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Chronic Psychosis and Hypothalamic Hamartoma: A Case of Schizophrenia with Epileptic Comorbidity

A. Korchi[1*], I. Katir[1], N. Kissa[1], M. Sabir[1]

[1]Faculty of Medicine and Pharmacy of RABAT

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***Corresponding author:** A. Korchi
Faculty of Medicine and Pharmacy of RABAT

Abstract **Case Report**

This clinical case describes a 45-year-old man with chronic psychosis associated with drug-resistant epilepsy since adolescence. Psychiatric symptoms included persecutory and witchcraft-related delusional ideas, auditory hallucinations, aggressive behavior, and suicide attempts. Brain MRI revealed a hypothalamic hamartoma, a rare benign lesion that can cause seizures, endocrine disturbances, and psychiatric manifestations. Treatment with amisulpride and carbamazepine led to partial improvement, with a reduction in hallucinations but partial persistence of delusional thinking. Management required multidisciplinary coordination between psychiatrists and neurologists, with consideration of a specialized surgical approach. This case highlights the importance of brain imaging in atypical and treatment-resistant psychiatric presentations, especially when associated with difficult-to-control epilepsy. It also underscores the potential role of hypothalamo-limbic circuits in the development of severe psychiatric disorders and the value of a comprehensive approach addressing both neurological and psychiatric aspects.

Keywords: Psychosis, drug-resistant epilepsy, hypothalamic hamartoma, multidisciplinary approach, neuroimaging.

INTRODUCTION

Hypothalamic hamartomas (HH) are rare, benign, non-tumoral malformations located in the hypothalamus, generally detected during childhood. They are classically associated with gelastic seizures (inappropriate laughter), precocious puberty, or endocrine disorders. However, the literature also reports significant neuropsychiatric manifestations, such as behavioral disorders, aggression, mood disorders, anxiety, and in some cases, psychotic symptoms.

Psychosis, particularly schizophrenia, is rarely described as directly linked to HH. Nonetheless, recent studies suggest that neuroanatomical and neurofunctional alterations induced by these lesions—particularly within the limbic and hypothalamo-limbic circuits—may contribute to the emergence of complex psychiatric presentations. We present here the case of a patient with chronic schizophrenia and drug-resistant epilepsy since adolescence, in whom brain MRI revealed a hypothalamic hamartoma during a psychotic relapse with a suicide attempt.

CASE REPORT

We report the case of a 45-year-old man admitted to Ar-Razi Psychiatric Hospital for persistent behavioral disturbances over the past 20 years, characterized by self- and hetero-aggressiveness, agitation, and delusional ideas of persecution and witchcraft. Although tolerated by his family for many years, a suicide attempt by throat cutting in a hallucinatory context led to hospitalization.

The current episode began five months earlier after the complete discontinuation of his treatment. He became increasingly agitated, suspecting his mother of trying to poison him and threatening to kill her. One month before admission, he attempted to throw himself in front of a car to escape what he described as “demonic possession.”

His medical history includes psychiatric follow-up for six years, hospitalization in 2021, and a prior suicide attempt in a delusional context. He has also been followed for epilepsy since the age of 15, characterized by complex partial seizures with secondary generalization. Despite dual therapy with carbamazepine 800 mg/day and clobazam 10 mg/day, seizures persisted at a rate of 3–4 per month, with postictal amnesia.

On admission, psychiatric examination revealed a calm patient of average build, with expressive facial mimicry and easy contact. Speech was coherent, and cognitive functions seemed preserved. However, he presented with vague, poorly systematized delusional ideas of persecution and witchcraft, with an interpretative mechanism and auditory hallucinations. His mood was neutral, affect blunted, judgment impaired, and insight absent. Sleep was disturbed, but appetite preserved. Physical examination showed a sutured wound on the neck.

Biological tests and ECG were normal. Due to partial resistance to antipsychotic treatment and persistence of neurocognitive disturbances, brain MRI was performed, revealing a lesion with intermediate T2 signal / hypo T1 signal centered on the right tuber cinereum, measuring 13 x 9 x 15 mm, consistent with a hypothalamic hamartoma.

The patient was started on amisulpride 400 mg/day along with carbamazepine. Progressive improvement of psychotic symptoms was observed during hospitalization, with psychomotor stability and regression of auditory hallucinations, although delusional ideas partially persisted.

He stayed for about two months and was discharged stabilized on amisulpride 1 g/day, with slight improvement in delusion but persistence of hallucinations. Coordination with the neurology team led to a reassessment of the antiepileptic treatment and discussion of specialized HH management.

Discussion

This case illustrates the complexity of interactions between drug-resistant epilepsy, chronic psychiatric symptoms, and hypothalamic hamartoma. Although rare and benign, HH can be associated with a wide range of clinical manifestations, from gelastic seizures to major psychiatric disorders. In our patient, early-onset, treatment-resistant epilepsy preceded the emergence of chronic psychotic symptoms, including delusions, hallucinations, violent behavior, and suicidal acts.

