

Introduction to Oncology

Nursing and the Care of the Oncology Patient

Cancer Basics: The Beginning

The following introductory information is directly cited from the NCI web page “What is Cancer” facts sheet. For additional information double click <http://www.cancer.gov> for direct access to additional cancer basics imaging and facts sheets.

What is cancer?

Cancer is a group of many related diseases. All cancers begin in cells, the building blocks that make up tissues. Cancer that arises from organs and solid tissues is called a solid tumor. Cancer that begins in blood cells is called leukemia, multiple myeloma, or lymphoma.

Normally, cells grow and divide to form new cells as the body needs them. When cells grow old and die, new cells take their place. Sometimes this orderly process goes wrong. New cells form when the body does not need them, and old cells do not die when they should.

Cellular replication in normal cells is a direct result of the needs of the organism. Cells transition through a cell cycle. Cyclins are proteins that activate various phases of the cell cycle. Cells will go through checkpoints to “inspect” their work. Differentiation is related to immortality. Remember that differentiated cells have a “biological clock” allowing them a certain number of times that they can divide before they must die. Cancer cells can originate from a single

abnormal (clonal cell) that does not have a “biological clock”. Cancer cells can affect tumor suppressor genes which are proteins that would normally suppress the cell cycle until the “work can be corrected”. After cancer colonies are about 1 mm in diameter they already have a blood supply that allows them to continue to proliferate at a rate that is not compensated for by the rate of cell death (Lowitz & Casciato, 2004).

To review in detail the cell cycle - click the following hyperlink
http://en.wikipedia.org/wiki/cell_cycle

The extra cells form a mass of tissue, called a growth or tumor. Tumors can be either benign or malignant. Benign tumors do not spread to other parts of the body, and they are rarely a threat to life. Malignant tumors can spread and may be life threatening.

What is primary cancer?

Cancer can begin in any organ or tissue of the body. The original tumor is called the primary cancer or primary tumor. It is usually named for the part of the body or the type of cell in which it begins.

What is metastasis and how does it happen?

Metastasis means the spread of cancer. Cancer cells can break away from a primary tumor and enter the blood stream or lymphatic system. When cancer cells spread and form a new tumor in a different organ, the new tumor is a metastatic tumor. The cells in the metastatic tumor came from the original tumor. This means that if breast cancer spreads to the lungs, the metastatic tumor in the lung is made up of cancerous breast cells. In this case, the disease in the lungs is metastatic breast cancer generally look the same as the cancer cells in the breast.

To review the process of metastasis click the following hyperlink
Video of spread <http://www.mayoclinic.com/health/cancer/mm00638>

Where does cancer spread?

Cancer cells can spread to almost any part of the body. Cancer cells frequently spread to lymph nodes near the primary tumor. This is called lymph node involvement or regional disease. Cancer that spreads to other organs or to lymph nodes far from the primary tumor is called metastatic disease. This may be referred to as distant disease.

The most common sites of metastasis from solid tumors are the lungs, bones, liver, and the brain. Some cancers tend to spread to certain parts of the body. Lung cancer often metastasizes to the brain or bones, and colon cancer frequently spreads to the liver. Prostate cancer tends to spread to the bones. Breast cancer commonly spreads to bones, lungs, liver and brain. However, each of these cancers can spread to other parts of the body as well.

Since blood cells travel throughout the body, leukemia, multiple myeloma, and lymphoma cells are usually not localized when the cancer is diagnosed. Tumor cells may be found in the blood, several lymph nodes, or other parts of the body such as the liver or bones. This type of spread is not referred to as metastasis.

Some people with metastatic cancer do not have symptoms. Their metastases are found by x-rays and other tests performed for other reasons. When symptoms of metastatic cancer occur, the type and frequency of the symptoms will depend on the size and location of the metastasis. For example, cancer that spreads to the bones is likely to cause pain and can lead to bone fractures. Cancer that spreads to the brain can cause a variety of symptoms, including headache, seizures, and unsteadiness. Shortness of breath may be a sign of lung involvement. Abdominal swelling or jaundice can indicate that cancer has spread to the liver.

