CASE REPORT

Hyponatraemia-associated catatonia

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SUMMARY

This is a case report highlighting the impact of low sodium levels on one's mental status. For the first time in the literature, we see two discrete episodes of catatonia associated with hyponatraemia occurring in the same patient. On both occasions, his serum sodium levels were depleted; he became profoundly psychotic with catatonic symptoms. Recovery, with complete resolution of all symptoms, was associated with normalisation of these levels. This case exemplifies the profound effect that electrolyte imbalances, in particular sodium depletion can have on a patient's mental status and is for the first time, we see this effect on two temporally unrelated occasions in the same patient.

BACKGROUND

Catatonia is a syndrome of altered motor behaviour accompanying many general medical and neurological conditions.¹ It manifests with significant behavioural changes (from mildly decreased speech output to complete mutism) that interfere with the ability to communicate.² Hyponatraemia is the most common electrolyte abnormality detected, yet hyponatraemia-induced catatonia is an uncommon presentation of this condition. This case highlights the association of the two conditions, particularly as it occurs twice in the same individual.

CASE PRESENTATION

A 69-year-old man was referred to the emergency department for increased confusion, anxiety, pallor and altered mental state. History revealed that he had memory problems and confusion for an unknown length of time. He had also suffered some sleep disturbance and related feeling very depressed since the death of his wife one and a half years earlier.

A collateral history from his daughter described the recent onset over a few weeks of altered behaviour which, when analysed, seemed to suggest impaired thought processing with obscure decision-making, some paranoid ideation and the occurrence of visual and auditory hallucinations which were distressing for him.

Medical examination revealed a tender epigastrium and a low haemoglobin (Hb) of 9.9. It was also noted at this time that his serum sodium level was 125.

Precipitating factors included the death of his wife one and a half years earlier which led to depression. There was also a suggestion of new-onset cognitive impairment which was felt to represent pseudodementia in the context of mood disturbance. He had

been prescribed escitalopram and was also using non-steroidal anti-inflammatory drugs for arthritis-related pain.

He was not found to have any significant psychiatric history of note other than the recent depression that he had suffered following the death of his wife one and a half years earlier. He was treated by the general practitioner for low mood with sleep disturbance at this time.

He had an extensive medical history including hypothyroidism, arthritis, hypertension, myocardial infarction, implanted cardiac defibrillator and congestive cardiac failure.

There was no known family history of mental health problems.

The man had been living independently and was functioning well on his own up until he was admitted to the hospital. His daughter lived nearby and provided good social support when required.

Mental state examination found him to be restless, confused and perplexed. Hypersalivation and a tendency to stiffness were noted. Speech was slow but normal in form. His mood was subjectively and objectively depressed. Thought pattern exhibited slow cognitive style with circumstantial speech and thought content revealed experiences of profound derealisation. He described auditory hallucinations in the form of a group of people providing a running commentary on his actions and frightening visual hallucinations of various well-formed images, but in particular of an elderly Chinese woman who was very angry with him and who was attacking him verbally. He had poor insight into his condition.

Treatment was commenced with medical management of anaemia, the discontinuation of escitalopram and the commencement of haloperidol and benzodiazepines on an as-required basis for agitation.

INVESTIGATIONS

Mini-Mental State Exam 25/30 indicating mild impairment.

Montreal Cognitive Assessment 20/30 indicating moderate impairment.

Geriatric Depression Scale 15/15.

Physical exam—tender epigastrium.

Blood tests—full blood count, low Hb; urea and electrolytes, hyponatraemia 125; liver function tests, normal; thyroid function tests, normal; creatine kinase (CK), 229 (elevated slightly); C-reactive protein, 22 (slightly elevated).

Oesophagoduodenoscopy—active upper gastrointestinal bleed.

CT brain—normal brain parenchyma and ventricular systems.

CrossMark

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Lumbar puncture—normal.

DIFFERENTIAL DIAGNOSIS

Bleeding duodenal ulcer

F05 Delirium, not induced by alcohol and other psychoactive symptoms.

F05.1 Delirium superimposed on dementia.

F06.1 Organic catatonic disorder.

F44.2 Dissociative stupor.

F32.3 Severe depressive episode with psychotic symptoms.

Organic cause—Neuroleptic malignant syndrome/Creutzfeldt Jakob disease/encephalitis.

TREATMENT

The patient was treated medically with the transfusion of 2 units of red cell concentrate and intravenous proton pump inhibitor for his active upper gastrointestinal bleed.

He was reviewed by the consultant the following day who found him to be psychically retarded, perplexed, distractible and restless. The patient was unable to give a history or interact during the consultation. The overall impression was that of an acute confusional state superimposed on a background of depression and possible dementia, with some features of Lewy body dementia. Treatment with alprazolam was recommended and commenced.

