Review Article

Medical nutrition therapy for adult intestinal failure: A review of current perspectives

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Intestinal failure (IF) is a clinical condition resulting from intestinal resection or disease-associated malabsorption or dysmotility. It is characterized by the reduction of functional gut capacity below the minimum needed for adequate digestion and absorption of nutrients. The priority of management is maintaining optimal nutritional and metabolic support until maximum bowel adaptation. The nutritional intervention is the cornerstone of treatment for IF, incorporating the parenteral nutrition (PN) support, enteral nutrition (EN) and oral feeding regimen. The individualized nutritional strategy requires a multidisciplinary approach and should be tailored to the specific pathophysiological characteristics.

Key Words: intestinal failure, adult, nutritional management

INTRODUCTION

The term of intestinal failure (IF) was coined in 1950s and first defined by Fleming and Remington as a state of "reduction in the functioning gut mass below the minimal amount necessary for adequate digestion and absorption of food".[1] Since then, with the greater awareness and recognition of this medical condition over the next few decades, the concept of IF has been continuously expanded and enriched. The National Health Service (NHS) in England has renewed definition of IF in 2008, which comprises a group of disorders characterized by an insufficiency to maintain adequate nutrition via the intestines. The condition results from various causes, including obstruction, abnormal motility, major surgical resection, congenital defect or disease-related loss of intestinal digestion and absorption.[2] In 2015, the European Society for Clinical Nutrition and Metabolism (ESPEN) defined IF as "the 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".[3]

IF is a long-recognized complication resulting from the short bowel syndrome, which leads to malabsorption after extensive resection of the small intestine. Apart from this rare condition,[4] IF is actually a common complication in various clinical settings, as the definition of IF has been revised and encompassed a constellation of medical conditions when the nutritional demands of the body are not met by the gastrointestinal absorptive function.[5] The wide range of etiologies comprise anatomic loss and/or functional impairment in intestinal motility or absorptive capacity, including Crohn's disease, vascular ischemia, radiation enteritis, intestinal obstruction, motility disorders, congenital defects and so on.[5] Moreover, as one of the clinical manifestations of multiple organ failure, IF has been considered prevalent among critical ill patients, albeit remained undiagnosed to some degree.[6] The acute gastrointestinal injury (AGI) was defined by the Working Group on Abdominal Problems (WGAP) of the European Society of Intensive Care Medicine (ESICM) in 2012 as malfunctioning of the GI tract in intensive care patients due to their acute illness.[7] Four grades of AGI severity were identified, with presence of gastrointestinal failure in patients with AGI grades III and IV.7 A meta-analysis estimated the prevalence of AGI in the critically ill patients at 40%.[8] Another recent investigation showed that the symptoms of gastrointestinal dysfunction developed in 60% of mechanically ventilated patients.[9]

Based on the metabolic change, clinical course and outcomes, etiopathogenesis and dependence on parenteral nutrition, IF was further categorized into functional, pathophysiological and clinical classifications (Table 1).[3] The type 1 IF is a short-term and usually self-limiting condition which lasts for less than 1 month. The postoperative ileus, acute intestinal obstruction, anastomotic leak or intestinal injury often accounted for the commonest etiologies of type 1 IF, occurring in 10-50% of postoperative patients.[10,11] The type 2 IF usually refers to IF related to a prolonged unstable condition which lasts for greater than 1 months and requires parenteral nutrition. It often involves patients with complex Crohn's disease, intestinal fistulas, or abdominal sepsis.[10,12] The type 1 and 2 IF

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Table 1. Classification of intestinal failure

Functional classification	Determined by the onset, metabolic and expected outcome criteria			
Type 1	• Acute, short-term, transient and usually self-limiting condition			
Type 2	• Prolonged acute condition with unstable metabolism, requiring complex multi-disciplinary care and parenteral nutritional support for weeks or months			
Type 3	• Reversible or irreversible chronic condition with stable metabolism, requiring parenteral nutritional support for months or years.			
Pathophysiological classification	Determined by the pathophysiological mechanism and etiopathogenesis (various underlying gastrointestinal or systemic diseases)			
	• Short bowel • Intestinal fistula • Intestinal dysmotility • Mechanical obstruction • Extensive small bowel mucosal disease			
Clinical classification of chronic IF	Determined by the requirements for energy and the volume of intravenous supplementation			
Intravenous energy supplementation (the mean of kcal/kg/d)	The mean volume of intravenous supplementation (ml)			
	≤1000	1001-2000	2001-3000	>3000
0 (A)	A1	A2	A3	A4
1-10 (B)	B1	B2	B3	B4
11-20 (C)	C1	C2	C3	C4
>20 (D)	D1	D2	D3	D4

comprise acute IF, while the type 3 IF refers to a chronic stable condition that requires long-term parenteral support. It may be irreversible and occurs as a consequence of short bowel syndrome. The frequently reported etiologies for type 3 IF include loss of length of small bowel from a massive small bowel resection or loss of absorptive capacity of the small bowel due to the inflammatory bowel diseases.[13]

