Managing Psychosocial Issues in the ICU

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LRCCP BASIC CRITICAL CARE CLASS

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Agenda

- Goal: To provide nurses with recommendations to assist in the identification and care of patients with co-occurring mental and physical health disorders by providing strategies to identify potential mental health issues, improve communication, identify safety concerns, and improve outcomes for this vulnerable patient population.
  - Depression/Suicide attempt
  - Delirium
  - Substance abuse
  - Dementia with behavioral disturbances
  - Personality disorders
  - Safety (Environment of care)
  - Interventions to manage difficult patients

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Objectives

- Identify different types of mental illness
- Identify treatment options for ICU patients
- Recognize safety concerns for mental health patients in the ICU

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Mental Health and Substance abuse Issues Statistics

- Nearly 1 in 5 adults are diagnosed with a mental illness
- 5.2% of the population are considered to have serious mental illness
- 13.48 per 100,000 population people successfully committed suicide in 2020
- 1.2 Million estimated suicide attempts in 2020
- Arkansas was at 18.4 per 100,000 people in 2019
- Crisis line calls increases 730% since schools and businesses first closed because of the pandemic
- Almost 21 million American adults battled with substance abuse
- In 2020 about 2.6 million Americans reported using meth in the past 12 months

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ICU patients with psychosocial issues

- Depression/Suicide attempt
  - Physical
  - Chemical
- Delirium
  - Metabolic
  - Substance induced
- Substance abuse
- Dementia with behavioral disturbances

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Depression/Suicide attempt

- Monitor/treat for at least 24 hours post suicide attempt
- 1:1 observation or start the q5 or q15 minute observations
- Psychiatric consult as soon as possible
- Maintain safe environment
  - Mitigate ligature risk as much as possible
  - Remove contraband
  - Limit and monitor visitation
- Educate patient and family that all interventions in place are to ensure patient safety
- Listen

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Delirium

- Acute state of confusion marked by the following:
  - Fluctuating level of consciousness
  - Perceptual disturbances
  - Sleep disturbances
  - Increased or decreased psychomotor activity

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Delirium

- Causes
  - Infection
  - Medications
  - Oxygenation
  - Electrolyte imbalance
  - Sleep disturbances
  - Trauma
  - Alcohol withdrawal

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Delirium

- Assessment
  - Confusion Assessment Method (CAM)
  - Mini Mental Status Exam (MMSE)
- Treatment
  - Treat underlying cause
  - Antipsychotics may be necessary for behavior

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Delirium

- Nursing Management
  - Safe structured environment
  - One step commands
  - Patience
  - Frequent reorientation
  - Reduce distractions
  - Family Support

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Substance Abuse
Delirium Tremens

- Delirium tremens
  - Severe withdrawal from alcohol, too unstable to be on a psychiatric unit or medical floor
  - Hallucinations, agitation, global confusion, disorientation, fever, hypertension, diaphoresis, tachycardia, hypertension
  - Has a mortality rate up to 37% without appropriate treatment
  - Mortality rate with appropriate treatment ranges from 5-15%

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Substance Abuse
DTs

- Treatment
  - Manage fluids and electrolytes
  - Benzodiazepines
  - Thiamine- can help prevent Wernicke encephalopathy
  - Magnesium- can diminish the severity of withdrawal symptoms

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Substance Abuse
DTs

- Nursing management (Alcohol withdrawal and DTs)
  - Accurate alcohol intake history
  - CAGE Assessment
  - Clinical Institute Withdrawal Assessment (CIWA)
  - Administer medications as prescribed
  - Closely monitor physical signs and symptoms
  - Limit visitation - monitor items brought into the room, remove all contraband
  - Keep the patient as comfortable as possible
  - Psychiatric Consult
  - Listen

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Substance Abuse
Substance induced psychosis

- Treatment
  - Psychiatric consult
  - Stabilize medically
  - Maintain safe environment
  - Limit visitation

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Dementia with Behavioral Disturbances

- Dementia - Impaired ability to remember, think, or make decisions that interferes with completing everyday activities
- Signs and symptoms
  - Memory loss
  - Attention deficit
  - Communication deficit
  - Poor reasoning and judgment
  - Agitation
  - Sleep disturbances
  - Hallucinations
  - Poor mobility

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Dementia with Behavioral Disturbances

- Assessment
  - Medical history from patient and family if possible
  - MMSE
  - Clock Drawing
- Treatment
  - Treat medical reason for admission
  - Provide medications as prescribed
  - Psychiatric consult for behavior management
- Nurse management
  - Routine assessments
  - Maintain safe environment
  - Frequent reorientation
  - Patience
  - Listen

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Personality Disorders

Cluster A (odd/eccentric)	Cluster B (Dramatic)	Cluster C (Anxious)
Paranoid	Antisocial	Avoidant
Schizoid	Borderline	Dependent
Schizotypal	Histrionic	Obsessive-Compulsive (not the same as OCD)
	Narcissistic	

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Cluster A
Characteristics

- Social awkwardness and social withdrawal
- Paranoid, suspicious of being deceived by others
- Secretive
- Rarely have close relationships
- Odd speech and behavior
- Similar behaviors as someone diagnosed with schizophrenia, but usually less extensive and less intrusive to daily functioning

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Cluster A
Management

- Be professional
- Clear expectations
- Empathetic to fears
- Avoid direct challenge to paranoid ideation
- Avoid over involvement in personal issues
- Tolerate odd beliefs and behaviors

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Cluster B
Characteristics

- Dramatic
- Erratic
- Very difficult to form healthy relationships
- Promiscuous behavior
- Intense emotions
- Disregard for social norms and lack of empathy (Antisocial)
- Emotional instability, impulsive behaviors, self-harm, attention seeking (Borderline)
- Need to be center of attention, frequent mood swings (Histrionic)
- Self-centered, exaggerated self image (Narcissistic)

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Cluster B
Management

- Be Professional
- Be honest
- Carefully investigate concerns and motives
- Clear communication with clear limits
- Nonpunitive
- Avoid excessive familiarity
- Schedule regular visits
- Patience (expect and tolerate angry outburst)
- Maintain firm boundaries
- Be aware of personal feelings
- Focus on facts and objective issues
- Validate concerns

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Cluster C
Characteristics

- Anxious
- Fearful
- Overlaps symptoms of anxiety and depressive disorders
- Disregard for rules
- Lack of empathy
- Fear of being alone
- Do things to get others to like or accept them
- Preoccupied with perfection, orderliness and control of relationships

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Cluster C
Management

- Validate concerns
- Encourage reporting of symptoms
- Provide reassurance
- Schedule regular check-ups
- Set realistic limits on availability
- Avoid rejecting the patient or making them feel rejected
- Provide thorough explanations
- Encourage patient to participate in treatment

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Safety
Environment of Care

- Risk Assessment for the unit (The whole unit or designated psych rooms)
- Screen patient on admission or as soon as possible
- Limit visitation to the same as a behavioral health unit
- 1:1 observation
- Q15 minute or Q5 minute checks
- Room checks for contraband
- Educate visitors on contraband items
- Limit cord length on beds and pumps
- Limit medical equipment in the room to only essentials
- Garage doors covering medical gases and outlets if not necessary
- Avoid complacency
- Situational awareness

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Tips for Managing Disruptive Patients in Medical Settings

- Patience and listen
- Create a pass off to share between caregivers and sitters (effective interventions, ineffective interventions)
- Set firm boundaries
- Be honest and treat the patients with respect
- Nonjudgmental
- Simple commands
- Reorientation
- Reduce distractions
- Validate concerns
- Continuity between shifts
- Psych consult

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Case Scenario #1

Richard, a 44yr old, unemployed, homeless man attempted suicide by stabbing himself in the chest. He is silent when you greet him and explain why you are there. He never looks up but stares out the window and refuses to talk to you.

How will you respond to this patient? How will you prepare the room? What instructions will you provide the sitter?

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Case Scenario #2

Nellie, age 35, recently became widowed and attempted suicide this morning by ingesting a bottle of sleeping pills. You are her RN for the nightshift and her husband arrives requesting to spend some time alone with his spouse.

How will you respond to this patient? How will you prepare the room? What instructions will you provide the sitter?

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Case Scenario #3

Heather, age 70, was admitted to the ICU with what appears to be acute psychosis. Her family states she has no psychiatric history. She is hallucinating and having a difficult time following simple commands. Heather is unstable on her feet but will not stay in the bed.

How will you respond to this patient? How will you prepare the room? What instructions will you provide the sitter?

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References

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- Assessment and management of Personality Disorders. American Family Physician. https://www.aafp.org/afp/2004/1015/p1505.html
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Hematological Disorders
• *Hematologic and Immunologic System A&P*

- Assessment is essential
  - History of predisposing factors
  - Assess and explore depth of any personal or genetic history –
    - Bleeding
    - Chronic Anemia
    - Systemic Diseases
  - Medication History
    - Anticoagulants
    - Anti-platelet agents
    - Steroids and non-steroidal agents
  - Physical signs
    - Pale or jaundiced
    - Unexplained bruising, clotting, ecchymosis, petichiae

Hematological Disorders
• *Hematologic and Immunologic System A&P*

- Structures, Components and Functions
- Bone Marrow
  - Spongy material
    - Hematologic and immunologic cells
      - Originate, mature and then released into bloodstream
- Liver
  - Removes non-functional erythrocytes from the bloodstream
- Spleen
  - Removes damaged erythrocytes from the bloodstream
- Cellular components
  - Red blood cells (erythrocytes)
  - Platelets (thrombocytes)
  - Coagulation factors
  - Stem cells – all cells from the bone marrow originate from stem cells
    - Daughter cells are produced
      - Lymphoid stem cells – produce β cells for the immune system
      - Myeloid stem cells – produce erythrocytes and thrombocytes

Hematological Disorders
• *Hematologic and Immunologic System A&P*

- Erythrocyte production stimulated by hypoxia
  - Kidney produces erythropoietin
  - Erythropoietin is produced to stimulate stem cell production
  - Erythrocytes form
    - Hemoglobin is required to carry oxygen
      - ATP production
      - CO_2 removal

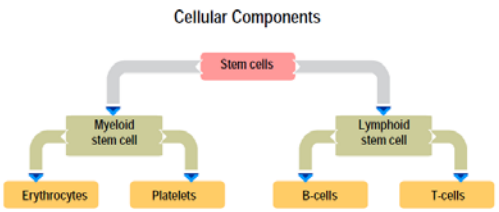
- Thrombocyte or platelet production and coagulation mechanism
  - Stimulated/activated from tissue or blood vessel injury
    - Platelets rush to site of injury
    - Cytokines are released
    - Additional platelets are activated
    - Coagulation mechanism continues
    - Immune response is stimulated

Cellular Components

ESSENTIALS OF CRITICAL CARE ORIENTATION

Cellular Components

Lesson: Hematologic System Anatomy & Physiology
Topic: Structures, Components and Functions



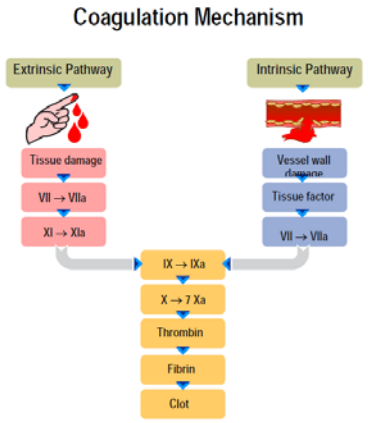
Hematological Disorders
• *Hematologic and Immunologic System A&P*

