Advances in Social Science, Education and Humanities Research, volume 586
Proceedings of the 2021 International Conference on Public Relations and Social Sciences (ICPRSS 2021)

A General Review of the Catatonia

Zhilin Huang[1, a, *, †] Hangjie Liu[2, b, *, †] Yihan Shen[1, c, *, †]

[1] *The Affiliated High School to Hangzhou Normal University, Zhejiang, China*
[2] *The International Department of the High School Affiliated to Shaanxi Normal University, Shaanxi, China*
[*]*Corresponding author. Email: [a] guanghua.ren@gecacademy.cn, [b]1320475939@qq.com，[c]2829980863@qq.com*
[†]*These authors contributed equally.*

ABSTRACT
This article is an overview of catatonia (catatonia is any condition of abnormal motor activity thought to be caused by a psychiatric disorder). In the DSM-5, catatonia is not recognized as its own disorder but rather is listed as a symptom of other psychiatric conditions, such as schizophrenia, bipolar disorder, post-traumatic stress disorder, and depression.), and including its etiology, impacts, treatments, and purpose is to help common people (who are not familiar with the concept of catatonia) understand catatonia. Pricing research illustrates the relationship between catatonia and other disorders, like Schizophrenia, autistic spectrum disorders, and Parkinson's disease, various impacts on different patients and several treatments, Electroconvulsive therapy, Benzodiazepines, and Antipsychotics. Some recommendations, in the end, might be helpful to future investigation. The treatments that were illustrated in the passage were all worked well in the treatment process. This article might be helpful for those who want to understand catatonia.

Keywords: *catatonia, treatment, causes.*

1. INTRODUCTION

1.1. Definition

Catatonia refers to motor syndrome [1]. In previous psychological research, catatonia is not considered an independently diagnosable mental illness. Emil Kraepelin previously noted this condition as a kind of clinical symptom of dementia named dementia praecox [2]. Subsequently, Eugene Bleuler reorganized the condition of dementia praecox with some associated chronic symptoms and separated it from progressive disorders, which were renamed independently by schizophrenia. At the same time, he classified catatonia as a subtype of schizophrenia [3].

Catatonia could be found among various other mental or somatic disorders, and Nearly 10 percent of psychiatric inpatients in hospitals have catatonic symptoms. For example, patients with catatonic symptoms are often accompanied by bipolar disorder, schizophrenia, and post-traumatic stress disorder [1].

The clinical manifestations of catatonia are diverse, which is associated with around 40 different symptoms. According to some studies, the specific clinical manifestation of catatonia included automatic obedience, posturing, paralytic, rigidity, imitation, repetitive motion and actions that cannot be stopped, tachycardia, and hyperthermia [3, 4].

1.2. Etiology

The etiology of catatonia is complicated and remains unsure till nowadays. A range of potential causes of it includes mental illnesses, like mood disorders, encephalitis, and schizophrenia. And the causes also include medical conditions, like infection, exposure to toxins, and a severe vitamin B12 deficiency. The side effect of certain medications was used to treat mental illnesses, withdrawal from medications like clozapine, Brain abnormalities.

In DSM-5 (The Diagnostic and Statistical Manual of Mental Disorders), scholars made a case for a separate category for catatonia as a distinct clinical-diagnostic entity, catatonia is not recognized as its own disorder but rather is listed as a symptom of other psychiatric conditions, such as schizophrenia, bipolar disorder, post-traumatic stress disorder, and depression [5]. This broad, all-inclusive view of catatonia was unsuccessfully challenged, and it eventually found its way into DSM-5, although in disguise and not without ambiguity [6]. Meanwhile, Catatonia appears in three forms in DSM 5: “Catatonia associated with another mental disorder (catatonia specifier),” “Catatonic disorder due to another

medical condition," and "Unspecified catatonia." The list of signs/symptoms is identical in the first two forms, while the third is a "wastebasket" category for catatonia of uncertain origin or incomplete presentation [7]. This ambiguity in DSM 5 clearly demonstrates the uncertainty surrounding the entire concept of catatonia. In literature, cases of catatonia that have been effectively treated with other therapies have been described. These include transcranial magnetic stimulation NMDA receptor antagonist zolpidem, antiepileptics, and atypical antipsychotics [7].

1.3. Diagnosis

The diagnosis condition of abnormal motor activity in DSM-5 is thought to be caused by a psychiatric disorder; it happens most often with people who have mood disorders or psychotic disorders.

