Case Report

THE JOURNAL OF
MEDICINE ACCESS

Challenges of treating catatonia in the community setting without access to electroconvulsive therapy

The Journal of Medicine Access
2023, Volume 7: 1–8

DOI: 10.1177/27550834231220504
journals.sagepub.com/home/map
Sage

Tyler Torrico[1,2], Shahzeb Shaheen[2], David Weinstein[2], Ranjit Padhy[2] and Md. Towhid Salam[2,3]

Abstract

Catatonia is a psychomotor syndrome resulting from an underlying psychiatric or medical disorder commonly observed in inpatient psychiatric units. While benzodiazepines and electroconvulsive therapy (ECT) are effective treatment options, the unavailability of ECT in many community psychiatric hospitals in the United States negatively affects patient outcomes. We present a 25-year-old African American male with a psychiatric diagnosis of schizophrenia complicated by malignant catatonia who was admitted to a community psychiatric hospital. He required intensive medical stabilization with supportive management, and transfer requests to ECT-equipped hospitals were initiated. While awaiting transfer for 148 days, the patient's symptoms did not fully remit with lorazepam (even with 36 mg daily in divided doses) and other psychotropic medication trials, including antipsychotics and mood stabilizers. After nearly 5 months of inpatient stay, he was successfully transferred, received ECT treatment, and experienced rapid resolution of catatonia. After discharge, to obtain three monthly sessions of maintenance ECT, he had 5-h one-way ground transportation arranged to an out-of-county ECT-equipped facility. There was no relapse in catatonia by the 2-year follow-up. This report highlights a significant healthcare disparity when attempting to manage severe catatonia within community hospital settings without access to ECT in the United States. Alternative treatments, including antipsychotics, had minimal impact on symptoms and possibly increased morbidity in this case while awaiting ECT. Treatment at our designated safety net hospital still required referral to 14 ECT-equipped hospitals before successful transfer. This case highlights the urgent need for ECT availability in more community hospitals to treat patients with refractory psychiatric conditions, including catatonia. ECT is an essential psychiatric treatment that, for certain conditions, has no appropriate alternatives. We propose that access to ECT be considered in the determination of safety net hospital systems, with improved ability to transfer patients who are suffering from treatable life-threatening mental health conditions.

Plain Language Summary

Challenges of Treating Catatonia without Access to Electroconvulsive Therapy

Catatonia is a complex psychiatric condition characterized by abnormal movements, behaviors, and withdrawal from regular activities. Electroconvulsive therapy (ECT) and benzodiazepines are first-line treatments for catatonia. However, ECT is not widely available, particularly in community mental health centers. We present a case of benzodiazepine-resistant catatonia that was initially treated at a community hospital that did not have access to ECT. We made a substantial number of referrals to ECT-equipped hospitals to transfer the patient; however, he was not able to be transferred until hospital day 148. The patient received ECT and experienced rapid resolution of symptoms. This report

[1]Noninvasive Neuromodulation Unit, Experimental Therapeutics Branch, Intramural Research Program, National Institute of Mental Health, Bethesda, MD, USA
[2]Department of Psychiatry, Kern Medical, Bakersfield, CA, USA
[3]Department of Population and Public Health Sciences, Keck School of Medicine of USC, University of Southern California, Los Angeles, CA, USA

Corresponding author:
Tyler Torrico, Department of Psychiatry, Kern Medical, 1700 Mount Vernon Avenue, Bakersfield, CA 93306, USA.
Email: TylerTorrico@gmail.com

highlights a significant healthcare disparity when attempting to manage catatonia within community hospital settings without access to ECT in the United States. ECT is an essential psychiatric treatment that, for certain conditions, has no appropriate alternatives. We propose that access to ECT be considered in the determination of safety net hospital systems, with improved ability to transfer patients who are suffering from treatable life-threatening mental health conditions.

