DOI: 10.4149/BLL_2023_113

Bratisl Med J 2023; 124 (10)
742 – 747

CLINICAL STUDY

Catatonia during COVID-19 pandemic – diagnostic and therapeutic approach

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ABSTRACT
Catatonia can be defined as an etiologically heterogeneous syndrome, with predominant psychomotor disturbances. Historically, the concept of catatonia has been associated with mental disorders, especially schizophrenia. However, nowadays our understanding of catatonia has evolved to recognize it as neuropsychiatric syndrome that can arise from diverse etiological factors ranging from neurological to systemic diseases. Furthermore, there is now a recognized association between catatonia and a broader spectrum of mental disorders. Catatonia as a secondary neuropsychiatric syndrome may be a clinical manifestation of COVID-19 also due to the known neuroinvasive potential of the SARS-CoV-2 virus or in connection with the overall somatic alteration of the patient. In clinical practice, co-infection with SARS-CoV-2 could impede the process of diagnosing and treating catatonia as the primary psychopathological syndrome. The administration of benzodiazepines and electroconvulsive therapy could endanger the patient's physical health with active COVID-19 infection. Management of catatonic syndrome associated with COVID-19 is a challenge and requires a comprehensive therapeutic approach. The article demonstrates the above-mentioned difficulties of treatment through two case presentations *(Tab. 2, Ref. 29)*. Text in PDF *www.elis.sk*
KEY WORDS: catatonia, COVID-19, SARS-CoV-2, neuropsychiatry, diagnosis, differential.

Introduction

Catatonia is a complex neuropsychiatric syndrome that has been associated with several mental disorders, most notably as a subtype of schizophrenia, but it is linked to a number of general medical conditions. Mean prevalence of catatonia is 9.2 % among subjects diagnosed with a variety of psychiatric or medical conditions. Catatonia occurs in patients with bipolar disorder, autism, schizophrenia, major depressive disorder or mixed psychiatric conditions (1). Most common general medical conditions related to catatonia are due to CNS-specific diseases. These include inflammation of the central nervous system (CNS), including infectious, autoimmune and paraneoplastic causes. Strokes and small vessel disease, brain tumors, epilepsy, neurodegenerative diseases, and metabolic derangements, while certain drugs have also been associated with catatonia (2). Even though catatonia is poorly understood, it is conceptualized as a disorder of cerebral motor network dysfunction (3) and reduced GABA activity, and glutamate abnormalities and dopamine dysfunction have been postulated as a cause of catatonia as well (4). Catatonia is characterized by predominant psychomotor disturbances with varying presentation. According to the Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-5), it is diagnosed when three or more signs are present (5) (Tab. 1).

Conceptual changes in the understanding of catatonia as a separate entity are visible in the new classification systems (DSM 5, ICD-11), where catatonia is described as a specifier or separate syndrome. For psychiatric practice, the most significant change is the exclusion of catatonia as a subtype of schizophrenia in the ICD-11 classification (unlike its classification as F20.2 Catatonic schizophrenia in ICD-10). Instead, catatonia is now recognized as a distinct condition that can arise due to various causes, including mental disorder, psychoactive substances (including medication) and health condition (as a secondary syndrome) (6).

Benzodiazepines remain to be the first-line treatment of catatonia, while electroconvulsive therapy (ECT) is used when there is insufficient response to benzodiazepines, as well as in malignant or delirious catatonia and neuroleptic malignant syndrome (7). Ben-

Tab. 1. Group of symptoms of catatonia according to DSM-5 (2013).

Stuporous catatonia	Excited catatonia
• Mutism • Lethargy or stupor • Posturing • Catalepsy • Grimacing • Waxy flexibility • Negativism	• Agitation • Echolalia • Echopraxia • Motor repetition • Mannerism

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zodiazepines and ECT are the only treatments for which there is clinical evidence (8). Response rates to benzodiazepines and ECT are high, regardless of the cause of catatonia (7). There does not seem to be evidence for the use of antipsychotics as therapy for catatonic patients without any underlying psychotic disorder (8).