Catatonia is often confused with a variety of other conditions that have a similar sign, although the full clinical picture is normally distinguishable [8]. Parkinson's disease is associated with catatonia, but tremor is notably absent in catatonia. Extra-pyramidal side-effects (EPSE) are also similar to catatonia. More important is that if therapists use benzodiazepines, postural instability can be exacerbated [9]. As a result, it is so necessary to distinguish between EPS and catatonia. Except for these diseases, some illnesses can have a proper solution to finding differences with catatonia, such as neuroleptic malignant syndrome, locked-in syndrome, stiff-person syndrome, etc. Until recently, it was thought of as a type of schizophrenia. But doctors now understand that other mental illnesses and some conditions that throw off your body's metabolism also can make you catatonic [10]. The common perception is that catatonia has become a separate clinical entity although not yet an independent clinical diagnosis [11]. This ambiguity in DSM 5 clearly demonstrates the uncertainty surrounding the entire concept of catatonia.

2. RELATIONSHIP WITH OTHER DISEASES

2.1. Catatonia and Schizophrenia

Catatonia is highly associated with schizophrenia. For a long time, it has been considered a subtype of schizophrenia. According to the statistics and studies, about 20 percent of patients with schizophrenia have catatonic symptoms [12]. In contrast, with the prevalence of catatonia among psychiatric patients, some psychologists have proposed that catatonia should be separated from schizophrenia and as an independent disorder in psychiatric classification [13].

There are some reasons to support the opinion that catatonia could be a separate disorder. Statistically, more than half of catatonic patients do not have symptoms of schizophrenia [14]. Catatonia is also strongly associated with other psychiatric disorders such as mood disorder and bipolar disorder (43%) [1, 6]. It suggests that schizophrenia is not necessary to trigger catatonia to provide the basis for separating the catatonia from schizophrenia.

Based on a report, patients with catatonia may respond significantly by injecting intravenous amobarbital or benzodiazepines, and those agents could treat catatonia. Moreover, about 70% of patients only respond to lorazepam rather than some antipsychotic agents. As a result, it is reasonable to distinguish between catatonia and schizophrenia [15].

2.2. Catatonia and autistic spectrum disorders

Autism spectrum disorder is characterized by impaired social interaction, communication, and imagination and is associated with stereotypical, repetitive patterns of behaviour [16]. Catatonia is distinguished by repetitive movements, mutism, posturing, and frantic agitation. These signs are also frequent in autism yet usually do not amount to a diagnosis of catatonia unless there is a sharp and sustained increase of these symptoms lasting days or weeks [17]. Firstly, the association between catatonia and autistic spectrum disorders is found in Wing's (1981) case series on Asperger syndrome. In her sample of 34 cases, 17 percent reported a coma was considered to be psychotic, and Gillberg (1985) described a case that one 14-year-old boy was diagnosed with Asperger's syndrome [18, 19]. In a follow-up study of children and adults with autism spectrum disorders, Gillberg and Steffenburg (1987) reported prevalence of "catatonia" is estimated at 6.5 percent when their population had reached adulthood [20]. Compare with other diseases. This seems to be a significant growth rate.

After this research, Realmuto and August proposed that catatonia in people with autism spectrum disorders is directly related to autism, rather than separate comorbidity [21]. However, they went on to study the importance of bipolar disorder and "organic defects" as risk factors for catatonic disorders and discussed three case studies. Again, these cases were in young adults (16 to 21 years old) with autism spectrum disorders. Bell (1997) describes the case of "John", who developed a catatonic state at the age of 21. His symptoms included postural freezing and retention. If anyone tried to get him to leave, he would become extremely excited and hurt himself. The man had a history of long trips in the past, a habit that recurred five years after the onset of a catatonic state, and the records showed that his statements might change periodically [22].

In conclusion, catatonia and autistic spectrum disorders have a close connection. Researchers need to value this relationship.

