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Rapidly progressive dementia: probable sporadic Creutzfeldt–Jakob disease in a Yoruba Nigerian woman with rapidly progressive dementia: a case report

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Abstract

Background Creutzfeldt–Jakob disease is a neurodegenerative disorder that can present with neuropsychiatric features such as dementia; it is a rare cause of rapidly progressive dementia. There are no previously reported cases of probable or confirmed Creutzfeldt–Jakob disease in Nigeria.

Case presentation A 68-year-old Nigerian Yoruba woman with known hypertension presented with a 6-week history of progressively worsening decline in memory, incoherent speech, irrational behavior, visual hallucinations, acute insomnia, and inability to independently perform activities of daily living. Examination revealed impairment in immediate recall, short- and long-term memories, and a Mini-Mental State Examination score of 2/30. She had an ataxic gait with abnormal jerky movements of the upper limbs. Brain magnetic resonance imaging and electroencephalography were consistent with Creutzfeldt–Jakob disease. She subsequently experienced sudden clinical deterioration following seizure episodes and features suggestive of aspiration pneumonia, and she died within 14 weeks of the onset of the illness.

Conclusion This case highlights the pattern and progression of Creutzfeldt–Jakob disease as a cause of dementia in an elderly Nigerian woman and the need to have a high index of suspicion of the diagnosis in patients presenting with rapidly progressive neuropsychiatric symptoms and dementia. Early diagnosis allows caregivers and patients to prepare well for the future, as the disease remains incurable.

Keywords Dementia, Creutzfeldt–Jakob disease, Nigeria

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Background

Rapidly progressive dementia (RPD) is used to describe cognitive disorders that rapidly progress to dementia within less than 1–2 years, but more commonly over weeks to months [1]. There are a wide range of causes, some of which are treatable if identified early, including autoimmune, infectious, and toxic-metabolic encephalopathies. A rare cause of RPD is Creutzfeldt–Jakob disease (CJD), which is a transmissible spongiform encephalopathy associated with the deposition of abnormally folded protein (prion) in the brain. Other prion diseases include Gertsmann–Sträussler–Schneinker (GSS) disease, kuru, fatal familial insomnia,

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and variable protease-sensitive prionopathy. Four different forms of CJD have been recognized: variant, iatrogenic, familial, and sporadic. The most common form is the sporadic form, accounting for 85% of all cases of CJD, with an incidence of approximately 1–1.5 people per million annually. Familial CJD accounts for 10% of cases, whereas iatrogenic and variant forms account for the remaining cases [2, 3]. Several cases of CJD have been reported outside Africa [4–7]. However, within the African continent, a few cases have also been reported. These include a case of iatrogenic CJD from a dura mater graft in South Africa [8] and four definite, seven probable, and two possible cases of CJD reported in Kenya within a 14-year period [9]. Other risk factors associated with sporadic CJD include surgeries and blood transfusion. It is an occupational risk among health workers [10]. Abnormal prion protein in the brain results in spongiform alteration, neuronal loss, and gliosis, leading to rapidly progressive dementia; myoclonus; pyramidal and extrapyramidal signs; and cerebellar ataxia, with a median survival of 4.5 months from symptom onset [7, 11, 12]. To the best of the authors' knowledge, there are no previously reported cases of probable or confirmed CJD in Nigeria. We report a case of probable sporadic CJD in a 68-year-old Nigerian woman with rapidly evolving symptomatology corroborated by electroencephalogram (EEG) and neuroimaging findings.

Case presentation

The patient was a 68-year-old female Yoruba woman with a prior normal social life, who was referred to the neurology outpatient clinic with a history of progressive decline in memory for a duration of 6 weeks, as reported by her husband. She was unable to perform her activities of daily living, including cooking, bathing, eating, and managing funds, and had a history of talking out of context with irrational behavior. She often missed her way around the home, occasionally urinated in inappropriate places, and could not remember recent events or remote events.

There was a history of visual hallucinations, as she was reported to be seeing persons who were not physically present, but she had no tremors or body stiffness. There was no history of antecedent head trauma or stroke, and the husband reported no preceding history of depression or background mental illness. There was a history of poor memory in her older sister, who was in her late 60s and subsequently died approximately 5 years after onset of her symptoms. However, the details of the diagnosis could not be ascertained.

She was diagnosed with hypertension approximately 10 years prior to the onset of her symptoms and was compliant with her medications. She neither consumed alcohol nor smoked cigarettes.

