Case Report

Relapse of postictal psychosis following 14-year symptom-free period

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Abstract

Neuropsychiatric sequelae in patients with epilepsy have been vastly studied and documented. These may be affective, cognitive or psychotic. Certain risk factors may predispose some epileptics more to these sequelae. In general, good epileptic control may minimize these outcomes. We present in this report, a case of postictal psychosis (PIP), superimposed on delirium, in a 68-year-old woman, with history of a single previous psychotic illness following a cluster of seizures. This report shows a collaborative management of the neuropsychiatric complications of temporal lobe epilepsy (TLE), by the neurology, geriatric medicine and psychiatry teams.

Key words: Postictal; Interictal psychosis; Delirium; Temporal; Lobe, Epilepsy.

Introduction

Postictal psychosis (PIP) is the development of psychosis (eg. presence of hallucinations, abnormal /bizarre behaviour or affect) within seven days of seizure activity[1,2] and represents approximately 25% of the psychosis of the epilepsy population.[1] The risk factors for PIP include age above 30 years, localisation-related epilepsy, bilateral seizure or interictal foci, clustering of seizures, secondary generalisation, and perhaps, a family history of mood disorders, but not psychosis.[2,3] The most consistent risk factors of PIP include: bilateral or widespread CNS injury, including encephalitis, head injury, bilateral interictal epileptiform activity, borderline intelligence and EEG slowing.[4,5,6] Others include seizure clusters, including tonic clonic seizures, partial epilepsy, especially TLE. The onset of sleep disturbance may be a reliable warning sign of an imminent psychosis. The laterality of seizure focus, age of onset and duration of epilepsy were not significant risk factors in the predisposed.

The psycho-pathogenesis of PIP is poorly understood, but may involve bifrontal, bitemporal hyperperfusion[7], and lateral temporal hyperperfusion.[8] This case report presents some clinical features of PIP, co-morbidities, therapeutic options, and the place of collaborative management by relevant medical specialties.

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Case report

Our patient is a 68-year-old married lady with background history of a psychotic illness associated with late onset partial seizures with secondary generalization in 1993, aged 52. She remained seizure-free on carbamazepine, until summer 2009 when she relapsed after carbamazepine was discontinued due to low white blood cell (WBC) count. The patient admitted that she took her anti-epileptic erratically prior to it being stopped. About two weeks after discontinuing carbamazepine, patient developed a sudden speech arrest, followed by facial twitching for approximately two minutes. Family collateral described her as being in a trance-like state, with tremulous limbs, but without jerking movements, incontinence, tongue biting or loss of consciousness. Over the next 10 minutes, the patient responded very slowly to calls. At a local hospital ED, the patient was noted to be very confused and agitated. She was reported as feeling slightly unwell earlier in the day, and complained of being constipated. In the first 10 days of admission, the patient had several clusters of seizure, an average of three episodes per day, each lasting an average of three to five minutes. Each seizure episode progressed from an initial period of apparent confusion, to speech arrest, with staring into space, lasting about five minutes, then to facial twitching movements. There was no associated incontinence, tongue biting or frothing. The patient always reported amnesia for these episodes, except once when she described her seizure experience like being in a movie, where ward staffs were like actors. She denied *déjà vu* or *jamais vu* experiences. She denied olfactory, visual, auditory or tactile hallucinations. The patient was started on three antiepileptics: oxcarbazepine, clobazam, and levetiracetam. Dose of oxcarbazepine was reviewed when the patient became neutropenic. She was also treated for a urinary tract infection. Psychiatric review revealed sudden onset of paranoia, behavioural disturbance and apparent confusion, following a two-day seizure-free interval. The patient was difficult to engage in interview, making circular movements with her hands. She thought the ward staff wanted to poison her food, and that a bomb was planted in the hospital. She also exhibited delusions of reference, and auditory hallucination. These psychotic symptoms occurred in clear consciousness and were clearly distinguishable from periods of apparent confusion. An impression of postictal psychosis was made. Quetiapine was commenced and dose titrated with therapeutic benefits.

