Nutrition in Clinical Practice
Volume 29 Number 3
June 2014 397–401

DOI: 10.1177/0884533614528543
ncp.sagepub.com
hosted at
online.sagepub.com

Total Body Sodium Depletion and Poor Weight Gain in Children and Young Adults With an Ileostomy: A Case Series

Megan O'Neil, MS, RD[1]; Daniel H. Teitelbaum, MD[2]; and Mary Beth Harris, MPH, CSP, RD[1]

Abstract

Patients with high-output small bowel ostomies are at risk for total body sodium depletion (TBSD), defined as a urine sodium level <10 mmol/L. Failure to thrive (FTT) as a consequence of TBSD has been reported in neonates with ileostomies; however, this has not been well described in older children. The records of all children beyond the age of infancy with a small bowel ostomy cared for in our Children's Intestinal Rehabilitation Program from 2010–2012 were reviewed. Four patients between the ages of 18 months and 19 years were identified as having TBSD. All 4 patients experienced unintentional weight loss, despite adequate energy intake based on calculated needs, which was associated with a urine sodium level ≤10 mmol/L. With the supplementation of sodium, either enteral or intravenous, all patients demonstrated improved weight gain and correction of TBSD. The following cases suggest that the relationship between TBSD and FTT may extend well beyond the neonatal period and possibly into adulthood. We advise that patients of all ages with high stoma output have routine urine sodium levels checked, particularly in the setting of weight loss or poor gain. Furthermore, instances of TBSD should be treated with sodium supplementation. Further research is needed to better understand the relationship between TBSD and FTT and to establish intervention guidelines. (*Nutr Clin Pract*. 2014;29:397-401)

Keywords

pediatrics; ostomy; acid-base equilibrium; water-electrolyte balance; hyponatremia; nutritional support; parenteral nutrition; enteral nutrition

Background

Patients with small bowel ostomies (ie, jejunostomies or ileostomies) are highly susceptible to fluid and electrolyte abnormalities.[1,2] Among the electrolytes, ileostomy losses of sodium are the greatest at levels of 100–120 mEq/L/d.[1,3] A sodium deficit of 7% and a significant decrease in urinary sodium excretion have been seen in otherwise healthy adult patients with ileostomies.[4] Patients with high-output stomas or long-established ileostomies are at a greater risk for sodium losses, potentially leading to total body sodium depletion (TBSD). While a precise measure of total body sodium (TBS) levels is challenging, many have used the definition of TBSD as a urine sodium level <10 mmol/L, thus reflecting strong retention of the body's sodium reserves. Sodium losses are of greater concern in infants and young children due to proportionately greater obligatory losses and immature renal conservation of fluid and electrolytes as well as special requirements for growth.[5,6]

Failure to thrive (FTT) associated with TBSD has been reported in neonates with ileostomies.[5,7] In fact, TBSD results in an inability to become anabolic despite energy delivery exceeding energy needs. Thus, it is a common practice to check urine sodium levels in neonates, with expected sodium losses demonstrating poor weight gain,[5,7] and to supplement sodium in those with documented depletion. However, FTT associated with TBSD has not been well described outside of the neonatal period. The following 4 cases illustrate our experience of weight loss in the presence of TBSD in children and young adults with an ileostomy.

Methods

A retrospective screening of all children with a small bowel ostomy cared for in our Children's Intestinal Rehabilitation Program from 2010–2012 was completed. All children beyond

From [1]Clinical Nutrition, C.S. Mott Children's Hospital, University of Michigan, Ann Arbor, Michigan; and [2]Section of Pediatric Surgery, Department of Surgery, C.S. Mott Children's Hospital, University of Michigan, Ann Arbor, Michigan.

Financial disclosure: None declared.

This article originally appeared online on April 3, 2014.

Corresponding Author:
Mary Beth Harris, MPH, CSP, RD, Clinical Nutrition, C.S. Mott Children's Hospital, University of Michigan, 1500 East Medical Center Drive, MPB D5225, SPC 5718, Ann Arbor, MI 48375, USA.
Email: harrismz@med.umich.edu.

the age of infancy with >1 episode of TBSD were selected for this study. Patients with renal insufficiency were excluded from the study, as their urine sodium levels might not be a good reflection of TBS levels. Of the 11 patients with a small bowel ostomy, 4 were identified as having TBSD, defined as a urine sodium level <10 mmol/L, and comprised the patients reported in this series. The 7 patients excluded did not have regular urine sodium levels checked; therefore, TBS status was unknown. The study was approved by our hospital's institutional review board (HUM00066381).

