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The challenges of managing and following-up a case of short bowel in eastern europe

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ABSTRACT

INTRODUCTION: This article reflects on the plight of patients with short bowel syndrome (SBS) in developing countries. SBS is life threatening, rare, complex and often not considered a priority by healthcare planners in the developing countries because of the high cost of treatment. Data was collected and analyzed from 3 different hospitals in two different countries (Romania and Austria) from November 2013 to February 2016

CASE PRESENTATION: The patient had an emergency surgery for volvulus as a result of an extensive ischemic necrosis, with just 80 cm of the bowel left and no ileocecal valve after enterectomy. Despite intensive care and surgeries for anastomotic joint ischemic necrosis and intestinal adhesion with just 70 cm of the intestine left after primary anastomosis, the patient remained in a catabolic state (metabolic acidosis, severe malabsorption and loss of nutrients, water and electrolytes through diarrhea) and was transferred overseas where two more surgeries (intestinal stomas) and good intensive care helped to achieve enteral autonomy at the optimal time.

DISCUSSION: This immune-deficient patient was exposed to various types of bacteria (*Klebsiella pneumoniae*, *Pseudomonas aeruginosa*). Two years after surgery an acute enterocolitis with salmonella infection and resultant intestinal failure treated in patient's country of origin failed to achieve enteral nutrition warranting a second overseas transfer.

CONCLUSION: The lack of sufficient mucosal surface followed by long time intestinal adaptation process is crucial in determining bowel functional capacity. Long time hospital stay and cost was reduced through a parental home healthcare management training scheme.

1. Introduction

We followed an individual case of short bowel syndrome (SBS) and tried to illustrate what children and parents had to go through in a developing country with a less financed healthcare system. These cases are rare, complex and often not considered a priority by healthcare planners in government. Medical records were gathered from 3 different hospitals in 2 different countries and this case was followed up from November 2013 to February 2016. Patient's weight and height were regularly monitored, while hypercaloric and hyperproteic diet were structured to improve nourishment and to gain weight. The patient initially was placed on total parenteral nutrition (TPN), then followed by parenteral nutrition (PN) combined with enteral feeding, and later progressed to just enteral feeding [1]. The complex and complicated nature of this case after enterectomy coupled with life threatening enterocolitis with salmonella infection 2 years after surgery were all part of the problems we encountered. Most of these patients die because of inadequate monitoring system due to the lack of healthcare funding. Parents had to seek extra medical help for their child from the West. In order to reduce hospital stay and cost, the parents were involved and trained on how to continue patient's healthcare man-

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Table 1
Laboratory test- short bowel syndrome (SBS).

