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Review Article

Short bowel syndrome

Ketan Vagholkar*, Madhavan Iyengar, Suvarna Vagholkar, Amish Pawanarkar, Sharique Ansari, Shamshershah Pathan, Ankit Bhupatkar

Department of Surgery, D.Y. Patil University School of Medicine, Navi Mumbai 400706, Maharashtra, India

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***Correspondence:**
Dr. Ketan Vagholkar,
E-mail: kvagholkar@yahoo.com

ABSTRACT

Short bowel syndrome is one of the most complex sequel to resection of extensive lengths of the small intestine. The nutritional depletion caused exerts deleterious effects on every organ system of the body. Identifying and managing this complex problem is the biggest challenge to the clinician. The pathophysiology and therapeutic approach to short bowel syndrome is discussed.

Keywords: Short bowel syndrome treatment

INTRODUCTION

Resection of large segments of the small bowel for various gastrointestinal disorders is commonly encountered in surgical practice. Mesenteric vascular occlusion, intestinal tuberculosis, Crohn's disease are common indications for extensive small bowel resection. In the pediatric population necrotizing enterocolitis is a commonly encountered indication of small bowel resection. The average length of small intestine is approximately 6 feet. The exact cut off for short bowel syndrome (SBS) to develop remains arbitrary ranges approximately from 100 to 200 cms. Understanding the physiological basis for SBS is pivotal in determining an algorithm for the management of this challenging entity.

Anatomy

The small bowel extends from the duodenojejunal junction to the ileocaecal junction (IC). The duodenum is fixed and ends at the ligaments of Treitz. The proximal two fifth is the jejunum and the distal three fifth is ileum. Vascular considerations of the small bowel may not always be pertinent as the gross morphology of small intestine during the surgical operation may necessitate resection of grossly gangrenous portions. Nutritional implications depend on which portion of small bowel is significantly sacrificed. The IC junction which contains the IC valve is important in improving efficacy of the intestinal transit time. If the valve is lost, the transit time is hastened with more loss of fluid and nutrients. Colon is also an important determinant of optimum haemostasis in the G.I. tract. Absence of the colon adversely affects the absorption of water, metabolism of carbohydrates serving as an energy source and alteration of ions which may predispose to stone formation.

Pathophysiology

Resection of large segments of the small intestine initiates a wide range of physiological changes there by initiating a cascade of adaptations.[1,2]

The acute phase may last up to three to four months. It is associated with malnutrition, fluid and electrolyte loss though the GI tract. There is transient fluctuation in liver function test (L.F.T.).

The adaptation phase begins 2-3 days after bowel resection and may extend for 12 to 18 months. Maximum bowel adaptations may occur in this phase. Villous hyperplasia, increase in the depths of the crypts followed

by dilatation of intestine are the common changes seen in this phase.

In the maintenance phase the absorptive capability of the G.I. tract increases maximally. However it will never match that of the normal intestine.

Nutritional alterations with significant loss of jejunum and ileum.

The absorption of proteins, carbohydrates, vitamins and minerals may remain unaffected as the ilium may take over this function. The loss of enteric hormone production by the jejunum results in weakening of enzymatic digestion. Biliary and pancreatic secretions decrease significantly. However surprisingly, the gastrin levels rise causing high acid secretion leading eventually to mucosal injury of the small intestine. The dwindling intra luminal pH level impedes effective activity of pancreatic enzymes. As a result of a large osmotic load unabsorbed nutrients are delivered to the ilium and colon resulting in severe diarrhea.[3]

Resection of significant portion of the ileum impedes the absorptive capacity for water and electrolytes in addition to altering the intrahepatic circulation of the bile salts, loss of fat soluble vitamins, fat malabsorption and steatorrhea. The net result being excessive loss of fluids, electrolytes and fat soluble vitamins.[4]

With respect to colon an intact I.C. junction is very important.[5] If the I.C junction is resected then colonization of the small bowel by the colonic (flora) bacteria will worsen the diarrhoea and nutrient loss. With extensive small bowel gone but with residual colon the absorptive capability of the colon for water increases many fold. The colon also has an inherent capability to metabolize undigested carbohydrates into short chain fatty acids. These are utilized as an efficient fuel source for the colon. However the intact colon with loss of small intestine increases the incidence of urinary calcium oxalate stones. Oxalate is normally bound to calcium and is rendered insoluble by the time it reaches the colon. After extensive small bowel resection much of the calcium is bound by free intraluminal fats. As a result free oxalate is delivered to the colon where it is absorbed which eventually results in saturation of urine with calcium oxalate crystals resulting in stone formation

Etiology

Small bowel syndrome is commonly encountered in mesenteric vascular accidents, Crohn's disease and tuberculosis. However mesenteric vascular disease continues to be the leading cause of SBS.[6]

Clinical presentation

SBS presents with weight loss, fatigue, malaise, lethargy, severe diarrhoea with resultant dehydration, electrolyte imbalance, protein caloric malnutrition accompanied with loss of vital minerals and vitamins. Vitamin A deficiency may present as night blindness and xerophthalmia Vitamin D deficiency may be associated with paresthesia and tetany. Vitamin E can cause paresthesia, ataxic gait and visual disturbances. Easy bruisability and prolonged bleeding may be due to vitamin K deficiency. Varied types of anaemia due to vitamin B_{12}, folic and iron deficiency are commonly seen.

