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CATATONIA- A CLINICAL IMPOSTER

Amit Chail, Hapreet Singh, Sargundeep Singh* and Bharat Khadka

Department of Psychiatry, Armed Forces Medical College, Pune.

***Corresponding Author: Sargundeep Singh**
Department of Psychiatry, Armed Forces Medical College, Pune.

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ABSTRACT
Catatonia is a neuro-behavioural syndrome with autonomic, behavioral and motoric manifestation accompanying many medical and neurologic disorders.[1] It was first described in Karl ludwig Kahalbum in 1874 in his book Die Katatonie oder das Spannungsirresein.[2] During the early part of 20th century, it was considered a type of schizophrenia. However in the 1970s, it begin to be identified with many other disorders like mania and depression, as a toxic response and in general medical and neurologic illnesses.[3] Presently in ICD-10 it continues to be a part of Schizophrenai but in DSM-V it has been made a separate entity.[4] The draft version of ICD 11 also places it as a separate entity.[5] Two subtypes of the syndrome have been identified i.e retarded type and excited type.[6] Medical complications of the syndrome include aspiration, dehydration, pulmonary emboli, acute renal failure, cardiac arrest and death.[7] There are various conditions which mimics catatonia like Non-catatonic stupor, Encephalopathy, Stroke, Stiff-Person syndrome, Locked-in syndrome, Malignant hyperthermia, Status epilepticus etc.[8] Becuase of being not so common presentation, it is often confused with other diseases, so here we present a 21 years old student, who presented with two episodes of unresponsiveness and abnormal behaviour, initially was being managed as a case of Seizure disorder and Ictal Psychosis, however subsequent ward observation revealed otherwise.

KEYWORD: Catatonia, Psychogenic non epileptic seizures, Catatonia mimics, Bipolar Affective Disorder, Echopraxia, Stupor.

INTRODUCTION
No other disease entity in Psychiatry has undergone such transitions in conceptual and contextual understanding as Catatonia. The term "Catatonia" was coined by Karl Kahlbaum, who described it as a distinct disease entity. This viewpoint was reformulated by Emil Kraepelin who subsumed Catatonia as a subtype of dementia praecox.[9] Various authors identified different forms of catatonia based on presentation or etiology like hypokinetic, agitated, cyclic, chronic, malignant, lethal and organic or drug induced.[6,8] There are various conditions which mimics catatonia.[8] Various models such as motor circuitry model, Neurotransmitter model (implicating role of GABA), Epilepsy model, Endocrine model, Genetic model and immune model have been proposed, however the etio-pathogenesis remains elusive.[10] The excited form is relatively rare manifestation and clinically difficult to identity.[6] Adequate and timely treatment with benzodiazepines and other modalities including judicious use of Electro-convulsive therapy and anti-psychotics remain crucial to successful remission of symptoms.[11,12]

CASE REPORT
21 years old, student, studying in graduation second year, with no past or family history of psychiatry illness was brought by family members, with two episodes of unresponsiveness, staring in space, frothing from mouth. The family members also gave history that in one of the episodes he had involuntary voiding of urine. The episodes lasted for around two minutes each and occurred within a span of 4 hours. In addition the family members also reported that since three days patient was irritable and excessively religious.

