Diarrhoea is a problem of intestinal water and electrolyte imbalance. It results when excess water and electrolytes are actively transported into the lumen (secretory diarrhoea) or when water is retained in the lumen by osmotically active agents (osmotic diarrhoea). Gastrointestinal motility also contributes to diarrhea.

Diarrhoea is acute when increased number of stools persists for less than 2 weeks. Although often mild, acute diarrhea can lead to severe dehydration as a result of large fluid and electrolyte losses. Macro and micro nutrient deficiency is rare. Management is with replacement of fluids and electrolytes (oral or parenteral) to prevent dehydration. Milk and dairy products is best avoided for 24 to 48 hours as they can worsen the diarrhea.

Home-prepared ORSs supplements include salted rice water, salted yogurt-based drinks, and vegetable or chicken soup with salt. Hyperosmolar fluids like soft drinks, sweetened fruit drinks etc are best avoided. Oral feeds are maintained despite the loose stools.

**PRINCIPLE OF ORS**

The presence of glucose in the intestinal lumen enhances sodium absorption which is the basis for the use of glucose in oral rehydration solutions during diarrhea. Zinc supplementation is recommended as an addition to ORS. It reduces the severity and duration of diarrhea.

Resistant starch recently has shown to reduce fecal fluid loss and shorten the duration of diarrhea in adolescents and adults with cholera.

**Chronic diarrhoea** is defined as passage of loose stools three or more times a day that lasts for at least four weeks. The chronic nature of diarrhea can lead to potentially serious complications, including dehydration and malnutrition.

Causes of small bowel diseases presenting as chronic diarrhoea include:

- Intraluminal causes: parasites (giardiasis, opportunistic infections in HIV positive patients (cryptosporidiosis, microsporidiosis) and small intestinal bacterial overgrowth (SIBO)

- Brush border enzyme deficiency e.g. infective enteritis, celiac disease, tropical sprue: lactose and fructose intolerance

- Mucosal diseases e.g. Celiac disease, tropical sprue, immunoproliferative small intestinal disease etc

- Transmural diseases; e.g. Crohn’s disease, tuberculosis

- Miscellaneous conditions: Mixed Connective Tissue Disorder, Amyloidosis

- Chronic Pancreatitis, pancreatic resection

- Neuroendocrine tumours: Gastrinoma, Zollinger Ellison syndrome, VIPOMA

- Resection of small intestine, post gastric surgery
The principles of management of chronic small bowel diarrhea are largely based on the site and nature of the disease, the extent of involvement of the small bowel.

On most occasions, the management of chronic diarrhea is essentially treating the underlying cause. Despite this, several chronic intestinal disorders manifest with occult or overt nutritional deficiency which influences the recovery of patients.

The principles of nutrient management in chronic small diarrhea will be highlighted taking into consideration three representative clinical situations:

A. Celiac Disease

Celiac disease is a clinical entity in which the patient is intolerant to wheat and its related products to a protein called gluten. These patients present with long standing diarrhea, manifesting as malabsorption to various vitamin and nutrient deficiencies.

The disease often affects the proximal portions of the small intestine, with reduced absorptive surface. Though gluten free diet is the corner stone in management of celiac disease, nutritional support and correction of nutrient deficiency is an important component of its management (discussed below).

B. Crohn’s disease

Crohn’s disease affects any part of the gastrointestinal tract from the mouth to the anus. It is a transmural disease that involves the terminal ileum and colon in 35-45% of cases, the terminal ileum in 25-35%, the colon in 15-25% and proximal small bowel or diffuse distribution throughout the small bowel in the remaining 5-10%.

Malabsorption and maldigestion resulting in nutrient deficiency is not uncommon in CD (Table 1).

Maldigestion is often due to SIBO resulting in reduced fat and fat soluble vitamin absorption. Inflammation of the small intestine in CD results in increased nutrient loss with transudation of proteins and fluid from the affected mucosa. There is also reduction in absorptive surface. Resections of small intestine including distal ileum results in further loss of absorptive surface.

