INVITED REVIEW

Acute Intestinal Failure

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Abstract

Acute intestinal failure (AIF), "reduction of gut function below the minimum necessary for the absorption of macronutrients and/or water and electrolytes, requiring parenteral nutrition", is common, but very often neglected part of multiorgan dysfunction syndrome (MODS) in the critically ill patients. It is now increasingly being recognized as a cause of prolonged ICU and hospital stay and poor outcome. Multidisciplinary team approach, systematic approach to management with treatment of sepsis, early mobilization and enteral feeding with prokinetics if required, control of intra-abdominal pressure and surgical intervention, when mandated, can help treat AIF and improve patient outcomes.

Keywords: Acute intestinal failure, Critically ill, Enteral feeding, Intra-abdominal pressure, Multiorgan dysfunction syndrome, Parenteral nutrition, Short bowel syndrome.

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बलमारोग्यमायुश्च प्राणाश्चाग्नौ प्रतिष्ठिताः।
अन्नपानेन्धनैश्चाग्निर्ज्वलति व्येति चान्यथा ॥ ३४२ ॥

(Balamarogyamayushcha pranashchangnau pratishthita, annapanendhanaishchagnirjwalati vyeti chanyatha)

Charaka Sutra Sthana, Chapter 28, verse 342

Strength, health, longevity, and vital breath are dependent on the power of digestion including metabolism. When supplied with fuel in the form of food and drinks, this power of digestion is sustained; it dwindles when deprived of it.

Introduction

The adverse effect of gut dysfunction on outcomes of the critically ill patients is being increasingly recognized. In a study looking at gastrointestinal (GI) complications in mechanically ventilated patients, Montejo found a high incidence of these complications, which led to feeding intolerance, malnutrition, and prolong hospital stay and poorer outcomes.[1] Mentec et al. reported that high gastric residual volume (GRV) signified upper digestive intolerance, which led to increased incidence of nosocomial pneumonia, prolonged ICU stay, and increased ICU mortality.[2] In the critical care unit, acute intestinal failure (AIF) can be a presenting symptom or can more commonly be a part of multiorgan dysfunction syndrome (MODS).

Acute intestinal failure can be very incapacitating for the patients and is caused by an anatomical problem (i.e., short bowel syndrome), or a physiological disorder of the GI system[3] increasing the mortality, morbidity, and financial burden. In comparison, the resection of large bowel does not significantly alter the patient outcomes.

There is no evidence to suggest that biomarkers may help to diagnosis of syndrome, AIF may remain underdiagnosed in the ICU but may be the cause of sepsis and multiple organ failure.[4] Moreover, none of the available scoring systems for MODS include the adverse impact of the intestinal dysfunction (i.e., its various functions, such as digestive, endocrinological, immunological, and barrier function).[5] In this review, we discuss the definition, causes, and management of AIF in the critically ill adults.

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Evolution of the Concept, Definition, and Terminologies

Before the MODS and systemic inflammatory response syndrome (SIRS) were described in 1990s, Dr Metchnikoff suggested that the entrance of microbes and associated toxins from the bowel into the body was an important cause of early death.[6] Irving and colleagues were the first to describe the pathophysiology and clinical condition called "intestinal failure" in 1980s.[6]

In 1981, Fleming and Remington defined "intestinal failure" (IF) as a state of "reduction in functioning gut mass below the minimal amount necessary for adequate digestion and absorption of food".[3]

Intestinal failure is defined 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*.[7] The group suggested that *the reduction of gut absorptive function that doesn't require intravenous supplementation to maintain health and/or growth, can be considered as "intestinal insufficiency"*.

Intestinal failure can be classified further into functional and pathophysiological varieties (Tables 1 and 2). The various contributors to the etiopathogenesis are summarized in Table 3.