2.3. Catatonia and Parkinson disease

Parkinson's disease (PD) is a neurological disorder due to degeneration of dopaminergic neurons in substantia nigra, manifesting as rigidity, bradykinesia, and tremors [23]. Rigidity consists of a stiff position which the patient attempts to maintain despite efforts to be moved. Catatonic rigidity is not typically accompanied by cogwheeling or tremor, which helps catatonia to be differentiated from parkinsonian rigidity [24]. It is rarely reported that catatonia patients were accompanied by PD. Here is a case of a patient with PD and catatonia: A 55-year-old male farmer from a rural background with no family history of medical or psychiatric illness had been diagnosed with Idiopathic PD and catatonia; at first, he was diagnosed with Idiopathic PD and stated treatment with the combination of medicines, through several courses treatment, a diagnosis of catatonia was made in the absence of fever and elevated white blood cell count and treatment with electroconvulsive therapy (ECT) was used. In this case, Treatment with electroconvulsive therapy (ECT) improved symptoms of both PD and catatonia [25]. Although it is still unclear what is the true mechanism of catatonia, ECT is a considerable way of treatment. And till today, there are only three cases in which a patient diagnosed with PD with catatonia was treated with ECT. In these three cases, ECT all worked [26-28].

3. TREATMENT

3.1. Electroconvulsive therapy

Since the 16th century, the convulsive intervention has been used to treat mental disorders, until today, in the form of electroconvulsive therapy (ECT) [29]. However, the use of ECT declined after the use of pharmacotherapy for severe mental disorders during the 1970s and 1980s [30]. In the treatment for medication-resistant and very severe life-threatening clinical conditions, the main indication for ECT also transformed from first-line to last-resort treatment [30, 31]. In the literature review published in World J Psychiatry, ECT results were effective in all forms of catatonia, even after pharmacotherapy with benzodiazepines has failed. The results were superior to any other treatment in psychiatry because that Response rates ranged from 80% to 100% [32]. As a result, further understanding of ECT's mechanism of action in catatonia may facilitate the development of other brain stimulation techniques, such as transcranial magnetic stimulation and deep brain stimulation.

Hawkins et al. (1995) reviewed the treatment of 178 catatonic patients from 270 clinical records. Fifty-five patients (approximately 30%) received ECT alone, and 85% of them achieved catatonic remission [33]. The effects of electro spasticity in different subtypes of catatonia and maintenance of electro spasticity in the long-term prevention of recurrence of catatonia deserve further study [32].

3.2. Benzodiazepines

The use of benzodiazepine anxiolytics and hypnotics continues to excite controversy. Views differ from expert to expert and from country to country as to the extent of the problem, or even whether long-term benzodiazepine use actually constitutes a problem, and benzodiazepines are frequently prescribed to patients with different psychiatric disorders [34, 35]. So benzodiazepines are also prescribed to patients with catatonia. One of the most dramatic clinical phenomena is the response of catatonia to treatment with benzodiazepines. While benzodiazepines are extremely safe medications when used in the short term, several issues should be kept in mind during BZP treatment for patients with catatonia. They include 1) the risk of hypoventilation in obese patients or those with obstructive sleep apnea, 2) falls in elderly patients or those with balance problems after they start to move about following resolution of their catatonia, and 3) the potential, albeit small, for previously immobile patients to switch into a more exciting form of catatonia [12]. To discover the effectiveness of benzodiazepines, researchers had also treated the patients with catatonia with both benzodiazepines and Electroconvulsive Therapy. In this research, full recovery was achieved in catatonia by benzodiazepine plus ECT combination [36].

3.3. Antipsychotics

Catatonia was always linked with some psychotic disorders such as schizophrenia, so antipsychotics are often added to the treatment process. However, there are many debates on the use and effectiveness of antipsychotics [37]. The effectiveness of clozapine in the experiment of England has been proven. The vast majority of patients who received clozapine showed the remission of catatonia [38]. Despite the clozapine is beneficial for remission, it takes a long time to relieve symptoms, about seven weeks [38]. For the effectiveness of olanzapine, the experiment results are not clear enough but show the possibility of benefits on treatment. It needs further clinical trial with combination or contrast with other antipsychotics [39, 40].

Based on some cases, some classic antipsychotics, second-generation antipsychotics, and haloperidol have fewer benefits and even deteriorate the catatonia. Catatonic conditions may not abate and worsen to become NMS [1, 39, 41]. In a study, three catatonic patients developed the NMS (3.6%) among eighty-two patients who received antipsychotics. Compared to other kinds of patients with the treatment of antipsychotics, the incidence of NMS occurred was higher (0.07-1.8%) [1, 12, 42].

As a result, it should be cautious about choosing the kind of antipsychotics. The methods of Electroconvulsive therapy or benzodiazepines could be more recommended to use in the treatment process [37, 38].