Nutrition support is an important aspect of management of patients with IBD. Nutritional deficiency depends on the site of involvement. When CD affects the proximal intestine, malabsorption of carbohydrate is common. The high intraluminal osmotic loads results in diarrhoea and flatulence from metabolism by colonic flora. Protein calorie malnutrition is seen in 80% of CD, hypoa1buminemia in 25-80% and anemia in 29%. Mucosal

<table>
<thead>
<tr>
<th>Nutritional parameters</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight loss</td>
<td>65-76%</td>
</tr>
<tr>
<td>Growth retardation</td>
<td>40%</td>
</tr>
<tr>
<td>Hypoa1buminemia</td>
<td>25-80%</td>
</tr>
<tr>
<td>Iron</td>
<td>39%</td>
</tr>
<tr>
<td>Folate</td>
<td>54%</td>
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<tr>
<td>Vit B12</td>
<td>48%</td>
</tr>
<tr>
<td>Calcium</td>
<td>13%</td>
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<td>Magnesium</td>
<td>14-88%</td>
</tr>
<tr>
<td>Potassium</td>
<td>6-20%</td>
</tr>
<tr>
<td>Zinc</td>
<td>40-50%</td>
</tr>
</tbody>
</table>

Table 1: Prevalence of malnutrition in Crohn’s disease

The absorption of various nutrients and water is different in various regions of the small intestine. Thus, damage to the lining of the small intestines due to various causes can result in poor absorption of essential nutrients

DIGESTION OF NUTRIENTS

Carbohydrates: The digestion is in the proximal gut. The brush border enzymes (lactase, sucrase, maltase, trehalase, alpha dextrinase) play a role in its digestion in the duodenum and jejunum. The end products glucose, galactose and fructose are transported across the intestinal mucosa.

Proteins: Further to the digestion of proteins by pepsinogen in the stomach, the hydrolysed proteins i.e. amino acids and peptides are absorbed in the duodenum and small intestine, the latter after being reduced to oligopeptides and amino acids by brush border proteases in presence of pancreatic enzymes.

Lipids: The digestion of dietary fat is in the presence of lipase and colipase produced by the pancreas and intraluminal biliary phospholipid and bile salts. The absorption of fat is in the form of monoglycerides, re-esterified as triglyceride within the epithelial cells and enters the blood stream as chylomicron after combination with protein.

Water and electrolytes: Water movement into or out of the lumen of the GI tract is passive. Its absorption follows the osmotic gradients created by electrolyte movement. The cells of the villus tip promote fluid absorption, while the cells in the crypt region promote fluid and solute secretion. Potassium is passively absorbed in the jejunum and ileum when its luminal concentration rises because of absorption of water. The ileum is the primary site of carrier mediated bile salt and B12 absorption.

Thus, the proximal 100 to 200 cm of the jejunum is the primary site of carbohydrate, protein and water soluble vitamin absorption. Fat absorption occurs over a larger length of small bowel.
hemorrhage results in iron deficiency. Involvement of the ileum results in decreased bile acid absorption, and reduction in bile acid pool manifesting as steatorrhea. Fistula causes loss of zinc, fluids and electrolytes. Unabsorbed fats in CD, binds to calcium and magnesium causing loss of both minerals.

Vitamin B12 (terminal ileal involvement/resection of terminal ileum), and folate deficiency (SIBO, high homocysteine levels) is not uncommon. Low blood levels of biotin, thiamine, Vitamin A, C, E and β carotene has been documented in 40 - 90% of patients in absence of clinical signs and normal dietary intake. Fat soluble vitamins deficiency is seen in SIBO (30%) in CD. Other deficiencies observed include selenium, Vit D and calcium deficiency, latter due to steroids.

In CD, SIBO results in deconjugation of bile salts by bacteria. The effect of altered flora in the SI results in progressive damage to the mucosa with depletion of disaccharidases.

Correcting nutrient deficiencies

Subclinical deficiencies of nutrients may play a role in perpetuation of CD. There is a need to correct caloric and protein deficiency by correcting the diet itself. Specific deficits of vitamins A, D, E and K need correction. Correction of osteoporosis requires high dose of 25-OH vitamin D levels (50 000 IU several times weekly of ergocalciferol). Vit B12 supplement is recommended on a monthly basis. Zinc and magnesium deficiency requires correction.

Dietary manipulation such as use of elemental diet can serve as an adjuvant or even as a substitute to decrease dependence on various immunomodulatory drugs in CD. It is specially indicated in those who are steroid dependent, or are at risk to osteoporosis. Poor acceptance is a major drawback. Inflammation is a major factor in growth failure in children with CD and enteral nutrition may play a role in the primary treatment of CD and growth failure.

Elemental diet in CD:

Elemental formulas i.e. those containing amino acids instead of proteins, pre-digested liquid diet have been used in CD. These are non-antigenic to the intestinal mucosa and can induce remission in CD, results similar to that of steroids. Fish oil rich in ω-3 fatty acids is also known to have a therapeutic effect in CD especially in combination with antioxidants.