Case Presentations

Case 1

The first case was a 19-year-old female patient with intestinal failure secondary to ulcerative colitis after total colectomy with J-pouch and ileoanal anastomosis, multiple small bowel resections, and creation of an end ileostomy due to several small bowel obstructions. The patient presented to our clinic with a 2.2-kg weight loss and a body mass index (BMI) of 17 kg/m^2, despite receiving 130% of her estimated energy needs from a combination of parenteral nutrition (PN) and reported oral intake based on a 24-hour recall, following a 3-month period of persistent weight gain. The amount of sodium in her PN regimen was 161 mEq (3.4 mEq/kg/d), the reported ileostomy output was approximately 2200 mL/d (46 mL/kg/d), and serum and urine sodium levels were 138 and <10 mmol/L, respectively. After clinical evaluation, caloric provision from PN was determined to be appropriate, but the patient was instructed to add 1.5 teaspoons (154 mEq of sodium) of table salt to foods daily, bringing the total supplemental sodium intake to 315 mEq (6.6 mEq/kg/d). One month later, the patient returned to the clinic and demonstrated a weight gain of 4.8 kg, BMI of 18.8 kg/m^2, urine sodium level of 129 mmol/L, and a stable serum sodium level and oral intake. The reported ileostomy output was approximately 2600 mL (50 mL/kg/d). With the long-term goal of weaning the patient off PN in mind, PN was decreased from 6 to 5 d/wk to decrease energy input by approximately 15%, subsequently decreasing her supplemental sodium intake to a daily average of 269 mEq (5.1 mEq/kg/d). Approximately 1 month later, the patient demonstrated a further weight gain of 1.2 kg and an improved BMI of 19.2 kg/m^2, with a urine sodium level of 10 mmol/L. The serum sodium level was 139 mmol/L. She denied any changes in oral intake or ileostomy output. The caloric content of PN was decreased by another 20%, reducing PN to 4 d/wk and subsequently decreasing the supplemental sodium intake to a daily average of 246 mEq (4.6 mEq/kg/d). The following month, she demonstrated a weight loss of 3.2 kg and a BMI of 18 kg/m^2, despite reporting an "excellent appetite." Her urine sodium level was requested but never received due to noncompliance. The serum sodium level was 137 mmol/L, and the estimated ileostomy output was approximately 2800 mL (56 mL/kg/d). PN energy input was increased by 10%, PN sodium provision was unchanged, and the addition of salt to foods was reinforced due to suspicion of noncompliance. Approximately 2 months later, the patient demonstrated a weight gain of 5.1 kg, BMI of 19.8 kg/m^2, and serum and urine sodium levels of 139 and 79 mmol/L, respectively. Furthermore, the patient reported that she had not been infusing PN for approximately 1 month. Due to a combination of noncompliance with PN, adequate weight gain, improved oral intake, and appropriate laboratory results, the patient's supplemental PN was discontinued. Three weeks later, the patient's weight was down 3.3 kg with a BMI of 18.7 kg/m^2, and she reported no changes in oral intake. The serum sodium level was 141 mmol/L, and the urine sodium level decreased to 10 mmol/L. Ileostomy output was approximately 2200 mL (42 mL/kg/d). To provide more consistent and precise sodium supplementation, the patient was prescribed sodium chloride tablets at a dose of 120 mEq of sodium (2.3 mEq/kg/d). The following month, the patient demonstrated a 2.3-kg weight gain with a BMI of 19.5 kg/m^2. Her urine sodium level increased to 137 mmol/L, while the serum sodium level remained stable. Over the next 8 months, the patient maintained her weight and a BMI >19 kg/m^2, and the decision was made to take down her ileostomy. One month after the operation, the patient demonstrated a weight loss of 4.4 kg. Serum sodium and urine sodium levels were collected and were 136 and 10 mmol/L, respectively. It was recommended that she resume the sodium chloride tablets per her previous dose of 120 mEq (2.3 mEq/kg/d), and 5 days later, she had an improved urine sodium level of 163 mmol/L. Her weight was not obtained at that time; however, 3 weeks later, her weight increased by 2.2 kg. Figure 1 depicts urine sodium levels in relation to weight from the time of the initial documented TBSD to the time that the patient was being followed for FTT.

