genetic testing for this person was classified as a variant of uncertain significance.

Uniquely, this case report describes an adult who has been tested, as opposed to paediatric cases which comprise much of the literature in this area. The notable case described by Verhowen of a patient who presented with a late onset of seizures, similar symptoms, progression and prior failed trials of medication as the case presented here, also benefited from genetic testing in later life. In addition, this patient responded well to sodium valproate which is known to be beneficial in cases with SLC6A1. This is most likely related to the GABA mediation. The findings of this case demonstrate the SLC6A1 gene-related disorder provides a unifying explanation for this diverse clinical phenotype, previously thought to be a constellation of syndromes and co-morbid symptoms.

The individual in this case study failed to respond to three adequate trials of medication for ADHD, all which have a robust evidence base. This suggests a possible alternate pathway for the development of ADHD features in cases such as this, as the majority of individuals with moderate-severe ADHD symptoms achieve symptomatic relief with pharmacological intervention

Conclusion: This case highlights the relevance of genetic testing in adults and the reporting of variants of uncertain significance that could possibly lead to reclassification of a variant.

Parkinson's Disease With Psychosis: A Case Report of Neuropsychiatric Manifestations

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Aims: Parkinson's disease (PD) is mainly a movement disorder, although 30% of PD patients may also suffer from psychosis, which may impair quality of life. PD psychosis (PDPsy) may result following approximately 10 years of treatment using dopaminergic agonists. PDPsy is characterized by recurrent and continuous hallucinations and delusions lasting for at least 1 month.

Methods: A 59-year-old female was admitted under Section 2 of the Mental Health Act (MHA) due to concerns raised by the police and her family regarding her mental health. The police were particularly alarmed after she repeatedly contacted them, expressing paranoid delusions. Her confusion and memory issues further contributed to the concerns leading to her admission. She was under the Early Intervention Psychosis team at the time of admission but her engagement with them was very erratic. She was started on quetiapine 50 mg ON but was non-compliant. She was diagnosis with PD 12 years prior to admission, and was still under the neurology team, and being regularly reviewed. The medication prescribed for PD were Sinemet 12.5/25 2 tabs QDS and ropinirole 8 mg BD.

Upon admission, the patient reported feeling monitored via 16 satellites connected to her television, broadcasting signals worldwide. She denied calling the police and instead suggested that her phone had been hacked. The police reported that she alleged that she had been sexually assaulted by her ex-partner or individuals organized by him while being drugged, though she had no memory of these events when questioned. She believed she was being followed, a notion she first experienced on a train to Cornwall a year prior. She also alleged that her ex-partner frequently entered her home at night to steal from her. She, also, exhibited delusional beliefs regarding her YouTube presence, asserting that her ex-partner manipulated satellites to influence her views online. Risk factors identified included medication non-compliance, poor insight, risk of falls due to Parkinson's disease, potential financial exploitation due to engagement with strangers on social media, and vulnerability.

Results: The differential diagnosis considered for this patient:

Delusional Disorder.
Late-Onset Psychosis.
Parkinson's Disease Psychosis.
Alzheimer's Disease with Psychosis.
Brief Reactive Psychosis.

A literature search showed that management of PDPsy involves a balance between reducing PD medication and introducing an antipsychotic for symptom management.

Conclusion: This case highlights the diagnostic challenges of psychosis in Parkinson's disease and underscores the importance of a multidisciplinary approach in managing psychiatric symptoms in neurodegenerative conditions.

Case Report: The Forgotten Functions of the Hindbrain

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Aims: This case highlights the cognitive functions of the cerebellum which is often forgotten about.

Methods: An 80-year-old lady with a previous mental health diagnosis of Bipolar Affective disorder (BIPAD). She presented to the general hospital in July 2024 with a sudden onset of confusion and aggressive behaviour. She was referred to the Psychiatric Liaison Service (PLS). On assessments, she presented with no clear mood or psychotic disorder suggestive of relapse in her BIPAD. She scored 8 out 30 on the Mini-Addenbrooke's Cognitive Examination (M-ACE) – losing marks on attention, memory – registration/recall, verbal fluency and the clock drawing test showed neglect. She lacked insight into her presentation and thus was in hospital under Deprivation of Liberty Safeguards (DoLS). Collateral history from her son corroborated that this was not a relapse in BIPAD. He enquired about an MRI head which revealed a small old infarct in the left cerebellar hemisphere.

Results: Cerebellar cognitive affective syndrome (CCAS; Schmahmann's syndrome) would explain the timeline of symptoms, assessment findings and collateral history. CCAS is characterized by deficits in executive function, linguistic processing, spatial cognition, and affect regulation. Neuropsychiatric features include impairments in attentional control, emotional control, psychosis spectrum disorders and social skill set. The deficits suggest a disruption of the cerebellar modulation of neural circuits that link frontal, parietal, temporal, and limbic cortices with the cerebellum which is known as Cerebellum-Cerebrum cortex loop. Movement, co-ordination, and cognition are intricately connected within the brain, however the role of the cerebellum in cognition has often been ignored.

