ISSN: 2475-5435

Research Article

International Journal of Psychiatry

Management of Catatonia at Bernese Jura Hospital, Mental Health Pole in Bellelay, Switzerland

Makenga Bof Jean Claude[1,2,3*], Biyong Issack[4], Bugnon Carine[1], Karamalla Akram[1], Masse Benjamin[1] and Lucchelli Juan Pablo[1,5]

[1]*Hôpital Jura Bernois, Pôle Santé Mentale, l'Abbaye 2 | 2713 Bellelay ; Switzerland*

[2]*Université Libre de Bruxelles (ULB), Route de Lennik 14, Laeken, Brussels, Belgium*

[3]*Université Notre Dame du Kasayi (UKA), Kananga, RDC*

[4]*Institute of Psychotraumatology and Mediation, Grand-rue, 1A, 2000 Neuchâtel, Switzerland*

[5]*Laboratoire de Psychopathologie, E.A. 4050, Université de Rennes 2, France*

[*]Corresponding Author
Makenga Bof Jean Claude, Hôpital Jura Bernois, Pôle Santé Mentale, l'Abbaye 2 | 2713 Bellelay ; Switzerland.

Submitted: 2025, Feb 05; **Accepted**: 2025, Mar 05; **Published**: 2025, Mar 14

Citation: Makenga Bof, J. C., Biyong, I., Bugnon, C., Karamalla, A., Masse, B., et al. (2025). Management of Catatonia at Bernese Jura Hospital, Mental Health Pole in Bellelay, Switzerland. *Int J Psychiatry, 10*(1), 01-13.

Abstract

Background

Catatonia is a neurogenic motor and behavioral syndrome characterized by a range of physical manifestations, from profound immobility to excessive motor activity. It is often associated with stupor and other behavioral abnormalities.

Objective

This study aimed to determine the prevalence of catatonia signs, evaluate the treatment methods used, and identify predictive factors for catatonia in affected patients.

Methods

This retrospective, descriptive, and analytical study included all patients diagnosed with catatonia based on DSM-5 criteria and treated at the Bernese Jura Hospital, Mental Health Center, between January 1, 2017, and December 31, 2020. Data were collected from medical records, and statistical analysis was performed using STATA version 16 software.

Results

A total of 27 patients with a median age of 59 years [49; 72] were included in the study. Excited catatonia was observed in 74.1% of patients, while delayed catatonia was present in 25.9%. Half of the patients were over 60 years of age. The most prominent catatonia signs included grimacing (85%), mannerisms (77%), stereotypy (66%), and severe psychomotor agitation (51%). The primary reasons for hospitalization were depression (41%), encephalitis or autoimmune disorders (33%), and metabolic disorders such as hyponatremia (11%). The most frequently used treatments were lorazepam (44%), diazepam (33%), and zolpidem (11%). Discharge diagnoses were predominantly bipolar I disorder (85%), other acute psychotic disorders with delusional features (81.5%), and unipolar major depressive disorder (29.6%).

Patients with excited catatonia predominantly exhibited three or more DSM-5 criteria. Remission of catatonia signs occurred within 24 hours for 75% of patients treated with lorazepam. Oxazepam achieved 100% remission in patients with excited catatonia. Lorazepam, zolpidem, and diazepam were effective for both types of catatonia, with 25% efficacy in delayed catatonia and 75% in excited catatonia. The risk of developing catatonia was 14 times higher in patients aged 40 and older, 7 times higher in men, and 2 times higher in patients who had taken antipsychotics for more than 24 hours.

Conclusion

This study highlights that long-term antipsychotic use can induce catatonia. The primary therapeutic approach remains the rapid administration of lorazepam. Severe psychomotor agitation, combined with other typical signs, confirms excited catatonia. Zolpidem is a viable alternative after the failure of other benzodiazepines, and electroconvulsive therapy should be considered for patients unresponsive to benzodiazepines or those with malignant features.

Keywords: Catatonia, Major Depression, Psychotic Depression, Mood Disorders, Antipsychotics, Lorazepam, Oxazepam, Zolpidem

1. Introduction

Catatonia is a transnosographic neuropsychiatric syndrome characterized by a spectrum of physical manifestations, ranging from profound immobility to excessive motor activity [1,2]. Clinically, its emotional aspects vary between psychomotor retardation and extreme excitability [1,2]. Psychomotor signs include stupor, agitation, and pathognomonic features such as verbigeration and waxy flexibility [1,2]. Historically, catatonia was classified as a subtype of schizophrenia due to its volitional disturbances. However, changes in nosology now recognize its high prevalence in mood disorders, its overlap with delirium, and its frequent comorbidity with medical conditions [1,2].

The Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5), published in February 2015, includes psychomotor abnormalities as diagnostic criteria or specifiers for three psychiatric disorders: mood disorders, schizophrenia spectrum disorders, and delirium [1,2]. Additionally, the DSM-5 diagnostic criteria for autism spectrum disorders identify psychomotor signs of catatonia as a primary diagnostic feature [1,2]. The classification of catatonia has evolved over time, with recent changes in the DSM-5 reflecting a broader understanding of its clinical presentation [3]. Alongside the DSM-5, the Bush-Francis Catatonia Rating Scale (BFCRS), first introduced in February 1996 and revised in 2007, is widely used for assessment [3-6].

According to the DSM-5, the diagnosis of catatonia requires the presence of at least three of the following symptoms: stupor, catalepsy, waxy flexibility, mutism, negativism, posturing, mannerisms, stereotypy, agitation, grimacing, echolalia, and echopraxia [2-4]. The prevalence of catatonia varies across populations, ranging from less than 10% to over 60% [7]. In acute psychiatric inpatient settings, the estimated incidence is between 5% and 20% [8-10]. Although there is no uniform treatment, benzodiazepines and electroconvulsive therapy (ECT) are the most widely studied and effective interventions [11,12]. Early recognition and prompt treatment are critical, as certain etiologies and the syndrome itself can be life-threatening [13].

The primary objective of this study is to determine the prevalence of catatonia signs and their diagnostic features. Specific objectives include evaluating treatment indications and methods, describing the clinical course, and identifying predictive factors for catatonia in affected patients.