Characterized as the inability to maintain fluid, energy, protein, electrolytes or micronutrient balance, IF is a devastating condition with multiple complex complications and comorbidities. The management of IF requires an extensive multidisciplinary approach provided by the gastroenterologists, surgeons, nutrition physicians or registered dietitians, nutrition support nurses and pharmacists.[14] Medical nutrition therapy, as an indispensable life-sustaining management for IF, can enhance the prospect of achieving enteral autonomy and intestinal adaptation. The nutrition program should be customized and tailored to the individual patients by route and quantity, depending on the onset time, metabolic effect, degree, classification and stage of their gastrointestinal dysfunction.[10] The goal of nutritional management for IF is to provide adequate macronutrients and micronutrients, maintain rehydration and electrolyte balance, with weaning from parenteral nutrition and achieving enteral autonomy in the long run.[15]

NUTRITIONAL SCREENING AND ASSESSMENT

At baseline, nutritional risk screening should be the commencement of a routine clinical protocol that results in a plan of intervention if the screening result is positive.[16] The present screening tools that are most frequently recommended include the Nutritional Risk Screening 2002 (NRS 2002) for the inpatient setting, the Malnutrition Universal Screening Tool (MUST) for the ambulatory setting and the Mini Nutritional Assessment (MNA) for hospitalized geriatric patients. All of them combine dynamic parameter, typically recent weight loss, body mass index (BMI), food intake, and a grading of on-going disease severity.[16,17] The Global Leadership Initiative on Malnutrition (GLIM) criteria, a two-step model for risk screening and malnutrition diagnosis assessment, has been positioned within the nutritional care.[18] It consists three phenotypic criteria (non-volitional weight loss, low body mass index, and reduced muscle mass) and two etiologic criteria (reduced food intake or assimilation, and inflammation or disease burden).[18]

Moreover, patients with nutritional risk should subsequently undergo a more comprehensive nutritional assessment to determine the hydration and nutritional status of a patient with IF and to estimate the individual requirements.[14] Anthropometric measurement provides important indicators for nutritional assessment, including body weight (actual, ideal, adjusted), BMI, arm circumference, skin fold thickness as well as body composition assessed by dual-energy X-ray absorptiometry (DXA) or bioelectrical impedance analysis (BIA). In addition, a wide range of laboratory parameters are incorporated in the nutrition assessment and help to detect vitamins and trace elements deficiencies, electrolyte disturbance, dehydration and protein-energy malnutrition.[17] Moreover, clinical evaluation constitutes another essential component of nutritional assessment. The aspects influencing food intake, digestion, absorption and metabolic expenditure should be scrutinized, such as sex, age, presence of concurrent sepsis, the intestinal anatomy (e.g., remaining bowel length, enterostomies, presence of ileocecal valve) and so on.[14]

Following the detailed nutritional screening and assessment, the nutritional care plan might be implemented, with the route, quantification and duration of nutritional support depending on the patients' metabolic condition, underlying diseases and stage of IF. The screening and assessment should be repeated regularly to evaluate the efficacy, safety and feasibility of the nutrition intervention, and to guide and improve the management for patients with IF.

DEFINING NUTRITIONAL NEEDS

The indirect calorimetry has been recommended to measure accurate energy expenditure,[17] as is for critical ill patients in ICU. ESPEN has recommended caloric intake of 25 to 35 kcal/kg ideal body weight/day for patients with IF depending on estimated requirement in different disease phase,[17] if the indirect calorimetry is not available. Besides, the sufficient intake of either protein or equivalent quantity of amino acids is considered advantageous, with ingestion as high as up to 1.5 g/kg actual body weight/day.[17] In addition, the micronutrients (vitamins and trace elements) and electrolytes should be administered in the medical nutritional therapy to meet the metabolic needs. Patients with acute IF (type 1) might have concomitant sepsis, systemic inflammatory response syndrome (SIRS) or/and experience surgical trauma, which consists the classic ebb and flow phase of metabolic alteration.[19-21] Accordingly, the nutritional care should be also tailored to the evolution of the metabolic response to injury. In early acute stage of IF, the 'ebb' phase is characterized by hemodynamic instability and hormonal changes (especially insulin resistance), which benefits from a relatively hypocaloric, high-protein strategy to minimize muscle catabolism.[19] In contrast, in a later or prolonged stage of IF (type 2, type 3, and late phase of type 1 IF), the "flow" phase occurs after resuscitation from a state of shock, which involves an increased metabolic turnover and breakdown of lean muscle tissue.[19] This hypercatabolic condition leads to a significantly increased energy expenditure, which needs more aggressive calorie delivery and continuously sufficient protein provision.[19]

