Oncologic Emergencies

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CENTRAL CONNECTICUT CHAPTER

Objectives

- Outline the pathophysiology of oncologic emergencies.
- Describe management strategies of oncologic emergencies.
- Apply management strategies of oncologic emergencies to case studies.

Introduction

Metabolic	Structural
Sepsis	Cardiac tamponade
Disseminated Intravascular Coagulation	Increased intracranial pressure
Syndrome of Inappropriate Antidiuretic Hormone Secretion	Spinal cord compression
Hypercalcemia	Superior vena cava syndrome
Tumor Lysis Syndrome	Bowel Obstruction & Perforation
Hypersensitivity & Anaphylaxis	Pneumonitis

Sepsis

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What is Sepsis?

Sepsis occurs when chemicals released into the bloodstream to fight an infection trigger inflammatory responses throughout the body. This inflammation can trigger a cascade of changes that can damage multiple organ systems, causing them to fail (Mayo Clinic, 2018).

GET AHEAD OF SEPSIS

KNOW THE RISKS. SPOT THE SIGNS. ACT FAST.

FOR PATIENTS AND FAMILIES

PROTECT YOURSELF AND YOUR FAMILY FROM SEPSIS.

WHAT IS SEPSIS?

Sepsis is the body's extreme response to an infection. It is a life-threatening medical emergency. Sepsis happens when an infection you already have triggers a chain reaction throughout your body. Infections that lead to sepsis most often start in the lung, urinary tract, skin, or gastrointestinal tract. Without timely treatment, sepsis can rapidly lead to tissue damage, organ failure, and death.

IS SEPSIS CONTAGIOUS?

You can't spread sepsis to other people. However, an infection can lead to sepsis, and you can spread some infections to other people.

WHAT CAUSES SEPSIS?

Infections can put you or your loved one at risk for sepsis. When germs get into a person's body, they can cause an infection. If you don't stop that infection, it can cause sepsis. Bacterial infections cause most cases of sepsis. Sepsis can also be a result of other infections, including viral infections, such as COVID-19 or influenza.

Anyone can get an infection, and almost any infection, including COVID-19, can lead to sepsis. In a typical year:

- At least **1.7 million** adults in America develop sepsis.
- Nearly **270,000** Americans die as a result of sepsis.
- **1 in 3** patients who dies in a hospital has sepsis.
- Sepsis, or the infection causing sepsis, starts outside of the hospital in nearly **87%** of cases.

WHO IS AT RISK?

Some people are at higher risk for sepsis:

- **65+** Adults 65 or older
- People with weakened immune systems
- People with chronic medical conditions, such as diabetes, lung disease, cancer, and kidney disease
- People with recent severe illness or hospitalization
- Sepsis Survivors
- Children younger than one

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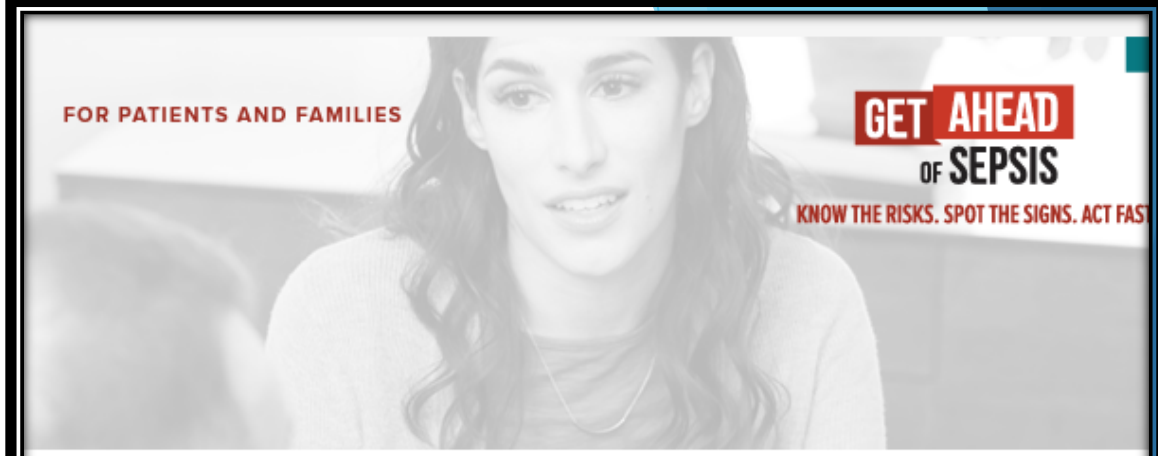
www.cdc.gov



FOR PATIENTS AND FAMILIES

GET AHEAD OF SEPSIS

KNOW THE RISKS. SPOT THE SIGNS. ACT FAST

HOW CAN I GET AHEAD OF SEPSIS?

As a patient, you can take specific steps to reduce your risk of sepsis, including caused by COVID-19, such as:

1 PREVENT INFECTIONS

Talk to your healthcare professional about steps you can take to prevent infections that can lead to sepsis.

- Take good care of chronic conditions
- Get recommended vaccines

2 PRACTICE GOOD HYGIENE

Remember to wash your hands and keep cuts clean and covered until healed.

- Wash your hands
- Keep cuts clean and covered until healed

3 KNOW THE SIGNS AND SYMPTOMS

A patient with sepsis might have one or more of the following signs or symptoms. A medical assessment by a healthcare professional is needed to confirm sepsis.

- High heart rate or low blood pressure
- Fever, or shivering, or feeling very cold
- Confusion or disorientation
- Shortness of breath
- Extreme pain or discomfort
- Clammy or sweaty skin

4 ACT FAST

Sepsis is a medical emergency. ACT FAST.

If you or your loved one has an infection that's not getting better or is getting worse, ACT FAST. Get medical care IMMEDIATELY either in-person, or at minimum, through telehealth services. Ask your healthcare professional, "Could this infection be leading to sepsis?" and if you should go to the emergency room for medical assessment.

Understanding Sepsis

Early identification of the pathways of sepsis is key!

SIRS – Systemic Inflammatory Response Syndrome

SIRS Criteria
Any of the TWO Following:

- ❑ Temp >100.9°F or <96.8° F
- ❑ HR > 90 beats per minute
- ❑ Respirations > 20/min
- ❑ WBC > 12,000 or < 4000; or 10% Bands on differential

RN's will often be the **FIRST** to recognize potential sepsis in a patient. Time is critical…
Do not delay!

- Notify the provider that patient meets SIRS criteria.
- Expect orders for **Blood Cultures** and **Lactic Acid**

Blood Cultures

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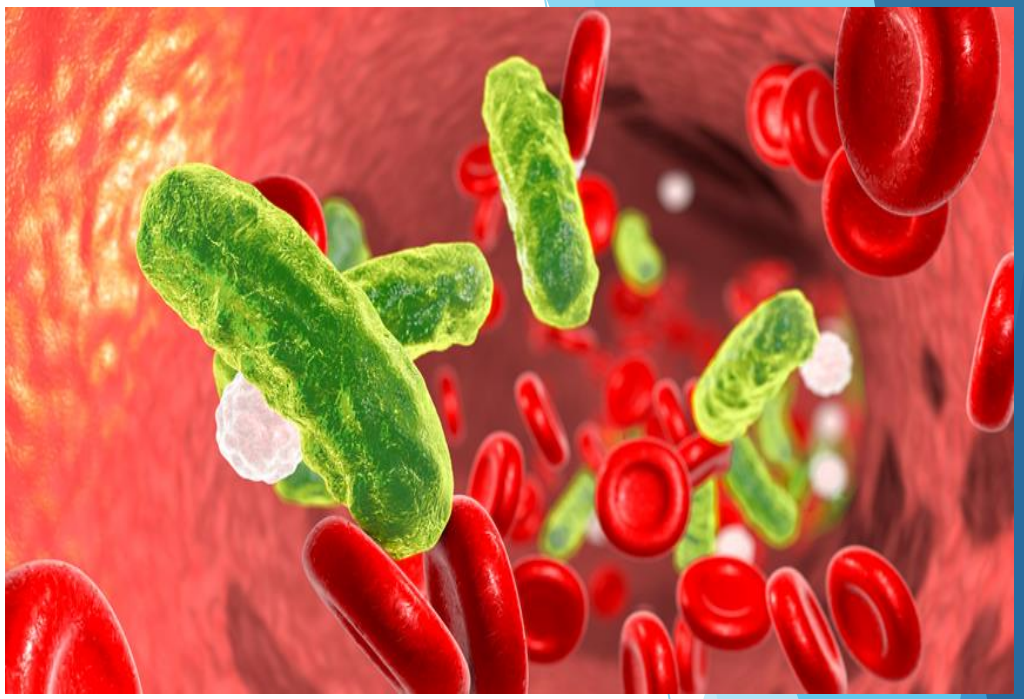
Sepsis

- **Sepsis** is Systemic Inflammatory Response Syndrome (**SIRS**) due to an infection ***without*** organ dysfunction.
  - **Early detection** of sepsis is ***vital*** in stopping the progression of Severe Sepsis and Septic Shock.