On neurological examination, she was conscious, with orientation to person only. Immediate recall, short-term memory, and long-term memory were impaired, with a Mini-Mental State Examination score of 2/30. She had quadriparesis with Medical Research Council (MRC) muscle power grade 3. Hypertonia, hyperreflexia, and extensor plantar response were observed in the right lower limb.

An examination of other systems revealed essentially normal findings. An initial assessment of rapidly progressive dementia to rule out an acute psychotic disorder was made. She underwent brain magnetic resonance imaging (MRI) and a series of investigations, and the results are shown in Table 1 and Figs. 1 and 2.

She was seen at a follow-up clinic visit 2 weeks later. She was observed to have developed persistent purposeless abnormal jerky movement of the upper limbs, which was worse on the right, with restlessness, ataxia, unnecessary shouting, and poor sleep. She had deteriorated further and was not oriented in person anymore. An assessment of rapidly progressive dementia, probable sporadic Creutzfeldt–Jakob Disease (CJD), was made.

The plan was to perform a cerebrospinal fluid (CSF) 14-3-3 protein assay and CSF and/or olfactory mucosa real-time quaking-induced conversion (RT-QuIC) seeding assays. She was also reviewed by the psychiatrists, who commenced her on tablet risperidone 0.5 mg at night and tablet levetiracetam 250 mg twice a day for seizure prophylaxis.

She presented at the emergency department 5 weeks later with a complaint of 2 days of fever, difficulty breathing, and an episode of generalized tonic–clonic convulsion, and she also experienced progressively worsening altered sensorium a few hours before presentation.

On examination, she was in respiratory distress, and she was tachypneic with a respiratory rate of 40 cpm; her oxygen saturation was between 89 and 90% on intranasal oxygen. There were coarse crepitations in both the middle and lower lung zones bilaterally.

She had a Glasgow Coma Scale (GCS) score of 9/15, with global hypotonia and hyporeflexia. A chest X-ray revealed multiple patchy opacities in both lung fields, with more in the right lung field, and an assessment of aspiration pneumonia. A repeat brain MRI scan was ordered, but the relatives declined due to financial constraints. She was started on intravenous antibiotics and continued on tablet levetiracetam 500 mg twice a day via a nasogastric tube and prophylactic anticoagulation with subcutaneous Clexane 40 mg daily.

Table 1 Summary of the investigation results at admission

Blood glucose	**FBG: 4.4 mmol/L, 2HPP: 7.6 mmol/L**
Thyroid function test	fT_3: 1.4 ng/ml (normal range: 0.8–1.9)
	fT_4: 9.0 ug/dl (normal range: 5–13)
	TSH: 1.5 mIU/ml (normal range: 0.3–4.2)
FBC, ESR (on admission)	PCV: 42%
	WBC: 16,600 cells/mL
	Neutrophils: 96%
	Lymphocytes: 04%
	Platelets: 107,000/mL
Urine microscopy, culture, and sensitivity (on admission)	Macroscopy
	Microscopy: < 5 WBC/Hpf, nil RBC, nil cast/crystals, nil yeast
	Culture-yielded no growth after 24 hours of incubation
Blood culture	Blood culture yielded no growth after 5 days of incubation
Serology	RVS: Negative
	HbSAg: Negative
	Anti-HCV: Negative
12-lead electrocardiogram	Sinus rhythm, normal axis
Lipid profile	Total cholesterol: 2.6 mmol/L
	LDL: 1.6 mmol/L
	HDL: 0.5 mmol/L
	Triglycerides: 1.3 mmol/L
Blood electrolytes, urea, and creatinine	Creatinine: 101 µmol/L (normal range: 50–132 µmol/L)
	K: 3.1 mmol/L (normal range: 3–5 mmol/L)
	HCO_3: **16 mmol/L** (normal range: 20–30 mmol/L)
	Na: **155 mmol/L** (normal range: 135–145 mmol/L)
	Urea: **13.6 mmol/L** (normal range: 2.5–5.8 mmol/L)
Liver function tests (on admission)	Total bilirubin: 10 (normal range: up to 20 umol/L)
	Conjugated bilirubin: 6 (normal range: up to 5 umol/L)
	Alkaline phosphatase: 93 IU/L (normal range: 60–170 IU/L)
	GGT: **83 IU/L** (normal range: 4–28 IU/L)
	AST: **103 IU/L**(normal range: < 40 IU/L)
	ALT: **128 IU/L**(normal range: < 40 IU/l)
	Total protein: 78 (normal range: 58–80 g/L)
	Albumin: 36 (normal range: 35–50 g/L)
	Globulin: 42 (normal range: 20–45 g/L)
Clotting profile	INR: 1.1
	PT test: 13 seconds (control: 12 seconds)
	PTTK test: 27.5 seconds (control: 33 seconds)