2. Methodology

2.1 Study Framework

Our study was conducted at the Bernese Jura Hospital, Mental Health Center in Bellelay, Switzerland. The Mental Health Center (PSM SA) is a local center of excellence in mental health, with the primary goal of maintaining and reintegrating patients into their social, family, and professional environments. To achieve this, the center is integrated into regional and national healthcare networks and actively pursues a clinical research program.

2.2 Study Design and Population

This is a retrospective, descriptive, and analytical study covering the period from January 1, 2017, to December 31, 2020 — a total of four years. The study population included all patients diagnosed and treated for catatonia during this period, based on the DSM-5 criteria. Patients with an uncertain diagnosis or unsuccessful diagnosis of catatonia were excluded.

2.3 Data Sources and Collection

Data were collected from patient medical records within the PSM system, using Cariatides, a hospital software developed by the Swiss Management System in collaboration with Swiss hospitals and clinics. A pre-established form was used for data collection, divided into four sections:

- Section 1: Patient identification (encoding).
- Section 2: Clinical information.
- Section 3: Biological data and treatment details.
- Section 4: Complications and outcome processing.

Data collection took place from June 8, 2021, to July 7, 2021 — a period of 30 days (1 month).

3. Variables Studied

a) Dependent Variable

The diagnosis of catatonia, as defined by the DSM-5, was considered the dependent variable. According to the DSM-5 criteria, a diagnosis of catatonia requires the presence of at least three of the following symptoms (or a minimum of two to four signs persisting for several hours):

- Stupor (reduced psychomotor activity or reactivity to the environment).
- Catalepsy (passive acceptance of body positioning by the examiner).
- Waxy flexibility (slight resistance to positioning by the examiner).
- Mutism (lack of verbal response, excluding patients with established aphasia).

- Negativism (unwarranted resistance to instructions or external stimuli).
- Posturing (voluntary maintenance of a position against gravity for an extended period).
- Mannerisms (odd, purposeful movements).
- Stereotypy (repetitive, non-goal-directed movements).
- Agitation (excessive, aimless motor activity not influenced by external stimuli).
- Grimacing.
- Echolalia (mimicking another person's speech).
- Echopraxia (imitating another person's movements).

b) Independent Variables

Independent variables included anthropometric data, reasons for hospitalization, length of stay, paramedical assessments, treatment details, remission of clinical signs, resumption of antipsychotic treatment, discharge diagnosis, and discharge treatment.

c) Data Analysis and Processing

Data were extracted and analyzed using STATA version 16 software for integration and statistical modeling. Descriptive statistics and univariate analysis were performed using medians and interquartile ranges (IQR) for continuous variables, and counts with percentages for categorical variables. Missing data were managed using the "pattern missing criterion."

Proportions of catatonia were stratified by baseline characteristics, and differences between subgroups were compared using Pearson's chi-square tests or Fisher's exact tests, as appropriate.

Univariate and multivariate logistic regression models were used to assess the risk of developing catatonia based on baseline characteristics with a p-value ≤ 20% in univariate analysis. Adjusted odds ratios (OR) with 95% confidence intervals (95% CI) were calculated using the maximum likelihood method.

Competing models were compared using goodness-of-fit statistics, including Deviance (-2log(L)), Akaike Information Criterion (AIC), Corrected Akaike Information Criterion (AICc), and Bayesian Information Criterion (BIC). The most parsimonious model was selected based on the minimum values of these statistics.

A two-sided p-value < 0.05 was considered statistically significant, and 95% confidence intervals were reported where applicable.

d) Ethical Considerations

The study was approved by the Cantonal Research Ethics Commission of the Directorate of Health, Social Affairs, and Integration of the Canton of Bern (approval number DSSI-CCER: 2021-00677). Participants were informed of the study's objectives, procedures, risks, and benefits, and provided informed consent before participation. For participants who did not respond within 30 days, it was assumed they did not exercise their right to refuse, and their coded non-genetic data were used for statistical analysis (Article 33, Paragraph 2 LRH). An exceptional authorization request under Article 34 LRH was made for unreachable or deceased patients. Data confidentiality and anonymity were maintained throughout data processing, analysis, and presentation. The study adhered to the principles outlined in the Declaration of Helsinki II.

4. Results

4.1 Characteristics of the Study

A total of 27 patients diagnosed with catatonia were included in the study, with a median age of 59 years [49; 72]. The majority were female (55.6%), with a female-to-male sex ratio of 1.3. Two types of catatonia were observed: the excited type (74.1%) and the delayed type (25.9%).

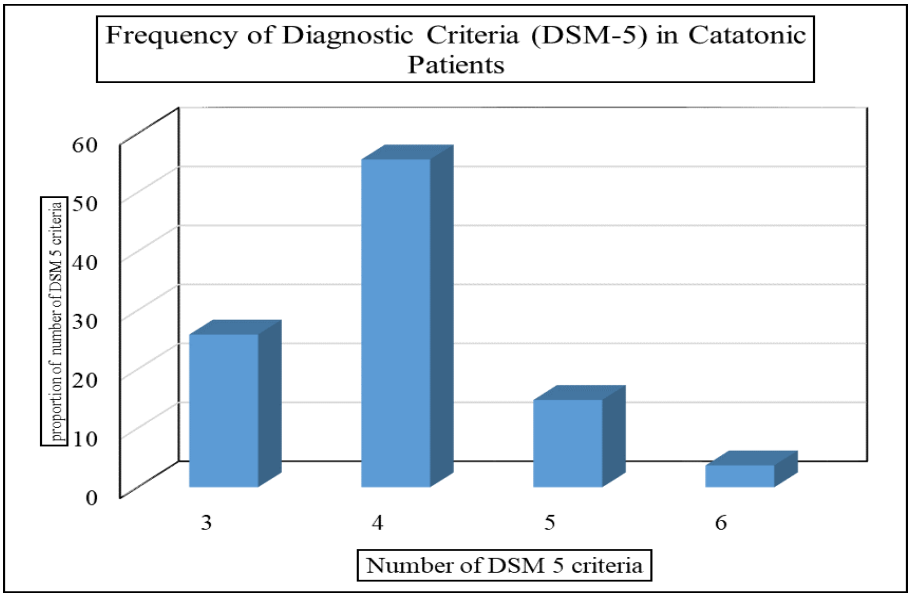

Figure 1: The Frequency of Diagnostic Criteria (DSM-5) in Catatonic Patients, HJB, PSM, Bellelay 2017-2020.