Nursing Considerations:

- Monitor Temp, HR, respirations, WBC counts
- Assess patient for possible sources/signs of infection
- Check blood pressure
- Notify provider. Anticipate sepsis care orders

Examples of Infection:

- Pneumonia/ Respiratory
- UTI/ GU/ GI
- Skin/Wound Infection
- Abscess
- Joint Infection
- Pressure Ulcers, Lines, Drains, Incisions, Wounds

***Documented POSITIVE Cultures**

- **Blood**
- **Urine**
- **Other**

Severe Sepsis

Severe Sepsis is SIRS due to an infection ***with*** organ dysfunction

SEVERE SEPSIS INTERVENTIONS:
(within 3 hours of time zero)

- **Initial Lactate Level drawn**
- Blood cultures x2
- Administer antibiotics
- NS or LR **@ 30mL/kg** for SBP<90 or MAP <65

If Lactic Acid >2.0 → Lactic Acid Level Needs to be Repeated within ***FOUR HOURS*** of initial value.

Notify Provider of symptoms and possible Severe Sepsis Criteria.

Time Zero – Onset of Severe Sepsis

- Must have all three clinical criteria occurring **within 6hrs** of each other.
- Last criterion determines "time zero".

Criteria for Severe Sepsis

1. Documentation of suspected/actual infection by MD/APRN/PA
2. 2 or more SIRS Criteria:
    1. T>100.9 (38.3c) or <96.8 (36.0c)
    2. HR >90
    3. RR>20
    4. WBC>12K or < 4K or >10% bands
3. One or more s/s of Organ Dysfunction: (*not due to chronic condition*)
    1. SBP < 90 or MAP < 65
    2. Creatinine > 2.0
    3. Bilirubin > 2.0 mg/dL
    4. Platelet Ct < 100,000
    5. INR > 1.5 or a PTT > 60 sec
    6. Lactate > 2.0 mmol/L
    7. Acute resp failure with intubation, CPAP, or BiPAP

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Septic Shock

Septic Shock is Severe Sepsis ***with*** acute organ dysfunction ***and*** circulatory failure.

Time Zero – Onset of Septic Shock

- Presence of Severe Sepsis clinical criteria ***AND*** persistent hypotension 1 hour after 30ml/kg crystalloid fluid administration

OR

- Presence of Severe Sepsis clinical criteria ***AND*** Lactate level >=4.0 mmol/L (even if normal BP)

SEPTIC SHOCK INTERVENTIONS:

- If not already done:
  - **Lactic level, Blood Cultures x2, Broad spectrum antibiotics** (within 3 hours)
  - **NS or LR @30 mL/kg for SBP <90, MAP<65, or Lactate >=4.0** (within 3 hrs)
- **Vasopressor admin** (within 6 hrs)

Patient will likely need Critical Care

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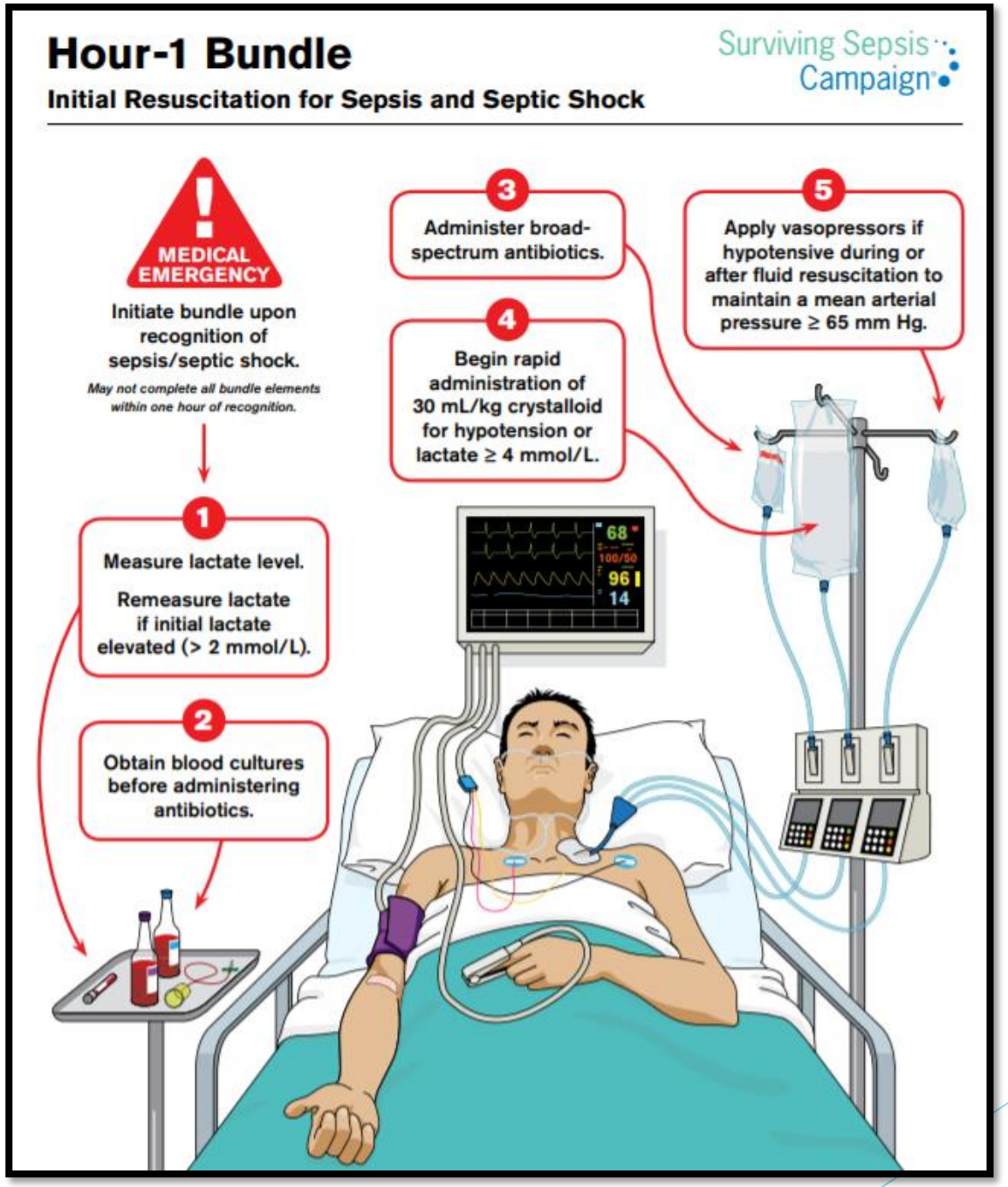
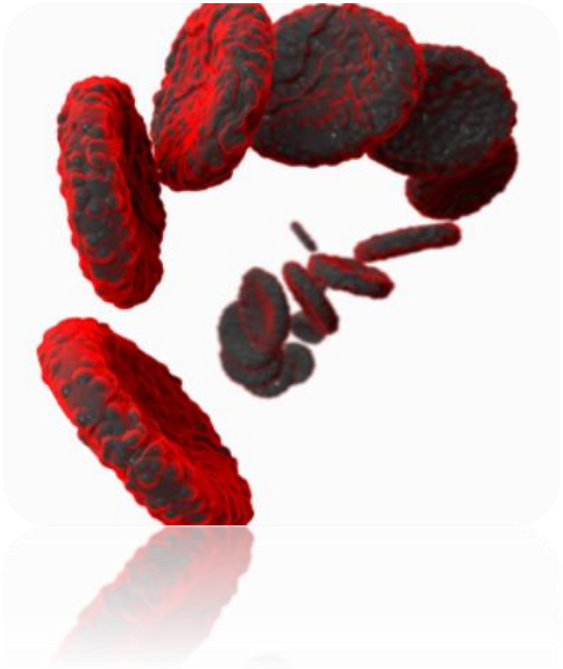

Hour-1 Bundle
Initial Resuscitation for Sepsis and Septic Shock
Surviving Sepsis Campaign
MEDICAL EMERGENCY
Initiate bundle upon recognition of sepsis/septic shock.
May not complete all bundle elements within one hour of recognition.
1
Measure lactate level.
Remeasure lactate if initial lactate elevated (> 2 mmol/L).
2
Obtain blood cultures before administering antibiotics.
3
Administer broad-spectrum antibiotics.
4
Begin rapid administration of 30 mL/kg crystalloid for hypotension or lactate ≥ 4 mmol/L.
5
Apply vasopressors if hypotensive during or after fluid resuscitation to maintain a mean arterial pressure ≥ 65 mm Hg.

