Síndrome do Intestino Curto: Uma Entidade de Gestão Complexa - A Propósito de um Caso Clínico e Revisão da Literatura

Short Bowel Syndrome: A Complex Management Condition – A Case Report and Literature Review

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Resumo:

A síndrome do intestino curto (SIC) é caracterizada por um estado de malabsorção de macro e micronutrientes que habitualmente se desenvolve após resseção de intestino delgado. Parece faltar uma correlação entre a quantidade de intestino ressecado e os sintomas desenvolvidos com a adaptação da estrutura e função intestinal. Reportamos o caso de um homem de 59 anos, submetido a resseção de 100 cm de intestino delgado tendo posteriormente desenvolvido síndrome de má absorção. Dos 95 dias de internamento hospitalar, várias complicações associadas à SIC ocorreram e de difícil gestão. Apesar de ser expectável um mau prognóstico, o doente apresentou melhoria significativa do estado nutricional após nutrição entérica e parentérica. À data de alta, apresentava um ganho ponderal de 9 kg. A SIC é uma patologia pouco reportada na literatura dado tratar-se de uma entidade complexa. Pretendemos com este caso reportar todo o processo de recuperação do doente e rever a fisiopatologia desta entidade

Palavras-chave: Nutrição Entérica; Nutrição Parentérica Pseudo-Obstrução Intestinal/tratamento.

Abstract:

Short bowel syndrome (SBS) is characterized by a state of macro and micronutrient malabsorption which usually develops after small bowel resection. There is a weakly correlation between the amount of resected intestine and the clinical symptoms with the adaptation of bowel structure and function. We report a case of a 59-year-old man, who underwent a 100 cm small bowel resection developing malabsorption syndrome afterwards. Throughout the 95 days spent in hospital many complications of SBS with complex management arose. Although the patient was expected to have a poor prognosis, his nutritional status improved significantly through continuous parenteral and enteral nutrition. At discharge he had recovered carrying a 9 kg weight gain. SBS is a non-common disease reported in the literature as being a complex entity. The aim of this case is to report the process of the patient´s recovery and to provide a review of the physiopathology of this entity.

Keywords: Enteral Nutrition; Parenteral Nutrition; Short Bowel Syndrome/therapy.

Introduction

Short bowel syndrome (SBS) is a rare disorder mainly caused by extensive intestinal surgical resection of the small intestine leading to a severe reduction of the absorptive intestinal surface. Hence, this entity can lead to severe and life-threatening complications. The management of SBS is complex and challenging and it demands a dedicated multidisciplinary team to overcome the morbimortality.[1]

Case Report

A 59-year-old white man was submitted to cephalic duodenopancreatectomy in addition to a 100 cm small bowel resection owing to a later rejected pancreatic neoplasia suspicion. Subsequently, he developed weight loss, anorexia and postprandial diarrhoea with undigested foods. These symptoms were interpreted as malabsorption syndrome (MS) and throughout the following months, several hospitalizations were necessary requiring total parenteral nutrition (TPN). Within the several endoscopic procedures with biopsies performed, no abnormalities were revealed. Eight months after, he was presented to our hospital with his aggravated clinical condition, with a weight loss of 38 kg and bedridden.

On admission, body weight was 36 kg with a body mass index of 12.0 kg/m². He was noticeably pale, cachexic, with

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mucosal dryness, multiple bruises and bilateral pretibial oedema. He had no travel history, was not under any medications and there was not a dietary trigger. He undertook full laboratory tests (Table 1), including a stool test compatible with osmotic diarrhoea. Endoscopic study and computed tomography (CT) enterography were normal. There was no symptomatic improvement of dejections after ertapenem 7-day administration due to urinary tract infection. As diarrhoea stopped with enteral nutrition (EN) suspension, SBS was assumed. Once our patient was severely malnourished and it was not possible to use EN alone as he had severe diarrhoea, he was supported through low dose TPN. A slow increase in nutritional intake along with close monitoring of serum electrolytes and measurement of thiamine was made. In parallel with TPN, the early addition of small amounts of low-fat diet was started, along with multi-vitamin supplementation including folic acid and thiamine. After 48 hours full dose TPN was achieved, our patient presented

Table 1: Full laboratory results.

