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Case Report

Two Cases of Hyponatremia Following Bowel Preparation for Colonoscopy

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Summary

Polyethylene glycol is a safe agent for bowel cleansing in preparation for colonoscopy. However, it can cause considerable electrolyte abnormalities. We herein report two cases of hyponatremia because of bowel preparation using polyethylene glycol. **Case 1**: The patient, a man in his 70s, was admitted to our hospital for an endoscopic mucosal resection of an adenoma of the large intestine. After the preparation, the patient developed consciousness disturbance. His serum Na level was 122 mEq/L, indicating a marked degree of electrolyte abnormality. Syndrome of inappropriate antidiuretic hormone secretion (SIADH) was inferred as the cause of hyponatremia. **Case 2**: A patient in his 70s, based on a positive fecal occult blood test result, was scheduled to undergo colonoscopy. A blood test on the day of the colonoscopy showed his serum sodium level as 107 mEq/L. The patient was found to have chronic hyponatremia, which prevented development of clinical symptoms. However, because of preservation of ADH secretion despite the hyponatremia, a diagnosis of SIADH was made. These cases suggest that clinicians should be aware of the risk of hyponatremia during bowel preparation for colonoscopy.

Key Words: Hyponatremia, Colonoscopy, Bowel preparation, Bowel prep hyponatremia

Introduction

In preparation for colonoscopy, various oral agents such as polyethylene glycol, magnesium citrate, and sodium phosphate are used for bowel cleansing. Among them, polyethylene glycol is a safe agent: it is useful in patients with heart or renal failure. However, it is sometimes known for causing electrolyte abnormalities [1]. This report describes two cases of hyponatremia caused by bowel preparation using polyethylene glycol.

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Case Presentation

Case 1

The patient, a man in his 70s, had earlier undergone four colonoscopies with no difficulty encountered during preparation. He had been treated for hypertension and had been prescribed thiazide diuretics. In month Y 20XX, he was admitted to our hospital for endoscopic mucosal resection of an adenoma of the large intestine. His serum sodium (Na) level on admission was 140 mEq/L. A sodium picosulfate solution (0.75%, 10 mL) was administered orally as a bowel preparation at 9 p.m. on day 1. Polyethylene glycol (2 L) was administered orally on day 2 (6 a.m. to 8 a.m.). However, because the first administration was insufficient, addi-

Table 1 Laboratory data of Case 1

Blood Cell Count		Hormons	
WBC	8.30 $\times 10^3$/μl	TSH	0.41 μIU/ml
Neutro	60.1 %	FT3	1.83 pg/ml
Lymph	30.8 %	FT4	1.91 ng/dl
Mono	3.8 %	Cortisol	0.41 pg/ml
Eos	4.1 %	ACTH	12.6 pg/ml
Baso	1.2 %	BNP	59.5 ng/dl
RBC	3.55 $\times 10^6$/μl	ADH	3.5 pg/ml
Hb	10.6 g/dl		
Ht	30.1 %	Urinalysis	
PLT	14.3 $\times 10^4$/μl	UN	291 mg/dl
		Creatinine	36 mg/dl
Blood Biochemistry		Na	82 mEq/l
AST	19 IU/l	K	31 mEq/l
ALT	14 IU/l	Cl	40 mEq/l
LDH	190 IU/l	Uosm	390 mOsm/kg
ALP	98 IU/l		
BUN	12 mg/dl		
Creatinine	0.40 mg/dl		
Na	122 mEq/l		
K	2.8 mEq/l		
Cl	86 mEq/l		
Glucose	159 IU/l		
Posm	262 mOsm/kg		

tional polyethylene glycol (2 L) was administered orally. After preparation, the patient developed consciousness disturbances while awaiting his colonoscopy.

Examination results showed his blood pressure as 115/64 mmHg, heart rate as 83/min, body temperature as 36.5℃, and SpO_2 as 97% (room air). Neurological findings were the following: Japan Coma Scale (JCS) I-2, Glasgow Coma Scale (GCS) E4V4M6, difficulty in object naming, median eye position, rapid eye movement (nystagmus), normal eye closure, midline tongue protrusion, able to raise both upper limbs, and resting tremor (5 Hz) in the upper right limb. Table 1 presents results of blood and urine tests conducted when the consciousness disturbance occurred. His serum Na level was 122 mEq/L, potassium (K) was 2.8 mEq/L, and chloride (Cl) was 86 mEq/L, indicating considerable degrees of electrolyte abnormalities. Despite the presence of hyponatremia and low osmolality, his antidiuretic hormone (ADH) level was 3.5 pg/mL, indicating that it was being secreted without suppression. Urinalysis showed urine osmolality of 390 mOsm/kg and urine sodium of 82 mEq/L, indicating no urinary dilution. Arterial blood gas analysis showed that metabolic acidosis was compensated by respiratory gasex change. Brain magnetic resonance imaging (diffusion-weighted images) showed no sign of acute cerebral infarction, although FLAIR images showed high signal intensity in the white matter, indicating chronic ischemic changes (Fig. 1). However, this was unlikely to reflect the cause of his consciousness disturbance. Based on these findings, because no apparent cause was identified for his consciousness disturbance other than hyponatremia, syndrome of inappropriate antidiuretic hormone secretion (SIADH) was inferred as the cause for the hyponatremia occurrence. It has been speculated that the suffering of colonoscopy pretreatment stimulates non-osmotic vasopressin secretion. It is because he got hyponatremia. After a diagnosis of hyponatremic encephalopathy was made, 0.9% NaCl was started to correct his hyponatremia 3 hr after onset of consciousness disturbance. His consciousness disturbance improved. Moreover, his tremor disappeared after correction of his Na levels 8 hr after onset. The following day, we re-examined brain magnetic resonance images to check there is no findings which sus-

