Behavioral presentations of focal onset seizures: A case series

ABSTRACT

Seizures can have varied presentations and may have different etiological factors. A multidisciplinary approach should be used to treat them. It becomes difficult to diagnose seizures if they have an atypical presentation, particularly so when they manifest as behavioral disturbances. This case series demonstrates four cases with different psychiatric and behavioral manifestations of seizures where the diagnosis had to be done in a multi-disciplinary approach. Two of the cases highlight the fact that in the light of normal investigations, paying attention to detailed history is of prime importance.

Keywords: Complex partial seizures, fugue, myoclonus, temporal lobe epilepsy

“People think that epilepsy is divine simply because they don’t have any idea what causes epilepsy. But I believe that someday we will understand what causes epilepsy, and at that moment, we will cease to believe that it’s divine. And so it is with everything in the universe”

—Hippocrates

A seizure is “a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain while epilepsy is defined by any one of the following: Two or more unprovoked seizures occurring more than 24 h apart or one unprovoked seizure with a probability of further seizures (at least 60%) after two unprovoked seizures, occurring over the next 10 years or diagnosis of an epilepsy syndrome.

Although the most common and marked feature of epilepsy is a seizure, psychiatric symptoms such as behavioral problems, poor social adjustment, cognitive dysfunctions

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: WKHLRPMedknow_reprints@wolterskluwer.com

How to cite this article: Maggu G, Dhamija S, Chaudhury S, Rohatgi S, Saldanha D, Jain S. Behavioral presentations of focal onset seizures: A case series. Ind Psychiatry J 2021;30:S204-9.
like memory impairment, and learning disabilities are also found. Problems related to mental health may continue to persist even if the patient achieves seizure freedom or reduction in seizure frequency. According to International League Against Epilepsy 2017 classification seizures can be classified on basis of the onset. The classification has been further elaborated based on awareness and motor signs. Although terms such as “simple and complex partial seizure” are no longer in use according to newer classification, we are using these terms as most of the literature is based on these terms.\textsuperscript{[2]}

On the basis of anatomical localization, temporal lobe is the most common site with a prevalence of 66\%. Temporal lobe seizures may take the form of simple and complex partial seizures (CPS), with both occurring in some 70% of patients. Simple partial seizures are brief and usually last for a few seconds to 2 min. During a simple partial seizure, the patient remains conscious while CPS are associated with some degree of additional impaired awareness. Both complex and simple partial seizures are associated with cognitive symptoms. They may present with gross disorders of thought and emotion and scalp electroencephalographic (EEG) during a partial seizure can be normal. Temporal lobe seizures produce the most diversified auras of all, so they are of particular interest to the psychiatrist as they often contain symptoms that imitate a psychiatric disorder which can lead to psychiatric referral.\textsuperscript{[3,4]} This case series attempts to highlight some of the unusual psychiatric manifestations of seizure disorders.

**CASE REPORT**

**Case 1**

A 20-year-old guard was initially hospitalized with a history that he had wandered away from his night sentry duty post and subsequently was found in an unconscious state in a village about 3 km away. He was unable to account for his whereabouts in the night. He was found in his uniform. He had a deep lacerated wound on his fingers and multiple abrasions on both his legs. On an initial examination by a physician, he was noted to be confused and had difficulty in expressing himself about the event, but after a few hours was found to be normal. His physical examination was within the normal limits apart from the injuries to his extremities. He was therefore referred for psychiatric evaluation with a provisional diagnosis of conversion reaction. A detailed history and narcoanalysis revealed that while on night duty the patient saw a tall black figure and he followed that figure. He did not remember anything thereafter until he found himself in the hospital with injuries to the extremities and headache. There was no past or family history of mental illness or fits. He denied any stress at work or at home. According to his administrative authorities, he was a good worker and had never been punished. However, he was awaiting disciplinary action for the absence from his guard duty without permission. Physical examination was within the normal limits. His mental status examination (MSE) showed a kempt cooperative individual who was in touch with reality. His speech was relevant and coherent. There were no features of anxiety, depression, and psychosis. His biological functions were normal. His behavior in the ward was normal. Hemogram, urinalysis, liver function tests, blood sugar, serological test for syphilis, fundoscopy, computed tomography (CT) scan head, and EEG were normal. However, given the history of transient visual hallucinations, followed by aimless wandering, lacerations and abrasions to the body, unconsciousness, followed by confusion, aphasia, and headache, a diagnosis of CPS was considered and confirmed by neurophysician. He responded well to tablet carbamazepine and on review after 6 months, he remained symptom-free.

