WATER INTOXICATION IN PSYCHIATRIC PATIENTS IN SINGAPORE

L H Peh, G S Devan, P W Eu

ABSTRACT

The syndrome of water intoxication may occur in psychiatric patients and various hypotheses regarding its aetiology have been postulated. Twenty-seven patients in Woodbridge Hospital were found to have this syndrome. The aim of the study was to describe the clinical and biochemical findings of this group of patients. 70.4% had schizophrenia, 25.9% had mental retardation and 3.7% had a history of alcohol dependence. Many of them were on antipsychotic medication. The symptoms of water intoxication included polyuria, nausea, tremors, weight gain, disorientation, coma and fits. A majority of the patients had hyponatraemia during the acute stages and the osmolality of urine and plasma were correspondingly low. A few patients had abnormalities in electroencephalogram and computerised axial tomography of brain. The management of patients with water intoxication is discussed briefly.

Keywords : Water intoxication, Psychogenic polydipsia, Hyponatraemia.

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INTRODUCTION

The syndrome of water intoxication was comprehensively reviewed by Barlow and De Wardener in 1959 (1) and recently by Illowsky and Kirch (2). Early symptoms include polydipsia, polyuria, headache, blurred vision, vomiting, diarrhoea, weight gain, excessive perspiration and incoordination while severe cases may develop delirium, coma and convulsions. Biochemical investigations often include hyponatraemia and a decreased urine specific gravity, and low serum and urine osmolality. The prevalence amongst psychiatric patients was estimated by Jose (3) to be 6.6% for psychogenic polydipsia and 3.3 % for water intoxication in a mental hospital. The criteria for water intoxication has not been clearly defined but usually would include excessive water drinking with clinical symptoms and biochemical changes as described.

Psychotic patients may develop water intoxication while on neuroleptic medication and in some cases the water intoxication has been associated with SIADH (Syndrome of inappropriate secretion of anti-diuretic hormone). Such medication includes haloperidol, chlorpromazine, thioridazine and fluphenazine. Smith and Clark (4) speculated that antipsychotic drugs, by blocking dopaminergic activity, and through denervation supersensitivity cause aberrations of dopamine-dependent central nervous system functions such as thirst, drinking behaviour and anti-diuretic hormone secretion. The anticholinergic effect of such medication leading to thirst has also been implicated. It has also been postulated by Singh et al that in schizophrenic patients who are not on any medication, a hyperdopaminergic mechanism underlying the illness may be responsible for water intoxication not due to SIADH (5)

In the Asian context, the condition of water intoxication has hardly been reported. Singapore, with its two and a half million people of mixed ethnic groups of Chinese, Malay and Indian, is served by one main mental hospital with a bed capacity of 2,300 and the majority of the patients are psychotic and about half of these are long-staying patients.

In a one-month survey conducted by the authors in this hospital, there were 27 cases of water intoxication. We present the clinical data and biochemical findings of these patients as follows:-

MATERIALS AND METHODS

Nurses in all the wards were informed to notify the authors of inpatients who had symptoms suggestive of water intoxication, or had excessive drinking warranting fluid restriction or actually documented water intoxication before. Effort was made in visiting each ward to ensure that such patients were not left out in the study.

The case records of these patients were then looked into for demographical and clinical data which included the diagnosis of mental illness, physical illness, biochemical investigations carried out in the past, and the symptoms displayed. In all the cases, urine specific gravity, urine and serum osmolalities were done. In cases with suspected SIADH, inferred from an inappropriately

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concentrated urine in the presence of a hypo-osmolar plasma, the patients were to be investigated for a possible underlying cause of the SIADH. Where available, other investigations which had already been carried out such as electroencephalogram (EEG) and computerised axial tomography of the brain (CAT Scan), have also been recorded.

RESULTS

(A) Clinical Phenomenology

1. There were a total of 27 inpatients with the syndrome of water intoxication. Most of these were chronically ill psychiatric patients, and 19 (70.4%) of them were diagnosed to have schizophrenia (meeting DSM III criteria) and 7 (25.9%) had mental retardation and one (3.7%) had a history of alcohol dependence syndrome. 90% of those with schizophrenia had a duration of psychiatric illness of over 10 years, the mean being 17.9 years. The average duration of symptoms of excessive water drinking was 3.7 years in these patients, with an average interval of 12.9 years from the start of schizophrenic illness to these symptoms.