	Results	Reference value
Hemogram		
Hemoglobin	5.6 g/dL	13-18 g/dL
MCV	108 fL	87-103 fL
MCH	34.3 pg	27-35 pg
Leukocytes	6.3 x109/L	4-11 x 109/L
Platelets		
Platelet count	83 x 10^9/L	150-400 x 10^9/L
ARC	0.126 x 10<<12>>/L	0.025-0.090 x 10<<12>>/L
ESR	7 mm/1h	0-20 mm/1h
Iron study		
Serum iron	54 µg/dL	53-167 µg/dL
Serum ferritin	2463.3 ng/mL	20.0-250.0 ng/mL
Serum transferrin	< 75 mg/dL	200-360 mg/dL
Lipid panel		
Total cholesterol	69 mg/dL	< 200 mg/dL
HDL cholesterol	21 mg/dL	> 60 mg/dL
Triglycerides	110 mg/dL	< 150 mg/dL
Liver tests		
Albumin	12,9 g/L	38-51 g/L
AST	51 U/L	10-37 U/L
ALT	54 U/L	10-37 U/L
ALP	185 U/L	30-120 U/L
GGT	73 U/L	10-49 U/L
Total bilirrubin	0.50 mg/dL	< 1.20 mg/dL
Direct bilirrubin	0.23 mg/dL	< 0.40 mg/dL
LDH	276 U/L	135-225 U/L
Pancreatic enzymes		
Serum lípase	< 3 U/L	7-60 U/L
Serum amílase	14 U/L	22-80 U/L
Coagulation study		
PT	18.9 s	10.1-13.6 s
aPTT	44.9 s	24.2-36.4 s
Ionogram		
Sodium	138 mEq/L	135-145 mEq/L
Potassium	2.7 mEq/L	3.5-5.1 mEq/L
Magnesium	1.48 mEq/L	1.55-2.05 mEq/L
Phosphate	2.8 mg/dL	2.7-4.5 mg/dL
Ionized calcium	2.2 mEq/L	2.26-2.64 mEq/L

	Results	Reference value
Renal function		
Serum Creatinine	0.54 mg/dL	0.67-1.17 mg/dL
Serum Urea	25 mg/dL	10-50 mg/dL
CPR	15.5 mg/L	< 3.0 mg/L
Vitamin B12	1611 pg/mL	187-883 pg/mL
Folic Acid	2.4 ng/mL	2.2-17.5 ng/mL
Vitamin D	27 ng/mL	> 30 ng/mL
Thyroid test		
TSH	3.39 µUI/mL	0.35-4.94 µUI/mL
T4	0.57 ng/dL	0.70-1.48 ng/dL
Stool test		
Presence of leukocytes in the *stool*	Negative	
Potassium	51 mEq/L	
Sodium	16 mEq/L	
Osmotic gap	156 mOsm/Kg	
Detection of fat in the stool	Negative[a]	
Detection of reducing substances	Positive	
Stool Culture	Negative for *Salmonella, Shigella, Clostridioides difficile, Campylobacter*	
Parasitology test	Negative for parasites	
Viral markers		
HBV		
HBsAg	Non reactive	
Anti- HBc	Nonreactive	
Anti- HBs	Positive (640.09 UI/L)	
HCV		
Anti-HCV	Nonreactive	
HIV	Nonreactive	
Treponemal tests	Negative	

ALP: alkaline phosphatase; ALT: alanine transaminase; anti-HBc - hepatitis B core antibody; anti-HBs - Hepatitis B surface antibody; anti-HCV – hepatitis C antibody; aPTT: activated partial thromboplastin; ARC: absolute reticulocyte count; AST: aspartate transaminase, CPR: C-reactive protein; ESR: erythrocyte sedimentation rate; HDL: high-density lipoprotein, HIV - human immunodeficiency vírus; GGT: gamma-glutamyltransferase; HBsAg - Hepatitis B surface antigen; LDH: lactate dehydrogenase; MCH- mean corpuscular hemoglobina; MCV: mean corpuscular volume; PT - prothrombin time; T4: thyroxine; TSH: thyroid-stimulating hormone a Note that when the patient undertook this sample he was in food break.