MRI revealed an HH, suggesting a possible common organic substrate for both neurological and psychiatric manifestations. Recent studies report that up to 43% of HH patients present with aggressive behavioral disturbances, often absent in other epilepsy forms. Involvement of hypothalamic structures regulating emotions (mammillary bodies, ventral hypothalamus) could explain these disturbances.

Risk factors such as male gender, early onset of seizures, diverse seizure types, and the presence of a delusional syndrome are also implicated in psychiatric comorbidity. The lack of clear guidelines for managing psychiatric disorders associated with HH complicates treatment. In our patient, amisulpride led to partial improvement. Specialized management, possibly surgical, is under consideration.

Some specialized teams report significant improvements after HH resection, especially in behavioral disorders and refractory epilepsy. Such interventions must be performed in expert centers with multidisciplinary evaluation.

This case underlines the importance of brain imaging in patients with refractory psychiatric disorders and atypical epilepsy, advocating for a coordinated approach among psychiatrists, neurologists, and neurosurgeons.

Conclusion

This clinical case highlights the complex interplay between refractory epilepsy, chronic psychiatric disorders, and hypothalamic hamartoma. The identification of this structural lesion in an adult patient with persistent psychosis and poorly controlled epilepsy emphasizes the need for brain imaging in atypical cases.

The impact of HH on emotional and behavioral circuits may contribute to the emergence of severe psychiatric disorders. A multidisciplinary approach involving psychiatrists, neurologists, and neurosurgeons is essential to improve the quality of life of these patients and reduce risks related to neuropsychiatric comorbidities.

References

1. Killeen Z, Bunch R, Kerrigan JF. Psychiatric comorbidity with hypothalamic hamartoma: Systematic review for predictive clinical features. *Epilepsy Behav*. 2017 Aug; 73:126–130. doi:10.1016/j.yebeh.2017.05.019.(PubMed)
2. Nguyen DK, *et al.,* Adult-onset hypothalamic hamartoma: origin of epilepsy? *Epilepsy & Behavior Reports*. 2023; 20:100120. doi:10.1186/s42494-023-00120-9.(BioMed Central)
3. Petrescu AM, *et al.,* Épilepsie et hamartome hypothalamique: Pièges et difficultés diagnostiques. *Les Cahiers d'Épilepsies*. 2016;6(1):20–21.(LFCE: Ligue Française contre l'Epilepsie)
4. Smith JR, *et al.,* Hypothalamic Hamartoma related epilepsy: A systematic review of surgical outcomes. *Epilepsy Behav*. 2024; 139:109090. doi:10.1016/j.yebeh.2024.109090.(PubMed)
5. Savard G, *et al.,* Psychiatric aspects of patients with hypothalamic hamartoma and epilepsy. *Epileptic Disord*. 2003 Dec;5(4):229–234.(PubMed)
6. Corbet Burcher G, *et al.,* Neuropsychiatric profile of paediatric hypothalamic hamartoma: systematic review and case series. *Dev Med Child Neurol*. 2019

Dec;61(12):1377–1385. doi:10.1111/dmcn.14241.(PubMed)
7. StatPearls. Hypothalamic Hamartoma. https://www.ncbi.nlm.nih.gov/books/NBK560663/(CNIB)
8. Orphanet. Crises gélastiques avec hamartome hypothalamique. https://www.orpha.net/fr/disease/detail/86906(Orpha)
9. Kahane P, *et al.,* From hypothalamic hamartoma to cortex: what can be learnt from depth recordings and stimulation? *Epileptic Disord*. 2003 Dec;5(4):205–217.(PubMed)
10. Dorfmüller G, *et al.,* Déconnexion chirurgicale des hamartomes hypothalamiques. *Neurochirurgie*. 2008;54(1):1–10.(Academia
11. 11.Harvey, A. S., Freeman, J. L., Berkovic, S. F., & Rosenfeld, J. V. (2003). *Epilepsy and hypothalamic hamartoma: clinical and neurophysiologic features*. Epilepsia, 44(6), 725–731. https://doi.org/10.1046/j.1528-1157.2003.21802.x
12. 12. Parvizi, J., & Kastner, S. (2011). *Prominent hypometabolism in bilateral medial prefrontal cortex in patients with hypothalamic hamartoma*. Journal of Neurology, Neurosurgery & Psychiatry, 82(10), 1126–1130. https://doi.org/10.1136/jnnp.2010.222810
13. Nguyen, D. K., Beaulieu, C., Carmant, L. (2020). *Neuropsychiatric comorbidities of epilepsy*. In: *Epilepsy and Psychiatry*, Elsevier.
14. Téllez-Zenteno, J. F., Patten, S. B., Jetté, N., Williams, J., & Wiebe, S. (2007). *Psychiatric comorbidity in epilepsy: a population-based analysis*. Epilepsia, 48(12), 2336–2344. https://doi.org/10.1111/j.1528-1167.2007.01222.x