Social Isolation and Anomalous Proprioceptive Experiences in Schizophrenia: A Case Study

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Aims. Integral to Bleuler's concept of schizophrenia, anomalous beliefs regarding the self are crucial to maladaptive social functioning. In schizophrenia, a predisposition to unusual bodily experiences, coupled with reduced awareness and increased social isolation, leads to hallucinations and delusions. Proprioceptive hallucinations, a subset of bodily hallucinations, present a challenging diagnosis due to their subjective nature, often resembling genuine bodily perceptions. We present the case of a 42-year-old man with untreated psychotic illness, manifesting perceptual abnormalities in the modality of proprioception.
Methods. Mr. X was referred to Early Intervention in Psychosis (EIP), believing that all his joints were dislocated despite a normal neurological examination, Magnetic Resonance Imaging (MRI), and blood tests. Pertinently, childhood adversities and a seven-year history of prodromal and schizophrenia symptoms, chronic marijuana usage, potentially triggered by separating from his ex-partner, were present. At assessment, Mr. X recalled a delusional memory from age 5, seemingly heralding the onset of his illness. He displayed thought disorder, poor sleep and lacked insight. Olanzapine titrated to 15mg omni nocte (ON) improved sleep, but insight remained poor.
Results. This case of rare proprioceptive hallucinations presenting in middle-age underscores the impact of positive schizophrenia symptoms on social impairment, suggesting a link between unusual bodily experiences and social isolation. Proprioception, encompassing joint perceptions, muscle force, and effort, contributes to body image by combining with exteroception. Interactions with others, influenced by our bodily sense, are crucial for adaptive social functioning. The social deafferentation hypothesis posits that loneliness in schizophrenia may heighten susceptibility to bodily aberrations. The psychological formulation and the chronic use of marijuana on Mr. X's psychopathology, although not thoroughly explored, cannot be overstated.
Conclusion. Proprioception, vital for body image and social interactions, contributes to maladaptive functioning. The potent link between positive schizophrenia symptoms and social impairment needs exploring.

Paediatric Catatonia as a First Presentation of Autism: A Case Report

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Aims. Catatonia is a rare neuropsychiatric syndrome in children. It is characterised by mutism, stupor, posturing, negativism, and rigidity. Historically, catatonia was associated only with psychosis, however catatonic symptoms are being recognised as more prevalent in people with Autism Spectrum Disorder (ASD). Our case report highlights the importance of investigating the potential underlying psychopathology and/or neurodevelopmental condition as this may guide management.
Methods. We present a case of a boy in early adolescence who was admitted to the Emergency Department for abnormal slowing of movement and stuttered speech. He described losing all interest in his hobbies, lying down for long periods of time, sometimes being unresponsive and 'freezing' in place. On examination, his symptoms were consistent with catatonia: mutism, grimacing, abnormal gait, and ambitendency were all present. He was investigated extensively to rule out medical and neurological causes, all of which were normal. He was assessed and managed by the Centre for Interventional Paediatric Psychopharmacology and Rare Diseases (CIPPRD). After appropriate treatment, he was discharged from the hospital and was managed jointly by CIPPRD and the local Child and Adolescent Mental Health Service (CAMHS). This assessment revealed that the presentation of catatonia occurred during a depressive episode on a background of ASD and underlying Intellectual Disability. He was prescribed fluoxetine as opposed to benzodiazepines or antipsychotics, which led to the catatonic symptoms receding. The neurodevelopmental review revealed that his pattern of social communication and speech after catatonia improvement was consistent with ASD, which was then formally diagnosed.
Results. Untreated catatonia can be fatal. Early diagnosis and management are crucial to avoiding complications. Our case report highlights the challenge of treating paediatric catatonia and the diagnostic and therapeutic importance of understanding underlying psychopathology to decide treatment. Studies have shown that in this population, assessing and treating the underlying psychopathology as opposed to sole use of the lorazepam is essential.
Conclusion. Catatonia in paediatric and adolescent populations may be a first presentation of emotional and behavioural problems underlying autism spectrum disorder (ASD). When treating catatonia, consideration of the underlying psychopathology may warrant alternative pharmacological treatments to the traditional lorazepam challenge test and antipsychotics. The course of catatonia and associated comorbid affective and/or psychotic disorders may fluctuate with environment and therefore a biopsychosocial therapeutic model is warranted.

Navigating Complexity: A Case Report on Interplay of Intellectual Disability (ID), Schizophrenia, Clozapine Treatment and Chemotherapy

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Aims. The use of Clozapine treatment requires rigorous and mandatory monitoring due to the side effects profile of Clozapine.

Certain clinical situations may pose a dilemma for clinicians such as concomitant use of clozapine during myelosuppressive chemotherapy. There is limited evidence-based data regarding Clozapine and chemotherapy. We report on a case of a clozapine-stabilized, Schizophrenia patient with Mild ID who was diagnosed with High Grade B-cell Non-Hodgkin's Lymphoma (NHL) requiring chemotherapy. The challenges of this complex case are detailed in this paper.