Many patients with catatonia had 4 criteria (55%) and 25% had 3 criteria.

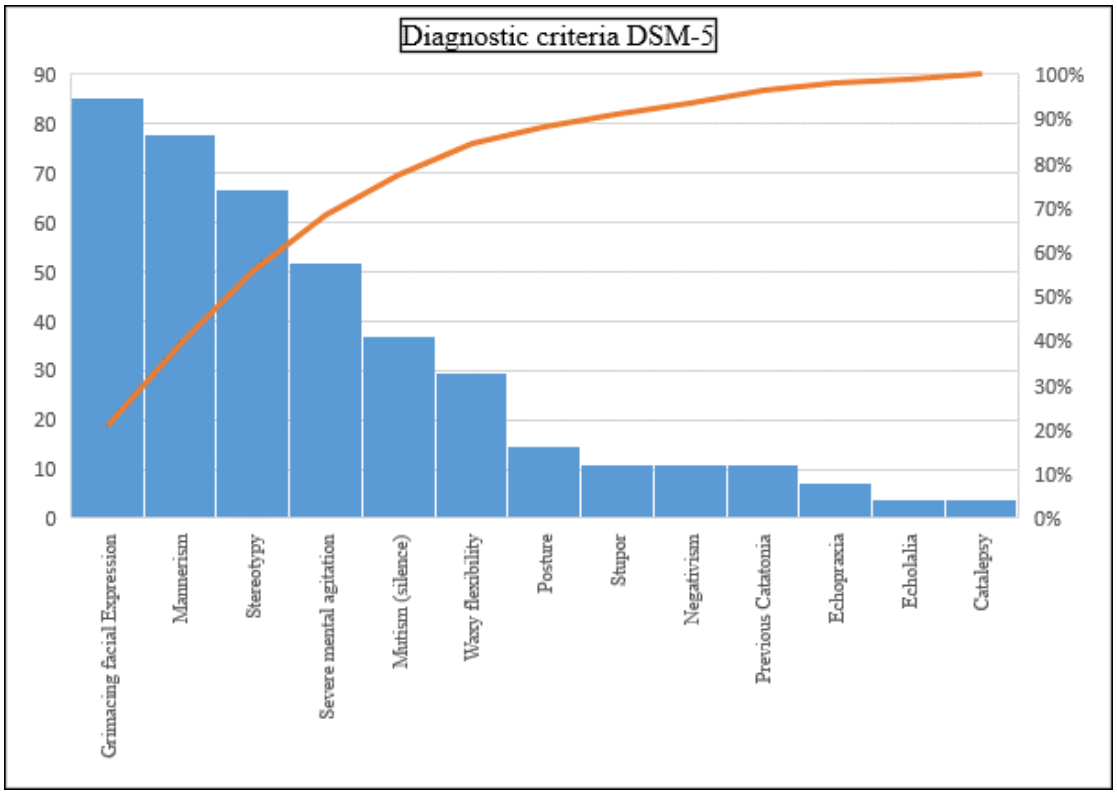

Figure 2: Diagnostic Criteria (DSM-5) in Catatonic Patients, HJB, PSM, Bellelay 2017-2020.

The most prominent criteria for catatonia were: Grimacing facial expression (85%), Mannerism (77%), Stereotypy (66%), and Severe psychomotor agitation (51%). The main reasons for hospitalization were: Depression (41%), Encephalitis, Autoimmune disorders (33%), Metabolic disorders (hyponatremia) (11%).

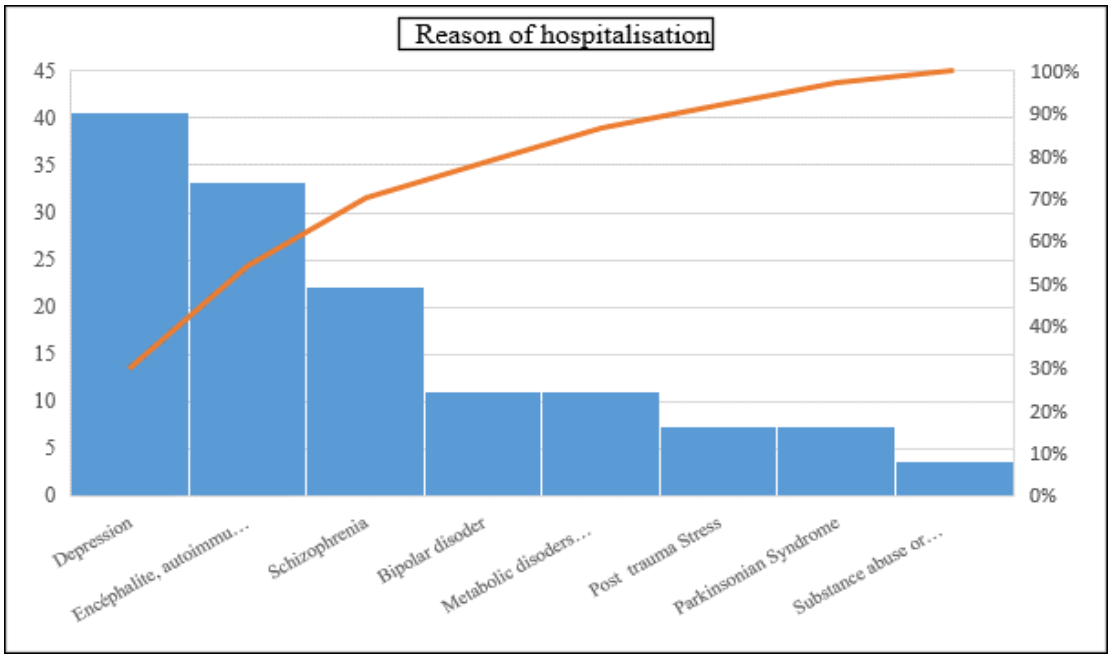

Figure 3: Raison of Hospitalisation in Catatonic Patients, HJB, PSM, Bellelay 2017-2020.

The main reasons for hospitalization were: Depression (41%), Encephalitis, Autoimmune disorders (33%), Metabolic disorders (hyponatremia) (11%).

