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CASE REPORT

Hyponatremic encephalopathy due to polyethylene glycol-based bowel preparation for colonoscopy: A case report

Yuan Zhao, Hai-Sheng Dong

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Yuan Zhao, Hai-Sheng Dong, Department of Critical Care Medicine, Shanghai TCM-integrated Hospital, Shanghai 200082, China

Corresponding author: Hai-Sheng Dong, MD, Doctor, Department of Critical Care Medicine, Shanghai TCM-integrated Hospital, No. 230 Baoding Road, Hongkou District, Shanghai 200082, China. hsdong06@163.com

Abstract

BACKGROUND

Adequate bowel preparation is critical for colonoscopy screening. At present, the most widely used intestinal cleaner recommended at home and abroad is Polyethylene glycol (PEG). Intestinal cleansers can cause electrolyte disturbances and hyponatremia. However, hyponatremic encephalopathy due to hyponatremia induced by PEG solution, although rare, can lead to serious irreversible sequelae and even death.

CASE SUMMARY

In this report, we discuss a case of neurological dysfunction due to hyponatremia, also known as hyponatremic encephalopathy, observed in a 63-year-old woman who underwent PEG-based bowel preparation for colonoscopy. She was eventually transferred to our intensive care unit for treatment due to her Glasgow Coma Scale score of 9/15 (Eye opening 2; Verbal response 1; Motor response 6) and abnormal laboratory tests.

CONCLUSION

Physicians should be thoroughly familiarized with the patient's history before prescribing PEG for bowel preparation, and timely identification of patients with hyponatremic encephalopathy is essential as delayed treatment is associated with poor neurological outcomes. An intravenous infusion of 3% sodium chloride is recommended at the onset of early symptoms. The goal of treatment is to adequately treat cerebral edema while avoiding serum sodium correction beyond 15 to 20 mEq/L within 48 h of treatment to prevent osmotic demyelination syndrome.

Key Words: Polyethylene glycol; Intestinal preparation; Hyponatremic encephalopathy; Hyponatremia; Brain; Case report

Core Tip: Hyponatremic encephalopathy caused by the use of polyethylene glycol (PEG) solution for intestinal cleaning is rare, which can lead to irreversible sequelae and even death. This case is a female with neurological dysfunction due to hyponatremia induced by the use of pegylated for bowel preparation before colonoscopy. The patient was in a coma with the brain suggesting cerebral edema, and was transferred to the intensive care unit for treatment with 3% sodium chloride injection. The patient's medical history should be fully understood before using PEG for bowel preparation, as both untimely and overtreatment can lead to serious complications.

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INTRODUCTION

Colonoscopy is a gold standard for diagnostic screening of colon disorders[1]. High-quality bowel cleansing is a prerequisite for colonoscopy. Polyethylene glycol (PEG)-based solution is the most widely used intestinal cleansing agent, although several cases have been reported to cause hyponatremia[2-4], it is a relatively safe option for patients at risk of electrolyte imbalance and dehydration[5]. As the most common electrolyte abnormality, hyponatremia is closely related to the brain. Indeed, several neurological disorders are frequently associated with hyponatremia, and hyponatremia itself can involve central nervous system dysfunction[6]. Thus, hyponatremic encephalopathy induced by PEG solutions is of concern to us, although rare and potentially serious. Hyponatremic encephalopathy was first proposed by Arieff *et al*[7] and is defined as neurological symptoms associated with hypotonic cerebral edema[8], is most commonly seen in the intensive care unit (ICU), with a prevalence of only 1% of postoperative patients[9,10]. Its clinical manifestations include headache, nausea and vomiting, seizures, and decreased consciousness, *etc*, but the severity depends on the degree and rate of serum sodium reduction. At present, relevant guidelines indicate that hypertonic saline is considered to be a safe and effective treatment for acute or symptomatic hyponatremic encephalopathy[11,12]. However, hyponatremic encephalopathy is the result of multiple factors, and the current studies on its clinical characteristics and treatment are limited[7,13]. Here, we describe a case of a 63-year-old female who presented with acute hyponatremic encephalopathy and seizures after bowel cleansing with PEG for colonoscopy.