OUTCOME AND FOLLOW-UP

In spite of the correction of the upper GI bleed, the patient became increasingly confused and disorientated. He stopped communicating and fell into a state of generalised rigidity and stupor which was interspersed with periods of rhythmic jerking movements believed to represent pseudoseizures. He stopped eating and drinking and remained either seated or lying for prolonged periods with a fixed blank stare. He was completely unresponsive to external stimuli. Treatment with midazolam and oral haloperidol was initiated with minimal effect. Haloperidol was discontinued after a brief period due to the possibility of neuroleptic malignant syndrome, given the slight elevation in CK. Midazolam was changed to diazepam and his symptoms eventually resolved completely with diazepam 10mg intravenously three times a day.

He was discharged home 3 weeks after his initial presentation and was followed up in the community by the community mental health nurse and at regular Out Patient Department clinics.

A review of his blood test results during his admission showed a presenting hyponatraemia level of 125 with subsequent normalisation of sodium levels. This was then followed by a spike of hypernatraemia to a level of 153 before it eventually normalised again. Symptom resolution occurred after sodium levels were normalised.

He presented again 3 years later with acute onset of confusion, slurred speech and paranoia. Sodium levels on admission were found to be reduced to a level of 116. Shortly after his admission, he became catatonic once more and exhibited stupor, negativism, waxy flexibility and echolalia. Sodium levels were corrected and he recovered very quickly on this occasion. He was found to be fully orientated and alert within 7 days of his admission. Symptom resolution was again associated with correction of sodium imbalance.

DISCUSSION

Hyponatraemia is the most common electrolyte abnormality encountered in clinical practice. It is associated with multiple

poor clinical outcomes and is often managed suboptimally because of inadequate assessment and investigation.³ There are many physical causes of low sodium levels including tumours, infection and trauma. Many commonly prescribed drugs such as thiazide diuretics, selective serotonin reuptake inhibitors, proton pump inhibitors, ACE inhibitors and loop diuretics are strongly associated with this side effect. Hyponatraemia can manifest in an array of clinical symptoms, including those of catatonia.

Catatonia is a neuropsychiatric disorder characterised by 'immobility, mutism, negativism (active or passive refusal to follow commands), mannerisms, stereotypies, posturing, grimacing, excitement, echolalia, echopraxia, muscular rigidity and stupor, sometimes punctuated by sudden violent outbursts, panic or hallucinations'.⁴

There are only a handful of published case reports presenting data on hyponatraemia and catatonia. While one case series⁵ suggests that hyponatraemia in a psychotic patient may be a physiologic sign of catatonia and may be due to abnormalities in vasopressin transmission and secretion, other studies would suggest that the opposite is true. Anglin *et al*⁶ carried out a literature review looking at neuropsychiatric symptoms in patients with Addison's disease and found catatonia to be a rare but documented complication of the disease, suggesting that hyponatraemia can cause catatonia in susceptible patients. Grover *et al*⁷ described a case of venlafaxine-induced hyponatraemia which was temporally related to the onset of catatonia and delirium, again suggesting a causative role for hyponatraemia. This is given further credence by a similar case described by Novac *et al*² who also attempted to elucidate the underlying mechanism of this process. They hypothesise that self-related processing, which is a vertical processing system involved in the integration of bodily functions and basic emotional systems, is the responsible pathway and they also suggest that the neurochemical dysfunction potentiates the observed response.

On review of our patient's blood tests during his admission, it is also noted that sodium levels fluctuated significantly to a peak of hypernatraemia shortly after his admission before normalising. Resolution of symptoms did not occur immediately with the correction of the Na levels but required treatment with benzodiazepines. This is reflected in similar cases^{1 2 8} where correction of the hyponatraemia did not immediately result in resolution of the symptoms of catatonia, but also required treatment with benzodiazepines.

While there were the confounding factors of anaemia, depression and pseudodementia in the patient's initial presentation, his subsequent episode presented a very clear-cut association of hyponatraemia and catatonia with delirium which resolved with the correction of the electrolyte imbalance and treatment with benzodiazepines.

This case illustrates the somatic mechanisms of psychic disturbances and highlights the importance of assessing patients for a physical cause when presenting with new-onset psychotic symptoms. It also reminds us of the need to be cautious when prescribing for the elderly, given the profound morbidity that

Learning points

- ▶ Search for an underlying physical cause in a patient with new-onset psychosis.
- ▶ Be vigilant in relation to side effects of medication.
- ▶ Liaise with primary care especially when a drug-related adverse reaction is encountered.

can be associated with the use of certain commonly prescribed drugs.

Contributors The first (EM) and second (MY) authors reviewed the case notes and collated the data. The first author carried out the literature review and wrote the article. The third author (LL) helped to proof-read the article and the final author (GM) oversaw the work and suggested that it be published.

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