Case Study: Small bowel perforation secondary to ileal tuberculosis: intensive care unit case study

Veldsman L, MNutr, BScDiet, RD(SA), Charlotte Maxeke Johannesburg Academic Hospital
Correspondence to: Lizl Veldsman, e-mail: lizlveld@gmail.com
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The following case study was discussed at the South African Society for Parenteral and Enteral Nutrition (SASPEN) workshop on critcal care nutrition held at the 25th Congress of the Nutrition Society of South Africa and the 13th Congress of the Association for Dietetics in South Africa in Johannesburg in September 2014. It is a reflection of the general opinion of the audience, followed by a rationale of the latest literature on the topic. Here is a summarised discussion of the case.

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Introduction

Nontraumatic terminal ileal perforation is a common cause of obscure peritonitis, and since the clinical features are similar to any other acute abdominal condition, patients are often misdiagnosed, resulting in delayed surgical intervention. Apart from intestinal tuberculosis, other common causes of ileal perforation include typhoid fever, non-specific inflammation, obstruction (e.g. Crohn's disease) and radiation enteritis.[1] Intestinal tuberculosis or tuberculosis enteritis commonly affects the ileocecal region, possibly because of the increased physiological stasis, abundant lymphoid tissue and high absorptive capacity.[2] Tuberculosis enteritis may lead to perforation of the terminal ileum in 1-15% of cases, which usually occurs as a blowout of the small bowel secondary to distension caused by distal strictures or adhesions.[2,3] Presenting symptoms include severe abdominal pain (100%), fever (57%), vomiting (42%), constipation (58%), dehydration (71%), tenderness (86%), distension (68%) and rigidity (32%).[2] The treatment for tubercular peritonitis is the same as that for peritonitis due to other causes, and involves resuscitation, nasogastric aspiration, intravenous fluids, antibiotics and surgery, often necessitating the formation of an ileostomy.[2,4] Two important postoperative complications directly impacted on the patient's nutritional care plan in this given case report. The one was a high-output ileostomy and the other, Guillain-Barré syndrome.

High-output ileostomy

Patients often develop a high-output ileostomy (> 2 000 ml/day)[5] after stoma formation following bowel surgery.[5,6] Although an early high-output ileostomy often resolves spontaneously, some patients require ongoing medical treatment in the form of anti-diarrhoeal drugs, octreotide and proton-pump inhibitors.[5,6] Patients with a persistent high-output stoma are at risk of significant fluid and electrolyte imbalances, as well as protein energy malnutrition due to insufficient nutrient absorption.[5]

Guillain-Barré syndrome

Guillain-Barré syndrome is usually an acute-onset, monophasic, immune-mediated disorder of the peripheral nervous system, characterised by symmetric muscle weakness, loss of sensation and the loss of deep tendon reflexes.[7,8] Guillain-Barré syndrome is often preceded by an infection in the upper respiratory tract or gastrointestinal tract. Organisms that are usually implicated in the development of Guillain-Barré syndrome include *Campylobacter jejuni*, cytomegalovirus, Epstein-Barr virus, *Mycoplasma pneumonia* and *Haemophilus influenzae*.[8-10] Other factors associated with the development of Guillain-Barré syndrome include the stress of surgery, immunisation and parturition.[10] Both intravenous immunoglobulin and plasma exchange are effective in the treatment of Guillain-Barré syndrome.[11] The goals of nutrition therapy for patients with Guillain-Barré syndrome are the same as those for any other critically ill patient. Since weaning from the ventilator is often difficult, optimal nutrition therapy plays a very important role; firstly, since nutritional repletion is essential to build up the musculature, and secondly, to avoid overfeeding, as this may lead to an increased ventilator load.[12] However, despite the delivery of optimal nutrition therapy, Guillain-Barré syndrome patients often lose a significant amount of weight due to various factors, including stress-induced catabolism, prolonged bed rest, as well as certain medications, e.g. corticosteroids.[12] Furthermore, Guillain-Barré syndrome patients often present with oropharyngeal weakness and subsequent swallowing dysfunction, as well as autonomic nervous system dysfunction, which, in turn,

may lead to gastrointestinal dysmotility, and in severe cases, ileus.[7,11] Dysmotility is frequently managed with nasogastric suctioning, the administration of erythromycin or neostigmine, and in severe cases, the suspension of enteral nutrition.[7]

