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WORD COUNT	4376	TIME SUBMITTED	25-AUG-2022 01:35PM
		PAPER ID	89846277

Name of Journal: *World Journal of Clinical Cases*
Manuscript NO: 78604
Manuscript Type: MINIREVIEWS

A concise review on short bowel syndrome

Lakkasani S *et al*. A Concise Review on Short Bowel Syndrome

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Abstract

Adults have approximately 20 feet of the small intestine, the primary site for absorbing essential nutrients and water. Resection of the intestine for any medical reason may result in short bowel syndrome (SBS), leading to loss of major absorptive surface area and resulting in various malabsorption and motility disorders. The mainstay of treatment is personalized close dietary management. Here we present SBS with its pathophysiology and different nutritional management options available. The central perspective of this paper is to give a concise review of SBS and the treatment options available, along with how proper nutrition can solve major dietary issues in SBS and help patients recover faster.

Key Words: Short bowel; Small bowel resection; Malabsorption; Nutrition; Intestinal transplantation

Lakkasani S, Seth D, Khokhar I, Touza M, Dacosta TJ. A concise review on short bowel syndrome. *World J Clin Cases* 2022; In press

Core Tip: This is a very informative review about the etiology, pathophysiology and management of short bowel syndrome with newer treatment options based on extensive literature review and expert opinion.

INTRODUCTION

Malabsorptive disorders may result from the loss of bowel mass secondary to surgical resection of the small intestine. One of the rare disorders called short bowel syndrome (SBS) (< 180-200 cm of remaining small bowel) can occur[1,2]. SBS can also be caused by inflammatory bowel syndrome, vascular diseases, neoplasms, *etc.* The condition may present clinically depending mainly upon the remaining anatomical portion of the intestine and its related function, which makes the clinical representation of the disease variable[3].

Since the length and function of the intestine are compromised, it leads to loss of nutrients, fluid, and weight loss due to malabsorption. Symptoms of electrolyte disturbances and deficiencies of micronutrients and vitamins occur clinically. Patients with SBS may experience abdominal pain, diarrhea, dehydration, and malnutrition[2,37]. Patients' lifestyles may be impaired due to secondary lactose intolerance, gastric acid hypersecretion, metabolic acidosis, biliary and renal oxalate calculi, and dehydration[1,2].

SBS occurs in around 15% of patients with intestinal resection. 3/4th of these patients have a massive intestinal resection, and 1/4th have multiple sequential resections. Overall, the prevalence of SBS is 3-4 per million[4].

Nutritional management becomes crucial, and total parenteral nutrition (TPN) plays a vital role in SBS. TPN usually contains the missing nutrients and some micronutrients to make up for the loss of the bowel[2,5]. The treatment is planned individually depending upon the length of the small bowel, clinical symptoms, number of resections, *etc.* Anti-secretory and anti-diarrheal, digestive enzymes, *etc.*, are prescribed to slow the transit time and maximize nutrient absorption[6,7]. A team of dietitians and physicians educate the patient regarding the revised nutrition plan to restore the nutrients as soon as possible

and avoid long-term complications such as metabolic diseases, bone disorders, and vitamin deficiencies[7]. The goal of treatment is to achieve tolerance for oral feeds[5].

Rich nutrient supply, continuous diet monitoring, and regular follow-up are essential for a favorable outcome in patients with SBS. The central perspective here is to present a brief review on SBS and to highlight how the right nutrition can solve major dietary issues in patients with SBS and help them recover faster by reducing complications and improving the quality of life and if the conservative management fails, how can intestinal transplantation benefit.

METHOD OF SEARCH

The data included in this review has been gathered from extensive PubMed and Medline searches using keywords such as SBS, parenteral nutrition, nutrient deficiencies, electrolyte imbalance in SBS, and intestinal transplantation until 2020. The data has been inspired by various online case reports, review articles, and clinical trials. The search also included multiple articles and abstracts outlining the pathophysiology and treatment of SBS. This article highlights the SBS pathophysiology, importance of parenteral nutrition, treatment modalities, nutritional deficiencies, and adult prognosis.