Table 1: Classification of intestinal failure

Functional classification (based on onset, metabolic, and expected outcome criteria)	
Type I	Acute, short-term, and usually self-limiting
Type II	Prolonged acute condition, often in metabolically unstable patients, requiring complex multidisciplinary care and intravenous supplementation over periods of weeks or months
Type III	Chronic condition, in metabolically stable patients, requiring IV supplementation over months or years. It may be reversible or irreversible
Pathophysiological classification (due to gastrointestinal or systemic diseases)	
	Short bowel
	Intestinal fistula
	Intestinal dysmotility
	Mechanical obstruction
	Extensive small bowel mucosal disease

Table 2: Clinical classification of chronic intestinal failure

IV energy supplementation (kcal/kg body weight)	*Volume of the IV supplementation (mL)*			
	≤1,000	*1,001–2,000*	*2,001–3,000*	*>3,000*
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

On the basis of the requirements for energy and the volume of the intravenous supplementation (IV), chronic intestinal failure is categorized into 16 subtypes

The other ways to classify AIF are as follows:

Time of Presentation

- Congenital, e.g., gastroschisis.
- Acquired, e.g., due to surgical complications.

Speed of Onset

- Rapid, e.g., mesenteric ischemia.
- Prolonged, e.g., Crohn's disease.

Underlying Pathology

- Benign.
- Malignant.

Localization

- Localized to GI tract.
- Systemic disease.

Duration

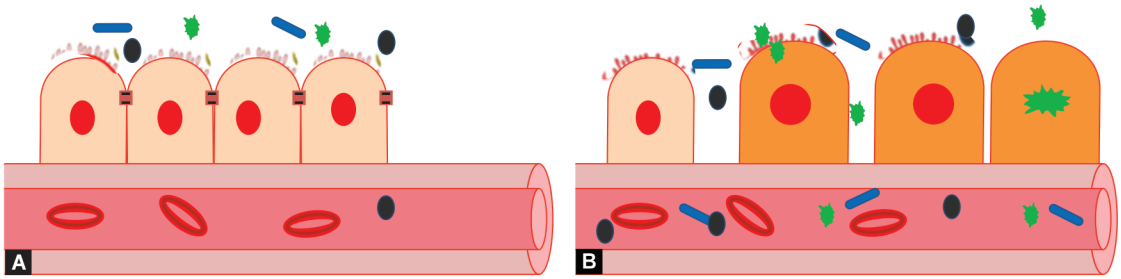
- Short-term.
- Long-term.

Table 3: Mechanisms of intestinal failure

Short bowel (reduced absorptive mucosal surface)	
Concomitant mechanisms	Increased intestinal losses of fluids and electrolytes (adjunctive mechanism in the case of end-jejunostomy)
	Restricted oral/enteral nutrition (to reduce intestinal losses)
	Disease-related hypophagia
	Lack of adaptive hyperphagia
	Accelerated gastrointestinal transit time
	Small bowel bacterial overgrowth
Intestinal fistula (bypass of large areas of absorptive mucosal surface)	
Concomitant mechanisms	Increased intestinal losses of fluids and electrolytes
	Disruption of the enterohepatic cycle
	Restricted oral/enteral nutrition or total fasting (bowel rest) to decrease fistula output
	Impaired intestinal peristalsis and increased metabolic demand related to concomitant sepsis and inflammation
Intestinal dysmotility: Restricted oral/enteral nutrition or total fasting from intolerance due to feeding-related exacerbation of digestive symptoms or to episodes of non-mechanical intestinal obstruction	
Concomitant mechanisms	Malabsorption due to small bowel bacterial overgrowth
	Increased intestinal secretion of fluids and electrolytes in the obstructed segments
	Increased intestinal losses of fluids and electrolytes due to vomiting, gastric drainage, and/or diarrhea
Mechanical obstruction: Incomplete or total fasting (bowel rest)	
Concomitant mechanisms	Increased intestinal secretion of fluids and electrolytes in the obstructed segments
	Increased intestinal losses of fluids and electrolytes with vomiting or gastric drainage
Extensive small bowel mucosal disease: Inefficient absorptive and/or nutrient losing mucosal surface	
Concomitant mechanisms	Increased intestinal losses of fluids and electrolytes
	Restricted oral/enteral nutrition
	Disease-related hypophagia

