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Sporadic Creutzfeldt-Jakob Disease Presenting as Stroke and Wernicke Encephalopathy: A Report of Two Cases

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Abstract

Sporadic Creutzfeldt-Jakob disease (sCJD) is a rare and fatal condition whose early clinical heterogeneity often leads to misdiagnosis as more common, potentially manageable disorders. We report two retrospective cases of unique adult patients with probable sCJD whose initial presentations mimicked ischemic stroke and Wernicke encephalopathy with paraneoplastic autoimmunity. Case 1 involves a woman in her early 70s who was treated for a presumed ischemic stroke for several months before the onset of rapidly progressive dementia. Cortical ribboning on magnetic resonance imaging (MRI) and markedly elevated cerebrospinal fluid (CSF) total-tau and 14-3-3 with a positive real-time quaking-induced conversion (RT-QuIC) assay supported the diagnosis. Case 2 describes a woman in her late 50s initially treated for suspected Wernicke encephalopathy. Following a positive P/Q-type voltage-gated calcium channel (VGCC) antibody test, she was treated for paraneoplastic cerebellar degeneration before CSF RT-QuIC and 14-3-3 later confirmed sCJD. sCJD should remain on the differential for rapidly progressive neurological decline, even when early features suggest stroke, thiamine deficiency, or paraneoplastic autoimmunity.

Categories: Neurology
Keywords: diffusion-weighted imaging, positive real-time quaking-induced conversion (rt-quic) assay, rt-quic, sporadic creutzfeldt-jakob disease, stroke mimic, wernicke encephalopathy

Introduction

The spontaneous misfolding of normal cellular prion proteins into the PrPSc form is the cause of sporadic Creutzfeldt-Jakob disease (sCJD), an uncommon and consistently fatal neurodegenerative condition, with an estimated yearly frequency of almost one case per million and usually starting between the ages of 50 and 70 [1]. It typically presents with rapidly progressive cognitive impairment, often accompanied by myoclonus, cerebellar ataxia, visual disturbances, and pyramidal or extrapyramidal signs [2]. The main diagnostic tests are cerebrospinal fluid (CSF) real-time quaking-induced conversion (RT-QuIC), the most specific biomarker (specificity 99%-100%; sensitivity 73%-97% depending on assay generation); brain magnetic resonance imaging (MRI) with diffusion-weighted imaging (DWI), a highly sensitive imaging marker (sensitivity roughly 80%-98% across studies); and CSF 14-3-3 protein and total tau, which are less specific and are best used as adjuncts rather than as single diagnostic tests [2-4]. Patients with sCJD often progress to akinetic mutism or a vegetative state, losing the ability to move, speak, or respond to their surroundings [1, 2], and survival is about four months in classic cases but longer in some subtypes [1, 5]. Systemic infections such as aspiration pneumonia have been reported as the most common cause of death [5]. sCJD could mimic more common, sometimes treatable conditions, such as ischaemic stroke and Wernicke encephalopathy [6-7]. Each of these mimics can delay the diagnosis of a condition whose only treatment is supportive and palliative care. To show this variability, we report two cases reviewed for rapidly progressive neurological decline. Both cases were classified as probable sCJD based on the clinical course, MRI, and a positive CSF RT-QuIC.

Case Presentation

Case 1 was a woman in her early 70s whose only documented medical history was a COVID-19 infection and orthostatic hypotension (no syncope or hypoxic-ischemic event), who presented with acute-onset imbalance when walking and a tendency to fall backwards during exertion; the head CT was negative. After a three-day hospital admission, the work-up was reported as negative, but she regained partial mobility with the help of a walker (although she still fell backwards, even with it) and remained stable for four to six weeks until a sharp decline with frequent backward falls, inability to care for herself independently, development of urinary incontinence, and disorganized (“word salad”) speech. During an 11-day admission, an empiric treatment with carbidopa-levodopa produced a transient improvement in alertness. Brain MRI showed nonspecific hyperintensities in the bilateral basal ganglia, putamen, caudate, and thalami, as well as the frontal cortex, which could reflect either the sequela of hypoxic-ischemic insult or a “subacute spongiform encephalopathy” as an alternative (the first outside MRI had been read as suspected hypoxic-ischemic

encephalopathy, and the follow-up outside MRI report added subacute spongiform encephalopathy as an alternative).