**Case 2**

A 23-year-old female student of MBA was brought to psychiatry OPD by her father, who was a general practitioner, with the complaints of periods of abnormal behavior during which she appeared anxious and “lost” and subsequently had a muddled recollection of the events. Her first episode occurred 2 years back. For a few minutes, she was fearful and did not know what was happening to her, after which she complained of a severe headache. She took paracetamol tablet, went to sleep, and woke up symptom-free. About a year later, she had another episode lasting for a few hours. These two episodes occurred while she was at her college hostel and she had not told her parents about it. The last episode occurred a day earlier while she was at home on a leave. As per her father, she appeared quiet and morose in the morning. Thinking that she may be missing her friends, her father suggested that they should go shopping, which she used to like. She went and put on her mother’s clothes, unlike her normal self. Although she had been driving for many years, that particular day, she was unable to drive, so her father took over. In the market, she seemed disinterested and was not answering properly. On returning home, she remained quiet and aloof. In view of this behavior, father gave her a tablet of alprazolam (0.5 mg) at night. She woke up in the morning with no complaints, but she did not remember the events of the previous day. Her father, 56 years of age was a practicing general physician. Her mother was 48-year-old homemaker and a known case of case of bipolar disorder. She was the eldest of two siblings. Her younger brother was studying medicine. After completing her B.Sc., she opted for business studies and had appeared for her final MBA examinations few days back.
Her physical examination was within the normal limits. On MSE, she was anxious and worried about her episodes of abnormal behavior. An EEG was done which revealed a background activity consisting of 9 Hz alpha activity which was responding to eye opening bilaterally. There were generalized 4 Hz spike and wave discharges, which lasted for 2–3 s at two places. Phase reversal was seen at F3 [Figure 1]. In view of abnormal EEG record, which was suggestive of right frontal epileptic activity, she was started on tablet carbamazepine 200 mg which was titrated up to 600 mg and she showed a good response to the drug.

**Case 3**

A 31-year-old Security force personnel having about 13 years of service was initially hospitalized for psychiatric evaluation for the following incident. One day, under the influence of alcohol, he fired in the unit lines for which he required psychiatric counseling and help. His brother, a Naik was killed by another colleague about 3 years back and since then, he had bouts of aggression. Once, while he was on leave due to his anger issues while sleeping on the roof of his home, he woke up and instead of using the staircase had directly jumped off the roof and sustained injuries. The patient reported that, he did not remember the exact circumstances of the events. He denied prolonged and excessive consumption of alcohol. He said on that particular evening he had consumed 2 pegs of rum and had gone to sleep. According to his colleagues, he woke up in the middle of the night, and he fired one round from his rifle in the air for no apparent reason. At that moment, he was overpowered and disarmed by his colleagues. Since, he was not talking properly he was allowed to sleep. In the morning, when he woke up, he was talking normally but had complete amnesia for that episode. His physical examination was within normal limits. His MSE showed that he was kempt, cooperative, in touch with reality and rapport could be established with ease. His speech was relevant and coherent. His affect was anxious and mildly depressed. There were no features of psychosis. Relevant investigations including CT scan brain were normal. EEG showed left frontal and anterior temporal spikes and wave [Figure 2]. With a diagnosis of focal seizure nonmotor in onset (behavioral), he was started on tablet phenytoin 300 mg HS, to which he had a good response. On review after 6 months, he was symptom free and was abstinent from alcohol.

**Case 4**

A 31-year-old male patient was bought to psychiatry OPD by family members with acute onset, complaints of disturbed sleep, appetite, muttering to self, and gesturing in the air from the past 3 days. He had a history of alcohol consumption from the past 4 years in an on and off pattern. His recent pattern of drinking was about 90–180 ml 3–4 times a week and his last drink was about 7 days before the admission and on the day of admission, he had no withdrawal symptoms. He had no significant family history of any major illness. He had a well-adjusted pre-morbid personality and his early developmental period along with childhood history was also unremarkable. He was admitted and all his routines blood workup was done which revealed an elevated serum glutamic oxaloacetic transaminase (174 U/l) and serum glutamic pyruvate transaminase (132 U/l) with normal bilirubin. Apart from this, all his blood parameters were within the normal limits. His ultrasonography revealed Grade I fatty liver. His non contrast computed tomography (NCCT) head was normal. On MSE, the patient had a kempt appearance with normal psychomotor activity and was fearful. He reported 2nd person auditory hallucinations which were derogatory in content. In a further interview, the patient gave a vivid
explanation about seeing a flying statue of a deity that was wrapped in his mother’s clothes and subsequently, he caught up that flying statue and he burnt it. He further described that the family deity is punishing him for his wrong deed. Although his family members denied any such incident, the only thing they corroborated was that he burnt his mother’s clothes without any apparent reason. Because of psychotic symptoms and concurrent use of alcohol, a diagnosis of alcohol-induced psychosis with dependence syndrome was kept. At that time, he was started on olanzapine 5 mg which was up titrated to 20 mg, but he had no response to that medication even after 1 month.

