

Oncologic Emergencies

Cancer involves multiple tissues in a wide range of locations. There are metabolic diversities among various tumors as well as metastatic potentials that can result in a variety of complications. Most patients will die from metastatic complications of the disease and related complications that may occur at any time. The overall goal is to prevent, reverse, or minimize life-threatening complications through prophylaxis, early detection, and effective management (Henke Yarbro, Hansen Frogge, Goodman, 2002).

Critical Thinking!

Management Factors in the Evaluation and Treatment of an Oncologic Emergency (Henke Yarbro, Hansen Frogge, Goodman, 2002).

Signs and Symptoms:

1. Are the *Signs and symptoms* caused by the tumor?
- 2 How quickly are the *Signs and symptoms* progressing?

Natural history of the Primary Tumor:

1. Is there a previous diagnosis of malignancy?
2. What is the disease-free interval between the diagnosis of the primary tumor and the onset of the emergency?
3. Has the emergency developed in the setting of terminal disease?
- 4.

Efficacy of Available Treatment:

1. No prior therapy versus extensive pretreatment
2. Should treatment be directed at the underlying malignancy and/or the urgent complication?
3. Will the patient's general medical condition influence the ability to administer effective treatment?

Treatment and Goals:

1. Potential Cure?
2. Is prompt palliation required to prevent further debilitation?
3. What is the relative risk versus the benefit ratio?
4. Should treatment be withheld if the patient is terminal with limited chance of response to antitumor therapies?

Most Common Oncologic Emergencies:

- DIC
- Hypercalcemia
- Malignant pleural effusion
- Cardiac tamponade
- Septic shock
- Spinal cord compression
- Superior vena cava syndrome
- SIADH
- Tumor lysis syndrome

Two Key Concepts when caring for people with cancer:

- Identify patients at risk for complications and assess with each interaction
- Educate the family regarding risk and how to assess and report with the onset of complications

Disseminated Intravascular Coagulation: (Escalante, Manzullo & Weiss, 2007; Henke Yarbro, Hansen Frogge, Goodman, 2002; Hopper Friend & Pruett, 2001).

Scope of the problem:

Disseminated intravascular coagulation (DIC) represents an inappropriate and exaggerated over-stimulation of normal coagulation. Both thrombosis and hemorrhage may occur simultaneously making it difficult to treat.

Approximately 10% of persons with cancer will experience this complication. It is almost always secondary to an underlying disease process or malignancy

Risk Factors:

- Infection or sepsis (most common) may be bacterial, fungal, and viral. Sepsis from **gram-negative bacteria** is the most common.
- Intravascular hemorrhage (blood transfusion reaction)
- **Acute leukemia** and **adenocarcinomas** (lung, breast, stomach, and prostate)
- Liver disease that results in liver failure
- Prosthetic devices (shunts)
- Non-cancer causes; pancreatitis, heat stroke, trauma, burns, **drug or transfusion reaction, anaphylaxis**

Pathophysiology:

Once the coagulation system is activated, thrombin, the main proteolytic enzyme of blood coagulation is produced.

Steps in the coagulation process:

1. fibrinogen is consumed by thrombin-induced clotting and plasmin-induced fibrinolysis
2. decreased macrophage-clearing limits removal of activated clotting factors
3. rapid coagulation exceeds the ability of the liver to clear fibrin split products (FSPs) and excess FSPs inhibit clotting
4. consumable factors I, II, V, and VIII are depleted by microvascular clotting

Things to Consider: ((Henke Yarbro, Hansen Frogge, Goodman, 2002; Escalante, Manzullo & Weiss, 2007).

- Acute DIC is characterized by microvascular thrombosis and bleeding from multiple sites occurring simultaneously.
- Often is fatal
- May go unrecognized until severe hemorrhage (hint: DIC panels)
- Look at labs!!
CBC (decreased Hgb, Hct, WBC are variable)
Red Cell smear (evidence of microangiopathic hemolytic anemia)
Thrombocytopenia
Decreased fibrinogen (remember it **gets used up quickly, less than 150mg/dl**)
Increased FSPs (**earliest sign***** equal to or > 40ug/ml)
Increased thrombin time (increased by 3 or more seconds)
Activated partial thromboplastin time (by 5 or more seconds)

Signs and symptoms:

- Anxiety, restlessness
- Tachycardia, tachypnea, and headaches
- Conjunctival hemorrhage and periorbital petechie
- Oozing blood
- Bleeding gums

Late changes:

- Changes in mentation
- Frank hematuria
- Joint pain
- Hemoptysis
- Tarry stools, or melena

Treatment: (Henke Yarbro, Hansen Frogge, Goodman, 2002; Escalante, Manzullo & Weiss, 2007).