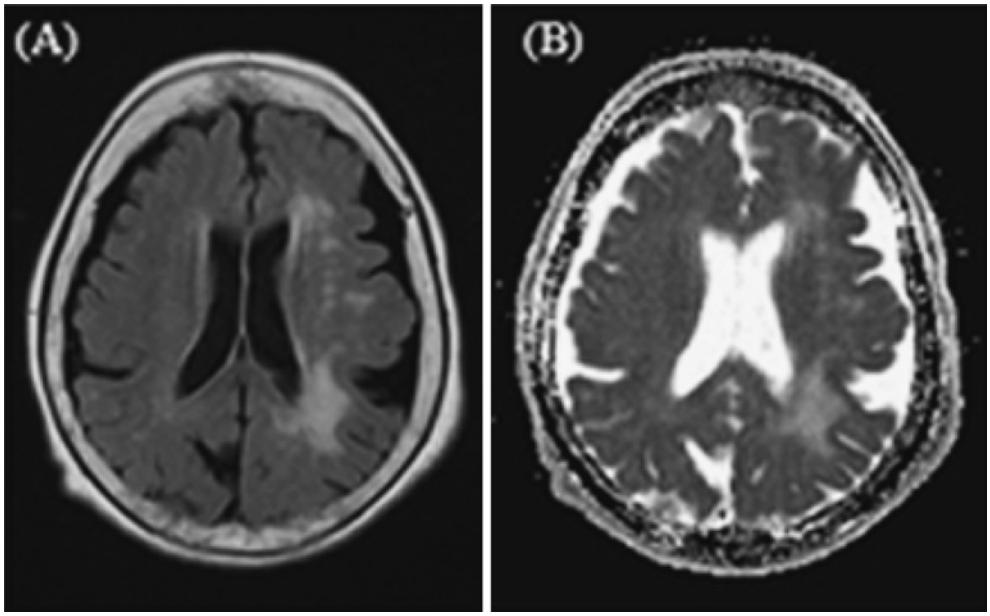

Figure 1 Brain magnetic resonance images.
The MRI shows chronic ischemic changes and no new stroke changes. There was no evidence that caused disturbance of consciousness.
A: diffusion-weighted images
B: FLAIR images

pect osmotic demyelination syndrome. Thereafter, no major problem occurred.

Case 2

The patient, a man in his 70s, was scheduled to undergo colonoscopy in month Z 20XX because of a positive finding from a fecal occult blood test. He had an implanted cardiac pacemaker because of a complete atrioventricular block. He had been followed up regularly at our hospital. For his hypertension, he had been taking bisoprolol fumarate 2.5 mg (one tablet) and had been under a strict salt restriction. A blood test taken during month Z-1 revealed that his serum sodium level was 126 mEq/L. Sennoside 24 mg (two 12 mg tablets), which is an irritative purgative, was administered orally as an initial bowel preparation at 9 p.m. on the day before the colonoscopy. Polyethylene glycol (2 L) was administered orally from 6 a.m. to 8 a.m. on the day of the colonoscopy. A blood test administered on the day of the colonoscopy revealed his serum sodium level as 107 mEq/L. He was admitted to our department for additional examination.

Examination on admission revealed his blood pressure as 128/60 mmHg, heart rate as 76/min, body temperature as 36.3℃, and SpO_2 as 97% (room air). No neurological abnormality was found (JCS I-1, GCS E4V5M6). Table 2 presents results of blood and urine tests on admission. Despite hyponatremia and low osmolality, his ADH level was 3.4 pg/mL (within the normal range), indicating that ADH was being secreted without suppression. Urinalysis showed urine osmolality of 486 mOsm/kg, indicating a lack of urinary dilution. Results indicated that the patient had chronic hyponatremia, which prevented the development of clinical symptoms. In addition, he had underlying arrhythmia. Additional blood tests revealed a mildly elevated concentration of brain natriuretic peptide, but echocardiography showed normal cardiac function, thereby excluding the possibility of heart failure. Given the preservation of ADH secretion despite the hyponatremia, a diagnosis of SIADH was made. Results suggest that the patient had chronic hyponatremia because of strict salt restriction and treatment with an angiotensin-receptor antagonist. However, ADH secretion was induced by the bowel cleansing for colonoscopy leading to a loss in fluid. After admission, 0.9% NaCl was started to correct the hyponatremia. His sodium level returned to normal on day 2. No major diffi-

