Case Report

# Supersensitivity Psychosis with Acute Dystonia

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## Abstract

### Introduction
Supersensitivity psychosis is a phenomenon that occurs with chronic usage of antipsychotics secondary to treatment resistance. At this time, there are no standardized guidelines regarding the management of supersensitivity psychosis.

### Case Presentation
We present a case of a patient with schizoaffective disorder who developed supersensitivity psychosis and acute dystonia in response to discontinuing psychotropic medications, including high-dose quetiapine and olanzapine. The patient presented with excessive anxiety, paranoia, bizarre thoughts, and generalized dystonia affecting the face, trunk, and extremities. We treated the patient with olanzapine, valproic acid, and diazepam, which alleviated the psychosis back to baseline and significantly improved the dystonia. Despite compliance, the patient returned for inpatient stabilization due to depressive symptoms and worsening of the dystonia. During the second admission, the patient required further modification of psychotropics and supplemental electroconvulsive therapy.

### Conclusion
In this paper, we discuss the proposed treatment of supersensitivity psychosis, including the role that electroconvulsive therapy may play in alleviating supersensitivity psychosis and associated movement disorders. We hope to expand the knowledge of additional neuromotor manifestations in supersensitivity psychosis and the management of this unique presentation.

### Keywords
supersensitivity psychosis; antipsychotic withdrawal; schizoaffective disorder; schizophrenia; electroconvulsive therapy (ECT); dopamine; dopamine supersensitivity; olanzapine; dystonia

## Introduction
Schizophrenia is a debilitating mental illness that requires continuous antipsychotic treatment.[1] Patients with schizophrenia are often subject to intermittent psychotic episodes, with rates as high as 80% within 5 years post-diagnosis.[1] Although guidelines recommend continuous antipsychotic medication usage among patients with schizophrenia, there are reported cases of unexpected psychotic symptoms believed to be secondary to a supersensitivity to dopamine.[1] Dopamine supersensitivity psychosis occurs due to the upregulation of D2 receptors and enhanced dopamine affinity, secondary to chronic antipsychotic usage.[2] Thus, dopamine supersensitivity psychosis is proposed to cause rebound psychosis and an acquired tolerance to antipsychotics.[1,2] Movement disorders are also linked to dopamine sensitivity, particularly tardive dyskinesia.[2] There are 4 clinical features used to categorize dopamine supersensitivity psychosis. There is a rapid relapse of symptoms after reducing, discontinuing, and switching antipsychotics. There is usually co-occurring tardive dyskinesia, indicative of dopamine supersensitivity.[2] Furthermore, patients often develop tolerance to medications and doses that were previously

therapeutic.[2] Fourth, there are associated psychotic exacerbations in response to stressors.[2] Although dystonia is a well-known complication from long-term antipsychotic usage[3], we present a case where an acute dystonic reaction and psychosis were associated with the abrupt discontinuation of antipsychotics.

## Case Description

A 53-year-old male with a history of schizoaffective disorder presented to the emergency department due to muscle contractions and bizarre movements. The patient's symptoms progressed as the patient abruptly discontinued psychiatric medications 1 week prior. His regimen consisted of quetiapine 400 mg 2 times daily (BID), olanzapine 20 mg at bedtime (QHS), lamotrigine 100 mg 3 times daily (TID), lithium 300 mg QHS, and benztropine 0.5 mg QHS. The patient was only on quetiapine 25 mg BID and alprazolam 2 mg QHS upon arrival at the emergency department. The patient presented at the time with paranoia, bizarre behavior, significant anxiety, and multiple somatic delusions. He denied hallucinations and mood symptoms. However, he was noted to be responding internally. Physical examination demonstrated involuntary contractions in the trunk and extremities, facial grimacing, bizarre grunting, and an antalgic gait. Alprazolam was discontinued. The patient was started on valproic acid 500 mg BID and olanzapine 5 mg BID. Treatment with benztropine or diphenhydramine did not reduce his muscle contractions. Diazepam 10 mg TID was initiated, which provided temporary relief. However, the patient began having increased muscle contractions, limiting his daily activities. Neurology was consulted and found that the patient was experiencing an acute dystonic reaction. Laboratories, head CT, brain MRI, EEG, and cerebrospinal fluid (CSF) analysis were unremarkable. Medical causes of dystonia were not found. During his hospitalization, the patient continued psychotropics and experienced a gradual reduction in his dystonia and psychosis. Medications were increasingly optimized to olanzapine 7.5 mg BID, diazepam 5 mg BID, and valproic acid 250 mg BID.