- Platelets, FFP
- Antithrombin III concentrate to neutralize excess thrombin and slow DIC
- Controversial heparin therapy as continuous infusion or SQ
- Heparin contraindicated if intracranial bleeding, open wounds or recent surgery
- E-aminocaproic acid (controversial to maintain platelet and fibrinogen levels)
- Effective control is measured by normal coagulation screen and platelet count

Nursing Monitoring: (Henke Yarbro, Hansen Frogge, Goodman, 2002).

- Vital signs, urine output, blood loss
- Suppress cough, vomiting (meds) to prevent increased ICP
- Monitor for 1). cardiogenic shock, 2). hypovolemia, 3). hypoxia, 4). hypotension, 5). oliguria

The following two links will guide you through case studies on DIC. Click on the following links to participate:

<http://faculty.alverno.edu/bowneps/MSN621/student%20tutorials/tutorial%20final%20DIC.ppt>

<http://www.ons.org/publications/journals/CJON/Volume7/Issue4/0704479.asp>

Cardiac Tamponade: (Grannis, Cullinane & Lai, 2007; Henke Yarbro, Hansen Frogge, Goodman, 2002).

Scope of the Problem:

cardiac tamponade is a life threatening oncologic emergency. This situation occurs from an excess accumulation of fluid in the pericardial sac (pericardial effusion). This fluid causes an increase in pressure around the heart and a decrease in blood flow to the heart.

Risk Factors:

1. malignancy (most common cause; lung 40%, breast 23%, lymphoma 11%, leukemia 5%)
2. constriction of the pericardium from tumor (**Hint:** where is the tumor located? Review radiology reports ex. Hilar masses, left lung lobe masses)

Physiological Alterations:

- Amount of fluid surrounding the heart varies and may range from 50ml – 1 liter.
- As excess fluid accumulates it compresses the right ventricle and therefore it is unable to fill
- The amount of blood leaving the left side of the heart is less
- Severity is based on how rapidly and the amount of fluid that is accumulating

Signs and symptoms

- Variable depending on the rate and amount of fluid that is accumulating
- Must also consider baseline cardiac function
- Look for signs associated with;
 - decreased cardiac output
 - compression of the heart by other structures (cough, retrosternal chest pain, dyspnea)
 - venous congestion,
 - weak heart sounds
- chest fullness and discomfort

Hint: look to diagnostic tests to identify

- echocardiography (**most sensitive**)
- CXR
- CT
- MRI
- Serum labs (Hct, arterial blood gases may reveal bleeding or respiratory alkalosis)

The following is a case study review of cardiac tamponade with illustrations of imaging and EKG readings.

<https://www.thoracic.org/sections/clinical-information/critical-care/critical-care-cases/cases/cardiac-tamponade.html>

Treatment: (Grannis, Cullinane & Lai, 2007; Henke Yarbro, Hansen Frogge, Goodman, 2002).

- Pericardiocentesis with possible sclerosing
<http://en.wikipedia.org/wiki/Pericardiocentesis>
- Pericardiotomy
- Pleuropericardial window
- Pericariectomy <http://en.wikipedia.org/wiki/Pericardectomy>

Nursing: (Henke Yarbro, Hansen Frogge, Goodman, 2002).

- Assess cardiac output
- Look at ordered EKG
- Administer aggressive fluid therapy (hypovolemia)
- Oxygen
- Evaluate perfusion
- Look at ABG's (acid-base imbalances?)
- Potential for infection r/t procedures (look at labs; WBC's, blood cultures, other signs of infection?)
- Antibiotic therapy
- Sterile dressing changes

The following hyperlink will connect with an overview and case study of the assessment and nursing management of cardiac tamponade. Click on the following link:

<http://www.ons.org/publications/journals/ONF/Volume30/Issue2/3002224.asp>

Malignant Pleural Effusion: (Grannis, Cullinane & Lai, 2007; Henke Yarbro, Hansen Frogge, Goodman, 2002; Prommer & Casciato, 2004).