Sometimes a person's primary cancer is discovered only after the metastatic tumor causes symptoms. For example, a man whose prostate cancer has spread to the bones in his pelvis may have lower back pain before he experiences any symptoms from the primary tumor in the prostate.

Metastatic cancers may be found before or at the same time as the primary tumor, or months or years later. When a new tumor is found in a patient who has been treated for cancer in the past, it is more often a metastasis than another primary tumor.

Review of Key Points

- Cancer occurs when cells become abnormal and grow without control
- The place where the cancer started is called the primary cancer or the primary tumor
- Metastatic cancer occurs when cancer cells spread from the place where the cancer started to other parts of the body
- When cancer spreads, the metastatic cancer has the same type of cells and the same name as the primary tumor
- The most common sites of metastasis are the lungs, bones, liver, and brain
- Treatment for metastatic cancer usually depends on the type of cancer as well as the size and location of the metastasis.

Colorectal Cancer

Incidence:

Colorectal cancer (CRC) is the second most common cause of cancer mortality next to lung cancer (Alberts & Goldberg, 2004; Ellenhorn, Cullinane, Coia, & Alberts, 2005). According to statistics quoted from Alberts & Goldberg, 2004, colon cancer rates third in frequency among primary solid tumors in both men *and* women. Nearly 800,000 new cases are diagnosed annually worldwide. Both incidence and mortality have declined since 1985. The risk for colorectal cancer increases with age. Only 3% of colorectal cancers occur in those < 40 years of age. The incidence is 19 per 100,000 for those <65 years and 337 per 100,000 among those > 65 years. It is estimated that 147,500 new cases of colorectal cancer will develop in the United States per year and 57,000 persons will die from this disease. The average person will have a 5% lifetime risk of developing colorectal cancer.

Etiology:

There are multiple factors that begin the transformation of normal colorectal cells to cancer cells. The exact cause of colon and rectal cancers are still unknown. The main risk factors for CRC are age and family history. The relative risk of developing CRC is associated with the following;

- Polyps
- Dysplasia
- Malignant potential of adenomas
- Diet
- Inflammatory bowel disease
- Family history of colorectal cancer (first-degree relative)
- Genetic predisposition (two major syndromes)
- Smoking
- Personal or family history of cancer (breast, endometrial, ovarian)
- Exposure to asbestos (brake mechanics)
- HPV infection of the columnar mucosa

(Alberts & Goldberg, 2004; Ellenhorn et al., 2005; Griffin-Sorbel, 2001; Takimoto & Allegra, 2001)

Polyyps:

According to statistics from Berg, 2001, it is believed that cancers of the colon and rectum begin with the formation of a polyp. Approximately 30% of all people in their 50's will produce polyyps. This number increases to approximately 50% in people in their 70's. Adenomas are classified by their structure as either

pedunculated (stalked or tubular), sessile (flat or villous), or a combination of the two (tubulovillous). Most polyps will not become cancerous (only about 5% do) (Berg, 2001; Alberts & Goldberg, 2004; Takimoto & Allergra, 2001). Polyps may undergo a cellular transformation under the right conditions. A simplified version of the transformation theory is that a polyp will first enlarge. Polyps will then become more dysplastic and later develop into carcinoma insitu. Eventually the dysplastic polyp invades into and/or through the colon wall, and spreads to distant sites (Berg, 2001). Sessile polyps are associated with a higher frequency of malignant transformation. Polyps greater than 2 cm will have up to a 40% likelihood of undergoing a malignant transformation. If a polyp is going to become malignant it can take 5 to 10 years (Alberts & Goldberg, 2004).

Types of Polyps:

Histologically polyps are classified as neoplastic or non-neoplastic. It is important to note that non-neoplastic polyps have no malignant potential. These types include (Alberts & Goldberg, 2004);

- Hyperplastic
- Mucous retention
- Hamartomas (juvenile polyps)
- Lymphoid aggregates
- Inflammatory polyps

Neoplastic polyps (70% adenomatous) have malignant potential and are classified as follows ();

- Tubular (75-85%)
- Tubulovillous (mixed) (10-25%)
- Villous (<5%)

Classifications are based on the presence and volume of villous tissue.

