



ISSN: 2476-8642 (Print)

ISSN: 2536-6149 (Online)

www.annalsofhealthresearch.com

African Index Medicus, Crossref, African Journals
Online & Google Scholar

C.O.P.E & Directory of Open Access Journals

Annals of Health Research

IN THIS ISSUE



- Fetal Echocardiography Audit
- Diabetes-Related-Stress and Glycaemic Control
- Severity of Anaemia in Haemodialysis Care
- Professional Autonomy in Critical Care Nursing
- Plasma Lipids in Sickle Cell Anaemia
- Medication Adherence in Anti-Retroviral Therapy
- Benign Prostatic Hyperplasia
- Central Obesity in Diabetes mellitus
- Decision-Delivery-Interval in Caesarean Section
- Catatonia in Uraemia and Depression

**PUBLISHED BY THE MEDICAL
AND DENTAL CONSULTANTS ASSOCIATION
OF NIGERIA, OOUTH, SAGAMU, NIGERIA.**

www.mdcan.outh.org.ng

CASE SERIES

Catatonia in Renal Failure and Major Depressive Disorder: A Case Series

Fasesan OA*¹, Alakeji IA²

¹Benjamin Carson College of Medicine, Babcock University, Ilishan-Remo, Ogun State, Nigeria

²St Andrew's Healthcare, Northampton, England, United Kingdom

*Correspondence: Dr OA Fasesan, Benjamin Carson College of Medicine; Babcock University, Ilishan-Remo, Ilishan-Remo, Ogun State, Nigeria.
E-mail: fasesano@babcock.edu.ng; ORCID - <https://orcid.org/0000-0002-6138-0997>.

Summary

Catatonia is an acutely evolving and severe medical emergency. It occurs in several medical and psychiatric disorders but is often missed or misdiagnosed. There are majorly three different categories; the retarded, excited and malignant types. The latter is the most severe due to the associated autonomic instability. Much is unknown about the pathophysiology, but dysfunction of cortico-cortical modulation and a "top-down modulation" of the basal ganglia resulting from a deficiency of GABA in the cerebral cortex, resulting in motor dysfunction and glutamate hyperactivity and dopamine hypoactivity at the D2 receptor have been proposed. Benzodiazepines, especially Lorazepam, are reportedly effective, but reports of other benzodiazepines, Zopiclone and trials of mood stabilisers are documented. Electroconvulsive therapy is the suggested next line of action in case benzodiazepine fail, while the use of antipsychotic in catatonia is inconclusive. We present a case series of catatonia in 1) acute on chronic renal failure, complicated with uraemic encephalopathy and 2) severe depressive disorder with psychotic features. Physicians are encouraged to have a high index of suspicion to forestall the lethal complications.

Keywords: *Benzodiazepines, Catatonia, Depressive disorder, Electroconvulsive Therapy, Neuropsychiatric syndrome.*

Introduction

Catatonia is an acute, severe, and life-threatening neuropsychiatric syndrome with motor, vocal, behavioural, cognitive, and affective manifestations. ^[1, 2] Karl Kahlbaum first described catatonia in 1874 in his monograph "Die Katatonie oder das Spannungsirresein." ^[3] Due to its varying presentations and symptoms, catatonia has been underreported, creating the impression that it is a rare disorder. ^[2] Given the challenges encountered while making its diagnosis, underdiagnosing catatonia in mental illness or

general medical condition is inevitable. ^[1] Although the DSM-V tended to the under-recognition and discrepancies of catatonia in the DSM-IV, its presentation is still overlooked. ^[1]

The prevalence of catatonia differs among studies and patient populations. Psychopathologists quickly adopted Kahlbaum's terminology after he publicised the symptom profile documentation and distinction. Within a few years, the incidence of catatonia was 6-38% of hospitalised psychiatric illness, ^[2] whereas it ranged from 1.6-5% in patients with medical conditions. ^[4] In a more recent publication, 9-17% of patients with acute psychiatric illnesses have catatonia, with an

estimated prevalence among psychiatric inpatients of about 10%.^[5]

