Oncologic Emergencies

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PSONS Fundamentals of Oncology
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The Basics

- **Oncologic emergencies are <u>life-threatening</u> medical emergencies and must be treated as such!**
- **Why do they occur?**
  - Malignancy
  - Treatment of malignancy
- **When do they occur?**
  - Early in disease process (initial manifestation of malignancy itself)
  - Late in disease process (manifestation of treatment of malignancy)

The Basics

Metabolic Emergencies
- Tumor Lysis Syndrome (TLS)
- Systemic Inflammatory Response Syndrome (SIRS) and Septic Shock
- Disseminated Intravascular Coagulation (DIC)
- Thrombotic Thyrombocytopenia (TTP)
- Hypercalcemia
- Inappropriate Antidiuretic Hormone Secretion (SIADH)
- Anaphylaxis/Hypersensitivity

- Structural Emergencies
  - Spinal Cord Compression
  - Superior Vena Cava (SVC) Syndrome
  - Increased Intracranial Pressure (ICP)
  - Cardiac Tamponade

(Maloney, 2016; Vogel 2016)

Tumor Lysis Syndrome (TLS)

Tumor Lysis Syndrome

- **Metabolic Imbalance**
- **Caused by breakdown of malignant cells**
  - - Large number of rapidly proliferating cells killed
  - - Cell lysis, rupture of tumor cell membranes
- **Intracellular components released into bloodstream**

(Maloney, 2016; Zobec, 2008)

Tumor Lysis Syndrome

Who is <u>most</u> at risk?

Patients with large tumor burden that is highly responsive to chemotherapy (resulting in rapid cell kill).

- Risk Factors:
  - Tumor-related
    - High-grade lymphomas
    - Hematologic malignancies (acute or chronic leukemia's with ↑ WBC)
    - Tumors with high growth fractions (anticipated to be responsive to treatment)
  - Patient-related
    - Large tumor burden/bulky tumors
    - Elevated LDH
    - Pre-existing renal dysfunction
  - Treatment-related
    - Chemotherapy & biologic agents
    - Radiation therapy

(Maloney, 2016)

Tumor Lysis Syndrome

•Onset:

- Usually within 12-72 hrs. after initiation of antineoplastic therapy

Duration:

- May persist for 5-7 days post-therapy

(NCCN, 2016; Holmes Gobel, 2013)

Tumor Lysis Syndrome

- **Clinical Presentation:**
- Asymptomatic or only experiencing only vague symptoms
- Detected in blood chemistries

(Maloney, 2016)

Tumor Lysis Syndrome

- **So……**

- **What are the signs and symptoms of:**
- Hyperkalemia
- Hyperphosphatemia
- Hyperurecemia
- Hypocalcemia
- **???**

Tumor Lysis Syndrome

General management:

- **Prevention Strategies**
  - Recognition of at-risk patients
  - Hydration
  - Prevention of hyperuricemia
  - Frequent monitoring of electrolytes

- **Intervention Strategies**
  - Hydration
  - Control of hyperuricemia
  - Aggressive correction of electrolytes
  - Management of acute renal failure

(Maloney, 2016)

Tumor Lysis Syndrome

- **Prevention:**

- **Hydration**
  - IV Normal saline or 5% dextrose
  - Begin 24 – 48 hours prior to therapy
  - Ensure urine output >150 – 200 ml/hr

- **Diuresis**
  - Typically used if urine output not maintained by hydration alone
  - Loop diuretics or osmotic diuretics

(Holmes Gobel, 2013)

Tumor Lysis Syndrome

Prevention:

- **Monitor serial lab values**
  - Serum potassium, phosphorous, calcium, uric acid
  - Renal function studies – BUN & creatinine

- **Frequency of monitoring**
  - Prior to initiation of therapy
  - Every 8 – 12 hours during the first 48 – 72 hours of treatment

(Holmes Gobel, 2013)

