Netherlands
The Journal of Medicine

PHOTO QUIZ

Altered mental status in a young male

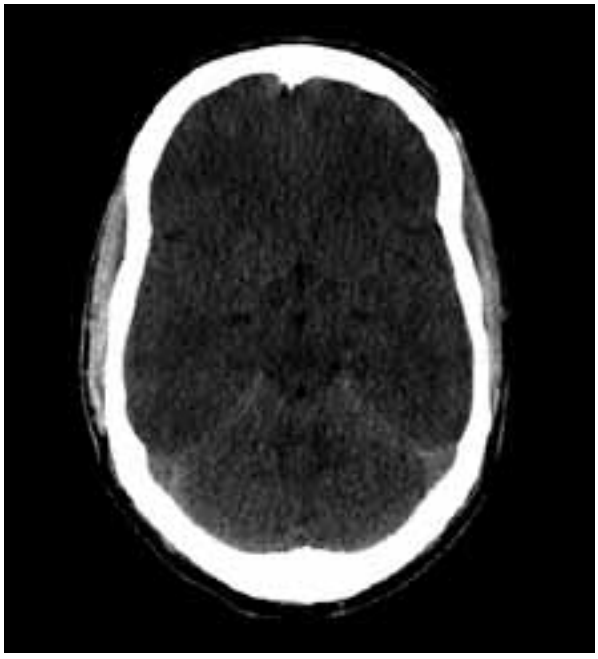
A.D. Pols, P.S. van Dam, G.E.L. van den Berk*

Department of Internal Medicine, Onze Lieve Vrouwe Gasthuis, Amsterdam, the Netherlands,
*corresponding author: g.e.l.vandenberk@olvg.nl

CASE REPORT

A 28-year-old male was brought into the Emergency Department of our hospital by ambulance because of an altered mental status. He had attended a dance party the night before and after going to sleep at 7 am, his girlfriend had not been able to wake him up at 10 am in the morning. She suggested that he might had used recreational drugs at the party. The patient had no medical history and was not taking any medication. Physical examination showed a blood pressure of 130/75 mmHg, heart rate of 70 beats/min, ventilation rate of 16 breaths/min, and a temperature of 35.1 °C. At physical examination he was sweating excessively, his pupils were myotic and only slightly reactive to light. At arrival the Glasgow Coma Scale (GCS) was E1M4V1. The rest of his neurological examination revealed no abnormalities. The capillary refill and skin turgor were normal suggesting an euvolaemic state. There were no signs of head trauma. His ECG showed no abnormalities. Blood glucose was 5 mmol/l, further laboratory evaluation revealed a sodium content of 122 mmol/l and a urinary sodium concentration of 148 mmol/l. Soon after arriving, the patient awoke and was physically aggressive and had to be sedated using 5 mg of midazolam and after that with 50 mg of promethazine.
To rule out intracerebral causes, a non-contrast CT was taken, showing the image as published here (*figure 1*).

Figure 1. *Axial slide of a CT scan of the brain*



WHAT IS YOUR DIAGNOSIS?

See page 284 for the answer to this photo quiz

ANSWER TO PHOTO QUIZ (PAGE 281)

ALTERED MENTAL STATUS IN A YOUNG MALE

DIAGNOSIS

Before the CT scan, we considered several diagnoses to explain the comatose state of our patient. 3,4-Methylenedioxymethamphetamine (MDMA) use with excessive water intake could explain the hyponatraemia and the comatose status. The non-contrast CT taken showed extensive brain oedema. The urine toxicology screening was positive for amphetamines. Combined with the laboratory results the diagnosis of MDMA-induced syndrome of inappropriate antidiuretic hormone synthesis (SIADH) causing symptomatic hyponatraemia with brain oedema was made. MDMA is a party drug that has become more popular over the last decade. Its effects on neurotransmitters are on the serotonin, noradrenalin and dopamine system giving the '3E' effect of euphoria, empathy and energy. The adverse effects can be serious and even fatal. The most dangerous of these being hyperthermia, hepatotoxicity and rhabdomyolysis.[1] It is also associated with long-term damage as significant deficits in neurocognitive function (particularly immediate and delayed verbal memory) and increased psychopathological symptoms.[2] Acute lowered serum sodium levels can also occur, leading to brain oedema with cerebral damage as shown in this case report. ADH release from the neuropituitary has been proven to be affected by serotonin in animal experiments. MDMA, being a serotonin agonist, can stimulate ADH release.[3] Combined with excessive fluid intake it can lead to dangerously low sodium levels which can cause brain oedema.

Fluids were restricted, but as the sodium concentration dropped further, the patient was treated with hypertonic fluid infusion (NaCl 3%). Approximately 12 hours later, the sodium concentration normalised and the patient recovered fully.

REFERENCES

1. Hall AP, Henry JA. Acute toxic effects of 'Ecstasy' (MDMA) and related compounds: overview of pathophysiology and clinical management. Br J Anaesth. 2006;96:678-85.
2. Rogers G, Elston J, Garside R, et al. The harmful health effects of recreational ecstasy: a systematic review of observational evidence [review]. Health Technol Assess. 2009;13:iii-iv, ix-xii, 1-315.
3. Henry JA, Fallon JK, Kicman AT, Hutt AJ, Cowan DA, Forsling M. Low-dose MDMA ('ecstasy') induces vasopressin secretion. Lancet. 1998;351:1784.