Keywords
Community psychiatry, healthcare disparity, severe mental illness, amantadine, aripiprazole, case report

Date received: 1 March 2023; accepted: 24 November 2023

Background

Catatonia is a complex syndrome characterized by abnormal movements, behaviors, withdrawal from regular activities, and affective symptoms including aggression and fear. The causes of catatonia are diverse and broader than schizophrenia alone. Catatonia is frequently associated with bipolar spectrum illness, major depressive disorder, autism spectrum disorder, or underlying medical conditions;[1,2] however, this list is not exhaustive. Catatonia is common and likely under-recognized, with its incidence in the inpatient setting estimated to be between 5% and 20% among community and private hospital settings in the United States and other countries.[3–6] Optimizing outcomes in catatonia depends on prompt diagnosis and treatment, as well as treatment of the underlying cause. Failure to recognize and treat catatonia is associated with poor outcomes, especially malignant catatonia, which has an increased risk of fatality.[7]

Electroconvulsive therapy (ECT) and benzodiazepines are first-line treatments for catatonia. However, benzodiazepines have fair but inconsistent efficacy.[8,9] Despite this, many community psychiatry hospitals in the United States have no availability of ECT, creating a healthcare disparity for those with severe mental illness who often benefit the most from ECT.[10] We present an abbreviated clinical course of a patient with schizophrenia complicated by severe catatonia to elucidate the challenges faced in community psychiatric hospitals.

Case report

Psychiatric history and the baseline state of health

We describe a 25-year-old black male with an extensive psychiatric history, including schizophrenia (age of onset, 16 years), three previous catatonia episodes (resolved with lorazepam and antipsychotics), multiple psychiatric hospitalizations, and a substance use history of alcohol use disorder, and cannabis use disorder. The patient has no significant medical history. Due to the severity of schizophrenia, he previously had a year-long legally designated conservator to assist him with decision-making for his psychiatric care and housing; however, this expired 4 months before his hospital admission. The patient was clinically stable, living at home with his mother on an outpatient medication regimen which included haloperidol 5 mg twice daily, lorazepam 1 mg twice daily, benztropine 1 mg twice daily, clozapine 100 mg in the morning and 200 mg at night, and fluoxetine 20 mg daily. On presentation to the hospital, the patient weighed 95 kg.

Abbreviated catatonia clinical course and medication treatments

One week before hospitalization, the patient had alcohol intoxication and stopped taking psychotropic medications. A family member brought the patient to the emergency department, who reported that the patient had not been speaking, eating, or drinking for 4 days. The clinical presentation was consistent with malignant catatonia, as the patient was mute and exhibited staring behaviors; physical examination revealed mild muscle rigidity and vital signs were significant for tachycardia to 146 beats per minute (bpm), tachypnea at 30 breaths per minute, and blood pressure at 125/96 mmHg. On presentation, he scored 19 on Bush-Francis Catatonia Rating Scale (BFCRS). Laboratory studies significant for creatinine kinase (CK) elevated to 1426 unit/L. Lumbar puncture cerebrospinal fluid (CSF) analysis, brain magnetic resonance imaging (MRI), and electroencephalogram (EEG) were unremarkable and did not reveal any organic etiology. The patient was subsequently admitted to a community hospital that does not have ECT available as a treatment modality, although it was clinically indicated. In addition, clozapine was not able to be resumed due to the refusal of oral medications. Therefore, alternative treatments for catatonia were pursued while internal medicine simultaneously provided supportive management.

On admission, he was administered lorazepam 2 mg intravenous (IV) for catatonia. Despite continuing lorazepam 2 mg IV three times daily for three additional days, the patient developed new symptoms of spontaneous agitation, worsening muscle rigidity, urinary incontinence,

Table 1. Sequential referrals to transfer the patient to electroconvulsive therapy (ECT) equipped facilities.

Referral number	Hospital day	Facility type	California general location	Distance (miles)
1	33	Public hospital	Southern California	258
2	35	Public hospital	Southern California	156
3	35	Non-profit	Middle Central Valley	236
4	35	Public hospital	Southern California	111
5	35	Private	Bay Area	256
6	35	Private	Southern California	126
7	35	Non-profit	Middle Central Valley	276
8	36	Non-profit	Southern California	85
9	118	Non-profit	Southern California	101
10	121	Private	Bay Area	295
11	123	Private	Bay Area	265
12	125	Private	Southern California	174
13	125	Public hospital	Middle Central Valley	371
14[a]	127	Non-profit	Bay Area	277

[a]Indicates the accepting facility.

and diaphoresis. The patient's symptoms had slow general improvement over the following 9 days, with limited speech and normalizing vital signs with down-trending CK to 88 unit/L.