Tumor Lysis Syndrome

- Prevention and/or control of hyperuricemia:

Allopurinol
or
Rasburicase

(Maloney, 2016)

Tumor Lysis Syndrome

- **Allopurinol (Oral or IV)**
  - Blocks uric acid production by inhibiting xanthine oxidase (liver enzyme)
  - Prevents uric acid precursors from converting to uric acid, ↓ risk uric acid crystallization
  - Dosing:
    - Oral: 300 mg/m^2 /day (not to exceed 600 mg/day)
    - IV: 200 – 400 mg/m^2 /day
    - Begin 2 – 3 days prior to chemotherapy
    - Continue for 10-14 days

(Holmes Gobel, 2013)

Tumor Lysis Syndrome

- **Rasburicase (Elitek®) IV**
  - Converts uric acid into allantoin (which has a much greater solubility than uric acid)
  - NCCN recommendations for preventative therapy indications
  - Uric acid levels usually decrease within 4 hours of injection
- **FDA approved dosing**
  - 0.2 mg/kg IV as a 30 minute infusion daily for up to 5 days
- **NCCN dosing recommendations**
  - One dose frequently adequate
  - Doses of 3 – 6 mg IV usually effective

(Sanofi-Aventis US (2016) Elitek Package Insert.; NCCN, 2016).

Tumor Lysis Syndrome

Metabolic Abnormality	Management
Hyperuricemia	• Hydration, urinary alkalinization • Oral allopurinol or IV allopurinol • Rasburicase • Hemodialysis for significant renal compromise
Hyperkalemia	**Mild (Potassium<6.5 mEq/L):** • Sodium polystyrene sulfonate orally or by enema **Potassium >6.5 mEq/L or cardiac changes:** • IV calcium gluconate or calcium carbonate • IV sodium bicarbonate, hypertonic glucose & insulin accompanied by sodium polystyrene sulfonate • Loop diuretics & aggressive hydration

(Holmes Gobel, 2013)

Tumor Lysis Syndrome

Metabolic Abnormality	Management
Hyperphosphatemia	• Phosphate-binding agents • Aluminum-containing antacids • Hypertonic glucose plus insulin • Aggressive hydration
Hypocalemia	• Appropriate management of hyperphosphatemia • IV calcium gluconate or calcium chloride to treat arrhythmias

(Holmes Gobel, 2013)

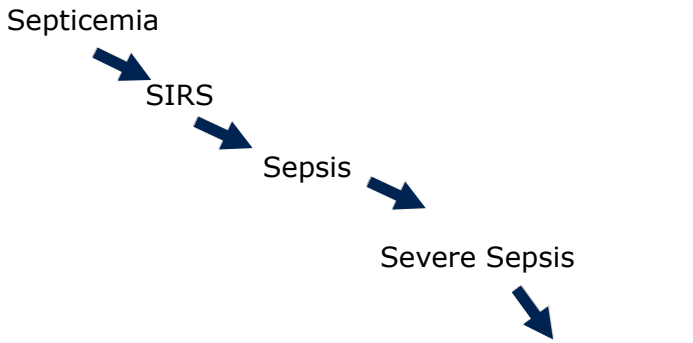
Tumor Lysis Syndrome

- **Nursing Interventions:**
- **Can you name a few?!**
  - For monitoring
  - For managing fluid balance
  - For patient and family education

Septic Shock

Septic Shock

- **Occurs on a continuum!!**



(Maloney, 2016)

Septic Shock

- **Septicemia**: Invasion of blood by microorganisms
- **Sepsis**: Systemic response to infection (vasodilation, displacement of intravascular volume)
- **Septic Shock**: Vascular collapse caused by vasodilation, leakage intravascular volume into interstitial space

What are some of the criteria used to identify where a patient may be on the SIRS continuum?

