NUTRITIONAL CHALLENGES IN MYELOFIBROSIS PATIENTS TREATED WITH STEM CELL TRANSPLANTATION

Melissa Levine
Sodexo Dietetic Internship
2012-2013
# TABLE OF CONTENTS

Introduction ........................................................................................................................ 1

Discussion of the Disease ................................................................................................. 2-22
   I. Myelofibrosis ........................................................................................................ 2-4
   II. Symptoms .......................................................................................................... 4-6
   III. Methods of Diagnosis ...................................................................................... 7-8
   IV. Prognosis .......................................................................................................... 8-9
   V. Palliative Treatment Options .......................................................................... 9-12
   VI. Curative Treatment: Stem Cell Transplantation ........................................ 12-21
   VII. New Treatment Options .............................................................................. 21-22

Medical Nutrition Therapy ................................................................................................ 23-50
   I. Goals of Medical Nutrition Therapy ................................................................... 23
   II. Nutrition Requirements for Adults .................................................................... 23-28
   III. Nutrition Management and Support ................................................................ 29-31
   IV. Treatment Side Effects and Nutritional Interventions .................................... 31-38
   V. GVHD and Nutritional Interventions ................................................................ 38-41
   VI. Nutrition Care Process .................................................................................... 41-50

Presentation of the Patient ............................................................................................. 51

Medical and Nutritional Hospital Course ......................................................................... 52-57

Critical Comments ........................................................................................................ 58-59

Patient Update ................................................................................................................. 60

Summary .......................................................................................................................... 61

Medication Bibliography ................................................................................................ 62-69

References ....................................................................................................................... 70-72

Appendix ........................................................................................................................ 73-78
INTRODUCTION

My previous knowledge of the nutritional implications involved in cancer treatment was somewhat rudimentary; I knew the basics in that it involved taste changes and weight loss, but beyond that I was never fully aware of the nutritional struggles patients face and how they can affect their overall chance of survival. I was also unaware of the side effects of medications that ease symptoms but cause other unintended nutritional issues. Finally, I was unaware of the challenges care teams face in prioritizing diagnoses and nutritional interventions when the treatment to cure the cancer leads to other medical complications.

When my oncology rotation at Mount Sinai was approaching, I was given a variety of articles to study before beginning. Through these articles, my interest in stem cell transplantation increased as I was intrigued by how much of a role nutrition plays in patient care in this population. Once my rotation started, I began to see and appreciate the interactions between the dietitian and patients and also the dietitian and the other disciplines involved in patient care. The team approach was especially interesting to experience, and – as I learned – patient care in this population is often about prioritizing interventions to best meet the needs of the patient, and therefore communication is key.

For all of these reasons and more, I chose the topic of nutritional management in patients treated with stem cell transplantation. In entering into the research portion of this paper, I wanted to learn as much as possible about how to manage symptoms and keep a patient nutritionally sound in the short- and long-term post-treatment. This paper offers a thorough review of the research available now and what we can expect in the future.
**DISCUSSION OF THE DISEASE**

**I. MYELOFIBROSIS**

Myelofibrosis (MF) is a myeloproliferative neoplasm (MPN) characterized by progressive bone marrow fibrosis and hematopoiesis. The word myelofibrosis stems from the prefix “myelo-” meaning “from the marrow” and “fibrosis” meaning “formation of scar tissue.” In normal individuals, hematopoietic stem cells (immature cells) are created in the bone marrow and gradually mature into one of two specialized cells: a myeloid stem cell or a lymphoid stem cell. Lymphoid cells become white blood cells while myeloid cells become one of three mature cells. These mature cells include red blood cells that carry oxygen throughout the body, white blood cells that fight infection, and platelets that create blood clots.¹

![Cell differentiation diagram](image)

Individuals with an MPN have slow-growing cancer of the blood – also known as a chronic leukemia – in which “large numbers of abnormal red blood cells, white blood cells, or platelets grow and spread in the bone marrow and the peripheral blood”.¹ In short, the bone marrow –
which produces the body’s cells – produces cells that develop and/or function in an abnormal way. The six MPN’s include:

- Chronic myelogenous leukemia (CML) – hyperproliferation of abnormal white blood cells (granulocytes) in the bone marrow
- Polycythemia vera (PV) – hyperproliferation of red blood cells in the bone marrow
- Essential thrombocytopenia (ET) – hyperproliferation of platelets in the bone marrow
- Chronic neutrophilic leukemia (CNL) – too many blood stem cells become neutrophils
- Chronic eosinophilic leukemia (CEL) – hyperproliferation of white blood cells (eosinophils) in the bone marrow
- Myelofibrosis (MF) – abnormal blood cells and fibers form in the bone marrow

MF is the most rare of the MPN’s and is the most difficult to diagnose due to its similarity in symptoms to those of the other MPN’s. MF has a yearly incidence of 5-10 cases per 1 million people (or <2 people per 100,000) with an average age of 65. As defined by the National Cancer Institute, in primary MF “large numbers of blood stem cells become blood cells that do not mature properly (blasts).”² A main feature of MF is the production of too many megakaryocytes, which are giant cells in the marrow that break up into fragments and produce hundreds to thousands of platelets. Platelets are small blood cells that stick to the site of a blood vessel injury and form a plug to seal off the injured vessel to stop bleeding. “Normally, new platelets are made to replace used platelets in the body.

With MF, extra magakaryocytes are made and many die off too early which causes excess platelets to be released into the blood and chemicals called
cytokines to be released into the marrow. The cytokines stimulate the development of collagen and fibrous connective tissue in the marrow”. This fibrous tissue inside the bone marrow then becomes increasingly thick and slows the blood-forming tissue’s ability to make blood cells. This leads to a severe reduction in the creation of red blood cells in the bone marrow. In order to make up for the low number of blood cells made in the bone marrow, the liver and spleen begin to make the blood cells. This often leads to splenomegaly, a major characteristic of MF, and less commonly leads to hepatomegaly.

There are two types of MF, which are characterized mainly by the disease course. The first is primary myelobibrosis (PMF), which presents as a “de novo” disorder, and the second is secondary MF, which evolves from polychthemia vera (post-PV MF) or essential thrombocythaemia (post-ET MF). Both types of MF are MPN’s and are characterized by bone marrow fibrosis, osteosclerosis, extramedullary hematopoiesis (EMH), splenomegaly, abnormal cytokine expression, and leucoerythroblastic anemia”.

II. SYMPTOMS

The main characteristics of MF are collagen fibrosis in the bone marrow leading to anemia, leucopenia (low WBCs) or leukocytosis (high WBCs), thrombocytopenia (low platelets), and extramedullary haemopoiesis (blood formed outside of the bone marrow) leading to splenomegaly. Constitutional symptoms of MF often arise from splenomegaly and include pain/fullness below the ribs on the left side, early satiety, fatigue, dyspnea, bruising easily, fever, night sweats, pruritus, and weight loss.

A. Collagen fibrosis

“Clonal myeloproliferation in MF is accompanied by bone marrow fibrosis, from which the name of MF is derived historically. Although fibrosis is recognized as a secondary
phenomenon, it remains pathognomonic for MF. In the prefibrotic stage, the bone
marrow displays marked hypercellularity with several classes of atypical megakaryocytes
and granulocytes, followed by reticulin collagen fibrosis or osteosclerosis in the fibrotic
stage. The fibrotic stage is typically associated with leukoblastosis, hepatic
splenomegaly, and extramedullary hematopoiesis (EMH), particularly in the spleen but
also at other sites. Cellular abnormalities in MF are detected in a peripheral blood smear,
which typically shows nucleated red blood cells and immature granulocytes. The
development of reticulin and/or collagen fibrosis in the bone marrow space in MF
contributes to insufficient hematopoiesis followed by worsening cytopenias, resulting in
significant morbidity and mortality”.

B. Anemia, leucopenia, thrombocytopenia

Anemia, leucopenia, and thrombocytopenia are caused by the basic feature of MF: blood
cells are produced in the bone marrow and do not mature. In MF patients, “one blood
stem cell acquires the ability to reproduce without regulation, producing large numbers of
immature blood cells”. These cells look like tear-drop shaped red cells and do not
function properly. “The body continues to produce these abnormal, non-functional cells,
leaving little space for healthy cells. At the same time, these cells release chemicals that
cause the bone marrow to become fibrous or fill with scar tissue, further interfering with
the ability to produce healthy blood cells. In addition, these abnormal cells may be
produced in other areas of the body, most often the spleen or liver, which results in an
enlarged spleen or liver that can be felt by the physician”. In MF, too few red blood cells
are made due to the fibrosis, whereas too many white blood cells and platelets form.
C. Extramedullary Hematopoiesis and Splenomegaly

Extramedullary hematopoiesis is a result of the bone marrow’s inability to produce new blood cells once the fibrous tissue forms within the bone. It has also been shown that MF causes the bone marrow to leak, and cells are released into the blood that are normally not in the blood. These cells tend to gravitate towards the spleen where they take up residence and begin hematopoiesis. This gradually causes the spleen to become enlarged, which is a key characteristic of MF. “Splenomegaly, resulting from extramedullary hematopoiesis, accounts for some of the most debilitating symptoms of MF, whether primary or secondary to PV or ET. It contributes to the morbidity associated with MF by causing early satiety, dysregulated gastrointestinal function, portal hypertension, decreased physical activity, distressing abdominal pain, and worsening of assorted cytopenias secondary to splenic sequestration. About 10% of MF patients present with severely symptomatic splenomegaly at diagnosis; another 50% will develop it within 4 years.”

![Normal spleen](image1.png) ![Splenomegaly](image2.png)
III. METHODS OF DIAGNOSIS

Many of these characteristics and symptoms are used to diagnose MF. However, due to the rarity of MPN’s and the similarities between them, there have been difficulties over the years in setting specific diagnostic criteria. The World Health Organization (WHO) has well-established diagnosis criteria, which is detailed in Table I. A more basic approach which is used more often in practice is describe in Table II.

TABLE I. The 2008 World Health Organization (WHO) diagnostic criteria for primary myelofibrosis
Diagnosis requires meeting all three major criteria and two minor criteria.

<table>
<thead>
<tr>
<th>Major criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Presence of megakaryocyte proliferation and atypia,* usually accompanied by reticulin and/or collagen fibrosis, or in the absence of significant reticulin fibrosis, the megakaryocyte changes must be accompanied by an increased bone marrow cellularity characterized by granulocytic proliferation and often decreased erythropoiesis (ie, prefibrotic cellular-phase disease)</td>
</tr>
<tr>
<td>2. Not meeting WHO criteria for polycythemia vera,† BCR-ABL1+ chronic myelogenous leukemia,‡ myelodysplastic syndrome,§ or other myeloid neoplasms</td>
</tr>
<tr>
<td>3. Demonstration of JAK2V617F or other clonal marker (eg, MPLW515L/K), or in the absence of a clonal marker, no evidence that the bone marrow fibrosis or other changes are secondary to infection, autoimmune disorder, or other chronic inflammatory condition; hairy cell leukemia or other lymphoid neoplasm; metastatic malignancy; or toxic (chronic) myelopathies.¶</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Minor criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Leukoerythroblastosis**</td>
</tr>
<tr>
<td>2. Increase in serum lactate dehydrogenase level**</td>
</tr>
<tr>
<td>3. Anemia**</td>
</tr>
<tr>
<td>4. Splenomegaly**</td>
</tr>
</tbody>
</table>

*Small to large megakaryocytes with an aberrant nuclear/cytosplasmic ratio and hyperchromatic, bulbous, or irregularly folded nuclei and dense clustering.
†Requires the failure of iron replacement therapy to increase hemoglobin level to the polycythemia vera range in the presence of decreased serum ferritin. Exclusion of polycythemia vera is based on hemoglobin and hematocrit levels; red cell mass measurement is not required.
‡Requires the absence of BCR-ABL1.
§Requires absence of dyserythropoiesis and dysgranulopoiesis.
¶Patients with conditions associated with reactive myelofibrosis are not immune to primary myelofibrosis, and the diagnosis should be considered in such cases if other criteria are met.
**Degree of abnormality could be borderline or marked.

TABLE II. Diagnostic criteria for MF: diagnosis requires A1 + A2 and any two B criteria

| A1 | Bone marrow fibrosis ≥ 3 (on 0–4 scale) |
| A2 | Pathogenetic mutation (e.g. in JAK2 or MPL), or absence of both BCR-ABL1 and reactive causes of bone marrow fibrosis |
| B1 | Palpable splenomegaly |
| B2 | Unexplained anemia |
| B3 | Leuko-erythroblastosis |
| B4 | Tear-drop red cells |
| B5 | Constitutional symptoms* |
| B6 | Histological evidence of extramedullary haematopoiesis |

*Drenching night sweats, weight loss >10% over 6 months, unexplained fever (>37.5°C) or diffuse bone pains.
A. Gene Mutations – JAK2

JAK2V617F is noted in the diagnostic criteria above. The presence of a JAK2 mutation is found in approximately 45-68% of patients diagnosed with MF and should therefore be screened using a molecular genetic assay during the diagnostic process. The gene mutation causes abnormal signaling in the JAK pathway, which regulates blood cell production.

IV. Prognosis

Prognosis in MF is important, especially in determining the course of medical action to take with each individual patient. With options ranging from managing symptoms to chemotherapy and/or stem cell transplantation, an accurate assessment of a patient’s prognosis is critical. Prior to 2009, the Lille Score model was used to assess prognosis. In 2009, Cerantes et al published the International Prognostic Scoring System (IPSS), which uses five risk factors to estimate survival from the time of diagnosis (see Table III below). In 2010, Passamonti et al modified the scoring system to include a feature to make it dynamic, aptly called the Dynamic IPSS in which risk factors are assessed throughout their disease, not just at diagnosis. There is a further modification called the DIPSS Plus which shows additional risk factors for platelet count of < 100 x 10⁹/l, RBC transfusion dependence, and unfavorable karyotype. Studies have shown that those with unfavorable an karyotype are at high risk of leukemic transformation.

<table>
<thead>
<tr>
<th>Variable</th>
<th>IPSS</th>
<th>DIPSS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt;65 years</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Constitutional symptoms</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Hemoglobin &lt;100 g/l</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Leukocyte count &gt; 25 x 10⁹/l</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Circulating blasts ≥ 1%</td>
<td>✓</td>
<td>✓</td>
</tr>
</tbody>
</table>

DIPSS-Plus: add 1 point to the DIPSS RISK GROUP* (low = 0; intermediate 1 = 1, intermediate 2 = 2 and high risk = 3) in addition for: Platelet count <100 x 10⁹/l, RBC transfusion need, Unfavorable karyotype
<table>
<thead>
<tr>
<th>Risk Group</th>
<th>Predictors (n)</th>
<th>Median Survival (yrs)</th>
<th>Predictors (n)</th>
<th>Median Survival (yrs)</th>
<th>Predictors (n)</th>
<th>Median Survival (yrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>0</td>
<td>11.3</td>
<td>0</td>
<td>Not reached</td>
<td>0</td>
<td>15.4</td>
</tr>
<tr>
<td>Intermediate-1</td>
<td>1</td>
<td>7.9</td>
<td>1 or 2</td>
<td>14.2</td>
<td>1</td>
<td>6.5</td>
</tr>
<tr>
<td>Intermediate-2</td>
<td>2</td>
<td>4</td>
<td>3 or 4</td>
<td>4</td>
<td>2</td>
<td>2.9</td>
</tr>
<tr>
<td>High</td>
<td>≥3</td>
<td>2.3</td>
<td>5 or 6</td>
<td>1.5</td>
<td>≥4</td>
<td>1.3</td>
</tr>
</tbody>
</table>

*Note that this is the risk group NOT the sum of points.

V. Palliative Treatment Options

Pharmaceutical MF treatment aims to relieve symptoms and reduce the risk of complications and therefore is only considered palliative and often short-lived. As noted above, some of the main characteristics of MF are splenomegaly from extramedullary haemopoiesis and anemia, along with a variety of other constitutional symptoms including pain/fullness below the ribs on the left side, early satiety, fatigue, dyspnea, fever, night sweats, pruritus, weight loss and increased risk of infection. The only potential cure for MF is allogeneic stem cell transplantation (ASCT).

Below is a review of the palliative treatment options for the most common symptoms of MF:

A. Splenomegaly

1. Hydroxycarbamide/Hydroxyurea (HU)

HU, an oral chemotherapeutic myelosuppressive agent, is the most commonly used drug therapy to treat an enlarged spleen, although clinical support is limited to a few studies showing its efficacy. Regardless, it remains widely used and is often the first line of therapy when patients are diagnosed with MF, specifically because diagnosis normally presents with an enlarged spleen. The spleen becomes enlarged in MF patients because hematopoiesis takes place there when the bone marrow fails to produce enough blood cells. Splenomegaly is often targeted first to treat because it causes the most constitutional symptoms such as early satiety and weight loss. However, “the primary side-effect of hydroxyurea is suppression
of blood counts, particularly the white blood cells (neutropenia) and platelets (thrombocytopenia). Neutropenia and thrombocytopenia respectively place patients at risk for infection and bleeding”. Therefore, the patient’s degree of cytopenia is considered when administering HU.