Fluctuating levels of consciousness was noted over a few days, but without lateralizing signs, hence a CVA was outruled. Patient was dehydrated, and was intermittently agitated and aggressive to staff. Review of electrolytes revealed hyponatraemia (Na+ = 125mmol/l), and mild hypokalaemia (K+ = 3.2mmol/l). There was also a transiently raised

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C-reactive protein (CRP), and a sustained, raised gamma-GT. EEG showed features of simple partial seizure activity involving the right hemisphere, with evidence of bilateral cerebral dysfunction. MRI brain showed mild generalised cerebral atrophy, with chronic ischaemic changes, but no evidence of mesial temporal sclerosis. The patient was transferred to a single room with 1:1 nursing care, and was rehydrated. Her mental state became stable after three weeks of agitation, aggression, and frank psychosis.

Past medical history

Patient was diagnosed with TLE in 1993. She also has history of hypertension, hypercholesterolaemia, chronic benign neutropaenia, hysterectomy and cholecystectomy. She was maintained seizure-free on carbamazepine until two weeks prior to admission, when it was discontinued because of low WBC count. Patient stated that her first episode of seizure was following an increase in dose of her hormone replacement therapy (HRT). There is no history of childhood febrile convulsion or CNS infections.

Past psychiatric history

Patient's first contact with the psychiatric service was in 1993, when she had a 12 day hospital admission for her first episode of postictal psychosis. Her symptoms remitted with a combination of thioridazine, chlorpromazine and phenytoin. She required no antipsychotic prophylaxis until the index admission.

Family/personal history

Patient is an only child of her parents and described a happy childhood. She has a science degree, and is a retired teacher. One of her daughters has a history of bipolar affective disorder.

Pre-discharge review

Patient's cognitive function was normal (MMSE = 30/30), and her mood normothymic (Geriatric Depression Scale (GDS) score = 11/30). She was appropriate at interview, with no psychotic symptoms, and not suicidal. She denied any recollection of her being agitated, aggressive or behaving abnormally in the preceding weeks. She had 17/24 on the standard profile score of the Rivermead Behavioural Assessment, which was indicative of poor memory. Patient was given relevant information leaflets and advice. The neurologist's plan was for monotherapy on levetiracetam in the long term.

Post-discharge follow-up

At a haematology review two weeks post discharge, patient remained seizure free, with no psychotic symptoms.

Discussion

Psychiatric disorders may be identified in 25-50% of patients with epilepsy, more often in patients with poorly controlled seizures.[9] PIP often complicates chronic epilepsy, especially in patients with seizure clusters that include tonic-clonic seizures, bilateral cerebral dysfunction, and a family history of psychiatric illness.[6] Patients with intractable temporal lobe epilepsy, which started several years before, are commonly affected.[1] The diagnostic criteria are as below.

PIP may present a characteristic of a lucid interval of 2.5 to 48 hours without apparent symptoms; between the last seizure and the onset of psychosis.[2] Cases of suicide during episodes of PIP have been reported. [6,10] The patient's symptoms were complicated by co-existing delirium, probably due to dehydration, and electrolyte disturbances. This is a cognitive disorder characterised by acute onset, of a fluctuating course of altered consciousness and disturbances in orientation, memory, attention, perception and behaviour.[11]

Table 1: Criteria for PIP[3]

1. Episode of psychosis within one week after a seizure
2. Psychosis more than 24 hours and less than three months
3. Delusions, hallucinations in clear consciousness, bizarre or disorganized behaviour, formal thought disorder or affective changes
4. No evidence of anti-epileptic drug (AED), toxicity, neuroconvulsive status epilepticus, recent head trauma, alcoholic or drug intoxication, withdrawal or prior psychotic disorder

As in this case, another study showed resolution of PIP with low dose quetiapine.[1]

Prior to her first seizure and postictal psychosis, in 1993, this patient had experienced days of sleep disturbance following bereavement. She became paranoid, aggressive, confused, and had EEG features of complex partial seizures. This episode settled within 12 days of inpatient treatment with thioridazine, lorazepam and chlorpromazine. Thereafter, she remained seizure-free on carbamazepine until the index admission, which lasted seven weeks and responded to quetiapine, oxcarbazepine, levatiracetam and clobazam. However, caution should be exercised to avoid long-term antipsychotic treatment in PIP as they have potential to reduce seizure threshold. It is unclear if hormone replacement therapy (HRT) is of aetiological significance in this patient's first episode of PIP. Oestrogen is known to be epileptogenic, while progesterone is protective of seizures. The history of bipolar affective disorder in this patient's daughter supports evidence three, that a family history of affective disorder may be associated with PIP.

