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Creutzfeldt-Jakob as an example of Prion Disease: III. Prion-mimic Diseases

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Abstract

Physicians caring for patients with rapidly progressive brain conditions need to be aware of sporadic Creutzfeldt-Jakob disease (CJD) but should consider other potential treatable neurological disorders before making the diagnosis of this rare, inexorable, and untreatable disease. Data are provided that may help physicians identify patients for whom life-extending treatment can, and should, be given. In particular, the neuropathological study that had indicated that heightened awareness of CJD had led to a tendency to over-diagnose this condition, and that many of the misdiagnosed patients suffered from potentially treatable diseases, will be presented. The resulting revised consensus criteria should decrease the number of false-positive diagnoses.

Abbreviations

AD Alzheimer's disease; AE: Autoimmune encephalitis; AM: Akinetic mutism; AT: Atomoxetine; BC: Bromocriptine; CAA: Cerebral amyloid angiopathy; CJD: Creutzfeldt-Jakob disease; CNS: Central nervous system; EEG: Electroencephalogram; FE: Fungal encephalitis; CSF: Cerebrospinal fluid; CVD: Cerebrovascular disease; DAF: Dural arteriovenous fistulas; DLB: Dementia with Lewy; DS/VS: Dorsal striatum/ventral striatum; EPS: Extrapyramidal symptoms; FTD: Frontotemporal dementia; GPe/vp: Globus pallidus externus/ventral pallidum; GPi/vp: Globus pallidus internus/ ventral pallidum;HE: Hashimoto's encephalitis; HE: Hepatic encephalopathy; LD: Levodopa; LD: Lyme disease; LIS: Locked-in syndrome; MD: Mixed dementia; MRI: MP: Methylphenidate; Magnetic resonance imaging; MS: Multiple sclerosis; NDD: Neurodegenerative diseases; NIA: (U.S.) National Institute on Aging; NMS: Neuroleptic malignant syndrome; NPDPSC: (U.S.) National Prion Disease Pathology Surveillance Center; PD: Parkinson's disease; PD: Prion disease; PMD: Prion-mimic diseases; PML: Progressive

multifocal leukoencephalopathy; RPD: Rapidly progressing dementia; RT-QuIC: Real-time quaking-induced conversion; sCJD: sporadic CJD; SCI: Spinal cord injury; SLE: Systemic lupus erythematosus; SNc/vta: Substantia nigra pars compacta/ventral tegmental area; stn: Subthalamic nucleus; UMNS: Upper motor neuron syndrome; VD: Vascular dementia; WE: Wernicke's encephalopathy; WHO: World Health Organization; ZPD: Zolpidem.

Keywords

Cerebral amyloid angiopathy; cerebellar signs; Creutzfeldt-Jakob disease; dural arteriovenous fistulas; extrapyramidal signs; fungal encephalitis; Hashimoto's encephalitis; hepatic encephalopathy; mitochondriopathies; prion diseases; prion-mimic diseases; prionopathy; progressive multifocal leukoencephalopathy; pyramidal signs; rapidly progressing dementias; systemic lupus erythematosus; Wernicke's encephalopathy.

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Physicians caring for patients with rapidly progressive brain conditions need to be aware of sporadic Creutzfeldt-Jakob disease (sCJD) but should consider other potential treatable neurological disorders before making the diagnosis of this rare, inexorable, and untreatable disease. This article aims to provide data that may help physicians identify patients for whom life-extending treatment can, and should, be given. After defining what are prion-mimic diseases and why they are important, the available diagnostic tools are reviewed. In addition, the neuropathological study that had indicated that heightened awareness of CJD had led to a tendency to over-diagnose this condition, and that many of the misdiagnosed patients suffered from potentially treatable diseases, will be presented. It is hoped that the resulting revised consensus criteria would decrease the number of false-positive diagnoses. The treatable prion-mimic conditions identified by the above neuropathological study are also succinctly described.

What are prion-mimic diseases?