In view of his nonresponse to olanzapine, his diagnosis and history were again reviewed. On further enquiry about the visual images, he explained that these images were intrusive in nature and he could get rid of this image by distracting himself by drawing the image of that deity. Also, he explained in detail that first he would get a thought about burning that deity, and then he would hear derogatory comments in form of a female voice stating “Why did you kill me.” He would never see these images while he was engaged in some work. During this interview, a jerky movement of one hand of the patient was observed. On further enquiry, he told that from past 3 years, he was getting this jerky movement in his hand, which he described as sudden and brief.

These jerky movements interfered with his work. He would get these jerks in his sleep, which would wake him up, but he never consulted any doctor. The description of these jerks was typical of myoclonus and an alternate diagnosis of focal seizure was considered. An EEG was done which came out to be normal, but on a clinical basis, he would get jerks of one hand of the patient was observed. During this interview, “non-epileptic” in origin.

While, an epileptic fugue, as described in our first case during an absence or complex partial non-convulsive status epilepticus or may occur in a post-ictal phase in patients with generalized seizures and commonly presents with a frank confusional state.

It has been described multiple numbers times dating back to 1889, when Charcot described a case with multiple fugue states, possibly of epileptic origin. Gastaut et al. in 1956 also described an interesting case report of a prolonged fugue state.

Similarly, Khwaja et al. reported a female patient of “Absence seizure” presenting with amnestic fugue.

Studies indicate that compared to the general population depression is 5 and 10 times more common in patients with controlled and uncontrolled seizures, respectively. Affective experiences are also a part of temporal lobe auras. The most commonly experienced emotion is anxiety, which is often described as fear and is sudden in nature as compared to pleasurable emotions (joy and ecstasy) which are found comparatively less. Emotive experiences during the ictus are very powerful, having a special quality that is not easy to explain and that is different from what the patient had experienced in the past, just as the patient described that she felt lost in our second case. Furthermore, emotions described by a patient with seizures are usually stereotyped, obscene, and often lack the profoundness of normal emotions. Temporal lobe epilepsy (TLE) in about 80% of cases presents with some form of aura manifesting with somatosensory autonomic or psychiatric symptoms or their combination; examples include some patients would often appear semireactive to their environment, picking up nearby objects like in our third case where patient fired his gun before being restrained by his colleagues.

“ Forced thinking (FT)” has been described in the literature as “parasitic” or as intrusive thoughts occurring due to seizure activity. It is a heterogeneous phenomenon that
was separated from the “psychic aura” by Penfield and Jasper. FT has been generally associated with frontal lobe epilepsy, however, it has been also been described as a psychic aura arising from the temporal lobe by some investigators in the literature. Gloor et al. reported a case where the patient experienced thinking or seeing characters from a comic book, similar to our 4th case, who reported seeing an image of a deity. FT originating in the frontal lobe may differ from that of temporal lobe origin, as frontal lobe FT is generally accompanied by an attempt to act on the thought (forced acts such as real behavior of the same content, vocalization, and gaze attraction), like our patient who described the incidence as he caught that deity floating in the air and burnt. FT from the temporal lobe involves the limbic system for expression. Therefore, the content of the FT described by a patient is much more intense and expressive, as well as accompanied by an emotional and affective reaction. Similarly, our patient was also emotionally aroused and distressed and he used to distract himself by drawing the image of that deity.

Our 4th case had some psychotic symptoms such as auditory hallucination (2nd Person). Psychosis is defined as the combination of the presence of hallucinations, delusions, and altered thought processes. The limbic structure is involved in psychosis as well as in TLE along with the frontal or parietal lobe. The prevalence of psychosis in the general population with epilepsy is about 2%-7%. The clinical picture may present a diagnostic dilemma like in our last case. On a neuro-psychiatric basis, Adachi et al. concluded that because of common clinical features and the linear distribution of time intervals, so-called “psychosis-epilepsy” and “epilepsy-psychosis” may represent the same condition. Psychotic episodes in epilepsy present with pleomorphic psychotic phenomena, conspicuous and diverse mood changes with a low incidence of schizophreniform symptoms. Psychosis can occur during the peri-ictal period, ictal or post-ictal phase. Postictal psychosis is the most common which accounts for 25% of epileptic seizures. The symptoms include confusion, catatonia, thought disorder, poorly systematized persecutory delusions, persecutory ideas, delusions of reference, grandiose and nihilistic delusions, along with visual, auditory, and somatic hallucinations. Treatment of seizures with anti-epileptics tends to improve psychiatric symptoms. As in our patient, treatment with anti-convulsant medication improved other symptoms as well.

