

## Polydipsia, hyponatremia and rhabdomyolysis in schizophrenia: A case report

Li-Chi Chen, Ya-Mei Bai, Meng-Han Chang

Li-Chi Chen, Meng-Han Chang, Department of Psychiatry, Taipei Veterans General Hospital, Taipei 112, Taiwan  
Ya-Mei Bai, Department of Psychiatry, Collage of Medicine, National Yang-Ming University, Taipei 112, Taiwan  
Author contributions: Chen LC collected the patient's clinical data and wrote the paper; Bai YM revised the paper; Chang MH collected the patient's clinical data.

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Correspondence to: Ya Mei Bai, MD, PhD, Department of Psychiatry, Taipei Veterans General Hospital, No. 201, Sec. 2, Shih-Pai Road, Taipei 112, Taiwan. [yambi@mail2000.com.tw](mailto:yambi@mail2000.com.tw)  
Telephone: +886-2-28344012

Fax: +886-2-28344012

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### Abstract

The prevalence of polydipsia among patients with schizophrenia is 6%-20%. Around 10%-20% of patients with polydipsia may develop hyponatremia and even complicated with rhabdomyolysis. Here we presented a 40-year-old man with schizophrenia, who had received paliperidone 15 mg/d for more than one year, and polydipsia was noted. In Jan, 2014, he developed hyponatremia (Na 113 mEq/L) with consciousness disturbance. After 3% NaCl (500 cc/d) intravenous supplement for three days, the hyponatremia was corrected, but rhabdomyolysis developed with a substantial elevation in the level of creatine kinase (CK) to 30505 U/L. After hydration, the CK level gradually decreased to 212 U/L. Both the hyponatremia itself and quick supplementation of NaCl can cause rhabdomyolysis. If rhabdomyolysis is not recognized, insufficient hydration or water restriction for polydipsia

may further exacerbate the rhabdomyolysis with a lethal risk. In this case, we highlight the possible complication of rhabdomyolysis with polydipsia-induced hyponatremia. In addition to monitoring the serum sodium level, the monitoring of CK is also important; and switching of antipsychotic may improve the polydipsia.

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**Key words:** Schizophrenia; Polydipsia; Hyponatremia; Rhabdomyolysis; Quetiapine

**Core tip:** We present a 40-year-old man with schizophrenia who had polydipsia for more than one year and later had hyponatremia related consciousness disturbance. Though the association of polydipsia with schizophrenia and/or neuroleptic treatment is already discussed in the literature, there were no articles as detailed as our article. It reviewed the possible mechanism associated with hyponatremia and rhabdomyolysis, the choice of antipsychotics, and the reasons of polydipsia in schizophrenia patient at a time.

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### INTRODUCTION

Polydipsia was prevalent in patients with psychiatric disorder, especially in chronic schizophrenia patient. The resulting complication of hyponatremia and rhabdomyolysis were also recorded in some case reports. Regarding the possible lethal risk of acute kidney injury, the management of polydipsia and the choice of antipsychotics should be concerned.

## CASE REPORT

This 40-year-old single man was diagnosed with schizophrenia with the presentation of auditory hallucination, persecutory delusions and disturbing behavior, self-talking, poor self-hygiene, and wandering out, when he was 21-year-old. He had a medical history of chronic hepatitis B. Due to poor family support he stayed in a nursing home for years. He had been prescribed paliperidone 12 mg/d since November 2012, and polydipsia (more than 3 L/d) was noted. In December 2013, the dose of paliperidone was titrated to 15 mg/d due to persistent auditory hallucination.

On Jan 24, 2014, he was observed to have disturbed consciousness by the staff at the nursing home, and was sent to the emergency room immediately. The initial laboratory evaluations showed hyponatremia (Na 113 mEq/L), and creatine kinase (CK) of 247 U/L. The Glasgow Coma Scale was E4V1M4, with normal vital signs and unremarkable results for other laboratory exams. The patient was treated with 3% NaCl (500 cc/d) for three days. His consciousness became clear on the second day, and on the third day, the follow-up sodium level was 132 mEq/L. He was transferred to the acute psychiatric ward on the fourth day (Jan 27, 2014) in stable condition.

However, the follow-up data at psychiatric admission showed elevated levels of CK (30505 U/L), serum alanine (ALT, 102 U/L) and aspartate aminotransferase (AST, 457 U/L). To prevent acute kidney injury causing by rhabdomyolysis, he was given 1000 CC NaCl 0.9% from the 4<sup>th</sup> to the 13<sup>th</sup> day. On the 7<sup>th</sup> day, the CK level decreased to 1081 U/L, and on the 16<sup>th</sup> day, CK was reduced to 212 U/L, and liver enzymes returned to a normal range. After admission, the patient was prescribed paliperidone 6 mg/d initially for his psychotic symptom, and polydipsia was still noted with total water intake of 4000 CC/d, although the behavior modification was done. On the 15<sup>th</sup> day, the antipsychotic was switched to quetiapine, and gradually titrated to 1200 mg/d. Polydipsia was not noted beginning the 21<sup>st</sup> day, and the patient was discharged on the 31<sup>st</sup> day. Polydipsia was still not noted at three months after hospitalization, and the psychiatric condition remained stable now.

## DISCUSSION

The clinical definition of polydipsia is water intake over 3 L/d. The prevalence of polydipsia among patients with schizophrenia is 6%-20%<sup>[1,2]</sup>. The possible mechanism may be related to the dry mouth side effect of anticholinergic drugs, compulsive behavior, or stress reduction<sup>[3-7]</sup>. It is also suggested the supersensitivity of the dopamine receptor induced by the long-term use of antipsychotic may stimulate the thirst center<sup>[4,8]</sup>. Previous studies showed 10%-20% of subjects with polydipsia may develop hyponatremia<sup>[7]</sup>. The complication of hyponatremia includes changes in consciousness, coma, seizure, and rhabdomyolysis. The exact etiology of rhabdomyolysis secondary to hyponatremia is unclear, but several mechanisms have been proposed. First, because of the lower osmolality of the extracellular fluid,

acute hyponatremia resulted in cells swelling, and after hours of the extrusion of intracellular potassium which lowered the transmembrane potential but cause the release of CK and myoglobin<sup>[9-11]</sup>. Second, the decreased extracellular sodium may disturb the Na-Ca exchange pumps. Calcium level increased inside the cell because of reduced of the sodium outside the cell to be exchanged which led to the cell death by releasing proteases and lipase<sup>[12-14]</sup>. To conclude, creatine kinase may elevate after 48-96 h of hypomatremia because of the lowered sodium level itself or the rapid correction of hyponatremia<sup>[9,13,15,16]</sup>. In our case, we assumed that hyponatremia was corrected rapidly by the supplement of 3% saline which led to the delayed rise of CK, ALT, and AST.

With regard to the possible relationship of polydipsia with the supersensitivity of the dopamine receptor induced by long-term use of antipsychotics<sup>[8,17]</sup>, risperidone, with a high affinity to D2 receptor blockade, has been reported to be associated with polydipsia<sup>[18,19]</sup>. There was also a case report discussing about olanzapine which caused the elevation in CK value<sup>[20]</sup>. Clozapine and quetiapine are weaker D2 antagonists, and have been suggested to be promising treatments for patients with polydipsia related to high potency antipsychotics<sup>[18,21-23]</sup>. Although two cases reported quetiapine-induced CK elevation in a neuroleptic-naïve patient<sup>[24]</sup>. In this case, the patient's polydipsia improved soon after switching paliperidone to quetiapine, not only in the acute psychiatric ward setting, but also after discharge and for three months in the nursing home (up to this writing).

In conclusion, both the hyponatremia itself and quick supplementation of NaCl can cause rhabdomyolysis. If rhabdomyolysis is not recognized, insufficient hydration or water restriction for polydipsia may further exacerbate the rhabdomyolysis with a lethal risk. In this case, we highlight the possible complication of rhabdomyolysis with polydipsia-induced hyponatremia. In addition to monitoring the serum sodium level, the monitoring of CK is also important; and switching of antipsychotic may improve the polydipsia.

## COMMENTS

### Case characteristics

An 40-year-old male with diagnosis of chronic schizophrenia.

### Clinical diagnosis

Psychogenic polydipsia, schizophrenia, hyponatremia.

### Treatment

Quetiapine 1200 mg.

### Experiences and lessons

The authors highlight the possible complication of rhabdomyolysis with polydipsia-induced hyponatremia.

### Peer review

The case report is interesting.

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