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Letter to the Editor (Case report)

Consciously-perceived generalized epileptiform discharges in a man with schizoaffective disorder

Epilepsy and psychotic disorders such as schizophrenia co-occur more often than would be expected by chance, and there are several syndromes — the "psychoses of epilepsy" — in which seizures and epilepsy are known to give rise to psychotic signs and symptoms (for a review see Trimble (1991)). Here, we describe a man with schizoaffective disorder and epilepsy whose case demonstrates a new type of epilepsy-related psychotic phenomena; highlights the complex interplay between neurology, psychiatry and pharmacology; and offers support for a "neural hypersensitivity" model of psychosis (Heinks-Maldonado et al., 2007).

1. Case report

The patient, a man with schizoaffective disorder characterized by longstanding psychotic symptoms of paranoid and religious delusions and auditory hallucination, had been treated with multiple psychotropic medications, including clozapine, for many years. He had suffered a head injury at age 25 after obeying command auditory hallucinations to jump off a building, but had a normal head CT and no neurologic deficits. He experienced a generalized tonic–clonic seizure at age 33. Valproate was added to his regimen for both neurologic (antiepileptic) and psychiatric (mood stabilization) purposes.

At age 38 he was noted to be confused and lethargic, and was found to have elevated venous ammonia. His psychiatric medications included valproate, clozapine, risperidone, olanzapine, lithium, citalopram, and gabapentin. Hyperammonemia was attributed to valproate, which was tapered, and replaced with oxcarbazepine. Hyperammonemia resolved, and the patient improved. Neurological exam following these medication changes was notable for mild myoclonic jerking. Myoclonus was considered most likely an effect of one or several of his medications, perhaps unmasked by valproate discontinuation. However, no medication changes were made since the myoclonus was not bothersome to the patient. He was discharged home.

One month later, while taking the same medications, the patient had his second lifetime seizure. He was admitted to a medical hospital where oxcarbazepine and gabapentin were increased. He was then admitted to our psychiatric hospital, where myoclonic jerking was noted to be worse than at his previous admission, and upsetting to the patient. Several medication changes were made to try to ameliorate the myoclonus: gabapentin was increased, then decreased when it was found to worsen myoclonus; oxcarbazepine was continued at lower dose; lamotrigine was added. Clozapine was continued. After several days, myoclonus was diminished. However, the patient reported episodes he called "mini seizures." These episodes consisted of a difficult-to-describe sensation in his head and body with inability to talk and visual illusions of a "messed up version" of his surroundings. Episodes lasted a few seconds and occurred several times per hour. Some episodes seemed to coincide with brief periods of thought blocking, disorientation and/or stuttering. Although these episodes were considered likely to be psychotic phenomena, to characterize them with certainty and determine whether they might be epileptic, the patient was referred for continuous video-encephalography (EEG) monitoring.

During the first day of monitoring, the patient pushed the event button 38 times to report his typical "mini seizures". 29 of these 38 reported events were preceded by generalized spike-and-wave epileptic discharges (Fig. 1). These discharges were otherwise rare. Background EEG was normal. For the 2nd day of monitoring, after the patient was started on topiramate, he reported only 13 mini-seizures, 6 of which were preceded by epileptiform discharges. By the day of discharge, mini-seizures and epileptiform discharges had nearly resolved.

Over the past four years he has continued to take topiramate. He remains chronically psychotic, but has been free of "mini seizures" and overt seizures. He still reports occasional myoclonic jerks.

2. Discussion

This case emphasizes that epileptic events can masquerade as psychiatric phenomena. While it is known that temporal lobe seizures can be misdiagnosed as psychiatric disorders, e.g. (Thompson et al., 2000), we could not find other reports of isolated epileptiform discharges causing psychotic symptoms.

Based on clinical findings and EEG, we believe this patient's convulsions, myoclonus, and "mini seizures" may have been related to clozapine (Gouzoulis et al., 1991; Malow et al., 1994; Pacia and Devinsky, 1994; Sajatovic and Meltzer, 1996; Wong and Delva, 2007), though other medications may have played a role, and an underlying generalized epilepsy syndrome cannot be ruled out. The relationship between his seizures and prior head injury was likely coincidental, since traumatic brain injury does not cause generalized epilepsy. In accord with prior reports, this case suggests that valproate (Foster and Olajide, 2005; Malow et al., 1994; Pacia and Devinsky, 1994) and topiramate (Navarro, 2001) are effective against clozapine-related epileptic events. In disagreement with prior reports (Landry, 2001), this patient's myoclonus appeared to worsen on gabapentin and possibly oxcarbazepine. Because these drugs are effective for partial epilepsy and may worsen generalized epilepsy, this is unsurprising.

