Journal of Enterocolitis

Review

Intestinal Failure and Management in Inflammatory Bowel Diseases

Gizem Dağcı, Filiz Akyüz

Division of Gastroenterohepatology, Department of Internal Medicine, Istanbul University, Faculty of Medicine, İstanbul, Türkiye

Cite this article as: Dağcı G, Akyüz F. Intestinal Failure and Management in Inflammatory Bowel Diseases. *J Enterocolitis*. 2025;4(Suppl 1):S87-S92.

Corresponding author: Gizem Dağcı, e-mail: gizemdagci22@hotmail.com

Received: January 19, 2025 **Accepted:** February 27, 2025

DOI: 10.14744/Jenterocolitis.2025.48430

Abstract

Intestinal failure (IF) is characterized by a decline in intestinal function that impairs the absorption of essential macronutrients, fluids, and electrolytes, necessitating parenteral or enteral nutritional support to sustain normal physiology and growth. Although advancements in diagnostic techniques and therapeutic approaches have reduced the prevalence of short bowel syndrome in inflammatory bowel disease (IBD), IBD remains a leading benign cause of chronic intestinal insufficiency. The management of these conditions—incorporating nutritional, pharmacological, and surgical strategies—is tailored to the specific anatomical and functional impairments of the intestine. A multidisciplinary approach, including supplementation with essential vitamins, minerals, and trace elements, is crucial for maintaining physiological stability. Total parenteral nutrition (TPN) and enteral nutrition should be administered under optimal conditions to carefully selected patients. However, intestinal transplantation has yet to achieve significant success rates in current clinical practice.

Keywords: Crohn's disease, intestinal failure, nutritional support

INTRODUCTION

The European Society for Clinical Nutrition and Metabolism (ESPEN) defines intestinal failure (IF) as a decline in intestinal function below the minimum level required for the absorption of macronutrients, water, and electrolytes, necessitating parenteral support to maintain health and growth. Although IF is uncommon, it imposes significant financial and personal burdens on both individuals and society.

Five pathological mechanisms are generally responsible for the occurrence of IF: short bowel syndrome (SBS), intestinal fistulas, dysmotility disorders (chronic intestinal pseudo-obstruction), small bowel mucosal disease, and mechanical obstruction.[1] While these conditions can contribute to the development of IF, short bowel syndrome accounts for over 60% of IF cases caused by benign etiologies and remains the leading indication for intestinal transplantation worldwide.[2-4]

INCIDENCE, PREVALENCE, AND ETIOLOGY

The annual incidence of IF is 2–3 per million, with a prevalence of 4 per million. The development of IF depends on the length of the intestine lost, its localization, and the adaptation process following the loss.[5,6]

CLASSIFICATION

The classification of intestinal failure is based on anatomical factors, such as the length and segment of resection, and functional factors, including the duration of functional loss (Table 1).[7-9]

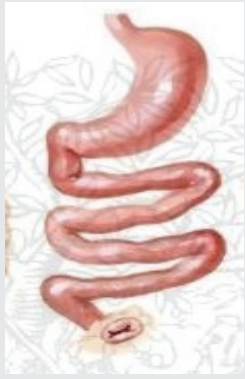
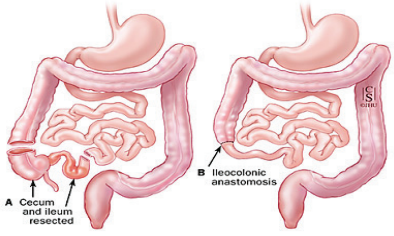
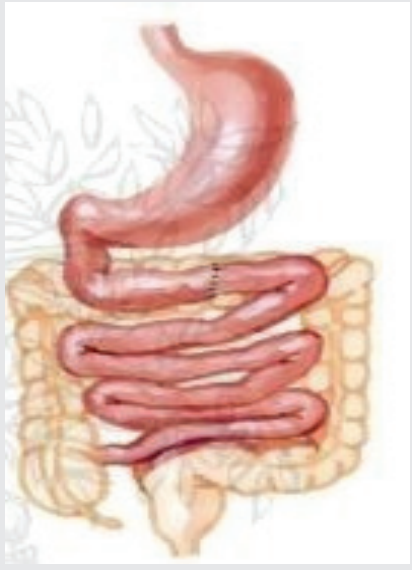
PATHOPHYSIOLOGY

