+ and symmetric. Coordination and gait were normal. She scored 30/30 on mini-mental state exam (MMSE) (8).

When asked about the excoriations, patient casually states she is infested with insects that live within her. She can both see and feel the insects moving underneath her skin and attempts to pick them out with tweezers. She first started seeing the bugs in her eyelids two years ago and has since been plagued by them. She further details having woken up to the sensation of her mouth feeling full and saw dead maggots coming out of her mouth when she looked in the mirror.

She reports frustration from having visited 12 to 14 physicians over the last two years for this concern without resolution. She underwent multiple tests for parasites, all of which have returned negative. She has consulted with various specialists including primary care, infectious disease, ENT, and ophthalmologists, bringing samples of the removed insects to these visits, all of which have been unsuccessful. Notably in those consultations, she had scored 4/6 on PHQ-2 and 6/6 on GAD-2. She has taken many pictures of these insects, spending several minutes looking through her phone for such photo, however ultimately did not share them. Husband states that they have an upcoming appointment with psychiatry. She denies any suicidal or homicidal intent.

To investigate neuropathy as the somatic interpretation of bugs, an electromyography and nerve conduction velocity test was ordered. Relevant conditions such as iron deficiency anemia and restless leg syndrome are well managed by primary. Otherwise, recent labs investigating reversible causes of dementia (TSH, free T4, HbA1C, and vitamin B12) were within normal limits and patient has an automatic implantable cardioverter-defibrillator incompatible with magnetic resonance imaging (8). Patient and her husband were scheduled to return in six months after muscle and nerve testing and establishment of psychiatric care to reassess mentation.

Discussion

Here we share a unique case of pseudodementia secondary to delusional parasitosis (Ekbom syndrome), a somatic subtype of delusional disorder, whose clinical picture was confounded by a recent SAH. Cognitive dysfunction is a well-recognized complication of structural brain injury following an intracranial hemorrhage and typically affects attention, executive function, and memory (9, 10). These impairments are supported by objective findings on cognitive testing and neurologic examination (8, 11, 12). Pseudodementia is a reversible form of cognitive impairment that is caused by psychiatric illnesses, including mood, anxiety, and psychotic disorders, that clinically resemble true neurodegenerative processes. Psychiatric conditions may similarly impair attention, memory, and information processing (13). To differentiate them, the workup for memory concerns includes a detailed history collection, cognitive and psychiatric screening tools, and a comprehensive neurologic and physical exam. Despite subjective complaints, our patient demonstrated no measurable cognitive deficits (8). Studies have shown that elderly patients with depression indeed perform worse on cognitive screening tools compared to elderly patients without depression (14-16). Thus, even though cognitive deficits in pseudodementia may fluctuate compared to neurodegenerative processes, it is unexpected that both cognitive screening tools and neurologic exam were unremarkable in this patient presenting with acute memory concerns after subarachnoid hemorrhage.

This diagnostic obscurity is explained by the type of pseudodementia. Ekbom syndrome -delusional parasitosis rarely presents to neurology (17-19). It is characterized by the presence of a fixed, false belief of parasitic infestation persisting for at least one month in the absence of objective evidence of infestation (20, 21). Yet despite the functional impact of delusions, behavior may appear normal when delusions are not being discussed or acted upon. The realization that our patient was experiencing parasitic delusions was not uncovered until physical exam revealed a unique distribution of round excoriations of the ears and along the lateral surfaces of the forearms, and right lower leg. Once the topic of delusional parasitosis was introduced, the patient became immediately fixated on parasites and was unable to be redirected to other subjects. Other classic features such as the "specimen" or "digital pics" sign, in which patients bring multiple specimens or photographs to prove infestation, and frequent healthcare visits, were revealed which further clarified the diagnosis (22-24).

Memory concerns are not a typical presenting complaint seen with delusional parasitosis (25). It is likely that the recent subarachnoid hemorrhage served as a psychological trigger for memory loss. Additionally, the presence of a known structural brain insult may bias clinicians toward attributing new or ongoing symptoms to that injury, delaying consideration of alternative diagnoses. This case challenges the tendency to reflexively attribute symptoms to a recent neurologic insult and underscores the importance in considering underlying psychiatric etiologies. Although our patient presented with recent concerns of memory loss, closer evaluation revealed that these symptoms had been present for 18 months and coincides with her two-year history of delusional parasitosis. Hence, the relation of her delusions to impaired concentration and disrupted attention due to delusional preoccupation paints a psychotic-related pseudodementia clinical picture. Ekbom's syndrome is a rare psychotic disorder whose association to pseudodementia is poorly described (7). Using objective cognitive testing, careful physical examination, and attention to behavioral and psychiatric features we were able to distinguish neurocognitive disorders from psychiatric mimics.

The patient later followed up with psychiatry whose assessment agreed with ours and found a strong depression component. They initiated therapeutic alliance and started discussion of starting treatment with second generation antipsychotics. With this case, we underscore the diagnostic complexity that arises at the intersection of neurologic and psychiatric processes and highlight the im-

Continued: Delusional Parasitosis (Ekbom Syndrome) After Subarachnoid Hemorrhage

portance of a comprehensive evaluation in patients with subjective cognitive impairment to avoid anchoring bias and misattribution of the underlying etiology.

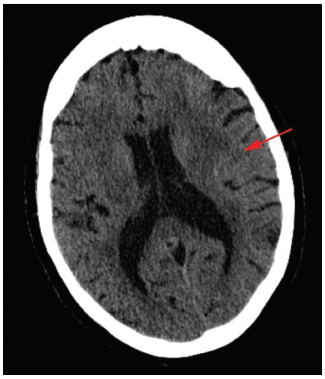

Figure 1. Non-contrast computed tomography (CT) of the head showing trace left sylvian fissure subarachnoid hemorrhage. A small 5.6 mm hyperdense region (red arrow) was noted on non-contrast CT imaging of the head which was suggestive of a trace subarachnoid hemorrhage of the left sylvian fissure.

