



A CASE REPORT ON FOCAL IMPAIRED SEIZURE ASSOCIATED WITH INTERMITTENT PSYCHOTIC DISORDER

Pharmacy

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ABSTRACT

Introduction: Focal impaired seizure is an Complex partial seizures (CPS), where focal (start in one region of the brain) and are accompanied with impairments in consciousness. The majority of them start in the temporal region and have automatisms, an aura, and reduced consciousness. The most typical incorrect diagnosis for CPS that develop in the temporal region is primary psychiatric illness. **Case report:** A 23-year-old male patient arrived with brief episodes of hallucinations, violent behaviour, and altered levels of awareness. According to ICD10 criteria, he was diagnosed with acute and transitory psychotic illness, and he was treated with injectable haloperidol 10mg BID, then oral olanzapine 10mg. Both the electroencephalogram and the brain's computed tomography were normal. He returned to the outpatient clinic after 15 days and complained of aggressive behaviour and sensory misinterpretations. It was advised to do a video electroencephalogram, however owing to financial limitations, this was not done. Despite the lack of EEG data, the diagnosis was changed and he was put on oral carbamazepine due to a strong clinical suspicion of complex partial seizures. Antiepileptic treatment went well for him, and the symptom remission has held. **Conclusion:** Persons who appear with psychosis require a thorough diagnostic analysis to rule out any other options.

KEYWORDS

Focal impaired seizures, Complex partial seizures, Psychiatric illness

INTRODUCTION:

Epileptic patients were once referred to as "lunatics," which led to the comorbidity of mental symptoms with epilepsy[1]. Complex partial seizures, also known as temporal lobe seizures (TLE) or psychomotor seizures, are focal seizures that begin in one hemisphere of the brain and are linked to reduced consciousness. Aura, reduced consciousness, and automatisms are characteristics of CPS [2]. CPS of temporal origin is the epilepsy that most frequently exhibits psychopathology overall [1,2]. Epilepsy affects between 34.7 and 54.3 people out of every 100,000 people. The prevalence of TLE, 10.4 cases per 100,000 people, makes it the most prevalent form of partial epilepsy [3]. It is possible to assume that between 20 and 30% of epileptic patients have psychological disorders overall [4]. CPS is frequently mistaken as a main mental condition as a result of emotional, behavioural, and cognitive symptoms. These symptoms typically co-occur with others that are unusual for the diagnosis of primary mental disorder, including macropsia, micropsia, gustatory and olfactory hallucinations, strong, fleeting delusions, and dejavu phenomena [5]. CPS begins with an aura, then transitions into ictal, postictal, and interictal phases. Epigastric churning, dejavu phenomena, persistent intrusive thoughts, hallucinations of foul scent, dread, and tachycardia are some of the symptoms of aura. Automatisms (semipurpose, seemingly automatic motions of the mouth and hands) are then seen in an ictal state. Examples include plucking at buttons, chewing, and swallowing, as well as repeated hand motions and lip smacking. Patients typically have a trance-like condition, a semireactive appearance, and no memory of automatisms. Nonetheless, in certain cases, automatisms may cause them to move, run, or behave violently without being provoked [5,6].

An altered state of consciousness with motoric activity known as a fugue or twilight state is followed by amnesia for the incident and one's own identity. Complex partial seizures non-convulsive status epilepticus (CPNSE) can cause this condition, which can remain for hours or even days [7]. Due to its clinical appearance, which includes visual and auditory hallucinations, delusions, illusions, concepts of reference, paranoia, and puzzled, confused, and agitated behavior that may continue for hours to months, CPNSE is also known as ictal psychosis [8]. Around 5% of all convulsive and non-convulsive episodes, and 35% of non-convulsive episodes, were complex partial status epilepticus (CPSE) events [9]. According to other research, CPSE accounts for 10% to 40% of all non-convulsive status epilepticus cases, which make for 20% of all status epilepticus cases [10]. Postictal condition might manifest as dysphasia or bewilderment. During a lucid interval of 1 to 6 days, postictal psychosis may follow the seizure and makes up 25% of all psychosis associated with epilepsy [11]. The distinctions between postictal psychosis and CPNSE are shown in Table 1.

Case Report:

A 23-year-old male from Vijayawada, India, appeared with a history of bizarre behaviour, hallucinations, and aggressiveness that had started a few hours earlier. The beginning was sudden and gradual in character. His wife claims that he abruptly got up, began picking at his clothes and looking in a drawer for something, and stopped answering her calls. Also, he was reportedly smelling like a dog. He grew hostile and assaulted her violently when she stepped in. He appeared to be lost in thought and not pay attention to his surroundings. There was no history of substance misuse, head trauma, falls, or fever. All of the patient's systems, including the nervous system, were normal when examined. As the patient didn't react to any queries and seemed confused and confused, it was impossible to develop rapport. He was talking to himself and seemed disturbed and delusional. He was accepted, and additional examination found that, despite his limited speech, he frequently claimed to hear God speaking to him and to be able to see Him, which gives him unlimited power. Family relatives were only seldom recognized by him. He frequently lost his sense of time, people, and location. There were, however, instances of unintentional aggression that seemed to have no motive. Electrolytes, a brain computed tomography (CT), and an electroencephalogram were all within normal ranges during regular tests (EEG). The International Classification of Mental and Behavioural Disorders (ICD 10) criteria were used to diagnose the patient with acute transient psychotic illness. Haloperidol 10 mg IM intramuscularly was administered twice daily for two days. Sleep, aggression management, and orientation all showed improvement. Unexpectedly, all of the psychotic symptoms disappeared within two days. Due to modest extrapyramidal side effects, he was later transferred to oral olanzapine 10 mg OD, and he was released after 3 days. After 15 days, he returned with concerns of strange behaviour. His wife claims that he entered a restaurant violently and damaged the furnishings. The patient had no recollection of acting violently. It was discovered during a history lesson that the current incident only lasted a short while. The patient's descriptions of the symptoms' nature include a sense of twisting in the belly and the perception that the raindrops had grown very huge because it was pouring that day. While thinking about his wife's possible infidelity at the same time, he forgot the later instance of hostility that his wife had described. Vital signs were normal during the test, and the mental status examination (MSE) appeared to be completely normal. Further questioning revealed that the woman had experienced several instances of transient abnormal behaviour during the previous two years. He was unable to use the higher level medical facility's recommended video EEG monitoring owing to budgetary limitations. On the basis of a clinical suspicion of CPS, he began taking oral carbamazepine 200 mg BID, which was eventually raised to 1000 mg daily. Olanzapine was tapered off concurrently. Over the past two years, the patient has been kept on 1000 mg of oral carbamazepine every day without experiencing a symptom recurrence.