Case study

A 51-year old female (recumbent length of 172 cm, an estimated weight of 50 kg and a usual weight of 56 kg ± 1 month prior as reported by the family) was admitted to the medical ward of a Johannesburg Hospital on Thursday, 3 July 2014, with severe abdominal pain, nausea and vomiting. The symptoms had begun one week previously, but increased in severity over the last three days. The patient reported the last instances of flatulence and defecation being two days prior to hospitalisation. On examination, she was febrile, appeared dehydrated and showed signs of peritonitis.

A laboratory evaluation revealed a positive human immunodeficiency virus (HIV) status (CD4 count of 132 cells/mm^3), leucocytosis (13.9 x 10^9/l), hyponatraemia (128 mmol/l), and pre-renal failure (urea 17.1 mmol/l and creatinine 203 µmol/l), which responded to adequate fluid resuscitation in the ward. An X-ray examination of the chest showed free air under the diaphragm, followed by abdominal sonography, suggesting features of a perforated bowel. There was free fluid in the peritoneal cavity, with multiple, dilated, fluid-filled loops in the small bowel. Nasogastric decompression was performed, and the patient was prepared for surgical exploration. A laparotomy, performed on Saturday, 5 July, with a midline incision, revealed purulent peritoneal fluid, mainly in the pelvis, as well as a perforated gangrenous loop of terminal ileum 50 cm from the ileocaecal valve, with a distended proximal bowel. Approximately 15 cm of necrotic small bowel was resected, and a double-barrel ileostomy brought out. This was followed by a thorough peritoneal washout and abdominal closure. A provisional diagnosis of a small bowel perforation secondary to ileal tuberculosis was made, and later confirmed by histopathology, and the patient was transferred to the intensive care unit (ICU) for further medical management.

On arrival in the ICU, the patient was haemodynamically stable, with a mean arterial pressure of 73 mmHg, and was placed on a low dose of inotropic support, i.e. adrenaline at 0.04 µg/kg/minute. A laboratory evaluation revealed an elevated C-reactive protein (CRP) of 134 mg/l, leucocytosis of 13.1 x 10^9/l and hypoalbuminaemia of 22 g/l. On physical examination, she was febrile (38.8°C). Her pulse rate was 115 beats/minute and her respiratory rate 24 breaths/minute (managed on 40% facial mask oxygen). The patient's blood glucose levels ranged from 8-10 mmol/l.

She was immediately placed on the following therapy:

- Empiric antitubercular treatment of rifafour 4 tablets *per os,* daily, and pyridoxine 25 mg *per os,* daily.
- A broad-spectrum antibiotic, i.e. tazobactam 18 g, over 24 hours, intravenously, for seven days.
- An antifungal medicine, i.e. fluconazole 400 mg, intravenously, daily.
- An analgesic, i.e. morphine 1-2 mg, intravenously, four hourly.
- An antithrombotic agent, i.e. clexane 40 mg, subcutaneously, daily.
- An ulcer prophylactic, i.e. ulsanic 1 g *per os,* six hourly.

The patient was kept *nil per os* and received intravenous fluid therapy in the form of Balsol®.