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Subacute Progressive Dysphagia, Dysphonia, Failure to Thrive, and Hyperekplexia: A Case of Concomitant Anti-Hu (ANNA-1) and GAD65 Encephalitis

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Background:

Anti-Hu (or ANNA-1) antibodies are paraneoplastic-associated antibodies that target neuronal cells and are strongly correlated with small cell lung cancer. The clinical phenotype most frequently consists of limbic encephalitis, seizures, multifocal sensory neuropathy, cerebellar ataxia, seizures, dysautonomia, GI dysmotility (with or without pseudo-obstruction), progressive encephalomyelitis/myelopathy, or movement disorders including but not limited to chorea, pseudoathetosis, supranuclear palsy, and opsoclonus-myoclonus (Mueller et al, 2023; Dash & Pandey, 2018). Anti-GAD65 antibodies target glycine receptors and may be associated with a clinical phenotype of limbic encephalitis, stiff person syndrome spectrum of disorders, parkinsonism, seizures, cognitive impairment, myelopathy, brainstem dysfunction and cerebellar ataxia (Dash & Pandey, 2018; Muñoz-Lopetegi et al, 2020). Anti-Hu and GAD65 antibodies rarely co-occur, though there are a few individual case reports documenting this in the literature (Zong et al, 2023).

Case:

AW s a 77 year old female with past medical history significant for remote rectal cancer reportedly in remission per her husband, mild dementia (well-functioning at baseline), COPD secondary to chronic heavy tobacco use, OSA, HTN, and HLD. She presented to our hospital directly from the ENT outpatient clinic for emergent tracheostomy placement with hospital admission and workup for progressive dysphagia and dysphonia after she'd been found to have bilateral vocal cord paralysis on laryngoscopy that day. Her husband described that up until around early March 2025, AW had been at her baseline which is independently ambulatory, communicative, and independent in her activities of daily living. Around late March/early April, her voice "started changing, like she was becoming hoarser and quieter." Her swallowing ability particularly with solid foods also began to deteriorate resulting in decreased PO intake, and over those two months she'd lost about 20 pounds despite her still endorsing a normal appetite. She'd also had brief moments lasting seconds to minutes a few times weekly where her husband noticed she'd say things that didn't make sense or seemed unrelated to conversation. She would continue with conversation like normal and seeming at her baseline after those moments passed. Her husband also mentioned a brief hospitalization in April 2025 (roughly one month after onset of symptoms) where she'd presented for shortness of breath and was treated for pneumonia, but reportedly her dysphagia had not been addressed at that time.

Of note, the day after her present admission the patient briefly went into atrial fibrillation with RVR with a brief narrow complex tachyarrhythmia and brief asystole, with successful chemical cardioversion after two attempts. She remained awake and alert throughout the event, but an MRI brain with and without contrast was ordered that day given her history, and general neurology was consulted the next day (two days after admission) for her dysphagia, dysphonia, and failure to thrive.

Her history did not reveal any clear preceding trauma or inciting event to explain her vocal cord paralysis and other progressive symptoms. Her physical exam revealed a frail African-American woman with prominent bilateral ocular proptosis and a very exaggerated startle response. She could mouth words but given her severe dysphonia with vocal cord paralysis in addition to the trach in place on supplemental oxygen, she was unable to verbalize. She was moderately bradyphrenic but cooperative with the exam and was able to follow simple commands when asked. She was mildly spastic in her bilateral upper more so than lower extremities and initially noted to have reduced reflexes throughout. However, on our exam about 5 days after the initial consult, she showed signs of hyperreflexia in her lower extremities (bilateral crossed adductor and weak crossed pectoral reflexes). Although she was frail she had no focal weakness, she had 5- to 5/5 strength in all her upper and lower extremity muscle groups. Her cranial nerve exam revealed bilateral horizontal gaze palsies and inability to accommodate, but she had preserved vertical gaze. Her tongue was midline on protrusion, and her palatal rise appeared minimal/reduced but symmetric. Her face was symmetric, and sensation was intact throughout to light touch and pinprick. She had no ataxia on finger-to-nose-finger but did have a slightly increased physiologic action tremor and was slow to reach out.

Her MRI was reviewed and read as negative other than mild/moderate white matter disease. However, there was a small area of T2 hyperintensity noted in her dorsal pons just anterior to the fourth ventricle, questionably extending slightly above and below to the superior medullary junction. There was a question as to whether it was artifactual or not.

Results:

Several labs were ordered including TSH, vitamin B12 level, thiamine level, myasthenia gravis (MG) panel, CK, ESR, and CRP. It was decided to not delay treatment of possible Guillain-Barre syndrome with 3 days of IVIg given her progressive and severe course. She completed treatment with IVIg on 5/18/25 with little to no improvement in her symptoms. After the neurology team changed on 5/19/25, a repeat MR brain with and without contrast was ordered, which showed the same chronic white matter disease, as well as "moderate improvement of the pontomedullary T2 hyperintensity" which was still readily present on this follow up scan. Her exam remained stable as above, with the exception that she was very drowsy during some morning exams. Her increased startle response (hyperekplexia) remained prominent

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and even calling out her name would result in a startle/opening her eyes wide and fixating on the examiners. She remained cooperative with follow up exams and nodded/shook her head appropriately in response to questions. The decision was made to pursue a lumbar puncture but because she had trouble holding still, it had to be attempted under anesthesia (successfully performed on 5/23/25). The differential on her LP was normal; she had 1 TNC, a protein of 27, and glucose of 89.

As her serum labs continued to result and other CSF labs pended, for about a week and a half there was no clear answer for her persistently poor exam (see Table 1 for list of lab results, in order of result). A CT chest/abdomen/pelvis with contrast was pursued given the possibility of malignancy – this did not show any evidence of malignancy but did show an enlarged right hilar lymph node which hematology/oncology felt was likely reactive and less likely related to malignancy. Select rheumatology labs resulted weakly positive (RNP and Sjogren antibodies, see Table 1). The rheumatology team was consulted but normal complement levels and negative ANA made a primary rheumatologic diagnosis unlikely, and it was thought that her home hydralazine (for hypertension, which she was receiving in the hospital) may have contributed to the abnormal RNP and Sjogren antibodies. After discussion with her and her husband about a low likelihood of infection (given her normal LP result, and no infectious signs), she was treated with 5 days of high dose IV steroids (ended 5/28/2025). She was documented to have had some subjective clinical benefit, but no substantial changes to her exam or functionality.

