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Case Report and Literature Review

Atypical First-Episode Psychosis with Prolonged Catatonia and Neuroleptic Malignant Syndrome: Diagnostic Challenges and Improvement with Clozapine: A Case Report and Literature Review

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Abstract:
Introduction: Schizophrenia is a long-term mental illness that usually shows typical positive symptoms, but some patients have unusual symptoms that make diagnosis and treatment difficult. Catatonia and neuroleptic malignant syndrome (NMS) can occur in schizophrenia, yet atypical early-onset cases are not well described. This case explores the first episode of atypical psychosis with prolonged catatonia and NMS, focusing on diagnostic challenges and response to clozapine.

This case report describes a 24-year-old Hispanic male, admitted with unclear depression, suicidal thoughts, disorganized behavior, and unclear psychosis, without hallucinations or delusions. Later developed prolonged catatonia overlapped with NMS following treatment with antipsychotics. Extensive medical, neurologic, autoimmune, and infectious evaluations were negative. Multiple treatments, including benzodiazepines and several antipsychotics, resulted in minimal improvement. After failure of standard treatments for five months, clozapine was started and slowly increased, following guidelines. The patient showed marked improvement, including improved speech, behavior, affect, and participation in therapy. No significant adverse effects were reported.

Methods: We developed and searched keywords in multiple databases (PubMed, Google Scholar, etc.) after obtaining the patient's informed written consent. We then reviewed the literature thoroughly.

Results: This case showed that first-episode psychosis with prolonged catatonia and overlapping NMS, with atypical psychosis, is hard to diagnose, did not respond to standard treatments, and improved significantly only with clozapine.

Conclusion: This case highlights the need to rule out medical and neurological causes in atypical first-episode schizophrenia and suggests earlier clozapine use and more research on pathophysiology to guide treatment of complex cases to reduce healthcare costs.

Background: Schizophrenia is a chronic, debilitating psychiatric illness that affects 1% of the global population[1,2,3]. It is more prevalent in males than in females and occurs mostly during late adolescence to early adulthood[1,2,3]. Schizophrenia ranked among the top 10 causes of global disability and causes increased premature mortality[1,2,3]. The concept of schizophrenia has changed over time and is still evolving. In the late 1800s, Emil Kraepelin described the illness as "dementia praecox," believing it was one disease that started early in life and led to progressive mental decline[4]. However, later, Eugen Bleuler renamed it schizophrenia and argued that it was not one single disease but a group of related disorders, defined mainly by core problems in thinking, emotions, and social connection rather than inevitable deterioration[4]. Over time, psychiatrists added subtypes and symptoms to improve diagnosis, but no clear brain marker or single gene has been found for schizophrenia, and the research is still ongoing [4].

The course of Schizophrenia follows three stages: a) prodromal stage, which is associated with social withdrawal and baseline function declining, b) active stage, which is characterized by full-blown psychotic features, and c) residual stage, which consists of negative symptoms and cognitive deficits[5]. According to the *Diagnostic and Statistical Manual of Mental Disorders,* Fifth Edition *(DSM-5)*, Schizophrenia is diagnosed with certain criteria; presence of two or more of the active phase symptoms such as delusions, hallucinations, disorganized speech, grossly disorganized or catatonic behavior and negative symptoms for a significant portion of a time during a one-month period with the persistence of signs of disturbance for at least six months associated with functional impairment in social, academic and occupational fields[6,7].

Studies found that around one-third of people with schizophrenia do not respond well to at least two standard antipsychotic medications and are considered to have treatment-resistant schizophrenia (TRS)[8,9]. TRS can be present from the first episode of schizophrenia, or it may develop later as the illness progresses[8,9].

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About 40% of patients with treatment-resistant schizophrenia respond to clozapine, and current evidence suggests it is the most effective treatment when other antipsychotics fail[8-10]. Only a few studies have looked at clozapine as a first-line treatment. While it has been tested early in illness, research is limited and does not clearly show better long-term outcomes compared with other antipsychotics[10].