Scope of the problem:

Occurs when a malignant process prevents reabsorption. Fluid accumulates in the intra-pleural space. A pleural effusion may be the initial sign of cancer or it may signal advanced disease. Typically, malignant effusions recur unless the underlying disease is controlled or cured. Approximately 50% of cancer patients will develop a malignant pleural effusion during the course of their disease. One-third of all patients will have a bilateral pleural effusion.

Occurs most commonly with: (Henke Yarbro, Hansen Frogge, Goodman, 2002; Prommer & Casciato, 2004; Grannis, Cullinane & Lai, 2007).

- lung
- breast
- adenocarcinoma, unknown primary
- leukemia
- lymphoma
- reproductive tumors
- GI tumors
- Genitourinary tumors (renal cell, prostate)

Common causes:

- Implantation on the pleural surface leading to increased permeability
- Obstruction of lymphatic flow by tumor preventing fluid reabsorption
- Tumor obstruction of primary vessels leading to increased capillary hydrostatic pressure.
- Necrotic tumor cells shed into the pleural space increasing colloid osmotic pressure
- Thoracic duct perforation

Signs and symptoms:

- Dyspnea
- Orthopnea
- Dry, non-productive cough
- Chest pain, heaviness
- Tachypnea
- Dullness to percussion
- Restricted chest wall expansion
- Impaired transmission in breath sounds

Hint: look at the following ordered tests for clues

- CXR
- Examination of pleural fluid
- CT
- Note: grossly **bloody** effusion is the **strongest indicator of malignancy**. Laboratory analysis is the only way to definitively diagnose the effusion as malignant

Treatment: (Henke Yarbro, Hansen Frogge, Goodman, 2002; Grannis, Cullinane & Lai, 2007; Prommer & Casciato, 2004).

- Small asymptomatic effusions may be left alone (**remember:** most recur anyway)
- Chemotherapy etc. for a particular tumor (response to treatment will often see a reduction or resolution of the effusion)
- If the tumor is chemo-resistant or refractory to systemic treatment, pleurodesis may be performed.
- Thoracentesis provides short-term relief of symptoms
- Thoracostomy tube
- Pleuroperitoneal shunt
- Pleural stripping
- External beam XRT

For a case study review and imaging on malignant pleural effusion click on the following link:

http://rad.usuhs.mil/medpix/tf_case.html?mode=pt&pt_id=4063#top

Hypercalcemia: (Messman & Monahan, 2001; Escalante, Manzullo & Weiss, 2007; Henke Yarbro, Hansen Frogge, Goodman, 2002; Carlson, 2004).

Scope of Problem: Hypercalcemia is the most common metabolic problem or metabolic complication of malignancy. It can be life threatening. Approximately 10-40% of patients with cancer will experience this problem as a late complication of cancer. Cancer-induced hypercalcemia most often develops rapidly, requiring prompt diagnosis and treatment. Hypercalcemia of malignancy occurs when more calcium is reabsorbed from bone than is deposited in bone. There are two primary causes;

1. **Humoral hypercalcemia of malignancy** (80% of cases. May or may not have bone metastasis but have tumors that secrete hormones and cytokines that cause calcium resorption from bone and hypercalcemia)
2. **Local Osteolytic Hyperclacemia** (occurs primarily in patients with extensive osteolytic bone metastasis. Tumor cells do not resorb bone but produce factors that induce local bone absorption, osteolysis, and hypercalcemia. Local production of parathyroid hormone-related peptide by tumor cells enhance tumor growth in the bone)

The majority (99%) of total calcium is bound in bones and teeth. Normally only 1% of calcium is found in the serum and the majority is **ionized calcium** which is filtered in the kidney. The kidney regulates and maintains serum calcium levels at a normal range;

Women: **8.9-10.2** mg/dl

Men: **9.0 - 10.3** mg/dl

Hypercalcemia is diagnosed when the serum calcium exceeds **11.0** mg/dl

Key: a mathematical calculation must be performed to identify the true estimate of ionized serum calcium. The following formula corrects for changes in serum albumin concentrations (low levels of albumin will give you a distorted calcium value). You should allow for 0.8 mg/dl for each g/dl change in serum albumin.

Corrected Serum Calcium = measured total serum calcium value (mg/dl) + (4.0- serum albumin value (g/dl)) x 0.8

Individuals who develop hypercalcemia may be asymptomatic or have a number of signs and symptoms that may be misinterpreted as terminal cancer or side effects of treatment. Note that there is often little relation between symptoms and calcium level.

Signs and symptoms: (Henke Yarbro, Hansen Frogge, Goodman, 2002).

Central Neurological:

- Impaired concentration
- Confusion
- Apathy
- Drowsiness or lethargy
- Late: obtundation, coma

Peripheral Neuromuscular:

- Muscle weakness
- Hypotonia
- Decreased or absent deep-tendon reflexes

Gastrointestinal:

- Increased gastric acid secretion
- Nausea, vomiting, and anorexia
- Constipation
-

Renal:

- Polyuria
- Polydipsia
- Dehydration (thirst, dry mucosa, decreased or absent perspiration, poor skin turgor, concentrated urine)

Cardiovascular:

- Slow cardiac conduction (prolonged P-R interval, widened QRS, shortened QT, ST intervals)
- Bradycardia (with rapid increases)

- Bradyarrhythmias can progress to bundle branch block, atrioventricular block with complete heart block

Assessment and Grading: (Escalante, Manzullo & Weiss, 2007; Henke Yarbro, Hansen Frogge, Goodman, 2002).

The diagnostic work-up for a patient suspected of having hypercalcemia includes a history and physical, laboratory tests.

Graded as follows:

- Mild = >10.3 to <12 mg/dl
- Moderate = 12 to 14 mg/dl
- Severe = 14-16 mg/dl
- Life-threatening = 14 to 16 mg/dl

Note: all values assume a normal serum albumin. When there is less serum albumin there is less albumin to bind with calcium.

Treatment:

The goal of therapy is to:

- correct dehydration
- increase renal excretion of calcium with vigorous saline diuresis
- inhibit calcium resorption from the bone with antiresorptive agents
- treat the underlying malignancy

Saline Diuresis:

- administration of oral or intravenous saline containing fluids (usually IV)
- the rate of NS is based on the severity of the hypercalcemia, severity of hypercalcemia, severity of dehydration, and cardiovascular tolerance
- four to five liters of NS over 24 hours results in modest decreases in serum calcium
- a loop diuretic such as lasix (furosamide) may be added after fluid balance has been restored

The following is an interactive case study on hypercalcemia. Click on the following link to participate: **NOTE:** you must click on the pink ex. **T1, T2** etc. for additional information

http://www.labmedicine.com/2003/Issue_06/1000895.html

Pharmacological Intervention: (Henke Yarbro, Hansen Frogge, Goodman, 2002; Escalante, Manzullo & Weiss, 2007).

Bisphosphonates: (etidronate, zolendronate, pamidronate)

- prevents precipitation of calcium phosphate
- zolendronate is usually the drug of choice but pamidronate may also be used (both are intravenous)
- serum creatinine must be closely monitored when using zolendronate and dose adjustments are based on a nomogram (calculation based on Ht, wt, age, and serum creatinine)
- calcitonin may be combined with one of the above if the patient's serum creatinine is $> 1.3 \text{ mg/dl}$
- a corticosteroid may be added to enhance and prolong the effects of the calcitonin

Infection: (Ito, 2001; Wujcik, 2001; Henke Yarbro, Hansen Frogge, Goodman, 2002).

Scope of the Problem:

Infections are responsible for approximately 50% to 75% of cancer deaths. These infections may result from a malignancy, intensive therapy, hospitalizations, or a combination of these factors. An **immunocompromised** host refers to someone who has one or more defects in their natural defense mechanisms. These defects may predispose them to severe and sometimes life-threatening infections.

Causes:

- disruption in protective barriers (skin, mucous membranes)
- changes in the normal flora (due to antibiotic usage)
- obstruction due to tumor
- **neutropenia** (neutrophil count $< 1,000 \text{ cells/mm}^3$ or $< 500 \text{ cells/mm}^3$ (more significant risk))

Note: look at your total WBC count and your ABS neutrophil count on your CBC. If you do not see an ABS # then you can calculate one by doing the following:

$$\text{ANC} = \text{WBC} \times (\% \text{ PMN} + \% \text{ bands})$$

- immunosuppression
- impaired immune function caused by viral infections and AIDS
- Nutritional disturbance
- Cachexia, obstruction, or direct tumor invasion of nutritional pathways can add to the risk of infection
- Cancer therapies
- Surgery, radiation, and chemotherapy can affect the body's defense systems and predispose them to infection

Common sites of infection:

- Mouth and pharynx
- Respiratory tract
- Skin and soft tissue
- Intravascular catheters
- Perineal region
- Urinary tract
- Gastrointestinal (GI) tract

The following is a nice simplistic but comprehensive overview of neutropenia, neutropenic precautions and the use of growth factor support. Click on the following link:

<http://www.realnurseed.com/t1000.htm>

Types of Infection and Specific Treatments: (Henke Yarbro, Hansen Frogge, Goodman, 2002; Ito, 2001; Wujcik, 2001).

Bacterial:

Gram-negative bacteria (Esherichia coli, Klebsiella pneumoniae, and Pseudomonas aeruginosa)

- Primary causes of infection in granulocytopenic patients

Gram-positive bacteria (Staphylococcus aureus and Staphylococcus epidermidis)

- Treatment is immediate empiric antibiotic therapy
- Highly specific therapy is initiated once an organism is identified
- Combination antibiotic therapy usually includes a beta-lactam antibiotic and an aminoglycoside

The following table lists the most common bacteria and the causative agents as well as the recommended drug therapies. Click on the following link:

<http://www.merck.com/mmpe/sec14/ch170/ch170a.html#CACJEHIIH>

Review the following short oncology case study on infection by clicking the following link:

<http://www.ons.org/publications/journals/CJON/Volume11/Issue2/1102189.asp>

Fungal Infections: (Ito, 2001; Wujcik, 2001; Henke Yarbro, Hansen Frogge, Goodman, 2002).

Fungal infections are an increasingly important cause of infections for cancer patients. Factors contributing to fungal infections include the following:

- Prolonged **granulocytopenia**
- Implanted vascular access devices
- Administration of **parenteral nutrition**
- **Corticosteroids**
- Prolonged **antibiotic therapy**

Candida is the most common cause of fungal infections. **Aspergillus**, **Cryptococcus**, **Histoplasma**, **Physomyces**, and **Coccidioides** species are other fungi that can cause serious infection.

Treatment selection may be made difficult if the practitioner is unable to culture the organism. There are also a limited number of effective agents available. Some common treatments are;

1) **Amphotericin B** is the drug of choice

Nephrotoxicity is the major side effect. Other side effects include:

- Fever
- Chills
- Rigors
- Nausea
- Vomiting
- Hypotension
- Bronchospasm
- Seizures

2) **Azole's**: fluconazole, ketoconazole, micronazole

Note: there is becoming increasing resistance to the **azole** group of antifungals. If there is no resolution of the organism after a course of therapy a sensitivity to the drug may be ordered and run by the lab to check for drug specific sensitivity or resistance.

The following link is an excellent overview of fungal infections specific to the oncology patient with an introduction to new antifungal agents. Click on the following link:

<http://www.supportiveoncology.net/journal/articles/0304290.pdf>

Viral Infections: (Henke Yarbro, Hansen Frogge, Goodman, 2002; Ito, 2001; Wujcik, 2001).

Most viral infections in cancer patients are caused by herpes simplex virus (HSV), varicella zoster virus (VZV), cytomegalovirus (CMV), and hepatitis A or B virus. Various treatments are as follows;

- Acyclovir, Valcyclovir, famciclovir for HSV and VZV
- Vidarabine is effective for HSV and VZV if used early
- Ganciclovir and foscarnet are used for CMV

Protozoa and parasites: (Ito, 2001; Wujcik, 2001; Henke Yarbro, Hansen Frogge, Goodman, 2002).

Protozoal infections are associated with defects in cell-mediated immunity. They can be difficult to treat in the immunocompromised host. They can also quickly become life-threatening. These organisms include;

Pneumocystis carinii

- Fatal if untreated
- Treat with Bactrim, pentamidine, dapsone
- high-risk patients (AIDS) are sometimes treated prophylactically

Toxoplasma gondii:

- treat with pyrimethamine plus sulfamethoxazole

Cryptosporidium

- no known treatment

(Henke Yarbro, Hansen, Frogge, Goodman, 2002).

Treatment: (Henke Yarbro, Hansen Frogge, Goodman, 2002).