Disseminated Intravascular Coagulation (DIC)



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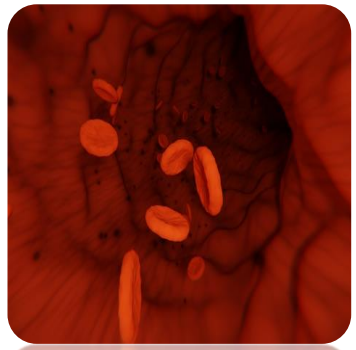
DIC

- DIC is usually secondary to an underlying disorder
  - Acute and chronic leukemia
  - Lymphomas
  - Solid cancers
  - Infection and sepsis
  - Transfusion reactions
  - Severe allergic reactions
  - Liver disease



- DIC is a bleeding disorder caused by a tendency to clot
  - A triggering factor produces damage to the small blood vessels of the body
  - The damage stimulates the coagulation pathways to form clots
  - The formation of clots depletes platelets and clotting factors

- Develops rapidly (within hours or days)
  - Excessive blood clotting in small vessels
  - Quickly leads to serious bleeding

https://www.msdmanuals.com/en-nz/professional/multimedia/video/v34912054

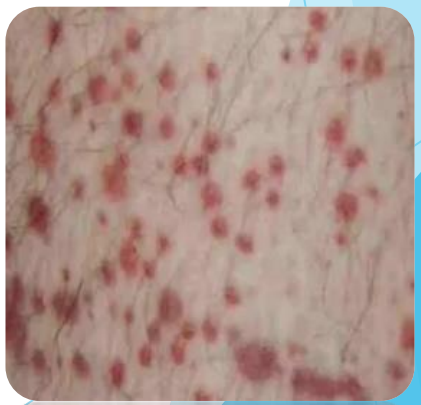
Symptoms

Lab findings:

Prothrombin time (PT)
Activated partial thromboplastin time (aPTT)
INR
D-dimer assay

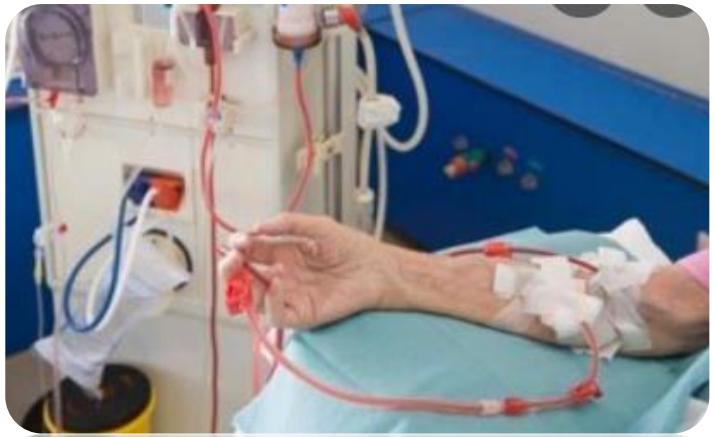
Platelets
Fibrinogen

- Petechiae, ecchymosis, bleeding, and hemorrhage
- Possible hypotension, tachycardia
- Hypoxia, dyspnea, tachypnea
- Abdominal pain and distention, tarry stools
- Hematuria
- Headache, change in mental status
- Thrombus formation: check for signs of DVT or renal impairment



Medical Management

- Treatment of underlying cause
- Fluid replacement
- Blood products – FFP, Platelets, PRBCs
- Management of bleeding
  - Fibrinolytic agents (e.g. aminocaproic acid, tranexamic acid/TXA)
- Management of clotting
  - Anticoagulants (e.g. Heparin, low-molecular weight heparin)
    - Platelets must be > 50,000
  - Plasmapheresis



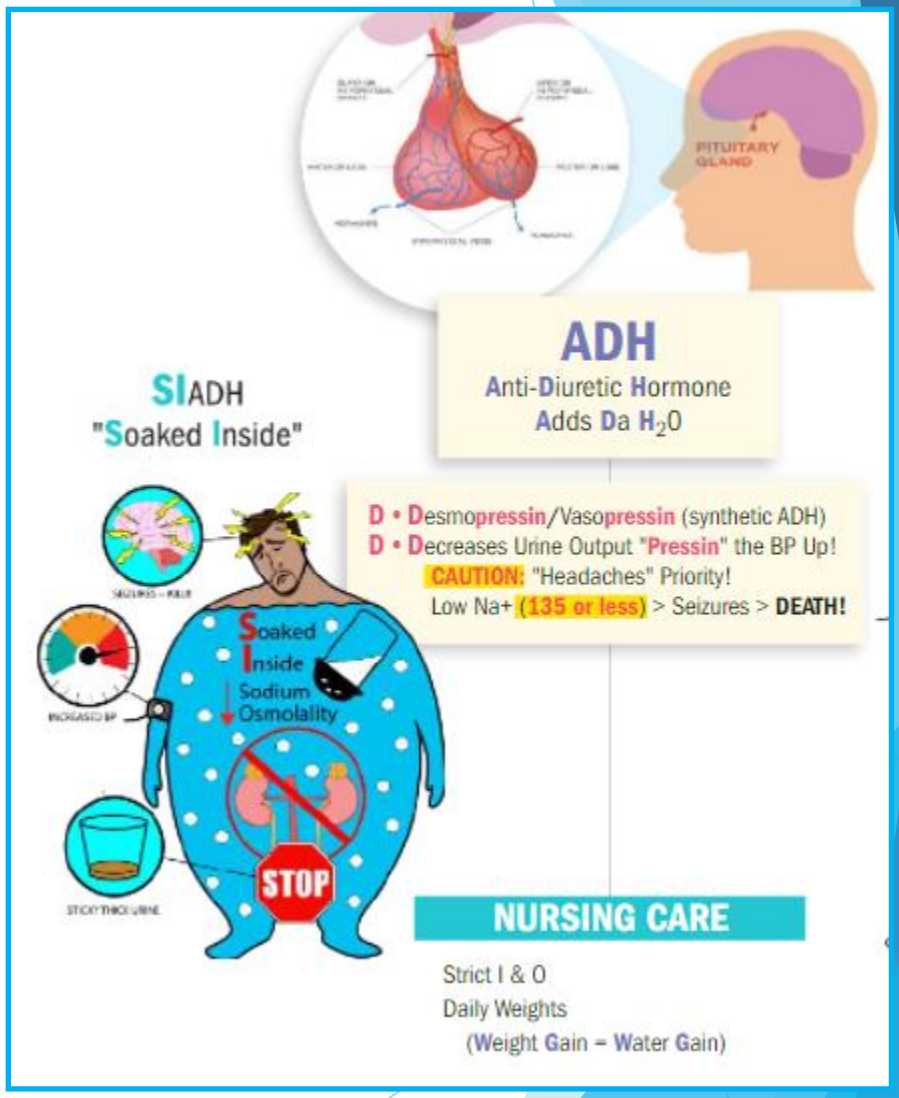
Nursing Care

- Nursing assessment includes:
  - Hemodynamic status
  - Oxygenation
  - Fluid status
  - Signs of bleeding or thrombosis
  - Tissue perfusion
  - Anxiety
- Patient education
  - Teach strategies to prevent or minimize the risk of bleeding
  - Review safety precautions
  - Avoid taking medications that could interfere with platelet function

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

ADH

- Secretion of ADH is regulated by receptors in the kidneys, heart, and hypothalamus in response to:
  - Elevated serum osmolality
  - Low blood pressure or volume
- Inappropriate ADH secretion
  - Malignancy (lung – esp. SCLC 15-50%, head and neck, brain, pancreas, colon, bladder, prostate, ovary, endometrium)
  - Severe brain trauma (surgery, trauma)
  - Drug induced (eg. vinca alkaloids, platinum compounds, cyclophosphamide, ifosfamide, melphalan, methotrexate, imatinib)



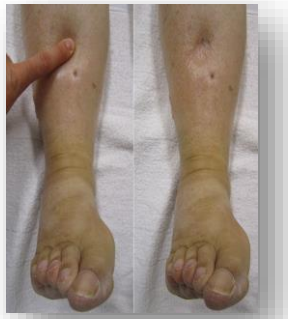
Symptoms

Worsen as sodium levels decrease!

