IJCI International Journal of Clinical Innovation
ijcinnovation.org
VERSION OF RECORD

CASE REPORT

Sporadic Creutzfeldt- Jakob Disease: Case Series of Refractory Convulsive Status Epilepticus and Ataxic Variant in a Health Care Provider

Enzo M. Fortuny, MD [1]*; Laura Alejandra Ochoa Torrico, MD [1]; Mario Camargo Villarreal, MD [1]; Erick Gonzalez Delgado, MD [1]; Fatima Urquieta Zabala, MD [1]; Andres Camargo Jordan, MD [1]

[1] Clinica Medical Neuro Center, Santa Cruz de la Sierra, Bolivia

* Corresponding author: Enzo M. Fortuny | enzofv89@gmail.com | ORCID https://orcid.org/0000-0001-9270-861X

PUBLICATION

Article ID:	IJCI-2026-0006
Specialty:	Neurology
Issue:	Volume 1, Issue 1

ARTICLE HISTORY

Received:	July 15, 2026
Accepted:	September 7, 2026
Published:	September 8, 2026

ABSTRACT

BACKGROUND:

Sporadic Creutzfeldt-Jakob disease (sCJD) is a rare, rapidly progressive, and fatal prion disease characterized by cognitive decline, myoclonus, cerebellar dysfunction, and characteristic neuroimaging abnormalities. Seizures are uncommon, and refractory convulsive status epilepticus represents a particularly rare complication. Case Presentations: The first case involved a 35-year-old man with several months of behavioral changes, sleep disturbances, gait instability, ataxia, dysarthria, and generalized myoclonus. Brain MRI demonstrated bilateral caudate and basal ganglia diffusion abnormalities with cortical involvement, and CSF 14-3-3 testing provided supportive evidence for probable sCJD. On hospital day 3, he developed recurrent generalized tonic-clonic seizures progressing to refractory convulsive status epilepticus requiring intubation, continuous EEG monitoring, levetiracetam, phenobarbital, midazolam, and propofol for seizure control. The second case involved a 50-year-old man with progressive sleep and behavioral disturbances followed by dysarthria and profound ataxia. MRI demonstrated bilateral caudate and lenticular hyperintensities with pulvinar and medial thalamic involvement producing a hockey-stick pattern. CSF 14-3-3 was positive, supporting a clinical suspicion of sCJD, although RT-QuIC and neuropathological confirmation were unavailable.

CONCLUSIONS:

These cases illustrate two clinically suspected presentations of sCJD, including one complicated by refractory convulsive status epilepticus and another with an ataxic-predominant phenotype. Acute deterioration, new seizure activity, or worsening myoclonus in patients with suspected sCJD should prompt EEG evaluation because status epilepticus, although rare, may require aggressive neurocritical care management.

KEYWORDS

Creutzfeldt Jakob Disease; Prion Disease; Encephalitis; Status Epilepticus; Case Report

HOW TO CITE

Enzo M. Fortuny, Laura Alejandra Ochoa Torrico, Mario Camargo Villarreal, Erick Gonzalez Delgado, Fatima Urquieta Zabala, Andres Camargo Jordan. Sporadic Creutzfeldt- Jakob Disease: Case Series of Refractory Convulsive Status Epilepticus and Ataxic Variant in a Health Care Provider. Int J Clin Innov. 2026;1(1).

Publication information

AUTHOR IDENTIFIERS

Enzo M. Fortuny | Email: enzofv89@gmail.com | ORCID: https://orcid.org/0000-0001-9270-861X

Laura Alejandra Ochoa Torrico | Email: lauraochoat22@gmail.com | ORCID: https://orcid.org/0009-0007-0502-9872

Mario Camargo Villarreal | Email: drmariocamargo@gmail.com | ORCID: https://orcid.org/0009-0007-4124-2400

Erick Gonzalez Delgado | Email: eglezmd@gmail.com | ORCID: https://orcid.org/0009-0000-1258-7775

Fatima Urquieta Zabala | Email: urquieta.f@gmail.com

Andres Camargo Jordan | Email: camargojordan.andres@gmail.com | ORCID: https://orcid.org/0009-0001-9288-475X

FUNDING

This study was not supported by any sponsor or funder.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest

AUTHOR CONTRIBUTIONS

Enzo M. Fortuny; Writing original and revised drafts. Andres Jordan Camargo; Data Collection and clinical management. Erick Gonzalez; Data Collection and patient management. Laura Alejandra Ochoa Torrico; Analysis, supervision, data collection, writing - review and editing. Fatima Urquieta Zabala; Analysis, supervision, data collection. Mario Camargo; Conceptualization.