- Coagulation Mechanism and Fibrinolysis
  - Two main functions
    - Oxygenation
    - Homeostasis
      - Process of clotting/coagulation
      - Fibrinolysis

Hematological Disorders
• *Hematologic and Immunologic System A&P*

- Coagulation Mechanism and Fibrinolysis
  - Coagulation and Fibrinolysis
    - Homeostasis
      - Self regulation
        - Clotting and fibrinolysis
          - Blood protein breaks down clot
          - Fibrin Split Products (FSP)/ Fibrin Degradation Products (FDP) – fragments created when fibrin is broken down
        - Coagulation Cascade of events
          - 12 factors and several co-factors (plasma proteins
          - Always ends with the production of Thrombin (Factor IIa)
          - Intrinsic pathway – response to vessel wall damage
          - Extrinsic pathway – response to tissue damage
            - Thrombin splits Fibrinogen (Factor I); fibrin forms, blood clots

Coagulation Mechanism



Hematology Drugs

CLASS of DRUG	NAME of DRUG	THERAPEUTIC USE
ANTICOAGULANTS		
Indirect Thrombin Inhibitors	Unfractionated Heparin	Treatment or prevention of venous thrombosis Adjunct to thrombolytic therapy with ACS
	Low Molecular Weight Heparins Ardeparin (Normiflo) Endoxaparin (Lovenox) Dalteparin (Fragmin)	Prevention of venous thromboemboli
	Fondaparinux (Arixta)	Prevention of venous thromboemboli with risk of HIT. Synthetic agent not from animal proteins
Direct Thrombin Inhibitors	Hirudin Derivatives Lepirudin (Refludan) Bivalrudin (Angiomax)	Heparin substitute for patients at risk or experiencing HIT
	Argatroban (Acova, Novastan)	Heparin substitute for patients at risk or experiencing HIT
Vitamin K Antagonist	Warfarin (Coumadin)	Prevention of venous thromboemboli development

Hematology Drugs

CLASS of DRUGS	NAME of DRUG	THERAPEUTIC USE
Antiplatelet Drugs		
Cyclooxygenase Inhibitor	Aspirin	At antiplatelet dose (75-325 mg) Primary and secondary prevention and treatment for ACS. Secondary prevention in stroke
Adenosine Diphosphate Receptor Antagonists	Ticlopidine (Ticlid) Clopidogrel (Plavix)	Reduction of athereosclerotic events in patients with recent stroke or MI Vascular disease
	Dipryridamole (Persantine)	Thromboembolism prevention following cardiac surgery
Glycoprotein IIb/IIIa Receptor Antagonist	Abciximab (ReoPro) Eptifibatide (Integrelin) Tirofiban (Aggrastat)	Acute Coronary Syndrome with or without percutaneous coronary intervention (PCI)
Thrombolytic Agents	Streptokinase (Kabikiane, Streptase) Alteplase (tPA) Reteplase (rtPA) Teneteplase (TNKase)	ACS, DVT, PE Adjunct to PCI Thrombotic Stroke
Antifibrinolytic Agents	Aminocaproic Acid (EACA, Amicar, Epsilson)	Coagulopathies with hemorrhage

Hematological Disorders
• *Hematologic and Immunologic System A&P*

- Immunologic Basics
  - White Blood Cells
    - Neutrophils
      - 55-70% of total white blood count
      - Live less than a day
      - Bone marrow constantly produces neutrophils
      - Infection protection
    - Lymphocytes
      - T lymphocytes
        - Regulate function of other immune cells
        - Directly attack various infected cells and tumors
      - B lymphocytes
        - Make antibodies
        - Target bacteria, viruses and foreign materials

Assessment, Diagnostic Tests and Monitoring Systems

Hematology Studies

Coagulation Studies

Immunology Studies

Hematological Disorders

• Assessment, Diagnostic Tests and Monitoring Systems

- Hematology Studies
  - Complete blood count
    - Platelet count
    - Hematocrit and Hemoglobin
    - RBC (red blood cells)
  - Mean Corpuscular Volume (MCV)
    - Normocytic
    - Microcytic
    - Macrocytic
  - Mean corpuscular Hemoglobin Concentration (MCHV)
  - Cell distribution Width (RDW)
  - Other tests
    - Reticulocyte
    - ESR (erythrocyte sedimentation rate)
    - Bilirubin levels

Red Blood Cell Laboratory Tests

Red blood Cell (RBC)	Male: 4.6-6.0 million/mm³ Female: 4.0-5.0 million/mm³
Mean Corpuscular Volume (MCV)	Male: 78-100 cubic micrometers Female: 78-102 cubic micrometers
Mean Corpuscular Hemoglobin Concentration (MCHC)	25-35 pg
RBC Distribution Width (RDW)	31-37%
Erythrocyte Sedimentation Rate (Sed Rate)	11.5% - 14.5%
Hematocrit (Hct)	Male: 37-49% Female: 36-46%
Hemoglobin (Hgb)	Male: 13-18g/100 ml Female: 12-16g/100 ml
Hemoglobin Electrophoresis	Hgb A_1 = 95-98% Hgb A_2 =1.5% Hgb F<2%
Methemoglobin	< 1% of total Hemoglobin
Reticulocyte Count	0.5-2.5% of total RBC count
White Blood Cells (WBC)	4,500-11,000/mm³
Polymorphonuclear (PMN) or Granulocytes Leukocytes	Neutrophils 45-75% 2,000-7,000 Eosinophils 0-4% 0-400 Basophils 0-3% 0-200
Mononuclear Leukocytes	Lymphocytes 25-40% 1700-3500 Monocytes 4-6% 200-600

Hematological Disorders

• Assessment, Diagnostic Tests and Monitoring Systems

- Coagulation Studies
  - PT (Prothrombin Time)
  - PTT (Partial Thromboplastin Time)
  - Bleeding Time test
  - Platelet count(thrombocyte)
  - Fibrinogen (Factor I)
  - FSP (Fibrin split products)/FDP (fibrin degradation products)
    - D-dimer
  - TT (Thrombin Time)
  - ACT 9aCTIVATED Clotting Time)

Coagulation Studies

TEST	NORMAL RANGE	PARAMETER MEASURED
Platelet Count	150,000-400,000/mm^3	# of Circulating Platelets, Measures amount not functional ability
Prothrombin Time (PT)	11-15 seconds	Extrinsic & Common coagulation Pathways Evaluation of Warfarin Therapy
International Normalized Ratio (INR)	0.7 -1.8	Standardized method of reporting the PT Evaluation of Warfarin Therapy
Activated Partial Thromboplastin Time (aPTT)	PTT 60-70 seconds	Intrinsic & common coagulation Pathways Evaluation of Heparin Therapy
Anti-factor Xa	0 units/ml DVT tx: LMWH 0.4 1.1 U/ml DVT prophylaxis: < 0.45 U/ml	Low Molecular Weight Heparin Monitoring
Bleeding Time	Depends on system IVY 1-8, Duke 1-3 min	Normal Platelet and Tissue function and Bleeding
Activated Clotting Time (ACT)	70-120 seconds	Intrinsic & common coagulation Pathways Evaluation of Heparin Therapy
Fibrinogen	200-400 mg/dL	Circulating Fibrinogen
Thrombin Time (TT)	14-16 sec	Common Coagulation Pathway and Quality of the functional Fibrinogen Evaluation of tPA
Fibrin Degradation (Split) Products	2-10 mcg/ml	Degree of fibrinolysis
D-Dimer	<2.5 mcg/ml	Specific Fibrin Breakdown Product

Hematological Disorders

• Assessment, Diagnostic Tests and Monitoring Systems

- Immunology Studies
  - All White Blood Cells
    - Neutrophils
    - Eosinophils
    - Basophils
    - Mast cells
    - Monocyte/macrophages
    - B lymphocytes
    - T cells

Immunologic Studies

White Blood Cells (WBC)	4,500 – 10,00 /mm^3
Neutrophils	2.0–7.0×10 ^{9}/l (40–80%)
Eosinophils	0.02–0.5×10 ^{9}/l (1–6%)
Basophils	0.02–0.1×10 ^{9}/l (< 1–2%)
Mast cells	Located in different tissues
Monocyte/Macrophages	0.2–1.0×10 ^{9}/l (2–10%)
B lymphocytes	1.0–3.0×10 ^{9}/l (20–40%)
T Cells CD4+(T4) CD8+ (T8)	600 – 1200 /mm^3 (30-60%) 200 -1000/mm^3 (15 – 40%)

Pathologic Conditions

Anemia

Thrombocytopenia

Disseminated Intravascular Coagulation

Immunocompromise

Other Coagulopathies

Hematological Disorders
• *Pathological Conditions*

- Anemia
  - Low blood count
    - Poor oxygen delivery
  - Etiology
    - Blood loss
      - Trauma, surgery, GI Bleed,
      - Increased bleeding from DIC, anticoagulants, excessive blood draws
    - Under production of RBC
      - Malnutrition, chronic illness, malignancies and cancer therapy
      - Organ dysfunction – liver or renal
    - Early elimination/destruction of RBC
      - Mechanical – CPB, IABP, mechanical valves
      - Immune response hemolytic anemia – sickle cell anemia/disease, G6PD deficiency, thrombotic thrombocytopenia purpura (TTP)

Hematological Disorders
• *Pathological Conditions*

- Anemia
  - Symptoms
    - Tachycardia
    - Weak pulses
    - Orthostatic hypotension
    - ECG changes
    - Increased rate of respiration and work of breathing
    - Pale skin and mucous membranes
    - Decreased urine output
    - Decreased LOC
  - Primary goal
    - Adequate oxygen delivery
  - Treatment
    - Administration of packed red blood cells
    - Supplemental oxygen
    - Minimize activity
    - Optimize cardiac output and pulmonary function

Hematological Disorders
• *Pathological Conditions*

- Sickle Cell Disease (SSD)
  - Hemolytic anemia
  - Hemoglobin S from both parents
  - RBC have "sickle" shape
    - Decreased oxygen carrying capacity
    - Microvascular hypoxia and clotting
    - Vessel occlusion
  - Lab values
    - Low RBC, HCT, Hgb
    - Elevated reticulocyte counts with microcytic anemia
  - Symptoms
    - Severe pain, fever, fatigue, tachycardia, tachypnea, hematuria
  - No cure; only symptom management
    - Hydration
    - Oxygen administration
    - Pain medication administration

Hematological Disorders
• *Pathological Conditions*

- Sickle Cell Crisis Patterns
- Four recognized patterns
  - Bone Crisis
  - Acute Chest Syndrome
  - Abdominal Crisis
  - Joint Crisis

Hematological Disorders
• *Pathological Conditions*

- Hemophilia
  - X chromosome inherited bleeding disorder
  - Females can be carriers; but only males have disease
  - Low levels of Factor VIII or IX
    - Treatment – to prevent bleeding risk
    - If bleeding – limit blood loss
    - Transfuse if necessary
      - RBC's, FFP, Platelets, and Cryoprecipitate
      - May be given sterile factor concentrates of Factor VIII
      - Recombinate human factor VIIa has been approved for use of bleeding with hemophilia
      - DDAVP or antifibrinolytics may also be used

Hematological Disorders
• *Pathological Conditions*

- Thrombocytopenia
  - Fairly common in the critically ill
  - Abnormally low platelet count
  - May not know the cause – idiopathic thrombocytopenia purpura

