REVIEW ARTICLE

Rapidly Progressive Dementia: A Quick Review

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Abstract

Rapidly progressive dementias (RPDs) involve cognitive deterioration over weeks to months, sometimes extending up to 2 years. Although Creutzfeldt–Jakob disease (CJD) is the most well-known cause, around 44% of cases arise from non-CJD conditions, including infections, autoimmune disorders, Alzheimer's disease, vascular pathology, and toxic encephalopathies. Prompt and accurate diagnosis is vital, as certain forms are potentially reversible. Key diagnostic tools include CSF biomarkers (14–3–3 protein, tau, neurofilament light chain), advanced neuroimaging (MRI with DWI/FLAIR, PET), and EEG. Treating RPD as a neurological emergency and employing a multidisciplinary approach can improve outcomes, with ongoing research into novel biomarkers and precision medicine offering further promise for early detection and targeted therapy.

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Introduction

The term rapidly progressive dementia (RPD) encompasses a large group of disorders characterized by rapid cognitive decline in about 1–2 years.[1] Prompt identification and comprehensive assessment are vital for determining the underlying cause and initiating timely management. This article aims to outline the major differential diagnoses, clinical presentations, underlying mechanisms, neuroimaging characteristics, and the role of recent biomarkers. A thorough understanding of these elements enables clinicians to achieve accurate diagnoses, guide effective treatment, and improve overall patient outcomes.

Dementia Diagnosis

"Major cognitive impairment" is the current term for what was previously called "Dementia" (Fig. 1).

Causes of Rapidly Progressive Dementia

The importance of identifying reversible causes of dementia was analyzed at the University of California, San Francisco (UCSF), which revealed that a significant proportion of RPD cases were due to non-CJD etiologies (Fig. 2 and Table 1).[2]

The progression of dementia varies among different etiologies. Based on the time from the first symptom to full dementia syndrome, etiologies can be grouped as given in Figure 3.[3]

Autoimmune Causes

- Comorbid symptomatologies such as seizures, psychotic behavior, movement disorders, and ataxia, when present alongside cognitive decline, should prompt consideration of autoimmune encephalitis. The most common forms include NMDA receptor encephalitis, anti-VGKC, and GABA-B receptor encephalitis. Early recognition and treatment are crucial, as prompt immunotherapy can improve outcomes and prevent irreversible brain damage.
- NMDA receptor encephalitis is the most recognized form, presenting with rapid cognitive decline, psychiatric manifestations, seizures, and abnormal movements. LGI1 encephalitis typically involves the limbic system, leading to memory impairment, confusion, and characteristic faciobrachial dystonic seizures.
- CASPR2 encephalitis also affects the limbic regions and may present with cognitive and behavioral symptoms along with peripheral nerve hyperexcitability.
- GABA receptor encephalitis (involving either GABA-A or GABA-B antibodies) is marked by cognitive decline, seizures, and psychiatric features.

Table 1: Etiologies of RPD

V	Vascular
I	Infectious
T	Toxic/metabolic
A	Autoimmune
M	Metastasis
I	Iatrogenic
N	Neurodegenerative
S	Systemic

- DPPX encephalitis, although uncommon, can produce similar rapid deterioration, often accompanied by gastrointestinal symptoms (Table 2).

Diagnostic Criteria for Possible Autoimmune Encephalitis

Subacute onset, with rapid progression over less than 3 months, characterized by working memory deficits (such as short-term memory loss), changes in mental status, or psychiatric symptoms.

At least one of the following:

- New focal CNS findings.
- Seizures not explained by a previously known seizure disorder.
- CSF pleocytosis.
- MRI features suggestive of encephalitis.
- Reasonable exclusion of alternative causes (e.g., HSV encephalitis)
- Diagnosis can be made when all three of the criteria are met.