DATA AVAILABILITY

The data that support the findings of this study are not publicly available due to patient and family privacy concerns but are available from the corresponding author upon reasonable request.

ETHICS

Not Required / Not Applicable

CONSENT

Obtained

Probable and Suspected Sporadic Creutzfeldt-Jakob Disease: Case Series of Refractory Convulsive Status Epilepticus and an Ataxic-Predominant Presentation

Case Series

Running title: Sporadic CJD With Status Epilepticus

Enzo M. Fortuny; Andres Camargo Jordan; Erick Gonzalez Delgado; Laura Alejandra Ochoa Torrico; Mario Camargo; Fatima Urquieta Zabala

Clinica Medical Neuro Center, Santa Cruz de la Sierra, Bolivia

Corresponding author: Enzo M. Fortuny — enzofv89@gmail.com — +591-76852960

ORCID: 0000-0001-9270-861X

Abstract

Background: Sporadic Creutzfeldt-Jakob disease (sCJD) is a rare, rapidly progressive, and fatal prion disease characterized by cognitive decline, myoclonus, cerebellar dysfunction, and characteristic neuroimaging abnormalities. Seizures are uncommon, and refractory convulsive status epilepticus represents a particularly rare complication.

Case Presentations: The first case involved a 35-year-old man with several months of behavioral changes, sleep disturbances, gait instability, ataxia, dysarthria, and generalized myoclonus. Brain MRI demonstrated bilateral caudate and basal ganglia diffusion abnormalities with cortical involvement, and CSF 14-3-3 testing provided supportive evidence for probable sCJD. On hospital day 3, he developed recurrent generalized tonic-clonic seizures progressing to refractory convulsive status epilepticus requiring intubation, continuous EEG monitoring, levetiracetam, phenobarbital, midazolam, and propofol for seizure control. The second case involved a 50-year-old man with progressive sleep and behavioral disturbances followed by dysarthria and profound ataxia. MRI demonstrated bilateral caudate and lenticular hyperintensities with pulvinar and medial thalamic involvement producing a hockey-stick pattern. CSF 14-3-3 was positive, supporting a clinical suspicion of sCJD, although RT-QuIC and neuropathological confirmation were unavailable.

Conclusions: These cases illustrate two clinically suspected presentations of sCJD, including one complicated by refractory convulsive status epilepticus and another with an ataxic-predominant phenotype. Acute deterioration, new seizure activity, or worsening myoclonus in patients with suspected sCJD should prompt EEG evaluation because status epilepticus, although rare, may require aggressive neurocritical care management.

Keywords: Creutzfeldt Jakob Disease, Prion Disease, Encephalitis, Status Epilepticus, Case Report

Introduction

Creutzfeldt Jakob Disease (CJD) is a rare, rapidly progressive and fatal neurodegenerative disorder within the family of spongiform encephalopathies. It has an estimated incidence of 1 case per million/year. The disease is caused by prions, or proteinaceous infectious particles (PrP) homologous to the cellular prion protein (PRPc) which is highly expressed in human central nervous system cells under normal conditions. Contact with prions induces a series of conformational changes in PRPc and conversion to PRPsc , followed by templated polymerization resulting in loss of function and neuronal loss(1). This process later results in astrocytic and microglial activation which contributes to the spongiform degeneration seen on histopathology. CJD has an unrelenting clinical course and is invariably fatal, with 70% of patients dying within the first year of diagnosis. Six clinical and pathological forms of Sporadic Creutzfeldt-Jakob disease (sCJD) have been described, each relating to distinct molecular features: MM1, MM2, MV1, MV2, VV1, VV2. VV2 has been associated with an ataxic form of sCJD, known as the Brownell-Oppenheimer variant, which accounts for 10% of cases. This form is characterized by predominantly ataxic symptoms and classic EEG findings are usually absent.

