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Ruben Hummelen, Laus JMM Mulder, Adriaan Dees

ABSTRACT

Introduction: Ecstasy (XTC) is a widely used synthetic drug, which can cause severe complications. Case Report: A 19-year-old male who had visited a rave party and used XTC presented at the hospital with acute hyponatremia and seizures. Initial treatment with hypertonic saline failed to prevent new seizures. When the hyponatremia persisted and we concluded that the patient was also volume overloaded due to polydipsia, we decided to administer a low dose of a loop diuretic. Within hours, more than six litres of urine was produced and the patient made a complete recovery with normalization of the serum sodium level. Conclusion: The combination of XTC use and excessive fluid intake can result in hypervolemia, along with treatment refractory hyponatremia. This case report emphasises the potential benefits of diuretics in the management of XTC-related hyponatremia.
CASE REPORT

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Keywords: Ecstasy, Hyponatriemia, Syndrome of inappropriate ADH secretion (SIADH)

INTRODUCTION

Ecstasy (XTC) or 3,4-methylenedioxymethamphetamine (MDMA) is an illicit synthetic drug. It is reported that XTC has been used by approximately 16 million Americans [1]. The drug is (ab)used for its euphoric and psychedelic effects. The desirable influences can be enhanced in electronic dance music or so-called rave party settings. Even though severe side effects of XTC are uncommon, potentially lethal adverse events have been reported, including hyperthermia, renal failure, non-traumatic rhabdomyolysis, hyponatremia, cerebral edema, coma, mediastinal emphysema and severe hypoglycaemia [2–4]. Herein, we describe a case of acute, symptomatic hyponatremia due to the combination of XTC and polydipsia.

CASE REPORT

A 19-year-old male presented at the emergency department of the hospital on Saturday morning after a rave party. The patient arrived by ambulance after having had a seizure at the party. Other attendees of the
party mentioned that the patient had consumed a large amount of water and (diluted) beer that night and had lost consciousness for a short time. On admission he was orientated with a maximum Glasgow Coma Scale (GCS) score of 15. He denied having used illicit drugs and his medical history was unremarkable. On physical examination the patient's blood pressure was 147/117 mmHg and the heart rate regular at 114 beats per minute. His pupils were mildly dilated. The cardiovascular and respiratory examination were normal, no crackles were heard. The patient, however, appeared hypervolemic. His face had an edematous appearance and he visited the toilet several times while awaiting the results of the initial investigations. Shortly after being admitted, he experienced tonic-clonic seizure, for which midazolam was administered. To rule out intracranial pathology, a brain computed tomography (CT) scan was performed which showed evidence of cerebral swelling (Figure 1).

Laboratory test findings on admission were as follows: sedimentation rate in the first hour: 2 (normal range <15), serum hemoglobin 8.6 mmol/l (normal range 8.5–11.0), leucocytes 13.0x10^9/l (normal range 4–10), sodium 119 mmol/l (normal range 135–145), creatinine 71 umol/l (normal range 65–110), osmolality 265 mOsmol/kg (normal range 275–300) and CK 1147 U/l (normal range <200). The serum ethanol level was 0.4 (normal range < 0.5/00), while the urine osmolality was 460 mOsmol/kg and the urine sodium level was 113 mmol/l. Urine toxicology screens for XTC and methamphetamine were positive. A diagnosis of acute, symptomatic hyponatremia associated with XTC intoxication was made. The patient was initially treated with hypertonic saline (50 ml of 3% NaCl per hour). The neurologist started the patient on intravenous Depakine to prevent new seizures.

Several hours after admission, the patient’s sodium level remained stable at 120 mmol/l. Meanwhile the patient experienced two additional seizures. Due to the patient’s refractory clinical condition, we decided to treat him with a low dose loop diuretic, so 10 mg of furosemide was administered intravenously. A diuresis of 6900 ml in six hours then followed and the hyponatremia was gradually corrected, reaching 137 mmol/l the next morning (24 hours after admission). The patient was discharged from the hospital the following day, without any sequelae.

DISCUSSION

Acute, symptomatic hyponatremia associated with XTC intoxication, as presented here, is rare. This may in turn lead to cerebral edema and seizures with potential fatal consequences. Mechanisms include acute kidney injury secondary to non-traumatic rhabdomyolysis, and the syndrome of inappropriate anti diuretic hormone secretion (SIADH), which can be triggered by MDMA and its metabolites [4–6]. A study from Amsterdam showed that females are particularly susceptible to developing dilution hyponatremia, which may be related to the effects of estrogen on anti-diuretic hormone (ADH) release [7]. Recent investigations have also confirmed that MDMA potentiates the effects of water loading on hyponatremia [8]. Since acute symptomatic hyponatremia requires urgent treatment, we treated the patient with hypertonic saline (5% at a rate of 50 ml/hour). The Adrogue–Madias formula was used to estimate the anticipated rise in the patient’s serum sodium level following treatment [9]. This regimen was the preferred treatment at the time. In recent years, however, a 100 ml bolus of 3% NaCl has become the standard instead of a continuous infusion [4, 10]. The bolus alternative may facilitate a simpler and more rapid management in the emergency department. The use of isotonic saline solutions is not advised since it may exacerbate volume overload and ADH secretion.

Our patient, however, did not well respond to treatment. We realized that SIADH might not have been the sole mechanism in this case. Ecstasy as well as hyperthermia stimulate feelings of thirst. In addition, the advice and common belief at parties where XTC is consumed is to drink large amounts of fluids to avoid hyperthermia. For our patient, this meant that the consumption of hypotonic liquids could explain the refractory character of the course of the disease. The serum sodium level of 119 mmol/l would have developed on the night of the party. During this time the patient’s total body water would have increased by a factor 1.18 (natrium 140/119) or 18%. In males, total body water is approximately 60% of body’s weight. Following this logic, with the patient weighing 80

Figure 1: Computed tomography scan of the brain demonstrating diffuse swelling and edema.
kg, he would have had an intake of approximately 8.6 L (18% of 60 L). This estimation does not include the loss of body fluid from perspiration or urination during the time period in question. The patient had an edematous appearance on arrival and experienced an extensive diuresis after receiving a small dose of furosemide. Therefore, we believe it is likely that the patient had been in a hypervolemic state.

Patients with SIADH are usually euvoolemic and fluid restrictions are the preferred treatment. In contrast with an euvoolemic state, hyponatremia along with a hypervolemic state is usually observed in cirrhosis, heart failure and end-stage renal failure. The patient made a full recovery. The case confirms the clinical course of patients in previous reports. Loop diuretics, and even the osmotic agent mannitol, have been used to manage cerebral edema due to XTC before [2, 11].

CONCLUSION

The case demonstrates that in refractory hyponatremia due to ecstasy, a low dose diuretic can be considered to counter potential volume overload due to polydipsia.

REFERENCES

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