  - Etiologic causes:
    - Decreased platelet production
    - Decreased platelet survival
    - Excessive consumption of platelets
    - Splenic sequestration of platelets
    - Platelet dilution (massive volume administration)

Hematological Disorders
• *Pathological Conditions*

- Thrombocytopenia
  - Signs and Symptoms
    - Laboratory signs
    - Integumentary signs
    - Renal signs
    - Gastrointestinal signs
    - Neurological signs
    - Miscellaneous signs

Hematological Disorders
• *Pathological Conditions*

- Thrombocytopenia
- Diagnosis
  - Low platelet count w/ prolonged bleeding; PT and PTT normal
    - **ITP - origin unknown; most common**
    - **Heparin Induced Thrombocytopenia (HIT) – immune reaction to Heparin**
      - body produces antibodies which lead to platelet destruction
      - Platelet count experiences an unexplained drop > 50% of baseline
      - Thromboemboli form
    - **Thrombotic Thrombocytopenia Purpura (TTP) –congenital, idiopathic, or secondary**
      - Platelet count is low but microvascular thrombi form as well
      - CNS and Renal most common sites
      - Administration of platelets not recommended
    - **Hemolysis with Elevated Liver enzymes and Low Platelets (HELLP)**
      - Result of pregnancy induced hypertension (PIH) or preeclampsia
      - Severe vasoconstriction causes destruction of RBC's and platelets

Hematological Disorders
• *Pathological Conditions*

- Disseminated Intravascular Coagulation (DIC)
  - Occurs secondary to other major illnesses
    - Obstetrical complications
    - Sepsis
    - Tissue Trauma
    - Liver disease
    - Blood transfusion
    - Cancer
    - Viral hemorrhagic fevers
    - Venomous snake bite
    - Pancreatitis
  - Patient history will reveal a concurrent condition known to precipitate DIC and spontaneous bleeding in the absence of a known coagulopathy
    - Bleeding
    - Clotting

Coagulation Cascade
• *Disseminated Intravascular Coagulation*

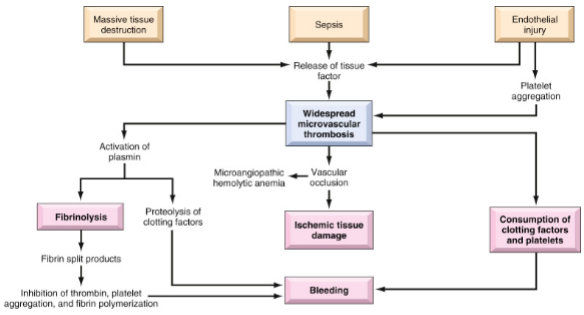


FIG. 37.3

Pathophysiology of Disseminated Intravascular Coagulation.

(From Kumar V, Abbas AK, Aster JC. *Robbins and Cotran Pathologic Basis of Disease* . 9th ed. Philadelphia: Elsevier; 2015.)

Hematological Disorders
• *Pathological Conditions*

- Disseminated Intravascular Coagulation
  - Abnormal coagulation studies confirm the diagnosis of DIC
    - Elevated
      - PT
      - PTT
      - Fibrin split products/ fibrin degradation products
      - D-Dimer
    - Decreased
      - Platelets
      - Fibrinogen
  - Lab tests assess
    - Increased coagulant activity
    - Increased fibrinolytic activity
    - Impaired regulatory function
    - End-organ failure

•DIC is a "consumptive coagulopathy"

Hematological Disorders
• *Pathological Conditions*

Test	Value
Prothrombin time (PT)	>12.5 s
Platelets	<50,000/mm ^{3} , or at least 50% drop from baseline
Activated partial thromboplastin time (aPTT)	>40 s
d -dimer	>250 ng/mL
Fibrin degradation products (FDP)	>40 mg/mL
Fibrinogen	<100 mg/dL

Hematological Disorders
• *Pathological Conditions*

- Disseminated Intravascular Coagulation
  - Signs and symptoms

System	Signs Related to Hemorrhage	Signs Related to Thrombi
Integumentary	Bleeding from gums, venipunctures, and old surgical sites; epistaxis; ecchymoses	Peripheral cyanosis, gangrene
Cardiopulmonary	Hemoptysis	Dysrhythmias, chest pain, acute myocardial infarction, pulmonary embolus, respiratory failure
Renal	Hematuria	Oliguria, acute kidney injury
Gastrointestinal	Abdominal distention, hemorrhage	Diarrhea, constipation, bowel infarct
Neurologic	Subarachnoid hemorrhage	Altered level of consciousness, ischemic stroke

Hematological Disorders
• *Pathological Conditions*

- Disseminated Intravascular Coagulation
- Medical Management
  - PREVENTION
  - Once DIC identified
    - Maintain organ perfusion
      - Support blood pressure and circulating volume
      - Administer inotropes and fluids
      - Overt hemorrhage – replace RBC's
    - Slow down the consumption of coagulation factors
      - Platelets <50, 000 administer platelets
      - Fibrinogen low – administer cryoprecipitate
      - PTT pronlonged – administer fresh frozen plasma (FFP)
    - Use of heparin is controversial
    - Use of antithrombin III has been approved in the US

Hematological Disorders
• *Pathological Conditions*

- Disseminated Intravascular Coagulation
- Nursing Management
  - Driven by the specific cause of DIC
    - Supporting patient's vital functions
    - Initiating bleeding precautions
    - Providing comfort and emotional support
    - Constant surveillance for complications
- Nursing Diagnoses
  - Deficient Fluid Volume active blood loss
  - Decreased cardiac output related to alterations in preload
  - Risk for infection
  - Anxiety related to threat to biologic, psychologic or social integrity
  - Compromised family coping related to a critically ill family member

Hematological Disorders

Questions?

Glycemic Emergencies

Patti Esmail MSN/ED RN CCRN-K
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Hypoglycemia

Pathophysiology

- Decreased serum glucose level to 50mg/dl or below
- Glucose production (feeding or liver gluconeogenesis) lags behind glucose use
- Decreased clearance of insulin or oral hypoglycemic agents
- Drug interactions

Etiology

- Insulin therapy
  - Dose greater than body needs
  - Sudden rotation of sites
  - Interruption of enteral tube feedings
- Oral hypoglycemic therapy – esp sulfonylurea agents
- Insufficient caloric consumption
- Uncompensated strenuous physical exercise
- Potentiation of hypoglycemic medication
- Excessive alcohol intake

Etiology - continued

- Decreased requirements
  - Recovery from physiological stress
  - Weight loss
  - Immediate post partum
  - Decrease steroid dose
- Use of pentamidine
  - Causes pancreatic islet cell necrosis
    - Acute release of insulin
- Other health problems
  - Liver disease
  - Pancreatic islet cell tumor
- Beta adrenergic blockers
  - Inhibit the adrenomedullary symptoms
  - Impairs recovery from hypoglycemia
    - Inhibits glyconeolysis

Signs and symptoms

- Headache
- Fatigue
- Hunger
- Irritability
- Pallor

Diagnostic findings

- Serum glucose levels lower than 50 mg/dl

Goals of Care

- Hypoglycemia and the associated sequelae are corrected

Management

- Anticipate the potential course of events for the patient
  - Potential complication
    - newly diagnosed diabetics
    - When insulin – food – activity balance has not be achieved or disrupted
  - Administer glucose either oral or IV
    - Remeasure glucose 20 – 30 minutes after treatment
    - Readminister glucose if needed
    - Discontinue insulin infusion if present

Diabetes KetoAcidosis (DKA)

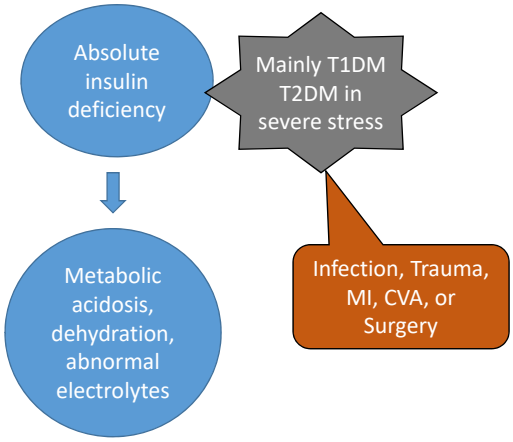
Pathophysiology

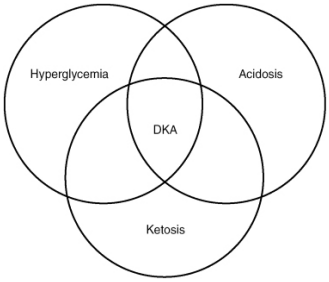
- Most serious metabolic complication of insulin dependant diabetes mellitus
- DKA is a state of insulin deficiency
  - Results in an altered metabolism of carbohydrate, fat, and protein and hyperglycemia
    - Decreased insulin level with gluconeogenesis and increased insulin resistance
      - Exaggerated hepatic glucose production

Pathophysiology - continued

- Ketosis and metabolic acidosis result from increased synthesis of ketones and lactic acidosis
- Glycosuria
  - Fluid and electrolyte imbalance
  - Osmotic diuresis
    - Loss of
      - Sodium
      - Potassium
      - Chloride
- Altered mental status
  - Hyperosmolality
  - Cellular dehydration
  - Acidosis
  - Possible impaired oxygen disassociation

Pathogenesis of DKA





Etiology

- **Diagnosed diabetes**
  - Insufficient exogenous insulin
    - Missed dose or insufficient for needs
  - Infection or trauma
  - Poor compliance
    - Alcohol or drug use
    - Educational gaps
    - Psychosocial distress or disease
    - Adolescence
  - Medication side effects
    - Increase gluconeogenesis – steroids
    - Decreased insulin resistance – thiazide diuretics, phenytoin
- **Undiagnosed diabetes mellitus**
  - Positive family history of diabetes

Signs and Syptoms

- Hypotension
- Tachycardia
- Tachypnea
- Fruity odor to breath (ketosis)
- Kussmaul's respirations
- Nausea, vomiting, abdominal cramping
- Decreased skin turgor
- Dry mucous membranes

Diagnostic findings

- Elevated plasma and urine glucose levels
  - Plasma glucose - > 250 mg/dl
  - Presence of glucose in urine
- Metabolic acidosis
  - Arterial pH - < 7.3
  - Serum HCO_3 - < 18 mEq/dl
- Positive results for serum and urine ketones
- Azotemia
- Anion gap: Na^+ - (Cl^-+HCO_3^-) >10
- ECG – may reflect hypokalemia
  - Potassium may be normal
  - Flate T waves
- Hypocalcemia in 30% of patients
- Hyperosmolality

Goals of Care

- Restore Acid – Base balance
- Normalize and maintain blood glucose level
- Restore fluid balance
- Infection, if present, is resolved

