Strategies for early metabolic disturbances in patients with an end jejunostomy or end ileostomy. Experience from a specialized Home Parenteral Nutrition (HPN) center

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Abstract

Introduction: An end stoma syndrome is usually the result of an intentional surgical intervention in the course of staged treatment or a complication of surgery. These patients most frequently suffer from water and electrolyte disturbances, malnutrition syndromes caused by malabsorption of trace elements and/or vitamins, and undernutrition.

Aim: To present early metabolic disturbances observed in patients with an end jejunostomy or end ileostomy syndrome on the first day of their hospitalization in a specialist Home Parenteral Nutrition (HPN) center.

Material and methods: The study included 142 patients with an end stoma syndrome (76 women and 66 men), hospitalized between 2004 and 2014. Patients were divided into two main groups. Group A consisted of 90 patients with an end jejunostomy and group B consisted of 52 patients with an end ileostomy.

Results: After comparing the patients with an end jejunostomy vs. those with an end ileostomy, significant differences were found as regards pH (7.34 vs. 7.39, $p = 0.043$) and BE (3.24 vs. –0.86, $p = 0.005$). Depending on the lack or possibility of oral food intake, patients in the end jejunostomy group had different levels of the markers phosphate, Mg, Ca, urea, and creatinine, with all of these parameters within normal laboratory limits. When the end ileostomy group was divided into subgroups depending on the lack or possibility of oral food intake, differences in C-reactive protein activity were found (55.6 vs. 25.7, $p = 0.041$).

Conclusions: Patients with an end jejunostomy syndrome are more prone to metabolic acidosis with significant alkali deficiencies.

Introduction

An end stoma syndrome is usually an acquired state, which is most often the result of an intentional surgical intervention in the course of staged treatment or a complication of surgery. This condition can lead to metabolic disturbances. These complications depend on the location of the end stoma and the length of the remaining functional portion of the digestive tract, as well as the type of therapy and an association with other organ failures. Early disturbances involve excesses and deficiencies as regards electrolyte balance, acid-base balance and carbohydrate and fat metabolism, while late metabolic disturbances include liver complications, e.g. parenteral nutrition-associated liver disease (PNALD), hepatic steatosis (fatty liver), hepatic cholestasis, cholelithiasis (gallstones), acalculous cholecystitis, renal complications, metabolic bone damage and secondary osteoporosis. Treating patients with an end stoma syndrome requires knowledge of the physiology and pathophysiology of the digestive tract, as well as the consequences of metabolic disturbances. Such

knowledge makes it possible to implement appropriate care [1]. From a clinical point of view, an end stoma syndrome is often accompanied by short bowel syndrome. This means that the medical team must be able to implement proper dietetic treatment, maintain water-electrolyte balance and monitor concomitant metabolic disturbances [2]. Proper monitoring of metabolic disturbances enables the medical personnel to obtain necessary information about the patient's clinical condition and to implement appropriate medical treatment [3, 4].

Aim

The aim of this work was to compare early metabolic disturbances observed in patients with an end jejunostomy or end ileostomy syndrome on the first day of their hospitalization in a specialist Home Parenteral Nutrition (HPN) center.

Material and methods

Between 2004 and 2014 medical records of 142 patients (76 women and 66 men) aged 18–85 were analyzed retrospectively. These patients were admitted to a specialist clinical nutrition center due to an end jejunostomy or end ileostomy syndrome that required further long-term nutritional treatment. All patients were fed parenterally via central or peripheral venous access during the post-operative period. Time of parenteral nutrition after surgery and at the same time waiting for transfer to a specialist HPN center lasted from 2 to 7 weeks. The patients received mainly ready-to-use (RTU) products, with calorie content ranging from 25 to 35 kcal/kg b.w. as per the European Society of Parenteral and Enteral Nutrition (ESPEN) guidelines. The patients were divided into two main groups:

– group A – 90 patients with an end jejunostomy; the average length of intestine was 70 ±40 cm,
– group B – 52 patients with an end ileostomy; the average length of intestine was 250 ±45 cm.