Pathophysiology

The GI tract performs numerous roles for the normal functioning of the body, such as, being a bacterial reservoir in the body, secretion and absorption of digestive juices, immunological function via gut-associated lymphoid tissue (GALT), and a mucosal barrier function. Any factors that impair these functions would cause AIF. Acute intestinal failure can occur either primarily due to direct



Figs 1A and B: Intestinal barrier function: (A) Normal intestinal barrier preventing intrusion by bacteria and allergens; (B) Inflammation promoting paracellular and intracellular intrusion of pathogens

consequence of organ injury or secondary to hypoperfusion. A vicious cycle of hypoperfusion and further organ injury is set in due to inflammatory response depending on the severity of the insult[5] (Fig. 1).

The intestinal mucosa barrier comprises mechanical, biological, and immune barriers and the factors that impair these barriers can lead to translocation of bacterial/endotoxin into blood and other normally sterile tissues. Hypoperfusion and ischemia/reperfusion injury alter the mucosal barrier and immune-inflammatory reaction, via release of biologically active factors into the blood as well as mesenteric lymphatics. Gut microbiota or products, such as, damage-associated molecular patterns (DAMPs) in lymphatic ducts and endotoxins in portal blood, can trigger distant organ dysfunction in patients with GI failure or dysfunction and can lead to poor outcomes.[8]

Reduction of transepithelial electrical resistance also attenuates the intestinal barrier function.[9] Increased sympathetic tone partially induces increased intestinal permeability leading to secondary AIF similar to that seen in traumatic brain injury.[5] Hormonal mediators of GI motility may be relevant in pathophysiology of AIF usually expressed as delayed gastric emptying.[10] During early phase, decreased plasma concentration of orexigenic (appetite-increasing) hormones (e.g., ghrelin) and increased anorexigenic hormones (e.g., PYY) has been demonstrated.[8]

Observational data demonstrate an association between change of the intestinal microbiome and critical illness. This can cause further GI dysfunction and worse clinical outcomes, though this needs to be confirmed by adequately powered studies.[8]

Epidemiology

The prevalence of AIF is difficult to quantify as it remains dependent on the definitions used.[5] Postoperative ileus is type I IF and probably occurs in 10–30% of postoperative patients.[3] Type II IF occurs most often due to anastomotic leak or intestinal injury at the time of surgery, with postoperative complications in the range of 32–50%. Other common causes are volvulus, intestinal ischemia due to hypotension or bands and trauma, etc.[11]

Different GI signs may be observed in up to 60% of mechanically ventilated patients.[5,12] Three or more GI symptoms may occur in about 20% of patients during their ICU stay, out of which clinically important GI bleeding is seen 2.6% of patients.[13]

Scoring Systems

Acute intestinal failure is commonly seen in critically ill patients; however, due to lack of consensus on determination of its severity, it is difficult to include it in MODS scoring systems. A systematic review was carried out to study 14 gastrointestinal dysfunction scoring tools (GDSTs) to develop a new tool to define AIF for critically ill patients. A marked variation was observed between these GDSTs due to lack of emphasis in the use of objective laboratory parameters and gut-specific biomarkers, limiting its applicability in critically ill patients.[14]

Assessment

Acute intestinal failure is often misdiagnosed in ICU patients owing to its complexity and lack of validated markers for monitoring. There is no valid scoring system for AIF and it is not integrated in MODS scoring system though its importance is known for a long time.[14] Thus, a systematic approach to assessment of AIF should be applied, which incorporates clinical assessment, radiology, biomarkers, etc.

Clinical Assessment

Evaluation of the GI tract is difficult, as abdominal signs and symptoms are not always related only to the GI tract. Signs and symptoms like pain and abdominal distension may be subjective and difficult to evaluate in mechanically ventilated patients. No particular GI symptom has been shown to correlate with mortality, but as the number of concomitant GI symptoms, such as, vomiting, diarrhea, and bowel distension increase, the patients have increasing mortality.[12] Clinical signs and symptoms may not correspond with objective assessment of GI function.