2. Bulsulfan and Melphalan

Bulsulfan and melphalan are cancer-fighting alkylating agents and are often used in low doses in patients when HU is no longer indicated due to cytopenia. Alkylating agents “work by reacting with the proteins that bond together to form the very delicate double helix structure of a DNA molecule, adding an alkyl group to some or all of them. This prevents the proteins from linking up as they should, causing breakage of the DNA strands and, eventually, the death of the cancer cell. This phenomenon is essentially a mutation that takes away the cancer cell’s ability to multiply”. With the low dose administration, the disease slows and more normal blood cells can be produced in the bone marrow, therefore helping to reduce the size of the spleen.

3. Splenectomy

Removal of the spleen is rarely indicated but is done if drug therapies do not work, leaving the spleen extremely enlarged or causing severe anemia, low platelet count or portal hypertension. Splenectomies have not proven to rid the body of the disease state and can cause a variety of other complications.
B. Cytopenia

1. Interferon (IFN)-α and PEG-INF α

“The interferons are a group of natural proteins that are produced by human cells in response to viral infection and other stimuli. They were first described in 1957, and were named for their ability to interfere with viruses that are replicating”.\textsuperscript{13} Initially, INF-α was used due to its cytoreductive properties. It has been shown to “reverse cytopenias and bone marrow abnormalities in patients with earlier forms of MF, prior to the advent of extensive fibrosis”.\textsuperscript{5} However, due to the inconvenient dosing schedule and increased risk of toxicity, its use is limited. However, a study published in 2009 showed the efficacy of pegylated (PEG) INF-α in achieving hematologic responses and placing the majority of patients in remission or experiencing major positive responses.\textsuperscript{14} To date, this is the only study using PEG INF-α in treatment for MF, however interest is growing in this area.\textsuperscript{5}

C. Anemia

As described above, MF results in the proliferation of too few red blood cells and an overabundance of white blood cells and blood platelets, therefore causing severe anemia. Treatments for anemia aim to either replenish the red blood cells and/or reduce the symptoms of anemia, such as fatigue and weakness.

1. Red blood cell transfusions

MF patients often experience anemia, either through lack of hematopoiesis in the bone marrow due to fibrosis, ineffective production of RBC in extramedullary sites (i.e. the spleen), or from pharmaceutical drugs such as HU that reduce the
extramedullary hematopoiesis, among other factors. However, this can often result in iron overload, which requires iron-chelating agents. The iron-chelating agents help prevent organ damage from the iron-overload and have been proven to “significantly improve the overall survival of red blood cell transfusion-dependent PMF patients.”

2. Erythropoietin and Androgen Therapy

Erythropoietin is a hormone that stimulates the production of red blood cells in the bone marrow. In states of chronic inflammation, such as cancer, serum erythropoietin (EPO) levels in the blood can get very low and therefore cause anemia. Recombinant human erythropoietin (rHuEPO) provides an alternative to blood transfusions and is often used to improve red blood cells levels. Danazol, an androgenic steroid, is another drug used to treat anemia and/or relieve the symptoms of severe anemia. This treatment has approximately a 33% success rate, may cause masculinizing effects in women and can cause liver damage; therefore, this is not a first line therapy for patients.

VI. CURATIVE TREATMENT: STEM CELL TRANSPLANTATION

Stem cell transplantation (SCT) has proven to be the only curative therapy for MF patients. The main goal of SCT is to replace the damaged bone marrow and diseased cells with healthy cells in order to restore normal hematopoietic and immunologic function. While there are three types of STC, listed below, the only options for MF patients is allogeneic.

A. Autologous SCT (Auto-SCT)

In this transplant, the transplant recipient and donor is the same person. Healthy stem cells are taken from the patient before the cancer reaches that area. Those cells are then
harvested and implanted later when the procedure is indicated. This type of transplant carries a much lower risk of rejection; however, it does carry some risk involving the potential to reinfuse the diseased cells and therefore causing a relapse. Another disadvantage is that auto-STC lacks the graft versus cancer effect.

a. The graft versus cancer/tumor effect takes place in allo-STC patients. When the donor’s healthy cells become established in the recipient’s body, the donor’s cells may begin to recognize the recipient’s cells as foreign and therefore destroy them. This can help destroy leftover or new cancerous cells.

B. Syngeneic SCT

Syngeneic SCT is when the recipient receives stem cells from an identical twin. This carries the same risks an autologous SCT carries in that the immune system between the donor and recipient are so similar that the transplant will not help to destroy anything. Therefore, effort is made to destroy as many cancer cells as possibly before the transplant.

C. Allogeneic (Allo-SCT)

This type of transplant involves transferring stem cells from a donor to the recipient. The ideal donor is a human leukocyte antigen (HLA)-genotypic match from a sibling, a match identified through the National Marrow Donor Program (NMDP). The four types of stem cell transplantation considered allogeneic are:

a. HLA-matched relative (most often a sibling)
b. HLA-matched unrelated donor
c. HLA miss-matched family member
d. Unrelated umbilical cord blood
The main advantage is that the donor cells can create an anticancer environment and infuse disease-free cells into the host; therefore there is a greater chance for the graft versus cancer effect. The main disadvantage is that the recipient has a much higher risk of getting graft versus host disease (GVHD), along with prolonged immunosuppression and the risk that it carries, including increased susceptibility to infection and graft rejection.\textsuperscript{16} 

a. GVHD, in short, is a condition that can happen after allo-SCT when the donor’s cells see the recipient’s cells as foreign. This can be good, as described above in the graft versus cancer effect, however in GVHD the donor’s cells also attack healthy cells. This most often occurs in the skin, GI tract and liver. See below for more information on GVHD.
D. Sources of Stem Cells

1. Bone marrow

Bone marrow is the spongy tissue in the center of bones that creates the cells that circulate throughout our body.\textsuperscript{31} Cells are harvested from the pelvic bone, and then the cells are filtered, stored, and then frozen. Bone marrow stems cells were the first stem cells ever used in practice.

2. Peripheral blood

Stem cells are taken from the blood in this case. Normally, there are few stem cells found in the peripheral blood since they develop in the bone marrow.\textsuperscript{31} However, donors are given a “hormone-like substances called growth factors” prior to the harvest day.\textsuperscript{31} This helps more stem cells grow and move from the marrow to the blood. A special machine is used to collect the peripheral blood and then separate the stem cells out so that the rest of the blood goes back to the donor. “Since 1994, peripheral stem cells have been the most frequent source of cells used for transplant. They are associated with accelerated engraftment and result in reduced length of hospitalization.”\textsuperscript{16}

3. Umbilical cord blood

“Around 30% of unrelated hematopoietic stem cell transplants are done with cord blood.”\textsuperscript{31} The umbilical cord is taken after a baby is born and stored for later use. The cord can be from the recipient him/herself or a donor.”“Stem cells collected from umbilical cord blood have the advantage of being immunological immature and can therefore be used with broader HLA disparity, but the quantity of stem
cells from umbilical cord blood is often too low for adults and these transplants have been associated with delayed engraftment.”

E. Patient Selection

HSTC is not indicated for everyone with MF due to the high risk of morbidity and mortality in SCT patients, therefore the prognostic scoring systems are essential when deciding whether a patient should go through with the procedure. With that noted, MF is increasingly being treated with allo-SCT as shown in the trends generated by Center for International Blood and Marrow Transplant Research. With advances in SCT and the fact that it is the only curative option, patients and doctors seem to be turning to it more and more.

F. Course of Therapy

In allo-SCT, patients first receive high doses of chemotherapy called myeloablative therapy and/or radiation therapy in order to destroy the diseased bone marrow. The high dose chemotherapy normally takes place for about five to seven days. After a day of rest, healthy hematopoietic stem cells from a donor are infused into the MF patients. The donor is someone whose stem cells are compatible with those of the patient, who is often a sibling who is a match but can be someone unrelated as well. The new cells are supposed to grow and multiply in the bone marrow, creating a healthy number of red cells, white cells and platelets. There is also new research in reduced intensity SCT that is being tested in an effort to lower treatment-related mortality (TRM).

G. Side Effects of Conditioning

Medications for chemotherapy and the effects of radiation can be harsh on one’s body. See the “Medication Bibliography” for a list of medications, their uses, mechanisms of
action and nutritional side effects. Below are the major effects chemotherapy and radiation can have on the body during treatment.

a. **Gastrointestinal Tract** – Common problems include mouth sores, nausea, diarrhea, cramps, nausea and vomiting.

b. **Hair** – Hair loss can occur during treatment and is usually temporary.

c. **Heart** – Depending on a variety of factors, including current health status, the heart can be affected by treatment. Tests prior to transplant are often conducted to check the strength of the heart.

d. **Lungs** – A tissue reaction within the lungs to treatment can cause interstitial pneumonitis pneumonia. This can be very severe and prevent the efficient exchange of oxygen in the lungs.

e. **Skin** – Severe rashes may develop.

f. **Blood Vessels** – Leaky blood vessels may develop and cause fluid accumulation in the lungs, leading to congestion, poor oxygen exchange and shortness of breath.

g. **Liver** – Sinusoidal obstructive syndrome (SOS) may occur due to blocked blood vessels in the liver, which may result in liver damage and accumulation of fluid in the abdomen.

h. **Immunosuppression** – The conditioning treatment causes a suppressed immune system due to cells (both healthy and not) killed during therapy. This, in turn, causes a low WBC count and therefore the result is immunosuppression.
H. Nutritional Side Effects of Allo-SCT

1. Oral Muscositis and Esophagitis

These are a form of GVHD and therefore occur due to the new healthy cells attacking healthy epithelial cells within the patient’s body. Cracked lips and ulcer-like mouth sores form and lead to inflammation of the epithelial tissue within the mouth and esophagus. This can be extremely painful and result in an inability to consume any energy by mouth. Intravenous (IV) opioids are often needed for pain management. Symptoms may occur within days of receiving chemo or radiation. Healing often takes place once marrow engraftment occurs and absolute neutrophils reach >500cm$^3$.\textsuperscript{15}

2. Xerostomia and Dysgeusia

Altered salivation in which saliva becomes thick can be due to conditioning treatments but is also associated with drugs such as antiemetics, antidepressants, and opiate-containing pain medications.\textsuperscript{15} Impaired taste (or sometimes diminished taste) are also effects of conditioning treatments and medications such as morphine and antibiotics. Xerostomia often leads to dysgeusia. These changes are normally temporary.\textsuperscript{15} These also lead to low energy intake throughout treatment.

3. Nausea and Vomiting (N/V)

These are caused by a large variety of factors, including conditioning treatments, medications, GI infections, intestinal GVHD, and fluid/electrolyte imbalances.\textsuperscript{15} Antiemetics are the first course of treatment for N/V and have variable success depending on the cause and the patient’s tolerance. Both symptoms lead to
nutritional deficiencies, as appetite decreases when this occurs and nutrients taken in are not absorbed properly.

4. Anorexia

Anorexia throughout treatment is caused by all of the factors listed above (mouth pain, altered taste, N/V, electrolyte imbalances, medications) along with early satiety from delayed gastric emptying and loss of appetite, GI infections, intestinal GVHD, and general discomfort from treatment.¹⁵

5. Diarrhea and Steatorrhea

Diarrhea is associated with all of the symptoms listed above, along with lactose intolerance, laxatives, and supplemented magnesium salts.¹⁵ Steatorrhea and fat malabsorption may develop from liver and GI GVHD.¹⁵

6. Sinusoidal obstructive syndrome (SOS)

SOS is a common side effect of high dose chemotherapy and radiation; however, its pathogenesis is not completely understood as its incidence has been documented to occur in less than 5% of patients to more than 70% depending on the population studied and the difference in conditioning therapy used prior to transplant.²¹ It is understood to cause hepatic vein obstruction due to subendothelial edema and fibrin deposits, which prevents blood from flowing out of the liver and back to the heart, which then leads to liver toxicity and damage.²⁰

Basically, liver dysfunction occurs due to blocked blood vessels through the liver, which may result in liver damage and accumulation of fluid in the abdomen.¹¹ Clinical symptoms include weight gain with ascites, increased serum bilirubin and jaundice, and hepatomegaly, all of which can lead to hepatorenal failure and
encephalopathy. Those with a history of liver disease (hepatitis C, hepatoc fibrosis, cirrhosis) and second transplants are at a higher risk of developing this.\textsuperscript{15,20}

7. Renal Complications

There are a variety of factors that may lead to renal complications from SCT treatment. The most common include side effects of chemotherapy, radiation, and medications during these treatments and post-SCT, including cyclosporine, tacrolimus, amphotericin, aminoglycosides, bancomycin, and trimethoprim-sulfamethoxazole are associated with renal damage. Other causes include radiocontrast agents, extreme dehydration, and hepatorenal syndrome.

8. Infection

Infections throughout cancer treatment are common due to treatment causing immunosuppression. Patients are at an increased risk for viral, bacterial, and fungal infections. One study that measured the long-term survival of patients post-allo-SCT found that serious infections are the third leading cause of death. More specifically, it found that it accounts for 4-20% of deaths beyond two years post-transplant. To evaluate immunocompetence, the following calculation can be done to determine the total lymphocyte count (TLC)\textsuperscript{19}: \textit{TLC} = (WBC \times \%\text{lymphocytes})/100

G. Graft Versus Host Disease (GVDH)

“Graft-versus-host disease (GVHD) is a complication that can occur after a stem cell or bone marrow transplant in which the newly transplanted donor cells attack the transplant recipient's body.”\textsuperscript{33} GVHD only occurs in allo-SCT. GVHD can develop in the oral/mouth area, skin, GI tract or liver (these are described in detail in the Medical
Nutrition Therapy section below). Depending on when the GVHD develops, it can be
categorized as acute or chronic:

1. **Acute GVHD**

   This occurs within 100 days post-transplant in 40-50% of patients and usually
   affects the skin, liver and GI tract. See the “Medications Bibliography” section at
   the end of this paper. Medications to help with symptoms include topical steroids,
   antiemetics, pain medications, fluid and electrolyte replacement, and
   antiidiarrheals. See Table XIV for staging parameters of acute GVHD. This can
   be helpful for the medical team to determine a patient’s disease state and course
   of action prior to performing a biopsy.

2. **Chronic GVDH**

   This occurs beyond 100 days post-transplant in 40-70% of patients and can affect
   the skin, liver, GI tract, eyes, musculoskeletal, nervous, hematopoietic, and
   pulmonary symptoms. Medications for this are listed in the “Medications
   Bibliography” section, as well.

**VII. NEW TREATMENT OPTIONS**

As described above, the majority of patients with MF are positive for the JAK2 gene mutation,
which has led to a large amount of research into the use of JAK2 inhibitors for the treatment of
MF. Ruxolitinib (Jakafi) is “the first JAK inhibitor and currently the only drug approved by the
FDA to treat symptoms and signs of MF; including an enlarged spleen, night sweats, itching and
bone or muscle pain. It is indicated for treatment of patients with intermediate- or high-risk
MF”. Side effects include thrombocytopenia and anemia. While there is much excitement
surrounding Jakafi, there are few studies that prove its effectiveness. As such, it is still considered an experimental drug in most practices.
MEDICAL NUTRITION THERAPY

I. GOALS OF MEDICAL NUTRITION THERAPY

The main goal of medical nutrition therapy for patients receiving stem cell transplantation is weight maintenance. Weight is a significant indicator of nutritional status in this patient population, as it can show how well a patient is tolerating treatment or whether his/her disease state is worsening. Since most treatments cause a variety of factors that lead to the combination of increased needs and inadequate intake (both orally and through nutrition support as described below), weight is extremely important to monitor and treat. In addition to weight, it is important to educate patients on the importance of nutrition therapy throughout their treatment. Patients may be put on a variety of diets depending on their symptoms, and it is important for the patient and his/her family to understand the significance behind the treatment in order to remain compliant. This specifically applies when patients are in a neutropenic state and need to follow a low bacteria diet. Adequate protein intake and maintenance of nutrition-related labs including electrolyte balances are important to monitor and treat, as shifts may be seen throughout treatment due to certain medications.

II. NUTRITION REQUIREMENTS FOR ADULTS

A. Energy

Due to the individuality of treatment courses, patient response, pre-transplant nutritional status, post-transplant symptoms and other health-related conditions, there are not set guidelines on energy requirements for cancer patients or hematopoietic transplant patients. Despite the lack of evidenced-based guidelines, it is well recorded that energy requirements are increased in patients who receive SCT. The requirements vary due to a variety of reasons including the length/strength of the conditioning treatments, potential
infections/fever, and development of GVHD causing metabolic shifts, along with with common symptoms that cause a decrease or inadequate energy intake (including nausea, vomiting, diarrhea, early satiety, etc…). Studies over the past thirty years have shown that requirements can range anywhere from 30-50 kcals/kg/day throughout the course of treatment and are generally calculated using one of the following categories:¹⁹

- Obese patients: 21-25 kcals/kg
- Non-ambulatory or sedentary adults: 25-20 kcals/kg
- Slightly hypermetabolic patients or those who need to gain weight, or are anabolic: 30-35 kcals/kg
- Hypermetabolic or severely stressed patients or those with malabsorption: 35 kcals/kg or greater as needed
- Basal Energy Expenditure* (BEE) x activity/injury factor (1.5-2)
  *BEE is calculated using the Harris-Benedict formula:
  - BEE Men = 66.5 + (13.75 x kg) + (5.003 x cm) - (6.775 x age)
  - BEE Women = 655.1 + (9.563 x kg) + (1.850 x cm) - (4.676 x age)
  o The factor of 1.5-2 takes into account both the activity factor and the injury factor

The most commonly used calorie goal is 30-35 kcals/kg/day with higher energy requirements reserved for only the most severe cases of malnourishment and hypermetabolic activity during GVHD.²³ Once metabolic complications are resolved, energy needs decrease back to a patient’s regular intake, likely 25-30 kcals/kg/day. Due to the daily changes in metabolism and energy needs in cancer patients, a metabolic cart is not normally indicated. The main goal of nutrition is to ensure adequate intake during a time when intake can be compromised for a variety of reasons.