On 5/30/25, her serum encephalopathy panel resulted positive for Anti-Hu (ANNA-1) and Anti-GAD65 antibodies, consistent with a probable paraneoplastic syndrome that correlated clinically with her cranial nerve and behavioral abnormalities. The prognosis for Anti-Hu encephalitis is unfortunately poor, and this antibody is highly associated with small cell lung cancer. She was recommended to undergo colonoscopy for routine cancer screening, as well as a PET-scan after discharge given no evidence of malignancy on the other screening tests completed while inpatient. Additional symptomatic treatment including PLEX or pulse-dosed steroids was also discussed with her family, but they felt that PLEX would be too hard on her body given how weak she'd become, and they opted to prioritize her discharge to IPR. However, They were all in agreement they wanted to pursue any further workup and treatment(s) that would provide her with the best chance of survival, including outpatient testing for suspected occult malignancy. The patient's family and the team agreed to initiate IV solumedrol 250mg daily for 3 days every 3 weeks, and work towards discharge to IPR.

Conclusion:

This case is unusual for a few reasons including that the patient was positive for both anti-Hu and anti-GAD65 antibodies which rarely co-occur, although a few individual case reports exist documenting this (Zong et al, 2023). Anti-Hu antibodies are strongly associated with malignancy, while GAD65 antibodies are most associated with autoimmunity and may be positive but considered to be clinically "silent," or a "bystander" antibody in patients without neurologic symptoms (Wang et al, 2020). Anti-Hu antibodies may also be considered clinically "silent" but this is less frequent and is correlated with patients having low antibody titers (Dalmau et al, 1990; Rossi et al, 2025). Autoimmune hyperekplexia is well documented to occur in cases of anti-glycine receptor antibody positivity in the stiff person spectrum of clinical disorders but has not been documented as specifically associated with GAD65 antibodies. Likewise, anti-Hu (ANNA-1) antibodies have not been described as associated with hyperekplexia, although Anti-Ri (ANNA-2) has rarely been associated with increased startle. However, anti-Ri antibodies are molecularly distinct from anti-Hu antibodies (Erlich et al, 2004).

In our patient's case, it seems probable that her hyperekplexia, dysphagia, and dysphonia are related to the damage to her pontomedullary brainstem region as manifested by the hyperintense T2 lesion seen on MRI. Hyperekplexia localizes to this region of the brainstem, as do other cranial nerve functions including that of cranial nerves IX and X (Brown et al, 1991; Kellett et al, 1998; Gambardella et al, 1999). Anti-GAD65 antibodies are associated with imaging abnormalities characterized by T2 hyperintensities including in the brainstem (approximately 29% of cases with related imaging findings), while any imaging findings on MRI with anti-Hu encephalitis are considered rare, but possible (Pittock et al, 2006; Saiz et al, 2009). However, anti-Hu encephalitis is often clinically associated with brainstem findings, so it remains unclear at this point whether her symptoms are related to the presence of only one versus both antibodies (Pittock et al, 2006; Budhram et al, 2021). It is also interesting that no malignancy was identified in this patient during admission despite anti-Hu being highly malignancy-associated. However, 10-20% of anti-Hu positive patients may initially screen negative for cancer (Lucchinetti et al, 1998).

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Table 1: Relevant lab results

Relevant serum labs (wnl unless "H" = high, "L" = low)
Sodium level 120-low 130s most of admission
CBC neg other than Hgb 8-9
Hgb a1c 5.5%
ESR 65
CRP 1.1 (H)
ANA IFA positive but titer neg, homogenous pattern
TSH 0.84
CK 38
Aldolase 5.2
Thiamine level 149.9
SPEP, UPEP neg
IgG level 1137
GM1, GQ1b neg
Voltage gated calcium channel level 6.1
NMO, MOG neg
Lupus panel neg other than RNP (4.8, H) and Sjogren antibodies (1.4, H)
MG panel neg
Cryptococcal antigen neg

Relevant CSF Results
Clear, colorless, TNC 1, RBC 1
Protein 27, glucose 89
1 oligoclonal band (MS panel)
Cytology and cytometry neg
CSF IgG index wnl
Encephalopathy panel positive for Anti-Hu (ANNA-1) and Anti-GAD65 antibodies

The Aberrant Evolution of Sleep Medicine: A Neurologist's Perspective

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"Sleep is of the brain, by the brain, and for the brain,"*

If the brain is the province of neurology, then neurologists should be natural stewards of sleep medicine. The brain may be shared with neuropsychologists, neurosurgeons, and psychiatrists, but extending its domain to cardiologists, otolaryngologists, or dentists requires some creative stretching, unless, of course, we choose to extract one of the 80 plus known sleep disorders and hand it over. That disorder is sleep apnea, the bread and butter of today's sleep providers. Neurologists can be faulted for allowing this shift, but the reasons are rooted in the history of the field itself.

In the 1970s, neurology began to transform from a specialty known for diagnosing untreatable diseases into one with real therapeutic power. Dopamine for Parkinson's disease, triptans for migraine, immunomodulators for MS, and new anticonvulsants for epilepsy ushered in a new era. While neurologists were rightfully energized by these advances, the modern field of sleep medicine was quietly taking shape.

A small group of neurologists, joined by pioneering PhDs, embraced the burgeoning science of sleep. By 1980, newly recognized disorders, including sleep apnea, led to a rapid expansion of sleep laboratories. Professional sleep societies emerged, conferences multiplied, and the field gained momentum. Early CPAP machines were cumbersome and poorly tolerated, opening the door for ENT physicians to surgically enlarge pharyngeal airways and for pulmonologists to take on CPAP management. In the search for non surgical treatments for sleep apnea, dentists introduced mandibular advancement devices. Neurologists, already stretched and with little sleep training in medical school or residency, often stepped back. Excessive daytime sleepiness, even narcolepsy, was viewed as rare and left largely unaddressed.

As connections between sleep apnea, restless legs, and insomnia with depression and dementia became more widely recognized, psychiatrists also entered the arena.

I recall giving a sleep disorders lecture a few years ago when a cardiologist approached me afterward. He invited me to speak at their weekly conference, this after I had just predicted that cardiology would one day fully embrace sleep medicine once they opened their own sleep labs. And indeed, the rest is history.

To be clear, sleep medicine benefits from participation across specialties. Even a basic recognition of sleep as an essential biological function is a step forward. The true pioneers of sleep medicine, neurologists and non-neurologists alike, worked tirelessly to highlight the importance of healthy sleep and the dangers of its neglect. Yet the influx of other specialists largely concentrated on sleep apnea.

There is, however, far more to sleep medicine than sleep apnea. Yes, every specialty has its "bread and butter," and apnea reliably pays the bills. It is relatively easy to diagnose, highly rewarding to treat, and supported by abundant management tools. For some clinicians, it has become so routine that much of the care is handled by ancillary providers.

