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Surgical Options in Short Bowel Syndrome

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Abstract

Short bowel syndrome is characterized by a significant reduction in the effective intestinal surface by an anatomical or functional loss of the small intestine. It mainly occurs after extensive bowel resection, intestinal intrinsic disease or surgical bypass. The main complications are mal-digestion, malnutrition, dehydration and, potentially, lethal lesions. The management, based on appropriate and individualized nutritional support, is complex and requires a multidisciplinary approach including dietary, fluid and pharmacological management, co-morbid disease management and, sometimes, surgery. Numerous surgical procedures to relieve the consequences of massive intestinal resection have been devised, but many have not been employed in human clinics and have remained in the experimental surgery field. The choice of technique is dictated by the patient's underlying pathophysiology and includes such factors as age, intestinal transit time, length of remnant bowel, presence of intact colon, degree of small bowel dilation, and others. Autologous intestinal reconstruction includes various techniques which manipulate the bowel surgically to facilitate the bowel adaptation process and restoration of enteral nutrition. The most accepted techniques are those elongating the bowel that may be applied only on dilated bowel. However, the intestinal transplantation can be a curative alternative for patients in whom intestinal rehabilitation attempts have failed and who are at risk of life-threatening parenteral nutrition complications.

Keywords: Short bowel syndrome, Short gut, Surgical management.

Introduction

Short bowel syndrome results from surgical resection, congenital defect, or disease associated loss of absorption, and is characterized by the inability to maintain protein-energy, fluid, electrolyte or micronutrient balances when on a conventionally accepted normal diet[1]. SBS patients cannot absorb enough water, vitamins, minerals, proteins, fat, calories, and other

nutrients from food. What nutrients the small intestine has trouble in absorbing depend on what small intestine section has been damaged or removed.

Etiology and Epidemiology

There are many causes of SBS [2,3], the main form of surgery is to remove a portion of the small intestine. This surgery can treat intestinal diseases, injuries, or birth defects. Currently, mesenteric vascular accidents are the main cause in adults, followed by inflammatory bowel disease (Crohn's disease), and radiation enteritis. In children, the main causes are congenital and perinatal diseases. In infancy, SBS most commonly occurs following surgery to treat necrotizing enterocolitis[3]. SBS may also occur following surgery to treat several pathologies: cancer and damage to the intestines caused by cancer treatment; inadvertent gastroileostomy after gastric surgery; intestinal hernia; intestinal injury from trauma or from loss of blood flow due to a blocked blood vessel; intussusception; midgut volvulus; omphalocele; intestinal pneumatosis; gastroschisis, among others. Even if a person does not undergo surgery, disease or injury can damage the small intestine. SBS incidence and prevalence are unknown because no reliable databases are available. Estimates are based on information from home parenteral nutrition (PN) registries, for which SBS is generally the most frequent indication. Two studies, limited to SBS patients, have reported the majority of patients being female and > 50 years of age [4,5]. A multifactorial etiology, uncertainly, intestinal length and varying definitions of SBS all make the comparing of reports difficult. In the U.S., annual prevalence of home PN has been estimated to be approximately 120 per million, of whom about 25% have SBS [6]. Each year SBS affects about three in every million people [3]. These numbers do not reflect patients with SBS who do not survive, were able to be weaned from PN during index hospitalization, or were able to be successfully weaned from home PN. Thus they, likely underestimate SBS prevalence.

SBS occurs in about 15% of adults who undergo intestinal

resection; nearly 75% result from a single massive resection, and 25% from multiple resections [7]. Approximately 70% of those with newly acquired SBS are eventually able to be discharged from the hospital [8]. Reports have demonstrated 2-year and 5-year survival rates for SBS at over 80% and 70%, respectively [9,10]. Survival rates are lower in the end-jejunal and ultrashort (remnant bowel 10% which an ileocecal valve, or 20% with no ileocecal valve) SB groups. PN dependence at 1, 2 and 5 years has been reported to be 74%, 64% and 48%, respectively [5]. Other factors which affect survival in SBS include patient's age, primary disease process, comorbid diseases, presence of chronic intestinal obstruction, and the experience of the team that manages the patient [11].

Prognostic Factors

The main factors involved in the prognosis of SBS-related bowel failure include the anastomotic region of the resected bowel, residual bowel length, presence of an underlying pathology, presence or absence of a colon and of the ileocecal valve, the nature of the main disease [2] and the patient's age [12].

