risk of relapse. Currently, there is limited clinical experience of managing lithium in this context.

Methods. 49 yr old female diagnosed with schizoaffective disorder well-maintained for several years on aripiprazole depot and 800mg lithium carbonate (Priadel) with therapeutic levels in treatment range (0.4–0.8mmol/L). Severe obesity (BMI 41kg/m^2) despite dietary modifications and metformin trial, and recently diagnosed with diabetes. Family history of cardiovascular disease and diabetic related complications with early mortality were additional factors in her request for bariatric surgery. Multidisciplinary discussion including patient, psychiatrist, mental health pharmacist, specialist bariatric dietician and GP prior, to ensure sharing of relevant information pertinent to re-titration and monitoring of lithium therapy and risks of toxicity and relapse.

Results. Patient underwent sleeve gastrectomy with discontinuation of lithium 72 hours prior to surgery. Stomach pouch capacity reduced to 120ml and advised daily fluid intake 500–1000ml in first two weeks. Lithium therapy re-commenced when fluid intake adequate and renal function within normal limits. Formulation changed to liquid for 6–8 weeks to avoid disruption to the healing line, and the dose gradually re-titrated with close monitoring of serum lithium levels. Stabilised on reduced dose of 400mg Priadel at 3 months with therapeutic levels. At 6 months BMI reduced to 32kg/m^2, antihypertensive and metformin discontinued and maintained remission of schizoaffective disorder.

Conclusion. Sleeve gastrectomy is an increasingly common procedure to treat obesity, with potential long-term positive physical health outcomes and reduction in mortality which may have a role in addressing health inequalities for SMI patients. Psychiatrists need to be aware of key aspects of bariatric surgery particularly relating to safe and effective prescribing of psychotropic medication including potential change to liquid or orodispersible formulation in the post-operative period, close monitoring of serum lithium levels in the short and medium term due its narrow therapeutic index, and consideration of longer-term dose adjustments due to ongoing weight loss.

A Suspected Case of Kluver-Bucy Syndrome in an Adolescent Male Following SARS-CoV-2 Infection

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Aims. We present a case of suspected Kluver-Bucy syndrome in an adolescent male, following a SARS-CoV-2 (Covid-19) infection. To the best of our knowledge, KBS has not been associated with Covid-19 before.

Methods. A 15-year-old male with a background of autism spectrum disorder (ASD) was reviewed in a children and adolescent mental health outpatient clinic. The young person was non-verbal, and history was taken from his next of kin. In the last four weeks, he had developed acute onset hyperphagia with weight gain (88th percentile for age), new onset physical and verbal aggression, and hyperorality, whereby the young person was exploring household objects with his mouth. A degree of hypersexuality was also noted in the form of rubbing and touching of the genital area.

There was no history of trauma or epilepsy; recent traveling or environmental change; psychosocial stressors or new medications, operations, or immunisations in the past year. The young person had a Covid-19 infection the month before the symptoms started. He was immunised against Covid-19 and this was the second time he contracted the infection, the first being 1 ½ years ago with full recovery.

The sudden onset of hyperphagia, aggression, hyperorality, and hypersexuality with the only known precipitating factor the recent Covid-19 infection, raised clinical suspicion for Kluver-Bucy syndrome. Six months later, the symptoms were milder but still present and no other cause had been identified. Due to ASD features, visual field testing, brain imaging, or routine blood tests were either not possible or required sedation and are being arranged with the support of his general practitioner.

Results. Kluver-Bucy syndrome is a rare neurological disorder characterised by a distinct constellation of behavioural and cognitive symptoms resulting from bilateral lesions or dysfunction in the temporal lobes, particularly the amygdala. Patients often exhibit alterations in their behavioural repertoire, including hyperorality, hypersexuality, disinhibited behaviour, and visual agnosia. The presentation has been associated with temporal lobe infarcts, epilepsy, and herpes simplex encephalitis. The differential diagnosis was based on the fulfilment of clinical criteria for KBS, while other differentials included metabolic causes or behavioural manifestations related to ASD. Although investigations to explore other causes of symptoms are still being arranged, clinical suspicion for KBS was based on the presence of diagnostic criteria and the recent viral infection.

Conclusion. Research is needed to identify potential associations between SARS and neuropsychiatric manifestations, while clinicians should be aware of the possibility of such complications.

A Case Report of a 16 Year Old With Catatonia With No Clear Medical or Psychiatric Cause

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Aims. 16 year old male with previous diagnoses of autism spectrum disorder and severe anxiety disorder was referred to Child and Adolescent Mental Health Services (CAMHS). His presentation included increasing anxiety, difficulty articulating his thoughts and emotions, difficulty completing tasks, school non-attendance, reduced food intake and possible auditory hallucinations. Risperidone was trialled in the community however refused after 5 days due to "brain fog". He was seen by CAMHS community team twice weekly for 3 months prior to his emergency detained admission to adolescent psychiatric inpatient unit, due to no oral intake for 72 hours.

Family history included schizophrenia, bipolar disorder, depression and anxiety.

Methods. Upon admission, symptoms observed included reduced verbal communication, psychomotor retardation, low mood, agitation, sleep difficulties, ambitendency, echolalia and poor oral intake. He had a Bush-Francis rating score of 8 and given a

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.

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