(Maloney, 2016)

Septic Shock

- **Risk Factors:**
  - Neutropenia
  - Infection
  - Medical devices
  - Mucositis
  - Hospitalization
  - Corticosteroids or other immunosuppressants
  - Splenectomy
  - Age
  - Poor nutritional status
  - Concurrent immunosuppressive disease
  - Type of malignancy

(Maloney, 2016)

Septic Shock

- **Pathophysiology:**

1. Infection (can be bacterial, viral, or fungal)
2. Endotoxins and other cellular components released
3. Vasodilation
4. Increased vascular permeability
5. Decreased arterial/venous tone
6. Clot formation
7. End-organ damage
8. Cell death

(Maloney, 2016)

Septic Shock

- **Clinical Presentation:**

Sepsis
- confusion, agitation
- tachycardia, hypotension
- tachypnea, hypoxia on RA, decreased breath sounds
- decreased UO
- warm, dry, flushed skin
- nausea/vomiting

→ Septic shock
- obtunded, coma
- arrhythmias, tachycardia, hypotensive
- SOB, decreased breath sounds, crackles/wheezes, ARDS, pulmonary edema
- oliguria or anuria, ARF
- cold, pale, mottled skin
- decreased GI motility, jaundice

(Maloney, 2016)

Septic Shock

Laboratory manifestations:

Sepsis
- long PT and aPTT
- decreased fibrinogen and platelets
- hyperglycemia
- leukocytosis
- elevated lactic acid
- + blood cultures
- WBCs in urine

Septic shock
- elevated LFTs
- increased BUN and/or creatinine
- decreased hematocrit and/or hemoglobin
- hypoglycemia

(Maloney, 2016)

Septic Shock

- **Diagnosis:**
- BMP
- CBC
- Coagulation studies
- Lactic Acid
- ABGs
- Anything else to help determine potential site of infection

Like what?

(Maloney, 2016)

Septic Shock

- **Managment:**
- Fluid resuscitation (crystalloids)
- Broad spectrum antibiotics (within 45 minutes)
- Oxygen therapy
- Vasopressor therapy
- Supportive therapy

Like what?

Septic Shock

- **Nursing Management:**

- Prevention and early recognition!!!!
- Frequent vitals and assessments
- Maintain adequate oxygenation
- Administer fluids and antibiotics on time
- Educate!

(Maloney, 2016)

Disseminated Intravascular Coagulation (DIC)

Disseminated Intravascular Coagulation

- **Definition:** Generalized activation of the hemostatic system, which results in widespread intravascular deposition of fibrin in the microvasculature and the simultaneous consumption of coagulation factors and platelets.

- DIC is never a primary diagnosis. It ALWAYS is a symptom of an underlying disease.

(Maloney, 2016; Viele, 2008)

Disseminated Intravascular Coagulation

Causes:

- Sepsis
- Severe infection
- Vascular abnormalities
- Severe allergic reactions
- Severe immunologic reactions
- Malignancy (both solid and liquid)

(Maloney, 2016; Viele, 2008)

Disseminated Intravascular Coagulation

- **Basic pathophysiology:**

- Overactivation of coagulation cascade from certain proteins
  - can be intrinsic (blood vessel damage)
  - can be extrinsic (tissue damage)
- Clots begin to form and are deposited throughout the body's vasculature
- Because of excessive clotting, clotting factors and platelets are all used up!
- This means there is no more clotting factors and platelets for normal clotting anymore, which allows for abnormal bleeding!