Residual bowel length correlates with the patient's degree of nutritional autonomy; that is, independence of PN. Patients with a very short residual bowel length are more likely to develop kidney failure [13] and liver failure [14], and depend more easily on PN. Intestinal resections that affect less than 33% do not imply protein-calorie malnutrition; loss of up to 50% allow nutritional tolerance with no nutritional support; those over 75% probably require PN to avoid malnutrition [15].

The anatomy of the remnant bowel, that is, if a colon or ileum continues, also affects the prognosis. Patients who are at high risk of losing nutritional autonomy are those with duodenostomy, <35 cm of residual jejunum in patients with jejunal-ileal anastomosis, <60 cm of residual bowel in patients with jejuno-colic or ileal-colic anastomosis, or <115 cm remnant with terminal jejunostomy [16].

Functionality does not depend only on length; 150 cm of pathological intestine can function worse than 75 cm of healthy intestine. Some definitions of SBS and intestinal failure have been based on measures of the remnant bowel's functional capacity. These measures include loss of fecal energy [17], or citrulline plasma levels [18].

Ileocecal valve loss is a major factor in patients who have undergone ileal resection. The ileocecal valve acts as the main barrier against colic reflux from the colon to the small intestine, and helps regulate not only the fluids and nutrients that leave the ileum to the colon. If the ileocecal valve is intact, intestinal transit slows down, but experimental studies have questioned this fact [19]. Resection of the terminal ileum and ileocecal valve is associated with bacterial overgrowth, a relevant aspect of SBS.

If the colon remains, it can sometimes prove to be a critical determining factor for patients who are independent of intravenous support because the colon in SBS patients becomes a major digestion organ. The colon helps maintain fluids and electrolytes as it can increase its absorption capacity 5-fold after small intestine resection. It is also able to absorb proteins and retrieve malabsorbed carbohydrates through bacterial metabolism, which converts them into short-chain fatty acids [17]. The colon slows down intestinal transit and stimulates

intestinal adaptation. Regarding PN dependence, presence of at least half a colon is the equivalent of 50 cm of small intestine [2]; thus patients can be grouped into two subgroups: those with a continuous colon (that is, jejunum-colon or jejunum-ileum-colon) and those with no colon, in whom a short bowel will end in stoma (enterostomy). However, the presence of a colon increases the incidence of calcium oxalate renal lithiasis.

Small Bowel Syndrome Types

There are three SBS patient groups depending on the type of anastomosis and whether the colon is present or not [2]. The first group is formed by those patients for whom resection affects part of the jejunum, ileum and colon, and they present terminal jejunostomy. The second group is formed by patients who have undergone ileal resection, which often involves the ileocecal valve, and thus have jejunal-colic anastomosis. Finally, other patients predominantly display jejunal resection with more than 10 cm of terminal ileum and remnant colon (jejunal-ileal). This last group of patients is quite scarce, whose management is similar to those with jejunal-colic anastomosis. Thus both groups enter the series of patients with SBS and preserved colon.

Intestinal Adaptation

Following resection, intestinal tissue undergoes morphological and functional changes to compensate for lost resected bowel function. These changes are mediated by multiple interactive factors, including intraluminal and parenteral nutrients, gastrointestinal regulatory peptides, hormones, cytokines and growth factors, many of which have been well characterized in animal models [20], in addition to tissue factors that include immunity, blood flow and neural influences [21,22]. The amount of remaining small bowel is the most important predictor of the adaptive potential; neither structural nor functional adaptive changes have been demonstrated in human or animal models, and more extreme resections result in end-jejunostomy [22]. Bowel adaptation is a remodeling process which, involve compensatory mechanisms that aim to improve the remnant bowel's absorptive capacity following intestinal loss. Increased intestinal mass and surface area occur through enterocyte and crypt cell proliferation, increase in microvilli with taller villi and deeper crypts, and hyperplasia and hypertrophy of smooth muscle layers [22,23]. Adaptation is an ongoing process that begins 24-48 hours after surgical resection, and rapidly progresses during the initial 4-24 months following intestinal loss, and can even take years to complete [5,24]. Following intestinal resection, the bowel's motor activity is also disrupted for a few months [25]. Studies have demonstrated a shorter duration of the migrating motor complex cycle and feed pattern after resection [26]. Since bowel dilatation is a natural sequel of adaptation, surgery should be deferred unless dilatation causes recurrent episodes of bacteremia [27]. Bowel dilatation occurs more commonly in younger patients, who are more often candidates for surgical rehabilitation than adults [28].