- Thirst
- Anorexia
- Nausea
- Fatigue
- Headache
- Muscle cramps or weakness

Mild Hyponatremia
(131-134mEq/L)

- Weight gain
- Oliguria
- Increasing neurologic manifestations



Moderate hyponatremia
(126-130 mEq/L)

- Papilledema
- Delirium
- Hypoactive reflexes
- Ataxia
- Gait disturbances
- Seizures
- Coma
- Death

Severe hyponatremia
(<120 mEq/L)

Treatment

- Treat underlying cause
  - Malignancy or discontinue medication
- Free water restriction (500-1,000 mL/day)
- Demeclocycline – promotes water excretion
- Hydration with 3% hypertonic saline
- Loop diuretics (e.g. furosemide)
- Vasopressin receptor (V2) antagonists (e.g. tolvaptan, conivaptan) – inhibits effects of vasopressin water reabsorption

Nursing Management

- Early recognition of risk
- Monitor intake/output
- Daily weight
- Monitor lung sounds
- Monitor sodium levels
- Monitor mental status and neurologic signs
- Seizure precautions

Hypercalcemia

Hypercalcemia of Malignancy

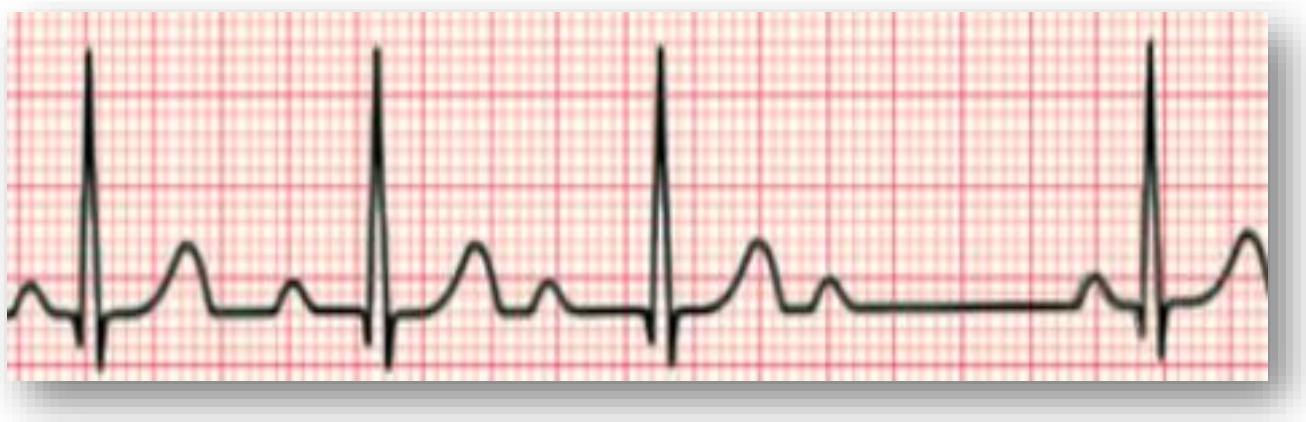
- Increased serum calcium levels (>10.5 mg/dL) in patients with cancer
- Most common oncologic emergency
  - ~20-30% of all cancer patients
  - Higher risk in solid tumors that metastasize to the bone (breast, lung, prostate), multiple myeloma, lymphoma
- Hypercalcemia may occur by several mechanisms:
  - Humoral: systemic release of parathyroid hormone-related protein by tumor resulting in bone resorption and renal retention of calcium
  - Osteolytic: Stimulation of osteoclasts by bone metastases leads to greater amount of calcium being released from bone than kidneys are able to excrete

    ***Primary cause**
  - Systemic secretion of vitamin D by tumor resulting in overproduction of calcium

Symptoms

- **Early:** Lethargy/weakness, nausea, constipation, polyuria, thirst, and hypoactive deep tendon reflexes

- **Late:** Mental status changes, ileus, heart block, renal failure, seizures, and coma



Management

- Aggressive hydration (3 - 4 L IV fluid/day) to facilitate calcium excretion
- Loop diuretics when patient is rehydrated to facilitate calcium excretion and prevent fluid overload
- Medications to prevent bone resorption (usually bisphosphonates)
  - Denosumab for patients with renal insufficiency
- Calcitonin to enhance calcium clearance in urine
- Glucocorticoids to reduce absorption of calcium through intestines
- Antineoplastic therapy to correct underlying cause of hypercalcemia
- Replace electrolytes lost to hydration and diuretic therapy

Tumor Lysis Syndrome (TLS)

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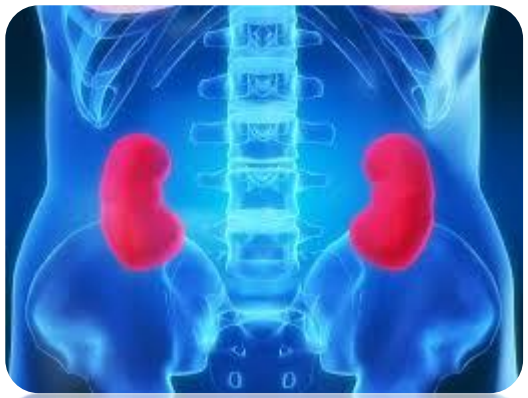
Pathophysiology

- Rapid breakdown of cells in response to anticancer treatment
- Intracellular contents released into circulation
- Body unable to maintain normal homeostasis because of excess cellular by-products
- Metabolic imbalances and multisystem organ dysfunction may result
- Can occur within 6 hours of cancer therapy initiation

Hyperuricemia

- Increased uric acid (>6.0 mg/dL females, >7.0 mg/dL males)
- Crystal precipitation can occur in the distal renal tubules resulting in decreased renal function
  - May lead to acute renal failure
- Assess for:
  - Nausea, vomiting, anorexia
  - Hematuria, flank pain, cloudy urine, sediment in urine
  - Fluid overload and oliguria



Hyperkalemia

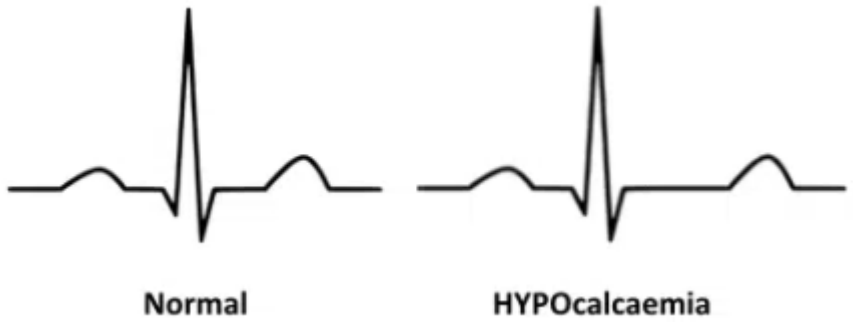
- Increased potassium (> 5.0 mEq/L)
- Can lead to life-threatening arrhythmias
- Assess for:
  - Nausea, vomiting, diarrhea, and anorexia
  - Muscle weakness, cramps, and parasthesias
  - Arrhythmias and EKG changes

Hyperphosphatemia

- Increased phosphorus (> 4.5 mg/dL)
- Malignant cells often contain more phosphorus (approx. 4x more than normal cells)
- Assess for:
  - GI symptoms: nausea, vomiting, and diarrhea
  - Lethargy
  - Seizures

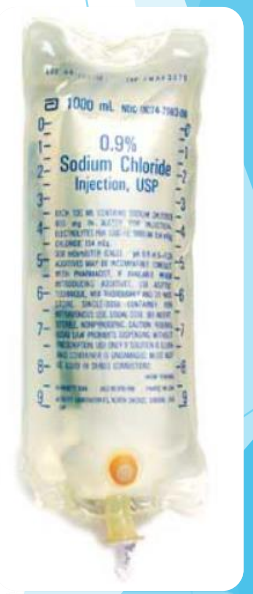
Hypocalcemia

- Decreased calcium (<9 mg/dL)
- As the amount of phosphorus in the bloodstream increases, it begins to bind with calcium, leading to a rapid drop in serum calcium levels
- Assess for:
  - Agitation
  - Hypotension
  - Severe muscle cramping, twitching, and tetany
  - Cardiac arrhythmias



Prevention is Key!!!

