Clinical Profile, Course, and Outcome of Secondary Catatonia: A Case Series

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Catatonia is a complex neuropsychiatric syndrome characterized by a cluster of motor abnormalities associated with disturbances in mood, behavior, and thought. Its nosological status had undergone a paradigm shift, from Kraepelin-Bleuler's view as a subtype of schizophrenia to the current approach of considering it a dimension in various psychotic, affective, and general medical disorders.[1] A broad range of symptoms and signs have been described in catatonia, based on which excited and retarded subtypes have been defined. The immobility and poor oral intake seen in the retarded subtype and the severe psychomotor agitation seen in the excited subtype can progress to severe life-threatening complications, thereby underscoring the need for rapid diagnosis and treatment. Secondary catatonia or catatonic disorder due to a general medical condition has been reported with various neurological and medical disorders.[2,3] However, inadequate and inconsistent description of signs of catatonia, lack of controlled data, and inadequate description of temporal association between the putative medical etiology and the catatonic syndrome are some limitations of earlier reports making any interpretation of the causal relationships arduous. It remains underdiagnosed and inadequately treated in general hospital settings, emphasizing the crucial role of consultation-liaison psychiatrists. We report the clinical presentation, course, and treatment outcomes in patients presenting with secondary catatonia in a general hospital setting.

Methods

This case series is from a tertiary care hospital with a general hospital psychiatry unit. All patients diagnosed with secondary catatonia (catatonic disorder due to a general medical condition) as per the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5), between January 2016 and December 2021, were included. Sociodemographic details, clinical features, course of the symptoms, medical co-morbidities, biochemical investigations, electroencephalogram and neuroimaging findings, and treatment details were extracted through a retrospective chart review. All patients presenting with catatonic symptoms were screened and assessed for the severity using Bush Francis Catatonia Rating Scale (BFCRS) as a part of the standard care. This case series was exempt from review by the Institutional Ethics Committee because the Committee does not require review for case series involving <10 cases.

Results

We found seven patients (three male and four female), with a mean age of 30.1 years (SD 8.4), diagnosed with secondary catatonia (**Table 1**). All patients had the retarded subtype. Mutism was the commonest symptom and was present in all the patients, followed by staring in six and posturing and withdrawal in four. The median duration of catatonic symptoms was 10 days (range: 1–90 days). The mean score on BFCRS at presentation was 15.6 (SD 6.4) (the maximum possible

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TABLE 1.

The Clinical Profile and Treatment Details of Patients with Organic Catatonia[a].

Sl. No.	Age, Sex	Medical Morbidities, Duration	Past Psychiatric Illness	Catatonic Symptoms	Features of Delirium	Duration of Catatonic Symptoms (days)	EEG Findings	MRI (brain)	Treatment
1	18/F	Enterobacteriaceae encephalitis 4 weeks	Nil	Staring, waxy flexibility, posturing, mutism, withdrawal	Yes	30	Diffuse slowing, maximal over left fronto-central region	Normal	Oral Lorazepam 8 mg/day
2	24/M	Post-dengue recurrent fever and arthritis 1 month	Nil	Mutism, posturing, waxy flexibility, immobility, staring, negativism	No	1	Not done	Normal	Oral Lorazepam 10 mg/day
3	28/M	Localization-related epilepsy 2 weeks	ADS	Mutism, posturing, staring	No	10	Diffuse, intermittent slowing without epileptiform discharges	Normal	Oral Lorazepam 12 mg/day
4	38/M	MRSA sepsis, EJV thrombosis, T2 DM 2 weeks	ADS, TDS	Immobility, mutism, staring, posturing, echolalia, negativism, waxy flexibility, excitement, mitgehen, gegenhalten, grasp reflex	Yes	90	Mild, diffuse polymorphic slowing without epileptiform abnormalities	Normal	Oral Lorazepam 8 mg/day
5	41/F	? Autoimmune encephalitis, RHD, Hypothyroidism 1 month	ATPD in the past	Immobility, mutism, staring, negativism, rigidity, gegenhalten, grasp reflex, withdrawal, ambitendency	No	12	Diffuse slowing in the theta range and bilateral delta slowing in the frontal region	Chronic infarct over the posterior aspect of right putamen. Essentially normal	Oral Lorazepam 6mg/day ECT
6	26/F	SLE, Anemia? Steroid-induced catatonia 1 week	Nil	Mutism, immobility, staring, rigidity, ambitendency	Yes	1	Mild generalized slowing	Non-specific white matter changes in centrum ovale and bilateral frontal and right parietal regions	Intravenous Lorazepam 4mg/day
7	36/F	SLE, Libman Sacks endocarditis 4 years	Nil	Mutism, staring, immobility, withdrawal, mitgehen	No	10	Generalized beta activity (lorazepam-induced)	Chronic infarct in the right posterior cerebellar hemisphere, T2/FLAIR hyperintensities in bilateral periventricular deep white matter	Oral Lorazepam 10mg/day ECT

