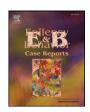
FISEVIER

Contents lists available at ScienceDirect

# **Epilepsy & Behavior Case Reports**

journal homepage: www.elsevier.com/locate/ebcr



## Case Report

# Homicide during postictal psychosis

Stephan Eisenschenk <sup>a,\*</sup>, Harry Krop <sup>b</sup>, Orrin Devinsky <sup>c</sup>

- <sup>a</sup> Department of Neurology, University of Florida, 1149 Newell Drive, Rm L3-100, Gainesville, FL 32611, USA
- <sup>b</sup> Community Behavioral Services, 1212 N.W. 12th Ave., Ste. B, Gainesville, FL 32601, USA
- <sup>c</sup> Department of Neurology, NYU School of Medicine, 223 East 34th Street, New York, NY 10016, USA



#### ARTICLE INFO

Article history:
Received 2 April 2014
Received in revised form 4 April 2014
Accepted 6 April 2014
Available online 4 May 2014

Keywords: Postictal psychosis Epilepsy Homicide Medical-legal

#### ABSTRACT

Postictal psychosis is characterized by a fluctuating combination of thought disorder, auditory and visual hallucinations, delusions, paranoia, affective change, and aggression including violent behavior. We present a case of homicide following a cluster of seizures. The patient's history and postictal behavior were his consistent with postictal psychosis. Contributing factors resulting in homicide may have included increased seizure frequency associated with a change in his AED regimen seizure frequency. The AED change to levetiracetam may also have increased impulsiveness with diminished mood regulation following discontinuation of carbamazepine. There is evidence that he had a cluster of seizures immediately prior to the murder which may have resulted in the postictal disinhibition of frontal lobe inhibitory systems. This homicide and other violent behaviors associated with postictal psychosis may be avoided with earlier recognition and treatment.

© 2014 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/3.0/).

## 1. Introduction

Postictal agitation and psychosis were initially described by Esquirol in 1838 and expanded upon by Jackson in 1873 [1,2]. Interest was rekindled in epilepsy-related agitation and psychosis in the 1950s–1960s [3–5] with a focus on postictal psychosis during the past few decades [6–10]. Logsdail and Toone's diagnostic criteria for postictal psychosis [7] are widely accepted with some modifications and include the following: 1) episode of confusion or psychosis immediately after a seizure; often there is a lucid period after the seizure followed by development of psychosis within hours up to one week; 2) psychosis lasting 24 h to 3 months; 3) some combination of disorientation, delirium, hallucinations, and delusions; although these may be present with 'clear consciousness'; and 4) no evidence of interictal psychosis, antiepileptic drug (AED) toxicity, nonconvulsive status epilepticus, head trauma, or alcohol/drug intoxication or withdrawal.

Postictal psychosis is characterized by a fluctuating combination of thought disorder, auditory and visual hallucinations (either one may predominate), delusions (grandiose, religious, persecutory), paranoia, affective change (mania or depression), and aggression [8,9,11]. Religious and violent behavior can be prominent [12,13]. Directed violent behavior is a rare feature of postictal confusion, but well-directed violent attacks occurred during 22.8% of episodes of postictal psychosis [14].

We present a case in which the patient killed his mother following a cluster of seizures. His history and postictal behavior were consistent with postictal psychosis possibly exacerbated by postictal disinhibition.

## 2. Case report

The patient is a 29-year-old right-handed Caucasian man with epilepsy since age 12. Initially, seizures were characterized by recurrent face slapping for 5 to 10 s. Subsequently, his events evolved to include rapid kicking, oral automatisms, and brief vocalization lasting 15–30 s. There was no definite loss of awareness during the events. During child-hood, seizure frequency was several times per week, typically occurring after sleep onset or immediately prior to awakening. Seizures were not preceded by an aura, but he occasionally reported 'déjà vu' when he awoke from sleep. He had no risk factors for epilepsy. There is no significant family history of psychiatric disease.

Seizures remained refractory to multiple AEDs, including phenytoin, carbamazepine, valproate, and levetiracetam, until adulthood. His seizures began to increase in frequency and severity; seizure clusters impaired cognition and reduced spontaneous behavior for hours to days. During the two years before the homicide, he began having episodes of paranoia and agitation following seizure clusters. His wife became concerned for her safety. Cocaine would exacerbate his seizures and postictal paranoia. He frequently used marijuana that he believed would reduce seizure activity.