Those diagnosed as mentally retarded were of an average age of 34.7 years at the time of study and the average duration of symptoms of excessive water drinking was 4.5 years, with these symptoms starting at an average age of 26.8 years. Three of these patients had also been diagnosed as psychotic and the symptoms of excessive water drinking started at about 11.5 years after the start of psychotic symptoms.

2. The number of patients with water intoxication was higher in women: 17 (63.0%) were female in this series. Chinese patients made up the majority of patients, 25 (92.6%), and there was one Indian patient (3.7%) and one of other race (3.7%). None was of Malay race.

3. With regards to physical illnesses, 7 (25.9%) had an array of diagnoses including epilepsy (2), non-toxic diffuse goitre (1), liver cirrhosis (1), pulmonary tuberculosis (1), anaemia (1) and Sturge-Weber Syndrome (1). From examination of case records, one of the patients with epilepsy was likely to have fits as a complication of his water intoxication. However he continued to receive anticonvulsant treatment at the time of study.

4. Medication prescribed to the patients at the time of study included Chlorpromazine (17 patients), Thioridazine (3), Haloperidol (10), Trifluoperazine (1), Fluphenazine Decanoate (21), Flupenthixol Decanoate (1), Amitriptyline (1), Lithium carbonate (6), Carbamazepine (5) and Diazepam (9).

(B) Clinical Presentation

Some of the patients were habitual in excessive water consumption (18 or 66.7%) and the others were episodic in this behaviour (9 or 33.3%). The amount of water consumed per day could not be accurately determined, but in 9 patients it was roughly estimated to be over 3 litres. Symptoms included polyuria (6 patients), nausea (14), tremors (1), weight gain (8), disorientation (5), coma (3) and fits (11). It could not be accurately determined whether at the time of excessive water consumption these patients were acutely psychotic, as the case records of some of the chronic patients were not kept in detail.

(C) Biochemical Findings

1. Sodium Levels: Majority of these patients had hyponatraemia during the acute stages of water intoxication, with sodium levels improving with fluid restriction and on the low side even during intervals between episodes of water intoxication (see Tables I, II and III).

2. Osmolality Levels: The osmolality of urine during water intoxication were low, and those of plasma were correspondingly low (see Tables IV and V). Only in one patient was there a hypo-osmolar plasma together with a normal concentrated urine. This, however, is not strongly suggestive of SIADH as in that case the urine will be inappropriately concentrated.

3. Urine Specific Gravity: This was found to be low even in the non-acute stages and on the average the urine S.G. was 1.009.

Table I
Sodium Levels during acute stages of Water Intoxication (available in 16 out of 27 patients)

Sodium Level (mmol/L)	No.	%
<115	4	25.0
115-119	6	37.5
120-124	2	12.5
125-129	3	18.7
130-134	0	0.0
≥135	1	6.3
Total	16	100.0

Table II
Sodium Levels on Recovery (available in 16 out of 27 patients)

Sodium Level (mmol/L)	No.	%
<115	0	0.0
115-119	0	0.0
120-124	0	0.0
125-129	1	6.3
130-134	6	37.5
≥135	9	56.2
Total	16	100.0

(D) Other Investigations

1. Electroencephalogram (EEG): This was carried out on 10 patients, with 5 of them showing abnormalities. These included non-specific bilateral disturbances in 4 patients and generalised slowing down of activity but with no focal activity in another. No baseline records of EEG were available before the onset of symptoms of water intoxication.

2. Computerised Axial Tomography of Brain (CAT Scan): This was done on 10 patients, with 3 of them

showing abnormalities. These were: cerebral atrophy with normal ventricles in a patient with alcoholic liver cirrhosis and cerebral atrophy with enlarged ventricles in two schizophrenic patients who had negative features.