The most frequent clinical presentation is a prodromal phase characterized by behavioral changes, followed by rapidly progressive dementia accompanied by myoclonus, ataxia, aphasia and akinetic mutism. (2, 3). Imaging findings may be variable depending on specific molecular subtypes of the disease, but characteristic MRI findings include cortical ribboning, pulvinar changes and basal

ganglia hyperintensities on DWI or T2/FLAIR(4). Definitive diagnosis requires confirmation via biopsy or autopsy, but these represent significant logistical challenges due to the difficulty of eradicating prions from surgical equipment with standard sterilization procedures, in addition to exposure risks in healthcare personnel. Fortunately, significant improvements in molecular biology techniques have facilitated this disease's work up. CSF assays for protein 14-3-3 can provide supportive evidence for CJD in the appropriate clinical context; however, 14-3-3 is not disease-specific and may be elevated in other causes of rapid neuronal injury(5). Seizures are rare in sCJD and represent an atypical presentation (approximately 10% of cases), and have scarcely been described in the literature(6). Seizures in this population may manifest as generalized tonic-clonic seizures, focal motor seizures, and in rare cases, convulsive or non-convulsive status epilepticus (NCSE)(6). Sleep disorders, such as sleep talking and walking have also been described in sCJD, often times preceding the onset of cognitive and motor symptoms. The following case series describes a young patient with probable sCJD whose hospital admission was complicated by refractory convulsive SE requiring treatment with multiple anti-epileptic drugs (AED's) as well as orotracheal intubation and sedation with propofol. The second case describes a patient presenting with frequent and worsening parasomnias along with mood and behavioral changes, followed by motor and cognitive symptoms more commonly seen in sCJD

Case Presentation

Case #1

35-year-old man with no past medical or family history presents due to 4 months of bilateral upper and lower extremity weakness, ataxia, dysarthria and vertigo. Patient is a healthcare provider at a rural hospital who routinely performs obstetric and gynecological procedures. His family reports worsening depression and anxiety in the months preceding the other symptoms. These mood disorders were accompanied by progressively worsening sleep talking and somnambulism that began at the same time. Patient was evaluated 3 months ago at an outside hospital where a CT head was unremarkable, as well as brain MRI/MRA which revealed a small area of possible DWI

restriction in the bilateral caudate nuclei, but otherwise within normal limits. He was subsequently discharged after the remainder of his work up was negative. He presents to our hospital for reevaluation for persistent dysarthria, ataxia and gait instability. On physical examination, patient is alert, oriented only to self and place. Patient exhibits right-left confusion, bilateral dysmetria and ataxia. Generalized myoclonus with decreased muscle tone is noted on all extremities. Motor strength is 3/5 on upper extremities and 2/5 on lower extremities. Complete blood count (CBC), comprehensive metabolic panel (CMP) and urine toxicology screening were within normal limits. Patient was admitted for evaluation. Repeat MRI brain was performed and revealed DWI hyperintensities in the basal ganglia and diffuse cortical edema **(Image 1).**

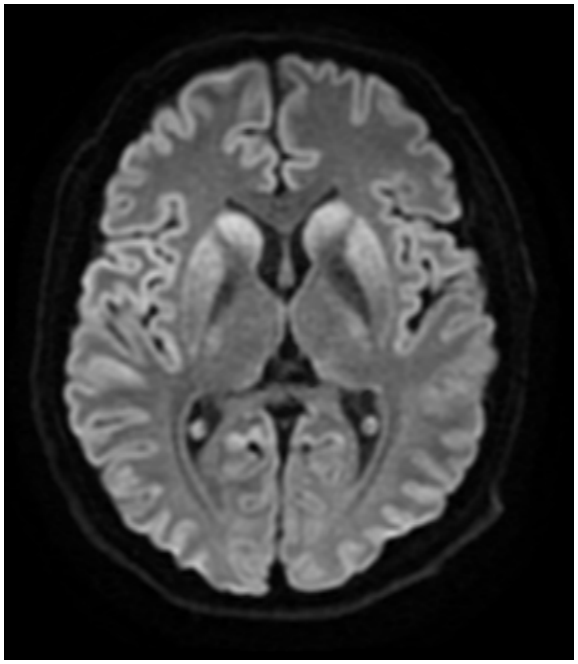

Image 1. MRI brain without contrast obtained on admission. Axial slice on DWI sequence demonstrates bilateral hyperintensities of the caudate and striatal nuclei along with insular cortical ribboning.

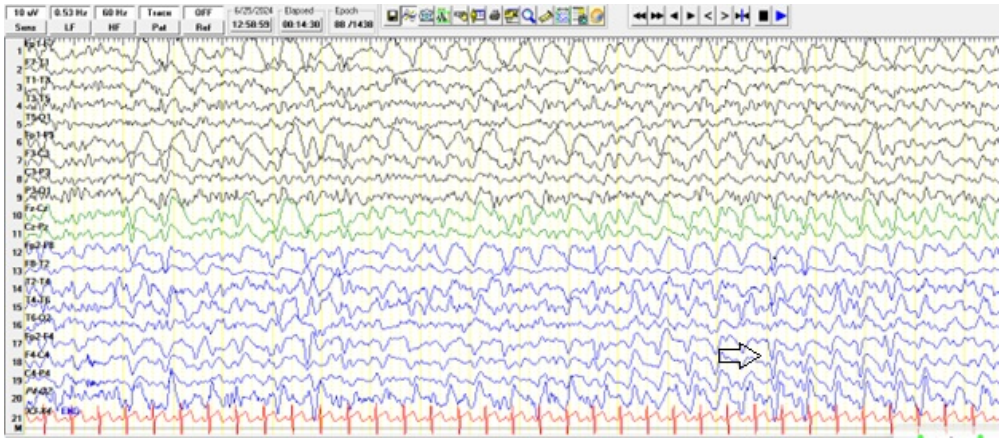

Image 2. EEG: Frequent bursts of synchronous bilateral anterior rhythmic delta activity with periodic right-sided lateralized discharges with triphasic morphology (arrow).

EEG was performed and revealed generalized slowing with frequent bursts of synchronous bilateral anterior rhythmic delta activity and periodic right-sided lateralized discharges with triphasic morphology **(Image 2).** Lumbar puncture was initially unremarkable, with normal CSF chemistry, negative gram stain, and negative bacterial/viral/fungal cultures. He was started on methylprednisolone 1 g/day IV for 5 days due to suspected autoimmune encephalitis while awaiting CSF results. On post admission day 3, patient exhibited an episode of generalized tonic clonic seizures, lasting approximately 10 minutes. He was treated with Midazolam 10 mg IV with temporary seizure control, but recurred minutes after and experienced two more episodes of tonic clonic seizures. He was loaded with Levetiracetam 3 g IV, followed by 1 g every 12 hours, and upgraded to the ICU on continuous EEG where he required orotracheal intubation as his seizures persisted. Patient was started on a phenobarbital infusion at 20 mg/kg due to breakthrough seizures despite treatment with levetiracetam and midazolam IV boluses. Continuous EEG continued to show breakthrough seizures, and control was eventually obtained by adding a continuous IV propofol infusion until burst suppression was achieved. Autoimmune encephalitis panel obtained during initial lumbar puncture returned negative. A Protein 14-3-3 assay in CSF was ordered and was positive (53,100 U/mL; reference <1,450 U/mL), providing supportive evidence for sCJD in the appropriate clinical context. Patient required vasopressor support due to hypotension despite adequate fluid balance, likely secondary to profound sedation with phenobarbital and

propofol. He was later transferred to an outside hospital on Post admission day #7 at the request of his family due to insurance coverage purposes. Patient was intubated, hemodynamically stable with no visible seizures on EEG at the time of transfer. One year after discharge from the outside hospital, he's continued to experience cognitive and neurological decline, eventually requiring tracheostomy and percutaneous endoscopic gastrostomy tube placement. Patient is currently unresponsive in persistent vegetative state.

