Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Psychiatry

Exploring the Complexities of an Intricate Connection: Psychosis in Focal Epilepsy: Case Report

S. Taky-Eddine[1*], Z. Ennaciri[1], A. Oumoussa[1], I. Adali[1], F. Manoudi[1]

[1]Hospital Ibn Nafis, Service Universitaire Psychiatrique, CHU Mohamed VI, BP 2360, Avenue Ibn Sina, Marrakech, Marocco

DOI: 10.36347/sjmcr.2024.v12i06.012 | **Received:** 21.04.2024 | **Accepted:** 26.05.2024 | **Published:** 04.06.2024

***Corresponding author:** S. Taky-Eddine
Hospital Ibn Nafis, Service Universitaire Psychiatrique, CHU Mohamed VI, BP 2360, Avenue Ibn Sina, Marrakech, Marocco

Abstract **Case Report**

***Background*:** Epilepsy is still a real mental health problem; although most epilepsies are curable, their psychopathological consequences are often significant and complex to manage. In this framework, the association of epilepsy with psychotic disorders has long been known. ***Case Report*:** To discuss the links between epilepsy and psychosis, we report the observation of a 42-year-old man, treated for complex focal epilepsy, admitted to a psychiatric department for attempting suicide by phlebotomy in a postictal psychosis under the commands of auditory hallucinations and a severe state of psychomotor agitation. ***Discussion*:** Psychotic symptoms in epilepsy can be part of intercritical, post-critical or alternative psychoses. In our patient's case, the psychotic symptoms were post-critical. Delusional themes are often mystical, fueled by auditory and unusual visual hallucinations. Negative disorders are rare. ***Conclusion*:** Epileptic psychoses have not been identified as nosographic entities in the psychiatric classification systems (DSM-V and ICD-10), which poses a problem in recognizing these disorders. Therefore, a collaboration between psychiatrists and neurologists is necessary to understand this complex comorbidity better, avoid diagnostic errors, and optimize management.

Keywords: Case report, Comorbidity, Diagnosis, Focal epilepsy, post ictal Psychosis.

INTRODUCTION

Epilepsy has long been associated with various cognitive and psychiatric manifestations. Among these, psychosis stands out as a complex and intriguing aspect, creating a web of challenges for patients, caregivers, and healthcare professionals alike.

It has been well established that patients with epilepsy have an increased rate of developing a psychotic disorder. In the general population, the risk of a psychotic disorder is 0.4%–1%, but this risk is increased to 7.8% among patients with epilepsy [1].

At the present time, there is no internationally-validated classification and no clear diagnostic criteria. For example, DSM-5 classification does not include a specific category for these disorders. This lack of consensus probably has consequences on diagnostic and therapeutic decisions [2].

In this article, we delve into the intricate relationship between epilepsy and psychosis, exploring the underlying mechanisms, clinical manifestations, and the implications for diagnosis and treatment. Literature pertaining to psychosis of epilepsy is also reviewed and discussed.

CASE PRESENTATION

This article explores the clinical case of Mr. A, a 42-year-old patient, without a family history of follow-up in a psychiatric setting, who has been battling epilepsy since the age of thirty. He also has a 20-year history of depression, for which he currently take a daily dose of 20 mg of paroxetine. Additionally, he has cannabis addiction with a successful withdrawal four years ago.

The progression of his epilepsy, from generalized tonic-clonic seizures to focal seizures with associated psychotic symptoms, poses a unique set of challenges for diagnosis and management.

The patient initially presented generalized tonic-clonic seizures and focal seizures, occurring at a frequency of 2 to 3 attacks per week. Electroencephalogram (EEG) findings revealed spikes in the bilateral temporal region, predominantly on the right side. However, brain MRI angiography showed no abnormalities. Carbamazepine was prescribed, resulting

in the successful cessation of generalized seizures for several years while focale seizures were poorly controlled.

Approximately three years ago, the patient's clinical picture evolved, with the emergence of focal seizures accompanied by gestural automatisms, hallucinations, headaches, and post-ictal amnesia. The complexity of the case deepened when the patient experienced a day before his admission to our facility an epileptic attack followed 24 hours later by a psychotic episode marked by delirium, with persecutory, megalomaniacal, and religious themes, and intense emotional participation with high anxiety.

