Introduction

This self-learning module is available to oncology nursing staff. Knowledge of critical cancer complications is essential. This module will assist in preparing nurses for unique oncologic emergencies.

Purpose

To assist oncology nurses in providing safe and emergency care of critical oncology complications.

Objectives

1. Discuss the etiology of oncology complications
2. Name the categories of oncology complications
3. Discuss assessment, treatment, and nursing interventions for the oncology emergencies

Directions

Read the module and complete the post-test. Submit the post-test to your educator. A passing score of 80% is required.
CRITICAL CANCER COMPLICATIONS

Oncologic complications occur in patients with cancer and may result directly from the disease. Frequently they are an indication of progressive or advancing disease. Oncologic complications also occur as a result of treatments of chemotherapy or radiation for cancer. Oncologic emergencies are acute and life threatening.

Nurses are in a key role to accurately assess patients who are at high risk for oncologic complications. Patients may be hospitalized on a medical, surgical, oncology, or critical care unit. Assessment is essential because a change in the patient’s condition may be subtle or dramatic. Once the oncologic complication is analyzed, decisions about the aggressiveness of treatment can be made. The overall goal is to prevent, reverse, or minimize life-threatening oncologic complications through prophylactic measures, early detection, and effective management.

The Oncology Nursing Society (ONS) in the Core Curriculum for Oncology Nursing (4th edition, 2005) divides oncologic emergencies into two main categories: metabolic and structural. This module will be organized according to the ONS categories with the addition of hypersensitivity (anaphylaxis) and tumor lysis syndrome.

<table>
<thead>
<tr>
<th>ONS CORE CURRICULUM ONCOLOGIC EMERGENCY CATEGORIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>STRUCTURAL</td>
</tr>
<tr>
<td>Cardiac tamponade</td>
</tr>
<tr>
<td>Increased intracranial pressure</td>
</tr>
<tr>
<td>Spinal cord compression</td>
</tr>
<tr>
<td>Superior vena cava syndrome</td>
</tr>
<tr>
<td>METABOLIC</td>
</tr>
<tr>
<td>Disseminated intravascular coagulation (DIC)</td>
</tr>
<tr>
<td>Hypercalcemia</td>
</tr>
<tr>
<td>Hypersensitivity reaction (anaphylaxis)</td>
</tr>
<tr>
<td>Malignant pleural effusion</td>
</tr>
<tr>
<td>Sepsis</td>
</tr>
<tr>
<td>Syndrome of inappropriate antidiuretic hormone</td>
</tr>
<tr>
<td>Tumor lysis syndrome</td>
</tr>
</tbody>
</table>
Cardiac Tamponade is the compression of the cardiac muscle by fluid accumulation within the pericardial sac. Normally the pericardial sac holds 25-35ml of fluid. Fluid accumulates because of pericardial constriction by a tumor or postirradiation pericarditis. Compression of the myocardium prevents adequate cardiac filling during diastole that reduces blood flow to the ventricles and reduces stroke volume. Ultimately, the cardiac output is decreased. This reduction of coronary artery flow may develop into myocardial ischemia. Cancers at greatest risk for the development of cardiac tamponade include: breast cancer, lung cancer, lymphoma and leukemia. Any cancer has the potential for metastatic spread to the pericardium via direct tumor extension, lymphatic invasion, or hematogenous dissemination. Patients with pericardial effusions are also at great risk for tamponade. Compensatory mechanisms including increased heart rate and vasoconstriction are activated in an effort to maintain adequate blood flow.

The severity of cardiac tamponade depends on the amount of fluid in the pericardium, the rate of accumulation, and the degree of pericardial compromise. Gradual fluid accumulation of 50ml or less allows the pericardium to stretch and accommodate. However, 100 to 200ml of fluid may cause severe cardiac impairment if the accumulation occurs rapidly. Small or slowly developing effusions may be asymptomatic. As fluid increases, the most common symptoms are dyspnea, tachycardia, retrosternal chest pain (usually relieved by sitting up and leaning forward) and a nonproductive cough. Tamponade due to radiation pericarditis may also be exhibited by fever and pleuritic chest pain. Other classic signs of cardiac tamponade are pulsus paradoxus and jugular venous distention from increased venous pressure. The point of maximal impulse may shift laterally from the fifth intercostal space due to cardiac enlargement.
The clinical finding in cardiac tamponade of pulsus paradoxus is a classic feature. Pulsus paradoxus is an abnormal finding of a weaker pulse during inspiration, resulting from a greater than normal (10mm Hg) decrease in systolic blood pressure during the inspiratory phase of normal respiration.