SBS typically manifests clinically when the remaining small intestine measures less than 150–200 cm. Nutritional outcomes are significantly influenced by residual anatomy, as well as patient- and treatment-related factors. The distribution of micro- and macronutrient absorption within the intestinal lumen is summarized in Table 2.

Jejunal resection alone is generally well tolerated in cases where the jejuno-ileostomy is intact (i.e., the colon and ileocecal valve remain functional). A small intestine longer than 35 cm may be sufficient for adequate absorption. However, if the colon remains intact but ileocecal resection has been performed (jejuno-ileo-colostomy), patients may experience bile salt diarrhea, fatty diarrhea, and vitamin B12 deficiency. In such cases, a minimum of 60 cm of small intestine is required. In cases of end-jejunostomy, at least 100 cm of small intestine should be preserved. Additionally, an adaptation protocol should include at least 200 cm of small intestine along with partial colon resection.[6,10,11]

If less than 60 cm of bowel remains or only the duodenum is preserved (i.e., massive resection), total parenteral nutrition (TPN) and oral therapy support are required at home.

Table 1. Classification of Short Bowel Syndrome[3,4,10]

SHORT BOWEL SYNDROME CLASSIFICATION		
Anatomical Classification	**Type I:** Terminal jejunostomy (if the remaining bowel length is <50 cm).	
	Type II: Jejunoascendingostomy, jejunotransversostomy, jejunodescendostomy.	
	Type III: Jejunoileotransversostomy.	
Functional Classification	**Type I** Acute and short-term. A self-limiting condition, usually seen in the perioperative setting and/or with critical illness, where patients require PN for several days or weeks.	
	Type II: Prolonged acute condition, usually occurring in metabolically unstable patients and requiring complex multidisciplinary treatment and parenteral support for weeks to months.	
	Type III: Chronic condition found in metabolically stable patients who require intravenous substitution for months or years.	

MAIN POINTS

- Inflammatory bowel disease is one of the most common benign causes of short bowel syndrome.
- Managing patients with short bowel syndrome requires a multidisciplinary approach.
- Treatment options include medical nutrition therapy, symptomatic agents, teduglutide, and, in selected cases, intestinal transplantation at specialized centers.

Table 2. Distribution of micro and macronutrient absorption within intestinal and colonic lumen

Duodenum	Ca, Mg, Fe, Zinc
Jejunum	Disaccharides, Protein, Fat-soluble vitamins A, D Water-soluble vitamins B1, B2, B6, C, Folic acid
Ileum	Fat, B12, Bile salts
Colon	Water and electrolytes

ANATOMICAL AND PHYSIOLOGICAL ADAPTATION TO RESECTION

Diagnostic Challenges in Intestinal Failure

The diagnosis of intestinal failure aims to confirm SBS or chronic intestinal failure (CIF) by evaluating postoperative bowel anatomy, including intraoperative bowel length and radiological assessments. It also involves assessing the absorptive capacity of the remaining intestine, identifying specific nutrient deficiencies, and detecting any complications related to enteral nutrition (EN) or parenteral nutrition (PN), if present.

TREATMENT OF SHORT BOWEL SYNDROME DUE TO INFLAMMATORY BOWEL DISEASE

Nutritional Support Therapy

Patients with a limited ileal resection (<100 cm), with or without a right hemicolectomy, are generally able to tolerate solid food during the late postoperative phase. In SBS patients who have undergone a colectomy, the diet may include any fat-to-carbohydrate ratio, provided it contains low levels of mono- and disaccharides. Conversely, for SBS patients without a colectomy, the diet should prioritize complex carbohydrates, minimize mono- and disaccharides, and incorporate medium-chain fatty acids. The addition of soluble fiber to enhance intestinal absorption is not recommended for SBS patients.