The values in bold letters are deranged

FBG fasting blood glucose, *2HPP* 2-hour postprandial, *FBC* full blood count, *INR* international normalized ratio, *PT* prothrombin time, *PTTK* partial thromboplastin time with kaolin, *PCV* packed cell volume, *LDL* low-density lipoprotein, *HDL* high-density lipoprotein, *HbSAg* hepatitis B surface antigen, *anti-HCV* hepatitis C antibody, *GGT* gamma glutamyl transferase, *AST* aspartate aminotransferase transaminase, *ALT* alanine aminotransferase

The results shown in Table 1 revealed leukocytosis with neutrophilia, hypernatraemia, elevated serum urea, and deranged liver enzymes.

The patient's condition, however, deteriorated during admission, with her GCS score decreasing to 6/15, approximately 9 days after admission. She was later admitted to the intensive care unit (ICU), intubated, and placed on ventilatory support.

While in the ICU, the patient was persistently febrile and had severe dyselectrolytemia (hypokalemia, hypomagnemia, hypernatremia, and hypocalcemia) accompanied by acute symptomatic focal seizures that

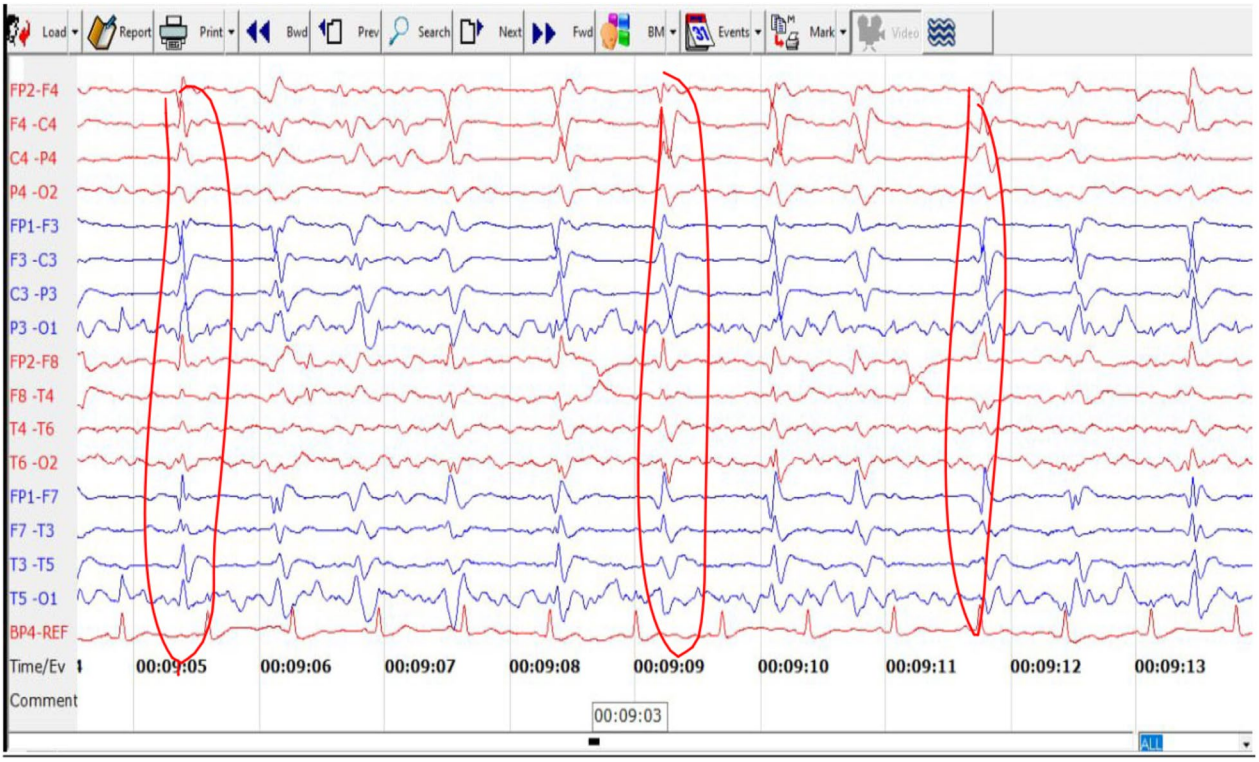

Fig. 1 Electroencephalogram of the patient showing a short interval and generalized periodic sharp and slow wave complexes (highlighted within the red lines)

were controlled. Cardiopulmonary physiotherapy and neurorehabilitation was commenced.