By hospital day 11, prominent symptoms of retarded type catatonia persisted. This included bizarre posturing, staring, and, most concerning, severe negativism with inanition. The patient had inconsistent adherence to oral lorazepam, and a legal petition to administer involuntary psychotropic medications through intramuscular (IM) and IV routes was granted by the court on the grounds of lack of capacity. Furthermore, a petition for conservatorship was pursued and granted during this hospitalization. On hospital day 11, spontaneous behavioral agitation became more concerning, and another attempt to treat underlying schizophrenia with clozapine was unsuccessful due to the refusal of oral medication. On day 17, due to increasing agitation (physical combativeness, use of four-point soft restraints for safety), chlorpromazine 50 mg four times daily was initiated, which was chosen to allow IM administration for refusal of oral formulation.

On hospital day 18, the patient had prominent agitation and received an as-needed dose of haloperidol 5 mg intramuscularly. The following day, severe muscle rigidity developed, and the patient became mute and incontinent of urine. The patient's health continued to decline with clinical concerns of reemerging malignant catatonia versus neuroleptic malignant syndrome (NMS), with the patient having tachycardia up to 164 bpm, tachypnea of 35 breaths per minute, and CK of 2398 units/L. The patient was administered increased supportive measures, which included IV fluids, foley catheterization, lorazepam 4 mg four times daily, and discontinuation of antipsychotics.

After the removal of antipsychotics from the treatment regimen, symptoms of malignant catatonia versus NMS resolved over a period of days with supportive management. However, severe retarded type catatonia persisted, which included severe withdrawal, at times including no oral intake for multiple days. By hospital day 33, due to a lack of response from continued titration of lorazepam (up to 16 mg daily), olanzapine titration was attempted as another alternative treatment, which was slowly titrated up to 20 mg daily. At this point in hospitalization, requests to transfer the patient to an ECT-equipped facility were initiated (Table 1).

The patient suffered another episode of malignant catatonia versus NMS, including CK peaking at 105,510 units/L on day 60. The patient received intensive supportive care, discontinuation of antipsychotics once again, and over a period of days, had a resolution of malignant catatonia versus NMS (Figure 1). Amantadine 100 mg three times daily was added as another alternative treatment for the patient's catatonia at this time.

The patient continued to suffer refractory retarded type catatonia but had acceptable oral intake and intermittent ability for brief conversations. On hospital day 77, aripiprazole 2.5 mg daily was started (titrated to 7.5 over 20 days), which was chosen because of its D2 partial agonism, which may be less likely to worsen catatonia or convert to malignant catatonia.[11,12] Due to refractory symptoms, lorazepam was titrated to 6 mg IV six times daily (36 mg daily) on hospital day 87.

Additional referrals to ECT-equipped facilities were initiated (Table 1). Through hospital day 145, the patient had no improvement but did not have any new medical compromises, and weekly laboratory studies were reassuring. On hospital day 146, the patient's blood pressures were slightly lower than the previous trend, including heart rates in the high 50s and blood pressures in the 100s over 60s. In response, lorazepam was decreased to 32 mg daily. On this day, the patient weighed 81 kg, having lost 14 kg (30.8 pounds) since the start of hospitalization.