Case 2

The second case was a 12-year-old female patient with intestinal failure secondary to dysmotility with chronic intestinal pseudo-obstruction who underwent a colectomy and creation of an end ileostomy. The patient presented to our clinic for a routine visit with an unexpected 5.2-kg weight loss and a BMI of 15.1 kg/m^2. Both serum sodium and urine sodium levels were collected and were 135 and <10 mmol/L, respectively. At that time, the patient was admitted to the hospital and began receiving 154 mEq (4.5 mEq/kg/d) of sodium supplementation via an enteral bolus of 250 mL of normal saline 4 times daily. Additionally, the patient was started on a combination of enteral feeds and intravenous hydration of D5 0.45NS titrated to a fluid goal of 75 mL/h. Over the first 4 days of admission, ileostomy output averaged 2100 mL (59 mL/kg/d). The electrolyte content of ileostomy output was 115 mmol/L of sodium, >10 mmol/L of potassium, and 35 mmol/L of chloride, showing an expected high sodium loss. On hospital day 5, her repeat urine sodium level was

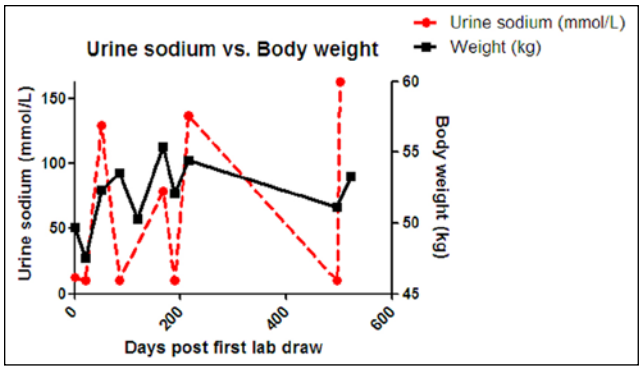

Figure 1. Graphic display of urine sodium levels (broken line) in relation to body weight (black solid line) for case 1. Note the relationship between each loss in body weight with a decline in the urine sodium level. Days represent the range of laboratory results and weights available during the time that the patient was being followed for failure to thrive.

203 mmol/L, the patient gained 0.67 kg, and her BMI increased to 16.3 kg/m^2. After a 3-week hospital course, the patient was discharged home on a modified oral diet, controlling ostomy output, and PN containing 185 mEq (4.7 mEq/kg) of sodium. After the patient was discharged, her repeat urine sodium level was requested but not received due to noncompliance. Over the next 10 months, the energy content and electrolyte composition of her home PN were slowly weaned as oral intake increased. The patient then presented to the clinic with an unexpected 2.5-kg weight loss with a BMI of 18 kg/m^2. Both serum sodium and urine sodium levels were collected and were 136 and <10 mmol/L, respectively. The sodium content of PN was increased from 134 mEq (3 mEq/kg) to 181 mEq (4.2 mEq/kg), and PN calories were increased by 10%. Ileostomy output was not quantified at home due to the patient's refusal. Two weeks later, both serum sodium and urine sodium levels were collected and were 140 and 154 mmol/L, respectively, and the patient gained 1.1 kg (BMI, 18.4 kg/m^2). See Table 1 for a chronological display of this patient's serum sodium level, urine sodium level, and weight.

Case 3

The third case was a 19-year-old female patient with a history of Hirschsprung disease and gastrointestinal dysmotility with an end ileostomy and long-term PN dependence. The patient presented to our clinic with an unexpected weight loss of 1.1 kg and a BMI of 16.2 kg/m^2, despite receiving 100% of her estimated energy needs via PN in addition to minimal oral intake. Ostomy output was not quantified at home due to the patient's refusal. Serum sodium and urine sodium levels were 139 and 10 mmol/L, respectively. The amount of sodium in PN was increased by 20 mEq to maximize the sodium concentration to 154 mEq/L, providing a total of 385 mEq (9 mEq/kg); however, the PN energy content was left unchanged. Three weeks later, the patient presented with a 3.75-kg weight gain and an improved BMI of 17.6 kg/m^2 and denied any change in her oral intake. At that time, serum sodium and urine sodium levels were 142 and 86 mmol/L, respectively. The patient was seen in the clinic 5 weeks later and gained another 4.3 kg (BMI, 19.3 kg/m^2) and reported improved oral intake. The serum sodium level was 140 mmol/L, and the urine sodium level was requested but never received due to noncompliance. After clinical evaluation, caloric input from PN was decreased by 20%, but the sodium content was left stable at 385 mEq

Table 1. Chronological Display of Changes in Each Patient's Serum Sodium Level as They Relate to Urine Sodium Level and Body Weight Changes.