Vascular Dementia

Strokes can contribute to rapidly progressive dementias, often presenting as multi-infarct dementia (MiD), which involves a stepwise cognitive decline resulting from recurrent strokes.[4] Both large vessel and small vessel pathologies may manifest as rapidly progressive cognitive deterioration. According to a meta-analysis, 10% of individuals have dementia before the first stroke, 10% develop dementia after the initial stroke, and 33% experience dementia following recurrent strokes.[5]

Strategic infarcts—lesions in specific brain regions—can produce acute memory loss and mimic other dementias. Key locations include the bilateral posterior cerebral artery,[6] thalamus,[7] basal forebrain aneurysm rupture,[8] angular gyrus,[9] and caudate nucleus.[10]

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Table 2: Common autoimmune encephalitis

Antibodies	*Features*
Anti-NMDAR antibodies	The mean age of patients with NMDAR antibodies is approximately 20 years
Anti-LGI-1 antibodies	The mean age of patients with this is closer to 60 years
Anti-AMPAR, GABABR antibodies	These are associated with limbic encephalitis symptoms, which include confusion, behavioral changes, seizures, and memory disturbance in older patients
Anti-Hu antibodies	Paraneoplastic autoimmune encephalitis caused by small-cell lung cancer
Anti-Ma antibodies	Paraneoplastic autoimmune encephalitis caused by testicular germ cell tumors

Other antibodies associated with AE include anti-GQ1b, anti-DPPX, anti-CASPR2, anti-RI, anti-Yo, and anti-CV2

DSM-5 criteria for major neurocognitive disorder (previously dementia)

A. Evidence of significant cognitive decline from a previous level of performance in one or more cognitive domains:
- Learning and memory
- Language
- Executive function
- Complex attention
- Perceptual-motor
- Social cognition

B. The cognitive deficits interfere with independence in everyday activities. At a minimum, assistance should be required with complex instrumental activities of daily living, such as paying bills or managing medications.

C. The cognitive deficits do not occur exclusively in the context of a delirium

D. The cognitive deficits are not better explained by another mental disorder (e.g., major depressive disorder, schizophrenia)

Fig. 1: Diagnosis of dementia–DSM-5

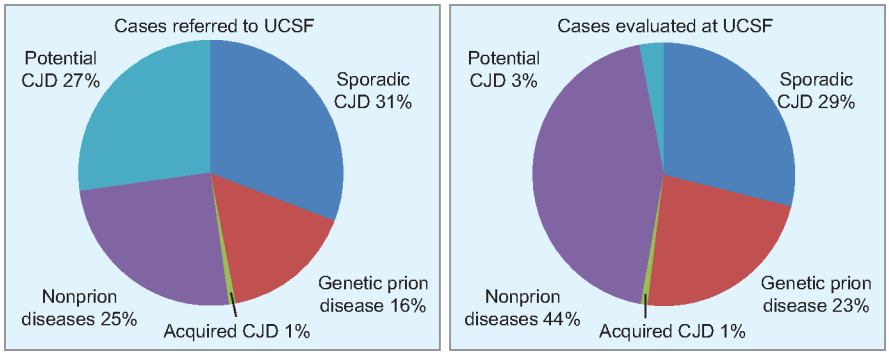

Fig. 2: Cases referred vs. cases evaluated at the tertiary care center, University of California, San Francisco (UCSF)

Leukoaraiosis (white matter hyperintensities) is increasingly recognized for its role in cognitive decline and is associated with hypertension, arteriosclerosis, amyloid angiopathy, Fabry's disease, vasculitis, and post-radiation status.[11] Clinically, it is linked with executive dysfunction and episodic memory impairment,[12] gait disturbances,[13] slowed cognitive processing,[14] depression,[15] and migraine.

COVID-19 and Rapidly Progressive Dementia

COVID-19 infection can mimic rapidly progressive dementia (RPD) through delirium, post- or parainfectious encephalitis, encephalomyelitis, cerebral hemorrhage, and infarction. Cognitive impairment is a common neurological manifestation. The pandemic-related lockdowns worsened cognitive deficits in patients with preexisting dementia. The long-term impact of COVID-19 on cognition is still under study, with potential mechanisms including:

- *Neuroinflammation*: Systemic inflammation may trigger brain inflammation.
- *Vascular effects*: COVID-19 can induce vascular changes contributing to cognitive decline.
- *Direct viral invasion*: SARS-CoV-2 may occasionally invade the central nervous system.