4. CONCLUSION

In conclusion, the paper defines the catatonia more clearly, explore the relationship between catatonia and other diseases such as Schizophrenia, autistic spectrum disorders, and autistic spectrum disorders through between summarize variety previous research paper. And in the treatment part, this study sums up the information of electroconvulsive therapy, benzodiazepines, and antipsychotics. Since the causes of catatonia are varied and unsure so that researchers and therapists still need to focus on finding all of them and try to exclude mistakes in the future, what is more, since therapists might misdiagnosis so many similar symptoms about catatonia and some other diseases, it is necessary not only to be sure of the importance of the future study of catatonia but also educate public more about this disease. Although this paper is mainly an overview, which does not include own experiment or data, reading broad about literature and searching the materials can still help and guide people's future study on catatonia.

REFERENCES

[1] 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.

[2] Bartolommei, N., Lattanzi, L., Callari, A., Cosentino, L., Luchini, F., & Mauri, M. (2012). Catatonia: a critical review and therapeutic recommendations. Journal of Psychopathology, 18, 234-246.

[3] Wilcox, J. A., & Reid Duffy, P. (2015). The syndrome of catatonia. Behavioral Sciences, 5(4), 576-588.

[4] Walther, S., & Strik, W. (2016). Catatonia. CNS Spectrums, 21(4), 341-348.

[5] Francis, A., Fink, M., Appiani, F., Bertelsen, A., Bolwig, T. G., Bräunig, P., ... & Wachtel, L. (2010). Catatonia in diagnostic and statistical manual of mental disorders. The journal of ECT, 26(4), 246-247.

[6] Ungvari, G. S. (2014). Catatonia in DSM 5: controversies regarding its psychopathology, clinical presentation and treatment response. Neuropsychopharmacol Hung, 16(4), 189-194.

[7] Bartolommei, N., Lattanzi, L., Callari, A., Cosentino, L., Luchini, F., & Mauri, M. (2012). Catatonia: a critical review and therapeutic recommendations. Journal of Psychopathology, 18, 234-246.

[8] 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), 391-398.

[9] Ahuja, N., & Cole, A. J. (2009). Hyperthermia syndromes in psychiatry. Advances in psychiatric treatment, 15(3), 181-191.

[10] Sanil Rege. (2020). Catatonia – Pathophysiology, Diagnosis and Management. Retrieved from: https://psychscenehub.com/psychinsights/catatonia-pathophysiology-diagnosis-and-management/

[11] Parker, G., McClure, G., & Paterson, A. (2015). Melancholia and catatonia: disorders or specifiers?. Current psychiatry reports, 17(1), 1-6.

[12] Rosebush, P. I., & Mazurek, M. F. (2010). Catatonia and its treatment. Schizophrenia bulletin, 36(2), 239-242.

[13] Heckers, S., Tandon, R., & Bustillo, J. (2010). Catatonia in the DSM—shall we move or not? Schizophrenia Bulletin, 36(2), 205–207.

[14] Peralta, V., Cuesta, M. J., Serrano, J. F., & Martinez-Larrea, J. A. (2001). Classification issues in catatonia. European archives of psychiatry and clinical neuroscience, 251(1), I14-I16.

[15] 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.

[16] Wing, L. (1996). Autistic spectrum disorders. BMJ, 312, 327–328.

[17] Dhossche, D. M., Reti, I. M., & Wachtel, L. E. (2009). Catatonia and autism: a historical review, with implications for electroconvulsive therapy. The journal of ECT, 25(1), 19-22.

[18] Ropper, A. H., Adams, R., Victor, M., & Samuels, M. A. (2005). Adams and Victor's principles of neurology. McGraw Hill Medical.

[19] Gillberg, C. (1985). Asperger's syndrome and recurrent psychosis—A case study. Journal of Autism and Developmental Disorders, 15(4), 389-397.

[20] Realmuto, G. M., & August, G. J. (1991). Catatonia in autistic disorder: a sign of comorbidity or variable expression?. Journal of Autism and Developmental Disorders, 21(4), 517-528.

[21] Leiknes, K. A., Schweder, L. J. V., & Høie, B. (2012). Contemporary use and practice of electroconvulsive therapy worldwide. Brain and behavior, 2(3), 283-344.