Despite all the interventions, there was no improvement in her clinical status, and the overall poor prognosis and terminal nature of the illness was discussed with her next of kin (husband); a decision was made to take her off the mechanical ventilator owing to poor response to treatment in the context of the terminal nature of the illness and the accumulating hospital bills, which had taken a heavy toll on the family. The consequences of the decision were explained to the husband and the patient's relations, with input from the social workers and legal unit of the hospital.

The patient was extubated 24 days after admission and transferred to the medical ward on intranasal oxygen support via nasal prongs with close vital sign monitoring; she died a day after leaving the ICU. An autopsy was requested, but the family declined.

Discussion

Creutzfeldt–Jakob disease (CJD) was first described in 1920 by Hans Creutzfeldt and was later described in 1921 and 1923 by Alfons Jakob. The disease progresses inexorably to death once clinical symptoms appear. The patient was diagnosed with probable sporadic CJD because of the presentation of rapidly progressive dementia, ataxia, myoclonic jerks of the upper limbs, brain MRI findings (cortical ribbon sign, high signal intensity in the left cerebral frontal gyrus, straight gyrus, insula, medial occipitotemporal gyrus, and caudate lobe), and typical EEG findings (periodic sharp and slow wave complexes). This assessment of probable sporadic CJD was performed via the Centers for Disease Control (CDC) diagnostic criteria highlighted in Table 2.

Our patient was 68 years old and fell into the age range of 60–80 years, which is typical of the age range of sporadic CJD described in a previous study [15]. She presented with talking out of context, irrational behavior, inappropriate behavior, and visual hallucinations, which were symptoms suggestive of psychiatric illness. The initial neurologic manifestation in this patient was poor memory, but she gradually developed ataxia, myoclonic jerks, and poor sleep within 2 weeks as shown in Fig. 3. Microglial activation, synaptic and neuronal loss are responsible for the rapid neurologic decline.The absence of neurons may be observed in the striatum, cerebral cortex and thalamus with the progression of the illness [16]. Other common causes of rapidly progressive dementia, such as