The patient also had a transient rise in C-reactive protein (CRP) level (47.5mg/l), which is explained by the UTI. The sustained high gamma glutamyl transferase (GGT) levels, may be related to patient's treatment with anti-epileptic drugs Expert opinion suggests that sleep disturbance may precede onset of seizures and psychosis. Low dose neuroleptics is recommended at the first sign of psychosis or sleeplessness.[12] The finding of residual memory deficits as shown by the Rivermead test is of prognostic importance, and may predispose the patient to future cognitive impairment.

Prognosis

In most patients given anti-psychotic drugs, the short-term outcome of PIP is favourable, but in the long term, progression to severe mood disorders, or to poor prognosis interictal psychosis is possible.[13] The 15 year seizure-free interval between the patient's two episodes of PIP suggests a good prognosis, despite a family history of bipolar affective disorder. However, a progressive increase in duration of individual episodes may be antecedent to intractable, treatment-resistant complex partial seizures. Treatment adherence is of critical importance here.

Conclusion

The patient's history shows that PIP can develop following the first seizure episode, and supports evidence that family history of affective disorders is a risk factor for PIP. Diagnostic

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difficulty may arise where delirium co-exists with PIP. Cognitive impairment may be a long-term outcome in this patient. Both atypical and typical antipsychotics were of therapeutic benefit in this patient. Regular follow-up, and a collaborative approach by relevant specialties, will optimize outcome.

Declaration of interest: None.

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Book Reviews

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Developmental Disabilities from Childhood to Adulthood: What Works for Psychiatrists in Community and Institutional Settings

Eds. Dryden-Edwards R & Combrinck-Graham L. Johns Hopkins University Press, Baltimore 2010. 376 pages. ISBN: 978-0801894183

Textbooks focusing on the psychiatry of intellectual disability are uncommonly published, and tend to mainly emanate from the UK, where this area of specialization has prominently developed. This postgraduate textbook from the US is multi author, mainly written by clinical practitioners, and mainly psychiatrists.

The psychiatry of intellectual disability has not developed in the US as a sub-speciality of psychiatry, and this is somewhat reflected in the overall broad perspective that this book presents. Based on the biographical information given on each author, it would appear that, in general the authors treat people with intellectual disabilities as part of their practice, but not exclusively, and this is reflected in the style and detail of the book.

The definition of intellectual disabilities is somewhat confusing, and in this book, the term is used interchangeably with developmental disabilities and mental retardation. This is unfortunate, as a general psychiatry readership would benefit from clear definitions and term usage. In general the editorial input to each chapter appears 'light-touch', with each chapter differing significantly in detail, style and layout. Also, in general the book does not, in any of its chapters deal with the important issue of psychiatric diagnostic classification in people with intellectual disabilities.

The book deals with its subject matter by adopting a life-span approach, and is divided in to five parts looking at developmental disabilities in general, aetiology and assessment, community living, interventions, and ending with a section looking at advocacy, as well as ethical and legal issues. Case examples are occasionally given, but overall the style of writing is quite a dense prose, with very few illustrations and no colour. It is generally well referenced and has a full index; its glossary is surprisingly incomplete. There is no preface or forward, and as a result the reader is left without a clear understanding of the purpose and intended readership of the book.

Of course exposure to different diagnostic and therapeutic approaches is a welcome feature of this book. The chapter on acquired brain injury in children is particularly good. While this is topic is not usually covered in this type of textbook, it is nonetheless a particularly informative chapter. The neurobiology of brain injury was well described and illustrated. The topic of autism is covered comprehensively in at least two chapters, with the section on assessment and psychopharmacological approaches particularly strong. There are also good case examples.

Chapters covering community living and services, as well as the legal and practical aspects of special education were of course written with a US readership in mind, but nevertheless exposed to me the similarities and differences between the US and the Irish situation.

In summary, this is a welcome addition to the small but growing library of intellectual disability psychiatry. The book is very suitable for a specialist intellectual disability readership. However, its dense prose and unfriendly layout, unclear definitions and limited discussion of diagnostic classification makes it less recommended for a potential general psychiatrist reader.

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