The echocardiogram is the most accurate tool for diagnosing cardiac tamponade. Cardiac ultrasound can determine the presence, location, and approximate quantity of fluid. CT, MRI and TEE may also be helpful in diagnosing this condition. The immediate goal of treatment is the removal of pericardial fluid to relieve impending circulatory collapse. Pericardiocentesis is usually effective in relieving signs and symptoms of cardiac tamponade but fluid generally reaccumulates in 24 to 48 hours. Malignant effusions are typically exudates from the irritation of serous membrane caused by sloughed cancer cells. Malignant effusions are usually serosanguineous.

The mortality rate for cardiac tamponade has been reported as 25%. This rate increases to 65% if the tamponade goes unrecognized. The overall prognosis of the patient may be poor, but the response and comfort of the patient is improved.

**INCREASED INTRACRANIAL PRESSURE**

Increased intracranial pressure results when the volume of any of the three components within the skull and meninges is increased. These components include the brain, cerebral spinal fluid, and cerebral blood volume. If increased intracranial pressure is not successfully treated, brain herniation and death can occur.
The most common oncologic etiology for increased intracranial pressure is brain metastases. 20-40% of cancer patients develop brain metastases. Lung and breast cancers are the solid tumors that most frequently metastasize to the adult brain.

Other risk factors for increased intracranial pressure that may occur in oncology patients are cerebral hematomas, hemorrhage, or infection. Patients with thrombocytopenia or platelet dysfunction can cause cerebral bleeding. Patients receiving radiation therapy to the brain may experience cerebral irritation. Patients with lymphoma, leukemia, central nervous system tumors or an Ommaya reservoir are at risk for infection. Syndrome of inappropriate diuretic hormone and CNS infections can cause cellular edema resulting in increased intracranial pressure. Patients with head and neck cancer may experience increased intracranial pressure from compression of the internal jugular veins.

General symptoms for increased intracranial pressure include headache, nausea, vomiting, change in level of consciousness, hemiparesis, dysphagia, ataxia, and seizures. Change in the level of consciousness is a classic sign of increased intracranial pressure. Visual findings of increased intracranial pressure include blurring, changes in pupil size and light accommodation, and papilledema (a late sign). Advanced intrcranial pressure can cause hemiparesis, hemiplegia, decreased reflexes, and decorticate and decerebrate positioning. Cushing’s triad, another late sign of increased intracranial pressure, is a combination of hypertension, bradycardia, and irregular or slow respirations. Posturing and Cushing’s triad are associated with the terminal phase of brain herniation. Vital signs for increased intracranial pressure reflect high blood pressure, low pulse and increased respiration.

Patients with cancer who develop neurologic changes should be evaluated immediately for brain metastases. Both CT scanning and MRI are used to evaluate brain metastases. Lumbar puncture should be avoided due to the risk of herniation.

Rapid reduction of cerebral edema is the immediate treatment goal. Corticosteroids can rapidly decrease peritumoral edema. Improvements in symptoms may be seen with 24 hours. Corticosteroids are thought to decrease capillary permeability and promote extracellular fluid resorption. Patients at risk for herniation are started on high doses such as 100mg dexamethasone IV. Maintenance doses may be as high as 30mg four times a day. Osmotic diuretics such as Mannitol may also be needed to decrease cerebral edema by increasing plasma osmolarity and drawing extracellular fluid back into the plasma where it can be excreted by the kidneys. Loop diuretics such as furosemide are used to decrease CSF production and enhance the excretion of sodium and water from the brain. Fluids, electrolytes and hemodynamics must be monitored closely. Normal intracranial pressure is 4 to 15 mm Hg. Anticonvulsants such as phenytoin, carbamazepine, and phenobarbital are given to manage seizures when appropriate. Emergent surgical intervention is needed for the patient with life threatening increased intracranial pressure. Tumor resection, shunts, or ventriculostomy can treat the edema. Radiation and chemotherapy are primarily used to treat brain metastases once the acute symptoms are controlled.