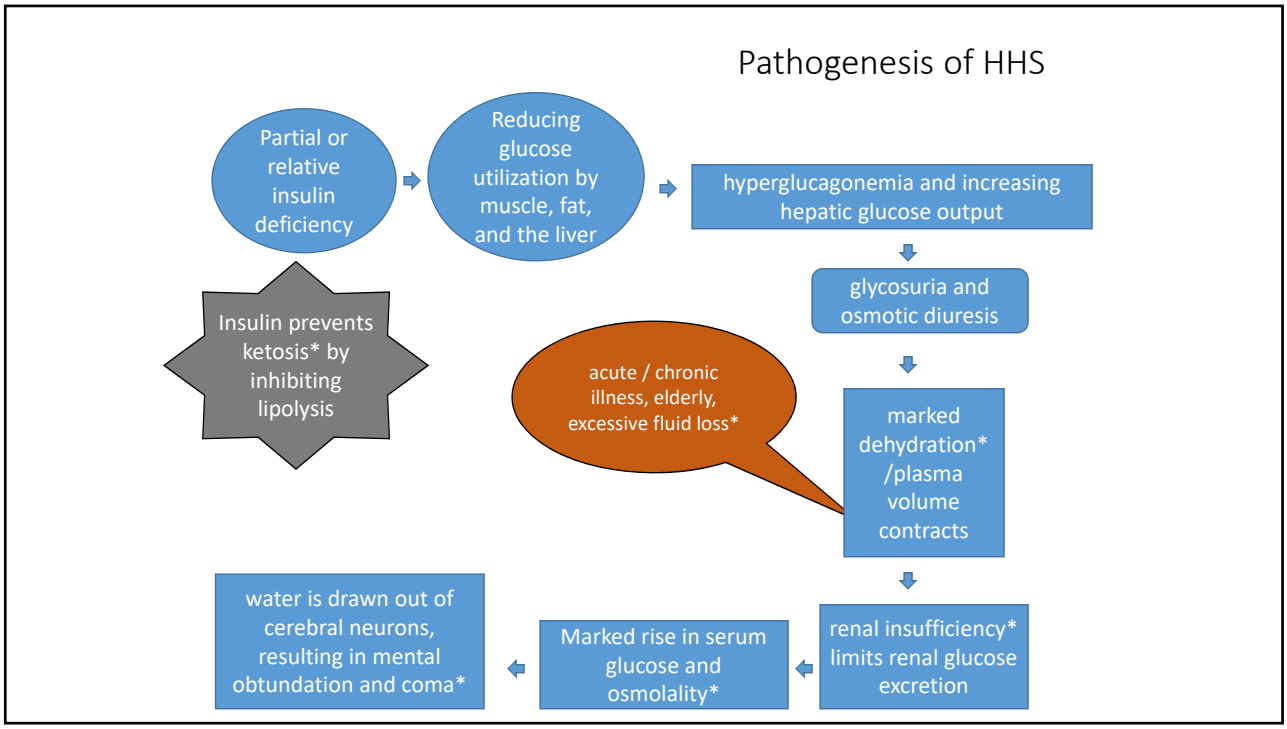
Management

- Anticipate the potential problems
  - Reoccurs easily in diabetic patients if
    - Medication compliance
    - Diet
    - Sick day management
      - Not well understood
  - Reoccurrence most common in teens and patients with new diagnosis

Hyperglycemic Hyperosmolar Syndrome (HHS)

Pathophysiology

- Life-threatening hyperglycemic emergency
  - Relative insulin deficiency
  - Hyperosmolality
    - Hyperglycemia
    - Hypernatremia
  - Fluid shifts intracellular to extracellular because of hyperosmolality
- Severe dehydration
  - Osmotic diuresis
    - Extracellular volume depletion
- Severe electrolyte losses
- Alterations in neurologic status without ketosis
  - Coma from cellular dehydration



Etiology

- Inadequate insulin secretion or action
- Advanced age and dehydration
- Other illnesses that increase glucose production or contributes to dehydration
- Lack of ready access to fluid or inability to recognize or express the need for fluid
- Disruption of medication regimen of insulin or oral hypoglycemic agents
- Use of medications known to elevate glucose levels
- Crisis more common in late middle aged and elderly patients with renal or CV disease

Signs and symptoms

- Lethargy, fatigue, coma
- Polydipsia, polyuria, polyphagia
- Flushed skin and dry mucous membranes
- Tachycardia, hypotension
- Shallow, rapid respirations

Diagnostic Findings

- Severely elevated glucose levels
  - > 600 mg/dl
- No ketosis
- Sodium and potassium levels often severely depleted requiring replacement
- Plasma hyperosmolality
  - > 330 mOsm/kg
- If acidosis is present, usually caused by lactic acid or renal dysfunction

Goals of Care

- Correct dehydration
- Correct hyperglycemia
- Restore peripheral tissue perfusion

Management

- Pharmacology
  - Fluid replacement
  - IV Insulin infusion
- Treatments
  - Hourly serum glucose monitoring
  - Strict management of I&O

Let's Compare DKA vs HHS

Definitions

Diabetic Ketoacidosis (DKA)

Absolute insulin deficiency

- Lipolysis , ketogenesis, acidemia
- Hyperglycemia, glycosuria, osmotic diuresis, volume depletion
- Type 1 Diabetes (Type 2 DM occasionally)
- Mortality <5%

Hyperglycemic Hyperosmolar State (HHS)

- Relative insulin deficiency
- No ketogenesis
- Hyperglycemia, glycosuria, osmotic diuresis, volume depletion
- Type 2 Diabetes
- Older patients
- Mortality 11%

Note: death in these conditions is rarely due to the metabolic complications of hyperglycemia or ketoacidosis but relates to the underlying precipitating illness.

History

DKA: acutely, ~ 24 hrs

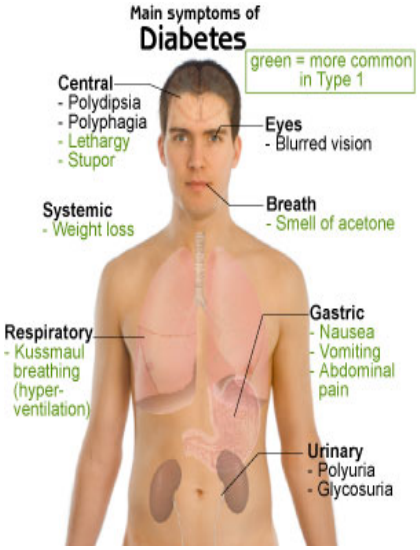
HHS: days to weeks

Polyuria, polydipsia, weight loss

Dehydration, weakness

Nausea/ Vomiting, abdominal pain **(DKA)**

Mental status change and coma **(HHS)**



Physical findings

Poor skin turgor secondary to dehydration

Tachycardia, hypotension, shock

Hyper/ normo/ hypothermic

Abdominal pain * **(DKA)**

Kussmaul respirations, fruity odor (DKA)

Alteration in mental status and **coma (HHS)**

In some patients, focal neurologic signs (hemiparesis, hemianopsia) and seizures may be the dominant clinical picture **(HHS)**

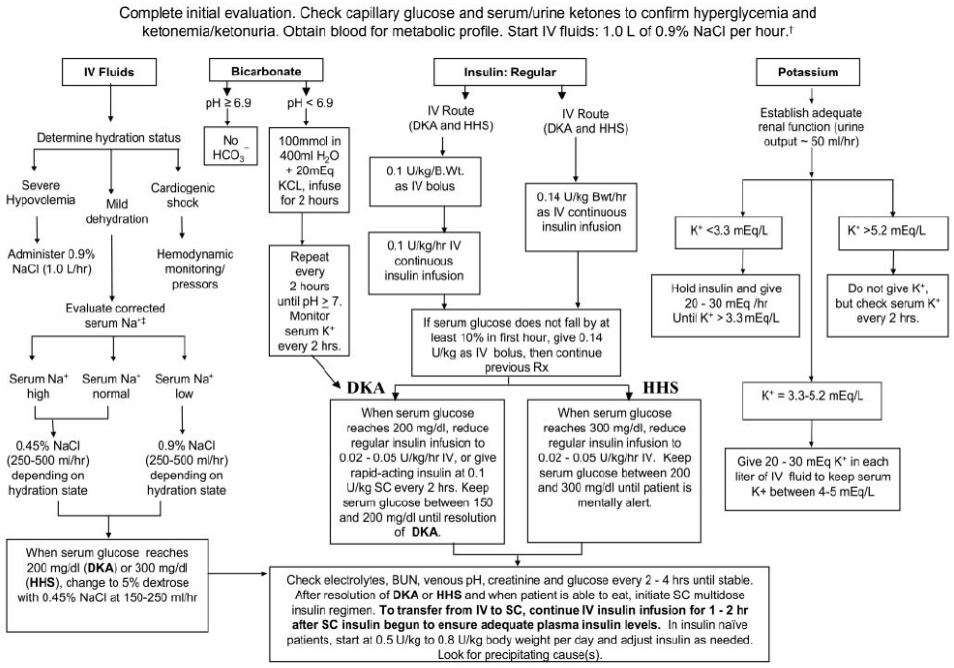
Diagnostic Criteria for DKA and HHS

Diagnostic criteria for diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS)

	DKA			
	Mild	Moderate	Severe	HHS
Plasma glucose (mg/dL)	>250	>250	>250	>600
Arterial pH	7.25-7.30	7.00-7.24	<7.00	>7.30
Serum bicarbonate (mEq/L)	15-18	10 to <15	<10	>18
Urine ketones*	Positive	Positive	Positive	Small
Serum ketones*	Positive	Positive	Positive	Small
Effective serum osmolality (mOsm/kg)●	Variable	Variable	Variable	>320
Anion gapΔ	>10	>12	>12	Variable
Alteration in sensoria or mental obtundation	Alert	Alert/drowsy	Stupor/coma	Stupor/coma

* Nitroprusside reaction method.
● Calculation: 2[measured Na (mEq/L)] + glucose (mg/dL)/18.
Δ Calculation: (Na+) - (Cl- + HCO3-) (mEq/L). See text for details.

DKA/HHS protocol



Fluid Therapy

Initial fluid therapy is directed towards expansion of the intravascular and extravascular volume and restoration of renal perfusion

1st Hour: 1-1.5 L 0.9% NaCl
(caution in CHF patients)

Fluid Therapy

- Selection of fluid therapy based on:
  - State of hydration
  - Serum electrolyte
  - Urinary output
  - Improvement in BP
  - Laboratory values
  - Clinical examination
- Monitor patient condition closely
- Excess fluid replacement may lead to ARDS and cerebral edema.

