CASE REPORT

Intermittent catatonia and complex automatisms caused by frontal lobe epilepsy in dementia

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SUMMARY

An 82-year-old man was admitted to the emergency department following bizarre behaviour. Police had noticed him driving erratically through his village. He did not stop when instructed, drove slowly home and appeared 'vacant' on questioning. While in hospital, he had approximately 15 episodes of catatonia, involving rigidity, negativism, mutism except echolalia and perseveration, automatic obedience and utilisation phenomena, lasting 2–20 min each. Between episodes, he was amnesic but otherwise well. Electroencephalography demonstrated bifrontal slowing with left-sided emphasis, and captured two focal onset partial seizures with the clinical correlate of the syndrome described above. He improved rapidly on levetiracetam and lorazepam, was discharged and received a diagnosis of dementia by his community mental health team shortly afterwards, based on chronic short-term memory loss, functional decline and MRI changes. This case has implications for our understanding of the neural correlate of catatonia, specifically frontal lobe pathway dysfunction.

BACKGROUND

Catatonia is an interesting clinical syndrome that we feel is suboptimally understood by general medical clinicians and psychiatrists alike. We feel that the neurological aetiologies of catatonic symptoms are often overlooked, to the detriment of patients, as the cause is assumed to be 'psychiatric'. This particular case illustrates thorough and informative management of a memorable presentation.

CASE PRESENTATION

An 82-year-old man presented to the emergency department with a 1-day history of bizarre behaviour. His wife had reported him missing after he went outside to move his car and did not return. Police noticed him driving slowly and erratically through his village; he did not pull over when instructed and drove home. He did not get out of the car when initially instructed and did not acknowledge the presence of the officers. He eventually got out of the car and slowly tried to open his front door with his car key. He was vacant on questioning. His wife reported two episodes during the previous evening, both lasting <10 min, when the patient was vacant but motionless.

The patient had crashed his car 9 months ago, following which he had an event resembling a grand mal seizure. Levetiracetam 25 mg once daily

(OD) was started following this, though this dose is unlikely to have provided adequate prophylaxis against seizures. He had received a diagnosis of severe depression without psychotic symptoms in the fortnight following this by a community mental health team, but was felt to have been in remission recently. He had recently scored 17/30 on a Montreal Cognitive Assessment, losing marks for recall (0/5), orientation (3/6), attention (3/6) and visuospatial/executive (3/5).

He had a medical history of hypertension, hypothyroidism, osteoarthritis, asthma and hypercholesterolaemia. He was taking mirtazapine 45 mg each night (ON), levetiracetam 25 mg OD, quetiapine 25 mg ON and nitrazepam 10 mg ON, as well as hydralazine, amlodipine, atorvastatin, cholecalciferol and thyroxine.

On arrival in the emergency department, his Glasgow Coma Score was 14/15. His Abbreviated Mental Test Score was 5/10. Respiratory, cardiac, gastroenterological and neurological examinations were unremarkable. His speech was fluent, and he was co-operative. He had no memory of recent events. He was aware of his location, but confabulated reasons for his admission. A psychiatric assessment was requested.

By the time of mental state examination by a psychiatrist, he was non-verbal and able to follow gaze but not instructions. His limbs were rigid and facial expression was blank. He automatically grabbed the assessor's hands and a cup when presented with them, but would not let go. Five minutes after this, he was re-examined; he had reverted to the mental state and examination findings identified on arrival. His mood became bright and responsive, and there were no psychotic symptoms or neurological signs.

A further 14 similar episodes were documented in the 24 hours following admission, varying between 2 and 20 min in duration. Each episode self-resolved. Additional observed features included echolalia, perseveration, negativism, utilisation phenomena and automatic obedience.

INVESTIGATIONS

Full blood count, renal function, electrolytes, thyroid function, bone profile and C reactive protein were unremarkable. ECG showed sinus rhythm. MRI of his brain showed parenchymal volume loss commensurate with age, small irregular deep white matter T2/fluid-attenuated inversion recovery signal hyperintensities consistent

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with chronic microangiopathic changes and bilateral lacunar infarcts.

Electroencephalography (EEG) demonstrated bifrontal slowing with left-sided emphasis, and captured two focal onset partial seizures with the clinical correlate of the syndrome described above.