She was discharged on dual antiplatelet therapy for 21 days and treated for a presumed stroke without a lumbar puncture. After a few more weeks in a rehabilitation center, she lost the ability to walk and stopped talking. Both the rehabilitation specialists and the outpatient neurologist believed that her rapid decline was not compatible with vascular etiology, so she was referred to our facility. On admission, she was awake and alert but expressed only one to three words at a time with neologisms, could not repeat, followed simple commands inconsistently, and was impulsive, grabbing at the examiner and nearby objects. Tone was increased in all limbs, reflexes were 3+, and a resting tremor was present in both arms. CT angiography of the head and neck showed no flow-limiting stenosis, dissection, or perfusion abnormality in a vascular territory. B12 and thyroid-stimulating hormone (TSH) were normal. Imaging on admission showed diffusion restriction with a cortical “ribboning” pattern (the initial MRI report described no acute abnormality, but on further review the next day the radiologist described bilateral diffusion hyperintensity in the caudate, putamen, and thalami with cortical ribbon signal in the cingulate and posterior frontal cortex, most consistent with Creutzfeldt-Jakob disease).

EEG was consistent with mild diffuse background slowing without any epileptiform discharges. Lumbar puncture showed an elevation of CSF total-tau protein (~19,500 pg/mL vs. a reference range of < 1,150) and 14-3-3 protein (~50,400 AU/mL vs. a reference range of < 2,000). The RT-QuIC test was also positive. A brief treatment with IV steroids (given empirically to cover potential autoimmune or inflammatory encephalopathy while send-out tests were pending) improved her energy level without affecting her neurological examination. The presumed ischemic stroke was a reasonable starting point at the outside hospital. There was an acute onset of sensory/motor imbalance and partial recovery, followed by plateaus over four to six weeks, a negative head CT (although this cannot rule out ischemia in the early phase), the outside MRIs interpreted as non-specific and more in keeping with hypoxic/ischemic injury, and antiplatelet treatment was started. There was no history of any vascular risk factors in this patient; there was no territorial perfusion abnormality on CT angiography.

Bilaterally involved signals of the cortex and deep nuclei did not correspond to one vascular territory and were stable since the previous outside MRI that was done about three months earlier, which is not usually true for infarction. Finally, the patient had been progressively deteriorating over weeks to months, and it was considered by the rehab team and the patient’s outpatient neurologist to be incompatible with an ischemic stroke. Hypoxic-ischemic injury was unlikely because of the presence of orthostatic hypotension without syncope and in the absence of any hypoxic-ischemic episode. As part of the differential diagnosis, white matter disease, atypical infection, and paraneoplastic syndrome were also considered, and the plans included MRI, EEG, serologies, paraneoplastic panel, and lumbar puncture. Prion disease (sCJD) was suspected due to diffusion restriction in the cortex and deep gray matter, as well as the clinical picture and CSF findings. As the clinical syndrome, imaging, and CSF biomarkers (including a positive RT-QuIC) met criteria for probable sCJD, the patient and her family decided to pursue comfort-focused care treatment. By discharge, she was mute, did not follow commands or move her limbs spontaneously, and had no myoclonus. She was discharged to a hospice care setting with a Do Not Resuscitate/Do Not Intubate order (Figure *1*).

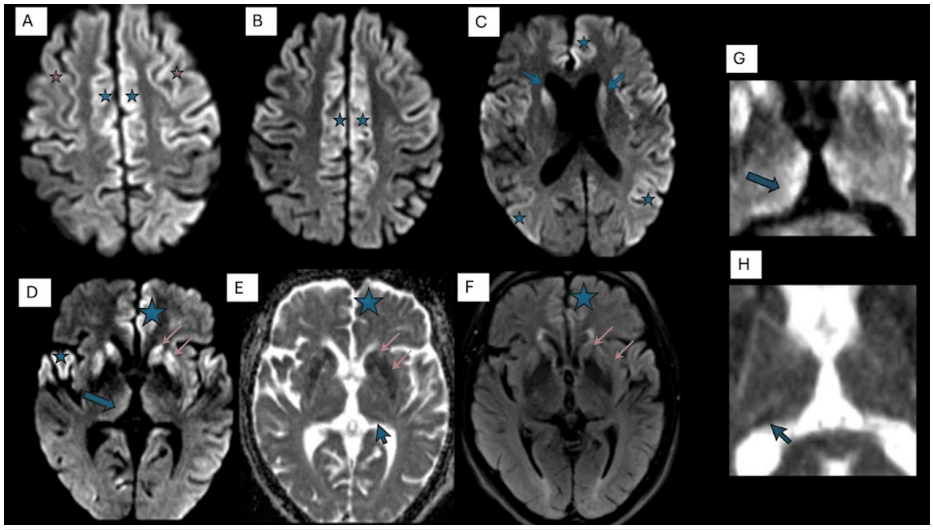


FIGURE 1: Brain MRI findings.