Prevention is the most critical step in care

- meticulous hand washing
- avoidance of crowds and infected individuals
- if granulocytopenis is present, avoid invasive procedures
- adequate nutrition
- meticulous personal hygiene
- avoid skin trauma

Assessment:

- determine the ANC
- culture from all potential sites of infection before giving antibiotics
- if an infection is present monitor for patient's response to therapy and be alert for signs of *septic shock* http://en.wikipedia.org/wiki/Septic_shock

The following link is a good overview of viruses specific to the oncology population. Click on the following link:

<http://asheducationbook.hematologylibrary.org/cgi/content/full/2006/1/368>

Treatment of the Patient with Neutropenia and Fever: (Henke Yarbro, Hansen Frogge, Goodman, 2002).

A patient with an infection should be treated specifically for the causative organism. In some instances in which the infection develops rapidly and is serious empiric therapy may be initiated immediately. Other precautions may include;

- 1) isolation and protected environment (refer to hospital policy)
- 2) administration of colony-stimulating factors (neupogen, neulasta, granulocyte-macrophage colony-stimulating factor)
- 3) supportive care to maintain fluid and electrolyte levels may be required

The following is a case study of the management of a patient with neutropenic fever. Click on the following link:

[http://www.ons.org/publications/journals/CJON/Volume7/Issue2/0702218.a
sp](http://www.ons.org/publications/journals/CJON/Volume7/Issue2/0702218.asp)

Also consider reviewing the NCCN Practice Guidelines in Clinical Oncology for the most current recommendations on growth factor support and infection management. Click on the following link and look under guidelines for supportive care:

http://www.nccn.org/professionals/physician_gls/PDF/myeloid_growth.pdf

Special Consideration for the patient with Gram-negative sepsis: (Henke Yarbro, Hansen Frogge, Goodman, 2002).

Septic shock develops in 27 to 46% of cases with gram-negative bacterium. Mortality can reach 80% in this population unless aggressively treated. The following are a list of treatment measures to reverse shock and sepsis;

- adequate oxygenation
- effective circulation with fluid replacement and vasoactive agents
- immediate broad-spectrum antibiotic therapy
- observation for complications of sepsis: DIC, renal failure, heart failure, GI ulcers, hepatic abnormalities

Malignant Cerebral Edema: (Wilkes, 2001; Henke Yarbro, Hansen Frogge, Goodman, 2002).

Cerebral edema results from an increase in the fluid content of the brain. Brain metastasis is the prime source of cerebral edema. Primary tumors that most often metastasize to the brain are;

- lung
- breast
- melanoma
- renal carcinomas

Physiology:

Cerebral edema may result from;

- direct injury to vascular endothelium
- dysplastic vascular structures within tumor
- biochemical alteration of capillary permeability
- less stable blood-brain barrier

If cerebral edema exceeds the limits of compensation than brain herniation may occur.

Clinical Signs and symptoms:

- headache
- nausea and vomiting
- weakness
- personality changes
- somnolence
- impaired cognition
- seizures
- gait disorder
- visual disturbance
- language disturbance
- hemiparesis
- sensory loss (unilateral)
- papilledema
- ataxia
- aphasia

Diagnostic Measures: (Henke Yarbro, Hansen Frogge, Goodman, 2002; Wilkes, 2001).

- complete neurologic exam
- CT scan
- MRI scan (best for visualizing edema)
- Stereotactic needle biopsy (if questioning definitive diagnosis)

To view imaging of a brain tumor with surrounding cerebral edema click on the following link:

<http://jjco.oxfordjournals.org/cgi/content/full/31/3/112/HYE023F1>

Treatment: (Wilkes, 2001, Henke Yarbro, Hansen, Frogge, Goodman, 2002).

- Aggressive therapy is necessary to sustain or restore optimal neurological function
- Most important treatment initially is the use of **glucocorticoids**
- High doses of **dexamethasone** (30 to 60 mg) is given in four to six divided doses to rapidly reduce the rate of fluid formation
- The dose is **tapered** once neurologic symptoms are controlled and reduced
- If patients **have seizures** then they are placed on anticonvulsant therapy
- Control of seizures avoids sudden increases in ICP
- **Mannitol** osmotherapy can be used to produce profound reductions of cerebral edema on a temporary basis

- **Surgical** decompression or debulking of the tumor is effective in reducing intracranial pressure and preventing further edema
- Unifocal brain tumors are potentially curable with **radiation** therapy
- Radiation causes an increase in brain edema but is used once the patient is stabilized to treat the underlying cause of edema
- **Chemotherapy** is limited in use due to its inability to cross the blood-brain barrier.