As discussed in Article I and II of this series, prion diseases are caused by misfolded proteins called prions that can transmit their abnormal structure to other normal prion proteins, leading to progressive brain damage and dementia. A key feature of prion diseases is their rapid progression with symptoms worsening quickly. On the other hand, prion-mimic diseases (PMD) are disorders that can present with symptoms similar to prion diseases, making them particularly difficult to distinguish clinically.

Why are prion-mimic diseases important?

Distinguishing between prion diseases and their mimics is crucial for accurate diagnosis and appropriate management. Many conditions can present with rapidly progressive dementia, personality changes, seizures and movement disorders like CJD.

Common prion-mimic diseases are numerous and may include:

- **Rapidly progressing dementias (RPDs) like Alzheimer′s disease (AD), dementia with Lewy bodies (DLB), and frontotemporal dementia (FTD):** They can cause rapid cognitive decline and, in case of FTD, personality changes and language difficulties.

- **Various types of encephalitis: Autoimmune encephalitis (AE)** - an inflammation of the brain caused by antibodies attacking brain cells, Hashimoto′s encephalitis (HE); hepatic

encephalopathy (HE), and Wernicke′s encephalopathy (WE) can potentially mimic CJD symptoms.

- **Dural arteriovenous fistulas (DAF):** These abnormal connections between arteries and veins in the brain, can potentially cause symptoms like seizures and neurological deficits that could mimic CJD.
- **Cerebral amyloid angiopathy (CAA):** Here, deposits of amyloid protein in the brain's blood vessels lead to strokes or other neurological problems, which can be confused with CJD.
- **Systemic lupus erythematosus (SLE):** This autoimmune disease can affect the brain and nervous system, causing symptoms like dementia and neurological deficits that can be similar to CJD.
- **Other neurodegenerative diseases (NDD):** Alzheimer's disease (AD), dementia with Lewy bodies (DLB), and other neurodegenerative conditions can also present with symptoms that overlap with prion diseases.
- **Mitochondriopathies:** These disorders affect the mitochondria, the energy-producing organelles in cells, causing progressive neurological damage that might mimic CJD.
- **Infections:** Certain infections, like progressive multifocal leukoencephalopathy (PML), fungal encephalitis (FE), and Lyme disease (LD), can also cause neurological symptoms that can be mistaken for CJD.
- **Various other diseases:** Cerebrovascular disease (CVD), neurosarcoidosis, and other less common conditions such as thyroid dysfunction are treatable diseases.

Diagnostic tools

Several tools help differentiate between prion diseases and their mimics, namely:

- **Magnetic resonance imaging (MRI):** These scans can reveal structural abnormalities in the brain, helping to identify conditions like strokes or dural arteriovenous fistulas.
- **Electroencephalogram (EEG):** It can detect abnormal brainwave activity, which may be suggestive of prion disease or other conditions.
- **Cerebrospinal fluid (CSF) analysis:** It can identify specific proteins or markers, like protein 14-3-3 or neural-specific autoantibodies, that may be helpful in differentiating between CJD and other diseases.
- **Real-Time Quaking-Induced Conversion RT-QuIC)):** A test that detects misfolded prion proteins, helping to confirm CJD.
- **Specific immunostaining:** This type of staining can be used to identify specific protein aggregates or autoantibodies that are characteristic of different conditions.

Neuropathological study points to revised consensus criteria

In 2011, Chitravas et al. performed a retrospective clinical and neuropathological review of prion-negative brain autopsy cases referred to the (U.S.) National Prion Disease Pathology Surveillance Center (NPDPSC) at

Case Western Reserve University from January 2006 through December 2009. Neurological signs, symptoms, and EEG results were compiled according to currently defined World Health Organization (WHO) criteria for sporadic sCJD (Table 1). Data were coded as missing when adequate documentation was unavailable. When available, brain MRI and CSF results were also obtained from the submitted medical records. All autopsy cases were included with both negative prion immunoblot and immunohistochemical analyses. Cases lacking immunoblot analyses were excluded unless the histopathologic findings were judged sufficient to explain the clinical illness.