Insulin Therapy for DKA and HHS

- Initial bolus (Regular insulin): 0.1 unit/kg
- Infusion (Regular insulin): continuous infusion at 0.1 unit/kg/hr
- This dose usually decreases plasma glucose at a rate of 50-75 mg/dl/hr
- If serum glucose does not fall by at least 10% in the first hr, give 0.14 unit/kg as IV bolus, then continue previous treatment

Pancreatitis

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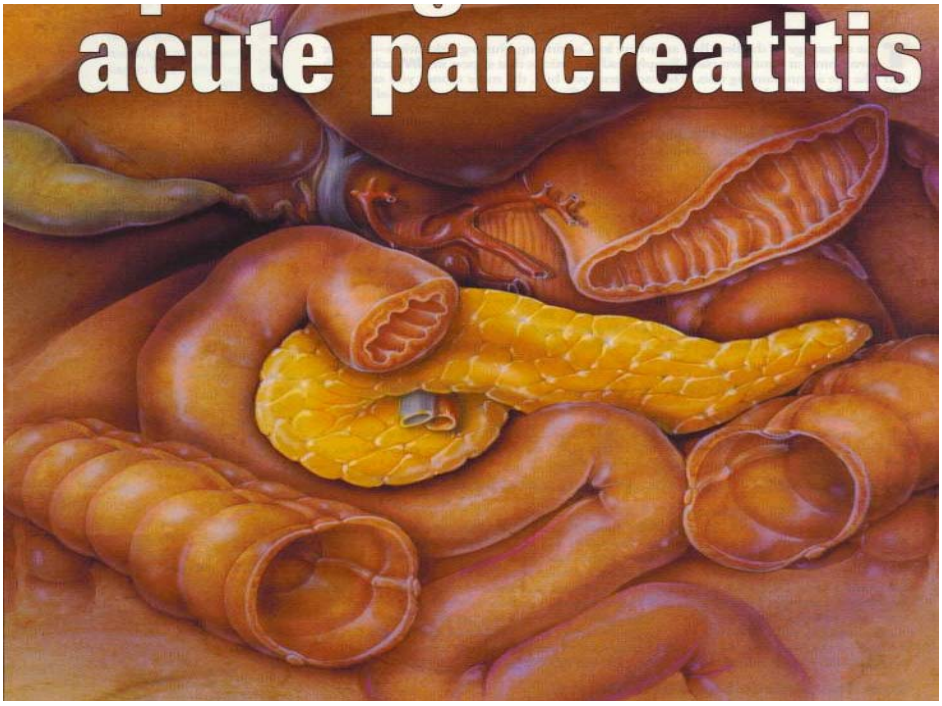
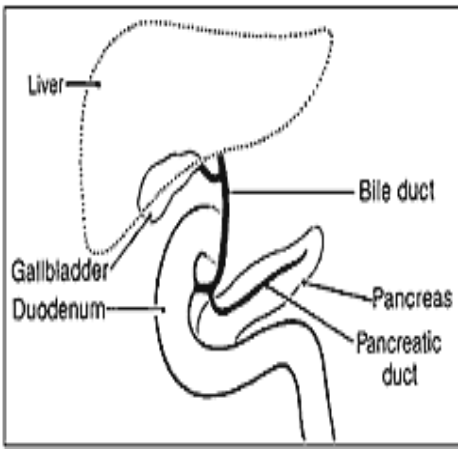


A & P

- Located parallel and beneath stomach
  - Common Duct
- Endocrine functions
  - Insulin by the
    - **Islets of Langerhans**
- Exocrine functions
  - Digestive enzymes
  - Secreted in response to stomach emptying into duodenum

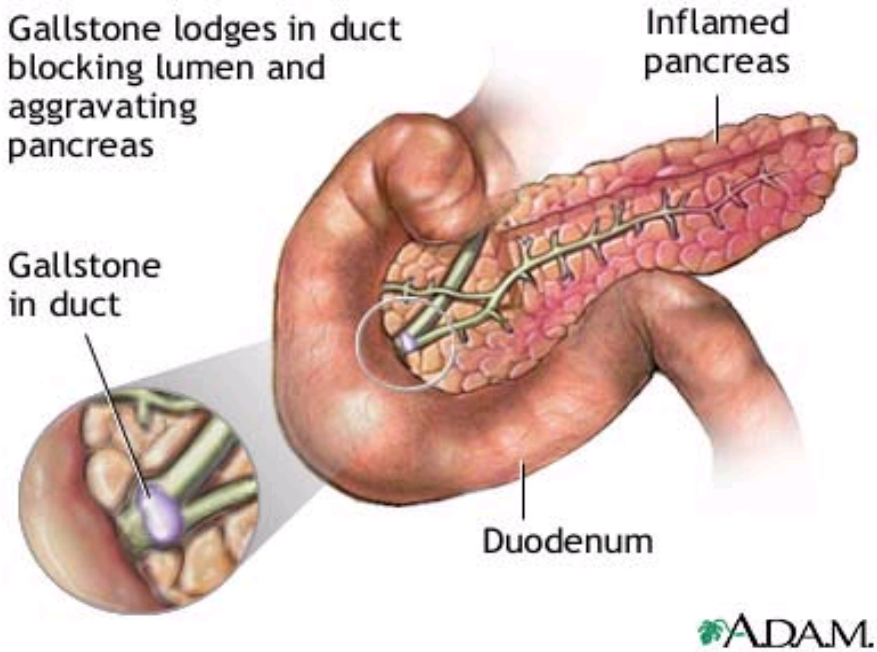


A & P

- Digestive enzymes
  - **Amylase: carbohydrates**
  - **Trypsin: protein**
  - **Lipase: fats**
- Bicarbonate ions
  - **Neutralize acid from stomach**
- Trypsin Inhibitor: prevent activation of enzymes prior to secretion into duodenum

PANCREATITIS

- PATHOPHYSIOLOGY
  - Diffuse inflammation, destruction, autodigestion
  - Normal flow of enzymes is blocked
    - **stagnation**
    - **premature activation**
    - **dyschylia: digestion of tissue and fat in and around the pancreas**



Etiology

- Gallstones or sludge (45%)
- Chronic alcohol ingestion (35%)
- Trauma: Endoscopic Retrograde Cholingiopancreatogram: ERCP, direct trauma, Performation of duodenal ulcer
- Metabolic problems:
  - **Hyperlipidemia**
  - **Hypertriglyceridemia (3rd leading cause)**
  - **Hypercalcemia (parathyroidism)**
  - **Renal failure (azotemia)**

Triglyceride of 1200 mg/dl

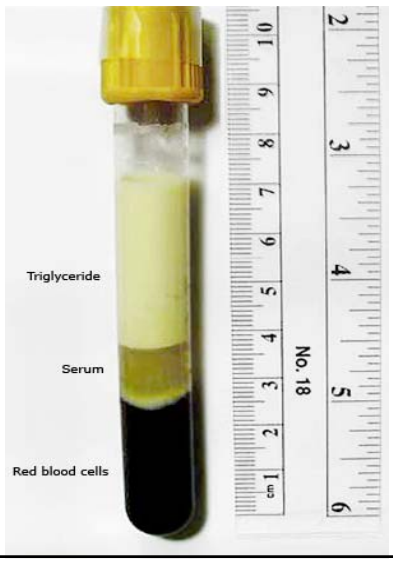
- Triglyceride is broken down into

  Toxic free fatty

  acids

Usually TG > 500

Associated with Acute Pancreatitis



Etiology: Drug-induced Pancreatitis (DIP)

- Top 100 prescriptions in US: 44 implicated in Acute Pancreatitis
  - Acetaminophen
  - Valproic Acid: Depakote
  - Opiates
  - Tetracycline
  - Steroids
  - Furosemide
  - Enalapril
  - Erythromycin
  - Mercaptopurine: Acute lymphatic leukemia
  - Azathioprine: Immuran, antirejection drug (renal)

Etiology

- Infection
  - Viruses
    - Hepatitis B
    - Herpes
    - Mumps
    - Varicella-zoster
  - Bacteria
    - Legionells
    - Leptospira
    - Salmonella
  - Parasitic

Forms of Acute Pancreatitis

- **Mild Edematous Form** (interstitial)
  - 95% of Cases
  - 5% mortality
  - Edema, minimal or no necrosis
  - Massive 3rd spacing of fluids = Hypovolemia

Forms of Acute Pancreatitis

- **Severe, hemorrhagic form: necrotizing pancreatitis**
  - 5% of cases
  - 50 % mortality

Severe, hemorrhagic form: necrotizing pancreatitis

- Extensive peri-pancreatic tissue necrosis of fat in omentum and retroperitoneum
- Hemorrhage: tissue necrosis, erosion of pseudocyst into veins & arteries
- SIRS, sepsis, multi organ dysfunction syndrome

Pancreatitis Pathophysiology

- Pancreatic and Peri-pancreatic Edema:
  - Loss of up to 6 L of fluid
- Auto digestion by pancreatic enzymes
- Severity depends on degree of inflammation and tissue destruction

Pancreatitis Pathophysiology

- 4 Major Physiologic Processes

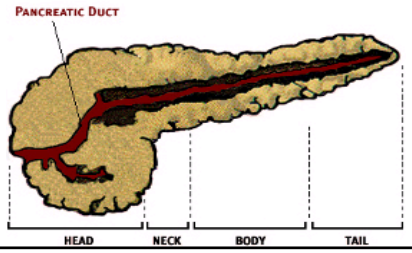
- #1 Lipolytic Process
- #2 Proteolysis
- #3 Necrosis of blood vessels
- #4 Inflammation

Pancreatitis Pathophysiology

- **Lipolytic Process: Lipase**
  - Hallmark of pancreatic necrosis
  - Enzymatic fat necrosis of endocrine and exocrine cells
  - Released fatty acids combine with ionized calcium to form soap-like product
  - Rapidly developing hypocalcemia

Pancreatitis Pathophysiology

- Proteolysis
  - Digestion of protein tissues of pancreas
- Necrosis of blood vessels
  - Results in bleeding
    - **from minor to major**

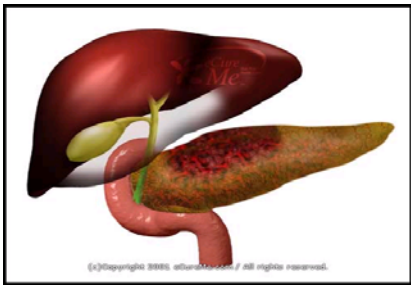


Pancreatitis Pathophysiology

- Release & Activation of Inflammatory Mediators
  - **Complement, kinins, histamine, prostaglandin, clotting factors**
  - **Systemic Inflammatory Response Syndrome (SIRS):**
    - **Vascular permeability, vasodilation, vascular status, DIC**

Necrotizing Pancreatitis
SIRS & MODS

- ARDS
- Acute Renal Failure
- Disseminated Intravascular Coagulation
- Cardiogenic Shock: release of myocardial depressant factor
- Hypovolemic shock
- Acute Liver Failure



Signs & Symptoms

- SEVERE Abdominal Pain
  - May radiate to all abd. quadrants & lumbar area
  - Difficult to achieve adequate pain relief

Signs & Symptoms

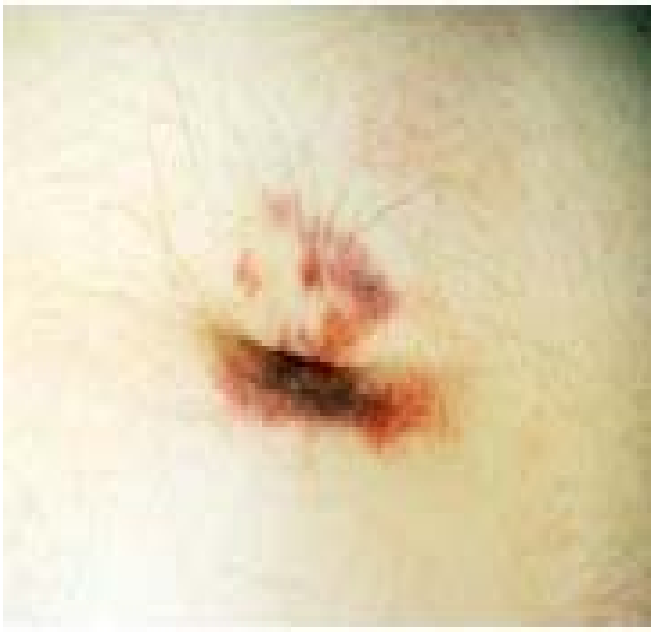
- Nausea & Vomiting
- Peritonitis
  - Rigid Abdomen:
  - Rebound Tenderness