Important nursing management of the patient includes assessing neurologic status often, positioning the patient with the head of the bed elevated 30 to 45 degrees, monitoring the patient for seizure activity and maintaining a calm, quiet environment. Actions that increase intrathoracic or intrabdominal pressures can restrict cerebral blood
flow and should be avoided. They include: valsalva maneuver, bending over, coughing, sneezing, extreme neck flexion or extension, lying on the abdomen and ventilator settings of positive end-expiratory pressure (PEEP). Unfortunately, many nursing interventions, although necessary, can further aggravate increased intracranial pressure (turning, range of motion). Spacing the activities and nursing care can decrease sustained elevation of increased intracranial pressure. Other important nursing actions to control increased intracranial pressure include fluid restriction, hyperventilation, sedation and control of temperature.

Prognosis for the patient with increased intracranial pressure depends on the severity and rapidity of the symptoms as well as early recognition and prompt initiation of appropriate treatment. Patient survival depends on the type and stage of cancer and response to the overall treatment.

**SPINAL CORD COMPRESSION**

A neoplasm in the epidural space can encroach on the spinal cord and result in spinal cord compression. Primary tumors of the spinal cord account for a small percentage of this condition. Metastatic disease is the most common cause. Patients with cancer that metastasizes to the bone are at greatest risk for spinal cord compression. Compression of the spinal cord leads to edema of the cord and ischemia and damages neural tissue. Lung, breast, and prostate cancers are associated with many of the spinal cord compression cases. Permanent neurologic deficits such as paralysis and loss of bowel and bladder control can occur if early detection and treatment are not instituted. The primary aspect of nursing care is early detection as response to therapy is directly related to the patient’s functional status at diagnosis. Very subtle changes in neurological function are significant and should be reported.
The cardinal signs and associated symptoms of spinal cord compression usually follow an established pattern of appearance. They include pain, motor weakness, and sensory loss. Back pain is the presenting complaint as most spinal metastases develops in the thoracic spinal region. This pain may radiate around the chest or abdomen and is aggravated by coughing, sneezing, straining (as with the valsalva maneuver) or straight leg raising. Motor and sensory involvement is manifested by hyperactive reflexes, positive Babinski signs, spastic weakness and bilateral sensory loss below the level of the compression. Loss of sphincter control is a late sign and is associated with a poorer prognosis. Sexual dysfunction may be manifested as impotence.

Testing for spinal cord compression may include x-rays, MRI, or myelography. Radiation therapy is the treatment of choice for spinal cord compression. The use of corticosteroids such as dexamethasone may also be used to reduce cord and nerve compression pain and edema.

Surgical interventions may be possible to remove the tumor. Recent research has also demonstrated a role for certain biphosphonates, particularly pamidronate (Aredia) to reduce the complications of bony metastases. Infusions of 90mg over a 2 hour infusion every 3 to 4 weeks may be helpful.

The degree of neurologic dysfunction at the time of diagnosis is the greatest predictor of outcome. Paraplegia and incontinence are preventable when treatment is begun in a timely manner.

**SUPERIOR VENA CAVA SYNDROME**
The superior vena cava is a major venous vessel that returns blood to the right atrium of the heart from the head, upper thorax, and upper extremities. Obstruction of the venous flow through this vessel results in impaired venous drainage, with engorgement of the vessels from the head and upper body. Compression of the superior vena cava can occur by:

- External compression by tumor or lymph nodes
- Direct invasion of the vessel wall by tumor
- Thrombosis of the vessel

As the venous pressure rises in the superior vena cava, blood is shunted to collateral venous pathways to facilitate return to the right atrium. Only about 3% of cases of superior vena cava syndrome have a benign cause. Nonmalignant causes of superior vena cava syndrome include aortic aneurysm, infectious agents, central venous catheters and thyroid conditions. Cancer is responsible for most cases. Most malignant cancers causing superior vena cava syndrome involve bronchogenic cancer, particularly small cell lung cancer.

The most frequent symptom of superior vena cava syndrome is dyspnea. If untreated, superior vena cava syndrome can lead to airway obstruction. The classical clinical picture also includes facial swelling, cough and chest pain. Progression of symptoms may lead to severe respiratory obstruction, paralyzed vocal cord, cyanosis of the upper extremity, drowsiness, unconsciousness, seizures and possible death. Chest films, MRI and CT scan may be helpful. Radiation therapy is the treatment of choice for this syndrome to reduce tumor size. Other adjunctive therapy for this condition includes diuretics to reduce edema of the head and neck, corticosteroids to reduce inflammation related to obstruction, and oxygen therapy for respiratory complications. Nursing interventions to promote patient comfort through positioning and elevation of the upper extremities will support decreased venous congestion. Additionally, preventing activities that increase intrathoracic or intracerebral pressure such as the Valsalva maneuver, vomiting, bending over or stooping is advised.