For the adaptation process, presence of nutrients inside the intestinal lumen is important. So beginning with oral or enteral nutrition must start as soon as possible. Nutrients also provide substrates to reproduce enterocytes and can stimulate the release of trophic factors [29-31]. Intestinal adaptation can be stimulated by secretions and gastrointestinal hormones.

Massive intestinal resections also determine changes in both the function and structure of gastric mucosa, and mucosa hyperchlorhydria and erosion stand out. Hyperchlorhydria agrees with the increase in oxyntic cells observed in gastric body glands. Intestinal adaptation can be facilitated because the patient eats more than is normal (hyperphagia), and the intestine is also able to adapt to accomplish more efficient absorption per surface unit by increasing its absorptive surface and/or slowing down gastrointestinal transit. Hyperphagia is a fundamental mechanism in humans, which occurs in more than 80% of SBS patients (>1,5 times basal energy usage levels).

Clinical Manifestations

The clinical symptoms of a patient with a short bowel generally come in three phases. Since virtually all the components that are useful for the organism, and most of water and electrolytes, are absorbed by the small intestine, the potential spectrum of short bowel clinical symptoms is almost infinite. The absorption of almost all nutrients is poor and, with time, this circumstance implies serious weight loss, tiredness, laxness and weakness as a result of the disorders that derive from poor fat, glycide and protein absorption.

Surgical Treatment

Surgically treating SBS is a therapeutic option that must be put into practice only when first-choice medical-diet treatment is not able to achieve acceptable patient nutritional status, serious complications appear because of the patient's condition which entails having to suspend this treatment, or the patient's quality of life with this treatment becomes intolerable.

The following general considerations about surgically treating SBS can be made:

1. The surgeon must undertake SBS prophylaxis when faced with a situation that implies massive intestinal resection. Exereses must be limited as much as possible (especially when they affect the ileum), the ileocecal valve must also be respected if at all possible, and termino-terminal anastomosis is recommended to avoid blind loop syndrome as this can harm compensation mechanisms. If the patient on resection treatment is a carrier of previous digestion bypasses that can help establish SBS, the suitability of eliminating them during the same surgical treatment must be assessed.
2. None of the operations considered in this epigraph must be done during responsible SBS resection surgery as the intestinal adaptation potential can render them unnecessary. Yet many of the techniques assayed in experimental surgery conduct intestinal resection and the procedure is used to relieve potential SBS following exeresis during the same surgical procedure.
3. Nor should surgical treatment be practiced in the initial syndrome phase, but after at least 6-12 months following resection and when patients cannot maintain 70% of their normal weight without PN; that is, in the last disease stage to allow mechanisms of compensation to be fully completed in the remnant bowel [32].
4. An intestine transplantation is the ideal solution to treat SBS; however, it is not routinely used in human clinics.

Numerous surgical procedures to relieve the consequences of

extensive intestinal resection have been devised [33-48], but many have not been employed in human clinics and have remained in the experimental surgery field. The tested surgical techniques have been grouped according to their objective: a) techniques to increase intestinal blood flow; b) techniques to control gastric hypersecretion; c) techniques to prolong intestinal transit; d) techniques to avoid bacterial overgrowth; e) techniques to increase the absorption surface, and, f) techniques to enlarge the absorption surface and to prolong transit time.

For SBS patients, surgery can play an important role in preventing, migrating and, in some cases, reversing intestinal failure. During intestinal resection, bowel length should be conserved to the fullest possible extent to avoid dependence of PN. Bowel salvage may be improved by initially preserving tissue of questionable viability and later reassessing by "second look" procedures. Once patients are stabilized, ostomy reversal and recruitment of distal unused bowel should be prioritized whenever feasible. Following progression to intestinal failure, surgical SBS management depends on the symptoms and anatomical characteristics of each individual patient.

