Correspondence

Acute polydipsia and water intoxication in first episode schizophrenia

†***David Zilles, Alkomiet Hasan, Oliver Gruber, Detlef Degner,*** *Department of Psychiatry and Psychotherapy, Georg August University, Goettingen, Germany*

Polydipsia and water intoxication are clinical phenomena long known in psychiatric literature. Prevalence rates in populations of chronic psychiatric patients are estimated to be over 20 and 1–5%, respectively [1], with evidence of an association with diagnosis of schizophrenia and chronicity. With respect to aetiology, there is evidence of a neuroendocrine dysregulation of antidiuretic hormone (AVP) secretion linked to psychosis [2,3].

A 26-year-old woman with first-episode schizophrenia (ICD-10: F20.0) repeatedly experienced conditions of anxiety due to delusions that something fatal would happen to herself or her friends and family. Therapy was initiated with quetiapine 100 mg/day and lorazepam. On the third day of the inpatient treatment she experienced increasing psychomotor agitation and showed sudden enuresis, encopresis and vomiting together with reduced vigilance. No epileptic seizure was observed. Blood pressure was 90/60 mmHg, pulse rate 60/min, blood glucose 131 mg/dl. In a routine blood sample taken in the morning (09:00 h) all parameters had been within the normal range except for a mild leukocytosis (sodium 141 mmol/l, potassium 3.5 mmol/l, calcium 2.49 mmol/l). At 13:00 h she was transferred to our Intermediate Care Unit, where a water intoxication with hyponatremia (112 mmol/l), hypokalaemia (3.0 mmol/l) and hypocalcaemia (1.96 mmol/l) was diagnosed. After electrolyte correction the patient was re-admitted to our psychiatric department. She stated that she had drunk six half-litre bottles of mineral water (sodium 17.5 mg/l) within half an hour. As an explanation she said she had been very agitated and in the past it had always helped her drinking a glass of water when being nervous. Antipsychotic therapy was continued with quetiapine (maximum daily dosage 700 mg) for a 3-week period and then switched to olanzapine due to lack of efficacy.

What is noteworthy about this case is the rapidness of the development of a severe symptomatic hyponatremia within less than 4 hours documented by two consecutive blood samples. In contrast to many reports about polydipsia as a more 'chronic' condition occurring preferentially in chronic schizophrenic patients, our patient experienced her first psychotic episode and had only one single event of polydipsia and hyponatremia. Neither before nor afterwards was there any deviation of the sodium levels. The patient was only treated with quetiapine for 2 days prior to the event and therefore an influence of the antipsychotic medication is extremely unlikely, particularly as quetiapine was uptitrated afterwards without any further complications. Regrettably, we did not examine AVP levels during acute psychosis to support or falsify the hypothesis of a neuroendocrine dysregulation in this case. However, the singularity of the event together with the statement of the patient leads to the assumption of a more psychological mechanism in terms of a psychotic-coping mechanism for stress reduction. An enhanced AVP response during the psychological stress in this acute psychotic state [2] yet may have contributed to the development of the severe hyponatremia.

In conclusion, potentially life-threatening behaviours can occur any time in acutely psychotic patients (also apart from suicidality). Thus, despite the existence of a recent normal blood test treating physicians should always consider de novo acute somatic conditions as they can (per-) acutely develop in psychotic patients.

References

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