Characteristics	n(%)
Treatment	
Lorazepam	
Yes	12(44.4)
No	15 (55.6)
Zolpidem	
Yes	3 (11.1)
No	24 (88.9)
Diazepam	
Yes	9 (33.3)
No	18 (66.7)
Oxazepam	
Yes	2 (7.4)
No	25 (92.6)
Duration of treatment of antipsychotics	
< 24H	24 (88.9)
≥ 24H	3 (11.1)
Complication	
Yes	2 (7.4)
No	25(92.6)
Remission Sign (hours)	
< 24	7 (25.9)
24 -48	10 (37.1)
49- 72	8 (29.6)
>72	2(7.4)
Rémission type of catatonia	
< 24	7 (25.9)
24 -48	11 (40.8)
49- 72	7 (25.9)
>72	2 (7.4)

The most used treatments: Lorazepam (44%), Diazepam (33%) and Zolpidem (11%).

Table 1: Treatment, Duration, Complication and Remission Of Catatonic Patients, HJB, PSM, Bellelay 2017-2020.

Characteristics	n(%)
Diagnostic exit	
Bipolar disorder	
Yes	23 (85.2)
No	4 (14.8)
Unipolar major depressive disorder	
Yes	8 (29.6)
No	19 (70.4)
Schizophrenia	
Yes	2 (7.4)
No	25 (92.6)
Schizo-affective disorder	
Yes	3 (11.1)
No	24(88.9)
Brief Psychotic disorder	
Yes	1 (3.7)
No	25(96.3)
Other acute psychotic disorder, predominantly delusional	
Yes	22 (81.5)
No	5 (18.5)
Output processing	
Benzodiazépine	
Yes	14 (51.8)
No	13 (48.2)
API	
Yes	16 (59.3)
No	11 (40.7)
APII	
Yes	5(18.5)
No	22 (81.5)
Mood stabilizer	
Yes	3(11.1)
No	24 (88.9)

API: first generation antipsychotic (typical neuroleptic); APII: second generation antipsychotic (atypical neuroleptic).

Table 2: Diagnostic Exit and Output Processing of Catatonic Patients, HJB, PSM, Bellelay 2017-2020.

Discharge diagnoses were dominated by: Bipolar disorder type I (85%), Other acute psychotic disorder, predominantly delusional (81.5%) and Unipolar major depressive disorder (29.6%). The most prescribed molecules at discharge were dominated by: API = 2 (59%), APII = 3 (18.5%) and Mood stabilizer = 4 (11%).

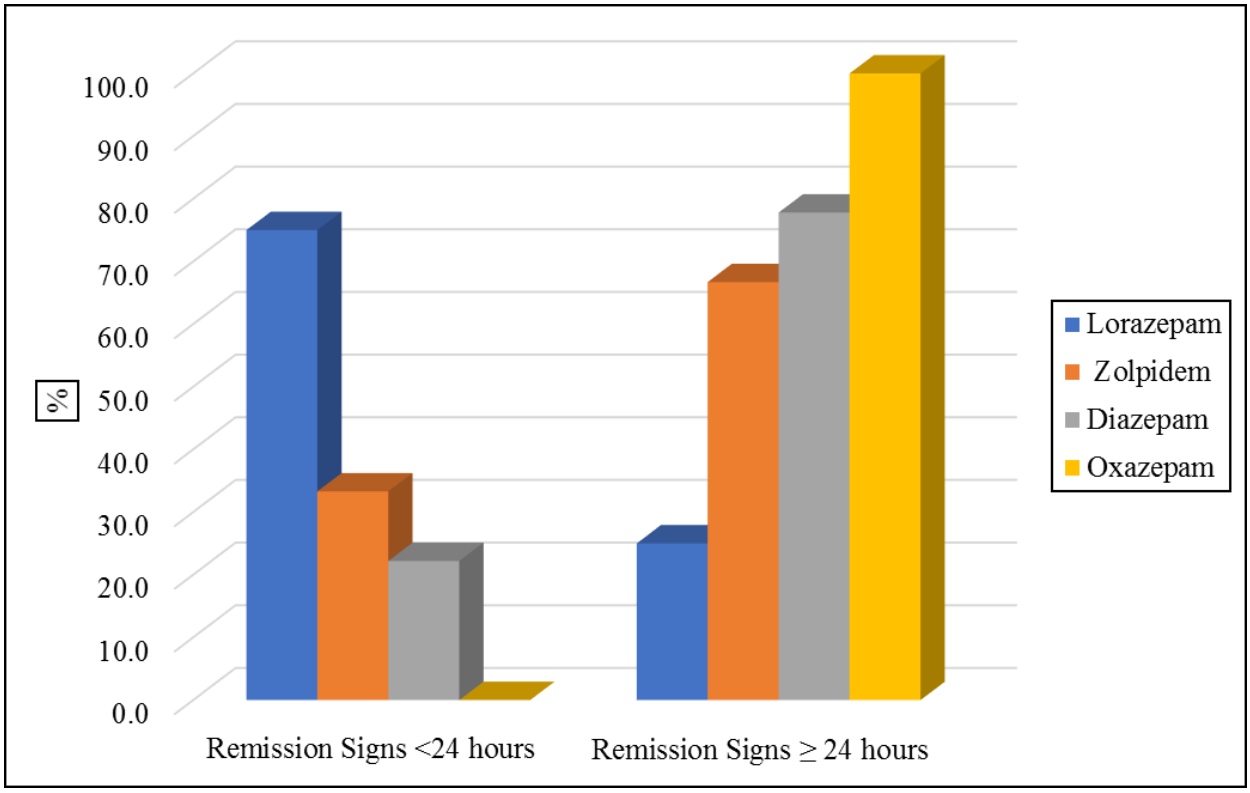

Figure 4: Time to Remission of Signs of Catatonia Depending on Treatment Initiated, HJB, PSM, Bellelay 2017-2020.

Remission of signs of catatonia was rapid within 24 hours for 75% of patients taking Lorazepam. In contrast, in almost all of those who took Oxazepam, signs of remission were observed after 24 hours of treatment.