PARENTERAL NUTRITION (PN)

In light of nutritional support, it is widely accepted that the preferred hierarchy generally ranges from oral feeding to enteral nutrition and to parenteral nutrition. However, most patients with type 1 and type 2 IF would be expected to depend on PN support preferably for a certain period and return to full intestinal autonomy with time; while the type 3 IF usually involves patients with established short bowel syndrome who demand long-term PN.[10] Hence, PN is the main nutritional support, temporarily for the early stage of acute IF (type 1 and type 2 IF) and persistently for the chronic IF (type 3 IF), if enteral nutrition cannot be adequately established.[22]

The PN formulations are often compounded as an "all-in-one" nutrient admixture, containing dextrose, amino acids, lipid, micronutrients, electrolytes and water. The amount of dextrose intravenously infused is usually 150-200g/day for adults, with increments made over 72–96 hours based on energy requirements and glycemic fluctuation.[23,24] The amino acid requirement for PN patients is usually quoted as 0.8–1.5 g/kg/day for adults,[24] depending on nutritional status (mild, moderate, or severe malnutrition), metabolism (normal, increased or elevated), the actual enteral ingestion and underlying clinical conditions.[25] The intravenous lipid emulsions are provided as another integral component in PN regimens, often prescribed with 1 g/kg/day for adults.[24,26] Although the soybean oil-based lipid emulsion is most commonly administered in PN algorithm, supplementation with omega-3 polyunsaturated fatty acids (PUFA) from fish oil has been currently recommended by ESPEN guidelines on PN for surgical and critically ill patients.[17,27] In addition, micronutrients (vitamins and trace elements) should be adequately supplemented via PN, especially when no oral diet or enteral nutrition is taken.[28] Still, the PN should also be adjusted with the aim of normalizing fluid, electrolyte and mineral balance.[29] As for patients with prolonged irreversible IF (type 3), home PN (HPN) support is indicated to maintain long-term fluid, nutritional, and metabolic balance, improve quality of life and promote rehabilitation.[28,30]

On the other hand, patients with dependence on long-term PN are at risk of various complications, including disturbances in fluid and electrolyte status, development of cholestasis and hepatic dysfunction, venous line complication and catheter related blood stream infection (CRBSI).[24,28,31] Careful monitoring of patients' condition and the PN formula can help prevent or treat these complications. In addition, the intestinal failure associated liver disease (IFALD), one of the most common and serious complications of long-term home PN, is prevalent in 15% to 40% of adults with IF.[32] IFALD is defined as the development of abnormal liver function and/or presence of radiological and/or histological liver abnormalities occurring in an individual with IF, with absence of other primary parenchymal liver pathology, other hepatotoxic factors or biliary obstruction.[33] Contributing factors including long-term PN, sepsis, lack of enteral nutrition, nutrients deficiency or excess may play a role in development of the IFALD.[32,33] ESPEN has recommended nutritional approaches aimed at minimizing PN overfeeding and optimizing enteral nutrition to prevent and/or manage IFALD.[33,34] Furthermore, the restriction of parenteral lipid administered to less than 1 g/kg/day with reduced omega-6/omega-3 PUFA ratio, minimizing PN caloric overfeeding, supplementation of choline, carnitine and taurine in necessity should be also considered.[33,34]

ENTERAL NUTRITION (EN)

Enteral feeding is the route of choice in all patients with a functioning gastrointestinal tract. The oral feeding or EN plays a crucial part in exerting trophic effect on the bowel, preventing mucosal atrophy, preserving the immune system function and preventing bacterial translocation.[17] In actual practice, the EN feeding strategies for IF vary a lot from person to person, regarding the length of residual bowel, presence of ileocecal valve, presence of colon in continuity and the digestive and absorptive function of the intestine.[35] Generally, for those who cannot eat enough for more than a few days, sip feeds of oral nutritional supplements or artificial tube feeding become essential.[36] However, both oral feeding and enteral nutrition might be limited in acute IF (type 1 and 2 IF) due to the presence of inflammation, strictures, obstruction, radiation damage, short bowel syndrome, and intestinal fistulas.[10,37,38] As for patients with IF due to the short bowel syndrome, especially for patients with a high stoma output, the PN is recommended to maintain optimal nutritional status, correct electrolyte disturbance and promote quality of life in the acute postoperative phase, until there is hemodynamic stabilization with a switch to EN and later to an oral diet.[39,40]