Methods. A 56-year old man with a diagnosis of Mild ID, Schizophrenia and OCD. The patient has been taking Clozapine since 2001 daily dose of 600-400 mg for the past 20 years. Unfortunately, he was diagnosed with High Grade NHL in 2023. The decision was reached to continue Clozapine while undergoing chemotherapy sessions with frequent blood monitoring. Towards the end of his chemotherapy his bloods showed dangerously low (Clozapine red alert) requiring stopping Clozapine. The patient started showing signs of relapse in his mental state and subsequently commenced on Olanzapine. He continued to show signs of relapse and didn't recover to his previous baseline; the treatment plan is adding another antipsychotic or considering re challenging Clozapine.

Results. This report contributes to a very limited literature on the concurrent use of clozapine with chemotherapy and the use of Clozapine "*outside license*". The main treatment options facing clinician is stopping or continuing clozapine during chemotherapy. The dilemma of taking the path of withdrawing a medication on which a patient is stabilized may compromise psychiatric stability, yet there is a valid argument that such inconvenience would present more favourable outcome than facing the serious haematological risks of neutropenia. There is a need for robust and close liaison between psychiatrists, oncologist, and haematologist on the various clinical considerations.

Conclusion. In summary, both clozapine and chemotherapy are known to cause neutropenia and agranulocytosis. The clinical decision to continue clozapine during chemotherapy could be challenging. Clinicians should be aware that psychotic decompensation in such patients would inevitably increase morbidity and perhaps mortality due to nonadherence to all proposed treatment, including chemotherapy. In the absence of guidelines and given the nature of treatment-resistant symptoms, clinicians should work in a multidisciplinary approach and carefully weigh the risks and benefits of continuing clozapine during chemotherapy.

A Case Study of Cognitive Impairment Associated With Levetiracetam

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Aims. In patients with cognitive impairment, it is important to assess the possible impact of medications on cognition. Levetiracetam is an antiepileptic medication used in the management of epilepsy. Its effect on cognition is unclear.

Methods. We present a case study of a 57-year-old female who developed cognitive impairment associated with levetiracetam.

She was referred to Memory Services from her GP due to cognitive impairment. Her past medical history included an optic nerve glioma which was surgically removed followed by radiotherapy, and meningiomas which were managed with stereotactic radiosurgery. She had no previous psychiatric history.

Following a first seizure, she was started on levetiracetam 250 mg BD. Over the following months, she developed worsening symptoms of poor memory, fatigue and lethargy, sleeping excessively, headaches, and subsequently, low mood and occasional suicidal thoughts. Levetiracetam dose was halved. When seen in Memory Services 3 months later, it was reported that there had been a gradual but partial improvement in her symptoms since the dose reduction. Addenbrooke's Cognitive Examination (ACE-III) score was 67/100. Short form mood scale was 3/15, below the threshold for depression. Blood tests were normal. MRI Head showed meningiomas and diffuse white matter hyperintensities, both unchanged from previous imaging.

The patient then started lamotrigine and stopped levetiracetam. On follow up (2 months after initial memory assessment), ACE-III score improved to 80/100 and it was reported that her symptoms had completely resolved.

Results. In this case, there is evidence to support a causal link between levetiracetam and the patient's cognitive impairment – there was a temporal relationship, dose response relationship, and reversibility, which are all in the Bradford Hill criteria for causation. Other causes were considered and deemed less likely, including depression; the mood symptoms were not the predominant symptoms and developed after the other symptoms, and the patient scored below the threshold score for depression on short form mood scale.

Regarding the aetiology in this case, one hypothesis is that there may have been risk factors that made this patient more susceptible to cognitive side effects from the biological effects of levetiracetam, such as previous neurosurgery and radiosurgery. Another hypothesis is that the levetiracetam may have triggered an atypical depressive episode which manifested predominantly with memory symptoms and tiredness.

Conclusion. This case study highlights the importance of reviewing medications when assessing cognitive impairment, and of obtaining a clear timeline of symptoms. There is a need for further research looking at the effect of levetiracetam on cognition.

An Interdisciplinary Approach to the Management of Ketamine Induced Uropathy

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Aims. This report describes the treatment of a patient with ketamine induced uropathy. This condition can be significantly debilitating due to its severe effect on the urinary system. This report outlines an interdisciplinary approach to the care of the patient involving addictions services, urology and primary care.

Methods. The patient presented with a history of inhalation of ketamine intermittently for four years and daily for three years. His highest daily use was 14 grams per day.

He developed multiple urinary symptoms including dysuria, urgency, incontinence, haematuria and abdominal and urethral pain. He had significant weight loss and suicidal thoughts. After