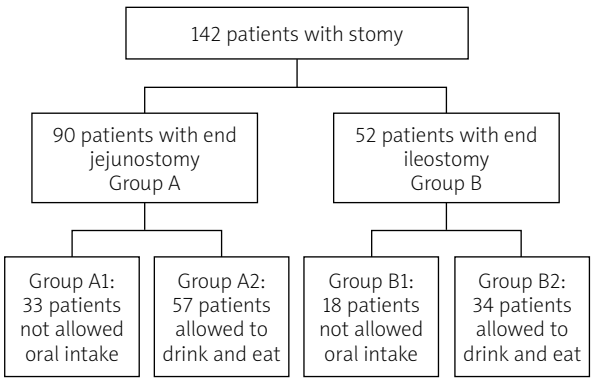

Figure 1. Diagram representing the groups included in the study

Each group was then analyzed comparing the patients allowed to take some food and fluids with those who were not allowed to take any food or fluids. Figure 1 shows the inclusion of the patients in the various groups.

The second stage of analysis involved group A (end-jejunostomy) patients (n = 90), who were divided into two subgroups: A1 and A2 according to Figure 1.

Subgroup A1 = 33 patients who were not allowed any oral intake of food or fluids in their primary treatment centers. Subgroup A2 = 57 patients who were allowed oral intake of flood and fluids in their primary treatment centers.

Medical records obtained from the patients' primary treatment centers revealed subgroup A2 patients (n = 57) to have received various oral diets depending on the subjective experience of the medical personnel from those centers. For 45 patients the initiation of oral feeding involved simple liquids that were gradually replaced with watery porridges, which were subsequently thickened and replaced with semi-liquid purees.

Only 12 patients in this subgroup were post-operatively hydrated with electrolyte-rich fluids (such as Gastrolyte or WHO formula of the oral rehydration salts) at up to 500 ml/day.

The third stage of analysis involved patients with an end ileostomy (group B, n = 52), who were divided into two subgroups: according to Figure 1.

Subgroup B1 = 18 patients who had not been allowed any oral food or fluid intake at their primary treatment centers. Subgroup B2 = 34 patients who had been allowed oral intake of food and fluids at their primary treatment centers.

Medical records obtained from the patients' primary treatment centers revealed subgroup B2 (n = 24) patients to have received various oral diets depending on the subjective experience of the medical personnel from those centers. For subgroup B2 patients the initiation of post-operative oral feeding involved simple liquids that were gradually replaced with porridges and eventually with a normal diet. No patients from this subgroup received post-operative hydration with electrolyte-rich fluids (such as Gastrolyte or WHO-approved oral rehydration salts).

Statistical analysis

SPSS IBM 21 for Windows was used for statistical calculations. Exploratory analyses (frequencies, comparison of mean values, percentage distribution) were conducted. For all statistical comparisons the non-parametric Mann-Whitney U-test was used.

Results

Differences in acid-base balance (pH and base excess (BE)) were observed between the groups. Group A

patients showed lower pH values and a significant base deficit in comparison with patients with group B. Moreover, both analyzed patient groups (A and B) demonstrated elevated liver enzymes (ALT, AST, ALP, GGTP), but no difference was noted between the groups. Results of analyses of selected laboratory parameters are presented in Table I.

The end-jejunostomy group A (subgroup A1 vs. subgroup A2) differed in terms of the following laboratory parameters: phosphate, Mg, Ca, urea, creatinine, total protein, cholesterol. These values were within the normal range for both subgroups (A1 vs. A2) (Table II).

Subgroups B1 and B2 showed elevated liver enzymes (ALT, AST, ALP, GGTP) exceeding the upper limit of normal. Moreover, both subgroups had elevated C-reactive protein (CRP) levels above the normal range, with subgroup B1 demonstrating significantly higher CRP levels in comparison to those in subgroup B2. Among the examined markers, differences were found only in CRP levels (Table III).