It is very unusual that this patient was able to sense and accurately report the occurrence of brief (1–2 ms) epileptiform discharges. Unless accompanied by a myoclonic jerk, such discharges do not typically have any subjective or objective correlate. Although we cannot rule out with certainty the possibility that this patient was actually feeling and reporting subtle myoclonic jerks, we believe this is unlikely because no muscle artifact was present in the EEG record and no movement was visible on video. We could find no mention of this phenomenon of "sensed spikes" in the literature, though it is known to occur rarely in clinical practice. We speculate that it is not coincidental that this patient was extremely psychotic and also had the unusual ability to sense epileptiform discharges. Perhaps this ability can be

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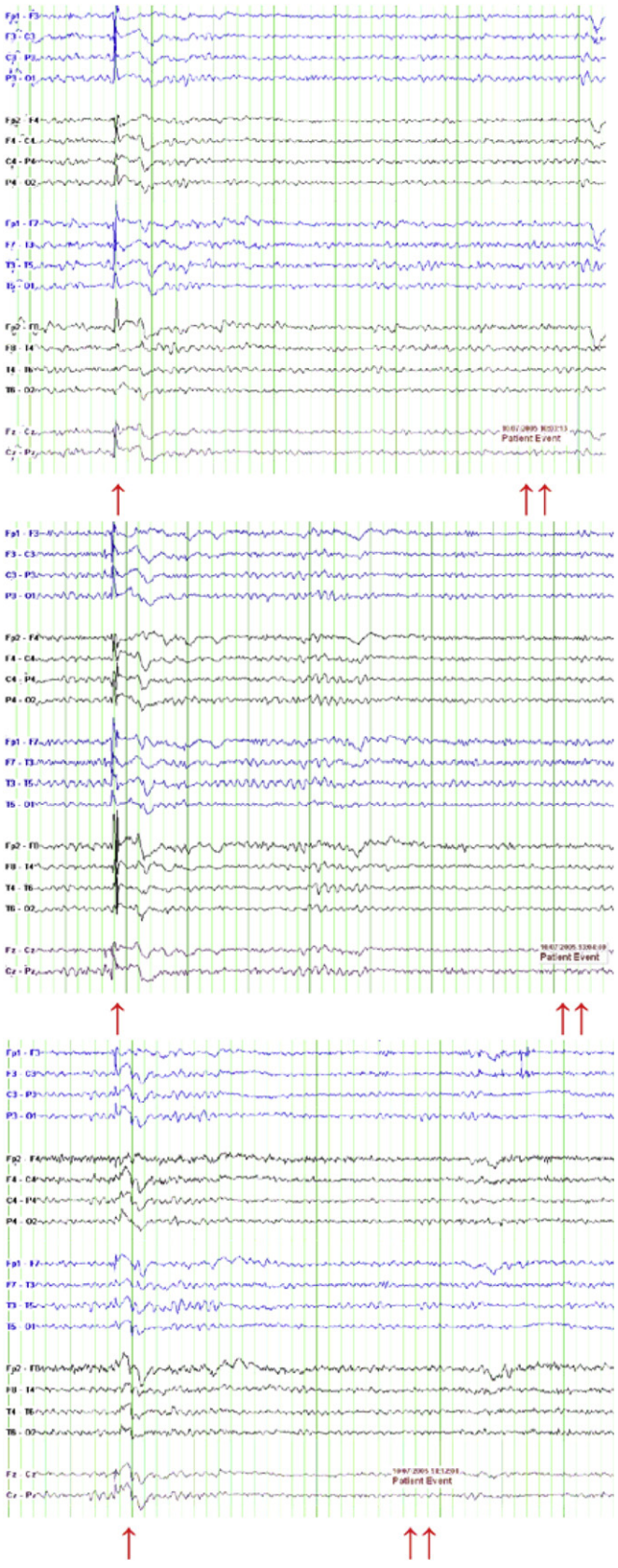

Fig. 1. Examples of brief generalized spike-and-wave epileptiform discharges (single arrow) followed within 5–7 s by the patient pushing a button (double arrow) to report his typical "mini seizure" characterized by psychotic symptoms.

understood as part of an overall neural hypersensitivity to internally-generated events in psychotic patients (Heinks-Maldonado et al., 2007), with the event in this case being an epileptiform discharge. This idea of neural hypersensitivity is derived from a model of psychosis that posits abnormally enhanced sensitivity to self-generated events such as thoughts, sensations or actions in psychotic patients. According to this model, failure to recognize these events as self-generated causes them to be misperceived as externally generated, resulting in such psychotic phenomena as hallucinations and delusions of bodily control (Blakemore et al., 2000; Frith and Done, 1989; Heinks-Maldonado et al., 2007). If the ability to sense usually-imperceptible neural events such as single epileptiform discharges is in fact more common in psychotic patients than in the general population, this could contribute to an understanding of the neural basis of psychosis.

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