Those polyps considered higher risk have the following features;

- Larger than 1 cm in diameter
- High-grade dysplasia
- Predominantly villous histology

Colonoscopic polypectomy and surveillance can reduce the incidence of colorectal cancer by 90% (Berg, 2001).

Dysplasia:

All polyps are classified as either low or high grade. Six percent of adenomatous polyps are considered high-grade dysplasia and only 5% contain invasive carcinoma at the time of diagnosis (Alberts & Goldberg, 2004).

Recommendations and Management of Polyps:

Newly detected polyps should be excised and additional polyps should be sought through colonoscopy. Colonoscopies have a high accuracy rate of 94% compared to a barium enema which has an accuracy rate of only 64%. A polypectomy can be done at the time of colonoscopy. The chance that multiple polyps will be identified when at least one known polyp has been located is between 40-50% (Berg, 2001). As a result of this incidence, if a polyp is detected on sigmoidoscopy a complete colonoscopy should be preformed.

To view a video on a colonoscopy revealing a cancer clink on the following hyperlink

<http://search.live.com/video/results.aspx?q=colon+polyp+picture+video&first=1&docid=1436058190098#first=1&docid=1436058190098>

Facts About the Role of Diet:

Those who consume a high fat, high caloric and low fiber diet are at increased risk for the development of colorectal cancer. Higher calcium intake and calcium supplementation prove to be protective. The regular use of aspirin (325mg/day) decreases polyps. Increased vitamins A, C, D, and E as well as beta-carotene do not appear to decrease the risk of colorectal polyp formation. It is believed that the higher incidence of rectal and sigmoid cancer in men is due to a higher consumption of alcohol. Postmenopausal women who have taken estrogen replacement therapy appear to have a lower risk for colorectal cancer compared to those who have not. Hormone replacement therapy is not advocated for the prevention of colon cancer since the risks of other serious conditions outweigh the benefits (Christiansen, 2001; Alberts & Goldberg, 2004).

Inflammatory Bowel Disease: A High Risk:

Both ulcerative colitis and Crohn's disease are a great risk for the development of colorectal cancer. According to literature quoted from Alberts & Goldberg in 2004, one percent of colorectal patients have had a history of ulcerative colitis. The risk for developing colorectal cancer varies based on the age of the patient at the onset of colitis as well as the extent of colonic involvement. The duration of active colitis is also a higher risk factor. The cumulative risk is 2% at 10 years, 8% at 20 years, and 18% at 30 years. Those with colitis should be screened with a colonoscopy annually or semiannually in order to determine the need for a proctocolectomy. Those considered for this procedure have had extensive colitis for 8 years or more. For those who are diagnosed with any degree of dysplasia a surgical referral should be made for consideration of a colectomy. Patients who have a diagnosis of Crohn's disease are also at an increased risk for colorectal

cancer but this risk is much lower than those with ulcerative colitis. The relative risk is increased by 1.5 to 2 times.

Genetic Factors:

Fifteen percent of all colorectal cancers occur in patients with a history of a first degree relative that has had a colorectal cancer. These people are more than twice as likely to develop a colorectal cancer (Alberts & Goldberg, 2004; Takimoto & Allegra, 2001; Griffin-Sobel, 2001; Ellenhorn et al., 2005; Berg, 2001). Colorectal cancer develops as a result of a collection of cells undergoing change. This change may occur as a result of a genetic alteration or “accident” which is either present at birth (germ line defect) or due to a change in normal genes caused by age or toxic exposure to substances that travel through the colon (somatic mutations) (Griffin-Sobel, 2001). Colorectal Cancer (CRC) is believed to be caused by a mix of both genetic and environmental factors. Approximately 95% of CRCs are believed to be sporadic. Fifteen to 25% of these sporadic cases will report a positive family history of CRC (Alberts & Goldberg, 2004). The remaining cases are felt to have a genetic factor (common exposure among family members, environmental insult or some combination of both) (Alberts & Goldberg 2004, Griffin-Sobel, 2001).

