

Explain the pathophysiology, etiology, and common causes of Tumor Lysis Syndrome
 Define the common lab findings, symptoms, pre-treatment considerations, and treatment options of Tumor Lysis Syndrome
 Describe the pathophysiology and risk factors of Disseminated Intravascular Coagulation (DIC)
 Differentiate between Acute DIC and Chronic DIC
 Identify the symptoms, common lab findings, treatment options, and supportive care of Disseminated Intravascular Coagulation (DIC)
 Identify the cancer types that are at risk for Spinal Cord Compression
 Differentiate between the symptoms of a cervical, thoracic, and lumbosacral spinal cord compression
 Explain how a spinal cord compression is diagnosed and the treatment options
 Define febrile neutropenia and the commonly seen symptoms
 Describe the assessment and management of febrile neutropenia as well as the consequences of delayed treatment and prevention tactics
 Understand the pathophysiology and risk factors of sepsis and septic shock
 Differentiate between early symptoms of sepsis, severe sepsis symptoms, and septic shock symptoms
 List the commonly seen lab results and treatment options of sepsis and septic shock

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What is TLS?
A condition in which serious electrolyte imbalances occur in the body as a result of rapid cell death and necrosis of tumor tissue
Pathophysiology
As tumor cells die, their normal intracellular components, such as potassium phosphate, get released into the bloodstream
This can cause high concentrations in the blood that the kidneys cannot eliminate quickly enough causing electrolyte and metabolic dysfunction
If not treated quickly, it can cause neurologic, GI, renal, and cardiac failure, and death
Most commonly occurs 48-72 hours after treatment

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Etiology

Typically associated with:
Leukemias with high white blood counts
Non-Hodgkin Lymphoma
Bulky, rapidly growing, treatment-responsive tumors

Causes
Chemotherapy
Cisplatin, cylarabine, etoposide, paclitaxel, fludarabine, hydroxyurea, intrathecal methotrexate Immunotherapy
Interferons, interleukins, rituximab, tumor necrosis factors
Hormonal Therapy
Tamoxifen
Corticosteroids
Surgery
Radiation

Laboratory Findings
Elevated:
Potassium >6 or an increase of 25% from baseline
Phosphorus >4.5 or increase of 25% from baseline
Uric Acid >8 or increase of 25% from baseline
Uric Acid >8 or increase of 25% from baseline

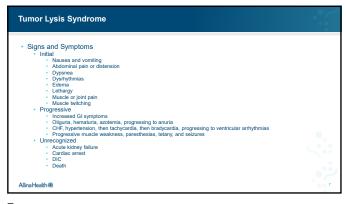
Calcium <7 or decrease of 25% from baseline

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Tumor Lysis Syndrome

• Pre-Treatment Considerations & Assessment

• Labs

• Electrolytes

• Renal function

• Liver function

• Baseline weight

• Baseline electrocardiogram

• Diet considerations

• Avoid foods high in potassium and phosphorus

• Medication review

• Avoid medications that may increase potassium (ACE Inhibitors, potassium sparing diuretics)

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Treatment

Hydration

Aggressive hydration beginning 24-48 hours prior to the initiation of treatment and for 72 hours after treatment

Hydration supports renal blood flow, maintains urine output, decreased concentration of soluble acids in the urine

Ensure urine output is greater than 150-200mL/hour

Allopurinol

Prohibits precursors of uric acid

Given prophylactically, even to those with low risk

Begin medication 1-2 days prior to treatment and for 2-3 days post-treatment

Rasburicase

Converts circulating uric acid into water soluble metabolite, allowing plasma and urine uric acid levels to decrease rapidly

Treatment Cont.

• Treatment Cont.

