Nutritional support of the patient receiving high-dose therapy with hematopoietic stem cell support

By Sheryl McDiarmid

Hematopoietic stem cell transplantation (HSCT) is an intensive therapy that is being used increasingly in an attempt to cure certain malignancies. One of the major adverse effects of this treatment is an inadequate oral intake that may result in dehydration and malnutrition. Factors that may contribute to inadequate oral intake include mucositis, nausea, vomiting, and anorexia. In addition, prior to transplant, many patients may have experienced, or continue to experience malnutrition associated with malignancy and its therapy.

Traditionally, total parenteral nutrition (TPN) has been the mainstay of nutritional support in this patient population. The blood and marrow transplant (BMT) team at the Ottawa Hospital has significantly decreased the use of TPN through the initiation of a comprehensive nutritional support program that uses a variety of interventions including oral supplementation and enteral feeding.

Understanding the causes and implications of malnutrition, and using tools that allow risk assessment and timely implementation of appropriate nutritional interventions, may facilitate full patient recovery parallel to hematopoietic recovery in the HSCT patient population.

Hematopoietic stem cell transplantation (HSCT) is being used increasingly as a treatment in an attempt to cure certain malignancies. Advances in stem cell technology and the use of colony-stimulating factors allow for rapid hematopoietic recovery that may result in a decrease in both length of hospital stay and resource utilization. However, HSCT is an intensive therapy that requires full use of supportive care interventions to ensure that the “whole” patient has recovered by the time blood counts normalize. One clinical condition that can extend the length of stay or result in re-admission post-HSCT is an oral intake inadequate to meet nutritional requirements and maintain hydration status. In general, the average length of stay of malnourished patients is twice as long as in diagnosis-adjusted well-nourished patients (Nixon et al., 1980; Ottery, 1995).

Adequate nourishment is necessary for a number of body functions. Delayed wound healing, reduced rate of drug metabolism, and impairment in physical and cognitive function have been reported in malnourished patients (Sullivan, 1995). Most nutritional deficits lead to suppressed immune responses, as the anabolic and catabolic pathways of the immune system require the same building blocks and energy sources as other physiological activities. Protein-calorie malnutrition disrupts the production of specific antibodies and complements and is a major cause of immunodeficiency.

Unfortunately, nutritional support interventions may not be given a priority throughout the transplant process, and are often only addressed when inadequate nutritional status interferes with discharge. To ensure an adequate nutritional status post-transplant, nutritional assessment and interventions must be initiated well before high-dose therapy (HDT) begins.

HSCT is rarely first-line therapy in the treatment of malignancies. Most patients undergoing this treatment have received prior courses of therapy, both chemotherapy and radiation therapy, for their malignancy. Often, patients are already in a malnourished state on presentation to the transplant centre, a state which will only be exacerbated by the toxic effects of HSCT. The identification of malnutrition has been based on objective measurements including weight, serum concentrations of proteins produced by the liver, anthropometric measurements, grip strength, energy, immunological functions, and the body-mass index. No single measurement is highly sensitive and specific in identifying malnutrition (Soubra, 1997). Progressive malnutrition leads to vital organ wasting with impairment of function. As the severity of malnutrition increases, there is an increase in morbidity and mortality (Grant, 1992). The ideal time to initiate specialized nutrition for patients undergoing HSCT is unclear (Mattox, 1999). However, nutritional support for individuals with cancer has been shown to decrease morbidity and mortality by preventing weight loss, increasing response to treatment, minimizing the side effects of therapy, and improving quality of life (Bloch, 1990).

As with any other illness, malnutrition cannot be treated if it is not recognized and correctly diagnosed. Patients at risk for developing malnutrition, or who are already in a state of malnutrition, must be identified, and nutritional support implemented, if optimal care is to be given (Grant, 1992). Ottery (1995) suggests that nutritional intervention in end-stage malnutrition is potentially no more successful than chemotherapeutic approaches in end-stage malignancy. Goals to maintain optimal nutritional status should be set with the patient during the initial pre-transplant assessment.

This article focuses on nutritional support of the patient being prepared for and receiving HSCT. The information presented in this paper is based on an extensive literature review, as well as clinical experience at our institution and other HSCT centres in Canada. Part One discusses causes and implications of malnutrition in the patient who has a malignancy and who has received, or is presently undergoing HSCT. Part Two reviews the components of nutritional screening and identifies the types of nutritional support available. The reader is also provided with an algorithm for selection of timely and appropriate nutritional support interventions (see Figure One).