**METABOLIC EMERGENCIES**

**DISSEMINATED INTRAVASCULAR COAGULATION**
Disseminated intravascular coagulation (DIC) is considered to be a bleeding disorder. Bleeding disorders are classified as congenital or acquired. DIC is an alteration in the blood-clotting mechanism, characterized by abnormal acceleration of the coagulation cascade, resulting in thrombosis. Hemorrhage occurs simultaneously as a result of the depletion of clotting factors. In patients with cancer, the incidence is usually related to the disease process or the treatment of cancer and often occurs with sepsis. The next most common cancers are APML (acute promyelocytic leukemia), lung, breast, stomach, pancreas, and prostate. The granules present in the leukemia cells of patients with APML contain potent activators of the clotting cascade which may trigger DIC.

The presenting signs and symptoms result from bleeding. Bleeding may range from oozing to frank bleeding or hemorrhage. Patients may have oozing from venipuncture sites, mucous membranes, needle puncture sites, or incisions. Petechiae, ecchymoses purpura, or hematomas may be evident. Profound menstrual or GI bleeding as well as epistaxis or hemoptyisis is possible. Intracerebral bleeding is critical and may be seen with mental status changes, stroke, visual changes, or headaches. Abdominal distention, blood in stools, or blood in urine may be observed. Clotting resulting from fibrin deposits in the microcirculation will impede blood flow. Early signs of clotting may include oliguria, hypoxemia, and cool and mottled extremities. Progression can cause severe tissue ischemia and lead to necrosis. Irreversible organ damage is a major concern.

The cardinal finding in DIC is thrombocytopenia, a decreased platelet count. In about 50% of these patients, the platelet count is less than 50,000/mm³. The PT is prolonged reflecting decreased levels of clotting factors. Fibrinogen level is decreased resulting
from the consumption of fibrinogen by thrombin-induced clotting and excessive fibrinolysis.

The only effective treatment is treating the underlying cause of the syndrome. Sepsis is treated with antibiotics. DIC can be controlled by administering platelets and fresh frozen plasma and cryoprecipitate. Heparin therapy which inhibits thrombin formation is controversial. The anticoagulation effects of heparin result from its prevention of the platelet aggregation. Nursing interventions including bleeding precautions and protecting the patient from injury. Avoidance of injections and lab draws is indicated.

**HYPERCALCEMIA**

Hypercalcemia is a metabolic condition that occurs when the serum calcium rises above the normal level of 11mg/dL. Lack of intervention can lead to renal failure, coma, or cardiac arrest. Hypercalcemia occurs in about 10-20% of oncology patients. Malignancies associated with this condition include cancers of the breast and kidneys,
small cell cancers of the lung, head, neck or esophagus, lymphoma, leukemia, and multiple myeloma. Patients with metastatic cancers treated with estrogens or antiestrogens may experience hypercalcemia, possibly from hormonal stimulation of the tumor.

Calcium is an essential inorganic element in the body. Most calcium is found in the skeletal tissue. The remaining 1% is found in the serum. One half of the serum calcium is ionized and one half is bound to circulating albumin. Under normal conditions the ionized calcium is in equilibrium with the protein bound calcium. Changes in serum albumin directly affect serum calcium levels. Only the ionized portion of calcium is capable of physiologic function. Calcium is responsible for bone and tooth formation, normal clotting mechanism, and cellular permeability. Calcium ion concentration regulates the contractility of the cardiac, smooth, and skeletal muscles and the excitability of the nerve tissue. Homeostasis is maintained through several body processes: GI calcium absorption, renal calcium absorption, and a balance of bone resorption of calcium and deposition of calcium through new bone formation. A balance is maintained between osteoclast (bone resorption) and osteoblast (bone formation).