The pathophysiology of catatonia is not clear.^[6] However, neuroimaging suggests an altered ventricular-brain ratio, decreased regional cerebral blood flow in the inferior prefrontal cortex and right parietal cortex, bitemporal hypometabolism, and deficits in the orbitofrontal cortex.^[7] Georg Northoff proposed that catatonia is due to dysfunction of cortico-cortical modulation and a “top-down modulation” of the basal ganglia resulting from a deficiency of Gamma Butyric Benzoic Acid (GABA) in the cerebral cortex, resulting in motor dysfunction.^[7] This may explain the dramatic response to benzodiazepines since benzodiazepines increase cortical GABA activity. Georg Northoff also proposed glutamate hyperactivity and dopamine hypoactivity at the D2 receptor.^[7] Catatonia is associated with several psychiatric disorders, such as schizophrenia, bipolar disorder, depressive disorders, eating disorders, post-traumatic stress disorder, and medical conditions such as encephalitis, stroke, paraneoplastic syndrome, seizure disorders, and metabolic imbalances.^[8] Irrespective of the underlying disorder, catatonia can lead to a host of medical complications involving any organ,^[9] such as hypoglycaemia, malnutrition, dehydration, urinary retention, urinary tract infection, skin infections, decubitus ulcers, ocular complications, deep venous thrombosis, hepatocellular damage, laryngospasm, pneumonia, rhabdomyolysis, multiple sclerosis, uraemia, metabolic ketoacidosis, and acute renal failure - a frequent complication of catatonia.^[10]

The possibility of morbidity and mortality in catatonia creates a double risk in a patient with underlying renal failure and severe depressive disorder. A high index of suspicion is required to avoid missing the diagnosis or mistaking it for another condition. When promptly diagnosed, catatonia responds well to treatment with benzodiazepines, particularly

intravenous lorazepam, in about 70% of cases, and about 80% of severe and complicated cases respond to electroconvulsive therapy (ECT).^[11] Other medications have also been tried in catatonia.

We report a case series of catatonia in two Nigerians with background diagnoses of acute on chronic renal failure and major depressive disorder, respectively. These reports highlight the challenges we encountered in managing these patients, which are common in many centres, particularly challenges likely engaged in resource-deprived, low-income countries. To our knowledge, this is the first report of catatonia in renal failure from this region.

Case Description

Case 1

Our team of mental illness experts was invited to "rule out background psychotic illness" in a 38-year-old man (ME) in the medical ward of a tertiary hospital in southwest Nigeria. He was observed to have mutism, staring into space, uncooperative (refusing food and medications), and intermittent agitation with associated brief fragmented speech. He was admitted 40 hours earlier with a 6-month history of frothy urine without reduced output. About a month before the presentation, he was diagnosed, for the first time, with severe systemic hypertension at a referral centre. There was an associated elevated renal biochemical profile necessitating six sessions of haemodialysis before referral to our facility on account of a faulty haemodialysis machine. His sleep had been poor for four days before the presentation, waking intermittently and brooding over his medical condition. Six hours before the presentation, he suddenly woke out of sleep late at night and uttered a few words out of context, such as "death" and "afraid". This was followed by mutism and staring blankly. While on admission, he gradually became less responsive and later refused food and oral medications. All attempts to use the

intravenous route for drug administration resulted in severe agitation with aggressive tendencies. This and a preceding history of talking out of context informed the decision to invite the mental health team. There was no reported personal or family history of mental disorder.

On examination, he was conscious but mute, maintaining an awkward rigid posture in bed and immobile, with clenched fists, resisting passive movement and a pathological pillow was observed at some point. His face was expressionless, and he did not maintain eye contact. His renal biochemical profile was markedly deranged at presentation. Other medical evaluations included abdominopelvic ultrasound, electrocardiography, full blood count with differentials, blood sugar, erythrocytes sedimentation rate, urinalysis, HbsAg, and cranial computerized tomographic scan. The patient had features that were in keeping with an acute on chronic renal failure, complicated with uraemic encephalopathy and was duly managed.

We diagnosed catatonia secondary to metabolic imbalances, and intravenous or oral lorazepam 2mg *statum* then, 2mg daily was prescribed. Unfortunately, the patient got oral bromazepam late in the night due to the non-availability of lorazepam at the hospital pharmacy and being fully aware of the consequences of further delay, given the underlying renal failure. Shortly after, the patient slept off, waking up a few hours later with severe motor excitement, necessitating a 4-point physical restraint. Meanwhile, rigidity, mutism and posturing had resolved entirely. During a dialysis session, the patient had another episode of severe motor excitement later that day. We administered intravenous diazepam immediately because of the agitation to prevent harm to self and others, and more so, the patient refused to receive oral medications. The agitation abated after the drug administration, but he exhibited verbal aggressiveness.