Criteria	
I	Progressive dementia
II A	Myoclonus
B	Visual or cerebellar problem
C	Pyramidal or extrapyramidal features
D	Akinetic mutism
III A	Typical EEG
B	Positive CSF 14-3-3

Source: World Health Organization
CSF: Cerebrospinal fluid; EEG: Electroencephalogram

Table 1 - World Health Organization criteria for the diagnosis of sporadic Creutzfeldt-Jakob disease (sCJD)

The following results were obtained:

- **Possible sCJD:** I and IIB and duration of less than 2 years.
- **Probable sCJD:** I, IIB, and IID either typical EEG and/or positive CSF 14-3-3 with a total disease duration of less than 2 years. Routine investigations should not suggest an alternative diagnosis.
- **Definite sCJD:** Neuropathologically confirmed diagnosis.

Potentially treatable disorders were defined as conditions for which therapy could either cure or prolong the life of the patient. These included immunological disorders, tumors, infections, and metabolic disturbances affecting the central nervous system (CNS). Neurodegenerative disorders such as AD, FTLD and the like were considered untreatable since symptomatic therapies are not known to modify the disease course.

Of the 1,106 brain autopsies considered in the above study, 352 (32%) were negative for prion disease (PD), 304 of which had adequate tissue for histopathological analysis. AD (154) and vascular dementia (VD; 36) were the two most frequent diagnoses. Seventy-one patients had potentially treatable diseases. Clinical findings included dementia (42 cases), pyramidal (20), cerebellar (14), or extrapyramidal (12) signs, myoclonus (12), visual disturbance (9) and akinetic mutism (AM; 5); a typical EEG occurred only once. Neuropathological diagnoses included immune-mediated disorders (26), neoplasia (25; most often lymphoma, infections (14), and metabolic disorders (6).

In summary, the above findings indicate that heightened awareness of CJD has led to a tendency to over-diagnose this condition, and that many of these misdiagnosed patients suffer from potentially treatable diseases. The application of revised consensus criteria should decrease the number of false-positive diagnoses. Especially promising are recently validated MRI features of prion-infected brains. Again, physicians caring for patients with rapidly progressive brain conditions need to be aware of sCJD but should consider other potential treatable neurological disorders before making the diagnosis of this rare, untreatable, and inexorable disease.

Treatable prion-mimic conditions

The treatable prion-mimic conditions identified by the above neuropathological study are succinctly described below:

Progressive dementia

Dementia is a general term for a group of progressive brain disorders that cause a decline in memory, thinking, and other cognitive abilities severe enough to interfere with daily life. It is not a single disease, but rather a set of symptoms caused by different underlying conditions that damage brain cells.

Key points about dementia are:

• **Progressive:** Symptoms worsen over time.
• **Not a normal part of aging:** While more common in older adults, it is not an inevitable consequence of aging, according to the (U.S.) National Institute on Aging (NIA).
• **Many types:** The most common types include AD, VD, FTD, DLB, and mixed dementia (MD).
• **Symptoms:** Include memory loss, difficulty with language and problem-solving, changes in mood and behavior, and challenges with everyday activities.
• **Impact:** Dementia affects not only the person living with it but also their families and caregivers.
• **No cure:** While there is no cure for most types of dementia, treatments can help manage symptoms and improve quality of life.

Common types of dementia:

• **Alzheimer's disease (AD):** The most common form, characterized by brain damage and buildup of abnormal proteins.
• **Vascular dementia (VD):** Caused by damage to blood vessels in the brain, often due to stroke or atherosclerosis.
• **Frontotemporal dementia (FTD):** Affects the frontal and temporal lobes of the brain, leading to changes in personality, behavior, and language.
• **Dementia with Lewy bodies (DLB):** Causes movement and sleep problems along with memory loss.
• **Mixed dementia (MD):** A combination of two or more types of dementia.

Pyramidal signs

Pyramidal signs are a set of neurological symptoms indicating damage or dysfunction in the pyramidal tract, which controls voluntary movements. These signs include weakness, spasticity, hyperreflexia (increased reflexes), and a Babinski sign (toes pointing upward when the sole of the foot is stimulated). Other signs may include decreased fine motor coordination and involuntary muscle contractions (clonus).