(Maloney, 2016; Viele, 2008)

Disseminated Intravascular Coagulation

- **Clinical Presentation:**
- Skin: pallor, petechiae, jaundice, ecchymosis, hematomas, acral cyanosis
- EENT: visual disturbances, scleral injection, periorbital edema, subconjunctival hemorrhage, eye and ear pain, petechiae on nasal and/or oral mucosa, epistaxis, tender and bleeding gums
- Cardiac: tachycardia, hypotension, weak peripheral pulses, color and temperature changes to extremities
- Respiratory: dyspnea, tachypnea, hypoxia, hemoptysis, cyanosis, SOB

(Maloney, 2016; Viele, 2008)

Disseminated Intravascular Coagulation

Clinical Presentation (continued):

- GI: tarry stools, hematemesis, abdominal pain, abdomina distension, guiac positive stools
- GU: hematuria, decreased UO
- Musculoskeletal: joint pain and stiffness
- Neuro: headache, restlessness, confusion, lethargy, altered LOC, obtundation, seizures, coma

(Maloney, 2016; Viele, 2008)

Disseminated Intravascular Coagulation

Diagnosis:

- clotting studies: platelet count, fribrinogen level, thrombin level
- clotting factors studies: PT, aPTT, INR
- fibrinolysis studies: fibrin degradation products, D-dimer, antithrombin
- Bilirubin
- BUN

(Maloney, 2016; Viele, 2008)

Disseminated Intravascular Coagulation

Diagnosis:

Platelet	Decreased
Fibrinogen	Decreased
Thrombin	Prolonged
Prothrombin Time	Prolonged
Activated	Prolonged
Fibrin	Increased
Antithrombin	Decreased

(Viele, 2008)

Disseminated Intravascular Coagulation

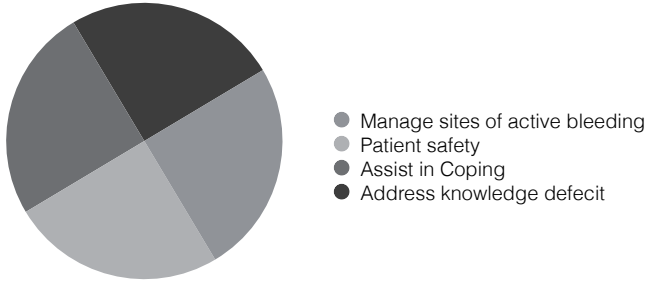
Managment:
1. Treat underlying cause!!

- May include:
  - Transfusions (platelets, FFP, cryopreciptate)
  - Anticoagulants
  - Fibrinolytic agents
  - Anticoagulant factor concentrates

(Maloney, 2016; Viele, 2008)

Disseminated Intravascular Coagulation

Nursing Management



(Vogel, 2016)

Hypercalcemia

Hypercalcemia

- **Definition:** abnormally high levels of calcium (>10.5mg/dL)
- Most common oncologic emergency!
  - -Occurs in 10-20% of all cancer patients
- Considered an emergency because, left untreated 50% of cases progress to renal failure, dehydration, coma, and death within days to weeks.

(Maloney, 2016; Jensen, 2008)

Hypercalcemia

- **Risk Factors:**
  - Solid tumors (lung and breast)
  - Liquid tumors (multiple myeloma)
  - Non-malignant conditions
  - Immobility
  - Use of thiazide diuretics
  - Overuse of dietary supplements

(Maloney, 2016; Jensen, 2008)

Hypercalcemia

- **Pathophysiology:**

Humoral (80%):
- Tumor cells produce PTH-rP
- Mimics real PTH
  - bone reabsorption of calcium
  - renal reabsorption of calcium
- PTH-rP is NOT controlled by the feedback mechanism that normal PTH is

Osteolytic (20%):
- Malignant cells invade and destruct bone
- These tumor cells release a variety of cytokines that promote calcium reabsorption
- Osteoclasts are also active at the cites of tumor cells, furthering calcium reabsorption

- Also, in certain lymphomas, vitamin D is converted into its active from which promote calcium absorption in the GI tract.