In the evening of the same day, he became agitated, and his speech was out of context. He appeared confused. We could not ascertain the presence of visual hallucination, as he was uncooperative, but he expressed some persecutory ideas. We diagnosed delirium due to electrolyte imbalances, otherwise called Dysequilibrium syndrome. We gave a *statum* dose of intramuscular haloperidol 5mg and continued him on oral dosage of 2.5mg *nocte*. The behavioural symptoms resolved entirely by the third day of our intervention, and he became cooperative with the medical team. He responded well to treatments by the managing team and was discharged home on the eleventh day.

Case 2

VB, a 50-year-old married mother of two teenagers, was managed for severe depressive disorder with psychosis. She has had numerous reoccurrences of symptoms since the index episode 10 years ago. She was on oral risperidone, sertraline and intramuscular long-acting antipsychotics due to poor adherence to medications and psychotherapy in the previous hospital. VB, on several occasions, defaulted from follow-up and has been attended to by different medical consultants in other hospitals. While on admission, VB complained of forgetfulness, which could not be substantiated after carrying out some psychological testing. Hence, she was considered to have pseudodementia due to the underlying history of depression. VB also had some religious preoccupation, claiming that rapture would soon take place and she would likely go to hell. During her last episode, VB presented to the emergency room with confused speech, restlessness, poor appetite, and poor sleep and was noticed to have an elevated blood pressure, 220/106mmHg. She was managed as a case of hypertensive emergency with background severe depression and was placed on oral Nifedipine 30mg XL *statum*. The attending medical casualty officer requested investigations such as echocardiography, electrocardiography and chest radiography.

The patient was referred to the cardiologist with the results of the requested investigations. The psychiatrist was invited, while VD was admitted after evaluation.

About four days into the admission, VB was observed to be selectively mute but responded mostly with non-verbal cues when asked questions. By the 6th day, she was met lying still in bed, non-responsive, rigid, and refusing food and medications. Catatonia was diagnosed in a patient with an underlying major depressive disorder. She was commenced on intravenous diazepam 10mg stat and 8-hourly intravenous infusions with added Vitamin B-complex. She was to be nursed in the left lateral position, and two-hourly vital signs monitoring was also ordered. By the following day, she was still mute, refusing food and lying still in bed, although occasionally changing position by herself. Due to the significant deteriorating clinical state (patient became completely mute and was no longer responding to verbal cues), intramuscular haloperidol 10mg *statum*, intramuscular biperidine 5mg *statum* for extrapyramidal side effects and intravenous diazepam 10mg 8-hourly were administered. Nursing care was ensured, including observing for rigidity, autonomic instability and hyperthermia. The next day, she was met sitting down on the bed without support and was being fed. She could respond verbally to questions, although she still had obvious hypokinesia. Intravenous diazepam 20mg *statum*, intramuscular haloperidol 10mg *statum*, and intramuscular biperidine 5mg *statum* were repeated following the initial assessment.

On the 8th day of admission, VB had improved significantly, as shown by better speech and movement and resolving hypokinesia. Parenteral medications were discontinued, and oral diazepam was commenced at 10mg three times daily and was subsequently tapered off. Oral Vitamin E 1000iu alongside other medications (Sertraline, Risperidone, Cognitol, Epilim, Biperidine, Vit B-complex) were recommenced. Intravenous fluid was

discontinued on the ninth day of admission. Family therapy and extensive psychoeducation were performed, and VB was discharged to the outpatient clinic on the 13th day of admission.

Discussion

Catatonia was a subtype of schizophrenia, later identified as a complex psychomotor disorder. Catatonia develops in other mental health diseases, such as substance-induced, brief psychotic, schizophreniform, and schizoaffective disorders. [12] Catatonia can be diagnosed independently of any syndromes or disorders, although it is usually associated with bipolar disorder. [13] Catatonia can be categorised into three subtypes: the retarded, excited, and malignant catatonia. Retarded catatonia is the more frequently observed subtype. [14] It characteristically presents with a paucity of movement, such as immobility, staring, mutism, rigidity, withdrawal, and refusal to eat, as well as bizarre features in posturing, grimacing, negativism, waxy flexibility, echolalia or echopraxia, stereotypy, verbigeration, and automatic obedience. [5] On the other hand, the hallmark symptom of excited catatonia is severe psychomotor agitation, [15] combativeness, or even delirium [16] with the potential to cause harm to the patient or others. [17] The last type, malignant catatonia, is dangerous and associated with autonomic instability. [18] It may be present in the neuroleptic malignant syndrome and can indicate the lethality of the underlying cause. Malignant catatonia can progress rapidly within days [18] with life-threatening complications such as hyperthermia, altered consciousness, and autonomic dysfunction. Therefore, there is a need for clinicians to have a high index of suspicion and act promptly to forestall the rapid evolution.