CBC	Post-operation I	Pre-operation II	Post-operation III	1-day	2-day	3-day	8-day	12-day	15-day	19-day	21-day	Normal values
WBC	$25{,}34 \times 10^3$	$30{,}12 \times 10^3$	19,48	12,85	10,23	15,23	17,21	–	–	–	–	3.4–9.5 × 10(9)/L
RBC	$3{,}25 \times 10^6$	$3{,}56 \times 10^6$	$3{,}48 \times 10^6$		3,38	3,78	3,95	–	10^{12}	–	–	4.20–5.10 × 10(12)/L
HGB	10,8	10,1	10,5	10,3	9,8	11,5	11,1	11,4	9,5	9,7	11,4	12.0–14.0 g/dL
HCT	32,4	30,9	31,5	30,9	28,6	33,5	32,7	33,1	26,4	–	33,1	35.8–42.4%
PLT	6,89,000	9,00,000	6,57,000	6,23,000	524,000	5,38,000	5,40,000	–	–	–	–	150–450 × 10(9)/L
Neut%	91,2	83,9	91,7	84,1	84,2	–	44,4	–	–	–	–	1.50–8.50 × 10(9)/L
Lymph%	4,2	4,9	4,3	4,5	4,3	–	57	62	–	–	–	1.50–6.50 × 10(9)/L
Mono%	1,9	1,8	1,6	1,5	1,4	–	2,6	–	–	–	–	0.00–0.80 × 10(9)/L
Eo%	0,3	0,2	0,1	0,1	0,3	–	0,1	–	–	–	–	0.00–0.65 × 10(9)/L
Cl	113	112	112	114	113	–	111	112	–	–	112	98–106 mmol/L
ESR	90	100	80	84	80	75	100	70	65	40	30	3 to 13 mm/h
Creatinine	210	190	141	1,2	<0,10	8	6	4	N	1,21	<0,10	0.0–0.7 mg/dL
Uric acid	2,0	2,2	2,1	2,0	2,0	2,2	2,1	2,2	N	2,3	2,2	2.4–6.0 mg/dL
Urea	9.0	10,45	12,33	11,4	10.9	9,3	9,77	8,3	N	9.65	7,49	7 to 20 mg/dL (2.5–7.1 mmol/L)
Amylase	–	–	–	–	–	293	196	–	78	58	54	0–137 U/L
Lipase	–	–	–	–	–	156	83	–	–	–	–	12–70 U/L
ALT	45	58	108	99	N	200	42	58	41	60	58	7 to 56 units per liter
AST	36	39	43	41	N	52	N		N	56	45	10 to 40 units per liter
Blood sugar	92	89	130	110	116	120	123	98	134	127	90	100–125 mg/dL
Ferritin	–	58	314	–	–	–	–	58	27	–	58	11 to 307 nanograms per milliliter
Transferrin saturation	–	15	–	–	–	–	–	15	12	–	15	15%–45%
GGT	98	87	79	84	N	80	112	109	67	60	52–	0–45 U/L
PCR	58	65	1,83,83	150	86,6	120	N	3	3,2	0,5	2,4	<15 IU/mL
PCT	0,74	0,32	9,43	4,77	–	–	–	–	–	–	–	<0.15 ng/mL
Serum protein	54	60	22	4,5	1,66	N	N	6	2,3	2,5	1,9	64–83 g per liter (g/L)
Sideremia	24	53	1,3	30	29	65	32	40	44	42	56	60–180 microg/dl
Cholesterol	–	–	–	–	15	–	N	–	–	–	–	<200 mg/dl
Triglycerides	–	–	–	–	56	–	N	–	–	–	–	<150 mg/dl
Thyroid values	N	N	–	–	–	–	N	–	–	–	–	TSH = 0,4–4,5 TT4 = 50–160 FT4 = 10–24 FT3 = 4–8.3
Urine test	–	–		–		–	N	–	–	–	–	negative
Urine specific gravity	1013	1010	1010	–	1013	–	–	–	–	–	–	1.005–1.030
Leukocyte	–	–	+	–	20	–	–	–	–	–	–	negative
Ph	5,6	6	5	–	–	–	–	–	–	–	–	4.5–8
RBC (erythrocyte)	–	–	–	–	5280/microl	–	–	–	–	–	–	≤2 RBCs/hpf
Protein	–	–	–	–	+++	+	–	–	+	–	–	≤150 mg/d
Stool test	–	*Pseudomonas aeruginosa*	–	–	*Klebsiella pn.* +++ *Pseudomonas ae.*+	–	N	N		E.Coli+++ Klebsiella oxytoca+++ Enterococcus sp.+++	Salmonella	–
Calprotectin	–	–	–		–	–	<100	–	–	Negative	–	<50
Occult bleeding	–	–	–	–	–	–	–	+	–	+	–	Negative
Abdominal R-ray with barium	–	–	–	–	–	O short stoma area stenosis	No pneumoperitoneum No air fluid levels, intestinal dilatation (4,7 cm)→ SBS/volvulus	–	–	–	–	–

CT	Free abdominal fluid in douglas pouch and between the intestinal loops; Air fluid levels and intestinal distention present. No pneu-moperitoneum..	–	–	–	–	–	–	–	–	–	–	–
Abdominal ultrasound	–	–	–	–	Subileus/Ileus with sufficient fluid between the intestinal loops. No intraabdominal abscess	–	–	–	–	–	–	–
Irigoscopy (Barium enema)	–	–	–	–	Retrograde transit within the transverse colon and the ilenum, anastomotic stenosis was excluded	–	No important area of intestinal stenosis	–	–	–	–	–
Soft tissue ultrasound/Doppler venous ultrasound of the neck and subclavian vein	–	–	–	–	–	–	Very well visualized jugular veins and without obstruction	–	–	–	–	–
Blood gas				–		–	–					
Ph	7,24	7,32	7,30		7,41			7,32	7,31	7,36	7,24	7,35–7,45
pCO2	46	38	45		47			–	N	–	–	38–42 mmHg
pO2	25	49	67		65			–	N	–	–	75–100 mmHg
Na	140	133	136		133			–	N	–	–	135–144 mEq/L
K	6,3	4,9	5,6		4,1			–	N	–	–	3.6–5.2 mEq/L
Cl^{+2}	–	–	–		100			–	N	–	–	97–106 mEq/L
Mg^{+2}	–	–	–		0,45			–	N	–	–	1.3–2.3 mg/dl
Phosphate	–	–	–		2,75			–	N	–	–	3.2–5.7 mg/dl
Ca	0,91	0,99	1,2		1,89			1,5	N	–	–	8.8–10.4 mg/dl
HCO3	19,7	18,5	19,7		15			18,5	N	18,5	14,7	21–28 mmol/l
BE	7,7	6,4	5,8		–			−6,4	−6,7	−3,6	−12,7	−2 to +2 mEq/L
LAC	2,5	2,6	2,3		–			–	N	–	N	4.5 to 19.8 mg/dL (0.5–2.2 mmol/L)
HEM	9,9	–	–		–			–	N	–	–	13.5 to 17.5 g/dl
D-dimer	–	1,35	–	–	–	–	–	1,35	0,63	–	1,35	0–50 mg/L
APTT	–	41,1	–	–	–	–	–	41,1	41,2	–	41,1	70 to 120 s.
Vitamin D3	–	25,2	–	–	–	–	–	25,2	28,5	–	25,2	30–60 ng/ml
Vitamin B12	–	–	–	–	–	–	–	–	N	–	–	200–900 picograms per milliliter (pg/mL).