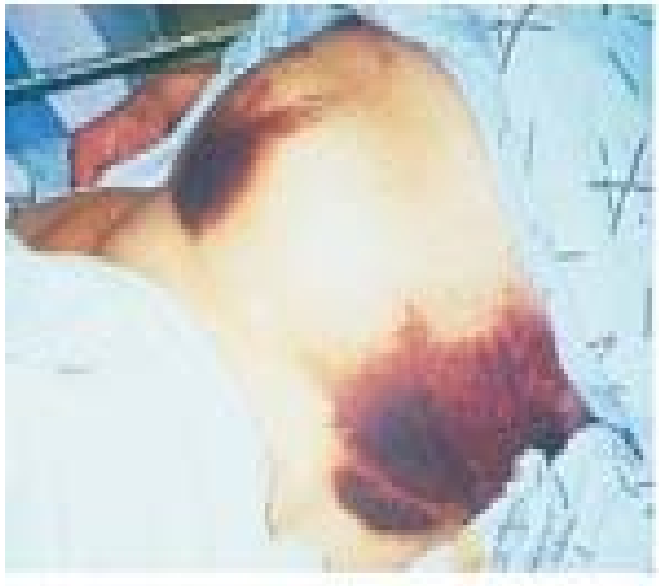
Signs & Symptoms

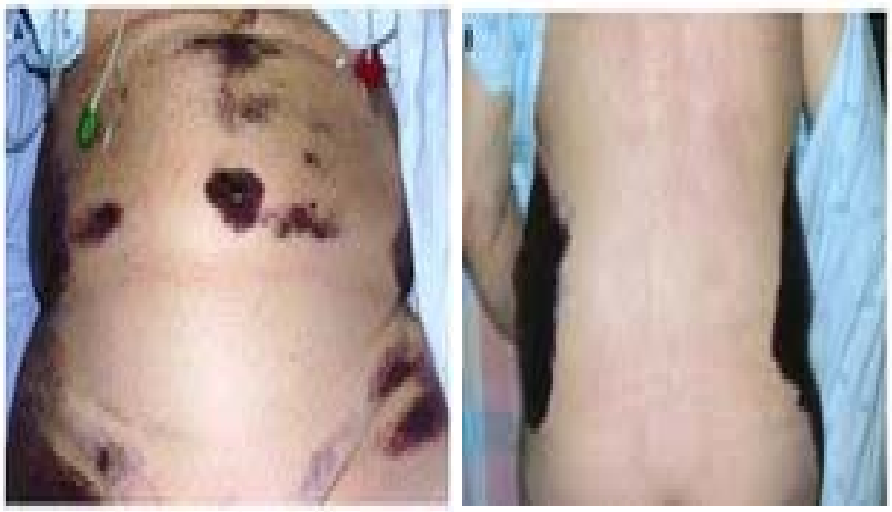
- Lowgrade Fever
- Diaphoresis
- Anorexia
- Diarrhea
- Dehydration
- Jaundice
- Steatorrhea

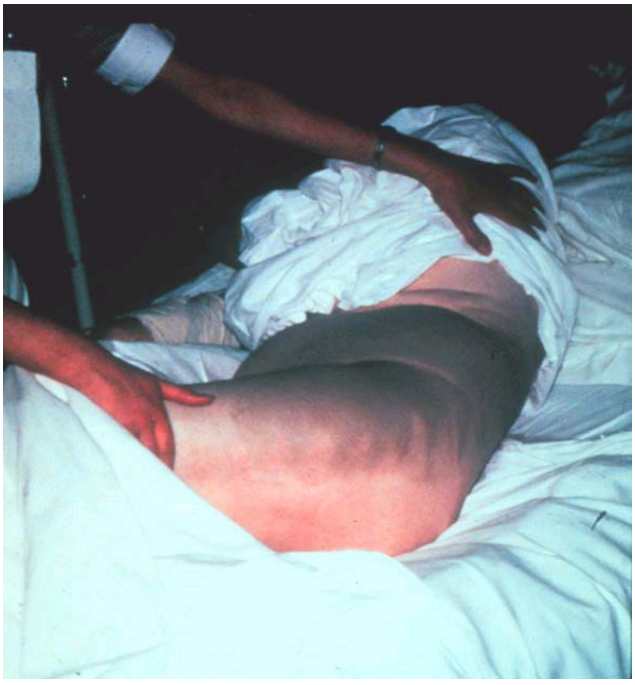
Signs & Symptoms

- Extravasation of Hemorrhage
  - Indicates severe, hemorrhagic pancreatitis
  - Halstead's sign: marbled abdomen
  - Cullen's sign: bluish discoloration of periumbilical area
  - Turner's sign: bluish discoloration of flanks









Diagnostic Studies

LAB	Normal Level
↑ Serum Amylase	25-125
↑ Serum Lipase	10-140
↑ Urine Amylase	< 17
↑ WBC	4.5-11
↓ Serum Ionized Calcium	4.25-5.25

Diagnostic Studies

- Ultra-Sonography: Pseudocyst or Abscess
- CT

Management

- Keep Pt NPO
- NG Tube for N&V
- Manage Pain
  - Fetal Position
  - Demerol for common duct stone
  - Morphine otherwise
- Treat Shock
  - Fluids to replace 3rd spacing
  - 6-8 Liters per day

Management

- Prevent hypocalcemia and hypomagnesemia
- Keep stomach pH close to neutral
- Urgent ERCP to clear biliary tract
  - ↓ morbidity and mortality in acute bilary pancreatitis

Management

- Pseudocyst formation or abscess
  - Pocket of pancreatic enzymes, fluids
  - Account for 70%-80% mortality
  - May be asymptomatic and resolve spontaneously
  - May require needle or endoscopic or surgical resection or drainage

Management

- Plural Fistulas and Pancreatic Ascites
  - Both result from pancreatic fluid entering plural space or abdomen
  - Treatment: ERCP with pancreatic duct stenting
  - Surgical correction
- Chronic Pancreatitis

Ranson's Criteria: Prognosis

- On Admission:
  - Age > 55 years
  - WBC count > 16,000
  - Blood glucose > 200 mg/dl
  - LDH > 350 IU/L
  - AST > 250 IU/L
- 2 or less = admit to Med/surg
- Greater than 3 = Severe Pancreatitis
  - Admit to critical care

Ranson's Criteria: Prognosis

- Helps to assess the progression
- At 48 Hours
  - Fall in Hct > 10%
  - Rise in BUN > 5 mg per dl
  - Serum calcium < 8 mg per dl
  - Arterial PaO2 < 60 mmHg
  - Base deficit > 4 mEq/L
  - Estimated fluid third spacing of >6 liters

Ranson's Criteria: Prognosis

- The presence of 3 or 4 signs on admission : mortality of 15-20%
- The presence of 7 or more: mortality approaches 100%

Pancreatitis: Big Risks

- Fluid Volume Deficit r/l to 3rd Spacing
- Hemorrhage
- SIRS
- Rupture of pseudocyst/abscess

LRCCP Basic Critical Care Course

Hematological Disorders

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Hematologic and Immunologic System Anatomy and Physiology

Structures, Components and Functions
Coagulation Mechanism and Fibrinolysis
Immunologic Basics

Liver Disorders

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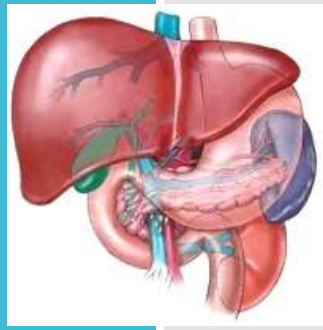
CHI St Vincent Critical Care Educator



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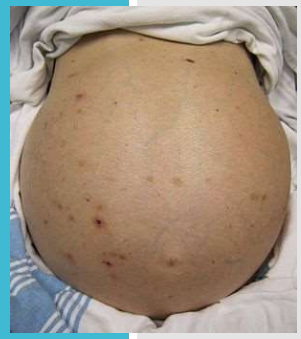
Acute (Fulminant) Liver Failure



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Acute Liver Failure: Pathophysiology

- Development of
  - hepatic encephalopathy within 8 weeks of symptoms
  - 2 weeks of onset of jaundice
- History of normal liver function
  - Normal serum alanine transaminase level
  - Indicates massive hepatocellular necrosis
  - Prolonged coagulation
  - Hypoglycemia can occur
- Except in acetaminophen toxicity
  - Mortality is 80-100% with out a liver transplant

3

Acute Liver Failure: Etiology and Risk Factors

- Acute Hepatitis A & B
- Autoimmune Hepatitis
- Acetaminophen toxicity
- Hepatotoxic drugs & substances
- Mushroom poisoning
- Viral Infections
  - Herpes virus family – esp in the immunocompromised
- Acute Wilson's disease and acute Budd-Chiari syndrome
- Bone marrow transplant
  - Veno-occlusive disease and graft vs host disease
- Reye's syndrome

4

Acute Liver Failure: Signs & Symptoms

- Vague flu-like symptoms, fever
- Jaundice
- Hyperventilation – respiratory alkalosis
- Hepatic encephalopathy
  - Rapid progression to hepatic coma
- Profound coagulopathy and hypoglycemia
- Hepatorenal syndrome
- Sepsis – metabolic acidosis
- Intracrainial hypertension
- Hyperdynamic circulation
- Systolic ejection murmur
- Eventual CV collapse
- Liver initially enlarged
  - Acute inflammatory stage
- Liver atrophies
  - Hepatocellular necrosis progresses

5

Acute Liver Failure: Diagnostic Studies

- Lab values
  - ↑ AST, ALT
  - To lesser degree alkaline phosphatase and GGT
    - If these values normalize but PT, INR and bilirubin rise – near complete hepatocellular necrosis
  - ↑ serum bilirubin, creatinine, BUN
  - Prolonged PT and INR
  - ↓ Factor V &VII
  - ↓ serum glucose, Hgb & Hct
  - ↑ serum lactate, serum ammonia & WBC
  - + results on cultures of body fluids
  - + for hepatitis
  - + urine toxicology screen
  - + stool guaiac test

6

Acute Liver Failure: Radiology Studies

- CXR
  - Bilateral infiltrates
  - Aspiration pneumonia
- CT head
  - Normal until late stages
- Cerebral perfusion scan
  - Decreased or absent flow late in process
  - Performed before liver transplantation to R/O brain death

7

Acute Liver Failure: Pressure Measurements

- Early signs
  - ↑ ICP, MAP and normal CPP
- Late signs
  - ↑ ICP, ↓MAP and ↓ CPP

8

Acute Liver Failure: Goals of Care

- Optimize liver function
- Stabilize for liver transplantation if appropriate
- Monitor and treat complications
  - Sepsis
  - Brainstem herniation
  - Renal failure
  - Respiratory failure
- Normalization of liver function, neurological function, renal function and vital signs

9

Acute Liver Failure: Management of Patient

- Very unstable with long recovery
- Transplant may be necessary
  - Cadaver
  - Living donor
- Skin care
- Pain management
- Nutrition
- Infection control
- Discharge planning
- Complex medication regimen
- Psychosocial issues

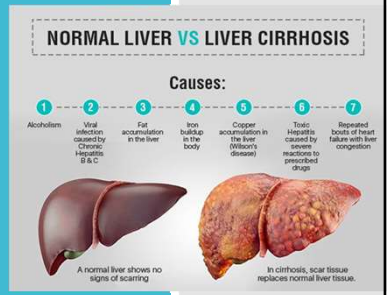
10

Cirrhosis Chronic Liver Failure: Pathophysiology

- Chronic, progresses slowly
- Fibrous connective tissue forms
- Nodular regeneration after necrosis
- Chronic inflammation
- Often irreversible
- 10 years after diagnosis
  - Probability of developing decompensated cirrhosis ~60%

12

Chronic Liver Failure: Cirrhosis



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Cirrhosis Chronic Liver Failure: Etiology and Risk Factors