Periodic catatonia, another subtype but not declared officially, is a rare form of catatonia where symptoms occur in phases and can resolve completely in between episodes. Like

the others, it poses a diagnostic challenge for clinicians because of its disappearance before treatment. [19]

The immobility, mutism, and rigidity in Case 1 raised the suspicion of the junior resident, who went ahead to elicit other features of catatonia. The first challenge he encountered was the audacity to pen down this diagnosis, knowing that he had to convince his superiors that he indeed identified this acclaimed 'exiled' syndrome. This was so because, in recent times, there is a general assumption that catatonia is rare following the introduction of antipsychotics. This could be because the symptoms when present in nonpsychotic mental disorders and other medical conditions are usually overlooked. Catatonia is not rare, though its incidence seems to have reduced. [20] Research has shown that catatonic symptoms or a higher risk of catatonia are associated with certain general medical conditions. Several documented medical case reports manifesting with catatonia include hyponatremia, cerebral venous sinus thrombosis, liver transplantation, renal conditions, strokes, neoplasms, infections, autoimmune disorders, neurodegenerative diseases, metabolic derangements, and certain drugs, among others. [10, 21] Other medical causes include meningitis, encephalitis, systemic bacterial, viral, or fungal infections and autoimmune processes, particularly N-methyl-D-aspartate receptor (NMDAR) encephalitis and systemic lupus erythematosus (SLE). [22]

The experience of the authors with Case 1 highlights the possibility of many missed or misdiagnosed cases of catatonia, particularly in medically ill individuals. This case was an exception because of the associated history of talking out of context and aggressive tendencies that prompted the medical team to invite the psychiatrists over. In addition, the availability of psychiatric services at the centre, and more importantly, the high index of suspicion of the psychiatry team ensured that the case was not mistaken for some other

differential diagnoses. The possibility of severe uraemic encephalopathy was not entertained as the catatonic symptoms resolved with the administration of benzodiazepines. We treated with the available benzodiazepines - bromazepam and diazepam. We would have used lorazepam, but this was not available.

While managing Case 2, we were also faced with the challenge of a lack of availability of lorazepam. Therefore, we commenced diazepam, but some hours afterwards, her clinical state deteriorated significantly despite administering 30mg of diazepam in divided doses while watching out for signs of respiratory emergency. This prompted us to commence antipsychotics. Further, she was given anticholinergic (Biperidine) to negate the extrapyramidal side effects of antipsychotics. There was appreciable improvement in less than twelve hours, and in a few days, she was discharged to the outpatient clinic.

Benzodiazepines are the drugs of choice for treating catatonia, rather than antipsychotics, irrespective of the cause, even in cases of catatonia due to other medical conditions. [5, 23] Prolonged benzodiazepine use increases GABA activity, but the risk of developing catatonia increases when benzodiazepines or clozapine are withdrawn [23] due to increased excitatory neurotransmission. [24] Despite this reaction, benzodiazepines are still the main form of treatment for catatonia. [1] Due to the swift onset of action, the penchant for the GABA-A receptor and the elongated duration of effect, intravenous lorazepam is favoured over other routes and other benzodiazepines. The role of antipsychotics in catatonia is controversial and inconclusive. There is heterogeneity in the response of catatonia to treatment, and there is a possibility that the aetiology of catatonia differs depending on the presentations. [25] Contrary to traditional guidelines, some catatonic conditions respond favourably to antipsychotics, as reported in Case 2. Certain studies reported that antipsychotics, especially the first generation, like haloperidol, also cause

catatonia.^[26] Some proved the efficacy of both the first and second generations ^[27] and others substantiated the need for antipsychotics in recurrent affective psychoses. ^[28]