Feeding Intolerance and Gastric Residual Volume (GRV)

An objective definition for feeding intolerance is lacking, leading to variable relationship between feeding intolerance and mortality in various studies.[5] Feeding intolerance, which can be subjectively defined as interruption of enteral nutrition because of a large GRV, abdominal discomfort, distension, emesis, or diarrhea, is observed in 30.5% of mechanically ventilated patients staying in ICU for at least 72 hours; and its development is associated with impaired outcomes.[15]

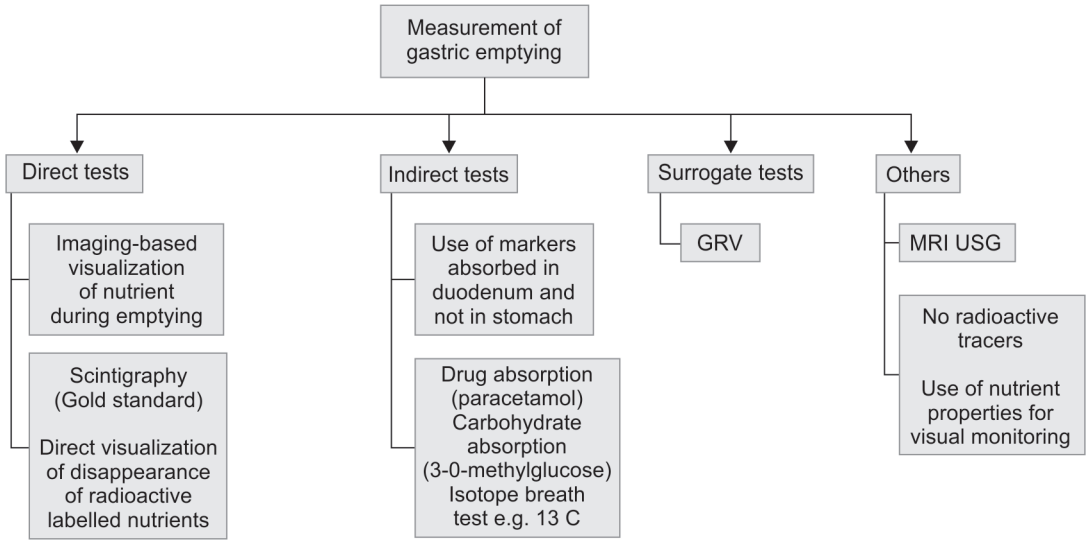
Large GRV (≥250 mL) during enteral feeding is a commonly used sign of feeding intolerance, though threshold of GRV remains unclear. A large GRV is highly predictive for delayed gastric emptying, but may be seen even when intestinal dysfunction is absent. Diarrhea may signify feeding intolerance, but evidence is lacking.[8]

Various methods like scintigraphy and paracetamol absorption test to assess gastric emptying are difficult to incorporate in routine practice and thus are used only for research purposes.[16] Flowchart 1 displays an approach to measurement of GRV.

Intra-abdominal Pressure

Increased intra-abdominal pressure (IAP) can lead to GI symptoms due to hypoperfusion of the intra-abdominal organs. It is an easy

Flowchart 1: Protocol for measurement of gastric residual volume



bedside tool. But there is no clear relationship between increased IAP and AIF. In mechanically ventilated patients, raised IAP without GI symptoms was not associated with mortality.[8]

Absorption of Nutrients

Diarrhea is common in critically ill patients, often due to malassimilation of nutrients.[5] Bomb calorimetry can be used to measure fecal weight and energy content, but this method is not widely available. Pancreatic exocrine insufficiency leading to fat malabsorption can lead to low fecal elastase levels and is seen in ICU patients. 3-O-methyl-glucose or C-glucose, a nutrient labeled with an isotope, is sampled from the blood after enteral administration to quantify nutrient absorption.[8] However, the results of the study can be confounded by factors like intragastric vs intraduodenal administration and delayed gastric emptying time.[8,17]