Inherited:

- Negative family history of CRC = 2% risk
- One first degree relative with CRC = 6% risk
- One first degree relative diagnosed < age 45 = 10% risk
- Two first degree relatives with CRC = 17%

There are two significant genetic syndromes related to CRC:

1. **HNPCC: hereditary nonpolyposis colon cancer** (genetic carrier) 70% (Alberts & Goldberg, 2004; Griffin-Sobal, 2001; Takimoto & Allegra, 2001)
 - More common than Familial adenomatous polyposis (1/500 people)
 - Clinically looks like sporadic CRC
 - Progression is accelerated
 - Screening colonoscopies should be done every 1 to 3 years
 - Recommended treatment is segmental colectomy
 - 30 to 50% will develop a second colon cancer

To view a picture of HNPCC click on the hyperlink
http://www.sabustin.org/page_1147679419025.html

2. **FAP: familial adenomatous polyposis** (genetic carrier) 100% (Alberts & Goldberg, 2004; Griffin-Sobal, 2001; Takimoto & Allegra, 2001)
 - characterized by signs and symptoms of polyps
 - usually pediculated
 - not usually present at birth
 - develop early in life (usually median age, 25 years)
 - spread through out the colon
 - develop symptoms by the age of 33 years

To view images and additional text information on polyps, FNP and HNPCC click on the following hyperlink

http://bhj.org/journal/2000_4202_apr00/sp_276.htm

Smoking:

Both genders who have smoked during the previous 20 years have three times the relative risk for small adenomas but not larger ones. Smoking for greater than 20 years was associated with 2.5 times the relative risk for larger adenomas. 5,000-7,000 colorectal cancer deaths in the United States are attributed to cigarette use (Alberts & Goldberg, 2004).

Clinical Facts about the Natural History of Colorectal Cancer:

(Alberts & Goldberg, 2004; Ellerhorn et al., 2005; Griggin-Sorbel, 2001; Takimoto & Allegra, 2001; Berg, 2001; Christiansen, 2001)

- 98% of colorectal cancers above the anal verge are adenocarcinomas
- cancers of the anal verge are often squamous cell or basaloid carcinomas
- carcinoid tumors cluster around the rectum and cecum and spare the rest of the colon
- 2/3 of colorectal cancers develop in the rectum
- Rectal tumors are detected by digital rectal examination in 75% of the cases
- Nearly 3% of colorectal adenocarcinomas are multicentric
- 2% of patients develop a second primary tumor in the colon
- The common clinical signs and symptoms relate to the size and location of the tumor
- Right-sided colonic lesions often result in dull and ill-defined abdominal pain, bleeding and anemia
- Remember that anemia is not a condition but merely a symptom which causes weakness, fatigue, and SOB
- Right sided tumors are more likely to result in anemia than obstruction

- Left sided tumors commonly lead to changes in bowel habits, bleeding, gas pain, decrease in stool caliber, constipation, increased use of laxatives, and colonic obstruction
- Metastasis to the regional lymph nodes are found in 40% to 70% of cases
- Venous invasion is found in up to 60% of cases
- Metastasis occurs most frequently in the liver, peritoneal cavity, and lung
- Metastasis is rarely to the brain
- Rectal cancers are 3 times more likely to recur locally
- Local recurrence of rectal cancers is mostly due to the anatomic restrictions of the rectum which prevent a wide excision. The rectum also lacks an outer serosal layer.
- Venous and lymphatic drainage of the rectum is directly into the inferior vena cava
- Venous drainage of the colon occurs into the portal vein and variable lymphatic drainage
- Rectal cancer often recurs first in the lungs
- Colon cancer more frequently recurs in the liver

Diagnostic Studies:

After a clinical diagnosis is made, pathologic confirmation is required. Various studies are performed to determine the extent of the disease (refer to staging module). Prognostic factors (tumor makers, genetic analysis, special histochemical staining etc.) are also considered prior to making a decision about the best treatment approach for the patient.

Biopsy Confirmation: if an obstructing lesion is present and the patient cannot undergo direct tissue sampling than cytology may be obtained (brush technique to obtain cells) (Alberts & Goldberg, 2004).

General Physical Evaluation: complete examination with digital rectal examination, CBC, LFT, and chest radiography (Alberts & Goldberg, 2004; Sweed, 2001).