B. Protein

Protein is needed to provide tissue repair after conditioning therapy (especially radiation) and to avoid loss of lean body mass.¹⁵ Requirements for adults after SCT are generally 1.2-1.5 g/kg/day depending on age, constitutional symptoms, energy intake, weight, height and muscle mass. However, many studies have noted that protein requirements can
reach 2 g/kg/day or more during specific treatment periods (chemo/radiation, high dose corticosteroid treatment) or due to progressive GVHD. Protein can help to repair tissue damage from conditioning treatments, promote tissue and cell growth (white and red blood cells and platelets), and help to meet increased demands caused by infection, wound healing and hypermetabolism. Protein may need to be further modified if hepatic, renal, or neurologic function is altered.

1. Glutamine

Glutamine is a non-essential amino acid that has been studied in recent years for use on SCT patients due to its proven ability to help gut function, repair tissue damage, improve immune response, and assist in other essential body processes, especially in times of stress. Glutamine “administration after BMT was indeed shown to exert positive effects on nitrogen balance, incidence of infectious complications, survival, duration of hospital stay, and need for TPN, although not univocally.” Nitrogen is necessary to keep organs functioning and repair wounds during times of injury. Glutamine provides one-third of this nitrogen. Additionally, glutamine has proven to help prevent and/or treat SOS by preserving hepatic function and protecting hepatocytes from oxidative stress during treatment. More studies need to be conducted on this therapeutic role of glutamine.

C. Fats

In healthy individuals, typical intake is 15-30% of daily energy. The recommendations for patients with cancer and/or undergoing STC should not exceed 60% of calories due to potential for hypercholesterolemia or hypertriglyceridemia. If high triglycerides are
apparent, lipid intake should not exceed 40% of total energy. “Lipids may be particularly useful in achieving the energy target if hyperglycemia develops as a consequence of steroid treatment or infection.” However, the minimum intake should not fall below 4-8% in order to prevent essential fatty acid deficiency. The essential fatty acids, linoleic (omega-6) and linolenic (omega-3), are necessary for the endogenous synthesis of other fatty acids, such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), which are needed for healthy skin, immune system, and neurologic function.

D. Carbohydrates

There are no specific carbohydrate recommendations for cancer patients eating a diet orally or when receiving enteral nutrition. Carbohydrates in general should be around 50-60% of total intake. If parenteral nutrition is involved, the infusion rate recommendation should not exceed 5 mg/kg/min. Hyperglycemia, often brought on by steroid use in cancer patients, may deem it necessary to restrict carbohydrates or plan for consistent intake. In cases where hyperglycemia persists, it may be necessary to increase protein and lipid calories in order to make up for the prescribed lower carbohydrate intake.

E. Fluids

Fluid needs are dependent upon a variety of factors in cancer patients and may change throughout the course of treatment. For adults, the standard recommendation is that fluid intake should exceed 1500 mL/day (generally 25-30 mL/kg). When a patient is experiencing fever, excessive GI losses, hypermetabolism, dehydration and during SCT conditioning, fluid needs are increased. Fluid restrictions may be necessary if a patient is experiencing edema or ascites. Intake/output should be documented daily for patients
with BMT, especially when symptoms involving fluid loss such as fever, emesis or diarrhea are present. Fluid needs will increase significantly in each of these conditions.

**F. Vitamins and Minerals**

According to A.S.P.E.N Nutrition Support Practice Manual 2nd Edition, there are no specific vitamin and mineral requirements for SCT patients.\(^{15}\) It notes, however, that patients should receive micronutrient supplementation in order to receive 100% of the dietary reference intakes for age throughout the first year post-transplant and/or as long as immunosuppressive medications are given. Iron is specifically not recommended due to the frequency of blood transfusions throughout the recovery period, as iron overload can occur. A.S.P.E.N provides the following guidelines for patients undergoing cytoreductive therapy and/or SCT:

1. **Vitamin C**

Vitamin C when used in parenteral nutrition helps to “promote tissue recovery via collagen biosynthesis after cytoreductive therapy.”\(^{15}\) For most adults, the recommendation is an additional 500 mg of vitamin C per day. Vitamin C is contraindicated in patients with serum ferritin levels > 100 mcg/L as it can cause oxidative damage with the free iron. In addition, it should not be used in patients receiving hemodialysis.\(^{15}\)

2. **Vitamin K**

Vitamin K deficiency may be seen in patients with malabsorptive disorders and should be supplemented in adults with an additional 10 mg/week.\(^{15}\)
3. Zinc

Zinc is a necessary supplement in patients who experience excessive diarrhea. A.S.P.E.N. notes that when stool volume exceeds 500mL in patients 20-40 kg and 1000mL in patients >40 kg, zinc should be supplemented at 1 mg/100 mL stool.

4. Electrolytes

Electrolytes are especially important to monitor due to medication administration and changes in GI losses. The following is a chart adapted from the A.S.P.E.N. guidelines on the causes of hyper- and hypo- electrolyte levels:15

<table>
<thead>
<tr>
<th>Electrolyte</th>
<th>Cause of Imbalance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Magnesium</td>
<td><strong>Hypomagnesemia</strong>: diarrhea, steatorrhea, malabsorption syndromes, medications (amphotericin, cyclosporine; foscarnet; tacrolimus), refeeding syndrome, severe malnutrition</td>
</tr>
<tr>
<td></td>
<td><strong>Hypermagnesemia</strong>: discontinuation of magnesium-wasting medication, excessive supplementation, renal insufficiency</td>
</tr>
<tr>
<td>Potassium</td>
<td><strong>Hypokalemia</strong>: GI losses, medications (amphotericin; corticosteroids; furosemide; foscarnet), refeeding syndrome, anabolism</td>
</tr>
<tr>
<td></td>
<td><strong>Hyperkalemia</strong>: excessive supplementation, potassium-sparing diuretics, renal insufficiency, tacrolimus</td>
</tr>
<tr>
<td>Sodium</td>
<td><strong>Hyponatremia</strong>: excessive GI losses, fluid retention, excessive IV or oral fluid/free water intake, hyperglycemia, medications (cyclophosphamide; cyclosporine; furosemide)</td>
</tr>
<tr>
<td></td>
<td><strong>Hypernatremia</strong>: inadequate fluid replacement during GI losses, aggressive diuretic therapy, fever</td>
</tr>
<tr>
<td>Phosphorus</td>
<td><strong>Hypophosphatemia</strong>: diabetes, medications (corticosteroids; cyclophosphamide; foscarnet; sirolimus), refeeding syndrome, renal acidosis, steatorrhea</td>
</tr>
<tr>
<td></td>
<td><strong>Hyperphosphatemia</strong>: excessive phosphorus intake, multiple myeloma, myelogenous leukemia, renal failure</td>
</tr>
<tr>
<td>Calcium</td>
<td><strong>Hypocalcemia</strong>: medications (corticosteroids; foscarnet; pamidronate), vitamin D deficiency</td>
</tr>
<tr>
<td></td>
<td><strong>Hypercalcemia</strong>: lymphoma, metastatic breast cancer, multiple myeloma, renal failure</td>
</tr>
</tbody>
</table>
III. NUTRITION MANAGEMENT AND SUPPORT

A. Oral Feedings

Oral feedings are the preferred form of nutrition management and support. Patients should continue consumption by mouth as long as their GI tract is functioning and they are well nourished. In general, energy dense foods (i.e. high calorie, high protein) are recommended, as early satiety can often be a conditional symptom of the conditioning treatment, SCT, or the cancer itself. Other restrictions and changes may be necessary depending on the conditional symptoms associated with the patients’ disease or treatment state. These restrictions are discussed in the next section under “Symptom Management.”

B. Enteral and Parenteral Nutrition

Nutrition support through EN and PN is often indicated in the SCT population due to the conditional symptoms reviewed in the next section that make it difficult for a patient to maintain a good nutritional status. A.S.P.E.N. states the following in regard to nutrition support therapy in SCT patients:

1. “Nutrition support therapy is appropriate in patients undergoing hematopoietic cell transplantation who are malnourished and who are anticipated to be unable to ingest and/or absorb adequate nutrients for a prolonged period of time. When parenteral nutrition is used, it should be discontinued as soon as toxicities have resolved after stem cell engraftment. Enteral nutrition should be used in patients with a functioning GI tract in whom oral intake is inadequate to meet nutrition requirements.”

2. “Nutrition support therapy is appropriate for patients undergoing hematopoietic cell transplantation who develop moderate to severe graft-vs-host disease
accompanied by poor oral intake and/or significant malabsorption.” The following table reviews the indications for PN and EN in patients post-SCT.

<table>
<thead>
<tr>
<th>Table IV. Indications for nutrition support in patients post-SCT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Enteral Nutrition Support</strong></td>
</tr>
<tr>
<td>• Functioning GI tract + inadequate oral intake x1 week or more</td>
</tr>
<tr>
<td>• Functioning GI tract + presence of oral/esophageal GVHD + inadequate oral intake</td>
</tr>
<tr>
<td>• Stable chronic GI GVHD + inadequate oral intake</td>
</tr>
<tr>
<td>• PN is contraindicated due to fluid retention and/or organ failure + functioning GI tract</td>
</tr>
<tr>
<td>• Central IV access not possible + enteral access possible + functioning GI tract</td>
</tr>
<tr>
<td>• Failure to thrive + functioning GI tract</td>
</tr>
</tbody>
</table>

*Table adapted from Graft-vs-Host Disease: Nutrition Therapy in a Challenging Condition* and *A.S.P.E.N Nutrition Support Practice Manual 2nd Edition*

Regarding access in EN, nasogastric (NG) feedings may be initially tried, however if patients are experiencing oral/esophageal GVHD or mucositis/esophagitis, NG tubes should not be used. Postpyloric access may be indicated in patients with delayed gastric emptying or frequent emesis. Percutaneous endoscopic gastrostomies (PEGs) are only indicated when a patient is no longer in a neutropenic state since it requires surgery. According to A.S.P.E.N. guidelines, patients must have platelets >50,000 units/L and absolute neutrophil counts >1000/mm³ as patients with numbers below this are in a neutropenic state. Patients in a neutropenic state are more susceptible to infections from surgery. Sometimes PEGs are placed two to three weeks prior to the conditioning treatment and transplant as a way to allow the body to heal from the surgery and provide a means to prevent malnourishment.
According to the A.S.P.E.N. guidelines, “PN is associated with improved long-term disease-free survival compared to hydration support in allogeneic patients receiving myeloablative conditioning regimens.”\textsuperscript{15} Indications for PN are listed in Table IV and supported in Table XV in which nutritional management of GI GVHD is described. Most patients have dual-lumen central venous access for several months post transplant in order to administer intravenous medications and blood products.\textsuperscript{15} As such, PN is often the first line of defense in a malnourished post-SCT patient due to the easy access of the central line. Some complications with PN include hyperglycemia, hypertriglyceridemia, essential fatty acid deficiency, cholestasis and gallbladder sludge, and infections.\textsuperscript{15} Glucose monitoring is especially important in patients receiving PN, and high doses of insulin may be necessary to control levels, especially when patients are on corticosteroids. Proper glucose control will help “decrease the number of bloodstream infections, renal failure requiring dialysis/filtration, red blood cell transfusions, and mortality.”\textsuperscript{20}

\textbf{IV. TREATMENT SIDE EFFECTS AND NUTRITIONAL INTERVENTIONS}

\textbf{A. Mucositis and Esophagitis}

Mucositis and esophagitis are an expected complication of myeloablative conditioning.\textsuperscript{15} Mucositis is the inflammation of the oral mucosa from chemotherapy or radiation and presents itself in the form of ulcerations or erythema (redness/rash of the skin). Mucositis normally arises in 7-10 days after chemotherapy treatment begins and can cause a significant decrease in intake due to the pain it causes.
Esophagitis is inflammation, redness and possible ulcerations within the esophagus. The degree in which patients experience symptoms is based on how intense the conditioning treatment is. The development of mucositis or esophagitis takes place over four phases: “an inflammatory phase caused by the release of cytokines during conditioning therapy; an epithelial phase, when cells cease dividing and die; an ulcerative phase when microbes and endotoxins translocate into the bloodstream; and a healing phase, which can be prolonged with deep lesions and is more complex in the gut than in the mouth.”

Patients say that mouth pain associated with mucositis is the “most debilitating” side effect of SCT and it is normally one of the first symptoms to occur. The pain often leads to the patient completely forgoing food and fluid intake, often leading to acute weight loss and dehydration. A thorough assessment of the mouth is necessary, as “alterations in the oral mucosa may include color changes of the tongue, lips, and gingiva; changes in moisture; and changes in integrity, including cracks, fissures, ulcers, blisters and lesions.” Opioids are normally used to treat the pain. Methotrexate, which is used to prevent GVHD, can exacerbate the ulcerations and pain. Mucositis is normally the first symptom post-SCT, often appearing within a week of the transplant as
neutrophil count drops significantly low. “Healing begins with marrow engraftment (absolute neutrophil count >500/cm$^3$)” which takes about 7-14 days after first appearing.$^{15}$

### Table V. MNT for Mucositis and Esophagitis$^{23}$

<table>
<thead>
<tr>
<th>Mucositis and Esophagitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Try soft or pureed foods</td>
</tr>
<tr>
<td>• Try a blenderized liquid diet – high calorie/high protein smoothies</td>
</tr>
<tr>
<td>• Offer smooth, bland, moist foods (custards, cream soups, yogurt, mashed potatoes)</td>
</tr>
<tr>
<td>• Offer soft, nonirritating, cold foods (popsicles, ice cream, frozen yogurt, slushes)</td>
</tr>
<tr>
<td>• Encourage frequent mouth rinsing to remove food and bacteria and to promote healing</td>
</tr>
<tr>
<td>• Avoid salty, spicy, and acid foods</td>
</tr>
<tr>
<td>• Avoid rough-textured foods</td>
</tr>
<tr>
<td>• Avoid alcohol and carbonated beverages</td>
</tr>
<tr>
<td>• Avoid extreme temperatures in foods</td>
</tr>
</tbody>
</table>

### B. Xerostomia and Dysgeusia

Xerostomia – in which saliva production decreases and saliva becomes viscous – occurs due to myeloablative regimens, antiemetics, antidepressants, and opiate-containing pain medications.$^{15}$ Dysgeusia, which is impaired taste, and hypogeusia, which is diminished taste, occur most often due to the conditioning treatments and are prolonged due to morphine and antibiotic medications. Altered salivation often precipitates dysgeusia because saliva is needed for taste perception. Medical nutrition therapy for these conditions involves the goal of comfort for patients who are in pain and creating a desire to eat despite difficulty chewing and tasting foods.

### Table VI. MNT for Xerostomia and Dysgeusia

<table>
<thead>
<tr>
<th>Xerostomia</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Moist foods (stews, casseroles, canned fruits) and liquids</td>
</tr>
<tr>
<td>• Add extra sauces, gravies, margarine, butter and broth to foods</td>
</tr>
<tr>
<td>• Encourage liquid with meals</td>
</tr>
<tr>
<td>• Add vinegar or pickles to foods (this induces salivation)</td>
</tr>
<tr>
<td>• Offer lemon-flavored, sugarless candy to help stimulate saliva</td>
</tr>
<tr>
<td>• Encourage good oral hygiene</td>
</tr>
<tr>
<td>• Try commercial saliva substitutes</td>
</tr>
<tr>
<td>• Offer clear liquids (tea, popsicles, slushes, warm broth)</td>
</tr>
</tbody>
</table>
Dysgeusia

- Try different flavors and spices to find acceptable ones
- Meats may be problematic and require alternate flavoring or preparation
- Add lemon or lime slices to water
- Dilute juices or other fluids if too sweet
- Use plastic utensils instead of metal if a metallic taste is present

C. Nausea and Vomiting

Nausea and vomiting are very common side effects of the conditioning treatment, supportive care medications (antibiotics, cyclosporine, interleukin-2, trimethoprim-sulfamethoxazole, and mycophenolate mofetil), GI infections, intestinal GVHD, and fluid and electrolyte imbalances.\textsuperscript{15} Antiemetics are often administered to treat and prevent nausea and vomiting. These are normally given via IV when symptoms are occurring and then given orally as symptoms improve.