Symptoms of hypercalcemia include restlessness, agitation, lethargy, and confusion. Skeletal muscles become hypotonic with decreased or absent deep tendon reflexes, ataxia and fatigue. The smooth muscle action of the GI system slows, leading to decreased motility anorexia, nausea, vomiting, constipation and later, ileus. Impaired cardiac muscle conduction and contractility can result in dysrhythmia and even cardiac arrest. Compensatory renal mechanisms of increased urinary calcium reabsorption leads to an inability to concentrate urine. This results in polyuria and polydipsia and possibly dehydration. Serum electrolytes are monitored for hypokalemia, hyponatremia, hypocalcemia and hypomagnesemia. Observe EKG for shortened QT intervals or prolonged PR intervals.

Adequate hydration is the primary treatment for hypercalcemia. Vigorous hydration (4-6 Liters/day) will raise glomerular filtration rate and interfere with calcium reabsorption in the proximal tubule. Once the patient is rehydrated, loop diuretics are sometimes used to enhance calcium excretion. Intravenous diuretics cause sodium diuresis which in turn causes calcium diuresis. Thiazides should not be administered to patients with hypercalcemia because these diuretics decrease urinary excretion of calcium by their action on the distal renal tubule. Pharmacologic therapy using biphosphonates which inhibit the action of osteoclasts; pamidronate, also a biphosphonate has become a first line agent for moderate to severe hypercalcemia. This medication also may prevent skeletal complications from bone pain (bone pain, spinal cord compression). Calcitonin is another agent which inhibits bone resorption and increases urinary excretion of calcium. With calcitonin, the risk of anaphylaxis is of great concern.

Immobilization should be avoided as it will increase resorption of calcium from the bones. Activity should be appropriate for the patient’s condition. A pain management program may be necessary. Dietary intake of calcium does not play a role in hypercalcemia of malignancy. Patients with cancer typically have reduced GI absorption of calcium so dietary restriction is not necessary. Hypercalcemia is reversible in 80% of cases if recognized and prompt attention is given. Without prompt treatment, this condition is associated with a 50% mortality rate. Nursing care of the patient is directed at early detection and support through treatment.
HYPERSENSITIVITY REACTION (ANAPHYLAXIS)

Hypersensitivity reactions or anaphylaxis is a life threatening immunologic response to a foreign substance or antigen. Chemotherapy, like other medications, may be recognized by the body’s immune system as “not self.” This may initiate a reaction of release of histamine and other inflammatory responses that trigger respiratory and cardiovascular collapse.

Risk factors associated with an increased risk of hypersensitivity include age, gender, Route of administration, protein composition, and heavy metal composition. Antineoplastic agents associated with hypersensitivity reactions can be classified as high risk, moderate risk and low risk. The following table outlines this information.

### Immediate Hypersensitivity Reactions: Predicted Risk of Chemotherapy

<table>
<thead>
<tr>
<th>High Risk</th>
<th>Low to Moderate Risk</th>
<th>Rare Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>L-asparaginase</td>
<td>Anthracyclines</td>
<td>Cytosine arabinoside</td>
</tr>
<tr>
<td>Paclitaxel</td>
<td>Bleomycin</td>
<td>Cyclophosphamide</td>
</tr>
<tr>
<td>Carboplatin</td>
<td>Chlorambucil</td>
<td></td>
</tr>
<tr>
<td>Cisplatin</td>
<td>Dacarbazine</td>
<td></td>
</tr>
<tr>
<td>Cyclosporine</td>
<td>5-Fluorouracil</td>
<td></td>
</tr>
<tr>
<td>Docetaxel</td>
<td>Ifosamide</td>
<td>Mitoxantrone</td>
</tr>
<tr>
<td>Etoposide</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Melphalan</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Methotrexate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Procarbazine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Teniposide</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Cancer Chemotherapy Guidelines and Recommendations for Practice
The priority for the nurse caring for the patient at risk of hypersensitivity or anaphylaxis is to assess and record baseline vital signs. This information is needed to assess hypotension or respiratory dysfunction. The signs and symptoms of hypersensitivity occur within minutes of initiating the agent and peak within 15 to 30 minutes. The most common effects include dyspnea, flushing, wheezing, urticaria, dizziness, agitation, and hypotension. Premedication regimens of corticosteroids, and receptor antagonists of diphenhydramine and cimetidine can reduce or eliminate this reaction. Nursing interventions to treat anaphylaxis include STOPPING THE INFUSION, providing oxygen, cardiac monitor and defibrillator, intubation supplies and administration of front line rescue drugs (epinephrine, dopamine). Always be crash cart ready when administering chemotherapy.