Discussion

Improper treatment of patients with a high-output end stoma syndrome may aggravate the existing nutritional deficiencies and cause life-threatening metabolic disturbances. In end stoma syndromes, a particularly dangerous complication is excessive loss of fluids through the intestinal stoma, leading to severe

Table I. Selected biochemical parameters in patients with an end jejunostomy (group A) and in patients with an end ileostomy (group B)

Parameter	Group A (n = 90) End jejunostomy		Group B (n = 52) End ileostomy		*P*-value
	Mean	SD	Mean	SD	A vs. B
pH (7.35–7.45)	7.34	0.33	7.39	0.06	0.043
BE (–2.5/+2.5)	–3.24	6.48	–0.86	5.7	0.005
Na (135–145) [mol/l]	135.6	4.62	136.1	5.2	NS
Cl (96–110) [mol/l]	99.8	7.87	99.1	6.4	NS
K (3.7–5.0) [mmol/l]	4.6	3.5	4.4	0.7	NS
Phosphate (2.5–5.0) [mmol/l]	3.49	1.18	3.8	1.1	NS
Mg (1.6–2.5) [mol/l]	1.74	0.63	1.7	0.3	NS
Ca (8.5–10.5) [mmol/l]	8.20	2.42	8.7	1.6	NS
Urea (19–30) [mg/dl]	42.1	33.2	40.4	30.2	NS
Creatinine (0.73–1.36) [mg/dl]	1.13	0.8	1.1	1.1	NS
Total protein (6.2–8.3) [g/l]	6.93	1	7	0.9	NS
Albumin (3.3–4.5) [g/l]	3.24	0.8	3.3	0.7	NS
Bilirubin (0.2–1.3) [mg/dl]	1.62	2.4	0.97	0.8	NS
ALT (16–60) [U/l]	71.5	68.2	80.3	120.5	NS
AST (17–59) [U/l]	44.3	32.1	51.7	52.3	NS
ALP (46–116) [U/l]	228.9	160.3	231.6	162.6	NS
GGTP (15–73) [U/l]	213	262	179.1	178.7	NS
Amylase (30–120) [U/l]	68.6	41.1	67.71	38.8	NS
Lipase (23–300) [U/l]	247.7	448	220.3	190.8	NS
LDH (82–227) [U/l]	283.5	220.8	242.5	122.9	NS
Total cholesterol (> 190) [mg/dl]	147.3	46.9	153	44.7	NS
Triglycerides (> 150) [mg/dl]	167.2	87.2	153.8	73.5	NS
CRP (0–10) [mg/dl]	42	51.8	36	40	NS

Table II. Selected biochemical parameters in patients (n = 90) with a jejunostomy depending on the lack (group A1) or possibility of oral intake of food and fluids (group A2)

Parameter	A1 = 33 No oral intake		A2 = 57 Oral intake		*P*-value
	Mean	SD	Mean	SD	
pH (7.35–7.45)	7.28	0.54	7.35	0.06	NS
BE (–2.5/+2.5)	–4.18	5.83	–2.7	6.82	NS
Na (135–145) [mmol/l]	135	5.64	135.5	3.9	NS
Cl (96–110) [mmol/l]	100.6	7.84	99.4	7.9	NS
K (3.7–5.0) [mmol/l]	5.1	5.7	4.2	0.6	NS
Phosphate (2.5–5.0) [mmol/l]	3.0	1.2	3.7	1.04	0.026
Mg (1.6–2.5) [mmol/l]	1.8	0.6	1.5	0.5	0.021
Ca (8.5–10.5) [mmol/l]	6.9	2.9	8.9	1.6	0.001
Urea (19–30) [mg/dl]	31.9	21.6	48	37.2	0.021
Creatinine (0.73–1.36) [mg/dl]	0.8	0.4	1.4	0.9	0.005
Total protein (6.2–8.3) [g/l]	6.6	0.9	7.1	1.07	0.033
Albumin (3.3–4.5) [g/l]	3.1	0.6	3.3	0.8	NS
Bilirubin (0.2–1.3) [mg/dl]	2.1	3.0	1.3	1.9	NS
ALT (16–60) [U/l]	70	30.6	72.3	67.4	NS
AST (17–59) [U/l]	43	36	44.7	29.9	NS
ALP (46–116) [U/l]	216	109.8	236.1	183.9	NS
GGTP (15–73) [U/l]	145.6	131.6	252.1	308.2	NS
Amylase (30–120) [U/l]	64.3	37.7	71.1	43.1	NS
Lipase (23–300) [U/l]	150.9	147.6	300.5	540.8	NS
LDH (82–227) [U/l]	283.8	197	283.3	235.2	NS
Total cholesterol (> 190) [mg/dl]	129.3	50.5	157.8	41.6	0.002
Triglycerides (> 150) [mg/dl]	148.9	88.3	177.8	85.5	NS
CRP (0–10) [mg/dl]	41.6	56.5	42.8	49.4	NS

