CATATONIA AND HYPONATREMIA : A CASE REPORT

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SUMMARY

We report a case of recurrent mania who developed catatonia and was found to be markedly hyponatraemic. The catatonic symptoms showed rapid resolution following correction of hyponatraemia suggesting a causal link between the two. It is suggested that the estimation of serum electrolyte levels should be an important part of the evaluation of cases of catatonia.

Catatonia is a syndrome with various possible causes (Gelenberg, 1976). Besides psychiatric illnesses, a wide variety of neurological and metabolic disorders and toxic and pharmacological agents are thought to be aetio-logically related to the clinical syndrome of Catatonia. Hypercalcaemia is the only electrolyte disturbance reported so far to cause catatonia (Carmen and Wyatt, 1977). We report here a case of mania who developed catatonia in the presence of marked hyponatraemia and showed rapid resolution of symptoms following correction of the hyponatraemia.

Case Report

RMS, a 45 year old, right handed, married man, with past history of four manic episodes over a period of 19 years, was admitted with one-month history of symptoms suggestive of a manic illness. On examination he showed cheerful mood, increased psychomotor activity, pressure of speech and grandiose delusions. He was treated with haloperidol tablets 10 mg b.d. and was fit enough to be discharged after two weeks.

Following discharge he discontinued treatment and had a relapse necessitating readmission after 6 weeks in a floridly manic state. Treatment with tablet haloperidol in the dose of 5 mg b.d. was restarted. On the sixth day of hospitalisation he was found to be sebrile (Temp. 38°C). Systemic examination was unremarkable and routine laboratory investigations were within normal limits. He was treated with cotrimoxazole and paracetamol for four days and haloperidol was discontinued. He was afebrile in two days time. As he continued to be psychotic, tablet haloperidol in the dose of 5 mg b.d. was restarted. Four days later he was found to be mute, lying in bed in knee-elbow position and repeatedly pulling down his ears, refusing feeds and making very few responses to verbal commands. He was actively negativistic and had parathetic rigidity. He was conscious, afebrile with stable vital signs, there was no dehydration the fundi were normal and general systemic examination revealed no abnormalities. A detailed assessment of higher mental functions was not possible because of the patient’s inaccessibility. An hour later he jumped out of the bed, tore his clothes and assaulted one ward attendant. He remained excited and combative over the next half an hour and had to be sedated. There was no history of excessive water intake or polyuria.

Laboratory investigations showed normal total and differential leucocyte count, haemoglobin 12.5 gm/100 ml, ESR...
20 mmHg/1st hour, blood urea 20 mg/100 ml, serum creatinine 1.3 mg/100 ml, blood sugar (random) 91 mg/100 ml, serum Na+ 116 mmol/L and serum K+ 4.3 mmol/L. EEG and ECG were within normal limits.

In view of the marked hyponatremia, intravenous normal saline drip was started. He received a total of 2200 ml over the next 24 hours. He was also given drinks containing table salt. The next day he was more accessible, responding to verbal commands and moving out of the bed. He had his meals but speech was limited to monosyllables. Repeat serum electrolytes estimation showed Na+ 129 mmol/L and K+ 4.4 mmol/L.

Following the resolution of catatonic symptoms he showed rapid improvement in his psychosis. The subsequent two weeks of hospitalization was uneventful. Over the next two years of regular follow-up he has been on small dose of HPL and remained asymptomatic.

Discussion

This was an established case of recurrent mania. He developed symptoms of catatonia for the first time in his fifth manic episode, four days after a non-specific febrile illness, while receiving haloperidol. He was found to be hyponatraemic without polydipsia or polyuria. The catatonic symptoms showed dramatic resolution following correction of hyponatraemia despite the continued use of HPL. The catatonia was unlikely to be of infective origin since he had mild fever for two days only and was afebrile for four days before the onset of catatonic symptoms. Some 80% of acutely ill patients with bipolar affective disorder are reported to exhibit clinical signs of catatonia (Taylor and Abrams, 1977). However, in the presence of severe hyponatraemia and remarkable recovery on Na+ replacement the catatonia was unlikely to be due to manic psychosis. A syndrome of catatonia in patients receiving high potency antipsychotic drugs has also been described (Geelenberg and Mandel, 1977). The onset of such symptoms, however, is often gradual and does not respond immediately to even discontinuation of antipsychotic treatment while this patient continued to be on the same dose of haloperidol that he was on before its onset. By the same token, he is unlikely to be a case of neuroleptic malignant syndrome since he was afebrile during the period of catatonia. Also he recovered while still on HPL. The cause of hyponatremia in this case remained elusive. He did not have excessive water-drinking and the syndrome of inappropriate secretion of ADH (SIADH) was unlikely to be the cause of hyponatremia, since hyponatremia in SIADH worsens with fluid load due to water retention while it improved in this case. Lastly catatonia is not reported to occur with SIADH. However, the urine and serum osmolality and urine Na+ were not measured.

Hyponatraemic encephalopathy is known to cause headache, blurred vision, weakness, much tremor and cramps, nausea and vomiting, diarrhoea, excessive salivation, restlessness, confusion, lethargy, psychosis, seizure, coma and death (Epstein, 1980). Focal neurological signs have also been described. Clinical differentiation of an exacerbation of a psychosis from early signs of hyponatraemic encephalopathy may be difficult. Psychiatric and manic symptoms have been reported with hyponatremia. The reverse is also true and has more often been the focus of attention. Hyponatremia and water intoxication have been reported in chronic schizophrenic inpatients (Goldman et al., 1988) and affective psychosis (Zubenko et al., 1988). Recently, Viette et al. 1988 have described a syndrome of psychosis, intermittent
hyponatraemia, and polydipsia (PIP). However, the association of hyponatraemia and catatonia has rarely been reported. Rowntree and Kay (1952) observed fluctuations in serum and urinary Na⁺ in two catatonic patients and Shulack (1944, 1946) reported remarkable improvement in cases of lethal catatonia on adrenal cortical extract and 10 mg/day of sodium chloride.

The onset, presentation, and course of catatonic symptoms and its relation to the serum Na⁺ level lead us to conclude that the catatonic symptoms were essentially related to hyponatraemic encephalopathy. It is suggested that estimation of serum electrolyte levels should be an important part of the evaluation of cases of catatonia.

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