Barrier Function

The breach in intestinal mucosa barrier, which comprises of mechanical, biological, and immune barriers, is difficult to be captured by a single biomarker. Increased intestinal permeability as seen in critical illness can be detected by invasive methods like tissue biopsy and electron microscopy. Double/triple sugar absorption tests performed for assessing paracellular permeability have limited utility in the critically ill, as their results can be incorrectly interpreted in the presence of renal and/or hepatic impairment, GI motility problems, and use of antibiotics.[8]

Biomarkers

Multiple biomarkers are available for early detection of AIF; however, these have not been found to be clinically useful, as multiple factors like the timing of sampling, coexistence of other organ dysfunction, extent of intestinal damage, and surgical stress may confound the laboratory values and limit its clinical interpretation. Diamine oxidase has been proposed as a specific marker for gut mucosal damage, but it has overlapping values in patients with and without suspected damage.[8]

Intestinal fatty acid-binding protein (I-FABP) has elevated levels in patients with mesenteric ischemia in contrast to patients with acute abdomen who have preserved gut perfusion. Increased D-lactate concentrations have been seen with increased intestinal mucosal and capillary permeability during mesenteric ischemia but also in patients with acute abdomen with and without mesenteric ischemia making it a non-specific marker.[5] The IN-PANCIA study found low levels of biomarkers citrulline and glutamine, in patients with small bowel dysfunction.[4] There is unclear role of various enterohormones (cholecystokinin, ghrelin, glucagon-like peptide-2, and peptide YY) for the diagnosis of AIF.[8]

Imaging

Static [abdominal X-ray, ultrasonography, computed tomography (CT scan), or magnetic resonance imaging (MRI)] and dynamic radiological studies can be used to assess the intestinal function.[8] Ultrasound as a bedside tool can be easily used for facilitation and confirmation of proper placement of feeding tubes. It can also be used for measurement of gastric emptying, bowel diameter, bowel wall thickness, peristaltic movements, and tissue perfusion using US Doppler study.[8]

Treatment

Importance of Multidisciplinary Approach

Acute intestinal failure is a debilitating condition affecting the physical, physiological, and psychological wellbeing of the patient. They have a prolonged ICU and hospital stay (ICU-LOS and hospital LOS) with multiple complications making their treatment very complex. This prolongation of hospital LOS leads to increased costs, compounded by lost income and worsening finances.[3] A multidisciplinary approach is vital in medically managed ICU patients, as well as in the pre- and postoperative surgical patients for the restoration to normalcy (Fig. 2). Issues relating to pain management, nutritional support, ICU acquired infections, and development of depression, which inhibit recovery, have to be addressed. A multimodal approach with multidisciplinary input may increase survival in these patients.

Initial Management of Intestinal Failure

The first important step in management of AIF is to distinguish the types as 1 and 2 on the basis of duration and severity. Treating the underlying causes and reversing the metabolic problems that may contribute to poor gut function need to addressed (Flowchart 2).

The management is aimed at reducing the severity of AIF, prevention and treatment of complications and achieving a good quality of life.[7] Treatment is mainly supportive and specific therapeutic intervention options are available, though limited. For achieving these goals, ESPEN focused on controlling sepsis, fluid, and electrolyte replenishment and, optimizing nutritional status, appropriate surgery and wound care, and active rehabilitation.[3,7,18] In critically ill patients, no specific management protocol has been shown to improve GI function, and subsequent morbidity and mortality. In perioperative GI surgical patients, ERAS protocol including use of epidural analgesia improve GI motility.[8]