- Alcoholism – Laennec's cirrhosis
  - Cirrhosis development preceded by reversible alcoholic hepatitis
- Postnecrotic cirrhosis
- Viral hepatitis
- Medication or toxin
- Autoimmune hepatitis
- Autoimmune diseases of biliary stasis
- Inborn errors of liver metabolism
- Nonalcoholic fatty liver disease
- Hepatic vein thrombosis
- Right sided heart failure

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Cirrhosis Chronic Liver Failure: Signs & Symptoms

- Fatigue
- Pruritis
- Muscle wasting, weight loss
- Abdominal distention
- Anemia, hematomas, ecchymosis
- Clay-colored stools
- Fetor hepaticus
- Altered mental status, asterixis
- Visible stigmata of liver disease
- Umbilical hernia, incisional hernia, splenomegaly
- Hyperdynamic circulation
- Possible pleural effusions
- Hepatic bruit

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Cirrhosis Chronic Liver Failure: Diagnostic Findings

- Labs
  - ALT, AST, alkaline phosphatase, GGT levels
  - Bilirubin level
  - PT, INR, platelet count
  - Blood ammonia level
  - Hgb/HCT, BUN, creatinine levels
  - Serum sodium
  - Hepatitis serology
  - Ascitic fluid

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Cirrhosis Chronic Liver Failure: Diagnostic Findings

- Abdominal CT
  - Liver volume decreased
  - Spleen volume increased
  - Ascites or tumor
  - Portal hypertension and collaterals
  - Portal vein patency
- MRI
  - Magnetic resonance venography
  - Magnetic resonance arteriography
  - Magnetic resonance cholangiopancreatography
- Abdominal Ultrasound
  - Size of liver and spleen
  - Portal vein patency
  - Hepatoma
  - Bile duct dilatation
  - Presence of small amounts of ascites

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Cirrhosis Chronic Liver Failure: Diagnostic Findings

- Invasive studies
  - ERCP
  - Upper GI endoscopy
  - Abdominal paracentesis
  - Liver biopsy

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Cirrhosis Chronic Liver Failure: Management of Patient

- 1st goal
  - Optimize liver function
- 2nd goal
  - Stabilize decompensation

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Cirrhosis Chronic Liver Failure: Potential Complications

- Portal Hypertension and splenomegaly
- Ascites
- Spontaneous bacterial peritonitis
- Malnutrition
- Hepatic encephalopathy
  - Four grades of alteration of mentation
  - Hepatic encephalopathy types
- Pulmonary complications
- Hepatorenal syndrome
- Infection or sepsis

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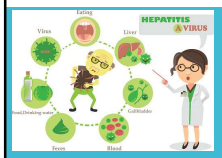
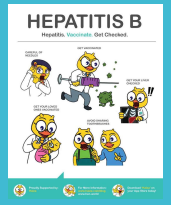
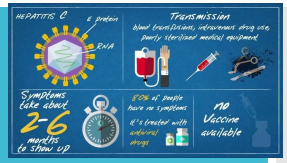
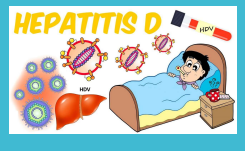
Hepatitis









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Hepatitis: Pathophysiology

- Acute inflammation of the entire liver
- May be a multisystem infection

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Hepatitis: Etiology and Risk Factors

- Viral
- Medication related
- Autoimmune

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Hepatitis A: (Infectious Hepatitis)

- Formerly known as Infectious Hepatitis
  - Fecal-oral transmission
  - Ingestion of raw or undercooked shellfish contaminated by sewage
  - Usually self-limiting

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Hepatitis B:

- Transmission
  - Mother-to-neonate transmission
  - Homosexual and heterosexual transmission
  - Parenteral transmission
    - IV med abuse
    - Blood transfusion or blood products
    - Hemodialysis
    - Exposure to contaminated equipment
    - Body piercings
    - Razors

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Hepatitis C: HCV

- Formerly called non-A, non-B hepatitis
- Transmission
  - Parenteral transmission
    - IV med abuse
    - Nasa cocaine use
    - Blood or blood product transfusion
    - Hemodialysis
    - Body piercings
    - Razors
    - Toothbrush sharing
    - Acupuncture
    - Health care exposure
  - Vertical transmission
    - 3% to 5% in HCV RNA-positive-monoinfected mothers
    - High as 19% in HIV-coinfected mothers

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Hepatitis D:

- Transmission
  - Parenterally
  - Coinfection with hepatitis B
  - May lead to fulminant hepatitis

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Hepatitis E:

- Epidemiology and clinical course similar to hepatitis A
- Prevalent among young adults

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Hepatitis: Autoimmune Hepatitis

- Idiopathic hepatitis
  - Chronic inflammation and plasma cells in liver tissue
  - Autoantibodies
  - Increased serum globulin levels
  - Associated with four antibodies
    - Antinuclear antibody (ANA)
    - Antismooth muscle antibodies (ASMA)
    - Antineutrophil cytoplasmic antibodies (ANCA)
    - Anti-Liver Kidney Microsomal Antibodies (anti-LKM)

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Hepatitis: Medication – related Hepatitis

- Form of medication allergy
  - Immune response is directed toward the liver cells
- Severe reaction produces diffuse necrosis and/or cholestasis

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Hepatitis: Categories

- Acute hepatitis
- Acute fulminant hepatitis
- Asymptomatic carrier state
  - Viral hepatitis
- Infected person unable to clear hepatitis antigen
  - Can transmit to others but suffer no liver damage
- Chronic hepatitis

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Hepatitis: Signs and Symptoms

- Anicteric cases
  - Not jaundiced
- Icteric cases
  - Jaundiced
  - Small proportion of cases
- Prodromal period associated with not feeling well

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Hepatitis: Diagnostic Studies

- Non-Invasive
  - Lab
    - WBC
    - Total bilirubin lever
    - ALT
    - AST
    - Alkaline phosphatase
    - GGT
    - Positive results on hepatitis serology testing
      - Hepatitis A (HAV)
      - Hepatitis B (HBV)
      - Hepatitis C (HCV)
      - Hepatitis D (HDC)
      - Hepatitis E (HEV)
    - Presence of autoimmune markers
      - ANA
      - ASMA
      - ANCA
      - Anti-LKM

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Hepatitis: Management of Patient

- 1st goal of care
  - Supportive
  - Follow treatment of pancreatitis
- 2nd goal of care
  - Optimize liver function and functional status
  - Treat underlying condition
  - Monitor for progression
    - Encephalopathy
    - Coagulopathy
    - Elevation of liver tests

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Hepatitis: Potential Complications

- Cirrhosis
- Ascites
- Encephalopathy
- Increased ICP
- Liver failure
- Infection or sepsis
- Renal failure
- Liver cancer
- Malnutrition

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Questions ?

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Acute Abdomen

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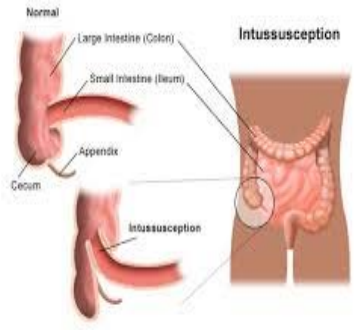
Definition of Acute Abdomen

- Sudden onset of abdominal pain
- This condition is associated with inflammation of the peritoneal cavity and usually requires emergent surgical intervention.
- Ideology of severe pain is less than 24 hours.
- Can be caused by a sudden inflammatory affect, perforation, obstruction, or an infarction of an intra-abdominal organ.
- Two-thirds of all cases are a result of a malignant tumor.
- Rare in adults is ileocolic intussusception (common condition in children).



Signs and Symptoms

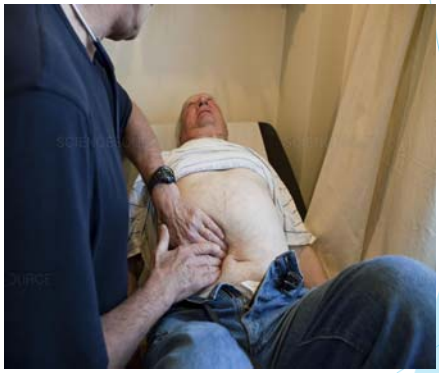
- Sudden or persistent severe abdominal
- Nausea and or vomiting
- Gastric reflux chronic and or severe
- Anorexia sudden weight loss
- Alteration in bowel patterns
- Fecal odor of gastric drainage (vomitus with fecal odor)
- Abdominal distention and bloating
- Bowel sounds either hyperactive or hypoactive
- Patient guarding of the abdomen
- Rebound tenderness
- Fever
- Pallor
- Tachypnea
- Dehydration
- Physical evidence of blunt force trauma (bruising)
- Penetrating trauma (Gun shot wound, knife wound, impalement of sharp objects

Considerations

- Acute abdomen conditions account for approximately 50% of all urgent admissions to the hospital.
- These patients must be assessed for abdominal tenderness, rebounding, and guarding.
- Preparation for surgical intervention by consulting a general surgeon per the attending will delay treatment if surgery is required.
- Patients who have underlying chronic conditions by have severe pain at presentation that is out of proportion of any physical findings.
- It is at this time that a thorough medical history and findings of previous abdominal surgeries can provide essential information for the healthcare team.

Physical Assessment

- A patient with sudden crampy pain and abdominal distention may have an intestinal obstruction caused by adhesions or an incarcerated hernia.
- Assess the patient for jaundice, skin lesions, scarring from prior abdominal surgeries.
- Does the patient have chronic liver disease.



Diagnostic Findings

Non-Invasive Diagnostic Findings:

1. Laboratory Studies – CBC results could show elevated WBC count and elevation in neutrophil and basophil band results. There can be an elevation in the alkaline phosphatase level, serum amylase, lactate acid . These patient may have low hemoglobin and hematocrit levels.
2. Arterial blood gas levels: Metabolic acidosis
3. Cultures of the blood and body fluids (peritoneal)
4. Abdominal x-rays (flat plate of the abdomen): alteration in the position, size, or structure of the abdominal contents. Free air or free fluid in the abdomen.
5. Abdominal ultrasonography: Masses (cysts, abscesses, tumors, foreign bodies, gallstones, infarctions.

Diagnostic findings

- **HIDA Scan**: is used for issues of the liver, gallbladder, and bile ducts.
- This scan is an imaging test used to view the liver, gallbladder, bile ducts, and the small intestine.

- **CAT Scan or MRI:** these diagnostic tests may be with or without contrast depending upon laboratory results and health history. These scans can discover vascular issues, infection, masses, cysts or pseudocysts.
- **EGD-Esophagogastroduodenoscopy:** bleeding from peptic ulcers, esophageal tears, strictures, foreign bodies.
- **Colon/sigmoidoscopy:** lower GI bleed, ulcerations, perforations abscesses and ischemia.
- **Abdominal paracentesis**: blood, bile pus, urine, or fecal material in the abdominal cavity.

Diagnostic Findings

Invasive Findings

- **Cholangiography:** inflammation of the bile duct system
- **ERCP:** biliary or pancreatic stones, obstruction of ducts (this procedure will require anesthesia)
- **Arteriography:** arterial bleeding or vessel infarction – this test my lead to the vessel being coiled for arterial bleeding
- **Peritoneoscopy (Laparoscopy):** bleeding, perforation, rupture, abscess, and ischemia.