These signs are further elaborated below:

• **Weakness:** Muscle weakness can manifest in various areas, depending on the location of the lesion in the pyramidal tract.

• **Spasticity:** This refers to increased muscle tone, resulting in stiffness and resistance to movement.

• **Hyperreflexia:** Reflexes become overly sensitive and exaggerated, including deep tendon reflexes (e.g., knee jerk).

• **Babinski signs:** This abnormal plantar reflex is a key indicator of pyramidal tract damage.

• **Other signs:** Loss of fine motor control, involuntary muscle contractions (clonus), and potential changes in speech or lower facial weakness depending on the corticobulbar tract involvement.

Causes and considerations

• **Damage to the pyramidal tract:** This can occur due to various factors like stroke, multiple sclerosis (MS), spinal cord injury (SCI), tumors, or infections.

• **Upper motor neuron syndrome (UMNS):** Pyramidal signs are a hallmark of upper motor neuron damage, as the pyramidal tract transmits signals from the brain to the spinal cord.

• **Bilateral vs. unilateral lesions:** If the corticobulbar tract (part of the pyramidal tract) is damaged on one side, it can lead to weakness in the lower part of the face on the opposite side. Bilateral damage to the corticobulbar tract can result in pseudobulbar palsy, affecting swallowing, speech, and emotional control.

Extrapyramidal signs

Extrapyramidal symptoms (EPS), also known as extrapyramidal side effects, are movement disorders caused by certain medications, especially antipsychotic drugs. These symptoms affect the motor system and can include involuntary movements, muscle stiffness, and tremors. They are classified into several types, including dystonia, akathisia, parkinsonism, and tardive dyskinesia.

Common signs and symptoms

• **Dystonia:** It involves sustained muscle contractions that cause abnormal postures, sometimes painful. Examples include twisted neck (torticollis), back arching, and stiff tongue.

• **Akathesia:** It is characterized by a feeling of restlessness and an inability to sit still. Individuals may pace, fidget, or have a strong urge to move.

• **Parkinsonism:** It mimics symptoms of Parkinson's disease (PD), such as tremors, stiff posture, slow movement, and a pill-rolling movement in the fingers.

• **Tardive dyskinesia:** It involves involuntary, repetitive movements, often affecting the mouth and tongue, such as tongue protrusion, cheek puffing, or lip smacking.

Other signs and symptoms

• **Rigidity:** Increased muscle stiffness, similar to Parkinson's disease (PD).

• **Bradykinesia:** Slowed movement and speech.

• **Tremor:** Shaking or rhythmic movement, especially in the hands.

• **Shuffling gait:** A shuffling or dragging of the feet while walking.

• **Mask-like facial expression**: Reduced facial expression, with a stiff or blank face.

• **Oculogyric crisis:** Involuntary eye movements, often upward and fixated, with difficulty repositioning.

Important considerations

• Extrapyramidal sign (EPS) can occur shortly after starting a medication or after long-term use.

• Some individuals may not be aware of these symptoms, but others close to them may notice the changes in behavior or movement.

• These symptoms can be distressing and may require treatment, such as adjusting the medication or using other medications to manage the EPS.

• Neuroleptic malignant syndrome (NMS): A serious but rare condition, can be associated with EPS and requires immediate medical attention.

Cerebellar signs

Cerebellar dysfunction presents with a range of symptoms and signs primarily affecting motor coordination, balance, and posture. Common symptoms include ataxia (difficulty with coordination and balance), dysarthria (slurred or abnormal speech), and

nystagmus (involuntary eye movements). Other signs may include dysmetria (difficulty with precise movements), tremors, and gait abnormalities:

Ataxia is the difficulty walking, often described as a wide-based, unsteady, or staggering gait. It includes:

- **Limb ataxia:** Incoordination and clumsiness in the arms and legs, making it difficult to perform precise movements like reaching or grasping.
- **Truncal ataxia:** Difficulty maintaining balance and stability of the trunk, leading to wobbling or swaying.

Dysarthria

- **Scanning speech:** Slurred, slow, and sometimes staccato-like speech with hesitations and pauses.
- **Dysarthria:** Difficulty with articulation and coordination of speech muscles.