General Aspects: Pain Management

Pain can be very distressing, even in mechanically ventilated patients, and when inadequately managed, can lead to development of chronic pain and posttraumatic stress disorder (PTSD). On the contrary, over-treatment of pain can lead to prolonged mechanical ventilation and associated complications. Optimization of pain management with support from pain management team, with understanding of GI effects of analgesics; can help. Also, a psychologist who understands addiction and the utility of nonpharmacological distraction techniques can be extremely helpful.[3] Stimulation of both opioid and α-2 adrenergic receptors inhibits GI motility. Thus, a combination of reduced opioid intake, early feeding, and multimodal analgesia can achieve faster recovery of GI motility.[8]

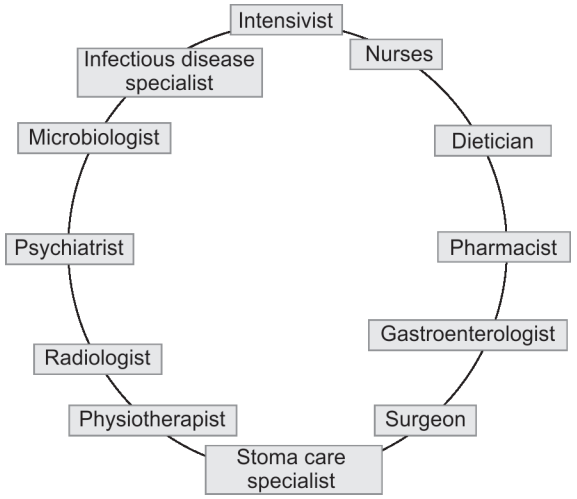


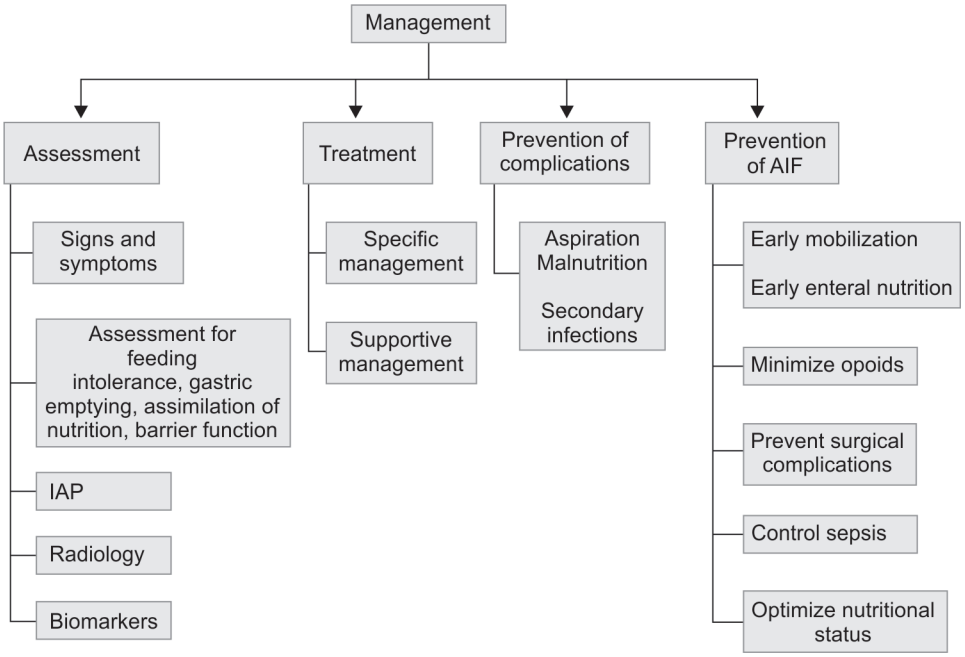
Fig. 2: Multidisciplinary approach

Nutritional Support

Regular nutritional assessment is vital since malnourished patients suffer from impaired immune response and poor wound healing. The aim is to provide adequate nutrition sufficient to meet their metabolic demands, building up the nutritional status, and ensure readiness for reconstructive surgery. Nutrition has to be individualized to the status of the patient for best optimization with options ranging from oral intake to gastric then jejunal nutrition to parenteral nutrition, in descending order.[3,7]