ETIOLOGY AND PATHOPHYSIOLOGY

There are various etiologies of SBS. The acquired form of SBS resulting from surgical resections could occur in patients with Crohn's disease requiring recurrent resections. A catastrophic vascular mesenteric ischemic event may require a massive enterectomy as well. Another etiology for SBS appears with malabsorptive conditions where the bowel length is unaffected, but the bowel function is reduced. Chronic intestinal pseudo-obstruction syndrome, refractory sprue, radiation enteritis, or congenital villous atrophy are causes of malabsorption[5]. Intra-abdominal trauma, neoplasm, radiation injury, and small bowel obstruction may also result in SBS[4,36].

In an adult, the average length of the small bowel measures between 275-850 cm, where the majority of nutrients are absorbed in the first 100 cm of the jejunum. Around 7

L of fluid get absorbed in the small bowel and 2 L in the large bowel. In SBS, adults have less than 180-200 cm of small bowel length remaining[8]. These patients are at risk of intestinal failure and nutrient deficiencies and thus require nutrition support. What is meant by intestinal failure (IF) is the inability to absorb sufficient energy, despite the increase in the intake or the failure to increase oral intake appropriately due to a reduction in the functionality of the gut, such that intravenous (IV) supplementation is needed to provide the patient with the required nutrients to maintain growth and proper health[39]. It has been defined as fecal energy loss that does not correlate with the remaining intestinal length[9]. IF has been classified into three types; Type 1 is short-term and self-limiting (acute). Type 2 is an acute condition that has been prolonged. The patients often require complex care and IV supplementation that may last for weeks or months to support the metabolically unstable patient. Type 3 refers to a reversible or irreversible IF that has become a chronic condition, where the patient is maintained metabolically stable through months or years of IV supplementation[39]. When intestinal failure occurs after bowel resection, trauma, infarction, congenital defects, or diffuse loss of absorptive surface due to a gastrointestinal (GI) disease, the condition is then termed SBS. This emphasizes that the pathophysiology of SBS involves functional impairment more than anatomical impairment.

SBS results following intestinal resection in adults, causing an inadequate intestinal length resulting in insufficient digestion and malabsorption of macro and micro-nutrients, water, and electrolytes. Several aspects determine the severity of this case and its manifestation. These include the loss of absorptive surface area, loss of site-specific transport processes, loss of endocrine cells and GI hormones, rapid intestinal transit time, colon removal, small intestinal dysbiosis because of altered motility, and the loss of ileocecal valve[10]. An individual becomes malnourished and requires supplemental dietary intervention to support his health[10]. The nutritional deficits depend on the anatomically resected portions of the intestine[11]. Furtherly, the most common types of SBS are 3, which include jejunoileal anastomosis (where parts of the jejunum or ileum are resected with an intact colon), jejunal-colic anastomosis (where the ileum is resected), and

terminal jejunostomy (where a stoma in the abdomen is formed following the complete resection of the ileum and colon while preserving part of the jejunum)[37]. These surgeries will create changes in the normal anatomy of the gastrointestinal tract and will furtherly affect its absorptive function. For that reason, further information regarding detailed nutritious support for the different types of SBS will be discussed in other sections.

INTESTINAL ADAPTATION

The process of intestinal adaptation begins after surgical resection to increase the absorptive function and continues for around two years. However, two schools of thought explain this process of adaptation. Some evidence indicates that the adaptation occurs with hypertrophy and the lengthening of the remaining intestine with increased diameter and height of the villus[12]. This happens in the presence of nutrients in the lumen, which stimulates the adaptation mechanism[12]. This indicates the importance of supplementing the patient with a complex diet as soon as possible to aid this process. Yet, another mechanism explains this adaptation *via* the upregulation of the peptide transporter (PepT1) in the colon of patients with SBS. Ziegler *et al*[34] compared to controls, expressed the increased presence of PepT1 in the colon of patients with SBS, 1.5-2.5 years following resection[34]. But that was not evident in their small intestine. Yet another study later showed that around 9.8 years following resection, the patients showed no difference in their expression of PepT1[35]. This may prove that the support of intestinal adaptation through the expression of this peptide may occur only in the early period following resection, rather than being a long-term process.