A lumbar puncture was not felt to be indicated as encephalitis was not suggested by observations, bloods or on imaging; and symptoms abated on medical management.

The Bush-Francis Catatonia rating scale was applied retrospectively. Ten of the 14 screening criteria were present (only two are required for diagnosis), with a total score of 25 over the full 23-item list.

TREATMENT

Intravenous levetiracetam was started at 500 mg two times a day (BD) and was converted to 1 g BD orally after 3 days. Oral lorazepam was started at 2 mg BD and stopped after 3 days.

OUTCOME AND FOLLOW-UP

The episodes ceased by day 4 of admission. The patient was discharged home and remains seizure-free. He was diagnosed with vascular dementia by his community mental health team based on the worsening of short-term memory deficits, executive dysfunction and functional decline in the context of the MRI findings and absence of further seizures or mood disorder. However, a diagnosis of vascular dementia requires the presence of dementia, cerebrovascular disease defined as the presence of focal neurological signs, and a time-dependent relationship between the two. Therefore, Alzheimer's may be a plausible differential. A single-photon emission CT scan has not been performed for pragmatic reasons but would provide further diagnostic clarity.

DISCUSSION

We have described an 82-year-old man with frontal lobe epilepsy (FLE) within dementia, presenting with complex automatisms and intermittent catatonic symptoms.

Frontal lobe seizures are uncommon, accounting for 1%–2% of all epilepsies.¹ The aetiology of FLE includes trauma, tumours, vascular malformations, encephalitis and cortical dysplasia although approximately half of the cases are idiopathic.^{2,3} Frontal lobe seizures are often short and followed by rapid recovery. They are heterogeneous, both clinically and electrophysiologically. One suggested classification is tripartite: focal clonic seizures involve the contralateral motor strip and manifest as contralateral clonic movements, whereas bilateral asymmetrical tonic seizures and complex motor seizures are thought to occur in different subdivisions of the frontal lobe and thus have different, often more complex, motor phenomena or bizarre vocalisations.^{4,5}

Frontal lobe seizures are not commonly known to manifest as catatonia, with only one prior case reported in medical literature.⁶ In that instance, a 47-year-old man presented with acute periodic catatonia with no history of psychiatric illness. He underwent multiple normal EEGs and repeated hospital admissions in a state of catatonic stupor, before a diagnosis of FLE was confirmed with two abnormal EEGs. Similar to our patient, these EEGs showed short bursts of seizure activity correlating with his periodic catatonia, and he was successfully treated with carbamazepine.

Catatonia has a wide range of psychiatric and organic causes, but the exact underlying pathophysiology remains unclear.

As reports of catatonia due to diffuse central nervous system pathology are much more common than those of catatonia caused by a single lesion,⁷ it is thought that catatonia is caused by pathway dysfunction as opposed to focal insult. Catatonia without identifiable neurological cause appears treatable with benzodiazepines and electroconvulsive therapy, providing insight into aetiology. Gamma-aminobutyric acid type A (GABA-A) agonists, such as lorazepam,⁸ alleviate catatonic symptoms in most patients, thus it is thought that dysregulation of GABA-A may be involved. It has also been suggested that N-methyl-D-aspartate (NMDA) hyperactivity can lead to dysregulation of GABA-A, and thus that NMDA antagonists may indirectly correct this.⁹ In 2005, a case was reported in which a patient with vascular dementia had catatonia refractory to multiple treatments¹⁰ before eventually responding to the NMDA antagonist memantine.

Our case indicates that catatonia should be a recognised, though rare, syndrome of FLE. It suggests that frontal pathways and regions play a primary role in catatonia, and that dementia is a predisposing factor, potentially by damaging frontal pathways and lowering seizure threshold. In such cases, anticonvulsants appear effective.

Learning points

- ▶ Catatonia has numerous neurological causes, including frontal lobe epilepsy.
- ▶ Identifying such causes requires a high index of suspicion and use of appropriate investigations.
- ▶ Patients with catatonia induced by frontal lobe epilepsy appear to have intermittent symptoms and respond well to anticonvulsants.
- ▶ Dysregulation of frontal lobe pathways and gamma-aminobutyric acid activity are likely to form part of the neural substrate of catatonia.

Contributors Both authors assessed the patient. RG reviewed the medical notes at length, performed the background literature search and prepared the manuscript. AL edited the first and subsequent drafts.

Competing interests None declared.

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