Table 2
Complication of SBS (case report).

I. Local	
INTESTINAL STOMA	PRIMARY ANASTOMOSIS
1. Peristomal skin excoriation	1. Wound infection (local abscess)
2. Granulomatosis	2. Intestinal obstruction (+)
3. Stomal retraction	3. Anastomotic leak,
4. Prolapse	4. Anastomotic stenosis (+)
5. Wound dehiscence	5. Anastomotic ischemia/necrosis (+)
6. Wound infection (local abscess)	6. Wound dehiscence
II. Systemic (intestinal stoma & primary anastomosis)	
1. Food intolerance (+)	13. Intestinal failure (+)
2. Vomiting (+)	14. Metabolic syndrome (+)
3. Bloating (+)	15. Enterocolitis (+)
4.Recurrent diarrhea (+)	16. Immune deficiency (+)
5. Gastro-intestinal reflux (+)	17. Weight loss (+)
6. Motility disturbances (+)	18. Hepatic, renal and circulatory insufficiency (+)
7. Failure to thrive (+)	19. Sepsis (+)
8.Malnutrition (+)	20. Anemia (+)
9.Malabsorption (+)	21. Hydrops and gallbladder sludge (+)
10. mMaldigestion (+)	22. Allergies
11. Dehydration (+)	23. Bone disease
12.Vitamin deficiency (+)	24. Drug toxicity (+)

Short, medium and long time prognosis are correlated with the underlined complications; local or systemic.

agement at home. The collaboration between our hospitals and the pediatric unit overseas was our last hope. Short bowel syndrome is defined as a patient requiring PN for more than 42 days due to gastrointestinal intolerance and residual small bowel length of less than 25% of predicted gestational age [2].

