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.

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Manganism – Unusual Presentation at Memory Assessment Service

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doi: 10.1192/bjo.2025.10760

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.

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Levetiracetam Induced Psychosis – A Case Study

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doi: 10.1192/bjo.2025.10761

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