There are some reports that second-generation antipsychotics reduce catatonia, cause catatonia, or precipitate NMS in some patients. ^[29] Second-generation antipsychotics like clozapine, olanzapine, risperidone, quetiapine and clozapine are effective in managing some catatonic schizophrenia.^[30,31] Other studies have reported the limitation in the effectiveness of the use of Lorazepam or electroconvulsive therapy (ECT) alone on catatonia in a controlled study of patients with chronic schizophrenia or chronic psychotic disorders.^[32] In a case of catatonia reported by Caroff et al., lorazepam was not effective during acute episodes of catatonia. Still, when combined with parenteral haloperidol, the frequent relapses resolved just as we did. ^[33] Also, Girish and Gill showed greater improvement in psychotic and catatonic symptoms from ECT compared with risperidone in a randomised trial of acute catatonic schizophrenia.^[34] Several authors published data suggesting that antipsychotic response varies depending on the clinical presentation and duration of symptoms; acute catatonic schizophrenia has a favourable prognosis and response to antipsychotic treatment,^[35] whereas catatonia correlated with severity, poor treatment response, and chronicity of schizophrenia in other studies. ^[36]

There is still a dearth of information on the treatment of catatonia, but the clinical evidence of the efficacy of benzodiazepines, such as lorazepam, and electroconvulsive therapy is overwhelming. ^[13] In management, the culture of thorough physical examination should be encouraged among medical and psychiatric residents, and all physicians should keep a high index of suspicion on catatonia, as it is associated with life-threatening complications, as stated above. There are several designed scales to assist in identifying it and for monitoring response to treatment. These include the Bush-Francis Catatonia Rating

Scale (BF CRS), the Modified Roger's Rating Scale (MRRS), and the Bush-Francis Catatonia Screening Instrument (BFCSI). ^[37] The Lorazepam Challenge test is a recommended invaluable tool. The *statum* dose of 1mg intravenous lorazepam should be administered while observing the patient for response; if there is no observable response after 5 minutes, another 1mg intravenous dose is administered. ^[38] If there is a positive response, the patient is treated with higher doses of lorazepam. ^[27] If negative, ECT or antipsychotics are advised. ^[39]

For Case 1, it was difficult to rule out the possibility of an underlying delirium, as noted by DSM-V that catatonic disorder due to another medical condition cannot occur exclusively during a delirium. As soon as the catatonia resolved, Case 1 met the criteria for delirium, which necessitated a course of low-dose antipsychotics. A recent study found that 30.2% of patients with delirium met the criteria for catatonia using BF CRS, while 12.7% met the criteria for catatonia using the DSM-V criteria.^[49] This exclusion needs to be reviewed as it does not appear evidence-based.

Conclusion

Catatonia is a complicated psychomotor disorder that occurs in psychiatric disorders and several other medical problems. Catatonia is a potentially fatal under-recognized and under-diagnosed disorder that mimics other medical conditions. Although benzodiazepines are the preferred medications for the treatment of catatonia, antipsychotics have been shown to be beneficial in circumstances where benzodiazepines failed. Clinicians must, therefore, maintain a high level of suspicion and act expeditiously to save lives in suspected cases of catatonia.

Acknowledgement: The authors acknowledge the patients and their relatives for their consent to use their data for this work.

Authors' Contributions: Both authors conceived and designed the study, did a literature review and

drafted the manuscript. FOA revised the draft for sound intellectual content. Both authors approved the final version of the manuscript.

Conflicting Interests: None.

Funding: Self-funded.

Publication History: Submitted 21 May 2023; Accepted 30 August 2023.