2. Case presentation

A 6 years old female patient arrived at our emergency unit in a serious condition: abdominal pain, fever, nausea, bilious vomiting, no passage of stool, abdominal distention, tachycardia and no history of surgery. Abdominal ultrasound and CT scan showed signs of intestinal obstruction. Emergency surgery was performed while common mesentery, intestinal obstruction due to volvulus with extensive ischemic necrosis of the ileocecocolic intestinal loop was discovered. Intestinal resection followed by side to side ileocolic anastomosis was performed with just 80 cm of the intestine left and without ileocecal valve. The post-operation status of patient was not encouraging. Pathology result; An ulcerative ileojejunocolic mucosa (Table 3). Blood pressure: 70/50 mmHg, urine output: 400 ml/day, gastric tube: 300 ml of blood strained fluid, central venous catheter was placed. Post-operation lab test; acidosis and anemia (Table 1). Parent requested transfer to a regional hospital after 24 h. Upon arrival the patient continued with total parenteral nutrition, antibiotics and antifungal agents. The patient remained in a severe catabolic state, metabolic acidosis, severe malabsorption and loss of nutrients, water and electrolytes through diarrhea (more than 10 stools/day), refusal of enteral feeding and signs of intestinal obstruction (seen on ultrasound and CT scan). Medial laparotomy was performed 7 days after, and ischemic necrosis of the anastomotic joint was discovered and resected followed by end to end anastomosis with just 70 cm of the intestine left. Meanwhile the increased bilious secretion was not encouraging. Despite all the effort to correct electrolyte and acid base imbalance, the patient developed intestinal failure, circulatory, renal and respiratory insufficiency. The antibiotics regimen were changed. The patient continued to show signs of paralytic ileus for several days, maldigestion, malabsorption, diarrhea, hydric-electrolyte imbalance and abdominal distention, and repeated ultrasound and CT scan showed signs of intestinal obstruction. Exploratory laparotomy was performed seven days after the second surgery. Intestinal adhesion was discovered and lysed; the anastomotic joint was intact, however, the patient's postoperative status did not improve. The patient remained febrile; the bilious aspirate (1000 ml/day), watery diarrhea (7–10 stools/day), metabolic imbalance, blood culture and peritoneal fluid culture were negative, stool culture was positive for pseudomonas aeruginosa, the intestinal failure continued. The pathology report after the second surgery demonstrated inflammatory changes, villous blunting (Table 4). Laboratory analysis showed anemia, reactive thrombocytes, inflammatory infectious syndrome (Table 1). The parents requested overseas transfer. The patient arrived overseas in a catabolic state (bilious vomiting, metabolic acidosis, pyrexia, confusion, speechlessness). Vitamin A, D, and E deficiency, selenium deficiency and axial hiatal hernia, gastro-esophageal reflux and hepatomegaly were also present. An upper GI study showed good contrast passage with 2 suspicious areas of possible stenosis. Swab showed multi-resistant germ (Klebsiella pneumonia) requiring antibiotics. PN and balancing of the electrolyte levels, fresh frozen plasma and albumin helped achieve anabolism; The patient was mobilized with the help of physical therapy. However, the patient continued to lose large amount of bilious fluid requiring further investigation by upper and lower endoscopy (gastroduodenoscopy and sigmoidoscopy) and biopsy. Visceral hernia and intestinal adhesion were solved through a medial laparotomy followed by peritoneal lavage (for colon and abdominal wall abscess), while two stomas (jejunostomy and colostomy) were placed at the stenotic sites to improve nutrition. The stomas were closed after 6 weeks. Correlated therapeutic management helped to reduce gastric secretion, improve digestive efficiency (against diarrhea, antibiotics, eubiotics etc). Bicarbonate, potassium, calcium and vitamin D deficiencies were corrected. Enteral feeding was gradually introduced and tolerated (dietary food with low lactose and fructose content). The parent's were trained to continue PN at home especially at night, smofkabien peripheral emulsion infusion for 18 months. Patient's condition improved and stool frequency reduced

Table 3
Pathology result (First Surgery).

MACROSCOPIC ASPECT	MICROSCOPIC ASPECT
COLOR: Dark pinkish jejunum and ileum, hemorrhagic serosa and leucorrhea (greenish intraluminal bowel content). Dark pinkish subileal ganglion group.	An ulcerative ileojejunocolic mucosa, with intestinal villi and ulcerative lesions. Lymphoid follicles, lymphocytic infiltration of submucosa and mucosa, intestinal dilatation, thrombotic vessels of the submucosa, and intestinal wall lesions mimicking enterocolitis follicles overlapping the ischemic lesions.
SIZE: 80 cm ileum and jejunum length, 4/3 cm lymphatic ganglion at ileocolic angle. Subileal ganglion group of 0.5–2 cm. Appendix of 10 cm and an omentum of about 12/15 cm.	Approximately 16 mesenteric lymphatic ganglions included in the block had the aspect of chronic lymphadenitis and with areas of hemorrhagic and thrombotic patches. Also present was thrombotic mesenteric vessels and an omentum without polymorphic modification.

Table 4
Pathology result (Second Surgery).

MACROSCOPIC ASPECT	MICROSCOPIC ASPECT
COLOR: Intestinal lumen; ash color with brownish elastic areas.	Increased villous height, crypt depth, intestinal epithelial hyperplasia, intestinal fragments with large surface of thrombotic extravasation.
SIZE: Intestinal tubular fragment of about 8.3 cm. Intestinal lumen of about 1.6 cm.	Hyperemic vessels with different stages of thrombus and highly infiltrated polymorphic inflammation (Ischemic modification).

to just 3 per day (no more watery diarrhea). Laboratory tests results improved. The patient was discharged with a right Broviac catheter. Good neuropsychological development, Blood gas follow-up was scheduled every fortnight. The patient went back to school.