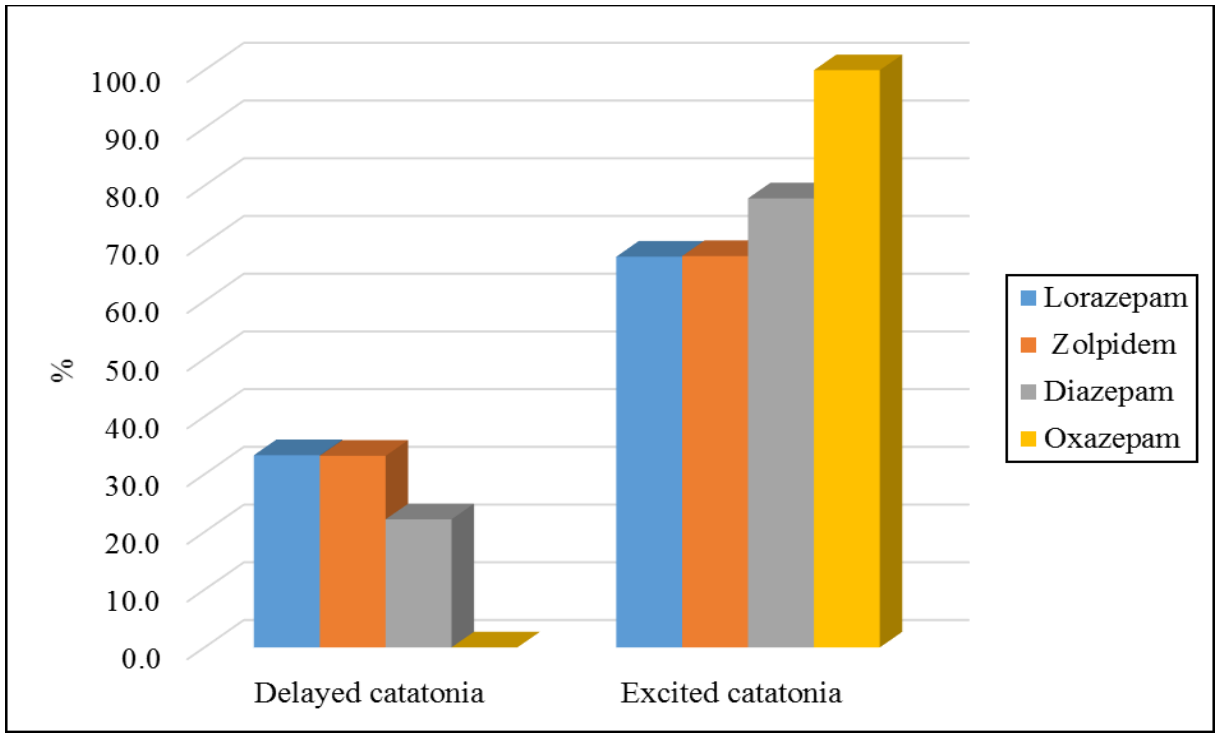

Figure 5: Remission of Signs of Delayed Vs Excited Catatonia According To The Type of Treatment, HJB, PSM, Bellelay 2017-2020

Remission was 100% with Oxazepam for patients with excited catatonia, however Lorazepam, Zolpidem and Diazepam worked on both types, similarly, 25% on delayed catatonia versus 75% on excited catatonia.

Predictors of catatonia risk	Univariate Model		Multivariate Model	
	OR brut (CI 95%)	P-value	Ajusted OR (IC95%)	P-value
Age (Years)				
< 40	1		1	
≥ 40	14.2(1.2-32.7)	0.038	23.8(1.7-53.6	0.002
Gender				
Male	7.3(1.7-72.6)	0.029	12.2(1.8-82.7)	0.007
Female	1		1	
Antipsychotic Duration (hours)				
≤ 24	1		1	
> 24	1.8(1.3-10;3)	0.012	11.7(1.8-82.7	0.008
HL=1.14, p=0.767				

Table 3: Predictors of the Risk of Developing Catatonia: Risk of having at Least 3 of DSM-5 Criteria, HJB, PSM, Bellelay 2017-2020

The results of the univariate analysis revealed three co-variables at the basis of the occurrence of catatonia. The risk of the occurrence of catatonia was 14 times higher for subjects aged 40 years and over, 7 times higher in men and 2 times higher for those who consumed antipsychotics for more than 24 hours. Using a multivariable regression including confounding variables on the risk factors for catatonia and the results are similar to those found on univariate analysis (Table 3)

5. Discussion

Our study had three primary aims: (1) to determine the prevalence of catatonia signs, (2) to evaluate the treatment methods used, and (3) to identify predictive factors for catatonia in affected patients. A total of 27 patients diagnosed with catatonia were included in the study, with a median age of 59 years [49; 72]. The prevalence rates were 25.9% for delayed catatonia and 74.1% for excited catatonia (Figure 5). Apart from anthropometric parameters and a few clinical features, the study population was largely heterogeneous.

All patients were diagnosed according to DSM-5 criteria, which recognize catatonic symptoms in various psychiatric and medical conditions. Catatonia is classified either as a specifier of another mental disorder (e.g., schizophrenia spectrum disorder, bipolar disorder, or depressive disorder) or as an associated diagnosis in cases of organic disorders, as reported in the literature by Sebastian Walther et al. in 2019 [2].

At the Bernese Jura Hospital, Mental Health Center (HJB, PSM), the diagnosis of catatonia was based on the presence of at least three of the 12 clinical signs defined in the DSM-5 [14, 15]. However, consistent benchmark definitions for catatonia remain lacking [16]. It is important to highlight the existence of multiple validated catatonia assessment scales, which can aid not only in diagnosis but also in monitoring treatment response [17]. Examples include the Bush-Francis Catatonia Rating Scale (BFCRS) and the North off Catatonia Scale [17-20]. Among these, the BFCRS is the most widely used in both clinical practice and research [17-20]. A revised version of the BFCRS, published in 2007, is particularly useful for assessing individuals with chronic schizophrenia [21]. Additionally, electroencephalographic (EEG) tracings may serve as a valuable diagnostic tool for catatonia [22].

