the clinical complications. How cholestanol interferes with cell functioning is not fully understood.

Treating CTX patients with chenodeoxycholic acid, a bile acid, restores the bile acid pool, which in turn decreases the synthesis of bile-acid precursors including cholestanol. Berginer *et al.*[6] have shown that patients on long-term treatment with chenodeoxycholic acid display improvement in dementia and other neurological signs.

This is the first southern African black patient reported with this condition. The gene for CTX is therefore found in southern Africa in both the black and white population groups. Patients presenting with xanthomas and a normal serum cholesterol level should be investigated for CTX since this is a potentially reversible condition which, if left untreated, may result in neurological deficits.

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Self-induced water intoxication

A case report

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Summary

A 19-year-old female schizophrenic with self-induced water intoxication is described. Factors of pathogenic significance included primary polydipsia and non-maximal urinary diluting capacity.

S Afr Med J 1988; **74:** 80-81.

Self-induced water intoxication is a condition found almost exclusively in patients with psychiatric disorders, especially of the schizophrenic type.[1] The two most important pathogenic factors are primary polydipsia (also referred to as compulsive water drinking or psychogenic polydipsia) and an inappropriate antidiuretic state.[2] Primary polydipsia alone does not usually lead to water intoxication because of the tremendous excretory capacity of the kidneys when vasopressin secretion is adequately suppressed. However, in the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) maximum water diuresis is prevented and water intoxication may ensue.[3]

Case report

A 19-year-old coloured female was admitted to the Psychiatry Unit, Tygerberg Hospital, with a sudden onset of agitation, verbal hallucinations, paranoid delusions and suicidal behaviour. She had been discharged from a psychiatric hospital 2 weeks previously, where she was diagnosed as suffering from schizophrenia and was treated with trifluoperazine 30 mg/d. When examined she was apathetic and non-responsive. Physical examination revealed nothing of note. The blood pressure was 120/70 mmHg, pulse 60/min and temperature normal. There were no localising neurological signs.

After admission to the ward she again became agitated and confused. She was noted to be drinking water almost continuously, was repeatedly seen going to the toilet and vomited once. Laboratory investigations performed the day after admission revealed the following serum values: sodium 124 mmol/l, potassium 3,7 mmol/l, chloride 92 mmol/l, urea 2,1 mmol/l and creatinine 65 μmol/l. Osmolality was 254 mmol/kg H_2O and glucose 4,7 mmol/l. Concomitant urine osmolality was 151 mmol/kg H_2O. Haematological tests were normal, as were liver function tests, chest radiography, ECG, electroencephalogram and computed tomography of the head.

The trifluoperazine was discontinued and fluid intake restricted. Within 4 days the patient's psychiatric state had returned to normal. Serum sodium value was 137 mmol/l and osmolality 275 mmol/kg H_2O. A standard water load test[4] performed at the time revealed no defect of water excretory capacity. Three months later there had been no recurrence of the polydipsia and her psychiatric state had remained normal.

Discussion

Disturbances in water balance in psychiatric patients have been recognised for over 50 years. Sleeper and Jellinek[5] observed that many schizophrenics exhibited polyuria — and that this ceased when fluid intake was restricted. Subsequently numerous cases of such patients developing water intoxication

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Accepted 10 Dec 1987.

have been reported (see Vieweg *et al.*[6]). In the present case the presence of profound hyponatraemia together with polydipsia, vomiting and confusion accord with a diagnosis of water intoxication.

Most of the patients in published reports of water intoxication displayed primary polydipsia (i.e. abnormal drinking behaviour caused by psychiatric or neurophysiological mechanisms in the absence of need) as well as impairment of urinary excretory capacity. In the present case, the urinary osmolality of 151 mmol/kg H_2O in the face of serum hypotonicity is less than maximally dilute, and indicates a mild impairment of excretory capacity. The most likely cause of this is SIADH.[3] The possibility that the clinical triad of psychosis, polydipsia and SIADH could result from a common underlying cerebral disorder has been suggested by Raskind *et al.*[7] These authors propose that a disturbance in limbic system function, possibly involving a hyperdopaminergic state, could explain the co-existence of psychosis, polydipsia and SIADH. As supportive evidence they cite experimental and clinical studies implicating the limbic-hypothalamic dopamine system of the brain in psychotic illness[8] and in central thirst and vasopressin secretory mechanisms.[9]

The role of medication in the development of water intoxication in our patient and other such cases is uncertain. Thus, the fact that the patient's symptoms improved after stopping the trifluoperazine accords with previous reports of neuroleptic-induced SIADH (see Jose *et al.*[10]). On the other hand, neuroleptic drugs have been shown not to have a pervasive stimulatory effect on vasopressin secretion,[11] and elevated plasma vasopressin values have been reported in medication-free psychotic patients.[12] Other drugs (not present in this case) associated with the development of SIADH in psychiatric patients include tobacco smoking,[13] carbamazepine[14] and thiazide diuretics.[15]

Whatever the mechanisms involved, heightened physician awareness of the condition is required, considering the implications. The prevalence of water intoxication in a state mental hospital has been estimated as 7%,[16] and nearly a fifth of deaths in schizophrenics aged under 50 years were attributable to self-induced water intoxication.[17]

This work was supported by the South African Medical Research Council.

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