- Identification of high-risk patients
  - Those undergoing induction chemotherapy
  - Tumors with high proliferation rates (e.g. Burkitt lymphoma, ALL, AML, diffuse large B-cell lymphoma)
  - Tumors highly sensitive to chemotherapy
  - Large volume of disease
  - Decreased renal function prior to starting chemotherapy
- Monitoring of lab work
  - Potassium, phosphorus, calcium, uric acid, BUN, creatinine
- IV hydration is 24 to 48 hours before treatment
- Uric acid lowering agents (e.g. allopurinol, rasburicase, febuxostat)



Management

- Hyperkalemia
  - Mild: Hydration, loop diuretics, sodium polystyrene sulfonate, dietary restrictions
  - Severe: Add to treatment regimen: hypertonic glucose, insulin and sodium bicarbonate
- Hyperphosphotemia
  - Aggressive fluid resuscitation, phosphate-binding agents and oral aluminum-containing antacids
- Hypocalcemia
  - No treatment unless symptomatic
- Dialysis for acute renal failure

Hypersensitivity & Anaphylaxis

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Pathophysiology

Hypersensitivity Reaction

Mediated by IgE

↓

B and T cells play a role in development of involved antibodies

↓

Initiates a cytokine cascade

↓

Results in an immune reaction

Anaphylaxis

IgE antibody developed after first exposure

↓

Next exposure IgE antibody binds to mast cells and basophils

↓

Release of inflammatory mediators – histamine, tryptase, leukotrienes, prostaglandins, and platelet-activating factor

↓

Causes systemic vasodilation, increased capillary permeability, bronchoconstriction and coronary vasoconstriction

Risk Factors

- Platinums (cisplatin, caroboplatin, oxaliplatin)
  - Can occur after multiple cycles of therapy
- Taxanes (paclitaxel, docetaxel)
  - Reaction to drug or diluent
- L-Asparaginase
  - Intradermal skin testing performed before administration
- Procarbazine
- Epipodophyllotoxins (etoposide, teniposide)
- Pegylated liposomal doxorubicin
- Cytarabine
- Monoclonal antibodies (rituximab, cetuximab, trastuzumab, bevacizumab, obinutuzumab, ofatumumab)
- Checkpoint inhibitors (pembrolizumab, nivolumab, atezolizumab, avelumab, durvalumab, ipilimumab)

Risk Factors (continued)

- Preexisting allergies, especially to food, drugs, bee stings, blood products, and contrast dye
- High doses of high risk agents
- IV administration of high risk agents
- History of hypersensitivity reactions
- IV administration
- Failure to administer premeds

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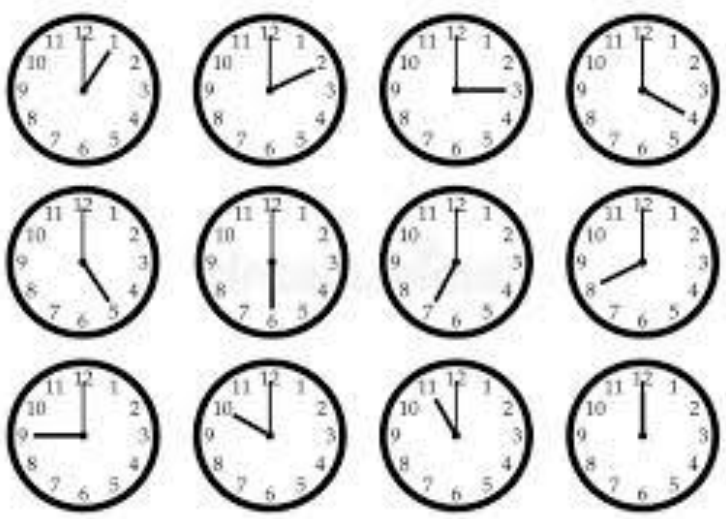
Hypersensitivity and Anaphylaxis

- HSRs are more likely to occur with the second dose of an agent but can occur with the first.
- Symptoms can range from itching at the injection site to systemic shock.
- The response usually occurs within 5-10 minutes of the initiation of therapy.
  - Quicker onset of symptoms increases the severity of the reaction.
- Delayed reactions can occur 10-12 hours after administration.



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Symptoms

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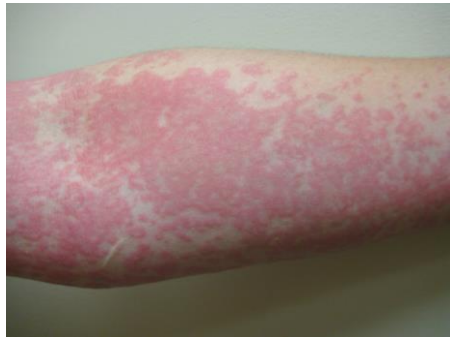

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- Fever
- Rigors
- Hives
- Flushing
- Itching (chest, feet, hands, etc.)
- Rash or flushing
- Chest tightness
- Difficulty breathing
- Hypotension
- Angioedema
- Back pain
- “I don’t feel right…”

Management of Hypersensitivity Reactions

- Antipyretics (e.g. acetaminophen)
- Opioids (e.g. meperidine)
- H1 antagonists (e.g. diphenhydramine)
- H2 antagonists (e.g. famotidine)
- Corticosteroids
- Epinephrine *First line for anaphylaxis
- Albuterol
- IV fluids

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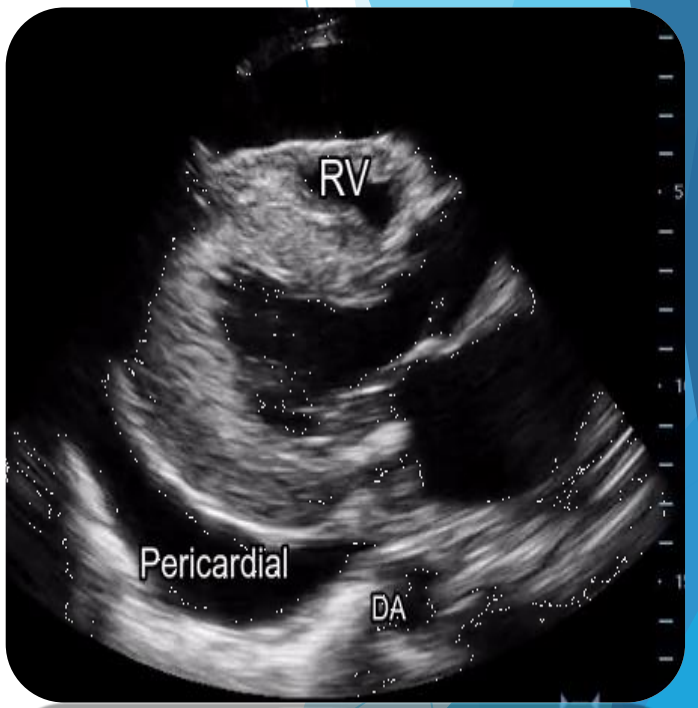
Cardiac Tamponade

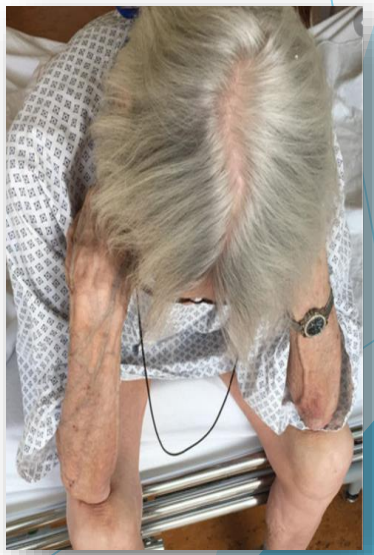
Pericardial effusion

- Pericardial pressure > 30 mm Hg from fluid accumulation (effusion) in the pericardial sac
- Pressure on chambers inhibits inflow of blood to the ventricles and reduces cardiac output
- Untreated can lead to:
  - Cardiovascular collapse
  - Shock
  - Death
- Patients at risk:
  - Cancers with involvement of the pericardium: Advanced lung cancer, breast cancer, malignant melanoma, leukemia, lymphoma
  - History of radiation to the chest
  - Chemotherapy-related effusions
    - Doxorubicin, daunorubicin, and cytarabine, cyclophosphamide



