Effective and Cheap Behavioral Modification Therapy to Manage Complicated Polydipsia and Seizures in a Chronic Mental Health Institute

Yen-Wen Chen[1], Weilun Chung[2], Hung-Yu Wang[2], Ching-Kuan Wu[2], Shih-Pin Hsu[1], Ping-Tao Tseng[2]

Abstract:

Purpose: Polydipsia is one of the most serious and complicated problems causing morbidity and mortality in chronic mental health institutes. The pathophysiology of polydipsia in chronic schizophrenia remains unclear; as a result, no effective methods exist to deal with this serious problem. This report describes a patient with schizophrenia with polydipsia and seizures who benefitted from a behavioral modification program at a chronic mental health institute.

Case report: A 56-year-old schizophrenic man did not have a history of physical illnesses or seizures and developed seizures following polydipsia. Despite drug adjustment, his polydipsia was uncontrolled and he suffered from generalized tonic convulsions. After introducing a "water restriction program," his polydipsia and seizures were controlled.

Conclusion: The "water restriction program" consisted of daily body weight monitoring and frequent checking of electrolyte data, both of which are inexpensive and simple. This program can be carried out by untrained nursing staff, who are the primary caregivers in chronic mental health institutes. Our case highlights an effective and inexpensive behavior modification program to deal with the difficult and complicated problems of polydipsia and seizures in chronic mental health institutes.

Key Words: polydipsia, neuropsychiatry, seizure, cognitive behavioral modification therapy, schizophrenia

Acta Neurol Taiwan 2016;25:41-44

PURPOSE

Polydipsia, also called "water overintake" or "excessive water drinking," is a troublesome problem in chronic mental health institutions and can cause a high mortality rate in this setting[1,2]. Polydipsia occurs in ~3–39% of inpatients in chronic mental institutes[2]. It is associated with cognitive impairment, frequently occurs several years after the patients' first episode of psychosis[3], and increases with disease severity[4]. It can

From the [1]Department of Neurology, E-Da hospital, Kaohsiung, Taiwan, [2]Department of Psychiatry, Tsyr-Huey Mental Hospital, Kaohsiung Jen-Ai's Home, Taiwan.
Received February 26, 2015. Revised June 12, 2015
Accepted March 3, 2016.
Corresponding to: Ping-Tao, Tseng, MD. Department of Psychiatry, Tsyr-Huey Mental Hospital, Kaohsiung Jen-Ai's Home, Taiwan. No. 509, Fengping 1st Rd., Daliao Dist., Kaohsiung City 831, Taiwan.
E-mail: ducktseng@gmail.com

produce numerous serious complications, such as seizures, rhabdomyolysis, or death[2,5,6].

Polydipsia's pathophysiology remains unclear. Hawken and colleagues have shown that social isolation, which is unfortunately frequent in chronic mental health institutes, is associated with polydipsia in animal models[7]. The administration of antipsychotic drugs is also believed to result in polydipsia[6]. On the other hand, accumulating evidence indicates that brain structure abnormalities[8] and an imbalance of neuroendocrine substances, such as glucocorticoids, arginine vasopressin, and anti-diuretic hormones, play an important role in polydipsia, regardless of normo or hyponatremia[9-12]. Not only physiological impairments, but also disease course can affect polydipsia; in a report by de Leon and colleagues, the chronicity of schizophrenia is also associated with polydipsia[2]. Furthermore, research has shown that polymorphisms in the dopamine-2 (D_2) receptor may also be associated with polydipsia in patients with chronic schizophrenia[13].

However, despite the seriousness of polydipsia in patients with schizophrenia, there are no effective and safe methods to manage it. Here, we describe one behavioral modification program introduced into a clinical setting and its successful resolution of polydipsia in one male patient with schizophrenia.