A study on adult patients found that when enteral nutrition (EN) was given early and continuously, it was possible to achieve enteral autonomy around 36 d post-surgery[44]. It has been discussed that enteral nutrition maximizes the saturation of carrier proteins, enhancing enteral absorption and thus beneficial at stimulating intestinal adaptation through 3 modes of action: mucosal hyperplasia, trophic GI hormone secretion, and production of the trophic pancreaticobiliary secretions[4,44]. The use of whole protein enhances intestinal adaptation and is preferred over hydrolysates[44]. It is

proposed that to promote intestinal adaptation, a diet of complex carbohydrates, whole proteins, and long chain triglycerides (LCTs) is recommended[44]. Yet, the loss of certain bowel sections would incur further re-adjustments to the EN composition. Lactose intolerance may occur following resection of proximal jejunum. However, there isn't strong evidence regarding this condition[44,45]. Fiber supplementation is recommended only in the presence of intact colon[46].

Intestinal adaptation is a process broken down into 3 phases[48]. Phase 1 is the hypersecretory phase (acute phase), which occurs after resection and may last for the first 1-2 mo (up to 6 mo)[48,49]. The adaptive response occurs gradually in phase 2, characterized by reduced fluid losses and improved absorption of micro and macronutrients. Phase 2 is achieved through intestinal hormones and growth factors that promote functional and structural changes, and the remaining bowel sections adapt to increase their functional capacity[48]. Phase 3 is the maintenance phase, which reached around two years post-resection, where maximum adaptation is attained[29].

Nutritional management during different SBS phases requires considering the physiological changes during adaptation. In phase 1, directly following resection, while in the hospital setting, the patient requires exclusive parenteral nutrition (PN) administration, where the patient is likely to experience type 2 or 3 intestinal failure[47,50,51]. Depending on the remaining bowel sections, individualized TPN composition helps avoid negative nitrogen balance and significant weight loss and maintain adequate fluid and electrolyte balance[50,51]. Following resection, enteral feeding shall be started as soon as the patient can tolerate it. Reducing the intake of simple sugars is recommended, avoiding hypotonic solutions, and having small meals frequently during the day[7]. As the oral intake is increased and the intestinal adaptation of the residual bowel is progressing, the TPN amount is decreased, and the frequency is reduced to every other day on week 1, then to 3 times in week 2, followed by two times in week 3[7,50]. The patient shall be placed back on TPN if there are lab abnormalities, weight loss reaches 1 kg/week, or diarrhea exceeds 600 g/day[50]. Usually, patients are supported with TPN until maximal adaptation is achieved, which may take up to 1 year

or longer[50]. If the residual bowel health is not optimal, the patient may require lifelong dependency on TPN. During phase 2 of adaptation, the goal is to wean the patient off PN and increase EN. PN is associated with complications like liver disease, bloodstream infections, or thrombosis due to the central line[51]. Phase 3 is characterized by stabilization, where the patient either does not require PN in the hospital or is clinically stable but requires home PN due to irreversible intestinal failure[51]. The probability of achieving enteral autonomy is linked to having longer bowel remnant, ileocecal valve preservation, resection at a younger age, absence of liver disease, and maintaining normal GI motility[51] (Table 1).

CONSEQUENCES OF SBS

The early phase of SBS is characterized by gastric hypersecretion due to loss of inhibitory hormonal feedback signals due to resection; this is attributed to a deficiency in hormones usually produced by the endocrine cells of the proximal GI tract, such as GLP1 and 2, neurotensin and peptide YY[13]. Malabsorption is the physiologic consequence of SBS, resulting in nutrient deficiencies (Table 2). Usually, the small bowel has a large functional reserve capacity, making resections of < 50% well tolerable; however, once they exceed 50%-70%, the patient would experience malabsorption requiring supplementation to enhance the absorption[9]. Patients who undergo terminal ileal resections would suffer from a deficiency in the absorption of vitamin B12-Intrinsic factor and thus would require supplementation if they have > 60 cm resection[12]. Steatorrhea is a consequence of terminal ileal resection due to unabsorbed and intestinal loss of bile salts (choleraic diarrhea). When reaching the colon, bile salts would stimulate secretory or choleretic diarrhea if > 100 cm of the terminal ileum is removed. Unabsorbed long-chain fatty acids in the colon cause severe secretory diarrhea[14]. The ileocecal valve prevents the reflux of colonic material into the small bowel, slows the transit time, and controls the contents that pass from the ileum into the cecum. This allows more time for absorption of the nutrients by the mucosa. Preventing content reflux into the small bowel also reduces the risk of bacterial overgrowth in the small bowel[15]. The necessity of home parenteral

nutrition home parenteral nutrition or IV fluids depends on the enterocyte function and colon preservation, which role lies in the absorption of water, electrolytes, and fatty acids. Patients with < 100-140 cm small bowel and no colon or < 40-60 cm jejunum-ileum anastomosed to a portion of the colon will probably require permanent long-term PN[16-18].