Date	Serum Sodium Level, mmol/L	Urine Sodium Level, mmol/L	Weight, kg
Case 1			
1/25/11	141	13	49.7
2/16/11	138	<10	47.5
3/16/11	138	129	52.3
4/20/11	139	10	53.5
5/25/11	137	N/A	50.3
7/13/11	139	79	55.4
8/4/11	141	10	52.1
8/31/11	141	137	54.4
6/10/12	136	10	51.1
6/15/12	140	163	N/A
7/6/12	N/A	N/A	53.3
Case 2			
5/11/11	136	126	39.8
7/6/11	135	<10	34.6
7/11/11	139	203	35.3
7/12/11	138	96	N/A
2/15/12	N/A	N/A	45.9
5/2/12	136	<10	43.4
5/15/12	140	154	44.5
7/11/12	138	79	46.9
Case 3			
2/3/12	139	130	44
2/27/12	139	10	42.95
3/19/12	142	86	46.7
4/25/12	140	N/A	51
6/8/12	142	111	N/A
7/9/12	N/A	N/A	52.3
Case 4			
12/4/11	144	98	7.82
12/16/11	132	<10	7.56
1/11/12	N/A	89	8.41
3/29/12	N/A	N/A	8.98
5/24/12	137	<10	8.73
6/28/12	N/A	46	9.1
7/31/12	N/A	<5	N/A
8/10/12	143	80	9.14

N/A, not available.

(7.5 mEq/kg). Approximately 6 weeks later, laboratory results were collected at an outside facility, and serum and urine sodium levels were 142 and 111 mmol/L, respectively. A weight check was requested but never received. Approximately 1 month later, the patient canceled her follow-up visit to our clinic but reported that her weight at that time was up by 1.3 kg with an improved BMI of 19.6 kg/m^2. See Table 1 for a chronological display of this patient's serum sodium level, urine sodium level, and weight.

Case 4

The fourth case was an 18-month-old male patient with a history of Hirschsprung disease complicated by a stricture at his anastomosis. He underwent an extensive lysis of small bowel adhesions and loop ileostomy formation. He presented to our clinic with a 0.3-kg weight loss and weight for length at the seventh percentile, despite receiving 100% of his minimum estimated energy needs via gastrostomy tube feedings. The sodium content of enteral nutrition (EN) was 13.8 mEq (1.8 mEq/kg), the estimated ileostomy output was 280 mL (37 mL/kg/d), and serum sodium and urine sodium levels were 132 and <10 mmol/L, respectively. Due to a low serum CO_2 level, he was prescribed 15 mEq of sodium bicarbonate twice daily (4 mEq/kg/d) for the treatment of his TBSD. Additionally, EN calories were increased by 10%, bringing the total sodium intake to 45 mEq (6 mEq/kg). One month later, he returned to the clinic and demonstrated a 0.85-kg weight gain, and the urine sodium level was 89 mmol/L. The serum sodium level and estimated ileostomy output were not obtained. The total sodium intake from EN and sodium bicarbonate supplements was 45 mEq (5.4 mEq/kg). Six weeks later, the patient was admitted to the hospital for 15 days secondary to viral-associated respiratory distress. At admission, his weight was up 0.3 kg, the total sodium intake was 46 mEq (5.3 mEq/kg/d), and the serum sodium level was 145 mmol/L. His urine sodium level was not collected. During his admission, sodium bicarbonate was held due to his nil per os status and was not resumed at discharge secondary to appropriate weight gain and laboratory results. At discharge, the patient's weight was down 0.2 kg. EN provided 1.9 mEq/kg of sodium daily. Two months later, he was seen in the clinic and demonstrated poor weight gain and had serum and urine sodium levels of 137 and <10 mmol, respectively. Sodium bicarbonate was resumed at the previous dose of 15 mEq twice daily (3.5 mEq/kg/d), bringing the total sodium intake to 48 mEq (5.5 mEq/kg). One month later, the patient demonstrated a 0.4-kg weight gain and an improved urine sodium level of 46 mmol/L. The following month, the urine sodium level was collected at an outside laboratory and was <5 mmol/L, despite the patient's mother denying changes in the EN regimen, ileostomy output, or weight. Subsequently, the sodium bicarbonate dose was increased to 25 mEq twice daily, increasing the total sodium intake to 68 mEq (7.5 mEq/kg). A week later, the patient presented to the clinic, and despite an improved urine sodium level of 80 mmol/L, his weight remained unchanged. He is currently stable and awaiting intestinal surgery. See Table 1 for a chronological display of this patient's serum sodium level, urine sodium level, and weight.

