with this medium attracting more funding than ever before. Additionally, as this disorder gains increased attention from the psychiatric world, there is a lack of any formal guidelines on the treatment of IGD, or for the superiority of any specific pharmaceutical treatment. The aim of this project focuses on reviewing the neurobiological mechanisms involved in IGD in order to garner a more robust understanding of the neural pathways involved.

Methods. Google Scholar and PubMed were explored using search terms including "Internet gaming disorder," "neurology," "imaging," "mechanisms," and "comorbid" in various permutations. Thirty-seven articles were included from 100 search results that addressed IGD neural and biological mechanisms, and their potential comorbidity with other mental disorders.

Results. The literature suggests that some of the neural findings in IGD are similar to those found in other addiction disorders, which include the following mechanisms: (i) Activation of brain regions associated with reward, as observed in cue exposure and craving studies. Neurotransmitter system studies further suggest the involvement of dopamine-mediated reward mechanisms. (ii) Decreased activity in areas responsible for impulse control and impaired decision-making. (iii) Reduced functional connectivity in brain networks related to cognitive control, executive function, motivation, and reward. Another study suggested that the severity of IGD and depression symptoms predict each other reciprocally. Neurologically, individuals with IGD exhibited enhanced rsFC between the left amygdala and the right dorsolateral prefrontal cortex, inferior frontal gyrus, and precentral gyrus compared to control participants. The baseline amygdala-frontoparietal connectivity negatively predicted the reduction in depression symptoms following a psychotherapy intervention. Other studies suggest that altered executive control mechanisms in attention deficit hyperactivity disorder (ADHD) would be a predisposition for developing IGD. Furthermore, according to the literature, it was indicated that engaging in Internet game playing was linked to reduced white matter density in brain areas responsible for decision-making, inhibiting behavior, and regulating emotions.

Conclusions. The literature findings regarding IGD's neural and biological pathways, as well as the association of these findings with other disorders such as depressive disorders and ADHD reflect behavioral patterns in individuals with IGD. These mechanisms can be utilized to maximize behavioral and pharmaceutical interventions.

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Diagnostic Challenges of Late-Onset Mania versus Dementia with Behavioral Disturbances: A Case Report

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Introduction. Late-onset mania presents as a diagnostic challenge given the interplay between psychiatry and neurology. Neurocognitive disorders, especially the behavioral subtype of Frontotemporal Dementia (FTD), are among one of the differential diagnoses in patients presenting later in life with manic-like symptoms. Here we discuss a case of new-onset manic symptoms in an elderly patient with no prior history of bipolar disorder and conduct a comprehensive literature review to assist with the diagnostic challenges of late-onset mania and dementia with behavioral disturbances.

Case. Mrs. X is a 67-year-old female with bipolar disorder diagnosed 2 months prior to admission who presented to the hospital after a mechanical fall and witnessed seizure-like activity thought to be due to benzodiazepine withdrawal. She was found to have new atrial fibrillation that was stabilized, and clonazepam was resumed. Psychiatry was consulted to assist with manic behaviors. On initial evaluation, Mrs. X presented as anxious, distractible, tangential with pressured speech, increased psychomotor activity, and paranoid towards her husband. She was treated for a urinary tract infection but otherwise workup was unremarkable. Head imaging demonstrated gray matter volume loss that appeared more prominent in the temporal and frontal lobes. Once transferred to the inpatient psychiatry unit, she presented with elevated mood, excessive jocularity, disinhibition, and poor insight. SLUMS of 18 noted poor attention, processing, and short-term recall. She was started on divalproex sodium titrated to 1500 mg daily (VPA level 108 mcg/mL) and olanzapine 25 mg daily. Observations throughout this time included minimal change in affect, elevated mood, memory deficits and social disinhibition. She did have less aggression, minimal paranoia towards her husband and circumstantial thought process although noted with confabulation.

Methods. We completed a comprehensive literature review utilizing PubMed and Google Scholar. Search terms included combinations of "late-onset", "bipolar disorder", "mania", "neurocognitive disorder", "dementia", "frontotemporal", "behavioral disturbances".

Discussion. Once Mrs. X's initial delirium resolved, she remained with manic-like symptoms. Collateral, brain imaging of reduced gray matter volume in the frontal and temporal lobes, and lack of response to high doses of a mood stabilizer and antipsychotic favor a probable neurocognitive disorder with behavioral disturbances. Literature reviewed helped to narrow down the differential diagnosis for Mrs. X and allowed for a more comprehensive treatment plan.

Conclusion. Diagnostic elucidation of late-onset mania relies on a comprehensive investigation into psychiatric and non-psychiatric etiologies, a detailed collateral history, and a neuropsychiatric lens. Neurocognitive disorders remain an important differential diagnosis.

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