The most important information needed by a patient who has had an anaphylactic reaction to chemotherapy concerns the recognition of signs and symptoms of anaphylaxis. If reactions should occur during administration of subsequent treatments, the patient needs to be able to report them immediately.

**MALIGNANT PLEURAL EFFUSION**

A pleural effusion is an abnormal accumulation of fluid in the pleural cavity. With cancer, the pleural space is the most common. Abnormal fluid accumulation results when the balance between secretion and reabsorption is altered. Malignant pleural effusion is debilitating and life threatening because increased pleural fluid affects respiratory function by restricting lung expansion, decreasing lung volume, and altering
gas exchange. Expansion of the pleural space leads to compression or collapse of the lung. Lung and breast cancers are most often associated with malignant pleural effusion. It is estimated that 5 to 10 liters of fluid pass through the pleural space in a day, but only 5 to 20 mL remains in the space at any given time. Lymphatic blockage or venous obstruction interferes with drainage of fluid. Necrotic malignant cells may shed into the pleural space and interfere with the fluid drainage process.

The onset of a malignant pleural effusion may be slow or rapid. Dyspnea is the most common reported symptom and is frequently accompanied by a non-productive cough and chest pain. Pleural effusion is usually detected by chest x-ray films. A thoracentesis is indicated for diagnostic and therapeutic measures. Milky or bloody fluids typically characterize malignancy. Treatment modalities include radiation therapy for lymphomas, chest tube insertion for repeated effusions, chemical sclerosing to prevent further fluid accumulations, and surgery (shunt, thoracotomy, pleurectomy). Nursing care of a patient with pleural effusion focuses on strong assessment skills and monitoring for recurrence as malignant pleural effusions tend to reoccur.

SEPTIC SHOCK

Septic shock is defined as sepsis induced hypotension (despite fluid resuscitation) and organ perfusion abnormalities. Septic shock can lead to irreversible multiorgan dysfunction syndrome and death. Definitions for Sepsis-related Clinical Conditions are described as:
Septic shock is the most common cause of noncoronary intensive care unit deaths in the United States. The incidence of sepsis in cancer patients is estimated at 45% with greater than 30% mortality. Early vital signs for shock are low blood pressure, high pulse, and increased respiration. Septic shock can be caused by bacterial, fungal, viral and protozoal organisms. Gram-negative bacteria (E. Coli, Klebsiella pneumonia, Pseudomonas aeruginosa) have historically been the primary organisms associated with septic shock. Due to the immunosuppression in cancer patients, endogenous flora is typically the source of infection. Patients with cancer are frequently immunosuppressed.
Neutropenia is caused by most antineoplastic agents as well as radiation therapy. Screening Criteria for Patients with severe sepsis is outlined as follows:

### Screening Patients for Severe Sepsis

A patient who meets the following 3 criteria has a positive screen suggestive of severe sepsis:

1. **INFECTION**—One or more of the following:
   - **DOCUMENTED**—Does the patient have positive culture results from blood, sputum, urine, etc.?
   - **ANTI-INFECTIVE THERAPY**—Is the patient receiving antibiotic, antifungal, or other anti-infective therapy?
   - **PNEUMONIA**—Is there documentation of pneumonia (x-ray, ultrasound, etc.)?
   - **WBCs**—Have WBCs been found in normally sterile fluid (urine, CSF, etc.)?
   - **PERFORATED VISCUS**—Does the patient have a perforated organ (bowel)?

2. **SIRS**—Two or more of the following:
   - **TEMPERATURE**—Is the patient’s temperature > 38°C (> 100.4°F) or < 36°C (< 96.8°F)?
   - **HEART RATE**—Is the patient’s heart rate > 90 bpm?
   - **RESPIRATORY RATE**—Is the patient’s respiratory rate > 20 breaths/min?
   - **WBC COUNT**—Is the patient’s WBC count > 12,000/mm³, < 4,000/mm³, or are there > 10% immature neutrophils (left shift)?