CASE PRESENTATION

Chief complaints

A 63-year-old female patient was brought to the local hospital in September 2021 due to a loss of appetite for the past month.

History of present illness

Therefore, a tumor was first suspected and a colonoscopy was performed in combination with the patient's medical history. This patient took PEG with 4.5 L water to clean the bowel before the colonoscopy. This patient was found unconscious and developed seizures the following afternoon, and a series of differential diagnoses were made to consider whether the patient had suffered from intracranial vasculopathy, cardiogenic causes, shock, or hypoglycemia. She was eventually transferred to our ICU for management.

History of past illness

The patient presented with gastric cancer 13 years ago and underwent radical gastrectomy. The patient subsequently developed liver metastases and received multiple chemotherapy regimens. In addition, the patient had a 10-year history of ulcerative colitis.

Personal and family history

The patient denied a family history of malignant tumors or other diseases.

Physical examination

This patient was admitted to the ICU with a Glasgow Coma Scale (GCS) score of 9/15 [Eye opening (E) 2; Verbal response (V) 1; Motor response (M) 6] and no signs of meningeal irritation. The pupil

examination revealed equal-sized and reactive to light.

Laboratory examinations

Laboratory results revealed serum sodium (114 mmol/L, reference: 136 - 144 mmol/L), potassium (3.4 mmol/L, reference: 3.6 - 5.2 mmol/L), chloride (82 mmol/L, reference: 101 - 111 mmol/L), bicarbonate (21 mmol/L, reference: 22 - 26 mmol/L), blood urea nitrogen (32.4 mg/dL, reference: 9 - 20 mg/dL), creatinine (0.44 mg/dL, reference: 0.6 - 1.2 mg/dL), glucose (187.2 mg/dL, reference: 70- 120 mg/dL). That means a total loss of 19.65 g Na deficit. The level of hematocrit decreased from 32.8% to 29.6%.

Imaging examinations

A computed tomography (CT) scan was performed and revealed cerebral edema (Figure 1).

FINAL DIAGNOSIS

After a series of differential diagnoses, combined with the patient's medical history, relevant examination, and treatment, the patient was finally diagnosed with hyponatremic encephalopathy.

TREATMENT

This patient did not have any history of sedatives. She was initially treated with 3% sodium chloride (NaCl) injection. The serum sodium gradually increased to 124 mmol/L after 6 h. The infusions ceased once this patient was free from further seizures.

OUTCOME AND FOLLOW-UP

The following afternoon, the patient's serum sodium returned to 135 mmol/L. Her neurological condition also improved dramatically during this period, with a GCS score of E4V5M6. A CT scan of the brain edema subsequently normalized (Figure 1). The patient was discharged home after 2 d of hospitalization. After a 1-mo of follow-up, the patient's neurological function recovered completely.

DISCUSSION

Acute hyponatremia is often accompanied by major neurologic manifestations[13,14]. An increasing number of studies have reported acute hyponatremia caused by the use of PEG-based solutions during colonoscopy preparation[15,16]. The main symptoms of hyponatremia are nausea, vomiting, headache, seizures, and coma. However, cerebral edema is rare[3,15]. We demonstrate again that bowel preparation with PEG for colonoscopy with PEG may lead to brain cerebral edema associated with hyponatremia. The patient's hyponatremia may be caused by several reasons. First of all, the patient had a history of gastric cancer and had undergone radical surgery at an early stage, which more or less affected gastrointestinal sodium absorption. Moreover, this patient had several high-risk factors, such as older age (> 60 years), female sex, and poor dietary intake. Hyponatremia is the most common electrolyte disorder in clinical practice, which is usually caused by excessive secretion of antidiuretic hormone (ADH) or infusion water retention. The syndrome of inappropriate ADH (SIADH) secretion can be caused by a variety of factors, such as ectopic secretion of ADH by tumor cells themselves, and the deterioration of the overall functional status of the patient can also induce ADH release independent of osmotic stimuli[17]. Therefore, hyponatremia is most likely the result of ectopic production of arginine vasopressin by the tumors in elderly patients in response to physiologic, non-osmotic ADH.