(Maloney, 2016; Jensen, 2008)

Hypercalcemia

- **Clinical Presentation:**

Mild (10.5-11.5mg/dL)
- GI: anorexia, N/V, abd cramping, low appetite
- Neuro: restlessness, poor concentration, lethargy, confusion
- Muscular: fatigue and weakness
- Renal: frequent urination, nocturia, polydipsia
- Cardio:orthostatic hypotention

Moderate (11.5-13.5mg/dL)
- GI: constipation, bloating, abd pain
- Neuro: psychosis, drowsiness, AMS
- Muscular: continued increased weakness
- Renal: dehydration
- Cardio:hypertension, ECG changes (long PR interval, wide QRS, short QT, short ST), arrhythmias,

Severe (>13.5mg/dL)
- GI: ileus
- Neuro: seizures, coma
- Muscular: ataxia and pathologic fractures
- Renal: oliguric renal failure and renal insufficiency
- Cardio: ECG changes (wide T waves, heart block, ventricular arrhythmias), cardiac arrest

(Maloney, 2016)

Hypercalcemia

- **Diagnosis:**

- BMP/CMP (including serum calcium)
- Serum albumin and prealbumin
- PTH

(Maloney, 2016)

Hypercalcemia

- **Management:**
- Tumor suppression is the only long term measure for reversal
- Hydration (usually 0.9% NS and 3-4L/day PO intake)
- Remove drugs that worsen hypercalcemia (thiazide diuretics)
- Diuresis (loop diuretics)
- Biphoshponates (zometa > pamidronate)
- Additional pharmacologic options
- Dietary recommendations

(Maloney, 2016; Jensen, 2008)

Hypercalcemia

- **Nursing Management:**
- Early recognition!
- Administer all necessary active treatment modalities
- Intervene to maintain patient safety, particularly if confused
- Intervene to maintain patient activity level
  - PT/OT
  - Frequent fall risk assessment
- Manage and monitor fluid and electrolyte balance

(Maloney, 2016; Jensen, 2008)

Anaphylaxis

Anaphylaxis

- **Definition:** an allergic reaction that potentiates a life-threatening emergency
- Can be generalized or localized.
- The most rapid and sever hypersensitivity reaction.

(Maloney, 2016)

Anaphylaxis

- **Risk Factors:** various antigens and routes of exposure

- Allergy testing
- Antibiotics
- Anesthetics
- Antineoplastics
- Blood products
- Insect venom
- Latex
- Contrast media for radiographic tests
- Foods
  - egg
  - fish
  - additives
  - peanuts
  - shellfish
  - milk

(Maloney, 2016)

Anaphylaxis

- **Pathophysiology:**

- First exposure to antigen causes IgE antibody to develop
- Upon repeat exposure to antigen, IgE antibody binds to and activates mast cells and basophils
- Inflammatory mediators are then triggered to release:
  - histamine
  - leukotrienes
  - prostaglandins
  - platelet activating factor
- These substances lead to:
  - systemic vasodilation
  - increased capillary permeability
  - bronchoconstriction
  - coronary vasoconstriction

(Maloney, 2016)

Anaphylaxis

- **Clinical Presentation:**

- Derm: flushing, itching, urticaria, morbilliform rash, angioedema
- Ophthalmologic: periorbital edema, infected conjunctiva, tears
- Resp: bronchospasm, chest tightness, tachypnea, throat/nasal itching, congestion, sneezing, dysphonia, horseness, dry cough, stridor, cyanosis, respiratory arrest
- GI: nausea, vomiting, diarrhea, abdominal pain
- Cardio: chest pain, tachycardia, diaphoresis, hypotension, cyanosis, dysrhythmias, palpitations, shock
- Neuro: headache, dizziness, uneasiness, lightheadedness, confusion, tunnel vision, loss of consciousness
- Other: metallic taste, feeling of impending doom

(Maloney, 2016)

Anaphylaxis

- **Managment:**

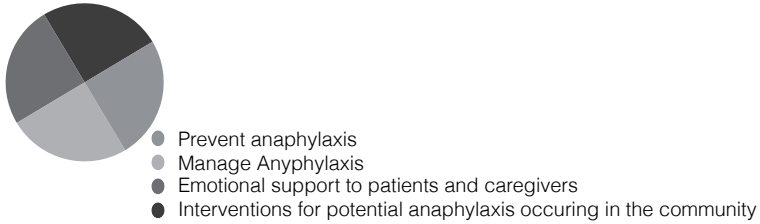
1. Epinepherine!!!