Infections and RPD

Several infections, including HSV encephalitis, coxsackie viral encephalitis, Lyme disease, syphilis, HIV, cryptococcosis, and prion diseases, can present as RPD, with Creutzfeldt–Jakob disease (CJD) being the classical prototype.

HSV encephalitis primarily affects the temporal lobes, leading to RPD via inflammatory neuronal damage. Neurological features may include aphasia, behavioral changes, seizures, and focal deficits.

Creutzfeldt–Jakob disease occurs in sporadic, iatrogenic, or familial forms, with sporadic CJD being most common. It progresses rapidly, with a median survival of 4.5–6 months and ~85% mortality within a year.[16] Onset typically occurs between 60 and 67 years.[17] Early signs include cognitive decline, behavioral and personality changes, motor and coordination difficulties, visual disturbances, and constitutional symptoms.[18] Cognitive deficits (confusion, memory impairment, poor concentration) appear first, while cortical involvement may cause aphasia, apraxia, or neglect. Motor signs include extrapyramidal and cerebellar symptoms and myoclonus; visual and sensory disturbances may also occur. Definitive diagnosis requires neuropathological confirmation via immunocytochemistry, Western blot for protease-resistant PrP, or identification of scrapie-associated fibrils (Table 3).

The diagnosis of CJD can be supported by a positive RT-QuIC assay in CSF or other tissues, which detects misfolded prion protein (PrPSc) through a fluorescent dye. RT-QuIC demonstrates high sensitivity (~92%) and specificity (~98%),[19] making it a reliable, less invasive alternative to brain biopsy, though repeat testing may be needed in cases of strong clinical suspicion due to rare false negatives. Brain MRI also aids diagnosis, with characteristic features including cortical ribbon sign, diffusion-weighted or FLAIR hyperintensities in the striatum, and thalamic changes such as the pulvinar and hockey stick signs.

Toxins and Dementia

Toxin-induced dementia arises from various exposures, with alcohol being a prominent cause, as seen in Marchiafava-Bignami disease, which damages the corpus callosum in chronic male alcoholics. Heavy metals—including mercury, arsenic, lead, toluene, and lithium—are significant contributors; mercury exposure can lead to Mad Hatter's syndrome (erethism),[20] marked by emotional, psychological, and motor disturbances. Chronic arsenic toxicity from contaminated groundwater can affect cognition and personality, especially in regions such as West Bengal and Bangladesh.[21] Certain medications, such as benzodiazepines, psychotropic drugs, and sodium valproate (via hyperammonemic encephalopathy),[22] may also cause cognitive and behavioral impairment. Additionally, radiation-induced encephalopathy from whole-brain irradiation in patients with metastatic tumors is a recognized cause of dementia.

Metabolic Causes

Cognitive impairment can result from various metabolic and endocrine disorders, many of which are reversible. Hypothyroidism, hyperthyroidism, and Hashimoto encephalopathy[23] may cause subacute cognitive decline, as can hypocalcemia, hypoparathyroidism, hypercortisolism, and repeated severe hypoglycemia in type 2 diabetes.[24] Other contributors include hepatic and uremic encephalopathy, while in chronic kidney disease, cognitive deficits are largely linked to hyperparathyroidism and anemia. Adult-onset inherited metabolic disorders—such as metachromatic leukodystrophy, adrenoleukodystrophy, adult polyglucosan body disease, cerebrotendinous xanthomatosis, Kufs disease, and rarer conditions such as advanced Wilson's disease, MELAS, and Leigh's disease—can also lead to dementia. Common, fully reversible causes include vitamin B1 and B12 deficiencies, frequently encountered in clinical practice.

Neurodegenerative Causes

Alzheimer's disease, frontotemporal lobar degeneration (FTLD), Dementia with Lewy bodies (DLBD), Corticobasal syndrome, and Progressive supranuclear palsy can cause RPD. Usually, Neurodegenerative diseases result in slowly progressive dementia exceeding 5 years.[25] Neurodegenerative dementias account for less than 5% of RPD.[26] Younger age of onset suggests FTLD, whereas DLBD occurs in older age. FTLD could also present with features of motor neuron disease. In conclusion, rapidly progressive neurodegenerative dementias with survival beyond 1 year typically represent non-CJD neurodegenerative dementias.[27] The terminal stage of late-life DLBD can sometimes resemble CJD.