Among trace elements, zinc deficiency should be corrected with supplementation of 220–440 mg/day. In cases of selenium deficiency, cardiomyopathy, peripheral neuropathy, and proximal muscle weakness may develop, necessitating careful monitoring. High doses of fat-soluble vitamins A and D (10,000–50,000 U/day) and vitamin E may be required for replacement. Vitamin K deficiency can manifest as hemolysis. In patients who have undergone colon resection (as colonic bacteria synthesize approximately 60% of vitamin K), a daily replacement dose of 1 mg should be administered. Since magnesium is primarily stored intracellularly, magnesium deficiency can occur even when blood levels appear normal.

Lactose should be eliminated from the diet of SBS patients only if clinical evidence of intolerance is present (e.g., a clear correlation between lactose intake and diarrhea or increased stoma output). Because patients with SBS have a negative calcium balance, a daily intake of 800–1,500 mg of Ca^{2+} is recommended.

To reduce steatorrhea and malabsorption, medium-chain fatty acids (MCFAs), which do not require micelle formation for absorption, should be incorporated into the diet to help minimize caloric deficits. A polymeric isotonic enteral diet is recommended as the primary option for managing SBS patients undergoing EN therapy.

Table 3. Phases of Intestinal Adaptation After Resection

Phases of Intestinal Adaptation After Resection	
1. Acute (Hypersecretory) Phase	**During the first postoperative days:** • Poor absorption of water, electrolytes, proteins, carbohydrates, fats, vitamins, and trace elements, with fluid loss reaching up to 5 liters per day (6-8 liters in patients with a jejunostomy). • **Gastric hyperacidity (GH):** Increased serum gastrin levels postoperatively, along with hyperacidity due to impaired secretion of vasoactive intestinal peptide (VIP). • **Proximal small bowel resection →** Significant reduction in serum cholecystokinin and secretin levels. • **Resection of the terminal ileum and proximal colon →** Reduced enterohormones (GLP-1, GLP-2) due to L-cell deficiency.
2. Residual Bowel Adaptation Phase	□ **Distal resection → Elicits a greater adaptive response compared to proximal resection (fluid loss <2.5 L/day).** □ **Within two years post-resection, 90-95% of the adaptation potential of the remnant intestine is achieved.** □ **Stabilization phase → Reduced diarrhea due to increased adaptation.** □ **Adaptation mechanisms → Include enterotrophic hormones and growth factors such as GLP-2**
3. Stable (Chronically Adapted) Phase	□ **CIF** develops in **50% of long-term intestinal failure cases**. □ **Patients remain metabolically stable** but may require **PN for months or years** (reversible CIF) or even lifelong support (irreversible CIF). □ **Home Parenteral Nutrition (HPN)** is the mainstay of therapy. At this stage, symptoms of **short bowel syndrome** are generally managed with medical treatment. □ **HPN discontinuation rates:** Approximately **50% in adults** and **73% in children**.

Table 4. Diagnostic Tests in Intestinal Failure

Diagnostic Tests in Intestinal Failure	
Diarrhea	Stool frequency, stool weight, and 24-hour steatorrhea/creatarrhea measurement
Absorptive surface area	D-xylose absorption test and/or serum citrulline (controversial)
Evaluation of Early onset nutrient deficiencies	Urinary zinc and magnesium concentrations, serum iron, total iron-binding capacity, ferritin, folic acid, calcium, phosphate, and copper analysis
Presence of Steatorrhea	Serum concentration of vitamins A, D, and E
Late-Onset Deficiencies	Vitamin B12 (liver stores are sufficient for 3-5 years)

Table 5. Symptomatic Treatment Approaches in Short Bowel Syndrome[1,10,17]