Conclusion: Clinicians tend to focus on the motor-coordination functions of the hindbrain, and this case report highlights cognitive functions of the hindbrain that are often forgotten. CCAS may be underdiagnosed due to the lack of awareness of the intricate

connections between the hindbrain and forebrain through the cerebellum-cerebrum cortex loop, responsible for cognitive function within the hindbrain. This can lead to inappropriate treatment plans being devised for patients, and subsequent negative impact on management outcomes and even quality of life.

Manganism – Unusual Presentation at Memory Assessment Service

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Aims: More than 200,000 clients are referred to memory assessment annually in the United Kingdom. Alzheimer's disease and vascular brain injury are found to be the main causes for the memory impairment among these clients. However, a minority of clients present with memory impairment due to metabolic causes.

Methods: Mr M, a 65-year-old Caucasian male was referred to memory assessment service due to memory problems for 7 months duration. He had evidence of amnesia, aphasia and apraxia. His executive functions, recognition, personality were intact. He scored 91/100 in Addenbrooke's cognitive examination. M also struggled with balance and tremors of his limbs.

He was diagnosed with liver impairment secondary to metabolic syndrome, type II diabetes, hypertension, long-standing cervical pain and heart block. He reported to sleep more than usual and was suffering from frequent episodes of constipation which was exacerbated by morphine. His partner reported that his cognitive symptoms coincides with constipation.

M was on treatment for mixed anxiety and depressive disorder with sertraline for 4 years. He was euthymic at presentation.

His laboratory work showed mild anaemia and low platelets. He was known to have a platelet disorder as well. Most recent HbA1c was raised but other basic blood investigations were largely within normal ranges.

His magnetic resonance imaging scan showed Symmetrical T1 high signal in bilateral globus pallidus on sagittal T1 weighted images. It was concluded that appearances could be due to manganese deposition consistent with history of hepatic dysfunction.

Small vessel ischaemic changes were seen in bilateral supra-tentorial white matter.

His electro encephalogram was in keeping with diffuse cerebral dysfunction.

Neurology multi-disciplinary meeting has concluded that the clinical presentation is one of a hepatic encephalopathy.

Results: Human physiological functions require many essential elements and manganese is identified as an essential element. Accumulation of manganese in excessive amounts in brain due to various metabolic derangements can causes central nervous system dysfunction known as Manganism. Manganism is an extrapyramidal disorder characterized by motor disturbances associated with neuropsychiatric and cognitive disabilities similar to Parkinsonism.

Manganese is cleared from the body by the liver. Chronic liver impairment hinders the clearing process causing accumulation of manganese in blood and brain. M was suffering from chronic liver impairment which was the most likely cause for manganese deposition in his brain.

Conclusion: It was concluded that M's cognitive impairment was due to hepatic encephalopathy and Manganism. Clinicians need to be aware of Manganism while assessing the patients with chronic liver impairment and neurocognitive dysfunction.

Levetiracetam Induced Psychosis – A Case Study

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Aims: Levetiracetam is a broad spectrum antiepileptic used in a variety of seizure disorders in both adults and children. Although a popular antiseizure medication, levetiracetam's association with new onset behavioural disturbance such as agitation, hostility, psychosis and mood symptoms has been widely reported in scientific literature. Seizure disorders themselves can present with psychiatric manifestations. We are reporting a case of an adolescent male where the interphase of physical and mental health came into play.

Methods: A 13-year-old male presented to A&E brought by his family following a referral from the epilepsy clinic due to two weeks history of bizarre behaviours including abnormal gait, tapping on the shoulders of his family members, talking to himself and generally being more irritable. From history, we noted he had been diagnosed with epilepsy (unspecified) for two years and recently his seizure activities increased in frequency, which prompted his neurologist to increase his antiseizure medication (levetiracetam from 1250 mg twice a day to 1500 mg twice a day) two weeks prior to his presentation, which coincided with the onset of his symptoms.

He reported experiencing intrusive and unpleasant thoughts about the safety of his family, experiencing multiple times of the day and to reduce the anxiety he was tapping on their shoulder, and checking the locks of the door and windows of the house, the thoughts and rituals corresponded to obsession and compulsion. He also reported thought broadcasting – people are able to know what he was thinking, and abnormal perception of hearing his own thoughts spoken aloud – appeared to be Gedankenlautwerden.

In the emergency department he underwent extensive blood (including auto-antibodies associated with first episode of psychosis) and radiological investigations to rule out acute neurological causes. The investigations did not yield any positive results, his levetiracetam level was also within therapeutic range.

The description of his seizures indicated that he experiences gustatory and olfactory auras with focal to generalised seizures followed by postictal transient paresis of the left arm, which has been consistent over the course of the two years he had the seizure.

Results: Diagnostic formulation was the acute onset obsessive-compulsive and psychotic symptoms are likely the direct result of the increase in the dose of levetiracetam which had a temporal relationship, differentials included psychiatric symptoms associated