The mainstay of management for children with SBS includes a prolonged course of PN and dietary adjustments until a degree of intestinal adaptation that is compatible with life and sustained growth has been achieved. These include vagotomy and pyloroplasty, recirculating small bowel loops, reversed small bowel segments (single and paired), periintestinal rings, pouch formation, and prejejunal or preileal colon transposition. Other researchers have attempted to grow small intestinal mucosa on colonic serosal patches, or have used vascularized abdominal wall pedicle flaps, mucosal denuded tubular colonic segments and prosthetic materials, bowel elongation techniques, neomucosa growth, among others.

Because of PN associated comorbidities, the initial goal in managing patients with intestinal failure is to enable PN weaning. Autologous gastrointestinal reconstruction (AGIR) further facilitates the adaptation process and attempt to reverse PN complications. The principle of AGIR relies on bowel dilatation resulting from post-resectional adaptation and the ability to manipulate this dilated bowel surgically to improve dysmotility and absorption, which thus facilitates enteral autonomy. Of the several reconstruction methods followed, tapening enteroplasty, Bianchi's longitudinal intestinal lengthening and tailoring (LILT) and serial transverse enteroplasty (STEP) have been more widely accepted. It is now well accepted that the probability of permanent PN dependency is fairly high with remnant bowel length <40 cm. In such cases, where the chance of spontaneous adaptation is minimal, the creation of controlled obstruction to actively generate bowel dilatation is suggested with a view to performing AGIR surgery later.

Different AGIR options must be tailored individually according to the length and type of residual intestine, but the combination of various techniques may prove beneficial in certain situations than a single procedure.

An antiperistaltic bowel loop, creates partial mechanical obstruction and delays myoelectric activity in the distal segment, which slows transit to enhance nutrient absorption. Reversed segment lengths of 10-15 cm for adults and around 3 cm for children have been suggested to provide maximum benefits, but

establishing the ideal length and location of the reversed bowel segment remains uncertain and produces variable results when clinically applied; e.g., this technique cannot be used when the remnant bowel is <25 cm [49]. For patients with rapid intestinal transit and who do not present bowel dilation, small bowel segmental reversal may cut PN requirements [49-52].

In the event of an oversmall remnant bowel precluding AGIR, isoperistaltic colon interposition can prove a useful technique in patients with ultra SBS. In exceptional circumstances, the colon plays a key role, similarly to those of the remaining small bowel. Tapering enteroplasty reduces the diameter of the dilated bowel and facilitates an early return of effective peristalsis.

The intestinal plication technique was designed by the circumferential plication of the dilated bowel after folding the redundant antimesenteric wall into the lumen to avoid a long anastomotic suture line of enteroplasty, and to also preserve the mucosal mass, which is a relevant aspect in SBS.

An important progress in SBS management came when Bianchi reported the longitudinal intestinal lengthening procedure, which doubled the length of the original segment [53], and with posterior procedure modifications.

For LILT and STEP, failure to achieve intestinal autonomy by conservative therapy is the main indication, and end-stage liver disease is the main contraindication. A sufficiently dilated intestinal segment is a common anatomical precondition for both procedures. STEP can be performed on shorter intestinal segments, and also on intricate segments such as the duodenum, which is technically not feasible by LILT. Both procedures offer a similar extent of intestinal lengthening (approximately 70%) and results in improved enteral nutrition and the reversal of PN complications. For carefully selected patients with rapid intestinal transit and a dilated bowel, LILT and STEP procedures may prove beneficial [49,52]. Outcomes following STEP and LILT are generally similar, and the choice between these procedures may depend on the surgeon's preference. From the technical perspective, STEP offers clear advantages over LILT as it is easily reproducible, and, above all, it can be performed primarily or repeatedly in patients who develop bowel redilation after a LILT or STEP procedure. STEP appears to have lower mortality and overall progression to transplantation rates. In children, STEP and LILT are both accepted procedures for non-transplant surgical SBS management.

Intestinal transplantation is a curative alternative for patients in whom intestinal rehabilitation attempts have failed and who are at risk of life-threatening PN complications [54]. The main goal of intestinal transplantation is to restore enteral nutrition. Intestinal transplantation has several variants depending on other concomitant organ failures, which need simultaneous replacement with intestines. Although intestinal transplantation is potentially life-saving for SBS, it should be reserved for patients with failed AGIR or for those who have no autologous enteral autonomy prospects [54,55].

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