As health care resources continue to shrink, nurses may increasingly find that they must assume responsibility for services previously provided by a nutritional support team. Nurses, with guidance from members of the nutritional support team, are in an excellent position to monitor patients’ nutritional status, recommend interventions, and evaluate their effectiveness.

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Figure One: Algorithm for nutritional support alternatives

Low risk patients (Stage A PG-SGA global assessment categories)
Does patient eat enough calories/day in regular diet?
Yes
Regular oral diet
No
Oral diet + supplements

Moderate risk patients (Stage B PG-SGA global assessment categories)
Can patient eat adequate oral diet + oral supplements?
Yes
Oral diet + supplements
No
Consider algorithm for high-risk patients

High-risk patients (Stage C PG-SGA global assessment categories)
Can patient eat adequate oral diet + oral supplements?
Yes
Oral diet + supplements
No
Gut can be used while patient receiving treatment
Monitor daily
Evaluate for enteral feeding
Vomiting under control
(restart here if tube is vomited up)

Does patient have mucositis?
Yes
Administer pain medication as ordered
Administer anxiolytic as ordered
No
Administer anxiolytic as ordered

Is patient thrombocytopenic?
Yes
Administer platelets as ordered then insert nasogastric tube
No
Insert nasogastric tube
MD verifies placement
Start enteral feeding according to protocol

Gastric Residuals High
Start Domperidone/metoclopramide as ordered
Decrease rate - if this rate does not meet caloric needs consider augmenting with TPN

Diarrhea increasing
Discontinue medications via feeding tube
If stool neg for C diff
administer Lomotil as ordered
If stool positive for C diff
administer Vanco as ordered

Diarrhea decreasing/status quo
Continue enteral feeding

Increase rate

Is vomiting under control?
Yes
Re-evaluate for enteral feeding
No
Consider TPN

* TPN is preferred nutritional supplement for patients with graft versus host disease involving the GI tract where patients need to be NPO to rest the gut
Part One: Selected causes of malnutrition associated with HSCT

Malnutrition is the most common secondary diagnosis in cancer patients (Ross, 1990). It results from a number of factors related to reduced oral intake, increased metabolic requirements of patients, systemic effects of treatment, and the tumour itself. Because of the intensity of therapy and the duration of symptoms, HSCT often negatively affects nutritional intake.

Inadequate intake is termed oligophagia and may result from many factors that include anorexia, nausea, vomiting, dyspepsia, mucositis, alteration in taste and smell, or learned food aversions.

Anorexia is a monumental barrier and is frequently the prime cause for weight loss and cachexia. Anorexia is a diminished appetite due to a reduced or absent sensation of hunger, despite the clear need for nutrients. Appetite is the psychological desire to eat that should normally accompany the physical manifestations of hunger, such as gnawing feelings of pressure and tension in the epigastric region. Appetite is developed through past experiences, family customs, and religion, and is responsible for the type of food that is preferentially sought. Frequently, anorexia is the cumulative result of other symptoms, but it may also be present without any identifiable reason. The emotional and psychological effects of cancer on a patient can have a great impact on appetite. The presence of uncontrolled pain can also greatly affect appetite, as well as oral intake. The need for patients who are anorexic to take multiple oral medications can compromise oral intake as they complain of not having enough room in their stomach for both food and medications. As well, medications themselves can be anorexigenic. Anorexia is most distressing, as patients undergoing high-dose therapy require 50-70% more calories and twice as much protein compared with normal intake.

Nausea and vomiting from antineoplastic chemotherapy and radiotherapy are among the most challenging side effects to control (Hecht, Lembo, & Chap, 1997). The combined effects of chemotherapy and the use of total body irradiation in the HSCT produce extensive tissue and organ damage, subsequently increasing the incidence and intensity of gastrointestinal effects. Nausea and vomiting are separate concepts, however, terms used to describe them are often used interchangeably. This can lead to confusion and inadequate understanding for prevention, education, and research. Rhodes, Johnson, and McDaniel (1995) describe nausea as a subjective, unobservable phenomena of unpleasant sensation experienced in the back of the throat and the epigastrium that may or may not result in vomiting. In contrast to vomiting, very little is known about the physiology of nausea. Nausea usually precedes vomiting, and suboptimal doses of emetic agents can evoke nausea alone (Andrews, Rapeport, & Sanger, 1988). Nausea is perceived as the most distressing side effect of antineoplastic treatment (Cooper & Georgiou, 1992). Sweating, pallor, salivation, dizziness, lightheadedness, and tachycardia often accompany it. Unlike nausea, vomiting may be objectively measured. Following vomiting, it is not uncommon for nausea to be alleviated and the patient to report feeling better.