Case #2

50-year-old man with no known past medical history or family history, who works as a motorcycle salesman, presents as a referral from an outside hospital. History is provided by the patient's partner. She reports that his symptoms began 2 months prior to presentation with progressively worsening changes in sleep behavior. She reports that the patient began talking in his sleep and subsequently sleepwalking. When awake, he would often report vivid dreams and nightmares. He subsequently began to experience changes in his behavior, becoming more irritable and ill-tempered with his partner. 1 month prior to presentation, he began to experience dysarthria and difficulty swallowing. On physical examination, patient is awake and alert, but confused to place and time. Horizontal nystagmus is noted on lateral gaze. Patient follows commands with prompting, and motor strength is intact despite reporting subjective lower extremity weakness. There was no clonus or fasciculations. Gait examination revealed a profoundly ataxic gait. Babinski sign was negative. CBC and CMP at presentation were unremarkable. FLAIR sequence on MRI brain obtained after arrival demonstrates bilateral hyperintensities of the caudate and lenticular nuclei, in addition to pulvinar hyperintensities in a hockey-stick pattern **(Image 3).** EEG performed during admission was unremarkable. A lumbar puncture was performed with suspicion for autoimmune/infectious encephalitis. CSF chemistry, gram stain and bacterial/viral cultures were negative, and he was started on methylprednisolone 1 g/day IV for 5 days with a presumptive diagnosis of autoimmune encephalitis. Patient's symptoms remained unchanged following the methylprednisolone pulse. Autoimmune encephalitis panel returned negative, while Protein 14-3-3

assay in CSF was positive (23,400; laboratory unit not specified in the available record), raising sCJD as the primary diagnosis. He had an otherwise unremarkable hospital stay, and was subsequently discharged home with family care. Patient has continued to decline neurologically. At this time, He is bed bound and requires 24-hour assistance with activities of daily living. His dysphagia has continued to progress and is now pending gastrostomy tube placement. Repeat EEG performed at an outside hospital during follow up remains unremarkable despite disease progression.

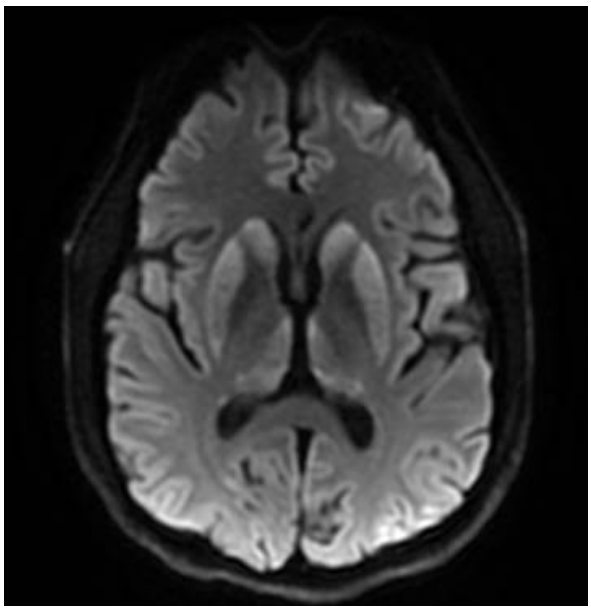

Image 3. MRI brain without contrast obtained during admission reveals bilateral symmetric caudate and lenticular nuclei hyperintensity in FLAIR sequence. There is bilateral pulvinar and medial thalamus hyperintensity producing a hockey-stick pattern.

Discussion

Diagnostic certainty in sporadic CJD (sCJD) can be categorized into definite, probable, and possible disease, based on clinical and laboratory criteria proposed by the WHO, as well as more recent revised criteria proposed by Hermann et al(7-9). Definite diagnosis requires neuropathological testing via autopsy or biopsy followed by immunocytochemical or western blot testing. This however poses significant logistical difficulties in most institutions due to the strict biosecurity protocols required to safely perform the procedure. Case 1 meets criteria for probable sCJD based on its rapidly progressive cognitive decline, myoclonus, cerebellar signs, characteristic