The patient was readmitted 2 months later due to a suicide attempt. The patient reported a depressed mood, recurrence of dystonia, insomnia, and increasing anxiety. The evaluation revealed bizarre behavior, mood-congruent delusions, severe anxiety, and internal preoccupation. The patient was compliant with medications consisting of valproic acid 250 mg BID, olanzapine 20 mg QHS, diazepam 5 mg in the morning and 10 mg QHS, and mirtazapine 15 mg QHS. Valproic acid was increased to 500 mg BID, and mirtazapine was increased to 30 mg QHS. Due to his history of recurrent depression and return of the dystonia, the patient was offered supplemental electroconvulsive therapy (ECT). Three ECT treatments were conducted. ECT treatment was performed utilizing right-side unilateral ultra-brief pulse stimulation. After a titration session, the seizure threshold was confirmed at 38 mC. The patient continued treatment utilizing the following parameters: 0.30 ms/60 hz, 8 sec, 800 mA (6x threshold) to provide a total of 230.4 mC. By the third treatment, a minor adjustment was done to optimize the response, and settings were adjusted to a total of 307.2 mC (8x threshold), which were tolerated well by the patient. No cognitive or memory deficits were noted during face-to-face evaluations. The Becks Depression Scale Score declined from a 37 at baseline to a 32, with a consistent Mini-Mental State Exam score of 29. During this time, the patient reported a reduction in depression and psychosis. Upon discharge, the patient had reduced dystonia, improved movement control, and a stable mood.

## Discussion

To our knowledge, this is the first case of acute dystonia occurring in supersensitivity psychosis, likely secondary to abrupt antipsychotic withdrawal. Antipsychotic usage is associated with increased D2 receptors in the basal ganglia, potentially implicating the development of various movement abnormalities.[2] Evidence-based management for dopamine supersensitivity psychosis is paramount as studies suggest that it may occur in as many as 30% of patients with schizophrenia and as many as 75% of patients with treatment-resistant schizophrenia.[2] Chouinard et al. proposed guidelines for preventing supersensitivity psychosis. They proposed assessing movements since they serve as indicators for early detection of excessive D2 blockade.[2] They proposed using antipsychotics, which were less likely to induce movement disorders, using the lowest therapeutic dose and the addition of low-dose antiepileptic medications.[2] For these reasons, we utilized olanzapine and valproic acid. Rebound psychosis and withdrawal-emergent dyskinesias have been reported in a patient dis-

continuing olanzapine, though symptoms were remitted upon restarting olanzapine.[4] Similarly, our patient improved with restarting olanzapine. Another report discusses how aripiprazole and lamotrigine were used to manage supersensitivity psychosis in treatment-resistant schizophrenia, supporting that antipsychotics and antiepileptic drugs may be effective in managing dopamine supersensitivity psychosis.[5] Although our patient was previously on lamotrigine, valproic acid was started due to faster titration. Furthermore, benzodiazepines may have additive benefits in the management of supersensitivity psychosis. Fukai et al. presented a case of supersensitivity psychosis treated with aripiprazole and clonazepam.[6] The authors proposed that benzodiazepine usage may reduce psychosis due to decreased concentration and activity of GABAergic interneurons among patients with schizophrenia.[6] Thus, diazepam may have played a role in the alleviation of both the patient's psychosis and dystonia.

ECT was used upon the second re-emergence of dystonia in this patient. ECT has shown promising evidence in alleviating movement disorders, including tardive dyskinesia and dystonia.[7] The mechanism of action for ECT is proposed to prevent supersensitivity of dopamine receptors, which often occurs secondary to chronic neuroleptic usage.[7,8] Studies suggest that ECT enhances dopamine transmissions and alters GABA levels.[7,8] Additionally, ECT is known to alter the blood-brain barrier, which can affect drug concentrations and their effects, including motor symptoms.[7] However, alleviation of movement symptoms has been shown to vary. A retrospective study by Yasui-Farukori et al. reported a 39% response rate with variable ECT sessions.[7] Thus, the evidence for ECT in antipsychotic-induced movement disorders requires more robust studies. We believe a combination of olanzapine, diazepam, and valproic acid best alleviated the patient's presenting symptoms. However, we also believe supplemental ECT contributed to this patient's stabilization.

## Conclusion

We present a case of supersensitivity psychosis and acute dystonia after abrupt withdrawal of psychotropic medications in a patient with chronic antipsychotic usage. We present supersensitivity psychosis with acute dystonia as an associated neuromuscular finding, which is the first such reported case to our knowledge. Our case report supports the use of second-generation antipsychotics and low-dose antiepileptic medications for treating symptoms of supersensitivity psychosis. We encourage clinicians to be familiar with dopamine sensitivity psychosis and its manifestations, especially in the context of psychosis after decreasing or discontinuing antipsychotics. We support the regular usage of standardized scales to monitor for movement disorders associated with supersensitivity psychosis. We also encourage practitioners to consider supplemental ECT in patients with supersensitivity psychosis and movement disorders, particularly in patients with recurrent depression, as in this case. We present this case to further expand the knowledge of supersensitivity psychosis and its manifestations and treatment modalities of this phenomenon.

### Conflicts of Interest

The authors declare that they have no conflicts of interest.

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