• Loop Diuretics

• May help prevent fluid overload and maintain urine output

• Oral Phosphate-Binding drugs (Albumin)

• Treatment for hyperkalemia and hyperphosphatemia

• Dialysis

• If renal failure occurs

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Disseminated Intravascular Coagulation (DIC)

DIC is a complex system disorder that involves the activation of coagulation pathways, leading to thrombotic and hemorrhagic events

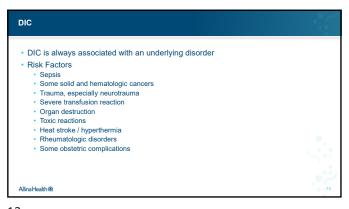
Pathophysiology

DIC causes hypercoagulation, triggering the coagulation pathways to be initiated inappropriately
The cascade begins with the release of tissue factor, causing the release of thrombin
This release then causes plasminogen to convert to plasmin, causing fibrinolysis (the breakdown of fibrin within clots)
This then causes excessive fibrin degradation products (FDPs), which causes bleeding
The final result is a situation of simultaneous hemorrhage and clot formation and is a lifethreatening event
Blood supply to vital organs is compromised

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Acute DIC

Acute DIC

Causes

Infection is the most common underlying cause of acute DIC

Infection may produce a systemic inflammatory response, activating the cytokine system that begins the hypercoaguillour causarde

Oncological Conditions

Acute promyelocytic leukemia (APL) is most often associated with acute DIC (85% of patients)

In APL, tissue factor is released directly from the promyelocyte blast cells into the blood stream, beginning the congulation pathway of events

Symptoms

Symptoms

Bleeding that occurs simultaneously form at least 3 unrelated sites is a hallmark sign of acute DIC

Chronic DIC

Causes

Solid metastatic mucinous adenocarcinomas and tumors of the prostate, breast, stomach, lung, and pancreas

Symptoms

Associated with minimal bleeding and diffuse thrombosis

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Symptoms
Initial
Bleeding
Anywhere from occult/oozing to frank hemorrhage
Renal
Acidosis
Hematuria
Oligoria
Uterine hemorrhage
Pulmonary
Upsprea
Hemoptysis
Cough
Theypnea
Hemoptysis
Cough
Trachypnea
Diminished breath sounds
Pleural friction rub

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Initial
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Symptoms Cont.

Initial

Integumentary
Jaundice

Petechiae
Skin necrosis of lower limbs
Thrombosis
Fever

Later Symptoms
Thrombus formation
Signs may be subtle but may manifest as organ dysfunction or failure occurs

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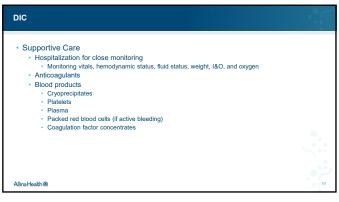
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    Laboratory Findings
    Decreased
    Platelets
    Fibrinogen level (severe cases, decreases slowly)
    Antithrombin III
    Plasminogen
    Alpha-2 antiplasmin level
    Protein C
    Elevated
    Fibrin degrading products (FDPs)
    D-Dimer
    Thrombin time
    Firbinopeptide A level
    Prolonged Results
    Protrombin time
    Partial thromboplasmin time
    Partial thromboplasmin time
    International normalized ratio (INR)

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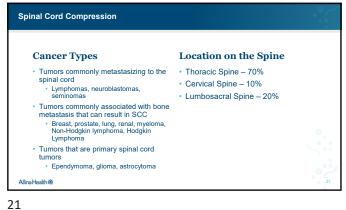
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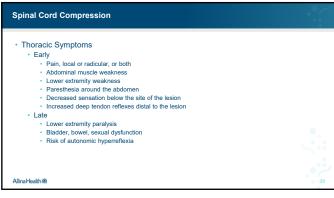




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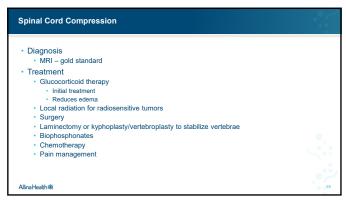
Spinal Cord Compression Cervical Symptoms Early Occipital headache, radiating to neck/shoulder, neck stiffness
 Neurogenic shock (hypotension, bradycardia) Paresthesia Lhermitte sign (tingling sensation on back and extremities when neck is flexed or extended) · Hyperactive deep tendon reflexes Late Quadriplegia Bladder, bowel, and sexual dysfunction · Autonomic hyperreflexia Lesion above C4, may have diaphragmatic dysfunction