- Additionally:
  - Oxygen therapy (100% non rebreather)
  - 0.9% NS given as fast as possible
  - corticosteroids
  - H1 receptor anatagonist

(Maloney, 2016)

Anaphylaxis

Nursing Management



(Vogel, 2016)

Increased Intracranial Pressure

Increased Intracranial Pressure

- **Definition:** a potentially life-threatening neurologic even that occurs with an increase in brain tissue, blood, cerebrospinal fluid, or all of these thing within the intracranial cavity, resulting in nerve cell damage, permanent neurologic defects, and/or death.

(Vogel, 2016)

Increased Intracranial Pressure

- **Oncology specific causes:**
- Tumors found within the intracranial cavity
- Leptomeningeal metastases
- Blood clots
- Infection
- Metabolic disorders

(Vogel, 2016)

Increased Intracranial Pressure

Oncology specific risk factors:

- Breast, lung, kidney, melanoma
- Leukemia, lymphoma, or neuroblastoma
- Primary tumors to the brain or spinal cord
- Thrombocytopenia, platelet dysfunction, or DIC
- Infection while immunocompromised
- SIADH
- History of radiation
- Occluded Ommaya reservoir

(Vogel, 2016)

Increased Intracranial Pressure

- **Pathophysiology:**
- Volume of blood, brain tissue, or cerebrospinal fluid increases
- Compensatory mechanisms take over
- Once these mechanisms fail, ICP increases and displacement or edema of brain tissue occurs
- CSF outflow becomes obstructed
- Cerebral perfusion decreases
- Brain injury and tissue necrosis

(Vogel, 2016)

Increased Intracranial Pressure

- **Clinical Presentation:**

Early:

- Headaches
- Visual disturbances
- Lethargy, apathy, confusion, restlessness
- Speech disturbances
- Decreased LOC
- Loss of appetite
- Nausea and vomiting

Late:

- Widening pulse pressure, increased BP, bradycardia
- Shallow respirations, Cheyne-Stokes respirations
- Papilledemia, poor concentration, decreased LOC, personality changes, hemiplegia, hemiparesis, seizures, pupillary changes
- GCS < 8
- Abnormal posturing
- Temperature elevations
- Cushing triad

(Vogel, 2016)

Increased Intracranial Pressure

- **Nonpharmacologic Management:**

- Surgery
- Hyperventilation
- Radiation therapy
- Removal of offending agent

(Vogel, 2016)

Increased Intracranial Pressure

Pharmacologic Management:

Chemotherapy or targeted therapy	Corticosteroids
Osmotherapy	Anticonvulsant therapy

(Vogel, 2016)

Increased Intracranial Pressure

- **Nursing Management**

(Vogel, 2016)

Spinal Cord Compression

Spinal Cord Compression

- **Definition:**
- A neurological emergency where the spinal cord or caudal equine is compromised by direct pressure, vertebral collapse, or both cause a direct extension or metastatic spread of malignancy.

(Schulmeister & Gatlin, 2008; Vogel, 2016)

Spinal Cord Compression

Cancers associated with spinal cord compression

Spinal Level	% Involvement	Associated Cancers
Cervical	10	Lung, breast, kidney, lymphoma, myeloma, melanoma
Thoracic	70	Lung, breast, kidney, lymphoma, myeloma, prostate
Lumbosacral	20	Lung, breast, kidney, lymphoma, myeloma, melanoma, prostate, GI

(Schulmeister & Gatlin, 2008)

Spinal Cord Compression

- **Risk Factors:**
- Cancers that have a natural history of spreading to the bone
- Cancers that have a natural history of spreading to the brain and spinal cord
- Primary cancers of the spinal cord
- History of vertebral compression fractures

