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Abstract

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CBS001

Detecting the invisible: catatonia in autoimmune encephalitis and how to intervene early

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Abstract: Anti-LGI1 autoimmune encephalitis can initially present with affective, psychotic, or behavioral symptoms, leadingto misdiagnosis as a primary psychiatric condition. Catatonia associated with this entity is rare and oftenunderrecognized. We report a case of delayed diagnosis, where catatonia emerged in the context of a progressiveneuropsychiatric syndrome later confirmed as anti-LGI1 autoimmune encephalitis
A 49-year-old male presented with an 8-month evolution of neuropsychiatric symptoms, including panic attacks,hallucinations, disorganized behavior, and faciobrachial dystonic seizures. He received multiple psychiatrictreatments with no sustained improvement. In early 2025, neurology evaluation included a brain MRI showingcortical atrophy and temporal FLAIR hyperintensities; EEG showed diffuse delta slowing and temporal sharp waves.CSF analysis revealed elevated protein levels without pleocytosis. Serology confirmed anti-LGI1 antibodies. InMarch 2025, psychiatry assessed catatonia using the Bush-Francis Catatonia Rating Scale (BFCRS), scoring 10 onscreening and 18 on severity. Treatment with lorazepam and quetiapine, and later instalation of memantine led to significant clinical improvement.Rituximab was later administered with favorable neurological stabilization.
The patient exhibited catatonic features including fixed gaze, rigidity, echolalia, automatic obedience, stereotypies,and disorganized speech. Marked clinical improvement followed immunotherapy and benzodiazepines.
This case highlights the importance of considering autoimmune encephalitis in patients with atypical psychiatricsymptoms and poor response to psychotropics. Early neurological evaluation and systematic screening forcatatonia can significantly improve diagnostic accuracy and outcomes. Multidisciplinary collaboration is crucial foridentifying catatonia as a possible manifestation of underlying neuroinflammatory disorders.

Disclosure of Interest: None Declared

CBS002

Catatonia in affective disorders

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Abstract: Catatonia is most commonly associated with schizophrenia and mood disorders, particularly depressive episodes; however, its occurrence during manic episodes of bipolar affective disorder remains relatively rare and insufficiently described. This presentation reports the case of a female patient with a long history of bipolar affective disorder and multiple psychiatric hospitalizations, who developed prominent catatonic symptoms during a severe manic episode.
During hospitalization, the patient exhibited marked psychomotor agitation, severely disorganized behavior, and repetitive motor automatisms, fulfilling criteria for an excited form of catatonia. The clinical course, diagnostic challenges, and therapeutic interventions are discussed in detail. The diagnostic work-up included laboratory testing, neuroimaging, and neurological hospitalisations to exclude organic causes of catatonic symptoms. Treatment strategies comprised pharmacotherapy tailored to both manic and catatonic symptoms, with careful monitoring of clinical response and adverse effects.

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In addition, a brief review of the available literature on catatonia occurring in the course of mania is presented. Published reports suggest that such cases are uncommon and often underrecognized, contributing to diagnostic uncertainty and therapeutic difficulties. This case highlights the need for increased clinical awareness of catatonic manifestations in manic episodes and emphasizes the importance of early recognition to optimize treatment outcomes. Further research is warranted to establish clear diagnostic and therapeutic guidelines for this challenging clinical presentation.

Disclosure of Interest: None Declared

CBS003

Substance-induced catatonia: the case of synthetic cannabinoids

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Abstract: Catatonia is a potentially life-threatening psychomotor syndrome that can complicate substance intoxication and may be missed in acute psychiatric and addiction settings. Synthetic cannabinoids (e.g., Spice/K2) are associated with severe neuropsychiatric presentations including agitation, psychosis, autonomic instability, and diagnostic ambiguity. This talk presents a clinical case of a 17-year-old male admitted with first-episode psychosis and behavioural disorganisation after synthetic cannabinoid use (positive K2 toxicology). Despite initial management for acute psychosis, the patient developed catatonic features including elective mutism, rigidity and impaired self-care, accompanied by escalating agitation and hallucinations. The case highlights key diagnostic dilemmas (catatonia vs delirium, intoxication, NMS/serotonin toxicity), practical bedside assessment, and evidence-based management principles, including early consideration of benzodiazepines, medical monitoring, and avoidance of potentially harmful treatment delays. The presentation emphasises that recognition of substance-induced catatonia enables timely intervention and prevention of complications such as dehydration, rhabdomyolysis, aspiration and cardiovascular instability.

Disclosure of Interest: None Declared

CBS004

How to sleep as a woman with ADHD and autism spectrum disorder

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Introduction: Sleep problems are highly prevalent in adults with neurodevelopmental disorders, with evidence of sex- and age-specific patterns. In women with ADHD, hormonal fluctuations across the reproductive lifespan interact with executive dysfunction, increasing vulnerability to sleep and cognitive disturbances. Women with ADHD report more severe sleep problems than men, including higher rates of insomnia and hypersomnia. Sleep disruption is also highly prevalent in autism spectrum disorder (ASD), affecting up to 80% of women, who are particularly prone to insomnia, circadian rhythm disturbances, and heightened sleep reactivity. Recent findings suggest poorer subjective sleep quality and more fragmented sleep, in autistic women, although age-related data remain limited. Menopausal transition may further compound these difficulties.
Objectives: To illustrate how perimenopausal hormonal transitions exacerbate sleep difficulties in women with ADHD and ASD through a case-based approach. To integrate clinical and scientific evidence highlighting the need for age- and sex-sensitive strategies in the management of neurodevelopmental disorders across the female lifespan.
Methods: This clinical vignette describes a 46-year-old woman with combined-type ADHD and ASD, who was referred for evaluation of persistent sleep difficulties during the perimenopausal transition. Diagnostic assessment included the DIVA-5, ADOS-2, RAADS-R, developmental history, collateral informant report, structured questionnaires (HSDQ, mood and anxiety), actigraphy, and clinical evaluation of hormonal status.
Results: The patient had longstanding ADHD and autistic traits. Perimenopause was marked by worsening insomnia, delayed sleep phase, and nocturnal awakenings with hot flushes. Actigraphy confirmed circadian disruption and frequent awakenings. Cognitive testing showed decline in working memory and planning. Sleep disturbance amplified anxiety, irritability, and neurodevelopmental symptoms. Methylphenidate improved attention but worsened sleep; lisdexamfetamine was better tolerated. Melatonin improved sleep timing but not maintenance. Menopausal hormone therapy (transdermal oestradiol, oral micronised progesterone) improved vasomotor symptoms but not sleep fragmentation.
Conclusions: This case illustrates how perimenopause can exacerbate sleep problems in women with ADHD and ASD. Literature supports high rates of insomnia and circadian rhythm disruption in both groups, with female-specific vulnerabilities. Recognition of reproductive transitions as periods of heightened risk is essential. Age- and sex-sensitive strategies may improve cognition, emotional wellbeing, and quality of life in women with neurodevelopmental disorders.

Disclosure of Interest: None Declared

CBS005

Pharmacological treatment of perinatal depression in women with ADHD

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Abstract: Perinatal depression affects 20–28% of women globally, with prevalence rates significantly higher among those with comorbid Attention-Deficit/Hyperactivity Disorder (ADHD), with self-reported symptoms exceeding 50% in clinical samples. Hormonal fluctuations during pregnancy and the postpartum period can exacerbate both ADHD symptoms and depressive episodes, leading to increased symptom severity. ADHD is linked to higher depression burden and greater risk of antidepressant treatment resistance