Nine months prior to the homicide, carbamazepine was discontinued and levetiracetam was introduced while valproate was continued.

<sup>\*</sup> Corresponding author.

Seizure clusters increased in frequency. Paranoia with delusions after seizure clusters was documented in his neurologist's records. Five months before the homicide, he became paranoid and very violent for several days after a seizure cluster. His family witnessed his delusional beliefs that aliens possessed women and that he was instructed to kill women. His family convinced him to go to the emergency room where he received several doses of lorazepam but had recurrent symptoms of paranoia and violence; subsequently, haloperidol 2 mg intravenously was administered with resolution of the paranoia and violent behavior.

In the days leading up to the homicide, based on interviews with the patient and family members, he was compliant with his AEDs. He did not utilize cocaine but drank two beers the night before the incident. He had several partial seizures shortly after sleep onset and again in the early morning hours prior to awakening. When he awoke, he experienced paranoid mentation and smoked marijuana which he believed would alter these distressing perceptions and went back to sleep. He had another seizure prior to awakening in the late morning and felt as if he was going to have more seizures. He perceived that his sister told him to leave the House because he was being told by 'Jesus' that he should kill her. His sister recalls that he appeared 'distant', similar to prior events when he developed postictal delusions.

He walked to his grandmother's home in the neighborhood to play cards. He abruptly left the house to go to his parents' home. On the way, he states that he 'blacked out' for an unknown duration of time. Upon arrival to his parents' house, he recalled 'Jesus' telling him to kill his mother. He remembers getting a knife and stabbing her in the neck and torso. He then remembers getting a gun from a nearby room and shooting his mother in the head and neck. Afterwards, he called the police but could not recall the specific conversation. Following his arrest, he recalls a clouded mental state with little memory for the next few days. Toxicology reports were positive only for cannabis.

His only prior criminal offense was possession of marijuana. His first psychiatric evaluation was at age 15 after he threatened to kill himself, apparently in relation to a seizure. His academic performance worsened, and he was in special education at age 15 and dropped out the following year. He worked at short-term jobs and married at age 22. He denied any prior violence associated with seizures, but he once banged on a window and scared his wife. His wife divorced him while he was incarcerated. A psychological assessment after the homicide, requested by his public defender, revealed a disheveled man who laughed inappropriately and who was disoriented, inattentive, and agitated. Although he described the postictal events as dream-like, he claimed that he stabbed his mother "before she could influence anyone else." He also described being controlled by his father, noting that his father had him chop his mother up with a sword. He did not remember his 911 call and admission of stabbing and shooting his mother. Neuropsychological testing revealed average intellectual functioning and intact executive functions but verbal memory deficits.

Shortly after incarceration, he was hospitalized for postictal psychosis and required four-point restraints, claiming that his mother took the form of a devil and tried to get him to kill his sister. Subsequently, his AEDs were converted back to valproic acid and carbamazepine. His seizure control improved, and his thought processes cleared with no evidence of a thought disorder or a psychotic process. His seizures remained well controlled while he was incarcerated.

Inpatient video-EEG monitoring was performed 3.5 years after the homicide; tapering of his AEDs led to four seizures. The first three were single nocturnal complex partial seizures on separate nights typical of events during seizure clusters. An aura of déjà vu and a euphoric feeling occurred in conjunction with two seizures. Ictal nonspecific leg movements and bicycling movements occurred as well as oral and manual automatisms with impaired consciousness. He had significant postictal confusion and agitation with biting gestures toward staff following each of these events, requiring wrist and leg restraints with a Posey vest for safety. He did not manifest paranoia or delusions even after discharge. The nocturnal events arose from stage II sleep with a

20- to 30-second delay after initial clinical onset prior to initial localization of ictal EEG over the left frontal region followed by secondary bilateral synchrony. On the morning of his discharge while awake, the patient had a fourth event: a simple partial seizure of paranoia and déjà vu with bashfulness. Electroencephalography findings revealed left frontal sustained discharges (FP1 and F3).

