Scholars Journal of Applied Medical Sciences
Abbreviated Key Title: Sch J App Med Sci
ISSN 2347-954X (Print) | ISSN 2320-6691 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Medicine and Pharmacy

Catatonia: Development of a Neuropsychiatric Entanglement through a Clinical Case

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DOI: 10.36347/sjams.2022.v10i10.039 | **Received:** 05.09.2022 | **Accepted:** 13.10.2022 | **Published:** 30.10.2022

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Abstract **Case Report**

Catatonia is a transnosographic and potentially fatal syndrome, most often associated with mood disorders or schizophrenia, but can also develop in autistic disorders, dementia, as well as in general medical conditions such as epilepsy, autoimmune encephalitis, hypercalcemia, hepatic encephalopathy, or diabetic ketoacidosis. The work we present is based on a clinical case of a patient with schizophrenia presenting a catatonic syndrome, of which a neurological cause was first evoked but after clinical investigations the diagnosis of schizophrenia was retained and currently the patient is stabilized on Clozapine. It is imperative to recognize a catatonic syndrome in order to treat it quickly, as some of the etiologies that cause this syndrome and the consequences of the syndrome itself can be life-threatening.

Keywords: Catatonia, schizophrenia, catatonic syndrome.

INTRODUCTION

Catatonia is a common yet misunderstood clinical syndrome. Linked to schizophrenia by Emil Kraepelin and then by Eugen Bleuler, it was not until the middle of the 19th century that catatonia was recognized in connection with other etiologies, in particular somatic affections [1]. Catatonia would correspond at the cerebral level to a dysfunction of the circuits involved in voluntary movement, in reaction to fear, most often in stuporous form but sometimes in furious form. The lateral orbitofrontal cortex, which is defective in catatonia, and which has GABA-A receptors, is strongly involved in this primitive reaction, which it inhibits by interaction with the medial orbitofrontal cortex and the amygdala [1].

Indeed, although reversible, its prognosis in case of delay in the implementation of an adapted treatment can become very severe, with a vital prognosis and risk of death by cardiovascular shock, dehydration/denutrition syndrome, acute respiratory distress syndrome, or even complications of decubitus with prolonged immobility, risk of pulmonary thrombosis and embolism, and urinary tract infections [2].

The major challenge of effective diagnostic and therapeutic management is therefore indisputable. The catatonic syndrome is transdiagnostic, which means that it can be found in the course of several psychiatric or non-psychiatric pathologies. The main non-psychiatric etiologies of catatonic syndrome are essentially neurological causes.

In this work, we will first see the semiology of the catatonic syndrome, in order to understand it and then the etiologies to which this syndrome can lead mainly neurological will be approached.

CASE PRESENTATION

We present the case of a patient named JK, 19 years old, without any psychiatric history, who presented since 02 months a brutal change of behavior contrasting with his previous state made of social withdrawal, tendency to isolation, tiredness, refusal to go out or to meet people, spending hours in front of the television with verbalization of remarks of persecution towards his friends with a school disinvestment. The evolution was marked quickly by the appearance of somatic complaints made essentially of headaches, he would have become more and more irritable, then the patient started to become clinophilic, not speaking any

more, keeping the lying position, immobile during hours, not trying to eat, it is thus that he became dependent on the others not even assuring his intimate toilet without the assistance of his family, which motivated to consult and a hospitalization in neurology.

In front of this catatonic syndrome, the diagnosis of a viral encephalopathy was evoked. A complete workup was done including a brain CT, a brain MRI, an angio-MRI, an EEG, and a lumbar puncture with a blood test including HIV serology, TPHA, VDRL, a thyroid workup, and a liver workup which came back without any particularities.

Given the persistence of the catatonic syndrome and the elimination of a neurological cause, the patient was admitted to a psychiatric hospital where the diagnosis of schizophrenia was retained. After the use of several antipsychotic molecules, the patient stabilized on Clozapine 400mg per day.

DISCUSSION

Catatonia is a syndrome composed of 12 psychomotor signs in the DSM-5. There are also other "classic" signs found in standardized evaluation scales for catatonic syndrome. The semiology of the catatonic syndrome results in fact from an interaction between three groups of signs and symptoms, motor signs marked by difficulties in starting and stopping a movement as in catalepsy, mutism and stereotypies; behavioral signs giving an impression of dependence on the environment, as in echo-phenomena, use or magnetization behaviors and neurovegetative signs. The DSM-5 defines catatonic syndrome by the presence of 3 of the 12 signs described (Table 1). The DSM-5 does not treat catatonia as an independent class but recognizes it as a specification of a disorder: catatonia may be associated with a psychiatric disorder, or due to another medical condition.

Tableau I
DSM-5.

Catatonie associée à un autre trouble mental (spécification « de type catatonique »)/dû à une autre affection médicale
1. Catalepsie : maintien contre la gravité de postures imposées par l'examinateur
2. Flexibilité cireuse : résistance légère et constante à la mobilisation passive exercée par l'examinateur
3. Stupeur : absence d'activité psychomotrice, pas d'interaction avec l'environnement
4. Agitation, non influencée par des stimuli externes
5. Mutisme : absence ou quasi-absence de réponse verbale (exclure si aphasie connue)
6. Négativisme : opposition ou absence de réponse à des instructions ou à des stimuli extérieurs
7. Prise de posture : maintien actif, contre la gravité, d'une posture adoptée spontanément
8. Maniérismes : caricatures d'actions ordinaires empreintes de bizarrerie, de solennité
9. Stéréotypies : mouvements non dirigés vers un but, répétitifs et anormalement fréquents
10. Expressions faciales grimaçantes
11. Écholalie : répétition des paroles de l'examinateur
12. Échopraxie : reproduction des mouvements de l'examinateur