But the broader universe of sleep disorders is intricate, time consuming, and intellectually rich, precisely the kind of mystery driven terrain that resonates with neurology.

Consider sleep disturbances associated with dementia and neurodegeneration, limb movement disorders, narcolepsy, insomnia, abnormal sleep architecture, and nocturnal events such as seizures, parasomnias, pain related awakenings, sleepwalking, and sleep related eating. These affect both children and adults. They are challenging, sometimes frustrating, but often rewarding, and they lie squarely within the neurological domain.

In stepping back from sleep medicine, neurology relinquished a fascinating, essential, and often treatable category of brain based disorders. The question now is whether the new era of neurology will reclaim this vital function of the brain.

Reestablishing neurology's leadership in sleep medicine will require:

1 Expanded sleep education in medical school and residency.

2. Greater neurologist participation in sleep fellowships and research.

3. Recognition of sleep as a neurologic vital sign.

4. Broader clinical engagement beyond sleep apnea.

*Hobson, J. Allan. Sleep is of the brain, by the brain, and for the brain. Nature 437, 1254-1256 (2005)

Guillain-Barré Syndrome: An Atypical Initial Presentation

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Introduction:

Guillain-Barré Syndrome (GBS) is an acute immune-mediated polyneuropathy characterized by a rapid, symmetrical weakness and absent or decreased deep tendon reflexes. It is an acquired neuromuscular paralysis seen to damage peripheral nerves. GBS is often triggered by a preceding infection such as an upper respiratory tract infection or gastroenteritis. Campylobacter jejuni is often cited as the most commonly identified infectious trigger.[1]

The disorder clinically presents with ascending weakness beginning in the lower extremities and progresses proximally over the course of days to weeks. Sensory findings typical of GBS include paresthesias and deep aching pain.[2] Diagnosis is primarily clinical and can be supported by cerebrospinal fluid (CSF) analysis and nerve conduction studies. CSF findings in GBS show albuminocytologic dissociation consisting of elevated protein, normal white blood cell count, and normal glucose.[2-4,8]

Two major subtypes of GBS exist: Acute Inflammatory Demyelinating Polyneuropathy (AIDP) and Acute Motor Axonal Neuropathy (AMAN). AIDP is a demyelinating subtype in which an immune attack targets the myelin sheath and Schwann cells.[5] This subtype appears with slowed conduction velocities, prolonged distal latencies, and temporal dispersion on nerve conduction studies. AMAN is an axonal subtype which involves primary axonal injury. Although AMAN does not manifest with substantial demyelination, it is often seen to have a worse short-term prognosis as it tends to progress faster.[5-6]

Early recognition and treatment with either plasma exchange or intravenous immunoglobulin (IVIG) is crucial as GBS has a narrow treatment window.[7] Treatments are most effective when initiated within the first two weeks of symptom onset. If this treatment window is missed, patients are at risk of rapidly progressing to life-threatening complications such as respiratory failure requiring mechanical ventilation or autonomic dysfunction.[6]

Case Presentation:

A 52-year-old female with a medical history of congestive heart failure, hyperlipidemia, gastroesophageal reflux disease, and obesity presented to the emergency department (ED) with severe back pain and an "electric band-like" pain running down her right leg. She denied a family history of autoimmune disease but reported a possible prior diagnosis of lupus in her early 30s. Imaging and diagnostic tests done at this visit revealed a right sided renal cyst, a chronic kidney stone, a urinary tract infection (UTI), and sciatica. The patient was discharged with appropriate treatment for her UTI symptoms.

Five days later, the patient returned to the ED with persistent back pain, a fall resulting in a left foot injury, gait instability, and new right leg pain and weakness distinct from her initial sciatic pain. Her symptoms initially involved the right leg but later progressed to involve the left leg. On presentation, she was hypertensive but otherwise hemodynamically stable. Urinalysis demonstrated ketonuria and hematuria, and foot radiographs revealed a displaced comminuted fracture of the left fifth digit. Following orthopedic and trauma consultation, CT imaging was obtained for further workup of the patient's worsening bilateral lower extremity weakness. CT imaging of the head, cervical, lumbar, and thoracic spine showed only mild degenerative changes and spondylosis.

The following day, the patient developed new neurological deficits, including right facial muscle weakness and tingling in the right upper extremity in an ulnar distribution. These symptoms progressed to a right facial droop and bilateral palmar paresthesias. After neurology consultation, a five-day course of IVIG was initiated, along with methylprednisolone. Following completion of IVIG therapy, the patient continued physical therapy for persistent lower extremity weakness, and a lumbar puncture was performed. Cerebrospinal fluid analysis showed albuminocytologic dissociation, supporting a diagnosis of Guillain-Barré Syndrome.

After completing steroid treatment, the patient's symptoms improved. She was expected to achieve a full recovery with continued rehabilitation at an inpatient rehabilitation facility. Given her unconfirmed history of lupus and autoimmune workup raising possibility for drug-induced lupus, the patient was advised to follow-up with an outpatient rheumatologist.

Discussion:

Guillain-Barré Syndrome is classically characterized by ascending, symmetrical weakness with areflexia. However, this case highlights an atypical presentation in which early symptoms were dominated by gait instability, recurrent falls, and asymmetric lower extremity pain.[2-3] These initial features, especially in the setting of concurrent diagnoses such as sciatica and urinary tract infection and in the absence of a preceding respiratory or gastrointestinal illness, contributed to a diagnostic challenge and delay in GBS recognition.[7]

This patient's presentation highlights the importance of maintaining a broad diferential diagnosis when evaluating patients with evolving neurologic symptoms. Furthermore, the case demonstrates how GBS symptoms can mimic musculoskeletal and radicular conditions. The patient's initial presentation with back pain and radiating leg pain led to a working diagnosis of sciatica. However, reconsideration of the diagnosis had to be made due to the patient's subsequent gait instability and falls as these symptoms point to broader neurologic involvement rather than isolated radiculopathy.

Early neurologic consultation and reassessment is crucial in cases like this in which symptoms rapidly progress. Furthermore, timely initiation of IVIG treatment and physical therapy led to the patient's clinical improvement. Although corticosteroids such as

Continued: Guillain-Barré Syndrome: An Atypical Initial Presentation

methylprednisolone are not typically indicated for GBS, the use of this medication was indicated for overlapping autoimmune processes particularly given the patient's reported but unconfirmed history of lupus.[9] In patients with neuromuscular weakness, continued therapy in an inpatient rehabilitation facility is crucial to address residual weakness and balance deficits and to optimize functional recovery.[10]

This case emphasizes the need for heightened clinical suspicion for GBS in patients with progressive gait instability and falls, even in the absence of classic features at presentation. Early recognition and prompt multidisciplinary management are essential to minimize morbidity and optimize patient outcomes.