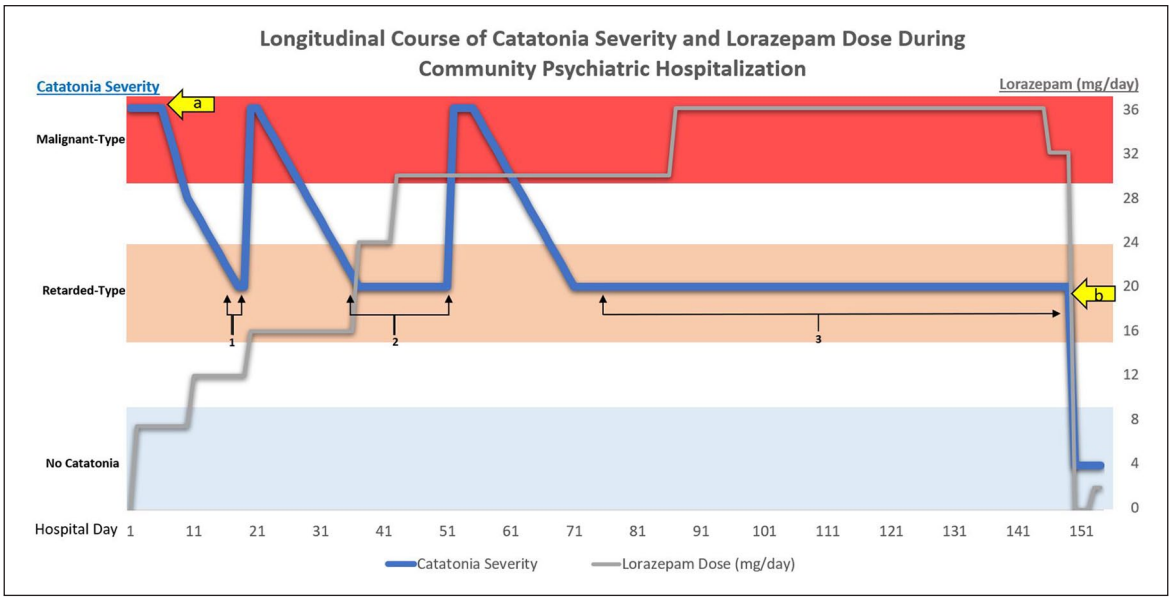

Figure 1. Malignant-type catatonia includes unstable vital signs and elevated creatinine kinase (CK). (1) indicates when chlorpromazine 50 mg four times daily was used and one administration of intramuscular haloperidol 5 mg. (2) Indicates olanzapine titration starting from 2.5 mg daily up to 30 mg. (3) indicates aripiprazole titration starting from 2.5 mg daily up to 7.5 mg daily. Low-dose aripiprazole is the only antipsychotic used that did not have a subsequent relapse into malignant-type catatonia. Yellow arrows: (a) indicates when electroconvulsive therapy (ECT) was a clinically indicated treatment and (b) indicates when ECT was administered.

Inpatient ECT course

On hospital day 148, the patient was finally accepted for transfer to an out-of-county hospital capable of providing ECT. This facility was located nearly 300 miles away from our hospital. The patient's conservator provided consent for ECT treatment. Over the following 4 weeks, he received three sessions weekly of bilateral ECT (pulse width 0.3 ms; 100 Hz; pulse duration 8 s; 800 mA, 383 mC). The patient tolerated ECT well and, during treatment, was administered methohexital 120 mg, succinylcholine 100 mg, flumazenil 0.4 mg, and midazolam 2 mg. Catatonia resolved quickly over these sessions, and the patient returned to normal speech and could attend to his activities of daily living.

Outpatient ECT and follow-up

After discharge, he returned to his home county and was recommended to obtain six sessions of monthly maintenance ECT. The patient's conservator arranged for ground transportation so the patient could attend these sessions, which included a 5-h one-way drive to each out-of-county session (approximately 300 miles). After 3 months, the patient continued to present clinically well, and in combination with considerations of the taxing transportation requirements, the last three sessions were canceled. There was no relapse in catatonia at the end of the 2-year follow-up, and the patient continues to be on a conservatorship status and has not required hospitalization or ECT since.

Discussion

This report presents a complex case of treatment-refractory catatonia with features of malignant and retarded subtypes. The prolonged hospital course in a community hospital with extensive morbidity highlights a significant healthcare disparity when attempting to manage severe catatonia in community hospital settings where ECT is unavailable. Inaccessibility to ECT led to a preventable increased disease burden and impaired opportunity to achieve optimal recovery. This is evident when considering that ECT was a clinically indicated treatment within the first 24 h of this hospitalization for this patient. Yet, it could not be administered to this patient until 148 days after admission despite attempts to transfer him to a facility where ECT was available.

