working diagnosis of catatonia. He responded positively to a lorazepam challenge therefore commenced on 1mg of lorazepam twice daily. Despite increasing doses, the catatonia worsened with severe psychomotor retardation, "psychological pillow" and nil food or fluid intake with Bush Francis score of 18. ECT was arranged as an emergency treatment but put on hold while tolerating all food and fluids requirements via nasogastric tube. Lorazepam dose was titrated to 3mg three times daily but signs seen of benzodiazepine toxicity therefore dose was reduced and ECT arranged for treatment resistance. Improvement seen on reduced dose prior to receiving ECT, therefore ECT put on hold again. His lorazepam dose was titrated up at a slower rate to 4mg three times daily which he was able to tolerate. His catatonia fully resolved at 12mg. Once stable, lorazepam dose was very gradually decreased until stopped. No evidence of catatonia returned.

Results. Medical and psychiatric causes of catatonia were explored.

Two positive blood anti-NMDA receptor tests two months apart; both 1/10 titre. This was discussed with the specialist neurology team in Oxford who advised this was an incidental finding with no clinical implication (1% of healthy population are positive).

Throughout admission, possible fleeting psychotic and depressive symptoms were noted, including not trusting food, hallucinations, worries about contamination and apathetic mood. However, these all improved as the catatonia was treated.

Conclusion. There was no clear underlying psychiatric or medical illness identified as a cause of the patient's catatonia. Catatonia has a higher prevalence in people with autism. At discharge he was well and reintegrated back to community life without requiring further medication.

Cognitive Decline With Low-Dose Procyclidine and Improvement Following Removal of Anticholinergic Burden

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Aims. A recent Cochrane review published in December 2023 concluded that "no trials found that interventions to reduce anticholinergic burden led to any other improvements in cognition compared to usual care". We describe the case of a 62-year-old lady who developed significant cognitive decline following the initiation of a low dose of procyclidine, which was rapidly reversed upon stopping the medication.

Methods. We present the case of a 62-year-old lady with a diagnosis of schizo-affective disorder, whose symptoms had been stabilized on a regime of lithium carbonate 500mg nocte, sulpiride 400mg BD and fluoxetine 20mg OD. When the patient presented to the outpatient clinic, she was noted to have bilateral coarse tremors and slight cogwheel rigidity. Procyclidine was started at a dose of 5mg OD to manage these extrapyramidal side-effects.

Following this, family members reported that the patient had difficulty initiating and following conversations. Short-term memory was affected and she was observed to have reduced attention span. These problems were reportedly getting worse with time, with a simultaneous decline in functional abilities. She was no longer able to carry out her daily shop, and family members ensured that she was no longer driving as they had concerns about her road safety. She stopped taking procyclidine after 1 month and notably, these problems ceased within one week of stopping the medication.

Cognitive testing confirmed that the patient was cognitively intact after procyclidine was stopped. The patient scored 96 on the 'Addenbrooke's Cognitive Examination' scale, which falls within the normal range. The 'Instrumental Activities of Daily Living' scale was administered to assess functioning at the time of the cognitive impairment. This returned a score of 1/8, indicating that there was significant functional impairment secondary to cognitive impairment when prescribed procyclidine. The 'Informant Questionnaire on Cognitive Decline in the Elderly' was administered to objectively quantify the extent of cognitive decline as noted by family. This returned a score of 4.3, confirming that the patient's cognition had indeed been worse when compared with her baseline.

Results. Our case report highlights the rapid improvement in cognition with the removal of anticholinergic burden in a 62-year-old female. Our report can, therefore, be a harbinger for more robust trials to determine the efficacy of interventions to reduce anticholinergic burden in preserving or improving cognition.

Conclusion. It is important to monitor for any change in cognition when prescribing anticholinergic medication in at-risk individuals.

Adult Onset Ornithine Transcarbamylase Deficiency: A Rare Cause of Psychosis

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Aims. Ornithine transcarbamylase (OTC) is an enzyme of the urea cycle catalyzing the condensation of carbamyl phosphate and ornithine to form citrulline. OTC deficiency leads to elevated serum ammonia and presents as different neurological or psychiatric symptoms. OTC deficiency is an X-linked inborn error of metabolism and most cases occur in neonatal period with severe presentation. Lesser known is the late-onset form that remains latent from infancy and only presents with intriguing symptoms mimicking psychiatric disease in adulthood.

Methods. Case report.

Results. We describe a case of adult-onset OTC deficiency in a 40-year-old man with borderline intellectual functioning and a psychotic episode following a protein rich meal. The case was first diagnosed as undifferentiated schizophrenia, until the genetic study was carried out.

Conclusion. Awareness of the adult onset ornithine transcarbamylase deficiency being a rare but possible differential diagnosis in a patient with acute psychiatric symptoms with hyperammonemia. Organic causes such as cerebral, metabolic, toxic causes of psychosis should be actively sought especially when encountering cases of acute psychosis.