Gastric hypersecretion	Ppi	20-40 mg i.v(p.o)
	Clonidine	2*75-150 µg s.c/p.o
	Octreotide	3-4*50-100 µg s.c
Hypermotility	Loperamide	4-6 mg p.o (maximum 16 mg/day)
	Diphenoxylate	4*2.5-7.5 mg (maximum daily dose 20-25 mg)
	Codeine	30 mg p.o
Secretory diarrhea	Octreotide	2-3*50-100 µg s.c
	Budesonide	3*3 mg p.o.
	Clonidine	2*75-150 µg s.c
Fat malabsorption	Pancrealipase	40000 IU with meals
Lactose malabsorption	Lactase replacement	According to seriousness
Bile acid malabsorption	Cholestyramine, Colestipol	2-4 g/day, 1-2g/day; with meals

TPN should initially be administered continuously for 10–12 hours postoperatively and then gradually tapered at 30–60-minute intervals. For patients requiring long-term TPN, Hickman catheters or central venous catheters with a subcutaneous reservoir should be used.[12] Transitioning to enteral nutrition while on TPN helps prevent intestinal atrophy and accelerates adaptation.[13,14] In some patients, this adaptation period may last 1–2 years.

Antisecretory drugs and antimotility agents are frequently utilized for symptomatic management (Table 5).[6,15-17]

Hormonal Therapy

According to ESPEN recommendations, in patients with SBS, if PN is still required and the patient remains stable after the postoperative adaptation period (typically 12–24 months after the last resection), intestinal growth factor therapy should be considered.

Before initiating treatment, all patients should undergo colonoscopy (to evaluate the presence of polyps, assess disease activity, and exclude neoplasia if the remnant colon and/or rectum is present), abdominal ultrasonography, and gastroscopy.[18] For SBS patients who are candidates for intestinal growth factor therapy, teduglutide (TED)—the only glucagon-like peptide-2 (GLP-2) analogue currently approved by the FDA and EMA—should be the first-line treatment.[19]

In a study conducted by Sato et al. involving 18 patients with Crohn's disease and TPN-dependent SBS, TED was administered at a dose of 0.05 mg/kg/day. Over a 4- and 8-week observation period, a >20% reduction in TPN volume was observed in five (31.8%) and seven (43.8%) patients, respectively.[20,21]

CHRONIC COMPLICATIONS IN SHORT BOWEL SYNDROME

Patients with intestinal failure may experience several chronic complications, including nephrolithiasis, cholelithiasis, bacterial overgrowth, choleretic diarrhea, intestinal failure-related liver disease (IFALD), osteomalacia, osteoporosis, and catheter-related complications (Table 6).[1,10,22,23]

SURGICAL TREATMENT

These treatments should be considered a last option before intestinal transplantation and should only be performed in experienced centers. So far, studies comparing medical and surgical treatments have not been conducted. Bianchi (longitudinal intestinal lengthening and tailoring, LILT) and STEP (serial transverse enteroplasty) are the most well-known approaches among autologous intestinal reconstruction procedures. These methods can be used in the presence of dilated intestinal segments and aim to increase the absorptive surface area.[24,25]

INTESTINAL TRANSPLANTATION

The principal indication for intestinal transplantation (IT) is parenteral nutrition-dependent short bowel syndrome, particularly when associated with the progression of liver disease. It is defined by the persistence of hyperbilirubinemia (>4.5 mg/dL) for more than two months despite intravenous lipid modification strategies, decreased synthesis function (subnormal albumin or INR), portal hypertension, hypersplenism, thrombocytopenia, or any combination of these persisting for more than one month without infection.[24-28]

Other major indications for intestinal transplantation include thrombosis of three out of four central veins (left subclavian, right subclavian, left internal jugular, and right internal jugular), invasive intra-abdominal desmoid tumors, acute diffuse intestinal infarction with liver failure, bacterial sepsis with fungal sepsis or septic shock, and a history of two or more episodes of catheter-related sepsis.[26-29]

INFLAMMATORY BOWEL DISEASES AND INTESTINAL TRANSPLANTATION

Ulcerative colitis (UC) is an uncommon indication for IT, accounting for less than 1% of patients on home parenteral nutrition and 1.3% of IT candidates in Europe. In contrast, Crohn's disease (CD) represents 11% of adult intestinal transplants, making it the second most frequent benign indication for the procedure.