Catatonia is a complex neuropsychiatric condition, characterized by psychomotor abnormalities that range from hypoactivity to hyperactivity, with a prevalence of 4-15% among schizophrenic patients[11-13]. The diagnostic tool for catatonia is the 23-item Bush-Francis Catatonia Rating Scale (BFCRS), rated on a Likert scale of 0-3[14]. In DSM-5, catatonia is removed as a schizophrenia subtype because catatonia occurs across many conditions and is not specific to schizophrenia and is better recognized and treated when diagnosed using a single set of criteria as a specifier[15]. During treatment of schizophrenia, a life-threatening complication called neuroleptic malignant syndrome (NMS) can occur, characterized by altered mental status (AMS), fever, muscle rigidity, and autonomic instability. NMS is associated with the use of high-potency typical antipsychotic agents but can also be associated with low-potency and atypical antipsychotic agents[16]. Previous research studies discussed the overlapping features of catatonia and NMS following the use of neuroleptic agents; however, there is limited information on atypical presentations of early onset of schizophrenia with minimal positive symptoms, along with prolonged catatonia and overlapping features of NMS[17,18]. This case report aims to focus on these diagnostic challenges, emphasize the need to rule out medical and neurologic causes, and show the role of Clozapine in improving symptoms in atypical and treatment-resistant first-episode schizophrenia.

Keywords: Schizophrenia, psychotic disorder, DSM-5, catatonia, NMS, neuroinflammation, antipsychotics, clozapine.

Methodology: Written informed consent was obtained from the patient prior to preparation of this case report. A focused narrative literature review was conducted to provide clinical context regarding atypical first-episode psychosis, catatonia, neuroleptic malignant syndrome (NMS), and clozapine treatment. Peer-reviewed articles were identified primarily through PubMed and Google Scholar using relevant keywords.

Case Presentation:

Patient Background

A 24-year-old Hispanic male with no known past psychiatric or medical history was brought to the emergency department for worsening disorganized speech and behavior, poor self-care, possible depression, and unclear psychosis. He did not clearly endorse hallucinations or fixed delusions at presentation. Urine drug screen was positive for cannabis. Initial CT head, CT abdomen, and chest X-ray were unremarkable.

The patient appeared disheveled, minimally verbal, with flat affect and severe thought disorganization. A complete mental status examination was limited throughout much of the hospitalization due to mutism, agitation, and catatonic features.

Initial Psychiatric Admission and Development of Catatonia

The patient was initially admitted to the acute unit of a psychiatric hospital. He initially refused medications, and a court order was obtained. He was started on haloperidol short-acting IM, titrated to 10 mg twice daily. After approximately one week, he developed worsening psychomotor slowing, mutism, posturing, rigidity, and negativism. His Bush–Francis Catatonia Rating Scale (BFCRS) ranged from 12 to 16, consistent with moderate catatonia.

An oral lorazepam challenge test was initiated; however, within days, he developed autonomic instability, altered mental status, fever, elevated creatine kinase (CK), leukocytosis, and rigidity. He was transferred to the hospital's medical floor for further management.

Medical Floor Course: Catatonia and NMS Overlap and Diagnostic Workup

On the medical floor, lorazepam IV was administered at 2 mg every 6 hours as a catatonia challenge, later reduced to 1 mg due to excessive sedation. Partial improvement in rigidity was noted, but the patient remained largely mute, agitated, and intermittently combative.

He developed features concerning NMS, including hyperthermia, severe rigidity, autonomic instability (heart rate 130–160 bpm), elevated CK, transaminitis, and leukocytosis. Antipsychotics were discontinued, and he was treated with IV 60 mg Q6H dantrolene for three days, leading to improvement in rigidity and laboratory abnormalities. However, catatonia persisted.

Due to ongoing agitation and line-pulling, short trials of atypical antipsychotics were cautiously attempted after initial NMS resolution, including olanzapine and ziprasidone, each given sequentially at low doses. These trials provided minimal benefit and were again followed by recurrence of NMS-like symptoms, requiring repeat discontinuation of antipsychotics and another short course of dantrolene.

Given prolonged catatonia and hyperreflexia with clonus, autoimmune encephalitis and neurologic causes were considered. Neurology and infectious disease were consulted. Extensive testing, including ANA (positive), HIV, syphilis, metabolic studies, EEG, and inflammatory markers, was performed; results were negative. Lumbar puncture and brain MRI were attempted but were initially unsuccessful due to extreme agitation. The patient was ultimately intubated to safely complete both procedures; the results were negative. A steroid trial was attempted with minimal clinical response.

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The patient remained on the medical floor for approximately 3 months with persistent catatonia and agitation, requiring continuous lorazepam treatment. During this time, he also tested positive for COVID-19, possibly associated with the extended hospitalization period, which further delayed transfer back to psychiatry. Throughout his stay on the medical floor, the patient was seen each day by a psychiatric consultation team.

Return to the Psychiatry Unit and Treatment Resistance

After medical stabilization and clearance of COVID-19, the patient was transferred to the psychiatric unit (including the geriatric unit for higher staffing needs). He continued to display catatonia, agitation, and disorganized behavior. Lorazepam was continued (IV on the medical floor, then transitioned to oral 2 mg every 6 hours PRN in psychiatric unit), totaling approximately six months of benzodiazepine exposure.