By the next morning (day 2 in the ICU), the patient's blood pressure began to improve and inotropic support was discontinued. She was fully awake, responsive and maintained adequate oxygen saturation via a facial mask, i.e. fraction of inspired oxygen 2 ($FiO_{2)}$ of 40**%**. However, she presented with progressive weakness, and after a neurological review, was diagnosed with postoperative Guillain-Barré syndrome. The patient was subsequently placed on a six-day course of immunoglobulin therapy, i.e. Polygam® 24 g, intravenously, daily, which contains ± 33.6 g sucrose as a stabiliser, and received an intravenous calcium replacement, i.e. calcium gluconate 10 ml 10%, over 10 minutes, for severe hypocalcaemia, with corrected calcium of 1.89 mmol/l. The nasogastric tube, still on free drainage, drained 300 ml over 24 hours. On physical examination, the abdomen was soft and tender, with positive bowel sounds. The attending physician prescribed a mixed-fluid diet.

By day 3 in the ICU (Monday morning, 7 July 2014), the patient's oral intake remained poor. She had now developed a septic abdominal wound, further complicated by a high-output ileostomy, i.e. three litres over the past 24 hours. On physical examination, her abdomen appeared to be tense and slightly distended. A laboratory analysis revealed severe hypoalbuminaemia, as well as hypokalaemia, hypocalcaemia and hypophosphataemia. Stool cultures were sent for *Clostridium difficile* testing. The result was negative, and the patient was referred to the dietitian for nutritional intervention.

Question 1: What are your decisions with regard to the patient's nutritional requirements on day 3 in the intensive care unit? What nutritional intervention do you suggest?

Parenteral nutrition (PN) was recommended, based on the following nutrition-related problems:

- *Grade II protein-energy malnutrition (PEM):* An estimated body mass index (BMI) of 16.9 kg/m^2, with significant weight loss (± 10.7% over one month) and little to no nutritional intake for > 5 days.
- *Increased nutritional requirements:* A hypermetabolic and hypercatabolic state, relating to abdominal sepsis, Guillain-Barré syndrome, HIV/acquired immune deficiency syndrome (AIDS) and abdominal tuberculosis.
- *Increased nutrient losses and severe malabsorption:* High-output ileostomy.
- *Other:* Post major gastrointestinal surgery, severe hypoalbuminaemia and abdominal distension.

Since there was no absolute contraindication to enteral nutrition, trophic feeding at 10-20 ml/hour was recommended in order to maintain gut trophicity. An isotonic semi-elemental enteral nutrition formula was chosen because of the abdominal distension, severe hypoalbuminaemia and significant nutrient malabsorption.

A total energy target of 25-30 kcal/kg actual body weight and 1.3-1.5g protein/kg ideal body weight was recommended. Owing to the risk of refeeding syndrome, based on PEM, low to no nutritional intake for > 5 days, hypokalaemia, hypophosphataemia and hypomagnesaemia, a decision was made to gradually initiate and advance the feeding in conjunction with a daily electrolyte replacement and micronutrient supplementation, and thiamine, in particular. The energy contribution from Polygam®, in the form of sucrose, was considered to be part of the total energy intake.

Rationale

Although the indications for PN remain largely unchanged, and two recent review articles[13,14] have suggested the need for PN in the case of significant nutrient malabsorption, e.g. high-output enterostomies of > 2 litres/24 hours, current clinical practice guidelines on its optimal timing are contradictory.[13] According to the European Society for Clinical Nutrition and Metabolism (ESPEN), patients who are not expected to be on normal nutritional intake within three days of the onset of disease should receive PN within 24-48 hours if enteral nutrition is contraindicated, or if it cannot be tolerated.[15] Despite a more delayed approach of 7-10 days, as recommended by the American Society for Parenteral and Enteral Nutrition (ASPEN), the general consensus remains that PN should be initiated as soon as possible if there is evidence of PEM on admission and enteral nutrition is not feasible.[13,15,16] Although serum secretory protein, e.g. serum albumin, is not a valid marker of nutritional status in critical illness, it can be used with other parameters, e.g. CRP, as a proxy indicator of inflammatory status, and hence disease severity, which, in turn, is a potent contributor to disease-related malnutrition.[17,18] Therefore, inflammatory status and disease severity can be used together with nutritional status and nutrient intake to identify patients at high nutritional risk who are likely to benefit from early goal-directed feeding.[19]