The European guideline recommends the use of hypertonic saline, usually 3% NaCl, for acute or symptomatic hyponatremia. Hypertonic saline is an effective and potentially life-saving treatment for brain edema induced by hyponatremia, because its high sodium concentration allows water to be diverted from the intracellular space[18].

Twelve cases of hyponatremia induced by PEG-based solutions have been reported (Table 1). Most patients were older than 60 years and most were female. Among them, one patient (case 3) had end-stage renal disease, and five patients (cases 2, 8, 9, 10, and 11) had their urination impaired due to thiazide diuretics. Cases 4 and case 6 had insufficient thyroid replacement and were older than 65 years, suggesting further exacerbation of their hyponatremia. In the presence of normal renal, thyroid, and adrenal function, patients 1 and 5 received an additional 4 L of rehydration and 3 L of PEG. Non-osmotic AHD stimulation combined with massive infusion is the main cause of hyponatremia in such patients. In addition, intestinal manipulation during bowel preparation for colonoscopy may lead to

Table 1 Clinical findings of patients with polyethylene glycol-related hyponatremia

Patient number	1	2	3	4	5	6	7	8	9	10	11	12
Reference	[3]	[3]	[3]	[3]	[3]	[3]	[3]	[15]	[15]	[15]	[18]	[16]
Age	59	62	51	73	70	65	69	53	79	68	64	71
Sex	F	F	M	F	F	F	F	F	F	F	F	M
History	Hysterectomy oophorectomy	Hypertension, Dyslipidemia	Diabetes, End-stage renal disease	Depression, Hypothyroidism	Stenosis of internal carotid artery, Hypertension, Osteoporosis	Breast cancer, Total thyroidectomy and radioiodine therapy	Dyslipidemia, Diabetes	Asthma, Osteoporosis, Hypertension	NA	Breast cancer, Nephrectomy, Esophageal dysmotility, Hypertension	Hypertension, Hyperlipidemia	Diabetic, Colon cancer (suspicion)
Prescription drugs	Estradiol, Aspirin	Thiazide	Amlodipine, Furosemide, Atenolol	Citalopram, Levothyroxine	Amlodipine, Clopidogrel, Ibandronic acid	Levothyroxine	Atorvastatin, Sitagliptin, Glimepiride, Metformin	Irbesartan, Chlorothalidone	Thiazides	Irbesartan, Hydrochlorothiazide, Tamoxifen, Ezetimibe	Thiazide	NA
Preparation methods	3L PEG and 4L weak tea	4L PEG	NA	255g PEG and 64 ounces gatorade	4L PEG and 3L clear water	4L PEG	4L PEG	NA	10mg Bisacodyl and PEG mixed with 120mL gatorade	2L Pico Prep and 1L PEG	4L PEG	7-8L PEG
Clinical presentation	Confusion	Seizure	Idioventricular rhythm, Cardiac arrest	Seizure	Seizure	Seizure	Seizure	Seizure	Nausea, Vomiting	Nausea, Vomiting, Malaise, Seizure	Seizure	Drowsy, Seizure
Blood pressure in mmHg	110/70	130/90	167/78	Normal	190/100	156/85	143/74	150/86	NA	150/70	NA	NA
Pulse in bpm	60	90	103	Normal	84	86	76	79	NA	84	NA	NA
Sodium in mmol/L												
Baseline	NA	138	138	NA	140	144	NA	NA	NA	NA	NA	NA
Lowest	120	116	122	117	110	127	113	115	118	106	114	111
Post-treatment	138	130	NA	131	138	141	135	NA	133	Normal	Normal	132
Potassium in mmol/L	4.6	3.9	5.1	3.3	3.4	4.3	3.4	2.5	NA	3.1	NA	NA
Chloride in mmol/L	NA	79	94	79	72	104	72	NA	NA	NA	NA	NA
Bicarbonate in mmol/L	17.2	26	20	21	17.3	17.3	17.3	NA	NA	NA	NA	NA
Urea in mg/dL	NA	2.5	24.3	6	11.8	14.6	11.8	NA	NA	NA	NA	NA
Creatine in mg/dL	0.9	0.6	7.7	0.6	0.67	0.71	0.67	NA	NA	Normal	NA	NA
Glucose in mg/dL	93	NA	95.5	NA	148	235	148	NA	NA	NA	NA	Normal
Brain	Normal	Cerebral	Not done	Not done	Normal	Normal	Small	Cerebral	Normal	Normal	Normal	Normal