Side effects directly related to administration of antineoplastic therapy may be compounded by psychological factors, so that nausea and vomiting may occur at other times as a result of learning processes. These clinical conditions are termed anticipatory and delayed nausea and vomiting. Conditioned nausea and vomiting occurring during or after drug administration may be difficult to distinguish from drug-induced effects. Despite gains achieved by antiemetic therapy, both anticipatory and delayed nausea and vomiting persist as problematic side effects. Morrow, Lindke, and Black (1991) argue that this is due to the use of more aggressive cytotoxic regimes that offset the improvements in antiemetic agents. As most patients undergoing HSCT have had previous chemotherapy, assessment of anticipatory and delayed nausea and vomiting could help in planning for nutritional support.

Dyspepsia describes a general abdominal discomfort following a meal. Gastroesophageal reflux disease (GERD) is the medical diagnosis, but patients frequently use terms such as acid indigestion and heartburn. The literature suggests that approximately 10% of the population have heartburn daily, and that more than a third have intermittent symptoms (Nebel, Fornes, & Castell, 1976). The prevalence of dyspepsia may increase in patients receiving cancer therapy as a consequence of the polypharmacy that often accompanies treatment (Otto, 1994). Assessment of dyspepsia is important because patients often self-medicate for this symptom. Drug interactions with antacids are well-recognized, so it is important that patients are informed to alert the health care team if they have GERD, so an appropriate therapy may be instituted.

Acute oral complications are a frequent and often serious problem associated with HSCT. The most notable complications include general mucosal inflammation and breakdown. This condition is known as mucositis. Conditioning regimens containing radiation, and the use of Methotrexate prophylaxis in the allogenic transplant population, increase the severity of mucositis. Sore mouths resulting from oral mucositis may also decrease oral intake, as patients are unable to initiate a swallow because of pain. Cytotoxic drugs may damage the basal layers of the oral mucosa, and combined with poor nutrition, can cause thinning of the epithelium and loss of dorsal tongue papillae. This atrophied epithelium is inadequate to resist the normal wear and tear of eating and talking.

Oligophagia may also be caused by indirect factors such as alterations in taste and smell. Assessment of the mouth should be undertaken in all patients. Many cancer patients complain of taste alterations, and they can be serious enough to affect nutrition (Wickham et al., 1999). Taste bud sensitivity may be modified by chronic changes in the local environment such as diminished saliva and/or candidiasis. Poor oral hygiene affects taste by allowing a physical barrier of debris to cover taste buds causing taste fatigue. This decaying material may also mask flavour detection (Otto, 1994). Unpleasant tastes can induce nausea and vomiting; whether this is a primary response or secondary as a result of prior exposure is unclear. Patients with altered taste may have decreased caloric intake and an increased incidence of weight loss compared with patients with normal taste (DeWys & Pascucci-Cimino, 1978). Medications such as antibiotics, antifungals, narcotics, and some chemotherapeutic agents have been implicated in taste alterations. Patients with dry mouth, generally secondary to radiation, have diminished salivary secretions. This is compounded if they become dehydrated or are febrile. Lack of saliva makes it difficult to chew and swallow many foods.

Learned food aversions, a result of the association between certain foods and unpleasant symptoms, can contribute to oligophagia. The incidence in the general healthy population has been estimated at 38% to 65% (Mattes, Arnold, & Boraas, 1987). To avoid this syndrome, patients were traditionally told to refrain from eating large amounts of food before therapy involving a drug known to cause nausea and vomiting. Studies now show that food aversions may form at any time during the 48 hours before or after the first therapy. It is not reasonable for patients to avoid eating for four days to decrease the risk of developing food aversions. Aversion to nutritional supplements may also develop by this mechanism. Nutritional supplements may have been prescribed to optimize oral intake prior to HSCT; it is important to determine if, and what type of supplements are an option once the need arises.

