

CASE REPORT**A case of postictal psychosis**

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INTRODUCTION

Patients with a long-standing history of seizures are more susceptible to develop major psychiatric disorders including chronic interictal psychosis and episodic psychotic states (1,2). The different psychotic syndromes associated with epilepsy are normally defined based on their chronological relation to the seizures. Interictal psychosis occurs without an antecedent seizure, or an increase in seizure activity (1), and results in persistent psychotic states of varying intervals (3). Postictal psychosis, on the other hand, is an episodic psychotic state that classically follows exacerbations of seizures, particularly clusters of complex partial seizures with or without generalization (4). A lucid period of 12 hours to 6 days after termination of seizure activity precedes the onset of psychiatric symptoms, which often remit spontaneously within days or weeks (5). Patients frequently present with auditory, olfactory, gustatory and/or visceral hallucinations, paranoid ideation, and cognitive dysfunction.

CASE REPORT

Ms. T. is a 31-year-old female who presented in a confused, but alert state after having experienced 3 episodes of seizure activity in 6 days. Upon interviewing she reported that she did not feel like her usual self and feared that something was wrong with her brain. Following her second seizure, she began having visceral hallucinations in her abdomen and groin. Within 24 hours of her third seizure, she had begun to hear voices emerging from her abdomen. The synchronicity of these two symptoms produced a deep anxiety that she was becoming homosexual. The patient

also expressed a feeling of responsibility for the suffering of innocent people and as a result, believed that she was being watched. Upon declaration of suicidal intentions, she was started on low dose haloperidol. Her psychoses resolved following two weeks of low dose haloperidol and strict administration of carbamazepine and lamotrigine.

Given our patient's previous hospital admissions for episodes of acute psychosis following a period of poorly controlled seizures, she has an increased risk for developing chronic psychosis from her repeated episodes of postictal psychoses (6,7). While there exists no published trials on the prevention of chronic psychosis, or the symptomatic or prophylactic treatment of postictal psychosis, treatment recommendations based on expert opinion have appeared in the literature in the field of neurology, which were followed for this patient. She was discharged on 200 mg carbamazepine BID, 75 mg lamotrigine BID and 0.5 mg risperidone BID. Follow-up as an outpatient was required in order to monitor her anticonvulsant levels, and to titrate the risperidone up to 1 mg BID, which was discontinued once she had achieved a prolonged period free of seizure activity and psychosis.

DISCUSSION

Epilepsy on average affects up to 1.5% of the general population, of which 0.8% has been admitted to hospital for schizophrenia and 1.5% for schizophrenia-like psychosis (8). Compared to the general population where the risk of developing schizophrenia is 1%, patients with a history of seizures have an increased risk of 2.5% (6). This risk further increases with the number of hospital admissions for epilepsy (6). A latency period of 8 to 15 years was noted between the onset of epilepsy and the patient's first hospital admission for psychiatric issues (5,7). Numerous epidemiological risk factors have been identified, including female sex, early onset epilepsy, long-standing epilepsy, intractable epilepsy, secondary generalization of seizures, and left and/or

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Figure 1a. CT scan, axial section of the brain showing a right middle cerebral artery infarct limited to the right temporal lobe.



Figure 1b. CT scan, axial section of the brain showing a right middle cerebral artery infarct limited to the right temporal lobe.

bilateral foci (1).

The propensity to develop postictal psychosis may have some link to the proximity of the seizure focus to limbic structures (8). While the neuropathology of epileptic psychoses remains undetermined, it is believed to be related to abnormal electrical discharges frequently localized to the temporal-frontal lobe and cerebellum (4,7), but can also occur with widespread electrical activity (1). It has been proposed that these epileptiform disturbances result in aberrant regeneration and wiring of neurons, cortical dysgenesis, diffuse brain damage and developmental lesions affecting the connectivity of limbic structures (1).

The patient has a history of seizures in early childhood following a cerebral infarct, compounded by a 10-year history of poorly controlled adult epilepsy. An MRI scan performed 3 years earlier confirmed a pre-existing right middle cerebral artery infarction with sparing of the basal ganglia. During her current admission she underwent computed tomography (CT) and an electroencephalogram (EEG). The CT revealed significant atrophy of the right temporal lobe, but no new lesions or recent infarcts. The EEG confirmed that she was having complex partial seizures with temporal lobe focus and secondary generalization.

While postictal psychosis has been well described in adults, the lack of ICD-10 or DSM-IV classification and the lack of treatment recommendations make the use of prophylactic neuroleptics experimental and problematic. When choosing an antipsychotic medication, it is important to consider the potential additive effect that occurs with the antiepileptic medication. This can increase the number of side effects and toxic effects experienced by the patient, in addition to the pharmacokinetic interactions that can occur during the absorption, distribution, excretion, and biotransformation, resulting in a reduced epileptogenic threshold (ET) and an increase in seizure activity (3). The extent of these adverse effects will vary based on

the specific medication, the dosage and duration of use (3). Nevertheless, these numerous unfavorable effects often lead to a high level of non-compliance among patients.

Typical neuroleptics with a low potency such as phenothiazine have been observed to decrease the ET (3). Conversely, highly potent typical neuroleptics like haloperidol demonstrate antipsychotic action at low dose with little effect on the ET, making it one of the safest antipsychotics used to treat postictal psychosis (3). However, haloperidol has a higher risk of extrapyramidal symptoms (EPS), and therefore its use as a first-line treatment for our patient's postictal psychosis was reserved solely for resolving active psychoses. She was then switched to risperidone, an atypical neuroleptic with less EPS side effects, minimal effects on the serum concentration of prolactin, and a positive effect on the negative and dysphoric symptoms of epilepsy that may accompany the psychosis of epilepsy (3).

Both haloperidol and risperidone are weak inhibitors of the hepatic microsomal P450 cytochrome oxidase system (CYP), limiting their effect on the metabolism of concomitantly administered antiepileptic drugs (3). On the other hand, carbamazepine can reduce the plasma concentration of haloperidol and risperidone via induction of CYP3A4, thus compromising the efficacy of psychiatric treatment (3). Lamotrigine, however, is metabolized predominantly by glucuronic acid conjugation with no effect on the cytochrome oxidase system, making it a relatively safe adjunct therapy.

The evaluation and treatment of epileptic patients who present with psychiatric manifestations prove to be complex issues. Although postictal psychoses is relatively rare, this case highlights the importance of seizure control in patients with long-standing epilepsy and the need for prophylactic and treatment protocols for patients at risk of developing epileptic psychoses.

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