CT/MRI findings		edema					vessel disease and tiny restriction focus in temporal lobe	edema				
Outcome	Complete recovery	Complete recovery	Death	Complete recovery	Complete recovery	Complete recovery	Complete recovery	Complete recovery	Complete recovery	Complete recovery	Complete recovery	Complete recovery

CT: Computed tomography; F: Female; M: Male; MRI: Magnetic resonance imaging; N/A: Not applicable; PEG: Polyethylene glycol.

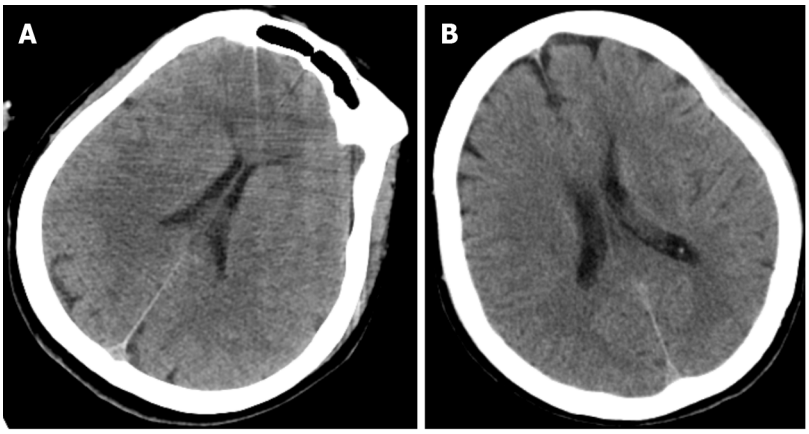

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Figure 1 Computed tomography scan of the head. A: Computed tomography (CT) scan of the head revealed cerebral edema; B: CT scan of the head showed that cerebral edema became normal after treatment.

increased ADH. Patient (case 12) received a double dose of PEG, which caused a severe internal environmental disturbance. All the patients recovered fully except for the patient who had dialysis-dependent chronic kidney disease and who died from cardiac arrest. This case report has several limitations. First, the single case itself has limitations, and high-quality evidence is needed to confirm. Second, some techniques, like non-invasive brain stimulation techniques, should be more widely used in clinical practice in the future; Third, not all aspects of hyponatremia encephalopathy were discussed.

CONCLUSION

Given this case report, physicians should be thoroughly familiarized with the patient's history before prescribing PEG for bowel preparation, and timely identification of patients with hyponatremic encephalopathy is essential, as delayed treatment is associated with poor neurological outcomes. An intravenous infusion of 3% sodium chloride is recommended at the onset of early symptoms while avoiding serum sodium correction beyond 15 to 20 mEq/L within 48 h of treatment to prevent osmotic demyelination syndrome.

FOOTNOTES

Author contributions: Zhao Y and Dong HS designed the study; Zhao Y performed the study, collected and analyzed the data and wrote the manuscript; All authors have read and approved the final manuscript.

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