Emergence of Self-Injurious Behaviour in a Student With Autism Following Peer Exposure: A Case Study

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Aims: People with autism often exhibit heightened sensitivity to environmental stimuli, including social and emotional cues. Exposure to maladaptive behaviours in peers may act as a powerful trigger, influencing emotional regulation and behavioural responses. This case highlights the impact of environmental modelling on self-harm behaviours in a student with autism.

Methods: We present the case of a student with autism, who developed self-injurious behaviours following exposure to a peer engaging in self-harm at college. The patient demonstrated increased emotional distress, repetitive rumination on the observed behaviour, and subsequent imitation of the act. No acute psychotic symptoms or major mood disorder were identified. The onset of self-harm appeared temporally linked to the environmental exposure rather than internal psychopathology.

Results: This case illustrates the vulnerability of autistic people to social modelling and environmental influences. Heightened empathic sensitivity, difficulties with emotional regulation, and concrete thinking styles may contribute to the internalization and replication of observed behaviours. Early recognition of environmental triggers, particularly within college settings, is crucial. Interventions focused on environmental modification, psychoeducation for caregivers and educators, and autism-adapted emotional regulation strategies were associated with a reduction in self-harm behaviours. This case emphasizes the importance of multidisciplinary collaboration and proactive monitoring of peer-related stressors in autistic populations.

Conclusion: This case underscores the profound environmental sensitivity of people with autism and the potential for peer behaviours to precipitate self-harm through social modelling. Clinicians, caregivers, and educators should remain vigilant to environmental risk factors and prioritize early intervention, college-based collaboration, and psychoeducation. Increased awareness of these vulnerabilities may help prevent self-injurious behaviours and promote safer, more supportive learning environments for students with autism.

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The Dopaminergic Tug of War: A Case of Psychosis at the Crossroads of Endocrinology and Psychiatry During Cabergoline Therapy

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Aims: Dopamine agonists like cabergoline are considered first-line therapies for prolactin-secreting pituitary microadenomas. Although efficacious from an endocrinological perspective, the use of dopaminergic stimulation can potentially exacerbate or unmask psychiatric symptoms, including psychosis. The management of emergent psychosis in this scenario is a challenge, as antipsychotic dopamine blockade can potentially worsen hyperprolactinemia. Aripiprazole, a dopamine D_2 partial agonist, is often preferred for its antipsychotic properties and relative prolactin neutrality; however, its efficacy in the treatment of severe psychosis while on dopamine agonist therapy is unclear.

Methods: A 29-year-old woman with a 12-year history of obsessive-compulsive symptoms, who had remained stable and had never been hospitalized, was diagnosed with hyperprolactinemia after the development of galactorrhea and menstrual disturbances. MRI showed a 6 mm pituitary microadenoma, and cabergoline 0.5 mg twice a week was started with good biochemical control. Shortly thereafter, she experienced severe psychiatric deterioration, including persecutory delusions, ideas of reference, refusal of food, severe self-neglect, and social withdrawal, requiring psychiatric hospitalization. Mental state examination showed prominent psychotic features coexisting with the patient's previous obsessive phenomena.

Aripiprazole was started at 5 mg/day and gradually increased to 20 mg/day over three weeks, chosen for its partial dopamine agonist properties and prolactin-sparing action. Despite optimal dosing, good tolerability, and maintenance of prolactin levels, there was little relief from psychosis or behavioral disturbance. Because of persistence of severity, aripiprazole was stopped and olanzapine started at titrated upto 15 mg/day. An SSRI was continued for obsessive symptoms. Cabergoline was continued under strict endocrinological follow-up.

Results: Within two weeks of the initiation of olanzapine, there was a marked improvement in persecutory delusions and behavioral disturbance, along with the return of appetite, sleep, and self-care, and partial recovery of insight. Serum prolactin levels remained within acceptable limits (46 ng/mL), and follow-up MRI at three months showed no progression of the tumor.

Conclusion: This particular case illustrates the shortcomings of aripiprazole in dealing with severe psychosis, especially in the setting of dopamine agonist therapy, even in the presence of theoretical endocrine benefits. In situations where endocrine function is substantially affected, psychiatric management may need to focus on effective antipsychotic therapy, with close interdisciplinary attention to minimize endocrine risk.

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Back to Basics: A Clinical Diagnosis of Behavioural Variant Frontotemporal Dementia (bvFTD)

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Aims: Behavioural variant frontotemporal dementia (bvFTD) is characterised by early and progressive changes in behaviour, personality and executive functioning. Neuroimaging and biomarkers can support diagnosis, these investigations may be normal in the early stages, increasing diagnostic uncertainty and potential misattribution to primary psychiatric illness. In such cases, a comprehensive neuropsychiatric assessment and collateral history remains key to diagnosis.

Methods: A woman in her 50s was referred to neuropsychiatry with a 3-year history of disinhibition, emotional blunting, aggression, repetitive behaviours, and decline in social functioning. Assessment included neuropsychological testing (ACE-III and RBANS Update), brain MRI, FDG-PET, DAT scan, EEG, and CSF analysis (including autoimmune and infectious panels). Genetic testing was also

undertaken. The case was reviewed by a dementia Neuroradiology multidisciplinary team and nuclear medicine consultant.

Results: Despite clear and progressive behavioural and functional deterioration, structural and functional neuroimaging, EEG, and CSF investigations were initially unremarkable. Neuropsychological testing demonstrated multi-domain cognitive impairment, with particularly marked deficits in visuospatial ability and memory (≤1st percentile on RBANS). Behavioural features included public disinhibition, rigidity, aggression, loss of empathy, and impaired insight. No primary psychiatric disorder, delirium, or alternative neurological diagnosis was identified. A clinical diagnosis of probable bvFTD was reached based on the behavioural profile, collateral history, and progression. Minimal symptomatic improvement was observed with the prescription of Quetiapine and Carbamazepine. Subsequent genetic testing identified a pathogenic C9orf72 hexanucleotide repeat expansion, confirming the diagnosis.

Conclusion: This case highlights how behavioural variant frontotemporal dementia may be diagnosed clinically despite normal imaging and biomarker studies, with genetic testing serving as confirmatory tool. Careful behavioural evaluation, collateral history, and longitudinal follow-up remain essential in complex neuropsychiatric presentations. This case supports early consideration of genetic testing in diagnostically challenging cases of suspected bvFTD.

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Recurrent Small Bowel Obstruction Following Combined Use of Clozapine and Tirzepatide: A Case Illustrating Additive Gastrointestinal Hypomotility

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Aims: Aims (Background)

Clozapine is associated with severe gastrointestinal hypomotility, constipation, ileus, and bowel obstruction through anticholinergic and antiserotonergic mechanisms. Tirzepatide, a dual glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide-1 (GLP-1) receptor agonist, delays gastric emptying and intestinal transit as part of its pharmacodynamic effect. Both agents independently impair gastrointestinal motility, yet their combined use is increasing as tirzepatide is prescribed to manage clozapine-associated metabolic dysfunction.

We hypothesised that the pharmacological combination of clozapine and tirzepatide may produce additive gastrointestinal hypomotility, increasing the risk of small bowel obstruction (SBO) in vulnerable patients.

Methods: Methods (Case Report)

We describe a single case (n=1) of a 54-year-old Black British woman with paranoid schizophrenia treated with clozapine since 2016, type 2 diabetes mellitus, and chronic constipation despite regular laxative therapy. Her medical history included extensive abdominal surgery for sigmoid volvulus with subtotal colectomy and ileostomy in 2012, followed by reversal in 2013.

Tirzepatide was initiated in March 2025 by the diabetes team for poor glycaemic control. Weight, glycaemic indices, clinical presentation, and radiological findings were reviewed using electronic health records.

Results: Results (Discussion)

Following initiation of tirzepatide, significant metabolic improvement occurred, with weight reduction from 71.5 kg to 64 kg and Hbs-782c improvement from 86 mmol/mol to 43 mmol/mol within four months.

In May 2025, approximately two months after starting tirzepatide, the patient was admitted with acute abdominal pain and vomiting. Computed tomography (CT) imaging demonstrated markedly dilated small bowel loops (up to 63 mm) with an abrupt transition point, consistent with SBO. A second SBO occurred in October 2025, again confirmed radiologically with dilated loops and a transition point, without evidence of malignancy, perforation, or ischaemia.

There was no documented history of SBO in the years preceding tirzepatide initiation. Both episodes were managed conservatively. Tirzepatide was discontinued following the second obstruction.

Conclusion: Conclusion

This case highlights a potential pharmacodynamic interaction between clozapine and tirzepatide, resulting in additive gastrointestinal hypomotility and recurrent SBO in a high-risk patient. While tirzepatide provided substantial metabolic benefit, its use alongside clozapine may pose significant gastrointestinal risk. Careful patient selection, baseline bowel assessment, and close monitoring are essential when prescribing GLP-1/GIP agonists to individuals receiving clozapine.

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Psychosis-Related Refusal of Life-Sustaining Insulin: Clinical and Legal Challenges at the Mental Health Act–Mental Capacity Act Interface

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Aims: Background

People with severe mental illness experience significantly higher rates of physical morbidity and premature mortality, with diabetes mellitus being a major contributor. When psychotic symptoms impair insight and decision-making, refusal of essential medical treatment can present acute and recurrent risks. In such situations, clinicians must navigate the interface between the Mental Health Act (MHA) and the Mental Capacity Act (MCA), two statutory frameworks that were not designed to address sustained community-based enforcement of physical healthcare in the context of fluctuating mental capacity.

Methods: Case Report

We describe the case of a patient with a chronic psychotic disorder and insulin-dependent diabetes mellitus who repeatedly refused insulin during psychotic relapses, leading to recurrent episodes of diabetic ketoacidosis requiring emergency hospital admission. During periods of florid psychosis, the patient lacked capacity to make decisions regarding insulin administration, with refusal directly driven by delusional beliefs. Despite multidisciplinary involvement, optimisation of psychiatric treatment, and repeated capacity assessments, adherence could not be sustained in the community. Each crisis was managed reactively through emergency admission, with no clear mechanism for anticipatory or preventative intervention once the immediate medical emergency resolved.