altered mental status with visual hallucination and dysmetria. A cranioencephalic-CT scan was performed excluding any abnormality. Only hypomagnesemia (1.13 mEq/L) was identified on the biochemical investigation. Hence, a clinical diagnosis of Wernicke encephalopathy secondary to re--feeding syndrome (RFS) was done. Treatment with thiamine (500 mg three times/day) and reduction of TPN dose was instituted with full recovery of the neurological symptoms one week later. At that time, every two days, the TPN dose was augmented until a full dose was achieved three weeks after. Posteriorly, a TPN dose achieving nutritional needs was administered with no complications.

During hospital stay, the patient started to tolerate larger amounts of EN supplementation using polymeric and hypolipidic enteral formula with medium-chain triglycerides (MCT), soluble fibres and maltodextrin. As there was a positive test for reducing substances, corn starch diluted in water was included in his diet. A slow but progressive clinical improvement started settling, with a total weight gain of 9 kg and faecal consistency improvement, allowing TPN suspension at day 75.
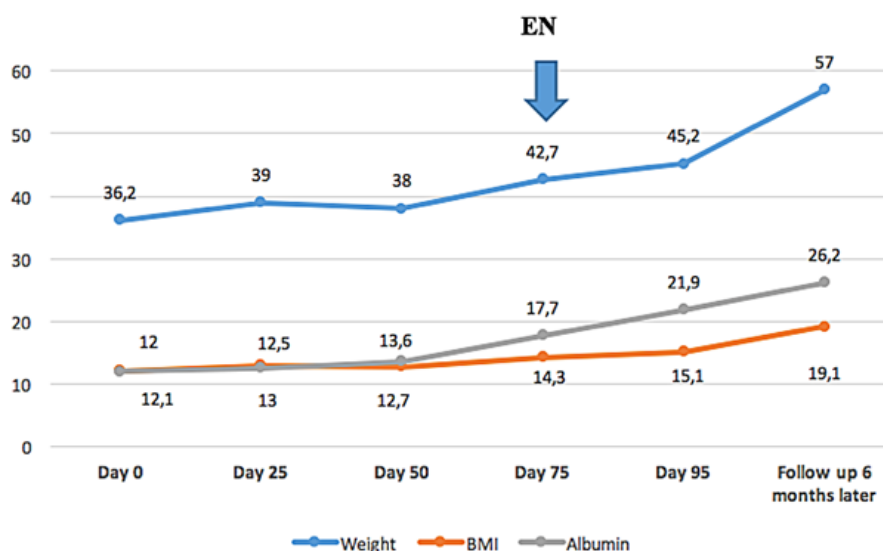
When discharged, on hospital day 95, the patient weighted 45.2 kg, he was able to walk and displayed improvement of clinical and biochemical findings. He was consuming a high energy, low-fat diet with food supplements adjusted to his preferences (including eggs white with sugar to improve serum albumin), and enteral nutritional supplements with MCT. To prevent clinical relapse and ensure continuous weight gain at home, a personalized nutritional plan was elaborated consisting of 2500 kcal, distributed into 20% of protein, 20% of lipids using MCT oil and 60% of carbs, in six meals/day, supplementation with maltodextrin (285 kcal/day) to achieve the energetic needs, and soluble fibre to regulate bowel function. Table 2 describes the parenteral and enteral support over time in more detail (kilocalorie along with protein and non-protein intake dosage). At the patient's six-month follow-up visit he had put on a 12 kg weight gain (Fig. 1 and Table 3) and was maintaining a good nutritional state. One year after, his weight was steady, he was able to drive and no intercurrences were registered.