A. Axial diffusion-weighted image (DWI) at a high supraventricular level demonstrates asymmetrically increased signal throughout the neocortex, more pronounced in the bilateral frontal lobes (red asterisks) and cingulate gyri (blue asterisks). B. Axial DWI at the level of the cingulate gyri demonstrates asymmetrically increased signal throughout the neocortex, more pronounced in the cingulate gyri (blue asterisks). C. Axial DWI at the level of the caudate heads demonstrates diffuse asymmetric increased signal throughout the neocortex, including the cingulate gyri and temporo-occipital cortices (blue asterisks), with additional increased signal in the bilateral caudate heads (blue arrows). D–F. Corresponding axial images at the level of the basal ganglia. DWI (D) demonstrates diffuse asymmetric cortical restricted diffusion, more pronounced in the cingulate gyri and temporal lobes (blue asterisks), with additional involvement of the bilateral caudate nuclei and putamina (orange arrows). Restricted diffusion involving the bilateral dorsomedial thalamic nuclei and pulvinar demonstrates the hockey-stick sign. The corresponding apparent diffusion coefficient (ADC) map (E) demonstrates markedly decreased signal in the bilateral caudate nuclei and putamina (orange arrows), with additional decreased signal in the cerebral cortex (blue asterisk) and pulvinar (blue arrow), confirming true restricted diffusion. The corresponding T2-weighted fluid-attenuated inversion recovery image (F) demonstrates less pronounced hyperintense signal in the involved regions. G–H. Magnified axial DWI (G) and corresponding ADC map (H) at the level of the basal ganglia. The DWI demonstrates the hockey-stick sign, with increased signal involving the dorsomedial thalamic nuclei and pulvinar (blue arrow). The corresponding ADC map demonstrates matching decreased signal in the pulvinar (blue arrow).

Case 2 was a woman in her late 50s with a history of hypertension, hyperlipidemia, and poorly controlled type 2 diabetes who was admitted to an outside hospital with complaints of progressive gait instability and frequent falls, short-term memory impairment, and blurred vision for five weeks (symptom onset about two months before admission to our institution). At that facility, she was diagnosed with Wernicke encephalopathy and received high-dose intravenous thiamine with limited improvement. Thiamine was given several weeks after symptom onset, and toxic metabolic encephalopathy was also suspected. She denied excessive alcohol consumption, and serum vitamin B1, B12, folate, and vitamin D levels were normal when measured (after thiamine had been given; pre-treatment values were not available to us). The heavy metal screen, syphilis serology, and urine drug screen were negative. On imaging, she had a diffusion restriction in the bilateral caudate nuclei, as well as thalamic hyperintensity on T2-weighted sequences. The results of the serum autoimmune/paraneoplastic panel were positive for P/Q-type voltage-gated calcium channel (VGCC) antibodies (exact titer unavailable from the outside facility), and the patient was treated with intravenous methylprednisolone and plasmapheresis under the presumption of paraneoplastic cerebellar degeneration with some objective improvement in ataxia and dysmetria.

Chest, abdominal, and pelvic CT scans and transvaginal ultrasound showed no definite malignancy (the ovaries were not visualized on ultrasound). On admission to our institution (approximately two months after symptom onset), repeat imaging showed no changes but still showed diffusion restriction in the bilateral basal ganglia, thalami, and left > right mesial temporal lobes/hippocampus, as well as fluid-attenuated inversion recovery (FLAIR) hyperintensities in the periaqueductal gray matter with suspected cerebellar and mesial frontal cortical involvement. Figure *2* shows asymmetric cortical diffusion restriction, greater in the right hemisphere, involving the cingulate, temporal, and parietal cortex. EEG showed mild diffuse background slowing without epileptiform activity. The results of CSF studies for infection were unremarkable: normal cell count and glucose concentration (WBC 1, RBC 0; cultures and herpes simplex virus, varicella-zoster virus, West Nile virus, Epstein-Barr virus, HIV, hepatitis, and tuberculosis testing negative; cytology and flow cytometry not obtained because of inadequate CSF). The CSF autoimmune and paraneoplastic antibody panel was negative.