In addition, due to the acuity of illness, multiple attempts were made to alleviate the patient's symptoms through alternative treatments. However, the quality of evidence for efficacy in alternative treatments is low.[11] These treatment decisions did not alleviate the symptoms and resulted in adverse effects. This case study highlights the challenges clinicians face in community hospitals when ECT is unavailable as a treatment modality; clinicians and patients are placed in challenging positions when access to the standard of care treatment is impaired.[13]

The American Psychiatric Association classifies ECT as an essential procedure, distinguishing it from an elective treatment.[14] It is important to note that the events described in this case report (prior to external transfer) occurred at a

safety net hospital for the state of California. Safety net hospitals in the United States are designated health centers that have legal obligation to provide medical care for individuals regardless of their insurance status or ability to pay. Generally, safety net hospitals are capable of providing most standard of care treatments or are able to transfer patients out to a higher level of care for a specific service within a reasonable time frame.[15] The inability to perform a similar transfer in a reasonable amount of time for a psychiatric condition highlights a significant disparity for this vulnerable population. Concerningly, catatonia is one of the few psychiatric conditions with risk of fatality. Another psychiatric condition with a high risk of fatality is suicide from untreated depression, which is also most effectively, efficaciously, and rapidly treated with ECT.[16] This emphasizes further that many patients who receive inpatient psychiatric hospitalization at community safety net hospitals also experience healthcare disparity from inability to access ECT. In general, neuromodulation for the treatment of psychiatric conditions, in both the inpatient and outpatient settings, has been less accessible to those without private health insurance or able to afford for out-of-pocket services.[17] In this case, we were unable to receive acceptance for transfer to an outside ECT-equipped facility until the 14th referral. There are at least 4411 hospitals in the United States that provide ECT (as of 2019), but the availability of the service is not evenly distributed, with most services provided in the Midwest.[18]

ECT was first utilized in 1938 by Ugo Cerletti[19] and Lucio Bini in Italy and used in the United States in 1940 by David Impastato.[20] The technique has been significantly refined in the following decades, with substantial improvements to its safety profile and reducing ECT-associated mortality complications.[21] Despite this, its use has generally declined.[22] Sensational media reports of "shock therapy" and movie and television coverage of ECT significantly impacted the American perception of ECT.[22] Furthermore, the rise of Scientology and antipsychiatry movements attacked ECT, deepening the American public's negative stigma about the regular use of ECT.[22] The potential for cognitive-related adverse reactions to ECT has also been a significant deterrent for ECT treatments. Simultaneously, the widespread development and use of psychotropic medications were held in a more favorable view and has left ECT use generally limited to academic and private institutions for multiple decades.[23] Electroconvulsive therapy use for inpatients in the United States has fallen dramatically in recent decades, and this decline is associated with the decreased capability of hospitals to conduct ECT internally.[24] Contemporary use and frequency of ECT practice vary worldwide, with many countries having higher utilization than the US.[25]

There are currently significant barriers to initiating an ECT service in the community setting in the United States, including a lack of well-trained psychiatrists and ECT practitioners, a lack of a champion within the institution, and a lack of physical space.[13] Due to the inaccessibility of ECT in some urban hospitals,[26] there have been investigations into the effectiveness of other psychotropics that may be able to treat catatonia. Medications such as amantadine, aripiprazole, valproate, and carbamazepine have been studied the most; but these have limited generalizability due to the observational nature of the reports.[11] High-quality, evidence-based treatment protocols for catatonia are yet to be developed.[27] Of note, even lorazepam (and other benzodiazepines) has failed to consistently provide relief of symptoms in controlled studies.[9,28] Furthermore, there is no clinical consensus about treatment protocols for utilizing benzodiazepines before and after ECT treatments.[29] There are previous reports of using up to lorazepam 30 mg daily for efficacious treatment of catatonia;[30] however, the patient we present did not respond to 36 mg daily.

There are some suggested treatment algorithms published that describe the treatment of catatonia; however, these algorithms are generally from observational reports which limit their generalizability.[11] Utilizing more medications to treat catatonia increases the risks for adverse reactions, which is of particular concern when adding antipsychotics. Catatonia is often observed in patients with schizophrenia, and the general recommendations for catatonia management include treatment of the underlying disorder.[31] However, using dopamine-blocking agents is associated with converting catatonia into the malignant type.[32,33] In this case, interestingly, olanzapine, chlorpromazine, and haloperidol appeared to precipitate malignant catatonia, but low doses of aripiprazole did not. It is unclear if this may be due to its mechanism of action as a D2 partial agonist, or if it is only because low doses were used in comparison to the other antipsychotic trials described in this case report.