There are many variables contributing to the potential for malnutrition in patients undergoing HSCT. The extent of the need for nutritional support depends on the overall goals of the patient and the health care team. The intent of HSCT is curative, so supportive care resources should be maximized to augment this treatment. Nutritional support interventions should be used as a strategy to optimize response to therapy and enhance recovery post-HSCT.
Part Two: Nutritional screening and nutritional support interventions

When planning nutritional care during HSCT, an individualized nutritional assessment and program of nutritional support are essential (Vaughn & McTierman, 1986). Although assessment of nutritional status is difficult, and no single indicator appears to be valid in isolation (Reilly, Martineau, Moran, & Kennedy, 1995), there are many assessment tools available. Given the need for a rapid, but valid assessment, Dr. Faith Ottery’s Patient-Generated Subjective Global Assessment Tool (PG-SGA) is a well-developed and validated tool (Ottery, 2000). The tool takes five minutes to complete. The patient fills out the first part of the form (history of weight, food intake, symptoms, and functional capacity). Next, the clinician completes the remainder of the form based on findings from the physical examination. The PG-SGA score is derived by adding the patient score to the clinician’s score, and categorizing the patients as well-nourished (Stage A), moderately malnourished (Stage B), or severely malnourished (Stage C). Table One summarizes the PG-SGA global assessment categories. Once an assessment is performed, it is critical that this information is used to develop a plan that involves all healthcare team members, followed by implementation, evaluation, and revision as needed.

Although some patients who are candidates for HSCT may begin their journey in the well-nourished category, many patients are in the Stage B or Stage C category when they first present to the transplantation centre for evaluation. The potential for all patients to develop a plan that involves all health care team members, followed by implementation, evaluation, and revision as needed.

Although some patients who are candidates for HSCT may begin their journey in the well-nourished category, many patients are in the Stage B or Stage C category when they first present to the transplantation centre for evaluation. The potential for all patients to become moderately or severely malnourished exists, and it is only through adequate assessment, intervention, and evaluation that nutrition will be optimized in the HSCT patient.

Now that you have all this information, how do you decide on what nutritional support interventions should be recommended to provide adequate nutrition and maintain or improve status? This section will discuss three nutritional support interventions: oral supplementation, enteral feeding, and total parenteral nutrition.

Oral supplementation

The motto of oral supplementation should be “make every mouthful count.” Maximizing oral intake for hospitalized patients is challenging, so it is essential that patients be made aware of the importance of adequate nutrition during the pre-HSCT phase, when most of their time is spent out of the hospital. Many health care providers assume that people are well-informed about the principles of good nutrition, however, now is the time to talk about nutrition basics. Patients can become so overwhelmed by the technological interventions that they minimize the impact of sound health maintenance practices, such as nutritional intake. The provision of written material on sound nutritional advice, combined with a dietary consult, may assist the patient to choose appropriate nutritional interventions for specific eating problems. Changes in food texture, timing of meals and snacks, increasing nutrient density of foods, and using high-calorie, high-protein food and/or supplements are examples of interventions that may be required.

Most clinicians are aware of commercial supplements that are available in hospitals. However, homemade supplements may be as nutritionally sound and should be encouraged to increase variety. The dietician can provide patients and family members with information on how to prepare homemade supplements. As an increasing proportion of care is delivered in the outpatient setting, interventions that can be customized to be carried out in the home will be essential. Forty per cent of the total length of stay for the HSCT process in our institution is spent in the outpatient setting.

When oral food consumption becomes inadequate, oral intake may need to be supplemented to meet caloric requirements. The two approaches to nutritional supplement that may be considered are enteral feeding and total parenteral nutrition (TPN).