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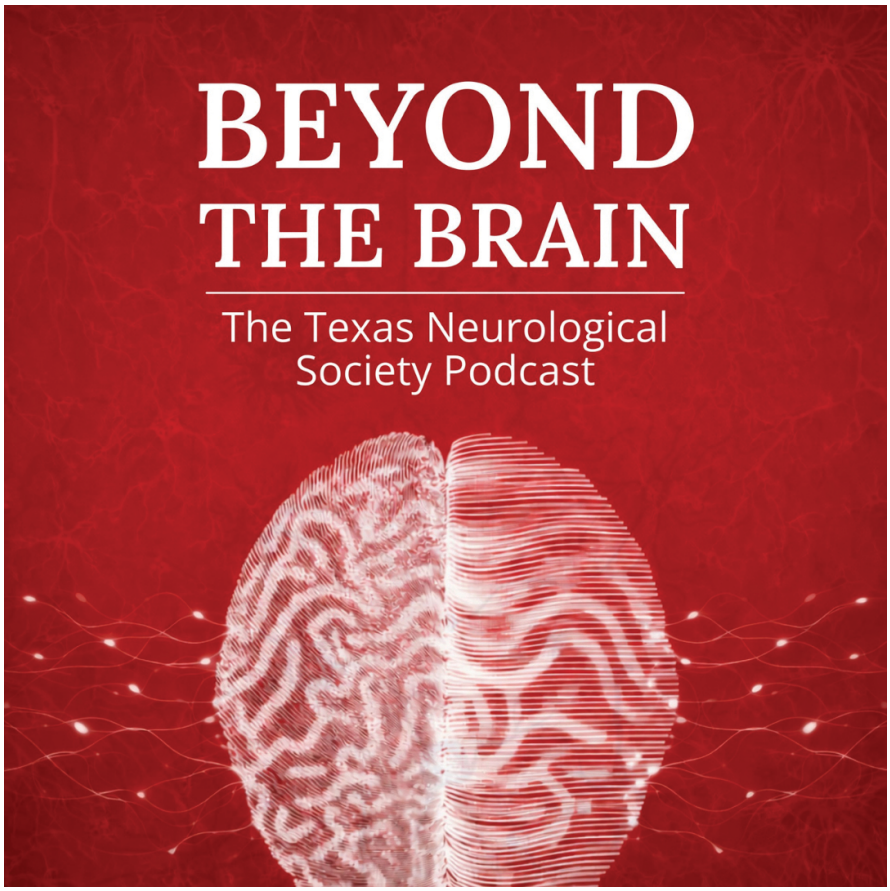

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Geographic Access to Thrombectomy-Capable Stroke Centers in Texas: Population Coverage Within 30- and 60-Minute Drive Times

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ABSTRACT

Background: Timely access to thrombectomy-capable stroke centers is critical for patients with acute ischemic stroke caused by large-vessel occlusion. Texas is geographically large, rapidly growing, and contains substantial urban-rural variation, making statewide access planning particularly relevant.

Objective: To estimate geographic population access to thrombectomy-capable stroke centers across Texas using 30-minute and 60-minute drive-time thresholds, and to evaluate variation across counties and Trauma Service Areas (TSAs).

Methods: A statewide geospatial analysis was performed using 2025 population estimates and identified thrombectomy-capable stroke centers in Texas. Modeled drive-time catchments of 30 and 60 minutes were generated. Population coverage was calculated at the state, county, and TSA levels. Regional findings were descriptively summarized.

Results: The statewide population included 31,689,512 residents. An estimated 16,021,147 residents were located within a 30-minute drive time of a thrombectomy-capable center, corresponding to 50.52% population coverage. At the 60-minute threshold, covered population increased to 25,950,644 residents, corresponding to 81.84% statewide coverage. At the TSA level, the highest 60-minute coverage was observed in TSA E (96.23%), followed by TSA O (90.74%), TSA P (87.66%), and TSA Q (86.84%). Lower 60-minute coverage was observed in TSA H (14.58%), TSA J (2.42%), and TSAs K, R, and T (0%). County- and regional-level variation was also observed.

Conclusion: Most Texas residents are located within a 60-minute drive time of a thrombectomy-capable stroke center, although marked geographic disparities remained across counties and TSAs. These findings may help inform targeted stroke systems planning and strategies to improve equitable access in underserved regions.

1. INTRODUCTION

Mechanical thrombectomy is a standard-of-care treatment for eligible patients with acute ischemic stroke due to large vessel occlusion (LVO) and can substantially improve functional outcomes when delivered rapidly (Goyal et al., 2016). Large vessel occlusion accounts for approximately one-quarter to one-third of ischemic strokes and is associated with a disproportionate share of stroke-related disability and mortality (Waqas et al., 2020). Because treatment benefit declines with delay, timely access to thrombectomy-capable centers is now recognized as an essential component of modern stroke systems of care (Saver et al., 2016).

Prior U.S. geospatial studies have demonstrated substantial variation in travel time to thrombectomy-capable centers and have shown that access analyses can help inform emergency medical services routing, interfacility transfer networks, and strategic placement of stroke resources (Adeoye et al., 2019; Aldstadt et al., 2022). These findings support the value of state-level evaluations tailored to local geography and population distribution.

Texas is an especially important setting for such analysis. As one of the largest and most populous U.S. states, Texas combines rapidly growing metropolitan regions with expansive rural areas where travel distances may be substantial. In a time-sensitive condition such as stroke, understanding where residents can reach thrombectomy care within clinically meaningful time windows has direct relevance for statewide neurological care planning.

Contemporary Texas-specific estimates of population access to thrombectomy-capable stroke centers remain limited. We therefore performed a geospatial analysis of thrombectomy-capable centers in Texas to estimate: (1) the proportion of residents living within a 30-minute drive time, (2) the proportion living within a 60-minute drive time, and (3) geographic variation in access across counties, Trauma Service Areas and regional areas.

2. METHODS

2.1. Study Design

We conducted a cross-sectional geospatial analysis of population access to thrombectomy-capable stroke centers in Texas.

