The Vicious Cycle of Malnutrition and Gastrointestinal (GI) Disorders

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The World Health Organization (WHO) defines malnutrition as a deficiency, or excess of nutrient intake and/or impaired nutrient utilisation. Undernutrition can be classified into several categories: wasting (low weight for height), stunting (low height for age), underweight (either wasting or stunting, or both), and micronutrient deficiencies.[1]

While socioeconomic factors often contribute to malnutrition, there are instances where food provision alone does not resolve wasting. In these cases, undernutrition is likely to be associated with underlying diseases.[2] Disease-related malnutrition can lead to further reduction in oral intake, malabsorption with increased nutrient losses and altered metabolic demands.[3]

GI disorders are frequently investigated as potential causes of malnutrition, as a healthy digestive system is crucial for nutrient absorption.[2] Malnutrition's impact on the GI tract can lead to altered intestinal blood flow, pancreatic exocrine insufficiency, and villous atrophy, all of which reduce nutrient absorption.[3] Additionally, increased intestinal permeability can cause a loss of digestive enzymes, further impairing nutrient absorption. This enzyme loss can occur early in the course of reduced energy intake, potentially resulting in secondary lactose intolerance.[4] Several GI conditions are closely associated with malnutrition.

Inflammatory bowel disease (IBD) & malnutrition

Malnutrition can manifest in various forms in individuals with IBD, including protein-energy malnutrition, micronutrient deficiencies, and poor bone health.[5] Contributing factors include reduced oral intake, malabsorption, protein loss, and bacterial overgrowth.[6] According to Balestrieri *et al.,* the incidence of malnutrition in IBD patients ranges from 20% to 85%.[6] Malabsorption in IBD is likely to be related to mucosal alterations that disrupt epithelial integrity, and ileal inflammation can further impact nutrient absorption by reducing bile salt uptake.[6] Additionally, active inflammation leads to blood and protein loss.

Malnutrition is more common in Crohn's disease than in ulcerative colitis, probably due to the small bowel involvement in Crohn's, which affects nutrient absorption.[5, 6] Newly diagnosed Crohn's disease patients have a 60% incidence of protein-energy malnutrition, compared to 35% in ulcerative colitis patients.[7, 8] IBD patients often present with protein-energy malnutrition at diagnosis, and their nutritional status can fluctuate between periods of flare-ups and remission.

Various treatments are available for Crohn's disease, including dietary interventions. Exclusive enteral nutrition (EEN) is a first-line treatment for Crohn's disease, with remission rates between 60-80%.[9] The preferred feed is a standard polymeric feed, as the type of protein does not impact EEN's efficacy.[9] If polymeric feeds are poorly tolerated, peptide-based or amino acid-based feeds may be considered.

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Tolerance and palatability are crucial when selecting nutritional support feeds. Clinically, if polymeric feeds are not well tolerated and there is no history of cows' milk allergy, peptide-based feeds are often a suitable and well-tolerated alternative, despite limited evidence. For patients with a history of cows' milk allergy, amino acid-based feeds should be used.

Motility & malnutrition

The GI tract plays a central role in food digestion and absorption, with the enteric nervous system (ENS) regulating these functions. The ENS consists of a network of 200-600 million neurons that coordinate gut activities such as motility and peristalsis. Disruptions to this complex system can lead to undesirable symptoms, malabsorption, and malnutrition. Motility disorders, which include impaired coordination and limited peristalsis, can be categorised as either neuropathic or myopathic.[10]

A common motility disorder is gastro-oesophageal reflux disease (GORD), characterised by oesophagitis and symptoms such as faltering growth, feeding difficulties, discomfort, and haematemesis. Cows' milk protein allergy (CMPA) is closely linked to GORD, with Cavataio *et al.* finding that 40% of infants with GORD may have an underlying CMPA.[11] A trial of an extensively hydrolysed formula for 2-6 weeks can help determine if this improves GORD symptoms.[12] If symptoms persist and the infant is breastfed, excluding cow's milk from the maternal diet may be recommended.[12]

GORD is frequently reported in children with neurological disabilities (as high as 70%) and negatively impacts their nutritional status and quality of life.[13] The European Society for Paediatric Gastroenterology Hepatology and Nutrition (ESPGHAN) suggests that trialling polymeric whey-based feeds may benefit these patients due to potentially increased gastric emptying.[13] Although evidence is limited, peptide-based or extensively hydrolysed feeds might improve tolerance and gastric emptying.[14]