Carcinoembryonic antigen (CEA): this tumor marker is favored by most physicians as a means to identify early recurrence. It is most helpful if a pre-operative CEA is obtained and used as a prognostic indicator when determining if the primary tumor is associated with CEA elevation (Alberts & Goldberg, 2004; Ellerhorn et al., 2005; Griffin-Sorbel, 2001; Takimoto & Allegra, 2001; Berg, 2001; Christiansen, 2001). Metastatic tumor cells are most likely to result in CEA elevations. Remember if these levels are not elevated to begin with they cannot be considered a reliable marker that would indicate recurrent or progressive

disease. CEA are most helpful to identify hepatic recurrences (liver metastasis). An elevated CEA should prompt further testing with diagnostic scans. High pre-operative CEA levels usually revert to normal in 6 weeks post-operative after a complete resection.

CT or magnetic resonance imaging (MRI): (with contrast) of the abdomen and pelvis and may identify liver or intraperitoneal metastases.

Endoscopy or Barium Enema: indicated to assess the entire colonic mucosa (3% will have synchronous colorectal cancers and a larger number will have additional premalignant polyps (Alberts & Goldberg, 2004; Ellerhorn et al., 2005; Griffin-Sorbel, 2001; Takimoto & Allegra, 2001; Berg, 2001; Christiansen, 2001; Sweed, 2001)

EUS (Endoscopic Ultrasound): this significantly improves the preoperative assessment of the depth of invasion of large bowel tumors, especially rectal tumors. The accuracy rate is 95% for EUS and 70% for CT, and 60% for digital rectal exams (Alberts & Goldberg, 2004). In rectal cancer the combination of EUS, to assess tumor extent, and digital rectal examination, to determine mobility, should enable both precise planning of surgery as well as identify patients who may benefit from preoperative chemoradiation. Transrectal biopsy of perirectal lymph nodes can often be accomplished under ESU (Alberts & Goldberg, 2004).

Biological Markers:

1. CEA = cell-surface glycoprotein which is shed into the blood and is the best known serological marker for monitoring colorectal cancer disease status and for detecting recurrence (**note if this tumor marker is elevated pre-operatively. If it is, then it is considered a sensitive marker to monitor post-operatively. Sometimes you will see a low CEA even in the presence of colon cancer**). (Alberts & Goldberg, 2004; Ellerhorn et al., 2005; Griffin-Sorbel, 2001; Takimoto & Allegra, 2001; Christiansen, 2001)

2. Other markers that are new but of interest are (do not expect to routinely see these but you may in the near future):

- CA 19.9 (used as a complement to CEA)
- Monoclonal antibodies (anti-CEA, anti-TAG-72)
- Aneuploidy (presence of an abnormal number of chromosomes in the tumor cells

(Alberts & Goldberg, 2004; Ellerhorn et al., 2005; Griffin-Sorbel, 2001; Takimoto & Allegra, 2001; Christiansen, 2001)

Tumor markers and staging will be discussed in more detail in the preceding modules.

Management:

Surgery is the only universally accepted potentially curative treatment for colorectal cancer (Alberts & Goldberg, 2004; Ellerhorn et al., 2005; Griffin-Sorbel, 2001; Takimoto & Allegra, 2001; Christiansen, 2001). Curative surgery should excise the entire tumor with wide margins and maximize regional lymphadenectomy while preserving function when ever possible. Adjuvant chemotherapy (therapy after surgery) is indicated for those tumors that are stage III or greater (Alberts & Goldberg, 2004). It is currently controversial to give chemotherapy to patient's adjuvant with a high grade stage II tumor (Alberts & Goldberg, 2004). The determination for chemotherapy in this setting will be left to the discretion of the oncologist.

The most common chemotherapy drugs used to treat colorectal cancer are as follows (Alberts & Goldberg, 2004; Ellerhorn et al., 2005; Griffin-Sorbel, 2001; Takimoto & Allegra, 2001; Christiansen, 2001; NCCN Practice Guidelines <http://www.nccn.org>; Wilkes, 2001):

Most Common Agents:

1. 5-FU
2. capecitabine
3. floxuridine (FUDR for intrahepatic infusion)
4. irinotecan
5. oxaliplatin

Most Common Combinations:

- **5-FU/Leucovorin** (combination increases the efficacy)
- irinotecan (single agent or with 5-FU/Leucovorin) = **IFL**
- **FOLFIRI** (irinotecan, leucovorin, and continuous infusion 5-FU)
- **FOLFOX** (5-FU loading dose and 24 hour infusion, oxaliplatin)
- **FUFOX** (5-FU given in high dose as a 24 hour infusion, leucovorin and oxaliplatin)
- **IROX** (irinotecan and oxaliplatin)

Adjuvant therapies for rectal cancers are often required. It is commonly difficult to do a complete resection because of the anatomical confines of the pelvic bones and sacrum (Alberts & Goldberg, 2004). Surgeons often cannot achieve a wide-resection margin for rectal cancers. As a result, almost half of the recurrences occur in the pelvis (Alberts & Goldberg, 2004). For this group of individuals the most effective adjuvant therapy employs combined 5-FU chemotherapy and XRT to reduce the local recurrence rates (Alberts & Goldberg, 2004).

For ongoing updates and practice guideline information related to diagnosis and therapy click on the hyperlink <http://www.NCCN.org> and go to clinical practice guidelines in oncology.

Surgery (Alberts & Goldberg, 2004):

- 85% of patients diagnosed with colorectal cancer can undergo surgical resection
- incurable cancers may benefit from palliative resection to prevent obstruction, perforation, bleeding, and invasion of adjacent structures
- May be able to avoid by using colonic stents and laser ablation of the intraluminal tumors when the cancer is metastatic.

XRT (Alberts & Goldberg, 2004):

- may be the primary and only treatment modality for small, mobile, rectal tumors
- may be used in combination after resection of rectal tumors
- may be used in a palliative setting to relieve pain, obstruction, bleeding and tenesmus

Hepatic Arterial Infusion (Alberts & Goldberg, 2004):

Metastases in the liver derive their blood supply primarily from the hepatic artery. Hepatocytes derive blood primarily from the hepatic vein. FUDR is instilled into the hepatic artery. Possible complications with this procedure are;

- variable anatomy (may be difficult to insert)
- catheter migration
- biliary sclerosis
- gastric ulcerations

- progression of extra-hepatic disease is a common pattern of failure

Radiofrequency Ablation (Alberts & Goldberg, 2004):

Radiofrequency ablation is commonly referred to as RFA. RFA is a localized thermal technique that is designed to destroy tumor tissue by heating the tumor tissue to a temperature that exceeds 60 degrees Celsius. Necrosis occurs in the site of treatment. This procedure is often performed on patients with unresectable liver cancer or liver metastasis but may also be used to palliate a patient who may be experiencing pain. The interventional radiologist performs this procedure.

For additional information and imaging on hepatic arterial infusion and RFA click on the following hyperlink

<http://www.cpmc.org/advanced/liver/news/newsletter/newsletter-vol8.html>

Chemoembolization (Alberts & Goldberg, 2004):

Chemoembolization is a local regional approach used as a potential treatment option. Since a majority of tumors receive its blood supply from the portal vein, a catheter is placed into the hepatic artery and chemotherapy is administered into the hepatic artery. This type of therapy is used to treat hyper vascular tumors such as hepatocellular carcinomas and neuroendocrine tumors rather than colorectal metastases. Colorectal cancers are often hypo-vascular tumors that have not demonstrated a benefit from this procedure. May be used in the setting of metastatic disease in order to delivery high concentrations of chemotherapy to a specific area.

For additional information on preparing a patient for chemoembolization click on the hyperlink

<http://www.radiologyinfo.org/en/info.cfm?pg=chemoembol&bhcp1>

Nursing Implications (Hogan, 2001; Wilkes, 2005;)

- Assess the patient's **knowledge** of colon or rectal cancer
- Assess the patient's **understanding of treatment** options
- Educate and document the patient and family members about the **treatment regimen's purpose and schedule**, common **side effects** of the prescribed treatment, and **self care measures** to manage side effects