INSIGHT INTO NUTRITIONAL MANAGEMENT

Nutritional management of patients with SBS requires an individualized treatment approach. The primary purpose of intestinal rehabilitation is to improve the quality of life by enhancing the absorptive potential of the remaining intestine and reducing the long-term dependency on parenteral nutrition PN. Post-resection, all SBS patients will require parenteral nutrition. Some may need it for a short period until the post-adaptive phase, while other patients may need it long-term. Oral feeding (enteral nutrition EN) enhances the process of intestinal adaptation and is favored over parenteral nutrition, especially when the bowel activity is resumed, and diarrhea is limited to < 2 L/day in a patient with stable electrolytes and hydration[19,20]. A paper by Matarese presents an adequate approach to nutrition optimization for patients with SBS[19]. American Society for Parenteral and Enteral Nutrition guidelines are not specific to patients with SBS who would require greater nutritional requirements when compared to other patients on PN. The total caloric delivery recommended for patients with SBS is approximately 32 kcal/kg/day. The amount of lipids provided should not exceed 1 g/kg/day but should make up 20%-40% of the total calories provided. 100-120 g/day is the minimum recommended amount of carbohydrates provided in the form of dextrose, but patients may need 2.5-6 g/kg/day. The amount of proteins recommended is 1.5 g/kg/day[20,21]. End-jejunostomy may result in high stomal outputs, increasing the need for fluids to > 3 L/day, additionally the PN they receive[22]. As mentioned earlier, the dietary approach should be patient-oriented depending on the remaining portions of the intestine. However, patients may benefit from having several small meals daily to increase the net absorption - what is referred to as adaptive hyperphagia. A way to compensate for the

malabsorption is consuming a caloric amount that is more than 50% of a regular diet. Complex carbohydrates are preferred to avoid osmotic diarrhea[9].

After resection, patients may be transitioned from complete PN/IV to oral diet or tube feeding. A study showed that when continuous tube feeding is present alone or in combination with oral nutrition, more absorption of nutrients is observed than when oral nutrition is carried out alone. The study demonstrated increased total lipids, calories, and proteins in these patients[23]. Polymeric diets are more commonly used than elemental diets due to preferred aspects of polymeric diets that include less cost, less hyperosmotic, and better at enhancing intestinal adaptation[20] (Table 3).

End-jejunostomy puts patients at risk of dehydration and diarrhea. Since this is type 1 of SBS, characterized by the loss of the most significant portions from the GI tract (ileum and colon), it sets more serious malabsorptive issues than other SBS[37]. As these patients seem to be net-secretors, it is recommended to provide them with fluid amounts greater than their ostomy outputs (1.5-2 L/day)[9,27,37]. Patients may benefit from the sodium-glucose co-transport with oral rehydration solutions to maintain hydration. Yet, if half of the colon is maintained, then an oral rehydration solution is unnecessary[9,27]. This type of SBS may lead to losing the ability to produce hormones such as PYY, as these are made by L cells in the distal ileum and colon, and reduced increases in the GLP-2 levels postprandially (which function is inhibiting the gastric emptying). This furtherly contributes to accelerating gastric emptying and intestinal transit time[24,38]. Patients with type 1 SBS may benefit from the following management recommendations. The dehydration a patient experiences may be corrected with IV saline while the patient is on nil oral intake for 24-48 h; this helps relieve the thirst the patient experiences. The IV saline is then weaned off over 2-3 d while the reintroduction of oral fluids gradually[11]. Reducing the hypotonic fluids taken orally to less than 500 mL/day is also essential. Since most stomal outputs have a sodium concentration of around 100 mmol/L, the patient may benefit from having glucose/saline solution sips with a sodium concentration of at least 90 mmol/L[11].