Trials of bupropion XL 150 mg daily, valproic acid 500 mg BID, and olanzapine (up to 7.5 mg AM and 10 mg HS) were attempted for mood stabilization and psychosis, with minimal improvement. Given failure of multiple antipsychotics, prolonged illness, and severe functional impairment, the patient met criteria for treatment-resistant schizophrenia.

Clozapine Initiation and Response

Once the patient was able to reliably take oral medications, clozapine was initiated according to standard monitoring guidelines, starting at 12.5 mg and titrated slowly to 400 mg daily over several weeks. Olanzapine initially overlapped at a moderate dose and then gradually tapered off as clozapine was increased.
Clozapine was the first treatment associated with sustained, multidomain improvement. The patient became verbal, calmer, and more organized. Catatonic symptoms resolved, agitation markedly decreased and affect improved. He began participating in group therapy, interacting socially, sleeping better, and engaging in self-care. Cognitive function, including attention and memory, also improved. Side effects of drooling and constipation from clozapine were managed with glycopyrrolate and docusate sodium respectively. No other significant adverse effects were noted.
Lorazepam tapering was initiated approximately two weeks after clozapine stabilization and proceeded very slowly (about 10% per week) due to prolonged use. A taper schedule was provided at discharge for completion over the next 2 months.

Collateral History and Diagnostic Considerations

As the patient improved, he reported hearing mild voices prior to admission. Further details were not provided. He described a recent breakup four days before hospitalization, long-term cannabis use since adolescence, and depressive symptoms related to leaving military boot camp early and job loss. His mother, interviewed with a Spanish interpreter, reported behavioral decline, poor intake, possible video game addiction, and unclear past rehabilitation. Both histories were limited by recall issues and language barriers.

Timeline of Hospital Course and Treatments

Phase	Duration	Key Events	Treatments
Initial psych admission	1 week	Disorganized behavior and speech	Haloperidol IM up to 10 mg BID
Early catatonia	Days–1 week	BFCRS 12–16	Oral or IM lorazepam
Medical floor stay	3 months	Catatonia and NMS overlap, autonomic instability	IV lorazepam, dantrolene (2 episodes), alternative olanzapine & ziprasidone trials, LP/MRI, steroids
Return to psychiatry unit	2.5 months, pre-clozapine	Persistent catatonia, agitation, disorganized behavior and speech	Oral lorazepam, olanzapine, valproate, bupropion
Clozapine phase	2 months	Marked clinical improvement	Clozapine titrated to 400 mg, taper off olanzapine
Discharge	-	The patient was stable	Clozapine 400 mg, valproate, bupropion, lorazepam taper

Discussion:

Schizophrenia is a complex, chronic mental illness mostly presented with positive symptoms such as delusions, hallucinations, etc. during the first psychotic episode[19]. However, atypical presentations can complicate diagnosis and delay effective treatment, particularly when catatonia and medical complications such as neuroleptic malignant syndrome (NMS) are present.

Diagnostic Challenges and Differential Diagnosis

This case illustrates the diagnostic difficulty of an atypical first episode of psychosis complicated by prolonged catatonia and recurrent neuroleptic malignant syndrome (NMS). At presentation, the patient showed severe disorganization, agitation, and catatonia but did not clearly report hallucinations or fixed delusions. Mood disorder with psychotic features was considered; however, there was no sustained manic or major depressive episode that could explain the severity and long duration of symptoms.

Substance-induced psychosis was also considered due to cannabis use, but symptoms persisted for several months in a supervised hospital setting, making this diagnosis unlikely. Autoimmune and neurologic causes were evaluated because of prolonged catatonia, autonomic instability, hyperreflexia, and partial benzodiazepine response. Although ANA was positive,

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extensive workup including EEG, MRI brain, lumbar puncture, infectious testing, and a steroid trial did not reveal an alternative diagnosis. Other possible causes, including mood disorder, substance-induced psychosis, and autoimmune or neurologic conditions, were ruled out due to persistent symptoms and negative medical and neurologic workup.

The diagnosis of schizophrenia with atypical features was ultimately favored based on the patient's age, illness duration longer than six months, marked functional decline, disorganized speech and behavior, negative symptoms, catatonia, and later report of possible hallucinations. Prior studies describe schizophrenia as typically presented with clear positive symptoms such as hallucinations and delusions[7]. Tsuang et al. found that atypical schizophrenia may resemble bipolar disorder and is often linked to better outcomes[19]. In our case, bipolar features were not clearly present, aside from agitation. Huq et al. reported an atypical schizophrenia case of an elderly woman with preserved functioning despite positive symptoms[5]. In contrast, our patient showed prolonged catatonia, poor functional status and unclear psychotic features, highlighting the broad range of schizophrenia presentations. This case supports the understanding that schizophrenia may present without prominent early positive symptoms.