Neuropsychiatric complications of COVID-19

There are several reasons why the current coronavirus disease 2019 (COVID-19) pandemic might have neuropsychiatric consequences. Currently available data show that the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the causal agent of COVID-19, has a neuroinvasive potential. Its neurotropism is limited, and it can be neurovirulent in at least a subgroup of patients (9). SARS-CoV-2 infection can directly invade the CNS through the blood circulation and neuronal pathways, and indirectly affect the innate and adaptive immune system and cause neuroinflammation (10). A wide spectrum of parainfectious and postinfectious neuropsychiatric symptoms have been described as complications of COVID-19, although these appear more prominent during the post-acute phase. Potential mechanisms for neuropsychiatric complications of the virus include direct viral invasion of the CNS, cytokine dysregulation, virus-induced immune reaction and autoimmunity during or after acute infection, effects of immunomodulatory treatments, hypoxic brain injury and hypercoagulability (11, 12, 13, 9). A significant proportion of patients with COVID-19 develop confusion, delirium, agitation, altered consciousness and other neuropsychiatric symptoms, including cerebrovascular events, encephalopathy, encephalitis, cognitive symptoms, depression, anxiety, insomnia, and post-traumatic stress disorder (10). Encephalitis as a complication of COVID-19 is uncommon, with the incidence being less than 1 % in the general population of COVID-19 patients but rises greatly to 6.7 % in those who are severely ill. A few patients who were reported to suffer from encephalitis as a complication of COVID-19 are asymptomatic carriers of SARS-CoV-2 with symptoms of encephalitis being their only manifestation (14).

Catatonia can be present in patients with an active viral infection with involvement of the CNS (11). Catatonia associated with viral infection was manifested during influenza pandemic in 1918, at that time called *encephalitis lethargica* (15). Akinetic mutism, retrospectively regarded as catatonia, was first described at the beginning of the COVID-19 pandemic, in May 2020 (16). Later, catatonia syndrome in COVID-19 patients, was reported in several case studies with varying presentation (17, 18, 19, 20, 21, 22, 23, 24). The diagnosis of concurrent catatonia in COVID-19 patients should be considered when the work-ups to identify more common medical causes of encephalopathy are negative, there is no identifiable psychiatric etiology for catatonia and there is a positive response to benzodiazepines (18).

Diagnostics and therapeutical management in co-occurrence of catatonic syndrome and COVID-19 infection are a clinical practice challenge that the authors demonstrate in two cases. In addition, both patients were not vaccinated against COVID-19 at the time.

Case 1

A 49-year-old female patient with a past medical history of arterial hypertension and liver damage, without any known prior personal as well as family neuropsychiatric history. In November 2021, one week before the admission to the hospital, she reported anosmia and ageusia and was tested positive for COVID-19 with RT-PCR nasopharyngeal swab. She was prescribed azithromycin, mucolytics and alprazolam (later the patient admitted taking only two tablets). She presented to the emergency department with dizziness, elevated blood pressure and fever. After the physical state evaluation, she was prescribed antihypertensive treatment, vitamins, non-steroid agents and was sent home. There was also a history of sudden change in behavior in previous days, namely that the patient excessively measured her blood pressure. Next day she was brought to the emergency department again, *she did not communicate at all (mutism),* was somnolent (GCS was 10), while her vital signs were stable. Based on the latter symptoms, a neuroinfection was considered. As described by the neurologist *she was not responsive when addressed, she did not respond to pain stimuli, she was resistant to manipulation, she was forcefully closing her eyes (negativism),* however when asked, she was able to elevate her arms and legs. CT scan showed small chronic ischemic lesions. In laboratory testing, interleukin-6 was elevated (IL-6 = 190,01 ng/l), and blood count showed thrombocytosis. Due to the lack of cooperation and atypical neurological clinical presentation the liaison psychiatrist was called. During psychiatric examination the patient was still *psychomotorically inhibited (lethargy),* but was able to answer the questions asked, although she did not communicate spontaneously. She was not disoriented but there were depressive delusions present. Two hours after the psychiatric examination the patient was reexamined by a neurologist because she stopped communicating again, she responded only to severe pain stimuli and her eyeballs were deviated. She was preliminarily diagnosed with *an altered state of consciousness – coma (suspected stupor).* With a differential diagnosis of brainstem ischemia, she was admitted to the department of neurology. On the second day, brain MRI was performed with findings of chronic small-vessel leukoencephalopathy (Fazekas 1) and cortical atrophy. CT scan of lungs showed small ground glass opacities. Since the cerebrospinal fluid examination showed no abnormalities, neuroinfection was ruled out. On the third day, upon investigation, the main findings were leukocytosis and thrombocytosis. EEG was without any pathological findings. She was seen by a pneumologist due to hypoxemia and inflammation and was diagnosed with bilateral COVID-19 bronchopneumonia. She received a complex treatment for COVID-19, including corticosteroids, antibiotics and oxygen therapy due to mild hypoxemia. On the fifth day, due to the worsening of anxiety, she was evaluated by a psychiatrist again. The patient was worried about her relatives because she was not in touch with her family and was prescribed benzodiazepines to be used only in case of worsening anxiety, while she refused antidepressants. Later that day, she was reexamined by a psychiatrist, due to mutism and refusal of medication and was indicated for transfer to the psychiatric department. On the sixth day upon admission she