References

1. Edinoff AN, Kaufman SE, Hollier JW, Virgen CG, Karam CA, Malone GW, *et al.* Catatonia: Clinical Overview of the Diagnosis, Treatment, and Clinical Challenges. *Neurol Int* 2021;13:570-586. <https://doi.org/10.3390/neurolint13040057>.
2. Fink M. Catatonia: A Syndrome Appears, Disappears, and is Rediscovered. *Can J Psychiatry* 2009;54:437-445.
3. Kahlbaum KL. Die Katatonie oder das Spannungsirresein. 1874. Bernin: Verlag August Hirschwald; 1874.
4. Jaimes-Albornoz W, Serra-Mestres J. Prevalence and clinical correlations of catatonia in older adults referred to a liaison psychiatry service in a general hospital. *Gen Hosp Psychiatry* 2013;35:512-516.
5. Rasmussen SA, Mazurek MF, Rosebush PI. Catatonia: Our current understanding of its diagnosis, treatment and pathophysiology. *World J Psychiatr* 2016;6:391-398. <http://dx.doi.org/10.5498/wjp.v6.i4.391>.
6. Sienaert P, Dhossche DM, Vancampfort D, Hert MD, Gazdag G. A clinical review of the treatment of catatonia. *Frontiers Psychiatry* 2014; 5. <https://doi.org/10.3389/fpsy.2014.00181>
7. Northoff G. What catatonia can tell us about “top-down modulation”: a neuropsychiatric hypothesis. *Behav Brain Sci* 2002; 25: 555-604.
8. Takaoka K, Takata T. Catatonia in childhood and adolescence. *Psychiatry Clin Neurosci* 2003;57:129-137.
9. Reddick B, Stem TA. Catatonia, neuroleptic malignant syndrome, and serotonin syndrome. In: Stem TA, Herman JB, Eds. *Massachusetts General Hospital Psychiatry Update & Board preparation, 2nd Ed.* New York, NY: McGraw-Hill; 2004:217-224.
10. Fekete R. Renal failure in dementia with Lewy bodies presenting as catatonia. *Case Rep Neurol*. 2013;5:10-13. <https://doi.org/10.1159/000346594>.
11. Levenson JL, Pandurangi AK: Prognosis and complications. In *catatonia: from psychopathology to neurobiology* Edited by: Caroff SN, Mann SC, Francis A, Fricchione GL. Washington, DC: American Psychiatric Publishing. 2004. p. 161-172.
12. Tandon R, Heckers S, Bustillo J, Barch DM, Gaebel W, Gur RE, *et al.* Catatonia in DSM-5. *Schizophr Res* 2013;150:26-30. <https://doi.org/10.1016/j.schres.2013.04.034>.
13. Fink M. Rediscovering catatonia: The biography of a treatable syndrome. *Acta Psychiatr Scand* 2013;127:1-47. <https://doi.org/10.1111/acps.12038>.
14. Medda P, Toni C, Luchini F, Giorgi Mariani M, Mauri M, Perugi G. Catatonia in 26 patients with bipolar disorder: clinical features and response to electroconvulsive therapy. *Bipolar Disord* 2015;17:892-901.
15. Morrison JR. Catatonia. Retarded and excited types. *Arch Gen Psychiatry* 1973;28:39-41. <https://doi.org/10.1001/archpsyc.1973.01750310023005>.
16. Bhati MT, Datto CJ, O'Reardon JP. Clinical Manifestations, Diagnosis, and Empirical Treatments for Catatonia. *Psychiatry Edgmont Townsh.* 2007;4:46-52.
17. Fink M., Taylor M.A. The Catatonia Syndrome: Forgotten but Not Gone. *Arch Gen Psychiatry* 2009;66:1173. <https://doi.org/10.1001/archgenpsychiatry.2009.141>.