<table>
<thead>
<tr>
<th>Table VII. MNT for Nausea and Vomiting \textsuperscript{15, 18, 23}</th>
</tr>
</thead>
<tbody>
<tr>
<td>• A clear liquid diet may be indicated if symptoms are severe</td>
</tr>
<tr>
<td>• Try high-carbohydrate foods and fluids (crackers, toast, gelatin) as well as nonacidic juices – electrolyte-replenishing drinks are often indicated</td>
</tr>
<tr>
<td>• Antiemetics should be taken 30-45 minutes before a meal</td>
</tr>
<tr>
<td>• Try small, frequent meals</td>
</tr>
<tr>
<td>• Offer cold, clear liquids and solids</td>
</tr>
<tr>
<td>• Avoid overly sweet or high-fat foods</td>
</tr>
<tr>
<td>• Create a comfortable setting when eating</td>
</tr>
<tr>
<td>• Avoid strong or unpleasant odors</td>
</tr>
<tr>
<td>• Encourage adequate fluid intake</td>
</tr>
<tr>
<td>• Avoid offering the patient’s favorite food during periods of nausea, as it may cause a permanent dislike of the food</td>
</tr>
<tr>
<td>• Document foods/smells/events that may trigger nausea and try to avoid them when possible</td>
</tr>
</tbody>
</table>

D. Diarrhea and Steatorrhea

Diarrhea is often due to myeloablative treatments, antibiotics, intestinal infections, GI GVHD, lactose intolerance, and GI mobility agents. “Liver and intestinal GVHD may produce steatorrhea and fat malabsorption.”\textsuperscript{15} Diarrhea will normally occur within the first two weeks post-SCT as a result of the toxicity to the GI mucosa during the conditioning treatment, but should resolve itself if no other problems appear. After the
two week mark, if diarrhea occurs it is normally a sign of GI GVHD or an infection. It is important to monitor daily stool output, as diarrhea >500 mL/day is an indication for parenteral nutrition support.

<table>
<thead>
<tr>
<th>Table VIII. MNT for Diarrhea and Steatorrhea&lt;sup&gt;15, 20, 23&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Try a low-fat, low fiber diet</td>
</tr>
<tr>
<td>• Avoid caffeine</td>
</tr>
<tr>
<td>• Offer cold or room-temperature foods and beverages as they may be better tolerated</td>
</tr>
<tr>
<td>• Offer low lactose intake</td>
</tr>
<tr>
<td>• Encourage adequate fluids and electrolyte intake to prevent dehydration</td>
</tr>
<tr>
<td>• Avoid excessive fruit juice ingestion</td>
</tr>
<tr>
<td>• If large volume diarrhea occurs, add zinc to the PN solution</td>
</tr>
<tr>
<td>• Monitor copper levels and add to PN if needed</td>
</tr>
</tbody>
</table>

E. Early Satiety and Appetite Loss

Early satiety is a symptom of MF, as the enlarged splenomegaly causes pain/fullness below the ribs on the left side, which leads to a feeling of fullness and therefore loss of appetite. It can also be caused by delayed gastric emptying in patients post-SCT. In addition, the conditioning treatments (chemotherapy and radiation) often lead to appetite loss due to nausea. In general, patients simply do not feel inclined to eat, as they either do not feel hungry or food no longer appeals to them.

<table>
<thead>
<tr>
<th>Table IX. MNT for Early Satiety and Appetite Loss&lt;sup&gt;15, 20, 23&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Small, frequent meals</td>
</tr>
<tr>
<td>• Nutrient dense foods</td>
</tr>
<tr>
<td>• Create a comfortable, appealing environment when eating</td>
</tr>
<tr>
<td>• Avoid foods that cause aversions</td>
</tr>
<tr>
<td>• Avoid favorite foods as to avoid creating aversions</td>
</tr>
<tr>
<td>• Try smoothies, shakes, protein powders, added fats</td>
</tr>
<tr>
<td>• Prokinetics may be indicated for delayed gastric emptying</td>
</tr>
</tbody>
</table>

F. Anorexia

Anorexia, which is the lack of or loss of appetite for food, occurs from a variety of symptoms. Overall loss of appetite may occur during the conditioning treatment and immediately post-transplant and it may be related to each of the symptoms listed above. If a patient is experiencing severe mouth pain or no longer enjoys the taste of foods, oral
nutrition is likely to decrease. Early satiety will also result in decreased energy intake. Nausea, vomiting, and diarrhea will create a malnourished state that can mimic the symptoms of anorexia. According to A.S.P.E.N., “prolonged use of PN post-SCT has been associated with anorexia and delayed resumption of oral intake.”

<table>
<thead>
<tr>
<th>Table X. MNT for Anorexia</th>
</tr>
</thead>
</table>
| • Offer small, frequent meals of nutrient-dense foods  
• Maximize intake when appetite is most normal  
• Limit fluids with meals to avoid feeling full  
• Use carbohydrate supplements and protein powders  
• Create a pleasant mealtime environment with enhancing food aromas, colorful place settings, and foods with varied colors and textures  
• Assess nutritional status through calorie counts |

G. Liver Complications (SOS)

Sinusoidal obstructive syndrome, is a syndrome of “jaundice, weight gain, ascites and painful hepatomegaly developing within approximately 10-20 days after SCT.”

Medical nutrition therapy for SOS mainly involves restricting fluid and sodium intake. Therefore, patients receiving nutrition support should receive concentrated solutions without added sodium; for those who are eating orally, sodium should be restricted. Dietitians and pharmacists should work together to evaluate whether a patient can receive his/her medications via a dextrose-based solution rather than through IV solutions with sodium. When a patient has severe SOS and liver failure, copper and manganese levels need to be monitored since excessive blood levels can be toxic if biliary excretion is abnormal. In addition, iron should be monitored as patients with SCT receive many iron-containing blood products throughout their treatment. Iron overload can lead to hemosiderosis of the liver.

<table>
<thead>
<tr>
<th>Table XI. MNT for SOS</th>
</tr>
</thead>
</table>
| • Limit/eliminate sodium and fluid intake  
• Monitor copper and manganese – eliminate if elevated  
• Avoid iron supplements and iron-containing multivitamins |
H. Renal Complications

Renal complications arise from a variety of factors. The conditioning treatment may cause kidney damage, medications (cyclosporine, tacrolimus, amphotericin, aminoglycosides, vancomycin, and trimethoprim-sulfamethoxazole) are associated with renal damage and insufficiency, and damage may be part of SOS as heptorenal failure.

<table>
<thead>
<tr>
<th>Table XII. MNT for Renal Complications\textsuperscript{15, 20, 23}</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Maintain sufficient intravascular volume</td>
</tr>
<tr>
<td>• Correct electrolyte imbalances</td>
</tr>
<tr>
<td>• If the patient receives hemodialysis:</td>
</tr>
<tr>
<td>o Monitor Vitamin A status, as it is known to accumulate during renal failure</td>
</tr>
<tr>
<td>o Provide a complete complex of water-soluble vitamins and folic acid</td>
</tr>
<tr>
<td>o Ensure Vitamin K needs are met</td>
</tr>
<tr>
<td>o Provide additional protein to compensate for losses during filtration</td>
</tr>
<tr>
<td>o Control glucose levels</td>
</tr>
<tr>
<td>o Replete calcium and magnesium if levels are low</td>
</tr>
</tbody>
</table>

I. Infections

After taking a variety of immunosuppressive medications prior to the SCT, neutrophil counts are severely decreased, which means that the patient is in a state of immunosuppression. The state of immunosuppression is important in order to allow the new donor cells to establish themselves inside the body of the recipient without being rejected. During this period, the patient is at an increased risk for infection since there are not enough white blood cells to fight off foreign bacteria and harmful organisms. The medical nutrition therapy for this condition is the neutropenic (i.e. low bacteria) diet. The neutropenic diet is designed to reduce the risk of food-borne illness (food poisoning) in individuals whose immunity is low due to low white blood cell counts. There is not much evidenced-based research to support neutropenic diets; however, it remains a common practice to recommend following this diet as long as a patient is taking immunosuppressant medications including cyclosporine, tacrolimus,
methotrexate, and prednisone (in other words, as long as it is thought that the patient is still in an immune-compromised state).\textsuperscript{28} A.S.P.E.N. states that “Patients should receive dietary counseling regarding foods that may pose infectious risks and safe food handling during the period of neutropenia.”\textsuperscript{25} The following is a basic outline on a neutropenic diet provided by the Leukemia and Lymphoma society.\textsuperscript{29} A more detailed version can be found in the Appendix.

<table>
<thead>
<tr>
<th>Table XIII. MNT for Neutropenia\textsuperscript{27}</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Avoid all uncooked vegetables and most uncooked fruits. You may eat fruit that you can peel a thick skin off of, such as a banana or an orange. Cooked vegetables and canned fruits and juices are safe to eat.</td>
</tr>
<tr>
<td>• Avoid raw or rare meat and fish and uncooked or undercooked eggs. Cook meat until it's well-done. Thoroughly cook eggs (no runny yolks).</td>
</tr>
<tr>
<td>• Avoid salad bars and deli counters. Buy vacuum-packed lunch meats instead of freshly sliced meats.</td>
</tr>
<tr>
<td>• Consume only pasteurized milk, yogurt, cheese and other dairy products.</td>
</tr>
<tr>
<td>• Avoid soft mold-ripened and blue-veined cheeses such as Brie, Camembert, Roquefort, Stilton, Gorgonzola and Bleu.</td>
</tr>
<tr>
<td>• Avoid well water or boil it for one minute before drinking. At home, it's okay to drink tap water or bottled water.</td>
</tr>
</tbody>
</table>

V. GVHD AND NUTRITIONAL INTERVENTIONS

As noted above, the four types of GVHD include skin, oral, GI, and liver. The first line of defense against GVHD is medications – immunosuppressants such as cyclosporine, methotrexate and tacrolimus, steroids like prednisone, and pain medications including opioids. The impact that GVHD has on nutritional needs is usually significant but very difficult to manage. In general, “needs are increased due to hypermetabolism, tissue damage, fluid and electrolyte imbalances, and medication-induced effects.”\textsuperscript{15} Energy needs are normally at the higher end of the 30-50 kcal/kg range, however intake is normally significantly reduced due to the symptoms described above. Protein needs are especially increased to 1.5-2 g/kg when there is tissue damage, with the higher end usually reserved for those with severe GVHD.\textsuperscript{23} Cases of severe GVHD (especially
when in stage 3 or 4, see Table XIV below) are often indicators for EN or PN. GVHD can be diagnosed with a biopsy of the area affected.

<table>
<thead>
<tr>
<th>Table XIV. Staging of Acute GVHD</th>
<th>Liver</th>
<th>GI Tract</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Skin</strong></td>
<td><strong>Liver</strong></td>
<td><strong>GI Tract</strong></td>
</tr>
<tr>
<td>Stage 1: &lt;25% rash</td>
<td>Stage 1: bili 2-3 mg/dL</td>
<td>Stage 1: diarrhea &gt; 500 mL/day</td>
</tr>
<tr>
<td>Stage 2: 25-50% rash</td>
<td>Stage 2: bili 3.1-6 mg/dL</td>
<td>Stage 2: diarrhea &gt; 1000 mL/day</td>
</tr>
<tr>
<td>Stage 3: generalized erythroderma</td>
<td>Stage 3: bili 6.1-15 mg/dL</td>
<td>Stage 3: diarrhea &gt; 1500 mL/day</td>
</tr>
<tr>
<td>Stage 4: bullae (blisters)</td>
<td>Stage 4: bili &gt;15 mg/dL</td>
<td>Stage 4: ileus, bleeding</td>
</tr>
</tbody>
</table>

According to A.S.P.E.N. Guidelines “nutrition support therapy is appropriate for patients undergoing hematopoietic cell transplantation who develop moderate to severe graft-vs-host disease accompanied by poor oral intake and/or significant malabsorption.”25 While indicated in order to keep a patient nourished when oral feedings are not possible, PN has been associated with an increased incidence of GVHD due to decreased oral intake.25,31 Although not thoroughly understood, studies have shown that the incidence of GVHD (especially GI GVHD) decreases with a standard oral diet or with EN.32 The following reviews the types of GVHD and the nutritional management if applicable.

**A. Oral GVHD**

Mucositis is a major sign of oral GVHD and are extremely painful, often leading to significantly decreased energy intake.

**B. Skin GVHD**

This is characterized by a severe rash anywhere on the skin, specifically by a “pruritic, maculopapular, and erythematous rash.” If it advances, it can sometimes turn into ulcers, blisters and worse that may lead to the need for burn care. If chronic, it can lead to changes in skin color.15
C. Liver GVHD

“Liver GVHD presents as cholestasis, elevated alkaline phosphatase and serum bilirubin levels, jaundice, hepatomegaly, and, in severe cases, encephalopathy and hepatorenal failure.”¹⁵ This complication can be fatal.

D. GI GVHD

GI GVHD can affect the GI tract anywhere from the esophagus to the rectum. It can cause difficulty swallowing, nausea, vomiting, abdominal cramps, poor absorption of nutrients (including fat and protein malabsorption), problems with gut motility, weight loss, and diarrhea. GI GVHD can be life threatening in its effects on the GI tract, including GI bleeds, ulcerations, and mucosal sloughing.¹⁵ A.S.P.E.N. has created guidelines for the management of GI GVHD that can be followed in clinical practice.¹⁵ It is important to note that probiotic therapy – which is normally indicated in patients with diarrhea as it adds good mucosal bacteria to the GI tract – is not indicated in patients with GI GVHD.²⁰

<table>
<thead>
<tr>
<th>Phase</th>
<th>Clinical Symptoms</th>
<th>Diet</th>
<th>Nutrition Support</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Bowel rest</td>
<td>GI cramping Large-volume watery diarrhea or active GI bleeding Depressed serum albumin Severely reduced transit time Small-bowel obstruction or diminished bowel sounds Nausea and vomiting</td>
<td>Oral: NPO</td>
<td>PN with supplemental zinc and possibly copper</td>
</tr>
</tbody>
</table>
| 2. Introduction of oral feedings | Minimal GI cramping  
Diarrhea <500 mL/day  
Improved transit time (minimum 1.5 hours)  
Infrequent nausea and vomiting | Oral: isosmotic, low-residue, low-lactose beverages, initially 60 mL every 2-3 hours, for several days | PN  
Trophic* enteral feeds of semielemental formula if patient is unable to eat |
|---|---|---|---|
| 3. Introduction of solids | Minimal or no GI cramping  
Formed stool | Oral: allow introduction of solid food, once every 3-4 hours: minimal lactose, low fiber, low fat (20-40 g/day), low total acidity, no gastric irritants | Begin to cycle and decrease PN  
Advance feeds slowly (small boluses or continuous infusion) if patient is unable to eat |
| 4. Expansion of diet | Minimal or no GI cramping  
Formed stool | Oral: minimal lactose, low fiber, low total acidity, no gastric irritants; if stools indicate fat malabsorption: low fat | Nighttime supplemental PN if oral intake is less than needs or patient is unable to maintain weight owing to malabsorption  
Enteral feed schedule and formula are dependent on any residual GI symptoms |
| 5. Resumption of regular diet | No GI cramping  
Normal stool  
Normal transit time  
Normal albumin | Oral: progress to regular diet by introducing 1 restricted food per day: acid foods with meals, fiber-containing foods, lactose-containing foods. The order of addition will vary depending on individual tolerances and preferences  
Patients no longer exhibiting steatorrhea should have the fat restriction liberalized slowly | Discontinue PN  
Supplemental enteral feeds if patient unable to eat adequate nutrients |

*Trophic feeding is “a small volume of balanced enteral nutrition insufficient for the patient's nutritional needs but producing some positive gastrointestinal or systemic benefit”20. It is commonly used in the pediatric population.

**VI. NUTRITION CARE PROCESS**

The A.S.P.E.N. Clinical Guidelines for “Nutrition Support Guideline Recommendations in Hematopoetic Cell Transplantation” states the following: “All patients undergoing hematopoietic cell transplantation with myeloablative conditioning regimens are at nutrition risk and should undergo nutrition screening to identify those who require formal nutrition assessment with development of a nutrition care plan.”25 The Patient Generated Subjective Global Assessment (PG-SGA), as shown in the Appendix is a nutrition screen that can be used on oncology patients to assess their nutritional status. It has been “shown to be accurate at distinguishing well-
nourished patients from malnourished patients, and has a high sensitivity and specificity. It is a quick, valid, and reliable nutrition assessment tool that enables malnourished hospital patients with cancer to be identified and triaged for nutrition support.19 The PG-SGA is a good starting point for an assessment, however a more thorough and comprehensive assessment is needed initially and continually throughout treatment, as changes in nourishment can happen rapidly in the SCT population.