- Review with the patient and family when to **call** for medical/nursing advice
- Assess the likelihood of **nausea and vomiting** based on emetogenic potential of the regimen
- Identify **risk factors** for **nausea/vomiting** such as; female gender, younger age, chronic alcohol intake, history of motion sickness
- Identify the **underlying cause** of the symptom such as treatment induced constipation, opioid analgesia, hypercalcemia, or bowel obstruction, and initiate corrective measures
- Administer **appropriate antiemetic** prior to therapy and educate about regular administration thereafter.
- Consider the **level of emetogenic potential** of chemotherapy and educate patients to take prn meds according to the risk for developing nausea and vomiting
- Educate patients to keep a **daily log or diary** of side effects of therapy and pain level. This will enable the practitioner to make adjustments for future therapy if toxicity or intolerance develops. It will also provide a guide to enable the practitioner to make appropriate analgesia adjustments to ensure pain relief.
- Incorporate **nonpharmacologic interventions** (acupressure, relaxation techniques, guided imagery)
- Educate patient about potential **dietary modifications**; small bland meals, avoid greasy, fatty or rich foods or those that have an unpleasant odor or taste, cool or cold foods are often better tolerated, change to a clear liquid diet if experiencing vomiting or excessive diarrhea.
- Perform a baseline **oral exam** and educate regarding good oral hygiene (soft bristle tooth brush, saline/salt rinses QID).
- Evaluate the **mucosa for stomatitis** (ulcerations, sores) , yeast or bleeding.
- Educate patients to regularly perform an oral examination
- Avoid **oral irritants**; tobacco, alcohol, citrus fruits, and juices, spicy foods and commercial mouthwashes.
- Discuss the need for delay or dose modification if necessary
- Assess a patient's baseline **dietary intake** and **concomitant medications** , including any herbal or complimentary medications
- Note baseline GI abnormalities and identify and document new findings as such

- Evaluate the patient's risk for developing **taste changes**, anorexia, or cachexia; older age, alcoholism, history of smoking, anorexia, concomitant medications that cause diarrhea, nausea, vomiting, and taste alterations, malabsorption disorders, difficulty swallowing
- Assess **social situation** that may complicate patients care
- Monitor the patient's **weight** and report losses of excess of 5 lbs. Always consider that any sudden loss may impact the patients BSA and therefore the dosage of chemotherapy. Sudden weight loss may also signal malnutrition or potential dehydration
- If complaints of "**bad taste**" encourage patient to brush teeth prior to eating, experiment with seasonings to make food more palatable, recommend mints, hard candy, or cough drops to temporarily relieve symptom
- Assess the patient for **nausea prior to mealtime**. If present administer antiemetics before meals
- Encourage strategies to increase the **caloric and nutritional** value of oral intake: eat small meals, eat foods high in protein and calories (eggs, peanut butter, cheese, chicken, and milkshakes), limit liquids during meals, and maximize intake when feeling the best.
- Evaluate and offer liquid food supplements if needed
- Initiate **appetite stimulants** as needed; corticosteroids, megestrol acetate, hydrazine sulfate, metoclopramide, and dronabinol as ordered
- Instruct patient to avoid storing food in metal containers, and if taste changes are severe, to consider using plastic utensils instead of metal ones
- Assess baseline **bowel habits** and concomitant medication use
- Educate the patient about ways to prevent constipation
- Increase fiber intake, whole-grain foods, legumes, fresh foods, and raw vegetables, unless the patient is at risk for a narrow colon lumen
- Increase **fluid intake** to 2 to 3 quarts of liquids per day; juices, water, hot fluids.
- Follow a **prescribed bowel regimen**. Goal: one bowel movement every three days
- With **complaints of constipation** assess bowel sounds, rule out fecal impaction, advancement of bowel obstruction
- Treat constipation with laxatives, bowel stimulants, enemas (not recommended with low WBC or Platelet counts).