The situation escalated when the patient, in the throes of psychosis, experienced auditory hallucinations commanding him to end his life. In a state of great psychomotor agitation, the patient attempted suicide through phlebotomy, underscoring the severity of the psychiatric symptoms.

Given the dual nature of the patient's condition – epilepsy and psychosis – a comprehensive treatment approach became imperative. The introduction of Quetiapine at a dose of 300 mg and Levomepromazine at 200 mg, Alprazolam 02mg in addition to Carbamazepine 1000 mg resulted in a rapid clinical improvement, with a return to normalcy within two days. This pharmacological intervention addressed both the epileptic and psychotic components of the patient's presentation. For the disorders described above, the patient was hospitalized in our department for 15 days and discharged after the disappearance of the psychotic disorders and epileptic seizures.

After his discharge, the patient remains in psychiatric clinical remission. He had partial epileptic seizures but without any associated psychotic component. He was referred to the neurology department for optimization of his epilepsy management.

DISCUSSION

Mr. A's case, marked by psychosis and poorly managed seizures, underscores the significance for neurologists and psychiatrists to be aware of the categories encompassed within psychosis of epilepsy (POE) [3].

The onset of psychosis in relation to an ictal event is the basis for how different types of POE are categorized. The two main categories of POE are interictal psychosis and postictal psychosis. Other phenomena described in the literature include ictal psychosis in which symptoms are contemporary with a critical disorganization of all or part of the cerebral cortex, “forced normalization,” in which psychosis worsens the further from an ictal event an individual gets , then antiepileptic drug (AED) induced psychosis (certain AED including: ethosuximide, topiramate, felbamate, levetiracetam, zonisamide, vigabatrin, tiagabine, and lacosamide) [4].

As shown in Mr. A’s history of psychosis the delirium began 24 hours after the seizure which defines the postictal psychosis.

Postictal psychosis is diagnosed when there is a 24- to 48-hour—but no more than 7-day—delay between a seizure and the onset of psychosis and when the psychosis lasts between 15 hours and 2 months and there is not a more obvious reason for the psychosis (AED induced, status epilepticus, etc.) [5]. Postictal psychosis occurs in 3.7% of persons with epilepsy and is a self-limiting phenomenon. It appears in patients with refractory focal epilepsy, evolving for at least ten years. It often consists of complex focal temporal seizures and secondarily generalized seizures with frequently intercritical bitemporal abnormalities on the EEG [6], like we had seen in our case.

Generally, patients tend to experience features suggestive of mania, such as grandiose, religious delusions. It tends to be preceded by a period of insomnia [3], as seen in Mr.A.

The risk factors for the occurrence of postictal psychosis are:• drug-resistant focal epilepsy that has been present for at least 5 to 10 years;• the very recent resurgence of crises, readily secondarily generalized;• a temporal or frontotemporal focus, often bilateral;• a history of neurological conditions such as encephalitis or head trauma [4];• a greater prevalence of a family history of mood disorders, a personal history of psychiatric hospitalizations or suicide attempts;• a history of post-ictal psychosis [7].

In Mr.A’s case we have a poorly controlled temporal epilepsy evolving since 10 years.

Interictal psychosis is characterized by psychosis independent of the timing of seizure activity, and it occurs in 2.2% of persons with epilepsy. Phenomenologically, interictal psychosis closely resembles primary psychotic disorders, but it is differentiated by the psychosis beginning after the onset of epilepsy. Interictal psychosis presents with disorganized thought, paranoia, command auditory hallucinations, and negative symptoms [5].

Numerous theories have attempted to elucidate the complex interplay between epilepsy and psychosis: Spreading of paroxysmal activity within the structures of the limbic system is considered as the background for peri-ictal psychoses [10]. Repetitive bioelectrical discharges in the limbic system overstimulate the dopaminergic system (through excessive dopamine secretion and/or increased sensitivity of dopamine receptors), which may lead to the development of psychotic disorders [1]. According to another hypothesis,

a psychotic episode is a manifestation of a self-limiting autoimmune encephalitis subsequent to a transitional increase in permeability of the blood-brain barrier, leading to systemic blood antigens exposure [9].

