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Geschwind syndrome in remitted bipolar affective disorder without MRI brain findings: A case report

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Summary

Geschwind Syndrome (GS) comprises a constellation of behavioural traits traditionally associated with temporal lobe epilepsy. These include hyper-religiosity, hypergraphia, altered sexuality, and circumstantial thought processes. Increasingly, GS features are also observed in other neuropsychiatric conditions. We present a 26-year-old female with bipolar affective disorder in remission who developed GS-like features such as hyper-religiosity, compulsive writing, and atypical romantic ideation, without evidence of seizures, abnormal EEG, or MRI findings. She showed limited response to aripiprazole and partial improvement with behavioural interventions. This case highlights the need for increased recognition of GS features in psychiatric populations, independent of structural or seizure-related pathology.

Keywords: *Geschwind Syndrome, bipolar disorder, hypergraphia, hyper-religiosity, neuropsychiatry*

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INTRODUCTION

Geschwind Syndrome (GS) refers to a group of interrelated behavioural traits identified initially in individuals with temporal lobe epilepsy. Described by Norman Geschwind, this syndrome typically includes hyper-religiosity, hypergraphia, altered sexual behaviour, and circumstantial thought processes or viscosity (Geschwind, 2009). While GS has historically been linked with epilepsy, recent literature indicates that similar symptom patterns may manifest in a variety of neuropsychiatric conditions, including schizophrenia and schizoaffective disorder (Gama Marques et al., 2015; Mudgal et al., 2021; O'Connell et al., 2013). The underlying mechanisms of GS remain debatable, particularly concerning whether its origins are neurological, psychiatric, or a combination of both (Çolak et al., 2021). Here, we describe an unusual case of GS presenting in a patient with Bipolar disorder (BD) in remission, without any associated neuroimaging or electrophysiological abnormalities.

CASE PRESENTATION

A 26-year-old unmarried female from a Christian nuclear family presented to our psychiatric outpatient services with behavioural changes persisting over the past ten months. She had been diagnosed with BD three years ago and had experienced three manic and two depressive episodes. The most recent manic episode occurred approximately one year before the current presentation. Since then, she had remained euthymic and functional in daily activities, as assessed clinically using the Young Mania Rating Scale (YMRS) (score <7), and corroborated by family reports. However, her family had noted significant personality changes. She spent long hours praying, fasted three to four days a week, and became preoccupied with religious texts. She also expressed strong desires to marry a religious man, frequently consulting spiritual advisors and rapidly shifting romantic interests when rejected. Over the last year, she lost more than 12 kilograms due to frequent fasting and self-imposed dietary restrictions.

During her inpatient evaluation, she displayed a compulsive tendency to write, documenting her religious visions, personal thoughts, and romantic experiences in diaries and on paper scraps. On occasions when writing materials were unavailable, she wrote on her own body. She often initiated lengthy discussions with the treatment team on topics related to spirituality, morality, and ideal partnerships. Her speech was circumstantial but not disorganised. Her affect remained stable, and no hallucinations or delusions were elicited during assessment. Physical and neurological examinations were unremarkable. Cognitive functions were assessed using the Mini-Mental State Examination (MMSE) and were found to be within normal limits. The Bender Visual-Motor Gestalt Test indicated possible organicity. MRI brain and EEG revealed no abnormalities.

Based on the clinical presentation (characterised by hyper-religiosity, hypergraphia, altered romantic ideation,

and cognitive viscosity), a diagnosis of Geschwind Syndrome was made. Notably, this diagnosis was rendered in the absence of epilepsy or structural brain lesions, challenging the classical understanding of GS as an epileptic behavioural syndrome. The patient had been on aripiprazole, which was discontinued one month before admission. It was reinitiated at 10 mg/day following inpatient evaluation, as it had previously been effective during manic episodes. Psychoeducation sessions faced challenges as she often reverted to familiar themes. However, she agreed to reduce her fasting. After 2 weeks of ward stay, patient had resumed eating regularly. She was discharged with Aripiprazole 10 mg, and behavioural management was continued on outpatient basis. Patient continued to be on regular follow up with minimal improvement in behavioural symptoms.

DISCUSSION

This case exemplifies the clinical features of GS in a psychiatric patient without epilepsy or structural abnormalities, contributing to the expanding conceptualisation of the syndrome. The absence of classical neurological correlates raises questions about the underlying mechanisms of GS. Although GS was initially defined in the context of temporal lobe epilepsy, similar features have been reported in patients with schizophrenia, schizoaffective disorder, and now BD (Gama Marques et al., 2015; Mudgal et al., 2021; O'Connell et al., 2013). This case also aligns with growing evidence that BD and epilepsy share overlapping neurobiological mechanisms such as kindling, episodic progression, and pharmacological responsiveness to antiepileptic drugs (Wiglusz et al., 2015).

Neurobiological models suggest that the limbic system, especially the amygdala and hippocampus, plays a central role in mood regulation and religious/spiritual cognition. Chronic stress and mood dysregulation in BD are known to cause functional and structural alterations in these regions, although not consistently detectable on standard MRI. The concept of "allostatic overload" in BD posits that repeated affective episodes may sensitise neural circuits, potentially resulting in behavioural syndromes like GS even in remission phases (Çakır et al., 2015; Kapczinski et al., 2008).

In this case, the normal neuroimaging and EEG findings suggest that GS-like symptoms represent subtle functional dysregulation rather than structural damage. The Bender Gestalt findings indicated some organicity, though insufficient to establish a clear aetiology. The patient's circumstantiality, religious fervour, hypergraphia, and persistent romantic preoccupation align closely with descriptions of GS in literature.

Treatment for GS remains largely empirical. While aripiprazole helped contain her previous manic symptoms, it had minimal effect on the GS features in this case. This indicates that GS traits may not respond well to standard mood stabilisers or antipsychotics. Behavioural strategies like structured psychoeducation, cognitive redirection, and habit reversal training may hold promise in managing such presentations. Importantly, identifying GS in psychiatric settings requires clinicians to remain open to cross-diagnostic features and not dismiss atypical behaviours as residual or personality-related alone.

CONCLUSION

This case contributes to a growing body of evidence supporting the decoupling of GS from epilepsy. It also reinforces the need to broaden diagnostic frameworks in psychiatry to account for neurobehavioral syndromes traditionally associated with neurology.

Conflict of Interest: None to declare

Ethical Considerations: Does this study include human subjects? YES

Authors confirmed the compliance with all relevant ethical regulations.

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