Two years after surgery the patient was readmitted with the following symptoms: fever, bilious vomiting, watery diarrhea (9 stools/day), she was later transferred to a regional hospital as her condition worsened. The patient was afebrile, but in a serious condition, pale, thin, weak, whitish tongue, pharyngeal congestion, acetone breath, asthenic thorax, heart rate of 120 b/min, no pulmonary rales, painless palpable abdomen, watery diarrhea (9 stools/day), vomiting and no meningeal irritation, signs of dehydration > 15%, prerenal azotemia, hepatocytolysis (hydric-electrolytic imbalance, metabolic acidosis, no sign of meningeal irritation), carential syndrome (hyposideremia, hypocalcemia). Growth retardation and weight loss (from 23 kg to 18 kg) due to metabolic imbalance and prerenal insufficiency was also observed. The hydric-electrolytic imbalance was corrected, antibiotics, antiemetic, antisecretory, antidiarrheic and pain medications were administered. Ultrasound and x-ray of the thorax and abdomen were normal. Laboratory analysis: hepatocytolysis, acute gastroenterocolitis, renal insufficiency (prerenal type), hypopotasemia (moderate type), respiratory compensation metabolic acidosis, SBS, urinary tract infection, mild malnutrition, lymphopenia, iron deficiency (Table 1). Patient's health worsened so parents insisted on overseas transfer. The patient was admitted overseas for gastro-intestinal infection (enterocolitis), watery diarrhea, vomiting, metabolic syndrome, consciousness disturbances (confusion, speechlessness and hypotonic state). The amount of parenteral nutrition administered in patients country of origin was considered inadequate thus worsening the patient's health. Lab analysis; acute phase reactants on the rise, acute renal insufficiency (hematuria and proteinuria), cytolysis (Table 1). Abdominal ultrasound; renal changes due to toxic infectious nephritis probably caused by salmonella infection, SBS, moderate hepatosplenomegaly, pancreatic edema, moderate intraperitoneal fluid, hydrops and gallbladder sludge. Ultrasound of the soft tissues of the neck; no sign of obstruction. Abdominal x-ray showed intestinal dilatation (Table 1). Salmonella was confirmed in stool and antibiotics regimen was changed. HCO3 and potassium were administered, electrolyte imbalance was corrected and hydration was increased. Patient tolerated enteral feeding gradually (no more abdominal pain) and was discharged with port catheter, acid base values normalized. Complication of SBS: compared the difference between local and systemic intestinal stoma and primary anastomosis (Table 2).

3. Discussion

The term short bowel syndrome is a malabsorption state that occurs after the resection of a large portion of the small intestine [2]. This can also mean the need for prolonged PN as a result of intestinal failure. These patients require long term hospitalization and PN [3]. The degree of malnutrition depends on the remaining intestinal length, which is crucial in determining bowel functional capacity. With just 70 cm of the intestine left and no ileocecal sphincter capacity, severe malnutrition, watery diarrhea and metabolic acidosis in the acute phase of the disease was life threatening. This was followed by the adaptation phase from 2 to 4 days after bowel resection, which lasted for months. The second surgery is debatable as some experts argue that this is a paralytic ileus state often seen in the adaptation phase, some surgeons may prefer intestinal stoma instead of anastomosis at this stage. Stoma versus primary anastomosis is also debatable, though intestinal stoma had a better result in this case. Strategies that slow intestinal transit, improve peristaltic function, or enhance mucosal absorption function each has application in the management of SBS [4]. The last phase is the maintenance phase, here the absorption capacity of the intestine is at its maximum. The optimal goal in the management of SBS is to gain full enteral autonomy at the optimal time [5]. Long time TPN can lead to intestinal failure associated liver disease [6]. As enteral feeding was tolerated TPN was gradually weaned. Fluid and electrolyte imbalance were corrected during this process of bowel adaptation. To avoid metabolic bone disease calcium, vitamin D and alkaline phosphate levels were periodically checked. Soluble vitamins (A, D, E, K), and vitamin B12 were monitored closely for deficiency. Food with less osmotic load such as protein and fat helped provide additional stimulant for intestinal adaptation. Adequate PN and later enteral nutrition helped to reduce diarrhea from 10 to 3 stools per day. For maintenance of a good hydration and electrolyte balance normal or half saline, potassium, sodium bicarbonate were supplemented. Continuous sip of oral rehydration solution (ORS) through the day helped maintain a positive fluid balance. Extra intravenous fluid was needed when the enteral route was unable to meet the patient's nutritional need. Small bowel biopsy in SBS always demonstrate inflammatory changes, villous blunting, while the present of adherent or intracellular bacteria proves the presence of short bowel bacterial overgrowth [7]. Catheter related blood stream infection is another source of infection in SBS [8]. Bacterial translocation has been noted in animal models, but data supporting its occurrence in human is limited [9]. Bacterial overgrowth and impaired mucosal immunity puts SBS patients at risk of bacterial translocation [7]. Intestinal bacterial growth is controlled by many mechanisms such as: gastric acidity, pancreatic enzyme activity, enterocyte turnover, normal peristaltic activity and the presence of ileocecal sphincter [10]. These factors are altered in SBS Patients, so bowel dilatation with reduced peristalsis may develop adaptation mechanism to improve enteral adaptation, these factors on the other hand may favor bacterial overgrowth by reducing bowel ability to expel microorganism. Intestinal endotoxine increases in children without ileocecal valve and can also impair liver function by decreasing body's bactericidal defense mechanism [7]. Elevated d-lactic is responsible for acidosis, this condition is associated with confusion, speech disturbance, and severe metabolic acidosis seen in this case. Growth retardation is as a result of severe metabolic disturbances and impaired immune system.