For patients with type 2 SBS (jejunal-colon), long-term parenteral nutrition may be needed if: increased oral/enteral intake causes high volumes of diarrhea/stomal output that are socially unacceptable to the patient or if the patient is unable to absorb more than one-third of the oral energy intake, or if the absorption is 30%-60% with high energy requirements[11,42]. Patients with preserved colon would rarely be in negative water or sodium balance and thus rarely require supplementation[43]. If the patient becomes sodium depleted, they may sip a glucose-saline drink[11].

Different patients with SBS require parenteral nutrition for varying periods, depending on the remaining bowel length and the type of SBS resulting after resection. It has also been shown that the length of the remaining small bowel sections is predictive of the ability to wean PN later. Nutritional autonomy (achieving home PN-free status for 12 mo) and home PN cessation could be possible if the final small bowel length achieved following surgery is greater than or equal to 115 cm (for type 1 SBS), greater than or equal to 60 cm (type 2 SBS), and greater than or equal to 35 cm (type 3 SBS)[39,40].

Before weaning patients off PN or IV fluids, patients must maintain stable body weight and electrolyte levels as they can obtain around 80% of their daily energy requirements through oral feeding. Urine output should be > 1L/day on PN/IV-free nights and an enteral balance of 500-1000 mL/day. Weaning may be accomplished in one of two ways: reducing the number of PN/IV fluid days or reducing the volume of PN/IV fluid delivered during sessions. Dehydration is less likely to occur with the latter option[28].

Messing *et al*[29] carried out a study with 124 adult patients. They showed that the likelihood of PN weaning is related to factors such as the SBS types with colonic remnant (even if partial) as well as having a post-duodenal remnant of small bowel < 100 cm. At five years, the survival probability reported was 75%, and the PN-dependence was 45%. Survival was negatively related to type 1 SBS, small bowel length < 50 cm, and vascular etiology of SBS, but not negatively related to PN dependence[29]. Another study found that the median duration of home PN was 2.6 years, where nutritional autonomy was achieved by 13.8% in year 1, 24.5% in year 2, 34.1% in year 5, and 38.3% in year 10[40]. As

per this study, achieving PN autonomy was associated with younger age, greater length of the small bowel, and having a colon remnant[40]. The most recently published data showed that patients with home PN had a 5-year survival of 76%. The home PN-related deaths accounted for 11% of fatalities during home PN, with an incidence of 10 home PN-related deaths per 1000 home PN treatment years[41].

COMPLICATIONS

Malabsorption is caused by intestinal failure, which may result in malnutrition, diarrhea, weight loss, steatorrhea, electrolyte imbalance, and vitamin deficiencies. The patient requires long-term treatment and supplementation with minerals and electrolytes to prevent various complications of SBS. Among these complications, intestinal failure-associated liver disease and liver cholestasis are significant. To avoid this complication, oral feeding is essential. Also, it is an option to decrease dependence on soy-based lipid emulsions or switch them to fish oil-based emulsions[13].

Gallstone is a possible complication in patients with < 180 cm small bowel or absent ileocecal valve. The most frequent type of gallstone is calcium carbonate. Enteral feeding, limiting oral fasting, and reducing the use of narcotics and anticholinergics are methods used to limit the occurrence of cholelithiasis[13].

IV fluids reduce complications such as hyponatremia, dehydration, nephrolithiasis (from hyperoxaluria), and chronic renal failure. Regular hydration and maintaining a urine output of 800 mL/day and Urine sodium > 20 mmol/L are suitable actions for these patients. Magnesium malabsorption should be treated with > 1.5 mg/dL supplementation[7,13].

A patient presenting with ataxia, slurred speech, psychosis, and altered mental status may be a sign of developing D-lactate encephalopathy. The colon microbiota's fermentation of unabsorbed carbohydrates causes metabolic acidosis with a high anion gap due to D-lactic acidosis. Management of these patients includes stopping carbohydrate-based enteral feeds and administering antibiotics like metronidazole,

vancomycin, and clindamycin as they are active against D-lactate forming bacteria. Hydration and thiamine supplementation should also be considered[13].