3. **ACUTE ORGAN DYSFUNCTION**—One or more of the following:
   - **RESPIRATORY**—Does the patient require mechanical ventilation (PF ratio < 250, PEEP > 7.5)?
   - **CARDIOVASCULAR**—Does the patient require vasopressor support (systolic BP < 90 mmHg or Mean Arterial Pressure < 70 mmHg or 1 hour despite fluid bolus)?
   - **RENAL**—Does the patient have low urine output (eg, < 0.5 mL/kg/hr), increased creatinine (> 50% increase from baseline) or require acute dialysis?
   - **HEMATOLOGIC**—Does the patient have a low platelet count (< 100,000/mm³) or PT/PTT > upper limit of normal?
   - **METABOLIC**—Does the patient have a low pH with high lactate (eg, pH < 7.30 and plasma lactate > upper limit of normal)?
   - **HEPATIC**—Are the patient’s liver enzymes > 2x upper limit of normal?
   - **CNS**—Does the patient have altered consciousness or a reduced (Glasgow Coma score?

\[
\text{INFECTION} \times \text{SIRS} \times \text{ORGAN DYSFUNCTION} = \text{POSITIVE SCREEN SUGGESTIVE OF SEVERE SEPSIS}
\]

Prevention is the best precaution against septic shock. Measures to protect immunocompromised patients from infection are critical. Meticulous handwashing, low bacterial diets (fresh fruit, raw vegetables, flowers), and avoidance of crowds are necessary.
The goals of treatment are to maintain blood pressure and tissue perfusion and treat the underlying pathogen. Antibiotics, fluid replacement, oxygen, ventilatory support, blood products and Xigris may be indicated.

**SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE SECRETION**

The syndrome of inappropriate antidiuretic hormone secretion (SIADH) is a syndrome that causes a disorder of water balance. ADH (Antidiuretic hormone) regulates the body’s water balance. SIADH is characterized by elevated serum blood levels of ADH, excessive water retention, hypo-osmolality, and hyponatremia. The most common malignant disease associated with this syndrome is lung cancer (small oat cell carcinoma). Several chemotherapeutic agents including cisplatin, cyclophosphamide, vinblastine and vincristine have also been associated with SIADH.

Hyponatremia (serum sodium less than 135 mEq/L) may result in thirst, nausea, weight gain, muscle weakness, peripheral edema and oliguria. Currently, no drugs are available that suppress the synthesis or release of ADH from malignant tissue. However, the sodium level and patient’s neurological status need to be stabilized. Fluid restriction, electrolyte replacement and osmotic diuresis may be required.

**TUMOR LYSIS SYNDROME**

Tumor lysis syndrome is an oncologic emergency that occurs with rapid lysis of malignant cells. The resulting metabolic imbalance can quickly lead to fatal renal, cardiac, and neurologic complications. Tumor lysis syndrome occurs spontaneously in patients with high tumor burden or high growth capacity. It may occur anytime from 24 hours to 7 days after chemotherapy is initiated.

As malignant cells are lysed, intracellular contents are rapidly released into the bloodstream. This results in high levels of potassium (hyperkalemia), phosphate
(hyperphosphatemia), and uric acid (hyperuricemia). Hypocalcemia also occurs because of the inverse relationship between phosphate and calcium. Patients with lymphomas and leukemias are most at risk.

Early signs may include nausea, vomiting, anorexia, and diarrhea. This may be accompanied by muscle weakness, cramping, and later progress to tetany, paresthesias, convulsions, anuria, and cardiac arrest.

Prophylactic treatment with hydration to maintain a urinary output of 100 mL/hour may be used to promote the excretion of phosphate and uric acid. Allopurinol prevents the formation of uric acid. Diuretics are used to prevent volume overload. Kaexylate is used to eliminate potassium. Calcium gluconate is used to correct hypocalcemia.

In Summary- Successful management of patients with oncologic complications requires an advanced knowledge of oncology nursing. Nursing assessment and patient advocacy assist patient and families in dealing with the impact of cancer on their lives. Due to the urgent nature of oncologic complications, prevention, early recognition, decision making and prompt treatment are essential.

References
Oncology Nursing Society (ONS), Core Curriculum for Oncology Nursing. 2005.
Copyright 2002.

PICTURES FROM THE FOLLOWING WEBSITES:
Cardiac Tamponade- https://health.google.com/health/ref/graphic/18123
DIC- http://sites.google.com/site/nursing211fall09/wk-3-lung-surg-pleural-effusion resp-failure-ards-chest-trauma/211b-group-5
http://commons.wikimedia.org/wiki/File:DIC_With_Microangiopathic_Hemolytic_Anemia.jpg
Hypercalcemia- www.usfca.edu/fac-staff/ritter/Image40.gif
Superior Vena Cava Syndrome- http://bestpractice.bmj.com/best-practice/monograph/848/resources/image/bp/2.html