Rare Possibilities

Primary CNS lymphomas, Metastases, Sarcoidosis, SLE, Sjögren, Celiac disease, Intravascular lymphomas, Atypical psychiatric disorders can present as rapidly progressive dementia. Whipple's disease, caused by *Tropheryma whippelii,* presents as a neuropsychiatric syndrome that progresses rapidly over months. Common clinical features include diarrhea, abdominal pain, weight loss, fever, and lymphadenopathy. CNS involvement occurs in 5–45% of cases.[28] Cognitive impairment occurs in 71% of cases, whereas psychiatric signs are seen in 44%.[29] Ataxia has been reported to occur in 45% of Whipple's cases.[30]

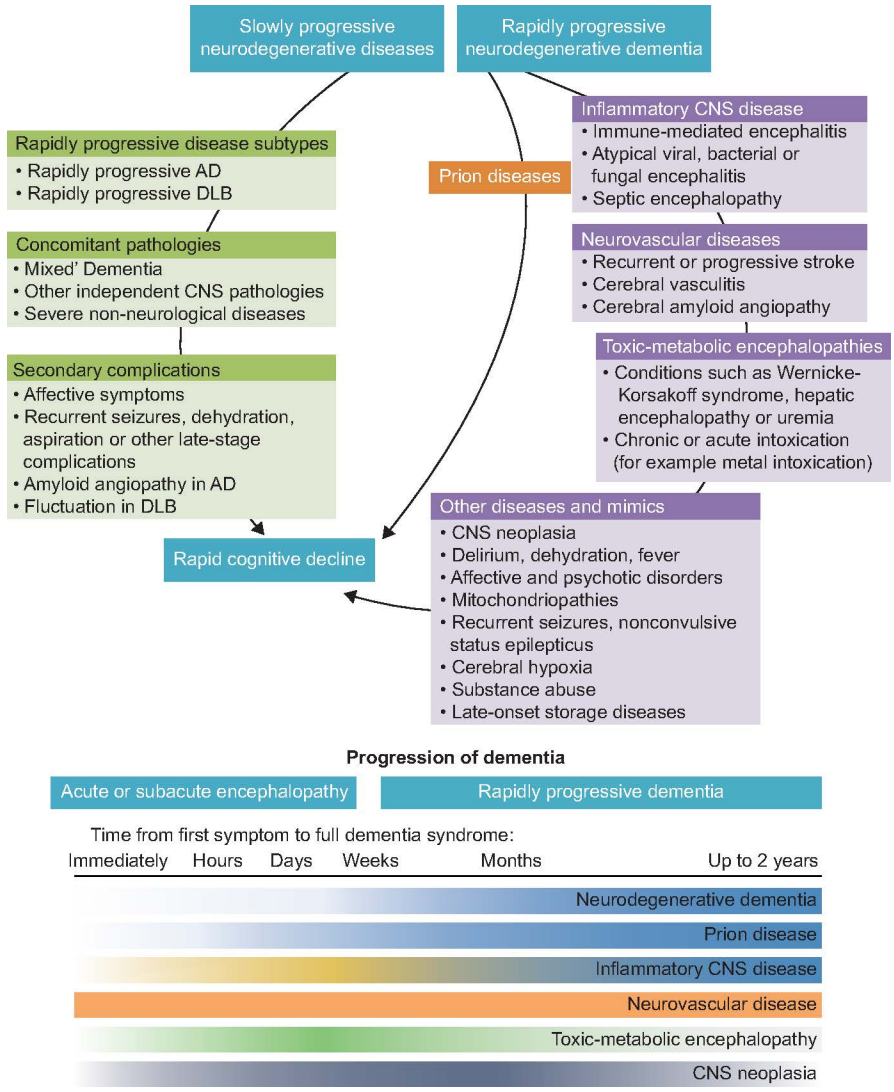

Fig. 3: Outlook of different etiologies of RPD and progression of dementia

Table 3: Diagnosis of probable CJD

Diagnostic criteria for sCJD	
1	RPD
2	At least two of the following: Myoclonus Visual or cerebellar signs Pyramidal/extrapyramidal signs Akinetic mutism
3	At least one of the following: Positive EEG findings Positive MRI findings Positive 14-3-3 protein test result
4	No indication of an alternate diagnosis