In the acute phase, early nutrition aimed to fulfil patient's entire caloric requirements from the beginning can be harmful, but in the early phase we do not yet know the optimal amount of calories and proteins required.[7] Unless contraindicated, enteral feeding is the route of choice as it prevents mucosal atrophy and thus helps to preserve the microbiome. It also contributes to the psychological health.[3] For patients at high risk of aspiration or with gastric feeding intolerance, international guidelines recommend the post-pyloric route for feeding.[8] Patients may need combined enteral and parenteral nutrition, as per the degree of dysfunction the GI tract; and if enteral nutrition does not meet the nutritional requirement. This may lead to over feeding, as well as complications associated with PN, such as, cholestasis and catheter-related bloodstream infections (CRBSI).

Flowchart 2: Management of acute intestinal failure



Fluids, Electrolytes Replenishment, and Glycemic Control

Fluid management should be aimed at optimizing electrolytes and hemodynamics with avoidance of both hyper- and hypovolumia. Capillary leak is observed in initial stage, leading to fluid shifts and bowel edema, impairing the bowel motility, and an increase in IAP. Dyselectrolytemia and dysglycemia interfere with bowel motility. There are insufficient data at present, to define the targets for specific glucose thresholds or serum electrolytes, to improve GI function.[8]

Gastrointestinal Motility Drugs

Prokinetic drugs (e.g., metoclopramide, domperidone, and erythromycin) are used to improve gastric emptying. Erythromycin is probably superior to metoclopramide but when combined, the effect of metoclopramide and erythromycin lasts longer, than either drug given alone. We do not have enough data the effects of GI tract or mortality and morbidity benefit.[8] Neostigmine is useful for the treatment of colonic paralysis. It has been used in the treatment of acute colonic pseudo-obstruction (Ogilvie's syndrome) also.[19] Polyethylene glycol and lactulose are suggested to prevent GI paralysis with limited evidence.[8,20]

Treatment of Intra-abdominal Hypertension

Intra-abdominal hypertension (IAH) can exacerbate bowel edema, but treatment aimed at treating IAH has not been shown to improve GI dysfunction or outcomes.[8]

Management of Sepsis

Sepsis is the leading cause of death in AIF, whether the source of infection is intra- or extra-abdominal. Clinical signs may be present in only 50% of patients; hence, a high index of suspicion for the presence of sepsis, abnormal labs, such as raised C-reactive protein (CRP) and leukocytosis or leukopenia are important pointers for the presence of sepsis. Other diagnostic radiological modalities like CT scan, ultrasound, MRI, radionucleotide studies, and fluoroscopy, can be used to identify source of infection. Appropriate and adequate antibiotic therapy should be given depending on the results of culture and sensitivity and local antibiograms.[7,18]

Stoma and Wound Care

High-output stomas (such as, enterocutaneous fistulae and complex ostomies), i.e., type II-prolonged AIF, are associated with poor outcomes.[7] Staff with sufficient expertise and resources should be assigned for their complex management, failing which morbidity of the patient increases.[3]

Surgery

Presence of abdominal sepsis mandates urgent intervention to remove source of sepsis, either by surgical or radiological drainage, a method being chosen which will lead to least physiological disturbance. If draining of collections needs resection of bowel, then the bowel ends should be brought out as stomas rather than attempting a reconstruction in the presence of peritonitis.[18] Constructive surgeries like intestinal reconstruction, abdominal wall reconstruction, and intestinal transplantation should be avoided and can be performed later for improvement in quality of life and are not lifesaving surgeries. These surgeries should be deferred where possible and should be undertaken once the patient is nutritionally stable, mobile, and physically and mentally in optimum status. The patients should be given much needed active mobilization and psychological support in the immediate postoperative period.[3,18]