A. Nutrition Assessment

1. Food/Nutrition-Related History

This includes the subjective evaluation of current food and nutrient intake, including allergies and intolerances, current medications and supplements, alternative medicines, nutrition knowledge, attitudes towards food/nutrition, food-related beliefs and behaviors, factors that affect access to food and medications, and physical activity.19 Understanding these factors helps assess where the patient is in their knowledge of food and nutrition in relation to health in general and their disease state, and it also helps to understand their willingness to change certain behaviors. For cancer patients specifically, this should reveal any potential impediments to food intake and tolerance. Past dietary treatment of the current disease – or any other disease states – should be recalled, along with the success and failures of those treatments. Dietitians should look at past use of enteral nutrition (EN), parenteral nutrition (PN), supplementation, and fluid intake. For patients with myelofibrosis, this information will help the dietitian later determine how to approach specific interventions. Specifically for dietitians treating patients
with MF who are treated with SCT should evaluate the following prior to, during and after treatment:\(^5\)

- Chewing/swallowing difficulties
- Mucositis and esophagitis
- Dental health
- Taste alterations
- Xerostomia
- Heartburn or reflux
- Nausea and vomiting
- Early satiety
- Appetite changes – calorie counts/food diaries may be necessary
- Anorexia
- Changes in bowel habits

### 2. Anthropometric Measurements and Nutrition-Focused Physical Findings

Anthropometric measurements are crucial in assessing patients with MF, as weight loss >10% over 6 months is one of the main constitutional symptoms used in the diagnosis process. In addition, weight loss is a major effect of chemotherapy, radiation and SCT due to diarrhea, vomiting, nausea, early satiety, chewing/swallowing difficulties, and taste alterations. The following should be assessed initially, during treatment and after treatment in patients with MF:

- Height
- Weight
- BMI
- IBW
- %IBW
- Weight change over the past 1 month, 6 months, 1 year (voluntary/involuntary?)
- %UBW
- Skinfold measurements (subcutaneous fat)
- Mid-arm muscle circumference (lean body mass)
- Edema
In cancer patients, weight should be evaluated throughout treatment based on the patient’s usual body weight; it should not be evaluated using a reference standard such as ideal body weight. While changes in weight are often the most visually and numerically obvious changes and should certainly be a main focus, weight alone does not tell the entire story of the patient’s nutrition status. For instance, patients may experience significant fluid retention due to medications leading to ascites and/or edema. In this case, it is important to analyze the time frame of the weight change and look at physical changes in the patient’s body. Mid-arm muscle circumference along with skinfold measurements can help determine whether a patient is losing fat-free or fat mass, an important consideration in determining malnutrition.

3. Biochemical Data, Medical Tests and Procedures

Laboratory data for patients with MF and patients who undergo SCT should be assessed throughout a patient’s lifetime, as changes can signify changes in disease state and nutritional status and will therefore affect the treatment course. Below are a list of the common tests and procedures used to help diagnose and continually assess patients with MF, along with their normal lab values and what the patients values can tell us about their disease state:

<table>
<thead>
<tr>
<th>Lab</th>
<th>Normal Range</th>
<th>Likely Cause</th>
</tr>
</thead>
</table>
| Red Blood Cells (RBCs)   | M: 4.7-6.1 million/mm³  
F: 3.9-5.5 million/mm³ | Increased in polycythemia, dehydration, severe diarrhea  
Decreased in anemia, Fe deficiency, systemic disease (i.e. leukemia) |
| White Blood Cells (WBCs) | 3200-10,600/ulq                                   | Increased in leukemia, bacterial infection, hemorrhage, trauma or tissue injury, cancer  
Decreased in viral infections, chemotherapy, radiation, bone-marrow depression |
| Platelets                | 177-406 x 1000/ul  
thrombocytosis  
thrombocytopenia | Increased in malignancy (i.e. leukemia), polycythemia vera, post splenectomy, iron deficiency anemia  
Decreased in hemolytic/pernicious anemia, chemotherapy, infection, leukemia |
<table>
<thead>
<tr>
<th>Neutrophils</th>
<th>&gt;1,000/mm³</th>
<th>Low neutrophils due to immunosuppression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>Fasting: 70-99 mg/dL</td>
<td>Increased in diabetes, high blood sugar Decreased in hypoglycemia</td>
</tr>
<tr>
<td>Albumin</td>
<td>3.5-5.0 gm/dL</td>
<td>Increased in dehydration Decreased in edema, hepatic disease, malabsorption, diarrhea, malnutrition, low protein intake, over-hydration, some cancers</td>
</tr>
<tr>
<td>Prealbumin</td>
<td>18-38 mg/dL</td>
<td>Increased in acute catabolic state, hepatic disease, stress, infection, surgery, malnutrition, low protein intake</td>
</tr>
<tr>
<td>Magnesium</td>
<td>1.3-2.1 mEq/L</td>
<td>Increased in diarrhea, steatorrhea, malabsorption syndromes, medications (amphotericin, cyclosporine; foscarnet; tacrolimus), refeeding syndrome, severe malnutrition Decreased in discontinuation of magnesium-wasting medication, excessive supplementation, renal insufficiency, malabsorption, malnutrition</td>
</tr>
<tr>
<td>Potassium</td>
<td>3.5-5.5 mEq/L</td>
<td>Increased in GI losses, medications (amphotericin; corticosteroids; furosemide; foscarnet), refeeding syndrome, anabolism Decreased in excessive supplementation, potassium-sparing diuretics, renal insufficiency, tacrolimus</td>
</tr>
<tr>
<td>Sodium</td>
<td>136-144 mEq/L</td>
<td>Increased in excessive GI losses, dehydration, fluid retention, excessive IV or oral fluid/free water intake, hyperglycemia, medications (cyclophosphamide; cyclosporine; furosemide) Decreased in inadequate fluid replacement during GI losses, aggressive diuretic therapy, fever</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>2.3-4.3 mg/dL</td>
<td>Increased in diabetes, medications (corticosteroids; cyclophosphamide; foscarnet; sirolimus), refeeding syndrome, renal acidosis, steatorrhea Decreased in excessive phosphorus intake, multiple myeloma, myelogenous leukemia, renal failure</td>
</tr>
<tr>
<td>Calcium</td>
<td>8.4-10.2 mg/dL</td>
<td>Increased in medications (corticosteroids; foscarnet; pamidronate), vitamin D deficiency Decreased in lymphoma, metastatic breast cancer, multiple myeloma, renal failure</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.4-1.2 mg/dL</td>
<td>Increased in renal disease, muscle damage, starvation, diabetic acidosis</td>
</tr>
<tr>
<td>BUN</td>
<td>8-23 mg/dL</td>
<td>Increased in renal failure, dehydration, infection, diabetes, excessive protein/catabolism Decreased in malnutrition, hepatic failure, malabsorption, overhydration</td>
</tr>
<tr>
<td>ALT</td>
<td>M: 4-40 U/L  F: 4-31 U/L</td>
<td>Increased in liver dysfunction</td>
</tr>
<tr>
<td>AST</td>
<td>M: 10-37 U/L  F: 10-31 U/L</td>
<td>Increased in liver dysfunction</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>(fasting range, mg/dL) desirable &lt;150 borderline 150-199 high 200-499 very high &gt;500</td>
<td>Increased in hyperlipidemia from PN, hepatic disease, poorly controlled diabetes Decreased in malnutrition, malabsorption syndrome</td>
</tr>
</tbody>
</table>
4. Patient History

Assessing the patient’s history, including socioeconomic status, education level, social support systems, family situation, lifestyle and attitude towards their disease state will all affect nutrition status, both in the short term and long term. It is important to understand if a patient has the ability to follow nutrition recommendations outside of the hospital setting, which could strongly be affected by their economic status and support systems. Their attitude towards their disease state and nutrition recommendations may help one evaluate where the patient is in his/her readiness to learn. A patient’s prognosis and medical course are also extremely important factors to consider when giving nutrition recommendations. For instance, if a patient’s goal is comfort versus survival and palliative care is involved, it would be important to understand where the patient stands in receiving alternate forms of nutrition support through parenteral or enteral nutrition. All of these factors will influence the interventions and goals set later in the nutrition care process.

B. Nutrition Diagnosis

Nutrition diagnoses are often subjective and can vary from day to day depending on the nutritional status of the patient and the intervention priority, especially in the oncology population. As a patient’s condition evolves, so does the diagnosis. Diagnoses and interventions should be discussed with other areas of the medical team such as nurses, practitioners, doctors, nurses and speech pathologists in order to assess how realistic an intervention is. The following are examples of possible diagnoses throughout the course
of treatment for a patient with MF treated with SCT along with the potential reasons for the diagnosis (this list does not cover all potential diagnoses/causes):

- **MF patients prior to treatment:**
  - Inadequate energy intake – related to early satiety from splenomegaly
  - Unintended weight loss – related to inadequate intake from nausea or fatigue from low red blood cells
  - Excessive iron intake – related to increased iron levels from red blood cell transfusions

- **During conditioning treatments:**
  - Inadequate energy intake – related to increased needs and/or nausea/vomiting, early satiety, anorexia, taste changes
  - Unintended weight loss – related to increased needs from hypermetabolism
  - Increased nutrient needs – related to altered absorption (from N/V/D/C)
  - Altered nutrition-related labs (glucose levels) – related to steroid medications and/or nutrition support

- **Post-SCT:**
  - Inadequate energy intake – related to increased needs and/or nausea/vomiting, early satiety, anorexia
  - Inadequate protein intake – related to increased needs for tissue repair
  - Altered nutrition-related labs – related to electrolyte imbalances from emesis and diarrhea
  - Excessive fat intake – related to decreased needs from diarrhea/steatorrhea
  - Altered GI function – related to GI GVHD
  - Chewing/swallowing difficulty – related to oral GVHD
  - Food- and nutrition-related knowledge deficit – related to neutropenic diet

**C. Nutrition Intervention**

**1. Prioritizing Diagnoses and Identifying Goals**

The intake domain remains the most important area to tackle for a dietitian. It can often be the most acute problem, especially in oncology patients, and therefore it is commonly the most urgent. Weight changes are also extremely important to note in this population, as changes in weight often signify an underlying problem.
such as hypermetabolism, inadequate intake, or inability to consume sufficient nutrients orally.

Two aspects of the SCT population make it somewhat difficult to prioritize diagnoses. The first is that changes can take place so acutely that the most important diagnosis one day may differ from the most important diagnosis the next. For instance, a patient may have mucositis on Monday in which the goal might be to consume at least one high calorie nutritional supplement drink a day. Then Tuesday the patient may develop excessive diarrhea and nausea – in which the intervention would likely be to remain NPO until symptoms resolve – all while the mucositis still remains. The point is that goals may change daily and close monitoring is extremely important in this population. The second aspect – which is somewhat of a continuation of the first – is that multiple diagnoses may present themselves at once and it is up to the dietitian to prioritize based on experience and knowledge. Continuing with the previous example, during this time of mucositis, diarrhea and nausea, nutrition-related lab values may be altered as a side effect of these conditions and medications in which the goal may be to replete electrolytes and try to normalize lab values. Furthermore, a week later the patient may show signs of severe weight loss from the inadequate intake and increased needs in which the goal would be to increase intake and weight. The priorities and interventions will continually and quickly change based on the patient’s condition and needs.

Interdisciplinary practices are extremely important in order to prioritize goals. It is the job of the RD to convey the importance of nutritional care during this time to
other disciplines. In addition, it is just an important for the RD to understand the goals of care of the other disciplines in order to understand what are realistic interventions for the patient.

2. Planning and Implementing the Nutrition Intervention

Interventions for this patient population are discussed above in the section “Symptoms and Nutritional Interventions.” When implementing these interventions, it is important to discuss any dietary changes with the medical team in order to make sure nutritional and dietary goals are consistent with the medical goals. It is also important to educate the patient on the changes being made, as this will help with educating the patient on nutritional guidelines and will likely contribute to better compliance.

D. Nutrition Monitoring and Evaluation

1. Food/Nutrition-Related History Outcomes

Monitoring food intake will be one of the most important parts of this process. If a patient is in the hospital, interviewing is an efficient way of monitoring intake. If the patient were unable to convey his or her food intake, a 3-day calorie count conducted by a nurse would be a good option. When discharged from the hospital, SCT patients must follow up weekly with an outpatient doctor over an extended period of time. During these appointments, intake should be assessed along with food tolerance, diet adherence, physical activity, food supplies and availability, and overall nutritional quality of life.
2. Anthropometric Measurement Outcomes

The following measurements should be continually assessed throughout treatment and after treatment for an extended period of time to be determined by the medical team:

- Height
- Weight
- BMI
- IBW
- %IBW
- Weight change over the past month, 6 months, 1 year
- %UBW
- Skinfold measurements (subcutaneous fat)
- Mid-arm muscle circumference (lean body mass)
- Edema

In addition, nutrition-focused physical findings should be assessed, such as physical appearance, muscle and fat wasting, and swallow function.

3. Biochemical Data

The same labs noted above under “Biochemical Data, Medical Tests and Procedures” should be assessed continually throughout and after treatment.
PRESENTATION OF THE PATIENT

Patient: MS, 47 year old male

Admission date: November 7, 2012

Current diagnosis: Myelofibrosis (MF), Jak2 V617F mutation positive

Duration of present illness: 8 years from diagnosis

Admitted for: Allogenic stem cell transplantation (allo-SCT)

Past medical history: MS was diagnosed with MF – Jak2 mutation positive in 2004 when he presented with a ruptured spleen after a snowboarding fall. He was seen at Winthrop in Long Island where he had a bone marrow examination and was diagnosed with MF. From diagnosis to May 2012, MS was treated with long-course hydroxyurea (HU) 1,000 mg/day. Throughout his treatment, he had transfusion-dependent anemia and marked splenomegaly.

From May to August 2012, his therapy was switched to Jakafi 15 mg twice a day. In August 2012 his counts dropped significantly and the medication was discontinued. He was then admitted to Winthrop with fever and severe anemia. He received a packed red blood cell (PRBC) transfusion and was placed on low dose Jakafi which he took until 11/6/12. He has an 10/10 HLA identical sibling and was referred to MSH for allo-SCT.

Family history: no relevant family history

Social history:
- Married with three children
- Works as a high school teacher
- Non-smoker, denies alcohol/drug use
- Strong familial support system
MS was admitted to Mount Sinai on November 7, 2012 specifically for stem cell transplantation treatment. He was treated for 7 days with a combination of Fludarabine and Busulfan for his chemotherapy conditioning. After a day of rest (D-1), he received the stem cell transplantation infusion from his brother. Of note, MS has four brothers and one was a 10/10 HLA match. His brother’s cells were harvested peripherally the same morning of transplantation (#D 0), processed the same day, and then administered into MS. The below description navigates through MS’s hospital course with the nutritional interventions in ADIME note form throughout. MS’s hospital stay was from November 7-December 3, 2012. Nutrition saw him weekly throughout his stay. He continues to follow-up weekly in an outpatient clinic to monitor his engraftment.

The medications used are described in the “Medication Bibliography” section under “Patient Medications.” Events post-discharge are described in the “Summary Section” below.

**November 7: #Day-8**
- Admission
- Hickman catheter placed
- Chemotherapy plan:
  - Conditioning:
    - Fludarabine (Fludara) – Flu
    - Busulfan (Busulfex) – Bu
  - Post-SCT
    - Methotrexate (MTX)
- **Physical findings:** palpable splenomegaly
- **Conditioning Schedule:**
  - D-7: Flu + Bu
  - D-6: Flu + Bu
  - D-5: Flu + Bu
  - D-4: Flu
  - D-3: Flu
  - D-2: Flu
  - D-1: Rest day
  - D 0: SC infusion
- **GVHD/Immunosuppression:** Tacrolimus from D-1 and MTX IV on D+1, D+3, D+6
- **Medical goals during treatment:**
  - To be discharged home, engrafted and clinically well

**November 8: #D-7**

<table>
<thead>
<tr>
<th>Initial Nutrition Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Assessment:</strong> 48 y/o M with dx of MF since 2004, treated with HU then Jakafi until 8/12 when his counts dropped significantly; was then admitted to Winthrop with fever and severe anemia. Admitted to MSH 11/7 for allo-SCT from HLA-identical sibling. Noted conditioning treatment begins today. Visited pt at bedside with wife present. Pt</td>
</tr>
</tbody>
</table>
reports fair appetite since admit (does not enjoy hospital food but eats meals brought in by wife). Pt is with no complaints of nausea/vomiting or chewing/swallowing difficulty at this time, no recent wt loss (UBW~190#). Pt reports loose stool x1 today. Pt follows a regular diet at home. NKFA.

**BMT Day#: -7**
**Diet:** Low Bacteria
**Supplements:** None

**Relevant labs:** Ca 8.4
**Notable meds:** acyclovir, levaquin, ondansetron, dexamethasone

**Chemo:** fludarabine, busulfan

**Skin:** no breakdown noted
**Ext:** no edema noted

**Anthropometrics**

- **Ht:** 1.81m (71”)
- **Wt:** 85 kg (187#)
- **IBW:** 78 kg (+/- 10%)
- **%IBW:** 109%
- **BMI:** 26

**Estimated Nutritional Needs:**

- **Kcal:** 2120-2550 kcal/day (25-30 kcal/kg)
- **Pro:** 102-128 g/day (1.2-1.5 g/kg)

**Nutrition Diagnosis:**

1. Food- and nutrition-related knowledge deficit related to lack of prior exposure to accurate nutrition information as evidenced by no prior knowledge of low bacteria diet s/p chemo and SCT.

