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Diagnostic Anchoring in Psychiatry: ADHD Misclassified as Bipolar II Disorder in a Treatment-Resistant Patient: a case report

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Case Report

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Abstract

Background

Diagnostic anchoring, the tendency to maintain an initial diagnosis without adequate reassessment, is a well-recognized contributor to persistent misdiagnosis in psychiatry. In adults with unrecognized ADHD, overlapping features such as emotional dysregulation, impulsivity, and behavioral instability may lead to misclassification as bipolar II disorder. Once assigned, this diagnosis may persist across providers and treatment settings without sufficient re-evaluation, resulting in inappropriate pharmacologic treatment, delayed identification of ADHD, and ongoing functional impairment.

Case Presentation

A woman in her late 40s with a longstanding diagnosis of bipolar II II disorder, assigned in her mid-20s following a hypomanic episode during antidepressant treatment, presented after dozens of medication trials across multiple providers without sustained remission. Despite continued treatment within the bipolar II II diagnostic framework, including a therapeutic trial of lithium, no clinical improvement was observed. Subsequent systematic reassessment, including developmental history, the Adult ADHD Self-Report Scale (score 55), and DIVA 2.0 interview, supported a diagnosis of attention-deficit/hyperactivity disorder. Discontinuation of mood stabilizer and antipsychotic therapy and initiation of ADHD-targeted pharmacotherapy resulted in rapid and sustained improvement in attention, emotional regulation, and overall functioning. At two-month follow-up, PHQ-9 decreased from 24 to 6 and GAD-7 from 17 to 3.

Conclusions

This case illustrates how diagnostic anchoring can sustain misclassification of ADHD as bipolar II disorder across multiple providers and care settings over time. When treatment response is inconsistent with the established diagnostic framework, the diagnosis itself should be reconsidered rather than continuing pharmacologic adjustments within that framework. ADHD, particularly in women, may present with emotional dysregulation and behavioral features that overlap with mood disorders, increasing the risk of misdiagnosis and prolonged ineffective treatment.

Background

Diagnostic anchoring is a well-documented mechanism by which initial diagnostic impressions persist across providers and care settings despite accumulating contradictory evidence.[1,2] In psychiatry, where diagnosis relies on clinical interview rather than objective biomarkers, anchoring poses particular risk. Adult attention-deficit/hyperactivity disorder (ADHD), particularly in women whose presentations tend toward inattention, emotional dysregulation, and internalized symptoms rather than overt hyperactivity, are factors contributing to misclassification as bipolar II disorder.[5,6] Impulsivity and behavioral

dysregulation may be attributed to mood or personality pathology rather than a neurodevelopmental condition,[6] and antidepressant-related activation, including restlessness, agitation, and cognitive acceleration, can be misattributed to hypomania in patients with underlying ADHD.[7]

Case Presentation

The patient is a woman in her late 40s who presented for psychiatric evaluation with longstanding mood instability and dissatisfaction with her medication regimen. She lives with a family member and has no history of psychiatric hospitalization, self-harm, or suicide attempts, and no history of substance use. Medical history includes gastric bypass surgery for binge eating and weight management, hysterectomy, and sleep apnea that resolved following weight loss. The patient reported a belief that psychiatric illness was present in her family of origin; however, specific diagnoses were unknown and had not been discussed within the family. No formal family psychiatric history was available. All identifying details have been de-identified, and written informed consent was obtained prior to submission.

Her psychiatric history began in early adulthood with depressive symptoms, behavioral dysregulation, impulsivity, and academic failure. At age 19, an impulsive act led to her arrest and her first psychiatric contact, resulting in diagnosis of an unspecified mood disorder and trials of alprazolam and multiple selective serotonin reuptake inhibitors.

In her mid-20s, during up-titration of sertraline from 100 mg to 200 mg, she developed what was labeled in records as hypomania, characterized by subjective grandiosity without psychotic features. Symptoms resolved upon discontinuation of sertraline. The diagnosis of bipolar II disorder was assigned at that time and was not challenged across subsequent providers.

No spontaneous manic or hypomanic episodes were identified over the following two decades. The episodes the patient described as "hypomanic," including impulsive spending, repetitive phone number changes, irritability, and feeling out of control, were not consistent with a classical hypomanic presentation. Her psychiatric course continued to be characterized by chronic emotional dysregulation, cognitive overload, impulsivity, and functional instability.

