Brief report

A case series of catatonia: Lessons to learn

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Summary

Catatonia is a neuropsychiatric syndrome which is associated with a diverse range of psychiatric and medical disorders. Current nosological approach supports the view that catatonia is a separate clinical entity. We present a case series of patients with clear catatonic symptoms associated with or as a result of vitamin B12 and folic acid deficiency, hyponatreamia, Systemic Lupus Erythromatosis (SLE) and viral encephalitis, highlighting the importance of being aware of medical causes of catatonia. The patients with B12 and folic acid deficiency and hyponatreamia made a dramatic recovery after correction of the underlying cause, and the patient with SLE and viral encephalitis also showed a good clinical response following appropriate treatment. Early identification of underlying cause and initiation of treatment facilitates a better outcome in patients with catatonia.

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Introduction

Catatonia can be described as a neuropsychiatric syndrome, and its common clinical features are mutism, stupor, refusal to eat or drink, posturing and excitement or hypokinesia (1). Catatonia was described and introduced by Kahl Kahlbaum in 1874. Subsequently Emil Kreapalin included catatonia as a subcategory of dementia praecox and later considered it as a psychiatric condition often associated with schizophrenia (2). For more than a century catatonia was regarded as a subtype of schizophrenia. The DSM-5 does not recognise catatonia as an independent disorder, but identifies it as a specifier of other mental disorders or includes it under the 'unspecified' category for example, schizophrenia co-morbid with catatonia (3). The underlying aetiological factors for the syndrome are many and are commonly associated with mood disorders (4). Catatonia is also associated with many medical and neurological conditions (1,5,7). Prospective studies have demonstrated that catatonia in psychiatry is due to medical conditions in 20%-25% of presentations (6). Neuro-psychiatric conditions such as delirious mania, neuroleptic malignant syndrome, autistic spectrum disorders, anti-NMDA receptor encephalitis are also associated with catatonia (7).

Identification of the underlying organic cause, if any, should be a priority in the management of catatonia. Prompt treatment in the early phases of catatonia is crucial to obtain a lasting abatement of symptoms. We present an assortment of cases to highlight the organic causation of catatonia and their management, and the importance of early recognition of such cases.

Case 1

A 46-year old housewife presented with suspiciousness, irritability followed by gradual withdrawal from household activities for 6 months duration. Eventually she became mute and showed posturing. There was no past history of significant medical or psychiatric conditions. Mental state examination revealed evidence of self-neglect and waxy flexibility in addition to mutism. Other than mild pallor she was physically normal. Her haemoglobin level was 9.6g/dl with increased mean corpuscular volume (MCV). Her blood picture was very compatible with folate and vitamin B12 deficiency. All the other investigations including liver function tests, renal function tests, serum electrolytes, C-reactive protein, thyroid function tests and computed tomography (CI) of the brain were normal. She did not respond to an array of treatment modalities such as an adequate course of risperidone, lorazepam, electro-convulsive therapy and chemical abreaction. However, she showed a marked clinical and functional improvement within 2 weeks after treatment with hydroxycobalamine 1 mg intra muscularly on alternative days for 10 days and folic acid 5 mg daily. Her haemoglobin level improved (12.5 mg/dl) and blood picture became normal in subsequent tests. However, we could not measure red cell folate and serum B12 levels due to practical difficulties. She has remained well for nearly four years up to now, without any psychotropic medication.

Case 2

A married male in his fifties had been commenced on fluoxetine for a depressive disorder. Two weeks later he presented with mutism and posturing. He was afebrile and the physical examination was normal apart from increased muscle tone with passive movements. His serum sodium level was 112 meq/l and potassium level was 4.1 meq/l, repeated serum sodium level remained low at 117meq/l. The rest of the investigations including liver function tests, serum creatinine, full blood count, chest x-ray, thyroid function tests were within normal limits. The reason for hyponatraemia was thought to be due to fluoxetine and it was discontinued. Catatonic

symptoms rapidly resolved after correction of hyponatraemia. Subsequently he was commenced on mirtazapine and the sodium levels remained normal.

Case 3

A seventeen year old school girl had been referred from a medical ward due to sudden onset mutism. She had fever for three days and then rapidly developed catatonic symptoms such as posturing and negativism. She developed a seizure on the day of admission to the psychiatry unit. She was febrile but the rest of the physical examination was normal except for catatonic symptoms. Electroencephalography (EEG) showed slow waves suggestive of encephalopathy and findings of the computerized tomography (CT) of the brain were also compatible with this. Cerebrospinal fluid analysis was indicative of viral encephalitis.

She was commenced on intravenous acyclovir and antiepileptic medication (sodium valproate). She made a slow but complete recovery.

Case 4

A sixteen year old school girl was referred with behavioral changes suggestive of schizophrenia. She was smiling to self and had been withdrawn from social activities over the previous three months. She also exhibited negativism and waxy flexibility. Her physical examination was normal except for a facial rash and catatonic features. She had been treated with risperidone at the local hospital with poor response.

Investigations revealed a high ESR of 118 mm/1st hour which remained high on subsequent measurements at 120 mm/ 1st hour. Double stranded DNA was positive, and CT of the brain and EEG both supported the diagnosis of cerebral lupus. Her catatonic symptoms improved after adding prednisolone to the treatment regime.

Discussion

Catatonia is a syndrome which cuts across several psychiatric diagnoses. Several medical, neurological and metabolic disorders are also associated with or aetiologically related to the clinical syndrome of catatonia (1,5,7). Pernicious anaemia is known to be associated with psychiatric conditions such as mood disorder, psychosis and dementia (8). An incidental finding of pernicious anaemia in a patient with catatonia has been reported (9). Vitamin B12 deficiency as a causative factor of catatonic symptoms has also been described in a case study where complete remission of symptoms was achieved following B12 replacement (10). Our first case supports the view that there is an association between catatonia and vitamin B12 /folic acid deficiency.

A case of catatonic symptoms related to hyponatreamia and rapid resolution of symptoms after correction of hyponatraemia has been reported (11). Our second patient's catatonic symptoms are likely to have been due to hyponatraemia which improved rapidly with the correction of sodium levels. The importance of performing serum electrolytes in such patients is highlighted here. Viral encephalitis and meningitis are relatively common conditions in countries like Sri Lanka. The presentation may vary from mild symptoms such as headaches to severe presentation such as delirium and seizures. It has been shown that neuropsychiatric manifestations are common during the acute phase of viral encephalitis, which needs to be considered in the differential diagnosis of patients who present with behavioural changes in emergency settings (12). Viral aetiology of psychosis has been hypothesised for many years. Our third patient developed catatonic symptoms following viral encephalitis and catatonic symptoms resolved following anti-viral treatment. This case illustrates the importance of considering the possibility of central nervous system infections in the assessment of catatonia.

Patients with systemic lupus erythematosus (SLE) may commonly present with psychiatric and neurological symptoms. Although rarely seen, patients with SLE presenting with catatonia have been previously reported (13). Our fourth patient presented with catatonic symptoms following cerebral lupus, and the catatonic symptoms improved with steroids. This case again illustrates the importance of excluding autoimmune disorders such as SLE and the need to perform relevant investigations in the assessment of catatonia, particularly in young female patients.

In conclusion, although many catatonic presentations are due to conditions such as mood disorders, it is mandatory to look for organic causes, as early detection and treatment of them will certainly improve the prognosis. It is noteworthy that our case series also supports the concept that catatonia is a neuropsychiatric syndrome as Kahlbhaum originally described.

Conflict of interest

None declared

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