2.2. Thrombectomy-capable Center Selection

Level I and Level II stroke facilities were included as thrombectomy-capable stroke centers based on the Texas Department of State Health Services stroke designation framework, in which Level I and II facilities represent advanced stroke centers with endovascular stroke capabilities (Texas Department of State Health Services [DSHS], 2025). A statewide list of Level I and Level II stroke centers in Texas was obtained from the Texas DSHS website (Texas Department of State Health Services, 2025).

2.3. Population and Geographic Data

Texas county boundary shapefiles and county population estimates were obtained from the United States Census Bureau (USCB) website (USCB, 2025). Counties were selected as the unit of population aggregation for statewide analysis. A county-to–Trauma Service Area (TSA) crosswalk file (Appendix 1) was used to group counties into TSA regions for secondary analyses.

2.4. Geospatial Analysis

Hospital coordinates were imported into ArcGIS Pro. Network-based drive-time service area analyses were performed us-

Continued: Geographic Access to Thrombectomy-Capable Stroke Centers in Texas: Population Coverage Within 30- and 60-Minute Drive Times

ing ArcGIS Network Analyst to generate 30-minute and 60-minute travel-time polygons around each thrombectomy-capable center using driving mode. Individual service areas were merged to create statewide 30-minute and 60-minute coverage layers.

Coverage layers were spatially intersected with Texas county polygons and associated population datasets. Covered population estimates within 30-minute and 60-minute travel-time catchments were calculated through spatial overlay and attribute summarization procedures. County-level covered populations were subsequently aggregated to derive statewide and Trauma Service Area–level estimates.

2.5. Outcomes

Primary outcomes were: (1) proportion of the Texas population residing within a 30-minute drive time of a thrombectomy-capable stroke center, and (2) proportion residing within a 60-minute drive time. Secondary outcomes were variation in estimated coverage across counties and aggregated variation across Trauma Service Areas.

2.6. Data Presentation

Results were summarized using descriptive statistics and displayed using maps and/or tables of statewide, county-level, TSA-level and region-level population coverage

3. RESULTS

3.1. Facility Identification and Distribution

Fifty-three thrombectomy-capable stroke centers were identified statewide. Forty-eight level I and 5 level II facilities. Facilities were concentrated in major metropolitan regions, particularly Houston, Dallas–Fort Worth, Central Texas, and the Rio Grande Valley. In contrast, large portions of West Texas and other rural areas had limited local center presence (Figure 1).

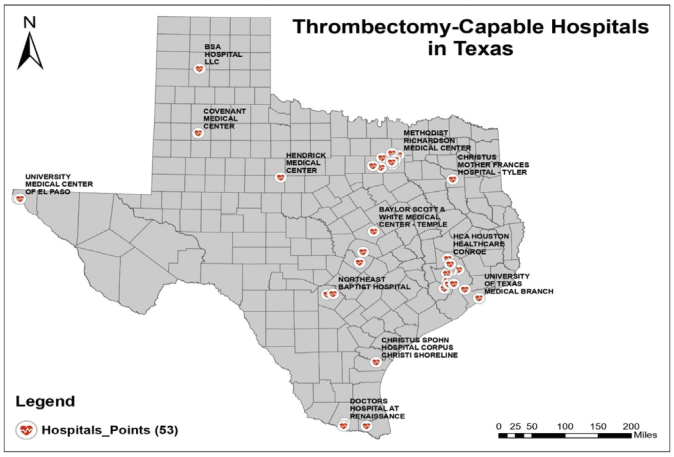

Figure 1: Geographic distribution of Thrombectomy-Capable Stroke Centers in Texas (2024)

3.2. Statewide Population Access

Using 2025 USCB estimates, the total population included in the statewide analysis was 31,689,512 residents. An estimated 16,021,147 residents were located within a 30-minute drive time of a thrombectomy-capable stroke center, corresponding to 50.52% statewide population coverage. At the 60-minute threshold, the covered population increased to 25,950,644 residents, corresponding to 81.84% statewide population coverage.

3.3. County-Level Coverage

County-level access varied substantially. At 30 minutes, 177 counties (69.7%) had no estimated coverage, decreasing to 94 counties (37.0%) at 60 minutes. Counties with high coverage (80–100%) increased from 5 (2.0%) at 30 minutes to 47 (18.5%) at 60 minutes. Highest 30-minute coverage was observed in Dallas (94.47%), Lubbock (93.04%), Tarrant (92.45%), Smith (82.04%), and Harris (81.88%). The detailed breakdown of county-level coverage is presented in Table I. The individual county coverage list is included in Appendix 1.

Table I: Distribution of Texas Counties by Population Coverage Category at 30-Minute and 60-Minute Drive-Time Thresholds to Thrombectomy-Capable Stroke Centers (n=number of counties=254)

County coverage category	30-min coverage, n (%)	60-min coverage, n (%)
80%–100%	5 (2.0%)	47 (18.5%)
60%–<80%	4 (1.6%)	18 (7.1%)
40%–<60%	7 (2.8%)	15 (5.9%)
20%–<40%	11 (4.3%)	23 (9.1%)
>0%–<20%	50 (19.7%)	57 (22.4%)
0%	177 (69.7%)	94 (37.0%)

Table I: Distribution of Texas Counties by Population Coverage Category at 30-Minute and 60-Minute Drive-Time Thresholds to Thrombectomy-Capable Stroke Centers (n=number of counties=254)

3.4. Trauma Service Area Coverage

Marked regional variation was observed across TSAs (Figures 2 and 3; Table II). At the 30-minute threshold, the highest population coverage was observed in TSA E (64.08%), followed by TSA Q (63.19%), TSA B (58.57%), TSA P (50.81%), and TSA O (46.55%). Intermediate coverage was observed in TSA V (42.64%), TSA L (33.38%), and TSA G (32.43%). Several TSAs demonstrated no estimated 30-minute coverage, including TSAs J, K, R, S, and T. TSAs C and M contained no assigned population in the current dataset and were retained for completeness of the statewide TSA framework.

At the 60-minute threshold, coverage increased substantially across most regions. TSA E demonstrated the highest 60-minute coverage (96.23%), followed by TSA O (90.74%), TSA P (87.66%), TSA Q (86.84%), TSA L (83.58%), and TSA G (82.94%). TSA B (78.33%) and TSA V (77.41%) also demonstrated high regional coverage. Per-

Continued: Geographic Access to Thrombectomy-Capable Stroke Centers in Texas: Population Coverage Within 30- and 60-Minute Drive Times

sistently low 60-minute coverage was observed in TSAs F (10.70%), H (14.58%), J (2.42%), K (0%), R (0%), and T (0%) (Figure 4).