MRI abnormalities, and positive CSF protein 14-3-3. Case 2 is clinically strongly suggestive of sCJD but does not strictly fulfill the 2017 criteria for probable sCJD because only one qualifying clinical feature (cerebellar signs) is clearly documented and RT-QuIC was unavailable(4, 8). Neuropsychiatric manifestations such depression and anxiety, especially if accompanied by rapid cognitive decline have been described in early disease onset. Both of the patients described above initially manifested with sleep disturbances and behavioral changes as perceived by their partners. Both patients later developed notable ataxia and dysarthria, consistent with cerebellar and basal ganglia involvement frequently seen in sCJD(1, 4). Bilateral caudate nuclei hyperintensity seen on DWI and T2 sequences on MRI brain is a classic radiological finding that is also present in these patients. A positive CSF protein 14-3-3 assay in the setting of a negative autoimmune encephalitis panel supports the diagnosis of prion disease in the appropriate clinical context, but 14-3-3 is a nonspecific marker of rapid neuronal injury and is not diagnostic by itself (5, 8). The differential diagnoses for patients presenting with rapidly progressive cognitive decline, movement disorders and seizures may include infectious meningoencephalitis, autoimmune/ paraneoplastic encephalitis, with other conditions such as Alzheimer’s disease, Corticobasal degeneration, Frontotemporal dementia as other possible but less likely causes of rapidly progressive symptoms(10). Real time Quaking induced Conversion (RT- QuIC) is a novel laboratory technique that is highly accurate at diagnosing Creutzfeldt-Jakob Disease, with an approximate sensitivity of 92% and a specificity of 100%(8, 11). This technique relies on detecting PrP using fluorescent dyes(11). This test however is still not widely available and could not be used in this patient for this reason. Status Epilepticus (SE) is a rare but potentially fatal complication that has been described in CJD(2, 10). SE in sCJD most commonly manifests as Non-convulsive SE (NCSE), with Convulsive Status Epilepticus and Epilepsia partialis continua being rarer. The patient described in this case experienced severe convulsive SE refractory to first- and second-line treatment, requiring sedation with propofol and phenobarbital to achieve seizure control. Because of its rarity, seizure disorders and SE in prion diseases have never been studied rigorously and management remains challenging. The profound molecular and synaptic structural changes induced in sCJD likely result

in poor response to antiepileptic drugs, a phenomenon that has been described in other neurodegenerative conditions such as Alzheimer's and Parkinson's disease, where seizure disorders are also more common(12). Neurologist and Neuro intensivists must remain vigilant of patients with sCJD as seizures may often manifest as NCSE without overt convulsions. Our patient exhibited generalized myoclonus on presentation, and focal motor seizures may be confused by worsening myoclonus in some patients. Changes on neurological exam should prompt evaluation with EEG. Although seizure activity can produce peri-ictal DWI restriction and T2/FLAIR hyperintensities(13, 14), the characteristic basal ganglia and cortical abnormalities in this patient were documented on MRI before the onset of status epilepticus, making a peri-ictal explanation unlikely. The patient meets criteria for probable CJD under the revised 2017 criteria based on rapidly progressive dementia with myoclonus and cerebellar signs together with characteristic MRI findings and positive CSF protein 14-3-3(8). The EEG showed triphasic/periodic discharges, but these should not be equated with the typical CJD EEG criterion of generalized periodic sharp-wave complexes. Seizures in this patient population may pose a considerable therapeutic challenge requiring general anesthesia for seizure control. Our second case was characterized by prominent ataxia and dysarthria, suggesting dysfunction of the cerebellum or its pathways. Brain MRI revealed classic findings of sCJD including T2 hyperintensities of the basal ganglia, medial thalamus and pulvinar nuclei, with no signs of cerebellar atrophy or signal changes(15). These clinical findings are consistent with an ataxic-predominant phenotype of suspected sCJD; however, a specific molecular subtype could not be established in the absence of PRNP genotyping or neuropathological confirmation. Periodic sharp-wave complexes often seen in other forms of sCJD may be absent in ataxic-predominant presentations, which was the case with this patient who had a normal EEG despite prominent motor symptoms. In both of these cases, diagnosis was delayed considerably as CSF Protein 14-3-3 assay is not available at our center, and requires samples to be sent to a laboratory abroad. More advanced molecular testing such as RT-QuIC assay are very highly specific for sCJD but unavailable at our center, as well as genetic testing to identify human PRNP gene mutations.

Conclusion

SE is a rare but potentially life-threatening complication in sCJD. Since SE most frequently manifests as NCSE in sCJD, physicians must have a high index of suspicion in patients who experience an acute change in mental status or sudden worsening of baseline myoclonus.

Conflict of Interest

The authors declare no conflicts of interest.

Consent for Publication Statement

Informed consent and consent for publication were obtained by the patient's next of kin and uploaded as supplementary materials

Funding Resources

This study was not supported by any sponsor or funder.