- Teach patient to dose and schedule **antidiarrheal** medications, assess that patient is taking medication as recommended. Record a 24 hour report of frequency of stools , onset, number of episodes, duration, description of stools, symptoms of dehydration (dizziness; orthostatic vs., dry mouth, mental status changes)
- Encourage and support patients to alter antidiarrheal medications as needed
- **Foods and liquids** to encourage during **episodes of diarrhea**;
 1. Bland, easily digested foods, BRAT diet (bananas, white rice, apple sauce, toast; gelatins, yogurt, sherbet; cooked foods at room temperature.
 2. small frequent meals
 3. boiled or baked potatoes, pasta, skinless baked chicken, and crackers: increase these types of foods once diarrhea resolves
- **Avoid:** foods that are raw or high in fiber or roughage, rich, heavily seasoned, greasy or very hot, milk products, alcohol, caffeinated beverages
- Teach the patient about perineal care
- Diarrhea may lead to a treatment delay or dose modification
- Monitor the patient and document ascites or jaundice and report
- Assess for signs and symptoms of **bowel obstruction** related to advancing disease (nausea, sporadic vomiting, abdominal pain, worsening constipation, and lack of bowel sounds)
- Consider and assess for **urethral obstruction** due to advancing disease (oliguria and elevated serum creatinine)
- Monitor the **patients bilirubin** levels and report abnormalities
- If **pruritis** due to advancing disease , provide symptomatic relief such as; applying topical anesthetics or corticosteroid preparations, cholestyramine.
- **Assess pain:** use pain rating scale, assess the patients pain by asking about; onset, location, duration, characteristics, aggravating/alleviating, rate, timing
- Include a 24 hour recall of all new pain episodes or complaints of uncontrolled pain
- Calculate the number of short acting doses of analgesia required in a 24 hour period.
- Reinforce the proper use of both short acting and long acting analgesia

- Educate patient and family about the potential **side effects of analgesia** (constipation, dry mouth, drowsiness, nausea and vomiting, sedation, and respiratory depression (not cessation) and self care measures to treat
- Utilize **nonpharmacologic methods to reduce pain**: heat and cold, rest, music, humor, hypnosis, acupuncture, progressive muscle relaxation, guided imagery, and distraction
- Always evaluate and document the effectiveness of care
- Evaluate the risk of **DVT**; colon cancer, sedentary lifestyle, history of coagulation problems or prior DVT, injury or trauma to vasculature or extremity, treatment used for malignancy, history of cigarette smoking
- Assess for chest pain or SOB (usually sudden onset that may be severe) rule out **pulmonary emboli**. If in question MD may do a D-dimer and if elevated CT angio. You cannot see a PE on CXR but one may be done first to *rule out* pneumonia or pleural effusion.
- Assess the calf for signs of **thrombosis**; warmth, pain, redness, unilateral swelling or edema, positive Homans' sign (pain with dorsi flexion)
- Encourage regular **exercise** and ambulation to prevent stasis
- Educate patient about **anticoagulation therapy**; drugs, therapeutic window, labs, anticipated length of therapy
- Teach self injection of **low-molecular weight heparin** if ordered
- Evaluate fear and anxiety with each interaction
- Encourage emotional support and refer to social services when necessary
- Educate patient about expected **hair loss** (usually 10-21 days after the initiation of therapy)
- Reinforce that hair loss is temporary and may involve thinning of body hair as well.
- Avoid excess or harsh hair treatments; hair dyes, bleach, permanent solutions, electric hair dryers, curling irons, elastics, frequent shampoos, and excess hair manipulations
- Head coverings should be worn all year round (protect skin and prevent excess heat loss)
- Assess the etiologies of **fatigue**; anemia, pain, emotional distress, insomnia, nutritional alterations, therapeutic regimen, presence of advanced disease
- Prescribe fatigue treatment based on the cause

- Teach the patient to rest as needed throughout the day, yet maintain a normal sleeping pattern at night.
- Encourage an exercise program
- Eat foods high in iron; liver, eggs, carrots, raisins
- Advise patient to; pace activities, avoid overexertion, schedule important activities at times of maximal energy, delegate activities, create strategies to overcome “energy drainers”
- Encourage patient and significant other to discuss concerns and fears related to **altered sexual function** and desire and discuss; create a sensual environment, explore alternative forms of sexual expression, vaginal lubricants, counseling if appropriate
- Educate the patient about hand-foot syndrome, be able to recognize this side effect and discuss management of this condition.

Click on the following links below to obtain more information and pictures on hand-foot (palmar-plantar erythrodysesthesia) syndrome.

<http://www.caring4cancer.com/go/cancer/effects/lesscommon/handfoot-syndrome.htm>

<http://www.ons.org/publications/journals/CJON/volume9/issue1/pdf/0901103.pdf>