Discussion

These 4 cases illustrate unintentional weight loss, despite an estimated adequate caloric intake, that was associated with a urine sodium level ≤10 mmol/L in the presence of sodium losses via ileostomy. With sodium supplementation, all patients demonstrated weight gain and an improved urine sodium level. All instances of weight gain corresponded with a urine sodium level >10 mmol/L, with the exception of 1 occurrence in case 1 when a weight gain of 1.2 kg occurred with a urine sodium level of 10 mmol/L. However, the patient could have been nearing TBSD, with weight yet unaffected as seen by a 3.2-kg weight loss the following month.

The connection between TBSD and FTT has been well described in premature and newborn infants[5,7] in whom difficulty regulating sodium balance is expected. Bower et al[5] reviewed the records of 11 premature infants with ileostomies, 6 of whom failed to gain weight despite receiving adequate caloric intake. All cases of growth disturbances were associated with persistent urine sodium levels <10 mmol/L, despite normal serum sodium levels. With the supplementation of sodium chloride or sodium bicarbonate directly proportional to the amount of ileostomy output, all 6 infants demonstrated weight gain and urine sodium levels >10 mmol/L. Similarly, in our 4 cases, serum sodium levels remained within the reference range and often remained stable or only mildly fluctuated, despite significant alterations in urine sodium levels with supplementation. This supports the evidence that urine sodium is a superior and/or more sensitive indicator of total body stores, even in older children, for the detection of TBS.

Although much has been published about sodium depletion in patients of various ages with established ileostomies,[1-3,8] there is a gap in the literature linking TBSD to weight loss in older children and adults. Our cases suggest that the relationship between TBSD and FTT is not exclusive to infants but rather extends into adolescence and possibly adulthood. Our cases are supported by an additional report by Tsao et al[9] in which a 56-year-old female patient with a loop jejunostomy secondary to Crohn's disease and multiple bowel resections struggled with weight loss and high-volume stoma output despite a high caloric intake. She had a normal serum sodium level of 140 mmol/L but a random urine sodium level of 10 mmol/L. After treatment with a glucose-saline solution containing 90 mEq/L, she demonstrated an improvement in her BMI by 1.7 kg/m^2.

Guidelines for the treatment of TBSD in patients with ileostomies are not well defined. In cases 1, 2, and 3, patients were consuming an oral diet in addition to PN, making it difficult to determine the total sodium intake. However, when the patient

in case 3 first presented with weight loss and TBSD, she was fed almost exclusively via PN and required 9 mEq/kg of sodium to achieve weight gain and a urine sodium level >10 mmol/L. In our patient fed exclusively via enteral feeds (case 4), the total sodium intake was easily quantified. Weight gain and urine sodium levels >10 mmol/L were achieved when the total sodium intake was 5.3–7.5 mEq/kg/d, and weight loss and TBSD occurred when the total sodium intake was <2 mEq/kg/d. Our data suggest that the optimal sodium intake for this patient population may be 5–10 mEq/kg/d; however, this may vary depending on individual daily ostomy losses. This recommendation is consistent with those by Schwarz et al[6,10] (6–10 mEq/kg/d) and Bower et al[5] (4–10 mEq/kg/d). We were able to successfully treat TBSD using parenteral and enteral routes and with sodium chloride and sodium bicarbonate, suggesting that treatment can be individualized based on clinical factors such as laboratory results and feeding modality. The appropriate duration of supplementation is also not well established, but our cases suggest that ongoing supplementation may be necessary to maintain sodium balance, particularly in the setting of high stoma output and possibly even after ileostomy takedown. When the patient in case 4 was hospitalized and sodium supplementation was discontinued, he lost weight and had TBSD within 2 months of discharge, suggesting that he would have benefitted from continuing sodium supplementation. The patient presented in case 1 lost weight and demonstrated sodium depletion shortly after her ileostomy takedown, suggesting ongoing poor sodium balance and the need for continued sodium supplementation. This finding is supported by Schwarz et al,[10] who demonstrated episodes of fluid and sodium depletion at 4–8 years of age in 3 children with ileostomies performed in the neonatal period, followed by ileoendorectal pull-throughs performed at 7–14 months of age.