A key consideration for IT in CD patients is the potential recurrence of inflammatory bowel disease (IBD) in the transplanted graft. Histological signs of CD have been detected in graft biopsies as early as 20 days after transplantation; however, the incidence of clinically significant recurrence remains relatively low, at approximately 10%. The effect of recurrent CD on long-term graft performance and patient survival, however, remains unclear.[30]

CONCLUSION

Intestinal failure may develop in Crohn's disease due to extensive involvement or enteroenteric fistulas, as well as from anatomical loss following resections. Providing adequate support for vitamins, minerals, and trace elements through a multidisciplinary approach is essential. TPN and enteral nutrition should be administered to suitable patients under appropriate conditions. Intestinal transplantation, however, has not achieved significant success under current conditions.

Table 6. Chronic Complications and Special Conditions in Intestinal Insufficiency[1,10,21,22]

Chronic Complications and Special Conditions in Short Bowel Syndrome/Intestinal Insufficiency		
Complication	**Etiology/ risk factor**	**Recommendation/ Treatment**
Nephrolithiasis and enteric hyperoxaluria	As a result of wide ileal resection (>60 cm), ileal bypass, fat and bile acids pass into the colon, decreasing free calcium and increasing oxalate	Limiting oxalate intake in the diet (50 mg/day, limiting the intake of foods such as spinach, chocolate, cola, cocoa), reducing fat intake and preferring medium-chain fatty acids, targeting daily calcium intake (1000-1,200 mg)
Cholelithiasis	Prolonged fasting, changes in body weight, drugs such as opiates, lipid emulsions (in TPN), alteration of enterohepatic circulation by the absence of ileum and/or ileocecal valve after surgical resection	Increase oral intake, cholecystectomy in cholecystitis
Bacterial overgrowth	Bile acid deconjugation, steatorrhea, and fat-soluble vitamin malabsorption associated with delayed intestinal adaptation	In the presence of clinical suspicion, intermittent-alternative antibiotic therapy for 7-10 days (**rifaximin 550 mg 3*1, 14 days-first choice.** An alternative: metronizadol 3*250 mg, TMP-SMX160/800 mg 2*1, ciprofloxacin 500 mg 2*1)
D-Lactic Acidosis	Only in patients with an intact colon; Decrease in colonic pH causes proliferation of gram-positive, acid-fast, D-lactate-producing anaerobes (e.g., Bifidobacterium, Lactobacillus); neurological symptoms such as visual disturbances, confusion	The diagnosis is made by determining the levels of D-lactate in the blood. Due to the similarity of the symptoms of D-lactic acidosis to those of Wernicke's encephalopathy, prophylactic thiamine administration
Liver Disease Associated with Intestinal Insufficiency	More than 50% of patients develop severe liver disease after >5 years of TPN. May manifest as cholestasis, steatosis, steatohepatitis	- Prevention; Cyclical application of PN, avoidance of overfeeding, hepatoxic drugs, and toxins**,** prevention of sepsis, preservation of intestinal residual, maintenance of oral/ enteral nutrition, evaluation of distal enteral nutrition reinfusion in patients without small bowel incontinence - Treatment; UDCA, linoleic acid, avoidance of excessive (>40 kcal/kg/ day) dextrose and lipid (>1 g/kg/day) infusion, intestinal transplantation
Catheter-related complications	Sepsis, thrombosis, occlusion: Fever, around the catheter or Erythema at the tunnel entrance, slowing of infusion rate, extravasation, induration at the exit site	- In prevention; appropriate catheter (tunneled, Hickmann) care and use. In treatment, warfarin, flushing with tPA, ethanol, or HCl in non-thrombotic occlusion
Metabolic Bone Disease	Calcium and vitamin D malabsorption, immobilization	Calcium and vitamin D monitoring, replacement, annual bone densitometry, intravenous bisphosphonate therapy if indicated

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – F.A., G.D.; Design – F.A., G.D.; Literature Review – G.D.., F.A.; Writing– G.D., F.A.,; Critical Review – F.A.