Discussion

SBS is a disabling malabsorptive condition whose definition is more accurately based upon intestinal dysfunction, such as a significant malabsorption of both macro and micronutrients. In its initial phases, after bowel resection, there are four main defining determinants: intestinal resection length; ileum and ileocecal valve losses; total/partial colon loss; continuity/discontinuity of the intestines.[2] The ileum exhibits a more robust adaptive response than the duodenum and jejunum. However, when these sections are involved as it happened in our case, it typically results in a more pronounced hypergastrinemia with iron, folate and other micronutrient deficiencies.[3] This entity is the most common cause of intestinal failure which is defined as "a reduction of gut function below the minimum necessary for the absorption of macronutrients and/or water and electrolytes, such that intravenous supplementation is required to maintain health and/or growth".[4] After intestinal resection, the intestinal adaptation begins within 24-48 hours by means of a compensatory process in the remaining bowel where structural and functional changes take place. This adjustment can take up to 2-3 years.[5,6]

Pertaining to management, TPN should be started once the patient is hemodynamically stable to prevent malnutrition, weight loss and negative nitrogen balance. Long

Figure 1: The patient´s weight, BMI and albumin over the time. BMI, body mass index (kg/m2), EN - enteral nutrition initiation.

Table 2: Parenteral and enteral support over time (Kcal along with protein and non-protein intake dosage).

Day	Oral diet	Enteral nutrition - oral suppelements	Parenteral nutrition
Day 2	Bland diet (<500 kcal)	X	500 mL | 350 kcal | 2 gN | 10 kcal/kg
Day 3	Bland diet (<500 kcal)	X	1000 mL | 650 kcal | 5 gN | 18 kcal/kg
Day 5	pasty diet without fiber or residues (2000 kcal)	oligomeric nutritional supplement (200 kcal | 9 g proteins) + 50 g soluble fiber	1000 mL | 650 kcal | 5 gN | 18 kcal/kg
Day 7	pasty diet without fiber or residues (2000 kcal) - patient refuse to eat almost diet - eat <25%	oligomeric nutritional supplement (200 kcal | 9 g prot) + 50 g soluble fiber + MCT oil + maize starch (40 g/day)	1320 mL | 901 kcal | 6.7 gN | 25 kcal/kg
Day 10	Keeps the same	Keeps the same	493 mL | 518 kcal | 2.5 gN | 15 kcal/kg
Day 13	Plus appetite - eat almost 50%	Keeps the same	840 mL | 875 kcal | 4.5 gN | 25 kcal/kg
Day 18	Keeps the same	Keeps the same	(After re-feeding syndrome) 333 mL | 350 kcal | 2.5 gN | 10.5 kcal/kg
Day 20	Keeps the same	Keeps the same	492 mL | 518 kcal | 3.7 gN | 15 kcal/kg
Day 23	Keeps the same	Keeps the same	492 mL | 518 kcal | 3.7 gN | 15 kcal/kg
Day 26	Keeps the same	Keeps the same	666 mL | 700 kcal | 5 gN | 20 kcal/kg
Day 30	Keeps the same	oligomeric nutritional supplement 2/day (400 kcal, 18 g P)	666 mL | 700 kcal | 5 gN | 20 kcal/kg
Day 35	Strats soft diet (2330 kcal)	Spots maize starch	666 mL | 700 kcal | 5 gN | 20 kcal/kg
Day 40	The same diet but without egg (hyperphosphatemia)	oligomeric nutritional supplement 3/day (600 kcal, 27 g P) + MCT oil (280 kcal) + sweet food/desserts/cake	999 mL | 1050 kcal | 7.5 gN | 30 kcal/kg
Day 47	Soft diet low fat (2000 kcal)	Keeps the same	1500 mL | 1600 kcal | 12 gN | 45 kcal/kg
Day 54 till Day 68	Keeps the same	Keeps the same	Keeps the same
Day 70	Keeps the same	Keeps the same	Peripheral access - 1300 kcal | 9.8 gN | 37 kcal/kg
Day 75 till Day 95	Keeps the same	Keeps the same	Stops parenteral nutrition
Hospital discharge (food plan for home)	2500 kcal. (20%P (125 g), 20% L (55 g), 60% Carbs (375 g))	50 g soluble fiber + maltodextrine (75 g/285 kcal)	-

g – grams; gN - quantity of nitrogen in grams; Kcal – kilocalories; Kg – kilogram; L – lipis; MCT – medium-chain triglycerides; mL- mililiters; P – proteins.