[a] ADS, alcohol dependence syndrome; ATPD, acute transient psychotic disorder; ECT, electroconvulsive therapy; EJV, external jugular vein; MRSA, methicillin-resistant *Staphylococcus aureus*; RHD, rheumatic heart disease; SLE, Systemic Lupus Erythematosus; T2 DM: Type 2 Diabetes Mellitus; TDS, tobacco dependence syndrome.

score is 69). Five patients had a continuous course of catatonic symptoms, and the remaining two had a fluctuating course. Three concurrently met the criteria for delirium. Two had systemic lupus erythematosus (SLE), two had encephalitis, and the other etiologies included sepsis, epilepsy, and post-dengue sequelae. Four patients had no structural abnormality in the MRI brain scans. Generalized slowing of electrical activity was the commonest EEG finding.

All patients were initially treated with lorazepam, with a mean dose of 8 mg (SD 3.2) daily. Four responded to

lorazepam within a day, and one had a gradual response. Two patients who did not respond to lorazepam were subsequently administered electroconvulsive therapy (ECT). Of them, one responded after ten sessions of ECT; however, she developed congestive cardiac failure after the 11th ECT session and required prolonged intensive care for a month, following which she recovered. The other patient developed delirium after the second ECT and subsequently had a cardiac arrest and required prolonged intensive care.

Discussion

In our center, we found seven cases of secondary catatonia over a five-year period. In general hospital settings, 20%–40% of patients with catatonic features have underlying organic etiology, which is often underdiagnosed and undertreated.[2,4] Etiologies include neurological conditions such as encephalitis, epilepsy, autoimmune encephalopathy, cerebrovascular events, and general medical conditions such as endocrine disorders, metabolic derangements, nutritional deficiencies, and so on.[2–5] Features that should raise the suspicion of underlying organic etiology in catatonic patients include acute presentation, absence of past psychiatric history, presence of autonomic instability, and poor or fluctuating response to lorazepam.[2,6]

The symptom profile of our patients is comparable to the earlier reports on secondary catatonia, with mutism being the commonest symptom.[4] However, another study[6] comparing the symptom profile and severity of catatonia due to affective, psychotic, or organic conditions found no significant difference across the groups. Moreover, mutism was also the most commonest and one of the first symptoms to appear with diverse causes of catatonia.[6] We found that delirium was a concurrent presentation with catatonia in three patients. Even though clear consciousness is considered a prerequisite for diagnosing catatonia as per DSM 5, patients with delirium frequently have catatonic symptoms. The need for concurrent diagnosis of these two conditions in the current nosological systems is debated.[7] If catatonia patients have delirium too, one should suspect that an underlying medical illness may be the cause, although further research is needed regarding this relationship. EEG findings of generalized theta to delta range slowing and occasional superimposed focal slowing in frontal/temporal lobes have been commonly reported in organic catatonia,[8] which are comparable to our findings. Increased likelihood of EEG abnormalities and the presence of these abnormalities during the early course have been considered potential indicators of underlying organic etiology in patients with catatonia.[5,8] Diffuse white matter changes and focal abnormalities in the frontal lobe/basal ganglia have been reported in the structural brain imaging studies of catatonia.[5,9] However, in structural imaging, we found no focal abnormality in our patients. Recognition of underlying causes is crucial in managing secondary catatonia.

The use of benzodiazepines is challenging in such situations, owing to variable responses and the concomitant use of medications for the underlying medical condition. ECT is considered an effective treatment option if there is a poor response to benzodiazepines; however, the risk of complications with ECT is a concern, as seen in our cases. A risk-benefit analysis should be carried out to weigh the possibility of complications of ECT due to concomitant general medical conditions versus the potentially life-threatening complications associated with catatonia.

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