Pt at moderate nutrition risk secondary to chemotherapy treatment initiation and need for neutropenic diet. Pt appears normal weight consistent with BMI 26. Continue with Low Bacteria diet. Extensive low bacteria diet education was given; pt and wife asked appropriate questions and demonstrated good understanding; compliance is expected. Will reinforce before discharge.

**Nutrition Interventions:**

1. Continue with low bacteria diet
2. Provided written and verbal low bacterial diet education
3. Provide supplement as needed
4. Low Ca noted, replete as medically appropriate
5. Encourage good po and fluid intake

**Monitor/Evaluation:**

1. Anthropometrics, labs, po intake and diet tolerance, nutrition-focused physical findings, BM

**November 13: #D-2**

MS began experiencing diarrhea 1-3x/day with nausea and little appetite. He was given Immodium to help with the diarrhea.

**Nutrition Follow Up**

Previous nutrition note reviewed. Pt reports feeling tired with no appetite, poor po intake (only ate soup yesterday brought in by wife). Soup provides ~400 kcals, 8 g protein. Pt with complaints of some nausea, diarrhea 1-3x/day. Pt denies vomiting, no chewing/swallowing difficulties at this time. Pt not consuming shake.

**BMT Day#: -2**
**Diet:** Low bacteria  
**Supplements:** shake - whole milk, ice cream, beneprotein

**Labs:** Ca 8.3  
**Notable meds:** acyclovir, levaquin, ondansetron, immodium, methylprednisolone  
**Chemo:** fludarabine  
**Skin:** no breakdown noted

**Anthropometrics**  
Wt (11/13): 86.8 kg  
Admitted wt (11/7): 83.7  
Wt change: 3.7% gain in 6 days

**Nutrition diagnosis:** Inadequate oral intake related to nausea, diarrhea, and appetite loss from high dose chemotherapy as evidenced by consumption of <25% of nutritional needs.

**Nutrition Interventions:**
1. Continue with low bacteria diet  
2. Pt not consuming shakes 2/2 nausea and diarrhea; will discontinue shakes  
3. Reviewed food options consistent with Low Fiber diet and encouraged to increase po intake as able  
4. Noted pt on Immodium – would continue until diarrhea resolves  
5. Wife to track po intake  
6. Encourage fluid intake

**Monitor/Evaluation:**
1. Anthropometrics, labs, po intake and diet tolerance, nutrition-focused physical findings, BM

**November 15:** #D-0 Stem cell infusion day  
- MS began feeling better overall with improved nausea. However, he began experiencing drenching night sweats during the night and had a rash over his back for two nights. He denied fevers, chills, and abdominal pain at this time. He still remained with palpable splenomegaly at this point. Any nausea he was feeling was likely due to Fludarabine and was being controlled by Ativan and Zofran. Received Benadryl for rash on back which helped with significant improvement.  
- MS was seen by Dermatology who noted “rash is mildly pruritic but not otherwise bothersome.”

**November 20, 2012, #D+5**  
#D+5: Mucositis pain developed. Palliative care intervened and added a morphine drip.

**Nutrition Follow Up**

Previous nutrition note reviewed, diarrhea has resolved at this time. Pt reports feeling okay with increasing appetite, fair po intake (cornflakes for breakfast x2, canned soup for lunch, hospital dinner). Pt with some complaints of pain when swallowing 2/2 sores on tongue. Per NP note, pt with mucositis. Pt with no complaints of N/V/D/C, +BM today.

**BMT Day#:** +5  
**Diet:** Low Bacteria  
**Supplements:** adding shake - vanilla soy milk, banana, 2 packets beneprotein

**Labs:** neutrophils 0.5  
**Meds:** prograf, tacrolimus, magic mouthwash, cancidas
**Anthropometrics**

Wt (11/20): 82.8 kg  
Wt (11/13): 86.8 kg  
Wt change: 4.6% x1 week (significant)

**Nutrition diagnosis:** Unintended weight loss related to diarrhea and appetite loss likely from high dose chemo and SCT as evidenced by significant weight loss of 4.6% loss x1 week.

**Nutrition Interventions:**
1. Continue with low bacteria diet
2. Provide supplement: shake (vanilla soy milk, banana, 2 packets beneprotein, provides 250 kcal, 18 g protein)
3. If sores on tongue continue, recommend Mechanically Altered, Low Bacteria Diet
4. Encourage good po and fluid intake

**Monitor/Evaluation:**
1. Anthropometrics, labs, po intake and diet tolerance, nutrition-focused physical findings, BM

---

**November 22, 2012, #D+7**

Neutropen administered to increase WBC. The chart below shows WBC trending throughout his treatment, with the red line showing the date that conditioning began and the yellow line showing this day (11/22) when Neupogen was administered. The WBC increased significantly once administered and helped ease the mucositis pain over the following week.

(Normal count: 4.5-11 x 10^3 uL)

Red line – conditioning treatment began  
Yellow line – pt started on Neupogen (11/22, #D+7)

**November 24, 2012, #D+9**

Pt reported worsening mouth and throat pain (“dry, scratching”) along with difficulty swallowing due to mouth sores; noted to be grade II mucositis. He began swabbing with viscous lidocaine and using magic mouthwash. Diarrhea resolved at this point with the use of loperamide.
November 25, 2012, #D+10
Pt had no BM x3 days and now presented with nausea and vomiting, triggered after he has eaten something. He was started on colace and senna. TPN noted to be a consideration if poor po intake continued.

November 26, 2012, #D+11
Pt reported marked improvement of mouth and throat pain and less difficulty swallowing compared to previous day. He still reported very poor appetite and almost no po intake. There was no fever or other constitutional symptoms at this point. Mucositis began improving with return of WBC. +BM

November 27, 2012, #D+12
Liver Consult - Liver was consulted secondary to cholestasis recognized in an increase in AST/ALT/bilirubin (as shown in the chart below). The etiology was likely hepatotoxicity from Fludarabine and Busulfan and indirect bilirubinemia from transfusions.
• At this point, pt denied any abdominal pain, but admitted to nausea, constipation, and diarrhea. Noted no history of ETOH, drug use, liver disease or family hx of liver disease. PT's WBC began uptrending on 11/26/12. Pt was being maintained on Prograf for immunosuppression.

November 28, 2012, #D+13
Pt reported feeling better however remained with poor appetite. He reported having improved stamina and one episode of vomiting. No fever or other constitutional symptoms were reported at this point.
Nutrition Follow Up

Previous nutrition notes reviewed. During RD visit yesterday, pt reports feeling better with less mouth pain. Notes he will attempt to increase intake. Pt seen at bedside this am, reports feeling okay with increasing appetite, improved po intake x2 days (consumed bagel with cream cheese this am). Pt reports not liking the shake; will discontinue. Pt with no c/o N/V/D/C, however per MD note pt with emesis yesterday. Pt’s wife monitoring intake – reports intake is improving.

BMT Day#: +13
Diet: Low Bacteria
Supplements: none

Labs: BUN 9, Alb 3.4, Abs neutrophils 3.55 (WNL)
Meds: tacrolimus, magic mouthwash, cancidas, colace, senokot

Anthropometrics
Wt (11/28): 82.2 kg   Wt (11/20): 82.8 kg
Wt change: >1%  
BMI: 25

Nutrition Interventions:
1. Continue with low bacteria diet
2. Discontinue supplement
3. Dietary options reviewed for nausea/diarrhea – focused on soft, bland, low fiber foods
4. Encourage good po and fluid intake

Monitor/Evaluation:
1. Anthropometrics, labs, po intake and diet tolerance, nutrition-focused physical findings, BM

November 29, 2012, #D+14
Neupogen discontinued
Pt reported feeling better with his appetite improving. The day before he ate 3 small meals with no nausea or vomiting as his mouth/throat pain improved. He denied diarrhea but experienced occasional soft stools at this point (he was not taking an anti-motility agent). He was walking around with increased energy.

November 30, 2012, #D+15
Pt reported feeling well and that mucositis pain in mouth and throat almost completely resolved. He is was tolerating a po diet well, +BM. No other complaints were offered.

December 3, 2012 – DISCARDED
Pt was discharged medically stable on low bacteria diet with no signs of GVHD. Low bacteria diet education was reinforced prior to discharge.
CRITICAL COMMENTS

MS was admitted in a relatively stable state nutritionally. He had not experienced any recent changes in appetite or weight. From my initial assessment, I was able to gather that the patient and his wife were well educated and understood the importance of nutrition in MS’s treatment. During that assessment, I stated that my goals were to keep his weight stable by offering supplements and food options when his appetite decreased or when he was unable to consume adequate amounts of food. I also explained the Low Bacteria diet in detail, and the patient and his wife asked appropriate questions.

After this initial assessment, I checked in with the patient weekly and monitored labs and weight closely on days between visits. The interdisciplinary aspect of MS’s care team was extremely helpful in monitoring this patient, as it was clear from their notes when and why certain labs and medications changed. In addition, nutrition and intake were always noted. As expected, MS’s intake dropped significantly as he began feeling the effects of the conditioning treatment. Oral nutrition supplements were offered to increase protein and calorie intake; however, the patient was not amenable to these shakes throughout his stay. During his period with mucositis, MS was not eating anything which was a cause of concern and prompted the MD to suggest TPN if his intake remained low. However, MS bounced back around #D+13 after the mucositis resolved and his WBCs increased. Nutrition support was never necessary.

When looking back on the nutritional interventions, it would have been better if MS had not experienced a period without intake. I understand that this is often unavoidable especially during times of nausea and mucositis pain, but I was at a loss when he first refused the supplemental shake due to a dislike of rice/soy milk (regular milk was not allowed due to his period of diarrhea). I believe it may have been that he was not familiar with those products and
therefore did not want to consume something foreign, especially during periods of nausea. If I were to do anything over, I would have explained the importance of not consuming dairy during his stay and the potential need for a non-dairy nutritional supplement during my initial assessment and education. I would have possibly had him try a shake before feeling sick so he could determine better whether he truly felt adverse to the taste or if it was the idea of rice/soy milk, as he was unfamiliar with them prior to his admission. More generally, I learned that in the future when working with this population, I should explain what to expect in terms of dietary changes beyond the neutropenic diet based on this experience and the knowledge I gained from writing this paper. This is a small change, but I believe it could make a big difference. I would like to note that I believe the continuous monitoring, emphasis on the importance of intake for success, and the formation of a trusting relationship with the patient helped contribute to his nutritional success. MS was discharged only 3% lighter than when he was admitted, and he left with the understanding of the importance of nutrition for his continued care.
PATIENT UPDATE

SCT patients are required to visit an outpatient clinic weekly for a period of time post-discharge, therefore MS has been monitored closely since December 3rd when he left Mount Sinai. While his stay at the hospital was relatively unremarkable in terms of complications, his status post-discharge has declined slightly. As of the latest note on January 24th (#D+70), MS currently has skin GVHD grade II-III and began taking Prednisone to help ease the symptoms. He was also noted to have increased liver function tests (see the chart below for his bilirubin trend post-discharge; the yellow shows stage I liver GVHD according to the criteria below) and diarrhea 1-3x/day (volume not available).

<table>
<thead>
<tr>
<th>Staging of Acute GVHD</th>
<th>Liver</th>
<th>GI Tract</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td>Stage 1: bili 2-3 mg/dL</td>
<td>Stage 1: diarrhea &gt; 500 mL/day</td>
</tr>
<tr>
<td>Stage 2: 25-50% rash</td>
<td>Stage 2: bili 3.1-6 mg/dL</td>
<td>Stage 2: diarrhea &gt; 1000 mL/day</td>
</tr>
<tr>
<td>Stage 3: generalized erythroderma</td>
<td>Stage 3: bili 6.1-15 mg/dL</td>
<td>Stage 3: diarrhea &gt; 1500 mL/day</td>
</tr>
<tr>
<td>Stage 4: bullae (blisters)</td>
<td>Stage 4: bili &gt;15 mg/dL</td>
<td>Stage 4: ileus, bleeding</td>
</tr>
<tr>
<td>GI Tract</td>
<td>Stage 1: diarrhea &gt; 500 mL/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Stage 2: diarrhea &gt; 1000 mL/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Stage 3: diarrhea &gt; 1500 mL/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Stage 4: ileus, bleeding</td>
<td></td>
</tr>
</tbody>
</table>

Since each of symptoms is a sign of increasing GVHD, the doctor increased his immunosuppressive regimen to include CellCept along with the Prograf and Prednisone. Per the MD’s notes in the outpatient clinic, MS continues with a good appetite, no weight loss, and is walking for exercise.
Summary

Myelofibrosis treated with STC is a complicated process that involves a variety of interventions and treatments that affect a patient’s nutritional status. When developing a nutritional plan in the SCT population, it can be difficult to weigh the pros and cons of the many nutritional interventions as a patient’s status can change daily. It is important to understand and follow the evidenced-based research set for this population – especially when considering nutritional support – in order to best serve the patient.
# Medication Bibliography

