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A case report of an unusual presentation of a patient with recurrent idiopathic catatonia

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ABSTRACT

Background: Catatonia-like presentations can be precipitated by multiple organic and medication-related causes. Psychiatric causes of catatonia are typically associated with underlying psychotic or mood disorders. Recurrent catatonia without other precipitating psychiatric diagnosis is rarely described.

Methods: We present the case of a man in his early 30′s with idiopathic recurrent catatonia, presented with patient consent.

Results: Our case presented in a catatonic state, having recently stopped using cannabis. No organic cause for his presentation was identified following extensive investigation and he was admitted for psychiatric assessment. During admission he slowly improved with benzodiazepine and electroconvulsive therapy (ECT) treatment, alongside psychological support. Despite near complete recovery, he significantly relapsed on 2 occasions requiring psychiatric re-admission over the subsequent 6 months. Thereafter, he had multiple relapsing episodes with decreasing severity during rehabilitative care. During admission we explored extensive differentials including mood disorders, schizophrenia or psychosis, drug abuse or poisoning, as underlying triggers for his catatonia. He had a finding of FIRDA (frontal intermittent rhythmic delta activity) on his second electroencephalogram (EEG), with no clinical correlate of seizure or structural abnormality. We found no evidence of any underlying psychiatric or organic cause for his presentation.

Conclusions: In contrast to classical descriptions of catatonia or recurrent catatonia, our case highlights the need for greater recognition of isolated idiopathic catatonia, as a diagnosis independent of mood disorder or schizophrenia. Furthermore, we evidence effective recovery with psychological support, benzodiazepines and ECT.

Introduction

We present a case of recurrent idiopathic catatonia with no psychiatric, organic, or other precipitating cause identifiable. Intriguingly our case has a recurrent cyclical nature, which does not fit canonical descriptions of periodic catatonia by Kahlbaum (Catatonia, 1973, Barnes et al., 1986, Peralta et al., 1997 Jan) or Leonhard (Perris, 1990, Leonhard, 1979). The patient has reviewed drafts of the manuscript and consented to the publication, following changes to clarify his personal details.

Our patient was a single man of Punjabi ethnicity in his early 30′s, living with his grandmother and working as a courier driver at first presentation. His only psychiatric history was of reported low mood and possible suicidal ideation two years prior. This was managed in primary care by his general practitioner (GP), requiring no medication or secondary care input. He completed secondary and further education to the age of 18, and had been employed from age 17 until 1 month prior to his first admission. He had no family history of any psychiatric disorder or neurological disease (including epilepsy) amongst his grandparents, parents' generation or in his sibling. He had no children. His father died aged 36, reported to be an alcoholic, with the cause of death reported by the family as 'heart attack'. Our patient was regularly smoking cannabis prior to admission. His case was first recognised when he was urgently referred by his GP for increasing isolation and withdrawal, having not left the house for 2–3 weeks. He reported that he had recently stopped smoking cannabis before becoming increasingly

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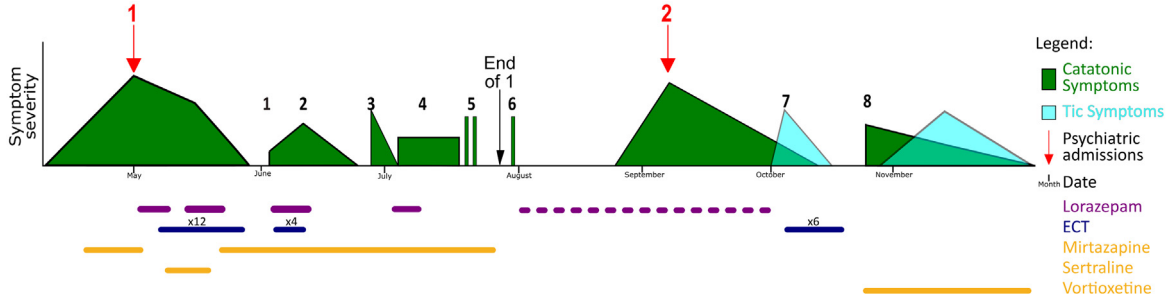