Among psychiatric disorders, it is now accepted that the primary etiology of the catatonic syndrome is a mood disorder and within mood disorders, it is the manic episode that seems to be the most involved [3]. Other psychiatric disorders can cause a catatonic syndrome, such as neurodevelopmental disorders and even obsessive-compulsive disorder and schizophrenia. On the other hand, it is important to emphasize the close link between the catatonic syndrome and the use of neuroleptics and the neuroleptic malignant syndrome. Indeed, among the risk factors, the use of a neuroleptic treatment and a history of neuroleptic malignant syndrome can be noted [4]. Conversely, catatonia is a risk factor for the development of neuroleptic malignant syndrome and may, according to some authors, be a subtype of pharmacologically induced catatonia. Similarly, in the presence of catatonia, the initiation of neuroleptic treatment may increase the risk of neuroleptic malignant syndrome. The prevalence of catatonia is 9.2% according to a meta-analysis of 74 studies (107304 patients). In 75 to 80% of cases, catatonia is associated with a psychiatric cause. In adults, catatonia is mostly associated with bipolar disorder (20.1%

prevalence) and postpartum psychosis (20.0%). Catatonia is more rarely associated with autism spectrum disorder (11.1%) and schizophrenia (9.8%) [5].

The main non-psychiatric etiologies of catatonic syndrome are essentially neurological and metabolic causes. Regarding metabolic etiologies, it is important to look for hypercalcemia, hepatic encephalopathy, homocystinuria, or diabetic ketoacidosis. There is also a rare genetic form of catatonic syndrome, called "periodic catatonia". Autosomal dominant transmission, it is characterized by the sudden onset of cyclic catatonic episodes, more or less regular and separated by periods of remission, with both signs of stupor and excitement [2]. As far as neurological etiologies are concerned, epilepsy is very common, and a catatonic syndrome may reveal a non-convulsive malignant state, sometimes being its only clinical manifestation. Autoimmune encephalitis can also be the cause of catatonic syndrome, in particular anti-GAD antibody encephalitis, or anti-NMDA receptor antibody encephalitis. It is even estimated that anti-NMDA receptor encephalitis is accompanied in 70% of cases by a catatonic syndrome. Finally, the catatonic syndrome can occur in the context of a cerebral vascular accident, a cranial trauma, or during the evolution of a Parkinson's disease.

The main causes of catatonia are summarized in Table 2.

Principales causes de catatonie.

Psychiatriques	Systémiques	Neurologiques	Toxiques
Dépression	Métaboliques (dysnatrémie/calcémie/	Encéphalite à HSV1	Neuroleptiques
Manie	thyroïdie, hyperurémie, intoxication au CO)	Encéphalites limbiques (anti-NMDAR	Tacrolimus
Schizophrénie	Infections (Syphilis, VIH, typhoïde)	notamment)	Corticoïdes
Autisme, Trouble envahissant	Auto-immunes (Lupus érythémateux)	Épilepsies frontale et/ou temporale	Disulfiram
du développement	Métaux lourds (intoxications au Cuivre,	Sclérose en plaques	Sevrages médicamenteux
Catatonie périodique	au Plomb et au Manganèse)	Démences (Dégénérescence	(Benzodiazépines, Zolpidem)
	« Stress physique » (Œdème aigu du poumon,	frontotemporale et maladie à corps	Intoxication ou sevrage en
	transplantation hépatique, chirurgie lourde…)	de Lewy diffus)	stupéfiants (notamment opiacés,
		Maladies neurométaboliques	amphétamines et cocaïne)

Organic causes would be responsible for 20 to 30% of catatonia. It is important to remember that the clinical presentation of patients with catatonia of organic cause is strictly identical to that of catatonia of psychiatric cause. Therefore, especially in the absence of a known psychiatric history, as in the case of JK, it is necessary to follow a comprehensive approach to search for an organic cause. Catatonia is therefore an opportunity to combine the views of the psychiatrist and the neurologist.

The first step is clinical. We will particularly look for weight loss, which will point to a deficiency or tumor cause, the existence of risk factors for HIV infection, the exhaustive list of treatments taken with or without a medical prescription, and the use of drugs. Concerning Mr JK, the interrogation did not objectify a weight loss, or a drug intake, or a consumption of psychoactive substances. The paraclinical workup will include, in addition to a standard blood workup: thyroid hormones, vitamins B12 and folates, CPK, drug assays, HIV serology, martial workup, urinary toxicology, ECBU and autoimmune workup, in particular for lupus erythematosus. An electroencephalogram and brain MRI will also be performed. In Mr. JK, serologies, CT and EEG are without abnormalities in favor of a viral encephalopathy. It seems legitimate to systematically perform a lumbar puncture, not only to look for infectious encephalitis (especially HSV1), but also for limbic encephalitis (especially anti-NMDA). Our patient's lumbar puncture did not reveal any abnormalities.

How the infection might lead to catatonia is not described in the literature. Possibilities include direct neurotoxicity or a psychological reaction to the infection [6].

In a systematic review of 47 cases of organically induced catatonia, 45 were secondary to known neurotropic viruses, suggesting direct neurotoxicity. The immunological response may also be important, since in some neurological disorders, such as meningoencephalitis, damage is primarily caused by an immune response.

CONCLUSION

Catatonia remains a subject of research for centuries, the diagnosis is clinical, based on a set of criteria grouped in the DSM5 and its etiologies are psychiatric and organic including neurological. Rapid diagnostic and therapeutic management is essential to avoid life-threatening complications.

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