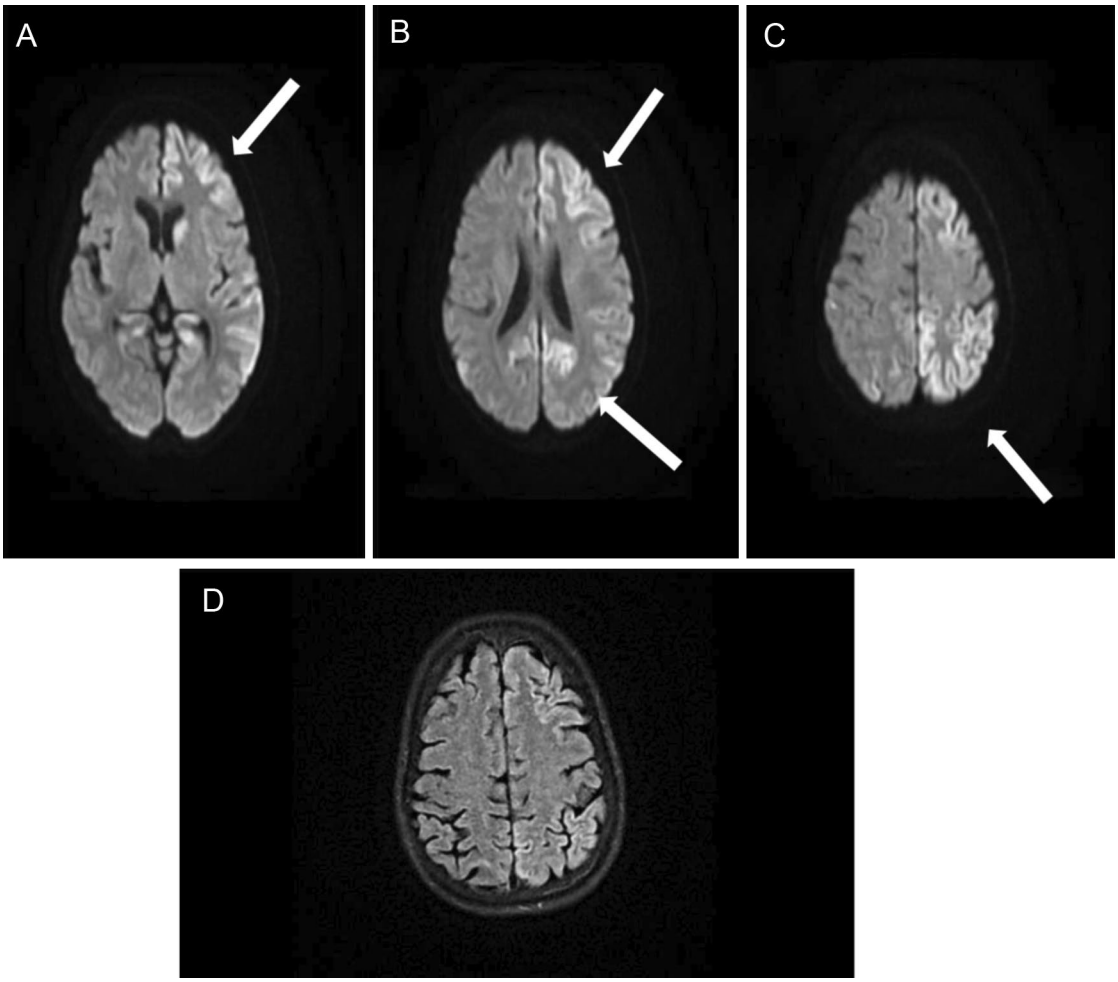

Fig. 2 **A**, **B**, and **C** are axial diffusion-weighted images of a 68-year-old woman who presented with poor memory/rapidly progressive dementia. The images show high cortical signals (cortical ribbon signs) asymmetrically affecting the cerebral hemispheres, with more signals on the left. The arrow in **A** shows the effects on the left cerebral frontal gyrus, straight gyrus, insula, medial occipitotemporal gyrus, and caudate lobe (deep gray matter); the arrow in the lower left in **B** shows the effects on the cuneus bilaterally, but more on the left; **C** shows the effects on the left superior and middle occipital gyri and the left inferior parietal lobule. The findings are less pronounced on fluid-attenuated inversion recovery images, as shown in panel **D**

thyroid dysfunction, hepatic encephalopathy, cerebral infarction, and HIV were excluded. A close differential is also fatal familial insomnia, in which there may be a strong family history, and insomnia, phobia, panic attacks, and hallucinations usually precede memory problems, unlike in CJD.

DWI and FLAIR sequences of brain MR images revealed that cortical ribbon signs affect various parts of the cerebral hemispheres and deep structures. This method has been reported to have high sensitivity (91%) and specificity (95%) [17]. The high cortical signals on DWI-MRI were more on the left. Unilateral imaging findings have been observed in the early stages of the disease, with global symmetric spread as the disease progresses [18]. Typical EEG findings in patients with CJD are that of periodic sharp and slow wave complexes, also demonstrated in our patient [19]. EEG findings vary depending on the stage of the disease, as it is said to emerge by 8–12 weeks from onset and disappear in the later stages [20, 21]. The patient later presented with features of aspiration pneumonia, from which she developed other complications that eventually led to her death. Aspiration pneumonitis has been reported as the most common cause of death by Zerr *et al.* [16] The rate of progression from illness onset to death in our patient was approximately 13 weeks, as shown in Fig. 3, which is less than the 20-week survival time [22] and greater than the 5.3-week survival time [7]. This difference

Table 2 Center for Disease Control's diagnostic criteria for Creutzfeldt–Jakob Disease (CJD), 2018 (adapted from the World Health Organization, WHO, [13], Zerr *et al*. [14] and the National CJD Research & Surveillance Unit)

Sporadic CJD
Definite: Diagnosed by standard neuropathological techniques; and/or immunocytochemically; and/or western blot confirmed protease-resistant PrP; and/or presence of scrapie-associated fibrils
Probable: Neuropsychiatric disorder plus positive RT-QuIC in cerebrospinal fluid (CSF) or other tissues
OR
Rapidly progressive dementia; **and** at least two out of the following four clinical features:
(1) Myoclonus, (2) visual or cerebellar signs, (3) pyramidal/extrapyramidal signs, and (4) akinetic mutism
AND a positive result on at least one of the following laboratory tests a typical EEG (periodic sharp wave complexes) during an illness of any duration a positive 14-3-3 CSF assay in patients with a disease duration of less than 2 years
High signal in caudate/putamen on magnetic resonance imaging (MRI) brain scan or at least two cortical regions (temporal, parietal, or occipital) either on diffusion-weighted imaging (DWI) or fluid attenuated inversion recovery (FLAIR)
AND without routine investigations indicating an alternative diagnosis
Possible: Progressive dementia; **and** at least two out of the following four clinical features: (1) myoclonus, (2) visual or cerebellar signs, (3) pyramidal/extrapyramidal signs, and (4) akinetic mutism
AND the absence of a positive result for any of the four tests above that would classify a case as "probable" **AND** duration of illness less than two years **AND** without routine investigations indicating an alternative diagnosis

