Hyponatraemic encephalopathy induced by single dose Indapamide

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1. Introduction
Hyponatraemia, defined as a serum [Na+] < 135mmol/l, is the most common electrolyte abnormalities, both in the community and in the hospital setting, (seen in 2% of hospital patients, and up to 30% elderly patients in a nursing homes). There are numerous factors which may directly cause hyponatraemia, including dehydration, concomitant cardiac/hepatic/renal disease, and certain medications, such as diuretics. It may cause a range of symptoms, depending on the speed and severity of the deficiency. With mild hyponatraemia, (>115mmol/l), patients may experience malaise – nausea, vomiting and generalised fatigue. Clinically significant hyponatraemia, [Na+]<115mmol/l, manifests itself through neurological symptoms: confusion, seizures, and ultimately coma. Chronic, asymptomatic hyponatraemia has been shown extensively to cause gait disturbances, falls and neurocognitive impairment.

Her past medical history also included asthma, ischaemic heart disease, and well-managed hypothyroidism. She had been a lifelong smoker, although she denied any new respiratory symptoms, or weight loss. She reported having commenced indapamide for hypertension one day prior to presentation.

2. The case of Ms. H
A 76 year-old lady was brought into A&E, having been found collapsed at home. On admission, GCS was 6/15. She remained quite confused, agitated and disorientated whilst in resus. She was euvoaemic, with an adequate cardiac output, and normal blood glucose. CT-head showed no acute intracranial abnormality to account for her presentation. Laboratory work up showed a profound hyponatraemia, [Na+]=108. Prior to this admission, her sodium levels were within normal ranges.

3. Laboratory work
On admission: K = 4.2mmol/l; corrected calcium = 2.13mmol/l. She was hyperphosphataemic (PO4 = 1.67mmol/l), hypercholesterolaemic (7.1mmol/l). Her urinary osmolality was 475OSm/kg; urinary [Na+] was 27; serum osmolality, 336. Her arterial blood pH 7.49; PaCO2 6.2kPa; HCO3- 26.5mmol/l; bicarbonate 17.7mmol/l; base excess -1.6mmol/l; 

4. Management
She was put onto fluid restriction, with a strict input/output chart. Indapamide was stopped. She had regular [Na+] checks, which showed gradual improvement in the hyponatraemia: 108mmol/l on admission, 111mmol/l the following day, 120mmol/l the next, 127mmol/l the next.

The management of hyponatraemia requires identification and appropriate management of the underlying cause. Commonly, patients with hyponatraemia secondary to SIADH are placed on a fluid restriction. Patients who present acutely in extremis with hyponatraemia (neurological symptoms) should receive hypertonic saline infusion. It is vital that [Na+] is not corrected too quickly, (no more than an increase of 8-12mmol/L over 24 hours) to prevent central pontine myelinolysis, (CPM), and subsequent permanent neurological dysfunction.

References: