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Case Report Open Access

Severe Hyponatraemia and Associated Seizure Following the Administration of Sodium Picosulfate/Magnesium Citrate (Picolax): A Case Report

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Abstract

Bowel preparation is known to cause minor electrolyte disturbances. We report the case of a patient with severe hyponatraemia resulting in a seizure, following the administration of picolax.

A 60 year old female patient with no significant past medical history and taking no regular medications presented with confusion following administration of picolax for an elective colonoscopy. On arrival her GCS was 14/15 but moments later she had a tonic-clonic seizure lasting two minutes. Following this, her GCS was 9/15. Laboratory tests revealed a serum sodium level of 119 mmol/L. Other investigations including CT head and lumbar puncture were normal. Following intravenous replacement of electrolytes, her electrolytes improved and GCS returned to 15/15.

Severe hyponatraemia and associated seizures following bowel preparation are rarely described in the literature. There are only nine reported cases of severe electrolyte disturbances following bowel preparation and seven that have caused seizures. We urge care to be taken when prescribing bowel preparation; particularly in those with pre-existing medical conditions and taking medications which can cause hyponatraeamia, and to council patients when prescribing bowel preparation on the possible side effects. We also urge clinicians to consider Polyethylene glycol based bowel preparations as opposed to sodium picosulfate for bowel preparation.

keywords: Seizure; hypokalaemia; Hypokalaemia; Sodium picosulfate; Picolax

Introduction

Bowel preparation is known to cause minor electrolyte disturbances. There are only nine reported cases of severe electrolyte disturbances following bowel preparation and seven that have caused seizures

Case Report

A sixty year old female patient with no significant past medical history and taking no regular medications presented with confusion, seizures and decreased consciousness following administration of sodium picosulphate/magnesium citrate (picolax).

The patient was to undergo an elective colonoscopy for investigation of abdominal pain, for which an ultrasound scan of the abdomen had not provided a diagnosis. She had taken picolax as bowel preparation with two litres of water as prescribed and had then developed profuse vomiting, followed by diarrhoea which lasted for two hours. She became confused and her husband concerned, brought her to Accident and Emergency (A&E).

On arrival to A&E her Glasgow Coma Scale (GCS) was registered as 14/15 (E4, V4, M6), she then had a tonic-clonic seizure which lasted for two minutes. Her GCS following this was calculated as 9/15 (E2, V1 M6) and pupils were equal and responsive to light. A blood glucose reading was 9.2 mmol/L (4.0-8.0). Physical examination was unremarkable, neurological examination was limited due to post-ictal state. An initial arterial blood gas revealed a metabolic acidosis (pH 7.09, pCO_2 4.6, pO_2 10.4 lactate 15.2, base excess-18.4, and bicarbonate of 10.6). Laboratory tests revealed a sodium level of 119 mmol/L. Other investigations to rule out causes of hyponatraemia including thyroid function tests, random cortisol level and serum and urine osmolality were sent in Accident and Emergency unit and are presented in Table 1. Initial bolus intravenous fluids were slowed down and an immediate CT head arranged which was unremarkable.

The following day, her GCS remained decreased at 10/15 (E4, V1, M5) and the patient developed a temperature of 38.3°C. She was commenced on aciclovir and ceftriaxone as a precaution and a lumbar puncture performed. The lumbar puncture revealed an opening pressure of 14 mmHg, WCC <1mm^3, glucose 4.7 (capillary glucose 5.6) and protein 0.23, it was negative for any organism. Empiric medications were thus discontinued. The patient was fluid resuscitated with isotonic fluids containing potassium chloride. Thirty-six hours after admission, serum sodium level was measured at 137 mmol/L.

Forty-eight hours after admission, GCS returned to 15/15. The patient had no recollection of events from after she took the bowel preparation. There were no neurological deficits noted. Ninety-six hours after admission, patient was discharged home once serum sodium level had stabilised. Repeat blood tests six weeks later revealed a normal sodium level of 138 mmol/L. She was advised not to drive for six months after having a provoked seizure.

Discussion

This case report describes a patient presenting with confusion and tonic-clonic seizure secondary to acute hyponatraemia following the administration of picolax. The acute drop in sodium level is well known to cause confusion, seizures, coma and even death secondary to cerebral oedema.