She subsequently reported trials of more than fifteen psychotropic agents across multiple providers, with at least seven recalled by the patient, including antipsychotics, mood stabilizers, antidepressants, and anxiolytics in dozens of combinations, with corroboration from available prescription records. A pattern in her treatment response was noted: antipsychotics provided some behavioral stabilization but did not address cognitive or depressive symptoms, and no regimen led to sustained remission.

She presented on aripiprazole 5 mg daily and clonazepam 0.5 mg as needed, reporting severe depressive symptoms (PHQ-9 = 24), severe anxiety (GAD-7 = 17), anhedonia, social withdrawal, and obsessive-compulsive spectrum symptoms including compulsive phone number changes, appliance-checking, and repetitive phone purchasing and returning. A seasonal depressive pattern, with consistent worsening each autumn, was noted. Mental status examination revealed cooperative presentation with

anxious affect, linear thought, and intact cognition. Treatment was initiated within the established bipolar II diagnosis, with lamotrigine added to aripiprazole.

Over the following three months of treatment with her most recent clinician, multiple trials remained within the established bipolar II diagnosis but with limited efficacy or significant adverse effects. Medications with reported prior partial benefit, including aripiprazole and sertraline, were retrialed; lithium was initiated and titrated to a therapeutic level of 0.6 mEq/L. Neither produced sustained improvement. Other trials were limited by adverse effects, including akathisia on aripiprazole, severe restlessness on the olanzapine/fluoxetine combination, and intolerance to cariprazine and fluvoxamine. A fluoxetine-related episode of restlessness, racing thoughts, and irritability was documented as hypomania, leading to its discontinuation. The patient's most impairing symptoms, including cognitive dysfunction, attentional difficulties, emotional dysregulation, and compulsive behaviors, persisted throughout.

Following three months of treatment resistance, the established bipolar II framework was challenged and the underlying diagnosis reconsidered. The Mood Disorder Questionnaire (MDQ), administered at intake and at reassessment, yielded a score of 7 on both occasions, meeting the minimum clinical threshold.[9] However, the MDQ is a screening tool, not a diagnostic instrument; a positive screen requires clinical corroboration. In the context of no documented spontaneous mood episodes across more than two decades, the MDQ result alone was insufficient in supporting the bipolar II diagnosis.[9]

The differential diagnosis included Bipolar II II disorder, Major Depressive Disorder with mixed features, Borderline Personality Disorder, Generalized Anxiety Disorder, obsessive-compulsive disorder, and ADHD. The Bipolar II II diagnosis was reconsidered on several grounds. Although the MDQ score of 7 met the screening threshold, it is not diagnostic and requires clinical corroboration.[9] The index hypomanic episode occurred during antidepressant up-titration and did not meet DSM-5 criteria for primary bipolar II disorder,[8] and no spontaneous hypomanic or manic episodes were documented over two decades. In addition, a therapeutic trial of lithium produced no clinical benefit across symptom domains. Borderline Personality Disorder was considered but was not supported given the absence of identity disturbance and persistent interpersonal instability. The patient’s compulsive behaviors, initially attributed to obsessive-compulsive disorder, lacked ego-dystonic intrusive thoughts and were more consistent with executive dysfunction and cognitive overload.[10] Overall, the clinical picture was most consistent with ADHD, supported by a longstanding pattern of attentional impairment, emotional dysregulation, and repeated antidepressant-induced activation.[6,7]

Reassessment included a detailed developmental history that revealed longstanding ADHD-consistent symptoms that had not been previously investigated: frequent classroom removal, behavioral dysregulation, and difficulty remaining seated. The Adult ADHD Self-Report Scale (ASRS) yielded a score of 55, consistent with ADHD.[3] The Diagnostic Interview for ADHD in Adults (DIVA 2.0) confirmed clinically significant symptoms with functional impairment across both childhood and adulthood.[4] Features previously attributed to bipolar II disorder, obsessive-compulsive disorder, and behavioral

dysregulation, including academic failure, occupational instability, impulsive decision-making, and compulsive ritualistic behaviors, were reconceptualized as symptoms of chronic, untreated ADHD.[10] The bipolar II diagnosis was removed and revised diagnoses included ADHD (combined presentation), Major Depressive Disorder (recurrent, seasonal pattern), Generalized Anxiety Disorder, and obsessive-compulsive spectrum symptoms.

Treatment

Following diagnostic reassessment, mood stabilizer and antipsychotic therapies were discontinued. Methylphenidate extended-release (Concerta) was initiated at 18 mg daily alongside continuation of buspirone 10 mg twice daily, sertraline 50 mg daily, and clonazepam 0.5 mg as needed. Two weeks later, methylphenidate was titrated to 27 mg daily.

Outcome and Follow-up

The patient's response was substantial. Within the first week, she reported marked cognitive quieting with near-complete resolution of chronic rumination and intrusive thoughts. By two weeks, sleep normalized, occupational functioning improved, and impulsive and compulsive behaviors resolved. The patient also reported resolution of maladaptive daydreaming, decreased emotional reactivity, and significant improvement in executive functioning. Residual symptoms at final follow-up included mild evening dysphoria as stimulant effects waned, consistent with methylphenidate extended-release's pharmacodynamic profile rather than underlying mood instability, and baseline anxiety, which remained present but was not acutely impairing. At two-month follow-up, the patient continued to demonstrate sustained progress on methylphenidate extended-release 27 mg daily. Repeat standardized assessment yielded a PHQ-9 of 6 and GAD-7 of 3, representing clinically significant reductions from intake scores of 24 and 17, respectively. Occupational and social functioning remained improved. Follow-up is ongoing.

Discussion

This case illustrates how diagnostic anchoring can sustain misclassification across providers and care settings over decades. This patient's course involved multiple clinicians, more than fifteen psychotropic medications across dozens of trials, and no sustained remission, a pattern seen in diagnostic anchoring.[1,2]

Research on diagnostic error demonstrates that anchoring persists across levels of training and experience, and that awareness alone is insufficient to prevent it; active reassessment strategies are required.[1,2]

Women with undiagnosed ADHD may be particularly vulnerable to diagnostic anchoring once symptoms have been attributed to a mood disorder. ADHD presentations characterized by emotional dysregulation, internalized distress, and executive dysfunction may be less likely to prompt reassessment once a mood

disorder diagnosis is established. In this case, diagnostic reconsideration was prompted only by persistent treatment resistance and a willingness to question the existing bipolar II diagnosis rather than continuing pharmacologic adjustments within the same framework.[5,6]

Limitations of this report include its single-patient design and limited follow-up duration. In addition, the absence of formal neuropsychological testing may limit diagnostic confirmation. Strengths include the use of multiple validated diagnostic instruments—the ASRS, DIVA 2.0, MDQ, PHQ-9, and GAD-7—to support both the original and revised diagnoses, the availability of corroborating prescription records to document the longitudinal treatment course, and repeat standardized outcome measures at two months confirming sustained treatment response within the minimal symptom range.

Conclusions

Diagnostic anchoring can sustain psychiatric misclassification for decades.[1,2] In adults with treatment-resistant presentations, foundational diagnostic assumptions should be reassessed rather than inherited from prior providers. ADHD, particularly in women, remains underrecognized and may present with features that overlap with mood disorder, increasing vulnerability to misdiagnosis.[5,6]

Patient's Perspective

“I remember telling you that at some point you would not want to work with me, because it had happened before. I think doctors get frustrated when you stop a medication or want to keep switching, but it happened because they didn’t feel right. Since starting the Concerta I feel so calm. The crazy rumination thoughts are gone. I feel like crying because my mind is quiet and not torturing me anymore. I used to daydream a lot — it was one way to stop the crazy thoughts. I would create another life in my head. I noticed the daydreaming has stopped. Little things used to be so hard. I used to make a mountain out of a molehill. I have not been impulsive once. I can think clearer, like the fog went away. I really wish that the people in my life that thought I was a lost cause could see me now.”

Declarations

Ethics approval and consent to participate

Written informed consent for participation and publication was obtained from the patient. All identifying details have been de-identified in accordance with journal guidelines.

Consent for publication

Written informed consent for publication of this case and accompanying patient perspective was obtained from the patient.

Competing interests

The author declares no competing interests.

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Author Contribution

LG was responsible for the clinical care of the patient, conceptualization of the case report, data collection, formal assessment, writing of the original draft, and reviewing and editing the final manuscript. LG is the guarantor.

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The author used artificial intelligence to assist with manuscript preparation and editing. All clinical content, diagnostic reasoning, patient history, and intellectual contributions are entirely the author's own. The author reviewed, edited, and takes full responsibility for the accuracy and integrity of all content.

Data availability

All data generated or analyzed during this study are included in this published article. No additional datasets were generated or analyzed beyond what is reported in the manuscript.

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