Numerous MRI studies have conducted whole brain analysis in psychoses of epilepsy, but the results were inconsistent. Some reported no significant cortical gray matter differences between patients with psychoses of epilepsy and those with non-psychotic epilepsy. However, there were some studies that had explored cortical gray matter differences between participants with temporal lobe epilepsy with psychoses and with temporal lobe epilepsy only. They reported significant bilateral volume reductions in the inferior, middle and superior temporal gyri and fusiform gyri, and unilaterally in the left parahippocampal gyrus and hippocampus [11]. In our case the brain MRI showed no abnormalities.

Regarding treatment of POE, we have concluded to these recommandations: In drug-induced psychosis, the offending agent should be avoided. In postictal and interictal psychosis, seizure control with antiepileptic drugs is reasonable; however, antipsychotics are also indicated because these agents decrease the duration of the psychotic episodes [8].

The recognition and treatment of psychosis in persons with epilepsy is recommended with the apparent dilemma between treating psychosis and opening the possibility of exacerbating seizures. Risperidone and quetiapine are relatively safe for patients with a history of seizures [6].

CONCLUSION

The presented case and the additional studies collectively underscore the need for a nuanced understanding of the relationship between epilepsy and psychosis.

Future research endeavors should focus on unraveling the intricate molecular and cellular pathways linking epilepsy and psychosis. Additionally, large-scale clinical trials investigating the efficacy of novel pharmacological interventions and personalized treatment approaches are imperative to optimize patient outcomes. Interdisciplinary collaboration between neurology and psychiatry remains paramount in providing holistic care for individuals grappling with the challenges of both epilepsy and psychosis.

REFERENCES

1. Jeżowska-Jurczyk, K., Kotas, R., Jurczyk, P., Nowakowska-Kotas, M., Budrewicz, S., & Pokryszko-Dragan, A. (2020). Mental disorders in patients with epilepsy. *Psychiatr Pol*, *54*(1), 51-68.
2. Tarrada, A., Hingray, C., Sachdev, P., Le Thien, M. A., Kanemoto, K., & de Toffol, B. (2019). Epileptic psychoses are underrecognized by French neurologists and psychiatrists. *Epilepsy & Behavior*, *100*, 106528.
3. Kanner, A. M., & Rivas-Grajales, A. M. (2016). Psychosis of epilepsy: a multifaceted neuropsychiatric disorder. *CNS spectrums*, *21*(3), 247-257.
4. Clancy, M. J., Clarke, M. C., Connor, D. J., Cannon, M., & Cotter, D. R. (2014). The prevalence of psychosis in epilepsy; a systematic review and meta-analysis. *BMC psychiatry*, *14*, 1-9.
5. Hilger, E., Zimprich, F., Pataraia, E., Aull-Watschinger, S., Jung, R., Baumgartner, C., & Bonelli, S. (2016). Psychoses in epilepsy: a comparison of postictal and interictal psychoses. *Epilepsy & Behavior*, *60*, 58-62.
6. Adelöw, C., Andersson, T., Ahlbom, A., & Tomson, T. (2012). Hospitalization for psychiatric disorders before and after onset of unprovoked seizures/epilepsy. *Neurology*, *78*(6), 396-401.
7. Eloge, J. (2019). Psychosis of Epilepsy: Psychosis in a Patient with a Negative Continuous Electroencephalogram. *American Journal of Psychiatry Residents' Journal*.
8. Sophie, D. (2020). Epilepsie de l'enfant, de l'adolescent et de l'adulte. Elsevier Masson, 136-138.
9. Toone, B. K. (2000). The psychoses of epilepsy. *Journal of Neurology, Neurosurgery & Psychiatry*, *69*(1), 1-3. https://jnnp.bmj.com/content/69/1/1.short (consulté le 5 avril 2022)
10. Benzahra, S. (2021). Schizophrénie et épilepsie: liens cliniques et psychopathologiques. http://ao.um5.ac.ma/xmlui/handle/123456789/19071 Consulté le: 12 avril 2022. [En ligne]. Disponible sur: [Google Scholar]
11. Hirakawa, N., Kuga, H., Hirano, Y., Sato, J., Oribe, N., Nakamura, I., ... & Onitsuka, T. (2020). Neuroanatomical substrate of chronic psychosis in epilepsy: an MRI study. *Brain Imaging and Behavior*, *14*, 1382-1387.