Other common signs

- **Nystagmus:** Involuntary, rhythmic eye movements that can be horizontal, vertical, or rotary.
- **Dysmetria:** Difficulty judging the distance and speed of movements, leading to overshooting or undershooting target.
- **Tremor:** An intention tremor, a tremor that worsens as a person approaches a target with a movement, is a common finding.
- **Hypotonia:** Decreased muscle tone, leading to floppy or weak muscles.
- **Posture and balance problems:** Difficulty maintaining a stable posture, often with a wide-based stance.
- **Cognitive and mood changes:** In some cases, cerebellar dysfunction can also be associated with cognitive and mood disturbances.

Signs related to specific cerebellar regions

• **Anterior lobe:** May cause gait and limb ataxia and can affect proprioception (sense of body position).

• **Posterior lobe:** May cause problems with eye movements, balance, and coordination of complex movements.

• **Flocculonodular lobe:** May cause balance and gait problems, as well as truncal ataxia.

Differential diagnoses

• It is important to differentiate cerebellar dysfunction from other conditions that can cause ataxia, such as sensory ataxia (due to nerve damage) or vestibular disorders (affecting the inner ear).

• Other possible causes of cerebellar dysfunction include stroke, trauma, infection, inflammation, and tumors.

Myoclonus

Myoclonus refers to involuntary, sudden muscle jerks or spasms. Symptoms can vary depending on the underlying cause and severity, but may include:

• **Jerky movements:** Brief, involuntary muscle contractions that can occur in any part of the body.
• **Seizure-like episodes**: In some cases, myoclonus can resemble seizures, with generalized body jerking or loss of consciousness.

• **Muscle stiffness:** Myoclonus can sometimes be accompanied by muscle stiffness or spasms.
• **Tremors:** Rhythmic, involuntary shaking movements.
• **Difficulty with coordination and balance:** Myoclonus can interfere with motor control and cause balance problems.
• **Sensitivity to stimuli:** Some people with myoclonus may experience increased sensitivity to sounds, light, or touch, which can trigger jerks.
• **Weakness:** In severe cases, myoclonus can lead to muscle weakness or paralysis.

It is important to note that myoclonus can be a symptom of various underlying conditions, including metabolic disorders, neurological diseases, and certain medications.

Visual disturbances

Vision disturbances refer to changes or abnormalities in eyesight, affecting the ability to see clearly. They can manifest in various ways. Types of vision disturbances include:

• **Blurred vision:** Inability to focus clearly, making objects appear fuzzy or indistinct.
• **Double vision:** Seeing two images of the same object, either in one or both eyes.
• **Floaters:** Small spots or specks that move in the field of vision.
• **Flashes of light:** Sudden bursts of light or shimmering sensations.
• **Loss of vision:** Partial or complete inability to see.
• **Halos around lights:** Rings of light appearing around sources of illumination.
• **Distorted vision:** Objects appearing misshapen, curved, or wavy.
• **Color blindness:** Difficulty distinguishing between colors or seeing them in muted tones.

Causes

Vision disturbances can be caused by a wide range of factors, including:

• **Eye conditions:** Glaucoma, cataracts, macular degeneration, retinal detachment.
• **Neurological disorders:** Stroke, migraine, brain tumor, multiple sclerosis (MS).
• **Medications:** Side effects of certain drugs, such as antidepressants and blood pressure medications.
• **Injuries:** Trauma to the head or eye.
• **Age:** Presbyopia (age-related nearsightedness).
• **Lifestyle factors:** Smoking, excessive alcohol consumption, lack of sleep.

Early diagnosis and treatment are crucial to prevent potential complications and preserve vision.

Akinetic mutism

Akinetic mutism (AM) is a rare neurological disorder characterized by the presence of an intact level of consciousness and sensorimotor capacity, but with a simultaneous decrease in goal-directed behavior and emotions, a profound apathy, lack of motivation, indifference to pain, thirst, or hunger. and the inability to initiate voluntary movement and speech, despite being conscious and alert. Patients are in a wakeful state of profound apathy, seemingly indifferent to pain, thirst, or hunger (Figure 1).