was mute, manifested active negativism and lethargy, but there was no posturing or catalepsy. During the psychiatric hospitalization, further information was obtained, namely that the patient's father had a history of one suicidal attempt after a huge financial loss. On the seventh day, parenteral olanzapine was administered (10 mg per day). The contact with the patient gradually improved but the depressive symptoms started to be more prominent in the clinical picture. However, the cooperation with the patient continued to be insufficient, she refused to eat, and a nasogastric tube had to be inserted. Afterwards, venlafaxine was added to the treatment (final dose was 112.5 mg per day). Psychodiagnostic examination did not confirm any signs of psychosis. The patient gradually recovered and was discharged after 15 days of psychiatric care with diagnoses of COVID-19, adjustment disorder F43.2 and other acute and transient psychotic disorders F23.8.

Case 2

A 42-year-old female patient with a long-term psychiatric history, diagnosed with paranoid schizophrenia, and history of clozapine-induced agranulocytosis, and unvaccinated against COVID-19. The family history is positive for mental disorders, her mother and sister are both diagnosed with schizophrenia. The patient's husband died due to acute leukemia 10 months prior to the current treatment. She was hospitalized in the psychiatric department in November 2021 due to acute relapse of schizophrenia. In clinical picture, the contact with the patient was limited, and there were visual and auditory hallucinations, incoherent thinking, paranoid delusions and negativism. She was treated with high doses of haloperidol and later, quetiapine was administered due to sedation and extrapyramidal syndrome. After seven days of psychiatric in-patient treatment, she tested positive for COVID-19 with RT-PCR nasopharyngeal swab. The patient displayed mild symptoms of COVID-19 (subfebrile temperature) without the need for complex treatment, however her condition got complicated with elevation of CRP (60,80 mg/l) and assumed urinary infection, which was managed empirically with sulfamethoxazole. Due to agitation, the patient was later treated with parenteral olanzapine (20 mg per day) and zuclopenthixol depot injection without any significant improvement. The cooperation with the patient continued to be difficult, she was aggressive towards the staff and *mute.* Due to food refusal, a nasogastric tube was administered. Clinical symptoms began to progress, the patient continued to be mute, *negativistic* and *stuporous*. Because of the epidemiological situation in the hospital, the electroconvulsive treatment (ECT) had to be postponed. On Day 27, the treatment with olanzapine was discontinued and the patient was instead treated with clonazepam and two applications of midazolam (5 mg intravenous bolus) with only a slight improvement in her ability to communicate. ECT was contraindicated by an internal medicine doctor because of persistent inflammation and fever as a result of relapsing urinary tract infection. The patient underwent specific antibiotic treatment (initially with sulfamethoxazole/trimethoprim, which later changed to ciprofloxacin and fosfomycin) and started rehabilitation of hypomobilization syndrome. Additional findings showed low concentrations of serum B12 (85 pg/ml) and mild anemia (hemoglobin 101 g/l) and required parenteral substitution. After full resolution of catatonia symptoms, we initiated titration of cariprazine up to 6 mg per day due to prominent negative symptoms, and the contact with the patient significantly improved and psychotic phenomena subsided. Prior to the end of inpatient treatment we observed insomnia and reactive sub-depressive mood (the loss of husband, length of hospital stay) therefore she was treated with 100 mg of trazodone with a positive effect. The patient was discharged from hospital after an 84-day treatment.

Tab. 2. Psychopathological symptoms of catatonia in presented cases.

Catatonic symptoms	
CASE 1	CASE 2
• Mutism • Lethargy, susp. stupor • Negativism	• Mutism • Stupor • Negativism

Discussion

Current revisions of classification systems reflect present-day scientific knowledge and the development in the concept of catatonia, which in the past was mainly understood as a subtype of schizophrenia. Numerous infectious diseases and immune dysregulation have been reported to have an interconnection to catatonia, however the possible underlying etiopathological mechanism remains unclear. Suggested mechanisms are direct neurotoxic effect, psychological reaction to the infection, or mediation by an acute-phase response (7). In real clinical practice, especially during COVID-19 pandemic, the diagnostic and therapeutic management of patients with COVID-19 infection and neuropsychiatric clinical symptoms was challenging. The aim was to evaluate the direct and indirect effects of COVID-19 infection on the manifestation and course of psychopathological symptoms of catatonia (Tab. 2). It is worth noting that none of the patients had developed productive symptoms, nor any of the more specific symptoms of catatonia, but the clinical presentation characteristics clearly indicated a catatonic syndrome.