BFCRS Items	DSM-5 Items	Definitions of signs of catatonia
1	9	Comotiontion/excitement - extreme hyperactivity with unintentional movements and extreme uncontrollable and emotional rections.
19		Ambiguity - appearance to be "stuck" in an indecisive or hesitant movement.
16		Automatic obedience – mecanical and reproducible conformity at the request of the examiner, even if it is dangerous.
23		Autonomous Anomaly - diaphoresis, palpitations or abnormal temperature, pulse, blood pressure, pulse or respiratory rate.
5	2	Catalepsy - passive induction of a maintained posture against gravity.
22		Fighting spirit – fight against other people, with or without risk of injury.
7	11	Echolalia - imitation of other people's words.
7*	12	Echopraxia – imitate the movements of another person.
18		Resistance – resistance to positioning by the examiner which increases in proportion to the force applied.
20		Grip reflex
6	10	Grimace – strange and inappropriate facial expressions in any situation.
15		Impulsivity - the patient suddenly adopts inappropriate behavior without provocation; thereafter, he can give no explanation, or only an easy explanation.
9	7	Mannerism: strange and circumstantial caricature of normal actions.
17		Movements - exaggerated movements in response to slight pressure.
3	4	Mutism - no or very little verbal response (exclude if known aphasia)
12	5	Negativism - opposing or not responding to external instructions or stimuli.
21		Perseveration - total or partial repetition of non-goal-oriented verbal actions or content.
5	6	Posture - spontaneous and active maintenance of a posture against gravity.
11		Rigidity - resistance through an increase in muscle tone.
4		Fixation/blinking - fixed gaze, reduced blinking and eyes wide open. Or increased blinking
8	8	Stereotypy: repetitive, abnormally frequent movements that are not directed towards a goal.
2	1	Stupor - no or markedly reduced psychomotor activity; no active relationship with the environment.
10		Verbalization - continuous repetition, without direction, of words, expressions or sentences.
13	3	Waxy flexibility - light and regular resistance to positioning by the examiner.
14		Removal - diversion of the examiner's gaze, lack of eye contact and/or refusal to take food or drinks when offered. Or social isolation

Table 4: Summary of the Definition of the items of the Bush Francis Catatonia Rating Scale (BFCRS) and the DSM-5 Criteria [2]

Our results indicate that 3 out of 4 patients exhibited excited catatonia, and 1 in 2 patients was over 60 years of age. The most prominent diagnostic criteria for catatonia were: grimacing facial expression (85%), mannerisms (77%), stereotypy (66%), and severe psychomotor agitation (51%). Indeed, it is well-established that catatonia, not otherwise specified, can occur in both men and women, with the age of onset often influenced by the presence of major mood disorders, psychotic disorders, or general medical conditions, as documented in the literature [2,23]. Our study reveals that women were more likely to develop catatonia, with a prevalence of 55.6% compared to 44.4% in men. This observation aligns with the findings of Kolevzon et al., who reported a higher incidence of catatonia in women than in men (13 women versus 3 men) [24]. It is important to note that catatonia can manifest across all age groups, including children, adolescents, adults, and the elderly, as demonstrated by Benarous et al. and McGuire et al. in their respective studies [25,26].

Contrary to the literature, which often emphasizes delayed catatonia as the most common presentation in hospital settings, our study found that 74% of patients exhibited excited catatonia, a less common form characterized by prolonged periods of psychomotor agitation [27]. This finding highlights the variability in catatonia presentations and underscores the importance of recognizing excited catatonia in clinical practice.

Several studies have identified grimacing facial expressions, mannerisms, stereotypy, and severe psychomotor agitation as key indicators of excited catatonia [28,29]. Our results corroborate these findings, with grimacing facial expression (85%), mannerisms (77%), stereotypy (66%), and severe psychomotor agitation (51%) being the most prominent signs observed in our patient cohort. These findings are consistent with those reported by Morrison and Zain et al., who described cases of excited catatonia marked by severe psychomotor agitation, rigidity, tense speech, and combativeness [30,31].

Regarding the reasons for hospitalization, the majority of patients (41%) were admitted due to depression, followed by encephalitis and autoimmune disorders (33%), and metabolic disorders such as hyponatremia (11%). Neerukonda et al. have highlighted anti-N-methyl-D-aspartate receptor encephalitis (anti-NMDA receptor encephalitis) as one of the most well-recognized autoimmune conditions affecting the brain, characterized by a range of progressive neuropsychiatric symptoms [32]. Our observations support this, as illustrated by the case of an 18-year-old woman with excited catatonia secondary to anti-NMDA receptor encephalitis. The patient presented with an acute psychotic state marked by profound disorganization and vivid visual hallucinations. Her hospital course was complicated by delayed and excited catatonia, autonomic instability, and sensitivity to several neuroleptics [32].

Although the association between recently onset catatonic symptoms and hyponatremia has rarely been reported in the literature, Peritogiannis and Rizos noted that hyponatremia can induce catatonic symptoms in patients, irrespective of the underlying mental illness [33]. This phenomenon is particularly relevant in patients with psychotic or mood disorders, which themselves can precipitate catatonic symptoms [33]. In our study, serum sodium levels were seldom assessed, and newly onset catatonic symptoms were primarily attributed to the underlying psychotic or mood disorder. However, measuring serum sodium levels could guide treating psychiatrists to pursue further investigations and initiate appropriate treatment [33].

The literature indicates that approximately 20% to 50% of catatonic patients suffer from depressive disorders, 28% exhibit features of mixed mania-depression, and 30% are diagnosed with schizophrenia [34-36]. Prajapati et al. suggest that the underlying mechanisms of catatonia involve decreased dopaminergic neurotransmission, reduced gamma-aminobutyric acid (GABA) activity, and altered N-methyl-D-aspartate (NMDA) signaling due to excessive glutamatergic activity [37].

Our study highlights that the most commonly used treatments were Lorazepam (44%), Diazepam (33%), and Zolpidem (11%). Discharge diagnoses were predominantly Bipolar I Disorder (85%), Other Acute Psychotic Disorders, Predominantly Delusional (81.5%), and Unipolar Major Depressive Disorder (29.6%). Rapid remission of catatonic symptoms within 24 hours was observed in 75% of patients treated with Lorazepam. Similarly, nearly all patients treated with Oxazepam showed signs of remission within 24 hours. These findings align with those reported by Zaman et al., who demonstrated significant improvement in catatonic symptoms, measured as a 50% reduction on the visual analogue scale, in 17 patients treated with Lorazepam and Oxazepam [38]. However, these authors caution against definitive conclusions regarding the efficacy of benzodiazepines in managing catatonia in patients with schizophrenia and other severe mental illnesses, citing limited data quality and availability [38].