Enteral feeding

Enteral feeding is a method of providing a nutrient solution into the GI tract by way of a tube. The route may be via a nasogastric (NG), nasointestinal (NI), gastrostomy, or jejunostomy tube. The use of enteral nutrition support has increased because of improvements in formulas and delivery systems, and because clinicians now have a greater appreciation of its merits. Using the gastrointestinal system whenever possible preserves gastrointestinal function and minimizes infections related to enteric pathogens. The motto of enteral nutrition

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<th>Table One: PG-SGA global assessment categories</th>
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<tr>
<td><strong>Category</strong></td>
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<tr>
<td>Weight</td>
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<tr>
<td>No weight loss or recent nonfluid weight gain</td>
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<tr>
<td>No weight stabilization or weight gain</td>
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<tr>
<td>Nutrient intake</td>
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<td>Nutritional impact symptoms</td>
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should be “if the gut works, use it” (Kaminski & Blumeyer, 1993). The gastrointestinal tract was once believed to be a passive conduit for nutrient absorption that functioned suboptimally in the critically ill patient and could easily be bypassed through the use of parenteral nutrition. It is now evident that the gut plays a central role in the injury response, and that the maintenance of normal gut function through enteral feeding and the provision of essential nutrients may hasten recovery and improve survival (Heinburger & Weisner, 1997; Wilmore, Smith, & Odwyer, 1988).

Bacterial translocation is the movement of bacteria and endotoxins, which normally colonize the gut, across the intestinal mucosa, causing invasion of organs and tissues. Impaired host immune defenses and gut mucosal injury, both of which are common in HDI, facilitate bacterial translocation. The presence of food in the gut helps inhibit mucosal penetration by pathogens. Food stimulates the production of secretions which help maintain GI tract barrier function and stimulates continual epithelial cell turnover, peristalsis, and defecation which decreases exposure to and subsequent colonization of the mucosa by pathogens (Phillips & Olsen, 1993).

There are four contraindications to enteral nutrition: bowel obstruction, shock, >90% bowel resection, and mesenteric ischemia (ASPLEN, 1987). Unless contraindicated, enteral nutrition may be initiated as soon as patients are unable to meet their daily nutritional needs through regular oral intake, including supplementation.

**Reasons for enteral feeding failures**

One of the main barriers to enteral feedings is the negative attitude by members of the health care team and patients. This may be overcome by reinforcing the idea of optimal nutritional support. While it may seem easier to be fed through the central venous catheter, this may not be optimal nutritional support for many patients. Members of the team have to be consistent in their approach to nutritional support.

The role of enteral nutrition should be valued at the same level as the choice of antibiotics or chemotherapy. Early counselling about nutritional support, and the provision of written information stressing the philosophy of nutritional support, can emphasize the positive attributes of enteral feeding in optimizing patient care and recovery. Nurses can help the patient cope with changes in body image by offering acceptance, emotional support, and education.

Other reasons for failure of enteral feeding include nausea and vomiting, which may result in expulsion of the feeding tube, diarrhea, and high gastric residuals. Assessment of the symptoms described by the patient should be undertaken to evaluate the success of enteral feeding. Nurses, physicians, dieticians, and patients may identify nausea and vomiting as the same symptom and may be reluctant to pursue enteral feeding. As discussed earlier, many more patients experience nausea than frank vomiting and there may be long intervals between episodes of vomiting (Cooper & Georgiou, 1992). Nausea is not automatically a deterrent to enteral feeding. In fact, vomiting may not be either. Gulley, Vander Pleeg, and Gullley (1993) described a protocol for the treatment of hyperemesis gravidus that uses continuous infusion of an iso-osmolar tube-feeding product. This group used the enteral feeding route to treat the symptoms that traditionally argue against the use of enteral feeding in oncology patients. Vomiting and expulsion of the feeding tube, if otherwise the enteral feeding is successful, should not be considered as the only criterion to stop. In our experience, patients who have been educated about the importance of optimal nutrition are not averse to having the feeding tube reinserted.

Diarrhea is a frequent complication of HDI, so it is difficult to establish the causative factor. Thomas (1994) suggests that the type of tube feeding formula is rarely the cause of diarrhea, and it is unnecessary to stop the tube feeding formula until the cause is identified. Of the various mechanisms of diarrhea, osmotic diarrhea is a relatively common cause (Greenwood, 1994). The major cause of osmotic diarrhea is the administration of medications that increase the osmotic load. While it may seem advantageous to give medication through the feeding tube, each medication must be assessed for the osmolality and/or sorbitol content to determine its propensity to cause diarrhea. Medications implicated by Thomas include magnesium antacids, cimetidine, potassium and phosphorous supplements, quinidine compounds, lactulose, and other laxatives. Many elixirs contain sorbitol, frequently in laxative dosages that cause diarrhea by stimulating GI motility and increasing the secretory process. If medications are suspected as the contributing factor, it may be reasonable to change to a nonelixir formula or to parenteral administration.