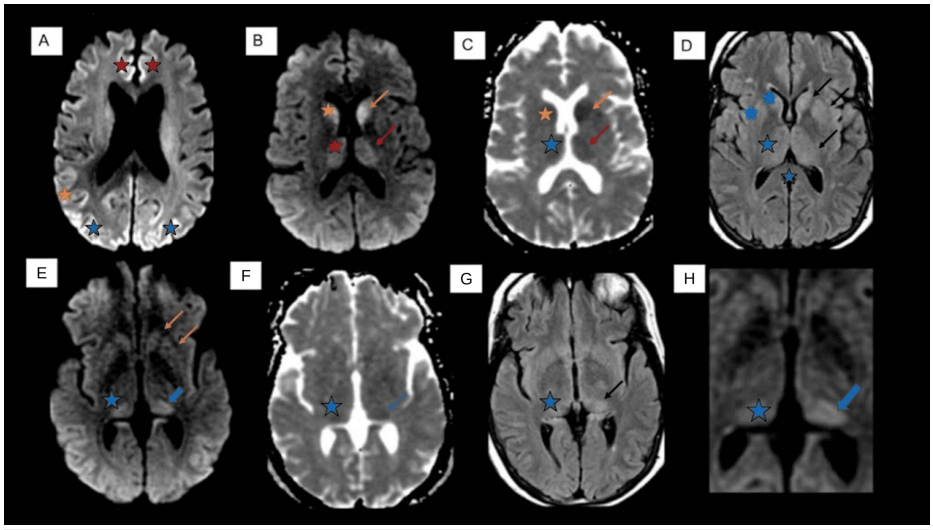


FIGURE 2: Brain MRI showing multifocal restricted diffusion in the cerebral cortex, basal ganglia, and thalami.

A. Axial diffusion-weighted image (DWI) at the level of the lateral ventricles shows diffuse, asymmetric cortical hyperintensity, more pronounced in the right hemisphere. The cingulate gyri (red asterisks), temporal cortex (orange asterisk), and parietal cortex (blue asterisks) are prominently involved. B-D. Corresponding axial images at the level of the basal ganglia. DWI (B) shows increased signal in the caudate nuclei (orange arrow) and putamina (red arrow), with the left greater than the right. The corresponding colored asterisks mark the less conspicuous contralateral involvement. The apparent diffusion coefficient (ADC) map (C) shows matching low signal in the same regions (arrows and asterisks), confirming restricted diffusion. The T2-weighted fluid-attenuated inversion recovery (FLAIR) image (D) shows less pronounced hyperintensity in the same regions, also left greater than right (black arrows and blue asterisks). E-G. Corresponding axial images at the level of the third ventricle. DWI (E) shows increased signal in the lentiform nuclei (orange arrows) and, more prominently, in the pulvinar nuclei of the thalami bilaterally (blue arrow, left; blue asterisk, right). The ADC map (F) shows matching low signal in the bilateral pulvinar nuclei (blue arrow, left; blue asterisk, right), confirming restricted diffusion. FLAIR (G) shows less pronounced hyperintensity in the bilateral pulvinar nuclei (black arrow, left; blue asterisk, right). H. Magnified axial DWI at the level of the thalami shows marked hyperintensity of the bilateral pulvinar nuclei (blue arrow, left; blue asterisk, right).

The CSF 14-3-3 protein concentration was markedly elevated (reported value 139,772 AU/mL vs a reference range < 2,000), and RT-QuIC was positive; CSF was sampled roughly two months after symptom onset. The positive serum VGCC antibodies did not explain her condition, and we discussed this with the patient and her family. There was no available disease-modifying treatment for this condition, so we recommended palliative care, and the patient and family wished to go home. Repeated imaging for monitoring the radiographic progression was suggested, but the patient and family wished to take time to decide, as it would not change management (Figure *2*). A summary of the clinical, EEG, imaging, and CSF findings for the two cases is provided in Table *1*.

