Atypical Presentation of Sporadic Creutzfeldt–Jakob Disease in a 59-Year-Old Male Patient

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Aims: Creutzfeldt–Jakob Disease (CJD) is a rare, fatal neurodegenerative disorder caused by prion proteins, leading to progressive brain damage. CJD has sporadic, variant, genetic, and iatrogenic forms, with sporadic being the most common, affecting 1–2 people per million annually. It typically presents with rapid cognitive decline, motor dysfunction, and personality changes, with no effective treatment available.

Methods: A 59-year-old physically fit, male with no past medical history presented to A&E with a two-week history of behavioural changes, aggression, left facial droop, disorientation, poor coordination, and mild dysarthria. He had repetitive speech and his GCS on admission was 14/15.

Stroke was suspected and CT scan showed hypoattenuation of the right frontal lobe. Contrast MRI showed multifocal areas of gyriform diffusion restriction affecting bilateral cerebral hemispheres. Initial differential diagnoses were stroke, vasogenic oedema, old brain injury, post-ictal changes, encephalitis.

EEG and initial blood tests were normal. CSF analysis revealed elevated proteins, normal glucose and lactate and no oligoclonal bands. CSF virology and cultures were negative. A protein assay from specialist services in Edinburgh confirmed a diagnosis of sporadic CJD.

The patient was discharged to a care home with clonazepam and a plan to introduce low-dose quetiapine if needed. Upon transfer, he became increasingly agitated, displaying verbal and physical aggression towards staff and residents, wandering and damaging property. His sleep was poor, and his aggression unpredictable, escalating late in the evening.

The care home implemented a low-stimulus environment with one-to-one care and covert medication administration. Initially treated with quetiapine and benzodiazepines, his aggression persisted, prompting introduction of haloperidol, which resulted in some reduction in aggression. He was later transferred to an inpatient ward.

On the ward, his dysphasia worsened, but he had no further behavioural challenges. He received regular physiotherapy, occupational therapy and specialised nursing care. Parkinsonism symptoms prompted the discontinuation of haloperidol, which was later reinstated at a lower dose due to return of restlessness. Quetiapine was gradually tapered off. At discharge, he was compliant with medication, required assistance with feeding and personal care, had severe dysphasia and some ataxia.

Results: This case demonstrates the atypical presentation of CJD with neuropsychiatric symptoms at onset, rather than the usual neurological signs. The patient's agitation and aggression required careful pharmacological adjustments and highlighted challenges in managing CJD's neuropsychiatric symptoms.

Conclusion: This case highlights the importance of considering CJD in the differential diagnosis for patients with rapid-onset neuropsychiatric disturbances. Early recognition and symptomatic management, although not curative, can improve the patient's quality of life during the disease's progression.

Clozapine-Induced Rhabdomyolysis in a Patient With Paranoid Schizophrenia: A Case Study Highlighting the Importance of Vigilance in Antipsychotic Therapy and Recurrence During Re-Challenge

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Aims: A 45-year-old Afro-Caribbean male with a history of paranoid schizophrenia, hypertension, Gilbert syndrome, epilepsy, vitamin D deficiency, mitral regurgitation, and penicillin allergy was admitted in March 2024 for clozapine titration following mental state deterioration. This was his first clozapine re-challenge since suspected clozapine-induced rhabdomyolysis in October 2022, during which CK levels had risen to 7442 IU/L, necessitating discontinuation.

Methods: Clinical findings: During titration in March 2024, CK levels rose to 7096 IU/L. The patient, engaging in vigorous exercise, reported mild myalgia but no severe symptoms or NMS.

Diagnostic focus: Clozapine-induced rhabdomyolysis was suspected. IV hydration was initiated, and CK levels decreased to 1500 IU/L after two days but later rose to 4500 IU/L. Clozapine was discontinued, and haloperidol was started, leading to CK normalization.

Second Re-challenge (August 2024): Re-challenged with clozapine, CK levels fluctuated but remained within acceptable limits (1016–833 IU/L), with no symptoms. The patient was encouraged to hydrate and avoid vigorous exercise.

Results: Clozapine is effective for treatment-resistant schizophrenia but risks rhabdomyolysis. The mechanism remains unclear but may involve direct myotoxicity or indirect factors like seizures. This case highlights the need for CK monitoring, especially during titration. Elevated CK levels exceeding 5000 IU/L necessitate clozapine discontinuation, IV hydration, and reassessment.

Re-challenging clozapine requires careful evaluation. This patient was successfully re-challenged under close monitoring with tailored CK testing, hydration, and activity recommendations. Vigilance and individualized care minimized risks while maintaining therapeutic benefits.

Conclusion: This case underscores the importance of routine CK monitoring, patient education, and interdisciplinary care during clozapine therapy, particularly in those with prior rhabdomyolysis. Personalized treatment strategies and adherence to evidence-based protocols ensure safety and optimize outcomes. Ethnicity- and sex-specific CK reference values should be developed to enhance clinical decision-making. Despite challenges, clozapine re-challenge is feasible with vigilant monitoring and risk mitigation strategies.