To review a comprehensive overview of the management of brain tumors click on the following link:

<http://www.emedicine.com/NEURO/topic625.htm>

Pulmonary Emboli: (Escalante, Manzullo & Weiss, 2007; Henke Yarbrow, Hansen Frogge, Goodman, 2002).

The lungs are a filter for blood returning from the systemic circulation and therefore, material too large to pass through the pulmonary capillary system may become lodged in the pulmonary vasculature. Depending on the size and composition of the material, these embolisms may be asymptomatic or result in hemodynamic collapse.

Pulmonary emboli (PE) are common and are life threatening they manifest primarily from venous thromboemboli but there may be other causes as well (air, marrow fat, sickled cells, and parasites). Cancer patients are considered a high risk group for the development of DVT and PE.

Autopsy reports have demonstrated that 80% of patients who have had a PE also had evidence of lower extremity thrombosis. On presentation only 15-20% had evidence of lower extremity DVT.

Signs and symptoms:

- Dyspnea (most common)
- Tachypnea(> 20/min.)
- Pleuritic chest pain or nonretrosternal chest pain
- Hemoptysis
- Pleural rub
- Arterial saturation of less than 92% on room air
- Low-grade temperature
- Tachycardia (>100/min)
- Chest radiograph with presence of PE

The diagnosis of PE remains one of the most difficult to diagnose since it has a similar presentation as other cardiac and pulmonary disorders.

Diagnostic work-up:

- Assess for clinical probability (risk factors and presence of symptoms of DVT)
- D-dimer (lab will be elevated in 90% of patients)
- Ventilation-perfusion scanning (V-Q)(non-diagnostic in 40% who are positive)
- Pulmonary angiogram (highly sensitive and specific for the diagnosis of emboli for the proximal pulmonary arteries; more distal emboli cannot be reliably detected)
- New: magnetic resonance pulmonary angiography
- EKG (maybe but plays a secondary role in diagnosis)

Note: you will most commonly see a D-dimer and CT angio or CT angio alone ordered as part of a work-up. If you see a CXR order it is because the MD needs to rule out other causes first and has a low suspicion of PE. If the CXR is negative he may then order the CT angio. You cannot see pulmonary emboli routinely on CXR.

Treatment: (Escalante, Manzullo & Weiss, 2007; Henke Yarbrow, Hansen Frogge, Goodman, 2002).

- Rapid institution of fractionated heparin or LMWH (Lovenox, Fragmin)
- If a high suspicion that therapy should be started even before results are back
- Maintain a PTT of 1.5 to 2 times the control value
- Monitor platelet counts
- Oral warfarin is started concurrently, and warfarin is continued for 3 to 5 days until warfarin reaches a therapeutic level (Goal = 2 to 3 times the control value)
- Warfarin is continued for 3-6 months
- The ideal duration of therapy is unknown but 6 months of therapy appears to decrease the risk of recurrent PE compared to 6 weeks.
- If high risk for recurrent PE then they may receive long-term or life-long therapy

The following is a case study about the presentation and management of pulmonary emboli. Click on the following link:

<http://www.ons.org/publications/journals/CJON/Volume11/Issue3/1103343.asp>

Spinal Cord Compression (SCC):

Review the following article on SCC. Click on the following link:

<http://www.ons.org/publications/journals/ONF/Volume30/Issue1/300134.asp>

The following link includes a case study on the SCC. Click on the following link:

http://www.nursingcenter.com/library/journalarticleprint.asp?Article_ID=537619

Superior Vena Cava Syndrome (SCVC):

The following article provides a comprehensive overview of SVCS. Click on the following link:

<http://www.ONS.org/publications/journals/ONF/Volume30/Issue4/pdf/513.pdf>

To view an image of SVCS syndrome click on the following link:

<http://www.nature.com/ncpcardio/journal/v4/n4/images/ncpcardio0850-f1.jpg>

<http://www.aboutcancer.com/svco.htm>

Syndrome of Inappropriate Antidiuretic Hormone (SIADH):

To review SIADH click on the following link:

<http://www.ons.org/publications/journals/ONF/Volume30/Issue3/3003381.asp>

Tumor Lysis Syndrome:

To review TLS and participate in a case study Q&A click on the following link:

<http://www.ons.org/publications/journals/CJON/Volume8/Issue4/pdf/415.pdf>

NOTE:

Try this fun and informative website to review case studies to test your self on a variety of oncologic emergencies. Click on the following link:

http://www.nursingcenter.com/library/journalarticleprint.asp?Article_ID=755801