A small amount of enteral nutrition, e.g. 250 ml/day, typically administered as a trophic feed at 10-20 ml/hour, is possible in most patients requiring PN unless enteral nutrition is an absolute contraindication, e.g. bowel left in discontinuity after damage control laparotomy, and offers additional non-nutritional benefits, such as being able to maintain gut integrity, attenuate oxidative stress, promote insulin sensitivity, increase brush-border enzymes, restore commensal bacteria, and stimulate bowel motility and immunoglobulin A secretion.[13,14,19] This may be of particular benefit to patients with Guillain-Barré syndrome since they often present with gastrointestinal dysmotility, resulting from autonomic nervous system dysfunction, further worsened by long-term immobilisation and the administration of opiates for pain control.[20,21]

Severely malnourished patients on PN and/or those at risk of refeeding syndrome should initially receive 10 kcal/kg actual body weight/day, progressively increasing the target to reach 25-30 kcal/kg actual body weight/day over 3-4 days.[14,15] Owing to the potential risk of overfeeding, especially in patients receiving a combination of PN and enteral nutrition, daily monitoring of actual energy intake, inclusive of non-nutritional energy sources, e.g. propofol, Polygam® and intravenous dextrose, is clearly warranted. Furthermore, PN should be gradually weaned over time when enteral nutrition reaches the energy target.[13,14,22-24]

Apart from achieving an adequate energy intake, optimal protein provision is as important, and may offer a significant mortality benefit.[25,26] ESPEN and ASPEN recommend a daily protein intake in the range of 1.3-1.5 g/kg and 1.2-2.0 g/kg, respectively.[15,16] ICU patients with a daily protein intake of 1.5 g/kg had a significantly lower mortality compared to those receiving only 0.8 g/kg or 1.1 g/kg, independently of energy intake, in an observational study by Allingstrup et al.[25] According to Puthucheary et al,[27] muscle wasting occurs early and rapidly during the first week of critical illness. Therefore, a high protein intake of 1.5 g/kg/day, combined with early mobilisation, may help to overcome anabolic resistance, especially in the acute phase of illness.[23] Appropriately designed randomised controlled trials are needed to confirm the optimal macronutrient distribution in the different phases of critical illness.[22,23]

An adequate micronutrient supply is another important component of optimal nutrition therapy. Henceforth, ESPEN recommends that all PN bags should include a daily dose of multivitamins and trace elements (grade C).[15] Critically ill patients, in particular, are at risk of developing micronutrient deficiencies. This is partly owing to increased requirements, but is also often as a result of pre-existing deficiencies, increased losses (e.g. high-output enterostomy), drug-nutrient interactions (e.g. vitamin B_6 in antitubercular treatment) and/or an inadequate intake.[28,29] Therefore, micronutrients should be included in the nutritional care plan, especially since they play an important role in substrate metabolism, cellular immunity, wound healing and antioxidant defences. The latter is particularly challenging in the critically ill patient.[15, 28-30] Furthermore, daily thiamine supplementation, together with the correction of fluid and electrolyte imbalances, plays an important role in preventing refeeding syndrome.[24,31] Therefore, in order to minimise fluid and electrolyte losses via the ileostomy, preference should be given to an isotonic enteral nutrition formula of osmolality near 300 mOsmol/kg, with a sodium concentration near 100 mmol/l, and preferably semi-elemental to facilitate nutrient absorption.[32,33]

Question 2: What are your decisions regarding glutamine in the current setting?

Given the fact that this was a malnourished patient, post major gastrointestinal tract surgery, with no signs of multi-organ failure, shock or renal and liver dysfunction, a decision was taken to add intravenous glutamine supplementation to the prescribed all-in-one PN regimen, in order to provide 0.2-0.4 g/kg/day of L-Glutamine. Furthermore, a decision was taken to avoid enteral glutamine supplementation.