The exact mechanism by which sodium depletion influences weight is unclear, but earlier studies have suggested ineffective glucose absorption due to impaired sodium-glucose cotransport via SGLT-1.[7] However, this cannot be the only mechanism, as FTT associated with TBSD occurs in patients receiving full PN, with negligible or no oral energy intake as described in case 3. Additionally, TBSD can be successfully treated by supplementing sodium intravenously as demonstrated in cases 3 and 4. The lack of anabolism due to sodium depletion may relate to a shutdown of anabolic pathways with resultant loss of weight gain and protein accretion. The mechanism may also be one of the contributing factors that cause patients with cystic fibrosis (with sodium losses) to suffer from significant FTT.[11,12]

The possibility of confounding factors affecting changes in weight cannot be ruled out by these case reports. At times, caloric input from PN or enteral feeds was adjusted concurrently with sodium input, complicating the effect that sodium alone would have on the patient's weight. Additionally, 3 of the 4 patients presented consumed foods and beverages by mouth, causing sodium and caloric intake to vary day to day. In future studies looking at TBSD, all oral sodium and energy intake should be accounted for using daily food journals, and when making changes to sodium intake, caloric intake should be held consistent whenever possible. Noncompliance with nutrition support regimens also has to be considered as a contributing factor to fluctuations in weight. This is particularly evident in case 1 in which noncompliance was observed with both PN regimen and obtaining follow-up urine sodium levels. Lastly, as this is a retrospective case study, the quality of weight gain was not directly addressed, and it is unclear if weight gain reflected increased muscle mass. However, none of the laboratory results or documented physical examination findings indicated fluid retention. In future studies, it would be beneficial to obtain additional anthropometric measurements, such as triceps skinfold and midarm muscle circumference, to determine changes in body fat and muscle mass.

Conclusion

This case report and other related studies suggest that patients of all ages with high stoma output should have routine urine sodium levels checked, particularly in the setting of weight loss or poor gain. In instances where sodium supplementation is necessary, the dose, type, and duration of treatment should be driven by careful and frequent monitoring of weight, ileostomy output, and laboratory serum and urine sodium levels. Further research is needed to better understand the relationship between TBSD and FTT and to establish intervention guidelines.

References

1. Gallagher ND, Harris DD, Skyring AP. Fluid and electrolyte disturbance in patients with long-established ileostomies. *Gut*. 1962;3:219-223.
2. Kennedy HG, Al-Dujaili EA, Edwards CR, Truelove SC. Water and electrolyte balance in subjects with a permanent ileostomy. *Gut*. 1983;24:702-705.
3. Hill GL, Mair WS, Goligher JC. Cause and management of high volume output salt-depleting ileostomy. *Br J Surg*. 1975;62:720-726.
4. Clarke AM, Chirnside A, Hill GL, Gillian P, Stewart MK. Chronic dehydration and sodium depletion in patients with established ileostomies. *Lancet*. 1967;2:740-743.
5. Bower TR, Pringle KC, Soper RT. Sodium deficit causing decreased weight gain and metabolic acidosis in infants with ileostomy. *J Pediatr Surg*. 1988;23:567-572.
6. Schwarz KB, Ternberg JL, Bell MJ, Keating JP. Sodium needs of infants and children with ileostomy. *J Pediatr*. 1983;102:509-513.
7. Sacher P, Hirsig J, Gresser J, Spitz L. The importance of oral sodium replacement in ileostomy patients. *Prog Pediatr Surg*. 1989;24:226-231.
8. Weise WJ, Serrano FA, Fought J, Gennari FJ. Acute electrolyte and acid-base disorders in patients with ileostomies: a case series. *Am J Kidney Dis*. 2008;52:494-500.
9. Tsao SK, Baker M, Nightingale JM. High-output stoma after small-bowel resections for Crohn's disease. *Nat Clin Pract Gastroenterol Hepatol*. 2005;2:604-608.
10. Schwarz KB, Keating JP, Ternberg JL, Bell MJ. Sodium balance following soave ileo-endorectal pull-through. *J Pediatr Surg*. 1977;12:945-953.
11. Laughlin JJ, Brady MS, Eigen H. Changing feeding trends as a cause of electrolyte depletion in infants with cystic fibrosis. *Pediatrics*. 1981;68:203-207.
12. Coates AJ, Crofton PM, Marshall T. Evaluation of salt supplementation in CF infants. *J Cyst Fibros*. 2009;8:382-385.