Gastroparesis, characterised by delayed gastric emptying, is another motility condition that can lead to malnutrition due to symptoms such as nausea, early satiety, postprandial bloating, and pain.[15] Moderate to severe cases often show signs of malnutrition, dehydration, and nutrient deficiencies, including calcium, iron, folate, magnesium, potassium, and fat-soluble vitamins. The use of proton pump inhibitors can further impact these micronutrient levels.[16] Patients who can eat may benefit from low-fibre and low-fat diets, with liquids often being better tolerated than solids. If oral feeding is not tolerated, enteral feeding might be necessary, often opting for jejunal feeding to bypass the stomach and improve tolerance.[17] In severe cases, parenteral nutrition (PN) may be required if other forms of enteral feeding fail.

Short bowel syndrome & malnutrition

Several factors can increase the risk of malnutrition in short bowel syndrome, including the length and quality of the remaining bowel, the presence of the ileum (which allows for greater adaptation), and the presence of the ileocecal valve and/or colon.[18] Early infant feeding is known to predict shorter hospital stays and reduced dependence on parenteral nutrition (PN).[19] Introducing enteral feeds encourages small bowel adaptation, resulting in mucosal hyperplasia, villous lengthening, and increased crypt depth.[20] Enteral tolerance also stimulates bile flow.

While many patients will require PN to meet their nutritional needs, it is essential to introduce and progress enteral feeds whenever possible, choosing feeds that are likely to be tolerated. If available, breastmilk should be the first-line choice for these patients due to its beneficial components, such as immunoglobulin A (IgA), human milk oligosaccharides, nucleotides and leukocytes, which help develop the baby's immune system. In the absence of breastmilk, a partially hydrolysed/whole protein formula feed that is low in lactose and contains medium-chain triglycerides (MCT), and has low osmolality should be considered.[19]

Verlato *et al.* conducted a survey on the feeding management of infants with short bowel syndrome, which was sent to 24 centres in 15 countries, achieving a 100% response rate. The survey found that 95.8% of centres used breastmilk as their primary feed. For 54.2% of respondents, the second choice was an extensively hydrolysed feed containing MCT.[18] The choice of feed is important to successfully establish and progress with oral feeding, with the aim of reducing PN and thus the risk factors associated with long-term PN.

CASE STUDY

Presenting symptoms

A 9-year-old boy was admitted to the ward from his local hospital with dehydration, severe malnutrition, electrolyte disturbances, distended abdomen, constipation, poor tolerance of feeds and developmental delay.

• **Weight:** 14.4 kg (< 0.4th centile) • **Height:** 130 cm (25th centile) • **BMI:** < 0.4th centile
Classified as severe malnutrition.

Biochemistry test results: Metabolic alkalosis, hypokalaemia, hypocalcaemia and bradycardia.

Past medical history: Ongoing poor oral intake. He had a very limited diet as his parents felt he had reacted to most foods. Most of his dietary intake was from homemade vegetable broth, thought to be better tolerated.

Nutritional rehabilitation

The patient was transferred to the gastroenterology ward for investigations and nutritional rehabilitation. He was bedridden and unable to bear weight. He was started on PN and developed refeeding syndrome, which was treated with intravenous (IV) potassium replacements. He required oral phosphate and bicarbonate supplements. Once he was stable a nasogastric tube (NGT) was inserted, and continuous polymeric feeds (1 kcal/ml) were started. He developed abdominal distention, pain and vomiting. The NGT was changed to a naso-jejunal tube (NJT) and a continuous 1 kcal/ml peptide-based feed containing 50% fat from MCT was started. This feed increased gradually each day and PN was weaned. After 2 weeks, the patient was tolerating full jejunal feeds. The feed rate was gradually increased, and the hours were decreased until he was having 16-hour feeds that meet his requirements.

Investigations & and diagnosis

Solid gastric empty study – found slow gastric emptying with semi-solid porridge.
Liquid gastric empty study – normal emptying times. Better emptying than with solids.

Further management

After 2 months of nutritional rehabilitation his weight had increased to 19 kg. He was started on a bite and dissolve diet and began to mobilise. Due to the results of the gastric emptying studies a gastric feeding trial was indicated. His feeding tube was changed to a NGT and the 1 kcal/ml peptide feed was trialled continuously. This was well tolerated. He was progressed to slow bolus feeds.