Rationale

According to the ESPEN guidelines on PN, the amino acid solution should contain 0.2-0.4 g/kg/day of L-Glutamine, i.e. 0.3-0.6 g/kg/day of alanyl-glutamine dipeptide.[15] Early high-dose glutamine supplementation given separate from nutrition therapy

offered no benefit, and may have been associated with increased mortality in critically ill patients with multi-organ failure in the recent REducing Deaths due to OXidative Stress (REDOXs) study by Heyland et al.[34] Therefore, until further safety and efficacy data are available from adequately powered randomised controlled trials, glutamine administration should be avoided in critically ill patients with multi-organ failure, and particularly in those with concomitant renal dysfunction.[35,36] However, a large body of evidence still remains that suggests the beneficial treatment effect of intravenous glutamine supplementation at 0.35 g/kg/day in critically ill patients not experiencing multi-organ failure, and receiving PN.[37] Furthermore, based on weak data from small studies, enteral glutamine supplementation of 0.3-0.5 g/kg/day should only be considered in burn or trauma patients, and not in general critically ill patients.[37]

Four days later (day 7 in the ICU), inotropic and vasopressor support, in the form of adrenaline, was restarted for new-onset hypotension, and the patient required intubation and mechanical ventilation for worsening arterial hypoxaemia [partial pressure of O_2 in arterial blood (PaO_2)/FiO_2 < 300 mmHg]. In addition, she presented with an elevated urea and creatinine, metabolic acidosis and a tapering urine output. Intravenous fluid therapy, in the form of 5% dextrose water with added sodium bicarbonate, prescribed at 60 ml/hour, was administered to improve renal perfusion and treat metabolic acidosis. Furthermore, she was placed on a continuous insulin infusion for worsening blood glucose control, i.e. 10-12 mmol/l on average. Ileostomy output remained high at 2.1 l/day. The patient remained *C. difficile*-negative.

Question 3: Would you make any changes, or additions, to the patient's current feeding prescription, in view of the clinical picture on intensive care unit day 7?

A total daily energy intake of 20-25 kcal/kg was recommended owing to the new bout of stress being experienced, i.e. severe sepsis with hypoperfusion, and subsequent multi-organ failure [acute lung injury, acute kidney injury (AKI) and hypotension requiring vasoactive support]. In an attempt to reduce the risk of overfeeding and to improve blood glucose control, the additional energy delivered via the 5% dextrose water and Polygam® infusion was taken into account. The protein target was reduced to 1.2 g/kg/day since an overzealous protein delivery can worsen azotaemia in AKI patients not on renal replacement therapy (RRT). However, due to the patient's hypermetabolic and hypercatabolic state, i.e. severe sepsis, metabolic acidosis, HIV/AIDS and tuberculosis, a protein restriction of less than 1 g/kg/day was strongly discouraged, as this would have aggravated the loss of lean body mass. An upper limit for vitamin C intake was set at 100 mg/day to prevent the development of secondary oxalosis. Owing to a persistently high ileostomy output, a decision was taken to continue PN together with a trophic enteral feed. An electrolyte-free PN regimen and a low electrolyte enteral nutrition formula was recommended, in order to further avoid the worsening of electrolyte derangement.

Rationale

ICU patients exhibit an increasing spectrum of intertwined pathophysiological processes, making an individualised approach to nutrition therapy essential. In addition, a patient's nutritional needs will constantly change during his or her ICU stay, depending on the stage of critical illness, i.e. acute versus chronic or recovery phase, and depending on the treatment modalities, e.g. daily RRT and surgery.[22]