dehydration. Patients with an end jejunostomy are particularly prone to this type of dehydration. A common treatment error in their case is when healthcare professionals allow the patients to take food and liquids orally without any restrictions [5]. This treatment error is often committed in inexperienced medical centers in which the patient's initial surgery and postoperative care take place. Based on the analyzed clinical data, end-jejunostomy patients who had received food and fluids orally in their primary hospitals (subgroup A2) showed elevated serum creatinine and urea levels as well as low serum magnesium levels. Abnormalities in these laboratory parameters suggest early renal failure due to dehydration caused by excessive stoma-related fluid loss.

The data suggest that in subgroup A2, with longer administration of foods and fluids orally in primary hospitals allowed, more serious metabolic disorders were observed. This relationship was not observed in subgroup B2 of patients with end ileostomy.

Patients with an end jejunostomy should be subjected to special restrictions regarding the consumption of fluids and food. A safe and recommended procedure is oral rehydration with an electrolyte-rich fluid (this WHO-approved formula contains 500 ml of distilled water, 20 ml of 40% glucose, 1 ampoule of 8.4% $NaHCO_3$,

Table III. Selected biochemical parameters in patients with an end ileostomy (n = 52) depending on the lack (group B1) or possibility of oral intake of food and fluids (group B2)

Parameter	B1 = 18 No oral intake		B2 = 34 Oral intake		*P*-value
	Mean	SD	Mean	SD	
pH (7.35–7.45)	7.4	0.07	7.39	0.06	NS
BE (–2.5/+2.5)	–0.8	5.7	–0.87	5.85	NS
Na (135–145) [mmol/l]	135.9	6.1	136.1	4.77	NS
Cl (96–110) [mmol/l]	98.6	7.6	99.4	5.82	NS
K (3.7–5.0) [mmol/l]	4.3	0.6	4.4	0.73	NS
Phosphate (2.5–5.0) [mmol/l]	3.5	0.8	4.0	1.24	NS
Mg (1.6–2.5) [mmol/l]	1.6	0.4	1.8	0.34	NS
Ca (8.5–10.5) [mmol/l]	8.5	1.6	8.9	1.66	NS
Urea (19–30) [mg/dl]	38	16	41.7	35.7	NS
Creatinine (0.73–1.36) [mg/dl]	0.9	0.3	1.2	1.43	NS
Total protein (6.2–8.3) [g/l]	6.8	1.0	7.1	0.87	NS
Albumin (3.3–4.5) [g/l]	3.2	0.7	3.4	0.66	NS
Bilirubin (0.2–1.3) [mg/dl]	0.8	0.5	1.0	1.0	NS
ALT (16–60) [U/l]	76.5	164.8	82.3	91.9	NS
AST (17–59) [U/l]	47.7	66.1	53.8	44.3	NS
ALP (46–116) [U/l]	226.6	159.4	234.3	166.5	NS
GGTP (15–73) [U/l]	164	139.7	187.2	197.7	NS
Amylase (30–120) [IU/l]	63.8	30.1	69.7	42.9	NS
Lipase (23–300) [U/l]	248	241	206.9	163.6	NS
LDH (82–227) [U/l]	244.7	94.2	241.5	136.3	NS
Total cholesterol (> 190) [mg/dl]	148	38.4	155.7	48.01	NS
Triglycerides (>150) [mg/dl]	165.8	76.7	147.4	72.18	NS
CRP (0–10) [mg/dl]	55.6	52.5	25.7	27.37	0.041

10 ml of 15% KCl, 20 ml of 10% NaCl). Up to 500 ml should be given daily.