Use of AI for Writing Assistance: Not declared.

Conflict of Interest: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study received no financial support.

REFERENCES

1. Pironi L, Cuerda C, Jeppesen PB, et al. ESPEN guideline on chronic intestinal failure in adults - Update 2023. *Clin Nutr*. 2023;42(10):1940-2021. [CrossRef]
2. Pironi L. Definitions of intestinal failure and the short bowel syndrome. *Best Pract Res Clin Gastroenterol*. 2016;30(2):173-185. [CrossRef]
3. Massironi S, Cavalcoli F, Rausa E, Invernizzi P, Braga M, Vecchi M. Understanding short bowel syndrome: Current status and future perspectives. *Dig Liver Dis*. 2020;52(3):253-261. [CrossRef]
4. Lakkasani S, Seth D, Khokhar I, Touza M, Dacosta TJ. Concise review on short bowel syndrome: Etiology, pathophysiology, and management. *World J Clin Cases*. 2022;10(31):11273-11282. [CrossRef]
5. Dibaise JK, Young RJ, Vanderhoof JA. Enteric microbial flora, bacterial overgrowth, and short-bowel syndrome. *Clin Gastroenterol Hepatol*. 2006;4(1):11-20. [CrossRef]
6. Jeppesen PB, Mortensen PB. The influence of a preserved colon on the absorption of medium chain fat in patients with small bowel resection. *Gut*. 1998;43(4):478-483. [CrossRef]
7. Grainger JT, Maeda Y, Donnelly SC, Vaizey CJ. Assessment and management of patients with intestinal failure: A multidisciplinary approach. *Clin Exp Gastroenterol*. 2018;11:233-241. [CrossRef]
8. Kappus M, Diamond S, Hurt RT, Martindale R. Intestinal failure: New definition and clinical implications. *Curr Gastroenterol Rep*. 2016;18(9):48. [CrossRef]
9. Pironi L, Arends J, Baxter J, et al. ESPEN endorsed recommendations. Definition and classification of intestinal failure in adults. *Clin Nutr*. 2015;34(2):171-180. [CrossRef]
10. Aksan A, Farrag K, Blumenstein I, Schröder O, Dignass AU, Stein J. Chronic intestinal failure and short bowel syndrome in Crohn's disease. *World J Gastroenterol*. 2021;27(24):3440-3465. [CrossRef]
11. Solar H, Ortega ML, Gondolesi G. Current status of chronic intestinal failure management in adults. *Nutrients*. 2024;16(16):2648. [CrossRef]
12. Leiberman D, Stevenson RP, Banu FW, Gerasimidis K, McKee RF. The incidence and management of complications of venous access in home parenteral nutrition (HPN): A 19 year longitudinal cohort series. *Clin Nutr ESPEN*. 2020;37:34-43. [CrossRef]
13. Cawsey SI, Soo J, Gramlich LM. Home enteral nutrition: Outcomes relative to indication. *Nutr Clin Pract*. 2010;25(3):296-300. [CrossRef]
14. Joly F, Dray X, Corcos O, Barbot L, Kapel N, Messing B. Tube feeding improves intestinal absorption in short bowel syndrome patients. *Gastroenterology*. 2009;136(3):824-831. [CrossRef]