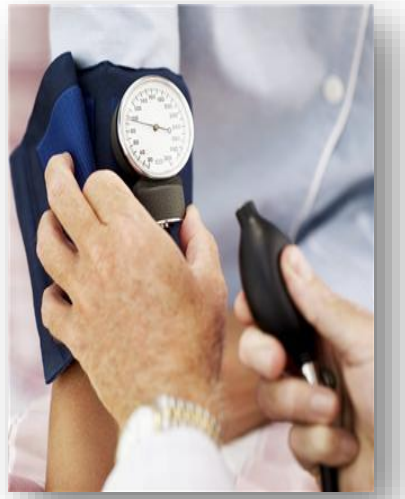
Symptoms

- Early "Effusion" period
  - Asymptomatic or mimics heart failure
    - Jugular venous distention (JVD)
    - Peripheral edema
    - Hepatomegaly and abdominal distention
    - Increased diastolic pressure
    - Tachycardia, chest pain
    - Fatigue, exertional dyspnea, and orthopnea
- Late
  - Dull chest pain/heaviness
  - Increasing dyspnea and "air hunger"
  - Tripod positioning
  - Nonproductive cough
  - Anxiety, agitation, and mental status changes
  - Cold sweats or confusion
  - Hiccoughs, dysphagia, or hoarse voice



Physical Assessment

- JVD (nonpulsating)
- Weak pulses and tachycardia
- Muffled heart sounds; possible pericardial friction rub
- Hypotension
- Narrow pulse pressure (↓systolic and ↑diastolic)
- Pulsus paradoxus (late sign)
- Decreased urine output (oliguria)
- Tachypena, orthopena



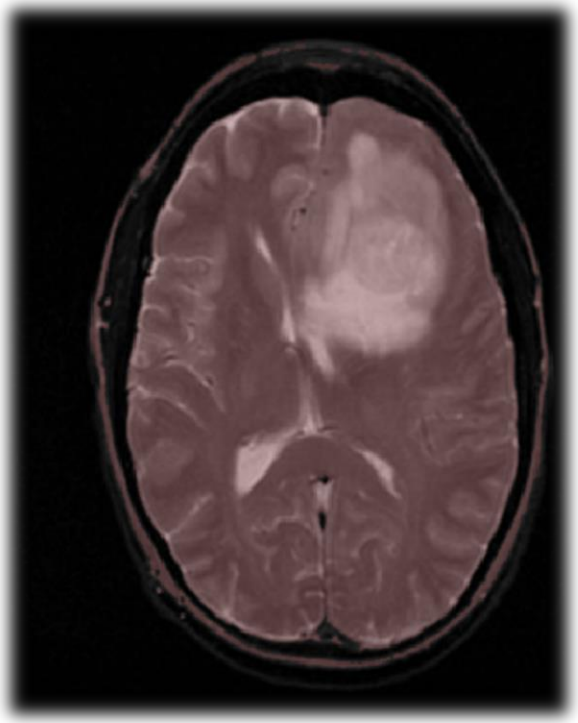
Treatment

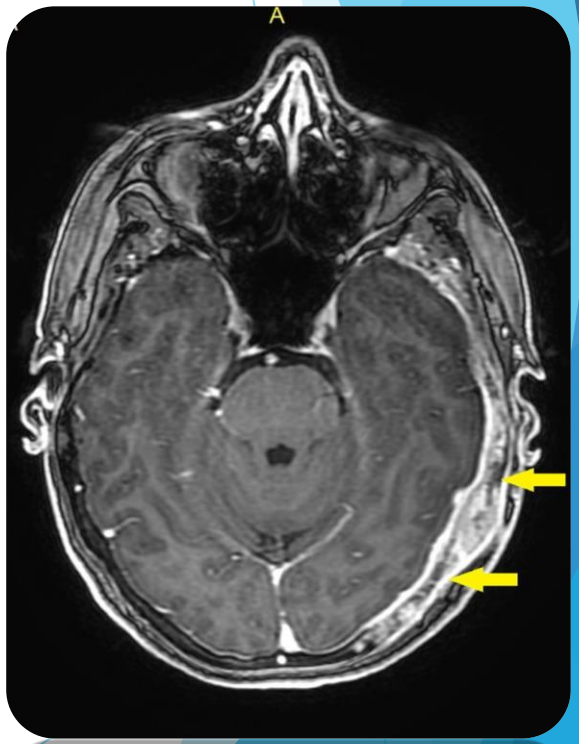
- Immediate removal of fluid
  - Pericardiocentesis
- Control reaccumulation of fluid
  - Pericardial window with drain
  - Sclerosis of mesothelium with agents that causes inflammation and subsequent sclerosis given via catheter (e.g. mitomycin, bleomycin)
- Chemotherapy or radiation therapy
- Glucocorticoids
- IV fluids

Increased Intracranial Pressure (ICP)

Causes

- Primary or metastatic tumors
- Leptomeningeal metastases
- Blood clots
- Edema of the brain tissue
- Infection
- Metabolic disorder

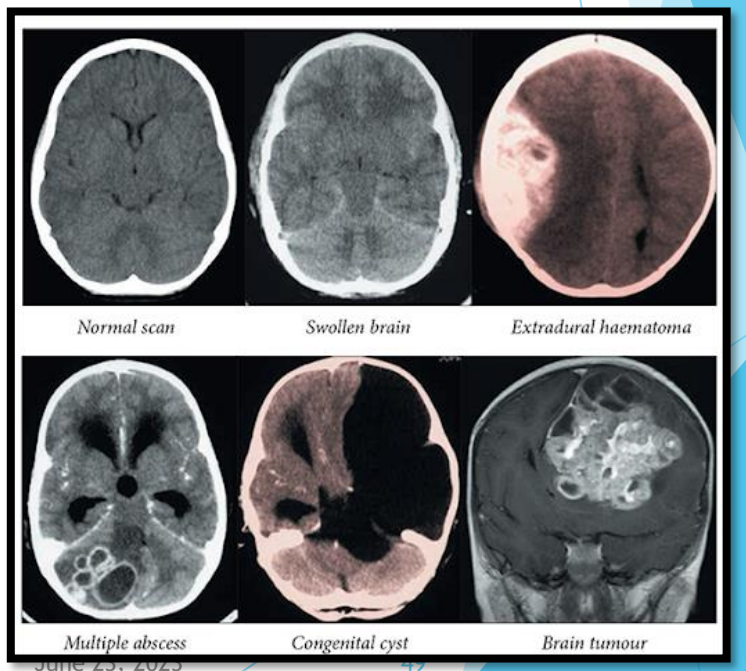




Patients at Risk

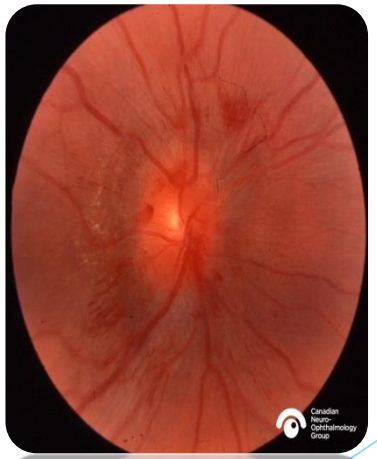
- Cancers with increased risk of brain metastases (e.g. lung, breast, kidney, melanoma)
- Primary brain or spinal cord tumors
- Leukemia, lymphoma, neuroblastoma
- Thrombocytopenia or DIC

- SIADH
- History of radiation therapy to the brain
- Occluded Ommaya reservoir
- Infections (e.g. encephalitis, meningitis)
- High dose Cytarabine