Diarrhea may be present in patients receiving antibiotics, due to altered colonization of the GI tract. This is true independent of whether the patient is enterally fed or parenterally fed. However, severe diarrhea may result in malabsorption of nutrients and parenteral nutrition may then be indicated.

Graft-versus-host disease (GVHD) is a complication of allogenic transplantation and may be exhibited in the skin, liver, and/or gastrointestinal tract. The incidence of acute GVHD, which occurs within the first 100 days after transplant, ranges from 20% to 80% depending on the degree of histocompatibility, the number of T-cells in the graft, the patient’s age, and the prophylactic immunosuppression regimen used. Gastrointestinal GVHD can be manifested by diarrhea with or without nausea and vomiting, abdominal pain, and/or ileus. Diarrhea associated with GVHD has a characteristic watery green appearance. Treatment for intestinal GVHD includes corticosteroids and gut rest until there is a decrease in symptoms. This precludes the use of enteral feeding during the acute phase.

Another complication of transplantation, again primarily in the allogenic setting, is veno-occlusive disease of the liver (VOD). VOD is a clinical syndrome characterized by painful hepatomegaly, fluid retention, and hyperbilirubinemia. Histologically, VOD is defined as progressive and concentric narrowing of small intralhepatic venules, associated with necrosis of hepatocytes. However, the mechanisms and cellular events involved in VOD are not fully understood. The clinical diagnosis of VOD is based on the presence of jaundice, hepatomegaly, and/or right upper quadrant pain and ascites, and/or unexplained weight gain. The literature does not report any studies using enteral feeding in this group of patients. The use of total parenteral nutrition is limited in this setting by both the known liver toxicity associated with the use of TPN, and the need to restrict fluid intake.

The most dangerous complication of enteral feeding is aspiration of the feed into the lungs (Burchett, 1994). In addition to assessing absorption, ensuring that gastric residuals are minimal reduces the risk of aspiration should the patient vomit. To prevent aspiration, elevate the head of the bed at least 30 degrees during feedings and for an hour after feeding (Bowers, 2000). Delayed gastric emptying is caused by sepsis, elevated blood sugar levels, diabetic gastroparesis, elevated body temperature, ischemia or surgical repairs, and the formula fat content (Thomas, 1994). Gastric feeding is appropriate for patients who have intact gag and cough reflexes and adequate gastric emptying. Nasoduodenal tubes and nasojejunal tubes are generally indicated for critically ill patients at risk for pulmonary aspiration or delayed gastric emptying (Bowers).

An algorithm for initiating nutritional support alternatives is offered as a decision-making tool (see Figure One). Feeding regimens vary, so know your facility’s policy regarding introduction of enteral feedings, care and maintenance of feeding tubes, and guidelines for nursing assessment and intervention.

**Total parenteral nutrition**

Total parenteral nutrition (TPN) has been the mainstay of nutritional support in patients undergoing HSC T (Herrmann & Petruska, 1993). Although studies have shown that most allogenic transplant patients need some form of TPN during their post-transplant course, TPN has not been uniformly successful in improving...
nutritional status or outcome (Szeluga, Stuart, Brookmeyer, Utermohlen, & Santos, 1987; Weisdorf, Hofland, & Sharp, 1984). TPN consists of a solution containing glucose, amino acids, vitamins, minerals, trace elements, and lipids. It is most often infused via a central venous catheter. Although TPN may sometimes be a life-saving modality for patients unable to tolerate an enteral diet, it is not without risks. Patients receiving TPN are subject to a greater number of technical and metabolic complications such as increased risk of infection, particularly fungal infections (Herrmann & Petruska, 1993). TPN should not be used indiscriminately, particularly in patients who have a functioning gut; however, there are two standard recommendations for instituting TPN in this population. These are: (1) when therapy is expected to last for a five- to seven-day period due to GI side effects, and (2) when patients are only able to consume, either orally or enterally, 60% of their nutritional requirements for a three- to five-day period (Mattox, 1999). In acute GVHD, nutrition support is of paramount importance to facilitate tissue repair and, as previously discussed, when GVHD affects the bowel, TPN may be indicated to facilitate bowel rest. In our institution, several patients have required long-term TPN to meet nutritional needs while the bowel is recovering from GVHD. The Ottawa Hospital has a program that teaches patients to infuse their own TPN in the home. A protocol that condenses the infusion so that TPN can be infused overnight for eight to 12 hours has allowed patients to carry on activities of daily living that would be limited by continuous infusion. This has been life-saving and life-sustaining for a number of patients.