**List of Patient’s Medications**

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Medication Name</th>
<th>Indications</th>
<th>Diet/Nutritional Side Effects</th>
<th>Monitor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antibiotic/Antiviral therapy</td>
<td>Acyclovir</td>
<td>To treat/prevent bacteria infections during immunosuppression</td>
<td>Ensure adequate fluid intake; may lead to N/V/D/C, abd pain</td>
<td>Monitor renal function, may increase BUN, Cr, AST, ALT</td>
</tr>
<tr>
<td>Bronchodilator</td>
<td>Albuterol (Proventil)</td>
<td>Allows pts to breathe more easily; also indicated to treat hyperkalemia</td>
<td>Sore dry throat</td>
<td>Monitor K+, Glu, Chol</td>
</tr>
<tr>
<td><strong>Antimalarial drug, Antiprotozoal agent</strong></td>
<td>Atovaquone (Mepron)</td>
<td>Treat/prevent pneumonia caused by a fungal infection called Pneumocystis carinii pneumonia (PCP)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Antibiotic</td>
<td>Bacitracin Polymyxin (Polysporin)</td>
<td>Wound healing</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Antineoplastic</td>
<td>Busulfan (Busulfex)</td>
<td>Chemotherapy; treats blood cancers</td>
<td>N/V/D/C, hair loss, mouth sores, appetite/weight loss, headache, dry mouth, insomnia, anxiety, dizziness, chest pain, joint pain, itchiness, dry skin, darkened skin</td>
<td>Monitor CBC, weight, neutrophils</td>
</tr>
<tr>
<td>Corticosteroid</td>
<td>Dexamethasone (Decadron)</td>
<td>Anti-inflammatory and immunosuppressant; used in cancer treatment to counteract chemo side effects</td>
<td>May lead to esophagitis, N/V, dyspepsia, peptic ulcer, bloating, GI bleeding/perforation Ca wasting with long term use - may lead to fractures/muscle wasting Steroid-induced diabetes May increase weight/appetite Negative N balance due to pro catabolism</td>
<td>Monitor electrolytes, adrenal function, glucose, BP, weight, bone density</td>
</tr>
<tr>
<td>Antihistamine</td>
<td>Diphenhydramine (Benadryl)</td>
<td>Treats allergic reactions</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Laxative</td>
<td>Docusate sodium (Colace)</td>
<td>Stool softener, for constipation</td>
<td>May cause bitter taste, nausea, diarrhea, cramps</td>
<td>Monitor electrolytes, I+O’s</td>
</tr>
<tr>
<td>Anticoagulant</td>
<td>Enoxaparin sodium (Lovenox)</td>
<td>Used to prevent and treat deep vein thrombosis or pulmonary embolism</td>
<td>-</td>
<td>Monitor renal function, CBC, platelet count</td>
</tr>
<tr>
<td>Catecholamine</td>
<td>Epinephrine</td>
<td>Treats severe allergic reactions</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Category</td>
<td>Drug Name</td>
<td>Main uses</td>
<td>Side effects</td>
<td>Monitoring/Precautions</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
<td>---------------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Histamine H2-receptor antagonist</td>
<td>Famotidine (Pepcid)</td>
<td>Treats ulcers, gastroesophageal reflux disease (GERD), and conditions that cause the stomach to produce too much stomach acid</td>
<td>Do not take with Fe, Mg, antacids; may decrease Fe and Vit B12 abs; may cause N/V/D/C</td>
<td>Monitor hepatic func, Vit B12 with long term use</td>
</tr>
<tr>
<td>Colony-stimulating factor</td>
<td>Filgrastim (Neupogen)</td>
<td>Treats neutropenia during/after cancer treatment by increasing the production and activity of neutrophils (WBCs)</td>
<td>May cause bone pain, splenomegaly, nose bleed, fever</td>
<td>Monitor neutrophils, white blood cells</td>
</tr>
<tr>
<td>Antineoplastic</td>
<td>Fludarabine (Fludara)</td>
<td>Chemotherapy; treats blood cancers; interferes with DNA synthesis</td>
<td>May cause stomatitis, dysphagia, N/V/D/C, hair loss, mouth sores, appetite/weight loss, headache, dry mouth, insomnia, anxiety, dizziness, chest pain, joint pain, itchiness, dry skin, darkened skin</td>
<td>Monitor CBC, weight, neutrophils</td>
</tr>
<tr>
<td>Hormone</td>
<td>Glucagon</td>
<td>Treats severe low blood sugar</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Mouthwash</td>
<td>Grade 0 mouthwash</td>
<td>Prevents/treats chemotherapy-induced oral mucositis</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Corticosteroid</td>
<td>Hydrocortisone</td>
<td>Anti-inflammatory</td>
<td>May lead to esophagitis, N/V, dyspepsia, peptic ulcer, bloating, GI bleeding/perforation Ca wasting with long term use - may lead to fractures/muscle wasting Steroid-induced diabetes May increase weight/appetite Negative N balance due to pro catabolism</td>
<td>Monitor electrolytes, adrenal function, glucose, BP, weight, bone density</td>
</tr>
<tr>
<td>Opioid</td>
<td>Hydromorphone (Dilaudid)</td>
<td>Treats moderate to severe pain</td>
<td>Avoid grapefruit; may lead to dry mouth, dyspepsia, gastritis, N/V, diarrhea, constipation</td>
<td>Monitor liver enzymes, sodium</td>
</tr>
<tr>
<td>Antineoplastic</td>
<td>Hydroxyurea (HU)</td>
<td>Longterm use chemotherapy</td>
<td>Increase fluid intake; may lead to anorexia, stomatitis, N/V/D/C</td>
<td>Monitor baseline and periodic CBC with diff and platelets; hepatic, renal function</td>
</tr>
<tr>
<td>Anti-epileptic</td>
<td>Levetiracetam (Keppra)</td>
<td>Used to treat seizures</td>
<td>May lead to anorexia; do not take with renal dysfunction; may decrease RBC, Hb, HCT, WBC,</td>
<td>Monitor baseline CBC, renal function</td>
</tr>
<tr>
<td>Category</td>
<td>Drug Name</td>
<td>Uses</td>
<td>Side Effects</td>
<td>Lab Monitoring</td>
</tr>
<tr>
<td>--------------------------</td>
<td>--------------------</td>
<td>----------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------</td>
<td>---------------------------------------------</td>
</tr>
<tr>
<td>Neutrophils, Platelets</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Benzodiazepine, Anxiolytic, Antiemetic</td>
<td>Lorazepam (Ativan)</td>
<td>Treats anxiety, anxiety with depression, and insomnia; also works to prevent/treat anxiety and the symptoms of anticipatory nausea and vomiting associated with chemotherapy</td>
<td>Caution when taking with grapefruit, chamomile, caffeine; may cause anorexia, decreased wt, increased appetite; caution with hepatic/renal function</td>
<td>Monitor CBC with diff, hepatic and renal function</td>
</tr>
<tr>
<td>Mouthwash</td>
<td>Magic Mouthwash</td>
<td></td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Mineral supplement</td>
<td>Magnesium chloride</td>
<td>Treats hypomagnesaemia</td>
<td>Take fiber, Fe, Fol separately by at least 2 hrs; may cause a chalky taste, N/V/D/C</td>
<td>Monitor Mg</td>
</tr>
<tr>
<td>Antineoplastic</td>
<td>Methotrexate</td>
<td>Treats several kinds of cancer, including cancer of the blood, bone, lung, breast, head, or neck; suppresses the immune system to help prevent transplant rejection</td>
<td>Encourage fluid intake; may decrease folate abs; may cause stomatitis, gingivitis, altered taste, N/V, diarrhea</td>
<td>Monitor baseline and periodic CBC with diff and platelets; hepatic, renal function, alb</td>
</tr>
<tr>
<td>Corticosteroid</td>
<td>Methylprednisolone</td>
<td>Anti-inflammatory</td>
<td>May lead to esophagitis, N/V, dyspepsia, peptic ulcer, bloating, GI bleeding/perforation Ca wasting with long term use - may lead to fractures/muscle wasting Steroid-induced diabetes May increase weight/appetite Negative N balance due to pro catabolism</td>
<td>Monitor electrolytes, adrenal function, glucose, BP, weight, bone density</td>
</tr>
<tr>
<td>Narcotic</td>
<td>Morphine</td>
<td>Treats moderate to severe pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antifungal (oral treatment)</td>
<td>Nystatin (Mycostatin)</td>
<td>Treats fungal infections</td>
<td>May cause GI distress, N/V, stomach pain, diarrhea</td>
<td>-</td>
</tr>
<tr>
<td>Antiemetic</td>
<td>Ondansetron (Zofran)</td>
<td>Prevents nausea and vomiting caused by cancer treatments</td>
<td>May cause dry mouth, abd pain, constipation, diarrhea</td>
<td>Monitor hepatic function</td>
</tr>
<tr>
<td>Opioid</td>
<td>Oxycodone (Roxicodone)</td>
<td>Treats moderate to severe pain</td>
<td>Avoid grapefruit; may lead to dry mouth, dyspepsia, gastritis, N/V, diarrhea, constipation</td>
<td>Monitor liver enzymes, sodium</td>
</tr>
<tr>
<td>Mineral supplement</td>
<td>Potassium chloride</td>
<td>Treats hypokalemia; may cause N/V, abd pain, diarrhea</td>
<td>May cause GI distress, N/V/, abd pain, diarrhea, flatulence; caution with renal dysfunction, dysphagia; do not take</td>
<td>Monitor K+</td>
</tr>
<tr>
<td>Class</td>
<td>Example</td>
<td>Description</td>
<td>Side Effects</td>
<td>Monitoring</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-----------------------</td>
<td>-----------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Corticosteroid</td>
<td>Prednisone</td>
<td>Treats inflammation and used to decrease some symptoms of cancer including mucositis, GVHD</td>
<td>May lead to esophagitis, N/V, dyspepsia, peptic ulcer, bloating, GI bleeding/perforation Ca wasting with long term use - may lead to fractures/muscle wasting Steroid-induced diabetes May increase weight/appetite Negative N balance due to pro catabolism</td>
<td>Monitor electrolytes, adrenal function, glucose, BP, weight, bone density</td>
</tr>
<tr>
<td>Antiemetic</td>
<td>Prochlorperazine (Compazine)</td>
<td>Treats severe nausea and vomiting</td>
<td>May cause dry mouth, abd pain, constipation, diarrhea</td>
<td>Monitor hepatic function</td>
</tr>
<tr>
<td>Laxative</td>
<td>Sennosides</td>
<td>Treats constipation</td>
<td>May cause electrolyte imbalances, N/V, cramps; need to consume adequate fluids</td>
<td>Monitor electrolytes, I+O's</td>
</tr>
<tr>
<td>Immunosuppressant</td>
<td>Tacrolimus (Prograf)</td>
<td>An immunosuppressant - used after allo transplants to suppress the patient's immune system so to lower the risk of organ rejection; helps treat/prevent GVHD</td>
<td>May lead to anorexia, increased appetite, decreased Fe; may cause oral candidiasis, stomatitis, dysphagia, dyspepsia, N/V/D/C, gastritis, hemorrhage, GI perforation, abd pain, flatulence, HTN, hyperglycemia, hyperkalemia, hypomagnesemia, nephrotoxicity, neurotoxicity</td>
<td>Monitor baseline and periodic - BP, electrolytes, Mg, K+, renal function, hepatic function, CBC with diff, platelets, drug level</td>
</tr>
<tr>
<td>Bile acid</td>
<td>Ursodiol</td>
<td>Dissolves gallstones and treats primary biliary cirrhosis</td>
<td>May cause N/V, stomach pain</td>
<td>Monitor liver enzymes</td>
</tr>
<tr>
<td>Antibiotic/Antifungal therapy</td>
<td>Voriconazole</td>
<td>To treat/prevent fungal infections during immunosuppression; treats mucositis, esophagitis</td>
<td>Take 1 hr before/after food; avoid SJW; may lead to dry mouth, N/V, abd pain, diarrhea; correct hypokalemia, hypocalcemia or hypomagnesemia prior to use. Do not take with hepatic dysfunction or renal dysfunction.</td>
<td>Monitor vision, hepatic, renal function, electrolytes</td>
</tr>
</tbody>
</table>
## MEDICATION BIBLIOGRAPHY
### MEDICATIONS TO EASE TREATMENT SIDE EFFECTS

<table>
<thead>
<tr>
<th>Drug</th>
<th>Brand Name</th>
<th>Use</th>
<th>Mechanism of action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorpromazine Hydrochloride</td>
<td>Chlor-Trimet®</td>
<td>Treatment of nausea and vomiting; psychoses; Tourette’s syndrome; mania; intractable hiccups (adults); behavioral problems (children)</td>
<td>Blocks postsynaptic mesolimbic dopaminergic receptors in the brain; exhibits a strong alpha-adrenergic blocking effect and depresses the release of hypothalamic and hypophyseal hormones</td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>Decadron®</td>
<td>Systemically and locally for chronic inflammation, allergic, hematologic, neoplastic, and autoimmune diseases; may be used in management of cerebral edema, septic shock, and as a diagnostic agent</td>
<td>Decreases inflammation by suppression of migration of polymorphonuclear leukocytes and reversal of increased capillary permeability; suppresses normal immune response</td>
</tr>
<tr>
<td>Diphenhydramine Hydrochloride</td>
<td>Benadryl®</td>
<td>Symptomatiac relief of allergic symptoms caused by histamine release that include nasal allergies and allergic dermatosis; milk nighttime sedation, prevention of motion sickness, as an antitussive; treatment of metoclopramide-or phenothiazine-induced dystonic reactions</td>
<td>Competes with histamine for H1 receptor sites on effector cells in the GI tract, blood vessels, and respiratory tract</td>
</tr>
<tr>
<td>Dronabinol</td>
<td>Marinol®</td>
<td>Treatment of nausea and vomiting secondary to cancer chemotherapy in patients who have not responded to conventional antiemetics; treatment of anorexia associated with weight loss in AIDS patients</td>
<td>Dronabinol is the principal psychoactive substance found in Cannabis sativa (marijuana); its mechanism of action as an antiemetic is not well defined, it probably inhibits the vomiting center in the medulla oblongata; has complex effects on CNS including central sympathomimetic activity</td>
</tr>
<tr>
<td>Droperidol</td>
<td>Inapsine®</td>
<td>Tranquilizer and antiemetic in surgical and diagnostic procedures; antiemetic for cancer chemotherapy; preoperative medication</td>
<td>Alters the action of dopamine in the CNS at subcortical levels to produce sedation and a dissociative state; also possesses alpha-adrenergic blockade effects</td>
</tr>
<tr>
<td>Granisetron</td>
<td>Kytril®</td>
<td>Prophylaxis and treatment of chemotherapy and radiation-related nausea and emesis; prophylaxis and treatment of postoperative nausea and vomiting</td>
<td>Selective 5-HT3 receptor antagonist, blocking serotonin, both peripherally on vagal nerve terminals and centrally in the chemoreceptor trigger zone</td>
</tr>
<tr>
<td>Hydroxyzine</td>
<td>Vistaril®</td>
<td>Treatment of anxiety; preoperative sedative; antipruritic; antiemetic</td>
<td>Competes with histamine for H1 receptor sites on effector cells in the GI tract, blood vessels, and respiratory tract</td>
</tr>
<tr>
<td>Drug</td>
<td>Brand Name</td>
<td>Indications</td>
<td>Mechanism of Action</td>
</tr>
<tr>
<td>-------------------------</td>
<td>------------------------</td>
<td>-----------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>Ativan®; Lorazepam</td>
<td>Management of anxiety; status epilepticus; preoperative sedation and amnesia</td>
<td>Depresses all levels of the CNS, including the limbic and reticular formation, by binding to the benzodiazepine site on the gamma-aminobutyric acid (GABA) receptor complex and modulating GABA, which is a major inhibitory neurotransmitter in the brain</td>
</tr>
<tr>
<td></td>
<td>Intensol®</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metoclopramide</td>
<td>Reglan®</td>
<td>Gastroesophageal reflux; prevention of nausea associated with chemotherapy;</td>
<td>Potent dopamine receptor antagonist; blocks dopamine receptors in chemoreceptor trigger zone of the CNS, preventing emesis; accelerates gastric emptying and intestinal transit time without stimulating gastric, biliary, or pancreatic secretions</td>
</tr>
<tr>
<td></td>
<td></td>
<td>facilities intubation of the small intestine and symptomatic treatment of</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>diabetic gastric stasis</td>
<td></td>
</tr>
<tr>
<td>Odansetron</td>
<td>Zofran® ODT; Zofran®</td>
<td>Prevention of nausea and vomiting associated with initial and repeat courses</td>
<td>Selective 5-HT3 receptor antagonist, blocking serotonin, both peripherally on vagal nerve terminals and centrally in the chemoreceptor trigger zone</td>
</tr>
<tr>
<td></td>
<td></td>
<td>of emetogenic cancer chemotherapy and prevention of postoperative and</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>radiation-induced nausea and vomiting</td>
<td></td>
</tr>
<tr>
<td>Prochlorperazine</td>
<td>ComproTM</td>
<td>Management of nausea and vomiting; acute and chronic psychosis</td>
<td>Blocks postsynaptic mesolimbic dopaminergic receptors in the brain, including the medullary chemoreceptor trigger zone; exhibits a strong alpha-adrenergic blocking effect and depresses the release of hypothalamic and hypophyseal hormones</td>
</tr>
<tr>
<td>Promethazine Hydrochloride</td>
<td>Phenergan®; Promethegan</td>
<td>Antiemetic; symptomatic treatment of various allergic conditions and motion</td>
<td>Blocks postsynaptic mesolimbic dopaminergic receptors in the brain; exhibits a strong alpha-adrenergic blocking effect and depresses the release of hypothalamic and hypophyseal hormones; competes with histamine for the H1-receptor</td>
</tr>
<tr>
<td></td>
<td>Tigan®</td>
<td>sickness; sedative</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medicine or Treatment</td>
<td>Mechanism of Action</td>
<td>Nutrition-Related Side Effects</td>
<td></td>
</tr>
<tr>
<td>-----------------------</td>
<td>---------------------</td>
<td>--------------------------------</td>
<td></td>
</tr>
<tr>
<td>Corticosteroids (systemic, topical, and mouthwash)</td>
<td>Anti-inflammatory response at arterial site inhibits IL-1 and decreases IL-2, which suppresses lymphocyte proliferation and decreases circulating lymphocytes</td>
<td>Fluid and sodium retention, hyperglycemia, hypertriglyceridemia, hypercholesterolemia, increased appetite, weight gain, muscle wasting, bone demineralization</td>
<td></td>
</tr>
<tr>
<td>Cyclosporine</td>
<td>Inhibits response of cytotoxic T cells to IL-2 and prevents T helper lymphocytes from producing IL-2</td>
<td>HTN, hyperglycemia, hyperkalemia, hypomagnesemia, hyperlipidemia, gingival hyperplasia, nephrotoxic, neurotoxic</td>
<td></td>
</tr>
<tr>
<td>Tacrolimus</td>
<td>Inhibits proliferation of cytotoxic T cells and synthesis of IL-2</td>
<td>HTN, hyperglycemia, hyperkalemia, N/V/D, hypomagnesemia, nephrotoxicity, neurotoxicity</td>
<td></td>
</tr>
<tr>
<td>Methotrexate</td>
<td>Antimetabolite, antineoplastic, immunosuppressant</td>
<td>Anorexia, N/V/D, stomatitis, mucositis, elevated liver function tests, renal failure, cirrhosis</td>
<td></td>
</tr>
<tr>
<td>Mycophenolate mofetil</td>
<td>Decreases lymphocyte activation and replication by suppressing enzymes in the purine salvage pathway, creating a purine deficiency and thus inhibiting T and B cell proliferation; suppresses antibody formation</td>
<td>N/V/D/C, GI bleeding, peripheral edema, sepsis</td>
<td></td>
</tr>
<tr>
<td>Sirolimus</td>
<td>Inhibits T and B cell proliferation while not affecting IL-2 production</td>
<td>Hypercholesterolemia, hypertriglyceridemia, HTN, peripheral edema</td>
<td></td>
</tr>
<tr>
<td>Thalidomide</td>
<td>Anti-inflammatory and immunosuppressive properties</td>
<td>Neuropathy, C, neutropenia</td>
<td></td>
</tr>
<tr>
<td>Antithymocyte globulin (ATG)</td>
<td>Decreases circulating lymphocytes</td>
<td>Abdominal pain, N/V/D, hyperkalemia, HTN, peripheral edema</td>
<td></td>
</tr>
<tr>
<td>Etanercept</td>
<td>TNF-α antagonist or binder</td>
<td>Abdominal pain, V, rhinitis</td>
<td></td>
</tr>
<tr>
<td>Ursodeoxycholic acid</td>
<td>Replaces native human bile acids, reduces class I HLA expression on hepatocytes</td>
<td>N/V/D, abdominal pain</td>
<td></td>
</tr>
<tr>
<td>Daclizumab</td>
<td>Humanized anti-IL-2 receptor antibody</td>
<td>V, edema, HTN, hypotension, fever, dyspnea, infection</td>
<td></td>
</tr>
<tr>
<td>Azathioprine</td>
<td>Inhibits RNA and DNA synthesis to prevent cytotoxic T and B cell proliferation and antibody production</td>
<td>GI hypersensitivity, hepatotoxicity, megaloblastic anemia, pancreatitis, infection, bone marrow suppression</td>
<td></td>
</tr>
<tr>
<td>Hydroxychloroquine</td>
<td>Interferes with antigen processing and presentation, proliferation, TNF-α production, and cytotoxicity, synergistic with cyclosporine and tacrolimus in vitro</td>
<td>N/V/D</td>
<td></td>
</tr>
<tr>
<td>Infliximab</td>
<td>Humanized chimeric antibody against TNF-α, IgG monoclonal antibody</td>
<td>Abdominal pain, N/V, worsening CHF</td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>Effect</td>
<td>Side Effects</td>
<td></td>
</tr>
<tr>
<td>------------------------------------------------</td>
<td>------------------------------------------------------------------------</td>
<td>-----------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Psoralen and PUVA</td>
<td>Interferes with antigen presentation and inflammatory cytokine production by Langerhan's cells, increases IL-10 production by keratinocytes</td>
<td>Increase in skin cancer, phototoxicity, N, hepatotoxicity</td>
<td></td>
</tr>
<tr>
<td>Extracorporeal photopheresis</td>
<td>Induces apoptosis in alloreactive T cells, normalization of CD4/CD8 ratios by decreasing CD8 cells, increases natural killer cells, decreases dendritic cells, photoinactivation of antigen presenting cells and T lymphocytes</td>
<td>GI upset, hypocalcemia (if citrate anticoagulant used)</td>
<td></td>
</tr>
</tbody>
</table>

N, nausea; V, vomiting; D, diarrhea, C, constipation; HTN, hypertension; Abd, abdominal; CHF, congestive heart failure; GI, gastrointestinal; TNF, tumor necrosis factor; IL, interleukin; HLA, human leukocyte antigen; IgG, immunoglobulin G.
REFERENCES


Dietary advice for patients with neutropenia

Revised January 2012

This publication has been produced and prepared by the London Haematology Dietitians Group for patients who may be at risk of food borne infection due to high dose chemotherapy and/or bone marrow transplant.

