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Brief Reports

Psychotic Depression Presenting as Status Epilepticus

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A case of status epilepticus was secondary to water intoxication, which in turn was secondary to a depressive psychosis.

Water intoxication occurs in approximately 50% of psychiatric patients who compulsively drink water, the majority (80%) of whom have schizophrenia (Jose *et al*, 1979). Thus most reports have focused on water intoxication in schizophrenic subjects in whom this potentially fatal syndrome occurs as a late complication. This has been described as the 'pre-terminal stage' by Arieti (1945), developing on average 12–13 years after first psychiatric contact and characterised by diminution in acute symptoms and the development of primitive habits (Vieweg *et al*, 1984). Water intoxication is also associated with other features of chronic or residual illness such as tardive dyskinesia, cerebral ventricular enlargement, poor response to neuroleptics. Previous reports have also suggested that in the majority of cases polydipsia occurs in response to delusional beliefs (e.g. to drown a parasitic worm, to propitiate the Gods, etc.; Singh *et al*, 1985). In addition, the actions of some psychotropic drugs, including tricyclic antidepressants, lithium and major tranquillisers, have been implicated in the aetiology (Illowsky & Kirsh, 1988).

Reports of water intoxication occurring in subjects other than chronic psychotic patients are rare, although a few cases of uncomplicated compulsive drinking have been described in neurotic subjects (e.g. Silber, 1984). In addition, Lee *et al* (1989) have recently reported the case of a 16-year-old Chinese girl who compulsively drank water in order to induce a state of altered consciousness. Apart from this addiction she had no other psychiatric illness and successful treatment consisted of simple advice to stop the activity.

We have only found three previous case reports (Singh *et al*, 1985) which describe water intoxication in patients with affective disorder and in all cases hyponatraemia developed as a late complication, occurring several years after the onset of illness and in subjects who had other disabilities such as personality disorder, poor social or occupational functioning. The case we present adds to this small literature on water intoxication in affective disorder and is also atypical and striking for a number of other reasons. Firstly, hyponatraemia-induced status epilepticus was the patient's first psychiatric presentation although she admitted to previous minor episodes of depression which had failed to come to medical attention. Secondly, our patient's stated reason for drinking excessively were not delusional but were in response to poor appetite. Thirdly, she had not been taking any psychotropic medication. Lastly, she made a complete recovery from the episode and, unlike previously described psychotically ill cases, showed no deterioration of functioning at follow up.

Case report

EK, a 58-year-old married woman with two children had never been admitted to hospital until December 1987 when she gave up her part-time job in the local post-office because of non-specific chest pain. She was admitted for investigation of possible cardiac pathology. However, although noted to be pale and tired, all investigations were normal and she was discharged from hospital two days later.

She was admitted for the second time in July 1988 in status epilepticus. Renal investigations on admission gave the following results: in serum, sodium 117 mmol/l, potassium 3.9 mmol/l, chloride 85 mmol/l, urea 2.2 mmol/l, creatinine 71 μmol/l, osmolality 241 mOsmol/kg; in urine, osmolality 429 mOsmol/kg, sodium 87 mmol/l, potassium 47 mmol/l, urea 114 mmol/l.

All other investigations including full blood count were normal. She was resalinated and regained consciousness after 14 hours. Twenty-four hours after admission, she was fully orientated and serum electrolytes also reverted to the normal range. Subsequent investigations including liver and thyroid function tests, gamma-glutamyl transpeptidase, urine culture and computerised tomography headscan, barium meal and chest X-ray were all within normal limits, as was the electroencephalogram. Renal response to water loading was not assessed.

When interviewed by the duty psychiatrist she complained of marked loss of appetite and had lost weight (4.5 kg in three months). To compensate for her poor appetite and to ensure adequate hydration, she had started drinking large quantities of water and tea. For three months before admission her fluid intake had gradually increased although precise details of this were not available either from the patient or her husband. Her husband did report, however, that she was drinking almost continually for two days before the first seizure. Thus her fluid intake was relatively constant and showed no fluctuation. She gave a six-month history

of early morning waking, anhedonia, poor concentration and gloomy thoughts concerning the future. She was not actively suicidal but did admit to feeling that life was not worth living. She had numerous physical complaints including pain in her back, arms, chest and abdomen. She had become increasingly withdrawn and easily confused and was unable to do the shopping or housework without her husband's assistance. She admitted to previous brief episodes of low mood over the past 10 years. However, these had been quite mild and had never caused her to stop work or seek treatment but were associated with an increase in fluid intake, although never previously to the degree on this occasion.

Her birth and upbringing in west Wales were unremarkable and she had moved to the Rhondda valley at age 15 years on account of her father's job as a Methodist minister. She left school unqualified and married her husband, a physics teacher, at the age of 19 years. Both her younger brother and her son had received psychiatric help for alcohol dependence. Her three older sisters have no psychiatric problems and both parents were deceased at the time of her illness. There was no family history of epilepsy and the patient herself had not previously had any seizures.

