Post-ictal psychosis in a patient with a history of nocturnal epilepsy

Aarti Datta  MBChB, Olakunle Oladinni  MBChB, BSc, MRCPsych

Psychiatric symptoms can occur following a seizure (post-ictal) in patients with epilepsy. Here, Drs Datta and Oladinni describe the case of a man with a history of nocturnal epilepsy, who developed psychotic symptoms following two successive seizures.

Predisposition to major psychiatric disorders as a result of seizures has been recognised for a number of years.1,2 Such disorders include post-ictal psychosis; an episodic psychotic state, which can affect up to 10 per cent of patients suffering from epilepsy.3,4 It usually manifests after a lucid interval, which lasts approximately three hours to six days – this is the period from the last seizure to the onset of psychiatric symptoms.5

Post-ictal psychosis often has a polymorphic presentation, including features of mood disturbance such as lability, depressive mood, grandiosity and fear. The latter may extend to the sensation of looming death. In addition, hallucinatory experiences, paranoia and thought disorder have been reported.6-8 Episodes tend to resolve spontaneously over a period ranging from a few days to a fortnight; benzodiazepines may shorten this period.9

Presentation
A 51-year-old man with a history of nocturnal epilepsy, diagnosed when he was 18 years old, was detained under Section 136 for ‘acting in a bizarre manner in the street outside his home’. During the initial assessment in the psychiatric unit, he presented with autonomic disturbance, in the form of excessive sweating and episodes of hyper/hypoventilation. He was therefore referred to the accident and emergency department, where the medical team felt that he was suffering from an ‘acute psychotic episode’ and as such was referred back for a full psychiatric assessment. He was found to be restless and incoherent. Although eye contact was present, rapport could not be established and he was unable to register information.

Collateral history from his wife revealed that the patient had previously been admitted to psychiatric hospital for a period of 10 days, following a nocturnal seizure that had left him feeling light-headed and confused. In addition, five days prior to his most recent detention, he had suffered his ‘first daytime fit’, which was unwitnessed, but during which he had bitten his tongue as well as bruised his limbs. He subsequently had a nocturnal seizure, before being encouraged by his wife to seek medical advice. He did not suffer any other injuries or show any behavioural changes immediately following this, but was said to be ‘groggy’. He had not been taking any regular medication and has not attended any neurology follow-up for over three years.

Admission to a medical ward was deemed necessary for a thorough medical work up and neurological observation as the risk of further seizures and a more chronic change in mental state remained high.10 His Glasgow Coma Scale (GCS) score had fallen from 13/15 to 10/15, and on the basis of this he was started on aciclovir and prednisolone and a CT head scan and lumbar puncture were advised in order to rule out encephalitis. He became more settled and communicative for about 24 hours and his GCS score increased to 15/15.

The following day, neurological observation was stopped after he became aggressive and unsettled on the ward, and was seen responding adversely to external stimuli; he began to lash out at staff and was threatening to other patients. Blood tests revealed no abnormalities of note, and his treatment for encephalitis was stopped. Owing to his chaotic presentation, neuroimaging could not be obtained and a psychiatric consultation was requested by the medical team.

During psychiatric review (over a period of about 48 hours on the medical ward), he presented with a poor attention and concentration and could not register any of the questions asked. Speech was intermittently incoherent and he was not orientated to time or to person. A subsequent neurology review suggested further investigations including EEG, lumbar puncture and MRI in order to rule out the diagnosis of anti-NMDA receptor encephalitis. Less than 24 hours following psychiatric review, he had what appeared to be a partial seizure with tonic
movements and 'twitching' of the limbs. His GCS score fell to 8/15. Airway protection was initiated and 10mg IV diazepam administered and a phenytoin infusion commenced. Repeat blood tests revealed a raised white cell count of 13.4x10^9 per litre with all other tests being unremarkable.

A transfer to a local neurology intensive care unit was arranged and intubation under sedation was carried out with the use of midazolam, propofol and a further bolus dose of diazepam. Aciclovir was recommenced and a further loading dose of phenytoin was administered. Risperidone 4mg four times daily was also administered as per the advice of the neuropsychiatry team.

A battery of investigations, including baseline blood tests, virology, blood cultures, lumbar puncture, EEG and an MRI, were unremarkable and the patient was subsequently admitted to a neurology ward where he remained settled. There were no concerns about his mental state and he also regained full capacity. Neuropsychiatry input continued, with advice sought regarding the use of risperidone. This was reduced from 16mg daily to 6mg daily and then to 3mg daily. The specialist epilepsy team recommended levetiracetam 500mg twice daily; the patient’s liver function tests deteriorated over the first 48-72 hours before recovering slowly. An ultrasound scan of the abdomen came back unremarkable. During this time on the ward, he was settled and compliant with treatment.

The patient was eventually taken off risperidone and discharged with neurology follow up. He was commenced on carbamazepine 100mg daily with a view to titrate the dose post-discharge. Psychiatry services were made aware that the patient had a history of non-compliance and that there was a risk of further psychotic episodes.