Malignant catatonia presents with more pronounced vital sign abnormalities, including hyperthermia, tachycardia, tachypnea, blood pressure fluctuations, and severe muscle rigidity and breakdown, leading to elevated CK serum levels. NMS presents similarly with hyperthermia and rigidity. However, NMS typically presents after the administration of antipsychotics, while malignant catatonia generally has a behavioral prodrome of psychosis, retarded type catatonia, or agitation. Still, it can be difficult to distinguish these two disorders, with estimates of up to one-third of cases being indistinguishable.[34] So, managing catatonia in patients with schizophrenia poses another challenge to manage both conditions concurrently with medications as the risks of malignant catatonia and/or NMS could be higher where ECT could relatively safely address symptoms of both schizophrenia and catatonia within a reasonable timeline.

The prevalence of catatonia in inpatient psychiatric units ranges between 5% and 20%.[3–6] Luccarelli et al.[35]

analyzed the 2019 National Inpatient Sample, an all-payors database of acute care hospital discharges, and found 13,630 encounters for catatonia, with the total charges for those admissions reaching US$1.15 billion and a total of 215,165 cumulative hospital days (equally divided is approximately 16 days per encounter). From this, we anticipate that catatonia cases in the community setting are likely to have a significantly longer length of hospitalization compared to other inpatient psychiatric treatments. For comparison, the case we describe includes a hospitalization length of 148 days, while schizophrenia treatment is typically between 7.4 and 11.1 days, bipolar disorder treatment between 5.5 and 9.4 days, and depression treatment between 4.4 and 8.4 days.[36]

Multiple case reports documented similar challenges in treating catatonia without access to ECT when the condition is not responsive to benzodiazepines.[37–40] Notably, in studies of community-based programs for persons with severe mental illness, Fenton et al.[41] found that "hospitalization is the single largest cost element in the array of services needed to provide community care" and Luccarelli et al.[35] concluded that catatonia is a rare but costly discharge diagnosis. Although the cost of introducing ECT into community settings may have initial significant financial expenses, it is likely that ECT's potential to reduce hospitalization length may ultimately be a cost-effective approach that simultaneously provides improved care to community patients. The cost-effectiveness of ECT for treatment-resistant depression has similarly been established.[42,43] Of course, community hospitals with ECT continue to have their own challenges in treating their patient populations;[44] however, the implementation of ECT is ultimately likely to reduce patient morbidity in the community psychiatry setting.

Conclusion

Community psychiatrists, without the ability to administer or quickly transfer patients for ECT, are placed in difficult situations balancing clinical obligations to help patients, recognizing system-based resource limitations, and watching their patients experience healthcare disparity. In this case of severe catatonia, antipsychotics, amantadine, and lorazepam 36 mg daily could not successfully treat catatonia in this patient, whereas ECT could, and attempts to deliver maintenance ECT revealed further treatment barriers in this setting. Treatment at our designated safety net hospital still required referral to 14 ECT-equipped hospitals before successful transfer. This case highlights the urgent need for ECT availability in more community hospitals to treat patients with refractory psychiatric conditions, including catatonia. Electroconvulsive therapy is an essential psychiatric treatment that for certain conditions has no appropriate alternatives. We propose that access to ECT be considered in the determination of safety net hospital systems, with improved ability to transfer patients who are suffering from treatable life-threatening mental health conditions.

Declarations

Ethics approval and consent to participate

The study protocol (22084) was approved by the Kern Medical Institutional Review Board with written informed consent obtained.

Consent for publication

Written informed consent for publication was obtained from the patient, for the information and images included in this case report.

Author contributions

Tyler Torrico: Conceptualization; Data curation; Formal analysis; Investigation; Project administration; Writing—original draft; Writing—review and editing.
Shahzeb Shaheen: Conceptualization; Data curation; Writing—review and editing.
David Weinstein: Conceptualization; Formal analysis; Writing—review and editing.
Ranjit Padhy: Supervision; Writing—review and editing.
Md. Towhid Salam: Conceptualization; Methodology; Supervision; Writing—original draft; Writing—review and editing.

Acknowledgements

None.

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This research was supported (in part) by the Intramural Research Program of the NIMH (annual report no. ZIAMH002955).

Competing interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Availability of data and materials

The data displayed during this study are not publicly available as they are part of the protected health information of the patient described. They are available upon reasonable request and approval from the hospital authority.