4. Conclusion

The high cost of managing SBS patients in the developing countries and the complicated nature of this disease limits parent's access to quality healthcare for their children. The main goal in SBS is to achieve intestinal autonomy at the optimal time because prolonged time of intestinal failure can lead to more complications or death. Reduce hospital stay and avoid hospital acquired infections. Reduce hospital cost through parental home healthcare management training.

Compliance with Ethical Standards

This article does not contain any studies with human participants or animals performed by any of the authors. The authors declare that they have no sources of funding. Informed consent was obtained from parents for this article's publication. This work has been reported in line with the CARE criteria and the paper above meets the CARE guidelines: consensus-based clinical case report guideline development [11].

Conflicts of interest

Authors declares no conflict of interest.

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Ethical approval

There is no ethical approval needed for this case.

Consent

Informed consent was obtained from all individual participants (parents) included in the study.

Author contribution

Prof. Dr Eugen Boia. Coodinator.
Dr. Henry Osakwe. Concept and designer.
Dr. Cristina Dragomir Data gathering.
Dr. Cristan Nicolescu Data analysis.

Guarantor

Dr. Henry Osakwe.

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References

[1] S.F.A. Dorney, M.E. Ament, W.E. Berquist, Improved survival in very short bowel of infancy with use of long-term parenteral nutrirtion, J. Pediatr. 106 (1985) 521.
[2] S.J. O'Keefe, A.L. Buchman, Short bowel syndrome and intestinal failure: consensus definitions and overview, Clin. Gastroenterol. Hepatol. 4 (2006) 6–10.
[3] C. Torres, J.A. Vanderhoof, 2004. SBS: clinical enteral nutrition and tube feeding Fouth ed 39. 451–463.
[4] L. D'Antiga, A. Dhawan, M. Davenport, Intestinal absorption and permeability in pediatric SBS: a pilot study, J. Pediatr. Gastroenterol. Nutr. 29 (1999) 588–593.
[5] D. Sigalet, D. Boctor, M. Brindle, Elements of successful intestinal rehabilitation, J. Pediatr. Surg. 46 (2011) 150–156.
[6] D.A. Kelly, Intestinal failure-associated liver disease: what do we know today? Gastroenterology 130 (Suppl 1) (2006) 570–577, 2.
[7] S.S. Kaufman, C.A. Loseke, J.V. Lupo, Influence of bacterial overgrowth and intestinal inflammation of parenteral nutrition in children with SBS, J.Pediatr. 131 (1997) 356–361.
[8] C.R. Cole, R. Frem, B. Schmotzer, The rate of bloodstream infection is high in infants with SBS: relationship with SBBO, enteral feeding, and inflammatory and immune responses, J. Pediatr. 156 (2010) 941–947.
[9] R.D. Berg, A.W. Garlington, Translocation of certain indigenous bacteria from the gastrointestinal tract to the mesenteric lymph nodes and other organs in a gnotobiotic mouse model, Infect. Immune. 23 (1979) 403–411.
[10] T.R.M. Ziegla, M.E. Evans, C. Fernandez-Estivariz, Tropic and cytoprotective nutrition for intestinal adaptation mucosal repair, and barrier function, Annu. Rev. Nutr. 23 (2003) 329–361.
[11] J. Gagnier, G. Kienle, D.G. Altman, D. Moher, H. Sox, D.S. Riley, et al., J. Clin. Epidemiol. 67 (1) (2016) 46–51.