When patients with an end stoma syndrome are losing more fluids through the intestinal stoma than through the urinary system, it is absolutely necessary to try to change such a clinical situation through intravenous fluid replacement and reduction or discontinuation of oral intake of food and fluids. Moreover, in these patients, it is advisable to use drugs that reduce secretion from the digestive tract, such as proton pump inhibitors (PPIs), H2-receptor antagonists, racecadotril, inhibitors of intestinal peristalsis and somatostatin analogues, which decrease pancreatic secretion [5]. Significant stoma fluid loss exacerbates shortages of energy reserves, as well as deficits of sodium, magnesium, calcium, trace elements and electrolytes. In patients with an end stoma syndrome, gastric emptying occurs faster because inhibitory hormones (gastric inhibitory polypeptide – GIP) are no longer secreted. This leads to increased hydrochloric acid secretion, with a possibility of ulcer formation in the stomach and duodenum. It must be emphasized that in cases where the end stoma was created from one of the first loops of the small intestine, patients are much more prone to dehydration than patients in whom the end stoma was created from the terminal loops of the small intestine [6].

In extreme cases, end-jejunostomy patients who receive food and fluids orally without any volumetric restrictions may develop severe dehydration, metabolic acidosis, and progressive renal failure.

On the other hand, there are many opinions in support of the benefits of trophic enteral feeding, which helps to reduce the incidence of septic complications associated with bacterial translocation. Trophic feeding helps rebuild a normal intestinal microflora by nourishing enterocytes and preventing atrophy of intestinal villi. Trophic feeding stimulates the immune system, as well as pancreatic and hepatic secretion [7]. In the analyzed material, in the group of patients with an end ileostomy who were not allowed to consume food or fluids we observed significantly elevated markers of inflammation. C-reactive protein elevation in subgroup B1 is likely due to a lack of stimulation of intestinal villi, which weakens the integrity of intestinal mucosa, giving rise to bacterial translocation and secondary inflammation. Improperly treated patients with an end stoma syndrome suffer from hypomagnesemia, hypokalemia and hypophosphatemia, which may possibly contribute to another acute metabolic complication: refeeding syndrome [8].

In the analyzed group of patients with end jejunostomy in 5 cases there were demonstrated features of severe malnutrition and electrolyte deficiencies. These patients were at risk of refeeding syndrome. Parenteral nutrition was progressively implemented after adjusting fluid and electrolyte imbalance.

Another metabolic disturbance that may occur in this group of patients is elevation of liver enzymes, which is most likely caused by excessive nutritional therapy in primary centers before admission to a specialist center [9, 10]. Having excluded a mechanical, viral, alcohol-related or parenchymal cause of liver problems in these patients, parenteral nutrition-associated liver disease (PNALD) should be suspected. Elevation of liver enzymes in the group of patients with an end stoma is most likely to be associated with excessive parenteral feeding in primary centers, rather than with the presence of an end stoma. This is consistent with our findings, as we have observed elevated liver enzymes ALT, AST, ALP and GGTP above the normal range in the analyzed clinical data. Excessive parenteral calorie intake from carbohydrates and/or fats is a common cause of liver damage in centers with little experience in nutritional treatment [11]. A synthetic trophic hormone acting on intestinal villi – glucagon-like peptide-2 – appears very promising for the prevention of metabolic complications of end stoma syndromes. Multicenter clinical trials have shown significant metabolic benefits in patients who received this drug [12]. Clinical experience in studying the effects of glucagon-like peptide-2 in multicenter clinical trials shows a reduction in stoma output, associated with a mechanism of increased intestinal absorption. Administration of glucagon-like peptide-2 significantly improved the quality of life of patients with short bowel syndrome and a concomitant end stoma [13, 14].

Conclusions

Patients with an end jejunostomy syndrome are more prone to metabolic acidosis with large alkali deficiencies. This is caused by loss of alkaline content. Patients with an end jejunostomy who are allowed to consume food and fluids without calorie or fluid restrictions may develop severe dehydration, hypomagnesemia and progressive renal failure.

Conflict of interest

The authors declare no conflict of interest.

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