## 3. Legal proceedings

Throughout his incarceration, he was a model inmate, well liked by fellow inmates and correctional officers. He understood that his attorney was considering an insanity defense and that if he were unsuccessful, he would receive a life sentence. The State believed that the defendant's actions were goal-directed because of anger with his mother. Two medical and psychological opinions stated that he met the criteria for a DSM-IV diagnosis of psychosis due to organic mental disorder that rendered him insane at the time of the offense. He was concerned with the potential of the death penalty if convicted and, thus, accepted a plea offer of 25 years.

## 4. Discussion

The evidence from witnesses and medical records overwhelmingly supports the idea that this homicide was due to a postictal psychosis, not a premeditated or environmentally provoked (e.g., argument) incident with his mother. He had an ictal left frontal seizure focus as documented by video-EEG; a clear history of postictal paranoia, agitation, and delusions; and recent changes in his AED regimen which led to an increase in the frequency and severity of seizure clusters. Although he had seizure clusters followed by postictal psychosis before the murder, he did not have consistent medical follow-up and never had prophylactic or rescue medications available to prevent or rapidly treat postictal psychosis. This family homicide may have been avoided with earlier recognition and treatment.

Although all patients with chronic treatment-resistant epilepsy are at risk for postictal psychosis, this man did not have additional risk factors such as bilateral or widespread CNS injury (e.g., encephalitis, head injury, intellectual disability, generalized EEG slowing) nor a family history of mood disorders, psychosis, or epilepsy [15,16].

There may have been a contributing role of AEDs in his postictal aggression. Changes in AEDs can be temporally related to the onset of psychosis through mechanisms other than changes in seizure activity [17]. Levetiracetam can cause behavioral side effects including agitation, psychosis, and aggressive behavior [18,19], but these are interictal adverse effects. This man's irritability and aggression were limited to the postictal state. However, patients on levetiracetam may have increased impulsiveness which may have been a contributing factor [20]. In addition, carbamazepine has mood-stabilizing effects, and its discontinuation may have increased the propensity for postictal mood dysregulation.

The patient exhibited aggressive biting behavior during postictal confusion. Directed violent behavior occurs in nearly a quarter of postictal psychoses, but this is a rare (<1%) feature of postictal confusion although studies have not consistently included violent actions such as cursing and menacing gestures [14]. This man often made biting gestures during postictal confusion. On the day of the homicide, the patient blacked out before arriving at his mother's home. It is likely that he had an additional seizure or seizures at this time that exacerbated his postictal delusions. Postictal disinhibition of frontal inhibitory systems following seizures may have exacerbated his underlying postictal psychosis, diminishing insight, self-regulation, and behavioral monitoring, thus, resulting in the extremely violent aggressive behavior against his mother that he was able to suppress earlier in the day against his sister.

There was no prior history of violence toward his mother specifically and no evidence of conflict with his family. A prosecution psychiatrist opined that an individual would not be aware of details about the attack nor contact the police during psychosis. Many observations and the

medical literature refute this argument. Delusions during postictal psychosis are often recalled by patients days or weeks after the psychosis has resolved. Although patients may manifest violent behavior, they may not understand why they cannot control the behavior and may feel guilt over the violent behavior as in this individual's case [14].

The prosecution psychiatrist also opined that this individual could not have postictal psychosis based on Logsdail and Toone's criteria since toxicology was positive for marijuana. However, this strict application of the criteria is arbitrary and lacks support from the medical or toxicological literature. There is no evidence that consuming marijuana would cause this aggressive behavior. He utilized homegrown marijuana on a nearly daily basis for several years without psychosis, and his delusions only occurred following seizure clusters. Further, many patients utilize marijuana for the resulting perceived seizure control and anxiolytic effects. In larger patient surveys, aggressive behavior has never been reported [21]. In patients with epilepsy, marijuana use has not been shown to have a positive or negative effect in controlled studies compared with other illicit drugs [22]. Substance abusers may demonstrate frontal neural dysfunction during tasks that require inhibition and performance monitoring [23]. This individual demonstrated a direct correlation with increases in seizures and postictal psychosis when utilizing cocaine in the distant past but not with his regular use of marijuana.