term necessity depends on the four factors abovementio-ned. Despite TPN being crucial for the initial survival, EN should be started as soon as possible and be slowly increa-sed depending on the stool output (primary determinant of EN tolerance). The European Society for Clinical Nutrition and Metabolism (ESPEN) guidelines state that early initia-tion of EN provides a better distribution and maximum ex-posure of the available intestinal surface-area nutrients. The continuous intake of dietary nutrients is essential to stimula-te remodelling not only via direct contact with the intestinal mucosa, but also due to the release of intestinal hormo-nes.[7,8] Moreover, while a patient is under PN or EN, monito-ring fluid balance and electrolytes are important measures.[7] Our patient maintained TPN until day 75, with unsuccess-ful weaning due to fluid/electrolyte abnormalities, food refu-sal, no decrease of daily dejections and other SBS related

Table 3: Progression of laboratory findings of the patient.

Biochemical parameters	Normal range	Day 10	Day 25	Day 40	Day 50	Day 55	Day 75	Day 95	6 months follow-up
Ionized calcium (mEq/L)	2.26-2.64	2.01	2.3	2.4	2.3	-	-	2.4	-
Inorganic phosphorus (mg/dL)	2.7-4.5	2.6	3.2	4.6	4.4	5.3	4.9	3.8	3.1
Serum potassium, (mEq/L)	3.5-5.1	2.8	4.6	5.6	4.7	5.3	3.7	3.6	4.6
Serum magnesium, (mEq/L)	1.55-2.05	1.4	1.5	1.7	-	-	-	1.51	1.7
Hemoglobin, (g/dL)	13.0-18.0	9.5	7.8	7.4	7.7	8.1	9.1	9.9	12.2
Platelet count (x10^9/L)	150-400	93	171	258	285	256	254	182	175
PT (s)	10,.1-13.6	13.8	14.1	-	-	13.3	-	-	-
aPTT (s)	24.2-36.4	36.9	34.7	-	-	30.3	-	-	-
AST (U/L)	10-37	65	31	-	-	18	-	16	41
ALT (U/L)	10 - 37	64	34	-	-	20	-	22	-
GGT (U/L)	10-49	126	170	-	-	45	-	-	-
ALP (U/L)	30 - 120	249	242	-	-	60	-	-	-

Legend: ALP: alkaline phosphatase; ALT: alanine transaminase; aPTT: activated partial thromboplastin; AST: aspartate transaminase; GGT: gamma-glutamyltransferase; PT - prothrombin time

complications ahead-mentioned. Also, electrolytes and micronutrients abnormalities were corrected after targeted supplementation for each deficit. Serum levels were closely monitored at regular intervals.

When a transition to oral feeding is possible it should be done in a slow and stepwise form. If colon continuity is maintained, an hypolipidemic and rich complex carbohydrates diet is recommended.[3] However, it is recommended that apart from the MCT supplementation, fat should be restricted to prevent steatorrhoea.[7] In our case, MCT were preferred once they are absorbed through both small and large intestine and do not require pancreatic lipolysis.[9] On the other hand, proteins typically do not entail changes in diet as nitrogen absorption is less affected by the decrease of absorptive surface in SBS.[9] As recommended by ESPEN guidelines, when our patient started oral protein, the intake was divided over the day. Eggs, poultry, fish and lean minced meat are also good choices for oral provision of protein.[7]

SBS originate numerous acute/chronic complications. The real challenge comes from managing the multiplicity of complications.[10] Systemic complications such as osteoporosis, nephrolithiasis, hepatic cholestasis and liver disease as well as D-lactic acidosis are not infrequent.[10,11]