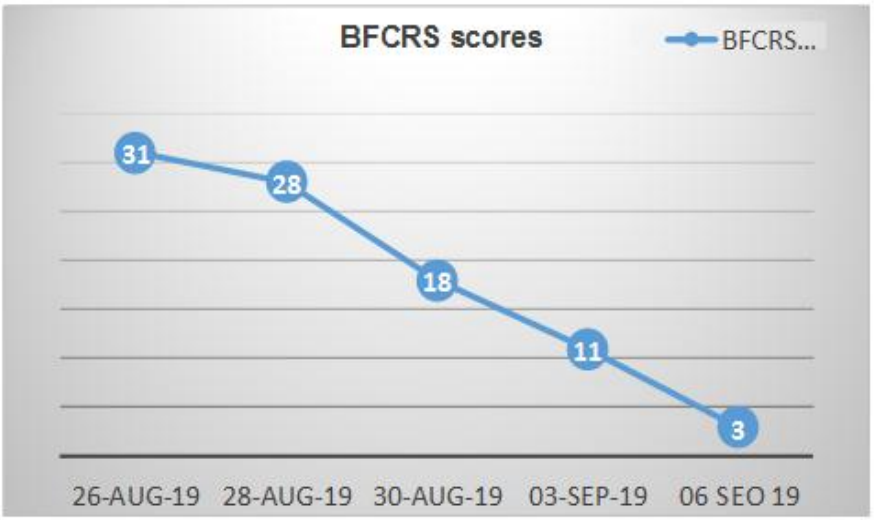
On evaluation, his vitals were Pulse- 70/min, B.P.- 132/82mm Hg. He was afebrile, other general Physical and systemic examination was essentially normal. Relevant hematological, biochemical Investigations were WNL. Patient was admitted with a provisional diagnosis of Seizure and possibility of Ictal psychosis with a differential diagnosis of Psychogenic Non-Epileptic Seizures (**PNES**).

On the second day of admission, he again had an episode of staring. CSF analysis, MRI Brain and EEG were normal. He was kept on close observation. In the absence of his clinical profile being of any specific medical

presentation, a psychiatric call was placed. Initial Mental Status Examination revealed an ill-kempt, unshaven, disinhibited individual with adequate eye contact. He was fidgety during the interview. Speech was increased in rate, tone & volume. He described his mood as "Bahut Badiya hai" with his affect fluctuating from euphoric to dysphoric. Thinking was increased in stream with delusions of grandeur present. There was no perceptual abnormality in a clear sensorium with disturbed bio-drives in form of reduced need for sleep, increased energy and reduced appetite.

The history was re-visited and on detailed history from family members revealed that since the past three weeks he was found to be more cheerful than usual and was noted to have increased confidence and reduced need for sleep. This progressed over 2-3 weeks, where in his behaviour had become disinhibited and he started performing religious rituals at odd hours. Clinical observation in Psychiatric ward and a high index of suspicion revealed other catatonic features in the form of stupor, mutism, posturing, echolalia and echopraxia.

A diagnosis pf Catatonia associated with Bipolar Disorder-I was made. His score on Bush-Francis Catatonia Rating Scale (BFCRS) was 31. In the background of provisional diagnosis of Affective Catatonia, he was started on Inj Lorazepam 4 mg TDS. Following the first dose of Lorazepam, he had a significant improvement, however the same was ill sustained. Injection Lorazepam was increased to 16 mg/day on the next day. His BFCRS score fluctuated with maximum of 22 and minimum of 8 (ill-sustained) by day 4 of admission. He persisted to have fluctuating and residual symptoms. So, additionally he was also given of 4 ECTs over next two weeks. After the second ECT, we added Olanzapine 10 mg Hs and Tab Lithium 300mg (1-0-2). He showed good response to the treatment within about ten days (03 ECTs) his catatonic features had reduced and his BFCRS score was 3. Lorazepam was tapered over two weeks.



DISCUSSION

Catatonia remains a commonly encountered clinical problem with high potential for medical co-morbidity. Signs of catatonia occur in up to 18% of psychiatric inpatients, 20% of patients with mania, 17% of adults with autism-spectrum disorders, and 30% of patients with delirium.[7] The pathobiology of catatonia is poorly understood, although abnormalities in gamma-aminobutyric acid and glutamate signaling have been suggested as causative factors. Because catatonia is common, highly treatable, and associated with significant morbidity and mortality if left untreated, physicians should maintain a high level of suspicion for this complex clinical syndrome.[6] Complete history with a thorough semiology analysis of patient symptoms and further complementary investigation are always necessary.[13] Quick application of diagnostic scales and a lorazepam challenge test should be performed to avoid delaying the diagnosis.[14] Our patient presented with semiology suggestive of an organic illness with possibility of a seizure being considered first. The affective especially manic stupor being a rare presentation was difficult to diagnose at the first instance. Detailed neurological evaluation including neuroimaging, EEG and CSF examination ruled out the possibility of an organic etiology. As seen in the index case above a high index of suspicion and prompt treatment helped to prevent any further complications.

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