Neurological findings

When considering the possibility of a direct involvement of the SARS-COV-2 in the CNS *in Case 1,* there was negative cytology and biochemistry in CSF findings. In the MRI scan there were chronic changes and neuroinfection was excluded by the neurologist, but specific autoimmune encephalitis antibodies and PCR of SARS-CoV-2 in CSF were not examined. However, there are a few reports of patients with viral encephalitis as a complication of COVID-19 with normal brain imaging results (14). There have been reports of SARS-CoV-2 found in cerebrospinal fluid (CSF). Despite these findings, inflammatory damage by SARS-CoV-2 cannot be ruled out even with absent CSF abnormalities as the virus dissemination is transient and its CSF titers may be extremely low (21, 22). What stood against the possibility of encephalitis diagno-

sis was EEG testing which did not show any specific epileptiform activity but there was no mention of other EEG changes.

In Case 2, there were no neurological findings and catatonia was considered as a feature of chronic schizophrenia. There was an absence of EEG and recent CT or MRI brain scan and therefore we cannot exclude certain neurological abnormalities.

Immune system abnormalities

The question remains whether the corticosteroid therapy in Case 1 was a contributing factor to treatment since the patient ended a 10-day dexamethasone course due to COVID-19 pneumonia (8 mg per day intravenously). On the other hand, this would indicate a possibility of immune dysregulation etiology because general treatment for autoimmune encephalitis lies in immunosuppression. The differential diagnostics of etiology of vascular changes and neurodegeneration not typical for age was not performed.

Systemic inflammation and cytokine storm are proposed as a likely etiology of COVID-19-associated neuropsychiatric symptoms (18).The patient *in Case 1* showed elevated IL-6 and was put on corticosteroid therapy due to COVID-19 pneumonia. The patient *in Case 2* showed leukocytosis and CRP elevation above 60 (with concurrent urinary tract infection), without any severe physical impairment due to COVID-19 infection. Disruptions to immune functioning and neuroinflammation in COVID-19 ultimately lead to brain lesions and accelerate the progression and worsening of the clinical outcomes of neuropsychiatric disorders (10). Given the central role of immune dysregulation in developing fatal SARS-CoV-2 infections, and providing that schizophrenia is already characterized by subclinical inflammation, the systemic hyper-inflammation triggered by SARS-CoV-2 infection may be more pronounced in people with schizophrenia (25). We speculate that aforementioned mechanisms might be responsible for catatonia presentation in the patient with schizophrenia in Case 2.

Impact of anxiety

One possible interpretation of catatonia is that the syndrome is an outward manifestation of extreme anxiety (26). There are reports of anxiety as a significant feature in catatonia and COVID-19 cases (24). We speculate that anxiety as a prominent symptom was manifested *in Case 1* since the patient suffered from major anxiety which persisted even after the resolution of catatonic symptoms, and antidepressant treatment with SNRI was required for state stabilization. The patient *in Case 2* also showed signs of increased anxiety due to traumatic events (loss of husband) in recent months, but the stress could not be described as acute. Rather, it could be considered a prolonged stressor that may have resulted in relapse even in the absence of pandemic. Nevertheless, contracting the disease in the beginning of acute treatment might have contributed to catatonia symptoms manifestation in a patient without catatonia in their history, i.e. the disease contraction could be the stressor that triggered the anxiety.

Additionally, we must emphasize the negative impact of COVID-19 measures in health care facilities on treatment outcomes, namely isolation of the patients and lack of contact with relatives. In Slovakia there were special departments for COVID-19-positive and COVID-19-negative patients established, which had a disrupting effect on the continuity of treatment (treatment initiated in one facility might need to be continued in another), while the reduced availability of patient beds in psychiatric departments shortened the in-patient care duration as well as time necessary for sufficient diagnostic process (e.g, in *Case 1,* cortical atrophy in early age without exclusion of neurodegenerative causes).