(Vogel, 2016)

Spinal Cord Compression

- **Pathophysiology:**
- Compression of spinal cord
  - Direct tumor pressure on cord
  - Tumor invasion of the vertebral column causing collapse & pressure on cord
- Compression leads to:
  - Edema
  - Inflammation
  - Mechanical compression
- Resulting in:
  - Direct neural injury to cord
  - Vascular Damage

(Kaplan, 2013)

Spinal Cord Compression

- **Clinical Presentation:**
- 90% of patients with SCC experience back pain as the first symptom.

Early signs:

- Neck pain
- Motor weakness and dysfunction
- Sensory loss

Late signs:

- Loss of sensation for deep pressure, vibration, and position
- Incontinence or retention
- Impotence
- Paralysis
- Muscle atrophy
- Loss of sweating below lesion

(Vogel, 2016)

Spinal Cord Compression

- **Back pain characteristics:**
- Localized: usually occurs at level of lesion, described as dull and constant, more severe with movement, coughing, weight bearing, during a Valsalva maneuver
- Radicular: along dermatomes
- Referred: in a non-radicular pattern
- May be a combination of all three!

(Schulmeister & Gatlin, 2008)

Spinal Cord Compression

- **Diagnosis:**
- MRI
  - Gold standard for diagnosis
  - Accurate, sensitive, and specific diagnostic for malignant spinal cord compression
- Other diagnostic tests
  - Spinal x-rays
  - CT scan
  - Bone scan and/or PET scan

(Vogel, 2016)

Spinal Cord Compression

- **Treatment:**
- Immediate and aggressive!

Nonpharmacologic:
- Radiation
- Surgery
- Surgery followed by radiation

Pharmacologic:
- Corticosteroids
- Chemotherapy
- Analgesics
- Bone remodeling agents

(Vogel, 2016)

Spinal Cord Compression

- **Nursing Management:**

- Manage pain and increase comfort
- Promote physical mobility
- Improve or maintain neurologic function
- Improve or maintain skin integrity
- Increase knowledge of disease process and therapeutic interventions
- Preserve self-image and role performance

(Vogel, 2016)

Cardiac Tamponade

Cardiac Tamponade

- **Definition:** A life-threatening emergency where an excessive amount of fluid accumulates in the pericardial sac that prevents full contraction and relaxation of the heart, decreasing cardiac output and function.

(Camp-Sorrell, 2008; Vogel, 2016)

Cardiac Tamponade

- **Risk factors and causes:**

- Primary tumors of the heart
- Tumors that are metastatic to the heart
- Having received > 4000 cGy radiation to a field that included the heart
- Comorbidities
- Trauma
- CVC perforation
- Infection
- Chemotherapy or biotherapy that increases capillary permeability
- Obstruction of mediastinal lymph nodes

(Camp-Sorrell, 2008; Vogel, 2016)

Cardiac Tamponade

- **Pathophysiology:**

1. Increase of intrapericardial pressure occurs (depending on cause)
2. Cardiac chambers are compressed and LV filling decreases
3. Pumping function of heart declines
4. CO decreases and BP drops
5. Impaired systemic perfusion occurs, possibly leading to cariogenic shock

(Vogel, 2016)

Cardiac Tamponade

- **Diagnosis:**

- CXR
- CT
- Echocardiogram
- EKG
- MRI
- Pericardiocentesis and cytology

(Vogel, 2016)

Cardiac Tamponade

- **Early Clinical Presentation:**
  - Retrosternal chest pain
  - Dyspnea with exertion
  - Muffled heart sounds
  - Tachycardia
  - Fatigue and malaise
  - Vague RUQ abdominal pain
  - Mild jugular vein distention
  - Or … may be asymptomatic!

