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Case Report

Levodopa induced hyponatremia in an elderly patient

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ABSTRACT

Hyponatremia is one of the most common electrolyte abnormalities encountered in clinical practice. Older persons have an increased risk of developing hyponatremia due to multimorbidity and frequent polypharmacy. We present a case of hyponatremia induced by levodopa in an 87-year-old lady not suffering from Parkinson's disease. This case highlights an important iatrogenic cause of hyponatremia and its sequelae.

Keywords: Levodopa, Case Report, Hyponatremia

INTRODUCTION

Hyponatremia is a common electrolyte imbalance and is defined as a serum sodium below 136 mmol/l.[1] It can vary in severity and most patients are asymptomatic. When symptomatic it may present with headache, vomiting, lethargy and altered mental status and can be associated with serious complications. Levodopa can rarely induce hyponatremia and there are only a few documented cases of such a presentation.

We present a case of an 87-year-old lady who developed hyponatremia secondary to the use of levodopa/carbidopa. Following a week from starting the medication the patient had a fall and was admitted to hospital with a left femoral fracture.

Admission was complicated with acute hyponatremia that was not responding to conventional treatment. A review of the diagnosis of Parkinson's disease and withdrawal of the medication resulted in resolution of the hyponatremia and recovery.

Our aim is to raise the awareness of such a rare complication and the dangers of iatrogenesis in the elderly.

CASE REPORT

The patient, an 87-year-old lady, presented to hospital after falling at a nursing home. She had been walking to the bathroom when she lost her balance and landed on her left hip. She did not sustain a head injury but was unable to bear weight on her left leg after the fall. On presentation, the patient was noted to have a shortened and externally rotated left leg and she was also noted to be confused and slightly agitated.

The patient had a past medical history of hypertension and anxiety. Her drug history included mirtazapine 15 mg nocte, mexazolam 1 mg daily, enalapril 10 mg twice daily, sennakot 15 mg nocte and lactulose 15 ml daily. It was also noted that the patient had been started on carbidopa/levodopa 110 mg twice daily and trihexyphenidyl 2 mg daily by her family doctor one week prior to admission for suspected idiopathic Parkinson's disease.

On review she had a normal fluid status and there was no evidence of dehydration or fluid overload. X ray confirmed a left subcapital femur fracture. Blood investigation results are shown in Table 1.

Table 1: Summary of blood investigations taken on admission to hospital.

Test	Patient test results	Normal range
Serum sodium (mmol/l)	124	136-145
Serum potassium (mmol/l)	4.57	3.5-5.1
Serum osmolality (mOsm/kg)	252	282-300
Urine osmolality (mOsm/kg)	555	50-1200
Thyroid stimulating hormone (mic/IU/ml)	1.936	0.3-3.0
Free thyroxine (pmol/l)	16.43	11.9-20.3
Serum cortisol (nmol/l)	772	140-690

A diagnosis of euvolemic hyponatremia secondary to the syndrome of inappropriate ADH (siADH) was made. The patient was admitted to a surgical ward and put on fluid restriction of 1 litre/day as part of the management of hyponatemia. Her treatment was also reviewed. Mirtazapine was tailed down slowly and enalapril was changed to amlodipine 5 mg daily. Despite this initial management, the patient remained hyponatremic with a sodium level of 122 mmol/l.

She was reviewed by the orthogeriatricians who examined the patient and noted that there were no signs of parkinsonism. Thus, the levodopa/carbidopa was tailed down over three days and then stopped. Within 24 hours, sodium levels increased from 119 to 124 mmol/l and normalised within one week of stopping the medication.

DISCUSSION

Hyponatremia is a serum of sodium of less than 136 mmol/l and is one of the commonest electrolyte disturbances encountered in clinical practice.[2] Hyponatremia is estimated to have a prevalence of 20% to 35% among hospitalized patients and is twice as high in elderly patients.[3] Reasons for this include multimorbidity and polypharmacy. It can be classified as mild, moderate or severe and in the most severe cases it can lead to delirium, coma, seizures and death.

It is hypothesised that levodopa-induced hyponatremia is cause by an inappropriate secretion of antidiuretic hormone (ADH). In animal studies, there is evidence that dopaminergic input to magnocellular ADH-secreting neurons facilitates ADH release. There is also evidence, however, that the system is functionally adaptive and this variability may explain the individual idiosyncratic susceptibility to the hyponatremic complications of levodopa.[4]

We could find three previous case reports of levodopa/carbidopa induced hyponatremia. The first case was that of a 68-year-old gentleman developing hyponatremia following treatment for Parkinson's disease (PD) with levodopa/carbidopa and amantadine.[5] Subsequently there was also a case report of 73-year-old woman with hyponatremia developing four days after starting levodopa following a diagnosis of PD. Recently, another case has been reported of a dose-dependent L-dopa/carbidopa induced hyponatremia presenting with hiccups in a patient who suffers from PD.

To our knowledge, this is the first case report where L-dopa/carbidopa induced hyponatremia occurring in a patient not suffering from PD.[6]

CONCLUSION

Acute severe hyponatremia is a rare adverse effect of treatment with levodopa/carbidopa. This case highlights the dangers of iatrogenesis, especially in elderly patients where complications can be more severe.

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