### **Conflict of interest**

None

#### References

- [1] Esquirol E. Des Maladies Mentales Considerées sous les Rapports Médical. Hygienique et Médico-Legal. Paris: Baillière; 1838.
- [2] Jackson JH. On temporary mental disorders after epileptic paroxysm. West Riding Lunatic Asylum Med Rep 1875;5:105–29.
- [3] Landolt H. Serial encephalographic investigations during psychotic episodes in epileptic patients and during schizophrenic attacks. In: deHass L, editor. Lectures on epilepsy. Amsterdam: Elsevier; 1958. p. 91–133.

- [4] Slater E, Beard A. The schizophrenic-like psychoses of epilepsy. Br J Psychiatry 1963;103:95–150.
- [5] Flor-Henry P. Psychosis and temporal lobe epilepsy: a controlled investigation. Epilepsia 1969: 10:363–95.
- [6] Ramani V, Gumnit R. Intensive monitoring of interictal psychosis in epilepsy. Ann Neurol 1982;11:613–22.
- [7] Logsdail S, Toone B. Post-ictal psychosis: a clinical and phenomenological description. Br J Psychiatry 1988;152:246–52.
- [8] Devinsky O, Abramson H, Alper K, FitzGerald LS, Perrine K, Calderon J, et al. Postictal psychosis: a case control series of 20 patients and 150 controls. Epilepsy Res 1995;20:247–53.
- [9] Kanner AM, Stagno S, Kotagal P, Morris HH. Postictal psychiatric events during prolonged video-electroencephalographic monitoring studies. Arch Neurol 1996;53: 258–63
- [10] Devinsky O. Postictal psychosis: common, dangerous, and treatable. Epilepsy Currents; 2008;8:31–4.
- [11] Kanemoto K. Postictal psychoses, revisited. In: Trimble MSB, editor. The neuropsychiatry of epilepsy. Cambridge. UK: Cambridge University Press: 2002, p. 117–34.
- [12] Gerard ME, Spitz MC, Towbin JA, Shantz D. Subacute postictal aggression. Neurology 1998:50:384–8
- [13] Kanemoto K, Kawasaki J, Mori E. Violence and epilepsy: a close relation between violence and postictal psychosis. Epilepsia 1999;40:107–9.
- [14] Kanemoto K, Tadokoro Y, Oshima T. Violence and postictal psychosis: a comparison of postictal psychosis, interictal psychosis, and postictal confusion. Epilepsy Behav 2010;19(2):162–6.
- [15] Alper K, Devinsky O, Westbrook L, Luciano D, Pacia S, Perrine K, et al. Premorbid psychiatric risk factors for postictal psychosis. J Neuropsychiatry Clin Neurosci 2001:13:492–9
- [16] Adachi N, Matsuura M, Hara T, Oana Y, Okubo Y, Kato M, et al. Psychoses and epilepsy: are interictal and postictal psychoses distinct clinical entities? Epilepsia 2002;43: 1574–82.
- [17] Weintraub D, Buchsbaum R, Resor Jr SR, Hirsch LJ. Psychiatric and behavioral side effects of the newer antiepileptic drugs in adults with epilepsy. Epilepsy Behav 2007;10(1):105–10.
- [18] Mula M, Trimble MR, Yuen A, Liu RS, Sander JW. Psychiatric adverse events during levetiracetam therapy. Neurology 2003;61:704–6.
- [19] Aggarwal A, Sharma DD, Sharma RC, Kumar R. Probable psychosis associated with levetiracetam: a case report. J Neuropsychiatry Clin Neurosci 2011;23(3):E19–20.
- [20] Helmstaedter C, Fritz NE, Kockelmann E, Kosanetzky N, Elger CE. Positive and negative psychotropic effects of levetiracetam. Epilepsy Behav 2008;13:535–41.
- [21] Gross DW, Hamm J, Ashworth NL, Quigley D. Marijuana use and epilepsy: prevalence in patients of a tertiary care epilepsy center. Neurology 2004;62(11):2095–7.
- [22] Hamerle M, Ghaeni L, Kowski A, Weissinger F, Holtkamp M. Cannabis and other illicit drug use in epilepsy patients. Eur J Neurol 2014;21(1):167–70.
- [23] Gruber SA, Yurgelun-Todd DA. Neuroimaging of marijuana smokers during inhibitory processing: a pilot investigation. Cogn Brain Res 2005;23:107–18.