### Table 1. Recommended pharmacotherapy for post-ictal psychosis

<table>
<thead>
<tr>
<th>Class of agent</th>
<th>Recommended drugs/daily doses</th>
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<tbody>
<tr>
<td>Antipsychotic drugs</td>
<td>Oral administration: Olanzapine 5mg-20mg, Risperidone 0.5mg-6mg, Quetiapine 50mg-600mg, Amisulpride 50mg-800mg daily</td>
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<tr>
<td></td>
<td>IV administration: Haloperidol 2.5mg-5mg daily, Droperidol 2.5mg-5mg daily</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>Lorazepam 0.5mg-2mg daily, Clobazam 10mg-60mg daily</td>
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### Discussion

There are diagnostic criteria that ultimately distinguish post-ictal psychosis from other psychoses associated with epilepsy, such as inter-ictal and pre-ictal psychosis. Features described by Logsdail and Toone include:
- Episodic psychosis developing within one week after the return to normal mental state post-seizure
- The episodic event spans between 24 hours and three months
- There cannot be evidence of anticonvulsant toxicity, a previous history of inter-ictal psychosis, EEG evidence of non-convulsive status epilepticus, recent head injury, or alcohol or illicit substance intoxication. However, change in mental state can be attributed to other aetiology such as infection or metabolic disturbance.

Our patient certainly met the majority of the criteria for this diagnosis; however, there was some difficulty in establishing whether there was an episode of inter-ictal psychosis. The presence of tongue biting suggests a more generalised nocturnal seizure. Interestingly, however, the current episode of psychosis in our patient followed that of both a daytime and nocturnal seizure and the episode of chaotic behaviour was prolonged, lasting up to 72 hours.

Risk factors for developing post-ictal psychosis and the chronicity of symptoms have been discussed in numerous studies. It has been widely suggested that patients with generalised tonic-clonic or partial secondary generalised seizures are at increased risk. Another finding that may be relevant to our patient is that some studies propose that post-ictal psychosis may not completely resolve between episodes when coupled with poor compliance with antiepileptic drugs (AEDs). Previous admission to a psychiatric unit is also considered a risk factor.

The overall propensity to develop post-ictal psychosis may arise from the structures of the limbic system, with connectivity affected as a consequence of aberrant neuronal regeneration. The lack of an encompassing neurological explanation for post-ictal psychosis can make this a difficult pathology to treat. The underlying mechanism in post-ictal psychosis has been postulated to be akin to Todd’s paralysis. However, in a study carried out by Fong et al, lateral temporal hyperperfusion was observed upon SPECT imaging, which is inconsistent with Todd’s paralysis phenomenon, which would predict the presence of hypoperfusion. Another possible
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A SPECT study involving four patients suffering from temporal lobe epilepsy and post-ictal psychosis revealed increased cerebral blood flow in the mesial frontal region during psychosis. Another report from the same group revealed hyperperfusion in both temporal and mesial frontal regions and the left lateral frontal region. This demonstration of increased cerebral blood flow is consistent with the findings of Fong et al. who showed a marked increase in cerebral blood flow over the right temporal and left basal ganglia in two patients with post-ictal psychosis.

The presence of hyperperfusion may be related to the psychosis. However, the difficulty in incorporating this finding into an established underlying neurobiological mechanism is the fact that seizures may separate cerebral perfusion from metabolic activity by altering cerebrovascular autoregulation.

Furthermore, increased perfusion can be related to increased inhibition or excitation; therefore, we remain unsure about the location of the primary disturbance. Metabolic studies, combined with neurotransmitter labelling using positron emission tomography (PET), would help to clarify these possibilities. It is in understanding and appreciating the various mechanisms that take effect in the post-ictal state that a unifying neurobiological explanation can be formulated.

Studies suggest that low-dose antipsychotic medication such as risperidone should be used in the early stages after signs and symptoms of post-ictal psychosis manifest. Although atypical antipsychotics appear to be the mainstay of management, low-dose haloperidol has been discussed in the literature as another successful psychotropic agent. The propensity to develop extrapyramidal side-effects with haloperidol has led to the suggestion that its use should be preserved for the resolution of active psychosis. Long-term use of antipsychotics is largely discredited in the available literature owing to lowering of the seizure threshold, hence preventative treatment of the episode through prompt administration of benzodiazepines has been recommended. 

The issue of non-compliance also needs to be addressed. In answer to the question, ‘had our patient been compliant with antiepileptic medication would he have been less likely to have developed post-ictal psychosis?’ it is tempting to answer ‘yes’ owing to the seemingly obvious benefits of seizure control in long-standing epilepsy. However, the use of levetiracetam may present with certain difficulties in terms of its association with certain psychiatric side-effects such as anxiety, irritability, agitation and mood changes. A study from 2005 suggests that the addition of vitamin B6 may reduce the emergence of psychiatric symptoms, although offering further medication may have proven unfruitful in this case, given the extent of the patient’s non-compliance.

Appropriate treatment during the acute phase of psychiatric manifestations of epilepsy continues to be a challenge. This case highlights the importance of joint working at the neurological and psychiatric interface to prevent a ‘pass the parcel’ approach to care that tends to occur, especially in settings without an established liaison psychiatry. This is crucial in order to recognise potentially life-threatening organic syndromes and to manage and prevent the psychiatric sequelae of epilepsy.

Declaration of interests
None declared.

Dr Datta is a CT1 Psychiatry Trainee and Dr Oladinni is a Consultant Psychiatrist, Surrey and Borders Partnership NHS Foundation Trust

References