[22] Eranti, S. V., & McLoughlin, D. M. (2003). Electroconvulsive therapy–state of the art. The British Journal of Psychiatry, 182(1), 8-9.

[23] Luchini, F., Medda, P., Mariani, M. G., Mauri, M., Toni, C., & Perugi, G. (2015). Electroconvulsive therapy in catatonic patients: efficacy and predictors of response. World journal of psychiatry, 5(2), 182-192.

[24] Wijemanne, S., & Jankovic, J. (2015). Movement disorders in catatonia. Journal of Neurology, Neurosurgery & Psychiatry, 86(8), 825-832.

[25] Ramesh, V., Sharma, A., Sharma, V., & Somani, A. (2019). Treatment of catatonia in Parkinson's disease with electroconvulsive therapy. Annals of Indian Academy of Neurology, 22(4), 501-503.

[26] Suzuki, K., Awata, S., Nakagawa, K., Takano, T., & Matsuoka, H. (2006). Catatonic stupor during the course of Parkinson's disease resolved with electroconvulsive therapy. Movement disorders: official journal of the Movement Disorder Society, 21(1), 123-124.

[27] Kamigaichi, R., Kubo, S., Ishikawa, K., Yokoyama, K., Ogaki, K., Usui, C., ... & Hattori, N. (2009). Effective control of catatonia in Parkinson's disease by electroconvulsive therapy: a case report. European journal of neurology, 16(2), e6-e6.

[28] Poyraz, B. Ç., Poyraz, C. A., Yassa, A., Arikan, M. K., Gündüz, A., & Kiziltan, G. (2016). Recurrent catatonia in Parkinson disease. Journal of clinical psychopharmacology, 36(1), 104-106.

[29] Wing, L. (1981). Asperger's syndrome: a clinical account. Psychological medicine, 11(1), 115-129.

[30] Gillberg, C., & Steffenburg, S. (1987). Outcome and prognostic factors in infantile autism and similar conditions: A population-based study of 46 cases followed through puberty. Journal of autism and developmental disorders, 17(2), 273-287.

[31] BELL , S . R . (1997) 'Catatonia in Autism: Prevalence and Short Case Study', in Living and Learning with Autism: Collected Papers from Conference Held at St Aidan's College, University of Durham, April 1997.Autism Research Unit, University of Sunderland.

[32] McCall, W. V. (2001). Electroconvulsive therapy in the era of modern psychopharmacology. International Journal of Neuropsychopharmacology, 4(3), 315-324.

[33] 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.

[34] Lader, M., Tylee, A., & Donoghue, J. (2009). Withdrawing benzodiazepines in primary care. CNS drugs, 23(1), 19-34.

[35] Uzun, S., Kozumplik, O., Jakovljević, M., & Sedić, B. (2010). Side effects of treatment with benzodiazepines. Psychiatria Danubina, 22(1), 90-93.

[36] Unal, A., Bulbul, F., Alpak, G., Virit, O., Copoglu, U. S., & Savas, H. A. (2013). Effective treatment of catatonia by combination of benzodiazepine and electroconvulsive therapy. The journal of ECT, 29(3), 206-209.

[37] Van Den Eede, F., Van Hecke, J., Van Dalfsen, A., Van den Bossche, B., Cosyns, P., & Sabbe, B. G. (2005). The use of atypical antipsychotics in the treatment of catatonia. European Psychiatry, 20(5-6), 422-429.

[38] England, M. L., Öngür, D., Konopaske, G. T., & Karmacharya, R. (2011). Catatonia in psychotic patients: clinical features and treatment response. The Journal of neuropsychiatry and clinical neurosciences, 23(2), 223-226.

[39] Francis, A. (2010). Catatonia: diagnosis, classification, and treatment. Current psychiatry reports, 12(3), 180-185.

[40] Marténvi, F., Metcalfe, S., Schausberger, B., & Dossenbach, M. R. (2001). An efficacy analysis of olanzapine treatment data in schizophrenia patients with catatonic signs and symptoms. Journal of Clinical Psychiatry, 62, 25-27.

[41] Pelzer, A. C., van der Heijden, F. M., & den Boer, E. (2018). Systematic review of catatonia treatment. Neuropsychiatric disease and treatment, 14, 317-326.

[42] Aderibigbe, Y. A., & Mathews, T. (1999). Epidemiology of neuroleptic malignant syndrome. Clinical neuropharmacology, 22(3), 151-158.