Alcohol dependence was an associated co-morbidity in our 4th case, which can itself cause withdrawal seizure (usually within 2–3 days) as well as, can increase in pre-existing epilepsy. For this reason, the patient's treatment should be done holistically with an emphasis on the treatment of comorbid alcoholism along with seizure control.

Furthermore, a decision for starting our patient on Divalproex Sodium was based on two reasons, the first one being, its role in the treatment of myoclonus and the second Divalproex Sodium being an anti-craving agent. Confusion in diagnosis in the last case also arose from the fact that loss of consciousness is a rare phenomenon in a patient presenting with myoclonus. However, myoclonic seizures occurring in rapid succession may be associated with impaired awareness and responsiveness. Mood, anxiety, and mild-to-moderate personality disorders along with frontal lobe dysfunction have been described in Juvenile Myoclonic Epilepsy (JME), a type of generalized myoclonic seizure disorder. Further, as we have discussed above, frontal lobe dysfunction can be associated with FT. A comparative study of mesial temporal sclerosis (TLE-MTS) patients and JME patients revealed that mood (23.7%), anxiety (13.7%), and psychotic (11.6%) disorders were the most frequently found psychiatric disorders in TLE-MTS group, while mood and anxiety disorders (25% and 21%, respectively) were the most common among JME patients.

CONCLUSION

The diagnosis of seizure and epilepsy is a clinical one, with the EEG findings supporting the diagnosis if positive, but not excluding it if negative. Up to 20% of patients with the clinical diagnosis of epilepsy will have a normal EEG. Therefore, differential diagnosis of seizure should be kept in mind when patients present unusually.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Scheffer IE, Berkovic S, Capovilla G, Connolly MB, French J, Guilhoto L, et al. ILAE classification of the epilepsies: Position paper of the ILAE Commission for Classification and Terminology. Epilepsia 2017;58:512-21.
2. van Rijckevorsel K. Cognitive problems related to epilepsy syndromes, especially malignant epilepsies. Seizure 2006;15:227-34.
3. Blair RD. Temporal lobe epilepsy semiology. Epilepsy Res Treat 2012;2012:751510.
4. Téllez-Zenteno JF, Hernández-Ronquillo L. A review of the epidemiology of temporal lobe epilepsy. Epilepsy Res Treat 2012;2012:630853.
5. Janszky J, Schulz R, Ebner A. Simple partial seizures (isolated auras) in medial temporal lobe epilepsy. Seizure 2004;13:247-9.
6. Kumar A, Sharma S. Complex partial seizure. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2021.
7. Williamson PD, Engel J Jr. Complex partial seizures. In: Engel J Jr., Pedley TA, editors. Epilepsy: A Comprehensive Textbook. Philadelphia, Pa, USA: Lippincott-Raven; 1997. p. 557-66.

8. Lu E, Poynta N, Burant CJ, Sajatovic M. Systematic literature review of psychiatric comorbidities in adults with epilepsy. J Clin Neurol 2021;17:176-86.

9. Silberman EK, Sussman N, Skillings G, Callanan M. Aura phenomena and psychopathology: A pilot investigation. Epilepsia 1994;35:778-84.

10. Akhtar S, Brenner I. Differential diagnosis of fugue-like states. J Clin Psychiatry 1979;40:381-5.

11. Loewenstein RJ. Dissociation debates: Everything you know is wrong. Dialogues Clin Neurosci 2018;20:229-42.

12. Goetz CG. Charcot at the Salpêtrière: Ambulatory automatisms. Neurology 1987;37:1084-8.

13. Raval CM, Upadhyaya S, Panchal BN. Dissociative fugue: Recurrent episodes in a young adult. Ind Psychiatry J 2015;24:88-90.

14. Gastaut H, Roger J, Roger A. The significance of certain epileptic fugues; concerning a clinical and electrical observation of temporal status epilepticus. Rev Neurol (Paris) 1956;94:298-301.