Parenteral Nutrition in small intestinal CD:

Short term TPN in CD is indicated in short bowel syn-

drome resulting from extensive intestinal resection, fistulae arising from diseased areas of the intestine or leaks, extensive stenosing CD to limit extent of resection, or to provide nutritional support until drug therapy effectively controls disease.

C. Short Bowel Syndrome (SBS):

This often results after extensive resection of the small intestine due to various causes. Typically this translates to a patient with <50-70 cm of small intestine when colon is intact or <100-150 cm of small bowel when colon is absent.

SBS often presents as chronic diarrhoea and malabsorption resulting in macro and micro nutrient deficiencies often requiring enteral or parenteral nutrition.

The establishment of daily calorie and fluid intake requirement is adjusted to suit patient tolerance, the clinical presentation, stool output, micronutrient levels, weight and hydration status of the individual.

Individual calorie goals are estimated using the Harris-Benedict estimation multiplied by activity and malabsorption factors. In general most stable adult SBS patients absorb only about one-half to two-thirds as much energy as normal. Thus dietary intake is increased by at least 50% i.e. hyperphagic diet. The increased quantity of food tends to be best tolerated when consumed throughout the day in 5 or 6 meal period.

Tube feeding is indicated while weaning off from parenteral nutrition. Nocturnal gastric tube feeding of a semi elemental or polymeric formula administered continuously via an infusion pump in small quantities is better tolerated than bolus tube feeding due to greater absorption of nutrients and a reduced occurrence of osmotic diarrhoea.

The optimal fluid components of the diet depend on the remaining bowel anatomy. Patients without a colon generally require the use of glucose-electrolyte ORS, to enhance absorption and reduce secretion. Those with intact colon can maintain hydration without excessive fluid loss with hypotonic fluids. An ORS with a sodium concentration of 90 to 120 meq/L provides optimal jejunal absorption. One to three litres of such fluid daily, sipped through the day may be needed to maintain adequate hydration. Regardless of bowel anatomy, hyperosmolar fluids such as concentrated sugars, fruit juices etc should be avoided as they potentiate stool output. Patients who have a bowel movement shortly after a meal may benefit from avoiding drinking fluids during meals. Parenteral fluids are necessary if the ostomy output exceeds fluid intake.

The provision of complex macronutrient in the diet of SBS is preferred. Complex CHO lower the osmotic load and potentially exert a positive effect on the adaptation process. In presence of a proximal jejunum, lactose is well tolerated.
and is not restricted unless the patient has milk intolerance. With respect to proteins, those with high biological values such as those found in beef, pork, poultry, the restriction of fat to 20-30% of the daily calories is recommended in those adults with an intact colon. This results in a reduction in steatorrhea, magnesium and calcium loss and a reduction in oxalate absorption (Tables 2 and 3).

Medium chain TG is an alternative energy source and is absorbed both from the large and small intestine. In patients with intact colon diet enriched with MCT improves overall energy and fat absorption. In absence of colon the improvement of fat absorption is offset by malabsorption of other macronutrients. MCT is directly absorbed and does not require intraluminal digestion. The provision of essential fatty acids (safflower oil or soybean oil) is important as deficiencies are common, particularly in the setting of low fat diets and malabsorption.

Soluble fibre supplementation has an advantage of enhancing intestinal adaptation and slowing of gastric emptying, though it may increase gas and bloating.

Glutamine is ‘conditionally essential’ amino acid that is a primary energy source for the enterocyte and has been shown to prevent mucosal atrophy and deterioration of gut permeability in patients receiving parenteral nutrition.

Micronutrient supplementation (water soluble vitamins, iron) is not necessary in presence of an intact proximal intestine. However, fat soluble vitamin and essential fatty acids needs to be supplemented. Supplemental zinc and occasionally selenium may be required in the presence of excessive stool losses. Lifetime administration of supplemental Vitamin B12 is necessary on a monthly basis, more so in those with > 50-60 cm resection of terminal ileum.

Hypomagnesemia is due to secondary hyperaldosteronism, reduced mucosal surface, binding of magnesium by unabsorbed fatty acids and loss via kidney. Oral magnesium can be given in doses of 12 to 24 mEq/day and is best given at night. In moderate to severe hypomagnesia (<1 mg/dL), parenteal magnesium may be necessary.

Summarising, the principles of supplementation of macro and micronutrients in chronic small bowel diarrhoea is dependent on the nature, site and extent of the disease. Most often symptoms are subclinical and replacement is necessary to hasten the mucosal recovery.

REFERENCES