A gastrostomy placement was scheduled, and he continued with small amounts of bite and dissolve foods orally.

Discussion

Gastroparesis can lead to poor tolerance of oral food, and due to the rarity of the disease, a prolonged diagnosis process can result in malnutrition. In this case, the patient responded well to peptide-based feeds, likely due to improved gastric emptying facilitated by the broken-down proteins and MCT fats.

References: **1.** World Health Organization. Malnutrition. Accessed online: www.who.int/health-topics/malnutrition#tab=tab_1 (May 2024). **2.** Thompson AJ, *et al.* (2021). Understanding the role of the gut in undernutrition: what can technology tell us? Gut.; 70(8): 1580-1594. **3.** Allen B, Saunders J (2023). Malnutrition and undernutrition: causes, consequences, assessment and management. Nutrition.; 51(7): 461-468. **4.** Saunders J, Smith T, Stroud M. (2011). Malnutrition and undernutrition. Medicine; 39(1): 45-50. **5.** Gerasimidis K, McGrogan P, Edwards CA. (2011). The aetiology and impact of malnutrition in paediatric inflammatory bowel disease. J Hum Nutr Diet.; 24(4): 313-26. **6.** Balestrieri P, *et al.* (2020). Nutritional Aspects in Inflammatory Bowel Diseases. Nutrients.; 12(2): 372. **7.** Sawczenko A, Sandhu BK (2003). Presenting features of inflammatory bowel disease in Great Britain and Ireland. Arch Dis Child.; 88(11): 995-1000. **8.** Weinstein AT, *et al.* (2003). Age and family history at presentation of pediatric inflammatory bowel disease. J Pediatr Gastroenterol Nutr.; 37(5): 609-613. **9.** Miele E, *et al.* (2018). Nutrition in Pediatric Inflammatory Bowel Disease: A Position Paper on Behalf of the Porto Inflammatory Bowel Disease Group of the European Society of Pediatric Gastroenterology, Hepatology and Nutrition. J Pediatr Gastroenterol Nutr.; 66(4): 687-708. **10.** Lehmann S, Ferrie S, Carey S. (2020). Nutrition Management in Patients With Chronic Gastrointestinal Motility Disorders: A Systematic Literature Review. Nutr Clin Pract.; 35(2): 219-230. **11.** Cavataio F, Carroccio A, Lacono G. (2000). Milk-induced reflux in infants less than one year of age. J Pediatr Gastroenterol Nutr.; 30 Suppl: S36-S44. **12.** Meyer R, *et al.* (2022). Diagnosis and management of food allergy-associated gastroesophageal reflux disease in young children-EAACI position paper. Pediatr Allergy Immunol.; 33(10): e13856. **13.** Romano C, *et al.* (2017). European Society for Paediatric Gastroenterology, Hepatology and Nutrition Guidelines for the Evaluation and Treatment of Gastrointestinal and Nutritional Complications in Children With Neurological Impairment. J Pediatr Gastroenterol Nutr.; 65: 242-264. **14.** Garzi A, *et al.* (2002). An extensively hydrolysed cow's milk formula improves clinical symptoms of gastroesophageal reflux and reduces the gastric emptying time in infants. Allergologia et Immunopathologia.; 30(1): 36-41. **15.** Krasaelap A, Kovacic K, Goday PS. (2020). Nutrition Management in Pediatric Gastrointestinal Motility Disorders. Nutr Clin Pract.; 35(2): 265-272. **16.** Bharadwaj S, *et al.* (2016). Management of gastroparesis-associated malnutrition. J Dig Dis.; 17(5): 285-294. **17.** Aguilar A, Malagelada C, Serra J. (2022). Nutritional challenges in patients with gastroparesis. Curr Op Nutr Metab Care.; 25(5): 360-363. **18.** Verlato G, *et al.* (2021). Results of an International Survey on Feeding Management in Infants With Short Bowel Syndrome-Associated Intestinal Failure. J Pediatr Gastroenterol Nutr.; 73(5): 647-653. **19.** Cernat E, *et al.* (2021). Short bowel syndrome in infancy: recent advances and practical management. Frontline Gastroenterol.; 12(7): 614-621. **20.** Andorsky DJ, *et al.* (2001). Nutritional and other postoperative management of neonates with short bowel syndrome correlates with clinical outcomes. J Pediatr.; 139(1): 27-33.