18. Northoff G, Wenke J, Pflug B. Increase of serum creatine phosphokinase in catatonia: An investigation in 32 acute catatonic patients. *Psychol Med* 1996;26:547-553. <https://doi.org/10.1017/S0033291700035625>.
19. Ghaffarinejad A.R., Sadeghi M.M., Estilae F., Zokaee Z. Periodic catatonia. Challenging diagnosis for psychiatrists. *Neurosci Riyadh Saudi Arab* 2012;17:156-158.
20. Hawkins JM, Archer KJ, Strakowski SM, Keck PE. Somatic treatment of catatonia. *Int J Psychiatry Med* 1995;25:345-369.
21. Oldham M.A. The Probability That Catatonia in the Hospital has a Medical Cause and the Relative Proportions of Its Causes: A Systematic Review. *Psychosomatics* 2018;59:333-340. <https://doi.org/10.1016/j.psych.2018.04.001>.
22. Rogers JA, Pollak T, Blackman G, David A. Catatonia and the immune system: A review. *Lancet Psychiatry* 2019;6:620-630. [https://doi.org/10.1016/S2215-0366\(19\)30190-7](https://doi.org/10.1016/S2215-0366(19)30190-7).
23. Rosebush PI, MacQueen GM, Clarke JT, Callahan JW, Strasberg PM, Mazurek MF. Late-onset Tay-Sachs disease presenting as catatonic schizophrenia: diagnostic and treatment issues. *J Clin Psychiatry* 1995; 56:347-353.
24. Lander M, Bastiampillai T, Sareen J. Review of withdrawal catatonia: What does this reveal about clozapine? *Transl Psychiatry* 2018;8:139. <https://doi.org/10.1038/s41398-018-0192-9>.
25. Ungvari GS, Caroff SN, Gerevich J. The Catatonia Conundrum: Evidence of Psychomotor Phenomena as a Symptom Dimension in Psychotic Disorders. *Schizophrenia Bulletin* 2010;36:231-238. <https://doi.org/10.1093/schbul/sbp105>.
26. Jabs BE, Pfuhlmann B, Bartsch AJ, Cetkovich-Bakmas MG, Stöber G. Cycloid psychoses-from clinical concepts to biological foundations. *J Neural Transm* 2002;109:907-919. <https://doi.org/10.1007/s007020200074>.
27. Peralta V, Campos MS, de Jalon EG, Cuesta MJ. DSM-IV catatonia signs and criteria in first-episode, drug-naive, psychotic patients: psychometric validity and response to antipsychotic medication. *Schizophr Res* 2010;118:168-175. <https://doi.org/10.1016/j.schres.2009.12.023>.
28. Muneer A. Aripiprazole in the treatment of refractory mood disorders: A case series. *Clin Psychopharmacol Neurosci* 2014;12:157-159. <https://doi.org/10.9758/cpn.2014.12.2.157>.
29. Van Den Eede F, Van Hecke J, Van Dalen A, Van den Bossche B, Cosyns P, Sabbe BG. The use of atypical antipsychotics in the treatment of catatonia. *Eur Psychiatry* 2005;20:422-429.
30. Yoshimura B, Hirota T, Takaki M, Kishi Y. Is quetiapine suitable for treatment of acute schizophrenia with catatonic stupor? A case series of 39 patients. *Neuropsychiatr Dis Treat* 2013;9:1565-1571.
31. Cieslak K, Feingold J, Antonius D, Walsh-Messinger J, Dracxler R, Rosedale M, *et al.* Low vitamin D levels predict clinical features of schizophrenia. *Schizophr Res* 2014;159:543-545.
32. Caroff SN, Hurford I, Bleier HR, Gorton GE, Campbell EC. Recurrent Idiopathic Catatonia: Implications beyond the Diagnostic and Statistical Manual of Mental Disorders 5th Edition. *Clin Psychopharmacol Neurosci* 2015;13:218-221. <https://doi.org/10.9758/cpn.2015.13.2.218>.
33. Girish K, Gill NS. Electroconvulsive therapy in lorazepam non-responsive catatonia. *Indian J Psychiatry* 2003;45:21-25.

34. Stompe T, Ortwein-Swoboda G, Ritter K, Schanda H, Friedmann A. Are we witnessing the disappearance of catatonic schizophrenia? *Compr Psychiatry* 2002;43:167-174.
35. Mimica N, Folnegovic-Smalc V, Folnegovic Z. Catatonic schizophrenia in Croatia. *Eur Arch Psychiatry Clin Neurosci* 2001;251:117-120.
36. Bush G, Fink M, Petrides G, Dowling F, Francis A. Catatonia. I. Rating scale and standardised examination. *Acta Psychiatr Scandi* 1996;93:129-136.
37. Dhossche DM, Watchel LE. Catatonia in psychiatric illnesses. In: Fatemi SH, Clayton PJ, Editors. *The Medical Basis of Psychiatry*. Totowa, NJ: Humana Press 2008.
38. Zisselman MH, Jaffe RL. ECT in the treatment of a patient with catatonia: consent and complications. *Am J Psychiatry* 2010;167:127-132.
39. Grover S, Ghosh A, Ghormode D. Do patients of Delirium have catatonic features? An exploratory study. *Psychiatry Clin Neurosci* 2014;68:644-651.



This open-access document is licensed for distribution under the terms and conditions of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by-nc/4.0>). This permits unrestricted, non-commercial use, reproduction and distribution in any medium provided the original source is adequately cited and credited.

Erratum

In the article titled “**A retrospective analysis of eclampsia as a major cause of maternal and perinatal mortality in Sagamu**” published in *Annals of Health Research* 2015; 1(2): 68-73, the authors were listed as Sotunsa JO, Inofomoh AI, Akinseku AK, Ani FI and Sule-Odu AO.

Our attention has been drawn to the error in one of the names and we wish to correct the names of the authors listed on the article as “Sotunsa JO, Inofomoh AI, **Akiseku AK**, Ani FI and Sule-Odu AO”.

-Editor-in-Chief