CASE REPORT

A Case of Excited-Type Catatonia: The Need for Early Recognition and Treatment

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Abstract

We report a case of catatonia of the excited-type in a 59 year old male with known schizophrenia, ischaemic heart disease and cerebrovascular disease. The main presenting features were excessive motor activity, bizarre posturing, impulsitivity, disorganised speech and confabulation. Regular daily use of a low dose of lorazepam was shown to be effective in this case with resolution of symptooms. Difficulties were encountered in trying to determine the exact cause of his catatonia.

Keywords: Catatonia, Excited, Management, Aetiology

Introduction

Catatonia is a neuropsychiatric syndrome of motor and behavioural dysregulation linked to various aetiologies but unified by a common underlying pathogenetic process that is yet to be fully understood. [1, 2] An essential feature of this condition is the rapid and effective response to benzodiazepines or electroconvulsive therapy (ECT), both of which are well documented in the literature. A severe form of this condition, known as malignant catatonia has been associated with a death rate as high as 9%. [3]Thus, it is important to recognize the clinical aspects of this condition. We therefore present a case on catatonia and the difficulties involved in uncovering the underlying cause.

Case Report

Mr. A is a 59 year old Caucasian male who presented with attempted self-harm. He was diagnosed with schizophrenia in the 1980s and is also known to have stable angina, and substance misuse with alcohol and cannabis. In this presentation, he was found wandering outside the police station threatening to kill himself due to a low mood. He had apparently left his wife 3 days ago and spent this time living off the streets.

A mental state examination instead revealed a labile mood with incongruent affect. He appeared irritable and easily excitable. His speech was pressured with confabulated contents that sometimes made no sense.

There was associated perseveration and at times, use of a foreign American accent. He appeared to have impaired concentration but scored 27/30 on a MMSE, losing the few points in orientation of time. During his admission, he displayed bizarre posturing, odd mannerisms and impulsitivity. At times, he would be observed pacing up and down the ward, speaking constantly to himself or anyone that passed by. He would also repeatedly attempt to abscond from the ward to contact emergency services with various "made up" scenarios, such as a broken ankle. Physical examination, routine blood sampling and urine toxicology tests carried out were unremarkable.

A speech and language therapy (SLT) assessment revealed impairments across a range of language modalities with semantic and word finding difficulties alongside his stereotypical speech. A neuropsychological assessment highlighted difficulties in orientation, attention, memory and reduced executive functioning. An occupational therapy assessment of his kitchen skills revealed him to be "chaotic" and unsafe.

Catatonia was suspected and the Bush-Francis Catatonia Rating Scale (BFCRS) was selected to assess this.[4] There are 2 components to the BFCRS; a screening component of 14 items, where having 2 or more listed symptoms is indicative of catatonia, and a severity component of all 23 items, each given a score of 0 to 3. He had 7 different symptoms in the screening component and scored a 13 overall on severity. Catatonia of the excited form was diagnosed. A trial of regular oral lorazepam of 1mg tds was initiated. A dramatic resolution of his symptoms ensued within the first day. His bizarre posturing and mannerisms were no longer present. Reassessment by SLT services showed complete resolution of his stereotypical speech with improved working memory and frontal executive functioning. Occupational therapy assessments in the kitchen and outdoors now reported that he was displaying appropriate living and social skills.

Further investigations were carried out to pinpoint the cause of his catatonia. His recent EEG was unremarkable however a MRI brain scan and a SPECT scan revealed multifocal areas of ischaemic changes in the subcortical deep and periventricular white matter with involvement beyond the frontal lobes of both hemispheres.

Discussion

Catatonia is commonly divided into an excited or withdrawn type depending on the patient's presenting signs, although the picture is often mixed. Classic withdrawn-type catatonia is characterised by stupor, mutism, negativism, and posturing while excited-type catatonia is attributed to mannerisms, rituals, disorganized speech, disorientation, aggression, and violence. Studies have shown the prevalence to range from 7-38% of psychiatric inpatients.[5] In another large study, clinicians diagnosed catatonia in 2% of 139 psychiatric inpatients. However, the research team identified catatonia in 18% of patients, pointing towards a trend of underdiagnosis.[6] There are a number of catatonic rating scales that can be used for screening purposes, as evaluated by Pascal et al.[4] The study concluded that the Bush-Francis Catatonic Rating Scale (BFCRS), Northoff Catatonic Rating Scale (NCRS) and the Braunig Catatonic Rating Scale (BCRS) are all reliable for use. The BFCRS was considered preferable and was used in this case for its validity, reliability and ease of use. [4]

There were several difficulties and thus,

learning points, encountered in diagnosing catatonia in this case. Firstly, his excitatory symptoms were mild and varied throughout the day. In this case, a longer period of detailed observation as an inpatient helped. He also has a history of making up symptoms to gain attention and later on admitted them to be false.

The treatment of catatonia is well established in the literature, consisting initially of benzodiazepines and/or ECT treatment.[6, 7] According to a review by Fink et al., the efficacy of using lorazepam alone in treating catatonia ranged from 80-100% in 4 studies.[6] A "trial" dose of lorazepam is usually given once catatonia is suspected. A low dose of 3mg/d is initially started. This is then titrated up as required, sometimes as high as 20-30mg/d. In this case, a dose of 1 mg tds was sufficient. ECT is reserved as second line treatment, with a reportedly high success rate ranging from 82%-96% in 5 different studies.[6] Recently, numerous case reports have also reported success in using NMDAR antibodies, amantadine or memantine, for patients refractory to both benzodiazepine and ECT treatment.[8] However, there are currently no large studies regarding this as of yet.[8] Importantly, the use of antipsychotics in catatonic patients is cautioned, as its use increases the risk of developing neuroleptic malignant syndrome.[7]

The underlying cause for catatonia should be sought and treated as part of the management process. This can be difficult as there are many reported causes of catatonia, broadly classified into neurological, psychiatric and medical. [9] More than one potential cause could be identified or none at all, in idopathic cases. [10] Thus, it is important to appreciate that catatonia is a syndrome not just restricted to psychiatric patients and medical conditions should always be considered, regardless of existing psychiatric conditions, such as in this case. [5] Important conditions to consider include epilepsy, encephalitis, cerebrovascular infarction or a mass lesion.[9] Endocrine abnormalities, electrolyte imbalances and traumatic brain injury can also present with catatonia.[5] All assessments should include a thorough history and physical examination, full blood count, urea and creatinine levels, liver function tests, thyroid function tests, urine drug screen, urinalysis, EEG and MRI brain scan. Other suggested tests if indicated, include vitamin B12 levels, folate, serum iron, HIV/AIDS serology, lumbar puncture and workup for systemic lupus erythematosus. [11]

It is most often difficult to pinpoint the exact cause of catatonia. In this case, it was felt that the possible aetiologies were the underlying ischaemic changes in both frontal lobes and his preexisting diagnosis of schizophrenia. However, he had not experienced positive psychotic symptoms for the past 3 years and is currently doing well without any antipsychotic medications. Thus, the accuracy of his previous diagnosis of schizophrenia made in the 1980s is now pondered upon as he is currently maintaining well without any antipsychotic medications.

Conclusion

Through this case report, we highlight the importance of recognizing and managing catatonia early. It is not limited to psychiatric patients and other underlying causes should be sought. This case has also shown the BFCRS to be helpful and accurate in screening for catatonia. A low dose of benzodiazepine alone was shown to be effective in resolving all catatonic symptoms in this patient.

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