Fig. 1. Pictorial timeline of 8 months of symptoms and treatment, across 2 admissions
Red arrows represent admissions 1 (admitted after a 3 week history of increasing catatonia at home) and 2 (admitted to the acute psychiatric ward with gradual decline in psychomotor skills, overall retardation and catatonia) as an acute psychiatric inpatient. Black arrow shows the end of admission 1. Key clinical events are demonstrated by numbers 1–8. Green filled areas denote catatonic features (height broadly represents severity, width denotes duration). Blue areas highlight the appearance of tic/hyperactive symptoms. Medication and treatments is shown by coloured bars, dashed bars shows 'as required' medication used intermittently.
Key events: [1] – Catatonic following home leave; [2] – Fluctuating symptoms, ECT and benzodiazepine responsive. Neurology input sought; [3] – Readmitted following home leave, catatonic and doubly incontinent; [4] – Relapses at home, without representing; [5] – Sudden-onset GCS 6, dystonic, laryngospasm. Seen in secondary care ED for the same symptoms on 2 consecutive days, resolved with IM procyclidine; [6] – Choking on tables: catatonic, transferred to an acute hospital from home, discharged the same day; [7] – Appearance of tics with lip smacking, rocking and akathisia; [8] – Relapse from home leave with fluctuating catatonia.
Abbreviations used: ECT – electroconvulsive therapy, GCS – Glasgow coma score, IM – intramuscular.

isolated. He was initially reviewed in the emergency department (ED), presenting with salivary dribbling, urinary incontinence and profound psychomotor retardation. On admission he was mute, stuporous, lying in bed staring at the ceiling and doubly incontinent requiring incontinence pads. He scored 26 (of 69) on the Bush-Francis Catatonia Rating Scale (BFCRS) on this first admission, positive for 7 of 14 screening features and 10 of 23 severity features. Hydration was challenging to maintain, achieved by offering oral fluids through a straw, which he managed slowly with assistance. He required high-calorie elemental diet drinks, following dietetic advice, to maintain his basic nutritional requirements. Extensive medical review and investigations (**Supplementary Table 1**) excluded organic causes, and he was transferred to our acute psychiatric hospital. The only finding of note was FIRDA (frontal intermittent rhythmic delta activity), on a second EEG undertaken towards the end of his first admission. An MRI was normal, undertaken to identify structural or pathological causes of FIRDA. In line with consensus for the investigation for NMDA (N-methyl-Daspartate) receptor encephalitis, (Warren et al., 2020, Dalmau et al., 2011) serum testing was performed, and being negative for NMDA receptor antibodies CSF testing was not carried out.

We initially diagnosed depression with catatonia, considering differentials of drug-induced or idiopathic psychosis, and/or drug withdrawal. He was treated with lorazepam (1–2 mg daily) augmented by electroconvulsive therapy (ECT) to good effect (all treatments, medications, doses and routes are detailed in **Supplemental Table 2**, summarised in Fig. 1). He showed gradual steady improvement by the 6th session of ECT, whereon he was able to mobilise, feed independently and articulate himself. He completed a total of 12 ECT sessions and lorazepam was stopped the week prior he was granted home leave, having regained capacity. Unfortunately he returned from home leave catatonic, with neck extension, mouth fixed open and an upward starring gaze (Fig. 1, **label 1**). Lorazepam and ECT treatment were restarted, with fluctuating symptoms prior to improvement by the 4th ECT session. Suspicions of illicit drug use whilst he was on leave were raised, however toxicology screen at this point was unobtainable. Furthermore, his family dynamics were challenging, where our patient's decompensations correlated temporally with family visits. Suspicions were raised of ulterior motives amongst some members of the family. A differential diagnosis of poisoning, from home-cooked food brought to the ward, was considered. At this point family visits and home food was suspended. He steadily improved and was discharged home. Five days after discharge he presented to secondary care with an acute chocking episode and catatonia, he was medically managed for oculogyric crisis, with a single dose of lorazepam. He was discharged the same day with acute psychiatry input, and reviewed at home without ongoing symptoms.

He remained fully well for one month at home, but represented with identical symptoms. Over the following year, our patient developed over 5 episodes of catatonic relapse, of varying intensities, some self-resolving. Other relapses necessitated further courses of lorazepam and/or ECT, with two further admissions and multiple ED attendances in varying degrees of stupor. From his third admission onwards, the episodes became milder, more self-contained and typically resolved by 48 hours. Doctors on call out of hours diagnosed dystonic reactions, cervical dystonias and oculogyric crises, successfully treated with procyclidine or lorazepam. A similar dystonic reaction was observed during the first admission and was treated similarly. Sertraline was suggested as the offending agent, and was discontinued with immediate effect. Repeated urine and blood toxicology during catatonic episodes disproved illicit drug use, including testing by ultra-performance liquid chromatography time-of-flight mass spectrometry (**supplementary table 1**). He tested positive for cannabis and cocaine once only, at the beginning of his first admission, but never again as an inpatient despite multiple urine drug screens. Blood toxicology demonstrated haloperidol during his third relapse, which he had not been prescribed for 9 months' at that point. It was considered by his treating team at the time that haloperidol use may have triggered his catatonia-like presentation, particularly in the context of an early dystonic reaction responding to procyclidine. The team could not establish where the patient would have obtained haloperidol and the patient denied knowingly using haloperidol. It was considered possibly a contaminant of the patient's intermittent illicit drug use, but unlikely the predominant driver for recurrent catatonia.