All four criteria must be satisfied to make a diagnosis of sCJD

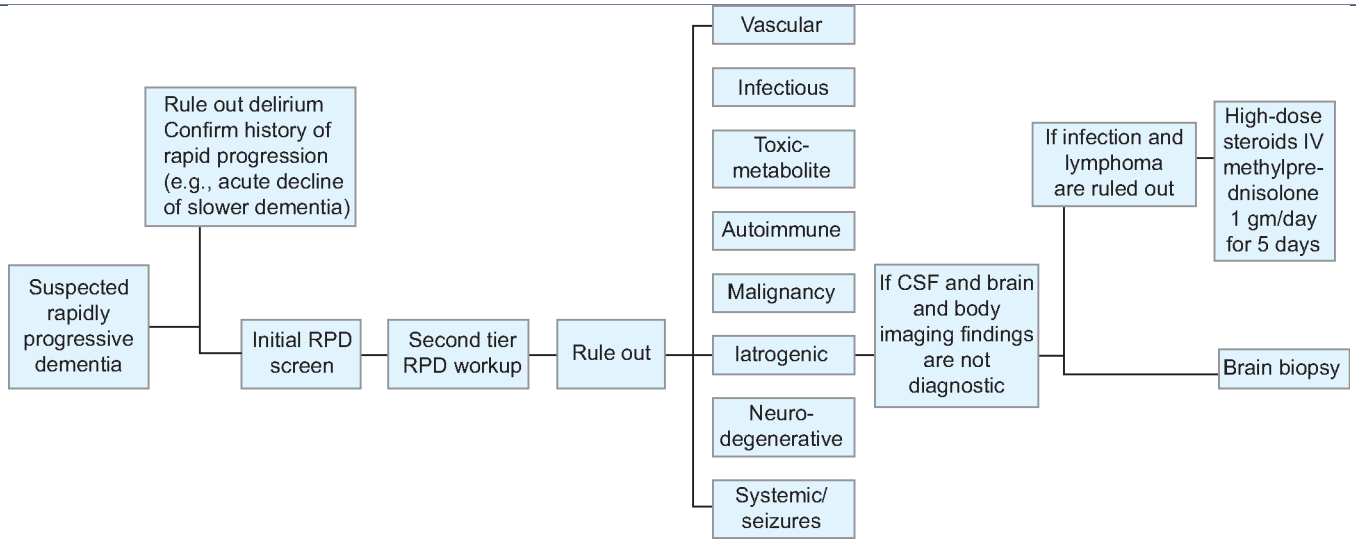

Fig. 4: Algorithm for evaluating RPD

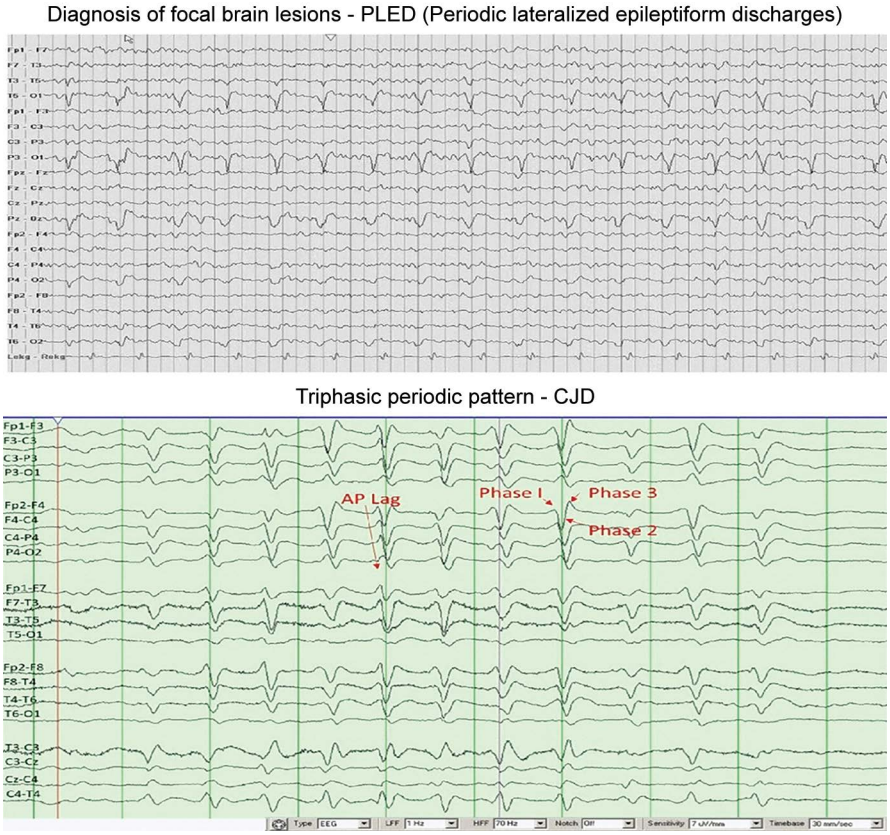

Fig. 5: Diagnosis of focal brain lesions–PLED (periodic lateralized epileptiform discharges) and triphasic periodic pattern–CJD

Diagnostic Algorithm for RPD

A structured diagnostic algorithm for rapidly progressive dementia is provided in Figure 4.

Role of Electroencephalography

Electroencephalography (EEG) helps to differentiate between various underlying causes and identify specific patterns associated with certain conditions.

Diagnosis of NCSE–Salzburg criteria

- Epileptiform discharges (ED) >2.5 Hz, or
- ED ≤2.5 Hz or rhythmic delta/theta activity (>0.5 Hz) and one of the following:
  - EEG and clinical improvement after intravenous antiepileptic drug, or
  - Subtle clinical phenomena, or
  - Typical spatiotemporal evolution (defined as incrementing onset - increase in voltage and change in frequency >1 Hz or change in location, or decrementing termination).
- If EEG improvement occurs without clinical improvement and if fluctuation without definite evolution occurs, those cases are considered possible NCSE.

Key Roles of EEG in RPD

- Differentiating delirium from dementia: Delirium often shows more pronounced diffuse slowing on EEG compared to dementia.
- Supporting diagnosis of prion diseases: In CJD, EEG may reveal characteristic periodic sharp wave complexes (PSWCs).
- Detecting seizure activity: EEG can identify epileptiform discharges and subtle seizures, including non-convulsive status epilepticus (NCSE) based on Salzburg criteria.
- Evaluating encephalopathies: Diffuse slowing helps assess brain dysfunction due to infections, toxins, or autoimmune disorders.
- EEG results are interpreted in conjunction with other investigations such as MRI, cerebrospinal fluid analysis, and laboratory tests to achieve an accurate diagnosis and guide management (Fig. 5).

Serum and CSF Markers of RPD

Serum and CSF biomarkers play an important role in the diagnosis of rapidly progressive dementias and related disorders. Increased proinflammatory cytokines (IL-6, IL-13, TNF-α, G-CSF) and specific antibodies—anti-NMDAR, anti-LGI1, anti-AMPA-R, and anti-GABA-BR—are indicative of autoimmune encephalitis, while anti-Hu and anti-Ma suggest paraneoplastic