DISCUSSION:

Aura in temporal lobe epilepsy might include abdominal churning, macropsia, olfactory hallucinations, and recurring intrusive thoughts. The twisting sensation in the belly described by the patient in this case is an autonomic aura. The phenomena of enormous bubbles might be classified as macropsia, which is a sensory misunderstanding; the notion of adultery is more likely to be a repetitive intrusive thought, and sniffing is an olfactory hallucination. As observed in this example, these symptoms are all part of the aura. Aura is caused by the activation of reciprocal connections between the mesial and neocortical temporal cortex; epigastric aura is more common in seizures with right temporal foci [13]. Picking at clothing, looking about, and acting aggressively are automatisms that originate in the mesial temporal lobe, and bitemporal spread signals a change in awareness [13]. Auras are typically followed by abnormally violent behaviour, which includes nondirected aggression that may be referred to as automatism. The symptoms of CPNSE with temporal origin might be very comparable to those of primary mental disease [14]. In the current example, the anomalous behaviour that lasted for two days and resembled a psychotic episode with changes in awareness, sparse speech, episodes of lethargy and hostility, delusions, and hallucinations can be regarded as a CPNSE of temporal origin. In the current case, the atypical CPNSE attacks displayed fluctuating and agitated behavior, with delusions and hallucinations mostly containing religious content. These symptoms could last for several hours, days, or even months, as stated in reference [14]. CPNSE is characterized by prolonged epileptic discharge on intracranial stereo-electroencephalography (SEEG) in the hippocampus and mesial temporal structures. Sometimes, there may be no changes observed in the scalp EEG despite the presence of CPNSE. Similarly, in the present case, the patient did not exhibit any changes in scalp EEG during the psychotic behavior. However, there was no video EEG or SEEG performed to confirm the possibility of CPNSE due to the lack of availability at the treating center. Therefore, the diagnosis was made based only on clinical suspicion. It should be noted that the psychotic behavior observed in this case cannot be considered postictal psychosis as no lucid interval was reported. As the lucid interval is not mentioned, the psychotic behaviour observed in the current instance cannot be classified as postictal psychosis. The episodic pattern of the anomalous behaviour, clinical suspicion, and improvement with antiepileptic medicine all support the presumptive diagnosis of CPS of temporal origin, even if we were unable to confirm it. It is well established that all antipsychotics lower the seizure threshold and raise the likelihood of having a seizure. This propensity appears to be dosage dependent and differs amongst antipsychotics. The least likely of the standard antipsychotics to cause a seizure is haloperidol. Nonetheless, clozapine is the most epileptogenic atypical antipsychotic, with seizures being documented in 0.3% to 5% of patients treated with it.

Similar to olanzapine, 0.24% of those without epilepsy experienced seizures when taking it [15]. According to Degner et al. [16], olanzapine doses over 20 mg are more likely to cause seizures, especially in those with epilepsy. In that case series, it was also noted that individuals receiving olanzapine saw a slight reduction or no change in the frequency of seizures, but those receiving haloperidol experienced an increase in seizures relative to baseline. Nonetheless, there is scant data that suggests antipsychotics can ameliorate the psychotic symptoms of epilepsy, including one RCT and a few descriptive investigations [17].

In this instance, it is possible to claim that the symptom improvement was brought about by spontaneous remission; in fact, using haloperidol may have caused the spontaneous remission to take longer to occur. If haloperidol hadn't been taken, the CPNSE in this case would have gone away in a matter of hours instead of lasting for two to three days. This demonstrates the value of video EEG in the department of psychiatry for accurate diagnosis. Despite the dose not surpassing 20 mg, the usage of olanzapine may have contributed to the lowering of the seizure threshold and the occurrence of a second seizure within 15 days.

CONCLUSION

In conclusion, this case emphasises the need of clinical history-taking, the necessity of video EEG monitoring in the psychiatric department, and the value of evaluating psychotic patients for conditions other than primary mental disorder. It also emphasises how crucial it is to take clinical suspicion and improvement from antiepileptic treatment into account when making the diagnosis of CPS, even when the EEG is normal.

Table 1: Shows contrasting features of postictal psychosis and CPNSE.

Postictal Psychosis	CPNSE
Lucid interval 12 hrs – 6 days [11]	No lucid interval [11]
Lasts for 12 hours – less than 2 months. [12]	Lasts for few hours to months. [12]
Episodes of psychosis often without confusion and delirium, following a cluster of seizures [12]	Major features are confusion, delirium and aggression [12]
No evidence of EEG changes [12]	Evidence of EEG changes [12]
Mental state characterised by delusions and hallucinations in clear consciousness [12, 13]	Hallucinations, mood symptoms and delusions with impaired consciousness [12, 13]

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