Key symptoms include:

• **Lack of spontaneous movement and speech:** Individuals with AM may be unable to move or speak on their own, even when alert and awake.
• **Impaired initiation and motivation:** They struggle to initiate goal-directed behaviors and tasks.
• **Apathy and indifference:** A lack of interest in their surroundings and a reduced emotional response.
• **Difficulty following commands:** While they may understand spoken commands, they may not be able to carry them out.

• **Intact consciousness and attention:** Despite the physical and behavioral limitations, individuals with akinetic mutism are generally aware and attentive.

Causes

Akinetic mutism can be caused by a variety of brain injuries or disorders, including (Figure 1):

• **Damage to specific brain areas:** The anterior cingulate cortex, striatum, and thalamus are often implicated.

• **Stroke:** Specifically, unilateral anterior cerebral artery occlusion can lead to akinetic mutism.

• **Trauma:** Brain trauma can damage the areas responsible for motivation and movement.

• **Encephalopathy:** In some cases, akinetic mutism can be a manifestation of encephalopathy, a general term for brain dysfunction.

• **Other neurological conditions:** Akinetic mutism can also be associated with conditions like fat embolism syndrome and certain infections.

Important distinctions

- **Locked-in syndrome (LIS):** While akinetic mutism and locked-in syndrome share some similarities, individuals with locked-in syndrome are physically paralyzed but can still communicate using eye movements or other cues. In contrast, patients with akinetic mutism may be able to follow someone's eyes or react to sounds, but they lack the motivation to initiate any movement or speech.

- **Catatonia:** Catatonia can also present with immobility and mutism, but it is often accompanied by symptoms like echolalia (repeating others' words) and posturing (holding unusual body positions). Akinetic mutism does not typically have these features.

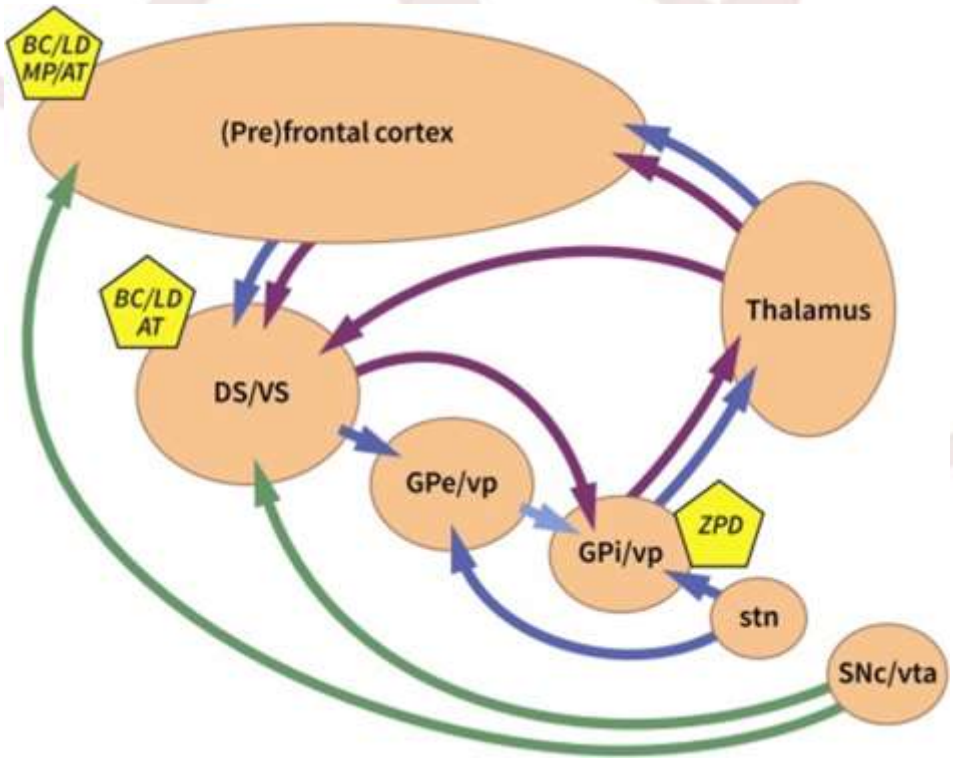

Figure 1: Schematic overview of frontal-subcortical circuitry associated with motivation and possible site(s) of

action of pharmacological interventions in akinetic mutism

(Purple arrows represent the direct pathway, blue arrows represent the indirect pathway, and green arrows represent dopaminergic pathways.