Causes of Acute Abdominal Pain

Inflammation

- Appendicitis
- Diverticulitis
- Cholecystitis
- Pelvic inflammatory disease
- Pancreatitis
- Pyelonephritis
- Intra-abdominal abscess

Perforation/rupture

- Peptic ulcer/gastric ulcers
- Rupture of sigmoid diverticula
- Ovarian cyst
- Ectopic pregnancy ruptured fallopian tube
- Aortic aneurysm (rupture, tearing)
- Appendix rupture
- Strangulated hernia

Locations of referred pain and its causes

- Right shoulder

Liver

Gallbladder

Right hemi diaphragm

- Left shoulder

Heart

Tail of the pancreas

Spleen

Left hemi diaphragm

- Scrotum and testicles

Ureter

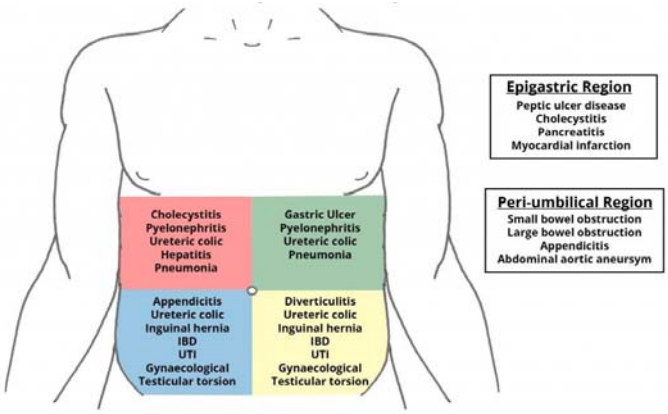
Causes of Acute Abdominal Pain

Obstruction

A condition in which digested material is not able to pass normally through the gastrointestinal system.

- Biliary colic (gallbladder attack) characterized by sudden pain by the right side of the abdomen or mid abdominal area. This is the result of a gallstone temporarily blocking the cystic duct.
- Ureteric colic: urinary stone either in the kidney or the ureter. Is generally associated with severe urinary system pain. This is excruciating pain that will not have any warning and the patient will seek emergent help due to the severity of pain. The pain is caused by the ureter being dilated, stretched and spasm.

Nonsurgical causes of acute abdomen



Endocrine and Metabolic Causes

- 30% of all causes of acute abdomen account for all acute abdomen pain resulting in hospitalizations
- Uremia
- Diabetic crisis, DKA, HHS (hyperosmolar hyperglycemic state)

Hematologic causes

- Sickle cell crisis
- Acute leukemia
- Blood dyscrasia
- Myocardial infection (ACS)

Toxins and Drugs

- Lead poisoning
- Heavy metal poisoning
- Narcotic withdrawal
- Black widow spider poisoning
- Malaria

Surgical causes

Hemorrhage

- Solid organ trauma
- Leaking or ruptured arterial aneurysm
- Ruptured ectopic pregnancy
- Diverticulum bleeding
- Arteriovenous malformation of the GI tract
- Intestinal ulceration
- Mallory-Weiss tear
- Ruptured spleen (trauma, cancer treatment)

Infection

- Appendicitis
- Cholecystitis
- Meckel's diverticulitis (congenital pouch lower part of the small intestine)
- Hepatic abscess
- Diverticular abscess
- Psoas abscess (pus collection)

Surgical causes - Perforations

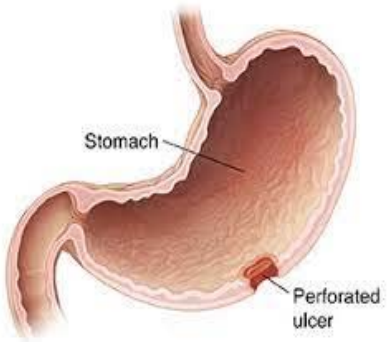
- Gastrointestinal ulcers
- Cancers
- Boerhaave syndrome – spontaneous perforation of the esophagus caused by an sudden increase in intra-esophageal pressure, due to severe straining or vomiting.

Mackler triad: chest pain

vomiting

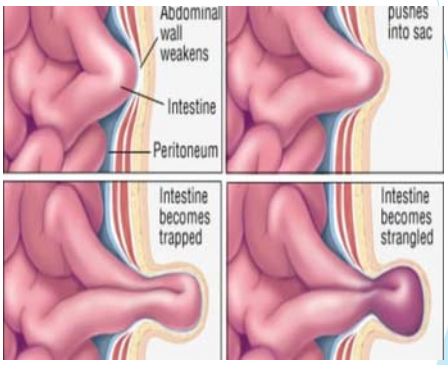
subcutaneous emphysema

- Mallory Weiss syndrome (non transmural esophageal tear



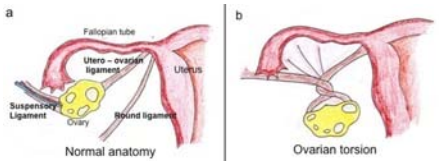
Surgical Causes - Blockages

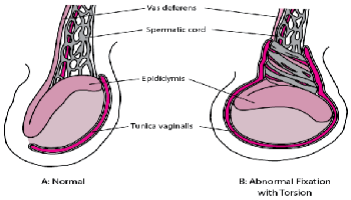
- Adhesion induction small or large bowel obstruction
- Sigmoid volvulus or twisting of the bowel
- Cecal volvulus
- Incarcerated hernias
- Inflammatory bowel disease (Chon's disease)
- Tumors
- intussusception



Surgical causes: Ischemia

- Mesenteric thrombosis or embolism
- Ovarian torsion (twisting of ligaments)
- Ischemic colitis
- Testicular Torsion
- Strangulated hernias





Abdominal Pain

- Abdominal pain is conveniently divided into visceral and parietal components
- **Visceral:** pain tends to be vague and poorly localized to the epigastrium, periumbilical region, or hypogastrium, depending on its origin from the primitive foregut, midgut, or hindgut. Usually the result of distention of a hollow viscus.
- **Parietal:** pain corresponds to the segmental nerve roots innervating the peritoneum and tends to be sharper and better localized. Referred pain is pain perceived at a site distant from the source of stimulus. For example, irritation of the diaphragm may produce pain in the shoulder.
- Determining whether the pain is visceral, parietal, or referred is important and can usually be done with a careful history.

Typical Clinical Features

Condition	History	Examination
Acute appendicitis	Nausea, vomiting, central abdominal pain that later shifts to right iliac fossa	Fever, tenderness, guarding or palpable mass in right iliac fossa, pelvic peritonitis on rectal examination
Perforated peptic ulcer with acute peritonitis	Vomiting at onset associated with severe acute-onset abdominal pain, previous history of dyspepsia, ulcer disease, non-steroidal anti-inflammatory drugs or glucocorticoid therapy	Shallow breathing with minimal abdominal wall movement, abdominal tenderness and guarding, board-like rigidity, abdominal distension and absent bowel sounds
Acute pancreatitis	Anorexia, nausea, vomiting, constant severe epigastric pain, previous alcohol abuse/cholelithiasis	Fever, periumbilical or loin bruising, epigastric tenderness, variable guarding, reduced or absent bowel sounds
Ruptured aortic aneurysm	Sudden onset of severe, tearing back/loin/abdominal pain, hypotension and past history of vascular disease and/or high blood pressure	Shock and hypotension, pulsatile, tender, abdominal mass, asymmetrical femoral pulses

Typical Clinical Features

Condition	History	Examination
Acute mesenteric ischemia	Anorexia, nausea, vomiting, bloody diarrhea, constant abdominal pain, previous history of vascular disease and/or high blood pressure	Atrial fibrillation, heart failure, asymmetrical peripheral pulses, absent bowel sounds, variable tenderness and guarding
Intestinal obstruction	Colicky central abdominal pain, nausea, vomiting and constipation	Surgical scars, hernias, mass, distension, visible peristalsis, increased bowel sounds
Ruptured ectopic pregnancy	Premenopausal female, delayed or missed menstrual period, hypotension, unilateral iliac fossa pain, pleuritic shoulder-tip pain, 'prune juice'-like vaginal discharge	Suprapubic tenderness, periumbilical bruising, pain and tenderness on vaginal examination (cervical excitation), swelling/fullness in fornix on vaginal examination
Pelvic inflammatory disease	Sexually active young female, previous history of sexually transmitted infection, recent gynecological procedure, pregnancy or use of intrauterine contraceptive device, irregular menstruation, dyspareunia, lower or central abdominal pain, backache, pleuritic right upper quadrant pain (Fitz-Hugh-Curtis syndrome)	Fever, vaginal discharge, pelvic peritonitis causing tenderness on rectal examination, right upper quadrant tenderness (perihepatitis), pain/tenderness on vaginal examination (cervical excitation), swelling/fullness in fornix on vaginal examination

How to assess for abdominal pain

- **Physical Assessment:**

Duration

Site and radiation

Severity

Precipitating and relieving factors (food, drugs, alcohol, posture, movement, defecation)

Nature (colicky, constant, sharp or dull, wakes patient at night)

Pattern (intermittent or continuous)

Associated features (vomiting, dyspepsia, altered bowel habit)

Management of patient

- **1st Goal of Care**: Restore hemodynamic equilibrium and fluid balance

Monitor I & O, hemodynamic parameters, administer IV fluids and blood products as appropriate, monitor respiratory system for effects of volume administration.

- **2nd goal of care:** Restore electrolyte balance

Monitor for and correct electrolyte imbalances

- **3rd goal of care:** Early identification of infectious processes

Minimize potential for hospital acquired infection, monitor for changes in clinical condition indicative of escalating infectious process (e.g., physical assessment, vital signs, laboratory results, blood cultures)

- **4th goal of care:** Pain management

Potential Complications

- **Sepsis** (e.g., from obstruction, perforation)
- **Myocardial infarction**
  - **Mechanism:** Increased myocardial workload reduced myocardial oxygen
  - **Management:** Monitor oxygen supply and demand, minimize myocardial workload
- **Dehydration**
  - **Mechanism:** Hemorrhage, third-spacing, nausea, vomiting, diarrhea, intraoperative losses
  - **Management:** Administer fluids as appropriate, monitor intake and output and venous pressures
- **Renal insufficiency**
  - **Mechanism:** Mechanism: Reduction in mean arterial pressure, infection, hypotension, nephrotoxic medications, hepatorenal syndrome
  - **Management:** Administer fluids as appropriate, monitor intake and output and renal function

Patient management

- Positioning: As the patient's condition and comfort dictate
- Skin care: Postoperative wound care and pressure relief are required, as patient is susceptible to skin breakdown from diarrhea, fistula formation, wound drainage, dehydration, hypotension, and malnutrition
- Pain management: epidural, pca, or nurse administered analgesics
- Nutrition: Initiate to meet increased metabolic demands of surgery, fever, wound healing. Etiology and the caloric requirements will determine enteral or parenteral route.

Geriatric population

- With age pain may present atypically.
- Delirium, decrease LOC, immobility
- Most common causes in geriatric population is cholecystitis, bowel obstruction (adhesions), diverticular disease, cancer complications, drug side effects
- Fever and tachycardia may not be present as in younger adults.
- Cardiac medications may mask an elevated heart rate due to infection.

Cancer

- For patients over the age of 70 acute abdominal pain can be associated with cancer than patients under the age of 50.
- 60% of colorectal cancer patients are older than 70 years old
- Symptoms in this population are less distinctive, and due to some patients development of mental decline discomfort and pain can be undiagnosed.
- Due to their co-morbidities minimally invasive surgeries are better served in this population.
- Decisions regarding aggressive treatment versus comfort care depending upon the extent of the cancer.

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