Transitional feeding

Nutritional support, like any other therapeutic intervention, requires ongoing evaluation with subsequent modification of therapy. Use of one form of nutritional support is not mutually exclusive, and patients may benefit from a combination approach. Ongoing assessment is necessary when working with patients receiving HSCT, as symptoms can change quickly. A patient may develop severe mucositis overnight, necessitating a change in texture of the diet, or the implementation of enteral feeding. The reverse is also true. We have found that with the use of peripheral blood stem cells once the neutrophil count recovers, a patient’s mucositis can improve dramatically within a 24-hour period so that oral intake can be commenced with a view to discontinuing enteral feeding.

The process of advancing from one route of feeding to another, or from one formula to another, is called transitional feeding. Due to the cost and potential metabolic and mechanical complications associated with TPN, it is highly desirable to make the transition from TPN to enteral or oral nutrition as soon as is medically feasible. Patients who have a viable GI tract with signs of GI tolerance are candidates for transition. Even when small amounts of nutrients are provided orally, the gut mucosal structure and function are enhanced. Figure Two provides an algorithm to determine transition from parenteral nutrition to enteral and oral nutrition.

There is a paucity of literature on the role of enteral feeding in HSCT. Most patients receiving HDT will continue to ingest oral food/fluids in varying amounts and should always be encouraged to increase oral intake. As more of the patient’s nutritional needs are met by oral intake, enteral feedings may be given as cyclic feeding. It is usually provided at night and is thought to increase the patient’s appetite and allow for increased ambulation during the day (Trujillo & Queen, 1994).

Nutritional and fluid intake should be monitored on a daily basis. Oral intake may progress while the patient continues to receive tube feeding at home. Patients and family members may be educated on how to administer enteral feeds at home. This shortens the length of hospitalization, as patients can increase their oral intake gradually, using enteral feeding to supplement their nutritional needs. Once the patient is consistently consuming two-thirds to three-quarters of protein and energy needs over a three-day period, the tube may be removed.

Enteral nutrition is gaining in popularity as studies have demonstrated the beneficial effect of providing nutritional support directly to the gut, the improved substrate utilization provided by enteral feeding, and the economical advantages (Johansson, Backman, & Jakobsson, 1996; Mainous, Block, & Deitch, 1994). However, further studies need to be undertaken within the HSCT patient population to determine both the patients most likely to benefit from enteral feeding, and when enteral feeding should ideally be commenced.

Implications for nursing practice

Nurses are in a key position to provide support to patients and families with regard to nutrition issues. There are many options for providing nutrition support interventions for the patient undergoing HSCT, but the most appropriate strategy for a patient should be based on a careful assessment of contributing factors with input from a multidisciplinary team. Nursing assessment and management of patients’ symptoms contributing to diminished oral intake and loss of nutrients is fundamental to the success of any intervention. Managing nausea, vomiting, and diarrhea requires excellent assessment skills and knowledge of pharmacological actions. Accurate pain assessment, followed by the administration of adequate analgesia, can assist patients to optimize their oral intake. Promoting and assisting the patients to perform good oral hygiene minimizes the possibility of bacterial introduction through the oral cavity, and also helps maintain the integrity of the oral mucosa which is so vital to the success of re-establishing adequate oral nutrition.

Nurses can also provide support to family members who are often frustrated with their inability to improve oral intake in patients. Often, frustration leads to pressure on the health care team to initiate TPN. Nurses can reinforce the benefits of oral nutrition and assist the patient and family to set goals and establish interventions that will meet both short- and long-term needs.

In terms of research, this paper provides an overview of the causes of malnutrition in the patient undergoing HSCT and reviews the types of nutritional support available. However, further research is needed in a variety of areas related to nutritional supplementation in this patient population. Although at the Ottawa Hospital we have been utilizing enteral feeding as a nutritional support strategy in our HSCT patient population for the past eight years, we have not evaluated the impact of our interventions on patient outcomes. Research that will provide evidence to guide practice in the area of nutritional support is warranted.
References