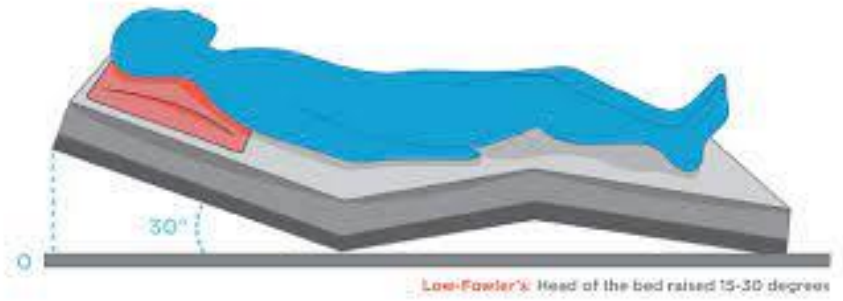
Symptoms

- Early
  - Persistent headache (worse in the am; bending over or during Valsalva maneuvers)
  - Nausea or vomiting (unrelated to food intake; often projectile)
  - Visual changes (blurring; double vision)
  - One-sided muscle weakness
- Late
  - Lethargy, apathy, confusion, decreased LOC
  - Speech alterations (e.g. slowed or delayed responses)
  - Papilledema *Cardinal sign of ICP
  - Widening pulse pressure
  - Abnormal breathing patterns
  - Temperature elevations
  - Coma/seizures
  - Abnormal posturing



Management

- History and neuro exam/checks are important!
- Surgery
  - Craniotomy to relieve pressure
  - Shunt placement to provide an alternative pathway for CSF
  - IT chemo
- Radiation therapy after pressure normalized
  - Used with caution – can cause herniation and death with ICP
- Chemotherapy after pressure normalized
- Supportive therapies
  - Administer dexamethasone
  - Elevate head of bed to 30 degrees
  - Maintain blood pressure and temperature
  - Monitor ICP
  - Maintain sodium level – isotonic fluid
  - Administer diuretics, cautiously
  - Restrict fluid intake, cautiously
  - Osmotic therapy (mannitol)
  - Administer anticonvulsants, if indicated



Spinal Cord Compression (SCC)

https://www.youtube.com/watch?v=hq-qmlW7eWQ

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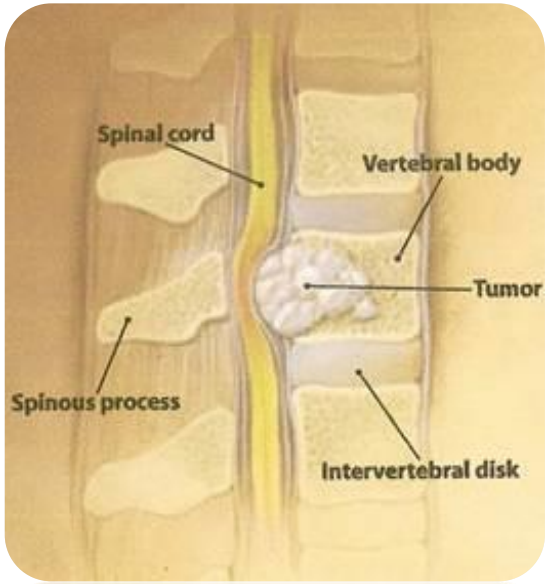
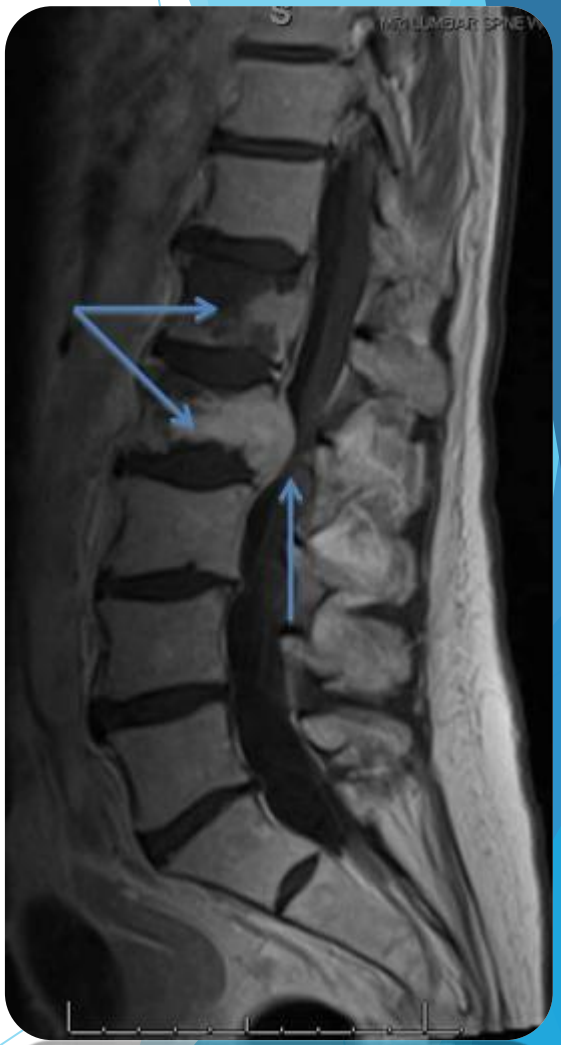
Mechanisms of Spinal Cord Compression

- 85% of SCC cases are caused by direct pressure on the cord resulting from metastasis to vertebral body
- 15% of SCC cases are caused by direct tumor involvement
- Two mechanisms:
  - Metastatic tumor mass in vertebral body expands into the epidural space
  - Metastatic tumor erodes and collapses vertebral body, displacing bone fragments into the epidural space





Cascading impact of Spinal Cord Compression

- Invasion of spinal cord leads to vascular physiologic response:

Symptoms

- Symptoms are progressive
  - May progress over months or progress rapidly over days or hours
- Back pain – dull/achy → radiating
- Sensory loss, numbness, tingling, and sensory changes
- Motor weakness or gait changes
- Autonomic dysfunction: Bowel and bladder retention/dysfunction/incontinence
- Paralysis

***MRI diagnostic procedure of choice

Treatment

Standard Treatment:

Steroids

- Usually dexamethasone
- Reduces edema, improves function, and relieves pain
- Loading dose followed by tapered dose after neurologic status stabilized
- Steroid treatment is typically the initial treatment until radiation therapy or surgery is instituted

Radiation Therapy
Surgery
Pain Management

Additional Options:

Chemotherapy
Hormonal Therapy
Bisphosphonates
CyberKnife

Superior Vena Cava Syndrome (SVCS)

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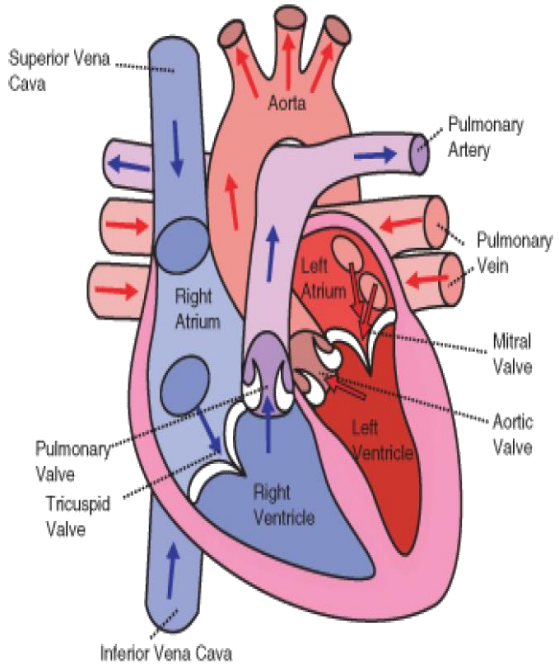
SVCS

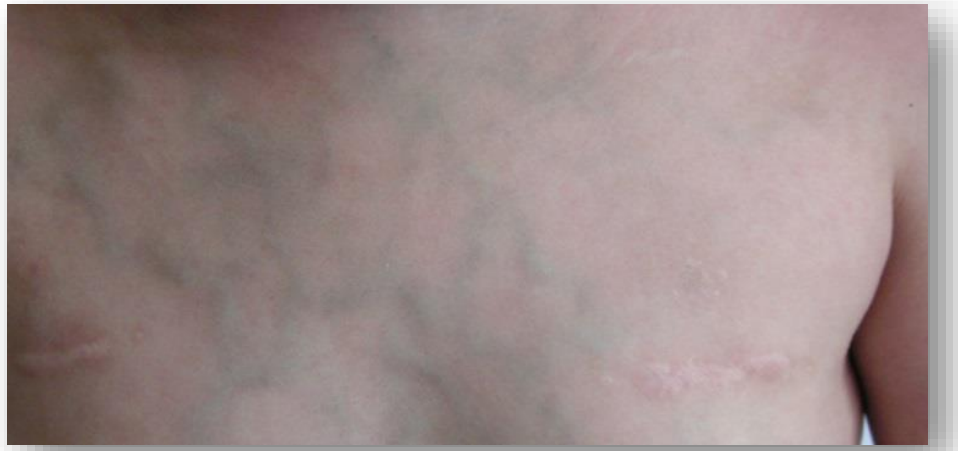
- A disorder of venous congestion and venous drainage in the upper thorax caused by obstruction of the superior vena cava (SVC)