Characteristic	Case 1	Case 2
Age/sex	Early 70s, female	Late 50s, female
Relevant comorbidities	None documented	Hypertension, hyperlipidemia, uncontrolled type 2 diabetes
Presenting feature	Gait imbalance, backward falls	Gait instability, ataxia, blurred vision, memory lapses
Initial working diagnosis	Ischemic stroke	Wernicke encephalopathy, then paraneoplastic cerebellar degeneration (serum P/Q-type VGCC antibody positive)
Disease course to sCJD suspicion	~3 months	~5–6 weeks
Key MRI finding	Cortical “ribboning” with basal ganglia/thalamic DWI change	Bilateral caudate and thalamic DWI change, later multifocal (basal ganglia, thalami, mesial temporal)
EEG	Mild diffuse background slowing, no epileptiform activity	Mild diffuse background slowing, no epileptiform activity
CSF 14-3-3/RT-QuIC	Markedly elevated 14-3-3 and total tau; RT-QuIC positive	Elevated 14-3-3; RT-QuIC positive
Disposition	Hospice (facility-based)	Palliative care, discharged home

TABLE 1: Comparative summary of the two cases

CSF: cerebrospinal fluid; DWI: diffusion-weighted imaging; RT-QuIC: real-time quaking-induced conversion; VGCC: voltage-gated calcium channel

Discussion

Applying the RT-QuIC-amended diagnostic criteria [2], Cases 1 and 2 met criteria for probable sCJD: a rapidly progressive neuropsychiatric syndrome with a positive CSF RT-QuIC, supported by typical MRI findings and elevated CSF 14-3-3 (and total-tau in Case 1). Their presenting diagnoses varied widely: stroke and Wernicke encephalopathy/paraneoplastic autoimmunity, respectively. Published cases show how variable the presentation of sCJD can be (Table *2*). No family history of dementia or prion disease was documented in either of the two records, but PRNP (Prion Protein gene) was not sequenced, so “sporadic” is used on clinical grounds. Case 1 shows the known overlap between sCJD and stroke. Like previously reported sCJD cases misdiagnosed as cerebrovascular disease [6, 8], distinguishing features included an atypical pattern of progression, ongoing deterioration as opposed to the usual post-stroke course, and an MRI pattern of basal ganglia, thalamus, and cortex involvement that does not fit within a single vascular territory. In this patient, admission MRI was initially reported as showing no acute abnormality, and the cortical and deep-nuclei diffusion restriction was recognized on re-review the next day: the diagnosis therefore rested on recognizing the cortical and deep-nuclei diffusion restriction as a prion pattern together with the CSF results (markedly elevated total-tau and 14-3-3 and a positive RT-QuIC), rather than on serial imaging. This resembles the reports of Oliver et al. and Al Taie et al., in which the diagnosis was established by autopsy or by clinical follow-up and prion-specific testing rather than by the initial scan [8, 9].

Reported case	Presentation	Initial misdiagnosis	How sCJD was confirmed
74-year-old woman, stroke-like decline [8]	Three-month history of stroke-like deficits, progressive decline	Ischemic stroke	Autopsy: spongiform encephalopathy, no pathologic evidence of infarct
56-year-old man, Irish tertiary center [9]	Waxing/waning hemiparesis with a non-corresponding parietal DWI lesion	Acute ischemic stroke	Clinical trajectory plus prion-specific workup
58-year-old man [10]	Cognitive decline, motor and behavioral change after alcohol cessation	Wernicke encephalopathy	Cortical DWI ribboning, positive RT-QuIC, elevated 14-3-3
MM1-subtype autopsy case [11]	Rapidly progressive dementia	CJD was diagnosed pre-mortem; Wernicke was not suspected	Autopsy: co-existing CJD and Wernicke encephalopathy pathology

TABLE 2: Comparison with representative published cases

sCJD: sporadic Creutzfeldt-Jakob disease; DWI: diffusion-weighted imaging; RT-QuIC: real-time quaking-induced conversion