Visual mapping demonstrated concentration of higher TSA-level coverage in major metropolitan and central corridor regions, with lower coverage persisting across several frontier and sparsely populated western and southern regions (Figures 2 and 3).

TSA	Total Population	30-min Coverage (%)	60-min Coverage (%)
A	431,338	21.48	65.03
B	538,916	58.57	78.33
C*	0	0.00	0.00
D	534,814	15.11	40.23
E	8,883,230	64.08	96.23
F	370,474	0.21	10.70
G	727,607	32.43	82.94
H	346,051	0.37	14.58
I	1,325,241	22.24	55.98
J	166,199	0.00	2.42
K	18,869	0.00	0.00
L	949,023	33.38	83.58
M*	0	0.00	0.00
N	518,638	1.20	21.62
O	2,759,082	46.55	90.74
P	3,076,969	50.81	87.66
Q	8,522,805	63.19	86.84
R	55,643	0.00	0.00
S	110,849	0.00	20.90
T	299,532	0.00	0.00
U	612,447	22.10	65.00
V	1,441,785	42.64	77.41

*Table II: Population Coverage Within 30-Minute and 60-Minute Drive-Time Thresholds to Thrombectomy-Capable Stroke Centers Across Texas TSAs. *TSAs C and M contained no counties/population in the current dataset and were retained for completeness of the statewide A–V TSA framework.*

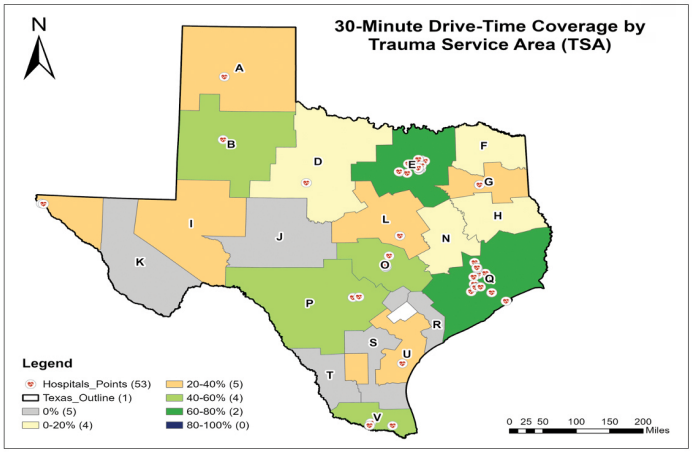

Figure 2: 30-Minute Drive-Time Coverage By TSA

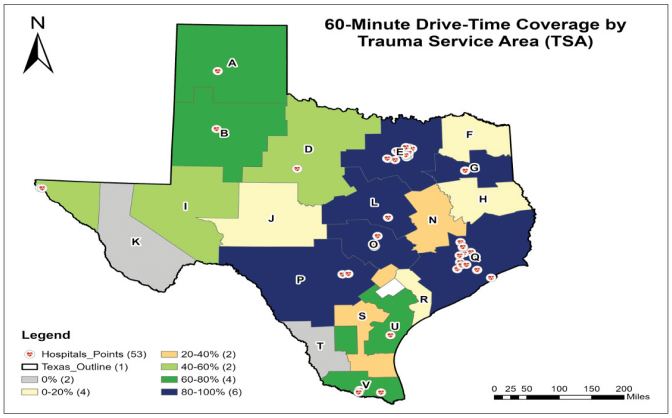

Figure 3: 60-Minute Drive-Time Coverage By TSA

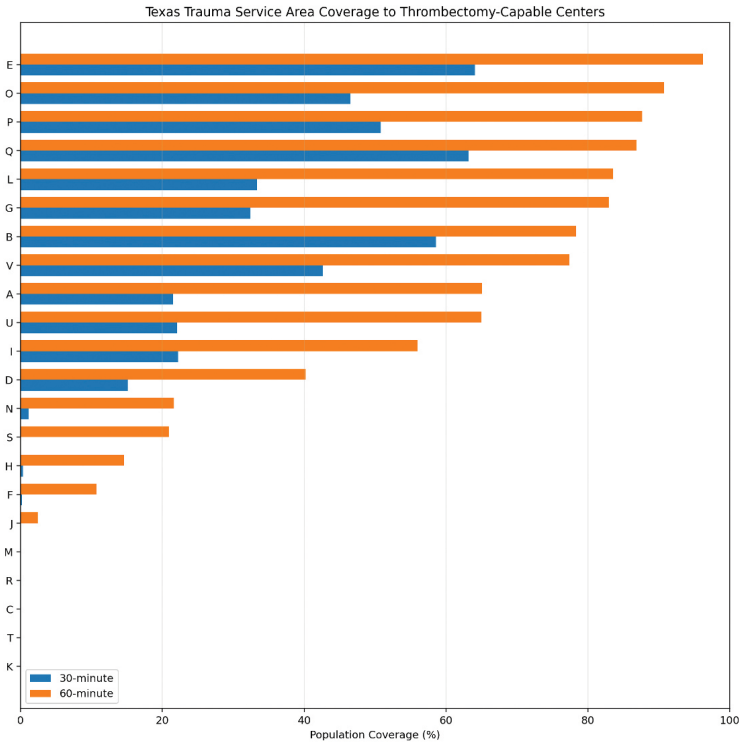

Figure 4: Comparison between 30- and 60-minute coverage per TSA

Continued: Geographic Access to Thrombectomy-Capable Stroke Centers in Texas: Population Coverage Within 30- and 60-Minute Drive Times

3.5. Regional Coverage Patterns

Trauma Service Area findings were grouped into broadly recognized geographic regions of Texas for descriptive interpretation. Highest estimated coverage was seen in the Dallas–Fort Worth/North Texas region (96.23%), followed by the San Antonio/South Central Texas region (87.66%), the Greater Houston/Gulf Coast region (86.84%) and Central Texas/Austin corridor region (80.65%) (Figure 5).

Good coverage was observed in South Texas/Border/Coastal Bend region (approximately 62%) and Panhandle/Northwest Texas region (approximately 61%). Moderate coverage was observed in the Northeast Texas region (approximately 58%) and West/Far West Texas (approximately 49%). Lower coverage was observed in East Texas (15%) (Figure 5). These groupings were used for descriptive presentation and do not represent formal administrative regional boundaries.