Calcium and magnesium deficiency can present as paresthesia and tetany. Low zinc levels can cause anorexia and diarrhoea.

Physical examination will reveal gross physical depletion, temporal wasting and loss of digital muscle mass, peripheral oedema, drying of skin, prominent ridges of nails and atrophy of lingual papillae. Essential fatty acids loss may lead to growth retardation, dermatitis and alopecia. Corneal ulceration and growth delay are caused by vitamin A deficiency.

Low levels of B complex vitamins can cause a range of symptoms such as stomatitis, cheilosis, glossitis, edema tachycardia, ophthalmoplegia, seizures, neuropathy and decreased tendon reflexes. Bow legs due to vitamin D deficiency. Petechial ecchymosis and purpura may be manifestations of vitamin K deficiency. As a result every organ system of the body will be significantly affected.[7,8]

Investigations

Complete blood count usually shows hypochromic microcytic anaemia. In severe cases megaloblastic anaemia is seen. Plasma albumin level usually falls thereby affecting the process of healing.

Free albumin levels may also be low but can be skewed by hydration status and renal function. Liver enzymes are altered in those patients in whom long term parenteral nutrition is administered.

Blood urea nitrogen and creatinine are significantly altered in patients with inadequate fluid intake. Trace elements are also depleted. Therefore the standard set of investigations comprises of complete blood count, liver profile, renal profile, peripheral smear and estimation of vitamin levels.[9] Imaging studies may help in diagnosing concomitant comorbid conditions which may impact the outcome in such patients.

Contrast enhanced CT scan may help in assessing the status of the bowel, liver and gall bladder. Cholelithiasis and fatty changes of liver are commonly encountered in patients suffering from SBS.

Chest x-ray is mandatory on a periodic basis in those patients who are on intravenous TPN.

Treatment

Aggressive supportive care is the initial mainstay of treatment.[9,10] A variety of protocols for fluid and electrolyte management have been proposed. Optimum correction of fluid deficit and electrolyte imbalance is of utmost importance. This ensures optimum function of the cardiovascular and renal system. Once hemodynamic stability has been achieved, parenteral nutrition (TPN) constitutes the most important treatment. TPN should provide adequate proteins, carbohydrates, fats, macro and micro nutrients.

If the oral route can be used then a proper calculated diet can be of immense help. The oral route has the advantage over TPN as it helps in better utilization of residual bowel as compared to TPN.[10,11] This includes improved local immunity, better assimilation and lesser chance of sepsis whereas TPN may provide a complete correction of fluids, electrolytes, nutrients and vitamins but may have deleterious effect on various other organ systems of the body. Fatty liver, gall stones, poor GI immunity and vascular complications due to long standing central lines add to the susceptibility to sepsis. Various other medical treatments have been tried out with beneficial effects.

Somatotropin which is the recombinant human growth hormone has a significant anabolic and anti-catabolic effect on a wide range of cells from ranging from hemopoietic to myocytes. It acts on specific receptors like IGF1 which have a tropic effect on the gut. The adult dosage is 0.1mg/kg/day SC for up to 4 weeks.[11,12]

Tedugludid which is an analogue of naturally occurring glucagon like peptide 2 is also being used. This is used to improve the intestinal absorptive capability by stimulating the effect on entero endocrine cells, sub epithelial myofibroblasts, enteric neurons of the submucosal and myenteric plexus.[12]

Decreasing gastric secretion by proton pump inhibitors helps in decreasing the severity of diarrhoea, administration of octreotide in early stages may significantly help in those patients with extensive resection of the small intestine and colon.[12,13]

A glutamine supplement exerts a very strong trophic effect on the gut. It induces hypertrophy and hyperplasia of the gut enterocytes thereby increasing the absorptive capability both qualitatively and quantitatively.[11]

The role of surgical intervention in SBS is variable.[14,15] A variety of operations have been tried but with mixed results. They are divided into two group's viz. intestinal/combined intestinal and liver transplant and non-transplant operations.

Transplant operations provide great hope to such patients, however the morbidity associated with these procedures is very high culminating to increased mortality in most cases.[14]

Non transplant procedures have been practised for quite some time. The type of operation is selected on the basis of certain criteria.[15-17]

1. Age of patient
2. Length of remnant bowel
3. Absence/presence of bowel dilatation
4. Functional status of remnant bowel
5. Intestinal transit time
6. Absence/presence of TPN related complications

Intestinal lengthening procedures, intestinal tapering for dilated segments, stricturoplasty and creation of intestinal valve or reversal bowel segments have been tried out.[18-21]

The STEP procedure which comprises of serial transverse enteroplasty has been tried out in children with promising results.[21-23]

CONCLUSION

SBS continues to be the most challenging aftermath of extensive bowel resection.

Aggressive supportive care with medical treatment continues to be the mainstay of treatment.

Surgical procedures can be performed only in a select group of patients. However the results may not be always promising.

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