RFS is a well described but often forgotten condition. It is a potentially fatal complication in malnourished patients receiving artificial nutrition. The underlying factors are metabolic and hormonal changes caused by rapid refeeding.[11] It is characterized by fluid and electrolyte disturbances caused by a shift of electrolytes and vitamins from the extra-cellular space after re-feeding with glucose and aminoacids. This leads to an increased cellular uptake and dramatic depletion of phosphate, potassium, magnesium and thiamine serum levels. Hypophosphatemia is the predominant alteration.[12] Diagnosis is made when clinicians come across a history of severe malnutrition, re-feeding, clinical symptoms and electrolyte abnormalities. In this case, the patient exhibited an altered mental state and associated hypomagnesemia which was coincident with an increased TPN dosage. Few case reports of SBS presenting with RFS are published in the literature. Xie Y *et al* and Satarpia L *et al* reported their experience in patients who were under TPN in whom Wernicke´s encephalopathy developed after PN dose was increased. The biochemical findings seen were slightly hypokalaemia (3.4 mEq/L) and severe hypophosphatemia (0.05 mmol/L). In both cases, after thiamine supplementation, a complete resolution of this entity was seen.[12,13] These

reports not only demonstrate the variety of biochemical findings that may exist but also alert physicians to the risk of incorrect management of TPN and need for regular supplementation of vitamins (particularly thiamine).

After confirming that the patient's oral ingestion was stable, we have also recommended avoiding high-fat diet and maintaining a low-oxalate diet in order to prevent kidney stone formation. Calcium oxalate stones are an unpleasant complication and have been reported in 60% of patients.[1,14]

Diarrhoea in SBS is of multifactorial aetiology. In our case, diarrhoea was likely triggered by the absence/reduction of absorptive mucosal area, decrease in intestinal transit time and gastric hypersecretion due to parietal cell hyperplasia and hypergastrinemia which denatures pancreatic enzymes and bile salts and contributes to maldigestion/malabsorption.[1] The ESPEN guidelines recommend the use of anti-diarrhoeal medication and H2-receptor antagonists/proton pump inhibitors to reduce the losses of water and electrolytes, faecal wet weight and to minimize the symptoms and consequences of diarrhoea.[8] Antimotility therapy includes: codeine, loperamide, tincture of opium, somatostatin or octreotide and cholestyramine.[7] In our case, we opted to treated our patient with 2 mg daily dosage of loperamide – to reduce intestinal motility and prolong transit time – as well as fibres and intravenous proton pump inhibitors (pantoprazole 40 mg) to reduce sodium excretion.

Nevertheless, it should be noted that small intestine bacterial overgrowth is able to yield these findings. Hence, this hypothesis was excluded after a negative stool test and the absence of clinical improvement with antibiotics. However, it is important to report that SBS patients who have motility disorders and who suffer from symptoms of bacterial overgrowth benefit from antibiotic treatment.[8]

A decreased fat-soluble vitamin absorption was assumed because these vitamins' absorption occurs in stomach and duodenum15 and our patient had coagulopathy. Although vitamin K deficiency is uncommon in patients with intact colon, its deficiency is frequent in patients who have taken broad-spectrum antibiotics recently, as it happened in the eight months after surgery.[16,17] After supplementation of 10 mg weekly, for 6 weeks, coagulopathy resolved.

Each nutritional program should be individualized and clinicians ought to be aware of the risks and complications, whether the nutritional support is enteral/parenteral. Although the initial survival depends on TPN, it should be followed by an aggressive attempt to use EN. Despite the few reports described, when a multidisciplinary team is present as it was in our case, a successful management with remarkable recovery is seen, even when it is not expected.[18-20] Special attention must be paid towards early correction of macro and micronutrients before full dose nutritional support can be initiated.

We consider this case significant due to the scarcity of cases reported in the literature perchance justified by this entity's intricacy. This case not only epitomizes the importance of appropriate nutrition care but also highlights the importance of how a close cooperation between the medical and nutrition support can notably contribute to a favourable prognosis.

In conclusion, nutrition therapy is crucial for the successful management of SBS. Patients with this entity require lifelong monitoring and management goals frequently change over time. Despite the complexity and difficult control, treatment becomes challenging and requires patience, persistence, attention to details and appropriate multidisciplinary intervention. We expect this case could be used as an example of the management for SBS and how difficult could be to treat it. ■

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