Delirium as differential diagnosis

Differential diagnosis should include delirium, given that various clinical patterns of delirium in patients with COVID-19 were observed (hypokinetic delirium; hyperkinetic delirium with or without dementia; hyperkinetic delirium with or without ARDS) (27). Also, there is a possibility of co-occurrence of delirium and catatonic features (2). *In Case 1,* hypokinetic delirium should have been considered, especially when the diagnosis of delirium in COVID-19 cases is more common and better documented than catatonia. The patient in Case 1 showed a partial worsening of psychiatric symptoms when overall inflammation markers started to rise, however her state fluctuated in severity of symptoms, not in disorientation (the patient showed only slight amnesia). Catatonic symptoms were more indicative of affectivity disturbances in view of the fact that anxiety continued to be dominantly present even after the patient's physical condition had improved. *In Case 2,* we did not suspect delirium as a possible explanation of the symptoms because the general physical condition was not severe and the patient had a prior history of schizophrenia spectrum disorder.

Other general medical conditions

Last but not least, other general medical conditions must be included as a cause of catatonia in the differential diagnostic process. *In Case 2,* the patient had signs of anemia (101 HGB mg/l) due to severe B12 deficiency that required parenteral supplementation. B12 deficiency has been reported as one of the causes of medical catatonia (2).

Therapeutic management

Identifying and treating the underlying disorder is essential in the treatment of catatonia (8)), although the rates of response to benzodiazepines and ECT are high regardless of the cause of the catatonia (7). The patient *in Case 1* did not receive any benzodiazepines for catatonia symptoms. Considering the patient's history *in Case 1* (first episode of mental disorder, rise of anxiety, depressive delusions) we could have considered it to be the first episode of MDD with psychotic features, which would have probably been responsive to the treatment with benzodiazepines. As benzodiazepines are respiratory-driven depressants and patients with COVID-19 are at risk of hypoxemic respiratory failure, they were not used in the treatment of the patient. However safe

administration of parenteral benzodiazepines in patients without respiratory compromise was demonstrated (23). The patient *in Case 1* was administered 10 mg of olanzapine instead and there was continuous improvement of symptoms described within several days. Some authors recommend that antipsychotics should be avoided altogether in catatonia patients, although there are case reports of successful treatment with aripiprazole, risperidone, olanzapine, ziprasidone and clozapine (28). *In Case 2,* there was just a slight response to benzodiazepines, but catatonia associated with schizophrenia is also less likely to respond to benzodiazepines and ECT than catatonia caused by affective disorders and general medical conditions (29).

The use of ECT was not required *in Case 1* due to the resolution of catatonia symptoms within a short period of time. *In Case 2,* ECT was contraindicated by an internal medicine doctor because of preexisting SARS-CoV-2 infection with the risk of possible progression to pneumonia and hypoxemia during the treatment. Another factor which complicated ECT administration were epidemiological adjustments in terms of COVID-19-free and COVID-19 infection departments. As indicated in literature, concurrent administration of ECT in catatonia can be safely permitted in the absence of respiratory compromise and ongoing close monitoring of the course of COVID-19 (23). Again, we speculate that the patient would benefit from initiating ECT and even at the beginning of the psychotic relapse; a prompt administration of ECT could have prevented further development into the catatonic state. The patient continuously showed improvement on pharmacological treatment wherefore ECT was not required anymore.

Conclusion

Catatonia is a possibly life-threatening condition with multiple complications that should be included in a differential diagnosis, despite being a rare neuropsychiatric complication of COVID-19. Differentiating catatonia from quantitative disturbances of consciousness due to COVID-19-associated physical alteration is challenging, especially for patients without prior psychiatric history. However, even in patients with a prior history of mental disorders with possible catatonia-like clinical picture, the complex approach should be applied when concurrent SARS-CoV-2 infection is present. The management of catatonic syndrome with COVID-19 presents a challenge and requires a comprehensive therapeutic approach. The aim of our case reports was to demonstrate possible difficulties in diagnostic evaluation and treatment management of catatonia in COVID-19. The cases underscore the widely accepted shift in the concept of catatonia which is no longer linked exclusively as a subtype to schizophrenia. Instead, catatonia is recognized as a neuropsychiatric syndrome that demands a multidisciplinary approach. Catatonic features can be present in viral infections of CNS, which includes also SARS-CoV-2 infection. We suggest the medical professionals should keep a high index of anxiety evaluation in patients with COVID-19 infection, preventing its extreme acceleration as a risk factor for catatonia development.

Limitation

In the described cases, standardized symptom scales such as BFCRS were not used. In Case 2, no imaging and neurological examinations were carried out. Another limitation is the lack of available follow-up information on patients.

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Received April 4, 2023.
Accepted April 17, 2023.