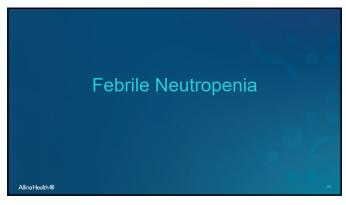


Spinal Cord Compression · Lumbosacral Symptoms Early Pain, local, or radicular, or both, in groin and down legs (sciatica)
Pain while raising legs straight Weakness in pelvic muscles & lower extremities Ataxia gait · Paresthesia and numbness Decreased reflexes Late Autonomic effects
 Bowel, bladder, sexual dysfunctions

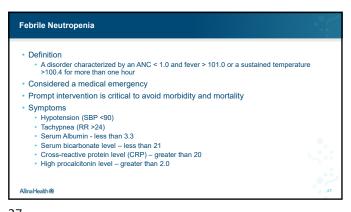
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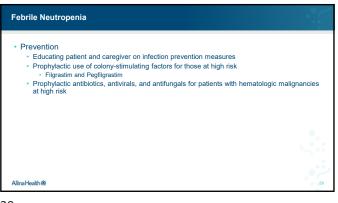


Assessments & Management
 Prompt intervention is essential
 Blood and urine cultures
 Chest X-ray
 VRE swabs (if indicated)
 Prompt administration of IV antibiotics
 Cefepime is the most common first-line treatment

 Consequences of Delayed Treatment
 Circulatory collapse
 Acute respiratory failure
 Sepsis and septic shock
 Death

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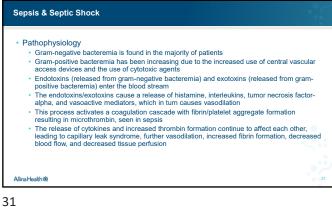
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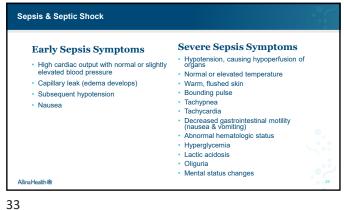


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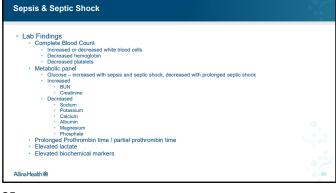
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Sepsis and Septic Shock Risk Factors Granulocytopenia Most common risk factor
 Increased duration and severity increases the risk Extreme age (>1 or <65) · Long intensive care hospitalizations Loss of skin or mucosal injury Malignancy-related immunosuppression
 Humoral immunity modifications Multiple myeloma Chronic lymphocytic leukemia Diabetes · Comorbid organ dysfunction · Presence of a vascular access device



Sepsis & Septic Shock Septic Shock Symptoms
 Cold, clammy skin
 Lethargy, progressing to coma Hypotension Weak pulse Tachyonea Decreased breath sounds with pulmonary edema/rales Persistent hematologic abnormalities Hyperglycemia Lactic acidosis Hyponatremia Hypokalemia Hypocalcemia Hypomagnesemia Hypophosphatemia Decreased albumin



Sepsis & Septic Shock **Sepsis Treatment Septic Shock Treatment** ICU monitoring for aggressive and continuous monitoring · Fluid resuscitation · Respiratory support Central venous pressure monitoring DVT prophylaxis Oxygen support & pulse oximetry Antibiotics Aggressive fluid resuscitation Antifungals Intubation and ventilation due to pulmonary edema, encephalopathy, and/or coma Blood products · Electrolyte replacement Supportive treatments
 Prophylaxis for stress ulcers · Nutritional support · Insulin support DVT prophylaxis
 Nutritional support
 Assessment/intervention for delirium
 Psychosocial support and education Vasopressors and inotropic drugs Activated protein C replacement

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