The metabolic bone disease may result in osteoporosis or osteomalacia. Patients receiving parenteral nutrition in a long-term manner are at risk of developing abnormal bone metabolism. Patients may complain of back pain, joint pain, atraumatic fractures, and loss of height. Examining the mineral bone density, checking mineral levels, vitamin D and PTH, and markers of bone turnover are helpful steps in evaluating this type of patient. Dietary and lifestyle changes can make a significant impact on these patients. Also, maintaining adequate levels of vitamin D and nutrition is essential. Finally, bisphosphonates may also help manage these patients[13].

INTESTINAL TRANSPLANTATION

Intestinal transplantation has long been an exciting aspect of gastroenterology that has faced several challenges until professionals could finally carry it out successfully. In 1959, Lillihei and coworkers reported the first canine model of intestinal transplantation at the University of Minnesota[30]. Later, in 1967, they reported the first formally published human intestinal transplantation. Until 1970, seven intestinal transplants were carried out, yet the most prolonged graft survival was 76 d. The first long-term survivors were cases carried out in Kiel in 1988 and Paris in 1989. There were cases carried out in 1989 in Innsbruck and 1990 and 1991 in London, Ontario, that resulted in death due to tumor recurrence or lymphoma, even though the grafts were functional[30].

SBS has been the most common reason for the need for a transplant, accounting for 60%-65% of all transplant cases[52]. Home PN is considered the primary treatment for patients with SBS. However, when it fails by contributing to liver disease, central venous catheter thrombosis of at least two central veins, frequent central line sepsis, or severe dehydration despite receiving fluids, the choice of undergoing intestinal transplantation is supported[8,30]. Three intestinal transplants are described: liver-intestine, isolated intestine, and multi-visceral transplant. The last two are the most used in adult patients. Irreversible IF with PN-associated life-threatening conditions and preserved liver

function is a significant indication for undergoing an isolated intestinal transplant. The isolated intestine includes the jejunoileal portion and could be in the presence or absence of a graft colon[52].

In the adult population, prolonged PN exposure accounts for the need for liver-containing intestinal grafts. For that reason, early recognition of intestinal failure and proper management and referring the patient for transplant option, with the withdrawal of PN before suffering from irreversible PN-associated liver disease, could serve to provide the patient the opportunity of isolated intestine transplant and avoid the necessity of liver-inclusive graft[52]. The 1-year graft survival of intestinal transplants performed in 6 US centers between January 2014 and June 2016 was in the range of 65.5%-83%. The average 3-year adult graft survival in these centers was 56.3%, ranging between 28.6% and 72.7%[53] (Table 4).

PHARMACOLOGICAL ADVANCEMENT IN THE TREATMENT OF SBS

Clinical trials are still on for administering GLP-2 to patients following small bowel surgery. Teduglutide, an enzyme-resistant GLP-2 analog, has been studied in clinical trials and used in patients with SBS. Teduglutide increases absorptive capacity and inhibits gut motility, stimulating crypt cell proliferation and inhibiting enterocyte apoptosis. Teduglutide has shown promising results, but in some patients, it has taken longer than usual[31]. In one of the 24-week studies, Teduglutide was well tolerated among patients with SBS, and it was seen to reduce the number of days of parenteral support in these patients[32]. One of the meta-analyses on teduglutide response showed that the response rate to teduglutide treatment was estimated to be 64% at six months, 77% at one year, and 82% at ≥2 years; while the weaning rate was estimated as 11% at six months, 17% at one year, and 21% at ≥ 2 years. Overall, the response rate was observed to be significantly increased between 6 mo and one year and then was found to be maintained afterward[33]. Clinical trials are still needed to assess the effective and practical dose calculations and length of the treatment required for SBS.

CONCLUSION

Expert recommendations are required while preparing the nutrition chart as the plan depends upon the existing length of the intestine, patient factors, and the nutrition available. Regular follow-up is essential to proper nutrition to look out for complications and patient compliance. The right nutrition plan is crucial in patients with SBS to accelerate the recovery time, which should be tailored to the patient's needs to lead a healthy lifestyle. A thorough understanding of gastrointestinal anatomy and physiology is crucial to planning out the nutrition and treatment in SBS patients. Intestinal transplantation could be considered for those in which conservative management fails.

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