15. Khwaja GA, Duggal A, Kulkarni A, Chaudhry N, Gupta M, Chowdhury D, et al. Recurrent prolonged fugue states as the sole manifestation of epileptic seizures. Ann Indian Acad Neurol 2013;16:561-4.

16. Salpekar J. Mood disorders in epilepsy. Focus (Am Psychiatr Publ) 2016;14:465-72.

17. Stefan H, Schulze-Bonhage A, Pauli E, Platsch G, Quiske A, Buchfelder M, et al. Ictal pleasant sensations: Cerebral localization and lateralization. Epilepsia 2004;45:35-40.

18. Beletsky V, Mirsattari SM. Epilepsy, mental health disorder, or both? Epilepsy Res Treat 2012;2012:163731.

19. Penfield W, Jasper H. Epilepsy and the Functional Anatomy of the Brain. London: Churchill Livingstone; 1954.

20. Gloor P, Olivier A, Quesney LF, Andermann F, Horowitz S. The role of the limbic system in experiential phenomena of temporal lobe epilepsy. Ann Neurol 1982;12:129-44.

21. Bonini F, McGonigal A, Trébuchon A, Gavaret M, Bartolomei F, Giussano B, et al. Frontal lobe seizures: From clinical semiotics to localization. Epilepsia 2014;55:264-77.

22. Cho YJ, Song SK, Jang SH, Chang JW, Lee BI, Heo K. Simple partial status of forced thinking originated in the mesial temporal region: Intracranial foramen ovale electrode recording and ictal PET. J Epilepsy Res 2011;1:77-80.

23. Oyeboye F. The neurology of psychosis. Med Princ Pract 2008;17:263-9.

24. Trimble MR, Schmitz B. The psychoses of epilepsy/ schizophrenia. In: Engel J Jr., Pedley TA, editors. Epilepsy: A Comprehensive Textbook. Philadelphia, Pa, USA: Lippincott-Raven; 1997. p. 2071-81.

25. Matsuura M, Adachi N, Oana Y, Okubo Y, Kato M, Nakano T, et al. A polydiagnostic and dimensional comparison of epileptic psychoses and schizophrenia spectrum disorders. Schizophr Res 2004;69:189-201.

26. Adachi N, Onuma T, Kato M, Ito M, Akanuma N, Hara T, et al. Analogy between psychosis antedating epilepsy and epilepsy antedating psychosis. Epilepsia 2011;52:1239-44.

27. Puryear LJ, Kunik ME, Molinari V, Workman RH Jr. Psychiatric manifestations of temporal lobe epilepsy in older adults. J Neuropsychiatry Clin Neurosci 1995;7:237-5.

28. Chaudhury S, Rohatgi S, Murthy PS, Soren S, Bakhla AK, Kiran C. A clinical study of post-ictal psychoses. Saudi J Health Sci 2015;4:99-103.

29. Beckmann A, Shaikh AA, Swash M, Scott DF. Seizure induction by alcohol in patients with epilepsy experience in two hospital clinics. J R Soc Med 1990;83:6-9.

30. Caviness JN. Treatment of myoclonus. Neurotherapeutics 2014;11:188-200.

31. Reoux JP, Saxon AJ, Malte CA, Baer JS, Sloan KL. Divalproex sodium in alcohol withdrawal: A randomized double-blind placebo-controlled clinical trial. Alcohol Clin Exp Res 2001;25:1324-9.

32. Mellers JD. Epilepsy. In: David D, Fleminger S, Kopelman M, Lovestone S, Mellers J, editors. Lishman’s Organic Psychiatry a Textbook of Neuropsychiatry. Chichester, West Sussex, UK: Wiley-Blackwell; 2009. p. 313-4.

33. Trinka E, Kienpointner G, Unterberger I, Luef G, Bauer G, Doering LB, et al. Psychiatric comorbidity in juvenile myoclonic epilepsy. Epilepsia 2006;47:2086-91.

34. Pascalicchio TF, de Araujo Filho GM, da Silva Noffs MH, Lin K, Caboclo LO, Vital-Dourado M, et al. Neuropsychological profile of patients with juvenile myoclonic epilepsy: A controlled study of 50 patients. Epilepsy Behav 2007;10:263-7.

35. de Araujo Filho GM, Yacubian EM. Juvenile myoclonic epilepsy: Psychiatric comorbidity and impact on outcome. Epilepsy Behav 2013;28 Suppl 1:574-80.

36. Noachtar S, Remi J. The role of EEG in epilepsy: A ×critical review. Epilepsy Behav 2009;15:22-33.