- Causes:
  - Occlusion by:
    - Extrinsic mass
    - Tumor invasion through the SVC
    - Thrombus around a venous access device
    - Thrombus within the SVC



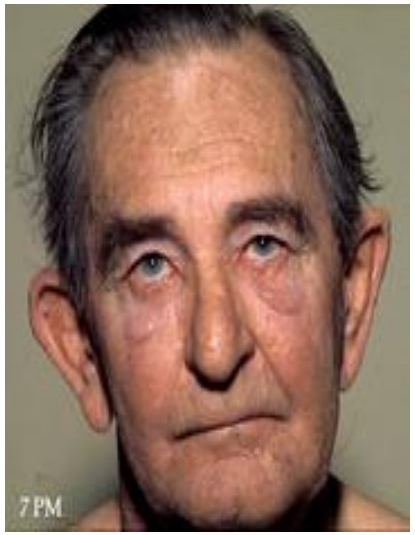
Mechanism of SVCS

- Usually has a gradual onset because collateral circulation develops to divert blood around the obstruction
- Collateral circulation bypasses site of obstruction
  - Redirects blood flow from upper thoracic venous system and the obstructed SVC to the inferior vena cava
  - Internal mammary, lateral thoracic, and superficial thoracoabdominal veins may become more developed



Symptoms

- Early symptoms occur when supine and subside after arising, which allows venous drainage
- Bending over, stooping, or lying flat may aggravate symptoms
- Headache
- Facial swelling
- Tightness or fullness in the neck
- Upper extremity edema
- Periorbital edema
- Dyspnea (one of the most common symptoms)
- Neck vein distension
- Swelling in hands and fingers
- Erythema of face and neck



Treatment

- Treatment is based on histologic diagnosis, rate of onset, and type of obstruction (intra- or extraluminal)
  - Radiation therapy
  - Chemotherapy
    - For chemo sensitive malignancies such as SCLC and NHL
  - Pharmacologic treatments (steroids, diuretics, thrombolytic therapy)
  - Surgery (SVC bypass, stent placement)
- Nursing Care
  - Monitor vital signs and support respiratory system
  - Fowler's position
  - Supplemental oxygen
  - Energy conservation
  - Monitor weight, fluid and electrolyte balance
  - Avoid over hydration (may exacerbate symptoms)
  - Avoid dehydration (may increase risk of thrombus)
  - Avoid invasive/constrictive procedures to involved extremity
  - Reduce anxiety

Bowel Obstruction

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Definition

- Cessation of forward movement of bowel contents
- Causes:
  - Malignant obstruction (e.g. colorectal cancer, ovarian cancer)
  - Postop intra-abdominal adhesions
  - Nonsurgical adhesions after peritonitis or RT
  - Hernias with colonic incarceration
  - IBD
  - Strictures from diverticulitis
  - Ileus from medication (e.g. opiods, antidiarrheals)

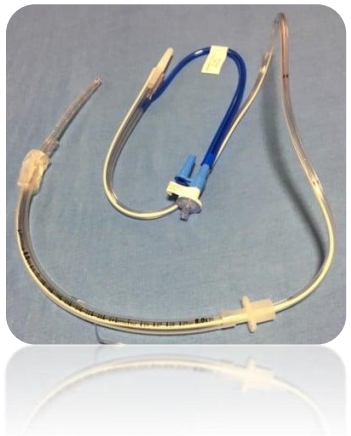
Symptoms

- Abdominal pain
  - Partial obstruction: cramping pain after eating
  - Complete obstruction: pain intensifies and comes in waves or spasms, with strangulation pain is constant and severe
- Nausea and vomiting
- Anorexia, appetite changes
- Constipation or paradoxical diarrhea
- Bloating, abdominal distention, abnormal bowel sounds
- Pyrexia, tachycardia, hypotension

Image: Microsoft Office Clip Art

Treatment

- Surgery and/or chemotherapy
- IV fluids
- Low dose steroids to decrease bowel wall edema
- Antiemetics
- Octreotide to decrease intestinal sections and stretching of bowel wall
- Dietary consult for possible TPN
- Pain control
- NG tube



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Bowel Perforation

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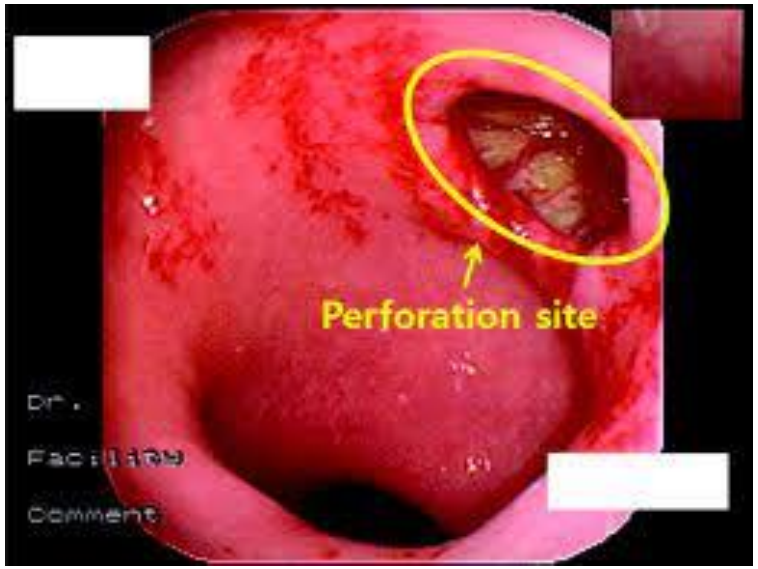
Pathophysiology

- A full-thickness injury of the bowel wall allowing bowel contents to leak out
- May cause peritonitis, abscess or fistula formation
  - Immunosuppression increases the risk for perforation
- Can quickly progress to sepsis



Treatment

- Surgical emergency
- Stent placement or closing of perforation via endoscopy
- Antibiotics
- Proton pump inhibitors
- Analgesics

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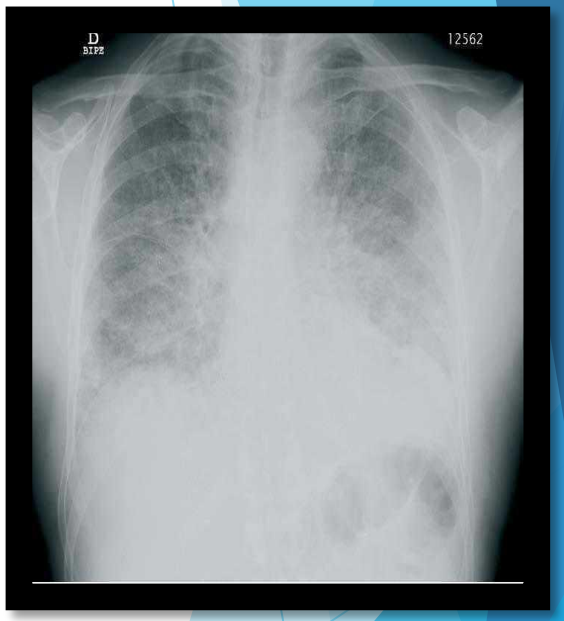
Pneumonitis

Causes and Symptoms

- Inflammation of the interstitial lung parenchyma with interstitial and alveolar infiltrates caused by a non infections source such as a chemotherapy (e.g. Rituximab, immunotherapy, taxanes) or radiation therapy to chest
- Radiation pneumonitis usually develops 4-12 weeks after radiation completion
- Symptoms:
  - Low-grade fever
  - Hypoxia
    - O2 sat < 90% or more than 4% decrease from baseline
  - Tachypnea and cyanosis
  - Bilateral rales or a pleural rub often heard
  - If rash present, consider pneumonitis may be from a hypersensitivity reaction



Treatment

- Glucocorticoids
- Inhaled bronchodilators
- Immunosuppression with infliximab with/without cyclophosphamide for severe immunotherapy-related pneumonitis
- Antibiotics
- Oxygen therapy
- Mechanical ventilation may be needed

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CHAPTER

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