Vasopressors and inotropes are routinely given to hemodynamically unstable patients to maintain adequate blood pressure and cardiac output.[38] Adrenaline acts as an inotrope at low doses by increasing cardiac contractility, and hence cardiac output. However, adrenaline acts as an inotrope and vasopressor at high doses. Vasopressors increase blood pressure by elevating the sensitivity of the gut to vasoconstriction, thereby increasing the risk of ischaemia and associated mortality.[38] However, the general consensus remains that vasopressors are not a contraindication to carefully monitored early enteral nutriton.[38,39] Previous studies have shown that early enteral nutrition in patients treated with vasopressors may improve gut perfusion and preserve bowel absorptive capacity.[39] Furthermore, Khalid, Doshi and DiGiovine[40] found that patients treated with vasopressors and fed within 48 hours of ICU admission had a lower hospital mortality of 34% vs. 44% (p-value < 0.001), and that the most severely ill patients and those receiving multiple vasopressors benefited the most from early enteral nutrition.

The ESPEN guidelines recommend a daily total energy intake of 20-25 kcal/kg body weight in the initial acute phase of illness, which should then be increased to 25-30 kcal/kg body weight once the patient progresses to the chronic or the recovery phase of illness.[22] This is in line with the recently published Kidney Disease: Improving Global Outcomes (KDIGO) recommendations of 20-30 kcal/kg body weight in patients with any stage of AKI,[41] as well as the recently published recommendations by McCarthy and Phipps[42] suggesting an energy intake of 20-30 kcal/kg body weight, with up to 35 kcal/kg body weight/day, if undergoing continuous RRT.

The protein target for patients with AKI should be calculated based on the underlying condition and treatment modality, i.e. no RRT versus intermittent RRT versus continuous RRT.[42] Restricting protein intake with the aim of preventing or delaying the need for RRT should be avoided (grade 2D).[41] The hypercatabolic patient, e.g. AKI secondary to severe sepsis, is more susceptible to malnutrition and increased protein losses owing to metabolic acidosis, uraemia, fluid and electrolyte imbalances, as well as physiological stress from infection, inflammation and tissue destruction.[42] Therefore, restricting protein intake to less than 1 g/kg/day may aggravate malnutrition in hypercatabolic patients. Lastly, vitamin C intake should be restricted to less than 100 mg/day in AKI patients not requiring continuous RRT since it may precipitate secondary oxalosis, leading to delayed recovery of renal function.[42]

Question 4: On day 7, the attending physician asks whether or not intravenous glutamine supplementation should be continued (or added if not yet prescribed). What do you suggest and why?

Glutamine supplementation (intravenous and enteral) should be discontinued on the basis of multi-organ failure, i.e. AKI, acute lung injury (PaO_2/FiO_2 < 300 mmHg), and hypotension requiring vasoactive support (see the rationale provided for question 3).

The patient was anuric the next day, i.e. day 8 in the ICU, and required daily RRT in the form of sustained low-efficiency dialysis (SLED). Intravenous fluid therapy was stopped, and fluid intake derived from the feeding prescription alone. The patient's blood culture grew vancomycin-resistant *Enterococcus faeciam* and the linezolid antibiotic therapy dose and frequency was escalated. The ileostomy output reduced to 600-800 ml/day.

Question 5: Will the patient's nutritional requirements have altered since the previous day (day 7)? If yes, explain why, and how you would adjust her feeding prescription?

Yes. The protein and micronutrient intake was increased to compensate for the increased losses of amino acids, peptides, water-soluble vitamins and trace elements via the dialysate. An energy- and protein-dense feed was recommended in order to achieve an adequate nutrient intake within a restricted fluid volume.