3. T4 was done in 8 patients, the results of which were all normal.

Table III
Sodium Levels in Non-Acute Stages (available in 6 out of 27 patients)

Sodium Level (mmol/L)	No.	%
<115	0	0.0
115-119	0	0.0
120-124	1	16.7
125-129	1	16.7
130-134	2	33.3
≥135	2	33.3
Total	6	100.0

Table IV
Urine Osmolality Levels (available in all 27 patients)

Urine Osmolality (m Osm/L)	No.	%
<200	14	51.9
200-499	11	40.7
500-1200	2	7.4
>1200	0	0.0
Total	27	100.0

Table V
Plasma Osmolality Levels (available in 26 out of 27 patients)

Plasma Osmolality (m Osm/L)	No.	%
<275	9	34.6
275-305	17	65.4
>305	0	0.0
Total	26	100.0

DISCUSSION

The diagnosis of water intoxication is based on clinical signs and symptoms, as well as biochemical findings of hyponatraemia and low plasma and urine osmolality in most of these patients. Hyponatraemia has been consistently reported in other cases eg. by Hariprasad et al, 1980 (6).

The terms used to identify psychiatric patients with polydipsia and polyuria with or without accompanying hyponatraemia have included "compulsive water drinking", "self-induced water intoxication" and "psychogenic polydipsia". Illowsky and Kirch (1988) felt that it was more accurate to limit the labels used to the simplest descriptive terms i.e. "polydipsia" and "hyponatraemia" in psychiatric patients (2), to avoid implying elements that are not necessarily involved, such as wilfulness, compulsion, or psychological drive.

Schizophrenia is the most frequent psychiatric diagnosis, being found in 69% to 83% of these patients (2) and in this study, 70.4%. Mental retardation and alcoholism have also been associated with polydipsia. Women have also been noted to have a higher prevalence of water intoxication (3). The association of water intoxication and Chronic Schizophrenics with negative features and venticular enlargement on CAT Scan has also been noted in other studies (2). This was found in 2 of our patients. Most of the EEG abnormalities noted were non-specific. Structural defects in the brain may have a part in the aetiology of water intoxication but this has yet to be proven.

The onset of polydipsia has been thought to develop 5 to 15 years after the onset of pychiatric illness. The mean interval reported in in our schizophrenic patients is 12.9 years between the onset of psychosis and the first symptoms of water intoxication. The mentally retarded patients who also had psychosis had a similar mean interval.

In terms of clinical presentation, most of the patients come to attention because of initially unexplained fits or coma. In some cases water seeking and water drinking was noted by staff, but this may be subtle, as the behaviour may be concealed by the patient. Weight gain, polyuria, disorientation, nausea, and fits or coma seem to be the commonest signs in water intoxication here.

The cause of water intoxication in our cases is difficult to determine but there are a number of possibilities. It might be related to the drugs that the patients were taking (7). Psychotropic drugs have been implicated in producing SIADH. However, in most of the patients there was no suggestion from the plasma and urine osmolality that there was SIADH, as both were correspondingly low. In the patient with pulmonary tuberculosis or alcoholic cirrhosis, these medical illnesses could have been a cause of SIADH but SIADH itself was not documented.

Psychosis itself could have brought about the polydipsia. At neurotransmitter level this polydipsia could have been explained by hyperdopaminergic activity. Animal experiments have shown that a hyperdopaminergic state is associated with increased fluid intakes and dopamine depletion is associated with decreased fluid intake; dopamine appears to be a common link between psychosis, thirst and drinking behaviour (2).

The management of patients with water intoxication lies in their careful evaluation. Identification of such patients includes a proper history of amount of fluid consumed and excreted, water-seeking behaviour, and signs and symptoms of water intoxication. The use of medication in relation to these episodes and focal neurological deficits should be elicited. Laboratory investigations should include electrolytes, plasma and urine osmolality, urine specific gravity. If SIADH is suspected, endocrine function, liver function test, Chest X-ray and CAT Scan may be ordered.

Restriction of fluids is necessary for cases of water intoxication. A useful method is that of prevention with

target weight procedure described by Goldman and Luchin, 1987 (8). Other methods of treatment have been described including the use of of high dose propranolol (9) and the infusion of hypertonic saline (10). However rapid correction of hyponatraemia can be dangerous as it can cause demylelination of the pons.

It is important to keep in mind that fatalities have been reported in water intoxication eg. by Raskind (11) and Rendall et al (10). If the psychiatrist or physician is alert as to its possibility then early diagnosis and prevention of water intoxication may help reduce fatalities.

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