Complications of Intestinal Failure

Acute intestinal failure can be associated with CRBSI due to central venous catheter (CVC) used to administer the parenteral nutrition, which can be prevented by simple measures, such as hand washing, chlorhexidine skin preparation, use of full sterile barrier precautions, removal of unwanted catheters, and avoidance of femoral arterial or venous site. Ethanol locks are effective at reducing CRBSI rates, but are not recommended by the ESPEN since they increase the risk of catheters getting blocked.[21] A meta-analysis of six randomized controlled trials found that use of taurolidine locks reduced CRBSI risk without an increase in catheter occlusion, as compared to heparin locks.[22]

In adult patients, IF-associated liver disease (IFALD) is uncommon. Severe liver dysfunction of multifactorial origins observed in <5% of patients.[21] It may be prevented by ensuring enteral intake, ensuring prompt replacement of nutrients, administration of cyclical PN; avoiding lipids for calorie intake in particular, adding fish oils, and medium- and long-chain triglycerides mixtures and lipid-containing monounsaturated fatty acids, and preventing sepsis.[23] Patients with AIF are at high risk of respiratory complications due to malnutrition weakened respiratory musculature. Risk factors for aspiration of gastric contents include increasing age, encephalopathy, upper abdominal surgery, presence of endotracheal and gastric tubes, vomiting, and supine position during feeding.

Adequate pain control with epidural analgesia, avoidance of opioid sedatives, head end elevation, early mobilization, ensuring presence of proper swallowing function before oral intake, and respiratory physiotherapy, can all minimize the risk of respiratory complications due to atelectasis and aspiration.[18]

Specific Management

Short bowel syndrome (SBS), which may occur due to extensive surgical resection or congenital diseases, is defined as total small bowel length of <200 cm and is the main cause of type III chronic intestinal failure (CIF) in adults.[7] Restricted oral or enteral nutrition, increased GI losses of fluids and electrolytes, hypophagia, lack of adaptive hyperphagia, accelerated GI transit time, and small bowel bacterial overgrowth contribute to the development of AIF. Short bowel syndrome -associated AIF can be reversed with intestinal rehabilitation programs, which involves coordinated approach with dietary and fluid modifications, symptomatic conventional medications, selective use of intestinotropic agents, and if required surgery.[24]

Intestinal fistulas occur spontaneously secondary due to underlying pathology in 15–25% of cases or as a result of bowel injury during surgery or inadvertent enterotomy and/or anastomotic leakage in 75–85% of patients. Fistula/s is/are abnormal communication between two parts of the GI tract or between the gut and the other organs, such as, vascular structure or skin. Parenteral nutrition plays a central role during metabolic instability. Fistuloclysis or refeeding enteroclysis are modalities of nutritional support after achievement of metabolic stability. Fistuloclysis is providing enteral nutrition into the intestine distal to the fistula opening, whereas refeeding enteroclysis, is a process of chyme collection from the proximal stoma and reinfusion into the distal stoma.[7]

Mechanical obstruction in the GI tract, which may be incomplete or complete, can lead to increased loss of fluids and electrolytes

into the lumen of obstructed segments and also loss of fluids and electrolytes during vomiting or nasogastric drainage. Replacement of fluid and electrolytes, as well as surgical management of obstruction when indicated, are the keys to management.[7]

In patients with extensive small bowel mucosal disease, the mucosa is intact but inefficient and leads to reduction of nutrient absorption. It may be caused by autoimmune enteropathy, intestinal lymphangiectasia, severe food allergy, protein-losing enteropathies, celiac disease, Crohn's disease, enteritis secondary to chemoradiation, and malabsorption of glucose–galactose.[7] The management is etiology based and beyond the scope of this review.

Conclusion

Acute intestinal failure is an extremely debilitating condition, which can lead to malnutrition, prolonged hospital stay, and poorer outcomes in critically ill patients. Lack of scoring systems and objective assessment tools can lead to misdiagnosis of AIF. A systematic approach to assessment of AIF with clinical assessment, radiology, and biomarker along with a multidisciplinary treatment approach can improve the outcomes in these complex patients.

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