Table 2 Laboratory data of Case 2

Blood Cell Count		Hormons	
WBC	6.80 $\times 10^3$/μl	TSH	1.220 μIU/ml
Neutro	79.7 %	FT3	3.62 pg/ml
Lymph	15.9 %	FT4	1.70 ng/dl
Mono	4.3 %	Cortisol	26.99 pg/ml
Eos	0.0 %	ACTH	17.9 pg/ml
Baso	0.1 %	BNP	64.7 ng/dl
RBC	4.72 $\times 10^6$/μl	ADH	3.4 pg/ml
Hb	14.0 g/dl		
Ht	39.1 %	Urinalysis	
PLT	25.7 $\times 10^4$/μl	UN	766.2 mg/dl
		Creatinine	87.04 mg/dl
Blood Biochemistry		Na	39 mEq/l
AST	61 IU/l	K	40 mEq/l
ALT	31 IU/l	Cl	24 mEq/l
LDH	296 IU/l	Uosm	486 mOsm/kg
ALP	317 IU/l		
BUN	19 mg/dl		
Creatinine	0.66 mg/dl		
Na	107 mEq/l		
K	4.3 mEq/l		
Cl	84 mEq/l		
Glucose	111 IU/l		
Posm	235 mOsm/kg		

culty occurred with this case thereafter.

Discussion

Oral bowel cleansing agents are used for bowel preparation in almost all patients undergoing colonoscopy. Although they are generally safe, rare reports have described cases of hyponatremia following bowel preparation. Hyponatremia caused by oral bowel cleansing agents was first reported by Schröppel et al.[2] Analysis of subsequently accumulated cases revealed the risk factors to be female gender, age of 65 years or older, chronic kidney disease, heart failure, history of electrolyte abnormality, use of thiazide diuretics, angiotensin-converting enzyme inhibitors, or antidepressants, and history of gastric resection. The incidence of hyponatremia caused by oral bowel cleansing agents has been reported as 1-6.3%[3-6].

The physiological mechanism of hyponatremia is thought to involve fluid loss from bowel cleansing preparations and as a result increase of ADH secretion, leading to the development of SIADH[7]. In addition, decreased intake of protein and sodium during fasting for bowel preparation might contribute to hyponatremia development[7]. Two patients reported herein were older adults, but it is noteworthy that the patient in Case 1 had no underlying diseases that might cause hyponatremia. However, in Case 2 the patient had chronic hyponatremia but had no readily apparent symptoms. Both patients developed hyponatremia after bowel cleansing preparation. Therefore, it was inferred that their hyponatremia resulted from SIADH because of fluid loss from bowel cleansing preparations. Furthermore, both patients had been fasting since 8 p.m. the day before colonoscopy, indicating decreased intake of protein and sodium. These findings suggest that intake of bowel cleansing solution within a short duration and the subsequent acute loss of body fluid in patients in those circumstances are the primary causes of "bowel preparation hyponatremia."

Acute hyponatremia is frequently associated with major neurologic complications including coma, permanent brain damage, and death[8,9]. Hyponatremic coma should be treated with slow intravenous infusion of hypertonic saline aimed at increasing the sodium level at a rate of 0.5 mmol/l/h, while not exceeding 12 mmol/l during the first 24 hr or 18 mmol/l during the second

24 hr to avoid osmotic demyelination, brain edema, and central pontine myelinolysis. Regarding patients of the present cases, their serum sodium levels were corrected slowly, with no readily apparent sequelae.

Colonoscopy is widely used as a secondary examination following a positive result obtained from a fecal occult blood test. It is indicated in a wide population, including young and old people. When performing bowel preparation, it is important to prevent hyponatremia by understanding that many factors can cause a sodium imbalance. To date, no detailed report has described re-administration and recurrence prevention of hyponatremia in patients who earlier developed hyponatremia following bowel preparation. However, SIADH caused by acute fluid loss can lead to hyponatremia. When performing colonoscopy in patients, especially in elderly patients, who are at an increased risk of hyponatremia, or in patients who are known to have chronic hyponatremia, it is important to instruct them to have sufficient salt and protein intake on the day before the colonoscopy. It is also important to instruct them to take bowel cleansing agents slowly.

Conclusion

Clinicians should be aware of the risk of hyponatremia during bowel preparation for colonoscopy. They should measure serum electrolytes appropriately for patients who develop a disturbance of consciousness or those with a history of chronic hyponatremia. A need also exists for assessment of the risks beforehand, and for exploration of individualized treatment protocols.

Conflict of interest

Not applicable.

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