ORCID iD

Tyler Torrico https://orcid.org/0000-0002-4817-8380

References

1. Taylor MA and Abrams R. Catatonia. Prevalence and importance in the manic phase of manic-depressive illness. *Arch Gen Psychiatry* 1977; 34(10): 1223–1225.
2. Takaoka K and Takata T. Catatonia in high-functioning autism spectrum disorders: case report and review of literature. *Psychol Rep* 2007; 101(3 pt 1): 961–969.
3. Taylor MA and Fink M. Catatonia in psychiatric classification: a home of its own. *Am J Psychiatry* 2003; 160(7): 1233–1241.
4. Van der Heijden FM, Tuinier S, Arts NJ, et al. Catatonia: disappeared or under-diagnosed? *Psychopathology* 2005; 38(1): 3–8.

5. Chalasani P, Healy D and Morriss R. Presentation and frequency of catatonia in new admissions to two acute psychiatric admission units in India and Wales. *Psychol Med* 2005; 35(11): 1667–1675.
6. Seethalakshmi R, Dhavale S, Suggu K, et al. Catatonic syndrome: importance of detection and treatment with lorazepam. *Ann Clin Psychiatry* 2008; 20(1): 5–8.
7. Connell J, Oldham M, Pandharipande P, et al. Malignant catatonia: a review for the intensivist. *J Intensive Care Med* 2023; 38(2): 137–150.
8. Luchini F, Medda P, Mariani MG, et al. Electroconvulsive therapy in catatonic patients: efficacy and predictors of response. *World J Psychiatry* 2015; 5(2): 182–192.
9. Zaman H, Gibson RC and Walcott G. Benzodiazepines for catatonia in people with schizophrenia or other serious mental illnesses. *Cochrane Database Syst Rev* 2019; 8(8): CD006570.
10. Demas ML. Electroconvulsive therapy and triaging during reduced access and the COVID-19 pandemic: a personal perspective. *J ECT* 2020; 36(4): 226–228.
11. Beach SR, Gomez-Bernal F, Huffman JC, et al. Alternative treatment strategies for catatonia: a systematic review. *Gen Hosp Psychiatry* 2017; 48: 1–19.
12. Huffman JC and Fricchione GL. Catatonia and psychosis in a patient with AIDS: treatment with lorazepam and aripiprazole. *J Clin Psychopharmacol* 2005; 25(5): 508–510.
13. Wilkinson ST, Kitay BM, Harper A, et al. Barriers to the implementation of Electroconvulsive Therapy (ECT): results from a nationwide survey of ECT practitioners. *Psychiatr Serv* 2021; 72(7): 752–757.
14. Maixner DF, Weiner R, Reti IM, et al. Electroconvulsive therapy is an essential procedure. *Am J Psychiatry* 2021; 178(5): 381–382.
15. Khoong EC, Cherian R, Rivadeneira NA, et al. Accurate measurement in California's safety-net health systems has gaps and barriers. *Health Aff* 2018; 37(11): 1760–1769.
16. Li M, Yao X, Sun L, et al. Effects of electroconvulsive therapy on depression and its potential mechanism. *Front Psychol* 2020; 11: 80.
17. Leiphart J, Barrett M and Shenai MB. Economic inequities in the application of neuromodulation devices. *Cureus* 2019; 11(9): e5685.
18. Patel RS, Sreeram V, Thakur T, et al. A national study for regional variation of inpatient ECT utilization from 4,411 hospitals across the United States. *Ann Clin Psychiatry* 2019; 31(3): 200–208.
19. Cerletti U. Old and new information about electroshock. *Am J Psychiatry* 1950; 107(2): 87–94.
20. Impastato DJ. The story of the electroshock treatment. *Am J Psychiatry* 1960; 116: 1113–1114.
21. Reti IM, Walker M, Pulia K, et al. Safety considerations for outpatient electroconvulsive therapy. *J Psychiatr Pract* 2012; 18(2): 130–136.
22. Lebensohn ZM. The history of electroconvulsive therapy in the United States and its place in American psychiatry: a personal memoir. *Compr Psychiatry* 1999; 40(3): 173–181.
23. Fink M. Die Geschichte der EKT in den Vereinigten Staaten in den letzten Jahrzehnten [History of electroconvulsive therapy in the United States in the last decades]. *Nervenarzt* 1993; 64(11): 689–695.