EN should be initiated gradually, only in the circumstances when the hemodynamic stability is obtained, ostomy output or diarrhea <2 L/day, and the intestinal activity is restored.[23,38,41,42] With regards to the EN procedure, the use of enteral tube feeding in combination with oral feeding has been suggested by ESPEN.[29] Continuous enteral feeding is believed to be of nutritional benefit, for providing a better distribution and maximum exposure of the available intestinal surface area to nutrients while stimulating gastrointestinal secretions and endogenous hormonal secretions that are important to accelerate the intestinal adaptation.[23,29,41] In view of enteral nutritional solution, ESPEN has recommended polymeric diets for patients with chronic IF who are dependent on the tube feeding.[29] Previous studies have shown that polymeric diets are generally well tolerated for they are often less costly and less hyperosmotic than elemental diets.[43] The evidence from an animal study also has demonstrated the beneficial effect of the polymeric diets on promoting the intestinal adaptation.[43] Otherwise, the use of elemental feeding solutions is recommended in the case of gastrointestinal intolerance of polymeric formulas.[29]

ORAL FEEDING

Oral feeding of regular whole food diets might be resumed to different degree in IF patients with different remaining absorptive capacity, as a result of intestinal adaptation.[44] It is recommended that all patients with IF refer to dietary counselling guided by expert dietitians to ensure high compliance.[29,37,45] In most cases, frequent small meals are recommended to improve the absorption of nutrients. The dietary plan, as well as the diet composition, food volume and numbers of intakes should be adjusted to individual needs.

Oral fluid restriction to less than 1 liter per day is recommended for patients with high stomal output. Still, the salt could be consumed with food ad libitum to make up for sodium depletion.[46,47] In order to reduce ostomy volume in patients with high output jejunostomy, patients are often advised to restrict oral fluid intake at meal time (no liquid for half an hour before and after food intake); whereas oral sip feeds between meals may help to increase overall energy intake.[48,49]

Moreover, an isotonic high sodium oral rehydration solution is preferable to replace the stoma sodium losses. Previous clinical discoveries have found that oral intake of hypotonic solutions could not quench the patients' thirst but causes large stomal sodium losses; while hypertonic fluids also cause stomal losses of water and sodium.[50] Excessive oral intake of hypotonic (e.g. water, tea, coffee or alcohol) or hypertonic (e.g. fruit juices, colas) solutions may all aggravate stomal losses by stimulating fluid secretion or increasing the fluid and sodium influx into the intestinal lumen.[29,51]

As for macronutrients intake, consumption of complex carbohydrates (polysaccharides) rather than simple carbohydrates (added sugar) is advised to alleviate symptoms of diarrhea, flatulence and bloating for patients with short bowel syndrome. Besides, a diet low in fat is beneficial to relive the symptoms of feeding intolerance and promote the bowel adaptation. It has been found that unabsorbed long chain fatty acids in the colon are likely to exacerbate diarrhea and depletion of water and salt by reducing intestinal transit time,[52] decreasing water and sodium absorption,[53] having impacts on intestinal bacteria and thereby reducing carbohydrate fermentation.[54] The fat can also bind to calcium and magnesium, and increase oxalate absorption, which predisposes to the rising risk of enteric hyperoxaluria and urolithiasis.[50,55] Alternatively, beyond restriction of total fat ingestion, substitution of the medium chain triglycerides for long chain triglycerides as a source of energy has been demonstrated to improve energy absorption from the bowel and reduce the risk of renal stones.[56-59]

In addition, on account of malabsorption caused by IF, taking additional vitamin or mineral supplements is suggested especially for patients with short bowel syndrome, including calcium,[23,60-62] magnesium,[23,42,62,63] iron,[62] zinc,[23,42,64] vitamin A, B12, C, D, E and K.[23,42,59,65] Regarding other dietary supplements, use of the glutamine, probiotics and dietary fiber in management of IF patients has been discussed but not widely recommended, due to the remaining controversy of their effects in promoting the intestinal rehabilitation process.[29]

CONCLUSION

IF is a constellation of conditions with a great variability, both in etiology and in the clinical manifestations. It usually results from intestinal resection or disease-associated malabsorption or dysmotility, and characterized by the inability to maintain protein-energy, fluid, electrolyte or micronutrients balance. The priority of management is maintaining optimal nutritional and metabolic support until maximum bowel adaptation. The nutritional intervention is the cornerstone of treatment in these patients, including PN support, EN and oral dietary intake. The individualized nutritional regimen of IF requires a multidisciplinary approach and should be tailored to the patients' own clinical manifestation, duration of the condition and the pathophysiological characteristics.

AUTHOR DISCLOSURES

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