Several studies have also highlighted the beneficial effects of Zolpidem in treating catatonia following the failure of benzodiazepines and/or electroconvulsive therapy (ECT) [39]. For instance, Mastain et al. were the first to report a dramatic and sustained improvement in catatonia resistant to ECT and benzodiazepines using Zolpidem in a 56-year-old woman with subcortical stroke-induced catatonia [39]. Unfortunately, in our study, no patients experienced treatment failure with benzodiazepines. Other studies, such as those by Sienaert et al., have also documented the efficacy of Zolpidem in managing catatonia [40]. Madigand et al. emphasize that first-line treatment for catatonia typically involves benzodiazepines such as Lorazepam and Diazepam or benzodiazepine-related agents like Zolpidem. In cases of treatment failure or malignant catatonia, ECT is recommended [41]. No ECT procedures were performed at HJB PSM during the study period. However, further investigation into Oxazepam, which in our study reduced signs of excited

catatonia within an average of 24 hours, is warranted.

Univariate analysis revealed three covariates associated with the occurrence of catatonia: the risk was 14 times higher in individuals aged 40 and over, 7 times higher in men, and 2 times higher in those who had taken antipsychotics for more than 24 hours. Multivariate regression analysis, accounting for confounding variables, yielded results consistent with the univariate analysis (Table 3). As previously discussed, catatonia can occur in individuals of all ages, including children, adolescents, adults, and the elderly, as noted in the literature [25,26]. The increased risk of catatonia in individuals aged 40 and over in our study may be influenced by the sample composition, and we refrain from making broader statistical inferences due to the small sample size. It is important to note that catatonia can develop even at advanced ages, and some patients may exhibit resistance to benzodiazepines, as demonstrated by Saito et al.

Contrary to our findings, several studies suggest that women are more likely to develop catatonia. Additionally, the use of antipsychotics has been implicated in the onset of catatonia in some patients, as demonstrated by Worku and Fekadu. This aligns with our observation of a 7-fold higher risk of catatonia in patients who had taken antipsychotics for more than 24 hours.

6. Limitations

The small sample size in our study limits the ability to perform more comprehensive statistical analyses and draw definitive conclusions. As a retrospective study, we did not assess causality or therapeutic efficacy but instead focused on describing our observations. Laboratory tests were not analyzed in depth to determine the role of autoimmune diseases in the occurrence of catatonia.

7. Conclusion

Catatonia appears to have diverse etiologies, yet the general therapeutic approach remains consistent, emphasizing the rapid administration of benzodiazepines such as Lorazepam. The risk of developing catatonia was twice as high in patients who took antipsychotics for more than 24 hours. Catatonic syndrome can occur at any age, and while women are often reported to be more affected, our study found a 7-fold higher risk in men aged 40 and over. Severe psychomotor agitation, combined with other typical signs of catatonia, confirms the diagnosis of excited catatonia. Zolpidem is a viable option following benzodiazepine treatment failure, and electroconvulsive therapy should be considered for patients who do not respond to benzodiazepines or present with malignant features.

Recommendations

We recommend the use of the Bush-Francis Catatonia Rating Scale-Revised (BFCRS-R). The BFCRS was found to be one-dimensional, except for three unsuitable items and one marginally unsuitable item. These three unsuitable items were removed, resulting in the revised version, BFCRS-R [20].

In cases where first- or second-line benzodiazepines fail, or in the presence of malignant catatonia, the use of zolpidem and/or electroconvulsive therapy (ECT) is recommended [29].
We strongly recommend prospective studies to further investigate the causes, effects, occurrence, and treatment of catatonia.

Author Contributions

MBJC and LJP planned, organized, and contributed to the study design. MBJC, LJP, BM, CB, AK and IB contributed to the review and editing of the research topic and the editorial summarizing it. All authors contributed to the article and approved the submitted version.

Conflict of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Acknowledgments

We thank the authorities of the Bernese Jura Hospital for allowing us to conduct this research. We also extend our gratitude to all the authors who contributed to the development of this manuscript and to the patients who participated in this study.

References

1. Edinoff, A. N., Kaufman, S. E., Hollier, J. W., Virgen, C. G., Karam, C. A., Malone, G. W., ... & Kaye, A. D. (2021). Catatonia: clinical overview of the diagnosis, treatment, and clinical challenges. *Neurology international, 13*(4), 570-586.
2. Walther, S., Stegmayer, K., Wilson, J. E., & Heckers, S. (2019). Structure and neural mechanisms of catatonia. *The Lancet Psychiatry, 6*(7), 610-619.
3. Tandon, R., Heckers, S., Bustillo, J., Barch, D. M., Gaebel, W., Gur, R. E., ... & Carpenter, W. (2013). Catatonia in DSM-5. *Schizophrenia research, 150*(1), 26-30.
4. Bush, G., Fink, M., Petrides, G., Dowling, F., & Francis, A. (1996). Catatonia. I. Rating scale and standardized examination. *Acta Psychiatrica Scandinavica, 93*(2), 129-136.
5. Wong, E., Ungvari, G. S., Leung, S. K., & Tang, W. K. (2007). Rating catatonia in patients with chronic schizophrenia: Rasch analysis of the Bush–Francis Catatonia Rating Scale. *International Journal of Methods in Psychiatric Research, 16*(3), 161-170.
6. Lee, J. W., Schwartz, D. L., & Hallmayer, J. (2000). Catatonia in a psychiatric intensive care facility: incidence and response to benzodiazepines. *Annals of Clinical Psychiatry, 12*(2), 89-96.
7. Zingela, Z., Stroud, L., Cronje, J., Fink, M., & Van Wyk, S. (2022). A prospective descriptive study on prevalence of catatonia and correlates in an acute mental health unit in Nelson Mandela Bay, South Africa. *PLoS One, 17*(3),