The information in this booklet is correct at the time of going to print. For further information please contact the patient information team who is the only person with full information about their diagnosis and medical history.

At all times patients should rely on the advice of their specialist who is the only person with full information about their diagnosis and medical history.

For further information please contact the patient information team on 020 7504 2200.

Leukaemia & Lymphoma Research,
39-40 Eagle Street, London WC1R 4HT

Leukaemia & Lymphoma Research

© All rights reserved. No part of this publication may be reproduced or transmitted, in any form or by any means, electronic or mechanical, including photocopying, recording or any information storage and retrieval system, without permission in writing from the London Haematology Dietitians Group or Leukaemia & Lymphoma Research.
Following chemotherapy there is a risk of infection from bacteria or fungus in foods. This is for two reasons: 1) The white blood cells (neutrophils) that would usually fight food poisoning bacteria are at a low level. This is referred to as neutropenia. 2) The gut lining acts as a barrier between bacteria and the bloodstream. Chemotherapy and radiotherapy damage the gut lining making it easier for any bacteria to enter the bloodstream.

During neutropenia, the following guidelines will help to reduce the risk of food poisoning whilst still allowing as varied a diet as possible. There is no single agreed definition of neutropenia or severe neutropenia; the levels quoted below are very widely used but if a doctor uses different levels, patients should be guided by those. The type of advice to be followed is dependent on the number of white blood cells in the bloodstream: this is known as the neutrophil count.

This booklet provides general advice on safe food handling and storage for all patients at risk of neutropenia. This advice should be followed at all times by patients, their families and friends. It also lists some foods that patients with neutropenia should avoid – for those with a neutrophil count of 0.5-2.0 × 10^9/litre and those with a neutrophil count below 0.5 × 10^9/litre.

Patients should check their neutrophil count with the doctor or nursing staff and follow the advice both in hospital and at home. Please note that the level of restriction required may vary dependent upon clinical condition and appropriate advice will be given. After a bone marrow or stem cell transplant, or when on immunosuppressive medication, it may be necessary to continue following these guidelines after the neutrophil count has recovered. This is because the immune system is weaker, increasing risk of infections. If any questions arise regarding this diet or eating in general, the doctor or nurse specialist can refer patients to a dietitian for specialist advice.
Food Preparation

Hands should always be washed with warm water and soap before preparing food.

Hands must be washed after going to the toilet, sneezing and after touching pets, hair, dirty washing, rubbish, ready-made or raw food.

Use a separate towel or use kitchen paper to dry hands, do not use a tea towel.

Cover any cuts and grazes with a waterproof plaster.

Keep pets away from work surfaces, food and your dishes.

Ensure any cloths or sponges are regularly bleached, disinfected or changed.

Avoid cross-contamination of food by changing or washing chopping boards and utensils between raw and cooked food.

Disinfect work surfaces regularly.

Wash fruit and vegetables before eating.

Wash can tops before opening them.

Pre-heat the oven to ensure food is cooked at the recommended temperature.

Cook meat until the juices are clear.

Cook all food thoroughly and ensure it is piping hot all the way through.

Do not reheat cooked food.

Always follow manufacturers’ guidelines and do not store

Pre-cooked food in the oven to ensure food is cooked at the

Keep pets away from work surfaces, food and your dishes.

Ensure any cloths or sponges are regularly bleached, disinfected.

Cover any cuts and grazes with a waterproof plaster.

Let cool.

Use a separate towel or use kitchen paper to dry hands, do not use a

Cleaning rigs, hot and dirty washing, rubbish, ready-made or raw food.

Hands must be washed after going to the toilet, sneezing and after

Pre-prepared food.

Microwaves can be used for defrosting and for heating prepared food.

Microwave cookers can be used for defrosting and for heating prepared food.

Cool food at room temperature within an hour after cooking and then chill or freeze.

Choose fresh prepared foods from reputable outlets. Avoid salami, pâté and processed meats in the fridge. Do not eat it.

Ensure food is piping hot when served and cooked all the way through.

Wash can tops before opening them.

Disinfect work surfaces regularly.

Boards and utensils between raw and cooked food.

Avoid cross-contamination of food by changing or washing chopping boards and utensils between raw and cooked food.

Disinfect work surfaces regularly.

Wash fruit and vegetables before eating.

Wash can tops before opening them.

Pre-heat the oven to ensure food is cooked at the recommended temperature.

Cook meat until the juices are clear.

Always follow manufacturers’ guidelines and do not store.

Pre-cooked food in the oven to ensure food is cooked at the

Keep pets away from work surfaces, food and your dishes.

Ensure any cloths or sponges are regularly bleached, disinfected.

Cover any cuts and grazes with a waterproof plaster.

Let cool.

Use a separate towel or use kitchen paper to dry hands, do not use a

Cleaning rigs, hot and dirty washing, rubbish, ready-made or raw food.

Hands must be washed after going to the toilet, sneezing and after

Pre-prepared food.

Microwaves can be used for defrosting and for heating prepared food.

Microwave cookers can be used for defrosting and for heating prepared food.
Specific food safety advice for a neutrophil count of 0.5 - 2.0 x 10^9/litre

This list provides all the relevant examples of high risk foods that should be avoided. Alternatively, you can check with your hospital for their policy regarding foods brought in by visitors and supply of ice cream.

<table>
<thead>
<tr>
<th>Avoid</th>
<th>Alternatives</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soft ripened cheese e.g. Brie, Camembert, goat's cheese, paneer and labnah</td>
<td>Processed cheese e.g. Dairylea, Kraft, Philadelphia, mesh and halloumi</td>
</tr>
<tr>
<td>Blue veined cheese e.g. Danish blue and Stilton</td>
<td>Vacuum-packed pasteurised and hard cheese e.g. cheddar and Edam</td>
</tr>
<tr>
<td>Vacuum-packed pasteurised and hard cheese e.g. cheddar and Edam</td>
<td>Raw or lightly cooked shellfish Well cooked shellfish e.g. prawn curry</td>
</tr>
<tr>
<td>Raw or lightly cooked meat, poultry or fish e.g. meat which is still pink and sushi; smoked items e.g. salmon or Parma ham, salami, caviar and oysters</td>
<td>Well cooked meat, poultry and fish; vacuum-packed cold meats such as turkey and ham; tinned meat and fish</td>
</tr>
<tr>
<td>Raw eggs or undercooked eggs e.g. homemade mayonnaise, homemade ice cream, mousse, egg-nog, meringue and hollandaise sauce</td>
<td>Hard boiled eggs; shop bought mayonnaise, ice cream and other products made with pasteurised egg</td>
</tr>
<tr>
<td>Probiotics, live or bio products e.g. live yoghurts, probiotic containing supplements and drinks</td>
<td>Pasteurised plain, fruit yoghurts e.g. thick and creamy or Greek yoghurts or yoghurt products e.g. lassi</td>
</tr>
<tr>
<td>Paté</td>
<td>Pasteurised paté and paste in tins or jars that do not need to be refrigerated</td>
</tr>
<tr>
<td>All unpasteurised dairy products e.g. unpasteurised cheese such as parmesan or milk sold on local farms</td>
<td>Any pasteurised milk, soya milk, Jersey milk, UHT milk and cheese products</td>
</tr>
</tbody>
</table>

Specific food safety advice for a neutrophil count below 0.5 x 10^9/litre

In addition to the advice on page five for food safety, it is recommended that you also follow the diet for severe neutropenia when your neutrophil count falls below 0.5 x 10^9/litre.

This means that you will have a few further restrictions to consider. Foods to avoid as well as the recommended alternatives are listed below.

<table>
<thead>
<tr>
<th>Avoid</th>
<th>Alternatives</th>
</tr>
</thead>
<tbody>
<tr>
<td>Raw unpeeled fruit or vegetables including salad items, stuffed vine leaves, fatoosh and taboulleh; raw dried fruit, products containing these e.g. muesli, Bombay mix, confectionary; damaged or over-ripe fruit or vegetables; unpasteurised or freshly squeezed fruit or vegetable juice or smoothies</td>
<td>Good quality fruit and vegetables that are well cooked or peeled; UHT or long-life fruit juices – in cartons or jars; pasteurised smoothies; tinned fruit; cooked dried fruit e.g. in fruitcake, flapjacks or cereal bars</td>
</tr>
<tr>
<td>Uncooked herbs, spices and pepper</td>
<td>Cooked herbs, spices and pepper</td>
</tr>
<tr>
<td>Unpasteurised or heat-treated honey</td>
<td>Pasteurised or heat-treated honey</td>
</tr>
<tr>
<td>Non-drinking water, bottled mineral or spring water, water from wells, water from coolers and water fountains</td>
<td>Freshly run tap/filtered/sterilised/carbonated water</td>
</tr>
<tr>
<td>Unpasteurised or “farm fresh” honey and honeycomb</td>
<td>Pasteurised or heat-treated honey</td>
</tr>
<tr>
<td>Unnecessarily large packets of food items from pick and mix, universal jars; deli counter foods e.g. olives, houmus, shawarma and baklava</td>
<td>Ideally, packets should be for personal use only e.g. butter, sweets, pickles, small packets of food, houmus and baklava</td>
</tr>
</tbody>
</table>

Food safety advice for a neutrophil count of 0.5 - 2.0 x 10^9/litre

Advise patients to avoid foods that are at risk of contamination or are high in risk of infections.

In addition to the advice on page five for food safety, it should be avoided and suitable alternatives provided.

**Food safety advice for a neutrophil count below 0.5 x 10^9/litre**

Specific food safety advice for a neutrophil count below 0.5 x 10^9/litre
If you have a poor appetite and are finding it difficult to manage your meals whilst neutropenic, try some of these nourishing snacks:

**Savoury**
- Cheese and crackers
  - Cheese spread or baked beans on toast
    - Jacket potato
    - Tinned spaghetti
    - Fried rice with cooked meat added
    - Rice porridge with cooked meat added
  - Sandwiches – try adding salad cream or mayonnaise
    - Breakfast cereals (at any time)
      - Toasted muffins
      - Meat dumplings/buns
      - Processed cheese such as Dairylea triangles, Kraft cheese slices
      - Roasted nuts, crisps and dips

**Sweet**
- Chocolate or plain, sweet biscuits
  - Sweets and chocolates – individual or bars
    - Thick and creamy style yoghurts and fromage frais
  - Chocolates or plain sweet biscuits

**Liquids**
- Soups – condensed and creamed varieties
  - Milky drinks – Ovaltine, cocoa, Horlicks and hot chocolate
  - Soups (build up to completion of prescribed nutritional supplements such as Fortisip, Ensure Plus, Resource Shake, Proven 1.5 or Freshun)

**Nutritional supplements**
- Fizzy drinks, long-life fruit juices, squashs and cordials
- Milkshakes
- Milky drinks – Ovaltine, cocoa, Horlicks and hot chocolate
- Soups – condensed and creamed varieties

**Energy drink**
- assistant_drink

**For up to 4 hours, refrigerated for up to 24 hours:**

Liquids podem

Nutritional supplements podem

If you are advised by your dietitian or other healthcare professional to take nutritional supplements these can be stored unopened at room temperature. Once opened these drinks should be stored refrigerated.

Nutrient rich, low fat, fruit juices, squashs and cordials cannot be added to these nutritional supplement products.

**Sweet snacks**
- Processed cheese such as Dairylea triangles, Kraft cheese slices
- Meat dumplings/buns
- Toasted muffins
- Breakfast cereals (at any time)
- Sandwiches – try adding salad cream or mayonnaise
- Rice porridge with cooked meat added
- FREE ice with cooked meat added
- Tinned spaghetti
- Cheese spread or baked beans on toast
  - Cheese and crackers
  - Malt loaf or fruitcake
  - Tinned fruit and cream/ice cream
  - Kheer made with pasteurised milk
  - Croissants
  - Peanut brittle
  - Individual or plain sweet biscuits
  - Buttered hot cross buns or sweet waffles with syrup
  - Chocolates or plain sweet biscuits
  - Thick and creamy style yoghurts and fromage frais
  - Sweets and chocolates – individual or bars
  - Chocolates or plain sweet biscuits

**Savoury snacks**
- Cheese and crackers
  - Cheese spread or baked beans on toast
  - Tinned spaghetti
  - Rice porridge with cooked meat added
  - FREE ice with cooked meat added
  - Tinned spaghetti
  - Cheese spread or baked beans on toast
  - Cheese and crackers
  - Malt loaf or fruitcake
  - Tinned fruit and cream/ice cream
  - Kheer made with pasteurised milk
  - Croissants
  - Peanut brittle
  - Individual or plain sweet biscuits
  - Buttered hot cross buns or sweet waffles with syrup
  - Chocolates or plain sweet biscuits
  - Thick and creamy style yoghurts and fromage frais
  - Sweets and chocolates – individual or bars
  - Chocolates or plain sweet biscuits

**Speciality snacks**
- Processed cheese such as Dairylea triangles, Kraft cheese slices
- Meat dumplings/buns
- Toasted muffins
- Breakfast cereals (at any time)
- Sandwiches – try adding salad cream or mayonnaise
- Rice porridge with cooked meat added
- FREE ice with cooked meat added
- Tinned spaghetti
- Cheese spread or baked beans on toast
  - Cheese and crackers
  - Malt loaf or fruitcake
  - Tinned fruit and cream/ice cream
  - Kheer made with pasteurised milk
  - Croissants
  - Peanut brittle
  - Individual or plain sweet biscuits
  - Buttered hot cross buns or sweet waffles with syrup
  - Chocolates or plain sweet biscuits
  - Thick and creamy style yoghurts and fromage frais
  - Sweets and chocolates – individual or bars
  - Chocolates or plain sweet biscuits

**Savoury snacks**
- Cheese and crackers
  - Cheese spread or baked beans on toast
  - Tinned spaghetti
  - Rice porridge with cooked meat added
  - FREE ice with cooked meat added
  - Tinned spaghetti
  - Cheese spread or baked beans on toast
  - Cheese and crackers
  - Malt loaf or fruitcake
  - Tinned fruit and cream/ice cream
  - Kheer made with pasteurised milk
  - Croissants
  - Peanut brittle
  - Individual or plain sweet biscuits
  - Buttered hot cross buns or sweet waffles with syrup
  - Chocolates or plain sweet biscuits
  - Thick and creamy style yoghurts and fromage frais
  - Sweets and chocolates – individual or bars
  - Chocolates or plain sweet biscuits

**Sweet snacks**
- Processed cheese such as Dairylea triangles, Kraft cheese slices
- Meat dumplings/buns
- Toasted muffins
- Breakfast cereals (at any time)
- Sandwiches – try adding salad cream or mayonnaise
- Rice porridge with cooked meat added
- FREE ice with cooked meat added
- Tinned spaghetti
- Cheese spread or baked beans on toast
  - Cheese and crackers
  - Malt loaf or fruitcake
  - Tinned fruit and cream/ice cream
  - Kheer made with pasteurised milk
  - Croissants
  - Peanut brittle