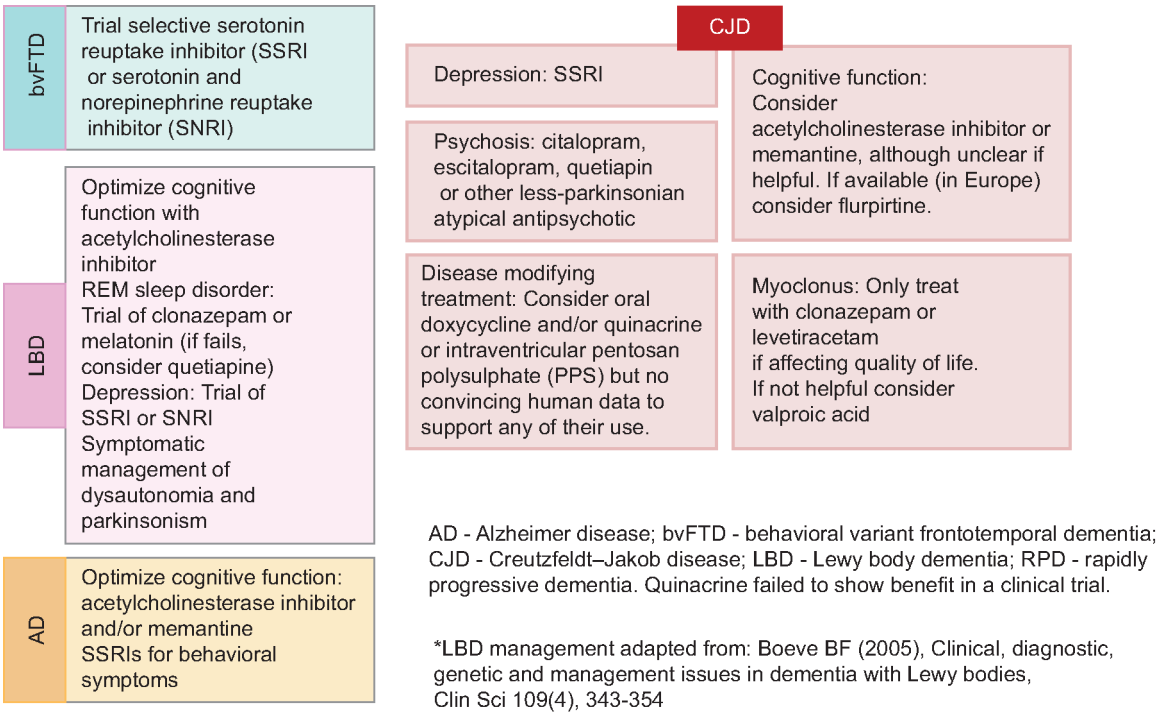

Fig. 6: Management algorithm for more common neurodegenerative causes of RPD

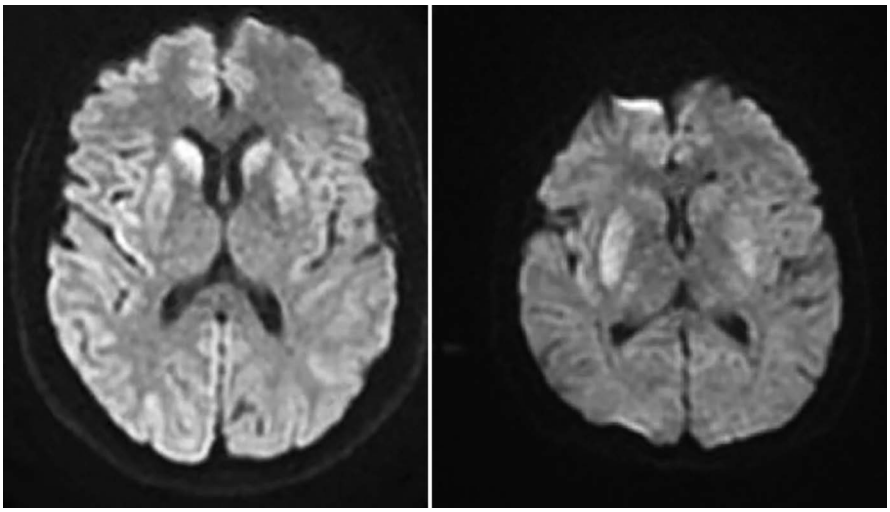

Fig. 7: MR Imaging showing striatal diffusion restriction in cases 1 and 2, respectively

variants. In Alzheimer's disease, t-tau, p-tau, and the t-tau/Aβ42 ratio serve as markers of neuronal and axonal injury. Neurofilament light chain (NfL) and YKL-40 reflect inflammatory processes, and MCP-1 is particularly elevated in vascular dementia. For CJD, CSF 14–3–3 protein demonstrates high sensitivity (92%) and specificity (80%). Although CSF NfL is nonspecific, it is frequently elevated in cases of RPD.