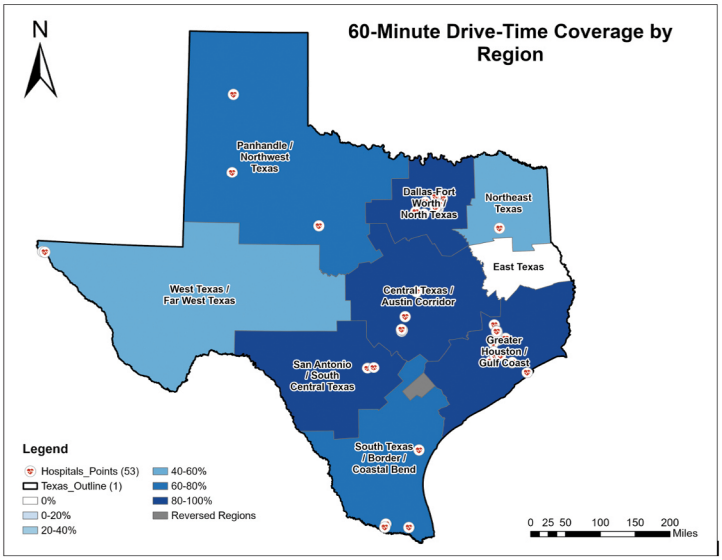

Figure 5: Comparison of 60-minute drive time regional coverage

4. DISCUSSION

In this statewide geospatial analysis, an estimated 50.5% of Texans resided within a 30-minute drive time and 81.8% within a 60-minute drive time of a thrombectomy-capable stroke center. These findings suggest that Texas has comparatively strong access to advanced stroke care despite its large land area and dispersed rural populations. Recent national analyses reported that approximately 80% of the US adult population had 60-minute access to advanced stroke care in 2022 (Schieb et al., 2025), while earlier estimates found that approximately 50% of the US population had timely ground access to thrombectomy-capable or comprehensive stroke centers in 2019 (Aldstadt et al., 2022). Against this benchmark, current Texas coverage appears favorable overall, although substantial intrastate variation remains across counties and Trauma Service Areas.

The favorable statewide 60-minute coverage appears to be driven primarily by high-access metropolitan regions containing large shares of the Texas population. TSA E, which includes the Dallas–Fort Worth area, demonstrated the highest 60-minute coverage (96.23%), while TSA P (San Antonio/South Central Texas) and TSA Q (Greater Houston/Gulf Coast) also exceeded 85% coverage. Because these regions are densely populated, strong access within a limited number of urban corridors substantially increases statewide population coverage. In contrast, lower modeled access in more sparsely populated western and frontier regions, including West/Far West Texas (~19%), exerts less influence on statewide averages despite representing large geographic gaps. This helps explain how Texas can achieve high overall 60-minute coverage while still retaining marked regional disparities.

Interpretation of lower-access areas should consider both geography and population size. Several regions with poor modeled access also had relatively small populations, including TSA K (population 18,869; 0% 60-minute coverage) and TSA J (population 166,199; 2.42% coverage), suggesting that some large geographic gaps occur in sparsely populated areas. By contrast, more populous regions with incomplete coverage may represent a greater public health priority. For example, TSA I contained 1,325,241 residents but only 55.98% 60-minute coverage, while TSA U included 612,447 residents with 65.0% coverage. Similarly, TSA V contained 1,441,785 residents and, despite relatively favorable coverage (77.41%), still leaves a substantial absolute number of residents beyond 60-minute access. These findings suggest that planning priorities should consider both equity for remote communities and the total population potentially underserved. They also suggest that statewide gains in thrombectomy access may be achieved through targeted system optimization rather than uniform expansion of new centers. Because several metropolitan TSAs already demonstrated high 60-minute coverage, existing urban hubs may continue to function as anchors for regional stroke networks. In contrast, remote low-coverage regions such as TSAs J and K may benefit more from faster transfer pathways, telestroke-supported triage, and streamlined interfacility transport than from immediate duplication of comprehensive services. For more populous regions with incomplete coverage, including TSAs I, U, and V, selective expansion of thrombectomy capacity or EMS routing redesign may offer greater population-level benefit.

Regionally, the greatest opportunities may differ across Texas. West and Far West Texas, where modeled access remained lowest and thrombectomy-capable centers were sparse, may warrant evaluation of strategically located existing hospitals for future thrombectomy-capable designation, supplemented by tele-stroke and rapid transfer pathways; whereas more populous South Texas and border regions may warrant priority consideration for future capacity expansion. High-performing metropolitan corridors such as

Continued: Geographic Access to Thrombectomy-Capable Stroke Centers in Texas: Population Coverage Within 30- and 60-Minute Drive Times

Dallas–Fort Worth, Houston, and San Antonio are likely to remain critical hubs within statewide referral networks.

Limitations of the study

Drive-time estimates were model-based and may not reflect real-world traffic conditions, weather, EMS availability, or use of air transport. Facility inclusion was based on publicly available thrombectomy-capable center designations and may not capture changes in certification status after 2025 or differences in procedural volume and 24-hour availability. Furthermore, population coverage estimates were derived from geographic aggregation methods and may not fully reflect within-county variation. Finally, we acknowledge that geographic access does not necessarily equate to receipt of thrombectomy or clinical outcomes.

5. CONCLUSION

Texas demonstrated strong overall geographic access to thrombectomy-capable stroke centers, with an estimated 81.8% of residents located within a 60-minute drive time. However, substantial disparities persisted across counties, Trauma Service Areas, and broadly defined geographic regions, particularly in parts of West Texas, East Texas, and selected border regions. These findings suggest that while Texas has a robust statewide endovascular stroke care network, targeted regional strategies may be needed to improve equitable access in underserved areas. Ongoing geospatial assessment may help guide future stroke systems planning in one of the nation's largest and fastest-growing states.

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The Neurological Translation Website

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Our team is excited to launch the Neurological Translation website, which is designed to teach clinicians how to perform the neurological and stroke examinations in Spanish. The site features recorded audio of key questions and commands required for conducting these exams in Spanish. It is freely accessible at: https://cskipworth.github.io/UTH-Stroke-PWA/index.html. We will continue to expand the platform by adding support for additional languages, including Vietnamese, Mandarin, Hindi, and Arabic. We also aim to further develop the website into a point-of-care application to assist clinicians in examining non-English speaking patients. We are grateful to the Texas Neurological Society for funding this project.