(Vogel, 2016)

Cardiac Tamponade

Late Clinical Presentation:

Tachycardia	Tachypnea	Low SBP and high DBP	Pulsus paradoxus
Increased CVP	Altered LOC	Oliguria	Peripheral edema
Diaphoresis	Cyanosis	Anxiety, agitation, and confusion	Hiccups
Hoarseness and dysphagia	Beck triad	Chest pain	Increased JVD

(Vogel, 2016)

Spinal Cord Compression

Treatment:

Goal is symptomatic relief

Nonpharmacologic:

- Pericardiocentesis
- Pericardial window
- Total pericardectomy
- Percutaneous balloon pericardiotomy
- Radiation

Pharmacologic:

- Pericardial sclerosis
- Chemotherapy
- Corticosteroids

(Vogel, 2016)

Spinal Cord Compression

Nursing Management:

- Maintain cardiac output
- Maintain optimal respiratory function
- Increase comfort
- Decrease anxiety
- Increase knowledge of disease process and therapeutic interventions
- Reduce risk for injury

(Vogel, 2016)

Superior Vena Cava Syndrome

Superior Vena Cava Syndrome

- **Definition:** Describes a pattern of physical findings that results from the obstruction of blood flow through the superior vena cava, due to tumor or thrombus, compromising venous drainage from the head, neck, upper extremities, and thorax.

(Mack & Becker, 2008; Vogel, 2016)

Superior Vena Cava Syndrome

- **Risk Factors:**
- Malignant diagnosis
- Presence of a CVC and/or pacemaker
- History of radiation to the mediastinum
- Other associated conditions
    - mediastinal fibrosis
    - fungal infection
    - aortic aneurysm
    - benign mass

(Mack & Becker, 2008; Vogel, 2016)

Superior Vena Cava Syndrome

- **Pathophysiology:**

1. Obstruction of the SVC occurs (depending on cause)
2. Venous pressure and congestion in the head, neck, thorax, upper extremities, and throat increases
3. Decreased cardiac filling and output occurs
4. Blood flow is diverted to smaller collateral vessels

(Mack & Becker, 2008; Vogel, 2016)

Superior Vena Cava Syndrome

- **Diagnosis:**

- CT
- MRI
- PET
- Contrast venography
- CXR

(Vogel, 2016)

Superior Vena Cava Syndrome

Early Clinical Presentation: Symptoms are more pronounced in AM or when bending over.

- Redness and edema in conjunctiva and around eyes and face
- Swelling of neck, arms, and hands
- Neck and thoracic vein distention
- Dyspnea
- Nonproductive cough
- Hoarseness, occasionally dysphagia
- Cyanosis of upper torso
- Nasal stuffiness and head fullness
- Breast swelling

(Vogel, 2016)

Superior Vena Cava Syndrome

Late Clinical Presentation:

- Symptoms of ICP
- Irritiability, altered mental status
- Stridor, signs of CHF
- Tachcardia, tachypnea, orthopnea
- Hypotension, no peripheral pulses
- Dysphagia, hoarseness, hemoptysis
- Progressive cyanosis, facial edema
- Horner syndrome

(Vogel, 2016)

Superior Vena Cava Syndrome

Treatment: Goal is relief of obstruction and addressing of underlying cause. Determined by rate of onset.

Nonpharmacologic:

- Radiation
- Removal of CVC*
- Percutaneous intravascular stent placement
- Surgical reconstruction of SVC
- Oxygen therapy

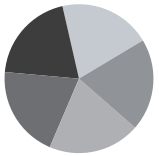
Pharmacologic:

- Chemotherapy
- Chemotherapy + radiation
- Corticosteroids
- Diuretics
- Thrombolytic therapy

(Vogel, 2016)

Superior Vena Cava Syndrome

Nursing Management:



- Maintain adequate gas exchange
- Maintain adequate cardiac output
- Decrease anxiety
- Increase knowledge of disease process and therapeutic interventions
- Prevent injury

(Vogel, 2016)

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