Poisoning was discounted, with recovery independent of exposure to home-cooked food. The treating team held multiple family meetings with all members of the family, and it became clear that despite tensions, the family demonstrated genuine concern for our patient's well-being. He was transferred to a rehabilitation unit following his third admission. Although his catatonia became more manageable throughout his rehabilitation, our patient returned to using illicit substances, including cocaine and cannabis. He was also drinking alcohol to excess and self-identified as an alcoholic, subsequently attending alcoholics anonymous. Throughout his rehabilitation, there was ongoing family anxiety and he developed non-epileptic (functional) seizures, which were temporally correlated with, and likely triggered by, growing family tensions. Early during his residential rehabilitation, he had multiple catatonic episodes, some requiring emergency secondary care admissions, however these catatonic episodes were significantly reduced in frequency

and intensity prior to discharge. Furthermore, catatonic features were well controlled and not exacerbated during the period he was using cannabis and cocaine, and drinking to excess. His treating team reported no temporal relationship to the use, or withdrawal, of illicit drugs or alcohol.

Discussion

Catatonia is a neuropsychiatric syndrome with a unique combination of mental, motor, vegetative and behavioural signs. (Dawkins et al., 2022 May 23, Penland et al., 2006) Catatonia has been strongly associated with schizophrenia and mood disorders. (Peralta et al., 1997 Jan) If catatonia is considered as a separate diagnostic entity, as Kahlbaum did, sensitivity to medications, recreational drugs, emotional stressors, infections or other insults, could all be reconciled as precipitating factors. The periodicity in periodic catatonia has raised questions regarding its relationship to other cyclic illnesses. The most obvious of these is bipolar disorder. The frequent presence of catatonic features in mood disorders is well recognized (Perris, 1990, Abrams and Taylor, 1976, Fein and McGrath, 1990). Patients with severe psychotic symptoms in the manic or depressive stages of bipolar disorder may indeed resemble the periodic cycling catatonic patient. Case reports since the 1960s and 1970s have reported successful treatment of cyclical catatonic illness with lithium citrate (Padhy et al., 2014, Gjessing, 1967, Sovner and McHugh, 1974, Pétursson, 1976, Sienaert et al., 2014). L. R. Gjessing was seminal in reporting periodic catatonia as an entity from the 1950s to 1970s. (Gjessing, 1974, Gjessing, 1975, Minde, 1966) Although their periodic patients differed greatly from our patient, in cycling more rapidly and having underlying mental health diagnoses, particularly mood disorders. Gjessing demonstrated a relationship with catatonia and the metabolic state of his patients, however these data have not been repeated. (Minde, 1966) Kahlbaum, however, does not mention or describe a periodic disease with recurrent episodes. Leonhard's classification on the other hand, describes periodic catatonias, but he ascribes it to a single diagnosis of schizophrenia, and classifies periodic catatonia as a subtype of schizophrenia. There is long-standing pressure to consider catatonia as a stand-alone entity. (Padhy et al., 2014) There has been some progress with the recognition of idiopathic catatonia, (Gazdag et al., 2017 Sep 22) independent of other organic or psychiatric disorders, with 'unspecified catatonia' recognised in the DSM-5 (Diagnostic and Statistical Manual of Mental Disorders), (Tandon et al., 2013) whereas catatonia remains a subtype of organic or psychiatric disorders in the ICD-11 (International Classification of Diseases and Related Health Problems 11). (Reed et al., 2019)

Our case presented a diagnostic and therapeutic challenge, initial working diagnoses of severe depression with catatonia, cannabis-induced or withdrawal-induced catatonic states were excluded, lacking mood symptoms and with repeated drug screens negative for drugs of abuse. This is particularly supported by his later return to cannabis, cocaine and alcohol use whist in residential care, once his catatonia had resolved. Neither this use, nor later abstinence, led to recurrence of any catatonia symptoms. The recurrent nature and rapid onset of his catatonic relapses, without clear precipitant, was challenging to for the patient and his treating team. It is recognised that benzodiazepine withdrawal may precipitate relapse. Indeed, lorazepam had been stopped a week prior to his first relapse. Subsequent relapses were not clearly related to benzodiazepine use or withdrawal (Fig. 1, points 3, 5, 6 and admission 2).

