

## Catatonia: extinct, lost, or forgotten?

Forouzan Elyasi\*

Department of Psychiatry, Psychiatry and Behavioral Sciences Research Center, School of Medicine, Mazandaran University of Medical Sciences, Sari, Iran



### Dear Editor

Catatonia is a neuropsychiatric syndrome that occurs in some primary psychiatric disorders (e.g., schizophrenia, mood disorders), or due to general medical conditions (e.g., neurological disorders, drug poisoning, metabolic disorders) (1). Although it is uncommon, but if it goes unrecognized in medical and surgical units (2), it can increase morbidity and mortality. Moreover, making a connection between signs observed across different systems (the motor, somatic, and psychiatric symptoms) could lead to misdiagnosis and a delay in treatment (3).

Our patient was a 48-year-old widow with a high school diploma, unemployed, and was from one of the northern cities of Iran. She had refused to talk and eat for the last 2 months prior to admission. She was brought to a psychiatric hospital by family members in September 2014. She complained about her weight loss, poor health, and inappropriate physical condition. In the referral letter, the psychiatry resident had noted that the patient was unconscious and did not make eye contact or verbal communication. Therefore, she was referred to the university general hospital for an investigation of her loss of consciousness. Following admission, patient's family left the hospital without notice or explanation. She was visited by emergency residents and physicians and was described as having loss of consciousness as well as lacking in verbal communication during physical examination. After initial laboratory tests, she underwent consultations with infectious diseases, internal medicine, and neurological specialists in order to understand the decreased level of consciousness. The results of the initial tests were as follows (Table 1):

The neurologist had noted that the patient had a history of major depressive disorders and was currently not conscious; she had rigidity in all limbs with the contraction lining of the bedridden. Rigidity was not reliable in the

examinations and the patient had a fever. The neurologist had also reported that a diagnosis of neuroleptic malignant syndrome according to CPK 354 IU/L was unlikely, and it was advised that the patient should have a sepsis work-up, be assessed for meningitis, undergo a brain magnetic resonance imaging scan, and have a consultation with infectious diseases specialists. In accordance with the normal CSF analysis, the infectious diseases specialists advised an assessment for neuroleptic malignant syndrome and a psychiatric consultation. In the initial psychiatric consultation which was conducted by the psychiatric resident, CPK, lactate dehydrogenase, and microglobulinuria tests were recommended to exclude neuroleptic malignant syndrome, and the only clinical feature mentioned was loss of consciousness. In a psychological consultation that we carried out several hours later by a consultation-liaison psychiatrist, we found that the patient had cachexia, bruxism, negativism, rigidity, gegenhalten, and stereotypic movements around the mouth. She also put her left hand in a particular position. Investigation of documents revealed that the patient had stopped taking a clozapine and fluphenazine decanoate injection 2 months in advance; hence the diagnosis of neuroleptic malignant syndrome was rejected. It was also found that she had been hospitalized three times, in 2005, 2008, and 2012, with a diagnosis of schizophrenia according to DSM-V criteria. Therefore, an early diagnosis of schizophrenia was proposed, but due to her failure to eat and severe malnutrition, a consultation with an oncologist was requested to exclude vitamin K deficiency and paraneoplastic syndromes as possible

**Table 1.** The results of the initial tests

		Unit
White blood cell count	11.1	10 <sup>3</sup> /uL
Hemoglobin	12/4	mg/dL
Platelets	228	10 <sup>3</sup> /uL
Glucose	96	mg/dL
Creatinine	1.3	mg/dL
Urea	104	mg/dL
Alkaline phosphatase	133	U/L
CPK	354	IU/L
Serum glutamic oxaloacetic transaminase	130	U/L
Serum glutamic pyruvic transaminase	78	U/L
CSF	Normal	

Abbreviations: CPK, creatinine phosphokinase; CSF, cerebrospinal fluid analysis.

**Received:** 26 December 2015; **Accepted:** 10 February 2016;

**Published online:** 29 August 2016

\***Corresponding author:** Forouzan Elyasi; Email: Forouzan.el@gmail.com

**Competing interests:** Author declares that she has no competing interests.

**Funding information:** There is none to be declared.

**Citation:** Elyasi F. Catatonia: extinct, lost, or forgotten? *Journal of Emergency Practice and Trauma* 2017; 3(2): 38-38. doi: 10.15171/jept.2016.02.



causes of her catatonia. The patient was transferred to the psychosomatic ward and after the rejection of medical causes as being responsible for catatonia; the patient received electroconvulsive therapy (ECT), with the written consent of her father, along with supportive therapy, such as total parenteral nutrition. The patient showed a favorable response to ECT and came out of catatonia after six sessions.

The described patient was brought into the hospital as having catatonic signs arising from a primary psychiatric disorder. The patient was examined by four physicians specializing in other medical disciplines as well as six residents in emergency medicine, neurology, internal medicine, infectious diseases, anesthesia, and psychiatry before being visited by a consultation-liaison psychiatrist. The diagnostic and therapeutic approach to the patient was such as a comatose patient. Catatonia is diagnosed using motor signs including negativism, immobility, stupor, posturing, waxy flexibility, mutism, abnormal movements, echopraxia, echolalia, and stereotypic movements (3). It appears that a large number of physicians do not have the experience required to identify and apply the terminology describing catatonia (4), and some even believe that catatonia no longer exists. Subsequently, these physicians do not recognize and treat this syndrome (5). If physicians rely on a large number of abbreviations according to the definitions in the references, detection of catatonia will improve (6). Undiagnosed catatonia can increase morbidity and mortality which demonstrates the need for effective screening of patients for its presence. Diagnosis can be reduced due to the lack of familiarity with catato-

nia in modern medicine. It is recommended that catatonia be included as an important and indispensable aspect of training for psychiatry and emergency medicine residents in Iran. There is also a need for training in other medical fields including internal medicine, neurology and infectious diseases. In addition, the presence of consultation-liaison psychiatrists in general hospitals may aid in the correct handling of catatonia.

#### **Ethical issues**

Not applicable.

#### **Author's contribution**

FE is the single author of the manuscript.

#### **References**

1. Fink M, Taylor MA. Catatonia: subtype or syndrome in DSM? *Am J Psychiatry* 2006; 163(11): 1875-76.
2. Carroll BT, Spetie L. Catatonia on the Catatonia on the consultation-liaison service: a replication study. *Int J Psychiatry Med* 1994; 24(4): 329-37.
3. Sadock BJ, Sadock VA, Ruiz P. Kaplan and Sadock's Synopsis of Psychiatry: Behavioral Sciences/Clinical Psychiatry. 11th ed. LWW; 2015. p. 343-6.
4. Kirkhart R, Ahuja N, Lee JW, Ramirez J, Talbert R, Faiz K, et al. The Detection and Measurement of Catatonia. *Psychiatry (Edgmont)* 2007; 4(9): 52-6.
5. van der Heijden FM, Tuinier S, Arts NJ, Hoogendoorn ML, Kahn RS, Verhoeven WM. Catatonia: disappeared or under-diagnosed? *Psychopathology* 2005; 38(1): 3-8.
6. Stompe T, Ritter K, Schanda H. Catatonia as a subtype of schizophrenia. *Psychiatr Ann* 2007; 37(1): 31-6.