Catatonia and NMS Overlap

This case also highlights the clinical overlap between catatonia and NMS. NMS was suspected when fever, elevated creatine kinase, leukocytosis, transaminitis, and severe autonomic instability developed after antipsychotic exposure and improved with dantrolene and discontinuation of antipsychotics[17,18].

In contrast, catatonic symptoms persisted despite resolution of laboratory abnormalities and muscle rigidity. The patient remained mute, withdrawn, and intermittently agitated, with elevated Bush–Francis Catatonia Rating Scale scores[14,15]. This suggests catatonia was a primary and ongoing condition rather than solely a manifestation of NMS[17]. The limited response to prolonged lorazepam indicated a moderate to severe and refractory form of catatonia[20].

Treatment Resistance and Role of Clozapine

In our case study, the patient met criteria for TRS, defined as inadequate response to at least two prior antipsychotic trials of adequate dose and duration, despite adherence, with persistent psychotic symptoms[8,9]. Clozapine was the first treatment associated with sustained improvement[21-23]. After titration, the patient showed resolution of catatonia and improvement in speech, affect, cognition, mood, sleep, and social functioning. As the patient's improvement was continued, the other antipsychotic (olanzapine) was gradually tapered. Clozapine's lower dopamine blockade and possible anti-inflammatory effects may explain its benefit in this complex presentation[21,23].

Role of Inflammation and Immune Factors

Evidence suggests that immune changes, inflammation, and gut–brain interactions may affect brain function in schizophrenia[23]. In this patient, such factors could have contributed to atypical psychosis, prolonged catatonia, and poor response to multiple antipsychotics, although research is limited. **Figure 1** illustrates how genetic, environmental, and lifestyle factors can alter gut bacteria, trigger immune activation, and lead to brain inflammation, potentially influencing symptom severity and treatment response. Future research should explore immune markers and interventions targeting inflammation or the gut–brain axis to help guide treatment in complex or treatment-resistant cases[24,25].

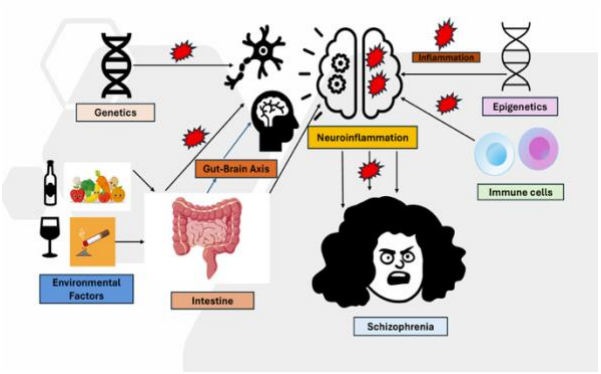

Figure 1: Proposed pathway illustrating how genetic predisposition, epigenetic changes, and environmental or lifestyle factors may alter gut microbiota, activate immune and inflammatory pathways, and contribute to brain inflammation, potentially influencing symptom severity, atypical presentation, and treatment resistance in schizophrenia.

Clinical Implications

This case shows that first-episode psychosis can sometimes present in unusual ways, including prolonged catatonia or poor response to typical antipsychotics. Clinicians should watch carefully for complications such as neuroleptic malignant syndrome and consider using clozapine earlier when multiple medications fail. Early recognition and tailored treatment may shorten hospital stays and support better recovery of daily functioning.

Limitations:

This report describes a single patient, limiting generalization. Diagnostic clarity was limited by unusual symptoms, incomplete or inconsistent collateral information, language barriers, reliance on retrospective history and the lack of long-term follow-up. The absence of biological markers for schizophrenia also makes the diagnosis less certain.

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Conclusion:
This case highlights an uncommon form of schizophrenia, marked by prolonged catatonia, unclear psychotic features, and repeated sensitivity to antipsychotics. Clozapine was the only treatment that led to sustained improvement in behavior, thinking, and mood. The case illustrates the wide range of schizophrenia presentations and emphasizes the need for flexible, individualized treatment strategies. Effective management required close multidisciplinary collaboration among psychiatry, neurology, internal medicine, and critical care teams to address diagnostic uncertainty, medical complications, and treatment resistance. More research is needed to understand the biological mechanisms behind atypical and treatment-resistant schizophrenia cases and to guide effective management.

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