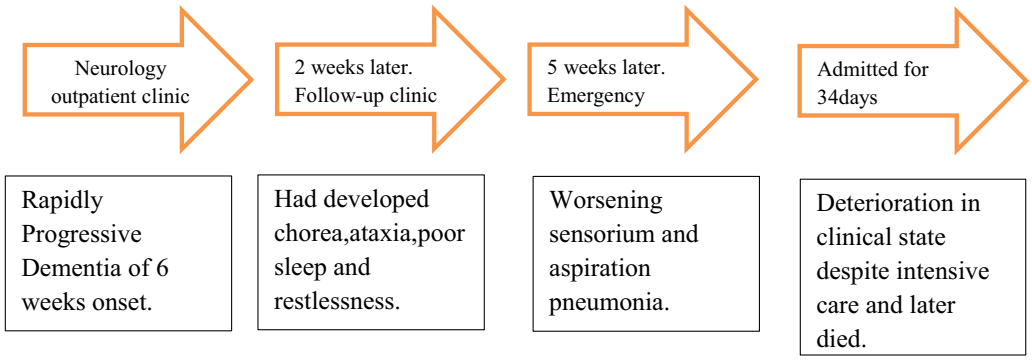

Fig. 3 Timeline of events

could be related to the various stages of presentation and the quality of palliative care received. Although a definite diagnosis of sporadic CJD can be made only by confirming pathologic prion protein deposition in the brain, the diagnosis of probable sporadic CJD is quite challenging considering the different patterns of presentation. Limitations in the definitive diagnosis of the patient include the unavailability of CSF 14-3-3 and RT-QuIC assays and cultural challenges in accepting autopsy for a definitive diagnosis.

However, it is essential to make an accurate diagnosis to differentiate it from other, reversible causes of dementia. The financial implications of the cost of investigations such as brain MRIs and all other blood tests were a major challenge, as the patient was not on the national health insurance scheme, similar to most other patients in developing countries [23]. Cultural obstacles encountered in the care of the patient include caregiver burden, since the spouse was elderly, and the fact that Yoruba people frown on taking family members to care homes. Most chronic neurological disorders are believed to be spiritual, and alternative care (traditional and spiritual) may be sought in addition to Western care. Increasing awareness of this condition among general practitioners and health care workers is recommended, as well as taking adequate clinical histories and making timely referrals to a neurologist.

Ethical dilemmas involving the escalation of treatment plans, the ceiling of care, and the withdrawal of ventilatory support also pose great challenges in the healthcare setting in developing nations because of the cultural and religious beliefs of patients with such terminal illnesses, coupled with the lack of a palliative care team in most hospitals in Nigeria. This was the case for this report's patient.

Unfortunately, there is no cure for CJD, and treatment focuses mainly on managing symptoms and providing supportive care. Owing to its rarity and complexity, CJD

remains an area of ongoing research to better understand its causes, progression, and potential therapeutic approaches. Studies and trials investigating the possibility of prophylactic and therapeutic interventions are still ongoing.

Conclusion

This case highlights an unusual presentation of Creutzfeldt–Jakob disease as a cause of dementia in a middle-aged Nigerian woman, as well as ethical and cultural challenges in its management. The diagnosis of CJD should be suspected in patients presenting with rapidly progressive neuropsychiatric symptoms and dementia. Diagnostic tests such as electroencephalography and brain magnetic resonance imaging with cerebrospinal fluid (CSF) analysis should be requested in such cases, as this could potentially help unravel the diagnosis, despite limitations in access to advanced diagnostic methods in most developing nations.

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Author contributions
AO, AF, and AA conceptualized and drafted the report. UC and SA analyzed and interpreted the investigation results. MA and MB critically revised the manuscript. All authors read and approved of the final manuscript.

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Availability of data and materials
Additional materials used during this study are available from the corresponding author upon request.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Written informed consent was obtained from the patient's next-of-kin for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests
The authors declare that they have no competing interests.

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