Abbreviations: AT: Atomoxetine; BC: Bromocriptine; DS/VS: Dorsal striatum/ventral striatum; GPe/vp: Globus pallidus externus/ventral pallidum; GPi/vp: Globus pallidus internus/ ventral pallidum; LD: Levodopa; MP: Methylphenidate; SNc/vta: Substantia nigra pars compacta/ventral tegmental area; stn: Subthalamic nucleus; ZPD: Zolpidem.)

Conclusions and take-aways

- Physicians caring for patients with rapidly progressive brain conditions need to be aware of sporadic Creutzfeldt-Jakob disease but should consider other potential treatable neurological disorders before making the diagnosis of this rare, inexorable, and untreatable disease.

- However, a neuropathological study has pointed to the need for revised consensus criteria. Such revised criteria should decrease the number of false-positive diagnoses.

- Prion-mimic diseases are disorders that can present with symptoms similar to prion diseases, particularly, making them difficult to distinguish clinically. These include, for example, rapidly progressive dementias like Alzheimer′s disease, dementia with Lewy bodies, and frontotemporal dementia that can also cause rapid cognitive decline, making them potential mimics. They also include autoimmune encephalitis, Hashimoto's encephalitis, vascular, granulomatous, and other diseases such as cerebrovascular diseases, neurosarcoidosis, and other less common conditions. Other conditions such as hepatic encephalopathy, Wernicke's encephalopathy, and thyroid dysfunction, which are treatable disorders, can also present with symptoms like Creutzfeldt-Jakob disease.

- Othe common prion-mimic diseases include: Autoimmune encephalitis, frontotemporal dementia, dural arteriovenous fistulas, cerebral amyloid angiopathy, systemic lupus erythematosus, vascular dementia, other neurodegenerative diseases, mitochondriopathies, and certain infections like progressive multifocal leukoencephalopathy, fungal encephalitis, and Lyme disease.

- Distinguishing between prion diseases and their mimics is crucial for accurate diagnosis and appropriate management.

- Treatable prion-mimic conditions identified by the above neuropathological study include: Progressive dementia, pyramidal and extrapyramidal signs, cerebellar signs, myoclonus, visual disturbances, and akinetic mutism.

References

1. Fymat AL (2025). "Creutzfeldt-Jakob disease as an example of prion disease: I. Prions", Journal of Neurology and Psychology 6(3):1-27.
2. Fymat AL (2025). "Creutzfeldt-Jakob disease as an example of prion disease: II. Prion diseases", Journal of

Neurology and Psychology 6(3):1-19.

3. Goedert M (2015). "Neurodegeneration: Alzheimer's and Parkinson's diseases: The prion concept in relation to assembled Aβ, tau, and α-synuclein". Science 349(6248): 1255555. doi:10.1126/science.1255555.
4. Groschup MH and Kretzschmar HA, eds. (2001). "Prion diseases diagnosis and pathogenesis". Archives of Virology Vol. 16. New York: Springer. doi:10.1007/978-3-7091-6308-5. ISBN 978-3-211-83530-2.
5. Olanow CW and Brundin P (2013). "Parkinson's disease and alpha synuclein: Is Parkinson's disease a prion-like disorder?". Movement Disorders 28(1):31–40. doi:10.1002/mds.25373.
6. Surmeier DJ, Obeso JA and Halliday GM. "Parkinson's disease is not simply a prion disorder". doi: 10.1523/JNEUROSCI.1787-16.2017.
7. Vascellari S and Manzin A (2021). "Parkinson's disease: A prionopathy?" Int J Mol Sci 22(15):8022. doi: 10.3390/ijms22158022.