CASE REPORT

Mr. A is a 56-year-old male who was diagnosed as having a schizoaffective disorder, with a first manifestation of auditory hallucinations, delusions of persecution, and mood components. Because of his chronically deteriorated personal function, he was hospitalized in an institute treating chronic mental health issues in Southern Taiwan. His psychotic symptoms remained stable under a sulpiride dose of 800 mg or 600 mg of chlorpromazine with lorazepam. The patient had no history of physical illness. The baseline serum laboratory data revealed sodium (Na): 138 mEq/L, potassium (K): 4.01 mEq/L, chloride (Cl): 104.1 mEq/L, creatinine: 0.9 mg/dL, Blood urea nitrogen (BUN): 11.6 mg/dL, glutamic oxaloacetic transaminase (GOT): 17 U/L, glutamate pyruvate transaminase (GPT): 19 U/L, preprandial blood sugar: 91 mg/dL, uric acid: 2.0 mg/dL, free thyroxin-4 (fT4): 4.7 μg/dL, thyroid-stimulating hormone (TSH): 2.382 μU/mL. The baseline electroencephalography (EEG) examination, which was performed at inter-ictal, revealed no significant slow wave or epileptic activities detected. All the laboratory data and EEG did not have significantly change during the treatment course. However, when treated with either first- or second-generation antipsychotics, he started to experience polydipsia each time his psychotic symptoms stabilized. After the water over-intake, he frequently developed water intoxication and hyponatremia, which is less than 120 mEq/L, with consequent generalized tonic convulsions despite of our prescription of anti-epileptic drugs (AEDs), for example carbamazepine 800 mg/day, and was sonly sent to another local general hospital emergently. The EEG was difficulty being performed because of the emergent condition and uncooperativeness. The "water restriction program" was started in 2008. The main procedure of this program includes daily monitoring of patients' body weight and weekly checking of electrolyte levels. Patients would be sent to isolation rooms if their body weight increased by over 4% of total body weight in one day, or if they showed any abnormal electrolyte data. Since then, Mr. A's epileptic activities were so significantly improved that his AED dosage was tapered down to its minimum. Finally, he received zotepine at 100 mg/day and valporate at 700 mg/day for his schizoaffective disorder, and clonazepam at 5 mg for his anxiety and insomnia. He now lives in the chronic mental health institute, without any recurrence of seizures to date. The followed EEG revealed no significant abnormal findings, neither slow wave nor epileptic activities.

DISCUSSION

The actual mechanism of polydipsia and complicated seizure in chronic schizophrenia is still unclear. There is evidence suggesting that this reversible polydipsia and polyuria in such situation might have less relationship with the diabetes insipidus[14]. Along with the worsen water intoxication, the precipitously decreasing serum sodium levels would finally result in the coma or seizure[14-17]. However, there are still no effective and safe methods to manage the serious problem of polydipsia in chronic care facilities. Some reports have suggested that medication adjustment[18-20] or administration of oral urea[21] might work in such situations. However, these strategies

show controversial effect in other patient with both schizophrenia and polydipsia in clinical practice. In our case report, we used a simple and inexpensive behavioral therapy, the "water restriction program," to successfully treat this problem. The core concept of this procedure is to frequently monitor the input/output of water in patients with chronic schizophrenia using the simplest and least expensive methods, which could be performed in any chronic-care institute. Besides this monitoring, and to avoid the risk of complicated hyponatremia and seizures by water intoxication, we also frequently check patient electrolyte levels. When the patient's body weight increases by an unusual amount, or their serum sodium level is lower than a specific threshold, the patient is kept in an isolation room in order to prevent them for drinking water, and thereby risk water intoxication. After introducing this program, this patient did not have any recurrent water intoxication or seizures despite being maintained on only low dose AEDs.

This program is modified from that of Bowen and colleagues[22]. In fact, this program is a simplified version, so that it can be carried out by most of the nursing staff, who are not trained in cognitive behavioral therapy (CBT). The simplified program focusses on preventing future excessive water intake, and reinforcing the effects of CBT through psychiatric re-education after each period of isolation.

There are some limitations in our case report. The first is that there is not any brain image examination in our hospital. Therefore, we could not exclude the possibility of abnormality of brain structure, for example the pituitary gland, according the record in our hospital. However, according to the patient's statement, there are not any significant abnormal findings in the brain image in other general hospital. The second is that we could not completely rule out the possible effect of carbamazepine on the electrolyte balance[23]. However, in the course of water restriction program, there is still intermittent hyponatremia detected despite that the carbamazepine has been shifted to valproate for a long time.

CONCLUSION

Our case highlights an effective and inexpensive behavioral modification program to deal with the difficult, prevalent, and dangerous problems of polydipsia and seizures in patients of chronic mental health institutes.

ACKNOWLEDGEMENT

The authors state that they have no conflict of interest or any financial sources or granting agency to disclose.