24. Case BG, Bertollo DN, Laska EM, et al. Declining use of electroconvulsive therapy in United States general hospitals. *Biol Psychiatry* 2013; 73(2): 119–126.
25. Leiknes KA, Jarosh-von Schweder L and Høie B. Contemporary use and practice of electroconvulsive therapy worldwide. *Brain Behav* 2012; 2(3): 283–344.
26. Hermann RC, Dorwart RA, Hoover CW, et al. Variation in ECT use in the United States. *Am J Psychiatry* 1995; 152(6): 869–875.
27. Pelzer AC, Van der Heijden FM and Den Boer E. Systematic review of catatonia treatment. *Neuropsychiatr Dis Treat* 2018; 14: 317–326.
28. Ungvari GS, Chiu HF, Chow LY, et al. Lorazepam for chronic catatonia: a randomized, double-blind, placebo-controlled cross-over study. *Psychopharmacology* 1999; 142(4): 393–398.
29. Delamarre L, Galvao F, Gohier B, et al. How much do benzodiazepines matter for electroconvulsive therapy in patients with major depression? *J ECT* 2019; 35(3): 184–188.
30. Pommepuy N and Januel D. La catatonie: résurgence d'un concept. Une revue de la littérature internationale [Catatonia: resurgence of a concept. A review of the international literature]. *Encephale* 2002; 28(6 pt 1): 481–492 (in French).
31. Fink M and Taylor MA. Catatonia: subtype or syndrome in DSM? *Am J Psychiatry* 2006; 163(11): 1875–1876.
32. Francis A. Catatonia: diagnosis, classification, and treatment. *Curr Psychiatry Rep* 2010; 12(3): 180–185.
33. Ohi K, Kuwata A, Shimada T, et al. Response to benzodiazepines and the clinical course in malignant catatonia associated with schizophrenia: a case report. *Medicine* 2017; 96(16): e6566.
34. Caroff SN, Mann SC and Keck PE Jr. Specific treatment of the neuroleptic malignant syndrome. *Biol Psychiatry* 1998; 44(6): 378–381.
35. Luccarelli J, Kalinich M, McCoy TH, et al. The occurrence of catatonia diagnosis in acute care hospitals in the United States: a national inpatient sample analysis. *Gen Hosp Psychiatry* 2022; 77: 141–146.
36. Stensland M, Watson PR and Grazier KL. An examination of costs, charges, and payments for inpatient psychiatric treatment in community hospitals. *Psychiatr Serv* 2012; 63(7): 666–671.
37. Oh J, Amber-Monta K, Sahota PC, et al. COVID-19-associated benzodiazepine-resistant catatonia responds to amantadine. *Prim Care Companion CNS Disord* 2022; 24(6): 22cr03394.
38. Lucchelli JP, Kourakou S, De Lucia Bové LP, et al. Lorazepam-resistant catatonia in an antipsychotic-naïve 24-year-old with psychotic symptoms. *Case Rep Psychiatry* 2020; 2020: 2469707.
39. Thomas C. Memantine and catatonic schizophrenia; letter to the editor. *Am J Psychiatry* 2005; 162(3): 626.

40. Carpenter SS, Hatchett AD and Fuller MA. Catatonic schizophrenia and the use of memantine. *Ann Pharmacother* 2006; 40(2): 344–346.
41. Fenton WS, Hoch JS, Herrell JM, et al. Cost and cost-effectiveness of hospital vs residential crisis care for patients who have serious mental illness. *Arch Gen Psychiatry* 2002; 59(4): 357–364.
42. Ross EL, Zivin K and Maixner DF. Cost-effectiveness of electroconvulsive therapy vs pharmacotherapy/psychotherapy for treatment-resistant depression in the United States. *JAMA Psychiatry* 2018; 75(7): 713–722.
43. Degerlund Maldi K, Asellus P, Myléus A, et al. Cost-utility analysis of esketamine and electroconvulsive therapy in adults with treatment-resistant depression. *BMC Psychiatry* 2021; 21(1): 610.
44. Prudic J, Olfson M, Marcus SC, et al. Effectiveness of electroconvulsive therapy in community settings. *Biol Psychiatry* 2004; 55(3): 301–312.