Throughout the multiple episodes of catatonia, our patient's physical observations were consistently within the normal range, he had no fever, and had a creatine kinase marginally above the normal range, excluding a diagnosis of neuroleptic malignant syndrome. Our patient had no personal or family history of autoimmune disease; his symptoms were generalised across his entire body, non-progressive, and eventually improved, excluding the rare diagnosis of stiff person syndrome. He experienced no symptoms of intrusive thoughts, images, or compulsive behaviours (suggestive of obsessive-compulsive disorder), throughout the admission to consider the patient's presentation as obsessive slowness. Our patient was and is functioning independently before, after, and throughout the admission when he was not catatonic, ruling out early-onset dementia or mental retardation. A neuropsychological evaluation was not completed to exclude personality disorder. His behaviour throughout the admission, however, was never problematic and never raised suspicion for personality disorder. The patient was cooperative throughout the admission, expressed frustration at his long hospitalisation, and was always looking forward to discharge. He demonstrated sincere interest in understanding his disorder and recovery to the treating team and visiting family. A secondary gain was considered but no gain was ever identified, making a diagnosis of malingering unlikely. Munchausen or fabricated illness were considered, as haloperidol was detected by ultra-sensitive mass spectrometry. However, haloperidol is rarely a drug of abuse so we discounted haloperidol self-intoxication. Haloperidol in the supratherapeutic range may mimic a catatonic presentation, nevertheless, for haloperidol use alone to replicate this presentation would require repeated dosing in a secure inpatient facility, which we deemed unlikely.

Non-convulsive status epilepticus should be considered in all cases of unexplained or non-psychotic catatonia. These are known to mimic catatonia. (Sutter and Kaplan, 2012, Ogyu et al., 2021, Silva Gadelho and Gama Marques, 2022) In our case, the initial EEG was undertaken whilst catatonic, performed by clinical neurophysiologists in conjunction with neurology colleagues, and was not suggestive of status. Furthermore, none of the EEG traces prior to ECT sessions whilst catatonic were suggestive of non-convulsive status epilepticus, nor did the subsequent EEG, showing FIRDA, suggest status epilepticus. In addition, his direct and clear (if slow) communication with his clinical team during his recovery, and lack of retrograde amnesia of his severe catatonic episodes, discount status epilepticus.

A psychosomatic disorder or conversion reactions could never be fully excluded. The periodic and cyclical nature of our patient's presentation prompted consideration of an unusual unspecific form of affective psychosis, for which treatment with a mood stabiliser such as lithium carbonate or lithium citrate was considered. These were never commenced due to an absence of a robust affective component, and the use of a mood stabiliser was not justified in our view.

Our case report has a number of limitations. Firstly, the confounding use of recreational drugs (particularly cannabis and cocaine) and alcohol made it challenging to fully exclude these as triggers or precipitants in the catatonic presentation. We believe the significant temporal separation of his drug and alcohol use from catatonic episodes provides evidence they are not relevant in his catatonia. Furthermore, the heavy drug and alcohol use (and subsequent withdrawal) 18 months following initial presentation and periodic catatonic episodes did not precipitate any catatonic episodes. Secondly, we have limited explanation for the finding of FIRDA on his second EEG. Whilst this finding is most commonly associated with structural brain abnormality, metabolic encephalopathy or seizures in adults, (Kim et al., 2021, Fariello et al., 1982, Mina et al., 2019) this was excluded by MRI and neurological review in our patient. Whilst EEG is requisite in exploring the aetiology of catatonia, non-specific abnormal EEG findings are not uncommon in catatonia without a medical diagnosis. (Hosseini et al., 2023) Given the diagnostic uncertainty in this patient we sought to have further outpatient neurology input, including further EEG and sleep EEG studies. These could have been expanded to further neuroimaging including PET. Unfortunately, whilst in recovery in the community (with capacity) he failed to attend many neurology outpatient appointments, including follow-up EEG studies. Lastly, genetic studies in this patient were not considered. However, there are small genome-wide studies suggesting regions of chromosomes 15 and 22 are associated with periodic catatonia in schizophrenia. (Stöber et al., 2001, Stöber et al., 2002) These associations have not, to date, demonstrated pathogenic variants. Given no evidence of pathogenic loci in periodic catatonia, no evidence

of schizophrenia in our patient and no psychiatric family history, genetic testing in our patient remains unlikely to be clinically justified outside a research setting.

We believe that the supportive psychological input and group therapy he recieved as an inpatient, and throughout his rehabilitation, contributed to the reduced severity and frequency of our patient's catatonic relapses. However, the protracted and relapsing nature of his illness complicated his management and caused distress to our patient, particularly with respect to its unpredictable nature.

Conclusion

Our key learning points from this case would be to consider all possible precipitants in patients with recurrent catatonia, including pharmacological, environmental, psychological and psychiatric. To investigate precipitants where possible, in order to exclude them. Where benzodiazepines alone are ineffective, we and others have found adjunctive ECT effective (Sienaert et al., 2014, Luchini et al., 2015, Pelzer et al., 2018). Lastly, we draw attention to the literature highlighting the benefit of lithium in catatonia driven by an underlying mood disorder. (Gjessing, 1967, Sovner and McHugh, 1974, Pétursson, 1976)

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.psycr.2023.100111.

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