e0264944.
8. Seethalakshmi, R., Dhavale, S., Suggu, K., & Dewan, M. (2008). Catatonic syndrome: importance of detection and treatment with lorazepam. *Annals of Clinical Psychiatry, 20*(1), 5-8.
9. Thakur, A., Jagadheesan, K., Dutta, S., & Sinha, V. K. (2003). Incidence of catatonia in children and adolescents in a paediatric psychiatric clinic. *Australian & New Zealand Journal of Psychiatry, 37*(2), 200-203.
10. Burrow, J. P., Spurling, B. C., Marwaha, R. (2021).Catatonia. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; PMID: 28613592.
11. Hawkins, J. M., Archer, K. J., Strakowski, S. M., & Keck Jr, P. E. (1995). Somatic treatment of catatonia. *The International Journal of Psychiatry in Medicine, 25*(4), 345-369.
12. Francis, A. (2010). Catatonia: diagnosis, classification, and treatment. *Current psychiatry reports, 12*, 180-185.
13. Freudenreich, O., Nejad, S. H., Francis, A., & Fricchione, G. L. (2011). Psychosis, mania, and catatonia. *Textbook of Psychosomatic Medicine: Psychiatric Care of the Medically Ill, Second Edition, Levenson JL (Ed), American Psychiatric Publishing, Washington, DC,* 219.
14. Fink, M., Shorter, E., & Taylor, M. A. (2010). Catatonia is not schizophrenia: Kraepelin's error and the need to recognize catatonia as an independent syndrome in medical nomenclature. *Schizophrenia bulletin, 36*(2), 314-320.
15. Brar, K., Kaushik, S. S., & Lippmann, S. (2017). Catatonia update. *The Primary Care Companion for CNS Disorders, 19*(5), 24215.
16. Sienaert, P., Rooseleer, J., & De Fruyt, J. (2011). Measuring catatonia: a systematic review of rating scales. *Journal of affective disorders, 135*(1-3), 1-9.
17. Bush, G., Fink, M., Petrides, G., Dowling, F., & Francis, A. Catatonia. I. Rating scale and standardized. *Acta Psychiatr Scand, 996*(93), t29-i36.
18. Subramaniyam, B. A., Muliyala, K. P., Suchandra, H. H., & Reddi, V. S. K. (2020). Diagnosing catatonia and its dimensions: Cluster analysis and factor solution using the Bush Francis Catatonia Rating Scale (BFCRS). *Asian Journal of Psychiatry, 52,* 102002.
19. Rooseleer, J., Willaert, A., & Sienaert, P. (2011). Rating scales for assessing catatonia; which ones are the best?. *Tijdschrift Voor Psychiatrie, 53*(5), 287-298.
20. Carroll, B. T., & Boutros, N. N. (1995). Clinical electroencephalograms in patients with catatonic disorders. *Clinical Electroencephalography, 26*(1), 60-64.
21. Walther, S., & Strik, W. (2016). Catatonia. *CNS spectrums, 21*(4), 341-348.
22. Kolevzon, A., Delaby, E., Berry-Kravis, E., Buxbaum, J. D., & Betancur, C. (2019). Neuropsychiatric decompensation in adolescents and adults with Phelan-McDermid syndrome: a systematic review of the literature. *Molecular Autism, 10*(1), 50.
23. Benarous, X., Raffin, M., Ferrafiat, V., Consoli, A., & Cohen, D. (2018). Catatonia in children and adolescents: new perspectives. *Schizophrenia Research, 200*, 56-67.
24. McGuire, E., Yohanathan, M., Lally, L., McCarthy, G. (2017). Hyponatraemia-associated catatonia. *BMJ Case Rep, bcr2017219487*.
25. Rasmussen, S. A., Mazurek, M. F., & Rosebush, P. I. (2016). Catatonia: our current understanding of its diagnosis, treatment and pathophysiology. *World journal of psychiatry, 6*(4), 391.
26. Allouard, L. (2017). La catatonie dans le trouble bipolaire, réflexions autour d'un cas.
27. Morrison, J. R. (1973). Catatonia: retarded and excited types. *Archives of general psychiatry, 28*(1), 39-41.
28. Zain, S. M., Muthukanagaraj, P., & Rahman, N. (2021). Excited catatonia—a delayed neuropsychiatric complication of COVID-19 infection. *Cureus, 13*(3).
29. Neerukonda, N., Bliss, M., Jafroodifar, A., & Leontieva, L. (2020). Olanzapine and lorazepam used in the symptomatic management of excited catatonia secondary to anti-N-methyl-D-aspartate receptor encephalitis. *Cureus, 12*(6).
30. Peritogiannis, V., & Rizos, D. V. (2021). Catatonia associated with hyponatremia: case report and brief review of the literature. Clinical Practice and Epidemiology in Mental Health: CP & EMH, 17, 26.
31. Taylor, M. A., & Fink, M. (2003). Catatonia in psychiatric classification: a home of its own. *American Journal of Psychiatry, 160*(7), 1233-1241.
32. Sienaert, P., Dhossche, D. M., Vancampfort, D., De Hert, M., & Gazdag, G. (2014). A clinical review of the treatment of catatonia. *Frontiers in psychiatry, 5*, 181.
33. Medda, P., Toni, C., Luchini, F., Giorgi Mariani, M., Mauri, M., & Perugi, G. (2015). Catatonia in 26 patients with bipolar disorder: clinical features and response to electroconvulsive therapy. *Bipolar Disorders, 17*(8), 892-901.
34. Prajapati, P. R., Fabius, D., & Pradhan, B. (2016). Clinical course and management of drug-induced catatonia and paranoid behavior in an adolescent. *Klinik Psikofarmakoloji Bülteni-Bulletin of Clinical Psychopharmacology, 26*(3), 310-315.
35. Gibson, R. C., & Walcott, G. (2008). Benzodiazepines for catatonia in people with schizophrenia and other serious mental illnesses. *Cochrane Database of Systematic Reviews*, (4).
36. Mastain, B., Vaiva, G., Guerouaou, D., Pommery, J., & Thomas, P. (1995). Favourable effect of zolpidem on catatonia. *Revue Neurologique, 151*(1), 52-56.
37. Madigand, J., Lebain, P., Callery, G., & Dollfus, S. (2016). Catatonic syndrome: From detection to therapy. *L'encephale, 42*(4), 340-345.
38. Saito, Y., Noto, K., Kobayashi, R., Suzuki, A., Morioka, D., Hayashi, H., & Otani, K. (2021). Catatonia as the initial manifestation of dementia with Lewy bodies. *The American Journal of Case Reports, 22,* e932018-1.
39. Manjunatha, N., Saddichha, S., & Khess, C. R. J. (2007). Idiopathic recurrent catatonia needs maintenance lorazepam:

case report and review. *Australian & New Zealand Journal of Psychiatry, 41*(7), 625-627.
40. Doran, E., & Sheehan, J. D. (2018). Acute catatonia on medical wards: a case series. *Journal of Medical Case Reports, 12*, 1-5.
41. Worku, B., & Fekadu, A. (2015). Symptom profile and short term outcome of catatonia: an exploratory clinical study. *BMC psychiatry, 15*, 1-8.