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	On Admission	Day 1	Day 2	Day 3	Day 4
Haemoglobin (g/L)	143	133	131	133	136
White Cell Count (×10^9/L)	15	14	9.3	8	6.7
Sodium (mmol/L)	119	117	137	133	132
Potassium (mmol/L)	3.1	3.7	3.4	3.4	3.9
Urea (mmol/L)	3.1	2	1.5	1.7	2.8
Creatinine (mmol/L)	48	45	57	49	46
Magnesium (mmol/L)	0.81				
Phosphate (mmol/L)	0.86				
TSH (mu/L)		0.44			
Adjusted Calcium (mmol/L)	2.34		2.36		2.36
Serum Osmolality (mmol/kg(L))	244				
Urine Osmolality (mOsm/kg)	508				
Random Cortisol (1400 hours) (nmol/L)	1000				

Table 1: Random cortisol level, serum and urine osmolality.

Severe hyponatraemia and associated seizures following bowel preparation are rarely described in the literature. A literature search was carried out on PubMed and MEDLINE using search terms, "picolax and hyponatraemia"; "picolax and seizure"; "sodium picosulfate and hyponatraemia"; "sodium picosulfate and seizure". This yielded seven publications, which described a total of nine cases and one population-based retrospective cohort study. One report was unable to be accessed [1]. A report [2] from New Zealand cites three cases of hyponatraemia and associated seizures after the administration of either sodium phosphate or picolax. The three female patients all had pre-existing medical co-morbidities including hypertension and depression, for which they were taking medications which could have contributed to hyponatraemia. A second report [3] from the Unites States of America again reports a female patient with pre-existing medical conditions, for which treatment could have contributed to hyponatraemia. A third report [4] from Ireland reports a female patient with no pre-existing medical conditions. The fourth report [5] from Korea describes a female patient with pre-existing medical conditions who was taking medications that can cause hyponatraemia. The fifth report [6] from Canada is a retrospective cohort study of 147,832 patients that looked at hospitalisation of patients with hyponatraemia following 30 days of administration of bowel preparation. Interestingly, the article compared sodium picosulfate and polyethylene glycol preparations and showed that sodium picosulfate was more likely to cause hyponatraemia. The sixth report from Ireland [7] looked at three case reports of symptomatic hyponatraemia in patients who had received sodium picosulfate. Only one of the three patients had a seizure. All three patients had pre-existing medical conditions and were taking medications that could have caused hyponatraemia.

Eight of the nine cases in the literature describe patients who have pre-existing medical conditions and are taking regular medication which could have contributed to hyponatraemia. Interestingly, all of the patients reported are female, although the authors do not know of a reported genetic link between gender and hyponatraemia causing seizures.

Our patient had no history of pre-existing medical conditions and was not taking any medications known to cause hyponatraemia. Hyperosmotic bowel preparation in the colon will draw isotonic fluid out of the bloodstream, leading to hypovolaemia and an increase in Ant-Diuretic Hormone (ADH). ADH may have further been increased by the nausea and vomiting described. The evidence for excess ADH is the urine osmolality reported as 508 mOsm/kg, which should be low (<100) in the case of hyponatraemia and hyposmolality. Unfortunately no urinary sodium was requested.

Furthermore, in the absence of other causes of hyponataremia such as Addison Disease, heart failure, liver failure or hypothyroidism, we believe that picolax administration was the cause of hyponatraemia for our patient as listed in the accompanying manufacturers leaflet which state "an uncommon side effect (less than 1 in 100 but more than 1 in 1000) is low levels of sodium or potassium in the blood with or without associated fits. Fits have occasionally been reported in epileptic patients". A 'Yellowcard' adverse reaction card was submitted to the MHRA.

Although the report describes a rare complication of a common mild effect of bowel preparation-we urge care to be taken when prescribing these medications; particularly in those with pre-existing medical conditions, and to council patients when prescribing bowel preparation on the possible side effects encountered. Polyethylene glycol based bowel preparations appear to be safer and cause less hyponatraemia than sodium picosulfate and this should be considered when prescribing bowel preparation. We also recommend not to drink the bowel preparation with free water alone as this can cause electrolyte imbalances and lead to hyponatraemia and seizures.

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