REFERENCES

1. de Leon J. Polydipsia--a study in a long-term psychiatric unit. Eur Arch Psychiatry Clin Neurosci 2003;253:37-39.
2. de Leon J, Verghese C, Tracy JI, Josiassen RC, Simpson GM. Polydipsia and water intoxication in psychiatric patients: a review of the epidemiological literature. Biol Psychiatry 1994;35:408-419.
3. Torres IJ, Keedy S, Marlow-O'Connor M, Beenken B, Goldman MB. Neuropsychological impairment in patients with schizophrenia and evidence of hyponatremia and polydipsia. Neuropsychology 2009; 23:307-314.
4. Poirier S, Legris G, Tremblay P, Michea R, Viau-Guay L, Mérette C, Bouchard RH, Maziade M, Roy MA. Schizophrenia patients with polydipsia and water intoxication are characterized by greater severity of psychotic illness and a more frequent history of alcohol abuse. Schizophr Res 2010;118:285-291.
5. Tolan P, O'Loughlin D, Botha J. Can seizures and rhabdomyolysis be a potentially serious complication of hyponatremia due to polydipsia? Aust N Z J Psychiatry 2001;35:386.
6. Chen LC, Bai YM, Chang MH. Polydipsia, hyponatremia and rhabdomyolysis in schizophrenia: A case report. World J Psychiatry 2014;4:150-152.
7. Hawken ER, Delva NJ, Beninger RJ. Increased drinking following social isolation rearing: implications for polydipsia associated with schizophrenia. PloS one 2013;8:e56105.
8. Nagashima T, Inoue M, Kitamura S, Kiuchi K, Kosaka J, Okada K, Kishimoto N, Taoka T, Kichikawa K, Kishimoto T. Brain structural changes and neuropsychological impairments in male polydipsic schizophrenia. BMC Psychiatry 2012;12:210.
9. Umbricht D. Polydipsia and hippocampal pathology.

BMC Psychiatry 1994;36:709-710.
10. Goldman MB, Torres IJ, Keedy S, Marlow-O'Connor M, Beenken B, Pilla R. Reduced anterior hippocampal formation volume in hyponatremic schizophrenic patients. Hippocampus 2007;17:554-562.
11. Goldman MB, Wood G, Goldman MB, Gavin M, Paul S, Zaheer S, Fayyaz G, Pilla RS. Diminished glucocorticoid negative feedback in polydipsic hyponatremic schizophrenic patients. J Clin Endocrinol Metab 2007;92:698-704.
12. Kishimoto T, Hirai M, Ohsawa H, Terada M, Matsuoka I, Ikawa G. Manners of arginine vasopressin secretion in schizophrenic patients--with reference to the mechanism of water intoxication. Jpn J Psychiatry Neurol 1989;43:161-169.
13. Matsumoto C, Shinkai T, De Luca V, Hwang R, Hori H, Lanktree M, Ohmori O, Kennedy JL, Nakamura J. Association between three functional polymorphisms of the dopamine D2 receptor gene and polydipsia in schizophrenia. Int J Neuropsychopharmacol 2005; 8:245-253.
14. Ellinas PA, Rosner F, Jaume JC. Symptomatic hyponatremia associated with psychosis, medications, and smoking. J Natl Med Assoc 1993;85:135-141.
15. Jose CJ, Barton JL, Perez-Cruet J. Hyponatremic seizures in psychiatric patients. Biological Psychiatry 1979;14:839-843.
16. Jose CJ, Perez-Cruet J. Incidence and morbidity of self-induced water intoxication in state mental hospital patients. Am J Psychiatry 1979;136:221-222.
17. Jose CJ. Generalized seizures from self-induced water intoxication. Psychosomatics 1984;25:153-157.
18. Diniz JB, Cordeiro Q, Zung S. Clozapine treatment for schizophrenia-related polydipsia. Rev Bras Psiquiatr 2010;32:318-319.
19. Amato D, Stasi MA, Borsini F, Nencini P. Haloperidol both prevents and reverses quinpirole-induced nonregulatory water intake, a putative animal model of psychogenic polydipsia. Psychopharmacology (Berl) 2008;200:157-165.
20. Rao N, Venkatasubramanian G, Korpade V, Behere R, Varambally S, Gangadhar B. Risperidone treatment for polydipsia and hyponatremia in schizophrenia: a case report. Turk Psikiyatri Derg 2011;22:123-125.
21. Kawai N, Ishikawa K, Nemoto K, Katano T, Takahashi S, Hori T, Asada T. Oral urea treatment for polydipsia-hyponatremia syndrome in patients with schizophrenia. J Clin Psychopharmacol 2009;29:499-501.
22. Bowen L, Glynn SM, Marshall BD, Jr., Kurth CL, Hayden JL. Successful behavioral treatment of polydipsia in a schizophrenic patient. J Behav Ther Exp Psychiatry 1990;21:53-61.
23. Van Amelsvoort T, Bakshi R, Devaux CB, Schwabe S. Hyponatremia associated with carbamazepine and oxcarbazepine therapy: a review. Epilepsia 1994;35: 181-188.