

# Treatment of catatonic schizophrenia and psychogenic polydipsia with clozapine: A case report

## Abstract

Psychogenic polydipsia is a common problem in patients with chronic schizophrenia with prevalence rates varying from 5%-10%. No definitive treatment guidelines exist for pharmacological treatment of psychogenic polydipsia. There have been conflicting reports on antipsychotics causing and being used for treatment of polydipsia. We report a unique case of a 53 year old patient presenting with schizophrenia with catatonic features and psychogenic polydipsia treated with Clozapine. Dramatic improvement was seen not only in the polydipsia and correction of hyponatremia but also in catatonic behavior within few weeks. Clozapine may be a possible alternative option for patients presenting with catatonia and psychogenic polydipsia especially in settings where there is no availability for Electroconvulsive therapy (ECT).

**Keywords:** psychogenic polydipsia, primary polydipsia, schizophrenia, clozapine, hyponatremia, catatonia

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## Introduction

Schizophrenia is a chronic debilitating psychiatric disorder that affects an estimated 1% of the adult population.<sup>1,2</sup> Psychogenic polydipsia is frequently seen in patients with chronic schizophrenia with prevalence rates varying from 5-10%.<sup>1,3,4</sup> It can cause dilutional hyponatremia resulting in acute brain edema. This has serious consequences which may result in delirium, seizures, coma and even death. The treatment options include various behavioral and pharmacological interventions. Due to the unclear mechanism and pathophysiology, very few controlled clinical studies have been done.<sup>5</sup> There are few treatment options and the evidence on their efficacy is weak and conflicting.<sup>1,6</sup> There have been conflicting reports on the use of Clozapine in patients with schizophrenia and psychogenic polydipsia. There are some reports which show that Clozapine can cause polydipsia and hyponatremia<sup>7</sup> while other reports have shown that Clozapine can ameliorate symptoms of polydipsia.<sup>5,8-10</sup>

We report a unique case of a 53 year old Caucasian man with past history of chronic schizophrenia and multiple past psychiatric hospitalizations. Patient presented with extreme stupor, mutism and polydipsia which was successfully treated with clozapine.

## Clinical presentation

Patient is a 53 year old Caucasian male with more than 30 year history of chronic schizophrenia and multiple past psychiatric hospitalizations including state hospitalizations, who were brought in to the emergency department from a residential facility due to inability to care for self and exhibiting odd and bizarre behaviour. The residential staff reported that the patient has not been eating, drinking or taking shower, socially disengaged, withdrawn and neglecting himself. Patient has been noncompliant with his medications for more than a month and slowly decompensating. On evaluation, patient was found to be in a state of stupor, mute, apathetic, aloof and listless. He actively maintained the same posture against gravity for a long duration of time.

Based on the aforementioned presentation of acute psychosis with catatonia, patient was diagnosed with schizophrenia, with catatonia<sup>11</sup>

[Diagnostic and Statistical Manual-5]. Patient was medically cleared and then transferred to the inpatient psychiatric unit for medication management and stabilization. He was started on Risperidone 2mg PO BID for psychosis, Ativan 1mg PO TID for catatonia and Cogentin 1mg PO BID for extrapyramidal symptom prevention. On the unit, patient was observed to be uncontrollably drinking excessive amounts of water and was evaluated by the medical team and neurology. After an extensive work up including Computerized Axial Tomography Scan (CAT scan) of the head to rule out other causes of polydipsia, patient was diagnosed with psychogenic polydipsia. He was placed on water restriction to 1 L/day and Q15 minute observation for verbal redirection in order to prevent water intoxication and hyponatremia. Patient became agitated and aggressive many times when he was redirected from drinking water from the water fountain and sink and had to be medicated often with short acting intramuscular doses of Haldol and Ativan. Haldol 2mg PO BID was added to his drug regimen on day 8 since no improvement was seen in his behavior. His catatonia responded poorly to Ativan and antipsychotics. His sodium levels were followed by the medical team due to concern of developing dilutional hyponatremia which can result in serious consequences including delirium, seizures, coma and even death. His sodium levels varied in the range of 128-131 mmol/l. Due to the concern for developing acute brain edema due to hyponatremia patient was put on constant 1:1 observation as he required constant verbal redirection.

Due to lack of improvement seen in patient's catatonic behavior and polydipsia, on day 14, Clozapine titration was initiated at 12.5mg PO BID while cross tapering of Risperidone was also initiated. Patient began to show improvement in both catatonic and polydipsic behavior in 7-8 days of starting Clozapine which was stabilized at 100mg PO BID. After 2 weeks of treatment with Clozapine patient became verbal, started interacting with staff and peers and his polydipsia resolved. His sodium levels trended upwards and were noted to return to normal limits. This effect has been sustained for 3 weeks now and patient has approached his baseline. He is compliant with his medications and weekly clozapine blood draws. He showed brighter affect with reactive mood. He became more conversational and conceptually organized with better cognitive flexibility. His activities of daily living

and instrumental activities of daily living also significantly improved. He has returned to his baseline level of functioning.

## Discussion

The pathophysiology of psychogenic polydipsia remains unclear and this limits the possibility of identifying an appropriate drug treatment. Treatment becomes very difficult especially in patients with schizophrenia who are actively psychotic and polydipsic due to limited insight and lack of responsiveness to verbal redirections. Most of the cases reported in the past have been related to psychogenic polydipsia in patients with schizophrenia without catatonic features. Our case is unique as clozapine not only improved polydipsia but also improved catatonia as our patient was refractory to treatment with Aripiprazole. In a study done by Canuso et al.<sup>8</sup> on patients with polydipsic hypo-osmolemic schizophrenia, the data showed that Clozapine dose of 300mg/day was sufficient to normalize plasma osmolality and was well tolerated. However it is noteworthy that it was an open label study done for 24 weeks on 8 male patients only. Patients were initially given conventional neuroleptics for the first 6 weeks and then switched to Clozapine doses of 300, 600, 900 mg/day depending on the tolerability. Our patient was stabilized at a dose of 200 mg/day with sodium levels and plasma osmolality returning to normal levels. This effect has been sustained for 3 weeks now. However in future, more studies with double blind randomized controlled trials and large sample size are needed to establish the efficacy of Clozapine in patients with concurrent schizophrenia and polydipsia symptoms. Clozapine may be an alternative to ECT in patients presenting with catatonia and polydipsia especially in settings where there is no availability for ECT or patients refusing ECT.

## Acknowledgements

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## Conflict of interest

The author declares no conflict of interest.

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