Newer Treatment Modalities

Quinacrine, an antimalarial drug, was tried for the management of RPD. The PRION-1 trial conducted at the National Prion Clinic in the UK found no benefit of Quinacrine on disease progression.[31] San Francisco trial also had the same results.

PRN 100, a monoclonal antibody, resulted in disease stabilization in three CJD patients. This research is ongoing. Brain autopsies also showed no neurotoxicity.[32]

The "Fastball" test, an at-home EEG-based device, has shown promise in detecting early signs of Alzheimer's disease within minutes, potentially aiding in earlier diagnosis (Fig. 6).[33]

Institutional Experience

Case 1: We had a 47/F, a homemaker, without any comorbidities, born to nonconsanguineous parents, without any family history, who presented with slowness of activities of daily life and stiffness of all four limbs for 6 months. History of decreased mood and anhedonia for 6 months. She has started misplacing things like her spectacles and room keys over the past 5 months. She had also started avoiding major discussions in the family. She was unable to utilize the mobile phone as she had previously. Over the last 1 month prior to presentation, she had a history of well-formed visual hallucinations as well. No history of fever. No history of prior TIAs. No history of loss of weight or appetite.

Overall, she had Parkinsonism, depression, psychosis, and rapidly progressive cognitive decline in the form of recent memory impairment and dysexecutive functions. On investigation, her total counts, serum B12, lipid profile, thyroid function tests, liver and renal function tests, and HbA1C were normal. ESR and CRP were elevated. MRI brain showed bilateral striatal diffusion restriction and T2/FLAIR hyperintensity.

The *second case* was a 55/F, known case of diabetes mellitus, who presented with Parkinsonism with postural instability and rapidly progressive cognitive decline over 8 months. There was no psychosis, cranial nerve involvement, weakness, myoclonus, dystonia, seizures, sensory involvement, or ataxia. Blood investigations, CSF, and EEG were noncontributory. The MRI brain was also similar to the previous presentation.

The probable differential diagnoses were autoimmune encephalitis, Hashimoto

encephalopathy, paraneoplastic syndrome, toxic encephalopathies such as carbon monoxide poisoning, and CJD (Fig. 7).

The breaking point in case 1 was CSF analysis, which showed elevated protein, normal glucose, and no cells. EEG was showing periodic sharp waves. CSF viral encephalitis panel was negative. CSF 14–3–3 is negative. CSF was positive for CASPR-2 antibodies. She was started on an IV pulse dose of steroids, which showed significant improvement in 1 week. She was continued on oral steroids and started on IV rituximab. Currently under follow-up. The second case had no such supporting investigations. On probing, she had a history of vomiting and loose stools prior to the onset of symptoms and was managed for hyponatremia. Hence, after excluding all possible causes, she was suspected of having developed extrapontine myelinolysis. She showed very minimal improvement with IV pulse steroids and was put on supportive therapy. Thus, RPDs need extensive workup to identify a possible treatable cause, as in case 1.

Conclusion

Rapidly progressive dementias (RPDs) present a diagnostic challenge due to their aggressive nature and the urgency for intervention. While the specter of prion diseases, such as CJD, often looms large in clinical considerations, it is crucial to recognize that RPDs encompass a diverse array of etiologies, many of which are treatable. These treatable conditions range from autoimmune encephalopathies and infectious diseases to metabolic disorders and toxic exposures. This article intends to provide a thorough overview of the wide range of differential diagnoses, highlighting the critical need for practicing neurologists to remain vigilant in identifying potentially reversible causes, by systematically evaluating each RPD case, and employing thorough diagnostic workups, we can strive to identify and address these treatable conditions, potentially preventing irreversible neurological decline and improving patient outcomes in the years to come.

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None.

Conflicts of Interest

None.

Informed Consent

Informed written consent was obtained from the patients for the publication of their data.

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