15. Woolf GM, Miller C, Kurian R, Jeejeebhoy KN. Diet for patients with a short bowel: High fat or high carbohydrate? *Gastroenterology*. 1983;84(4):823-828. [CrossRef]
16. Jeppesen PB, Høy CE, Mortensen PB. Deficiencies of essential fatty acids, vitamin A and E and changes in plasma lipoproteins in patients with reduced fat absorption or intestinal failure. *Eur J Clin Nutr*. 2000;54(8):632-642. [CrossRef]
17. Nehra V, Camilleri M, Burton D, Oenning L, Kelly DG. An open trial of octreotide long-acting release in the management of short bowel syndrome. *Am J Gastroenterol*. 2001;96(5):1494-1498. [CrossRef]
18. Pevny S, Pape UF, Elezkurtaj S, et al. De novo development of distal jejunal and duodenal adenomas after 41 months of teduglutide treatment in a patient with short-bowel syndrome: A case report. *JPEN J Parenter Enteral Nutr*. 2021;45(3):652-656. [CrossRef]
19. Pape UF, Iyer KR, Jeppesen PB, et al. Teduglutide for the treatment of adults with intestinal failure associated with short bowel syndrome: Pooled safety data from four clinical trials. *Therap Adv Gastroenterol*. 2020;13:1756284820905766. [CrossRef]
20. Siu RK, Karime C, Hashash JG, Kinnucan J, Picco MF, Farraye FA. Improved outcomes associated with teduglutide use in patients with both short bowel syndrome and Crohn's disease. *Crohns Colitis 360*. 2024;6(1):otae007. [CrossRef]
21. Sato T, Uchino M, Takeuchi J, et al. Short-term clinical evaluation of teduglutide for patients with Crohn's disease on home parenteral support for postoperative short bowel syndrome with intestinal failure. *Clin Nutr*. 2023;42(5):722-731. [CrossRef]
22. Chungfat N, Dixler I, Cohran V, Buchman A, Abecassis M, Fryer J. Impact of parenteral nutrition-associated liver disease on intestinal transplant waitlist dynamics. *J Am Coll Surg*. 2007;205(6):755-761. [CrossRef]
23. Iyer K, DiBaise JK, Rubio-Tapia A. AGA clinical practice update on management of short bowel syndrome: Expert review. *Clin Gastroenterol Hepatol*. 2022;20(10):2185-2194.e2. [CrossRef]
24. Selvaggi G, Gyamfi A, Kato T, et al. Analysis of vascular access in intestinal transplant recipients using the Miami classification from the VIIIth International Small Bowel Transplant Symposium. *Transplantation*. 2005;79(12):1639-1643. [CrossRef]
25. Mlaver E, Smith SR, Matar AJ, et al. Serial transverse enteroplasty in adults with parenteral nutrition dependence: A case series. *JPEN J Parenter Enteral Nutr.* 2024;48(8):965-973. [CrossRef]
26. Kaufman SS, Atkinson JB, Bianchi A, et al. Indications for pediatric intestinal transplantation: A position paper of the American Society of Transplantation. *Pediatr Transplant*. 2001;5(2):80-87. [CrossRef]
27. Secor JD, Yu L, Tsikis S, Fligor S, Puder M, Gura KM. Current strategies for managing intestinal failure-associated liver disease. *Expert Opin Drug Saf*. 2021;20(3):307-320. [CrossRef]
28. Johnson E, Vu L, Matarese LE. Bacteria, bones, and stones: Managing complications of short bowel syndrome. *Nutr Clin Pract*. 2018;33(4):454-466. [CrossRef]
29. Kaufman SS, Avitzur Y, Beath SV, et al. New insights into the indications for intestinal transplantation: Consensus in the year 2019. *Transplantation*. 2020;104(5):937-946. [CrossRef]
30. Limketkai BN, Orandi BJ, Luo X, Segev DL, Colombel JF. Mortality and rates of graft rejection or failure following intestinal transplantation in patients with vs without Crohn's disease. *Clin Gastroenterol Hepatol*. 2016;14(11):1574-1581. [CrossRef]