Case 2 presents two possible mimics: sCJD and Wernicke encephalopathy both cause ataxia, change in mental status, and sometimes overlapping MRI findings, with publications of sCJD patients who were empirically managed as Wernicke encephalopathy until CSF biomarkers or other findings led to the correct diagnosis [7, 10]; the overlap also runs in the opposite direction, as Wernicke encephalopathy has been mistaken for CJD [12], and an autopsied case showed coexisting CJD and Wernicke pathology in the same patient [11], indicating that lack of response to thiamine should prompt ordering DWI-MRI and CSF RT-QuIC as opposed to increased thiamine administration. Second, this case had P/Q-type VGCC antibodies, a less commonly described confounder. While VGCC antibodies are typically associated with paraneoplastic cerebellar degeneration and Lambert-Eaton myasthenic syndrome, most often seen in association with small cell lung cancer, in a series of paraneoplastic CNS disorders, VGCC antibodies were absent in cerebellar degeneration cases without small cell lung cancer but present in 12% of small cell lung cancer patients with paraneoplastic encephalomyelitis/sensory neuronopathy [13]; this patient had a negative malignancy workup. CSF RT-QuIC and 14-3-3 supported sCJD, suggesting that VGCC positivity was either incidental or due to nonspecific immune activation rather than a paraneoplastic process. The partial improvement in ataxia after methylprednisolone and plasma exchange, which likely reinforced the autoimmune diagnosis, is not expected in sCJD; it may reflect fluctuation, a nonspecific effect, or a coexisting immune-mediated component, and it cannot be resolved from a single case. It demonstrates that a partial response to immunotherapy should not end the search for an alternative cause when the course remains progressive.

DWI-MRI was abnormal in both cases, but two patients cannot establish which test performs best. In Case 1, the initial admission MRI was read as showing no acute abnormality, and in both cases the CSF markers were strongly abnormal, whereas EEG showed only nonspecific slowing. The “hockey stick” (dorsomedial thalamic and pulvinar) signal seen in Figure *1* is classically linked to variant CJD (vCJD), in which a pulvinar signal brighter than the anterior putamen is a strong indicator [2, 14]. vCJD is very unlikely here: the patient was in her seventies, whereas vCJD typically affects much younger patients; the thalamic signal accompanied prominent cortical ribboning and striatal involvement rather than standing out as the dominant finding; and thalamic (including pulvinar) involvement also occurs in sCJD, particularly in the VV2 and MV2 subtypes [2]. The same considerations apply to the pulvinar signal in Case 2. Larger studies indicate that MRI and CSF 14-3-3/tau are sensitive but less specific, whereas RT-QuIC is highly specific [2, 15]; a positive RT-QuIC supported the diagnosis in both of our cases despite nonspecific EEG or initial MRI findings. In both cases, once sCJD was considered probable, care focused on palliation because no disease-modifying treatment is available [2].

Limitations

This report has been done retrospectively using routinely generated medical records and has not been based on prospectively collected protocol-specific data. Also, independent re-review of the original imaging was not available in all cases. At the time of the last documented record, no genetic testing (PRNP codon 129 polymorphism) or neuropathological/autopsy confirmation was available for either of the patients, so by current criteria, both cases should be considered as "probable" rather than "definite" sCJD. No family history of prion disease was documented in the records reviewed, so the term “sporadic” rests on clinical grounds. In Case 2, pre-treatment thiamine levels and the exact outside serum VGCC antibody titer were not available.

Conclusions

sCJD remains an important but rare differential diagnosis of rapidly progressive neurological disease since its early presentation is very similar to ischemic stroke, thiamine deficiency, or paraneoplastic autoimmunity. Both described cases were first treated as the mimicking condition until the clinical course and MRI raised suspicion of sCJD, which CSF biomarkers then supported. Keeping a low threshold to perform DWI-MRI and CSF RT-QuIC/14-3-3 testing even when another diagnosis explains the presentation can help to establish the diagnosis faster and initiate appropriate palliative care.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. University of Florida Institutional Review Board (UF IRB) and UF Privacy Office issued approval (N/A; exempt per institutional policy). As both patients are deceased, neither written informed consent nor a formal waiver of consent could be obtained for the cases in this series. The authors made multiple documented attempts via telephone and messages to contact the listed next of kin for each patient using the contact information on file in the medical record but received no response. In accordance with the written policies of the University of Florida Institutional Review Board (UF IRB) and the UF Privacy Office (the institution where the patients were treated), retrospective case series of this size using de-identified clinical data do not constitute human subjects research and do not require formal IRB review, a formal consent waiver, or patient authorization. All patient data, clinical histories, and neuroimaging have been rigorously de-identified in compliance with HIPAA regulations to protect patient anonymity. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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