Statement of Ethics

This study was performed in accordance with the Declaration of Helsinki. According to applicable local and national guidelines, ethical approval was not required for this case series. Written informed consent was obtained from both patients' next of kin. Patient confidentiality was maintained by removing all identifiable information from the manuscript. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material.

Author Contributions

Enzo M. Fortuny; Writing original and revised drafts. Andres Camargo Jordan; Data Collection and clinical management. Erick Gonzalez Delgado; Data Collection and patient management. Laura Alejandra Ochoa Torrico; Analysis, supervision, data collection, writing – review and editing. Fatima Urquieta Zabala; Analysis, supervision, data collection. Mario Camargo; Conceptualization.

Data Availability Statement

The data that support the findings of this study are not publicly available due to patient and family privacy concerns but are available from the corresponding author upon reasonable request.

References

1. Ritchie DL, Smith C. Pathological spectrum of sporadic Creutzfeldt–Jakob disease. Pathology. 2025;57(2):196-206.

2. Bhatia P, Sonbol M, Jain D, Rincon-Flores N, Frontera A. Status Epilepticus in Creutzfeldt-Jakob Disease: A Case Report of an Unusual Presentation. Cureus. 2022;14(6):e26470.

3. Holt C, Sadek AT, Woodard J, Grinstead R. An Atypical Presentation of Sporadic Creutzfeldt-Jakob Disease in the Setting of Chronic Alcohol Use Disorder. Case reports in neurological medicine. 2025;2025:1332654.

4. Fragoso DC, Gonçalves Filho ALdM, Pacheco FT, Barros BR, Aguiar Littig I, Nunes RH, et al. Imaging of Creutzfeldt-Jakob Disease: Imaging Patterns and Their Differential Diagnosis. RadioGraphics. 2017;37(1):234-57.

5. Muayqil T, Gronseth G, Camicioli R. Evidence-based guideline: Diagnostic accuracy of CSF 14-3-3 protein in sporadic Creutzfeldt-Jakob disease. Neurology. 2012;79(14):1499-506.

6. Katsikaki G, Dagklis IE, Angelopoulos P, Ntantos D, Prevezianou A, Bostantjopoulou S. Atypical and early symptoms of sporadic Creutzfeldt - Jakob disease: case series and review of the literature. Int J Neurosci. 2021;131(9):927-38.

7. Uttley L, Carroll C, Wong R, Hilton DA, Stevenson M. Creutzfeldt-Jakob disease: a systematic review of global incidence, prevalence, infectivity, and incubation. The Lancet Infectious Diseases. 2020;20(1):e2-e10.

8. Watson N, Hermann P, Ladogana A, Denouel A, Baiardi S, Colaizzo E, et al. Validation of Revised International Creutzfeldt-Jakob Disease Surveillance Network Diagnostic Criteria for Sporadic Creutzfeldt-Jakob Disease. JAMA Network Open. 2022;5(1):e2146319-e.

9. Shimamura MI, Satoh K. Challenges and Revisions in Diagnostic Criteria: Advancing Early Detection of Prion Diseases. Int J Mol Sci. 2025;26(5).

10. Kojima G, Tatsuno BK, Inaba M, Velligas S, Masaki K, Liow KK. Creutzfeldt-Jakob disease: a case report and differential diagnoses. Hawaii J Med Public Health. 2013;72(4):136-9.

11. Green AJE. RT-QuIC: a new test for sporadic CJD. Pract Neurol. 2019;19(1):49-55.

12. Neri S, Mastroianni G, Gardella E, Aguglia U, Rubboli G. Epilepsy in neurodegenerative diseases. Epileptic Disorders. 2022;24(2):249-73.

13. Katramados AM, Burdette D, Patel SC, Schultz LR, Gaddam S, Mitsias PD. Periictal diffusion abnormalities of the thalamus in partial status epilepticus. Epilepsia. 2009;50(2):265-75.

14. Hufnagel A, Weber J, Marks S, Ludwig T, De Greiff A, Leonhardt G, et al. Brain diffusion after single seizures. Epilepsia. 2003;44(1):54-63.

15. Jurcau MC, Jurcau A, Diaconu RG, Hogea VO, Nunkoo VS. A Systematic Review of Sporadic Creutzfeldt-Jakob Disease: Pathogenesis, Diagnosis, and Therapeutic Attempts. Neurol Int. 2024;16(5):1039-65.