Pre-morbidly, EK described herself as self-confident, outgoing and someone who led a full and enjoyable life. She neither drank alcohol nor smoked. Her financial circumstances were secure, although her husband had lost a large proportion of his retirement 'lump sum' in the stock market crash of October 1987. During her illness, this fact had greatly concerned her.

Fourteen days after admission, she was transferred for psychiatric treatment. On arrival, she had little recollection of events leading to her admission and at interview appeared tense and agitated. Her speech was spontaneous and appropriate but rather abrupt in character with short sentences and minimal information given in response to questions. Her mood was depressed and thought-content included preoccupation with her illness, financial problems and the stigmatising effect of being a psychiatric patient. She expressed sensitive ideas of reference and persecutory ideation in connection with the comments she had made to the relatives of another patient, while on the medical ward. Following this she felt that the staff were talking about her and that she and her husband were going to be sued by these relatives and by the health authority. These ideas subsequently became delusional in intensity but rapidly responded to medication. She was correctly orientated and her memory was intact, although her performance on serial 7s and reverse months of the year was poor. She had limited insight into the psychiatric nature of her illness.

A diagnosis of psychotic depression was made and treatment with amitryptiline (100 mg nocte) commenced. Trifluoperazine (5 mg b.d.) was added and amitryptiline increased following the development of persecutory delusions. She responded rapidly to this regime and within four weeks of admission, her mental state had returned to normal. She was followed up as an out-patient for six months after which she was discharged without medication and symptom free.

An interview using the Schedule for Clinical Assessment of Neuropsychiatry (SCAN; Wing, 1988) was carried out one week after discharge and 10 weeks after presentation. Symptoms were scored for the episode of illness. The results are as follows: CATEGO-4 class R+, Index of Definition 8 (Wing & Sturt, 1978); tentative ICD-9 classification 296.2 (World Health Organization, 1978); ICD-10 diagnosis F31.0, severe depressive episode with psychotic features and mood congruent delusions (World Health Organization, 1988); DSM-III-R 296.24, major depression, single episode with psychotic features (mood congruent delusions) (American Psychiatric Association, 1987).

Discussion

We believe this to be the first case reported in which depressive illness has presented with status epilepticus induced by compulsive water drinking.

There have been three previous reports of the occurrence of self-induced water intoxication in depression (Singh *et al*, 1985), but the diagnosis of depression has always ante-dated the development of water intoxication and has developed in subjects with personality disorder or chronic illness.

EK stated that she was drinking excessively in a response to poor appetite and a wish to remain adequately hydrated. She complained of feeling very dry and having a choking feeling which she sought to alleviate by drinking. However, despite close questioning there was no evidence of any delusional belief system underlying her decision to drink excessively, and her explanation seems entirely plausible.

Eighty per cent of patients who develop water intoxication have schizophrenia but this is clearly not the diagnosis here. Although EK developed some persecutory delusions, these occurred late in the course of this episode, were not a prominent part of her illness and responded rapidly to treatment. A structured interview carried out just before discharge showed that the diagnosis was one of psychotic depression according to three different classifications, ICD-10, DSM-III-R and CATEGO-4.

In conclusion EK is clearly unusual in that she is not the type of chronic disabled patient with end-stage illness that is most commonly associated with water intoxication. Indeed, she remains a well integrated member of her local community. Her presentation was dramatic and life threatening but she made a complete recovery, and she was discharged from out-patient follow-up, and was free from symptoms and medication six months following admission.

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Leucopenia Secondary to Carbamazepine Despite Concurrent Lithium Treatment

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A manic–depressive patient developed leucopenia as a result of carbamazepine therapy, despite concurrent administration of lithium.

Haematological complications of carbamazepine therapy are well recognised (Pisciotta, 1975). Leucopenia is the most frequently reported haematological side-effect (Hart & Easton, 1982). In contrast, lithium is known to cause a leucocytosis (Shopsin *et al*, 1971), and it has been used in the treatment of agranulocytosis (Boggs & Joyce, 1983). Hence its effect should theoretically oppose the myelosuppressant effect of carbamazepine. We report a case of a 37-year-old man who developed leucopenia because of carbamazepine therapy despite concurrent lithium administration.

Case report

A 37-year-old male was admitted in September 1988 with mania (ICD–9, 296.0; World Health Organization, 1978). He was on lithium carbonate (1000 mg nocte) and his serum level was 0.82 mmol/l. He had a three-year history of manic–depressive psychosis and had been commenced on lithium in September 1987 after two previous episodes of mania which had required admission to hospital. His serum lithium in October 1987 was 0.67 mmol/l.

He required, however, readmissions in February 1988 and in August 1988, with further episodes of mania. His serum lithium concentration in February was 0.94 mmol/l and in August was 0.72 mmol/l. His episode in August was followed by three weeks of depression.

Family psychiatric history revealed a sibling had also been diagnosed as having manic–depressive psychosis.

On admission in September 1988, he was overactive, restless and had been spending money extravagantly. He had grandiose ideas but was not deluded. Therapy was commenced with haloperidol (10 mg t.d.s.) orally and the lithium was continued. His mood settled slowly but by three