Rationale

Critically ill patients with AKI often require RRT in the form of highly efficient modalities, such as SLED or continuous haemofiltration or dialysis, with potentially relevant effects on the nutrient balance.[43] A protein catabolic rate in the range of 1.4-1.75 g/kg body weight/day has been reported, especially in those treated with continuous RRT.[43-45] Furthermore, approximately 0.2 g amino acid is lost per litre of filtrate, amounting to a total daily loss of 10-15 g amino acid. In addition, 5-10 g of protein are lost per day, depending on the type of therapy and dialyzer membrane used.[41] A daily protein target of at least 1.5 g/kg body weight(1.5-2 g/kg body weight) is recommended in patients on intermittent dialysis, e.g. SLED, in order to achieve a less negative or nearly positive nitrogen balance and to offset losses via the dialysate.[42-45] Although the optimal dose of micronutrient supplementation in AKI is unknown, water-soluble vitamins, e.g. thiamine and vitamin C, as well as trace elements, especially copper and selenium, are easily removed by RRT. Hence, daily supplementation with a standard dose of multi-trace element preparations is recommended.[42-45]

The patient markedly improved over the next five days. The laboratory and blood gas analysis showed a gradual decline in septic markers, as well as an overall improvement in acid base status and renal function. By day 14, RRT was discontinued, and the patient no longer required inotropic and vasopressor support. Furthermore, after several trials of spontaneous breathing, the patient was successfully weaned from the ventilator. However, she remained immensely weak, and received daily physiotherapy to ensure adequate mobilisation. Her ileostomy output reduced to 300-400 ml daily. She was transferred to a high care unit and referred to the dietitian for nutritional rehabilitation.

Question 6: What would you suggest to ensure optimal nutritional and functional recovery?

This was the opinion of the audience, hence change sentence to: "A rehabilitation plan with the aid of a physiotherapist and dietitian should be implemented in an attempt to regain lean body mass and functional capacity. This plan should consist of daily mobilisation, combined with optimal nutrient delivery, i.e. energy 35-45 kcal/kg body weight and protein 1.5-2.5 g/kg body weight. Early on, the patient's swallowing ability and risk of aspiration should be assessed by a speech therapist, followed by the initiation of oral feeds, with the necessary consistency changes. Energy- and protein-dense oral nutrition supplements aid in achieving an adequate energy and protein intake, thereby facilitating weight gain. In the case of persistently inadequate oral feeds, enteral tube feeding should be switched over to intermittent feeding at night, rather than during the day, to allow the patient to freely partake in physiotherapy sessions during the day. Importantly, enteral nutrition should only be discontinued once 60-80% of the patient's nutrient targets have been met via the oral route alone. Additional micronutrient supplementation should also be considered, especially in the case of an established deficiency. Furthermore, the patient's ileostomy output must be monitored for possible diarrhoea or constipation, and the nutrition care plan adjusted accordingly.

Rationale

Optimal nutrition therapy continues to play a crucial role during the chronic and recovery phase of critical illness. Guillain-Barré syndrome, in particular, is associated with prolonged weakness.[46] Approximately 40% of patients diagnosed with Guillain-Barré syndrome need inpatient rehabilitation.[21] As mentioned previously, Guillain-Barré syndrome patients often lose a significant amount of weight, and this, together with prolonged immobilisation, makes such patients highly susceptible to the development of bed sores.[12,46] Furthermore, Guillain-Barré syndrome patients are at risk of muscle shortening and joint contractures.[46] Providing optimal nutrition therapy, in combination with daily physiotherapy, aids in rebuilding lean body mass, ultimately improving physical function and quality of life. According to Thibault and Pichard,[47] hospitalised long-stay patients in the post-acute phase of illness who are malnourished should receive 30-35 kcal/kg body weight. Higher energy and protein targets of 40-45 kcal/kg body weight and 2-2.5 g protein/kg body weight have previously been suggested in patients with Guillain-Barré syndrome in order to reduce muscle wasting.[20] Since Guillain-Barré syndrome patients often present with dysphagia secondary to oropharyngeal weakness and prolonged ventilation,[20] consistency changes may improve swallowing and reduce the risk of aspiration. Furthermore, Guillain-Barré syndrome patients who are in the recovery phase often present with constipation, mostly as a result of long-term immobilisation and the administration of opiates for pain control.[20,21] Therefore, regular monitoring of a patient's

ileostomy output is important.

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